American Petroleum Institute

Index and Abstracts of API Health-Related Research

13th Edition
1959-1994

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API STAFF

Karen Vaucrosson , Health and Environmental Sciences Department - Indexing

Edna Paulson, API Librarian - Indexing

Christine Sexsmith*, Health and Environmental Sciences Department - Indexing

Steven Riley, Information Systems - Database Management

*No longer employed with this organization.

INTRODUCTION

The Health and Environmental Sciences Department (HESD) of the American Petroleum Institute (API) sponsors petroleum health-related research in the areas of industrial hygiene and exposure assessment, toxicology, environmental biology, product safety, and community and occupational health. The reports and publications indexed and abstracted here are a compilation of research investigations/reviews conducted for API from 1959 through the end of 1994. For additional information including articles published in the peer-reviewed literature, on-line searching is recommended.

The Subject Index

The subject index of the 13th edition of **Index and Abstracts of API Health-Related Research** has been simplified to include a comprehensive list of key terms alphabetically arranged by "petroleum product of interest," "test conducted" or by "authors/organizations." The main headings in the subject index appear in **bold**. Subheadings appear in *italics*. Each study in the subject index is identified by its full title followed by a unique number (xx-xxxxx) in parentheses, which also identifies the study's abstract. Numbers are arranged numerically in the Abstracts Section.

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The Evaluation of the Carcinogenicity of Certain Petroleum Hydrocarbon Fractions (Revised: February 1983) with Supplemental Report (Revised: March 1983). (30-32851)

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Four-Week Oral Nephrotoxicity Screening Study in Male F344 Rats [by Borriston Laboratories Inc.]. Final Report. (32-30966)

Hydrocarbon nephropathy in male rats: Identification of the nephrotoxic components of unleaded gasoline (33-31098)

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Kidney Effects of Unleaded Gasoline. Comprehensive and Critical Summary of Observations in Rats and Mice. (30-32845)

Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Twelve-month toxicity evaluation report. Final report. [Vol. 1-3]. (33-31451)

Lifetime Dermal Carcinogenesis/Chronic Toxicity Screening Bioassay of Refinery Streams in C3H/HeJ Mice. Three-Month Toxicity Evaluation Report. [Vol. 1-2]. (32-32653)

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Hydrocarbon Toxicity: Acute, Subchronic and Chronic Effects in Relation to Unleaded Gasoline [(UG)] Exposure of Rodents, with Comments on the Significance to Human Health. (32-30192)

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Twenty-four month dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice (AP-190r). Final Report. (36-33220)

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Mutagenicity of API 83-18, heavy catalytic cracked naphtha (petroleum) (CAS 64741-54-4), in a mouse lymphoma mutation assay. Final report. (33-32804)

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A 28-Day Dermal Toxicity Study in the Rabbit. API 83-18, Heavy, Catalytically Cracked Naphtha (CAS 64741-54-4). Final Report. (32-32748)

Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 83-18: Heavy catalytic cracked naphtha (CAS 64741-54-4). (34-32776)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-18, heavy catalytically cracked naphtha (CAS 64741-54-4). Final report. (33-30593)

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Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of [11] refinery streams in C3H/HeJ mice. Twelve-month toxicity evaluation. (34-32865)

Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Three-month toxicity evaluation report. Final report. (33-32010)

Twenty-four month dermal carcinogenesis/ chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice (AP-190r). Final Report. (36-33220)

HEAVY CATALYTIC REFORMED NAPHTHA

Carcinogenicity Study

Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of [11] refinery streams in C3H/HeJ mice. Twelve-month toxicity evaluation. (34-32865)

Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Three-month toxicity evaluation report. Final report. (33-32010)

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Activity of API 83-06 (heavy catalytic reformed naphtha) in the acute in vivo cytogenetics assay in male and female rats. Final report. (33-30494)

L5178Y TK+/- Mouse Lymphoma Mutagenesis Assay. API 83-06, heavy catalytically reformed naphtha (CAS 64741-68-0). Final report [by Microbiological Associates Inc.]. (33-31641)

Mutagenicity Evaluation in the Mouse Lymphoma Forward Mutation Assay, API #83-06, Heavy Catalytically Reformed Naphtha. Final Report. (32-32460)

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A 28-Day Dermal Toxicity Study in the Rabbit. API [Sample] 83-06, Heavy Catalytically Reformed Naphtha (CAS 64741-68-0). Final Report. (32-32752)

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Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of [11] refinery streams in C3H/HeJ mice. Twelve-month toxicity evaluation. (34-32865)

Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Three-month toxicity evaluation report. Final report. (33-32010)

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Twenty-four month dermal carcinogenesis/ chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice (AP-190r). Final Report. (36-33220)

HEAVY THERMAL CRACKED NAPHTHA

Carcinogenicity Study

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Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Three-month toxicity evaluation report. Final report. (33-32010)

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28-day dermal toxicity in the rabbit. API 84-02, heavy thermally cracked naphtha (CAS 64741-83-9). Final report [by Tegeris Laboratories Inc.]. (33-31696)

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Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of [11] refinery streams in C3H/HeJ mice. Twelve-month toxicity evaluation. (34-32865)

Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Three-month toxicity evaluation report. Final report. (33-32010)

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A Brief Review of [Published Data on] the Toxicity and Carcinogenicity of Select Chlorinated Hydrocarbons. (32-30417)

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n-HEXANE

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Behavioral Evaluation of Petroleum Hydrocarbons. (30-32836)

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Mutagenicity Study

In-Vivo and In-Vitro Mutagenicity Studies. n-Hexane (Hexane UV). Final Report. (28-31628)

[Final Report on the] Mutagenicity Evaluation of n-Hexane in the Mouse Dominant Lethal Assay. (27-32179)

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A Comparison of the Rate of Development of Hexane Neuropathy in Weanling and Young Adult Rats. (30-32007)

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Experimental Studies of Hexacarbon Neurotoxicity. Final Report. (29-32291)

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Neuropathic Potential of n-Hexane in the Presence of Other Hexane Isomers. Final Report: [Phase 1 and Phase 2]. (30-30226)

Quantitative Analysis of Pathological Changes in the Cervical Spinal Cord of Control and n-Hexane-Treated Rats. Final Report. (30-30223)

Six-Month Continuous Inhalation Exposures of Rats to Hexane Mixtures...Phase II. (30-32846)

[Bio/Dynamics Inc. Project No. 77-1921] /A 26-Week Inhalation Toxicity Study of n-Hexane in the Rat. (28-30077)

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Four-Week Oral Nephrotoxicity Screening Study in Male F344 Rats [by Borriston Laboratories Inc.]. Final Report. (32-30966)

Six Month Continuous Inhalation Exposures of Rats to Hexane Mixtures. Phase I. (30-32858)

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The Effects of n-Hexane in Man and Animals. (30-31603)

Neurotoxicity Study

A Tale of Two Solvents: The Neurology of n-Hexane and Toluene. (30-32079)

Neurophysiological and Psychological Disorders and Occupational Exposure to Organic Solvents. (31-32980)

Neurotoxicity of organic solvents with special reference to the neurobehavioral effects: A [world] literature review (39-30623)

The Effects of n-Hexane in Man and Animals. (30-31603)

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A 13 week inhalation study of potential effects of commercial hexane on behavior and neuromorphology in rats. (37-31154)

An acute operant behavior study of inhaled commercial hexane in the Albino rat. (37-31153)

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An inhalation oncogenicity study of commercial hexane in rats and mice--2. Mice. Final report. (41-33232)

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A single-generation inhalation reproduction/fertility study on a commercial hexane. (33-32864)

Developmental toxicity study of commercial hexane vapor in CD(TM)-1 mice. (36-33318)

Developmental toxicity study of commercial hexane vapor in CD(TM) (Sprague-Dawley) rats. (36-33319)

Final Report, April 17, 1991. Commercial hexane test rule, 40 CFR 799.2155. Two-generation reproduction study of inhaled commercial hexane in CD(TM) (Sprague-Dawley) rats. (38-31216)

Final Report, April 17, 1991. Commercial hexane test rule. 40 CFR 799.2155. Two-Generation reproduction study of inhaled commercial hexane in CD(TM) (Sprague-Dawley) rats. (38-31217)

Metabolism/Pharmacokinetics

Disposition and pharmacokinetics of commercial hexane following IV bolus, dermal absorption or nose-only inhalation...Final report. (39-33107)

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CHO [(Chinese hamster ovary)]/HGPRT mutation assay of commercial hexane, Final Report. (37-31487)

Chromosome aberrations in Chinese hamster ovary (CHO) cells exposed to commercial hexane. (37-31152)

Subchronic in-vivo cytogenetics assay in rats using nose-only inhalation exposure to commercial hexane. Final report. (37-31830)

Neurotoxicity study

A 13 week inhalation study of potential effects of commercial hexane on behavior and neuromorphology in rats. (37-31154)

Reproductive effects

A single-generation inhalation reproduction/ fertility study on a commercial hexane. (33-32864)

Final Report, April 17, 1991. Commercial hexane test rule, 40 CFR 799.2155. Two-generation reproduction study of inhaled commercial hexane in CD(TM) (Sprague-Dawley) rats. (38-31216)

Final Report, April 17, 1991. Commercial hexane test rule, 40 CFR 799.2155. Two-Generation reproduction study of inhaled commercial hexane in CD(TM) (Sprague-Dawley) rats. (38-31217)

Toxicity study

A thirteen week inhalation toxicity study of commercial hexane in the rat and mouse. (37-31151)

Special pathology report...A thirteen-week inhalation toxicity study of commercial hexane in the rat and mouse. (37-31148)

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Neurotoxicity Study

Experimental Studies of Hexacarbon Neurotoxicity. Final Report. (29-32291)

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Neurotoxicity Study

A Comparison of the Rate of Development of Hexane Neuropathy in Weanling and Young Adult Rats. (30-32007)

Neuropathic Potential of n-Hexane in the Presence of Other Hexane Isomers. Final Report: [Phase 1 and Phase 2]. (30-30226)

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"Normal" Carboxyhemoglobin [COHb] Levels of Blood Donors in the U.S. (26-60048)

A survey and analysis of liquid gasoline released to the environment during vehicle refueling at service stations. (40-30807)

Acute exposure to methanol in fuels: A prediction of ingestion incidence and toxicity. (36-31914)

Acute exposure to methanol in fuels: A prediction of ingestion incidence and toxicity. (36-31914)

An exposure assessment for marketing and marine distribution workers in the petroleum industry with potential exposure to gasoline (41-31765)

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Carboxyhemoglobin Trend in Chicago Blood Donors, 1970-1974. 2430057)

Carboxyhemoglobin Trend in St. Louis Blood Donors, 1971-1975. CRC-APRAC Project No. CAPM-8-68. (27-30705)

Development and Persistence of Adaptation to Ozone Exposure in Ozone-Sensitive Southern California Residents. (28-33073)

Estimation of incremental benzene exposure associated with seven bulk gasoline storage facilities in North Carolina. (40-32761)

Exposure data on C_7 and C_8 aromatics during handling and production of motor gasolines. (33-31598)

Gasoline vapor exposure assessment at service stations. (40-31953)

Impact on Human Health of Petroleum in the Marine Environment. (32-32651)

Indoor vs. outdoor ambient benzene concentrations: Results of the EPA Total Exposure Assessment [Methodology] (TEAM) study. (35-31189)

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KEROSINE

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Sister chromatid exchange assay in Chinese hamster ovary (CHO) cells with API 83-07: Light catalytic cracked distillate. (35-32432)

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A (28--Day Dermal Toxicity Study in the Rabbit. API [Sample] 83-08, Light Catalytically Cracked Distillate (CAS 64741-59-9). (32-32753)

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Sister chromatid exchange assay in Chinese hamster ovary (CHO) cells with API 81-03 [(light catalytically cracked naphtha)]. Final report. (36-30045)

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28-Day dermal toxicity study in the rabbit [by Tegeris Laboratories Inc.]. API 81-04, light catalytically cracked naphtha (CAS 64741-55-5). Final report. (33-30747)

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28-Day dermal toxicity study in the rabbit [by Tegeris Laboratories Inc.]. API 83-04, light catalytically reformed naphtha (CAS 64741-63-5). Final report. (33-30597)

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Four-Week Oral Nephrotoxicity Screening Study in Male F344 Rats [by Borriston Laboratories Inc.]. Final Report. (32-30966)

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Toxicity Study

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Toxicity Study

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2-METHYL-2-PENTENE

Toxicity Study

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Carcinogenicity Study

Lifetime Dermal Carcinogenesis/Chronic Toxicity Screening Bioassay of Refinery Streams in C3H/HeJ Mice. Three-Month Toxicity Evaluation Report. [Vol. 1-2]. (32-32653)

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Toxicity Study

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Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Twelve-month toxicity evaluation report. Final report. [Vol. 1-3]. (33-31451)

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Carcinogenicity Study

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Clinical toxicology of the acute ingestion of methanol/hydrocarbon blends. A state-of-the-art review. (39-31093)

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The Effects of Oil on Estuarine Animals: Toxicity, Uptake and Depuration, Respiration. (26-60030)

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Uptake and Depuration of Specific Hydrocarbons from Oil by the Bi-valves Rangia cuneata and Crassostrea Virginica. (26-60072)

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Toxicity Study

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NO. 6 FUEL OIL, SPEC. GRAV. 0.92, SULFUR 0.2%

Toxicity Study

[Project No. 1443] Acute Toxicity Tests...API 78-8 No. 6 Heavy Fuel Oil (23.1 °API/ 0.2% S). (27-32816)

NO. 6 FUEL OIL, SPEC. GRAV. 0.95, SULFUR 0.8%

Toxicity Study

[Project No. 1443] Acute Toxicity Tests of API 78-7 No. 6 Heavy Fuel Oil (17.1 °API/ 0.8% S) (27-32774)

NO. 6 FUEL OIL, SPEC. GRAV. 0.99, SULFUR 2.7%

Toxicity Study

[Project No. 1443] Acute Toxicity Tests. API 78-6 No. 6 Heavy Fuel Oil (11.7 °API/ 2.7% S). (27-32814)

NO. 6 FUEL OIL, SPEC. GRAV. 1.04, SULFUR 1.2%

Toxicity Study

[Project No. 1443] Acute Toxicity Tests of API 79-2 No. 6 Heavy Fuel Oil (5.2 °API/ 1.2% S) (27-32813)

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Occupational Health

Guidelines on Noise (26-60119)

n-NONANE

Metabolism/Pharmacokinetics

Studies on the absorption of inhaled hydrocarbon vapors. Final report. (34-33036)

Toxicity Study

Petroleum Hydrocarbon Toxicity Studies--17. Animal Response to n-Nonane Vapor (27-31352)

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A Health Survey of Petroleum Asphalt Workers. (26-60093)
A case-control study of kidney cancer among petroleum refinery workers. Final report. (37-31336)
A mortality study of marketing and marine distribution workers with potential exposure to gasoline in the petroleum industry. (39-33438)
A nested case-control study of kidney cancer, leukemia and multiple myeloma in a cohort of land-based terminal workers exposed to gasoline in the petroleum industry. (41-32613)
An update of a mortality study of workers in petroleum refineries. (33-32726)
Aseptic Bone Necrosis Occurring in Persons Who Work under Pressure. (26-60042)
Assessment of the Healthy Worker Effect [(HWE)]. Final Report. (31-30449)
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Experiments in Human Work Capabilities Under Pressure Now Being Conducted at the Royal Naval Physiological Laboratory. (26-60033)
Experiments in Human Work Capabilities under Pressure, Now Being Conducted at the Institute for Environmental Medicine, University of Pennsylvania. (26-60117)
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Guidelines on Noise. (26-60119)
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Health Act of 1970 Upon the Operational Costs of Diving Services. (26-60039)
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Job code classification system--2. Production operations and marketing/transportation operations (33-30051)
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LA-8459-MS/Paraho Oil Shale Workers Occupational Health Study. (27-33273)
Medical Implications of the Occupational Safety and Health Act in the Diving Industry (26-60038)
Occupational Safety Problems in the Diving Industry. (26-60040)
Occupational Safety and Health Problems of the Diving Industry in Offshore Petroleum Production. (26-60031)
Overview of the Seminar's Objectives. [Seminar on Deep Sea Diving and the Petroleum Industry]. (26-60032)
Review of the Status of Biological Aspects of Coal Conversion Technology, 1977-Present. Final Report. (28-31863)
Statistical approaches for assessing exposures to chemicals. (33-31163)
Surveillance of Reproductive Health in the U.S.: A Survey of Activity Within and Outside Industry. (32-30187)
The Present and Projected Capabilities of the Diving Industry in the Support of Offshore Petroleum Production. (26-60035)
The Unknowns in Decompression Table Calculation and Their Implications in Occupational Safety and Health. (26-60041)
[Seven] Epidemiologic Studies of the Role of Gasoline (Hydrocarbon) Exposure in Kidney Cancer Risk (32-30411)

2,2,4,4,5,5,7,7-OCTAMETHYLOCTANE

Toxicity Study

Final Report. A four-week oral nephrotoxicity screening study in male F344 rats. (33-31097)

OCTANE

Mutagenicity Study

The L5178Y TK +/- mouse lymphoma mutagenesis assay with octane. Final report. (35-31365)

Metabolism/Pharmacokinetics

Studies on the absorption, tissue equilibria and excretion routes of inhaled hydrocarbon vapors and their metabolites (36-31430)

Toxicity Study

Final Report. A four-week oral nephrotoxicity screening study in male F344 rats (33-31097)

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The Effects of n-Octane Exposure on Schedule-Controlled Responding in Mice. (30-32080)

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n-OCTANE, DEUTERATED

Metabolism/Pharmacokinetics

Studies on the absorption of inhaled hydrocarbon vapors. Final report. (34-33036)

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A 14 day palatability and stability study in rats with benzene administered in drinking water. (37-32485)
Detectability and Irritability of Hydrocarbons [Stoddard Solvent and 70 Solvent] in Human Subjects. Final Report to American Petroleum Institute. Contract U-15-14-PS-5. (26-60025)
Human Sensory Response to Selected Petroleum Hydrocarbons. (30-32081)
Odor and taste threshold studies performed with tertiary-amyl methyl ether (TAME). (40-06424)
Odor threshold studies performed with gasoline and gasoline combined with MTBE, ETBE, and TAME. (41-01257)
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OIL MIST

Human Exposure Assessment

Oils Mist: [An] Evaluation of [Workplace] Sampling Procedures. (26-60118)

OZONE

Human Exposure Assessment

Development and Persistence of Adaptation to Ozone Exposure in Ozone-Sensitive Southern California Residents. (28-33073)
Numerical Modeling of Ozone Population Exposure: Application to a Comparison of Alternative Ozone Standards. Final Report. (32-32309)

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Duration of [Human] Pulmonary Hyperresponsiveness to Acute Ozone Exposure. Final Report. (32-31232)
Effects of Oxides of Nitrogen, Carbon Monoxide and Photochemical Oxidants on the [Electrocardiogram] during Exercise and on Cardiopulmonary Function. Final Report (APRAC Project CAPM-21-74). (26-60045)
Short-Term Responses of Healthy and Asthmatic Men to Ozone. Final Report, Phase 1: A Dose-Response Study of Healthy, Heavily Exercising Men Exposed to Ozone Concentrations near the Primary [National Ambient Air Quality] Standard [(NAAQS)]. (32-32755)

OZONE (REVIEW)

Human Exposure Assessment

Photochemical Smog. (26-60108)

Toxicity Study

A Unified Approach to the Use of Human Clinical and Animal Toxicologic Data; Application to the Establishment of Ambient Air Quality Criteria. Volume IA: Effect of Ozone on Airway Resistance (Raw) and Forced Expiratory Volume ($FEV_{1.0}$) in Humans. Final Report. (32-32503)
Oxidants: Air Quality Criteria Based on Health Effects. (26-60060)

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Polynuclear aromatic analyses and modified Ames tests on base lube [lubricating] stocks. (36-33219)

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Mutagenicity Evaluation of Petroleum Hydrocarbons. (30-31997)
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Toxicity Study

The Acute Toxicology of Selected Petroleum Hydrocarbons. (30-31530)
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PARAFFINIC BASE STOCK (VISCOSITY (SUS/100 °F) 331)

Analytical Chemistry/Methodology

Polynuclear aromatic analyses and modified Ames tests on base lube [lubricating] stocks. (36-33219)

Carcinogenicity Study

The Carcinogenicity of New and Used Lubricants. (30-32847)

Mutagenicity Study

In-Vivo and In-Vitro Mutagenicity Studies. Paraffinic Oil 79-3, 350 SUS/100 °F. Final Report. (28-31865)
Mutagenicity Evaluation of Petroleum Hydrocarbons. (30-31997)
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Toxicity Study

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PARAFFINIC BASE STOCK (VISCOSITY (SUS/100 °F) 485)

Analytical Chemistry/Methodology

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Carcinogenicity Study

The Carcinogenicity of New and Used Lubricants. (30-32847)

Mutagenicity Study

In-Vivo and In-Vitro Mutagenicity Studies. Paraffinic Oil 79-4, 550 SUS/100 °F. Final Report. (28-31866)
Mutagenicity Evaluation of Petroleum Hydrocarbons. (30-31997)
Polynuclear aromatic analyses and modified Ames tests on base lube [lubricating] stocks. (36-33219)

Toxicity Study

The Acute Toxicology of Selected Petroleum Hydrocarbons. (30-31530)
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PARAFFINIC BASE STOCK (VISCOSITY (SUS/100 °F) 65)

Analytical Chemistry/Methodology

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Carcinogenicity Study

The Carcinogenicity of New and Used Lubricants. (30-32847)

Mutagenicity Study

In-Vivo and In-Vitro Mutagenicity Studies. Paraffinic Oil 78-9, 70 SUS/100 °F. Final Report. (28-31864)
Mutagenicity Evaluation of Petroleum Hydrocarbons. (30-31997)
Polynuclear aromatic analyses and modified Ames tests on base lube [lubricating] stocks. (36-33219)

Toxicity Study

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PARAFFINIC BASE STOCK (VISCOSITY (SUS/100 °F) 990)

Analytical Chemistry/Methodology

Polynuclear aromatic analyses and modified Ames tests on base lube [lubricating] stocks. (36-33219)

Carcinogenicity Study

The Carcinogenicity of New and Used Lubricants. (30-32847)

Mutagenicity Study

In-Vivo and In-Vitro Mutagenicity Studies. Paraffinic Oil 79-5, 800 SUS/100 °F. Final Report. (28-31867)
Mutagenicity Evaluation of Petroleum Hydrocarbons. (30-31997)
Polynuclear aromatic analyses and modified Ames tests on base lube [lubricating] stocks. (36-33219)

Toxicity Study

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PARTICULATES (REVIEW)

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Toxicity Study

A 90-day feeding study in the rat with six different white mineral oils (N15 (H), N70 (H), N70 (A), P15 (H), N10 (A), and P100 (H), three different mineral waxes (a low-melting-point wax, a high-melting-point wax, and a high-sulphur wax) and coconut oil. July 1992. (39-32387)

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Neurotoxicity Study

Neurobehavioral Toxicology of Petroleum- and Shale-Derived Jet Propulsion Fuel No. 5 (JP5). (30-31533)

Toxicity Study

Comparison of the Subchronic Inhalation Toxicity of Petroleum and Oil Shale JP-5 Jet Fuels (30-31534)

PETROLEUM-DERIVED MIDDLE DISTILLATE

Toxicity Study

Renal Cortical Degeneration [of Mice] Associated with Chronic Dermal Application of Petroleum- and Shale Oil-Derived Middle Distillates. (30-31536)

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Fate and Effects

Bioaccumulation of polycyclic aromatic hydrocarbons and metals in estuarine organisms. (40-30741)

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Phenolics in Aquatic Ecosystems: A Selected Review of Recent Literature. (26-31781)

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Aquatic Toxicity

Polycyclic Aromatic Hydrocarbons [(PAH)] in the Aquatic Environment: Sources, Fates, and Biological Effects. (27-32178)

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Polycyclic Aromatic Hydrocarbons [(PAHs)] : A Review (28-30174)

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Polycyclic Aromatic Hydrocarbons [(PAHs)] : A Review (28-30174)

POLYMERIZATION NAPHTHA

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PROCESSING PRODUCTS OF COAL LIQUEFACTION (REVIEW)

Review of the Status of Human Community Health and Environmental Health Effects Research of Pollutants from Coal Liquefaction, Oil Shale, and Petroleum Technologies. A Final Report (29-32809)

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Human Exposure Assessment

Produced water radionuclide hazard/risk assessment. Phase I. (39-31094)

Risk Assessment

Produced water radionuclide hazard/risk assessment. Phase I. (39-31094)

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Toxicity Study

The Myocardial Toxicity [to Dogs] of Selected Aliphatic Hydrocarbons, [Including Propane (P), n-Butane, Isobutane, and a Mixture of These Hydrocarbons (A-46), and Mixtures of Isobutane with Methyl Chloroform, Trichloroethylene, Methyl Ethyl Ketone, and Methyl Isobutyl Ketone]. (30-32013)

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Odor and Taste

[A literature] review of published odor [threshold values (OTVs)] and taste threshold values [(TTVs)] of [the 276] soluble gasoline components (33-30589)

PROPYLENE

Metabolism/Pharmacokinetics

Studies on the absorption of inhaled hydrocarbon vapors. Final report. (34-33036)

RADIONUCLIDES

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Risk Assessment

Produced water radionuclide hazard/risk assessment. Phase I. (39-31094)

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Effects of Oil on Aquatic Organisms...A Review of Selected Literature (29-30299)

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A preliminary study of the effect of toluene on pregnancy [and in utero development] of the rat. (Inhalation exposure). (40-32425)

A single-generation inhalation reproduction/ fertility study on a commercial hexane. (33-32864)

A Single-Generation Inhalation Reproduction/ Fertility Study in Rats with Methyl t-Butyl Ether (MTBE). (32-30239)

Final Report, April 17, 1991. Commercial hexane test rule, 40 CFR 799.2155. Two-Generation reproduction study of inhaled commercial hexane in CD(TM) (Sprague-Dawley) rats. (38-31217)

Final Report, April 17, 1991. Commercial hexane test rule, 40 CFR 799.2155. Two-generation reproduction study of inhaled commercial hexane in CD(TM) (Sprague-Dawley) rats. (38-31216)

Inhalation reproduction range-finding study in mated rats with C_9 aromatic hydrocarbons. Final report. (35-31367)

Methyl tertiary butyl ether inhalation in rats: A single generation reproduction study. (35-30687)

Parental and Fetal Reproduction Inhalation Toxicity Study, in Rats, with Mixed Xylenes. Final Report. [Vol. I & II]. (31-31481)

Three generation [(F_0, F_1, and F_2)] reproduction/ fertility study in rats with C_9 aromatic hydrocarbons. (36-32642)

Toluene...The effect on pregnancy [and in utero development] of the rat. (Inhalation exposure). (40-32426)

Two-Generation Inhalation Reproduction/ Fertility Study on a Petroleum-Derived Hydrocarbon, Toluene [Vol. 1]. (32-32854)

[Bio/dynamics Inc. Project No. 79-2425] /An Inhalation Female Fertility Study with Benzene in Rats. (28-31212)

[Bio/dynamics Inc. Project No. 79-7342] /A Dominant-Lethal Inhalation Study with Benzene in Rats [and Effects on Male Reproductive Performance]. (28-31211)

[Litton Bionetics Inc. Project No. 21141-04] /Mutagenicity Evaluation of Diesel Fuel in the Mouse Dominant Lethal Assay. Final Report. (28-31346)

[Litton Bionetics Inc. Project No. 21141-02] /Mutagenicity Evaluation of Gasoline, API PS-6 Fuel (Unleaded) in the Mouse Dominant Lethal Assay. Final Report. (28-31344)

[Litton Bionetics Inc. Project No. 21141-03] /Mutagenicity Evaluation of Jet Fuel A in the Mouse Dominant Lethal Assay. Final Report. (28-31345)

[Litton Bionetics Inc. Project No. 21141-05] /Mutagenicity Evaluation of Toluene in the Mouse Dominant Lethal Assay. Final Report. (28-31347)

Dominant lethal assay

[Bio/dynamics Inc. Project No. 79-7342] /A Dominant-Lethal Inhalation Study with Benzene in Rats [and Effects on Male Reproductive Performance]. (28-31211)

[Litton Bionetics Inc. Project No. 21141-03] /Mutagenicity Evaluation of Jet Fuel A in the Mouse Dominant Lethal Assay. Final Report. (28-31345)

[Litton Bionetics Inc. Project No. 21141-02] /Mutagenicity Evaluation of Gasoline, API PS-6 Fuel (Unleaded) in the Mouse Dominant Lethal Assay. Final Report. (28-31344)

[Litton Bionetics Inc. Project No. 21141-05] /Mutagenicity Evaluation of Toluene in the Mouse Dominant Lethal Assay. Final Report. (28-31347)

[Litton Bionetics Inc. Project No. 21141-04] /Mutagenicity Evaluation of Diesel Fuel in the Mouse Dominant Lethal Assay. Final Report. (28-31346)

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A 14 day palatability and stability study in mice with benzene administered in drinking water. (37-32486)

A 14 day palatability and stability study in rats with benzene administered in drinking water. (37-32485)

API Exposure Classification Scheme for collection of industrial hygiene monitoring data. (40-30571)

API's Approach to Quality Assurance [in API Industrial Toxicology Studies]. (30-32064)

Dealing with uncertainties in a biologically based risk assessment model of cyclophosphamide-induced leukemogenesis. (41-32131)

Feasibility Analysis for an Historical Prospective Mortality Study of Employees Exposed to Downstream Gasoline in the Petroleum Industry. Final Report. (32-32565)

Feasibility of Case-Control Studies of Kidney Cancer and Hydrocarbon Exposure among Petroleum Company Workers. (32-32225)

Further development and application of an improved population exposure model. SYSAPP-86/061. (34-32923)

In Vitro Determination of Chemical Cocarcinogenesis in Balb/c 3T3-$A31_{SF}$ Cell Cultures. (31-30263)

Lack of Concordance of the *Salmonella*/ Microsome Assay with the Mouse Dermal Carcinogenesis Bioassay for Complex Petroleum Hydrocarbon Mixtures. (32-31299)

Meeting the Quality Assurance Challenges of the 1980's: Team Auditing by Toxicologists and QA Professionals. (31-32162)

Monitoring near refineries for airborne chemicals on the SARA [(Superfund Amendments and Reauthorization Act)] Title III Section 313 List--2. A Generic Study Design Protocol. (40-30722)

Statistical Evaluations in the Carcinogenesis Bioassay of Petroleum Hydrocarbon. (30-31983)

Stimulation and Inhibition of Chemotaxis in Abelson Virus-Transformed Macrophages, [in Relation to the Clearance of Air Pollutants in the Lung]. (31-30265)

Teratogenicity testing in vitro: Status of validation studies. (34-32774)

The Occurrence and Natural History of Experimental Skin Tumors. (30-31999)

The generation and analytical methods development work for the C_9 aromatic hydrocarbons program. Final report. (35-32077)

Validation of Behavioral Studies for Determining Neurotoxicity. Final Report. (31-31472)

Validation of Behavioral Studies for Determining the Toxicity of Petroleum Hydrocarbons. (30-32063)

[A review of] short-term toxicity tests for [atmospheric and aqueous] environmental carcinogens and mutagens (34-33031)

RISK ASSESSMENT

Dealing with uncertainties in a biologically based risk assessment model of cyclophosphamide-induced leukemogenesis. (41-32131)

Estimation of incremental benzene exposure associated with seven bulk gasoline storage facilities in North Carolina. (40-32761)

Produced water radionuclide hazard/risk assessment. Phase I. (39-31094)

RUBBER SOLVENT

Developmental Effects

Teratology Study in Rats. Rubber Solvent. Final Report. (26-60015)

Mutagenicity Study

Estimation of the Mutagenicity of Hydrocarbon Fractions Utilizing the Bacterial Assay Procedure with Mammalian Tissue Metabolic Activation. API Project PS-4 #6. (26-60103)
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Toxicity Study

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RUBBER SOLVENT (FILTERED)

Mutagenicity Study

Mutagenic Evaluation of Filtered Rubber Solvent (CHF-32-263). (26-60016)

SHALE DUST, RAW

Carcinogenicity Study

A Final Report [on a] 24 Month Inhalation Toxicity Study of Raw and Spent Shale Dusts in Rats and Monkeys (27-32466)

Developmental Effects

Teratology Studies in Rats. Raw Shale Dust. Final Report. (26-60007)

Toxicity Study

A Final Report [on a] 24 Month Inhalation Toxicity Study of Raw and Spent Shale Dusts in Rats and Monkeys (27-32466)
Long-Term Inhalation Studies with Raw and Processed [Oil] Shale Dusts. (30-31535)
Teratology Studies in Rats. Raw Shale Dust. Final Report. (26-60007)

SHALE DUST, RETORTED

Developmental Effects

Teratology Study in Rats. Retorted Shale Dust. Final Report. (26-60008)

Toxicity Study

Teratology Study in Rats. Retorted Shale Dust. Final Report. (26-60008)

SHALE DUST, SPENT

Carcinogenicity Study

A Final Report [on a] 24 Month Inhalation Toxicity Study of Raw and Spent Shale Dusts in Rats and Monkeys (27-32466)

Toxicity Study

Long-Term Inhalation Studies with Raw and Processed [Oil] Shale Dusts. (30-31535)

SHALE OIL

Analytical Chemistry/Methodology

Analysis of Shale Oils and Downstream Products. (27-33264)
Comprehensive Analysis of Shale Oil Products (Revised). (26-60063)

SHALE OIL (REVIEW)

Review of the Status of Human Community Health and Environmental Health Effects Research of Pollutants from Coal Liquefaction, Oil Shale, and Petroleum Technologies. A Final Report (29-32809)

SHALE OIL, CRUDE

Analytical Chemistry/Methodology

[API Project SPS-5-UOC(858/305)] Comprehensive Analyses of Shale Oil Products: Second Study. Final. (28-70300)
[Thirty-Day] Dermal Carcinogenesis Study of Shale-Derived Hydrocarbon Mixtures [(350-550 °F and 550-700 °F)] in Mice. (32-30626)

Carcinogenicity Study

The Determination of the Carcinogenicity of [Shale] Retort Oils and Downstream Products. (30-32849)

SHALE OIL, HYDROTREATED

Analytical Chemistry/Methodology

[API Project SPS-5-UOC(858/305)] Comprehensive Analyses of Shale Oil Products: Second Study. Final. (28-70300)
[Thirty-Day] Dermal Carcinogenesis Study of Shale-Derived Hydrocarbon Mixtures [(350-550 °F and 550-700 °F)] in Mice. (32-30626)

Carcinogenicity Study

Statistical Analyses of Crude Oil and Shale Oil Carcinogenic Test Data (30-32001)
The Determination of the Carcinogenicity of [Shale] Retort Oils and Downstream Products. (30-32849)

Toxicity Study

Twenty-Eight Day Subchronic Dermal Toxicity Study in Rats: API-HNC-1 (High-Nitrogen, [0.77%], Crude Oil); API-SFP-105 (0.05% N, Hydrotreated Shale Oil); API-SFP-119 (0.19% N, Hydrotreated Shale Oil); API-SFP-206 (0.06% N, Hydrotreated Shale Oil); API-SFR-1 ([1.6% N], Raw Shale Oil); API-SFR-2 ([2.1% N], Raw Shale Oil). (32-32652)

SHALE OIL, IN SITU

Analytical Chemistry/Methodology

[API Project SPS-5-UOC(858/305)] Comprehensive Analyses of Shale Oil Products: Second Study. Final. (28-70300)

Carcinogenicity Study

The Determination of the Carcinogenicity of [Shale] Retort Oils and Downstream Products. (30-32849)

SHALE OIL, RAW

Carcinogenicity Study

Lifetime dermal carcinogenesis bioassay of shale oil-derived streams in C3H/HeJ mice. Final interim report. (33-32009)
Statistical Analyses of Crude Oil and Shale Oil Carcinogenic Test Data. (30-32001)

Mutagenicity Study

In vivo sister chromatid exchange assay API RO-1 raw shale oil. (37-32484)
Sister chromatid exchange assay in Chinese hamster ovary (CHO) cells: API RO-1, raw shale oil. Final report. (33-30445)

Toxicity Study

Twenty-Eight Day Subchronic Dermal Toxicity Study in Rats: API-HNC-1 (High-Nitrogen, [0.77%], Crude Oil); API-SFP-105 (0.05% N, Hydrotreated Shale Oil); API-SFP-119 (0.19% N, Hydrotreated Shale Oil); API-SFP-206 (0.06% N, Hydrotreated Shale Oil); API-SFR-1 ([1.6% N], Raw Shale Oil); API-SFR-2 ([2.1% N], Raw Shale Oil). (32-32652)

SHALE OIL, RETORTED

Carcinogenicity Study

The Carcinogenicity of Raw and Spent Oil Shales and Retort Oils. (29-32356)
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Teratology Study in Rats. Shale Oil R01. Final Report [on LBI Project No. 20726-R01]. (27-32129)
Teratology Study in Rats. Shale Oil R03. Final Report [on LBI Project No. 20726-R03]. (27-32127)
Teratology Study in Rats. Shale Oil R04. Final Report [on LBI Project 20726-R04]. (27-32128)

Mutagenicity Study

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Lifetime dermal carcinogenesis bioassay of refinery streams in C3H/HeJ mice (AP-135R). (36-31364)

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Short-term dermal tumorigenesis study of selected petroleum hydrocarbon [processing fractions] in male CD-1 mice. Initiation and promotion phases. (36-32643)

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[A Final Report by Litton Bionetics Inc. on] Mutagenicity Evaluation Studies in the Mouse Lymphoma Forward Mutation Assay [with] Sweetened Naphtha, [API] Sample 81-08. (32-31233)

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LC_{50} acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 81-08, sweetened naphtha (CAS 64741-87-3). Final report [by International Research & Development Corp.]. (33-31827)

Lifetime Dermal Carcinogenesis/Chronic Toxicity Screening Bioassay of Refinery Streams in C3H/HeJ Mice. Three-Month Toxicity Evaluation Report. [Vol. 1-2]. (32-32653)

Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Twelve-month toxicity evaluation report. Final report. [Vol. 1-3]. (33-31451)

[A Hazleton Laboratories America Inc.] Dermal Sensitization Study in Guinea Pigs [by the] Closed Patch Technique [with the Use of] Sweetened Naphtha, API Sample 81-08. (31-31351)

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Teratology Study in Rats. Toluene. Final Report. (26-60019)

Toluene...The effect on pregnancy [and in utero development] of the rat. (Inhalation exposure). Toxicology Report Number TR401, June 1993. (40-32426)

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Absorption of petroleum products across the skin of the monkey and miniature pig. Annual report (Mar. 15, 1979-Mar. 14, 1980). (33-31034)

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Toluene...The effect on pregnancy [and in utero development] of the rat. (Inhalation exposure). Toxicology Report Number TR401, June 1993. (40-32426)

Two-Generation Inhalation Reproduction/ Fertility Study on a Petroleum-Derived Hydrocarbon, [i.e.,] Toluene [Vol. 1]. (32-32854)

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28-day dermal toxicity in the rabbit. API 84-02, heavy thermally cracked naphtha (CAS 64741-83-9). Final report [by Tegeris Laboratories Inc.]. (33-31696)

28-day dermal toxicity study in the rabbit: API 83-19, light alkylate naphtha (CAS 64741-66-8). Final report. (33-30498)

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Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-11, Straight-Run Middle Distillate (CAS 64741-44-2). (32-32857)

Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-06, Heavy Catalytically Reformed Naphtha (CAS 64741-68-0). (32-32860)

Acute Toxicity Studies of Catalytically Cracked Clarified Oil, API Sample 81-15. (30-31854)

Acute Toxicity Studies [by Hazleton Raltech Inc.] of Hydrodesulfurized Kerosine, [API] Sample 81-07. (30-31986)

Acute Toxicity Studies of Sweetened Naphtha, [API] Sample 81-08. (30-31990)

Acute Toxicity Studies of Vacuum Residuum, [API] Sample 81-14. (30-31989)

Acute Toxicity Studies [by Hazleton Raltech Inc. of] Hydrodesulfurized Middle Distillate, [API] Sample 81-10. (30-32348)

Acute Toxicity Studies [by Hazleton Raltech Inc. of] Hydrodesulfurized Middle Distillate, [API] Sample 81-09. (30-32347)

Acute Toxicity Studies of Light Catalytically Cracked Naphtha, [API] Sample 81-03. (30-31988)

Acute Toxicity Studies of Vacuum Residuum, [API] Sample 81-13. (30-31987)

Acute Toxicity Testing of Shale Oil and Process Materials. Final Report. (26-60027)

Closed-patch repeated insult dermal sensitization study of tertiary amyl methyl ether (TAME) in guinea pigs (Buehler method). (41-05416)

Dermal sensitization study in guinea pigs [by Hazleton Laboratories America Inc.] : API 83-04 light catalytically reformed naphtha (CAS 64741-63-5). Final report. (33-30496)

Dermal sensitization study in guinea pigs [by Hazleton Laboratories America Inc.] : API 83-05 full-range catalytically cracked reformed naphtha (CAS 68955-35-1). Final report. (33-30497)

Dermal sensitization study in guinea pigs [by Hazleton Laboratories America Inc.] : API 81-04 light catalytically cracked naphtha (CAS 64741-55-5). Final report. (33-30495)

Dermal Sensitization Study in Guinea Pigs. Closed Patch Technique. Hydrodesulfurized Middle Distillate, API Sample 81-09. (31-31352)

Dermal Sensitization Study in Guinea Pigs. Closed Patch Technique. Hydrodesulfurized Middle Distillate, API Sample 81-10. (31-31414)

Dermal Sensitization Study in Guinea Pigs. Closed Patch Technique. Vacuum Residuum, API Sample 81-13. (31-31415)

Dermal Sensitization Study in Guinea Pigs. Closed Patch Technique. Catalytic Cracked Clarified Oil, API Sample 81-15. (31-31417)

Dermal Sensitization Study in Guinea Pigs. Closed Patch Technique. Vacuum Residuum, API Sample 81-14. (31-31416)

Four-week dermal range-finding toxicity study in rats: API 81-15, catalytically cracked clarified oil (CAS 64741-62-4). Final report. (33-30442)

Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Twelve-month toxicity evaluation report. Final report. [Vol. 1-3]. (33-31451)

Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Three-month toxicity evaluation report. Final report. (33-32010)

Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of [11] refinery streams in C3H/HeJ mice. Twelve-month toxicity evaluation. (34-32865)

Lifetime Dermal Carcinogenesis/Chronic Toxicity Screening Bioassay of Refinery Streams in C3H/HeJ Mice. Three-Month Toxicity Evaluation Report. [Vol. 1-2]. (32-32653)

Renal Cortical Degeneration [of Mice] Associated with Chronic Dermal Application of Petroleum- and Shale Oil-Derived Middle Distillates. (30-31536)

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Thirteen Week Dermal Toxicity Study of a Petroleum-Derived Hydrocarbon in Rats: (API 81-15) Catalytically Cracked Clarified Oil (CAS 64741-62-4). Final Report. (32-32743)

Twenty-eight-day dermal toxicity study in the rabbit. API 84-01, light paraffinic distillate (CAS 64741-50-0). Final report [by Tegeris Laboratories Inc.]. (33-31642)

Twenty-Eight-Day Dermal Toxicity Study in the Rabbit of Hydrodesulfurized Middle Distillate, [API] Sample 81-10, [by Borriston Laboratories Inc.]. Final Report. (30-32296)

Twenty-Eight Day Subchronic Dermal Toxicity Study in Rats: API-HNC-1 (High-Nitrogen, [0.77%], Crude Oil); API-SFP-105 (0.05% N, Hydrotreated Shale Oil); API-SFP-119 (0.19% N, Hydrotreated Shale Oil); API-SFP-206 (0.06% N, Hydrotreated Shale Oil); API-SFR-1 ([1.6% N], Raw Shale Oil); API-SFR-2 ([2.1% N], Raw Shale Oil). (32-32652)

Twenty-Eight-Day Dermal Toxicity Study in the Rabbit of Hydrodesulfurized Middle Distillate, [API] Sample 81-09. Final Report. (30-32298)

Twenty-Eight-Day Dermal Toxicity Study in the Rabbit of Hydrodesulfurized Kerosine, [API] Sample 81-07. Final report. (30-32297)

Twenty-four month dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice (AP-190r). Final Report. (36-33220)

[A Hazleton Laboratories America Inc.] Dermal

Sensitization Study in Guinea Pigs [by the] Closed Patch Technique [with the Use of] Sweetened Naphtha, API Sample 81-08. (31-31351)

[A Tegeris Laboratories Inc.] (28--day dermal toxicity study in the rabbit. API 83-05 full range catalytically reformed naphtha (CAS 68955-35-1). [This] final report (33-30598)

[Hazleton Laboratories America Inc.'s] Acute Oral Toxicity Study in Rats, Acute Dermal Toxicity Study in Rabbits, Primary Dermal Irritation Study in Rabbits, and Primary Eye Irritation Study in Rabbits of API 83-05 Full Range Catalytically Reformed Naphtha. (32-31474)

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[Project No. 1599] Acute Toxicity Tests of API 79-4, Paraffinic Oil (550 SUS/100 °F). (29-33066)

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A two-year inhalation study of petroleum coke in [150 male and 150 female Sprague-Dawley] rats and [four male and four female Cynomolgus] monkeys. (34-32600)

Acute inhalation toxicity evaluation in rats: API 83-07, light catalytically cracked distillate (CAS 64741-59-9). Final report. (33-30549)

Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats, API 83-09, straight-run kerosine (CAS 8008-20-6). Final report. (34-30634)

Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 83-20: Light catalytic cracked naphtha (CAS 64741-55-55). (34-32777)

Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats, API 83-11, straight-run middle distillate (CAS 64741-44-2). Final report. (34-30635)

Acute inhalation toxicity evaluation in rats: API 83-08, light catalytically cracked distillate (CAS 64741-59-9). Final report. (33-30444)

Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 85-01: Stoddard solvent (CAS 8052-41-3). (34-32779)

Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 83-18: Heavy catalytic cracked naphtha (CAS 64741-54-4). (34-32776)

Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 83-12: Hydrotreated light naphthenic distillate (CAS 64742-53-6). (34-32775)

Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 84-02: Heavy thermal cracked naphtha (CAS 64741-83-9). (34-32778)

Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats, API 83-19, light alkylate naphtha (CAS 64741-66-8). Final report. (34-30636)

Acute Inhalation Toxicity Evaluation of a Petroleum-Derived Hydrocarbon in Rats...Light Catalytic Cracked Naphtha. API Sample 81-04. (31-30680)

Acute Inhalation Toxicity Evaluation of a Petroleum-Derived Hydrocarbon in Rats. Light Catalytic Reformed Naphtha...API Sample 83-04. (31-30613)

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Acute Inhalation Toxicity Evaluation of a Petroleum-Derived Hydrocarbon in Rats. API #83-06, Heavy Catalytically Reformed Naphtha. Final Report. (32-32169)

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Chronic Gasoline Toxicity [Tests on Laboratory Animals]. (30-31991)

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Duration of [Human] Pulmonary Hyperresponsiveness to Acute Ozone Exposure. Final Report. (32-31232)

Effects of Nitrate and/or Sulfate Aerosols upon Cardiopulmonary Function. Final Report [for First Year of Project]. (27-30842)

Effects of Oxidant Levels on Selected Health Characteristics of Persons in the Los Angeles Basin. First Annual Report. Volume 1. Data Collection. (26-60079)

Effects of Oxides of Nitrogen, Carbon Monoxide and Photochemical Oxidants on the [Electrocardiogram] during Exercise and on Cardiopulmonary Function. Final Report (APRAC Project CAPM-21-74). (26-60045)

Effects of Sulfate Aerosols upon Human Pulmonary Function. CRC APRAC Project CAPM-27-75. (27-30841)

Effects of Sulfur Dioxide on Human Subjects Exhibiting Peripheral Airway Impairment (26-60023)

Effects of Sulphate Aerosols upon Human Cardiopulmonary Function. CRC APRAC Project CAPM-27-75. (27-30839)

Experimental Human Exposure to Carbon Monoxide [at Concentrations of < 1, 25, 50, 100, 200, 500 and 1000 ppm for 0.5 to 24 Hr]. (26-60001)

Five-Day Pilot Inhalation Study in Rats and Mice. Benzene. Final Report. (31-30448)

Four-week subchronic inhalation toxicity study in rats. Final report. API 81-07, hydrodesulfurized kerosine (petroleum) (CAS 64742-81-0). API 81-09, hydrodesulfurized middle distillate (petroleum) (CAS 64742-80-9). API 81-10, hydrodesulfurized middle distillate (petroleum) (CAS 64742-80-9). (33-32724)

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Kidney Effects of Unleaded Gasoline. Comprehensive and Critical Summary of Observations in Rats and Mice. (30-32845)

LC_{50} acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 81-08, sweetened naphtha (CAS 64741-87-3). Final report [by International Research & Development Corp.]. (33-31827)

LC_{50} *acute inhalation toxicity evaluation of a* petroleum-derived hydrocarbon in rats. API 81-03, light catalytically cracked naphtha (CAS 64741-55-5). Final report. (33-31902)

LC_{50} Acute Inhalation Toxicity Evaluation of a Petroleum-Derived Hydrocarbon in Rats. Hydrodesulfurized Middle Distillate, API Sample 81-09. (30-32856)

LC_{50} Acute Inhalation Toxicity Evaluation of a Petroleum-Derived Hydrocarbon in Rats. Hydrodesulfurized Middle Distillate, API Sample 81-10. (30-32857)

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Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-18, heavy catalytically cracked naphtha (CAS 64741-54-4). Final report. (33-30593)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-07, light catalytically cracked distillate (CAS 64741-59-9). (33-30162)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 84-01, light paraffinic distillate (CAS 64741-50-0). Final report. (33-30595)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-12, hydrotreated light naphthenic distillate (CAS 64742-53-6). Final report. (33-30592)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-16, light paraffinic distillate solvent extract (CAS 64742-05-8). (33-31226)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. Final report. API 85-01, Stoddard solvent. (CAS 8052-41-3). (33-32723)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. Final report. API 83-20, light catalytic cracked naphtha (petroleum). (CAS 64741-55-5). (33-32722)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-15, hydrotreated heavy naphthenic distillate (CAS 64742-52-5). Final report [by Hazleton Laboratories America Inc.]. (33-32639)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 84-02, heavy thermally cracked naphtha (CAS 64741-83-9). Final report. (33-30596)

Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-11, Straight-Run Middle Distillate (CAS 64741-44-2). (32-32857)

Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-09, Straight Run Kerosine (CAS 8008-20-6). (32-32858)

Acute Oral Toxicity Study in Rats, Acute Dermal Toxicity Study in Rabbits, Primary Dermal Irritation Study in Rabbits, [and] Primary Eye Irritation Study in Rabbits of API 81-04, Light Catalytically Cracked Naphtha. (32-31708)

Acute Oral Toxicity Study in Rats, Acute Dermal Toxicity Study in Rabbits, Primary Dermal Irritation Study in Rabbits, and Primary Eye Irritation Study in Rabbits of API 83-04 Light Catalytically Reformed Naphtha. (32-31473)

Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-08, Light Catalytically Reformed Distillate (CAS 64741-59-9). (32-32859)

Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-06, Heavy Catalytically Reformed Naphtha (CAS 64741-68-0). (32-32860)

Acute Toxicity Studies of Light Catalytically Cracked Naphtha, [API] Sample 81-03. (30-31988)

Acute Toxicity Studies of Vacuum Residuum, [API] Sample 81-14. (30-31989)

Acute Toxicity Studies of Vacuum Residuum, [API] Sample 81-13. (30-31987)

Acute Toxicity Studies [by Hazleton Raltech Inc.] of Hydrodesulfurized Kerosine, [API] Sample 81-07. (30-31986)

Acute Toxicity Studies [by Hazleton Raltech Inc. of] Hydrodesulfurized Middle Distillate, [API] Sample 81-10. (30-32348)

Acute Toxicity Studies [by Hazleton Raltech Inc. of] Hydrodesulfurized Middle Distillate, [API] Sample 81-09. (30-32347)

Acute Toxicity Studies of Catalytically Cracked Clarified Oil, API Sample 81-15. (30-31854)

Acute Toxicity Studies of Sweetened Naphtha, [API] Sample 81-08. (30-31990)

Acute Toxicity Testing of Shale Oil and Process Materials. Final Report. (26-60027)

The Acute Toxicology of Selected Petroleum Hydrocarbons. (30-31530)

Twenty-eight-day dermal toxicity study in the rabbit. API 84-01, light paraffinic distillate (CAS 64741-50-0). Final report [by Tegeris Laboratories Inc.]. (33-31642)

[Hazleton Laboratories America Inc.'s] Acute Oral Toxicity Study in Rats, Acute Dermal Toxicity Study in Rabbits, Primary Dermal Irritation Study in Rabbits, and Primary Eye Irritation Study in Rabbits of API 83-05 Full Range Catalytically Reformed Naphtha. (32-31474)

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[Project No. 1443] /Acute Toxicity Tests [of] API #78-1, New Composite Motor Oil. (27-32131)

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[Project No. 1443] Acute Toxicity Tests [on] API #78-4, No. 2 Home Heating Oil (50% Cat) (27-32068)

[Project No. 1443] Acute Toxicity Tests of API 78-7 No. 6 Heavy Fuel Oil (17.1 °API/0.8% S) (27-32774)

[Project No. 1443] Acute Toxicity Tests...API 79-6 Diesel Fuel (Marketplace Sample). (27-32817)

[Project No. 1443] Acute Toxicity Tests of API 79-2 No. 6 Heavy Fuel Oil (5.2 °API/1.2% S) (27-32813)

[Project No. 1443] Acute Toxicity Tests...API 78-6 No. 6 Heavy Fuel Oil (11.7 °API/2.7% S). (27-32814)

[Project No. 1597] Acute Toxicity Tests of API 79-1, Naphthenic Oil (90 SUS/210 °F). (29-33065)

[Project No. 1598] Acute Toxicity Tests, API 79-3, Paraffinic Oil (350 SUS/100 °F). (29-33067)

[Project No. 1599] Acute Toxicity Tests of API 79-4, Paraffinic Oil (550 SUS/100 °F). (29-33066)

[Project No. 1600] Acute Toxicity Tests, API 79-5, Paraffinic Oil (800 SUS/100 °F). (29-33068)

[Project No. 1601] Acute Toxicity Tests of API 78-9, Paraffinic Oil (70 SUS/100 °F). (29-33104)

[Project No. 1602] Acute Toxicity Tests of API 78-10, Paraffinic Oil (150 SUS/100 °F). (29-33105)

[Project No. 1616] Acute Toxicity Tests of API 78-5, Naphthenic Oil (150 SUS/100 °F). (29-33106)

Oral

A 90-day feeding study in the rat with six different white mineral oils (N15 (H), N70 (H), N70 (A), P15 (H), N10 (A), and P100 (H, three different mineral waxes (a low-melting-point wax, a high-melting-point wax, and a high-sulphur wax) and coconut oil. July 1992. (39-32387)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-19, light alkylate naphtha (CAS 64741-66-8). Final report. (33-30594)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-18, heavy catalytically cracked naphtha (CAS 64741-54-4). Final report. (33-30593)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-07, light catalytically cracked distillate (CAS 64741-59-9). (33-30162)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 84-01, light paraffinic distillate (CAS 64741-50-0). Final report. (33-30595)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-12, hydrotreated light naphthenic distillate (CAS 64742-53-6). Final report. (33-30592)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-16, light paraffinic distillate solvent extract (CAS 64742-05-8). (33-31226)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. Final report. API 85-01, Stoddard solvent. (CAS 8052-41-3). (33-32723)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. Final report. API 83-20, light catalytic cracked naphtha (petroleum) (CAS 64741-55-5). (33-32722)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-15, hydrotreated heavy naphthenic distillate (CAS 64742-52-5). Final report [by Hazleton Laboratories America Inc.]. (33-32639)

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 84-02, heavy thermally cracked naphtha (CAS 64741-83-9). Final report. (33-30596)

Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-11, Straight-Run Middle Distillate (CAS 64741-44-2). (32-32857)

Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-09, Straight Run Kerosine (CAS 8008-20-6). (32-32858)

Acute Oral Toxicity Study in Rats, Acute Dermal Toxicity Study in Rabbits, Primary Dermal Irritation Study in Rabbits, [and] Primary Eye Irritation Study in Rabbits of API 81-04, Light Catalytically Cracked Naphtha. (32-31708)

Acute Oral Toxicity Study in Rats, Acute Dermal Toxicity Study in Rabbits, Primary Dermal Irritation Study in Rabbits, and Primary Eye Irritation Study in Rabbits of API 83-04 Light Catalytically Reformed Naphtha. (32-31473)

Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-08, Light Catalytically Reformed Distillate (CAS 64741-59-9). (32-32859)

Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-06, Heavy Catalytically Reformed Naphtha (CAS 64741-68-0). (32-32860)

Acute Toxicity Studies of Vacuum Residuum, [API] Sample 81-13. (30-31987)

Acute Toxicity Studies of Vacuum Residuum, [API] Sample 81-14. (30-31989)

Acute Toxicity Studies [by Hazleton Raltech Inc.] of Hydrodesulfurized Kerosine, [API] Sample 81-07. (30-31986)

Acute Toxicity Studies of Catalytically Cracked Clarified Oil, API Sample 81-15. (30-31854)

Acute Toxicity Studies [by Hazleton Raltech Inc. of] Hydrodesulfurized Middle Distillate, [API] Sample 81-10. (30-32348)

Acute Toxicity Studies [by Hazleton Raltech Inc. of] Hydrodesulfurized Middle Distillate, [API] Sample 81-09. (30-32347)

Acute Toxicity Studies of Light Catalytically Cracked Naphtha, [API] Sample 81-03. (30-31988)

Acute Toxicity Studies of Sweetened Naphtha, [API] Sample 81-08. (30-31990)

Acute Toxicity Testing of Shale Oil and Process Materials. Final Report. (26-60027)

Final Report. A four-week oral nephrotoxicity screening study in male F344 rats (33-31097)

Four-Week Oral Nephrotoxicity Screening Study in Male F344 Rats [by Borriston Laboratories Inc.]. Final Report. (32-30966)

Hydrocarbon nephropathy in male rats: Identification of the nephrotoxic components of unleaded gasoline (33-31098)

Hydrocarbon nephropathy in male rats. Temporal and morphologic characterization of the renal lesions. (33-31223)

Serial Sacrifice Study [by Borriston Laboratories Inc.] with [API PS-6] Unleaded Gasoline in Rats. Final Report. (32-31048)

The Acute Toxicology of Selected Petroleum Hydrocarbons. (30-31530)

The Chronic Toxicity of Lead. (26-60096)

Twenty-eight-day dermal toxicity study in the rabbit. API 84-01, light paraffinic distillate (CAS 64741-50-0). Final report [by Tegeris Laboratories Inc.]. (33-31642)

[Hazleton Laboratories America Inc.'s] Acute Oral Toxicity Study in Rats, Acute Dermal Toxicity Study in Rabbits, Primary Dermal Irritation Study in Rabbits, and Primary Eye Irritation Study in Rabbits of API 83-05 Full Range Catalytically Reformed Naphtha. (32-31474)

[Project No. 1443] Acute Toxicity Tests of API 78-7 No. 6 Heavy Fuel Oil (17.1 °API/0.8% S) (27-32774)

[Project No. 1443] Acute Toxicity Tests...API 79-7 Used Composite Motor Oil. (27-32772)

[Project No. 1443] Acute Toxicity Tests [on] API #78-4, No. 2 Home Heating Oil (50% Cat) (27-32068)

[Project No. 1443] Acute Toxicity Tests [of] API #78-1, New Composite Motor Oil. (27-32131)

[Project No. 1443] Acute Toxicity Tests [of] API #PS-6, Unleaded Motor Gasoline (27-32130)

[Project No. 1443] Acute Toxicity Tests of API #78-3 No. 2 Home Heating Oil (10% Cat) (27-32773)

[Project No. 1443] Acute Toxicity Tests of API #78-2 No. 2 Home Heating Oil (30-% Cat) (27-32771)

[Project No. 1443] Acute Toxicity Tests of API Jet Fuel A (27-32815)

[Project No. 1443] Acute Toxicity Tests...API 79-6 Diesel Fuel (Marketplace Sample). (27-32817)

[Project No. 1443] Acute Toxicity Tests of API 79-2 No. 6 Heavy Fuel Oil (5.2 °API/1.2% S) (27-32813)

[Project No. 1443] Acute Toxicity Tests...API 78-8 No. 6 Heavy Fuel Oil (23.1 °API/0.2% S). (27-32816)

[Project No. 1443] Acute Toxicity Tests...API 78-6 No. 6 Heavy Fuel Oil (11.7 °API/2.7% S). (27-32814)

[Project No. 1597] Acute Toxicity Tests of API 79-1, Naphthenic Oil (90 SUS/210 °F). (29-33065)

[Project No. 1598] Acute Toxicity Tests, API 79-3, Paraffinic Oil (350 SUS/100 °F). (29-33067)

[Project No. 1599] Acute Toxicity Tests of API 79-4, Paraffinic Oil (550 SUS/100 °F). (29-33066)

[Project No. 1600] Acute Toxicity Tests, API 79-5, Paraffinic Oil (800 SUS/100 °F). (29-33068)

[Project No. 1601] Acute Toxicity Tests of API 78-9, Paraffinic Oil (70 SUS/100 °F). (29-33104)

[Project No. 1602] Acute Toxicity Tests of API 78-10, Paraffinic Oil (150 SUS/100 °F). (29-33105)

[Project No. 1616] Acute Toxicity Tests of API 78-5, Naphthenic Oil (150 SUS/100 °F). (29-33106)

Review

A Brief Review of [Published Data on] the Toxicity and Carcinogenicity of Select Chlorinated Hydrocarbons. (32-30417)

A Review of the Toxicology of Lead (26-60061)

A Review of the Human Kidney Effects of Hydrocarbon Exposure (32-30410)

A Unified Approach to the Use of Human Clinical and Animal Toxicologic Data; Application to the Establishment of Ambient Air Quality Criteria. Volume IA: Effect of Ozone on Airway Resistance (Raw) and Forced Expiratory Volume ($FEV_{1.0}$) in Humans. Final Report. (32-32503)

A Unified Approach to the Use of Human Clinical Data: A Case Study of Nitrogen Dioxide and Sulfur Dioxide. (32-32502)

Air Quality Standards for Carbon Monoxide. (26-60062)

Air Quality Standards for Lead. (26-60064)

Ambient Air Quality Standards for Particulates...Review and Evaluation. (26-60057)

API Mineral Oil Review. (39-31651)

Arsenic. (26-60065)

Barium. (26-60110)

Benzene Toxicity. A Critical Evaluation (26-60074)

Cadmium, Zinc, and Mercury. (26-60115)

Cadmium: Environmental and Community Health Impact. (32-30599)

Chromium. (26-60113)

Clinical toxicology of the acute ingestion of methanol/hydrocarbon blends. A state-of-the-art review. (39-31093)

Copper. (26-60068)

Cytologic and Cytogenetic Effects of Benzene [on Human Health]. (26-60121)

Dioxins and furans...A primer: What they are and how to measure them. (37-31299)

Effects of Oil on Aquatic Organisms...A Review of Selected Literature (29-30299)

Facts and Opinions on the Role of Sulfur Dioxide in Causing Injury [to Human Health]. (26-60095)

Fluorosis of Livestock. (26-60107)

Gaseous and Particulate Sulfur Compounds in Urban Atmospheres. (26-60070)

Hydrocarbon Toxicity: Acute, Subchronic and Chronic Effects in Relation to Unleaded Gasoline Exposure of Rodents, with Comments on the Significance to Human Health. (32-30192)

Lithium. (26-60067)

Manganese. (26-60114)

Methanol health effects. Epidemiology literature review and search for study population--1. Critical review of the literature. 2. Search for study population. (39-31095)

Microparticulate Sulfates: Effects on Human Health. (26-60116)

Nickel. (26-60112)

Oxidants: Air Quality Criteria Based on Health Effects. (26-60060)

Particulates: Air Quality Criteria Based on Health Effects. (26-60058)

Phosphorus. (26-60066)

Polycyclic Aromatic Hydrocarbons [(PAHs)] : A Review (28-30174)

Response of Specific Airways Resistance to SO_2 in the Exercising Asthmatic: Dose Considerations. (32-32461)

Review of Inhalation Toxicology of Sulfuric Acid and Sulfates. (26-60024)

Study of the relationship between folate status and methanol toxicity. (41-30101)

Summation/Comments on Structure-Activity Relationships. (32-30422)

The Molecular Site of Benzene Toxicity. (26-60078)

The Renal Effects of Petroleum Hydrocarbons, Symposium (Boston 7/18-20/83). (32-01362)

Tin. (26-60069)

Vanadium: Environmental and Community Health Impact. (32-30600)

Vanadium. (26-60111)

Xenobiotic-Induced Kidney Lesions: Hydrocarbons. [A Synopsis of] the 90-Day and 2-Year Gasoline Studies. (32-30412)

[A Review on] Hematotoxicity in Humans (26-60073)

[A Review on] Experimental Benzene Intoxication (26-60077)

[This Compilation Based on the 1st API - Society of Toxicology "Toxicology of Petroleum Hydrocarbons" Symposium (Washington, D.C. 5/11-13/82).] (30-31529)

Subcutaneous

Effects of Subchronic Fourteen Day Exposure to Benzopyrene in B6C3F1 Male and

ABSTRACTS

24-30057

Carboxyhemoglobin Trend in Chicago Blood Donors, 1970-1974. An 18% reduction in the carboxyhemoglobin (HbCO) saturation in nonsmoking Chicago blood donors occurred between 1970 and 1974, indicating that donors in 1974 were being exposed to a lower average carbon monoxide (CO) concentration than was experienced by 1970 donors. In 1970, 74% of the nonsmokers in Chicago were being exposed to CO in excess of the amount permitted by the Federal air quality standards, but in 1974, only 41% of the nonsmokers were being overexposed. The observed reduction in HbCO correlates well with both the ambient CO levels recorded at the air monitoring stations and the reduction in CO emission from automobiles. If the current trend continues, Chicago should reach compliance with air quality standards for CO by 1985. The measurement of HbCO in a representative urban population is an accurate index of actual CO exposure and supplements the air pollution data provided by air monitoring stations. Map, graphs, tables, and 19 references. See also Abstract No. 21-30100.

■ R. D. Stewart; C. H. Hake; J. H. Kalbfleisch; T. A. Stewart; A. Wu. (Med. Coll. Wis.), *Archives of Environmental Health* 31(6):280-86 (Nov.-Dec. 1976).

Source: Not Available from API

26-31781

Phenolics in Aquatic Ecosystems: A Selected Review of Recent [(1950-79)] Literature covers analytical chemistry; synthetic sources of phenolics, e.g., natural gas plants, oil refineries, and chemical plants; natural phenolics, e.g., from leaf litter; chlorination of phenolics; toxicity of phenolics to bacteria, fungi, achlorophyllous protozoa, algae, and plants; acute and chronic effects on animals; modifiers of phenolic toxicity, including contaminants, structural and chemical properties, and environmental and biological factors; phenolics as nonspecific metabolic inhibitors; organismal effects, including development and growth, physiology and history, oxygen consumption, feeding, behavior, and other effects; populations and community effects; taste and odor problems; and environmental fate and effects, including transport, microbial and photochemical degradation, and uptake and depuration. Tables and about 375 references. (supported by API) See also Abstract No. 23-30918, 25-32531, and 26-31539.

■ A. L. Buikema; J. Cairns (Va. Polytech. Inst. & State Univ.); M. J. McGinniss (Metcalf & Eddy Inc.), *Marine Environmental Research* 2(2):87-181 (Apr. 1979).

Source: Not available from API

26-60001

Experimental Human Exposure to Carbon Monoxide [at Concentrations of < 1, 25, 50, 100, 200, 500 and 1000 ppm for 0.5 to 24 Hr] led to no untoward effects in sedentary males exposed to 100 ppm or less for 8 hr. Exposures producing carboxyhemoglobin saturations > 15-20% resulted in delayed headaches, changes in the visual evoked response, and impairment of manual coordination. Diagram, graph, and tables.

■ R. D. Stewart; J. E. Peterson; R. T. Bachand; E. D. Baretta; A. Herrmann; M. J. Hosko (Marquette Sch. Med. - Gen. Mot. Corp.), *Archives of Environmental Health* 21(2):154-64 (Aug. 1970).

Source: Not available from API

26-60002

The Toxicity of Water-Soluble Fractions of Four Test Oils for the Polychaetous Annelids *Neanthes arenaceodentata* and *Capitella capitata* were studied in the laboratory. The two refined oils, No. 2 fuel oil and bunker C residual oil, proved most toxic to both species. South Louisiana crude oil was less toxic than either of the refined oils, yet more toxic than Kuwait crude oil. The higher concentrations of toxic diaromatic compounds (naphthalenes) found in refined oils probably accounted for major differences between the toxicity of refined vs. crude oils. *Capitella capitata* was slightly more sensitive to three of the four oils than was *Neanthes arenaceodentata*. Both species appear to be quite similar to fish and crustaceans in their sensitivity to these four oils. Tables, graphs, and 14 references. (supported by Exxon Co. U.S.A. and American Petroleum Institute)

■ S. S. Rossi; J. W. Anderson; G. S. Ward (Tex. A&M Univ.), *Environmental Pollution* 10(1):9-18 (Jan. 1976).

Source: Not available from API

26-60003

Mutagenic Evaluation of 60 Solvent (CHF-33-148). 60 Solvent (CHF-33-148) showed no mutagenic activity in a series of in vitro and in vivo tests. The in vitro studies involved Ames assay with bacteria *Salmonella* and yeasts *Saccharomyces*, with and without mouse-liver tissue activation, and mouse lymphoma assay. In the in vivo studies the bone marrow cell chromosomes of rats were examined for induced aberrations. Tables. (supported by the American Petroleum Institute)

■ *API Medical Research Publication* (10/31/75) (150 p.).

Source: API Library

26-60004

Mutagenicity Evaluation of [Shale Oil] R-01. Draft Report. Shale Oil R-01 showed mutagenic activity in Ames assay on *Salmonella* bacteria activated with rat liver tissue, weak activity in the absence of activation and also in mouse lymphoma in vitro assays. No significant effect was observed in in-vivo studies of frequency of chromosome aberrations in rat bone marrow cells. Tables. (supported by the American Petroleum Institute)

■ *API Medical Research Publication* (Dec. 1975) (150 p.).

Source: API Library

26-60005

Mutagenicity Evaluation of [Shale Oil] R-03. Draft Report. Shale oil R-03 showed mutagenic activity in Ames assays on bacteria *Salmonella* with activation by rat liver tissue. It was not mutagenic tests with the bacteria without activation, nor did it show activity in in-vitro mouse lymphoma assays. In-vivo studies showed no activity with respect to chromosome aberrations in rat bone marrow cells. Tables. (supported by the American Petroleum Institute)

■ *API Medical Research Publication* (Jan. 1978) (150 p.).

Source: API Library

26-60006

Mutagenicity Evaluation of Retorted Shale. Final Report. Retorted shale showed no mutagenic activity in a series of in-vitro and in-vivo tests. The in-vitro studies involved Ames assay with bacterial *Salmonella* and yeasts *Saccharomyces*, with and without rat-liver tissue activation, and mouse lymphoma assay. In the in-vivo studies the bone marrow cell chromosomes of rats were examined for induced aberrations. Tables. (supported by the American Petroleum Institute)

■ *API Medical Research Publication* (July 1978) (60 p.).

Source: API Library

26-60007

Teratology Studies in Rats. Raw Shale Dust. Final Report. Exposing pregnant rats to air containing 17.7 or 101.5 mg/cu m of raw shale dust caused no adverse effects on either the mother or the embryos. Exposure was during days 6 through 15 of gestation for 6 hr/day. The dust was more than 90% below 10 microns particle size. The condition of the pregnant rats was evaluated on the basis of body weight, food consumption, and general behavior. Fetus effects were determined by removal of the uterus on the 20th day of gestation and examination for number and placement of implantations and resorptions, number of live and dead fetuses, soft tissue changes, and skeletal abnormalities. Tables. (Supported by the American Petroleum Institute)

■ *API Medical Research Publication* (Oct. 1978) (25 p.).

Source: API Library

26-60008

Teratology Study in Rats. Retorted Shale Dust. Final Report. Exposure of pregnant female rats to air containing 62.2 or 145.6 mg/cu m of retorted shale dust had no effects on the embryos, but caused

increased incidence of pulmonary changes in the mothers. The pregnant rats were exposed to the retorted shale on days 6 through 15 of gestation for 6 hr/day. Particle size of the dust was more than 90 per cent below 10 microns. Effects on the fetuses were determined by removing the uterus on day 20 of gestation and determining the number and placement of implantations and resorptions, the number of live and dead fetuses, soft tissue changes, and skeletal abnormalities. Although the exposed rats showed lung changes on necropsy, exposure to the dust had no effect on body weight or food consumption. Tables. (Supported by the American Petroleum Institute)
■ *API Medical Research Publication* (Oct. 1978) (7 p.).
Source: API Library

26-60009
Mutagenicity Evaluation of [Shale Oil] R-04. Shale oil R-04 showed mutagenic behavior in tests with several strains of *Salmonella* bacteria when activated with rat liver extracts, weak mutagenic effects in the mouse lymphoma cell assay, but no mutagenicity in in-vivo tests involving examination of rat bone cell marrow for chromosome aberrations, or in in-vitro tests with *Saccharomyces* yeasts. Tables. (supported by American Petroleum Institute)
■ *API Medical Research Publication* (Jan. 1978) (150 p.).
Source: API Library

26-60010
Mutagenicity Evaluation of Stoddard [Solvent] "Fuel". Stoddard Solvent showed no mutagenic activity in tests with *Saccharomyces* yeast and bacteria *Salmonella*, with or without rat liver activation, or in the mouse lymphoma assay, or in in-vivo tests involving examination of the chromosomes in rat bone marrow. Tables. (supported by American Petroleum Institute)
■ *API Medical Research Publication* (Jan. 1978) (150 p.).
Source: API Library

26-60011
Mutagenic Evaluation of Rubber Solvent (CHF-32-263). The rubber solvent CHF-32-263 did not induce significant genetic activity in in-vitro or in-vivo assays. The in-vitro tests were (1) Ames assays on bacteria *Salmonella* and yeast *Saccharomyces* exposed to concentrations of 1-500 µl of rubber solvent with and without activation by mouse liver microsomes, and (2) mouse lymphoma cell assays in the presence of 0.001 to 1.0% of the rubber solvent. In-vivo tests involved injecting rats with 0.166-1.66 ml/kg of rubber solvent and examining the bone marrow cells for chromosome aberrations. Additional tests indicated that rubber solvent did not induce dominant lethality when male rats receiving 0.083-0.83 ml/kg were mated with females after five consecutive days of treatment. Tables. (supported by American Petroleum Institute)
■ *API Medical Research Publication* (10/31/79) (150 p.).
Source: API Library

26-60013
Teratology Study in Rats. Xylene. Final Report. Exposing pregnant female rats to atmospheres containing 100 and 400 ppm of xylene for 6 hr/day on days 6-15 of gestation produced no adverse effects on the mothers and there was no evidence of fetal sex ratio variation, embryo toxicity, inhibition of fetal growth, or teratogenic potential. Tables. (Supported by American Petroleum Institute)
■ *API Medical Research Publication* (Apr. 1978) (38 p.).
Source: API Library

26-60014
Teratology Study in Rats. Unleaded Gasoline. Final Report. Pregnant female rats that were exposed to atmospheres containing 400 and 1600 ppm of unleaded gasoline on days 6-15 of gestation showed no evidence of adverse effects, no fetal sex ratio variation, and no embryo toxicity, inhibition of fetal growth, or teratogenic potential. Exposure was 6 hr/day. Tables. (Supported by the American Petroleum Institute)
■ *API Medical Research Publication* (Aug. 1978) (23 p.).
Source: API Library

26-60015
Teratology Study in Rats. Rubber Solvent. Final Report. Exposing pregnant female rats to atmospheres containing 800 and 1600 ppm of rubber solvent for 6 hr/day on days 6-15 of gestation produced no ill effects on the dams and no terata, fetal sex ratio variation, embryo toxicity, or inhibition of fetal growth. Tables. (Supported by the American Petroleum Institute)
■ *API Medical Research Publication* (June 1978) (17 p.).
Source: API Library

26-60016
Mutagenic Evaluation of Filtered Rubber Solvent (CHF-32-263). Filtered rubber solvent showed no genetic activity in Ames assays with bacteria *Salmonella* or yeast *Saccharomyces* or in-vitro mouse lymphoma cell assays or in in-vivo studies of rat bone marrow cells. Administering filtered rubber solvent at 0.083-0.83 ml/kg did not induce dominant lethality in females mated with these males. The in-vitro tests involved concentrations of 0.22-1.55% in the bacteria/yeast studies and 0.011-1.0% in the mouse lymphoma studies. In the bone marrow cell work concentrations ranged from .083 to 1.66 ml/kg of filtered rubber solvent. The bacteria/yeast tests were carried out with and without the presence of a mouse liver microsome activation system. Tables. (supported by American Petroleum Institute)
■ *API Medical Research Publication* (10/31/75) (150 p.).
Source: API Library

26-60017
Mutagenicity Evaluation of Kerosene. Final Report. Kerosine showed no mutagenic activity or germinal cell effects in a series of in-vivo and in-vitro tests. The in-vitro tests involved Ames assays on yeast *Saccharomyces* and bacteria *Salmonella* with and without activation by rat liver tissue, and attempted mutation induction in mouse lymphoma cells. In the in-vivo study the chromosomes of the bone marrow cells from rats treated with kerosine were examined. Tables. (supported by the American Petroleum Institute)
■ *API Medical Research Publication* (Mar. 1977) (150 p.).
Source: API Library

26-60018
Mutagenicity Evaluation of Xylene. Xylene did not show genetic effects in a series of in-vitro and in-vivo tests The in-vitro tests involved Ames assays with yeast *Saccharomyces* and bacteria *Salmonella* with and without rat liver activation as well as assay with mouse lymphoma cells. The in-vivo studies involve examining of bone marrow cell chromosomes of rats treated with xylene at 0.044-0.441 ml/kg. Tables. (supported by American Petroleum Institute)
■ *API Medical Research Publication* (Jan. 1978) (150 p.).
Source: API Library

26-60019
Teratology Study in Rats. Toluene. Final Report. Toluene showed no teratologic effect on pregnant rats breathing 100-400 ppm for 6 hr/day on days 6-15 of gestation. The rats showed no ill effects and there was no evidence of fetal sex ratio variation, embryo toxicity, inhibition of fetal growth, or teratogenic potential. (Supported by American Petroleum Institute)
■ *API Medical Research Publication* (Jan. 1978) (17 p.).
Source: API Library

26-60020
Mutagenicity Evaluation of Toluene. In-vitro and in-vivo studies of toluene gave results indicating that the compound does not have mutagenic properties. The in-vitro study involved Ames assays with yeast *Saccharomyces* and bacteria *Salmonella* with and without activation by rat liver tissue, as well as assay with mouse lymphoma cells. In the in-vivo studies the chromosomes of bone marrow cells from rats treated with toluene were examined for aberrations. Tables. (supported by American Petroleum Institute)
■ *API Medical Research Publication* (Jan. 1978) (150 p.).
Source: API Library

26-60021

Mutagenicity Evaluation of [Raw Shale] RS-101. Final Report. A series of tests indicated that raw shale RS-101 may have weak genetic activity. Ames assays with bacteria *Salmonella* and yeast *Saccharomyces* gave negative results but suspension tests showed an increase in mutation frequencies with dose for the TA-100 *Salmonella* strain when activated with rat liver. Weak and variable mutation increases were also obtained with two other bacteria strains. Mutation frequencies that increased at the high end of the dose range were also observed in mouse lymphoma assays. In-vivo work involving rat bone marrow cell chromosomes gave negative results except for one case. The results were, however, strongly suggestive of genetic activity. Tables. (supported by American Petroleum Institute)

■ *API Medical Research Publication* (May 1978) (150 p.).
Source: API Library

26-60022

Further Investigation on the Effects of Sulfur Dioxide on Human Subjects. Tests on human subjects showed a significant, reversible effect on pulmonary function of exposure for 120 hours to 3 ppm sulfur dioxide. Comparison of exposed subjects with controls demonstrated increased small airway resistance as shown by a difference in compliance at high respiratory frequencies. The pattern at both 1 and 3 ppm exposure was a large difference in compliance early in the tests becoming less different from the controls as exposure proceeded through 96 hours but showing a greater difference after 120 hours. This pattern suggests the effects of reflexive constriction of the airway in the early stages and breakdown in the adaptation processes in the later stages at the 3 ppm level. Subjects exposed for 48 hours to 3 or 6 ppm of sulfur dioxide did not show complete recovery of frequency dependence of dynamic compliance in 24 hours after cessation of exposure. Tables and graphs. (Sponsored by the American Petroleum Institute)

■ F. W. Weir; P. A. Bromberg (Ohio State Univ.), *API Medical Research Publication* (June 1972) (74 p.).
Source: API Library

26-60023

Effects of Sulfur Dioxide on Human Subjects Exhibiting Peripheral Airway Impairment do not appear to be significantly different from the effects on normal persons, as indicated in studies with persons showing definite small airway obstruction in the lungs. The subjects were exposed to atmospheres containing up to 3 ppm sulfur dioxide over a four week period. The work did show that significant, but minimal and reversible, effects occur in persons exposed to sulfur dioxide at 3 ppm, but not at concentrations below 1 ppm. A complementary blood study of the plasma from subjects exposed for up to 120 hours to up to 6 ppm of sulfur dioxide showed an increase of 1.1 nmoles of plasma S-sulfonate for each increase of 1 ppm of sulfur dioxide in the test atmosphere. Graphs, tables, and 10 references. (Sponsored by the American Petroleum Institute)

■ F. W. Weir; P. A. Bromberg; A. F. Gunnison; E. D. Palmes (Ohio State Univ.), *API Medical Research Publication* (Sept. 1973) (190 p.).
Source: API Library

26-60024

Review of Inhalation Toxicology of Sulfuric Acid and Sulfates. A review covers chemical mechanisms for oxidation of sulfur dioxide, observations on populations exposed to sulfates, experiments involving acute exposure of human, guinea pigs, mice, rabbits, rats, and cats, and chronic exposure studies on guinea pigs, cynomolgus monkeys, rats, dogs, and hamsters. It is concluded that acidic sulfate salt of submicron particle size may be hazardous but that the available information is not adequate. Graphs, tables, and 107 references. (Sponsored by the American Petroleum Institute)

■ M. C. Battigelli; J. F. Gamble (Univ. N.C., Chapel Hill), *API Air Quality Monograph* 75-25 (1975) (89 p.).
Source: API Library

26-60025

Detectability and Irritability of Hydrocarbons [Stoddard Solvent and 70 Solvent] in Human Subjects. Final Report to American Petroleum Institute. Contract U-15-14-PS-5. Incidence of eye and nose irritation in human subjects exposed to 0.60 mg/l. Stoddard solvent or 0.35 mg/l. of 70 solvent was only slightly higher than in controls during 30 minute exposure. The olfactory threshold for Stoddard solvent was 0.002 mg/l. and that for 70 solvent was 0.003 mg/l. in a series of 30 second exposures. The solvents had no post-exposure effects. ASTM distillation ranges were 322-402 °F for the Stoddard solvent and 328-401 °F for the 70 solvent. The Stoddard solvent consists mainly of paraffins (34.9%), monocycloparaffins (34.9%), and alkylbenzenes (22.0%). The solvent contains 74.7% alkylbenzenes, 9.3% monocycloparaffins, 6.9% paraffins, and 3.5% naphthalenes. In the tests on the human subjects irritation was determined by subjective reports and by polygraph records of eye blink, swallowing, and respiration rates. A flow-dilution olfactometer system was used for all exposures. Tables. (Supported by the American Petroleum Institute)

■ G. P. Cooper; L. Hastings; W. Tanski (Univ. Cinci.), *API Medical Research Publication* (2/1/76) (7 p.).
Source: API Library

26-60026

Teratology study in Rats. Stoddard Solvent. Final Report. Pregnant rats exposed six hours a day during days 6-15 of gestation to 100 or 400 ppm of Stoddard solvent showed no evidence of variation in sex ratio, embryo toxicity, or inhibition of fetal development due to the hydrocarbons. The study involved determining the condition of the visceral and thoracic organs of the anesthetized rats on the 20th day of gestation and examination of the contents of the uterus. Tables. (LBI Project No. 20698-2) (Sponsored by the American Petroleum Institute)

■ *API Medical Research Publication* (10/17/77) (6 p.).
Source: API Library

26-60027

Acute Toxicity Testing of Shale Oil and Process Materials. Final Report. Tests of four shale oils, four kerogens, and three spent shales on rats, rabbits, and guinea pigs, indicated that spent shale can have significant irritant effects on the eye mucosa of rabbits, shale oil can cause erythema and edema when applied to the skin of rabbits and application of all of the materials to the shaved skin of guinea pigs can cause deaths. The LD_{50} in rats was 8-10 g/kg for the shale oil. None of the materials appear to be sensitizers as determined by application to the skin of guinea pigs. In general, the shale materials did not seem to have serious toxic effects. Tables and graphs. (Supported by the American Petroleum Institute)

■ *API Medical Research Publication* (Mar. 1978) (100 p.).
Source: API Library

26-60028

Investigation of the Potential Hazards of Cancer of the Skin Associated with the Refining of Petroleum. Final Report. Extensive experiments with mice have shown that carcinogenic compounds are present in almost all refinery streams, that they arise from catalytic cracking but not thermal cracking, and that they are concentrated in the streams boiling at 675-1000 °F. The carcinogenicity of the condensed ring aromatic hydrocarbons can be increased by as much as a factor of 5 by the presence of accelerators such as alkyl aromatics and sulfur compounds which are not themselves carcinogenic. The effect of these accelerators can be counteracted by the presence of polycycloparaffin. Washing the skin of mice with aqueous solution of soap reduced the incidence of tumors caused by application of catalytically cracked oil but effectiveness was lost if the oil was applied with great frequency (more than once a week). Prior rinsing with oil did not increase effectiveness of the washing and in some cases had the opposite effect, suggesting that the oil in question contained accelerators. Applying a barrier cream to the skin of the mice prior to application of the carcinogenic oil was of no value. A formula for expressing the carcinogenicity of a petroleum fraction based on its content of benzopyrenes, Diels-Alder carcinogen, and "3-1/2 ring" carcinogens is proposed. Methods of fractionation, identification, and analysis of carcinogens in refinery streams are presented. Tables and 28 references (Supported by American Petroleum Institute)

■ *API Research Project MC-1 Report* (10/20/59) (200 p.).
Source: API Library

26-60029
Effects of Sulfate Aerosols upon Cardiopulmonary Function in Squirrel Monkeys. Report of Second Year's Work under APRAC Project CAPM-20-74. *Squirrel* monkeys have proven to be good indicators of the respiratory irritant potency of aerosols; and total-respiratory-resistance, forced-pressure perturbation, a good measurement technique. In the second year of work under contract to the Coordinating Research Council an esophageal balloon method for pulmonary resistance and dynamic compliance was also developed. Results obtained with squirrel monkeys include: ammonium bisulfate and zinc ammonium sulfate caused statistically significant increases in respiratory resistance at both 10 and 20 Hz. In 40% humidity the irritants had a greater effect on resistance than at 85% humidity but the results were not statistically significant; squirrel monkeys' response to histamine phosphate (a known challenge aerosol) was an 80% increase in resistance on injection of the aerosol directly into the mouth; exposure for ten minutes to 200 ppm sulfur dioxide gave 14-63% increase in resistance, in qualitative agreement with published results on guinea pigs; ranking of irritant sulfates by monkeys was the same as from published data on guinea pigs but the responses were quantitatively smaller; responses of individual monkeys showed variability comparable to that in humans. The experimental methods used are described in detail. Graphs, photographs, tables, and diagrams.
■ K. A. Bell; J. D. Hackney; E. L. Avol; R. M. Bailey; H. L. Greenberg, *API Medical Research Publication* (6/24/77) (96 p.).
Source: API Library

26-60030
The Effects of Oil on Estuarine Animals: Toxicity, Uptake and Depuration, Respiration. Experiments with sheepshead minnows, brown shrimp, and grass shrimp exposed to sublethal concentrations of oil hydrocarbons, showed that the respiratory response is species-dependent and dependent on hydrocarbon composition and concentration. The respiratory response is transitory, returning to normal when the animals are returned to oil-free sea water. In 4-hr exposure to 1 ppm of naphthalene or 1-methylnaphthalene the minnows accumulated 200 ppm 1-methylnaphthalene or 60 ppm naphthalene in their tissues, the concentrations dropping to 10 and 30 ppm, respectively, after 29 hours in clean sea water. Brown shrimp in contact with the water soluble fraction of No. 2 fuel oil similarly built up high concentrations of naphthalene and alkylnaphthalene which dropped to undetectable levels after 10 hours in oil-free seawater. Accumulation of methylnaphthalenes was considerably greater than that of either naphthalene or dimethylnaphthalenes. The 24 hour LC_{50} value of the three species for naphthalene, 1-methylnaphthalene, 2-methylnaphthalene and dimethylnaphthalenes was determined. The test material, provided by the American Petroleum Institute, included two crude oils (South Louisiana and Kuwait), a high aromatic No. 2 fuel oil, and a Venezuelan Bunker C oil. Tables, graphs, and 20 references.
■ J. W. Anderson; J. M. Neff; B. A. Cox; G. M. Hightower; H. E. Tatem (Tex. A&M Univ.), in *Pollution and Physiology of Marine Organisms*, ed. F. J. Vernberg and W. B. Vernberg, pp. 285-310 (New York: Academic, 1974).
Source: Not available from API

26-60031
Occupational Safety and Health Problems of the Diving Industry in Offshore Petroleum Production. A seminar on diving includes papers on experiments carried out at the Royal Naval Physiological Laboratory and at the University of Pennsylvania, functions performed by divers in offshore petroleum production, capabilities of the diving industry, description of a deepwater petroleum production system, implications of the Occupational Safety and Health Act of 1970, safety problems in diving, unknowns in decompression table calculation, aseptic bone necrosis in divers, and discussion of diving problems and solutions in offshore petroleum operations. (Sponsored by the American Petroleum Institute).
■ E. M. Smith; E. L. Beckman (Univ. Tex.), *API Medical Research Report* EA-7104 (1972) (155 p.).
Source: API Library

26-60032
Overview of the Seminar's Objectives. [Seminar on Deep Sea Diving and the Petroleum Industry]. This introduction covers briefly the value of divers, and their cost, safety problems, insurance, health problems, and the limits of deep sea diving. Photographs and graphs. (Sponsored by the American Petroleum Institute)
■ E. L. Beckman (Univ. Tex.), *API Medical Research Report* EA-7104, pp. 1-12 (1972).
Source: API Library

26-60033
Experiments in Human Work Capabilities Under Pressure Now Being Conducted at the Royal Naval Physiological Laboratory culminated in exposure of two men to a simulated depth of 1500 feet in an oxygen-helium atmosphere. Brain activity measurements showed unusual patterns as a result of compression but mental efficiency was unaffected. However, the subjects also developed tremors which interfered with manual dexterity. The sodium and potassium content of the subjects' urine also was affected by pressure changes. In other experiments rats were compressed to the equivalent of 4000 foot depths, which resulted in electrical brain activity much like that seen at 1500 ft in the human experiments. Photographs, graphs, and tables.
■ P. B. Bennett (R. Nav. Physiol. Lab.), *API Medical Research Report* EA-7104, pp. 13-42 (1972).
Source: API Library

26-60034
Functions Performed by Divers in Offshore Petroleum Production include those associated with pipeline operations, platform installation and maintenance, work on drilling rigs, and salvage. The various tasks performed by divers and the hazards associated with each task are described briefly, as are a number of diving fatalities. Causes of these accidents are indicated briefly.
■ M. Hughes (Oceaneering Int.), *API Medical Research Report* EA-7104, pp. 55-62 (1972).
Source: API Library

26-60035
The Present and Projected Capabilities of the Diving Industry in the Support of Offshore Petroleum Production. The long decompression time for bounced diving at depths in the vicinity of 600 feet make saturation diving preferable, and this technique is safer even at 200 feet. There is a need to develop appropriate equipment standards as well as a good heated suit and a comfortable helmet with good communication and satisfactory safety system. Diving limits appear to 300 feet for nitrogen-oxygen atmosphere, 2500-3000 feet for helium-oxygen, and 5000-6000 feet for hydrogen-oxygen. Experience in the Janus II 840-foot saturation dive and in the Physalie IV simulated 1700-foot dive is reported. It is also noted that baboons have been compressed to the equivalent of 4000 feet in a helium breathing mixture and monkeys have been kept for up to six hours at the equivalent of 2500 feet in a nitrogen-oxygen atmosphere. Photographs and graphs.
■ H. G. Delauze (COMEX), *API Medical Research Report* EA-7104, pp. 63-78 (1972).
Source: API Library

26-60036
Deep-Water Petroleum Production Systems. The Humble subsea production system has been designed to operate in depths of 1500 feet for the anticipated life of an oilfield. Maintenance of the equipment is conducted in the open sea environment. It has a manipulator system for opening and closing production valves, replacing valve operators, making and breaking connectors, and recovering control pods as part of maintenance. It can be operated from the surface by means of television cameras but the equipment also has a diving bell for inspection. Special operations will be done by means of a submersible or divers.
■ L. J. Snyder (Humble Oil & Refining Co.), *API Medical Research Report* EA-7104, pp. 79-81 (1972).
Source: API Library

26-60037
Implications of the Occupational Safety and Health Act of 1970 [OSHA] to Industry. A survey includes: who is covered by OSHA, employee and employer requirements and rights, health and safety standards, criteria for compliance, inspection procedures, penalties, effect on existing workman's compensation laws, and the mission of the National Institute for Occupational Safety and Health.
■ E. J. Fairchild (NIOSH), *API Medical Research Report* EA-7104, pp. 83-90 (1972).
Source: API Library

26-60038
Medical Implications of the Occupational Safety and Health Act in the Diving Industry fall into three categories: recognized dangers that result from the offshore marine environment; hazards related to decompression sickness; and problems requiring more information, such as chronic toxicology under high pressure, treatment of injuries and illness under high pressure, and possibility of acute toxicity (convulsions) at higher pressures than presently encountered. Graphs.
■ H. W. Gillen (Marine Technol. Soc.), *API Medical Research Report* EA-7104, pp. 91-97 (1972).
Source: API Library

26-60039
Implications in the Occupational Safety and Health Act of 1970 Upon the Operational Costs of Diving Services. Because the new safety law requires additional safety equipment and more frequent equipment testing, higher standards, more comprehensive record keeping and reporting, and more training of personnel, the costs of equipment, base facilities, administrative overhead, recruitment training, medical coverage, and equipment maintenance will trend upward. The cost trends of insurance and of research and development will be down. The trend of diver's wages and of the cost of consumables and transportation will be unchanged.
■ J. A. Lawrie (Ocean Syst. Inc.), *API Medical Research Report* EA-7104, pp. 99-105 (1972).
Source: API Library

26-60040
Occupational Safety Problems in the Diving Industry. The sources of danger to the diver are categorized as: the diver himself by taking risks; the contractor who, for example, does not have a deck decompression chamber; the type of boat or platform from which the diving is done, especially a moving boat; layout of equipment on the barge; inadequate communication among crew members and between management and barge crew members and divers before and during the job; weather and tide, which cause changes in temperature, current, visibility, and surface conditions. Photographs.
■ R W Honaker (J & J Marine Diving Co. Inc.), *API Medical Research Report* EA-7104, pp. 107-16 (1972).
Source: API Library

26-60041
The Unknowns in Decompression Table Calculation and Their Implications in Occupational Safety and Health. The types of data needed to be able to establish safe diving conditions are discussed. It is stated that twice as much bottom time as most diving companies current permit is probably safe.
■ P. O. Edel (Hydrospace Co.), *API Medical Research Report* EA-7104, pp. 117-23 (1972).
Source: API Library

26-60042
Aseptic Bone Necrosis Occurring in Persons Who Work under Pressure. A discussion states that aseptic bone necrosis among divers generally occurs as the result of failure to carry out correct decompression after a dive. The lack of good decompression data and the fact that bone lesions may occur without creating immediate symptoms complicates the problem. There is, however, no record of aseptic necrosis occurring from dives to depths of less than 38 FSW. However, divers in apparently good health may show symptoms in X-rays five years after a bad diving experience. Furthermore, there appears to be no relation to past decompression sickness. Treatment of necrosis consists of keeping the patient from putting any weight on the affected joints. If deterioration of the joint has set in surgery and prostheses is required, and this must be repeated after ten years. Because of the possible length of time between exposure and development of symptoms, the cost of this disease to the diving industry and to the insurance companies can be very high. It is recommended that research be directed to measurements of nitrogen and helium elution from body tissues, that mathematical models be abandoned, and that a central registry be set up containing X-rays of all divers and complete records of all diving activities for each person. X-rays patterns.
■ E. P. Kindwall (Saint Luke's Hospital, Milw.), *API Medical Research Report* EA-7104, pp. 125-40 (1972).
Source: API Library

26-60043
The Effect of a Rapid 4% Carboxyhemoglobin Saturation Increase on Maximal Treadmill Exercise. Tests on six physically fit firefighters showed that rapid increase in carboxyhemoglobin resulted in a decrease in the time to exhaustion in exercise tests, although in 16 of the 44 measurements exposure to carbon monoxide resulted in improved performance. The tests involved breathing 20,000 ppm carbon monoxide for 45 seconds followed by 30 ppm for four hours, after which the subjects were exercised to exhaustion on a treadmill. The carbon monoxide exposure also produced an increase in heart rate from 177.7 to 179.3, an increase in the volume of expired air from 127.2 to 131.4 l./min and a decrease in arterial oxygen pressure at 50% oxyhemoglobin saturation from 27.5 to 26.3 mm Hg of mercury. There was no evidence of adaptation and no significant changes in systolic time intervals. Graph, tables, and 22 references. (Supported by the Coordinating Research Council)
■ R. D. Stewart; P. E. Newton; H. V. Forster; C. L. Hake; J. Kaufman; M. H. Keelen; J. P. Klein; D. J. Stewart; A. Wu (Med. Coll. Wisc.), *API Medical Research Publication; Report No. CRC-APRAC-CAPM-22-75 MCOW-ENV* (1979) (43 p.).
Source: API Library

26-60044
Mutagenicity Evaluation of Raw Shale. Final Report. Raw shale showed a clastogenic effect in in-vivo tests involving chromosome aberrations in rat bone marrow cells. No mutagenic effects were found in in-vitro tests: (1) Ames assays using bacteria *Salmonella* and yeasts *Saccharomyces* with or without rat liver tissue activation; (2) mouse lymphoma assays. Tables (supported by the American Petroleum Institute)
■ *API Medical Research Publication* (May 1978) (150 p.).
Source: API Library

26-60045
Effects of Oxides of Nitrogen, Carbon Monoxide and Photochemical Oxidants on the [Electrocardiogram] during Exercise and on Cardiopulmonary Function. Final Report (APRAC Project CAPM-21-74). Exposure of human volunteers to 0.2 ppm ozone for two hours with intermittent light exercise and heat stress showed no adverse effect of ozone on arterial oxygenation or on pulmonary function. Separate blood measurements were made on samples drawn from the brachial artery and on "arterialized" blood collected from the earlobe. Diagram, graphs, tables, and 15 references.
■ W. S. Linn; J. D. Hackney, *API Medical Research Publication* (5/31/78) (50 p.).
Source: API Library

26-60048
"Normal" Carboxyhemoglobin [COHb] Levels of Blood Donors in the U.S. were surveyed by the Medical College of Wisconsin for the Coordinating Research Council Inc. and the U.S. Environmental Protection Agency in 1969-72. COHb analyses of venous blood samples taken from 29,000 adults residing in or near 17 large cities, from 1525 adults in 13 small communities in Vermont and New Hampshire, and from 4 donors voluntarily breathing CO-free air showed that 45% of all

the nonsmoking blood donors had COHb saturations of < 1.5%. Carbon monoxide analyses of alveolar breath samples from every tenth donor were also performed. Smoking was the most important factor responsible for the highest COHb saturations; other factors influencing the COHb saturation were geographical location, occupation, and existing meteorological conditions. None of the large urban communities had CO concentrations below enough to comply with the ambient air quality standards. Graphs, tables, and 15 references.
■ R. D. Stewart; E. D. Baretta; H. C. Dodd; K. K. Donohoo; S. A. Graff; J. H. Kalbfleisch; L. R. Platte; E. B. Stewart (Med. Coll. Wis.), *API Medical Research Publication* #4183 (May 1973) (240 p.).
Source: API Library

26-60049
[In a Two-Year] Investigation of the Effects of Carbon Monoxide on [40] Humans in the Driving Task, under contract to the Coordinating Research Council Inc., and funded by the U.S. Environmental Protection Agency, API, and the Motor Vehicle Manufacturers Association, the effects of 0, 7, 14, and 20% carboxyhemoglobin (COHb) levels on physiological performance, complex psychophysiological and psychomotor skills, and driving skills and judgement on the highway were determined. In general, no ostensible performance degradation was observed with COHb in normal driving tasks although subtle performance losses were observed, especially in information acquisition tests. With heavy information processing demands and/or other associated debilitating factors in driving, such as fatigue or alcohol, these subtle effects could be compounded into gross performance changes. Tables, charts, graphs, data forms, and 69 references.
■ F. W. Weir; T. H. Rockwell (Ohio State Univ.), *API Medical Research Publication* #4190 (Jan. 1973) (176 p.).
Source: API Library

26-60050
Effects of Oil and Chemically Dispersed Oil on Selected Marine Biota...A Laboratory Study. In a study by the Battelle Memorial Institute, Pacific Northwest Laboratories for API, the lethal dose of No. 2 fuel oil and South Louisiana and Kuwait crude oils was determined for 13 fish, 5 crustacea, 3 mollusca, and 2 algae by a flow-through bioassay method; the fuel oil was more toxic than the crudes. Sublethal and chronic effects were studied by observing alkane and methylnaphthalene uptake and depuration by oysters over a 28 day period. Uptake dropped after 2 days of continual exposure, apparently due to biological disturbances such as decreased feeding, but then increased directly with exposure. Depuration was rapid when the oysters entered clean water. The extraction and measurement procedures were devised and validated for C_{12}-C_{20} n-alkanes and methylnaphthalenes in biological tissues. Oysters exposed to Kuwait crude showed no pathological damage. Tables, graphs, and 81 references.
■ *API Medical Research Publication* #4191 (11/1/73) (87 p.).
Source: API Library

26-60051
[In] a Program for Upgrading the NO_2 Instrumentation Employed in the 1972 Chattanooga NO_2 Exposure Study by the Research Triangle Institute for the Coordinating Research Council Inc. chemiluminescent monitoring instrumentation for nitric oxide, nitrous oxide, and nitrogen oxides and automatic data acquisition systems were installed in appropriate shelters at seven of the nine mooring sites in Chattanooga. After checkout, chemiluminescent nitrous oxide field data were obtained for seven months from two sites and for six months at the other five sites during the year Apr. 1972 to Apr. 1973. Tables, flow chart, diagrams, photographs, graph, map, and 16 references.
■ C. E. Decker; T. M. Royal; J. B. Tommerdahl (Res. Triangle Inst.), *API Medical Research Publication* #4202 (1973) (99 p.).
Source: API Library

26-60052
A Survey of Eye Irritation and Lachrymation in Relation to Air Pollution was conducted by Copley International Corp. for the Coordinating Research Council Inc. It concluded that formaldehyde, acrolein, and peroxyacetyl nitrate may contribute to eye irritation but atmospheric levels of them may be too low to account for the observed degree; eye irritants are formed only by photochemical reactions; particulates do not cause eye irritation; atmospheric levels of irritants produce no visible changes in the eyes of humans or rabbits and do not deactivate sulfhydryl enzymes; lysozyme levels in the tears of sensitive individuals exposed to Los Angeles smog are lower than normal because of a subnormal flow of lysozyme tears; no objective method of measuring eye irritation has been devised and correlations with the oxidant level are questionable; individuals vary greatly in their sensitivity to eye irritation; and synthetic smog cannot duplicate the effects of natural smog. Additional research projects are suggested. Graphs, tables, diagrams, and 95 references.
■ K. W. Wilson (Copley Int. Corp.), *API Medical Research Publication* (Apr. 1974) (72 p.).
Source: API Library

26-60053
The Use of Panelists as Substitutes for Taxicab Drivers in Carbon Monoxide Exposure was investigated for the Coordinating Research Council Inc. and the U.S. Environmental Protection Agency by analyses of breath and limited blood samples from 30 pairs of taxi drivers and panelists who drove in New York City traffic for eight hours in two consecutive days. Both panelists and drivers attained similar blood carboxyhemoglobin (COHb) levels. This was true for both smokers and nonsmokers, though smokers had significantly higher COHb concentrations than nonsmokers. There was no consistent difference between the first and second day of driving in the levels of alveolar carbon monoxide. The use of alveolar air to indicate the probable levels of COHb was not a viable assumption since carbon monoxide concentrations in alveolar air did not correlate well with COHb under the conditions of this study. Expanded use of the finger-prick method will probably be more satisfactory. Tables and graphs.
■ A. W. Hoover; R. M. Albrecht (Columbia Univ.), *API Medical Research Publication* #4214 (July 1973) (31 p.).
Source: API Library

26-60054
The Effect of Carbon Monoxide Inhalation on Induced Ventricular Fibrillation in the Cynomolgus Monkey. In a study performed by Thomas Jefferson University for the Coordinating Research Council Inc., normal monkeys and monkeys with acutely induced myocardial infarction were immediately exposed to 100 ppm carbon monoxide for six hours. Blood carboxyhemoglobin at the end of the exposure period ranged from 7.6 to 12.3% with a mean of 9.2%. In noninfarcted air-breathing animals, ventricular fibrillation was induced electrically at 78.5 v; in air-breathing infarcted animals at 50.6 v; in noninfarcted animals exposed to carbon monoxide at 45 v; and in infarcted animals exposed to carbon monoxide at 30 v. Thus, both myocardial infarction and carbon monoxide inhalation enhance the vulnerability of the ventricle to induced fibrillation. The two effects appear to be additive since no significant interaction could be demonstrated. Tables, electrocardiograms, diagram, graph, and 16 references.
■ D. A. DeBias; C. M. Banerjee (Thomas Jefferson Univ.); N. C. Birkhead (U.S.V. Pharmaceutical Corp.), *API Medical Research Publication* #4228 (July 1973) (34 p.).
Source: API Library

26-60055
Laboratory Studies on the Effects of Oil on Marine Organisms: An Overview of studies conducted over a two-year period by Texas A&M University for the American Petroleum Institute showed that in bioassay tests with phytoplankton, crustaceans, and fish, the toxicities of oil-water dispersions and water-soluble fractions from refined oils were significantly greater than those from crude oils. Of the physiological responses studied in relation to the sublethal concentrations of hydrocarbons, respiration seemed most inconsistent, although heartbeat rate of fish embryos and growth rates appeared to be most productive. All species were shown to release accumulated hydrocarbons, including benzo[a]pyrene, when maintained in clean water. In uptake and release studies, the naphthalenes, which were shown to be quite toxic, were accumulated to the greatest extent in all test species and retained the longest, but the process was reversible. Graphs, tables, and 42 references.

■ J. W. Anderson (Tex. A&M Univ.), *API Medical Research Publication* #4249 (11/1/73) (70p).
Source: API Library

26-60057
Ambient Air Quality Standards for Particulates...Review and Evaluation. A review covers the physical and chemical properties of particulates; undesirable effects of particulates in relation to the establishment of air quality standards; particulates and reduction in visibility; soiling or nuisance problems from particulates; and existing ambient air quality standards for particulates. Graph, tables, and 33 references.
■ H. C. McKee (Southwest Res. Inst. - Texas Air Control; Board), *API Air Quality Monograph* 69-1 (Feb. 1969) (28 p.).
Source: API Library

26-60058
Particulates: Air Quality Criteria Based on Health Effects. A review covers the acute effects of the experimental inhalation of particulates, including physiological effects on the airways in terms of increased airway resistance, minor changes in compliance and disturbance of ventilation uniformity; the chronic effects of dusty air, including deposition of suspended matter (black pigment) in the lungs; and gas and particulate interaction, in which adsorption may modify the site of gas absorption in the respiratory system. Diagrams, table, and 30 references. (supported by American Petroleum Institute)
■ M. C. Battigelli (Univ. N.C. Sch. Med.), *API Air Quality Monograph* 69-2 (Feb. 1969) (15 p.).
Source: API Library

26-60060
Oxidants: Air Quality Criteria Based on Health Effects. A review covers smog photochemistry; total oxidant (ozone), including environmental considerations and health effects; nitrogen oxides, including environmental considerations and health effects; hydrocarbons, formaldehyde, and polynuclear hydrocarbons; and criteria for the control of photochemical smog and oxidant concentrations. Graphs, tables, and 51 references. (supported by American Petroleum Institute)
■ I. R. Tabershaw; F. Ottoboni; W. C. Copper (Univ. Calif. Berkeley), *API Air Quality Monograph* 69-5 (Feb. 1969) (36 p.).
Source: API Library

26-60061
A Review of the Toxicology of Lead covers the sources of environmental lead; body storage of lead; biochemical aspects of lead in the body; sources of lead overexposure; pediatric plumbism; adult plumbism; lead alkyl intoxication; and air quality criteria. Tables and 287 references. (supported by American Petroleum Institute)
■ T. J. Haley (Univ. Hawaii Sch. Med.), *API Air Quality Monograph* 69-7 (Feb. 1969) (53 p.).
Source: API Library

26-60062
Air Quality Standards for Carbon Monoxide. A review covers the existing levels of carbon monoxide and the health effects resulting from long-term, low-concentration exposure to carbon monoxide alone or combined with other pollutants, as related to the establishment of air quality standards and methods of measuring atmospheric carbon monoxide concentrations. Graphs, tables, and 20 references. (supported by American Petroleum Institute)
■ R. G. Smith (Wayne State Univ.), *API Air Quality Monograph* 69-9 (Feb. 1969) (20 p.).
Source: API Library

26-60063
Comprehensive Analysis of Shale Oil Products (Revised). Extensive analytical data and measurements are reported for four raw shales, four spent shales, and four shale oils. Data on the shales included Fischer assay, particle size distribution, mineral composition, "EPA Toxic Metals", elemental analysis, trace element analysis, surface area determination, benzene extractables content, electron photomicrographs, and qualitative X-ray data. For the shale oils the report presents hydrocarbon type analysis, trace element content, distillation data, "EPA Toxic Metals", refractive index, density, viscosity, pour point, BS&W, molecular weight, elemental analysis, and asphaltenes/carbenes/carboids/resins/oil fractionation data. The analytical methods used are described in detail. Tables, electron micrographs, and graphs. (Report to the American Petroleum Institute)
■ L. W. Burdett (Union Oil Co. Calif.), *API Report* (9/1/78) (75 p.).
Source: API Library

26-60064
Air Quality Standards for Lead. A review covers the effects of exposure to lead on human health; lead levels in biological samples; atmospheric lead levels; and proposed air quality standards. 51 references. (supported by American Petroleum Institute)
■ R. G. Smith (Wayne State Univ.), *API Air Quality Monograph* 69-11 (Feb. 1969) (15 p.).
Source: API Library

26-60065
Arsenic. A survey covers sources and uses of arsenic; occurrence of arsenic in air, water, soil, food, and drink; methods of analysis; chemistry and biochemistry of arsenic; arsenic toxicity and pharmacology; air quality standards for arsenic; and pertinent literature reviews. Tables and 87 references.
■ B. L. Vallee (Harv. Med. Sch.), *API Air Quality Monograph* 73-18 (1969) (36 p.).
Source: API Library

26-60066
Phosphorus. A review covers the presence of phosphorus in the environment; its production and industrial sources (including oil and petroleum products); methods of analysis; chemistry; nutrition and biochemistry of phosphorus and its derivatives; and toxic effects of phosphorus and its compounds. Tables and 39 references.
■ B. L. Vallee (Harv. Med. Sch.), *API Air Quality Monograph* 73-19 (1973) (39 p.).
Source: API Library

26-60067
Lithium. A survey briefly covers types of lithium minerals, lithium occurrence in water, soils, and plant and animal tissues, with data on lithium concentrations in water in cities that are close to lithium deposits and others that are at a distance, as well as concentrations in the different types of tissues of humans under treatment with lithium; analytical methods and chemical properties of lithium; biochemical effects, physiological effects, and toxicology. Tables and 37 references. (Supported by the American Petroleum Institute)
■ B. L. Vallee (Harv. Med. Sch.), *API Air Quality Monograph* 74-20 (Aug. 1974) (16 p.).
Source: API Library

26-60068
Copper. A survey covers the occurrence of copper in air, soils, rocks, and water, including atmospheric concentration data for many locations in the U.S.A. and concentrations in the waters of 16 U.S. river basins; uses, analytical methods, and chemical properties of copper; presence in biochemical systems such as enzymes and proteins and concentration in dry tissue of various plants; physiology and metabolism; nutritional requirements; copper toxicity and relationship to human disease. Tables and 41 references. (Supported by the American Chemical Institute)
■ B. L. Vallee (Harv. Med. Sch.), *API Air Quality Monograph* 75-21 (1975) (32 p.).
Source: API Library

26-60069
Tin. A survey covers the forms and concentrations of tin in rocks, plants, animals, and the air, including air analysis figures for 1966-67 for 137 sites in the U.S.; methods of analysis; physical and chemical properties; nutritional requirements; and toxicity. Tables and 24 references. (Supported by the American Petroleum Institute)

■ B. L. Vallee (Harv. Med. Sch.), *API Air Quality Monograph* 75-22 (1975) (13 p.).
Source: API Library

26-60070
Gaseous and Particulate Sulfur Compounds in Urban Atmospheres. A review covers the types and concentrations of gaseous and particulate sulfur compounds in the atmosphere, their sources and emission rates, the cycle of supply and removal of sulfur in the atmosphere, measurement of atmospheric sulfur dioxide and sulfate concentrations, relationship to urban weather factors, general characteristics of urban atmosphere and of urban aerosols, a model for urban sulfur aerosol formation, and vehicle emissions of sulfur oxides and sulfate. Graphs, tables, and 70 references. (Supported by the American Petroleum Institute)
■ E. Robinson (Wash. State Univ.), *API Air Quality Monograph* 75-23 (1975) (48 p.).
Source: API Library

26-60071
The Long-Term Effects of Sulfur Dioxide on Ciliary Activity in the Trachea. A research project sponsored by the American Petroleum Institute and carried out at the University of North Carolina was developed to identify the pathogenetic steps in sulfur dioxide injury. The respiratory surface was selected as the most probable site of effect. Rats, hamsters, and mice have been exposed to sulfur dioxide at 1 ppm, simultaneously with 1 mg/cu m of graphite or calcium sulfate dust, for 12 hr daily, 7 days/week, for up to 4 mo. The ciliary activity, the microflora of the respiratory surfaces, the weight of the whole animal body, the weight of the major parenchymal organs, the hematocrit,and the relative histology have been used as the indexes of injury. All indicators concurred to minimize significant pathological effect on the respiratory surfaces; apparently something different and more complex than the effect of sulfur dioxide on the surface of the respiratory apparatus is responsible for the casualties from air pollution exposure. Graphs, diagrams, tables, and 11 references.
■ M. C. Battigelli; D. A. Fraser (Univ. N.C.), *API Medical Research Report* EA7101 (1971) (72 p.).
Source: API Library

26-60072
Uptake and Depuration of Specific Hydrocarbons from Oil by the Bi-valves Rangia cuneata and Crassostrea Virginica. Laboratory studies were done on groups of clams (i.e., *Rangia*) and oysters (i.e., *Crassostrea*) by exposing them to oil-water emulsions of four test oils for 1-7 days. Some of the oysters were allowed to depurate for up to 52 days. South Louisiana crude oil, Kuwait crude oil, No. 2 fuel oil, and Venezuelan bunker C oil were used as the test oils. Detailed analyses were made of specific hydrocarbons present in the tissues of the animals. The levels of hydrocarbons accumulated in the tissues were related to the concentration of water-soluble fractions plus the amount of droplet dispersion. Both aromatic and saturated hydrocarbons are released from the tissues relatively rapidly, such that maintenance in clean water for periods of 24-52 days is sufficient to cleanse the tissues of detectable levels of hydrocarbons. Graph and tables.
■ J. W. Anderson, *U.S. National Technical Information Service* AD-783 990, pp. 690-708 (May 1973).
Source: Not available from API

26-60073
[A Review on] Hematotoxicity in Humans covers pancytopenia, leukemia, and other hematological diseases that appear to be associated with exposure to benzene; clinical manifestations and outcome; cellular effects of benzene in the blood; incidence of benzene-induced leukemia and studies on the relationship of leukemia to benzene exposure; case reports of other blood diseases apparently induced by benzene; individual factors in susceptibility to benzene, such as heredity, sex, age, and status of the hematopoietic system; and early detection of toxicity. Extensive tables summarizing individual reports of various types of benzene-related hematotoxicity are presented. 100 references. (Supported by the American Petroleum Institute)
■ B. D. Goldstein (N.Y. Univ.), *Journal of Toxicology and Environmental Health Supplement* 2: 69-105 (1977).
Source: Not available from API

26-60074
Benzene Toxicity. A Critical Evaluation is presented based on a review of world literature, including chapters on analytical techniques, benzene metabolism, the molecular mechanism of the toxicity, experimental intoxication, cytologic and cytogenic effects, and human hematotoxicity in separate chapter. 1003 references (Supported by the American Petroleum Institute)
■ S. Laskin B. D. Goldstein, *Journal of Toxicology and Environmental Health Supplement* 2 (1977) (147 p.).
Source: Not available from API

26-60075
Analytical Techniques [for Benzene] are reviewed, including methods for industrial gases, organic solvents, air, water, and biological systems. The best methods are nonvisible spectrophotometry when there are few interfering compounds,and gas chromatography in complex mixtures and biological systems. Tables and 271 references (Supported by the American Petroleum Institute)
■ C. A. Snyder (N.Y. Univ.), *Journal of Toxicology and Environmental Health Supplement* 2:5-22 (text), 107-18 (refs.) (1977).
Source: Not available from API

26-60076
[A Review of] Benzene Metabolism covering in-vivo and in-vitro studies including metabolism in humans and bacterial systems, concludes that the pathways are similar in humans and all animal species. The major differences arise from the conjugation of the final metabolites, sulfonation being the predominant form in human and glucuronide formation occurring only when the sulfonation route is heavily utilized. Chronic toxicity is related to the levels of metabolites present. Treatments that increase elimination of the metabolites and thus, decrease toxicity. Decreasing the rate of metabolism of benzene lowers the level of metabolites in some cases and sometimes decreases the chronic toxicity. Treatments do not, however, alter the acute toxicity of benzene. 90 references. (Supported by the American Petroleum Institute)
■ G. M. Rusch; B. K. J. Leong; S. Laskin (N.Y. Univ.), *Journal of Toxicology and Environmental Health Supplement* 2:23-26 (1977).
Source: Not available from API

26-60077
[A Review on] Experimental Benzene Intoxication covers acute, subacute, and chronic toxicity induced by inhalation; intoxication by parenteral and oral administration; the sequence of hemopoietic changes; the immunological effects; attempts to induce leukemia with benzene; embryonic and teratogenic effects; and biochemical and histochemical effects. Tables and 65 references. (Supported by the American Petroleum Institute)
■ B. K. J. Leong (N.Y. Univ.), *Journal of Toxicology and Environmental Health Supplement* 2:45-61 (1977).
Source: Not available from API

26-60078
The Molecular Site of Benzene Toxicity. A review of the literature on the molecular mechanism of benzene toxicity indicates that the action is probably not in DNA and RNA synthesis but in the process of protein synthesis. Evidence suggests that the primary toxic effect is inhibition of heme synthesis occurring at or before ALA (aminolevulinic acid) synthetase. Studies in vitro with rabbit reticulocytes suggest that there may be important relationships among heme synthesis, cyclic AMP, and pyridoxine metabolism. 35 references. (Supported by the American Petroleum Institute)
■ M. L. Freedman (N.Y. Univ.), *Journal of Toxicology and Environmental Health Supplement* 2:37-43 (1977).
Source: Not available from API

26-60079
Effects of Oxidant Levels on Selected Health Characteristics of Persons in the Los Angeles Basin. First Annual Report. Volume 1.

Data Collection. In an attempt to relate respiratory illness to air pollution, data on chronic and acute respiratory disease and asthma symptoms have been collected by means of questionnaires and telephone interviews in three communities in the Los Angles area. In addition, pulmonary function has been evaluated by measuring forced expiratory volume within 0.75 second in more than 5000 elementary school children in the three communities (90% of the school population). Details are presented on the populations surveyed, the questions asked, and the techniques used by the University of California at Riverside to monitor air quality in these communities. Photographs, graphs, and tables. (Report prepared for Coordinating Research Council and Environmental Protection Agency)
■ *API Publication* #4229 (4/1/73) (44 p.).
Source: API Library

26-60080
Studies on the Toxicity of Petroleum Waxes. Chemical and animal studies showed no carcinogenic hazard from petroleum waxes used for food packaging. Five waxes fed to rats at a 10% level in the diet over two years were harmless. Subcutaneous implantation of five waxes as discs and the aromatic and nonaromatic fractions of one wax as powders caused fibrosarcomas, which were ascribed to the physical rather than chemical properties of the waxes. Repeated skin application of benzene solutions of wax and aromatic and nonaromatic fractions had no carcinogenic effect on mice or rabbits. Dibenz[a,h]anthracene added to wax was extensively eluted in the gut of rats and mice but only 6-20% passed into the body and none of it was present as free hydrocarbon. Polycyclic aromatic hydrocarbons added to wax are eluted by both the fat and protein in milk, but very little of the eluted compound remains as free hydrocarbon. Elution is much greater from paraffin than from microcrystalline wax. Analysis of 36 wax samples showed the presence of benz[a]anthracene, benzo[e]pyrene, chrysene, fluoranthene, pyrene, or triphenylene in eight instances, at a maximum concentration of 0.64 ppm. (Supported by the American Petroleum Institute)
■ P. Shubik; U. Safiotti; R. Feldman; W. Lijinsky; A. Pietra; C. R. Raha; H. Ramahi; H. Rappaport; L. Tomatis; B. Toth (Chic. Mech. Sch.), *Toxicology and Applied Pharmacology Supplement* 4:1-59 (Nov. 1962).
Source: Not available from API

26-60082
Petroleum Hydrocarbon Toxicity Studies--1. Methodology. Techniques and equipment to be used in studies of the inhalation toxicity of a series of petroleum hydrocarbons are described, including procedures for acute inhalation toxicity of rats, subacute inhalation toxicity of rats and dogs, mouse upper respiratory tract irritation, and human sensory response procedures; equipment and techniques for supplying vapor-air mixtures to the test animals; sources, feed, and husbandry of the rats and dogs; analytical chemistry techniques; and procedures to determine health effects such as hematology and histological methods. Diagrams, graphs, tables, and 15 references. (Supported by the American Petroleum Institute.)
■ C. P. Carpenter; E. R. Kinkead; D. L. Geary; J. M. King; L. J. Sullivan (Carnegie-Mellon Inst. Res.), *Toxicology and Applied Pharmacology* 32(2):246-66 (May 1975).
Source: Not available from API

26-60083
Petroleum Hydrocarbon Toxicity Studies--5. [Studies of] Animal and Human Response to Vapors of Mixed Xylenes indicated a relatively low toxicity and suggested a hygienic standard of 110 ppm. The Lt50 was 92 min for rats inhaling 11,000 ppm, which is nearly saturated air. LC50 for rats in 4-hr inhalation was 6700 ppm. Dogs showed no effect at 530 ppm, lacrimation at 1200 ppm. Cats died within 2 hours on exposure to 9500 ppm. Exposure for 45 minutes to 15,00 ppm caused no increase in erythrocyte fragility in rats. Respiratory rates were depressed by 50 per cent or more in mice exposed for one minute to 12,000 ppm. Rats and dogs showed no ill effects in 65 days of six hour-per-day inhalation of 810 ppm. "Challenge" exposure tests of rats to 6700 ppm showed no protective effect of prior exposure to 810 ppm. A six-person panel determined the odor threshold to be about 1 ppm. Except for eye irritation in four of the six people, there were no common signs of discomfort in 15 minute inhalation of 460 ppm. Tables. (Supported by the American Petroleum Institute)
■ C. P. Carpenter; E. R. Kinkead; D. L. Geary; J. M. King; L. J. Sullivan (Carnegie-Mellon Inst. Res.), *Toxicology and Applied Pharmacology* 33:543-58 (1975).
Source: Not available from API

26-60084
Petroleum Hydrocarbon Toxicity Studies--6. Animal and Human Responses to Vapors of "60 Solvent". A standard of 90 ppm was proposed based on animal response results to "60 Solvent" vapor, even though 350 ppm was well tolerated by humans. Rats were affected at 170 ppm. The LC50 for rats that inhaled vapor for four hours was 4900 ppm, whereas 690 ppm caused no visble response. Similar responses occurred in beagles at 1900 and 820 ppm, respectively. Cats died within four hours at 4100 ppm. The Lt50 for rats was 150 minutes at 7700 ppm. There were no erythrocyte fragility effects in rats exposed to 9200 ppm. Upper respiratory tract irritation in mice was not observed below 1200 ppm. After 65 exposures of six hours per day, the only effects observed in beagles were slight increases in liver and kidney weights at 1200 ppm. In similar tests, rats showed kidney damage even at concentrations as low as 41 ppm. Human volunteers detected odor at 180 ppm. Five of the six volunteers thought that 340 ppm would be tolerable over an eight-hour working day. The "60 Solvent" has an ASTM boiling point of 263-319 °F, and contains 30% paraffins, 19% monocycloparaffins, and 51% alkylbenzenes. (Supported by the American Petroleum Institute)
■ C. P. Carpenter; E. R. Kinkead; D. L. Geary; J. M. King; L. J. Sullivan (Carnegie-Mellon Inst. Res.), *Toxicology and Applied Pharmacology* 34:374-94 (1975).
Source: Not available from API

26-60085
Petroleum Hydrocarbon Toxicity Studies--7. [Tests of] Animal and Human Response to Vapors of "70 Solvent" led to a proposed hygienic standard of 59 ppm. Inhalation of 810 ppm for eight hours caused no deaths in rats, whereas four-hour inhalation of 930 ppm caused some deaths in beagles. There were signs of central nervous system effects in cats that inhaled 370 ppm for six hours. No erythrocyte fragility effects were observed in rats exposed to 780 ppm. The lowest concentration that caused mouse respiratory tract irritation was 280 ppm. Exposure for six hours per day for 65 days caused body weight decreases in rats and in dogs as well as liver weight decreases in dogs. "Challenge" concentrations of 1100 ppm for six hours showed no effects of prior exposure to 70 Solvent in rats. Humans detected odor at between 0.1 and 1.0 ppm, and concentrations of 59 ppm had no serious effects. The "70 Solvent" has an ASTM boiling range of 315-411 °F and contains about 24% paraffins, 12% monocycloparaffins, and 64% alkyl benzenes. Tables. (Supported by the American Petroleum Institute)
■ C. P. Carpenter; E.R. Kinkead; D. L. Geary; J. M. King; L. J. Sullivan (Carnegie-Mellon Inst. Res.), *Toxicology and Applied Pharmacology* 34:395-42 (1975).
Source: Not available from API

26-60086
Petroleum Hydrocarbon Toxicity Studies--8. Animal and Human Response to Vapors of "140° Flash/Aliphatic Solvent". Exposure for eight hours to 43 ppm caused no ill effects in rats. Similarly, there were no ill effects in cats exposed for six hours to saturated vapor or to aerosol. Dogs exposed for eight hours to 33 ppm also showed no ill effects. Neither vapor nor aerosol caused respiratory tract irritation in mice. Exposure for six hours per day for 72 days caused no serious effects in rats or dogs. However, breathing aerosols of 0.5 μm droplets caused three of ten rats to die in the six hours. No erythrocyte fragility changes were observed in rats' blood after seven hours in approximately saturated vapor. Tests with humans gave an odor threshold of 0.6 ppm and no serious discomfort. The 140° Flash Aromatic Solvent has an ASTM boiling range of 363-402 °F, and contains 61% paraffins, 25% monocycloparaffins, and 11% dicycloparaffins. Tables. (Supported by the American Petroleum Institute)
■ C. P. Carpenter; E. R. Kinkead; D. L. Geary; J. M. King; L. J.

Sullivan (Carnegie-Mellon Inst. Res.), *Toxicology and Applied Pharmacology* 34:413-29 (1975).
Source: Not available from API

26-60087
Petroleum Hydrocarbon Toxicity Studies--9. Animal and Human Response to Vapors of "80 Thinner". Rats showed no ill effects in four hours exposure to 810 ppm. The LC50 for rats at four hours was 6200 ppm. Dogs showed no ill effects from four-hour inhalation of 480 ppm. Cats showed central nervous system depression after four-hour inhalation of 5500 ppm but there were no deaths. No change in erythrocytes fragility occurred in the blood of rats after 30 minute inhalation of 15,000 ppm. Exposure to four hours of 7800 ppm killed 14 of 16 rats within four hours. At 1000 ppm, there was 50% respiratory rate depression in three of six mice, no effect at 530 ppm. Rats and dogs that inhaled up to 390 ppm for 70 days, six hours per day, showed no significant pathological changes. Pre-exposure to 80 Thinner had no effect on the response of rats to six hours of 9700 ppm. Work with human volunteers established an odor threshold at 0.9 ppm and indicated that 100 ppm probably would be tolerated by most individuals and is thus, a reasonable hygienic standard. Tables. (Supported by the American Petroleum Institute)
■ C. P. Carpenter; E. R. Kinkead; D. L. Geary; J. M. King; R. C. Myers; D. J. Nachreiner; L. J. Sullivan (Carnegie-Mellon Inst. Res.), *Toxicology and Applied Pharmacology* 36:409-25 (1976).
Source: Not available from API

26-60088
Petroleum Hydrocarbon Toxicity Studies..10. [On the Basis of] Animal and Human Response to Vapors of "50 Thinner". A hygienic standard was suggested at 430 ppm for the thinner, which has an ASTM boiling range of 208-221 °F and is essentially a 2:1 mixture of normal heptane and toluene. Rats showed no ill effects in four hour exposure to 1300 ppm of the solvent. The LC50 for rats was 8300 ppm. Dogs showed no ill effects in six hours inhalation at 600 ppm. Central nervous system effects were observed in cats inhaling 7600 ppm for six hours. There was no effect on the erythrocyte fragility of rats that had been exposed to 25,000 ppm for seven minutes. The Lt50 was 16 minutes at 24,000 ppm. A 50 per cent respiratory rate depression was observed in five of six mice inhaling 11000 ppm. No pathological effects were observed in rats and beagles that inhaled 50 Thinner in concentration up to 600 ppm six hours per day for 65 days, based on blood chemistry, hematological findings, and body, liver, and kidney weight measurements. "Challenge" exposure tests on rats at 18,000 ppm for ninety minutes showed no effects of prior exposure to the hydrocarbon. The odor threshold in humans was 2.5 ppm. Tables and 13 references. (Supported by the American Petroleum Institute)
■ C. P. Carpenter; D. L. Geary; J. M. King; R. C. Myers; D. J. Nachreiner; L. J. Sullivan (Carnegie-Mellon Inst. Res.), *Toxicology and Applied Pharmacology* 36:427-442 (1976).
Source: Not available from API

26-60089
Petroleum Hydrocarbon Toxicity Studies--11. Animal and Human Response to Vapors of Deodorized Kerosene indicates a hygienic standard of 14 ppm, i.e., saturated air at 25 °C. No toxic effects were noted in eight-hour inhalation tests with rats, dogs, or cats in air saturated with deodorized kerosene. There was no upper respiratory tract irritation of mice on inhaling vapor or an aerosol of 6.9 mg/l. In four days of six-hour-per-day inhalation of 7-9.6 mg/l. aerosols of Deodorized Kerosene, rats were unaffected except for irritation of the extremities. There were no toxic effects in 65-day, six-hour-per-day exposure of rats and dogs to 14 ppm concentration as indicated by blood chemistry, hematological findings, and body, liver, and kidney weight. The odor threshold in humans was 0.09 ppm. The kerosene has an ASTM boiling range of 406-522 °F and contains 55.2% paraffins and 40.9% naphthenes by weight. Tables and 13 references. (Supported by the American Petroleum Institute)
■ C. P. Carpenter; E. R. Kinkead; D. L. Geary; J. M. King; R. C. Myers; D. J. Nachreiner; L. J. Sullivan (Carnegie-Mellon Inst. Res.), *Toxicology and Applied Pharmacology* 36:443-56 (1976).
Source: Not available from API

26-60090
Petroleum Hydrocarbon Toxicity Studies--12. Animal and Human Response to Vapors of "40" Thinner indicate a hygienic standard of 25 ppm for the hydrocarbon, which has an ASTM boiling range of 368-447 °F and contain about 35% paraffins, 35% mono- and dicycloparaffins, and about 25% alkylbenzenes. Rats and dogs showed no ill effects in eight-hour breathing of 33-43 ppm vapor or ten-day, six-hour-per day exposure to 31 ppm. There were no lasting effects on rats subjected to eight-hour inhalation of an 8.3 mg/l. aerosol. Exposing rats and dogs to 36 ppm for 65 days had no toxic effects as indicated by hematological findings, blood chemistry, and weight measurements of liver, kidney, and entire body. The odor threshold in humans was 0.17 ppm. (Supported by the American Petroleum Institute)
■ C. P. Carpenter; D. L. Geary; J. M. King; R. C. Myers; D. J. Nachreiner; L. J. Sullivan (Carnegie-Mellon Inst. Res.), *Toxicology and Applied Pharmacology* 36:457-72 (1976).
Source: Not available from API

26-60091
Petroleum Hydrocarbon Toxicity Studies--13. Animal and Human Response to Vapors of Toluene Concentrate. A hygienic standard of 480 ppm was recommended for Toluene Concentrate, which has an ASTM boiling range of 203-231 °F and consists of about 50% toluene, 15% naphthenics, and 35% paraffins. Rats showed no ill effects in four-hour inhalation of 1700 ppm. Dogs inhaled 760 ppm for six hours without ill effects. Central nervous system depression signs were observed in cats inhaling 7800 ppm for six hours. The LC50 for rats was 8800 ppm, and the Lt50 at 45,000 ppm was eleven minutes. Inhaling 20000 ppm for 45 minutes caused no change in osmotic erythrocyte fragility in rats blood. A 50% depression of the respiratory rate was obtained with mice at 8600 ppm. "Challenge" exposure tests on rats breathing 12,000 ppm for six hours showed that prior acclimation to Toluene Concentrate decreased mortality. Exposure of rats and dogs to 980 ppm of Toluene Concentrate or 65 days, six hours per day at 980 ppm produced no ill effects as indicated by hematology, clinical chemistry, micropathology, and body, liver, and kidney weight measurement. The odor threshold for humans was 2.5 ppm. Tables and 15 references. (Supported by the American Petroleum Institute)
■ C. P. Carpenter; D. L. Geary; J. M. King; R. C. Myers; D. J. Nachreiner; L. J. Sullivan (Carnegie-Mellon Inst. Rest.), *Toxicology and Applied Pharmacology* 36:473-90 (1976).
Source: Not available from API

26-60092
Mutagenicity Evaluation of Benzene...Final Report. In a study conducted by Litton Bionetics Inc. for the American Petroleum Institute, benzene was evaluated for genetic activity in a battery of genetic assays employing microbial cells, cultured mammalian cells and rat bone marrow cells (derived from animals treated in vivo). The microbial and cultured mammalian cells were treated under nonactivation and activation conditions (hepatic microsome preparations) so that biologically active metabolites, if produced, could be detected. This series of tests provided a good cross section of genetic end points including both gene mutations and chromosome mutations. The microbial tests and the mouse lymphoma assay showed negative results at all dose levels. A dose-related increase in chromosome aberrations was observed in the subchronic test in rat bone marrow cells. The effects at the higher two dose levels (0.053 and 0.16 ml/rat) were significant and indicate that benzene found in the bone marrow preparations from the benzene-treated rats was chromosome fragments. These probably resulted from chromosome breakage prior to cell division. Tables and graph.
■ *API Medical Research Publication* (July 1977) (134 p.).
Source: API Library

26-60093
A Health Survey of Petroleum Asphalt Workers. A survey was conducted by the American Petroleum Institute of the health of 462 asphalt workers in 25 oil refineries, and of 379 controls. Each worker had been engaged in asphalt work for at least five years, the average

being 15.1 yr. A physical examination, including a detailed medical and occupational history, was done on each subject. No significant differences in health were found between the two groups. Additional information concerning the health of their workers was obtained from paving companies, roofing manufacturers, and interstate truck operators who drive over asphalt highways. The health of these groups also gave no evidence that petroleum asphalt constituted a health hazard. Table and 11 references.
■ C. N. Baylor (Texaco Inc./Cornell Univ. Med. Sch. Bellevue Hosp.); N. K. Weaver (Humble Oil & Ref. Co./Tulane Univ./Univ. Tex.), *Archives of Environmental Health* 17(2):210-214 (Aug. 1968).
Source: Not available from API

26-60094
Trace Metals in Urban Aerosols. A study was conducted by the New York University Medical Center for the American Petroleum Institute and the Edison Electric Institute to characterize suspended and settled dusts in the New York City. Samples were analyzed for Pb, V, Cu, Cd, Cr, Fe, Mm, Ni, and Zn. Pb, V, Cu, Na, and K were use as tracers for dust associated with automotive sources, oil burning, incineration, and natural sources (sea salt and soil). The fractions of the total suspended particulates (TSP) associated with these sources in 1973-74 were: automotive, 30%; oil burning, 12% incineration, 5%; natural, 18%; and unassigned, 35%. The composition of dustfall samples was closely related to the composition of TSP. Current dustfall levels appear to be ~ 100 times lower than those reported in the 1930's. Analysis of human tissues at autopsy produced no indication of pathological lesions related to concentrations of heavy metals. An exponential lung model was used, along with measured values of Pb, Cr, and Ni in the New York City air and in the lungs and lymph nodes of New York City residents, to show that the biological half-times in the lung are: ~ 50 days for Pb; ~ 800 days for Ni; and ~ 1100 days for Cr. Tables, graphs, flow diagrams, maps, diagrams, and 203 references.
■ M. Eisenbud; T. J. Kneip; D. Bernstein; M. T. Kleinman; M. Lippman; R. Riddick (N.Y. Univ. Med. Cent.), *API Medical Research Publication* EA-7601 (10/10/75) (440 p.).
Source: API Library

26-60095
Facts and Opinions on the Role of Sulfur Dioxide in Causing Injury [to Human Health]. A review covers acute effects; chronic effects; mortality; morbidity; the effects of sulfur dioxide pollution on mortality; the effects of sulfur dioxide pollution on morbidity, including upper respiratory diseases, pneumonia, and bronchitis; and the absence of evidence that sulfur dioxide is a factor in health damage at the levels found in ambient air. 81 references. (Supported by the American Petroleum Institute).
■ Mario C. Battigelli, *API Air Quality Monograph* 69-10 (Feb. 1969) (20 p.).
Source: API Library

26-60096
The Chronic Toxicity of Lead. In an experimental study conducted by Hazleton Laboratories Inc. for the American Petroleum Institute to evaluate the effects of the ingestion of small amounts of lead acetate by laboratory animals (rats, dogs, monkeys, and rabbits) over a substantial part of their lifespan, 10 ppm (as lead) produced no detectable effects after 22 mo; thus, 10 ppm lead in the diet is a "no-effect" level. Minimal effects were observed in some animals at 50 ppm, and some histologic changes (principally in the kidney), but no functional changes, were detected in rats fed 100 ppm and 1000 ppm. These amounts of lead administered to the animals are far above the usual intake by man. In addition to the common chronic toxicity tests, the program included studies on reproduction, teratology, behavior, carcinogenicity, and metabolism, as well as special enzyme and electron microscope studies.
■ J. Clifford Jessup, *API Medical Research Report* EA-7102 (Apr. 1971) (9 p.).
Source: API Library

26-60098
Mutagenicity Study of Thirteen Petroleum Fractions. A study involving dominant lethality tests in mice and rats showed no pattern of decreased pregnancy or increased embryo loss, and thus no mutagenicity. VM and P Naphtha, Stoddard Solvent, Rubber Solvent, Mixed Xylenes, 60 Solvent, 70 Solvent, 140 Aliphatic Solvent, 80 Thinner, 50 Thinner, Deodorized Kerosene, High Aromatic Solvent, 40 Thinner, and Toluene Concentrate were injected into male Swiss-Webster white mice and Long-Evans rats. Females that mated with the injected males were subsequently examined for deciduomata (early deaths), late deaths and total implants following removal of the uterus. Corn oil and water were used as negative controls and triethylenephosphoramide as positive control. Most of the materials tested showed little deviation from the results obtained with the negative controls. Where deviations did occur, they were apparently random, unlike the consistent, reproducible differences in the positive control groups. Tables.
■ *API Medical Research Publication* (1973) (28 p.).
Source: API Library

26-60099
Rat Bone Marrow Cytogenetic Analysis [for] Unleaded Gasoline...Final Report. Male rats injected intraperitoneally with unleaded gasoline at low, intermediate, and high acute and subchronic dose levels were injected with colchicine 2 hr prior to sacrificing to arrest diving cells in the metaphase, and bone marrow cells from the femurs and tibias were subsequently examined for chromosomal aberrations. Acute dosages were 0.15 ml of a 1:5 dilution of the gasoline in acetone as the low dose; 0.1 ml of undiluted gasoline as intermediate; and 0.3 ml as high. Sets of test animals and controls were killed 6, 24, and 48 hr after dosing. In the subchronic studies, the rats were dosed once each day for five days, and killed 6 hr after the final dose. The dose levels were 0.06 ml of a 1:5 dilution of the gasoline in acetone; 0.19 ml of the same solution; and 0.13 ml of undiluted gasoline. The occurrence of chromosomal aberrations was within the normal background range, the highest being 1.3% at the 48 hr acute high dose.
■ *API Medical Research Publication* (Nov. 1977) (110 p.).
Source: API Library

26-60100
Further Studies of the Mutagenicity of Hydrocarbon Fractions [by] Utilizing Somatic Mutations in Rats. The cytogenetic study carried out by Hines Inc. for the American Petroleum Institute, involving measurement of chromosomal aberrations in the bone marrow and blood of rats exposed to three petroleum fractions, confirmed the previously observed mutagenicity of 60 Solvent and Rubber Solvent, and showed that a High-Aromatic Solvent was a less active mutagen than the other two solvents. The solvents also caused a deep depression revealed by temporary weight losses of the rats. In the tests on a single group of 50 male Long-Evans rats, the three solvents diluted in corn oil were injected intraperitoneally in three doses spaced three days apart, and the chromosome analyses were made on blood and bone marrow specimens at 1, 7, and 30 days after the treatment. Negative control rats received three corn oil/water injections on the same schedule, and positive control animals received a single dose of triethylenemelamine at the time of the third injections for the other two groups. Tables.
■ *API Medical Research Publication* (1978) (51 p.).
Source: API Library

26-60102
Mutagenicity Evaluation of Diesel Fuel. In tests carried out for the American Petroleum Institute, an unspecified diesel fuel was not mutagenic for Ames Salmonella tester strains in plate tests or in the Mouse Lymphoma Assay that measures forward mutation at the thymidine kinase locus, but it was clastogenic in the Rat Bone Marrow Cytogenic analysis, and thus should be considered a clastogen for animals. The predominant aberration was nonheritable chromosomal fragmentation; few arrangement types of aberration were observed. Extensive tabulations detail the test conditions and specific results obtained.
■ *API Medical Research Publication* (Jan. 1978) (148 p.).
Source: API Library

26-60103
Estimation of the Mutagenicity of Hydrocarbon Fractions Utilizing the Bacterial Assay Procedure with Mammalian Tissue Metabolic Activation. API Project PS-4 #6. Varnish makers' and painters' naphtha, Stoddard solvent, rubber solvent, 140 flash aliphatic solvent, 60 and 70 solvents, 50 and 80 thinners, mixed xylenes, deodorized kerosine, and high-solvency naphtha were tested for mutagenicity by the Ames in-vitro test, which utilizes two specially sensitive mutant strains of *Salmonella typhimurium* bacteria; the tests were carried out in the presence and absence of a microsomal liver extract from male rats. For all the solvents tested, the number of colonies reverting (mutating) to histidine independence was comparable with that in the negative control tests, run in the presence of dimethyl sulfoxide, indicating no mutagenic activity; several of the solvents had to be diluted 1:10 with dimethyl sulfoxide due to their toxicity to the bacteria. Tables.
■ *API Medical Research Report* (1978) (8 p.).
Source: API Library

26-60104
A Morbidity Study of Petroleum Refinery Workers at six refineries in 1973 was carried out by Tabershaw/Cooper Associates, Inc. by examination of medical absenteeism records, with the overall results for all refineries corrected for the difference in worker age distribution between the different refineries. The six refineries employed a total of 4777 men and had a total of 209 absences/yr/100 men, of which 163.2 absences were < 5 days and 21.2 days of absences > 5 days. The average frequency rate (ratio of number of illnesses to number of workers x 100) was 45.8 for all refineries; average frequency rates by illness were 15.3 for respiratory and 4.1 for digestive ailments, 4.1 for fractures, sprains, dislocations, etc., 0.5 for malignant neoplasms and lymphomas, and 0.4 for benign and unspecified neoplasms. Average severity rates (ratio of days lost to number of workers x 100) were 970.8 total for all refineries, 149.4 for circulatory, 141.9 for respiratory, and 104.4 for digestive ailments, and 17.4 for malignant neoplasms. Racve (white vs. non-white) did not appear to affect occurrence or duration of illnesses. Methods of obtaining the data and its validity are discussed. (Supported by the American Petroleum Institute)
■ *API Medical Research Publication* (4/28/75) (34 p.).
Source: API Library

26-60105
The Effects of Chronic Exposure to Low Levels of Carbon Monoxide on the Cardiovascular System of Dogs--1. Exposure to 100 ppm Carbon Monoxide. A study was undertaken at Jefferson Medical College with support from the Coordinating Research Council Inc. to obtain quantitative correlations between chronic exposure to low-level carbon monoxide (100 ppm CO, 23 hr/day for 14 weeks) and the physiologic parameters of the cardiovascular system and blood of normal dogs and of dogs with induced myocardial infraction and to determine the relationships between carbon monoxide exposure and the pathology of various body organs (heart, lungs, liver, kidneys, adrenals, and brain). No obvious signs were noted which could be interpreted as CO-induced. Throughout their exposure to CO, the animals were in clinically good health, and there were no characteristic alterations of the serum enzymes or the electrocardiogram that could be attributed to CO exposure. Hematologic parameters did not appear to change significantly during exposure. Carboxyhemoglobin averaged 14% at 100 ppm CO exposure. Diagrams, tables, graphs, photographs, and 48 references.
■ *API Medical Research Publication* #4096 (Oct. 1970) (81 p.).
Source: API Library

26-60106
Predicting The Carboxyhemoglobin [(COHb)] Levels Resulting from Carbon Monoxide [(CO)] Exposures. Data from 22 humans exposed to 50-200 ppm CO for 0.33-5.25 hr were fitted to the Coburn, Forster, Kane (CFK) equation for predicting COHb levels as a function of experiment duration and CO concentration, exercise level (sedentary to 300 kp-m/min), and sex. The equation satisfactorily predicted the COHb levels for both men and women at steady concentration levels of 50-200 ppm, even though the female subjects absorbed CO more rapidly than the male subjects; it also accurately summed the results of discontinuous exposures to CO. Thus, the CFK equation is a good theoretical model for the uptake and excretion of CO. A rational, efficient procedure for solving the equation by trial and error is outlined; a graph relating COHb saturation to exposure duration and concentration, prepared by solving the CFK equation, is presented; and the effects of several variables on the rate of CO uptake and equilibrium COHb levels are discussed. Tables, graphs, and 18 references.
■ J. E. Peterson; R. D. Stewart (Med. Coll. Wis.), *API Medical Research Publication* #4189 (June 1973) (46 p.).
Source: API Library

26-60107
Fluorosis of Livestock. A review covers the factors affecting the occurrence of fluorosis, including species other than cattle; diagnosing fluorosis, including the clinical aspects, acute fluorine toxicosis, and chronic fluorosis; the effects of ingesting up to 93 ppm of fluorine for up to 7.5 yr on the general condition of cattle, including the hair and skin, hoofs, soft tissues, blood, placental transfer, reproduction, and milk production; and methods of alleviating fluorine toxicity (e.g., with chemical food additives that act as inhibitors). Graphs, tables, photographs, and 30 references. (Supported by the American Petroleum Institute)
■ J. L. Shupe (Utah State Univ.), *API Air Quality Monograph* 69-4 (Feb. 1969) (29 p.).
Source: API Library

26-60108
Photochemical Smog. A review covers the nitrogen oxides, including atmospheric analyses, automatic monitoring in Los Angeles and Menlo Park, Calif., reactions with olefins, measuring total nitrogen compounds, and the phytotoxicity of nitrogen dioxide; oxidants, including the determination of ozone and oxidants in the atmosphere, damage to plants, and adverse effects on citrus trees; organic pollutants (especially polynuclear aromatic hydrocarbons), including detectors, urban pollution, Los Angeles smog, and eye irritation; and various national and state air quality standards. Tables and 91 references. (Supported by the American Petroleum Institute)
■ M. D. Thomas (Am. Public Health Assoc.), *API Air Quality Monograph* 69-6 (Feb. 1969) (40 p.).
Source: API Library

26-60110
Barium. A survey prepared for the American Petroleum Institute covers the geochemistry of barium in mineral deposits, in water, and in air; the presence of barium in marine organisms, in land plants and animals, and in mammals, especially man; human exposures to barium,, with data on geographic variation, barium in the lungs, and barium accumulation in human tissues with age; atomic structure, biochemical functions, and pharmacology of barium; barium metabolism; the toxicity of barium salts especially those used as pesticides and as smoke suppressants in diesel fuel; industrial exposures to barium; and air quality standards for barium. Tables and 20 references.
■ H. A. Schroeder (Dartmouth Med. Sch.), *API Air Quality Monograph* 70-12 (Oct. 1970) (25 p.).
Source: API Library

26-60111
Vanadium. A review compiled for the American Petroleum Institute covers the geochemistry of vanadium and niobium in the geosphere, in the atmosphere, and in the hydrosphere; vanadium and niobium in the biosphere; human exposures to vanadium and niobium, principally in food, and their accumulation with age; atomic structure, biochemical functions, and pharmacology; vanadium metabolism; toxicity of vanadium; the possibility that vanadium has a biological role in mammals essential for optimum function; and the tolerable limits of vanadium in air. Tables and 65 references.
■ H. A. Schroeder (Dartmouth Med. Sch.), *API Air Quality Monograph* 70-13 (Oct. 1970) (32 p.).
Source: API Library

26-60112
Nickel. A review prepared for the American Petroleum Institute covers the geochemistry of nickel; nickel in plants, animals, and man and its geographic variation; human exposures to nickel; atomic structure, biochemistry and pharmacology of nickel; nickel metabolism; nickel toxicity, including dermatitis, respiratory diseases, and cancer; industrial exposures; and proposed air quality standards. Tables and 39 references.
■ H. A. Schroeder (Dartmouth Med. Sch.), *API Air Quality Monograph* 70-14 (Oct. 1970) (24 p.).
Source: API Library

26-60113
Chromium. A review prepared for the American Petroleum Institute covers the geochemistry of chromium, molybdenum, and tungsten; chromium and molybdenum in the air, including the sources and particle sizes; chromium and molybdenum in living things, including plants, animals, and humans, and changes with age; chromium in human lungs; human exposures to chromium and molybdenum in food; the atomic structure and biological functions of chromium; chromium metabolism; pharmacology and toxicity of chromium, especially the hexavalent form; industrial exposures of workers to chromium and the concomitant health effects; and air quality standards for chromium. Tables and 38 references.
■ H. A. Schroeder (Dartmouth Med. Sch.), *API Air Quality Monograph* 70-15 (Oct. 1970) (28 p.).
Source: API Library

26-60114
Manganese. A review prepared for the American Petroleum Institute covers the geochemistry of manganese; manganese in plants and animals; human exposure to manganese in air, water, and food; atomic structure and biochemical functions of manganese; manganese and mammalian metabolism, homeostasis and accumulation from the environment; pharmacology and toxicology; and air quality standards for manganese. Tables and 44 references.
■ H. A. Schroeder (Dartmouth Med. Sch.), *API Air Quality Monograph* 70-17 (July 1971) (34 p.).
Source: API Library

26-60115
Cadmium, Zinc, and Mercury. A review prepared for the American Petroleum Institute covers the geochemistry of zinc, cadmium, and mercury; sources and effects of zinc, cadmium, and mercury in the air; their effects on plants, marine organisms, mammals, especially man, and their concentrations in man as a function of age; cadmium and zinc in human lungs; human exposures to zinc, cadmium, and mercury; their atomic structures; their role in human metabolism; human requirements for zinc; the pharmacology and toxicity of all three metals; zinc, cadmium, and chronic diseases, and the air pollution aspects of zinc, cadmium, and mercury, including tolerable levels. Tables, graphs, and 70 references.
■ H. A. Schroeder (Dartmouth Med. Sch.), *API Air Quality Monograph* 70-61 (July 1971) (40 p.).
Source: API Library

26-60116
Microparticulate Sulfates: Effects on Human Health. A review conducted by the American Petroleum Institute show that sulfuric acid mist appears to be more irritating than sulfur dioxide for equivalent sulfur concentrations. The irritating quality of the sulfates, for equivalent sulfur concentrations, varies depending on their particle size, composition, and water solubility. An interaction between sulfur dioxide and ozone or hydrogen peroxide exists and probably results in the formation of sulfuric acid. From the epidemiologic studies, it has been difficult to separate the effects of sulfur dioxide, sulfates, and particulates and their interactions. Some, but not all, of the studies showed that the sulfate component is the major irritative component. Since not all sulfates are equally irritating, if a sulfate level is proposed, it should be related to a specific sulfate. The identification of the sulfate levels that are acceptable, or the levels above which interference with health occurs, are not clearly defined. Table and 58 references.
■ B. G. Ferris (Harv. Univ.), *API Air Quality Monograph* 75-24 (1975) (27 p.).
Source: API Library

26-60117
Experiments in Human Work Capabilities under Pressure, Now Being Conducted at the Institute for Environmental Medicine, University of Pennsylvania, indicate that diving operations at depths well below 2000 ft of sea water are feasible with properly chosen breathing mixtures. In experiments in the pressure chamber complex at the University of Pennsylvania four men accomplished all tasks satisfactorily while breathing mixtures containing helium at pressures equivalent to 1200 ft of sea water and neon at 700 and 900 ft (equivalent to helium at 2000 and 4000 ft). With neon at 1200 ft, the subjects stopped exercising after ~ 27 min of a 30 min schedule. When breathing nitrogen at the equivalent of air at 500 ft of sea water depth, the subjects suffered impaired coordination and mental functioning as well as pulmonary stress. The experimental results have permitted plotting curves of pulmonary resistance and peak pulmonary ventilatory flow vs. for nitrogen, neon, and helium. These curves should permit prescribing optimal mixtures of inert gases for breathing under pressure. Problems encountered in the experiment were mainly skin itching and vestibular function derangement. No evidence of hypoxia was found in the experiment, in which oxygen concentration was always exactly equivalent to that of sea-level air. Decompression was smooth and without incident. At the highest pressure, 1200 ft, three of the four subjects developed vertigo and nausea indicative of vestibular or acoustic nerve disease, probably due to a viral infection transmitted from one subject to another. The symptoms cleared spontaneously while the subjects remained under high pressure. The experiments demonstrated that neither hydrostatic pressure nor a helium breathing mixture was physiologically or mentally limiting. Changes in pulmonary function generated by sharp rises in gas density were not severely limiting when the subject performed moderate work in a neon breathing atmosphere equivalent to helium at 5000 ft of sea water.
■ W. B. Wright (Univ. Pa. Inst. Environ. Med.), *API Medical Research Report* EA-7104, pp. 43-53 (1972).
Source: API Library

26-60118
Oils Mist: [An] Evaluation of [Workplace] Sampling Procedures. Sampling procedures sponsored by the American Petroleum Institute Subcommittee on Industrial Hygiene showed that collection on Whatman #41 paper, BM-2133, or on a glass fiber filter in a filter holder is suitable, with appropriate modifications in the presence of metal chips and dust, for oil mists without vapor; a better sample is obtained if the employee wears a personal sampler; a two-stage sampler will separate respirable from nonrespirable mist; electrostatic precipitation is a very good method, a silica gel, activated charcoal, or impinger method is suitable for determining mist plus vapor; and that the threshold limit value of 5 mg/cu m does not distinguish between respirable and nonrespirable mists. 12 references.
■ *API Med. Res.* EA-7201 (May 1972) (5 p.).
Source: API Library

26-60119
Guidelines on Noise include definition of measurement units; criteria for allowable noise levels for preventing hearing loss, based on OSHA standards, local ordinances, ambient noise levels in the community, and prevention of interference with speech; instruments for measuring sound levels, including portable meters to determine the frequency distribution, microphones, acoustic calibrators, vibration pickups and personnel dosimeters; errors due to temperature, humidity, wind, and electromagnetic fields; choice of instruments based on the type of noise, i.e., steady, broadband, fluctuating, intermittent, or impulsive; spot measurements and determination of isopleths; data recording; basic acoustical concepts, including sound pressure, power, directivity, and absorption and transmission loss; sound attenuation through dense woods, hills, buildings, etc.; estimating community noise levels for new plants; predicting in-plant noise levels from multiple sources of steady noise; noise reduction through proper plant layout and equipment specification; general noise reduction, including absorbent materials for enclosures,

mufflers and in-line silencers for piping noise, acoustical lagging, and damping of vibrations; and noise control techniques for furnaces, electric motors, control valves, piping, heat exchanger, fans in cooling towers, compressors, gears, flares, and gas turbines. Suppliers of noise- and vibration-control systems and materials are tabulated. Graphs, diagrams, tables, and 120 references.
■ *API Medical Research Report* EA-7301 (1973) (111 p.).
Source: API Library

26-60120
A Mortality Study of Petroleum Refinery Workers. A study of all the hourly, nonclerical, male workers employed for at least one year between 1/1/62 and 12/31/71 in 17 U.S. refineries, chosen to give a representative sample of size, geographic area, and ownership, was carried out under API sponsorship by tracing 94% of the 20,163 workers involved and obtaining the death certificates for 1145 of the 1165 deceased. Most of the men had been employed for at least ten years. The Standardized Mortality Ratio (SMR) was 69.1, compared with the general male U.S. SMR of 100. Mortality from cardiovascular-renal diseases, digestive cancer, and stomach or duodenal ulcers were higher for men with the least exposure to hydrocarbons. Mortality from respiratory and genital cancer increased with increasing exposure. Mortality from lymphomas was greater than expected, though not statistically significant. Extensive data are tabulated.
■ *API Medical Research Report* EA-7402 (9/15/74) (47 p.).
Source: API Library

26-60121
Cytologic and Cytogenetic Effects of Benzene [on Human Health]. A review of the effects of benzene on the individual cell as found in published experimental studies on various animals, chromosome studies in exposed humans, and studies on industrially exposed workers indicates that the last group furnishes the most reliable and consistent data with control group comparisons and better documented exposure conditions. However, the reported effects are both variable and contradictory, and it is impossible to distinguish carcinogenic from toxic effects. Clarification of the mode of action of benzene on the cell nucleus will require extensive and well-designed animal and in-vitro studies. This work was done under contract of the American Petroleum Institute with the New York University of Environmental Medicine.
■ S. R. Wolman (N.Y. Univ.), *Journal of Toxicology and Environmental Health Supplement* 2:63-68 (1977).
Source: Not available from API

26-60122
Skin Tumorigenesis in Mice by Petroleum Asphalts and Coal Tar Pitches of Known Polynuclear Aromatic Hydrocarbon Content. In studies partly supported by the American Petroleum Institute and partly by the National Cancer Institute, benzene solutions of eight petroleum asphalts and two coal-tar pitches of known polynuclear aromatic hydrocarbon (PAH) content were applied topically to Swiss mice. Epidermal carcinomas and papillomas were observed in over 90% of coal-tar pitch treated animals, i.e., there were 53 tumor-bearing animals out of 58 autopsied. Only one carcinoma and five papillomatous growths were observed in 218 mice treated with asphalts. The PAH content of the coal tar pitches were several orders of magnitude greater than that of the asphalts examined. For example, 10,000 ppm benzo[a]pyrene was found in the coal-tar pitches. An exceptionally high value of 27 ppm was found in one of the asphalts, whereas the other seven had levels of 0.1-2.5 ppm. However, because of the low tumor incidence in the asphalt-treated mice, no conclusions could be drawn as to a possible relationship between the PAH content of the asphalts and their tumorigenic properties. Spectrum, tables, photomicrographs, and 14 references.
■ L. Wallcave; H. Garcia; S. R. Wolman; R. Feldman; W. Lyinsky; P. Shubik (Univ. Neb. Coll. Med.), *Toxicology and Applied Pharmacology* 18(1):41-52 (Jan. 1971).
Source: Not available from API

26-60123
Petroleum hydrocarbon Toxicity Studies--2. Animal and Human Response to Vapors of Varnish Makers and Painters Naphtha. The suggested hygienic standard for inhalation of Varnish Makers and Painters Naphtha (VM&P naphtha) for man is 430 ppm. Inhalation of saturated vapor for more than a few minutes constitutes a hazard of life. Rats tolerated saturated vapor at room temperature for 7.5 min. The single 4-hr inhalation LC50 for rats was 3400 ppm, and the highest concentration producing no discomfort was 940 ppm. Rats that survived the 65-day inhalation schedule for 1200, 600, and 280 ppm were challenged with 5800 ppm for 6 hr. All treated rats were more resistant in terms of mortality than were controls. Cats responded with signs indicative to severe central nervous system effect when subjected to 4100 ppm for 4 hr but all cats survived. There were no dosage-related differences between the controls and rats and beagles that inhaled 2.8 mg/l. or 1.3 mg/l. for 6 hr daily, 5 days each week for 13 wk. The odor threshold for VM&P naphtha in sniff tests was 0.86 ppm. During a 15-min inhalation period, four of seven subjects had upper respiratory tract irritation at 880 ppm. (Supported by the American Petroleum Institute)
■ D. L. Geary; E. R. Kinkead; C. P. Carpenter; J. M. King; L. J. Sullivan (Carnegie-Mellon Inst. Res.), *Toxicology and Applied Pharmacology* 32(2):263-81 (May 1975).
Source: Not available from API

26-60124
Petroleum Hydrocarbon Toxicity Studies--3. Animal and Human Response to Vapors of Stoddard Solvent. The suggested hygienic standard for inhalation of Stoddard Solvent for man is 200 ppm. Inhalation of 1400 ppm (substantial saturation at 25 °C) caused the death of 1 of 15 rats at the termination of 8 hr. Beagle dogs had clonic spasms in 5 hr and cats died between 2.5 and 7.5 hr. There were no differences between the controls and groups of beagle dogs that inhaled 330 ppm, 190 ppm and 84 ppm 6 hr daily, 5 days/wk for 13 wk. However, rats exposed to 330 ppm for 65 days exhibited slight pathological changes in the kidney. The odor threshold in a sniff test is below 0.9 ppm. In a 15-min inhalation period, only slight eye irritation was reported by one of six persons at 150 ppm. (Supported by the American Petroleum Institute)
■ E. R. Kinkead; D. L. Geary; C. P. Carpenter; J. M. King; L. J. Sullivan (Carnegie-Mellon Inst. Res.), *Toxicology and Applied Pharmacology* 32(2):282-97 (May 1975).
Source: Not available from API

26-60125
Petroleum Hydrocarbon Toxicity Studies--4. Animal and Human Response to Vapors of Rubber Solvent. The suggested hygienic standard for inhalation of Rubber Solvent for man is 430 ppm. The LC50 for rats by 4 hr inhalation is 15,000 ppm. A concentration 2800 ppm caused no discomfort in rats in 4 hr while for dogs, the corresponding concentration was 1500 ppm. Cats exhibited central nervous system depression after a 4-hr inhalation of 12,000 ppm but survived. At the highest concentration obtainable, 45,000 ppm, the LC50 for rats was 4.3 min. Rats and dogs inhaled measured concentrations of up to 2000 ppm for 6 hr/day, 5 days/wk for 13 wk. The rats tolerated the highest level. In the dogs, the dosage related granulation nodules found in the lungs may have been somewhat exacerbated by the solvent vapor, but there was no similar lung pathology in rats that inhaled the same vapor. The odor of the solvent is detectable at 10 ppm. (Supported by the American Petroleum Institute)
■ D. L. Geary; L. J. Sullivan; C. P. Carpenter (Carnegie-Mellon Inst. Res.), *Toxicology and Applied Pharmacology* 33:526-28 (1975).
Source: Not available from API

27-00325
In-Vitro and In-Vivo Mutagenicity Studies. New Oil Composite [Motor Oil]. Final Report. "New Oil Composite" (motor oil) showed no mutagenic potential in the Ames test, the mouse lymphoma assay, or the in-vivo bone marrow test. The Ames test used selected strains of *Salmonella typhimurium* bacteria. In the lymphoma test, mouse lymphoma cells were exposed to the oil with and without metabolic activation. The in-vivo test measured the extent of abnormality in the chromosomes of the bone marrow cells of rats. Photomicrographs and tables.
■ *API Medical Research Publication* (8/17/79) (71 p.).
Source: API Library

27-30051
In-Vitro and In-Vivo Mutagenicity Studies. Jet Fuel A. Final Report. Although Jet Fuel A showed no mutagenicity in the Ames test, it was mutagenic in the mouse lymphoma assay and in the in-vivo bone marrow test. The Ames test used selected strains of *Salmonella typhimurium* bacteria. In the lymphoma test, mouse lymphoma cells were exposed to Jet Fuel A with and without metabolic activation. The in-vivo test involved determining the extent of abnormality in the chromosomes of the bone marrow cells of rats. Photomicrographs and tables.
■ *API Medical Research Publication* (8/13/79) (79 p.).
Source: API Library

27-30140
In-Vitro and In-Vivo Mutagenicity Studies. No. 2 Home Heating Oil. Final Report. No. 2 Home Heating Oil showed no mutagenic activity in the Ames test, the mouse lymphoma assay, and the in-vivo bone marrow test. The Ames test used selected strains of *Salmonella typhimurium* bacteria. In the lymphoma test, mouse lymphoma cells were exposed to the oil with and without metabolic activation. The in-vivo test measured the extent of abnormality in the chromosomes of the bone marrow cells of rats. Photomicrographs and tables.
■ *API Medical Research Publication* (7/24/79) (73 p.).
Source: API Library

27-30483
Inhalation/Teratology Study in Rats. Fuel Oil. Final Report. Pregnant female rats exposed to atmospheres containing 86.9 or 408.8 ppm of fuel oil showed no evidence of adverse effects, no variation in fetal sex ratio, and no embryo toxicity or inhibition of fetal growth. Exposure was 6 hr/day on days 6-15 of gestation. The test material was a 50:50 mixture of virgin and cracked gas oil with a boiling range of 437-610 °F. Tables. (supported by API)
■ *API Medical Research Publication* (Sept. 1979) (29 p.).
Source: API Library

27-30484
Inhalation/Teratology Study in Rats. VM and P Naphtha. Final Report. Pregnant female rats exposed to atmospheres containing 104.8 or 391.1 ppm of VM and P naphtha showed no evidence of adverse effects, no variation in fetal sex ratio, and no embryo toxicity or inhibition of fetal growth. Exposure was 6 hr/day during the 6th-15th days of gestation. The test material contained 55% paraffins, 33% cycloparaffins, and 12% alkylbenzenes. The boiling range was 244-304 °F. Tables. (supported by API)
■ *API Medical Research Publication* (Sept. 1979) (30 p.).
Source: API Library

27-30700
Studies on the Evaluation of Problems Associated with the Measurement of Low Concentrations of Carboxyhemoglobin. CRC-APRAC Project No. CAPM-26-75. Using a modified extraction chamber has made gas chromatography with a thermal conductivity detector a practical method for determining low carboxyhemoglobin concentrations in blood. Samples as small as 0.05 ml containing < 1% carboxyhemoglobin are satisfactorily analyzed. The spectrophotometric procedure using the CO-oximeter was rapid and easy to perform but less accurate, especially for the analysis of postmortem blood samples and when concentrations were below 3%. Commonly-used anticoagulants and preservatives did not interfere. Exposing samples to air caused significant carbon monoxide loss, but storage in air-tight syringes under refrigeration was satisfactory. Photographs, graphs, and tables.
■ F. R. Goldbaum (Armed Forces Inst. Pathol.) F.L. Rodkey (U.S. Nav. Med. Res. Inst.), *API Medical Research Publication* (30 p.).
Source: API Library

27-30704
Study of Synergistic Effects of Certain Airborne Systems in the Cynomolgus Monkey. No change in physiological effect on the monkeys resulted from combining any two of the pollutants nitrogen dioxide, sulfur dioxide, carbon monoxide, and lead chlorobromide, as compared with each agent alone. In the experiments, the monkeys continuously inhaled air containing the pollutants for 104 weeks. Combining calcium sulfate with nitrogen dioxide or with sulfur dioxide did not cause a change in effect. Effects on the monkeys were evaluated from lung function measurements and blood chemistry tests. Maximum pollutant concentrations were 6.78 ppm nitrogen dioxide, 9.7 ppm sulfur dioxide, 66 ppm carbon dioxide, 0.5 mg/cu m lead chlorobromide, and 9 mg/cu m calcium sulfate. Tables and 19 references. (supported by API)
■ W. M. Busey (Exp. Pathol. Lab. Inc.) A. A. Krumm (Hazleton Lab. Inc.), *API Medical Research Publication* (23 p.).
Source: API Library

27-30705
Carboxyhemoglobin Trend in St. Louis Blood Donors, 1971-1975. CRC-APRAC Project No. CAPM-8-68. The median carboxyhemoglobin in the blood of 771 nonsmokers in March 1975 was 1.4%, unchanged from March 1971, although estimated carbon monoxide emission from automobiles had decreased 10%. Carboxyhemoglobin levels were down 0.1% in downtown St. Louis and 0.2% in the semi-urban region, compared with 1971, but up 0.2% from 1.3 to 1.5% in the suburban-rural area, the region of lowest vehicle density. The latter results suggest that sources other than vehicle exhaust are the dominant factor in producing carboxyhemoglobin levels above 1.5%. Map, graphs, tables, and 18 references. (supported by Coord. Res. Counc. Inc.)
■ R. D. Stewart (Med.Coll.Wis./Milw.Co.Med.Cplx; P. E. Newton; A. Wu; J. H. Kalbfleisch; T. A. Stewart (Med. Coll. Wis./Milw. County Med. Complex), *API Medical Research Publication* (Oct. 1978) (21 p.).
Source: API Library

27-30765
A Twelve-Week Inhalation Toxicity Study of Benzene on Seven Species of Animals. Final Report. The mouse was the most sensitive species in tests involving exposure of all the animals tested to 361-388 ppm benzene for 6 hr/day, 5 days/week for 12 weeks. The mice showed blood platelet counts that decreased as exposure continued, but this effect, and ocular distress observed over the last eight weeks of exposure, were both reversed during a four-week recovery period allowed for some of the mice. Guinea pigs, some of which were held for postexposure recovery and an additional exposure to benzene, showed no ill effects, except for a weight loss which followed an exposure during week 37 and was subsequently reversed. Physical observations on the guinea pigs and on rats, rabbits, cats, dogs, and monkeys revealed no signs of treatment-related effects. Hematology measurements and differential bone marrow counts on rabbits, cats, dogs, and monkeys were unremarkable, and some variations in the results for rats and guinea pigs did not seem to be treatment related. Graph and tables.
■ *API Medical Research Publication* (11/26/79) (122 p.).
Source: API Library

27-30839
Effects of Sulphate Aerosols upon Human Cardiopulmonary Function. CRC APRAC Project CAPM-27-75. Human subjects exposed to ammonium sulfate at levels simulating the worst case atmosphere in the Los Angeles basin did not suffer any adverse short-term health effects. The tests involved 16 normal, allergic, or asthmatic subjects who were exposed to an atmosphere containing 100 µg/cu m of ammonium sulfate aerosol for two or three days, two hours per day, at 40 or 85% relative humidity. Effects were determined by a series of pulmonary function measurements and clinical observations. Prior research is reviewed. Graphs, tables, diagrams, photographs, and 42 references. (supported by Coord. Res. Counc. Inc.)
■ K. A. Bell; J. D. Hackney (Rancho Los Amigos Hosp.), *API Medical Research Publication* (5/31/77) (212 p.).
Source: API Library

27-30840
The Influence of Trace Metals in Disperse Aerosols on the Human Body Burden of Trace Metals. A relationship was found between the concentrations of cadmium in cigarette smoke and lead in urban air and the concentrations of these metals in human lungs. An association was noted between cadmium and occurrence of a respiratory bronchiolitis. Similar pathological effects were associated with copper and zinc. No

lung pathology was associated with lead. The data on lead in the lungs of New York City residents suggest a quasi-threshold concentration for lead in air: 1.3-1.4 µg/cu m. Below this concentration, lead in the lung is only slightly higher than the background level from ingestion. A size-selective particle sampler was developed for continuous sampling of urban air to obtain large samples over periods from hours to weeks. The average concentrations of iron, copper, manganese, zinc, cadmium, chromium, lead, and nickel in human tissues were determined by measurements on lungs, lymph nodes, liver, kidney, blood, bone, and trachea. Graphs, tables, diagrams, and 200 references. (supported by API)
■ D. M. Bernstein (N.Y. Univ. Med. Cent.), *API Medical Research Publication* (8/1/77) (310 p.).
Source: API Library

27-30841
Effects of Sulfate Aerosols upon Human Pulmonary Function. CRC APRAC Project CAPM-27--75. Human subjects exposed to ammonium sulfate, ammonium bisulfate, or sulfuric acid at levels simulating the worst atmospheric conditions in the Los Angeles Basin did not suffer any adverse effects. In the tests, up to 16 normal, allergic, or asthmatic subjects were exposed for two days, two hours per day, to atmospheres containing 100 µg/cu m ammonium sulfate, 85 µg/cu m ammonium disulfate, or 75 µg/cu m sulfuric acid. The ammonium sulfate tests were carried out at both 40 and 85% humidity and 88 °F. Effects were determined by a series of pulmonary function measurements and clinical observations. Graphs, diagrams, tables, and 35 references. (supported by Coord. Res. Counc. Inc.)
■ M. T. Kleinman; J. D. Hackney (Rancho Los Amigos Hosp.), *API Medical Research Publication* (7/31/78) (75 p.).
Source: API Library

27-30842
Effects of Nitrate and/or Sulfate Aerosols upon Cardiopulmonary Function. Final Report [for First Year of Project]. Five squirrel monkeys breathing 2.5 mg/cu m ammonium sulfate for one hour at 25 °C and 40% relative humidity ammonium sulfate showed increased total respiratory resistance at the 95% confidence level. Preliminary data on exposure of the monkeys to ~ 4 mg/cu m of ammonium sulfate suggested that increases in lung tidal volume, decreases in breathing rate, and possible changes in rate of nitrogen washout may result. At the lower concentration of ammonium sulfate exposure at 85% relative humidity did not cause a significant change in respiratory resistance. A prototype high-capacity aerosol generator for human exposure studies was designed. It can produce 500 µg/cu m ammonium sulfate aerosols with an aerodynamic mass median diameter of 0.35 µm and 2780 µg/cu m ammonium nitrate aerosols with 0.72 µm diameter. Graphs, tables, photographs, diagrams, and 65 references. (supported by Coord. Res. Counc. Inc.)
■ K. A. Bell; J. D. Hackney (Rancho Los Amigos Hosp.), *API Medical Research Publication* (6/1/76) (180 p.).
Source: API Library

27-30915
Carcinogenic Potential of Petroleum Hydrocarbons. A Critical Review of the Literature includes discussion of polycyclic aromatic hydrocarbons; epidemiological studies; carcinogenicity studies of crude oils, fuel oils and solvents, lubricating oils, white oils, waxes, high-boiling cracked aromatic oils, aromatic extracts, and bituminous residues; a discussion of the relationship between composition and carcinogenic potency; and methods of estimating and reducing carcinogenic hazard. Tables and 230 references. (supported by API)
■ E. Bingham; R. P. Trosset; D. Warshawsky (Univ. Cinci.), *Journal of Environmental Pathology and Toxicology* 3:483-563, 1980 (1979).
Source: Not available from API

27-30965
Final Report of the Study of the Relationship between Carboxyhemoglobin on Admission to the Subsequent Hospital Course of Patients Admitted to the Myocardial Infarction Research Unit at the Johns Hopkins Hospital. [CRC-APRAC Project No. CAPM-13-69]. A study of 140 heart attack patients showed that recent smokers had higher mortality than nonsmokers or ex-smokers when age was taken into account. The recent smokers showed evidence of larger infarcts than the nonsmokers but their hemodynamic function was relatively good. The results are consistent with both acute and chronic effects on the myocardium of long-term low-level exposures to carbon monoxide, but cannot be related to that factor by itself. Graphs and tables. (supported in part by Coord. Res. Counc. Inc.)
■ E. P. Radford M. L. Weisfeldt (Johns Hopkins Univ), *API Medical Research Publication* (Aug. 1975) (22 p.).
Source: API Library

27-30966
The Effects of Chronic Exposure to Carbon Monoxide (100 Ppm) on the Cardiovascular System of Monkeys. [CRC-APRAC Project No. CAPM-4-68]. Cynomolgus monkeys exposed to 100 ppm carbon monoxide showed changes in hematology and electrocardiograms after three weeks, in contrast to previously studied mongrel dogs. The electrocardiogram changes were greater in monkeys that had been subjected to induced myocardial infarction than in normal animals exposed to the carbon monoxide but, the effect began to wane after a peak at 12 weeks of exposure, compared to a peak at 21 weeks of carbon monoxide exposure in the normal monkeys. The electrocardiograms reflect altered atrial depolarization, and the waves bear a strong resemblance to those observed in pulmonary hypertension in man. In the monkeys not subjected to induced heart attack, the changes in the electrocardiogram were not observed until 21 weeks. Graphs and tables. (supported by Coord. Res. Counc. Inc.)
■ D. A. DeBias C. M. Banerjee N. C. Birkhead M. H. F. Friedman (Jefferson Med. Coll.), *API Medical Research Publication* (July 1972) (45 p.).
Source: API Library

27-31349
Petroleum Hydrocarbon Toxicity Studies--14. Animal and Human Response to Vapors of "High Aromatic Solvent" indicate a hygienic standard of 26 ppm for the solvent, which has a boiling range of 364-403 °F and consists of ~ 90% C_9-C_{11} alkylbenzenes and 9% indanes. At the maximum vapor concentration obtainable, 0.38 mg/l., 13 week exposure did not affect rats or dogs adversely, as indicated by hematology, clinical chemistry, micropathology, and weight measurement of body, liver, and kidney. Exposing rats for eight hours to an aerosol containing 8.7 mg/l. of the solvent resulted in death. Cats inhaling 8.2 mg/l. aerosol for six hours gave signs of central nervous system depression. Sniff tests with six human subjects indicated an odor threshold of 0.07 ppm. Tables and 16 references. (supported by API)
■ C. P. Carpenter; D. L. Geary; R. C. Myers; D. J. Nachreiner; L. J. Sullivan; J. M. King (Carnegie-Mellon Univ.), *Toxicology and Applied Pharmacology* 41:235-49 (1977).
Source: Not available from API

27-31350
Petroleum Hydrocarbon Toxicity Studies--15. Animal Response to Vapors of "High Naphthenic Solvent." A study led to recommendations that concentrations higher than 2.1 mg/l. should be avoided in workrooms and that workers continuously exposed to 0.61 mg/l. should undergo periodic physical examinations. The four-hour LC50 for rats was 5.3 mg/l. Rats tolerated 4.6 mg/l. and dogs tolerated 3.8 mg/l. for six hours without visible discomfort. In rats exposed to 5.5 mg/l. for 6 hours per day, 5 days per week, for 13 weeks, there was a lowered body weight gain initially and a 75% incidence of kidney tubules dilated with pink homogeneous debris. Exposure to 2.1 mg/l. or 0.61 mg/l. of solvent had no effect. Exposure to the solvent vapors at all concentrations caused no differences in urinalysis, hematology, or blood chemistry. The "high naphthenic solvent" has a boiling range of 315-361 °F and consists of 70% naphthenes and 29% paraffins. Tables and 14 references. (supported by the API)
■ C. P. Carpenter; D. L. Geary; R. C. Myers; D. J. Nachreiner; L. J. Sullivan; J. M. King (Carnegie-Mellon Univ.), *Toxicology and Applied Pharmacology* 41:251-60 (1977).
Source: Not available from API

27-31351

Petroleum Hydrocarbon Toxicity Studies--16. Animal Response to Vapors of "Naphthenic Aromatic Solvent." No deaths occurred among rats exposed to an atmosphere of the highest attainable vapor concentration of "Naphthenic Aromatic Solvent", a blend of "70 solvent" and "High Naphthenic Solvent" contrived to furnish equal amounts of aromatics and naphthenics. In an atmosphere saturated with vapor also containing aerosol, rats typically exhibited salivation, loss of coordination, and mild tremors. Rats subjected to these conditions (4.5 mg/l.) for 6 hours daily, 5 days per week, for 62 days gained significantly less weight than unexposed controls. Atmospheres containing 2.2 mg/l. or less of the solvent vapor had no effect. No solvent-related effects were noted in urinalysis, hematology, blood chemistry, or body weight measurements of the rats. Comparison with data on other solvents indicates that naphthenes are toxic, that they are especially hazardous because they do not give any irritative warning signs, and that the toxic effects of aromatic and naphthenic components in a mixture are strictly additive. Tables. (supported by the API)

■ C. P. Carpenter; D. L. Geary; R. C. Myers; D. J. Nachreiner; L. J. Sullivan; J. M. King (Carnegie-Mellon Univ.), *Toxicology and Applied Pharmacology* 41:261-70 (1977).

Source: Not available from API

27-31352

Petroleum Hydrocarbon Toxicity Studies--17. Animal Response to n-Nonane Vapor. The acute four-hour LC50 for rats breathing n-nonane vapor was 17 mg/l., and the rats tolerated 4.6 mg/l. for four hours without visible effect. Subjecting rats to inhalation of 8.4 mg/l. for 6 hours daily, 5 days per week, for 13 weeks caused salivation and lacrimation, and resulted in body weight gains significantly lower than those of controls. Rats subjected to a similar regime at 3.1 mg/l. or 1.9 mg/l. of the solvent vapor did not differ from the controls. No solvent-related effects were found in the blood, urine, or tissues of the rats. Tables and 17 references. (supported by the API)

■ C. P. Carpenter; D. L. Geary; R. C. Myers; D. J. Nachreiner; L. J. Sullivan; J. M. King (Carnegie-Mellon Univ.), *Toxicology and Applied Pharmacology* 44:53-61 (1978).

Source: Not available from API

27-31528

[Studies of the] Absorption, Distribution, and Excretion of Ethylbenzene [(EB)], Ethylcyclohexane [(ECH)], and Methyethylbenzene [(MEB)] Isomers in Rats showed that the mean quantity of the three compounds, all carbon-14 labeled, absorbed by three male Halan-Wistar rats during 6 hr exposure to a 1 mg/l. concentration was 48, 52, and 58 mg, respectively. Total (excretion and tissues) mean recoveries of 91, 80, and 76% were obtained for EB, ECH, and MEB, respectively. Mean total radioactivity remaining in the tissues was EB, 0.2%; ECH, 0.2%; and MEB, 0.3%. In general, the highest retained amounts were observed in carcasses, the G-I tract, and liver, with fat (EB and ECH) and plasma (MEB) containing relatively high amounts. Excretion of radioactivity was essentially complete for the three compounds within 24 hr after the start of exposure. The major routes of excretion after 48 hr (72 hr for the first study with EB) was through the urine (EB, 83%; ECH, 65%; and MEB, 71%), and expired gases (EB, 8%; ECH, 11%; and MEB, 1%). The remaining excretion routes of feces, CO_2, and tissues contained only minor amounts of the compounds. The quantity of MEB recovered in expired gases was 10-20% of that obtained by this route for the other two compounds. EB, ECH, and MEB are the principal components of "60 Solvent" petroleum hydrocarbon mixture. Diagram, graph, and tables.

■ B. H. Chin (Diamond Shamrock Corp.), J. A. McKelvey (Carnegie-Mellon Univ.), T. R. Tyler, L. J. Calisti, S. J. Kozbelt, L. J. Sullivan, *Bulletin of Environmental Contamination and Toxicology* 24:477-83 (1980).

Source: Not available from API

27-32068

[Project No. 1443] Acute Toxicity Tests [on] API #78-4, No. 2 Home Heating Oil (50% Cat) on laboratory animals produced moderately irritating primary skin effects, essentially no primary eye irritation, and no skin sensitization. The acute dermal test caused skin irritation but not systemic toxicity, but the subacute dermal test produced acute dermal corrosion and obvious treatment-related signs at the 10 ml/kg dosage level. Histopathologic examination of tissues confirmed dermal toxicity at all dosage levels. The oral median lethal dose of the test material was 21.2 ml/kg. The primary skin irritation and eye irritation tests were made on white rabbits; the skin sensitization tests, on guinea pigs; the acute dermal toxicity tests, on white rabbits; and the acute oral toxicity tests, on albino rats. Tables.

■ L. S. Beck; D. I. Hepler (Elars Biores. Lab.), *API Medical Research Publication* (7/17/80) (52 p.).

Source: API Library

27-32127

Teratology Study in Rats. Shale Oil R03. Final Report [on LBI Project No. 20726-R03]. Exposure of pregnant rats to air containing 0, 5.9, or 104.6 mg/cu m of shale oil R03 had no effect on the formation and development of the embryos, but caused significantly decreased food consumption and significantly decreased body-weight gain in the mothers at the 104.6 mg/cu m level. The pregnant rats were exposed to shale oil R03 on days 6 through 15 of gestation for 6 hr/day. The particle size of the aerosol was > 85% below 9 μ. The effects on the fetuses were determined by removing the uteri on day 20 of gestation and determining the number and placement of implantations and resorptions, the number of live and dead fetuses, soft tissue changes, and skeletal abnormalities. Tables. (supported by API)

■ *API Medical Research Publication* (Nov. 1978) (22 p.).

Source: API Library

27-32128

Teratology Study in Rats. Shale Oil R04. Final Report [on LBI Project 20726-R04]. Exposing pregnant rats to air containing 0, 5, or 100 mg/cu m of shale oil R04 caused mottled lungs in the mothers at the 5 and 100 mg/cu m levels, and produced evidence of embryo toxicity expressed as an increase in resorptions and accompanying parameters at the 100 mg/cu m level. The pregnant rats were exposed to shale oil R04 on days 6 through 15 of gestation for 6 hr/day. The particle size of the oil aerosol was > 97.9% below 9 μ. The effects on the fetuses were determined by removing the uteri on day 20 of gestation and determining the number and placement of implantations and resorptions, the number of live and dead fetuses, soft tissue changes, and skeletal abnormalities. Tables. (supported by API)

■ *API Medical Research Publication* (Feb. 1979) (27 p.).

Source: API Library

27-32129

Teratology Study in Rats. Shale Oil R01. Final Report [on LBI Project No. 20726-R01]. Exposing pregnant rats to air containing 0, 5, or 100 mg/cu m of shale oil R01 had no effect on embryo formation and development, but caused lung discoloration and decreased food consumption values in the mothers at the 100 mg/cu m level. The pregnant rats were exposed to shale oil R01 on days 6 through 15 of gestation for 6 hr/day. The particle size of the oil aerosol was > 98% below 9 μ. Effects on the fetuses were determined by removing the uteri on day 20 of gestation and determining the number and placement of implantations and resorptions, the number of live and dead fetuses, soft tissue changes, and skeletal abnormalities. Tables. (supported by API)

■ *API Medical Research Publication* (Feb. 1979) (27 p.).

Source: API Library

27-32130

[Project No. 1443] /Acute Toxicity Tests [of] API #PS-6, Unleaded Motor Gasoline showed a slight primary skin irritation but no primary eye irritation on rabbits, and no skin sensitization on albino guinea pigs. The acute dermal test produced skin irritation and no obvious signs of toxicity, although the death of one rabbit may be treatment-related. The subacute dermal test on rabbits caused acute skin corrosion and slight systemic toxicity; histopathologic examination determined dermal toxicity at all dosage levels. The oral median lethal dose for rats was 18.75 ml/kg. Tables.

■ L. S. Beck D. I. Hepler (Elars Biores. Lab.), *API Medical Research*

Publication (7/16/80) (51 p.).
Source: API Library

27-32131
[Project No. 1443] /Acute Toxicity Tests [of] API #78-1, New Composite Motor Oil. The test material caused slight primary skin irritation and virtually no primary eye irritation on rabbits, and no skin sensitization on albino guinea pigs. The acute and subacute dermal tests on rabbits produced skin irritation and acute dermal corrosion at the 8 ml/kg dosage level, respectively; neither test showed systemic toxicity. Histopathologic examination determined dermal toxicity at both the 8 and 4 ml/kg dosage levels in the subacute dermal test. The acute oral toxicity test on rats showed an oral median lethal dose of > 25 ml/kg. Tables.
■ L. S. Beck D. I. Hepler (Elars Biores. Lab.), *API Medical Research Publication* (7/17/80) (50 p.).
Source: API Library

27-32132
[API Project PS-8] /The Evaluation of the Carcinogenicity of Certain Petroleum Fractions. The skins of mice (usually in groups of 50) were treated with 50 mg of each of the 25 undiluted test materials, comprising two whole crude oils and several of their light to heavy fractions, except for the PS8-76C6 (1070 °F) and PS8-76D6 (1070 °F) fractions, which were dissolved in toluene and applied as 50% solutions, for 80 weeks or until papilloma, a benign tumor, developed. Histologic examination showed that 285 treated mice developed either sarcoma, basal cell, or squamous cell cancer; that 236 developed papilloma, fibroma, or keratoma (benign tumors); that 22 had dysplasia, which could be indicative of a precancerous stage; that an unexplained high incidence (26 out of 44 mice) of malignant tumors occurred in mice exposed to the PS8-76C3 saturated fraction; and that only the PS8-76D1 (OP-120 °F) and PS8-76C6 (1070 °F) fractions and the saturates from the PS8-76D5 (700-1070 °F) fraction had no carcinogenic effect. These studies, carried out by Gulf Science & Technology Co. for the American Petroleum Institute, do not confirm the prediction that the 550-700 °F and 700-1070 °F fractions and the oils and aromatics separated therefrom should exhibit tumorigenic activity after the skin painting because of their high polynuclear aromatics content. Tables, diagrams, and flow charts.
■ K. L. Stemmer (Univ. Cinci. Med. Cent.), *API Medical Research Publication* (Oct. 1979) (45 p.).
Source: API Library

27-32173
Inhalation/Teratology Study in Rats. Jet Fuel A. Final Report. Exposing pregnant female rats to air containing 102.5 and 394.7 ppm of jet fuel A for 6 hr/day on days 6-15 of gestation produced no adverse effects on the mothers and no evidence of terata, variation in sex ratio, embryo toxicity, or inhibition of fetal growth and development. Tables. (supported by API)
■ P. J. Knapinski, T. M. Lango, F. J. Mecler (Litton Bionetics Inc.), *API Medical Research Publication* (May 1979) (31 p.).
Source: API Library

27-32174
Teratology Study in Rats. Diesel Fuel. Final Report. Exposing pregnant female rats to air containing 101.8 and 401.5 ppm of diesel fuel for 6 hr/day on days 6-15 of gestation significantly reduced food consumption in the 401.5 ppm exposure group, but produced no other adverse diesel fuel-related effects on the mothers and no evidence of terata, variation in sex ratio, embryo toxicity, or inhibition of fetal growth and development. Tables. (supported by API)
■ F. J. Mecler R. P. Beliles (Litton Bionetics Inc.), *API Medical Research Publication* (Mar. 1979) (24 p.).
Source: API Library

27-32175
Teratology Study in Rats. Kerosene. Final Report. Exposing pregnant female rats to air containing 106.4 and 364.0 ppm of kerosine for 6 hr/day on days 6-15 of gestation produced no adverse effects on the mothers and no evidence of terata, variation in sex ratio, embryo toxicity, or inhibition of fetal growth and development. Tables. (supported by API)
■ F. J. Mecler R. P. Beliles (Litton Bionetics Inc.), *API Medical Research Publication* (Mar. 1979) (29 p.).
Source: API Library

27-32176
Teratology Study in Rats. High-Aromatic Solvent. Final Report. Exposing pregnant female rats to air containing 100 and 400 ppm of a high-aromatic solvent for 6 hr/day on days 6-15 of gestation produced no evidence of terata, variation in sex ratio, embryo toxicity, or inhibition of development of fetuses at 100 ppm exposure, but produced evidence of mucous membrane irritation at 100 and 400 ppm of exposure. At 400 ppm, there was a significant reduction in the food consumption and body weight; 12 pregnant rats died; and the mean fetal weight at Cesarean section decreased. There were too few full-term litters for a meaningful analysis of the direct effects on the fetuses at 400 ppm. Tables. (supported by API)
■ S. L. Makris, F. J. Mecler, R. P. Beliles (Litton Bionetics Inc., *API Medical Research Publication* (Feb. 1979) (24 p.).
Source: API Library

27-32177
Teratology Study in Rats. n-Hexane. Final Report. Exposing pregnant female rats to air containing 93.4 and 408.7 ppm of n-hexane for 6 hr/day on days 6-15 of gestation produced no adverse effects on the mothers and no evidence of terata, variation in sex ratio, embryo toxicity, or inhibition of fetal growth and development. Tables. (supported by API)
■ M. L. Ament, F. J. Mecler, R. P. Beliles (Litton Bionetics Inc.), *API Medical Research Publication* (Jan. 1979) (7 p.).
Source: API Library

27-32178
Polycyclic Aromatic Hydrocarbons [(PAH)] in the Aquatic Environment: Sources, Fates, and Biological Effects. Attempts to link PAHs to the high incidence of cancer in aquatic organisms have met with only limited success. PAH-contaminated water and fishery products represent minor sources of PAH toxicity for man. PAHs are acutely toxic to aquatic animals (e.g., dungeness crab) at concentrations of 0.2-10 ppm; are sometimes deleterious to aquatic organisms at concentrations of 5-100 ppb, inhibit the growth rates of certain bacteria and aquatic plants, and induce cancer-like growths or developmental anomalies in several aquatic animals (e.g., freshwater fish) and plants. The carcinogenicity, mutagenicity, and/or teratogenicity of some PAHs are attributed to the covalent binding of certain electrophilic metabolites of PAHs to cellular macromolecules. PAHs may be formed by high-temperature (e.g., 700 °C) pyrolysis of organic materials, low-to-moderate temperature (100-150 °C) diagenesis of sedimentary organic materials to form fossil fuels, and direct/indirect biosynthesis by microbes and plants. Fossil fuels such as petroleum, coal, and peat are rich in PAHs. A total of ~ 230,000 tons/yr of PAHs enter the aquatic environment via oil and oil products spillage, sewage effluents, deposition of airborne particulates, and surface runoff from land. The concentrations of PAHs in water and sediments may be reduced by photooxidation, evaporation, or biological transformation by aquatic bacteria, fungi, and animals. Tables, graphs, and 419 references.
■ J. M. Neff (Tex. A&M Univ.), *API Medical Research Publication* (Aug. 1978) (354 p.).
Source: API Library

27-32179
[Final Report on the] Mutagenicity Evaluation of n-Hexane in the Mouse Dominant Lethal Assay. Male mice exposed by inhalation to 100 and 400 ppm n-hexane for six hours a day, five days a week for eight weeks, showed that the test material did not induce dominant lethal mutations in the sperm cells of the treated mice at both dosage levels. Female mice mated with the exposed males showed no significant increases in either pre- or post-implantation loss of embryos at either dosage level. The sensitivity of the assay was confirmed by the significant induction of dominant lethal mutations in the positive control mice injected with 0.3 mg/kg of triethylenemelamine. Tables. (supported by API)

■ T. D. Nguyen D. J. Brusick (Litton Bionetics Inc.), *API Medical Research Publication* (Jan. 1980) (28 p.).
Source: API Library

27-32280
Job Code Classification System--1. Petroleum Refineries and Selected Petrochemical Operations. A job classification system was developed for the American Petroleum Institute by Tabershaw Occupational Medicine Associates P.A. for occupational health studies for which job data grouping would be useful. Possible applications include morbidity and mortality studies, qualitative and quantitative exposure and medical surveillance evaluations, exposure monitoring, and medical surveillance scheduling. Each job was classified into two variables: process and task. The process variable, which emphasizes where an individual is physically located rather than administratively assigned, was broken into 44 "units" (which are further refined with subheadings) that significantly differentiate between qualitative exposures from process, feedstock, and/or product streams. Tasks were divided into 11 major functions, many of which have subtask designation, and the majority of which are related to maintenance activities. A list of the process and task headings and subheadings, process/task matrix worksheets, and process list and task list worksheets are provided. Also see Abstract No. 33-30051.
■ *API Publication* (12/26/79) (91 p.).
Source: API Library

27-32296
Benzene, Xylene, and Toluene in Aquatic Systems: A Review of the literature covers industrial sources and uses of benzene, xylene, and toluene; their physical and chemical properties; analytical methods; mechanisms of their toxic action; factors affecting toxicity, including water salinity, temperature, presence of other toxic materials, life stage, organism size, and test species; toxicity to several species, including phytoplankton and 11 invertebrates including crabs; effects on fish fry, including changes in yolk utilization, growth, hatching, oxygen consumption, and feeding behavior; biological effects, including changes in histology, development and growth, oxygen consumption, behavior, feeding and assimilation, and cell permeability; uptake and depuration; degradation and metabolism; population, community, and ecosystem studies; taste and odor; and concludes that benzene will probably not be a major problem in aquatic systems, except during episodic spills, since it is volatile and easily degraded, which might also be the case for xylene and toluene, although toluene is taken up and does cause a taste and odor problem in fish. Tables, diagrams, and 157 references. (supported by API)
■ A. L. Buikema; A. C. Hendricks (Va. Polytech. Inst. State Univ.), *API Publication* (1980) (72 p.).
Source: API Library

27-32466
A Final Report [on a] 24 Month Inhalation Toxicity Study of Raw and Spent Shale Dusts in Rats and Monkeys shows no evidence for either a progressive fibrogenic or carcinogenic effect from a two-year exposure to 30 mg/cu m concentrations in air of the micronized dusts, despite indications of a lung burden that initiated inflammatory reactions in both species. A high incidence of mortality in the groups of rats was positively age-related, but not clearly treatment-related. The effects of dust exposures were dose-related, but were confined to the lungs and peribronchial lymph nodes in both species. There was no evidence of systemic toxicity. In the tests, 40 young cynomolgus monkeys and 520 Fischer 344 rats, equally divided between sexes, were exposed to air or shale dusts in 6 cu m chambers for 6 hr/day, 5 days/week for 104 weeks. Tables.
■ *API Medical Research Report* (3/6/80) (261 p.).
Source: API Library

27-32467
[A Review of the Published Literature on] Risks to the Offspring from Parental Occupational Exposures. covers the mutagenic and teratogenic characteristics of agent and transgenerational carcinogenesis; the possible environmental threats to pregnancy outcome posed by alcohol, smoking, drug exposure, and radiation; the possible threats to pregnancy outcome posed by occupational exposures to vinyl chloride, chloroprene, benzene, anesthetic gases, and petroleum-derived hydrocarbons; the difficulty in demonstrating the occurrence of teratogenesis and transgenerational carcinogenesis in workers and their families after workplace exposure to suspected agents; and the surveillance of spontaneous abortions as a strategy for environmental monitoring. 49 references. (supported in part by API)
■ J. F. Haas, D. Schottenfeld (Cornell Univ. Med. Col./Mem-Sloan Kettering Cancer Cent.), *Journal of Occupational Medicine* 21(9):607-13 (Sept. 1979).
Source: Not available from API

27-32610
The Evaluation of the Three-Month Inhalation Toxicity of Two Motor Fuels in experiments on male and female squirrel monkeys and male and female rats showed that, under the test conditions, air-diluted vapors of leaded and unleaded gasoline produced no effects beyond an increased platelet count in the rats after 3.0 mo of exposure, and in monkeys exposed to 6.35 mg/l. of unleaded gasoline, a small increase in respiratory rate after 1.5 and 3.0 mo of exposure. The diluted gasoline vapors did not affect the central nervous system, body weight, food consumption, and organ weight (except for a slight increase in liver weights for rats), or cause detectable histopathological effects or immunologic-based glomerular nephritis. The unleaded gasoline was an EPA reference fuel (Research octane number 93.2, Motor octane number 84.7) and the leaded fuel was an uncharacterized commercial product. Test animals were exposed to 6.35 or 1.57 mg/l. of the unleaded fuel or 1.53 or 0.42 mg/l. of leaded fuel for 6 hr/day for 5 days/week for 3 mo, with physiological tests and histological studies conducted 1.5 and 3.0 mo after the start of exposure. Table.
■ C. E. Ulrich (Huntington Res. Cent.), *API Medical Research Publication* (4/14/76) (214 p.).
Source: API Library

27-32771
[Project No. 1443] /Acute Toxicity Tests of API #78-2 No. 2 Home Heating Oil (30% Cat) caused moderately irritating primary skin effects on male and female albino rats treated with 0.5 ml/kg. The oil did not produce remarkable primary eye irritation on albino rabbits or skin sensitization on male albino guinea pigs. The application of 5 ml/kg of the test material on shaved areas of the albino rabbits' backs produced skin irritation but no obvious signs of toxicity during the acute dermal test. When tested for subacute dermal toxicity, the compound caused acute dermal corrosion on albino rabbits and resulted in obvious treatment-related signs in the 10 ml/kg treatment group. Histopathologic examination showed systemic toxic effects on the 10, 2.5, and 1.0 ml/kg treatment groups. This Elars Bioresearch Laboratories report for the American Petroleum Institute has determined that the dermal lethal dose for the test material is 5.9 ml/kg. The oral median lethal dose (Oral LD_{50}) on rats is 19.0 ml/kg. This places the test material in EPA toxicity category IV, Oral LD_{50} > 5000 mg/kg. Tables.
■ *API Medical Research Publication* (5/5/80) (48 p.).
Source: API Library

27-32772
[Project No. 1443] /Acute Toxicity Tests...API 79-7 Used Composite Motor Oil. The albino rabbits whose skins and lower eyelids were treated with 0.5 and 0.1 ml/kg of used composite motor oil, respectively, showed minimal skin irritation and no eye irritation. The test material did not produce skin sensitization on the animals. Acute dermal testing caused slight dermal irritation but no signs of systemic toxicity on albino rabbits treated with 0.5 ml/kg of the oil; rabbits treated with 0.8 ml/kg of the oil showed dermal irritation but no obvious treatment-related systemic toxicity in the subacute dermal test. The oral median lethal dose of the test material on rats is > 25 ml/kg. Tables. (supported by API)
■ *API Medical Research Publication* (8/25/80) (44 p.).
Source: API Library

27-32773
[Project No. 1443] /Acute Toxicity Tests of API #78-3 No. 2 Home Heating Oil (10% Cat) produced moderately irritating primary skin

effects and mildly irritating primary eye effects on male and female albino rabbits at 0.5 and 0.1 ml/kg dosage levels, respectively, and no skin sensitization on albino guinea pigs at a 0.5 ml/kg dosage level. The oil produced skin irritation but not systemic toxicity at a dosage level of 5 ml/kg, and acute dermal corrosion at 2.5, 4, and 10 ml/kg. Histopathologic examinations of the rabbit tissues confirmed dermal toxicity at all dosage levels and hepatic toxicity at the 10 ml/kg dosage level. The dermal lethal dose (LD_{50}) for this oil is 4.7 ml/kg. The acute oral toxicity test on male and female rats showed an oral median lethal dose (Oral LD_{50}) of 14.5 ml/kg. This places the test material in EPA toxicity category IV, Oral LD_{50} > 5000 mg/kg. Tables. (supported by API)
■ *API Medical Research Publication* (8/25/80) (52 p.).
Source: API Library

27-32774
[Project No. 1443] /Acute Toxicity Tests of API 78-7 No. 6 Heavy Fuel Oil (17.1 °API/0.8% S) on laboratory animals caused slightly irritating primary skin effects and was mildly sensitizing to the skin at a dosage level of 0.5 ml/kg. The oil also produced slight skin irritation but not systemic toxicity at a single application of 5 ml/kg, and acute dermal irritation and obvious treatment-related signs at the 8 ml/kg dosage level. Histopathologic examination confirmed dermal and hepatic toxicity at the higher dosage level. The test material was minimally irritating to the eyes at a dosage level of 0.1 ml/kg. The oral median lethal dose of this oil for rats is > 25 ml/kg. The primary skin and eye irritation, and acute and subacute dermal tests were conducted on albino rabbits, and the skin sensitization test on albino guinea pigs. Tables. (supported by API)
■ *API Medical Research Publication* (8/29/80) (45 p.).
Source: API Library

27-32813
[Project No. 1443] /Acute Toxicity Tests of API 79-2 No. 6 Heavy Fuel Oil (5.2 °API/1.2% S) produced slightly irritating primary skin effects on albino rabbits after a single, 0.5 ml/kg dose, and no skin sensitization on albino guinea pigs at a 0.5 ml/kg dosage level during ten treatments over three weeks. The acute dermal toxicity test produced three mortalities of the eight rabbits tested, each of which was treated with a single dose of 5 ml/kg of the oil; skin irritation; and severe signs of systemic toxicity. The subacute dermal test on rabbits caused acute dermal irritation and obvious treatment-related signs at the 1, 2, and 2.5 ml/kg dosage levels during the treatments over 12 days; histopathologic examination confirmed dermal and hepatic toxicity at all dosage levels. The dermal lethal dose (LD_{50}) for this oil is 1.90 ml/kg; the oral median LD_{50} for rats is 5.13 ml/kg. The test material produced mildly irritating primary eye effects on albino rabbits after a single dose of 0.1 ml/kg. Tables. (supported by API)
■ *API Medical Research Publication* (9/2/80) (52 p.).
Source: API Library

27-32814
[Project No. 1443] /Acute Toxicity Tests...API 78-6 No. 6 Heavy Fuel Oil (11.7 °API/2.7% S). In primary skin irritation and primary eye irritation tests on albino rabbits at 0.5 and 0.1 ml/kg dosage levels, respectively, API 78-6 was minimally irritating. In the acute dermal test on rabbits, a single dose of 5 ml/kg of the oil caused slight dermal irritation but no obvious signs of systemic toxicity during the 14 day post-dose period. Postmortem examination showed that two rabbits had slightly congested livers, which were considered to be treatment-related. In subacute dermal tests on rabbits at an 8 ml/kg dosage level in ten treatments over 12 days, there were obvious treatment-related signs and acute dermal irritation, which along with hepatic toxicity, were confirmed in histopathologic analysis; the dermal lethal dose (LD_{50}) for the oil is > 8 ml/kg. The acute oral toxicity test on male and female rats showed that the LD_{50} is > 25 ml/kg. The oil did not cause skin sensitization on albino guinea pigs at a dosage level of 0.5 ml/kg in 10 treatments over 3 weeks. Tables. (supported by API)
■ *API Medical Research Publication* (9/2/80) (47 p.).
Source: API Library

27-32815
[Project No. 1443] Acute Toxicity Tests of API Jet Fuel A on albino rabbits caused mildly irritating primary skin and primary eye effects after one dose of 0.5 and of 0.1 ml/kg, respectively, and no skin sensitization on albino guinea pigs at the 0.5 ml/kg dosage level over the three-week test. Acute dermal testing on albino rabbits with one 5 ml/kg dose produced slight skin irritation but no obvious signs of systemic toxicity. Subacute dermal testing at the 8 ml/kg dosage level over 12 days produced acute dermal corrosion and obvious treatment-related signs; dermal and hepatic toxicity and hyperplastic changes in the urinary bladder transitional epithelium were confirmed in histopathologic analysis. The dermal lethal dose (LD_{50}) is probably < 8 ml/kg. The oral median LD_{50} for rats is > 25 ml/kg. Tables. (supported by API)
■ *API Medical Research Publication* (9/12/80) (48 p.).
Source: API Library

27-32816
[Project No. 1443] Acute Toxicity Tests...API 78-8 No. 6 Heavy Fuel Oil (23.1 °API/0.2% S). Male and female albino rabbits treated with 0.5 and 0.1 ml/kg of the oil showed minimally irritating primary skin and eye effects, respectively. Albino guinea pigs showed no skin sensitization at a dosage level of 0.5 ml/kg. The acute dermal test on albino rabbits at a dosage level of 5 ml/kg produced slight dermal irritation but not systemic toxicity. Subacute dermal testing at the 8 ml/kg dosage level over two weeks caused slight dermal irritation and obvious treatment--related signs; histopathologic examination confirmed dermal and hepatic toxicity and proliferative changes in the transitional epithelium of the urinary bladder. The dermal lethal dose (LD_{50}) for albino rabbits is > 8 ml/kg; the oral median LD_{50} for rats is > 25 ml/kg. Tables. (supported by API)
■ *API Medical Research Publication* (9/12/80) (47 p.).
Source: API Library

27-32817
[Project No. 1443] Acute Toxicity Tests...API 79-6 Diesel Fuel (Marketplace Sample). Albino rabbits treated once with 0.5 ml/kg of the test material showed extremely irritating primary skin effects. Those tested for acute dermal toxicity after a single 5 ml/kg dose showed skin irritation but not systemic toxicity. The subacute dermal test on rabbits produced acute dermal corrosion and obvious treatment-related signs at the 4 and 8 ml/kg dosage levels for ten treatments over 12 days; histopathologic analysis confirmed dermal toxicity at both dosage levels and hepatic toxicity at the higher dosage level. The test material did not cause skin sensitization on albino guinea pigs, and was essentially non-irritating to the eyes of albino rabbits at dosage levels of 0.5 and 0.1 ml/kg, respectively. The oral median lethal dose for rats is 9.0 ml/kg. Tables. (supported by API)
■ *API Medical Research Publication* (9/2/80) (51 p.).
Source: API Library

27-33264
Analysis of Shale Oils and Downstream Products. Application of Exxon Research & Engineering Co.'s gas chromatography (GC)/UV procedure to 14 polynuclear aromatic N-heterocyclics (N-PNAs) led to the development of a general scheme for the isolation of polynuclear aromatic hydrocarbons (PNA) and N-PNA from shale oil liquids. Separation characteristics of the N-PNAs on silicic acid columns and in N-methylpyrrolidone/cyclohexane extractions were determined in model compound studies. Analyses of six samples of shale oil liquids showed that: three-ring PNAs occurred in diesel fuel marine precursor and product samples; higher boiling samples contained additional 4-7 ring PNAs; only a few N-PNAs were detected in all but the crude shale oil sample; the most commonly encountered species were carbazoles and benzocarbazoles; and unidentified N-PNAs were detected in isolated N-PNA fractions by high resolution mass spectrometry, especially for the crude shale oil in contrast with the in-situ oil. Tables and diagrams.
■ W. K. Robbins (Exxon Res. Eng. Co.), *API Medical Research Report* (10/1/80) (32 p.).
Source: API Library

27-33273
LA-8459-MS/Paraho Oil Shale Workers Occupational Health Study. In an occupational health and industrial hygiene study of oil shale

workers at the U.S. Department of Energy (DOE) Anvil Points Oil Shale Mine and Retorting Facility near Rifle, Colo., the Los Alamos Scientific Laboratory, under a contract with the American Petroleum Institute and DOE, determined that exposure to oil shale operations or its retorting products on a developmental scale did not, apparently affect the health of Paraho employees, but full-time employees would receive significant exposure to unhealthful conditions in some areas of the facility. Job-incurred injuries were the major health problem. Commercial scale operation of the Paraho direct combustion retorting process will require industrial hygiene control measures and periodic comprehensive medical evaluations, as well as the design of operations and equipment to avoid potential health and safety problems. Graphs, tables, diagrams, and 28 references.
■ J. Rudnick; L. L. Garcia; G. L. Voelz; H. F. Schulte (Los Alamos Sci. Lab.), *API Medical Research Report* (July 1980) (28 p.).
Source: API Library

28-30077
[Bio/Dynamics Inc. Project No. 77-1921] /A 26 Week Inhalation Toxicity Study of n-Hexane in the Rat. Neurological evaluation at the Albert Einstein College of Medicine of male and female rats after 8 and 18, 26, 28, 31, and 34 weeks of exposure to n-hexane vapors via inhalation at mean levels of 6, 26, and 129 ppm for 6 hr/day, five days a week, and at 5, 27, and 126 ppm for 21 hr/day, seven days a week, showed that the concentrations and patterns used in this test do not appear to have produced a toxic response in the eight groups of rats. Differences of significance ($p \leq 0.05$) were noted in the elevated weights of male rats exposed at 26 ppm at weeks 9, 16, 18, 24, and 26. In some mean hematologic parameters, significant differences (which were within normal biological limits) were noted in female rats at month three but not at month six; thus they do not seem to be treatment-related. In mean clinical chemistry parameters, at month six, the mean fasting glucose levels of male rats exposed at 6 and 129 ppm and the mean blood urea nitrogen levels of female rats exposed at 126 ppm were significantly higher and lower ($p \leq 0.05$), respectively, than the controls. Physical observations were generally comparable with the controls. Some variations of the incidence of abnormalities were apparent, but no markedly severe differences were noted. Tables. (supported by API)
■ *API Medical Research Publication* (12/19/78) (170 p.).
Source: API Library

28-30173
Mutagenicity Evaluation of Unleaded Gasoline: Final Report. Unleaded gasoline was considered nonmutagenic in a series of in-vitro and in-vivo tests conducted by Litton Bionetics Inc. The in-vitro studies involved genetic assays on bacteria (*Salmonella typhimurium*) and yeast (*Saccharomyces cerevisiae*) with and without activation by rat liver tissue, and on mouse lymphoma cells. In the in-vivo tests, an increase in chromosome aberrations at the 48 hr sacrifice time of the intermediate dose level in the bone marrow cells of rats was not considered to be treatment-related. Tables and graph. (supported by API)
■ *API Medical Research Publication* (Mar. 1977) (68 p.).
Source: API Library

28-30174
Polycyclic Aromatic Hydrocarbons [(PAHs)] : A Review covers the history and occurrence, metabolism, and biological activity (i.e., acute toxicity and carcinogenesis in vivo) of PAHs, their physiological disposition in laboratory animals and man, and the tumorigenicity of hydrocarbon diol-epoxide metabolites; recommends additional work on the ability of PAHs to influence the PAH-metabolizing system and the induction of the ultimate carcinogenic form of any substance, thus indicating the synergistic effect of PAHs on man; and suggests the possibility of determining whether workers in industries using PAHs have been exposed to these agents by examining their urine for the presence of mutagens via the Ames mutagenesis assay, as well as further studies on the absorption of PAHs from the human lung and skin into the systemic circulation, and from the gastrointestinal tract. 134 references. (supported by API)
■ M. S. Zedeck (Mem. Sloan-Kettering Cancer Cent.), *Journal of Environmental Pathology and Toxicology* 3:537-67 (1980).
Source: Not available from API

28-30724
American Petroleum Institute Project PS-22-GRD(840)/A "Comprehensive Analysis of Petroleum Coke Products" and one process water was made by the Gulf Research & Development Co. Four polynuclear aromatic hydrocarbons (PAHs), were present in the water sample, but at extremely low levels, and below the EPA recommended drinking water standards. Only the benzo(a)pyrene concentration was above 1 ppb. Although the water is not considered hazardous to handle, it is not recommended for drinking. Benzene extractables were 0.15-9.94% by wt for three coke samples consisting principally of carbon and hydrogen, but containing appreciable quantities of oxygen, sulfur, and nitrogen. Two extracts contained 11-544 ppm PAHs and one, 2-10 ppm. Cautious handling is suggested to prevent exposure to the carcinogenic properties indicated by the analysis. Two of the coke samples contained 56 and 113 ppm mercury, and the third, < 1 ppb. All three cokes contained 60-410 ppm of vanadium and nickel and 0.7-5.3 ppm of selenium. No arsenic was detected in any sample. Tables and photomicrographs.
■ *API Medical Research Publication* (6/10/80) (23 p.).
Source: API Library

28-30725
In-Vivo and In-Vitro Mutagenicity Studies. Quench Water from Delayed Process Coke. Liquid 7-1-100. A Revised Final Report. Liquid 7-1-100 showed no mutagenic potential in the Ames test, the mouse lymphoma assay, or the in-vivo bone marrow test conducted in 1979 for the American Petroleum Institute by Hazleton Laboratories America Inc. The Ames test used selected strains of *Salmonella typhimurium* bacteria. In the lymphoma test, mouse lymphoma cells were exposed to the test sample oil with and without metabolic activation. The in-vivo test measured the extent of cytogenic abnormalities in the chromosomes of the bone marrow cells of rats exposed via inhalation to the samples. Photomicrographs and tables.
■ *API Medical Research Publication* (1/20/81) (65 p.).
Source: API Library

28-30726
In-Vivo and In-Vitro Mutagenicity Studies. Fluid Process Coke. (Petroleum Coking Sample 6-1-468). A Revised Final Report. Petroleum coking sample 6-1-468 showed no mutagenic potential in the Ames test, the mouse lymphoma assay, or the in-vivo bone marrow test in a study made in 1979. The Ames test used selected strains of *Salmonella typhimurium* bacteria. In the lymphoma test, mouse lymphoma cells were exposed to the sample with and without metabolic activation. The in-vivo test measured the extent of cytogenetic abnormalities in the chromosomes of the bone marrow cells of rats exposed via inhalation to 0, 10, and 40 mg/l. of test sample in air. Photomicrograph and tables.
■ *API Medical Research Publication* (1/20/81) (74 p.).
Source: API Library

28-30727
In-Vivo and In-Vitro Mutagenicity Studies. Solid Condensed Emission Product from Delayed Coke Process. (Petroleum Coking Sample 3-1-134). Although petroleum coking sample 3-1-134 showed no mutagenicity in the Ames test and the mouse lymphoma assay, it was mutagenic in the in-vivo bone marrow test. The Ames test used selected strains of *Salmonella typhimurium* bacteria. In the lymphoma test, mouse lymphoma cells were exposed to the test sample with and without metabolic activation. The in-vivo test measured the extent of cytogenic abnormalities induced by inhalation at 10 and 40 mg/l. in the chromosomes of the bone marrow cells of rats. All tests were conducted for the American Petroleum Institute by Hazleton Laboratories America Inc. in 1979. Photomicrographs and tables.
■ *API Medical Research Publication* (1/20/81) (75 p.).
Source: API Library

28-30728
In-Vivo and In-Vitro Mutagenicity Studies. Delayed Process Coke. (Petroleum Coking Sample 4-1-140). A Revised Final Report. Although petroleum coking sample 4-1-140 showed no mutagenicity in the Ames test and the mouse lymphoma assay, it was mutagenic in the

in-vivo bone marrow test on rats after their five-day exposure, via inhalation, to 40 µg/l. of the test sample in air. The Ames test used selected strains of *Salmonella typhimurium* bacteria. In the lymphoma test, mouse lymphoma cells were exposed to the sample with and without metabolic activation. The in-vivo test measured the extent of cytogenic abnormalities in the chromosomes of the bone marrow cells of rats exposed to the test samples via inhalation. Photomicrographs and tables. All tests were performed for the American Petroleum Institute by Hazleton Laboratories America Inc. in 1979.
■ *API Medical Research Publication* (1/20/81) (70 p.).
Source: API Library

28-31209
[Bio/dynamics Inc. Project No. 78-7233] /A 26 Week Inhalation Toxicity Study of Heptane in the Rat. An inhalation toxicity study was performed on male and female rats exposed to a cumulative mean exposure concentration of 0, 398, and 2970 ppm of n-heptane for 6 hr/day, 5 days/week, for 26 weeks with selected animals being removed following 8, 17, and 26 weeks of exposure, and a two-week post-exposure recovery period. Labored or rapid breathing and slight prostration were noted during the first week of the study during the exposures only. Yellow staining of the anogenital fur and dry rales were noted during the weekly observations. These signs were more numerous and severe in the high-level exposure group. Neither hematology nor urinalysis gave evidence of treatment-related effects. The serum alkaline phosphatase levels at week 26 were significantly elevated in the female high-level rats and slightly elevated in the female low-level rats. There were no treatment-related deaths during the study. A report from the Albert Einstein College of Medicine on the neurological condition of the subject rats after the termination of exposure is included. Tables and graphs. (supported by API)
■ *API Medical Research Publication* (5/23/80) (78 p.).
Source: API Library

28-31210
[Bio/dynamics Inc. Project No. 78-7234] /A 26 Week Inhalation Toxicity Study of Toluene in the Rat. An inhalation toxicity study was made on rats exposed to cumulative mean concentrations of 0, 100, or 1481 ppm of toluene for 6 hr/day, 5 days/week, for 26 weeks, with selected animals being removed following 8, 17, and 26 weeks of exposure, and a two-week post-exposure recovery period. There were no treatment-related deaths during the study. The high-level group showed signs of central nervous system depression during week 2; and increased incidences of dry rales and staining of the anogenital fur. Body weights for the high-level male rats were significantly higher than for the control rats during the latter part of the study, but this response was not considered toxic. Decreased glucose levels were observed in exposed female rats, compared with the control group, and dose-related elevations in serum glutamic pyruvic transaminase levels were observed primarily in the females, but the absolute values were within normal limits for all animals except two. A report from the Albert Einstein College of Medicine on the neurological condition of the subject rats is included. Tables and graphs. (supported by API)
■ *API Medical Research Publication* (5/23/80) (86 p.).
Source: API Library

28-31211
[Bio/dynamics Inc. Project No. 79-7342] /A Dominant-Lethal Inhalation Study with Benzene in Rats [and Effects on Male Reproductive Performance]. Benzene exposure at 1, 10, and 30 ppm did not affect male reproduction performance or have a demonstrable dominant-lethal mutagenic effect. In a high-dose group (300 ppm), an observed increase in mean number of dead implants and mean mutagenic ratio were primarily related to the performance of a single male; the group values for mean dead implants and mutagenic ratio were not statistically significant. No gross or histopathologic lesions were noted during the evaluations of the testes in this high-dose male. The benzene was administered to rats of proven fertility for 6 hr/day, 5 days/week, for a ten-week treatment period. On the last day of dosing, each male was caged with two virgin, untreated females for seven consecutive days, after which the females were removed and replaced with another two untreated females. Tables. (study conducted for API and the Chemical Manufacturers Association)
■ *API Medical Research Publication* (Dec. 1980) (22 p.).
Source: API Library

28-31212
[Bio/dynamics Inc. Project No. 79-2425] /An Inhalation Female Fertility Study with Benzene in Rats showed that benzene administered by inhalation at 1, 10, 30, or 300 ppm to female rats during premating and mating periods and during the ensuing gestation and lactation periods, had no effect on reproduction. No treatment effects were seen in the 30 and 300 ppm dose level-pup survival data during lactation and during gross postmortem evaluations of these pups on day 21. The benzene was administered 6 hr/day, 5 days/week, during a ten-week premating treatment period and ensuing mating period. Mated females were exposed 6 hr/day during gestation to day 20; daily exposure was resumed on day 5 of lactation and continued until weaning (day 21 of lactation). Tables. (study conducted for API and the chemical Manufacturers Association)
■ *API Medical Research Publication* (Dec. 1980) (28 p.).
Source: API Library

28-31344
[Litton Bionetics Inc. Project No. 21141-02] /Mutagenicity Evaluation of Gasoline, API PS-6 Fuel (Unleaded) in the Mouse Dominant Lethal Assay. Final Report. A test gasoline (API PS-6 unleaded fuel), administered to CD-1 male mice via inhalation exposure at 400 or 1600 ppm for 6 hr/day, 5 days/week, for 8 weeks, did not reduce the fertility of the mice nor induce dominant lethal mutations in the sperm cells of the mice. The test gasoline did not significantly increase pre- or post-implantation loss of embryos in female mice mated with exposed males when statistically compared with those mated with negative control mice exposed to filtered air in identical dosing regimens. The assay sensitivity was confirmed by the significant induction of dominant lethal mutations in positive control mice injected once intraperitoneally with 0.3 mg/kg of triethylenemelamine. Tables. (supported by API)
■ *API Medical Research Publication* (June 1980) (31 p.).
Source: API Library

28-31345
[Litton Bionetics Inc. Project No. 21141-03] /Mutagenicity Evaluation of Jet Fuel A in the Mouse Dominant Lethal Assay. Final Report. Jet fuel A, administered to CD-1 male mice via inhalation exposure of 100 and 400 ppm for 6 hr/day, 5 days/week, for 8 weeks, did not reduce the fertility of the mice nor induce dominant lethal mutations in the sperm cells of the mice. Jet fuel A did not significantly increase pre- or post-implantation loss of embryos in female mice mated with exposed males when statistically compared with those mated with negative control mice exposed to filtered air in identical dosing regimens. The assay sensitivity was confirmed by the significant induction of dominant lethal mutations in positive control mice injected once intraperitoneally with 0.3 mg/kg of triethylenemelamine. Tables. (supported by API)
■ *API Medical Research Publication* (Dec. 1980) (38 p.).
Source: API Library

28-31346
[Litton Bionetics Inc. Project No. 21141-04] /Mutagenicity Evaluation of Diesel Fuel in the Mouse Dominant Lethal Assay. Final Report. Diesel fuel, administered to CD-1 male mice via inhalation exposure at 100 or 400 ppm for 6 hr/day, 5 days/week, for 8 weeks, did not reduce the fertility of the mice nor induce dominant lethal mutations in the sperm cells of the mice. The diesel fuel did not significantly increase pre- or post-implantation loss of embryos in female mice mated with exposed males when statistically compared with those mated with negative control mice exposed to filtered air in identical dosing regimens. The assay sensitivity was confirmed by the significant induction of dominant lethal mutations in positive control mice injected once intraperitoneally with 0.3 mg/kg of triethylenemelamine. Tables. (supported by API)
■ *API Medical Research Publication* (Dec. 1980) (38 p.).
Source: API Library

28-31347

[Litton Bionetics Inc. Project No. 21141-05] /Mutagenicity Evaluation of Toluene in the Mouse Dominant Lethal Assay. Final Report. Toluene, administered to CD-1 male mice via inhalation exposure at 100 or 400 ppm for 6 hr/day, 5 days/week, for 8 weeks, did not reduce the fertility of the mice nor induce dominant lethal mutations in the sperm cells of the mice. Toluene did not significantly increase pre- or post-implantation loss of embryos in female mice mated with exposed males when statistically compared with those mated with negative control mice exposed to filtered air in identical dosing regimes. The assay sensitivity was confirmed by the significant induction of dominant lethal mutations in positive control mice injected once intraperitoneally with 0.3 mg/kg of triethylenemelamine. Tables. (supported by API)
■ *API Medical Research Publication* (Jan. 1981) (39 p.).
Source: API Library

28-31614

The Effect of Sampling Duration on the Concentration of Particulate Organics Collected on [Gelman AE] Glass-Fiber Filters with high-volume samplers was studied at 6 hr, 24 hr, and 1 week sampling times followed by sequential Soxhlet extraction with cyclohexane, dichloromethane, and acetone and gas chromatographic analysis of each extract on 12 ft stainless steel columns packed with 6% Dexsil 300 on Chromosorb W, from 165 to 285 °C at 4 °C/min and flame-ionization detection. The acetone extracts appeared to be relatively unaffected by changes in sampling time, but the cyclohexane and dichloromethane extracts showed effects of a sampling-time dependence. Comparisons of the chromatograms of the cyclohexane extract showed losses of aliphatic hydrocarbons below n-nonadecane associated with longer sampling times; and increases in the number and mass of aliphatic hydrocarbons above n-nonadecane for the longer sampling times. Data from this study and published data suggest that there is a need to conduct shorter term sampling when studying specific compounds and extracts of particulate organic matter. Graphs, tables, chromatograms, and 13 references. (Supported by API)
■ G. P. Schwartz; J. M. Daisey; P. J. Lioy (N.Y. Univ. Med. Cent.), *Journal, American Industrial Hygiene Assocication* 42(4):258-63 (Apr. 1981).
Source: Not available from API

28-31628

In-Vivo and In-Vitro Mutagenicity Studies. n-Hexane (Hexane UV). Final Report. n-Hexane did not cause a mutagenic response in male albino rates exposed in the Mouse Lymphoma Forward Mutational test to doses of 110-240 µg/ml, but rats exposed in the In-Vivo Bone Marrow Cytogenetic Assay to 0, 150, 300, and 600 ppm concentrations of n-hexane, via inhalation for five consecutive days, exhibited chromosomal aberrations, particularly chromatid breaks and markers, at all treatment levels when compared with the negative control group. This result confirmed the findings of an earlier study which employed these tests plus the Ames test. All tests were conducted for the API by Hazleton Laboratories America Inc. in 1980. Tables and photomicrographs.
■ *API Medical Research Report* (5/5/81) (85 p.).
Source: API Library

28-31863

Review of the Status of Biological Aspects of Coal Conversion Technology, 1977-Present. Final Report. An Enviro Control Inc. survey of research sponsored by federal agencies and private organizations from 1977 through the present covers human health aspects, including toxicology, carcinogenicity, mutagenicity, and teratogenicity tests on materials (e.g., phenols, benzene, and polynuclear aromatic hydrocarbons) from coal gasification and liquefaction processes such as the Solvent-Refined Coal-I and -II processes; industrial hygiene surveys and the development of analytical techniques, instrumentation, and models to make the workplace safe; and medical surveillance and personnel protection and safety projects, such as those conducted by the Oak Ridge National Laboratory for an H-Coal pilot plant; environmental aspects, including projects on the identification, and environmental and ecological assessments of air, water, and solid pollutants associated with coal conversion processes, such as P. Cunningh's (Argonne Natl. Lab.) study, "Fractionation of Organic Samples from a HYGAS Pilot Plant"; risk assessment aspects; and recommendations for future research, notably in relation to the EPA premanufacturing notification rules. Tables and 304 references. (sponsored by API)
■ *API Medical Research Publication* (June 1981) (113 p.).
Source: API Library

28-31864

In-Vivo and In-Vitro Mutagenicity Studies. Paraffinic Oil 78-9, 70 SUS/100 °F. Final Report. Studies of the test material by Hazleton Laboratories America Inc. showed no mutagenicity in strains of *Salmonella typhimurium* at dose levels of ~ 40,000-240,000 µg/plate in the presence or absence of metabolic activation in the Ames and suspension tests. These tests are not conclusive for the mutagenic potential of the test material, because of the material's insolubility and inability to demonstrate toxicity for the bacterial strains. There was a mutagenic response at 8830-120,000 µg/ml, with or without metabolic activation, in the Mouse Lymphoma Forward Mutational test, although no dose-related effect was demonstrated. In-vivo Bone Marrow Cytogenetic assay showed no significant increase in structural mutations in the bone marrow cells of 50 male and female albino rats exposed to 500, 1000, and 2000 µg/kg of the compound for five consecutive days. Tables and photomicrographs. (sponsored by API)
■ *API Medical Research Publication* (6/19/81) (91 p.).
Source: API Library

28-31865

In-Vivo and In-Vitro Mutagenicity Studies. Paraffinic Oil 79-3, 350 SUS/100 °F. Final Report. In the Ames test and the suspension modification of the Ames test, the compound and dilutions prepared in ethyl acetate or in dimethyl sulfoxide were neither toxic nor mutagenic to *Salmonella typhimurium* strains at doses of 40,000-240,000 µg/plate in the presence or absence of metabolic activation. The material's insolubility and inability to demonstrate toxicity suggest that the mutagenic potential of Paraffinic 350 SUS/100 °F cannot be fully evaluated in these tests. In the Mouse Lymphoma Forward Mutational test, there was a nondose-related weak mutation response, with or without metabolic activation, at 908-12,712 µg/ml. In the in-vivo Bone Marrow Cytogenetic assay, the bone marrow chromosomes from 50 male and female sexually mature albino rats, which were exposed to 500, 1000, and 2000 mg/kg of the compound for five consecutive days, showed significantly increased aberration occurrences at the highest dose level. There were no observed dose-related response, no mortality after oral administration of the compound, and no significant decrease in mean mitotic indexes. These tests were conducted by Hazleton Laboratories America Inc. for API. Tables and photomicrographs.
■ *API Medical Research Publication* (6/19/81) (94 p.).
Source: API Library

28-31866

In-Vivo and In-Vitro Mutagenicity Studies. Paraffinic Oil 79-4, 550 SUS/100 °F. Final Report. Tests conducted by Hazleton Laboratories America Inc. showed that the test material was not mutagenic at dosages of 35,000-210,000 µg/plate, with or without metabolic activation, in the *Salmonella typhimurium* mammalian microsome plate and suspension assays. The mutagenic potential of Paraffinic 550 SUS/100 °F cannot be fully evaluated in the Ames test, because of the material's insolubility and inability to demonstrate toxicity. In the Mouse Lymphoma Forward Mutation test, the application of 9185-128,590 µg/ml of the compound on the cultures showed a nondose-related, nonlinear mutagenic response in the presence of metabolic activation. The solvents used in both tests were ethyl acetate and dimethyl sulfoxide, neither of which was toxic to the cells. In-vivo Bone Marrow Cytogenetic assays of 30 male and 30 female sexually mature albino rats, which were orally administered 500, 1000, or 2000 µg/kg of the test material for five consecutive days, showed no significant increase in structural mutation in the bone marrow cells. Tables and photomicrographs. (sponsored by API)
■ *API Medical Research Publication* (6/19/81) (92 p.).
Source: API Library

28-31867

In-Vivo and In-Vitro Mutagenicity Studies. Paraffinic Oil 79-5, 800 SUS/100 °F. Final Report. Studies by Hazleton Laboratories America Inc. of Paraffinic Oil 79-5, 800 SUS/100 °F and of a sample diluted with dimethyl sulfoxide showed that the test compound was not mutagenic in the *Salmonella typhimurium* mammalian plate (Ames test) and suspension assays at dose levels of 40,000-240,000 µg/plate in the presence or absence of metabolic activation. The insolubility of the test material and the inability to demonstrate toxicity for the bacterial strains suggest that the mutagenic potential of the sample cannot be fully evaluated in these tests. In the Mouse Lymphoma Forward Mutational test, there was a mutagenic response at 9288-130,032 µg/ml doses, although no dose-related effect was demonstrated in the presence or absence of metabolic activation. The in-vivo Bone Marrow Cytogenetic assay showed no significant increase in structural mutations in the bone marrow cells of 50 male and female albino rats exposed to 500, 1000, and 2000 µg/kg of the test material for five days. Tables and photomicrographs. (sponsored by API)

■ *API Medical Research Publication* (6/19/81) (92 p.).
Source: API Library

28-31868

In-Vivo and In-Vitro Mutagenicity Studies. Paraffinic Oil 78-10, 150 SUS/100 °F. Final Report. Concentrated Paraffinic Oil 78-10, 150 SUS/100 °F or 0.1 ml. of the solution prepared in dimethyl sulfoxide was not mutagenic in the *Salmonella typhimurium* mammalian microsome plate incorporation and suspension assays in the presence or absence of metabolic activation at doses of 40,000-240,000 µg/plate of the test material. The mutagenic potential of the test sample cannot be fully evaluated in the Ames test, because of the compound's insolubility and inability to demonstrate toxicity for the bacterial strains. The Mouse Lymphoma Forward Mutational test showed a mutagenic response, with or without metabolic activation, at doses of 8898-124,572 µg/ml. of the test material. A dose-related effect was not demonstrated. In-vivo Bone Marrow Cytogenetic assay showed no significant increase in structural mutations in the bone marrow cells of 50 male and female albino rats exposed for five days to 500, 1000, and 2000 µg/kg of the compound. These tests were conducted by Hazleton Laboratories America Inc. for API. Tables and photomicrographs.

■ *API Medical Research Publication* (6/19/81) (91 p.).
Source: API Library

28-33073

Development and Persistence of Adaptation to Ozone Exposure in Ozone-Sensitive Southern California Residents. Studies on 11 volunteer Angeleno subjects, who, except for one, adapted to ozone after exposure to ~ 0.47 ppm (UV calibration method) for 2 hr/day at 31 °C and at 40% relative humidity (plus subjection to intermittent moderate exercise and heat stress) for four successive days in the first week and for one day in each of the following five weeks after four- and seven-day rest intervals suggested that adaptation to ozone is not an important factor to consider in setting air quality standards. Adaptation was partially lost and almost completely lost after four and seven ozone-free days, respectively. In general, the effects of the four successive daily exposures were consistent with those from previous studies, with the most severe lung function response occurring on the second day, and adaptation developing by the fourth day. One subject's failure to adapt could be attributed to a persistent bronchial infection. Graphs, tables, and 16 references.

■ W. S. Linn; J. D. Hackney (Rancho Los Amigos Hosp. Inc.), *Coordinating Research Council, Inc. - Air Pollution Research Advisory Comm. P; CRC/APRAC Project CAPN-31-79(1-80)* (8/20/81) (28 p.).
Source: API Library

28-70300

[API Project SPS-5-UOC(858/305)] /Comprehensive Analyses of Shale Oil Products: Second Study...Final. Standard Oil Co. (Ohio)-refined samples of crude and hydrotreated shale oils, weathered gas feedstock, JP-5 precursor and product, JP-8 precursor and product, DFM precursor and product, hydrotreated residue (No. 6 fuel oil), and sulfuric acid sludge (from DFM treatment) from the Paraho site, and a dehydrated, in-situ shale oil sample from the Rock Springs, Wyo., Site 9 field were analyzed by Union Oil Co. of California researchers for refractive index, molecular weight, bottom sediment and water, density, pour point, viscosity, and solvent fractionation, by simulated distillation with a gas chromatograph, and for true boiling point distillation plus blending of fractions. The samples were also analyzed for carbon, hydrogen, nitrogen, sulfur, and trace element contents. In hydrocarbon-type analyses, the IBP-400 °F cuts and the weathered gas feedstock were analyzed by a Union mass spectrometric procedure; the 400-650 °F and 650-850 °F cuts, jet fuels, and diesel fuels, by a Union mass spectrometric/liquid chromatographic procedure, and the 850 °F+ cuts, by liquid elution chromatography. The results are tabulated and/or graphed.

■ M. R. Winward; L. W. Burdett (Union Oil Co. Calif.), *API Publication* 79-39 (Jan. 1981) (74 p.).
Source: API Library

29-30299

Effects of Oil on Aquatic Organisms...A Review of Selected Literature indicates that spills of light refined oil into freshwater or marine ecosystems will probably cause extensive mortality of nonmobile susceptible species such as those of phytoplankton, and may not acutely affect unconfined, highly mobile species such as fish. Both freshwater and marine organisms can recover from exposure to oil spills within a year unless recontamination occurs. In general, the acute toxicity of aromatic hydrocarbons to algae is inversely related to vapor pressure and aqueous solubility. Except for the four- and five-ring PAHs, the relatively lethal effects to marine fish and invertebrates apparently increase from monoaromatics to alkylated aromatics to dinuclear aromatics. Unlike algae, fish and invertebrates do not appear to be acutely affected by PAHs. Other findings are the apparent susceptibility of larvae of fish and most crustaceans to water-soluble fractions of crudes; an inhibition in the growth of some species of diatoms and green algae, but not of some species of microflagellates; and the apparent tolerance of most mollusks and polychaete worms to oil contamination. Tables and 195 references.

■ S. L. Burks (Okla. State Univ.), *API Medical Research Publication* (72 p.).
Source: API HESD Information Specialist

29-32291

Experimental Studies of Hexacarbon Neurotoxicity. Final Report. In-vitro enzyme studies of rat nerve, brain, and liver homogenates pre-incubated with different concentrations of 2,5-hexanedione (HD) or methyl n-butyl ketone (MnBK) showed that these n-hexane metabolites inhibited the activities of glycolytic enzymes phosphofructokinase (PFK) and glyceraldehyde-3-phosphate dehydrogenase (GAPDH), but not those of lactic dehydrogenase, succinic dehydrogenase, or transketolase. Other findings were reduced levels of adenosine triphosphate and creatine phosphate in cat sciatic nerve homogenate pre-incubated with 2,5-HD; and no GAPDH activity inhibition by non-neurotoxic 2,4-HD. The addition of dithiothreitol in the incubation medium could protect enzyme activity, thus suggesting that these toxins might be acting on sulfhydryl groups required for normal enzyme activity. Data from a morphological study of rats exposed to 0, 5, 25, or 125 ppm n-hexane at various frequencies for up to 6 mo did not meet the criteria of hexacarbon neuropathology. Graphs and tables. (supported by API)

■ P. S. Spencer (Albert Einstein Coll. Med.), *API Medical Research Publication* (July 1982) (36 p.).
Source: API HESD Information Specialist

29-32356

The Carcinogenicity of Raw and Spent Oil Shales and Retort Oils. An API-supported study at the Kettering Laboratory, University of Cincinnati Medical Center, suggests that raw shale oils from Anvil Points, Parachute Creek, and the CA Tract in Colorado, and the White River formation in Utah, have essentially the same potential for carcinogenicity, which is apparently greater than that of crude petroleum. Following a preliminary study of the oils, groups of 50 6-8 wk old male mice were treated topically twice weekly with 50 mg each of three samples of raw shale (RS) and four samples of spent shale (SS), all ground and applied in mineral oil, four samples of retort shale oil (RO), neat mineral oil, or 0.05 or 0.15% BaP in mineral oil for 80 wk or until

a skin lesion was diagnosed grossly as a papilloma. All the shale oils produced malignant dermal tumors which were not seen with BaP treatment, thus initiating the development of fibrosarcoma in RO-treated mice, but not in BaP-treated mice; RO and BaP produced papilloma, keratoacanthoma, basal or squamous cell carcinoma, or fibroma, with RP also producing hemangioma; the very low incidence of neoplasm in RS- or SS-treated mice could be due to the low concentration of PAHs in the oils, or to the prevention of epidermal absorption of the oils by their tight bind to the particulates. Tables, photomicrographs, and graphs.
■ *API Medical Research Publication* (July 1982) (108 p.).
Source: API HESD Information Specialist

29-32357
Mutagenicity Evaluation of Certified Cyclohexane. Final Report. A Litton Bionetics Inc. study for API showed that cyclohexane was negative in inducing chromosomal aberrations in rat bone marrow cells, and was inactive in the mouse lymphoma forward mutation assay. The frequency of structural aberration and percentages of cells showing one or more aberrations in either female or male rats subchronically exposed (five daily exposures) by inhalation to 96.6, 307.2, or 1041.6 ppm cyclohexane did not differ significantly from those in the negative controls. Positive controls acutely treated with 1 mg/kg of triethylenemelamine showed a significant increase in structural aberration frequency. The increases in the frequency of numerical aberrations in female rats at low and medium doses and in pooled males and females at low dose were not dose-related. Mouse lymphoma cells treated with cyclohexane for 4 hr showed no significant change in the mutant frequency at the thymidine kinase locus; cyclohexane produced small increases in mutant frequency at up to 5 µL/mL under activation conditions, and was completely lethal at 10 µL/mL. Tables and diagrams.
■ *API Medical Research Publication* (Apr. 1982) (44 p.).
Source: API HESD Information Specialist

29-32358
Chronic Benzene Toxicology. Final Report. In a study conducted by the New York University Medical Center for API, Sprague-Dawley rats, and AKR, C-57 B1, and Charles River CD-1 mice exposed to 100 or 300 ppm benzene vapor for 6 hr/day, 5 day/wk, until death, showed peripheral lymphocytopenia at 300 ppm, with the mice also developing anemia and the rats showing a trend toward anemia. At 100 ppm, AKR mice showed less marked lymphocytopenic and anemic effects and rats showed nonstatistically significant trends. All mouse strains developed granulocytoses at 300 ppm, with three deaths of CD-1 mice attributed to premyelogenous or myelogenous leukemia, which indicated a causal relationship between benzene exposure and myelogenous leukemia since there have been no reports of spontaneous myelogenous leukemia in mice or rats. Pharmacokinetic data showed that AKR mice had a more severe toxic response and faster rates of benzene elimination than rats at either 100 or 300 ppm. The increasing rates of elimination at 300 ppm suggested metabolic enzyme induction. Tables and graphs.
■ S. Laskin; B. Goldstein (N.Y. Univ. Med. Cent.), *API Medical Research Publication* (8/22/78) (37 p.).
Source: API HESD Information Specialist

29-32359
Mutagenicity Evaluation of API 78-5, 100 SUS/100 °F Naphthenic Oil. Final Report. In a Litton Bionetics Inc. study conducted for API, bone marrow cytogenetic assays of ten male and ten female Sprague-Dawley rats subchronically exposed (five daily doses) to API 78-5, 100 SUS/100 °F naphthenic oil at 0.5, 1.67, or 5 g/kg, showed that the test material is negative in inducing chromosomal aberrations in bone marrow cells. The structural aberration frequencies, percentage cells with chromosomal aberrations, and numerical aberrations in treated rats were not significantly changed from those in 0.9% saline-treated negative controls. Rats acutely treated with 1.0 mg/kg of triethylenemelamine showed a significant increase in structural aberration frequency. The test material was nonmutagenic at the thymidine kinase locus in the lymphoma cells of mice at 4 hr exposure to 1.90-1000 nL/mL in the presence or absence of rat liver S9 metabolic activation. Tables and diagrams.
■ *API Medical Research Publication* (June 1982) (44 p.).
Source: API HESD Information Specialist

29-32360
Mutagenicity Evaluation of API 79-1, 90 SUS/210 °F Naphthenic Oil. Final Report. A rat bone marrow cytogenetic assay conducted by Litton Bionetics Inc. for API showed that API 79-1, 90 SUS/210 °F naphthenic oil was negative in inducing chromosomal aberrations in the bone marrow cells of male and female Sprague-Dawley rats at five daily subchronic exposures of 0.5, 1.67, or 5 g/kg. The frequency of structural and numerical aberrations, and the percentages of cells showing one or more aberrations were similar to the negative controls; positive controls acutely exposed on a one-time basis to 1.0 mg/kg of triethylenemelamine showed a significant increase in structural aberration frequency. In the mouse lymphoma forward mutation assay, the test material was considered nonmutagenic, since the lymphoma cells treated with 15.6-1000 nL/mL of the test material for 4 hr in the presence or absence of rat liver S9 metabolic activation did not show significant changes in the mutant frequency at the thymidine kinase locus. Tables and diagrams.
■ *API Medical Research Publication* (June 1982) (42 p.).
Source: API HESD Information Specialist

29-32615
API Project PS-8-GRD-6156/Carcinogenic Potential of Petroleum Hydrocarbons...Preparation and Analysis of Fractions. This Gulf Research & Development Co. report describes the preparation of the apparatus, reagents, and materials, and the procedures used to fractionate two whole crude oils into six boiling range fractions each (i.e., overpoint-120 °F, 120-350 °F, 350-550 °F, 550-700 °F, 700-1070 °F, and 1070 °F plus residua); and details the steps taken to separate the 350-1070 °F fractions into maltenes and asphaltenes by n-pentane extraction, to remove the resins and oils from the maltenes by the SARA (saturates, aromatics, resins, asphaltenes) technique and to isolate the saturates and aromatics from the oils by HPLC, and to remove the mobile phases from the saturates and aromatics fraction (350-550 °F) by distillation; and gives the analytical data for each of these fractions. Diagrams.
■ *API Medical Research Publication* (5/31/77) (17 p.).
Source: API HESD Information Specialist

29-32668
API Project PS-8-GST-(989-300) and Project PS-8-GRD (543) Carcinogenic Potential of Petroleum Hydrocarbons...Polynuclear Aromatics in Petroleum Fractions. In this Gulf Science & Technology Co. continuation of an earlier work by Gulf Research & Development Co. for API [Abstract No. 29-32615], analysis of two samples each of the original, oil, and aromatics fractions in the intermediate (550-700 °F) and high (700-1070 °F) boiling ranges by various chromatographic and UV spectroscopic procedures showed that one or all of the 550-700 °F fractions contained 19 PAHs with concentrations of < 0.1-148 ppm, and that one or all of the 700-1070 °F fractions contained 21 PAHs with concentrations of < 0.1-167 ppm. Some of the PAHs, including all of the fluorene and most of the methyl phenanthrene, remained in the 350-550 °F oil fractions; some identified PAHs, including pyrene and BaP, and unknown PAHs were found in the overpoint-350 °F fractions. High resolution MS of four resin fractions (550-1070 °F) showed the presence of heterocyclic species. Other findings included higher concentrations of sulfur, nitrogen, and oxygen in the aromatics fractions (550-1070 °F) than in the oil fractions of the same boiling ranges or in the saturate subfractions. Tables, graphs, and diagrams.
■ *API Medical Research Publication* (12/21/81) (54 p.).
Source: API HESD Information Specialist

29-32681
Characterization of the Effects of Beta-Propiolactone [(BPL)] and Anthralin on Transformation of BALB/3T3 Cells. A New York University Medical Center study of the enhancement effects of 0.25, 0.5, or 1.0 µg/mL of anthralin in combination with 2.5, 5.0, or 10 µg/mL of BPL or of BaP (all of which were dissolved in acetone) on transformation and relative plating efficiencies (RPE's) in BALB/3T3 cell cultures, showed that anthralin is active both as a promoter and a cocarcinogen with BPL, a direct-acting carcinogen. Metabolic activation of the

carcinogen is thus not necessarily a prerequisite for cocarcinogenesis. The findings included higher transformation frequencies (TF's) for both BPL and the indirect-acting carcinogen BaP with anthralin as promoter/cocarcinogen than for BPL alone or BaP alone; higher TF's for 2.5 µg/mL each of BPL and BaP with 0.25 µg/mL anthralin as a promoter than with anthralin as a cocarcinogen; greater RPE enhancement for BPL and BaP with anthralin as promoter/cocarcinogen than for BPL alone or BaP alone, suggesting a possible protective effect; increased RPE's without TF decreases, indicating the independence of transformation and toxicity; and the inactivity of anthralin as a complete carcinogen. Tables, graphs, photomicrographs, and 103 references. (supported by API)
■ T. J. Kneip; A. Segal (N.Y. Univ. Med. Cent.), *API Medical Research Publication* (Oct. 1981) (70 p.).
Source: API HESD Information Specialist

29-32752
N-Nitroso Compounds in Airborne Respirable Particulate Matter. A New York University Medical Center analysis, by colorimetric reaction with Griess reagent and by spectrophotometry, of 3 yr (1/1/79-1/1/81) weekly and 1 yr monthly samples of respirable suspended particulates (RSP) collected from New York City and Sterling Forest, N.Y., respectively, suggested the presence of N-nitroso compounds in RSP. Mean values were 143 and 47 pmole/cu m for the weekly and monthly samples, respectively, and the respective ranges were 0-346 and 0-103 pmole/cu m. Apportionment models traced these airborne compounds to oil burning, secondary or sulfate-associated particulates, and other unidentified sources in New York City; a seasonal pattern showed winter maxima and summer minima in the airborne concentrations of the particulate compounds. Although no N-nitrosamines were detected, there is a need to identify the specific structure of the possible N-nitroso compounds, since published studies have shown that the carcinogenic potency of four alkyl N-nitrosamines to animals is roughly equal to that of BaP, and that the molecular concentration of N-nitrosamines is about 18 times that of particulate-borne BaP in New York City. Tables, graphs, diagrams, and 241 references. (supported by API)
■ *API Medical Research Publication* (Mar. 1982) (251 p.).
Source: API HESD Information Specialist

29-32809
Review of the Status of Human Community Health and Environmental Health Effects Research of Pollutants from Coal Liquefaction, Oil Shale, and Petroleum Technologies. A Final Report prepared by Dynamac Corp., Enviro Controls Division for API, which is based in part on a published literature search of computerized data bases and on unpublished literature data, summarizes, by category (e.g., cellular toxicology, organ/system toxicology, teratogenicity/reproductive effects, carcinogenicity and mutagenicity, epidemiological and clinical studies), and by technology (i.e., coal liquefaction, oil shale, petroleum, and generic), most of the 731 recently completed or ongoing research projects related to the human community and environmental health effects of pollutants from these technologies. The report gives some of the conclusions drawn, such as the order of mutagenicity and of carcinogencity (in skin-painting studies), which in general is coal liquids > shale oil > petroleum products; and identifies five areas in which adequate data are lacking, i.e., the definition of standard fractions and wastes from these standard fractions for testing; determination of the effective dose of a pollutant; the development of process-specific markers to follow pollutants as a function of technology; monitoring of the degree of water, air, and food chain contamination from liquid fuel technologies; and qualitative data on the teratological and reproductive effects of liquid fuels.
■ *API Medical Research Publication* (Aug. 1982) (304 p.).
Source: API HESD Information Specialist

29-32827
Use of the Ames Assay To Detect Diurnal Variations in Fractions of Extracted Particulate Organic Matter [(POM)]. A New York University Medical Center research team collected 6 hr samples of total suspended particulates (TSP) during 2/3-16/79 in New York City 50 m above ground level at the Center's residence hall and extracted these with dichloromethane (DCM) and acetone [Abstract No. 28-30651]. These extracts were combined into 12 hr composites and tested with the Ames *Salmonella typhimurium* (strains TA-98 and TA-100) assay, modified by incorporating L-Histidine and biotin in the bottom, rather than the top agar. Without microsomal activation and compared with positive controls treated with 1-20 µg of 4 nitroquinoline-N-oxide, there were significant day-night differences for TSP and DCM-extractable POM, and in the number of revertants per cubic meter of sampled air for the DCM-soluble POM with strain TA-98. This strain is sensitive to direct-acting frame-shift mutagens. There was no day-night difference with strain TA-100 for the DCM- or acetone-extractable POM. This pattern and evidence of a weekday-weekend difference indicated that emissions from fossil fuel combustion were the most significant sources of airborne mutagens. Tables, graphs, and 29 references.
■ *API Medical Research Publication* (June 1981) (54 p.).
Source: API HESD Information Specialist

29-33065
[Project No. 1597] Acute Toxicity Tests of API 79-1, Naphthenic Oil (90 SUS/210 °F). Tests by Elars Bioresearch Laboratories Inc. on male and female albino rabbits, whose hairs were shaved and certain parts of their bodies incised (not deep enough to cause bleeding) or left intact for the application of occlusive gauze patches with 0.5 mL of the test material for 24 hr, or with 5 g/kg for 6 or 24 hr, showed slightly irritating primary skin effects at 0.5 mL, and no toxic or lethal effects at 5 g/kg, thus establishing an acute and subacute dermal LD_{50} of > 5 g/kg. The test material was nonsensitizing on the skins of male guinea pigs treated similarly with up to 2.0 mL for 6 hr; controls treated with 0.5 mL of a 0.05% dilution of chlorodinitrobenzene in 70% ethanol showed sensitizing effects. Primary eye irritation tests on albino rabbits showed nonirritating effects at 0.1 mL; acute oral tests on albino rats showed an LD_{50} of > 5 g/kg. Tables. (supported by API)
■ *API Medical Research Publication* (6/24/82) (61 p.).
Source: API HESD Information Specialist

29-33066
[Project No. 1599] Acute Toxicity Tests of API 79-4, Paraffinic Oil (550 SUS/100 °F), which were conducted for API by Elars Bioresearch Laboratories Inc., showed minimum irritation to the shaven, lightly incised, or intact skins of albino rabbits after the application of occlusive gauze patches with 0.5 mL of the test material for 24 hr. Subacute and acute dermal toxicity tests at 5 g/kg for 6 hr/day for three consecutive weeks and for 24 hr, respectively, showed an LD_{50} of > 5 g/kg. The test material was nonsensitizing on the skins of male guinea pigs treated with up to 1.0 mL for 6 hr; controls treated with 0.5 mL of a 0.05% dilution of chlorodinitrobenzene in 70% ethanol developed sensitization. Other results showed no irritation to the eyes of albino rabbits at 0.1 mL doses, and an oral LD_{50} of > 5 g/kg for albino rats. Tables.
■ *API Medical Research Publication* (6/24/82) (60 p.).
Source: API HESD Information Specialist

29-33067
[Project No. 1598] Acute Toxicity Tests, API 79-3, Paraffinic Oil (350 SUS/100 °F). Tests by Elars Bioresearch Laboratories Inc. for API showed that the application of occlusive gauze patches containing 0.5 mL of the test material on the shaven, lightly incised or intact skins of albino rabbits for 24 hr produced minimal irritation, an irritation score of 0.33. Similar skin treatments with 5 g/kg for 6 or 24 hr showed no toxicity and established subacute and acute dermal LD_{50} values of > 5 g/kg. The skins of male guinea pigs treated similarly with 0.1, 0.5, 1.0, or 2.0 mL for 6 or 24 hr showed no irritation, compared with controls treated with 0.5 mL of a 0.05% dilution of chlorodinitrobenzene in 70% ethanol, which was a sensitizer. Other tests showed no eye irritation to albino rabbits at 0.1 mL; and no toxic affects or mortality among albino rats that received 5 g/kg orally, thus setting an oral LD_{50} of > 5 g/kg. Tables.
■ *API Medical Research Publication* (6/24/82) (61 p.).
Source: API HESD Information Specialist

29-33068
[Project No. 1600] Acute Toxicity Tests, API 79-5, Paraffinic Oil (800 SUS/100 °F). Six tests made by Elars Bioresearch Laboratories Inc. for API showed minimum irritation to the shaven, lightly incised, or intact

skins of albino rabbits after 24 hr treatments with 0.5 mL of the test material placed on occlusive gauze patches. The test material produced neither systemic toxicity nor mortality at 5 g/kg for 6 hr/day, for three days for three consecutive weeks, or for 24 hr, thus setting subacute and acute dermal LD_{50} values of 5 g/kg; showed no sensitizing effects on the skins of guinea pigs at up to 2.0 mL for 6 hr, three times a week for three weeks vs. controls treated with 0.5 mL of a 0.05% dilution of chlorodinitrobenzene in 70% ethanol, which developed sensitization; produced no eye irritation on rabbits at 0.1 mL; and had an oral LD_{50} of > 5 g/kg in albino rats. Tables.
■ *API Medical Research Publication* (6/24/82) (60 p.).
Source: API HESD Information Specialist

29-33104
[Project No. 1601] Acute Toxicity Tests of API 78-9, Paraffinic Oil (70 SUS/100 °F). Tests conducted by Elars Bioresearch Laboratories Inc. for API showed that 24 hr treatments on the shaven, incised, or intact skins of albino rabbits with 0.5 mL of the test material placed on occlusive gauze patches produced minimum primary skin irritation. One dosage of 5 g/kg produced no systemic toxicity or mortality after 24 hr, thus setting an acute dermal LD_{50} of > 5 g/kg. Subacute dermal toxicity tests at 5 g/kg for varying lengths of time showed slight skin irritation and inflammation upon repeated exposures over 21 days. The test material produced no skin sensitization on guinea pigs at 1.0 mL for 6 hr vs. controls, which developed sensitization after treatment with 0.5 mL of a 0.05% dilution of chlorodinitrobenzene in 70% ethanol; produced no eye irritation in albino rabbits at 0.1 mL; and established an oral LD_{50} of > 5 g/kg in albino rats. Tables.
■ *API Medical Research Publication* (6/24/82) (61 p.).
Source: API HESD Information Specialist

29-33105
[Project No. 1602] Acute Toxicity Tests of API 78-10, Paraffinic Oil (150 SUS/100 °F), which were performed by Elars Bioresearch Laboratories Inc. for API, showed that the application of occlusive gauze patches with 0.5 mL of the test material for 24 hr on the shaven, incised, or intact skins of albino rabbits, produced minimum primary skin irritation; established an acute dermal LD_{50} of > 5 g/kg; and produced mild skin irritation and inflammation at 5 g/kg compared with controls in the subacute dermal study. Similar treatments of guinea pigs with 0.5 mL for 6 hr showed no skin sensitization effects, whereas controls dosed with 0.5 mL of a 0.05% dilution of chlorodinitrobenzene in 70% ethanol developed sensitization. The test material produced neither eye irritation in rabbit at 0.1 mL, nor toxicity or mortality in rats at 5 g/kg, thus setting an oral LD_{50} of > 5 g/kg. Tables.
■ *API Medical Research Publication* (6/24/82) (72 p.).
Source: API HESD Information Specialist

29-33106
[Project No. 1616] Acute Toxicity Tests of API 78-5, Naphthenic Oil (150 SUS/100 °F). In these tests made by Elars Bioresearch Laboratories Inc. for API, certain body areas in albino rabbits were shaved and their incised or intact skins treated with the test material placed on occlusive gauze patches. The results showed slight primary skin irritation at 0.5 mL after 24 hr treatment; no systemic toxicity at a single dosage of 5 g/kg after 24 hr exposure, thus setting an acute dermal LD_{50} of > 5 g/kg; and slight-to-moderate irritation and inflammation at 5 g/kg for varying exposure periods in the subacute dermal toxicity study. Guinea pigs treated similarly with 0.5 mL for 6 hr showed no skin sensitization, whereas controls dosed with 0.5 mL of a 0.05% dilution of chlorodinitrobenzene in 70% ethanol developed skin sensitization. The test material produced no eye irritation in rabbits at 0.1 mL, and established an acute oral LD_{50} of > 5 g/kg in albino rats. Tables.
■ *API Medical Research Publication* (6/24/82) (62 p.).
Source: API HESD Information Specialist

30-30223
Quantitative Analysis of Pathological Changes in the Cervical Spinal Cord of Control and n-Hexane-Treated Rats. Final Report. Analysis of samples of spinal cord segments of the cervical (C5-C7) regions obtained in two previous studies from untreated Sprague-Dawley rats and those exposed to 500 ppm of n-hexane for 22 hr/day, 7 days/wk, for 1-6 mo, confirmed that treated rats develop an increased incidence of Type 1 changes (i.e., degenerating axons) and Type 2 changes (i.e., dystrophic axons) in the gracile tracts of the cervical region, compared with the controls. Type 3 changes in the motor nerve cells were virtually absent in all the rats. These and other results, notably the significant variability in the number of changes in both treated and untreated rats, suggest the efficiency of using multiple sample points, combined with a quantitative analysis, to describe the magnitude of neuropathological changes in a given population. Tables and graphs. (supported by API)
■ P. S. Spencer (A. Einstein Coll. Med., Yeshiva Univ.), *API Medical Research Publication* (10/5/82) (15 p.).
Source: API HESD Information Specialist

30-30225
Histopathological Analysis of Spinal Cord of Animals Exposed to Gasoline. Final Report. Male and female albino rats exposed by inhalation to 2056 ppm of unleaded gasoline for 6 hr/day, 5 days/wk, for up to 18 mo did not show nervous system degeneration in the areas examined. Gasoline treatment might have increased the incidence and prominence of naturally occurring age-related changes, such as a widespread axonal dystrophy in the rostral regions of the fasciculi gracilis. These changes affect mainly the spinal cord and medulla oblongata and could have a significance to man. In aging humans, changes in the fasciculi gracilis similar to those noted in the treated and control rats may be associated with decreased vibratory perception and increased sensory abnormalities of the lower parts of the legs. (supported by API)
■ P. S. Spencer (A. Einstein Coll. Med., Yeshiva Univ.), *API Medical Research Publication* (10/5/82) (10 p.).
Source: API HESD Information Specialist

30-30226
Neuropathic Potential of n-Hexane in the Presence of Other Hexane Isomers. Final Report: [Phase 1 and Phase 2]. The first part of this two-part study conducted for API involved the exposure by inhalation of Sprague-Dawley rats to 125 or 500 ppm of n-hexane alone and to combinations of 125 ppm of n-hexane and 125, 375, or 1375 ppm of C_6 isomers for 22 hr/day, 7 days/wk, for 1 to 6 mo. In the second part, rats were exposed to the same test materials individually or in combinations at concentrations of 500 ppm for the same length of time. Rats exposed to 500 ppm of n-hexane alone or a mixture of 500 ppm each of n-hexane and C_6 isomers showed neuropathological changes, expressed as hindlimb weakness, after 2 and 6 mo; the changes induced by exposure to the 500:500 mixture were similar in degree to those caused by exposure to 500 ppm of n-hexane alone. No neuropathological changes were noted at concentrations below 500 ppm. Tables.
■ P. S. Spencer (A. Einstein Coll. Med., Yeshiva Univ.), *API Medical Research Publication* (10/29/82) (26 p.).
Source: API HESD Information Specialist

30-31037
Mutagenicity Evaluation of Six Petroleum Substances in an In-Vivo/In-Vitro Urine Assay. This study conducted by Litton Bionetics Inc. for API showed that urine from rats, which were orally administered 5 mL/kg each of mineral oil U.S.P., jet fuel A, shale oil RO-1, API-PS-8-76D5, API-PS-8-76C5, and API-PS-8-76C6, produced negative mutagenic results for all but shale oil RO-1 in the Ames assay. Shale oil RO-1 and the positive control 2-acetylaminofluorene (2-AAF) were mutagenic in *Salmonella typhimurium* strain TA-98. According to an addendum to the final report, in another urine assay, toluene (purified) and API-PS-8-76C6 dissolved in toluene were inactive in *Salmonella* strain TA-98, compared with 2-AAF. Tables and 12 references.
■ *API Medical Research Publication* (Nov. 1982) (68 p.).
Source: API HESD Information Specialist

30-31038
Final Report: Testing of API Petroleum Fractions for Genotoxicity in C3H/10T1/2 Cells. API-supported tests on two high-boiling (C_5 029202, D_5 029203) petroleum fractions dissolved in 0.5% dimethylformamide, on four intermediate-boiling (C_2 029188, C_3 029190, C_{3a}

029192, and C_{3b} 029194) fractions dissolved in 1% acetone or 0.5% dimethyl sulfoxide, and on one gaseous (D_1 P58) fraction in C3H/10T1/2 cells, showed that all but the C_2 029188 and C_3 029190 intermediate fractions produced an insignificant response in the mutation assay. The high-boiling fractions produced weak, variable genotoxic effects in the transformation test, suggesting that the cytotoxic effects of the emulsified fractions may have been unrelated to genetic damage. Positive results for the high-boiling fractions in both tests suggest that the fractions contain genotoxic constituents, possibly because the variability in emulsion preparation may have caused more stable emulsions of smaller particle size in these tests than in those for which no such results were observed. Tables and graphs.
■ C. Heidelberger (Univ. South. Calif. Cancer Cent.), *API Medical Research Publication* (2/25/83) (29 p.).
Source: API HESD Information Specialist

30-31525
Occupational Exposure to Benzene in the Petroleum and Petrochemical Industries. A survey covers analytical techniques for the estimation of benzene, including GC, IR absorption from CO and CO_2 lasers (which detect ≥ 3 ppb benzene), measurement of excretion products in urine, and the monitoring of benzene in exhaled air, and in ambient air by areal and personal dosimetry; exposure levels in the petroleum industry (in oil recovery, gas processing, refining, transportation, and marketing (at gasoline service stations)), and in the petrochemical industry (from process vents, storage tanks, pumps, and valves, in chlorobenzene, alkylbenzene, styrene, and cyclohexene production units), and from the use of benzene as a solvent; exposure reductions by administrative and engineering controls, improved industrial hygiene, substitution of other solvents, closed systems in refineries and petrochemical plants, vapor recovery on trucks, and automatic gages on tanks and laboratory ventilating hoods; evidence that current exposure levels, in contrast with past high levels, will not have deleterious health effects; and benzene emissions from the natural environment. Tables and 49 references.
■ N. K. Weaver (API); R. L. Gibson (Gulf Oil Corp.); C. W. Smith (Shell Oil Co.), in *Carcinogenicity and Toxicity of Benzene*, ed. M.A. Mehlman (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 4) pp. 63-75 (1983).
Source: Not available from API

30-31529
[This Compilation Based on the 1st API - Society of Toxicology "Toxicology of Petroleum Hydrocarbons" Symposium (Wash., D.C. 5/11-13/82)] contains 20 papers, which will be abstracted separately, on the toxicity and other effects of petroleum, oil shale, their fractions and products, petrochemicals, hydrocarbons, and a contaminant derived from a hydrogenation catalyst with respect to laboratory animals and humans, and on the mutagenicity of automotive particulate exhaust.
■ *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) (1984) (298 p.).
Source: API Library

30-31530
The Acute Toxicology of Selected Petroleum Hydrocarbons. Primary eye and dermal irritation and acute dermal and subacute dermal toxicity in rabbits, dermal sensitization in guinea pigs, and acute oral toxicity in rats were determined for unleaded gasoline, Jet Fuel A, diesel fuel, No. 2 fuel oil, five paraffinic lubricating base oils, two naphthenic lubricating base oils, new and used motor oils, and four No. 6 fuel oils by Elars Bioresearch Laboratories Inc. for API in May 1979-Dec. 1980. The middle distillates were the most irritating and toxic of the petroleum hydrocarbon product streams, and No. 6 heavy fuel oil having a 5.2 °API gravity and containing 1.2% sulfur was the most toxic of all the materials tested. The amount of material required to produce toxicity and the degree of irritation produced, however, indicated that these petroleum hydrocarbons are relatively nontoxic and nonirritating on acute exposure. Tables.
■ L. S. Beck; D. I. Hepler; K. L. Hansen (Elars Biores. Lab. Inc.), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 1-16 (1984).
Source: Not available from API

30-31531
[A Series of Nine] Inhalation Exposures of [Sprague-Dawley] Rats to Aerosolized Diesel Fuel having a 0.9-11 μ mass median diameter at 0.5-6 mg/L with 15-20% of the fuel in the vapor phase was carried out for 2-6 hr at a frequency of 1-3/wk altered pulmonary free cell number, pulmonary function, responsiveness in startle reflex assay, and histopathology. Single exposures for 2 hr produced a concentration-related decrease in respiratory frequency during exposure, transiently decreased responsiveness in a startle reflex assay test after exposure, and influx of granulocytes into the lungs for several subsequent days. Diagrams, graphs, and tables.
■ W. Dalbey; S. Lock; S. Garfinkel; R. Jenkins; R. Holmberg; M. Guerin (Oak Ridge Natl. Lab.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 13-25 (Dec. 1982).
Source: API Library

30-31533
Neurobehavioral Toxicology of Petroleum- and Shale-Derived Jet Propulsion Fuel No. 5 (JP5). Gavage studies with 1-24 mL/kg of petroleum-derived JP5 (P-JP5) refined from a mixture of 55% Iranian crude, 25% Nigerian crude, and 20% Qatari crude and with 24 mL/kg of Paraho shale oil JP5 (S-JP5), and inhalation studies with 1100 mg/cu m (decene-equivalent) or P-JP5 or 1600 mg/cu m of S-JP5 for 6 hr/day and 5 days/week for ~ 6 weeks were performed with Sprague-Dawley rats. After gavage with either P-JP5 or S-JP5, the body weight and food and water consumption were reduced for 2-3 days; and home-cage activity increased markedly 2.5-6 hr after dosing. After dosing with P-JP5, there were no significant differences between control and experimental animals in the performance of a skilled motor test. With inhalation exposure to either P-JP5 or S-JP5, water consumption increased after 8 days and remained elevated for the remainder of the 30 day study; and there were no significant effects on tissue morphology or on hepatic or renal serum chemistries. Peak amplitudes or latencies for somatosensory evoked potentials did not change significantly during the 30-day exposure to S-JP5. Graphs and 31 references.
■ V. Bogo (Armed Forces Radiobiol. Res. Inst.); T. A. Hill (Nav. Med. Res. Inst.), and Others, in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 17-32 (1984).
Source: Not available from API

30-31534
Comparison of the Subchronic Inhalation Toxicity of Petroleum and Oil Shale JP-5 Jet Fuels involved exposure of female C57BL/6 mice and male and female beagle dogs and Fischer 344 rats to petroleum or shale JP-5 vapor at of 150 and 750 mg/cu m. The major sign of toxicity after inhalation of both fuels was fatty livers in mice and dose related renal tubular necrosis in male rats, which also experienced decreased body weight gains and increased kidney-organ weight ratios and slightly increased BUN and creatinine levels. With the exception of mild nasal inflammation and liver cell vacuolization in rats exposed to shale JP-5, the effects of both fuels were comparable. Graphs, tables, and photomicrographs.
■ C. L. Gaworski (Univ. Calif. Irvine); R. H. Bruner (Air Force Aerospace Med. Res. Lab.); M. J. Cowan (Nav. Med. Res. Inst.), and Others, in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 33-47 (1984).
Source: Not available from API

30-31535
Long-Term Inhalation Studies with Raw and Processed [Oil] Shale Dusts. Rats and monkeys were exposed 6 hr/day, 5 days/wk, for 2 yr, to

10 and 30 mg/cu m of respirable dusts of raw and spent Anvil Points, Colo., oil shale. Among the tests (including body weight changes, pharmacotoxic signs, palpation for tumors, pulmonary function, and hematological and clinical chemistry determinations on some or all of the animals), the only significant changes were found on histopathological examination of tissues from the animals at termination, in lungs and peribronchial lymph nodes, and were similar for both dust types. The severity of the response was dose-related. Thus, accumulations of pigment and particle-laden macrophages in and around end airways, with inflammations in the lung, were noted in both species, but these findings were not considered to indicate progressive fibrosis. No evidence of carcinogenic action or systemic toxic effects was observed. Tables, photomicrographs, and 12 references.
■ H. N. MacFarland (Gulf Sci. Technol. Co.); W. B. Coate (Hazleton Lab. Am. Inc.); D. B. Disbennett (API); L. J. Ackerman (Exp. Pathol. Lab.), *Annals of Occupational Hygiene* 26(1-4):213-26 (1982; 1983).
Source: Not available from API

30-31536
Renal Cortical Degeneration [of Mice] Associated with Chronic Dermal Application of Petroleum- and Shale Oil-Derived Middle Distillates. Male and female C3H mice, exposed percutaneously to shale- and petroleum-derived middle distillates for up to 60 weeks, were observed at terminal necropsy to have severe treatment-associated renal lesions. During the experiments, mice demonstrated a marked elevation in water intake, relative to cyclohexane vehicle controls, but urine production did not, on average, show a proportionate increase at the midpoint of the experiment. Elevated water intake was apparently due principally to loss of the barrier function induced by middle distillate alteration of the epidermis. Measurements on urine samples just before the experiment ended showed statistically significant treatment-related differences between treated and vehicle controls in the urine volume (elevated), osmolality (reduced), and protein (elevated). Grossly, the affected kidneys were pale, nodular and shrunken. The lesions resembled infarcts but were characterized by a loss of tubular parenchyma, preservation of relatively normal glomeruli and an absence of both inflammatory cells and fibrosis. Two hypotheses for renal toxicity are the possible formation of nephrotoxic metabolites by the skin, or indirect damage induced by cyclic episodes of severe dehydration. (Abstract only)
■ J. M. Holland; J. R. Easley (Oak Ridge Natl. Lab.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) p. 76 (Dec. 1982).
Source: API Library

30-31537
Interactive Effects of JP-5 Vapor Exposure and Elevated Temperature on Renal Lesion Induction [in Rats]. In studies of the effects of repeated exposure to JP-5 vapors at elevated temperatures, male F344 rats were exposed to 2900 mg/cu m JP-5 for 6 hr/day, 5 days/wk, for 60 exposure days in 30 L leach chambers at 38 °C. The 4 exposure groups were control, fuel, heat-control, and fuel + heat. Twenty-four hr urine, blood and selected tissues were obtained at termination of exposures. Urine and serum assays were divided into three groups for multivariate analysis of variance (MANOVA): (I) Serum: CPK, AST, ALT, and glucose; (II) Urine: LDH, AST, Alk. Phos., and GGT activities; and (III) Urine volume, urine GGT, serum alb/glob, creatinine clearance, and fractional urea clearance. MANOVA (I) was significant for temperature (T) and for fuel (F) effects with no interactive effect. MANOVA (II) and MANOVA (III) were significant for the T, F, and interactive effects. Kidneys from the rats exposed to room air both at 25 and 38 °C were unremarkable. Kidneys from the rats exposed to the fuel at 25 °C had the usual proximal tubular necrosis, but the kidneys from rats exposed to fuel at 38 °C evidenced minimal cellular alterations. This lack of pathologic response with combined exposure is consistent with the interactive effects found in the biochemistry data. By both pathologic and biochemical criteria, the toxic response to JP-5 may be significantly altered according to environmental conditions. (Abstract only)
■ L. L. Pitts (Medtronic Inc., Physiol. Res. Lab. Div.); R. H. Bruner (Air Force Aerospace Med. Res. Lab.), and Others, in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) p. 77 (Dec. 1982).
Source: API Library

30-31588
Evaluation of Techniques for Mutagenicity Screening of Petroleum Hydrocarbons. Ames assays by Arthur D. Little Inc. of liquid petroleum samples PS8-80C2, PS8-76C3, and PS8-76D4, with flash points of -10 to +500 °F and boiling at 120-700 °F, and of tar samples PS8-76C5 and PS8-76D5, both having a flash point of 700 °F and boiling at 700-1070 °F, were unsuccessful, due either to the insolubility or separation of the samples in the top agar layer, or to a failure to observe complete positive responses with all the positive control chemicals, with or without metabolic activation, at the concentrations used. Tar samples could be solubilized in dimethyl sulfoxide by sonication, and liquid samples could be emulsified by vortexing, but they also separated from the top agar overlay. Modified preincubation assays incorporating sonication for tar samples and end-over-end rotation for liquid samples showed a mutagenic response with PS8-76D4 and PS8-76C5, and an enhanced toxicity with PS8-80C2. A modified Ames assay of gas sample PS8-80D1 showed toxicity at higher concentrations, but no mutagenicity at any of the doses tested. Tables.
■ *API Medical Research Publication* (8/16/82) (97 p.).
Source: API HESD Information Specialist

30-31598
Carcinogenic Potential of Petroleum Coke and Process Products. Tests by Elars Bioresearch Laboratories Inc. showed that the thrice a week application of 100 µL each of two solid petroleum cokes suspended at a 25% concentration in mineral oil, of a solid condensed emission, and of an aqueous condensed emission to the shaved skins of male and female mice, was not carcinogenic to the animals. The same amount of BaP in mineral oil at 0.05 and 0.15% applied twice a week was carcinogenic for the mice, which showed a dose-related increase in neoplastic cutaneous lesions. Compared with mice treated with 100 µL of mineral oil only, the coke suspension-treated mice survived just as long and had an increased incidence of epidermal acanthosis. As expected, both of these test groups developed low incidence of mammary gland tumors, subcutaneous fibrosarcomas, and other spontaneous neoplasms, which occurred with approximately equal frequency. Mice treated with the aqueous condensed emission had an increased incidence of hyperkeratosis, compared with untreated, shaved mice. Tables. (See also Abstract. No. 30-32011.)
■ *API Medical Research Publication* (2/24/82) (171 p.).
Source: API HESD Information Specialist

30-31603
The Effects of n-Hexane in Man and Animals. Translation into English of Erdoel Kohle, Erdgas, Petrochem. Brennst.-Chem. vol. 36, issue 1, 37 pages. A compilation of available information on the toxic effects of n-hexane shows that spark ignition engines and emissions of industrial solvents are the major sources of n-hexane in the environment; that the body retains ~ 7% of the n-hexane inhaled with air; that biotransformation occurs primarily in the liver and yields primarily the toxic metabolite 2,5-hexanedione, which is the cause of the observed neurotoxicity of n-hexane; that the main metabolite in animals is the less toxic 2-hexanol; that acute poisoning occurs at 1500-2500 ppm n-hexane by action on the central nervous system to produce a narcosis-like syndrome; that chronic low-level inhalation leads to symmetric polyneuropathology; that recovery occurs 6-36 mo after the end of exposure; and that the (West German) maximum workplace concentration of 100 ppm n-hexane may be too high to protect the worker. Table.
■ W. R. Delbrueck; A. Kluge (DGMK), *Dtsch. Ges. Mineraloelwiss. Kohlechem. e.V. Ber.; API Publication* 174-2 (May 1982; 1983) (133 p.).
Source: API HESD Information Specialist

30-31639
Final Report: A Comparison of SMR, PMR, and PCMR [(Standardized Mortality Ratios, Proportionate Mortality Ratios, and Proportionate Center Mortality Ratios)] in a Cohort of [Heavy Equipment Operator] Union Members, Potentially Exposed to Diesel Exhaust Emissions shows that the PMR or PCMR, based on a reasonably sufficient death ascertainment, is useful in hypothesis generation, but not

as a reliable measure of the magnitude of risk. A comparison of cause-specific SMRs, and of PMRs or PCMRs (those based on all deaths and those based on deaths known to the union only), was made by Environmental Health Associates Inc., for the Coordinating Research Council Inc., based on the mortality experience of a cohort of 34,156 union members [Abstract No. 30-31644]. For the entire cohort, both types of PMRs were poor indicators for cancer risk, and produced false positives. PCMRs, however, appeared better than PMRs for assessing the direction of site-specific cancer risk, but tended to overstate the magnitude of risk. Neither PMRs nor PCMRs, particularly those based on deaths known to the union only, were sensitive in detecting trends in dose-response analysis. Tables, graphs, and 21 references. See also Abstract No. 30-31645.
■ O. Wong; R. W. Morgan (Environ. Health Assoc. Inc.), *API Medical Research Publication* (5/11/83) (55 p.).
Source: API HESD Information Specialist

30-31644
Final Report: The Mortality among Members of a Heavy Construction Equipment Operators Union with Potential Exposure to Diesel Exhaust Emissions was studied by Environmental Health Associates Inc., for Coordinating Research Council Inc., by comparing the mortality experience of a cohort of 34,156 male members [Abstract No. 30-31639] and several subcohorts to that of the U.S. white males, adjusted for age and calendar time, by using the Standardized Mortality Ratio (SMR). Over-all mortality for the entire cohort and several subgroups was significantly lower than expected, but the study suggested several potential health problems. Mortality from liver cancer, emphysema, and accidental deaths was significantly high for the entire cohort. Although mortality from lung cancer for the entire cohort was similar to the expected incidence, a positive trend by latency was observed for lung cancer. For length of union membership, lung cancer SMR rose in magnitude from the less than 5 yr group to the 10-14 yr group, and remained constant thereafter; retirees showed significant mortality excess. Tables and 15 references. See also Abstract No. 30-31645.
■ *API Medical Research Publication* (4/18/83) (54 p.).
Source: API HESD Information Specialist

30-31645
Final Report: The Cancer Incidence among Members of a Heavy Construction Equipment Operators Union with Potential Exposure to Diesel Exhaust Emissions was studied by Environmental Health Associates Inc., for the Coordinating Research Council Inc., by comparing cases for 30,449 person-years between 1/1/72-12/31/77 with those cases included in the Cancer Incidence System of the California Tumor Registry, which covers the counties that form the San Francisco/Oakland Standard Metropolitan Statistical Area of the Census Bureau. The only statistically significant result was the excess of cancer of the oropharynx, but this excess was based on a small number of cases, and therefore was difficult to interpret. The observed incidences of digestive and respiratory cancers were slightly lower than, and identical to, the expected, respectively. An excess of kidney cancer was not statistically significant. Data were included on all malignant neoplasms, except for in-situ tumors and basal and squamous cell carcinoma of the skin. Tables and 11 references. See also Abstract No. 30-31639 and 30-31644.
■ *API Medical Research Publication* (4/18/83) (22 p.).
Source: API HESD Information Specialist

30-31646
The Carcinogenic Potential of Key Petroleum Products was studied at the Eppley Institute by skin painting Eppley Swiss male mice three times weekly with API Petroleum "Test Materials", identified as API 79-6, 78-2, 78-3, and 78-4, until death or sacrifice after 62 wk; and with PS-6-Gasoline (G) and PS-6-Gasoline 10% Bottoms (B) for 2 yr. Significant increases in skin ulceration, hyperkeratosis, and ectoabscesses and in multipathological disorders of the kidney, lung, liver, lymph node, and spleen were observed at different frequencies in treated groups, compared with the untreated control. The measured survival of the "Test Materials"-treated groups was 14:50 (28%), compared with 112:122 (92%) in the untreated group; and of the G- and B-treated groups, 2 and 10%, respectively, compared with 30% (29:97) in the untreated control. Treatment with 0.05 and 0.15% BaP was highly carcinogenic to these mice. Tables.
■ M. M. Jacobs (Univ. Nebr. Med. Cent., Eppley Inst. Res. Cancer Allied Diseases), *API Medical Research Publication* (5/10/83) (71 p.).
Source: API HESD Information Specialist

30-31647
An Organ Compartment Model of Lead Biokinetics. A biokinetic model of lead metabolism has been developed from data obtained in controlled single-dose exposures and chronic (12-45,000 μg of lead per 1 kg of body weight per day) lead exposures of infant and juvenile baboons for 2.5-15 mo. The model, which was fitted to blood and organ clearance data after single exposures, to dynamic blood lead measurements at constant exposures, and to steady-state blood lead-organ lead concentrations, consists of a short-term gut-extracellular fluid compartment, and blood, liver, kidney, and bone compartments. Blood lead is accurately fitted for periods of rapid change and tissue lead levels are well fitted for data from animals which have not been used to develop the model. The model has been tested for prediction of human organ burdens by comparison with a substantial body of autopsy data. The fits are satisfactory, but a respiratory compartment is desirable to complete the model. Tables, diagrams, and graphs.
■ T. J. Kneip; R. P. Mallan; N. H. Harley (N.Y. Univ. Med. Cent.), *API Medical Research Publication* (25 p.).
Source: API HESD Information Specialist

30-31854
Acute Toxicity Studies of Catalytically Cracked Clarified Oil, API Sample 81-15. Acute oral toxicity tests by Hazleton Raltech Inc. on male and female albino rats dosed with 3.20-7.81 g/kg of the tar-like liquid test material for up to 14 days showed LD_{50} values of 5.27 and 4.32 g/kg of body weight for the male and female rats, respectively, at respective 95% confidence limits of 4.03-6.95 and 2.65-5.47 g/kg. The pharmacotoxic signs observed included hypoactivity, ataxia, and prostration. Acute dermal tests on rabbits treated with 2.0 g/kg of the tar-like semi-solid material for 24 hr showed LD_{50} values of > 2.0 g/kg, and no signs of systemic toxicity. In the primary dermal irritation tests on rabbits at a dosage level of 0.5 mL, the tar-like semi-solid material could not be wiped off and caused increased skin irritation. Observations following a single application of 0.1 mL of the black viscous liquid test material to one eye of albino rabbits included the presence of brown or light brown material around the eye, and scattered red foci throughout the lungs. Tables.
■ *API Medical Research Publication* (Aug. 1982) (38 p.).
Source: API HESD Information Specialist

30-31982
Skin Carcinogenic Potential of Petroleum Hydrocarbons--1. Separation and Characterization of Fractions for Bioassay. In studies initiated by API, samples for bioassay [Abstract No. 30-32000], i.e., a domestic Gulf Coast naphthenic crude oil (C) and a foreign high-sulfur paraffinic crude oil (D), were distilled into straight-run fractions corresponding to naphtha, kerosine, gas oil, heavy vacuum gas oil, and asphalt. After removal of high-molecular-weight, condensed nonhydrocarbons and polar nonhydrocarbons, the kerosine, gas oil and vacuum gas oil fractions were further separated by HPLC on 5 ft columns packed with Biosil A into a saturated-hydrocarbon and an aromatics fraction. Several of the lower-boiling fractions and all of the distillate fractions boiling at > 550 °F were analyzed for individual 4-6 ring polynuclear aromatics by a complex scheme involving LC on deactivated alumina, thin-layer chromatography (TLC) on cellulose, HPLC on Bondapak C_{18}/Corasil, TLC on acetylated cellulose, and UV spectroscopy. Detailed analyses are tabulated for each distillate fraction. The work was performed by Gulf Research & Development Co. Graphs, tables, flow diagrams, and 16 references.
■ R. W. King (Sun Tech Inc.); S. C. Lewis (Exxon Corp.); S. T. Cragg (API); D. W. Hillman (Diamond Shamrock Corp.), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp.

123-38 (1984).
Source: Not available from API

30-31983
Statistical Evaluations in the Carcinogenesis Bioassay of Petroleum Hydrocarbon. Among the useful measures of mouse skin tumorigenicity is the time to response of a tumor, which provides a good measure of response at high exposure rates, but which is unreliable at low exposure rates. The Kaplan-Meier (KM) or product-limit method, is a generally useful graphical method for revealing distributions of the time-to-tumor, unbiased by differences in mortality rates among different test groups. KM-derived estimates of the median-time-to-tumor (T50) can serve as a useful summary statistic for carcinogenic potency estimation. Weibull model-based estimates of T50 generally show good accord with the T50 values from the KM method, and also allows estimation of confidence limits. Relative potency estimation, relative to a pure reference standard such as BaP, is also a useful measure of tumorigenicity. The application of these methods at the Oak Ridge National Laboratory is illustrated for ORNL skin tumorigenicity data and a skin-painting data base developed by the Kettering Laboratory under contract to the American Petroleum Institute. Tables, graphs, and 17 references.
■ J. M. Holland; E. L. Frome (Oak Ridge Natl. Lab.), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 151-66 (1984).
Source: Not available from API

30-31986
Acute Toxicity Studies [by Hazleton Raltech Inc.] of Hydrodesulfurized Kerosine, [API] Sample 81-07, on male and female albino rats, which were given a single dosage of 5.0 g/kg of the clear liquid test material, showed an oral LD_{50} of > 5.0 g/kg of body weight. At the 5.0 g/kg dosage level, there was no mortality; and pharmacotoxic signs included hypoactivity, excess salivation, and diarrhea. Acute dermal tests on male and female rabbits treated with 2.0 g/kg of the test material showed an LD_{50} of > 2.0 g/kg of body weight. Except for one rabbit that exhibited diarrhea, none showed signs of systemic effects. In the primary dermal irritation tests at a dosage level of 0.5 mL, the rabbits showed no visible lesions. Primary eye irritation tests on albino rabbits treated once with 0.1 mL of the test material showed no visible lesions, distended colon with gas in one animal, and scattered red foci in the lungs of another. Tables.
■ *API Medical Research Publication* (Aug. 1982) (33 p.).
Source: API HESD Information Specialist

30-31987
Acute Toxicity Studies of Vacuum Residuum, [API] Sample 81-13. Studies by Hazleton Raltech Inc. showed an oral LD_{50} of > 5.0 g/kg of body weight in male and female albino rats dosed with 5.0 g/kg of the tar-like semi-solid test material, pharmacotoxic signs (e.g., dark brown or black stains in the anal region), and no mortality at the 5.0 g/kg dosage level; a dermal LD_{50} of > 2.0 g/kg of body weight in male and female rabbits treated with 2.0 g/kg of the test material, the adherence of the material to the skin of the rabbits, and diarrhea among two female rabbits; no visible lesions among rabbits treated with 0.5 mL of the test material in the primary skin irritation tests; and necropsy observations such as alopecia, and black material surrounding the eyes of rabbits treated once in the eye with 0.1 mL of the very thick, dark brown liquid test material. Tables.
■ *API Medical Research Publication* (Aug. 1982) (33 p.).
Source: API HESD Information Specialist

30-31988
Acute Toxicity Studies of Light Catalytically Cracked Naphtha, [API] Sample 81-03. Acute oral toxicity tests by Hazleton Raltech Inc. on male and female albino rats dosed with 5.0 g/kg of the clear liquid test material showed no mortality at this dosage level; pharmacotoxic signs such as hypoactivity and diarrhea; no visible lesions upon necropsy; and an LD_{50} of > 5.0 g/kg of body weight. Acute dermal tests on male and female rabbits exposed to 2.0 g/kg of the test material showed no signs of systemic effects, except diarrhea in one rabbit; no visible lesions, except a mild distention of stomach with gas, and a mild skin irritation in one animal on necropsy; and an LD_{50} of > 2.0 g/kg of body weight. In the primary skin irritation tests at 0.5 mL, one rabbit showed subcutaneous hemorrhage; and there were no visible lesions on necropsy. Necropsy findings after primary eye irritation tests on rabbits at 0.1 mL showed no visible lesions in seven animals, a severe reddening in the right salivary gland of one animal, and a pitted cortical surface of the right kidney in another. Tables.
■ *API Medical Research Publication* (Aug. 1982) (32 p.).
Source: API HESD Information Specialist

30-31989
Acute Toxicity Studies of Vacuum Residuum, [API] Sample 81-14, by Hazleton Raltech Inc. on male and female albino rats orally dosed with 5.0 g/kg of the black tar-like semi-solid test material, showed no mortality at this dosage level; hypoactivity, diarrhea, and brown and black stains in the anal area; an LD_{50} of > 5.0 g/kg of body weight; and, on necropsy, no visible lesions, and a moderately dilated renal pelvis in one animal. Acute dermal tests on male and female rabbits treated with 2.0 g/kg of the tar-like semi-solid showed an LD_{50} of > 2.0 g/kg of body weight; no signs of systemic effects; and a reddening of the skin and muscle layers in one animal, and no visible lesions in the rest of the animals upon necropsy. Rabbits exposed at 0.5 g/kg showed no visible lesions in the primary skin irritation tests. Necropsy findings after primary eye irritation tests on rabbits treated with 0.1 mL of the brown tar-like semi-solid material showed alopecia, no visible lesions, and black or dark brown material around the treated eyes. Tables.
■ *API Medical Research Publication* (Aug. 1982) (33 p.).
Source: API HESD Information Specialist

30-31990
Acute Toxicity Studies of Sweetened Naphtha, [API] Sample 81-08. Acute oral toxicity tests by Hazleton Raltech Inc. on male and female albino rats dosed with 5.0 g/kg of the colorless liquid test material showed no mortality at this dosage level; pharmacotoxic signs such as diarrhea; an LD_{50} of > 5.0 g/kg of body weight; and necropsy findings such as mild dilation of the renal pelvis. Acute dermal toxicity tests of male and female rabbits exposed to 2.0 g/kg of the test material showed no signs of systemic effects; an LD_{50} of > 2.0 g/kg of body weight; and, on necropsy, no visible lesions in six rabbits, and the presence of mild crust in two animals. There were no visible lesions in rabbits treated with 0.5 and 0.1 mL of the test material in the primary skin and primary eye irritation tests, respectively. Tables.
■ *API Medical Research Publication* (Aug. 1982) (32 p.).
Source: API HESD Information Specialist

30-31991
Chronic Gasoline Toxicity [Tests on Laboratory Animals]. Groups of 100 each of $B6C3F_1$ mice and Fischer 344 rats were exposed to 67, 292, and 2056 ppm of an EPA unleaded reference motor fuel containing 2% benzene for 6 hr/day, 5 days/week, and 103-113 weeks; sacrificed at 3, 6, 12, and 18 mo; and examined. There were no consistent compound-related changes in mortality, pharmacotoxic signs, or hematological or biochemical indexes in either species. Both sexes of rats and male mice exposed to 2056 ppm showed significant reductions in body weight gains; gross necropsies showed compound-related increases in liver nodules and masses in female mice exposed to 292 and 2056 ppm. Male rats showed renal carcinomata in the cortex or near the renal poles at all dose levels, with some indications of a dose/response relationship. Female mice exposed to 2056 ppm showed hepatocellular adenomas and carcinomas, which were attributed to gasoline exposure, in the last 6 mo of the study. (See also 32-32227.)
■ H. N. MacFarland (Gulf Sci. Technol. Co.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 78-86 (Dec. 1982).
Source: API Library

30-31992
A Mechanistic Evaluation of the Pulmonary Toxicology of Nickel Subsulfide, a contaminant formed during the use of nickel-based

hydrogenation catalysts with sulfur-containing feeds, was performed with 8-10 week old male A/J and B6C3F$_1$ mice, which were intratracheally instilled with nickel subsulfide particles of 1.8 and 13.3 μm mean median dia at various dose levels. Single exposures to coarse particles gave a LD_{50} 12 times as great as that with fine particles, i.e., 50 vs. 4 mg/kg of body weight. Exposures once per week for four weeks gave a two-fold increase in the LD_{50} with coarse particles, compared with fine particles, i.e., 2 vs. 1 mg/kg. Repeated exposure to fine nickel subsulfide caused a cumulative lethality equivalent to the single-exposure regime. Lavaged macrophages from lungs of mice exposed to fine nickel subsulfide showed reduced cellular functions 14 days after a single intratracheal instillation. In-vitro studies showed that particulate nickel subsulfide was about ten-fold more toxic than soluble nickel chloride and nickel subsulfide solubilized in fetal bovine serum. Tables, photomicrographs, and 13 references.
■ G. L. Fisher (Battelle Columbus Lab.); G. L. Finch (Univ. Calif. Berkeley); Others, in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 49-60 (1984).
Source: Not available from API

30-31993

Oil and Related Contaminant Effects on Waterfowl Immune Defenses. Adult male mallards were orally given 1.0-12.0 ml/kg of body weight of a South Louisiana crude oil (SLCO), 1.0-12.0 ml/kg of Bunker C fuel oil (BCFO), 0.006-0.05 ml per 5 ml of daily drinking water of a dispersant (Corexit 9257), and various oil/Corexit combinations daily for 28 days. Resistance to bacterial challenge against *Pasteurella multocida* was significantly reduced in mallards that received 2.5 or 4.0 mg/kg of BCFO, but not in mallards receiving SLCO or 50:1 oil/Corexit mixtures. Most of the deaths from BCFO occurred within 24 hr of inoculation. The ingestion of oil or oil/Corexit mixtures had no effects on mallard antibody-producing capabilities, as indicated by direct spleen plaque cell-forming assays. Tables, graphs, and 24 references.
■ T. E. Rocke; T. N. Yuill; R. D. Hinsdill (Univ. Wis.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 97-106 (Dec. 1982).
Source: API Library

30-31994

Percutaneous Absorption of Benzene was studied by topically applying ~ 0.5 ml of carbon-14-labelled benzene at 5-10 μL/sq cm of skin to rhesus monkeys, a miniature pig, and adult men and analyzing urinary excretions over the next 2-4 days. In all cases, total absorption averaged < 0.2%, with 0.14% in the monkey, 0.09% in the miniature pig, and 0.05% in man. Peak excretion of radioactivity occurred in the first 2 hr and rapidly decreased thereafter. In-vitro studies with a diffusion chamber showed average absorptions of 0.19% in the monkey, 0.23% in the miniature pig, and 0.10% in man. Benzene absorption in vitro was a function of the contact time with the skin. The application of progressively larger doses that persisted on the skin for ≤ 3 hr showed 10-100 times greater absorption. Total absorption was directly related to the length of time benzene remained on the skin. The in-vitro method studied appeared to be a valid means of assessing the percutaneous absorption of benzene. Tables and graphs.
■ T. J. Franz (Univ. Wash. Seattle), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp, 61-70 (1984).
Source: Not available from API

30-31995

The Induction of Xenobiotic Metabolism in Rats on Exposure to Hydrocarbon-Based Oils. The application of Kuwait Crude Oil to the skin of male Sprague-Dawley rats increased dermal BaP 3-hydroxylase 15 fold and diphenyloxazole hydroxylase 6 fold, to levels comparable to those obtained with 3-methyl cholanthrene. The maximum induction of BaP 3-hydroxylase occurred 24 hr after application of the Kuwait Crude at 50 μl/9 sq cm of dorsal skin. The oral administration of 0.5 ml of Kuwait Crude increased BaP 3-hydroxylase activity 5 fold in the liver and lung, and 25 fold in the kidney and small intestine. The activity of 7-ethoxyresorufin O-deethylase was similarly induced, but aminopyrine N-demethylase remained essentially unchanged. Application of No. 2 fuel oil and a used crankcase oil induced BaP 3-hydroxylase and diphenyloxazole hydroxylase activity to a lesser extent. Dermal and hepatic BaP 3-hydroxylase activity was inhibited by a α-naphthoflavone, but not by metyrapone, suggesting that the induced enzyme is cytochrome P_{448}. In-vitro studies were also performed. Tables, graphs, and 18 references.
■ A. D. Rahimtula (Mem. Univ. Newfoundland) P. J. O'Brien; J. F. Payne (Fisheries & Oceans Can.), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 71-79 (1984).
Source: Not available from API

30-31996

Inhalation Cytogenetics in Mice and Rats Exposed to Benzene. Bone marrow cells were harvested from Charles River CD-1 mice and S-D, CD rats after five consecutive daily inhalation exposures of 1, 10, 30, or 300 ppm of benzene for 1, 2, 4, 8, or 13 weeks. There were no statistically significant increases in the frequency of chromosomal aberrations in the mice, compared with controls, with exposures of 1-30 ppm; exposures of 300 ppm significantly increased the percentage of aberrant cells and the average number of aberrations per cell, with the greatest number of aberrations occurring in the males. Rats exposed for 13 weeks showed no statistically significant increases in the frequency of chromosomal aberrations, for males, but showed significant over-all variations in the percentage of aberrant cells and in the average number of aberrations per cell for females. Table and 19 references. (Sponsored by the American Petroleum Institute)
■ T. A. Cortina; E. W. Sica; Others (Hazleton Lab. Am. Inc.), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 81-86 (1984).
Source: Not available from API

30-31997

Mutagenicity Evaluation of Petroleum Hydrocarbons. The American Petroleum Institute test battery, consisting of the Ames *Salmonella* assay, the L5178Y mouse lymphoma assay, and rat bone marrow cytogenetics were used to evaluate the mutagenic activity of various petroleum base stocks, fuels, and lubricants. Data for five paraffinic base stocks and two naphthenic base stocks were negative for the Ames assay, were equivocal for the mouse lymphoma assay, and were mainly negative for the rat bone marrow cytogenetics assay. Unleaded gasoline, kerosine, Stoddard solvent, and an unused motor oil composite showed no mutagenic activity in the three assays. Diesel fuel was clastogenic in rat bone marrow cells. Jet fuel A caused bone marrow cell chromosome aberrations in rats; No. 2 fuel oil caused similar results. Three crude shale retort oils containing 1800-4250 ppb of BaP and 1200-3650 ppb of benzo [a] anthracene gave positive Ames assay results and mouse lymphoma tests but negative results in the bone marrow cytogenetics assay. A battery of three mutagenicity tests appears to be more reliable in predicting carcinogenicity than the Ames test alone. Tables, graphs, and 20 references.
■ f C. C. Conaway (Texaco Inc.); C. A. Schreiner (Mobil Oil Corp.); S. T. Cragg (API), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 89-107 (1984).
Source: Not available from API

30-31998

Mutagenicity of Automotive Particulate Exhaust. The Influence of Fuel Extenders, Additives, and Aromatic Content was studied by collecting the exhaust from four spark-ignition vehicles operated on a chassis dynamometer and testing for mutagenicity in the Ames *Salmonella* bioassay. With the gasoline vehicle, estimates of genotoxic combustion products emitted per mile of vehicle operation were consistently lower (than for indolene alone) in all tests conducted on indolene (unleaded

gasoline) blended with ethanol or methanol and with a commercial gasohol; this was due to reductions in the particle emission rates and in the mass of organic material associated with the particles. Addition of MTBE increased the mutagenicity of particle extracts from one car, but had no effect on the others studied. Other tests, with a diesel engine coupled to an eddy-current dynamometer and operated at steady state on No. 2 diesel with 10% blends of ethanol, butanol, and SRC-II were less mutagenic than those obtained from control diesel fuel tests, but these fuel extenders led to increased emissions of genotoxic materials per mile of simulated driving. Tables and 20 references.

■ C. R. Clark (Inhalation Toxicol. Res. Inst.); W. F. Marshall (Bartlesville Energy Technol. Cent.); Others, in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 109-22 (1984).

Source: Not available from API

30-31999

The Occurrence and Natural History of Experimental Skin Tumors. A discussion covers the determination of tumorigenicity with the mouse skin bioassay; experimental squamous cell carcinomas and their evaluation by considering properties such as malignancy, latent period, pathogenesis, growth pattern, and the occurrence of metastases; the importance of considering the vehicle or solvent for a potential carcinogen, since toluene and mineral oil seem to induce different types of tumors in mice when used as solvents for BaP; and experimental data, taken from the Kettering Laboratory, on the induction of skin tumors in mice with crude oils, aromatics, and asphalts. Tables and photomicrographs.

■ K. L. Stemmer; W. Barkley (Univ. Cinci. Med. Cent.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 162-69 (Dec. 1982).

Source: API Library

30-32000

Skin Carcinogenic Potential of Petroleum Hydrocarbons--2. The Carcinogenesis of Crude Oil, Distillate Fractions and Chemical Class Subfractions, as previously obtained [Abstract No. 30-31982], was studied via classical mouse skin cancer bioassays, in which undiluted 50 mg samples were applied to the skin of C3H/HeJ mice twice per week for ≥ 18 mo or until tumors developed. Both crude oils, the low-sulfur, naphthenic C and the high-sulfur, paraffinic D, were moderately carcinogenic. Distillate fractions boiling at < 120 °F or > 1070 °F were inactive. Fractions boiling at 120-700 °F showed low activity. The 700-1070 °F fraction, known to contain relatively large amounts of carcinogenic polynuclear aromatics, was strongly carcinogenic. All aromatic subfractions were active and showed the full range of potencies. The lower-boiling saturates subfraction showed unexpected activity. There were substantial differences in activity between oils C and D and among the fractions from each. The work was carried out at the Kettering Institute under contract-sponsorship by API. Flow diagram and tables.

■ S. C. Lewis (Exxon Corp.); R. W. King (Sun Tech Inc.); S. T. Cragg (API); D. W. Hillman (Diamond Shamrock Corp.), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 139-50 (1984).

Source: Not available from API

30-32001

Statistical Analyses of Crude Oil and Shale Oil Carcinogenic Test Data were made by applying 50 mg each of the test oils directly to the skins of mice twice per week for 80 weeks, with observations for an additional 40 weeks; and by applying 5 mg of the samples (diluted in 30:70 by vol cyclohexane in acetone) three times per week for 78 weeks, with observations for an additional 22 weeks. The South Louisiana and Kuwaiti crude oils tested showed a wide range of carcinogenic potencies. Tests on five raw and two hydrotreated shale oils showed that hydrotreating or upgrading of shale oil significantly reduced its carcinogenic potency. In high-dose studies, the carcinogenicity of a 0.25% nitrogen, hydrotreated shale oil did not differ significantly from that of the two crudes, but a 0.33% nitrogen, hydrotreated shale oil was more potent than the crudes. High-dose studies also showed that hydrotreated shale oils exhibited higher nontumor mortality than crude oils. The experimental data analyzed were contributed by Tosco Corp., the American Petroleum Institute, and the U.S. Department of Energy. Tables, graphs, and 11 references.

■ R. M. Coomes; K. A. Hazer (Tosco Corp.), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 167-86 (1984).

Source: Not available from API

30-32002

Carcinogenic Potential of Petroleum Cokes and Process Products - 1. Coking Process and Sample Characterization. To assess worker exposure during petroleum coking operations, a delayed coke, a fluid coke, process water, and a solid condensable material accumulated in the discharge piping between the coke drum and the blowdown system were analyzed. The three coke samples consisted mainly of carbon and hydrogen and contained 1-4 wt % oxygen, sulfur, and nitrogen. The fluid coke and solid condensable material contained 56 ppm and 113 ppm, respectively, of mercury. All three solids contained 60-410 ppm of vanadium and nickel. Benzene extractables comprised 0.15 wt % of the fluid coke and 9.94 wt % of the condensable material. The delayed and fluid cokes contained 11-544 ppm of PAHs. BaP levels ranged from 10 ppm for the condensable material to 440 ppm for the delayed coke. The process water sample showed metal levels that were below EPA standards for drinking water and contained very low levels of four PAHs; only BaP was present at > 1 ppb.

■ J.D. Watts (Texaco Inc.) & D.I. Hepler (Elars Biores. Lab.) in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 225-26 (Dec. 1982)

Source: API Library

30-32003

Inhalation Teratology of Jet Fuel A, Fuel Oil, and Petroleum Naphtha in Rats. Pregnant female Charles River rats were exposed to 100 or 400 ppm each of jet fuel A, fuel oil, and petroleum naphtha from day 6 through day 15 of gestation in dynamic inhalation chambers. Jet fuel A produced signs of eye irritation at both exposures, but produced no evidence of test material-induced terata, variation in fetal sex ratio, embryotoxicity, or fetal growth inhibition. Petroleum naphtha produced signs suggestive of mucous membrane irritation at 400 ppm, but produced no changes in fetuses related to exposure. Exposure to fuel oil at both levels produced no changes in the adult females or the fetuses. Tables.

■ R. P. Beliles; F. J. Mecler (Litton Bionetics Inc.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 233-38 (Dec. 1982).

Source: API Library

30-32004

An Experimental Evaluation of Selected Petrochemicals for Subchronic Neurotoxic Properties. Adult Sprague-Dawley or Fischer-344 rats were exposed to various levels of selected petrochemicals, including n-hexane and its metabolites, compounds chemically related to n-hexane, and its metabolites, compounds chemically related to n-hexane, methyl ethyl ketone, methyl isoamyl ketone, cyclohexanone, and other high-volume petrochemicals. Rats inhaling 500 ppm of n-hexane for 22 hr/day, and 7 days/week showed central nervous system (CNS)/peripheral nervous system (PNS) giant axonal degeneration after 2 mo of exposure. No specific changes were found after 6 mo of similar treatment with 125 ppm of n-hexane or 500 ppm of n-hexane-free hexane isomers; the latter did not reduce or potentiate the neurotoxic effect of n-hexane when 500 ppm of each were administered at the same time. Treatment with 1500 ppm of gasoline for 6 hr/day and 5 days/week for 6-18 mo appeared to increase the incidence of degenerated nerve fibers normally found with age in the rostral gracile tract of the spinal cord, but did not induce specific pathological changes. (supported by API, Exxon Corp., Shell Int.

Res. Mij. B.V., Mobil Oil Corp., Texaco Inc., and Standard Oil Co.)
■ P. S. Spencer (Albert Einstein Coll. Med.), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 199-214 (1984).
Source: Not available from API

30-32005
The Inhalation Toxicity of n-Hexane and Methyl Ethyl Ketone. Fischer 344 rats were exposed to 3000, 6500, or 10,000 ppm of n-hexane or to 1250, 2500, or 5000 ppm of methyl ethyl ketone (MEK) for 6 hr/day, 5 days/week, for 13 weeks. There were no exposure-related decreases in body weight gains in male rats exposed to 10,000 ppm n-hexane, but there were no other clinical signs attributable to n-hexane exposure. Male rats exposed to 10,000 ppm of n-hexane showed slightly, but significantly, lower brain weights at necropsy. Rats exposed to 1250 and 2500 ppm of MEK showed an elevation in group mean body weight, and those exposed to 5000 ppm showed a depression in group mean body weight. There were no other clinical signs attributable to MEK exposure. Rats exposed to 5000 ppm of MEK showed increases in liver weight, the liver/body weight ratio, and the liver/brain weight ratio at necropsy. Clinical-pathological, special-neuropathological, and routine histopathological studies revealed no lesions that could be attributed to exposure to MEK. Tables, graphs, and photomicrographs.
■ F. L. Cavender (ToxiGenics Inc.); E. J. Gralla (Chem. Ind. Inst. Toxicol.); Others, in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 215-31 (1984).
Source: Not available from API

30-32006
Concentration-Related Effects of Hexane on Evoked Responses from Brain and Peripheral Nerve. Action potentials (APs) of the ventral caudal nerve, the brain stem auditory-evoked response (BAER) and the cortical somatosensory (SER)-auditory-, and visual-evoked responses were recorded for Fischer 344 rats exposed to 500, 1000, or 1500 ppm of n-hexane for 24 hr/day, 5 days/week, for 11 weeks. Concentration--related latency increases occurred within each sensory modality. The most severe unfavorable effects of hexane were on body weight, peripheral measures of grip strength and caudal nerve AP and SER; these measures did not show much, if any, recovery of function during post-exposure measurement. The latency of the fifth, but not the first, component of the BAER was prolonged, indicating an effect on central auditory tract conduction time. The amplitudes of response components varied and were rarely affected significantly by exposure. The hexanes studied contained ≥ 95% n-hexane with small amounts of methyl cyclopentane, 2-methyl- and 3-methylpentanes, and 2,2- and 2,3-dimethyl-butanes. Graphs and 34 references.
■ C. S. Rebert; S. S. Sorenson (SRI Int.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 272-85 (Dec. 1982).
Source: API Library

30-32007
A Comparison of the Rate of Development of Hexane Neuropathy in Weanling and Young Adult Rats. Weanling and young adult male Fischer 344 rats were exposed to 1000 ppm of 95% n-hexane containing small amounts of other hexane isomers for 24 hr/day for 11 weeks, initially at 7 days/week, and after 4 weeks, at 6 days/week. Within 2 weeks of exposure, rats of both ages showed significant reductions in body weight and grip strength. The subsequent effects of exposure on these indexes of toxicity were greater in the young adults than in rats first exposed as weanlings. The older rats showed earlier and more-severe signs of hindlimb flaccid paralysis. Rats of both ages showed similar effects on tail nerve conduction time and brain stem auditory-evoked response, with latencies increasing over the exposure period. Blood levels of 2,5-hexanedione, an apparent proximate neurotoxin, were 76 µg/mL in the weanlings and 70 in the adults after 1 week and 49 and 74, respectively, at the end of the exposures. Immature animals were not more susceptible to the effects of toxins than adults. Tables, graphs, and 21 references.
■ R. A. Howd; C. S. Rebert; J. Dickinson; G. T. Pryor (SRI Int.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 286-94 (Dec. 1982).
Source: API Library

30-32008
Neurobehavioral Effects of Subchronic Exposure of Weanling Rats to Toluene or Hexane. Male Fischer 344 rats were exposed to 900 or 1400 ppm of toluene or hexane for 14 hr/day and 7 days/week for 14 weeks. Toluene and hexane both inhibited weight gain. Hexane caused a neurotoxic syndrome characterized by reductions in grip strength, especially of the hindlimbs, motor activity, and startle response, and by increased latencies of several evoked potential components. Toluene did not cause the peripheral motor symptoms associated with hexane exposure, but it did depress a component of the brain steam auditory-evoked response and impaired the acquisition of a conditioned avoidance response and the acquisition of a tone-intensity discrimination task, when tested within hours after the daily exposure ended. Graphs and 19 references.
■ G. T. Pryor; J. Dickinson; R. A. Howd; C. S. Rebert (SRI Int.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC (Washington, DC: API)* pp. 295-304 (Dec. 1982).
Source: API Library

30-32009
Transient cognitive deficits and high-frequency hearing loss in weanling rats exposed to toluene. Weanling male Fischer 344 rats were exposed to 1200 or 1400 ppm of toluene for 14 hr/day, 7 days/week for 5 weeks, to determine whether learning deficits previously found [Abstract No. 30-32008] were due to residual pharmacologic effects or whether they were more-permanent cognitive deficits. Rats were trained with a conditioned avoidance response (CAR) during the last week of exposure or during the first or third week after exposures ended. None of the toluene-exposed rats was able to acquire the auditory CAR, although they learned visual and somesthetic CAR's. Hearing was unimpaired at 4 kHz, slightly impaired at 8 kHz, and markedly impaired at ≥ 12 kHz. The experimental data do not support the possibility that permanent cognitive deficits occur. Graphs and 16 references.
■ G.T. Pryor, J. Dickinson et al. (SRI Int.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 305-13 (Dec. 1982).
Source: API Library

30-32011
The carcinogenic potential of petroleum cokes and process products - 2. Bioassay. Similar to Abstract No. 30-31598.
■ D.I. Hepler, L.S. Beck & D.A. Wingate (Elars Biores.Lab.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 227-32 (Dec. 1982)
Source: API Library

30-32012
An Inhalation Teratology Study of Benzene in Rats. A study by Hazleton Laboratories America Inc. of four groups (40 rats per group) of pregnant Sprague-Dawley rats exposed to benzene vapor at concentrations of 1, 10, 40, and 100 ppm for 6 hr/day from days 6 to 15 of gestation showed that the test material did not produce teratogenic effects. Compared with controls, the exposed rats showed a slight fetotoxic effect at 100 ppm, as evidenced by a significant decrease in mean fetal body weights. The remaining material and fetal data did not show treatment-related effects. Tables. (supported by API)
■ W.B. Coate, A.M. Hoberman, R.S. Durloo (Hazleton Labs. Am. Inc.), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton,

N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 187-98 (1984).
Source: Not available from API

30-32013
The Myocardial Toxicity [to Dogs] of Selected Aliphatic Hydrocarbons, [Including Propane (P), n-Butane, Isobutane, and a Mixture of These Hydrocarbons (A-46), and Mixtures of Isobutane with Methyl Chloroform, Trichloroethylene, Methyl Ethyl Ketone, and Methyl Isobutyl Ketone]. A comparative study of the effects of the hydrocarbons on the myocardium was undertaken in anesthetized open-chest dog preparation. Concentrations of 5000-200,000 ppm were administered through the inlet of the respirator for 5 min periods, and the effects on the following parameters were studied: left ventricular pressure, myocardial contractility, aortic pressure, cardiac output, heart rate, stroke volume, and stroke work. With the exception of heart rate, all parameters studied were decreased under the influence of these hydrocarbons. A-46 was the most effective in reducing cardiac output, and P was the least toxic of the hydrocarbons studied. Isobutane was the most toxic to the myocardium with respect to contractility. No arrhythmogenic effect was observed with these agents. To investigate the possible use of these hydrocarbons, which are usually used with other solvents, as propellants, the interaction between isobutane and different solvents (methyl chloroform, trichloroethylene, methyl ethyl ketone, and methyl isobutyl ketone) was studied. No interaction was observed in so far as negative inotropic effect is concerned. (Abstract only.)
■ S. Zakhari (Dynamac Corp.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) p. 107 (Dec. 1982).
Source: API Library

30-32063
Validation of Behavioral Studies for Determining the Toxicity of Petroleum Hydrocarbons. The testing of such validation methods, as part of the API 1981-1982 Behavioral Studies, involved administering a single oral dose of 0, 0.3, 1.0, and 3.0 mg/kg of trimethyltin hydroxide, a neurotoxicant, to male C57BL/6N mice and performing active/passive avoidance learning test procedures and spontaneous locomotor activity tests, i.e., the Optovarimax graphical and digital recording of seven activity parameters. The active avoidance task was a sensitive and dose-responsive indicator of neurotoxity; the more-complicated discrimination tasks produced confounding results that were not useful in detecting neurotoxicity. Preliminary data from the spontaneous locomotor activity study showed that the parameters of total horizontal activity, rearings, jumps, and total time spent moving also provide dose--responsive, sensitive indications of neurotoxicity. The experimental data show that appropriately designed behavioral tasks can provide a sensitive and cost-effective means of assessing toxicant-induced functional changes, and that a first step in validating such studies has been achieved. Tables, graphs, and 15 references.
■ J. M. Cranmer (Univ. Arkansas Med. Sci.); S. C. Lewis (Exxon Corp.); R. K. TeVault (Standard Oil Co. (Indiana)); S. T. Cragg (API); P. T. Goad; D. L. Avery; R. D. Phillips, in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 337-53 (Dec. 1982).
Source: API Library

30-32064
API's Approach to Quality Assurance [in API Industrial Toxicology Studies]. The PS-47 Task Force on Quality Assurance, established by the API Toxicology Committee and consisting of member-company toxicologists, assists the API staff in drafting quality assurance policies and serves as a liaison between the Toxicology Committee and API quality assurance personnel. The PS-47 Task Force maintains a master schedule of active studies, performs study-specific quality assurance duties, and establishes and maintains quality assurance files and laboratory inspection files. An example is given of an API master schedule for immunologic activity of petroleum hydrocarbons.
■ R. M. Siconolfi (Gulf Oil Corp.); B. K. Hoover (API), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 271-79 (1984).
Source: Not available from API

30-32077
Toluene-Induced Hearing Loss in Rats Evidenced by the Brainstem Auditory-Evoked Response [(BAER)]. A group of 23 day old male Fischer 344 rats were exposed to 1200 ppm of toluene for 14 hr/day for 5 weeks and BAERs were evoked 2.5 mo after termination of exposure by subjecting the rats to 100 μsec clicks and 1 msec 8, 12, or 16 kHz tone pips. BAERs were recorded at eight intensity levels (0 to 60 db above threshold for elicitation of BAERs in control rats) by electrodes placed subcutaneously over the anterior portion of the nose and posterior skull. In the exposed rats, thresholds for the appearance of the BAER were elevated by 13-27 db over the controls exposed only to air. Latency/intensity functions with tone pip stimuli were consistent with the occurrence of sensorineural hearing loss in that latency differences between experimental and control rats increased with decreasing tone pip intensity. In the exposed rats, the amplitude of the fifth BAER component evoked by 16 kHz tones increased with intensity, but was greatly attenuated at high intensities. Weanling or young adult rats exposed to 1000 ppm of hexane for 24 hr/day for 11 weeks showed central nervous system conduction delays in the auditory pathway, but showed no peripherally mediated heating loss. The data are apparently the first to show that toluene is ototoxic [Abstract No. 30-32009]. Graphs and 16 references.
■ C. S. Rebert; S. S. Sorenson; Others (SRI Int.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 314-19 (Dec. 1982).
Source: API Library

30-32078
Depressant Effects Associated with the Inhalation of Uncombusted Diesel Vapor were exposed by exposing pathogen-free, male CD-1 mice to 0.204, 0.135, or 0.065 mg/L of diesel vapor for 8 hr/day for 5 consecutive days. The mice were tested before exposure, immediately after each 8 hr exposure, and 24 hr after the last exposure. There was a dose/response effect for the square-box activity test, the rota rod test, and the hot plate test, but there was no difference in test and control animal response in the corneal reflex and inclined plane tests. General observations showed vasodilation, ataxia, poor grooming habits, and, in some cases, tremor, the degree of the effects varying with the dose and the duration of exposure. Diesel vapor may thus act as a neurodepressant. The components of the diesel vapor studied were determined at Southwest Research Institute by GC/MS. Table, graph, and 16 references.
■ R. J. Kainz (Environ. Ind. Safety Consult.); L. E. White (Tulane Univ.), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 233-243
Source: Not available from API

30-32079
A Tale of Two Solvents: The Neurology of n-Hexane and Toluene. A review of literature on n-hexane and toluene neurology shows that acute exposure to high levels of either solvent is associated with a similar pattern of central nervous system depression. Chronic human exposure to solvent mixtures containing n-hexane causes a distal symmetrical polyneuropathy of the distal axonopathy type; the neuropathological features of the human neuropathy include giant-axonal changes that are readily produced in experimental animals exposed to n-hexane. Chronic, high-level exposure to toluene causes an unusual clinical profile indicating dysfunction of cerebellar and corticospinal pathways. There is no satisfactory animal model of toluene neurotoxicity. There are no reports of neuropathological changes caused by toluene in humans. 38 references.
■ H. H. Schaumberg (Albert Einstein Coll. Med.), in *Proceedings of the Symposium The Toxicology of Petroleum Hydrocarbons, May, 1982, Washington, DC* (Washington, DC: API) pp. 328-36 (Dec. 1982).
Source: API Library

30-32080

The Effects of n-Octane Exposure on Schedule-Controlled Responding in Mice. The effects of acute exposure to n-octane on schedule--controlled responding to milk presentation were studied with male Charles River CD-1 mice that were food-deprived to 80% of their free-feeding weight. Responding was maintained under a fixed-interval, 60 sec schedule of food presentation. With cumulative exposures, in which the concentration of octane was increased at 40 min intervals until responding stopped completely, octane generally had little effect at ≤ 1000 ppm; decreased the responding by ~ 50% at 3000 ppm and by 90% at 5600 ppm; and abolished the responding completely at 7000 ppm. With 4 hr exposures to single concentrations, octane had little effect at 500 ppm, increased responding slightly at 2000 ppm, decreased responding by 85% at 4000 ppm, and abolished responding completely at 7000 ppm. The concentration of octane expected to produce a 10% decrement in responding in 1 out of 1000 exposures was estimated to be < 13 ppm. Table, graph, and 27 references.

■ J. R. Glowa (Harv. Med. Sch.), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 245-53 (1984).

Source: Not available from API

30-32081

Human Sensory Response to Selected Petroleum Hydrocarbons. Human subjects were exposed to Stoddard solvent or "70 solvent" for 30 min periods to assess their sensory and irritant properties and their behavioral effects. The olfactory thresholds for Stoddard solvent and "70 solvent" were 0.002 and 0.003 mg/L, respectively. Only minor complaints of irritation were reported during 30 min exposures to 0.60 mg/L of Stoddard solvent or to 0.35 mg/L of "70 solvent". In a second study in which human subjects were exposed to mixed xylenes at 1-4 times the TLV, there was a higher incidence of mild eye irritation and a higher rate of eyeblink in exposed subjects than in controls, but there were no significant differences in respiration rates or in results of psychomotor function tests. The current TLV levels for the three solvents studied are reasonable for brief exposures and for the criteria used in this study, since the only consistent and significant effect of exposure was an increased incidence of eye irritation at the higher exposure levels.

■ L. Hastings; G. P. Cooper; W. Burg (Univ. Cinci. Coll. Med.), in *Applied Toxicology of Petroleum Hydrocarbons*, ed. H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, & M.L. Lane. (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 6) pp. 255-70 (1984).

Source: Not available from API

30-32296

Twenty-Eight-Day Dermal Toxicity Study in the Rabbit of Hydrodesulfurized Middle Distillate, [API] Sample 81-10, [by Borriston Laboratories Inc.]. Final Report. White male and female rabbits treated dermally with 200, 1000, or 2000 mg/kg/day of the test material three times a week for 28 days showed slightly, moderately, and severely irritating effects at the respective doses, as shown by the over-all mean irritation scores. Compared with untreated controls, the skins of the treated rabbits showed flaking, cracking, sloughing, swelling, thickening, or/and encrustation, and other treatment-related effects that increased in frequency and severity with increasing dosage. Histopathology showed skin lesions consisting of subacute acanthotic dermatitis with or without hyperkeratosis among some of the high-dose rabbits. One high-dose male rabbit showed minimal eschar formation. The deaths of one male and one female rabbit in the high-dose group on the second and third days of the study were considered treatment-related. Tables.

■ *API Medical Research Publication* (Aug. 1983) (134 p.).

Source: API HESD Information Specialist

30-32297

Twenty-Eight-Day Dermal Toxicity Study in the Rabbit of Hydrodesulfurized Kerosine, [API] Sample 81-07. Final report. Dermal tests by Borriston Laboratories Inc. on white male and female rabbits treated with 200, 1000, or 2000 mg/kg/day of API Sample 81-07 three times a week for 28 days showed that the first two dose levels were moderately irritating and the highest dosage was severely irritating. One death in the high-dosage group was considered treatment-related because of prior observations of cracked skin and a discharge from two places on the treatment area. All the rabbits were sacrificed due to severe dermal reactions. Gross pathology of the treated skins showed flaking, scab formation, necrosis, sloughing, fissuring, and other reactions. Histopathology of the treated skins of high-dose rabbits showed acanthotic dermatitis and hyperkeratosis accompanied by dermal microabscesses in six of 10 instances. Liver lesions consisted of acute multifocal necrosis. Tables.

■ *API Medical Research Publication* (Aug. 1983) (138 p.).

Source: API HESD Information Specialist

30-32298

Twenty-Eight-Day Dermal Toxicity Study in the Rabbit of Hydrodesulfurized Middle Distillate, [API] Sample 81-09. Final Report. A toxicity study by Borriston Laboratories Inc. on white male and female rabbits whose skins were treated with 200, 1000, or 2000 mg/kg/day of the test material three times a week for 28 days, showed minimal irritation at the lowest dosage, moderate irritation at the other two dose levels for male rabbits, and severe irritation at the highest dosage for female rabbits. Findings from gross pathology of the treated skins included flaking, cracking, thickening, dryness, swelling, and ulcerations. Histopathology of all high-dose rabbits showed skin lesions consisting of subacute acanthotic dermatitis and hyperkeratosis uncomplicated by underlying dermal infections, and liver lesions consisting of multifocal necrosis in one male rabbit. It was not established whether the dermal and other reactions shown by one male rabbit in the 1000 mg/kg group prior to its sacrifice were treatment-related. Tables.

■ *API Medical Research Publication* (Aug. 1983) (131 p.).

Source: API HESD Information Specialist

30-32347

Acute Toxicity Studies [by Hazleton Raltech Inc. of] Hydrodesulfurized Middle Distillate, [API] Sample 81-09, showed that the oral administration of 5.0 g/kg of the clear liquid to male and female albino rats produced hypoactivity and other pharmacotoxic signs, but no mortality. Necropsy findings included mildly enlarged cervical lymph nodes and no visible lesions. LD_{50} was > 5.0 g/kg of body weight. Acute dermal tests on male and female rabbits at 2.0 g/kg caused diarrhea in two males and impaired the use of the left hind leg of another. There were mild encrustation and reddening in the test sites, and no visible lesions on necropsy. LD_{50} was > 2.0 g/kg of body weight. Primary dermal irritation tests at 0.5 mL showed blanching in the abraded test sites of two rabbits and in the intact site of one, and subcutaneous hemorrhage in both sites of one rabbit. One death was not considered treatment-related. Necropsy showed no visible lesions, and mildly reddened lungs in one rabbit. Necropsy of rabbits whose eyes were treated with 0.1 mL of the test material showed no visible lesions. Tables and charts. See also Abstract No. 30-32298.

■ *API Medical Research Publication* (Aug. 1982) (99 p.).

Source: API HESD Information Specialist

30-32348

Acute Toxicity Studies [by Hazleton Raltech Inc. of] Hydrodesulfurized Middle Distillate, [API] Sample 81-10, of which 5.0 g/kg was orally administered to male and female albino rats, showed no mortality and an LD_{50} of > 5.0 g/kg of body weight. There were pharmacotoxic signs such as diarrhea; and severe corneal ulcerations, red exudates in the eyes, and no visible lesions, on necropsy. Findings from acute dermal tests on male and female rabbits treated with 2.0 g/kg of the yellow liquid included an LD_{50} of > 2.0 g/kg of body weight; diarrhea by one rabbit; and no visible lesions, and encrustation, reddening, or/and thickening of the test sites on necropsy. Findings from primary dermal irritation tests on rabbits at 0.5 mL included blanching, subcutaneous hemorrhage, fissuring, and desquamation in the test sites; and no visible lesions on necropsy. Rabbits whose eyes were treated with 0.1 mL of the test material showed no visible lesions on necropsy. Tables and charts. See also Abstract No. 30-32296.

■ *API Medical Research Publication* (Aug. 1982) (111 p.).

Source: API HESD Information Specialist

30-32836

Behavioral Evaluation of Petroleum Hydrocarbons. The University of Arkansas carried out a study for the American Petroleum Institute 12/1/80-9/16/81 to determine the suitability of selected behavioral methods for evaluating the toxic impact of petroleum compounds. Toluene (5.00 and 0.70 mL/kg of body weight), n-hexane (10.00 and 1.00 mL/kg), ethanol (3.00 and 0.43 mL/kg), and maize oil (20.00 mL/kg) were administered via gavage to male and female C57B1/6 mice. Techniques for measuring 13 distinct behaviors were assessed on the basis of spontaneous locomotor activity in a novel environment and in the home cage, circadian pattern of food and water consumption, active avoidance learning in the two-way shuttle cage, and neuromuscular endurance and coordination on a variable speed rotorod with aversive consequence. Spontaneous locomotor activity in a novel environment was the most reliable of the five experimental paradigms. Tables and 54 references.

■ J. M. Cranmer (Univ. Arkansas), *API Medical Research Publication* (June 1983) (144 p.).

Source: API HESD Information Specialist

30-32845

Kidney Effects of Unleaded Gasoline. Comprehensive and Critical Summary of Observations in Rats and Mice. Each of four test groups consisting of 100 male and 100 female Fischer 344 rats and 100 male and 100 female $B_6C_3F_1$ mice were exposed to 0-2056 ppm of unleaded gasoline vapor for 114 weeks. In rats, there was a spectrum of histomorphologic renal alterations, including tubular cell basophilia, proteinaceous cast formation, chronic interstitial inflammation, renal pelvic mineralization, nuclear alterations, tubular cell hyperplasia and primary renal neoplasia, mostly before 12 months. In the mice, there were congenital lesions such as hydronephrosis, cortical cysts, and spontaneous lesions associated with aging mice, i.e., cortical fibrosis, glomerulosclerosis, interstitial inflammation, proteinaceous casts, amyloidosis, and ossification of the renal parenchyma. Tables, photomicrographs, and 33 references.

■ D. N. Kitchen; W. H. Halliwell (Westpath Lab.), *API Medical Research Publication* (Sept. 1983) (57 p.).

Source: API HESD Information Specialist

30-32846

Six-Month Continuous Inhalation Exposures of Rats to Hexane Mixtures. Phase II. Studies by International Research & Development Corp. have shown that male albino rats exposed to 500 ppm n-hexane/500 ppm n-hexane-free hexanes (Group 9), and to 500 ppm n-hexane alone (Group 10) for 22 hr/day, 7 days a week for ~ 6 mo, developed an abnormal gait which increased in incidence and severity over time, notably in Group 10 rats. Rats in Groups 9 and 10, respectively, weighed 25 and 30% less than the controls and rats exposed to 500 ppm n-hexane-free hexanes alone (Group 8). Microscopic findings on necropsy showed a greater incidence of a trace-to-mild degree of atrophy of sciatic and/or anterior tibial nerve with a mild secondary atrophy of skeletal muscle in Group 10 rats than in Group 9 rats, and no evident nerve and muscle lesions in Group 8 rats. The toxicological significance of the renal lesions in all treated rats was considered equivocal, since the level of spontaneous renal disease obscured the compound relationship of typical hydrocarbon-induced renal disease. Tables, graphs, and diagram.

■ *API Medical Research Publication* (Nov. 1983) (206 p.).

Source: API HESD Information Specialist

30-32847

The Carcinogenicity of New and Used Lubricants. The University of Cincinnati Medical Center's Kettering Laboratory studies of several groups of 50 male mice dermally treated twice weekly for 104 wk with 50 mg each of new and used API samples of a composite motor oil, five paraffinic base stocks with viscosities of 64, 133, 331, 485, and 990 SUS, and two naphthenic base stocks with viscosities of 83 and 2008 SUS, showed that the paraffinic and naphthenic lubricant samples and the unused composite motor oil were relatively non-toxic. The naphthenic samples showed borderline carcinogenicity, and the used composite motor oil was slightly to moderately carcinogenic. The rest of the materials, including unused composite motor oil, did not give evidence of carcinogenicity. Controls treated with 0.05 or 0.15% BaP in toluene developed large numbers of high-grade tumors with unexpectedly short latent periods; one control treated with 50 mg of toluene developed histiocytoma. The incidence of internal lesions in all groups was not higher than expected; no metastatic lesions from skin tumors were seen. Early deaths were probably due to housing-related stress. Tables, diagrams, and graphs.

■ *API Medical Research Publication* (Oct. 1983) (170 p.).

Source: API HESD Information Specialist

30-32848

Serum Immunoglobulin Levels of CD Rats and CD-1 Mice Exposed to Benzene Vapor. Final Report. Basal serum immunoglobulin analyses on male and female CD rats and CD-1 mice exposed to 1, 10, 30, and 300 ppm benzene for up to 91 days suggest that serum immunoglobulin levels may not be useful as indicators of benzene exposure in humans unless some specific subclass is identified as being uniquely sensitive. Data on the mice suggest that benzene is a hapten and causes a trend towards early suppression of immunoglobulin levels, with the females showing suppression in IgA levels at 7 days and in the levels of the IgG subclasses at longer periods, and males showing effects at 14 days. A trend towards IgG level elevation in both sexes after 28 days of exposure may be due in part to the antigenicity of benzene. Mice receiving the immunosuppressive cyclophosphamide showed a dose-dependent reduction in the levels of all immunoglobulins. The only statistically significant dose-related effect in rats was a reduction in the IgG level on day 14. Graphs and tables.

■ *API Medical Research Publication* (Oct. 1983) (86 p.).

Source: API HESD Information Specialist

30-32849

The Determination of the Carcinogenicity of [Shale] Retort Oils and Downstream Products. Skin bioassays by the University of Cincinnati Medical Center's Kettering Laboratory on groups of 50 male mice treated twice weekly for 79 wk with 50 mg each of API samples of two shale oils and four shale oil-derived downstream products showed that the crude and in-situ shale oils were strongly carcinogenic, causing rapid development of usually multiple tumors, with short latent periods. The hydrotreated oil and residue were moderately carcinogenic and somewhat toxic. The diesel fuel marine precursor and product were only minimally tumorigenic but highly toxic, and produced a large number of early non-tumor deaths. Hydrotreatment apparently reduced the tumorigenic potential of the downstream products. There were no tumors observed in controls treated with 50 mg of toluene; controls treated with 0.05 or 0.15% BaP in toluene showed a high incidence of benign and malignant neoplasms; one untreated control developed a papilloma. The results did not provide an adequate explanation of the observed toxicity not related to tumorigenicity. Tables and graphs.

■ *API Medical Research Publication* (Nov. 1983) (97 p.).

Source: API HESD Information Specialist

30-32850

Effects of Subchronic Fourteen Day Exposure to Benzopyrene in B6C3F1 Male and Female Mice on Specific Immunological Parameters. Final Report. Studies by the Medical College of Virginia on B6C3F1 male and female mice exposed daily for 14 days to 5, 20, and 40 mg/kg of BaP and benzo[e]pyrene showed that BaP primarily inhibits the mice's ability to produce antibodies to the T-dependent antigen sheep erythrocytes. The parameters used to measure the functional aspects of cell-mediated immunity were unaltered, although there were effects on the T-lymphocyte mitogen. There were no biologically relevant changes in basal serum immunoglobulins, except for IgA levels in female mice. Changes in other toxicological parameters include a dose-dependent reduction in spleen macrophage phagocytosis in BaP-treated male and female mice; slight increases in body weight, primarily in mice treated with BaP; reduction in the erythroid elements in the hematological tests; slight decreases in serum total protein and albumin of BaP-treated female mice; and slight increases in liver and spleen weight of BaP-treated mice. Tables.

■ *API Medical Research Publication* (Oct. 1983) (215 p.).

Source: API HESD Information Specialist

30-32851

The Evaluation of the Carcinogenicity of Certain Petroleum Hydrocarbon Fractions (Revised: February 1983) with Supplemental Report (Revised: March 1983). The University of Cincinnati Medical Center's Kettering Laboratory studies on numerous groups of 50 mice dermally treated twice weekly for 80 wk with 50 mg each of 25 API samples of two whole crudes (C and D) and their distillates, and of the aromatic and saturated subfractions of some of the distillates, showed that the most carcinogenic fractions were PS8-76 D2 (120-350 °F), PS8-76 D5 (700-1070 °F), PS8-76 C4 (550-700 °F), and PS8-76 C5 (700-1070 °F) distillates, with the C4 being more carcinogenic than the D4 distillate. The incidence of tumors was greater for mice treated with some of the saturated subfractions than with the corresponding aromatics fractions. The incidence of hepatocellular carcinomas was not higher in the oil fraction-treated groups than in the untreated or toluene-treated controls. The malignant tumors totaled 522, of which 442 were squamous cell carcinomas, which in turn produced three cases of pulmonary metastases in mice in the PS8-76 C3 saturate, and PS8-76 D2 and D3 aromatics groups. Tables and graphs.

■ *API Medical Research Publication* (Oct. 1983) (399 p.).

Source: API HESD Information Specialist

30-32852

A 28-Day Dermal Toxicity Study of API [Vacuum Residuum] Sample 81-13 in the Rabbit initiated by Borriston Laboratories Inc. for the American Petroleum Institute 9/20/82 involved application of the sample to the shaved skin of New Zealand white rabbits at dose levels of 200, 1000, and 2000 mg/kg/day three times a week for a total of 12 applications over 28 days. Treatment-related clinical signs consisted of thin appearance, decreased food intake, flaking skin, and wheezing. Males and females exposed to 2000 mg/kg showed very slight to slight edema beginning on the first day, and edema was a consistent finding in all rabbits beginning on day 16. An apparent treatment-related suppression in body weight gain was present in the 2000 mg/kg males. Treatment-related gross pathology findings were confined to the treated skin and consisted of reddened or thickened skin. Microscopic treatment-related findings were generally confined to the treated skin. The sample had a 6.6 °API gravity and contained 4.46 wt % of sulfur and 6.5 wt % of asphaltenes. Tables.

■ *API Medical Research Publication* (Oct. 1983) (136 p.).

Source: API HESD Information Specialist

30-32853

A 28-Day Dermal Toxicity Study of API [Vacuum Residuum] Sample 81-14 in the Rabbit involved a sample having an 11.8 °API gravity and containing 0.7 wt % of sulfur and 1.2 wt % of asphaltenes. The results were similar to those of a previous study carried out by Borriston Laboratories for the American Petroleum Institute [Abstract No. 32-32854], except that the body weight gain was not affected. Tables.

■ *API Medical Research Publication* (Oct. 1983) (141 p.).

Source: API HESD Information Specialist

30-32854

A 28-Day Dermal Toxicity Study of API [Catalytic Cracked Clarified Oil] Sample 81-15 in the Rabbit involved a fraction boiling above 350 °C, having a 0.1 °API gravity, and containing 1.1 wt % of sulfur and 4.2 wt % of asphaltenes. Besides symptoms similar to those in a previous study by Borriston Laboratories Inc. for the American Petroleum Institute [Abstract No. 30-32853], one male died in the 1000 mg/kg/day dose group and two males and one female in the 2000 mg/kg group. Other additional symptoms included moderate to severe erythema outside of the dosed area, skin ulceration at the test site, increases in liver weight, and some liver damage. The mean body weight gains were significantly lower than those of the controls for most of the males at all doses (200, 1000, and 2000 mg/kg/day) and most test intervals over the 28-day test period. Microscopic treatment-related findings of the skin included subacute acanthotic dermatitis which had progressed to severe early multifocal papillomatosis. In high-dose animals of both sexes, changes in the lymphoid organs were obvious, and the bone marrow had slight to severe diffuse hypocellularity. Tables.

■ *API Medical Research Publication* (Oct. 1983) (150 p.).

Source: API HESD Information Specialist

30-32855

LC_{50} Acute Inhalation Toxicity Evaluation of a Petroleum-Derived Hydrocarbon in Rats...Hydrodesulfurized Kerosine, API Sample 81-07. A group of five male and five female albino rats 61 days old were exposed once for four hours to an atmosphere having a 5.2 mg/L API No. 81-07 sample aerosol-vapor concentration, and a similar group served as an air-exposed control, beginning 10/22/82. The main pharmacotoxic sign observed during and after the exposure was dyspnea. There was no apparent effect of exposure on body weight gain. There were no test-related macroscopic or microscopic changes in the rats after 14 days of recovery from the single exposure. The 362-535 °F fraction contained 0.07 wt % of sulfur, 47% of alkanes, 1% of olefins, 35% of aromatics, and 18% of saturates. The study was carried out by International Research & Development Corp. for the American Petroleum Institute.

■ *API Medical Research Publication* (Nov. 1983) (44 p.).

Source: API HESD Information Specialist

30-32856

LC_{50} Acute Inhalation Toxicity Evaluation of a Petroleum-Derived Hydrocarbon in Rats...Hydrodesulfurized Middle Distillate, API Sample 81-09. In a study started by International Research & Development Corp. on 8/17/82 for the American Petroleum Institute, male and female albino rats were exposed once for four hours to a 3.6 mg/L API No. 81-09 sample aerosol-vapor concentration, which produced 60% mortality. The LC_{50} was calculated to be 4.60 mg/L, with 95% confidence limits of 3.92-5.40 mg/L. In general, the main pharmacotoxic signs were dyspnea, nasal discharge, and alopecia. Body weight gain was generally depressed during the first week of the post-exposure observation period but appeared normal during the second week. The single-inhalation exposure of the rats was followed by acute inflammatory changes in the lungs of animals which died during the study and in animals at all exposure levels which survived the 14 day observation period. The 502-574 °F sample fraction contained 0.15 wt % of sulfur, 46.0% of alkanes, 2.5% of olefins, 26.5% of aromatics, and 25.0% of saturates. Tables.

■ *API Medical Research Publication* (Nov. 1983) (81 p.).

Source: API HESD Information Specialist

30-32857

LC_{50} Acute Inhalation Toxicity Evaluation of a Petroleum-Derived Hydrocarbon in Rats...Hydrodesulfurized Middle Distillate, API Sample 81-10. In a study initiated on 9/2/82 by International Research & Development Corp. for the American Petroleum Institute, a group of five male and five female albino rats was exposed once to an atmosphere containing 5.0 mg/L of an aerosol-vapor mixture of API sample No. 81-10 for four hours, which produced 40% mortality. Four additional groups were exposed to the sample for four hours to determine the LC_{50}, which was calculated to be 7.64 mg/L, with 95% confidence limits of 5.51-10.58 mg/L. The main pharmacotoxic signs were dyspnea, nasal discharge, and excessive salivation. Body weight gain was depressed during the first week for both sexes and generally normal for the males only during the second week. Acute inflammatory changes were observed in the lungs of animals which died and foamy alveolar macrophages in all of the animals which survived the 14 day observation period. These changes were consistent with the signs of respiratory distress. Diagram, graphs, and tables.

■ *API Medical Research Publication* (Nov. 1983) (85 p.).

Source: API HESD Information Specialist

30--32858

Six Month Continuous Inhalation Exposures of Rats to Hexane Mixtures. Phase I. A study initiated 2/3/81 by International Research & Development Corp. for the American Petroleum Institute involved the exposure of male albino rats for 22 hr/day over six months to atmospheres consisting of filtered air (group I) or containing 125 ppm of n-hexane (group II), 125 ppm of n-hexane + 125 ppm of mixed hexanes (group III), 125 ppm of n-hexane + 375 ppm mixed hexanes (group IV), 125 ppm of n-hexane + 1375 ppm of mixed hexanes (group V), or 500

ppm of n-hexane (group VI). The most significant pharmacotoxic sign was the abnormal gait in group VI, in which body weights were significantly depressed. Liver weights of group V and VI animals and kidney weights of group IV, V, and VI animals were quite different than for control animals. Differences between control and treatment group brain, spleen, heart, and lung weights were either related to changes in body weight gain or within the range of normal variability. A 212 p. appendix is included. Diagram, graph, chromatogram, spectra, and tables.
■ *API Medical Research Publication* (Nov. 1983) (71 p.).
Source: API HESD Information Specialist

31-02438
Toxicological Characteristics of Refinery Streams Used To Manufacture Lubricating Oils. An analysis of previously unpublished mouse skin-painting tumorigenicity studies on 46 clearly defined refinery streams used in formulating lubricating oils and sponsored by the American Petroleum Institute and member companies, mainly at the University of Cincinnati Kettering Laboratory, indicates that unprocessed vacuum distillates cause skin tumors; that treatment of distillates by modern, conventional solvent-refining, severe (but not conventional) acid treatment with fuming sulfuric acid or relatively severe hydrotreatment can reduce or eliminate tumorigenicity; that dewaxing, clay treatment, and hydrofinishing do not eliminate distillate tumorigenicity; and that most unused engine oils formulated from solvent-refined base stocks, with additives, are not carcinogenic. Published acute-toxicity and mutagenicity studies with similar results are also discussed. Diagram, flow diagram, tables, and 30 references.
■ M. L. Kane; E. N. Ladov; C. E. Holdsworth; N. K. Weaver (API), *American Journal of Industrial Medicine* 5:183-200 (1984) (18 p.).
Source: Not available from API

31-30263
In Vitro Determination of Chemical Cocarcinogenesis in Balb/c 3T3-A31$_{SF}$ Cell Cultures. *In vitro* assays, paralleling *in vivo* mouse skin studies, conducted by the New York University Medical Center on a subclone of these cultures have shown that the number of colonies exhibiting typically transformed characteristics (e.g., increased numbers of pleiomorphic cells in multilayered, irregular colonial patterns) increased substantially when treated simultaneously with a combination of 1, 2, 5, or 10 µg/ml of BaP as the carcinogen/initiator and 1,2, or 4 µg/ml of catechol, or 5, 10, or 20 µg/ml of pyrene, or 3.5, 7, or 14 µg/ml of fluoranthene, or 0.1 µg/ml of phorbol myristate acetate (PMA) as the cocarcinogen than when BaP alone or any of the cocarcinogens alone were used. Unlike the other cocarcinogens used alone, PMA, the classic tumor promoter, showed tumor-promoting and cocarcinogenic capabilities, depending on the protocol used. Graphs, tables, and 166 references.
■ N. Z. Baturay (N.Y. Univ. Med. Cent.), *API Medical Research Publication* (Feb. 1983) (187 p.).
Source: API HESD Information Specialist

31-30264
A Metabolic Model of Lead Kinetics Based upon Measured Organic Burdens during Chronic Exposure Experiments with [66] Infant and Juvenile Baboons, which was developed by the New York University Medical Center, shows that the biological half-life of lead in blood of the very young (< 14 mo) baboons is ~ 5 days and is shorter than that of older (15-24 mo) baboons after exposure to 12-45,000 µg of lead/kg/day for up to 15 mo. Other findings involving rapidly growing baboons include a high (33%) daily uptake of the circulating blood lead by the bones; a decrease in the fraction of ingested lead absorbed from 0.25 to 0.02 as exposure increases from 10 to 1000 µg/kg/day; uptakes of } 10 and 3%/day of the lead in blood by the liver and kidney, respectively; a transfer of equal amounts of lead back to the blood from the soft tissue compartments of liver and kidney during steady state conditions; the elimination of ~ 8%/day of the lead in blood in urine; and an accurate prediction by the model of the accumulation and retention of lead in adult humans. Tables, graphs, diagrams, and 128 references.
■ R. P. Mallon (N.Y. Univ. Med. Cent.), *API Medical Research Publication* (Feb. 1983) (173 p.).
Source: API HESD Information Specialist

31-30265
Stimulation and Inhibition of Chemotaxis in Abelson Virus-Transformed Macrophages, [in Relation to the Clearance of Air Pollutants in the Lung]. *In vitro* studies by the New York University Medical Center on Abelson leukemia virus-transformed murine alveolar macrophage RAW624.7 cell line have shown that the cells responded chemotactically to various dilutions of endotoxin-activated mouse serum, with the 1:100 dilution evoking the highest response. 12-o-Tetradecanoyl-phorbol-13-acetate, a promoter of skin tumors in mice, did not stimulate chemotaxis in the cells. Chemotaxis was inhibited by 0.0001-0.000001 M mepacrine, but not by indomethacin, which suggests that phospholipase A_2 activity may be involved in the chemotactic response. In the cytotoxicity tests prior to chemotactic assays, the treatment of cells with 8 µg/mL of pyrene and with 1 µg/mL of BaP for 24 hr did not significantly alter the chemotactic response. The results did not show whether environmental pollutants modify chemotaxis in this system, and a broad spectrum of air pollutants should be tested to determine its usefulness for in-vitro screening. Tables, graphs, and 55 references.
■ A. Morris (N.Y. Univ. Med. Cent.), *API Medical Research Publication* (June 1983) (36 p.).
Source: API HESD Information Specialist

31-30448
Five-Day Pilot Inhalation Study in Rats and Mice. Benzene. Final Report. Studies by Hazleton Laboratories America Inc. on 10 male and 10 female Sprague-Dawley rats and on 30 male and 30 female CD-1 mice exposed to 2000 ppm of benzene for 6 hr/day for five consecutive days showed no consistent exposure-related trends in weight loss in all the animals, except for a slight, statistically significant depression in the mean terminal body weight (adjusted for day zero) of male rats. Compared with the untreated controls, all of the exposed animals showed a significant decrease in total leukocyte count (white blood cells), although the differential counts were not significantly affected in either species. The red blood cell count and hemoglobin concentration were significantly lower in the exposed female mice than in the male mice. The statistically significant decrease observed in total serum protein in exposed male rats had no biological significance, and there were no consistent treatment-related trends in the gross pathology of either species on necropsy. Tables.
■ *API Medical Research Publication* (1/24/83) (100 p.).
Source: API HESD Information Specialist

31-30449
Assessment of the Healthy Worker Effect [(HWE)]. Final Report. A review of literature published in the *J. Occup. Med., Arch. Environ. Health, Br. J. Ind. Med., Scand. J. Work Environ. Health,* and *J. Epidemiol. Community Health* during Jan. 1971-June 1982, which identified 115 follow-up cohort studies, such as an epidemiological survey of eight oil refineries in the U.K. by L. Rushton and M. R. Alderson (U.K. Inst. Cancer Res.) [Abstract No. 28-32352], shows such a diversity in several criteria as to make the assessment of HWE very difficult. Relatively uniform cohort data from nine occupational cohorts, including refinery and rubber workers, and from two retrospective cohort mortality studies of petroleum workers by the Stanford Research Institute and Gulf Oil Corp., suggest that, over all, the HWE is on the high side (i.e., low standardized mortality ratios) compared with other occupational cohorts, but not consistently or remarkably so. Tables, graph, and 132 references.
■ B. MacMahon; R. R. Monson, *API Medical Research Publication* (8/16/83) (110 p.).
Source: API HESD Information Specialist

31-30613
Acute Inhalation Toxicity Evaluation of a Petroleum-Derived Hydrocarbon in Rats. Light Catalytic Reformed Naphtha...API Sample 83-04. Five male and five female rats, exposed once to 5.05 ± 0.12 mg/L vapor from sample 83-04 for 4 hours showed no significant toxic signs, and none died, during exposure or up to 14 days after. The histopathological examination of the lungs yielded only the minimal to mild pulmonary changes normal for nongerm-free rats. Individual and mean body weights were not affected by the exposure. The test material

had a boiling range of 114-279 °F and was composed of 49.0% paraffins, 1.5% olefins, 10.0% naphthenes, and 39.5% aromatics. Graph and tables.
■ *API Medical Research Publication* (Feb. 1984) (30 p.).
Source: API HESD Information Specialist

31-30614
Mutagenicity Evaluation Studies in the Rat Bone Marrow Cytogenetic Assay [and] in the Mouse Lymphoma Forward Mutation Assay. Vacuum Residuum...API Sample 81-13. Studies on sample 81-13 carried out by Litton Bionetics Inc. for API showed that the structural aberration frequency in the bone marrow cells of ten male and ten female Sprague-Dawley rats dosed with the sample did not differ significantly from the negative control at any tested dose (0.3, 1, and 3 g/kg/day, in 5 doses per day per os) during 11/23/81-2/9/82. A positive control substance, triethylenemelamine, significantly increased the structural aberration frequency. In the test material induced small but repeatable increases in the mutant frequency at the thymidine kinase locus in L5178Y mouse lymphoma cells only in the presence of rat liver S9 metabolic activation; with activation, concentrations of 250-1000 µg/mL consistently induced two- to three-fold increases in the mutant frequency. The test material was thus considered weakly active in this in-vitro test. The sample had a 650 °F ibp and contained 4.46 wt % of sulfur and 6.5% of asphaltenes. Diagrams and tables.
■ *API Medical Research Publication* (Feb. 1984) (48 p.).
Source: API HESD Information Specialist

31-30615
Mutagenicity Evaluation Studies in the Rat Bone Marrow Cytogenetic Assay and in the Mouse Lymphoma Forward Mutation Assay. Vacuum Residuum...API Sample 81-14. Tests on this sample by Litton Bionetics Inc. for API showed that the administration of 0.4, 1.3, or 4.0 g/kg/day in 5 daily doses by oral gavage during 12/4/81-4/29/82 did not induce chromosomal aberrations in Sprague-Dawley rat bone marrow cells. The tests were on 110 animals, including 50 positive control rats to which 0.75 or 1.0 mg/kg of triethylenemelamine was administered, significantly increasing the structural aberration frequency. In-vitro treatment by exposure for four hours to 62.5-1000 µg/mL of sample 81-14 significantly increased the mutant frequency at the thymidine kinase locus of the L5178Y mouse lymphoma cell only in the presence of Fischer 344 Aroclor-induced rat liver S9 microsomal metabolic activation mix (0.3 mL/10 mL) in tests carried out during 11/4/81-2/16/82. The test material was thus considered weakly active in the mouse lymphoma forward mutation assay with activation. The sample had a 662 °F ibp and contained 0.72 wt % sulfur and 1.2% asphaltenes. Diagrams and tables.
■ *API Medical Research Publication* (Feb. 1984) (42 p.).
Source: API HESD Information Specialist

31-30680
Acute Inhalation Toxicity Evaluation of a Petroleum-Derived Hydrocarbon in Rats...Light Catalytic Cracked Naphtha. API Sample 81-04. A 5.25 mg/L concentration of API light catalytic cracked naphtha sample 81-04 was administered to five young adult rats of each sex for four hours via dynamic, whole-body exposure methods on 9/21/83 by Litton Bionetics Inc. for the API. No animals died or showed clinical signs during the test or the following 14 days. No toxic signs of any significance were seen in animals of either sex that could be attributed to exposure to the test material. Individual and mean body weights were not affected. Histopathological examination of lung tissues yielded minimal pulmonary findings, none due to the test material vapor, which had a 104-380 °F boiling range and contained 29.6% of olefins, 35.8% paraffins, 14.2% naphthenes, and 20.3% of aromatics. Graph and tables.
■ *API Medical Research Publication* (Feb. 1984) (36 p.).
Source: API HESD Information Specalist

31-30681
Acute Inhalation Toxicity Evaluation of a Petroleum-Derived Hydrocarbon in Rats...Full-Range Catalytic Reformed Naphtha. API Sample 83-05. API catalytic reformed naphtha sample 83-05 vapor concentrations of 5.22 mg/L were administered to five young adult rats of each sex for four hours via dynamic, whole-body exposure methods by Litton Bionetics Inc. for the API in a study beginning 10/20/83. No animals died or showed clinical signs during exposure or the following 14 days. No toxic signs of any significance were seen in any of the animals that could be attributed to exposure to the test sample, and individual and mean body weights were not affected. Histopathological examination of lung tissues yielded minimal to moderate pulmonary findings. The possibility that these could be due to the exposure could not be ruled out. The API sample had a 132-389 °F boiling range and contained 31.0% paraffins, 5% naphthenes, 2.2% benzene, 1.5% of olefins, and 62.5% of aromatics. Graph and tables.
■ *API Medical Research Publication* (Feb. 1984) (32 p.).
Source: API HESD Information Specialist

31-31351
[A Hazleton Laboratories America Inc.] Dermal Sensitization Study in Guinea Pigs [by the] Closed Patch Technique [with the Use of] Sweetened Naphtha, API Sample 81-08, has shown that the test material is not a skin sensitizer, since the responses by the treated animals did not exceed the most severe naive or vehicle control response. The 10 male albino guinea pigs treated with 0.4 mL of a 50 vol/vol % mixture of the test material in paraffin oil once a week for 3 wk showed very slight-to-well-defined erythema and very slight edema. A very slight erythema was shown by 5 of the treated animals and 2 of 10 previously untreated naive controls after receiving single applications of 0.4 mL of a 25 vol/vol % mixture of the test material in paraffin oil; and by 2 of 10 vehicle controls after a single application of 0.4 mL of undiluted paraffin oil. All 20 positive controls are considered to have been sensitized because of the slight-to-severe reactions after three applications of 0.4 mL of 0.3% 2,4-dinitrochlorobenzene (DNCB) sensitizer in 80% ethanol/distilled water and a subsequent single application of 0.4 mL of 0.1 wt/vol % of DNCB in acetone. Tables.
■ *API Medical Research Publication* (May 1984) (14 p.).
Source: API HESD Information Specialist

31-31352
Dermal Sensitization Study in Guinea Pigs. Closed Patch Technique. Hydrodesulfurized Middle Distillate, API Sample 81-09. A study by Hazleton Laboratories America Inc. has shown that the test material was not a skin sensitizer, since the responses by the treated male albino guinea pigs did not exceed the highest naive or vehicle control response. The 10 animals treated thrice in three weeks with 0.4 mL of a 25 vol/vol % mixture of the test material in paraffin oil showed very slight-to-well-defined erythema and very slight edema. Very slight erythema developed in 5 of the treated animals and in 3 of 10 naive controls treated once with 0.4 mL of a 10 vol/vol % of API 81-09 in paraffin oil, and in 1 of 10 vehicle controls treated thrice with 0.4 mL of 100% paraffin oil. None of the vehicle controls showed dermal reaction in the challenge application. All 20 positive controls were considered to have been sensitized, since they showed slight-to-severe reactions after three applications of 0.3% 2,4-dinitrochlorobenzene (DNCB) in 80% ethanol/distilled water and one application of 0.1 wt/vol % of DNCB in acetone. Tables.
■ *API Medical Research Publication* (May 1984) (14 p.).
Source: API HESD Information Specialist

31-31412
A Dermal Sensitization Study [by Hazleton Laboratories America Inc.] in [50] Guinea Pigs [by the] Closed Patch Technique [with the Use of] API Sample 81-03 (light catalytic cracked naphtha), has shown that the test material is not a skin sensitizer, since the reactions of the treated animals did not exceed the highest response of the naive or vehicle controls. The 10 animals that received three treatments in three weeks of 0.4 mL of a 50 vol/vol % mixture of the test material in paraffin oil showed slight-to-well-defined erythema and edema. Four of these animals and 2 of 10 naive controls showed very slight erythema after one application of 0.4 mL of a 1 vol/vol % mixture of the test material in paraffin oil. One of 10 vehicle controls showed slight erythema after three treatments of 0.4 mL of 100% paraffin oil, and very slight skin irritation after one application of the same material. All 20 positive controls are considered to have been sensitized because of the slight-to-severe reactions after three treatments of 0.4 mL of 0.3% 2,4-dinitrochlorobenzene (DNCB) in 80% ethanol/distilled water and after

one application of 0.4 mL of 0.1 wt/vol % DNCB in acetone. Tables.
■ *API Medical Research Publication* (May 1984) (14 p.).
Source: API HESD Information Specialist

31-31413
A Dermal Sensitization Study in [50] Guinea Pigs [by the] Closed Patch Technique [with the Use of] API Sample 81-07 [(Hydrodesulfurized Kerosine)] has shown that the test material is not a skin sensitizer because the reactions by the treated animals did not exceed the highest naive or vehicle control response. Other findings by Hazleton Laboratories America Inc. were: very slight-to-well-defined erythema and very slight edema in all 10 guinea pigs treated thrice in three weeks with 0.4 mL of a 50 vol/vol % mixture of API 81-07 in paraffin oil; very slight erythema in five of the treated animals and 2 of 10 naive controls after one application of 0.4 mL of 10 vol/vol % of API 81-07 in paraffin oil; very slight erythema in 2 of 10 vehicle controls treated with 0.4 mL of 100% paraffin oil; and sensitization in all 20 positive controls after three treatments with 0.4 mL of 0.3% 2,4-dinitrochlorobenzene (DNCB) in 80% ethanol/distilled water and after one application of 0.4 mL of 0.1% DNCB in acetone. Tables.
■ *API Medical Research Publication* (May 1984) (14 p.).
Source: API HESD Information Specialist

31-31414
Dermal Sensitization Study in Guinea Pigs. Closed Patch Technique. Hydrodesulfurized Middle Distillate, API Sample 81-10. A study by Hazleton Laboratories America Inc. on 50 male albino guinea pigs has shown that the test material was not a skin sensitizer by the closed patch technique. The findings to support this conclusion were: very slight-to-well-defined erythema and edema in 10 animals treated thrice in three weeks with 0.4 mL of a 10 vol/vol % mixture of the test material in paraffin oil; no dermal irritation in any of the above animals, nor in the 10 naive controls after one application of the same test material; no skin irritation in the 10 vehicle controls after three and one treatments with 0.4 mL of 100% paraffin oil; and sensitization in all 20 positive controls, which showed slight-to-moderate reactions after three treatments with 0.4 mL of 0.3% 2,4-dinitrochlorobenzene (DNCB) in 80% ethanol/distilled water and after one application of 0.4 mL of 0.1 wt/vol % of DNCB in acetone. Tables.
■ *API Medical Research Publication* (May 1984) (15 p.).
Source: API HESD Information Specialist

31-31415
Dermal Sensitization Study in Guinea Pigs. Closed Patch Technique. Vacuum Residuum, API Sample 81-13. The results from a study by Hazleton Laboratories America Inc. on 40 guinea pigs have shown that API sample 81-13 (black, tarry semi-solid) was not a skin sensitizer by the closed patch technique. The 10 animals treated thrice in three weeks with 0.4 mL of the undiluted test material showed very slight-to-well-defined erythema and very slight edema. None of the animals in this group or in the 10 naive controls group showed any dermal irritation after a single application of the same test material. All 20 positive controls showed reactions after three applications of 0.3% 2,4-dinitrochlorobenzene (DNCB) in 80% ethanol/distilled water, and slight-to-moderate responses after one application of 0.1 wt/vol % of DNCB in acetone, thereby making them sensitized. Tables.
■ *API Medical Research Publication* (May 1984) (14 p.).
Source: API HESD Information Specialist

31-31416
Dermal Sensitization Study in Guinea Pigs. Closed Patch Technique. Vacuum Residuum, API Sample 81-14. A study on 40 guinea pigs by Hazleton Laboratories America Inc. has shown that the test material (black, tarry, viscous liquid) was not a skin sensitizer. Ten animals showed very slight edema and very slight-to-well-defined erythema after three applications in three weeks of 0.4 mL of the undiluted test material. None of the treated animals and none of the 10 naive controls showed dermal irritation after one application of the same test material. All 20 positive controls reacted after three treatments with 0.4 mL of 0.3% 2,4-dinitrochlorobenzene (DNCB) in 80% ethanol/distilled water, and were considered to have been sensitized, since they showed slight-to-moderate responses after one application of 0.4 mL of 0.1 wt/vol % of DNCB in acetone. Tables.
■ *API Medical Research Publication* (May 1984) (14 p.).
Source: API HESD Information Specialist

31-31417
Dermal Sensitization Study in Guinea Pigs. Closed Patch Technique. Catalytic Cracked Clarified Oil, API Sample 81-15. Results from a study by Hazleton Laboratories America Inc. on 40 male albino guinea pigs have shown that the test material (black, tarry, viscous liquid) was not a skin sensitizer by the closed patch technique. Ten animals treated thrice in three weeks with 0.4 mL of the undiluted test material showed very slight edema and very slight-to-well-defined erythema. One subsequent application of the same test material did not produce any skin irritation in any of these animals or in any of the 10 naive controls. All 20 positive controls reacted after three treatments with 0.4 mL of 0.3% 2,4-dinitrochlorobenzene (DNCB) in 80% ethanol/distilled water, and were considered to have been sensitized after they showed slight-to-moderate responses following one subsequent application of 0.4 mL of 0.1 wt/vol % of DNCB in acetone. Tables.
■ *API Medical Research Publication* (May 1984) (14 p.).
Source: API HESD Information Specialist

31-31471
A Draft Final Report [by Battelle Columbus Laboratories] on [Its] Development of a Method for Measurement of Heterocyclic [Polynuclear] Aromatic Compounds [(Hetero-PACs)] in Petroleum Samples (API Crude D, Wilmington petroleum, and shale oil) has shown that the method is not suitable for the analysis of oxygen-containing PACs (O-PACs) in petroleum or shale oil due to the overwhelming interference by coeluting hydrocarbon material of the same molecular weight. The method, which was used on 20 model hetero-PACs, is a fractionation scheme that uses silica gel chromatography in an open column with batch elution procedures, detection by MS. Of the five fractions generated, three contained the target PACs containing sulfur (S), oxygen, and low/higher polarity, low/higher basicity secondary/tertiary nitrogen (N). The detection limits observed for the isobutane-assisted, chemical ionization mode of low-resolution MS were ~ 0.5 ppm for N-PACs and } 5-10 ppm for O- and S-PACs in Crude D. Other results included the quantification of over 300 individual compounds and sets of isomers in each oil sample; and 10-50% and 5-10% shares of S- and N-PACs, respectively, in the S and N content of the fractions. Tables, graphs, and 57 references.
■ S. V. Lucas; J. S. Warner (Battelle Columbus Lab.), *API Medical Research Publication* (10/1/83) (203 p.).
Source: API HESD Information Specialist

31-31472
Validation of Behavioral Studies for Determining Neurotoxicity. Final Report. Results from spontaneous locomotor activity monitoring (SLAM), learning and memory assessment (LMA), and home-cage consummatory behavior monitoring (HCBM) studies by Intox Laboratories Inc. on several male mice, which received a single dose of zero or 0.3-3 mg/kg of trimethyltin hydroxide (TMT), or multiple doses of zero or 2-20 mg/kg/day of acrylamide for 4 wk, indicate that SLAM and LMA can provide a valid and sensitive assessment of petroleum-derived materials for functional toxicity. HCBM is not a valid or sensitive test for neurotoxic insult, since the only parameter that was significantly altered by acrylamide treatment was the total daily water intake. Findings included reduced locomotor activity in mice dosed with TMT vs. the zero-dose controls in the SLAM test, with the high-dose mice showing no recovery after several days; and mild neuronal degeneration and necrosis in the brains of five high-dose (TMT) mice in the LMA test. Tables, charts, graph, and 21 references.
■ *API Medical Research Publication* (May 1984) (170 p.).
Source: API HESD Information Specialist

31-31480
Cigarette Smoking, Benzene and Leukemia. A review of published studies on the relationship between leukemia and cigarette smoking suggests that cigarette smoking in itself may not cause human leukemia,

despite the presence of benzene in cigarettes. The International Agency for Research on Cancer has classified benzene as a human carcinogen, causing acute myelocytic leukemia and possibly other types of leukemia. Several epidemiologic studies world-wide have implicated benzene, a bone marrow suppressant, as a cause of human leukemia. A study by P. Newberne for API has shown that cigarettes also contain nitroso compounds and PAHs, such as BaP, which cause leukemia in laboratory animals. Recommendations include a further investigation of the smoking-leukemia relationship by evaluating the data compiled by either the National Cancer Institute or the American Cancer Society. Tables and 40 references.

■ P. Cole; H. Austin (Epidemiol. Resources Inc.); P. Newberne, *API Medical Research Publication* (10/24/83) (29 p.).
Source: API HESD Information Specialist

31-31481

Parental and Fetal Reproduction Inhalation Toxicity Study, in Rats, with Mixed Xylenes. Final Report. [Vol. I & II]. A Biodynamics Inc. study on 90 male and 180 female rats showed no mortality among the 150 rats treated daily with 0, 60, 250, or 500 ppm of technical grade mixed xylenes for 6 hr a day for a 131 day premating and a 20 day mating period, and for most of the gestation and lactation period for the females. In-life observations included no adverse treatment-related effects on body weight of F_0 adults before mating, or on maternal weight; significantly lower mating indexes among mid-dose males and females and high-dose females only vs. untreated controls; comparable pregnancy/fertility indexes between treated rats and controls; and no adverse treatment-related effects on the gestation length, parturition, litter size, or pup survival. Postmortem findings included no adverse treatment-related effects on the testes of F_0 males, or on tissues from high-dose pups; a statistically significant increase in mean kidney weight in high-dose F_0 females; and a lower mean number of fetuses with malformations per litter in the high-dose group vs. controls. Tables.

■ *API Medical Research Publication* (8/23/83) (542 p.).
Source: API HESD Information Specialist

31-32162

Meeting the Quality Assurance [(QA)] Challenges of the 1980's: Team Auditing by Toxicologists and QA Professionals. Team auditing is an effective way to ensure the continued productive contribution of toxicologists and QA professionals to the conduct of scientific research. This approach can offset QA constraints such as economic and political problems, increasing regulatory requirements and test complexity, and advances in toxicology, such as computerization. Another advantage of team auditing is the expansion of technical knowledge and skills gained by individual team members from the interchange. The major contributory factors to successful team auditing are the careful planning and execution of a QA audit, and the selection of the audit and team coordinator. The team auditing approaches used by API and Exxon Corp. are discussed.

■ B. K. Hoover (API); J. K. Baldwin (Exxon Corp.), *Journal of the American College of Toxicology* 3(2):129-39 (1984).
Source: Not available from API

31-32169

Effects of Toluene Exposure on Auditory Pathways. Final Report. Morphological examination by the Albert Einstein College of Medicine of samples of the upper medulla oblongata, lower and upper metencephalons, mesencephalon-metencephalon junction, and mesencephalon-diencephalon junction of glutaraldehyde-fixed brainstems of male and female rats exposed to 100 or 1500 ppm toluene for up to 27 wk, showed no toxin-related pathological changes compared with controls. The significance of shrunken and darkly-stained neurons in the corpus trapezoid of the brainstem of two male rats and of other males exposed to 100 or 1500 ppm toluene was not known. Comparable neurons were not present in the controls' tissues. Samples from both treated and control rats showed isolated non-specific alterations and scattered cellular distortion caused by extended storage. Table.

■ *API Medical Research Publication* (July 1984) (6 p.).
Source: API HESD Information Specialist

31-32980

Neurophysiological and Psychological Disorders and Occupational Exposure to Organic Solvents. A literature review of industrial exposures to organic solvents suggests that, of the solvents studied, only CS_2 provided clear evidence that long-term exposure leads to permanent structural or functional injury to the central nervous system (CNS) that is detectable by clinical, pathological, neurophysiological, or neuropsychological tests. The abnormal findings (impaired short-term memory, etc.) from long-term exposure of workers to trichloroethylene, toluene, styrene, white spirit, jet fuel, ethylene dichloride, methylene dichloride, xylene, chloroform, methyl chloride, or n-hexane, or mixtures of these, are questionable evidence of CNS impairment, in part because these deviations were also found after short-term exposure and there was no increase in severity with time. The literature methods used to study CNS function are not suitable for epidemiological studies of solvent-exposed workers. Published findings from short-term exposures of laboratory animals to solvents at very high levels do not confirm those in humans. 183 references. (supported by API and Br. Pet. Co. P.L.C.)

■ P. Grasso; M. Sharratt; D. M. Davies; D. Irvine (Brit. Pet. Co. P.L.C.), *Food and Chemical Toxicology* 22(10):819-52 (1984).
Source: Not available from API

32-01362

The Renal Effects of Petroleum Hydrocarbons, Symposium (Boston 7/18-20/83). This publication includes papers, most of which are abstracted separately, on the renal toxicity and carcinogenicity of hydrocarbons, gasoline, petroleum naphthas, mixed distillates, synthetic fuels, and chlorinated hydrocarbons to laboratory animals and humans.

■ *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) (1984) (302 p.).
Source: Not available from API

32-01363

Introduction to Petroleum Hydrocarbons. [An Overview of the] Chemistry and Composition in Relation to Petroleum-Derived Fuels and Solvents that can be useful in the study of the effects of these materials on living organisms covers the alkanes, alkenes, naphthenes, aromatics, and their isomers; petroleum and petroleum products; and the compositions and characteristics of the feedstocks for, and product streams from, pipe stills, alkylation units, visbreakers, catalytic reformers and cracking units, polymerization units, and light ends plants; the post-World War II changes in gasoline composition; and the properties of solvents and of gasoline vapor. Flow diagram and tables.

■ W.G. Domask (Exxon Co. U.S.A.) in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 1-25 (1984).
Source: Not available from API

32-30187

Surveillance of Reproductive Health in the U.S.: A Survey of Activity Within and Outside Industry, including reproductive outcome monitoring programs that are operational or under development by nine petrochemical and other industrial companies, indicates that industry-sponsored programs are wider in scope than reproductive surveillance systems aimed at the general population, since industry integrates exposure data into its programs, while the latter deal almost exclusively with birth defects. Although none of the nine firms has started statistical analysis yet, there are standard methods (the Poisson method and the cumulative summation technique) available for evaluating data collected in a monitoring program. Recommendations for improving industry monitoring programs include the use of a simple, brief annual questionnaire to improve data collection; and steps to measure and increase participation rates. The major strengths and limitations of nationwide systems for surveillance of birth defects are discussed. Map, survey forms, and 108 references.

■ M. Hatch; V. Stefanchik-Scott (Columbia Univ.); Z. A. Stein (N.Y. State Psychiatric Inst.), *API Publication* (Dec. 1983) (204 p.).
Source: API HESD Information Specialist

32-30192
Hydrocarbon Toxicity: Acute, Subchronic and Chronic Effects in Relation to Unleaded Gasoline [(UG)] Exposure of Rodents, with Comments on the Significance to Human Health. A review by Universities Associated for Research & Education in Pathology Inc. (UAREP) of International Research & Development Corp.'s findings from a chronic inhalation study on mice and rats suggests that human exposure to gasoline at levels experienced in the general population poses little, if any, carcinogenic risk. A study on a group of unexposed controls and three groups (100 animals for each sex) of female and male Fischer rats and mice exposed to 67, 292, or 2056 ppm of UG for 6 hr/day, 5 days/wk for ~ 27 mo, showed unexpected renal neoplasms in male rats at all exposure levels, and an excess of hepatocellular neoplasia in high-dose female mice. UAREPs conclusions include the clear significance of the finding in male rats, which also showed subchronic and chronic renal lesions whose link to the carcinogenic process is uncertain; and a possibility that some UG components represent relatively weak carcinogens, that act as non-genetoxic promoters and/or cocarcinogenic agents, in male rats. Tables, diagrams, and 664 references.
■ *API Medical Research Publication* (Dec. 1983) (276 p.).
Source: API HESD Information Specialist

32-30231
Evaluation of Four Air Sampling Methods Used for Monitoring Worker Exposure to Gasoline Vapors. An evaluation at 25 °C of four sampling methods, including a 150 mg charcoal tube (A), a 600 mg charcoal tube (B), and a Three-M Co. Passive Organic Vapor Monitor single-stage Type 3500 (C) and double-stage Type 3520 (D) at about 30, 60, and 90% relative humidity (RH) and at about 30 and about 375 mg/cu m of gasoline in air showed that each method gave valid results at the lower concentration under all conditions, but that only method B was valid for an RH of > 60% at the higher concentration. Average recoveries (%) for all conditions for methods A, B, C, and D were 104.4, 89.9, 98.1, and 109.4, respectively, at 30 mg/cu m of gasoline and were 79.1, 71.2, 65.6, and 71.7, respectively, at 375 mg/cu m. All sampling media were exposed for 7 hr and were analyzed by a GC method based on U.S. National Institute for Occupational Safety & Health P&CAM 127. The gasoline used was type PS-6 unleaded regular. Flow diagram, diagrams, tables, graphs, and chromatograms.
■ *API Medical Research Publication* (May 1984) (60 p.).
Source: API HESD Information Specialist

32-30234
Chronic Inhalation Toxicity Study of Petroleum Coke (Delayed Process) in Rats and Monkeys. Three groups, each consisting of 150 male and 150 female Sprague-Dawley derived rats and 4 male and 4 female Cynomolgus monkeys, were exposed to clean air, or 10.2 or 30.7 mg/cu m of petroleum coke in air for 2 yr. No monkeys died during the study. The mortality rate for male and female rats was similar for exposed and control animals. Body weights, and ophthalmologic, hematologic, and biochemical parameters showed no adverse exposure--related effects in either sex of either species. Rat bone marrow cells showed no differences in chromosomal aberrations in the exposed and control group. The lung was the only organ to show any exposure-related weight differences; this was due to deposition of test material and related tissue responses. The animals showed increased lung weight after 3 mo exposure to 30.7 mg/cu m or 18 mo exposure to 10.2 mg/cu m. Rat lungs showed low-level inflammatory responses at 10.2 mg/cu m and significant neoplastic changes at 30.7 mg/cu m.
■ *API Medical Research Publication* (Dec. 1984) (15 p.).
Source: API HESD Information Specialist

32-30235
A Nine Day Inhalation Toxicity Study of Methyl t-Butyl Ether in the Rat. Sprague-Dawley-derived rats exposed for 6 hr/day for 9 days to MTBE at cumulative mean-levels of 101 ppm (II), 300 ppm (III), 1020 ppm (IV), or 2970 ppm (V) showed significantly elevated mean phosphorus levels in Group IV and V fasted females. Both sexes of fasted rats in Group V showed statistically increased relative liver weights; unfasted high-dose-level males showed a similar trend. No compound-related effects were revealed by gross pathology. Microscopic pathology revealed chronic inflammation in the nasal mucosa and trachea in Group IV and V rats. Hematology and urinalysis showed no exposure-related effects. Tables.
■ *API Medical Research Publication* (Mar. 1984) (63 p.).
Source: API HESD Information Specialist

32-30236
An Inhalation Teratology Study in Rats with Methyl t-Butyl Ether (MTBE). Female rats exposed for 6 hr/day to MTBE at cumulative mean levels of 250, 1000, and 2430 ppm during day 6 to day 15 of gestation showed no evidence of maternal toxicity or teratogenicity. Fetuses recovered from exposed rats on day 20 of gestation showed no teratogenic response in external, soft-tissue, and skeletal examinations. There was no mortality at any exposure level. No adverse effects were seen in body weight, water consumption, or physical in-life observations. Mean food consumption was significantly lower during days 9 through 12 of gestation in each of the treated groups. Treatment-related effects were seen neither in maternal gross postmortem examination data (including liver weight), nor in uterine implantation, fetal size, fetal sex distribution, or fetal ossification-variation data. Tables.
■ *API Medical Research Publication* (Mar. 1984) (19 p.).
Source: API HESD Information Specialist

32-30237
An Inhalation Teratology Study in Mice with Methyl t-Butyl Ether (MTBE). Female mice exposed for 6 hr/day to MTBE at cumulative mean levels of 280, 1110, and 2710 ppm during day 6 to day 15 of gestation showed no evidence of maternal toxicity or teratogenicity. There was no mortality during the treatment or post-treatment periods. There were no adverse effects in maternal body weight, food and water consumption, liver weight, or physical in-life parameters. Uterine implantation data showed a slight, but not significant, increase in the number of resorptions. Fetal weight, crown-rump distance, sex distribution data, and ossification variation data showed no adverse effect of treatment. There was a slight increase in the incidence of fetuses with fused sternebrae, but this was not considered to be a teratogenic response.
■ *API Medical Research Publication* (Mar. 1984) (23 p.).
Source: API HESD Information Specialist

32-30238
The Metabolic Fate of Methyl t-Butyl Ether (MTBE) Following an Acute Intraperitoneal Injection. Analyses of expired air, urine, and feces of rats administered one dose of ~ 60 µcuries of carbon-14-labeled MTBE at an average concentration of 232 mg/kg of body weight and sacrificed at intervals of 5 min to 48 hr post-treatment showed that 102% of the administered dose was labeled MTBE, labeled CO_2, or labeled formic acid. About 2% of the total administered dose could not be quantitated. An average of 91.75% of the total dose was identified as expired MTBE. Expired labeled CO_2 averaged 7.45% of the administered dose. A total of 3.08% of the administered dose was eliminated as formic acid in the urine and feces. Peak blood levels occurred 5 min post-treatment at an average of 92.04 and 83.40 µg/mL of labeled MTBE equivalents in the males and females, respectively; this value dropped to 5.72 and 3.27 µg/mL, respectively, 48 hr post-treatment. Tables, graphs, diagram, chromatogram.
■ *API Medical Research Publication* (Mar. 1984) (100 p.).
Source: API HESD Information Specialist

32-30239
A Single-Generation Inhalation Reproduction/Fertility Study in Rats with Methyl t-Butyl Ether (MTBE). Male rats were exposed to nominal MTBE levels of 250, 1000, or 2500 ppm for 6 hr/day, 5 days/week during 12 week premating and postmating periods, and 6 hrs/day during mating periods, and females were similarly exposed during a 3 week premating period, during mating, during the 0-20 day gestation period, and during days 5-21 of lactation. No adverse effects were noted in weight, in-life physical observations, mating indexes, male fertility indexes, or reproduction data, including gestation length, litter size, and litter survival indexes. There were no adverse effects on pregnancy rates during the first litter interval; during the second, pregnancy rates were

slightly lower than controls in each group, but the differences were not statistically significant. Pups nursing mid- and high-dose females had slightly lower mean weights at days 14 and 21 of lactation, and pup survival indexes during day 0-4 of lactation were lower in the low- and mid-dose groups; only the second of these differences was statistically significant. Tables.
■ *API Medical Research Publication* (Mar. 1984) (51 p.).
Source: API HESD Information Specialist

32-30240
Mutagenicity Evaluation Studies in the Rat Bone Marrow Cytogenetic Assay [and] in the Mouse Lymphoma Forward Mutation Assay. Hydrodesulfurized Kerosine, API Sample 81-07. Final Report. Studies by Litton Bionetics Inc. on bone marrow cells sampled 6, 24, and 48 hr after 140 male and female Sprague-Dawley rats were exposed intraperitoneally to 3, 1, or 0.3 g/kg/day of the test material, have shown that API #81-07 does not induce chromosomal aberrations in the cells. Neither the structural aberration frequency, nor the percentages of cells showing one or more structural aberrations, differed significantly from the negative controls. The Student t-test showed the same results with rats of both sexes. Positive controls exposed once to 0.8 mg/kg of triethylenemelamine showed significant increases in structural aberrations. In vitro treatments of mouse lymphoma L5178Y cells with up to 37.5 nL/mL of API #81-07 without rat liver S9 metabolic activation, or with 62.5 nL/mL with activation for 4 hr, suggest that the test material is inactive in the mouse lymphoma forward mutation assay. There were no significant increases in the mutant frequency at the thymidine kinase locus. Tables and diagrams.
■ *API Medical Research Publication* (Dec. 1984) (44 p.).
Source: API HESD Information Specialist

32-30407
Non-Neoplastic Exposure-Related Renal Lesions in [Fischer 344 Male] Rats Following Inhalation of [67-2056 Ppm] of Unleaded Gasoline Vapors for 6 hr/day and 5 days/week for 3, 6, 12, 18, and 24 mo distinguished the treated rats from the controls. These lesions, analyzed semi-quantitatively after the 3-12 mo exposures, consisted of increased foci of regenerative epithelium in the renal cortex and dilated tubules with intratubular protein at the corticomedullary junction. These lesions could not be identified as exposure-related after the 18 and 24 mo exposures because of the onset of spontaneous chronic progressive nephropathy in exposed and control rats. Rats exposed to 292 and 2056 ppm of unleaded gasoline for 12-24 mo had striking linear mineral deposits in the renal medullae, which distinguished them from the controls. These mineral deposits appeared as early as 12 mo, but more consistently at 18 and 24 mo. Tables, photomicrographs, and 10 references.
■ W. M. Busey; B. Y. Cockrell (Exp. Pathol. Lab. Inc.), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 57-64 (1984).
Source: Not available from API

32-30408
The Effect of Certain Light Hydrocarbons on Kidney Function and Structure in Male Rats. Male rats exposed to 100 or 800 ppm Stoddard solvent for 6 hr/day and 5 days/week for 8 weeks or to 300 or 900 ppm of C_{10}-C_{11} Isoparaffin for 8 weeks showed kidney structural damage and a pattern of change in kidney function that correlated well with the structural damage. Changes in urine parameters occurred after 4 or 8 weeks of exposure to either solvent. The primary changes in exposed male rats included a reduction in urine concentrating ability, an increase in total urinary protein and glucose, and an increase in urinary excretion of epithelial cells. Changes in kidney function were not observed prior to 4 weeks of exposure. Changes in kidney function due to nephrotoxicity were not apparent in females exposed to these solvents. Tables, photomicrographs, and 36 references.
■ R. D. Phillips (Exxon Corp.); B. Y. Cockrell (Exp. Pathol. Lab. Inc.), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 89-105 (1984).
Source: Not available from API

32-30409
The Pathogenesis of the Nephrotoxicity of Volatile Hydrocarbons in the Male Rat. The exposure of male and female rats, mice, dogs, and guinea pigs to decalin produced kidney effects only in male rats. In 91 day inhalation toxicity studies at 5 and 50 ppm, decalin induced hyaline droplet formation in the cytoplasm of the proximate convoluted tubule epithelial cells; induced the formation of granular casts at the junction of the inner and outer band of the outer zone of the medulla; and exacerbated chronic glomerulonephropathy. Hyaline droplet formation in male rats in response to decalin administered by oral gavage at 10-100 mg/kg/day for 5 days or by inhalation exposure at 125 ppm for 5-31 days was dose-responsive, but not time-responsive. Exaggerated hyaline droplet formation led to dose-dependent, but not time-dependent, tubular cell necrosis in male rats administered decalin by oral gavage at 0.1 or 1.0 g/kg/day for 5 days. Tables and photomicrographs.
■ C. L. Alden; R. L. Kanerva; Others (Procter & Gamble Co.), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 107-20 (1984).
Source: Not available from API

32-30410
A Review of the Human Kidney Effects of Hydrocarbon Exposure as covered in case reports, case-control studies, and cohort mortality studies on exposure to hydrocarbon and other solvents, gasoline, diesel fuel, gasoline-based paint spray, and mineral turpentine, provided only circumstantial evidence of an association between hydrocarbon exposure and glomerulonephritis and tubular dysfunction. Although five out of six case-control studies showed a positive association between such exposure and glomerulonephritis, only one was free of serious methodologic flaws. Six historical cohort mortality studies of hydrocarbon-exposed workers revealed generally lower-than-expected death rates from kidney disease, excluding cancer. Three cross-sectional surveys of renal function in hydrocarbon-exposed workers showed no significant changes in glomerular function and showed inconsistent minor abnormalities in tubular function. Considered in aggregate, these studies show little evidence for a functionally significant kidney effect from chronic low-dose occupational exposures. Recommendations are given for future studies. Tables and 34 references.
■ S. C. Phillips (Exxon Corp. Res. Environ. Health Div.), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 185-202 (1984).
Source: Not available from API

32-30411
[Seven] Epidemiologic Studies of the Role of Gasoline (Hydrocarbon) Exposure in Kidney Cancer Risk for which data were mainly drawn from a large retrospective cohort study of Gulf oil refinery workers, but in which analyses were extended to data from the entire company and from communities surrounding the particular refinery in Port Arthur, Tex., were examined by Gulf Oil Corp. Although kidney cancer mortality in the U.S. has increased in recent years, the data sets on workers with potential exposure to gasoline did not show any increase of kidney cancer risk over and above what would have been expected in the U.S. There were several increases of kidney cancer risk in a case-control study, but none reached statistical significance. Tables.
■ C. P. Wen; S. P. Tsai; Others (Gulf Oil Corp.),in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 245-57 (1984).
Source: Not available from API

32-30412
Xenobiotic-Induced Kidney Lesions: Hydrocarbons. [A Synopsis of] the 90-Day and 2-Year Gasoline Studies. In one study, albino rats and

squirrel monkeys were exposed to the whole vapors of 0, 384, or 1552 ppm of unleaded gasoline or 103 or 374 ppm of leaded gasoline for 6 hr/day and 5 days/wk for 13 weeks. In another study, Fischer 344 rats and B6C3F$_1$ mice were exposed to the whole vapors of 0-2056 ppm of unleaded EPA reference gasoline containing 2% benzene for 6 hr/day and 5 days/wk for 24-26 months. Response was measured with a variety of tests. Although various changes occurred in such variables as body weight and clinical parameters, microscopic examination of tissue sections provided the most striking evidence of damage. Other studies showed that a 28 day gavage procedure would be satisfactory for administering solvents or compounds to produce the characteristic early renal lesions. Tables.

■ H. N. MacFarland, in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 51-56 (1984).
Source: Not available from API

32-30413

Neoplastic Renal Effects of [Inhaled] Unleaded Gasoline in Fischer 344 Rats. In the exposure of Fischer 344 rats to 0, 67, 292, and 2056 ppm of unleaded gasoline vapor [*API Medical Research Publication* (Sept.1983) 57 p.], primary renal neoplasms occurred in 0, 2, 6, and 6 rats, respectively. All but one neoplasm occurred at or near the final sacrifice date, which was over 26 months. There was 3 renal adenomas, 9 carcinomas, and 1 sarcoma in male rats and 1 sarcoma in a female rat. Included was 1 adenoma observed at the 18 month sacrifice period. The oncogenic effects of inhaled unleaded gasoline on the rat kidney are discussed. Tables and 16 references.

■ D. N. Kitchen (Bio-Labs Inc.), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 65-71 (1984).
Source: Not available from API

32-30414

Renal Toxicity of [Vapors of] Gasoline and Related Petroleum Naphthas in Male Rats. Chemical class was a major factor in severe renal toxicity in male Sprague-Dawley rats exposed to the total vapors of light straight run, light catalytic cracked, light catalytic reformed, heavy catalytic reformed, full range alkylate, and polymerization naphthas as well as to an unleaded gasoline blend for 6 hr/day and 5 days/wk for a total of 15 exposures in 21 days. The alkanes appeared to be the most effective in producing nephrotoxicity. The alkenes, which were structurally similar to the alkanes, also produced nephrotoxicity, but aromatic hydrocarbons did not. Only the light catalytic cracked and heavy catalytic reformed naphthas produced no severe renal toxicity. In 65 exposures of both male and female rats to the unleaded gasoline vapor in a 90 day study, the severity and incidence of the lesions were considerably greater than observed in the 15 exposure study. It is possible that the full-range alkylate naphtha was primarily responsible for the nephrotoxicity in both studies. Photomicrographs, tables, and 10 references.

■ C. A. Halder (Standard Oil Co. (Indiana)); T. M. Warne (Amoco Oil Co.); N. S. Hatoum (IIT Res. Inst.), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 73-88 (1984).
Source: Not available from API

32-30415

Toxicology of Mixed Distillate and High-Energy Synthetic Fuels. Fischer 344 rats, Golden Syrian hamsters, C57B1/6 mice, dogs, and monkeys inhaled 30-5000 mg/cu m of JP-4, JP-5, JP-5 from oil shale, JP-7, JP-8, JP-TS, marine diesel fuel from petroleum and oil shale, and JP-10 and RJ-5 synthetic cruise missile fuels. After 24 hr/day, 90 day exposure, male rats showed renal proximal tubule degeneration and multifocal dilatation near the corticomedullary junction. At 19 months postexposure, male rat kidneys showed marked medullary mineralized casts, multifocal and diffuse hyperplasia of pelvic urothelium, and advanced tubular degeneration compatible with "old-rat nephropathy." After one year of intermittent (6 hr/day) exposure, there was no extensive proximal tubular degeneration, but there were significant numbers of renal tumors. Neither toxic nephropathy nor renal tumors were found in female rats of other animals. JP-10 is exo-tetrahydrodi(cyclopentadiene), and RJ-5 consists of hydrogenated dimers of norbornadiene. Tables and 14 references.

■ M. G. MacNaughton (U.S. Air Force Aerospace Med. Res. Lab. Wright-Patterson Air Force Base); D. E. Uddin (U.S. Nav. Med. Res. Inst., Wright-Patterson Air Force Base), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 121-32 (1984).
Source: Not available from API

32-30416

Pathologic Findings in Laboratory Animals Exposed to Hydrocarbon Fuels of Military Interest. Male and female Fischer 344 rats and female C57-BL6 mice were continuously exposed for 90 days, to 150-750 mg/cu m of petroleum and oil shale-derived JP-5, 50-300 mg/cu m of petroleum and oil shale-derived diesel fuel marine, 500-1000 mg/cu m of JP-4, and 28.3-283 mg/cu m of decalin. They were also exposed to 556 mg/cu m of JP-10 and 30-150 mg/cu m RJ-5 [API "Renal Effects Pet. Hydrocarbons" Symp. (Boston 7/18-20/83), pp. 121-32 (1984)] for 6 hr/day and 5 days/wk for one year. In general, male rats developed a dose-related nephropathy not observed in females, controls, or mice. Male rats held for long-term, oncogenic evaluation after exposure were affected in a way compatible with the progressive nephrosis known to occur in old rats. Male rats exposed to JP-10 and RJ-5 for one year had significant increases in renal cell tumors, but those exposed for 90 days to the various distillate fuels failed to develop increased kidney neoplasia following lifespan observations. Photomicrographs and 16 references.

■ R. H. Bruner (U.S. Air Force Aerospace Med. Res. Lab., Wright-Patterson Air Force Base), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 133-40 (1984).
Source: Not available from API

32-30417

A Brief Review of [Published Data on] the Toxicity and Carcinogenicity of Select Chlorinated Hydrocarbons, i.e., chloroform, carbon tetrachloride, and hexachlorobutadiene, as determined mainly in mice and rats by injection, inhalation, and percutaneous application relates the mechanisms of toxic action (when known) to sex, strain, and species differences in target organ response, and notes that the differences are apparently more quantitative than qualitative. Tables and 58 references.

■ M. M. Lipsky (Univ. Md. Sch. Med.), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 141-51 (1984).
Source: Not available from API

32-30418

[A Review of] the Epidemiology of Renal Carcinoma in Humans with a Note on the Effect of Exposure to Gasoline does not support any association with exposure to gasoline in the general population or with working in an oil refinery. The renal cancer incidence is highest in Scandinavia, lowest in Asia, and higher in males than in females. Moderate urban-rural and intra-country differences are observed in some countries. Graphs, maps, tables, and 59 references.

■ J. Higginson (Univ. Assoc. Res. Educ. Pathol. Inc.); C. S. Muir (Int. Agency Res. Cancer); P. A. Buffler (Univ. Tex.), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 203-26 (1984).
Source: Not available from API

32-30419

Risk Factors from a Population-Based Case-Control Study of Renal Cancer. Exposure to petroleum, tar, and pitch products was a risk factor for 495 cases of renal cell carcinoma but not for 74 cases of cancer of

the renal pelvis in a 1/1/74-6/30/79 study of white residents, including 697 controls, of the Minneapolis/St. Paul standard metropolitan statistical area. The risk was greater for those exposed to these products 20 or more years than for those exposed for less time. In general, cigarette smoking and obesity were major risk factors. Tables and 31 references.
■ J. K. McLaughlin (Johns Hopkins Sch. Hyg. Public Health), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 227-44 (1984).
Source: Not available from API

32-30420
Kidney Cancer Epidemiology in Petroleum-Related Studies. Eight studies of petroleum workers in the refining and distribution segments of the petroleum industry do not suggest any kidney cancer excess in refining populations and are inconclusive for distribution workers. None of the studies contains actual exposure information; and interpretation is limited because of the potential confounding variables. The criteria were formalized by A. B. Hill and updated by the International Agency for Research on Cancer. Tables and 23 references.
■ G. K. Raabe (Mobil Oil Corp.), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 259-71 (1984).
Source: Not available from API

32-30421
An Evaluation of the Significance of Experimental Hydrocarbon Toxicity to Man. A critical review of papers given at the API "Kidney Effects of Hydrocarbons" Workshop (Boston 7/18-20/83) and of some other studies covers subjects such as human kidney lesions related to hydrocarbon exposure, including inhaled unleaded gasoline, and the significance of acute and chronic hydrocarbons-induced renal lesions in rodents to the effects on humans, and concludes that some components of unleaded gasoline may be relatively weak carcinogens, perhaps acting as promoters and/or co-carcinogens, and that the gasoline exposure of the general population presents little, if any, risk. Graph, photomicrographs, and 23 references.
■ B. F. Trump; M. M. Lipsky; Others (Univ. Md. Sch. Med./Univ. Assoc. Res. Educ. Pathol. Inc.), in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. 273-88 (1984).
Source: Not available from API

32-30422
Summation/Comments on Structure-Activity Relationships. Data on renal toxicity of gasoline in male rats, on the effects of hydrocarbons exposure on the human kidney [Abstract No. 31-30556 and 31-30557], and from the series of API studies reported in *Toxicology & Applied Pharmacology* indicate that highly aromatic materials do not seem to produce nephrotoxicity. Alkanes, particularly isoalkanes, seems to be quite efficient in producing nephrotoxicity. The effect of naphthenes is somewhat intermediate between isoalkanes and aromatic hydrocarbons. Some data on C_6 hydrocarbons show a nephrotoxicity effect produced by continuous exposure and a very weak effect produced by intermittent exposure to higher concentrations. Tables.
■ R. Scala, in *Renal Effects of Petroleum Hydrocarbons*, ed. M.A. Mehlman, C.P. Hemstreet III, J.J. Thorpe, & N.K. Weaver (Princeton, N.J.: Princeton Scientific) (Advances in Modern Environmental Toxicology; vol. 7) pp. S1-S4 (1984).
Source: Not available from API

32-30534
Mutagenicity Evaluation Studies [of Catalytically Cracked Clarified Oil (API Sample #81-15)] in the Rat Bone Marrow Cytogenetic Assay and in the Mouse Lymphoma Forward Mutation Assay. Final Report. Studies by Litton Bionetics Inc. have shown that API #81-15 is negative in inducing chromosomal aberrations in rat bone marrow cells, and is considered active in the mouse lymphoma forward mutation assay with or without metabolic activation. The frequency of structural and numerical aberrations, and the percentages of cells with one or more structural aberrations in bone marrow cells of 66 male and female rats treated per os with 0.1, 0.3, or 1 g/kg/day of API #81-15 for five days did not differ significantly from those of the 20 male and female negative controls treated with corn oil. The 20 male and female positive controls treated once with 1 mg/kg of triethylenemelamine showed significant increases in structural aberration frequency. In in-vitro tests on mouse cells, API #81-15 significantly raised the mutant frequency at the thymidine kinase locus, and at 125 nL/mL without activation; and, with activation, was converted to a more toxic form or forms that induced dose-dependent increases in mutant frequency at 1.95-31.3 nL/mL. Tables and diagrams.
■ *API Medical Research Publication* (Feb. 1985) (41 p.).
Source: API HESD Information Specialist

32-30535
Mutagenicity Evaluation Studies [of Hydrodesulfurized Middle Distillate (D), API Sample 81-10] in the Rat Bone Marrow Cytogenetic Assay and in the Mouse Lymphoma Forward Mutation Assay. Final Report. Studies by Litton Bionetics Inc. have shown that API #81-10 does not induce chromosomal aberrations in rat bone marrow cells, and is considered active in the mouse lymphoma mutation assay only with rat liver S9 metabolic activation. The 90 male and female rats treated once intraperitoneally with 0.3, 1, or 3 g/kg of the test material and the 30 male and female negative controls showed similar frequency of structural and numerical aberrations in their bone marrow cells and similar percentages of cells with one or more structural aberrations. The 10 positive controls treated with 0.8 mg/kg of triethylenemelamine showed significant increases in structural aberration frequency at two dose levels. With metabolic activation, API #81-10 was converted to a form or forms that induced a dose-dependent increase in the mutant frequency of 2.1-3.4-fold above the background in in-vitro studies on mouse lymphoma cells. Without activation, low to moderately high toxicities were induced at 3.91-62.5 nL/mL without inducing significant increases in the background. Tables and diagrams.
■ *API Medical Research Publication* (Feb. 1985) (43 p.).
Source: API HESD Information Specialist

32-30599
Cadmium [(Cd)] : Environmental and Community Health Impact. According to published studies, Cd enters the petroleum industry as a trace contaminant of crude oil (0.2-29 µg/L), or through the purchase of it or its salts as a raw or intermediate material. The industry contributes ~ 17 tons/yr of Cd to the U.S. environment, or < 0.6% of the nation's annual Cd consumption. Cd tends to concentrate in refined petroleum products such as gasoline (10-50 µg/L), certain No. 2 distillate fuel oils (< 7-< 100 ηg/L), and oil and gas additives (< 20-< 900 µg/L). Cd concentrations in aqueous effluents from 17 refineries are < 1-< 20 µg/L. Also reviewed at length are the other sources of Cd; its chemistry; its fate and transport; its toxicodynamics in humans and aquatic species; its effects on aquatic organisms, domestic animals, and plants and their relationship to the human food-chain; human exposure to Cd via respiratory and gastrointestinal routes, and its potential acute and chronic effects on major organs; and federal standards regulating Cd in the air, land, water, and food. Tables and graphs; glossary and 255 references, 31 p.
■ *API Publication* (Jan. 1985) (205 p.).
Source: API Library

32-30600
Vanadium [(V)] : Environmental and Community Health Impact. According to a published study by the National Academy of Sciences, the combustion of residual fuel oils in the U.S. discharged 12,400-19,200 tons of V into the atmosphere in 1969 and 14,100-21,800 tons in 1970. Other published studies show that the combustion of distillate fuel oils and the use of V as catalyst in refineries contribute very little V to the atmosphere or environment; refinery effluents apparently have low V levels; and V concentrations in crude oil residua are 0.03-213.7 ppm vs. < 50 µg/L for distillate fuels (including gasoline, diesel fuel, kerosine, jet fuels, and home heating oils), < 3-2 000 µg/L for oil and gas additives,

< 10-90 µg/L for multigrade (10W-40) motor oil, and 6-35 ppm for drilling fluids and muds. The other sources of V; its chemistry; its environmental fate and transport; its toxicodynamics in humans and aquatic and marine species; its effects on aquatic and terrestrial organisms; its potential human health effects; and federal standards regulating V, are reviewed at length. Tables and graphs; glossary, and 146 references, 24 p.
■ *API Publication* (Jan. 1985) (179 p.).
Source: API Library

32-30626
[Thirty-Day] Dermal Carcinogenesis Study of Shale-Derived Hydrocarbon Mixtures [(350-550 °F and 550-700 °F)] in Mice. The results of a study by Battelle, Pacific Northwest Laboratories on mice painted interscapulary twice or thrice weekly for 4 wk with 25 µL each of ten shale-derived hydrocarbon mixtures, have shown that all the test materials induced some degree of acute inflammation, proliferation of epidermis, and atrophy of hair follicles and associated structures. The most significant microscopic lesion observed at the application site was epidermal ulceration in over 50% of the mice treated twice or thrice weekly with SFR-1-F4 or SFR-2-F4. This result suggests these fractions' potential to cause significant dermal toxicity problems in a chronic study that uses the same treatment regime. For a chronic study, it is thus recommended that these fractions and the other test materials be diluted 1:1 in toluene or other suitable organic solvent, and 50 µL of these diluted materials be applied twice weekly. Tables, photographs, and appendix, 21 p.
■ R. A. Renne (Battelle, Pac. Northwest Lab.), *API Medical Research Publication* (Feb. 1985) (36 p.).
Source: API HESD Information Specialist

32-30964
The Evaluation of the Carcinogenicity of Certain Petroleum Fractions. Mouse skin bioassays by the Kettering Laboratory on groups of 50 male mice treated twice weekly for 104 wk with 25 or 50 mg of ten fractions and subfractions of crude oils "C" and "D" showed that C3 saturates boiling at 350-550 °F, C5 and D5 fractions boiling at 700-1070 °F, D2 naphtha and D2 10% bottoms boiling at 120-350 °F, and D4 fraction boiling at 550-700 °F may be considered moderately carcinogenic. The C2 10% bottoms boiling at 120-350 °F and C4 saturates boiling at 550-700 °F were somewhat carcinogenic; the C2 naphtha boiling at 120-350 °F and C5 saturates boiling at 700-1070 °F showed little or no tumorigenic potency. Histological examination showed that the C2 and D2 10% bottoms, D2 naphtha, C3 saturates, C5, D5, and the BaP-treated controls produced one or two metastases from skin to lung or to spleen or to lymph node. Among the positive controls, 90-100% of those treated with 25 or 50 mg of 0.05% BaP in toluene developed tumors, often malignant and multiple. Tables and diagrams.
■ *API Medical Research Publication* (Feb. 1985) (160 p.).
Source: API HESD Information Specialist

32-30965
Mutagenicity Evaluation Studies in the Rat Bone Marrow Cytogenetic Assay and in the Mouse Lymphoma Forward Mutation Assay. Hydrodesulfurized Middle Distillate, API Sample 81-09. Final Report. In the rat bone marrow cytogenetic assay by Litton Bionetics Inc., the test material did not induce chromosomal aberrations in the ten male and ten female Sprague-Dawley rats after an acute intraperitoneal exposure to 0.3, 1, or 3 g/kg/day of API sample 81-09. Neither the structural aberration frequency in bone marrow cells, nor the percentages of cells showing one or more structural aberrations, nor the frequency of numerical aberrations, differed significantly from the negative controls. The positive controls treated with 0.8 mg/kg of triethylenemelamine showed significant increases in structural aberration frequencies. The test material is considered to have borderline activity in the mouse lymphoma forward mutation assay in the presence or absence of rat liver S9 metabolic activation. In activation and nonactivation assays at 31.3-1000 nL/mL, small but significant increases in the mutant frequency were induced at high toxicity. Tables and diagrams.
■ *API Medical Research Publication* (Feb. 1985) (43 p.).
Source: API HESD Information Specialist

32-30966
Four-Week Oral Nephrotoxicity Screening Study in Male F344 Rats [by Borriston Laboratories Inc.]. Final Report. A three-phase study on groups of ten male Fischer 344 rats which received 0.5 and 2 g/kg of API PS-6-fuel (reference control) and 22 hydrocarbon materials orally for 5 days a week for 4 wk showed no correlations among histopathologic changes, gross necropsy findings, and/or kidney weight differences. The nephropathy ranking, from highest to lowest, of the test materials is: ~ 220-280 °F API gasoline fraction, API 83-19 (light alkylate naphtha), 2,2,4-trimethylpentane, 2,3-dimethylpentane, 2,2,5-trimethylhexane, API PS-6-fuel, a 145-220 °F API gasoline fraction, 2,3-dimethylbutane, API 81-03 (light catalytically cracked naphtha), and 2-methylpentane. The materials that are comparable to saline (negative control material) in necropathy scores are 2-methyl-2-pentene, pentane, m-xylene, 2-methylbutane, methylcyclopentane, n-hexane, toluene, 1,2,4-trimethylbenzene, 2-methylhexane, trans-2-pentene, API 83-04 (light catalytically reformed naphtha), a 280 °F-plus API gasoline fraction, and API 83-18 (heavy catalytically cracked naphtha). Tables, 149 p.
■ *API Medical Research Publication* (Feb. 1985) (206 p.).
Source: API HESD Information Specialist

32-31048
Serial Sacrifice Study [by Borriston Laboratories Inc.] with [API PS-6] Unleaded Gasoline in Rats. Final Report. A study on groups of ten male and female Fischer 344 rats dosed daily by gavage with 0.5 and 2 g/kg of the test material for 28 days showed mortality among the treated rats only. One death among the low-dose male rats was due to dosing error. Findings include treatment-related decreases in body weight for the sacrificed high-dose males and females; significant increases in serum creatinine levels with corresponding decreases in creatinine clearance rates among the high-dose males on days 22 and 29, which correlated with treatment-related severe nephropathy in these rats; a possible relationship between the serum protein changes in male rats on day 8 and the advanced nature of the nephropathy by day 8, which were treatment-related; pronounced renal changes among the treated males on day 29, with the low dose just as nephrotoxic as the high dose; and an apparent resistance of female rats to the treatment-related nephropathy shown by the males. Tables, appendixes, addenda, and 11 references, 162 p.
■ *API Medical Research Publication* (Feb. 1985) (181 p.).
Source: API HESD Information Specialist

32-31232
Duration of [Human] Pulmonary Hyperresponsiveness to Acute Ozone Exposure. Final Report. Pulmonary function tests by the Institute of Environmental Stress on 19 males and 7 females who were exposed twice to 0.25 ppm ozone at 12, 24, 48, and 72 hr intervals while exercising on a cycle ergometer, suggest that the subjects were hyperresponsive to ozone exposure for at least 24 hr following the initial exposure. The mechanism for this hyperresponsiveness in humans has not been established. Although there was a trend toward a greater pulmonary function response among subjects re-exposed after 48 hr, the magnitude of the increase in response was much smaller than at 12 or 24 hr intervals. This trend seems to support a previous study and suggests a possibility of the persistence of the "residual effect" of the initial exposure for 48 hr with a larger subject population, or upon exposure to a higher concentration. A persistent symptom of discomfort on taking a deep breath 12 or 24 hr after exposure indicates persistence of the irritant response to ozone exposure. Tables, 18 references, and appendix, 34 p.
■ L. J. Folinsbee (Inst. Environ. Stress), *API Medical Research Publication* (Aug. 1984) (59 p.).
Source: API HESD Information Specialist

32-31233
[A Final Report by Litton Bionetics Inc. on] Mutagenicity Evaluation Studies in the Mouse Lymphoma Forward Mutation Assay [with] Sweetened Naphtha, [API] Sample 81-08, has shown that the test material is inactive in the mouse lymphoma cell system. Sweetened naphtha did not induce significant increases in the mutant frequency at the thymidine kinase locus in L5178Y mouse lymphoma cells. The exposure of cells to 12.5-300 nL/mL of the test material for 4 hr in the

presence of rat liver S9 metabolic activation induced low-to-very high toxicities, but no significant, dose-dependent increases in the mutant frequency were observed. A wide range of toxicities as induced in the absence of metabolic activation, but there were only sporadic, non-repeatable increases in the mutant frequency. Tables, diagram, and appendix, 4 p.
■ *API Medical Research Publication* (Mar. 1985) (29 p.).
Source: API HESD Information Specialist

32-31299
Lack of Concordance of the *Salmonella*/Microsome Assay with the Mouse Dermal Carcinogenesis Bioassay for Complex Petroleum Hydrocarbon Mixtures. The results from three laboratory studies on a paraffinic and a naphthenic whole crude oil and 11 of their distillate fractions in five strains of *Salmonella typhimurium*, with or without rat liver S-9 activation, indicate that the methods used routinely to perform the *Salmonella*/microsome assay are not useful in predicting the dermal carcinogenic activity of typical complex petroleum mixtures. None of the 13 samples showed mutagenic activity in the *Salmonella*/microsome assay, although 9 were lightly-to-highly dermally carcinogenic in mice, with the 371-577 °C heavy oil fractions of both whole crudes showing the most tumorigenicity (largely carcinomas) in 80-90% of the mice. These two samples also contained the highest levels of PAH, which nevertheless evoked a negative response in the *Salmonella* assay. Also, the lone carcinogenic sample (a 288-371 °C gas oil fraction of a paraffinic, high-sulfur foreign crude) which yielded a mutagenic response in strain TA-1537, without activation, contained < 0.1 ppb BaP and was only slightly-to-moderately tumorigenic. Tables and 12 references.
■ S. T. Cragg (API); C. C. Conaway (Texaco Res. Cent.); J. A. MacGregor (Chevron Environ. Health Cent.), *Fundamental and Applied Toxicology* 5:382-90 (1985).
Source: Not available from API

32-31300
Mutagenicity Evaluation Studies in the Rat Bone Marrow Cytogenetic Assay [and] in the Mouse Lymphoma Forward Mutation Assay of Light Catalytically Cracked Naphtha, [API] Sample 81-03. Final Report. Studies by Litton Bionetics Inc. on ten male and ten female Sprague-Dawley rats exposed by inhalation five times, 24 hr apart, to 63, 297, or 2046 ppm of the test material have shown that API sample 81-03 is negative in inducing chromosomal aberrations in rat bone marrow cells. Neither the structural aberration frequency, nor the percentages of cells showing one or more structural aberrations in the bone marrow cells of the exposed rats, differed significantly from the negative controls. An acute exposure of control animals to 0.8 mg/kg of triethylenemelamine, the positive control substance, significantly increased the structural aberration frequency. The test material did not significantly increase the mutant frequency of the thymidine kinase locus in L5178Y mouse lymphoma cells. Concentrations of 7.81-125 nL/mL without rat liver S9 metabolic activation, and of 7.81-300 nL/mL with metabolic activation induced a wide range of toxicities without inducing significant increases in the mutant frequency. Tables, appendix, and diagrams, 23 p.
■ *API Medical Research Publication* (Feb. 1985) (54 p.).
Source: API HESD Information Specialist

32-31472
Thirteen-Week Inhalation Toxicity Study of C_4/C_5 Hydrocarbon Blends in Rats. Final Report. Studies by the IIT Research Institute on several male and female Fischer 344 rats which were exposed to 1000 and 4500 ppm of 50:50 mixtures of n-butane/n-pentane, or of isobutane/isopentane, for 6 hr/day, 5 days/wk for 13 wk, have shown that the body weights of those exposed to isobutane/isopentane were unaffected relative to those of the unexposed controls. Rats exposed to n-butane/n-pentane showed statistically significant decreases in body weights, with the males recovering towards the end of the exposure period. Necropsy showed gross lesions that were spontaneous and non-treatment-related, no effect on liver and kidney weights, and no evidence of hydrocarbon nephropathy. Tables, graphs, diagram, and appendixes, 33 p.
■ C. Aranyi (IIT Res. Inst.), *API Medical Research Publication* (Mar. 1985) (85 p.).
Source: API HESD Information Specialist

32-31473
Acute Oral Toxicity Study in Rats, Acute Dermal Toxicity Study in Rabbits, Primary Dermal Irritation Study in Rabbits, and Primary Eye Irritation Study in Rabbits of API 83-04 Light Catalytically Reformed Naphtha. Studies by Hazleton Laboratories America Inc. on male and female Sprague-Dawley albino rats showed an oral LD_{50} of > 5 g/kg of body weight, since there was no mortality at a dosage of 5 g/kg of the test material. Necropsy showed no visible lesions. The acute dermal toxicity test on male and female New Zealand white rabbits showed an LD_{50} of > 2 g/kg of body weight. Dermal irritation ranged from moderate to severe for erythema and edema, and from slight to marked for atonia, desquamation, coriaceousness, and fissuring. Necropsy showed thickened and crusted skin on the test area, and red foci on the cortex of one rabbit's kidney. Rabbits treated with 0.5 mL of the test material for 24 hr showed a primary dermal irritation index of 2.0. A 0.1 mL treatment in the primary eye irritation test showed corneal, iridal, and conjunctival irritation in rabbits whose eyes were left unwashed, but only conjunctival irritation in those whose eyes were washed. All irritation cleared within 7 days. Tables and addenda, 3 p.
■ *API Medical Research Publication* (Apr. 1985) (24 p.).
Source: API HESD Information Specialist

32-31474
[Hazleton Laboratories America Inc.'s] Acute Oral Toxicity Study in Rats, Acute Dermal Toxicity Study in Rabbits, Primary Dermal Irritation Study in Rabbits, and Primary Eye Irritation Study in Rabbits of API 83-05 Full Range Catalytically Reformed Naphtha. Male and female Sprague-Dawley albino rats dosed with 3.57-9.80 g/kg of the test material showed respective oral LD_{50} values of 6.62 and 5.39 g/kg of body weight at respective 95% confidence limits of 6.20-7.08 and 4.23-6.86 g/kg. All the male rats in the 7 and 9.80 g/kg groups and all the female rats in the 9.80 g/kg group died. All surviving rats, except those which showed hair loss, returned to normal. Necropsy findings included stained stomach mucosa among the highest dosed rats. Male and female New Zealand white rabbits showed an estimated dermal LD_{50} of > 2.0 g/kg of body weight. Clinical signs included slight-to-severe erythema and moderate-to-severe edema; necropsy results included reddened, thickened, and crusted skin on the test area. Rabbits showed a primary dermal irritation index of 3.1 at a 0.5 mL treatment. The primary eye irritation test at 0.1 mL showed corneal, iridal, and conjunctival irritation among rabbits with unwashed eyes, but only the last sign among those with washed eyes. Tables and addenda, 3 p.
■ *API Medical Research Publication* (Apr. 1985) (29 p.).
Source: API HESD Information Specialist

32-31511
Second Follow-Up Update, Independent Verification of Vital Status, and Clarification of Cohort Definition and Initial Data Collection of OH-1 Cohort. This second study update by SRI International, which updates through 12/31/80 the original study by Tabershaw-Cooper Associates Inc. and SRI's first study update on the vital status of all the cohort members from 17 refineries, suggests that, because of the low standardized mortality ratios (SMRs), the OH-1 cohort is at lower risk for all deaths and for many specific causes of death than its counterpart in the general population. Of the 20,169 individual records in the study cohort file, 19,991 were eligible study cohort members. Of those eligible, 15,935 were known to be alive on 12/31/80, 3349 were deceased, and 707 were of unknown vital status. The SMR for all causes of death was 78, with a 95% confidence interval of 76-81. All but 80 death certificates were obtained. Each non-neoplastic cause had an SMR below 100; the SMR for all cancers was 87. SMRs for specific neoplasms included 89 for brain cancer, 108 for benign and unspecified brain neoplasms, and 123 for multiple myeloma. The limitations of this update are discussed. Tables and 21 references.
■ *API Medical Research Publication* (Mar. 1985) (37 p.).
Source: API HESD Information Specialist

32-31512
Effects of Subchronic Fourteen-Day Exposure to Benzopyrene in B6C3F1 Female Mice on Host Resistance. Final Report [by the Medical College of Virginia]. Studies on B6C3F1 female mice, which

were exposed via subcutaneous injection for 14 days to 5, 20, or 40 mg/kg of BaP, and to 40 mg/kg of benzo[e]pyrene (BeP), and then challenged with *Listeria monocytogenes*, *Streptococcus pneumoniae*, Herpes Simplex Type 2 (HSV-2), Influenza A_2, or B16F10 melanoma, have shown a relationship between altered immune function and change in host resistance. BaP increased resistance to *L. monocytogenes*; decreased resistance to B16F10 melanoma, HSV-2, and *S. pneumoniae*; and did not significantly affect resistance to Influenza A_2. BeP caused no change in resistance to any of the pathogen or tumor cells. A single injection of 200 mg/kg of cyclophosphamide to positive control mice decreased resistance to all the pathogen and tumor cells. Two studies to amend the protocol showed no effect of benzopyrenes on natural killer cell activity, and no recovery of control mice from the effects on humoral immunity for 90 days after the 14-day exposure. Tables, graphs, charts, and appendixes, 41 p. See also Abstract No. 30-32850.
■ A. E. Munson; K. L. White; H. H. Lysy (Med. Coll. Va.), *API Medical Research Publication* (Apr. 1985) (159 p.).
Source: API HESD Information Specialist

32-31555
Industrial Hygiene Assessment of Petroleum Refinery Turnaround Activities. Final Report. Evaluations by Dynamac Corp. of worker exposures during performance of over 30 maintenance turnaround activities involving a thermal cracking unit and an alkane polymerization unit in a small refinery, and an FCC unit and a vapor recovery unit in a medium-sized refinery, have shown that exposures to certain agents were well below the U.S. Occupational Safety & Health Administration's permissible exposure limits (PELs). Such agents included aromatic hydrocarbons (BTX and cumene), PAHs, H_2S, mercaptans, phosphoric acid, sodium hydroxide, asbestos, and metal fumes. Several activities, such as cutting and welding inside a regenerator, generated noise at levels above PELs; and some job categories, such as sandblasting, caused excessive dust and noise exposures. Precautionary measures, such as the use of only earplugs during decoking of towers, were insufficient to provide adequate protection at some activities. Tables and appendixes, 30 p.
■ *API Medical Research Publication* (Mar. 1985) (71 p.).
Source: API HESD Information Specialist

32-31708
Acute Oral Toxicity Study in Rats, Acute Dermal Toxicity Study in Rabbits, Primary Dermal Irritation Study in Rabbits, [and] Primary Eye Irritation Study in Rabbits of API 81-04, Light Catalytically Cracked Naphtha. Administration by single-dose oral gavage of 6.76 mL/kg of body weight of this highly aromatic, low-olefin naphtha caused some clinical signs which had subsided by day five of the study. The estimated oral LD_{50} value was > 5.0 g/kg for both male and female rats. One application of the test material to the skin caused dermal irritation ranging from slight to severe for erythema and from slight to marked for atonia, desquamation, coriaceousness, and fissuring, and yielded an estimated dermal LD_{50} value of > 2.0 g/kg of body weight for both male and female rabbits. In the primary dermal irritation test on rabbits, the test material produced slight to severe levels of irritation after a 20 hr exposure, but all irritation had subsided by day 20. The primary dermal irritation index was 4.0. In the primary eye irritation test, the scores were 4.3, 0.3, 0.7, 0.0, and 0.0 after 1, 24, 48, and 72 hr, and 7 days, respectively, for rabbits whose eyes were untreated after one administration of the test material. The corresponding scores for rabbits whose eyes were rinsed right after exposure were 6.0, 0.0, 0.0, 0.0, and 0.0. Tables and addenda, 8p.
■ *API Medical Research Publication* (Apr. 1985) (24 p.).
Source: API HESD Information Specialist

32-31709
L5178Y TK+/- Mouse Lymphoma Mutagenesis Assay of API 83-08, [Light Catalytic Cracked Distillate] boiling at 150-400 °C and consisting mainly of C_9-C_{25} hydrocarbons, with a large amount of bicyclic aromatics, indicated that API 83-08 produced a positive response in the presence and absence of exogenous metabolic activation under the test conditions. Three nonactivated, in-vitro assays were made; in the first, one culture having 1% total growth exhibited a mutant frequency more than twice the mean mutant frequency of the solvent controls. In the second, the results were invalid. In the third, five cloned cultures having 0-86% total growth and representing three dose levels exhibited mutant frequencies more than twice the mean mutant frequency of the controls. In an assay activated with Aroclor-induced rat liver S-9 (microsomes), one cloned culture showed a mutant frequency significantly greater than the mean mutant frequency of the solvent controls. A dose-dependent response was noted in the treated cultures. Diagram, graphs, table, and appendix, 27 p.
■ *API Medical Research Publication* (Apr. 1985) (48 p.).
Source: API HESD Information Specialist

32-31710
L5178Y TK+/- Mouse Lymphoma Mutagenesis Assay of API 81-04, [Light Catalytic Cracked Naphtha] boiling at ~ -20 to +190 °C, consisting mainly of C_4-C_{11} hydrocarbons, and containing a relatively large proportion of unsaturated hydrocarbons, yielded results considered negative in the absence of exogenous metabolic activation and equivocal in its presence. In the first assay, where total growth was 26-122%, all the cultures that were cloned, either in the presence of Aroclor-induced rat liver S-9 or its absence, had mutant frequencies that were not significantly different from the mean mutant frequency of the solvent controls, so that this assay was considered negative. In a repeat assay, made because of the test article's "precipitous" toxic response, none of the nonactivated, cloned cultures, which showed 9-121% total growth, gave a positive result. Two of the S-9-activated, cloned cultures, which showed 4-79% total growth, gave mutant frequencies more than twice the mean response of the solvent controls in the repeat assay. The dose-response relationship for toxicity in the presence and absence of activation was not clear cut. Diagram, graphs, tables, and appendix, 31 p.
■ *API Medical Research Publication* (Apr. 1985) (57 p.).
Source: API HESD Information Specialist

32-31768
An L5178Y TK+/- Mouse Lymphoma Mutagenesis Assay of API 83-11 [(Straight Run Middle Distillate) by Microbiological Associates Inc.] has shown that API 83-11 produced a positive response in the presence and absence of Aroclor-induced rat liver S-9. After a two-day expression period in which the nonactivated and activated cultures were treated with 0.013-1.0 and with 0.0067-0.5 µL/mL of API 83-11, respectively, ten each of the nonactivated and activated cultures were cloned and treated with API 83-11 with respective concentrations of up to 0.56 and up to 0.16 µL/mL. There was a dose-dependent response, with four of the cloned nonactivated, and two of the cloned activated, cultures showing mutant frequencies which were more than twice the mean mutant frequencies of the solvent controls, with respective total cell growths of 0-10% and 4 and 9%. Mutant frequency increases in cultures with < 10% total growth are usually not considered significant, but for API 83-11, it is considered that cultures with 10% total growth would also show a mutant frequency that was at least twice that of the solvent control. Tables, graphs, diagram, and appendix, 26 p.
■ *API Medical Research Publication* (Apr. 1985) (46 p.).
Source: API HESD Information Specialist

32-31769
The Acute In-Vivo Cytogenetics Assay in Male and Female Rats of API 83-09 [(Straight-Run Kerosine)]. Final Report. A study by Microbiological Associates Inc. on 13 groups of five male and five female Sprague-Dawley rats each, which received a single intraperitoneal injection of 0.3, 1, or 3 gm/kg of API 83-09 in corn oil, has shown that the test material did not induce chromosomal aberrations in bone marrow cells. Among the high-dose group, three male rats died within 24 hr of injection, and one additional male died within minutes of colchicine administration for the 48 hr sacrifice. There were weight losses of 7-10% and 0-3% in both male and female rats 24-48 hr after treatment with 3 and 1 gm/kg of API 83-09, respectively. API 83-09 had no apparent effect on the mitotic index; and did not significantly increase the incidence of cells with aberrations, or the number of aberrations per cell relative to the controls treated with 5 mL/kg of corn oil. Control rats treated with 0.5 mg/kg of triethylenemelamine induced an average of

2.646 aberrations per cell, with 31.6% of all cells containing one or more aberrations. Tables and glossary, 9 p.
■ *API Medical Research Publication* (May 1985) (23 p.).
Source: API HESD Information Specialist

32-32118
CHO/HGPRT [(Chinese Hamster Ovary/Hypoxanthine-Guanine Phosphoribosyl Transferase)] Mammalian Cell Forward Gene Mutation Assay of API 81-15. The assay, which fulfilled all the criteria for an acceptable procedure, demonstrated the nonmutagenicity of API 81-15, a catalytically cracked, clarified petroleum oil (0.1 °API, 1.0753 sp gr, a 56.1 SUS/210 °F viscosity, > 350 °C bp), consisting mainly of > C_{20} hydrocarbons, and probably containing 5 wt % or more of 4- to 6-membered condensed ring aromatic hydrocarbons. In the assay, 81-15 was evaluated in duplicate cultures in the mutation assay at dose levels of 0.1, 1.0, 3, 10, and 30 µg/ml without activation, and also at 0.1, 1.0, 10, 100, and 200 µg/ml with activation by Aroclor 1254-induced male Sprague-Dawley rat liver homogenate (S-9). The positive controls evaluated concurrently were ethyl methane sulfonate, a mutagen not requiring S-9 activation, and dimethylnitrosamine, a mutagen requiring S-9 activation. Before the assay, 81-15 was submitted to two cytotoxicity prescreens. Tables, 2 p.
■ *API Medical Research Publication* (June 1985) (17 p.).
Source: API HESD Information Specialist

32-32165
Motor Fuel Chronic Inhalation Study...Unleaded Gasoline. Three groups, each containing 100 male and 100 female Fischer 344 rats and 100 male and 100 female B6C3F_1 mice, were exposed to 67, 292, and 2056 ppm API PS-6 unleaded gasoline vapors by whole-body inhalation exposure for 6 hr/day, 5 days/week for about 2 yr. A fourth, control group containing a similar number of animals was exposed to filtered air. There were no consistent and toxicologically significant pharmacotoxic signs observed in either rats or mice which could be attributed to the exposure conditions. The spontaneous mortality rate for rats and mice was not adversely affected by exposure to the vapors. There were no exposure-related abnormalities apparent from any of the various hematological or serum biochemical parameters evaluated in male or female rats. Kidney weights in the male rats exposed to 2056 ppm were elevated as early as the three month interim; this elevation was observed at all subsequent evaluation intervals. Kidney weights in female rats and male and female mice were not affected by gasoline exposure. The most significant histological finding was renal carcinoma in male rats at all the exposure levels. Diagrams, graphs, tables, and 26 references, 153 p.
■ *API Medical Research Publication* (9/15/83) (212 p.).
Source: API HESD Information Specialist

32-32166
Mutagenicity Evaluation of API #83-11 in the Mouse Lymphoma Forward Mutation Assay. In vitro treatments of the mouse lymphoma cell line, L5178Y, with API #83-11, a C_{11}-C_{20} straight-run petroleum middle distillate boiling at 205-345 °C, induced significant increases in the mutant frequency at the thymidine kinase locus only when metabolic activation was present. With activation by rat liver S9, exposure of the cells to 50-1000 n L/mL of #83-11 for 4 hr induced repeatable increases in mutant frequency of 2.3 to 5.9-fold above the background. Without activation, exposure of the cells for 4 hr to 100-1000 nL/mL of #83-11 induced a wide range of toxicities but not significant, dose-dependent increases above the background mutant frequency. The test material was insoluble at and above 500 nL/mL. Table, 1 p.
■ *API Medical Research Publication* (June 1985) (23 p.).
Source: API HESD Information Specialist

32-32167
Mutagenicity Evaluation of API 83-07 in the Mouse Lymphoma Forward Mutation Assay. Final Reports. In-vitro treatments of the mouse lymphoma cell line L5178Y with API #83-07, a C_9-C_{25} light catalytic cracked petroleum distillate boiling at ~ 150-400 °C, induced significant increases in the mutation frequency at the thymidine kinase locus only when metabolic activation was present. In the presence of rat liver S9 metabolic activation, assays of #83-07 at 2.5-25 nL/mL concentrations for 4 hr caused significant dose-dependent increases in the mutant frequencies above 5.0 nL/mL, where low to very high toxicities were induced. When the cells were exposed to 5-60 nL/mL concentrations #87-07 for 4 hr under nonactivation conditions, there were no significant increases in mutant frequency, and nondetectable to moderate toxicities were induced. A small increase in concentration from 60 to 80 nL/mL was excessively toxic; and the treatment could not be cloned. Table, 1 p.
■ *API Medical Research Publication* (June 1985) (22 p.).
Source: API HESD Information Specialist

32-32168
Mutagenicity Evaluation of API #83-04 in the Mouse Lymphoma Forward Mutation Assay. Final Report. In vitro treatments of the mouse lymphoma cell line L5178Y with API #83-04, a C_5-C_{11} light, catalytic reformed naphtha boiling at ~ 35-190 °C, induced no reliable increases in the mutation frequency at the thymidine kinase locus, with or without metabolic activation, and was therefore inactive in this cell system. Under nonactivation conditions, exposure to 25-128 nL/mL of #83-04 for 4 hr induced no significant increases in mutant frequency but did induce a wide range of toxicities; the 125 nL/mL concentration was too toxic to be used in the mutation assay. With metabolic activation by rat liver S9, 4 hr treatment of the cells with 25-150 nL/mL of #83-04 induced a wide range of toxicities. Sporadic increases in mutant frequency were induced, but the increases were borderline and dose-dependent. API #83-04 was soluble at all the concentrations tested. Table, 1 p.
■ *API Medical Research Publication* (June 1985) (24 p.).
Source: API HESD Information Specialist

32-32169
Acute Inhalation Toxicity Evaluation of a Petroleum-Derived Hydrocarbon in Rats. API #83-06, Heavy Catalytically Reformed Naphtha. Final Report. Whole-body exposure of 5 young adult rats of each sex to ~ 5 mg/mL in air of API #83-06, a C_7-C_{12} heavy naphtha boiling at } 90-230 °C produced no clinical signs, caused no animal deaths, and did not affect individual and mean body weights during the 4 hr exposure or during a subsequent 14 day observation period. The inflammatory lesions observed grossly and microscopically in the lungs of rats 14 days after exposure were of a type and intensity commonly observed in unexposed laboratory rats. The significance, if any, of the inhalation exposure in generating the lesions could not be established. Table and appendixes, 25 p.
■ *API Medical Research Publication* (June 1985) (34 p.).
Source: API HESD Information Specialist

32-32225
Feasibility of Case-Control Studies of Kidney Cancer and Hydrocarbon Exposure among Petroleum Company Workers. An Epidemiology Resources Inc. study indicates that it is feasible to conduct a case-control study of adenocarcinoma of the renal parenchyma among petroleum company workers exposed to hydrocarbons, notably those found in unleaded gasoline, by using existing cohorts of refinery workers in seven of the 11 oil firms that participated in the study and/or by using the mortality registries (death certificates) that all the firms have. Published cohort mortality studies from five of the 11 firms report 49 kidney cancer deaths during 1928-79; forthcoming results from the other firms suggest 53 more such deaths. The participants are Exxon Corp., Amoco Oil Co., Gulf Oil Corp., Mobil Corp., Shell Canada Ltd., Standard Oil Co. of California, Texaco Inc., Atlantic Richfield Co., Shell Oil Co., Sun Co. Inc., and Conoco Inc. The feasibility criteria, i.e., company willingness to participate, case ascertainment, control selection, exposure characterization, potential confounders, and study size, are discussed. Tables, charts, appendixes, and 83 references, 24 p.
■ *API Medical Research Publication* (5/24/85) (73 p.).
Source: API HESD Information Specialist

32-32227
A Chronic Inhalation Study with Unleaded Gasoline Vapor. Similar to Abstract No. 30-31991.
■ H. N. MacFarland (Unaffiliated); C. E. Ulrich (Int. Res. Dev. Corp.);

C. E. Holdsworth (API); D. N. Kitchen (Biolabs Inc.); W. H. Halliwell (Westpath Inc.); S. C. Blum (Exxon Res. Eng. Co.), *Journal of the American College of Toxicology* 3(4):231-48 (1984).
Source: Not available from API

32-32288
Activity of API 81-04 in the Acute In-Vivo Cytogenetics Assay in Male and Female Rats. Final Report. Tests by Microbiological Associates Inc. on 13 groups of five male and five female Sprague-Dawley rats, which received a single intraperitoneal injection of 0.3, 1.0, or 3.0 g/kg of API 81-04 and which were sacrificed 6, 24, or 48 hr after treatment, showed that the test material did not induce significant increases in the incidence or number of chromosome aberrations in the rats' bone marrow cells. API 81-04 is an ~ -20 to +190 °C light catalytic cracked naphtha consisting mainly of C_4-C_{11} hydrocarbons, with a relatively large proportion of unsaturated hydrocarbons. Clinical signs of toxicity among the rats treated with 3 g/kg included lethargy and increased tearing. There were no apparent change in ploidy, or effect on the mitotic index. The positive control rats treated with 0.5 mg/kg of triethylenemelamine showed an average of 0.438 aberrations per cell, with 16.6% of all analyzed cells containing one or more aberrations. Tables, 8 p.
■ *API Medical Research Publication* (May 1985) (24 p.).
Source: API HESD Information Specialist

32-32289
Mutagenicity Evaluation of 83-05 in the Rat Bone Marrow Cytogenetic Assay. Final Report. Intraperitoneal injection of a single 0.25, 0.83, or 2.5 g/kg dose of neat API 83-05 into groups of male and female rats, followed by extraction of the bone marrow and microscopic examination of the cells therefrom showed that 83-05 did not induce chromosomal aberrations under the assay conditions and is negative in this test on rat bone marrow cells. The negative controls were dosed once with corn oil and the positive controls once with triethylenemelamine in saline. API 83-05, a catalytic reformed naphtha of 44.0 °API, consists mainly of C_4-C_{12} hydrocarbons boiling at ~ 30-22 °C and containing a large proportion of aromatic and branched-chain hydrocarbons and possibly ≥ 10 vol % benzenes. Diagrams, tables, and appendix 22 p.
■ *API Medical Research Publication* (June 1985) (45 p.).
Source: API HESD Information Specialist

32-32309
Numerical Modeling of Ozone Population Exposure: Application to a Comparison of Alternative Ozone Standards. Final Report. A population-exposure modeling technique relying on a modified version of the National Ambient Air Quality Standards (NAAQS) Exposure Model (NEM) has been developed to simulate high ozone exposure episodes. Reductions in the computational effort and associated cost of such evaluations could be achieved, since the attainment-year exposure distribution could be derived analytically from the current-year distribution, provided that the attainment-year and current-year concentrations were related through linear rollback. Adjustments in the procedures by which the NEM is used were also made to improve the relative accuracy. When simulation of ambient ozone concentration patterns typical of those occurring upon the attainment of several different standards was applied to a two-day ozone episode in Los Angeles, the results confirmed the need to do explicit population exposure modeling to characterize the differences between attainment of alternative ozone standards. Also, estimating attainment-year ozone air quality by using linear rollback, as the EPA has done, could cause population exposure to be over- or underestimated, and the direction of the error would be unpredictable. Diagrams, graphs, maps, tables, and 35 references.
■ S. R. Hayes; C. Seigneur; G. W. Lundberg (Syst. Appl. Inc.), *API Publication* #4400 (Aug. 1984) (106 p.).
Source: API Library

32-32405
Thirteen-Week Inhalation Toxicity Study of a 0 to 145 °F Gasoline Distillate Fraction in Rats. Final Report. Studies by the IIT Research Institute on 152 male and female Fischer 344 rats which were exposed to 1000 or 4500 ppm of a 0-145 °F boiling cut of PS-6 gasoline for 6 hr/day, 5 days/wk, for 13 wk, showed no relation between the exposures and clinical signs such as diarrhea and redness of the eyes. The test material, composed mainly of C_4-C_6 hydrocarbons, with some traces of C_3 and C_7 compounds, did not affect the body weight of the exposed rats relative to that of the controls. Comparative necropsies on control rats exposed to filtered air and on the hydrocarbon-exposed rats showed no hydrocarbon nephropathy in male or female rats, because the liver and kidney weights of the males were unaffected by the exposure and because the statistically significant increases in the kidney and liver weights of the females were biologically nonsignificant. The gross lesions were spontaneous and unrelated to the exposures. Tables, charts, diagram, appendixes, and addenda, 44 p. See also Abstract No. 32-31472.
■ C. Aranyi (IIT Res. Inst.), *API Medical Research Publication* (May 1985) (87 p.).
Source: API HESD Information Specialist

32-32406
An Evaluation of the Potential of RO-1, 81-15, and PS8-76D5-SAT To Induce Unscheduled DNA Synthesis [(UDS)] in the In Vivo-In Vitro Hepatocyte DNA Repair Assay indicated that 81-15 is a genotoxic agent in this assay, and that the other test materials are not, since they showed no increase in UDS (amount of incorporated tritiated thymidine). These test articles may exhibit other toxic, mutagenic, or carcinogenic effects, however, because this assay is unresponsive to carcinogens that produce tumors in tissues other than the liver. In the tests, Fischer-344 male rats were exposed by gavage to 50-1000 mg/kg of the test articles suspended in corn oil, to corn oil alone, or to the positive control, 2-acetylaminofluorene, in corn oil, and were sacrificed 2 or 12 hr after the treatment. In the subsequent assay on primary rat liver cell cultures, the test article was positive if UDS was markedly elevated above that in the solvent control. Tables, 10 references, and appendix, 26 p.
■ *API Medical Research Publication* (June 1985) (41 p.).
Source: API HESD Information Specialist

32-32407
An Evaluation of the Potential of RO-1, 81-15, and PS8-76D5-SAT To Induce Unscheduled DNA Synthesis [(UDS)] in Primary Rat Hepatocyte Cultures indicated that RO-1 and 81-15 are genotoxic in the in vitro rat hepatocyte DNA repair assay, since an increase in UDS and in the percentage of cells in repair above solvent control values was observed for both, and that PS8 is not genotoxic. In the assays by the G.M. Williams method, which can detect, inter alia, PAHs, nitrosamines, and aromatic amines, the positive control was 2-acetylaminofluorene, and there was an untreated medium control, and a negative solvent control in the culture medium. The UDS assay was made at 5 noncytotoxic concentrations of RO-1, 6 noncytotoxic concentrations of 81-15, d 6 concentrations of PS8-76D5-SAT, at respective maximum concentrations of 5000, 1000, and 5000 µg/mL in a medium that contained 1% dimethyl sulfoxide but was serum-free. Tables and appendix, 44 p.
■ *API Medical Research Publication* (June 1985) (64 p.).
Source: API HESD Information Specialist

32-32408
Acute In-Vivo Cytogenetics Assay in Male and Female Rats of API 83-11. Final Report. Tests by Microbiological Associates Inc. on 13 groups of five male and five female Sprague-Dawley rats, which were given a single intraperitoneal injection of 0.3, 1.0, or 3.0 g/kg of API 83-11, a 205-345 °C straight-run petroleum middle distillate containing mainly C_{11}-C_{20} hydrocarbons, and which were sacrificed 6, 24, or 48 hr after injection, showed that the test material did not induce chromosome aberrations in bone marrow cells. Neither the incidence of cells with aberrations, nor the number of aberrations per cell, was statistically or significantly increased relative to the vehicle controls. There was no apparent change in the ploidy or on the mitotic index. The positive controls treated once with 0.5 mg/kg of triethylenemelamine showed an average of 2.358 aberrations per cell, with 35.4% of all cells analyzed containing one or more aberrations. Tables and glossary, 9 p.
■ *API Medical Research Publication* (June 1985) (23 p.).
Source: API HESD Information Specialist

32-32409

Acute In-Vivo Cytogenetics Assay in Male and Female Rats of API 83-19. Final Report. An acute cytogenetics assay conducted by Microbiological Associates Inc. on 13 groups of five male and five female Sprague-Dawley rats, which were sacrificed 6, 24, or 48 hr after receiving a single intraperitoneal (IP) injection of 0.3, 1.0, or 3.0 g/kg of API 83-19, a 90-160 °C light alkylate petroleum naphtha containing mostly C_7-C_{10} branched alkanes showed that API 83-19 did not induce chromosome aberrations in bone marrow cells. The incidence of cells with aberrations and the number of aberrations per cell were not significantly increased relative to the vehicle controls. There was no apparent effect in ploidy or on the mitotic index. Clinical observations among the high-dose rats included the death of four females and five males, weight loss, and crusty eyes and noses. A single IP injection of 0.5 mg/kg of triethylenemelamine given to positive control rats induced an average of 2.336 aberrations per cell, with 34.2% of all cells analyzed containing one or more aberrations. Tables and glossary, 9 p.

■ *API Medical Research Publication* (June 1985) (23 p.).

Source: API HESD Information Specialist

32-32459

Mutagenicity Evaluation of Catalytic Reformed Naphtha, API #83-05, in the Mouse Lymphoma Forward Mutation Assay. Final Report. In this assay, in vitro treatments of the L5178Y cell line with API 83-05 induced significant increases in the mutant frequency at the thymidine kinase locus only in the presence of rat liver S9 metabolic activation, and was evaluated as active. Under nonactivation conditions, the cells were exposed for four hours to 6.25-100 nL/mL concentrations of #83-05, and low to moderate toxicities were induced without inducing significant, dose-dependent increases above the background mutation frequency. Treatment with 150 nL/mL was excessively toxic. Treatments of 18.8-200 nL/mL induced mutant frequencies 2.1-fold to 4.2-fold above the background frequency. API # 83-05 boils at ~ 30-220 °C, and consists mostly of C_4-C_{12} hydrocarbons, including a relatively large proportion of aromatic and branched chain types, and possibly 10 vol % or more of benzenes. Diagrams and table, 2 p.

■ *API Health Environ. Safety Dep. Rep.* (July 1985) (24 p.).

Source: API HESD Information Specialist

32-32460

Mutagenicity Evaluation in the Mouse Lymphoma Forward Mutation Assay, API #83-06, Heavy Catalytically Reformed Naphtha. Final Report. In this assay, in vitro treatments of the L5178Y cell line induced significant increases in the mutant frequency at the thymidine kinase locus only in the presence of rat liver S9 metabolic activation, and was evaluated as weakly active. Under nonactivation conditions, the cells were exposed for four hours to 6.25-75 nL/mL of #83-06, and a wide range of toxicities was induced without inducing significant increases above the background mutation frequency. The test material was excessively toxic at 100 nL/mL. In the presence of metabolic activation, treatments of 6.25-75.0 nL/mL showed low to moderate toxicities and mutation frequencies about 1.9-fold to 2.2-fold above the background frequency. An additional activation assay at 10-120 nL/mL confirmed the weak mutagenicity of #83-06. API #83-06 boils at 90-120 °C, and consists mainly of C_7-C_{12} aromatic hydrocarbons. Tables, 2 p.

■ *API Health Environ. Safety Dep. Rep.* (July 1985) (23 p.).

Source: API HESD Information Specialist

32-32461

Response of Specific Airways Resistance (SRaw) to SO_2 in the Exercising Asthmatic: Dose Considerations. An evaluation of published and available data on exercising asthmatics exposed to SO_2 via oral (encumbered) breathing through a mouthpiece confirmed that SRaw values (percent increase or decrease from the preexposure control value) increased progressively as the exposure rate (D min) increased, that SRaw and Dmin were highly correlated, and that SRaw and the total exposure dose correlated very poorly. An evaluation of published data on exercising asthmatics exposed to SO_2 via oronasal (unencumbered) breathing showed the same pattern of results, except that there was a consistently smaller percent SRaw increase per unit Dmin increase than with the encumbered subjects. For both exposure modes, the dose/effect relationship (SRaw vs. Dmin) was consistently exponential in character, but a linear model could also be used to fit the data. For both these models, a matrix has been developed for directly estimating the combined SO_2 and ventilatory volume concentration (ppm or µg/L) required to achieve various levels of airway resistance increase. Graphs, tables, and 12 references, 17 p.

■ A. V. Colucci (Colucci & Assoc. Inc.), *API Health Environ. Safety Dep. Rep.* (Aug. 1982) (29 p.).

Source: API HESD Information Specialist

32-32502

A Unified Approach to the Use of Human Clinical Data: A Case Study of Nitrogen Dioxide and Sulfur Dioxide. An analysis of published data for the airway resistances (Raw, percent) to < 20 to ~ 260 µg/min NO_2 and SO_2 of healthy human subjects at or near rest identified the dose rate (Dmin, µg/min), rather than total inhaled dose or total exposure, as the function which correlated best with changes in Raw for these pollutants. For both compounds, the increases in Raw with increasing Dmin were linear, approaching zero at 12-19 µg/min for NO_2 and at 13-26 µg/min for SO_2. The Dmin vs. percent Raw curves were initially very close, but diverged quite rapidly thereafter. A 10% increase in Raw would be expected at 36 µg/min delivery rate for NO_2 and at 77 µg/min for SO_2; a 25% increase would be expected at corresponding values of 142 and 60 µg/ml. NO_2 was thus about twice as potent as SO_2 for the subjects tested. The curve fitting methods used are discussed. Graphs, tables, and 21 references, 13 p.

■ A. V. Colucci (Colucci Assoc. Inc.); E. J. Faeder (South. Calif Edison Co.); P. E. Brubaker (Exxon Corp., Res. Environ. Health Div.); R. P. Strieter (API Med. Biol. Sci. Dep.), *API Health Environ. Sci. Dep. Rep.* (Apr. 1982) (25 p.).

Source: API HESD Information Specialist

32-32503

A Unified Approach to the Use of Human Clinical and Animal Toxicologic Data; Application to the Establishment of Ambient Air Quality Criteria. Volume IA: Effect of Ozone on Airway Resistance (Raw) and Forced Expiratory Volume ($FEV_{1.0}$) in Humans. Final Report. An analysis of 13 published studies on normal, healthy human males exposed at rest and when exercising to ~ 0.2-20 µg/L of ozone for up to 4 hr showed that exposure rate (Dmin) and total exposure dose (Dt) correlate well with reported increases in Raw, that increasing exercise lessens (attenuates) the increases in Raw induced by an increased exposure rate, and that the extent of the reduction is critically dependent on the exercise level. An observed correlation between Dmin, simple ozone concentration, Dt, and percent increase in Raw indicates that either of these dose expressions is applicable to ozone-induced increases in airway resistance. The lesser levels of exercise that induced a doubling of the resting minute volume (Ve, 10-20 L/min) did not attenuate Raw values, but higher levels (Ve, 50-60 L/min) did. As with Raw, the aEV(1.0) values decreased with increases in ozone effective dose, but the over-all correlations of Dmin, Dt, and ozone concentration with changes in percent FEV(1.0) were lower than the changes observed in percent Raw. Graphs, tables, 23 references, and appendixes, 70 p.

■ A. V. Colucci (Colucci Assoc. Inc.), *API Health Environ. Sci. Dep. Rep.* (May. 1983) (101 p.).

Source: API HESD Information Specialist

32-32565

Feasibility Analysis for an Historical Prospective Mortality Study of Employees Exposed to Downstream Gasoline in the Petroleum Industry. Final Report. An Environmental Health Associates Inc. study, which was based on 26 completed questionnaires from six firms, including Chevron U.S.A. Inc., Mobil Corp., Texaco Inc., and Gulf Oil Corp.; and on-site visits to five of the firms, shows that a cohort of petroleum industry employees exposed to motor and aviation gasolines and middle distillates (jet and diesel fuels, kerosine, and No. 2 fuel oil) during the transportation and distribution of these products can be identified by using employment records. Other aspects discussed include the availability of collectible data at a limited number of centralized record retention locations; of adequate employment records for a mortality study; and of a cohort comprising ~ 57,876 persons with at

least 15 yr of maximum follow-up to provide } 935,387 person-years, which will be able to detect standardized mortality ratios as small as 136 for kidney cancer and 127 for leukemia; and the constraints, that will include limited data on the employees' smoking history, on a mortality study. Tables, appendixes, and 21 references, 53 p.
■ O. Wong; R. W. Morgan (Environ. Health Assoc. Inc.), *API Health Environ. Sci. Dep. Rep.* (4/8/85) (101 p.).
Source: API HESD Information Specialist

32-32651
Impact on Human Health of Petroleum in the Marine Environment. An examination of the literature indicates that human biochemical and physiological responses to acute exposure to natural crudes and their products tend to be transient and short-lived except at very high exposures. There is now no proof that chronic exposure by eating seafood contaminated with PAHs is related to the incidence of cancer and other diseases in humans, except for seafood grown near local petroleum hydrocarbon sources, creosote pilings, and other concentrated sources. The natural intake, depuration, and metabolic processes of marine organisms will cause PAH levels to drop rapidly if the source is removed. Current evidence indicates that a substantial portion of PAHs in seafood come from many sources, often from combustion. Petroleum is not generally considered a major PAH source, except for its presence in seafood harvested near and soon after an oil spill. Current information also suggests that coal- and shale-derived crude oils have higher PAH and nitrogenated PAH contents than natural crudes and refined products. Diagrams, tables, and 259 references, 29 p.
■ I. R. Politzer; I. R. Deleon; J. L. Laseter (Univ. New Orleans), *API Publication* (June 1985) (171 p.).
Source: API HESD Information Specialist

32-32652
Twenty-Eight Day Subchronic Dermal Toxicity Study in Rats: API-HNC-1 (High-Nitrogen, [0.77%], Crude Oil); API-SFP-105 (0.05% N, Hydrotreated Shale Oil); API-SFP-119 (0.19% N, Hydrotreated Shale Oil); API-SFP-206 (0.06% N, Hydrotreated Shale Oil); API-SFR-1 ([1.6% N], Raw Shale Oil); API-SFR-2 ([2.1% N], Raw Shale Oil). Male and female Sprague-Dawley rats were exposed dermally to 0.25 or 2.5 g/kg doses of the test articles five times per week for four weeks. HNC-1, SFP-105, and SFP-119 caused no detectable dermal responses. SFP-206 caused no detectable dermal response at the low dose, but fissuring was noted in two high-dose males on dose days 9 and 10-12. Females were not remarkable. SFR-1 caused no dermal response at the low dose. High-dose females were not remarkable, but the males showed erythema, desquamation, edema, and fissuring. The skin apparently adjusted, and the rats were not remarkable by dose day 19. Low doses of SFR-2 caused no detectable dermal response. High-dose males, but not females, showed signs of erythema, edema, desquamation, and fissuring. Skin responses were not remarkable by dose day 19. No animals died during the study, but there were scattered occurrences of chromodacryorrhea and other overt signs of toxicity. Body weights and changes and food consumption were measured, and blood samples obtained at the study's end were used for hematology and clinical chemistry evaluations. Chart, tables, and appendixes, 304 p.
■ *API Health Environ. Sci. Dep. Rep.* (Aug. 1985) (322 p.).
Source: API HESD Information Specialist

32-32653
Lifetime Dermal Carcinogenesis/Chronic Toxicity Screening Bioassay of Refinery Streams in C3H/HeJ Mice. Three-Month Toxicity Evaluation Report. [Vol. 1 and 2]. Measurement of body weight changes, clinical observations, and tumor incidence data during the application twice a week of 12 neat liquid and xylene-diluted semisolid petroleum fractions to the skin of ~ 800 mice, and gross pathological and histopathologic examinations on sacrificed animals after three months of the twelve month study showed that only API 81-15, at 10% in xylene, was carcinogenic. Squamous cell tumors were diagnosed at the painting site. All the test materials were locally dermatotoxic. No other test material-related carcinogenic or systemic toxic responses were apparent. API 81-15 was a C_{20} and higher catalytically cracked, clarified oil boiling at > 350 °C, and was likely to contain 5 wt% or more of 4- to 6-membered condensed ring aromatic hydrocarbons; it had a low content of asphaltenes and sulfur. The other samples included a PS-6 motor fuel (81-24), three #2 type fuel oils (83-01, 83-02, 83-03), two vacuum residua (81-13 and 81-14), two hydrodesulfurized middle distillates (81-09 and 81-10), a sweetened naphtha (81-08), a hydrodesulfurized kerosine (81-07), and a light catalytic cracked naphtha (81-03). Vol. 1, tables and appendix, 232 p; Vol. 2, all appendix, 320 p.
■ *API Health Envir. Sci. Dep. Rep.* (Sept. 1985) (580 p.).
Source: API HESD Information Specialist

32-32656
Cancerous Diseases in Aquatic Animals and Their Association with Environmental Pollutants: A Critical Review of the Literature in English concerning feral shellfish and fish from the west, east, and Gulf coasts of the U.S., the continental U.S., European countries, and other world areas showed that a final conclusion regarding the role of environmental pollutants in causing fish neoplasms (cancers) is not yet possible. Although there is no direct proof that a chemical agent can cause neoplasms in fish, a pollution etiology cannot be ruled out. As for shell-fish, existing studies of clams, oysters, and mussels have yielded no impressive evidence to support the pollution-neoplasm hypothesis. Several carefully designed and conducted studies providing an abundance of data on shellfish at U.S. and Canadian locations suggest that PAHs and/or petroleum do not directly cause neoplasms in feral populations of bivalve mollusks, and that PAHs do not cause neoplasms in clams. Further studies are recommended, especially on the possible link between viruses and neoplasms in fish and shellfish. Chart, tables, and 413 references.
■ M. C. Mix (Oreg. State Univ.), *API Publication* (1985) (252 p.).
Source: API Library

32-32731
Sister Chromatid Exchange [(SCE)] Assay in Chinese Hamster Ovary (CHO) Cells [Exposed to] PS-8-D5-Saturates. Final Report. To validate the SCE assay in CHO cells for complex petroleum hydrocarbon mixtures, the mutagenic potential of one such mixture (PS-8-D5-Saturates, no composition data) was measured by its ability to induce SCEs in CHO cell chromosomes. The assay was conducted both in the presence and absence of a metabolic activation system, Aroclor-induced rat liver S-9 homogenate, with triethylenemelamine (0.025 µg/mL) and cyclophosphamide (2.5 µg/mL) used as the respective positive controls. Relative to the controls, PS-8-D5 was nontoxic in all the concentrations tested (0.6, 1.3, 2.5, 5.0, and 10 mg/mL in DMSO), but it did induce statistically significant increases in SCE frequencies Doth in the presence of S-9 (at PS-8-D5 doses ≥ 1.3 mg/L) and in its absence (at ≥ 2.5 mg/L). Tables and appendixes, 34 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1985) (48 p.).
Source: API HESD Information Specialist

32-32743
Thirteen Week Dermal Toxicity Study of a Petroleum-Derived Hydrocarbon in Rats: (API 81-15) Catalytically Cracked Clarified Oil (CAS 64741-62-4). Final Report. API 81-15 (consisting mainly of C_{20} and higher hydrocarbons and likely to contain 5 wt % or more of 4- to 6-membered condensed ring hydrocarbons) was applied at 0 (group 1), 40 (group 2), 200 (group 3), and 400 mg/kg (group 4) levels to the skins of groups of 10 male plus 10 female Fischer 344 rats 5 times a week for up to 12 weeks. There was no evidence of edema in the treated groups and no erythema or edema in group 1 controls during the twelve weeks. Erythema could not be scored for the treated rats because the test material stained the skin. Because of mortality and the moribund condition of many test animals, the study was ended at twelve weeks for groups 2 and 3 and at 4 weeks for group 4. One male and 2 females died and 1 male and 3 females were sacrificed in extremis in group 3. Nine males and 10 females died, and 1 male was sacrificed in extremis in group 4. These results for groups 3 and 4 are considered treatment-related. Statistically significant, treatment-related changes in body weight, in hematology and clinical chemistry values, and in histopathology results for body tissues were observed for many of the animals. Tables, appendix, and addenda, 161 p.

■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1985) (187 p.).
Source: API HESD Information Specialist

32-32744
CHO [(Chinese Hamster Ovary)] /HGPRT [(Hypoxanthine-Guanine Phosphoribosyl Transferase)] Mammalian Cell Forward Gene Mutation Assay of API [Sample] RO-1 Retorted Shale Oil. Final Report. In preliminary cytotoxicity screening at API RO-1 doses of 0.3-2000 µg/mL in dimethyl sulfoxide (DMSO), the shale oil showed total cytotoxicity at ≥ 250 mg/mL without metabolic activation and at ≥ 1000 µg/mL with activation by Aroclor-induced rat liver S9 homogenate. The 100 µg/mL dose produced a 13.1% relative cell survival (RCS) without S9; 100 and 500 µg/mL produced 17.7 and 3.3.% RCS, respectively, with S9. Based on these findings, RO-1 was evaluated in duplicate cultures in the CHO/HGPRT mutation assay at 1, 3, 10, 30, and 100 µg/mL in DMSO without S9 and at 1, 3, 10, 30, 100, and 300 µg/mL with S9. The mutation frequencies observed, up to 4.7 fold without S9 and 16.78 fold with S9 over solvent controls, did not fulfill the criteria of a positive assay according to the test protocol. The biological significance of this response is uncertain, and the assay results are equivocal. Tables and appendixes, 41 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1985) (55 p.).
Source: API HESD Information Specialist

32-32745
L5178Y TK+/- Mouse Lymphoma Mutagenesis Assay. API [Sample] 83-09, Straight-Run Kerosine (CAS 8008-20-6). Final Report. Three valid LL5178Y TK+/- Mouse Lymphoma Mutagenesis assays were carried out with API 83-09, a 150-290 °C bp kerosine containing mainly C_9-C_{16} hydrocarbons (82% saturates, 15.5% aromatics, 2.5% olefins), in the presence or absence of an Aroclor-induced rat liver S9 exogenous metabolic activation system. In the first, nonactivated assay, two cultures that were cloned showed mutant frequencies (MF) which were more than twice the mean MF of the solvent controls. The Total Growth (TG) of these cultures was 1 and 15%. In the second assay, five nonactivated and one S9-activated cultures showed more than double mean MFs of the solvent controls, with 0-3 and 11% TG, respectively. In the third, nonactivated assay, one culture, having a 30% TG, showed a MF which was significantly greater than the mean MF of the solvent controls. Thus, under the test conditions, API 83-09 produced a positive response in the absence of S9 and an equivocal response in its presence. Tables, graphs, and appendixes, 34 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1985) (55 p.).
Source: API HESD Information Specialist

32-32746
L5178Y TK+/- Mouse Lymphoma Mutagenesis Assay of API [Sample] 83-19, Light Alkylate Naphtha. Final Report. Six L5178Y TK+/- Mouse Lymphoma Mutagenesis assays, in the presence and absence of an Aroclor-induced rat liver S9 metabolic activation system, were carried out with API 83-19, a 90-160 °C naphtha, produced via isobutane alkylation of C_3-C_5 monoolefins and containing mainly C_7-C_{10} branched hydrocarbons (99.5% saturates, 0.4% naphthenes). Due to technical problems, only two assays gave valid results. In these assays, none of the cultures that were cloned, whether treated in the absence or presence of S9, showed mutant frequencies (MF), which were significantly greater than the mean MF of the solvent control cultures. Both nonactivated and S9-activated cultures were generated in the 10-50% Total Growth range. Thus, under the test conditions, API 83-19 produced a negative response both in the presence and absence of exogeneous metabolic activation. Tables, graphs, appendixes, 41 p.
■ *API Health Environ. Sci. Dep. Rep.* (Aug. 1985) (64 p.).
Source: API HESD Information Specialist

32-32747
28-Day Dermal Toxicity Study in the Rabbit: API 83-11, Straight--Run Middle Distillate (CAS 64741-44-2). Final Report. Application thrice a week for 28 days of 200, 1000, and 2000 mg/kg of API 83-11, boiling at 205-345 °C and consisting of mainly $_{11}$-C_{29} hydrocarbons, to the shaved skins of groups of 5 male and 5 female rabbits caused erythema and edema, and cracked, flaky, and/or leathery skin at the test site on all treated groups. Minimal inflammatory changes were also present in the treated skins. These skin changes were usually accompanied by an increased granulopoeisis of the bone marrow. Moderate skin irritation appeared in 2000 mg/kg dose-level males and females, slight to moderate irritation in 1000 mg/kg males and females, and minimal irritation in 200 mg/kg males and females. No other treatment-related clinical signs were noted. Tables, addenda, and appendixes, 90 p.
■ *API Health Environ. Sci. Dep. Rep.* (Nov. 1985) (113 p.).
Source: API HESD Information Specialist

32-32748
A 28-Day Dermal Toxicity Study in the Rabbit. API 83-18, Heavy, Catalytically Cracked Naphtha (CAS 64741-54-4). Final Report. Application thrice a week for 28 days of 200, 1000, and 200 mg/kg of API 83-18, boiling at ~ 65-230 °C and consisting mainly of C_6-C_{12} hydrocarbons, to the skins of three groups of 5 male plus 5 female rabbits showed erythema, edema, and cracked, flaky, and/or leathery skin. Moderate skin irritation was observed for males and females in the high- and mid-dose groups. Low-dose males showed slight to moderate dermal irritation to 83-15, and low-dose females showed only slight irritation. The treatment-related slight to moderate proliferative changes, and slight to moderate inflammatory changes in the skin of high-dose males and females were accompanied by an increased granulopoiesis of the bone marrow. Treatment with 83-15 at the high dose level did not appear to have any direct effect, vs. the control group, on the other tissues evaluated from these rabbits. Tables, appendixes, and addenda, 94 p.
■ *API Health Environ. Sci. Dep. Rep.* (Nov. 1985) (118 p.).
Source: API HESD Information Specialist

32-32749
The Absorption of Petroleum Products across the Skin of Monkey and Man: Annual Report, Jan. 1, 1984 to Dec. 31, 1984. The absorption of carbon-14-labeled benzene and isooctane across the skins of two adult male and two adult female rhesus monkeys and of adult male humans was followed in vivo by measuring the urinary excretion of radioactivity after the exposure and was determined in vitro on human autopsy skin. Adsorption of pure benzene through the human palm and forearm was 0.003 and 0.002 µL/sq cm, respectively, after 10 and 20 sec exposures. Absorption through the human palm was 0.035 and 0.063 µL/sq cm for a 1 and a 3 min exposure, respectively. Monkey in vivo and human in vitro experiments showed that skin exposure to pure benzene or gasoline does not seem to lead to barrier damage and to enhanced benzene absorption. A 30 min exposure to benzene vapor led to the adsorption of 0.15 µL/sq cm in monkey skin. After the intravenous administration of isooctane to the monkey, 43% of the dose was excreted in the urine over three weeks. Topical application of isooctane to the monkey in vivo and to human autopsy skin in vitro caused respective adsorptions of 0.003 and 0.006 µL/sq cm. Tables.
■ T. J. Franz (Univ. Wash. Sch. Med.), *API Health Environ. Sci. Dep. Rep.* (1985) (18 p.).
Source: API HESD Information Specialist

32-32750
Sister Chromatid Exchange [(SCE)] Assay in Chinese Hamster Ovary (CHO) Cells. API 81-15, Catalytically Cracked, Clarified Oil (CAS 64741-62-4). Final Report. In vitro SCE assays showed that, in the presence of rat liver S-9-induced activation, 50, 100, 500, 1000, and 5000 µg/mL doses of API 81-15 significantly increased the frequency of SCEs in CHO cells over the solvent control. Tests on the nonactivated system showed that 5.0, 10, 50, 100, and 500 µg/mL of API 81-15 marginally increased the frequency of SCEs in the CHO cells over the solvent control, but the results were not statistically significant at any of the dose levels. The positive controls, triethylenemelamine or cyclophosphamide, produced significant numbers of SCEs both with and without activation. API 81-15 contains mainly > C_{20} hydrocarbons, is likely to contain 5 wt % or more of 4- to 6-membered condensed ring aromatic hydrocarbons, and boils at ≥ 350 °C. Tables and appendixes, 39 p.
■ *API Health Environ. Sci. Dep. Rep.* (Nov. 1985) (54 p.).
Source: API HESD Information Specialist

32-32751

A 28-Day Dermal Toxicity Study in the Rabbit of API [Sample] 83-07, a Light Catalytically Cracked Distillate (CAS 64741-59-9). Final Report. API 38-07 was applied at 250, 500, or 1000 mg/kg body weight to the shaved intact skin of three respective groups (5 males, 5 females each) of New Zealand White rabbits three times a week for a total of 12 applications over 28 days. No treatment-related clinical signs, other than skin irritation, were observed in 27 sample-treated rabbits that survived until study termination. The three deaths (one high-dose and two low-dose animals) were considered incidental to treatment. The irritation, observed in all sample-treated animals, consisted of erythema and edema, as well as cracked, flaky, and/or leathery skin at or around the test site, and was accompanied by an increased granulopoiesis of the bone marrow, which may be related to the stress or other factors associated with the severe skin irritation. The 1000 mg/kg dose was severely irritating to male and female rabbits; 500 mg/kg was severe in females but moderate in males, and 250 mg/kg was moderately irritating in all rabbits. API 83-07 consists mainly of C_9-C_{25} hydrocarbons, contains a relatively large amount of bicyclic aromatic hydrocarbons, and boils at ~ 150-400 °C. Tables and appendixes, 89 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1985) (113 p.).
Source: API HESD Information Specialist

32-32752

A 28-Day Dermal Toxicity Study in the Rabbit. API [Sample] 83-06, Heavy Catalytically Reformed Naphtha (CAS 64741-68-0). Final Report. API 83-06 was applied at 200, 1000, or 2000 mg/kg body weight to the shaved intact skin of three respective groups (5 males, 5 females each) of New Zealand white rabbits three times a week for a total of 12 applications over 28 days. No deaths occurred during the study, and no treatment-related findings, other than dermal irritation in all sample-treated animals, were observed. The irritation consisted of erythema and edema, as well as cracked, flaky, and/or leathery skin at or around the test site, and was accompanied by an increased granulopoiesis of the bone marrow. Treatment with API 83-06 did not appear to have any direct effect on the other selected nondermal tissues examined. The 2000 and 1000 mg/kg doses were severely irritating in both male and female rabbits; the 200 mg/kg dose was moderately irritating in males and slightly to moderately irritating in females. API 83-06 consists mainly of C_7-C_{12} aromatic hydrocarbons and boils at ~ 90-230 °C. Tables and appendixes, 87 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1985) (111 p.).
Source: API HESD Information Specialist

32-32753

A 28-Day Dermal Toxicity Study in the Rabbit. API [Sample] 83-08, Light Catalytically Cracked Distillate (CAS 64741-59-9). API 83-08 was applied at 200, 1000, or 2000 mg/kg body weight to the shaved intact skin of three respective groups (5 males, 5 females each) of New Zealand white rabbits three times a week for a total of 12 applications over 28 days. Two high-dose (2000 mg/kg) females were sacrificed *in extremis* during the first 2 wk of the study; the condition of one of these animals, including thin appearance, prostration, labored breathing, and loss of righting reflex, was attributed to API 83-08. Treatment-related thinness was also noted in three other high-dose rabbits (two males and one female). Other treatment-related clinical signs included a significant adverse effect on body weight gain of the 1000 and 2000 mg/kg doses and dermal irritation in all three dose groups. API 83-08 was a severe skin irritant at 1000 and 2000 mg/kg and a slight to moderate irritant at 200 mg/kg in both male and female rabbits. No treatment-related findings were evident in the hematology or the clinical chemistry data. API 83-08 consists mainly of C_9-C_{25} hydrocarbons, has a large portion of bicyclic aromatic hydrocarbons, and boils at ~ 150-400 °C. Tables and appendixes, 91 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1985).
Source: API HESD Information Specialist

32-32754

In Vivo Sister Chromatid Exchange [(SCE)] Assay, API 81-15, Catalytically Cracked, Clarified Oil (CAS 64741-62-4). Final Report. Five experimental groups of male and female B6C3F1 mice were exposed to 0.4, 2.0, and 4.0 gm/kg of API 81-15 in corn oil via a single intraperitoneal injection 4 hr after subcutaneous implantation of a 50 mg BRdU pellet; the bone marrow cells were arrested in metaphase with colchicine, collected 24-26 hr after pellet implantation, and examined microscopically. The negative control substance was corn oil and the positive control substance was cyclophosphamide. Under the test conditions, 81-15 induced a statistically significant and dose-responsive increase in mouse bone marrow SCEs. API 81-15 consists mainly of > C_{20} hydrocarbons, is likely to contain 5 wt % or more of 4- and 6-membered condensed ring hydrocarbons, and boils at above ~ 350 °C. Tables, 3 p.
■ *API Health Environ. Sci. Dep. Rep.* (Nov. 1985) (17 p.).
Source: API HESD Information Specialist

32-32755

Short-Term Responses of Healthy and Asthmatic Men to Ozone. Final Report, Phase 1: A Dose-Response Study of Healthy, Heavily Exercising Men Exposed to Ozone Concentrations near the Primary [National Ambient Air Quality] Standard [(NAAQS)]. A group of 24 well-conditioned young adult male volunteers, free of asthma or clinical respiratory allergies, were exposed to purified air containing 0.08-0.16 ppm ozone. Exposures were separated by 2 wk intervals, occurred in random order, and lasted 2 hr each. Subjects exercised heavily on bicycle ergometers, attaining ventilation rates of 68 L/min avg. Lung function was measured before and 1 and 2 hr after exposure, and airway responsiveness to a cold-air challenge was measured immediately after 2 hr exposures. Symptoms were recorded at 15 min intervals during exposure and for one-week periods following exposures. For the group as a whole, no significant harmful effects were observed, except for a slight respiratory irritant response after > 1 hr exposure to 0.16 ppm ozone. Two subjects showed possible responses at 0.14 ppm, end one of them also at 0.12 ppm ozone. The study does not confirm the previous finding [*J. Appl. Physiol. 54*:1345-52 (1983)] of significant irritant responses to ozone at the NAAQS concentration of 0.12 ppm. Tables, graph, 26 references, and appendixes, 16 p.
■ *API Health Environ. Sci. Dep. Rep.* 1 (Oct. 1985) (52 p.).
Source: API HESD Information Specialist

32-32854

Two-Generation Inhalation Reproduction/Fertility Study on a Petroleum-Derived Hydrocarbon, [i.e.,] Toluene [Vol. 1]. Toluene was administered by whole body inhalation exposure at 100, 500, or 200 ppm in air for parent Charles River CD rats of both sexes and at 2000 ppm for males and females bred with untreated counterparts in each of two consecutive generations. Each generation was mated once. A teratologic evaluation was conducted on selected F_1-generation fetuses. Treated adults of both generation were exposed for ~ 6 hr/day, 7 days/wk during an 80 day premating period and a 15 day mating period. Treated adult females were also exposed on days 1 through 20 of gestation and days 5 through 21 of lactation. Exposure at 2000 ppm, especially of female parents, caused a significant inhibition of the growth of the offspring over both generations, but exposure at 2000 ppm of the male parents alone or exposures at ≤ 500 ppm produced no consistent effect. There were no toluene-related effects on parental survival, appearance or behavior, fertility indexes, copulatory duration, gestation length, mean number of stillborn pups, or pup survival through lactation. Tables. Vol. 2 and Vol. 3 of this report consist entirely of appendixes.
■ *API Health Environ. Sci. Dep. Rep.* (July 1985) (20 p.).
Source: API HESD Information Specialist

32-32855

Evaluation of Micronuclei [(MN)] Frequency in the Peripheral Blood of Male and Female CD-1 Mice Exposed Chemically to Benzene for 90 Days. Final Report. A Brookhaven National Laboratory study on groups of 10 male and 10 female CD-1 mice exposed to 0, 1, 10, 30, and 300 ppm benzene for 6 hr/day, 5 days/wk, has shown significant increases in micronucleated polychromatic erythrocytes (mPCEs) and micronucleated normochromatic erythrocytes (mNCEs) in male and female mice at 300 ppm at all sample times, with the males showing greater MN induction than the females. Sample time did not significantly affect the frequency of mPCEs among the high-dose males and females,

as it did the rate of mNCEs, thus indicating that the ability of benzene to induce bone marrow genotoxic damage did not change with increasing exposure duration. Steady state conditions for MN induction in males and females were achieved by the 30-day sample time. Other results include a significant increase in micronucleated erythrocytes in female mice at 30 ppm at some sample times, but no such finding at 1 or 10 ppm at any sample time. Tables, photomicrograph, and 21 references, 76 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1985) (87 p.).
Source: API HESD Information Specialist

32-32856
Adapting the Ames Salmonella Assay to Complex Hydrocarbon Mixtures. Final Report. A study by Microbiological Associates Inc. has shown that its modifications to the standard Ames Salmonella assay can demonstrate the mutagenicity of some petroleum-derived hydrocarbon mixtures by increasing both the S-9 homogenate and reduced nicotinamide adenine dinucleotide (NADP) concentrations. Tests on five of the six hydrocarbon mixtures on the strain TA98 at 1000, 5000, 25,000, and 50,000 µg/plate and at 10, 100, 500, and 1000 µg/plate for the RO-1 mixture, with 10, 30, 60, 70, and 80% rat liver S-9 at different concentrations of NADP has shown a mutagenic potency rank of RO-1 > PS8-C5 = PS8-D5 = PS8-D4 > PS8-C3 (questionable), PS8-80C2 (negative). The use of hamster liver S-9 gave essentially the same results as when rat liver S-9 was used, except that the revertant count of the RO-1 samples with rat S-9 was about twice that with hamster S-9. The optimal concentrations for maximum revertant recovery were 80% h-9 homogenate and threefold and twofold NADP concentrations for rat and hamster liver S-9, respectively. Tables and charts, 31 p.
■ *API Health Environ. Sci. Dep. Rep.* (Sept. 1985) (48 p.).
Source: API HESD Information Specialist

32-32857
Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-11, Straight-Run Middle Distillate (CAS 64741-44-2). API 83-11, consisting mainly of C_{11}-C_{20} hydrocarbons and boiling at 205-345 °C had an estimated oral LD_{50} value > 5 g/kg body weight for both sexes when administered undiluted by oral gavage to male and female rats at a single dosage of 5 g/kg. The dermal LD_{50} value for 83-11 was > 2.0 g/kg of body weight for male and female rabbits when applied undiluted at a single dosage of 2.0 g/kg to shaved abraded and shaved nonabraded skin patches. The primary dermal irritation index was 3.2 for rabbits when applied at 0.5 mL per area to clipped abraded and clipped nonabraded skin patches. The primary eye irritation scores for rabbits were 3.3, 0.0, 0.0, 0.0, and 0.0, 1 hr, 24 hr, 48 hr, 72 hr, and 7 days, respectively, after exposure to one 0.1 mL dose of undiluted 83-11 in eyes that were then immediately flushed with water. The corresponding scores for unflushed eyes were 3.7, 1.0, 0.0, 0.0, and 0.0. API 83-11 was not a skin sensitizer in guinea pigs tested by the closed patch technique. Tables and appendix, 13 p.
■ *API Health Environ. Sci. Dep. Rep.* (Dec. 1985) (42 p.).
Source: API HESD Information Specialist

32-32858
Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-09, Straight Run Kerosine (CAS 8008-20-6). API 83-09, consisting mainly of C_9-C_{16} hydrocarbons and boiling at ~ 150-290 °C, had an estimated oral LD_{50} value > 5.0 g/kg body weight for both sexes when given by oral gavage to male and female rats at a single dosage of 5.0 g/kg. The dermal LD_{50} value for 83-09 was > 2.0 g/kg of body weight for male and female rabbits when applied at a single dose of 2.0 g/kg to shaved abraded and shaved nonabraded skin patches. The primary dermal irritation index was 5.5 for rabbits when applied undiluted at 0.5 mL per area to clipped abraded and clipped nonabraded skin patches. The primary eye irritation scores for rabbits were 2.0, 0.0, 0.0, and 0.0, and 0.0 1 hr, 24 hr, 48 hr, 72 hr, and f days, respectively, after exposure to one 0.1 mL dose of undiluted 83-09 in eyes that were then immediately flushed with water. The corresponding scores for unflushed eyes were 0.7, 0.0, 0.0, 0.0, and 0.0. API 83-09 was not a skin sensitizer in guinea pigs tested by the closed patch technique. Tables and appendix, 13 p.
■ *API Health Environ. Sci. Dep. Rep.* (Dec. 1985) (43 p.).
Source: API HESD Information Specialist

32-32859
Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-08, Light Catalytically Reformed Distillate (CAS 64741-59-9). API 83-08, consisting mainly of C_9-C_{25} hydrocarbons and boiling at ~ 150-400 °C, had an estimated LD_{50} value > 7.18 g/kg body weight for male, and > 6.79 g/kg for female, rats when given undiluted by oral gavage. The dermal LD_{50} value for 83-08 was > 2.0 g/kg for male and female rabbits when applied undiluted at a single dose of 2.0 g/kg to shaved abraded and shaved unabraded skin patches. The primary dermal irritation index was 6.9 for rabbits when applied undiluted at 0.5 mL per area to clipped abraded and clipped unabraded skin patches. The primary eye irritation scores were 4.7, 0.0, 0.0, 0.0, and 0.0 1 hr, 24 hr, 48 hr, 72 hr, and 7 days, respectively, after exposure to one 0.1 mL dose of undiluted 83-08 in eyes that were then immediately flushed with water. The corresponding scores for unflushed eyes were 4.3, 3.2, 2.2, 1.2, and 0.0. API 83-08 was not a skin sensitizer in guinea pigs when tested by the closed patch technique. Tables and appendix, 16 p.
■ *API Health Environ. Sci. Dep. Rep.* (Dec. 1985) (49 p.).
Source: API HESD Information Specialist

32-32860
Acute Oral Toxicity Study in Rats. Acute Dermal Toxicity Study in Rabbits. Primary Dermal Irritation Study in Rabbits. Primary Eye Irritation Study in Rabbits. Dermal Sensitization Study in Guinea Pigs. API 83-06, Heavy Catalytically Reformed Naphtha (CAS 64741-68-0). API 83-06, consisting mainly of C_7-C_{12} hydrocarbons and boiling at ~ 90-230 °C, had an estimated oral LD_{50} value of 4.82 g/kg of body weight for male, and 5.80 g/kg for female, rats when administered undiluted by gavage. The dermal LD_{50} value for 83-06 was > 6.0 g/kg of body weight for male and female rabbits when tested at 2.0, 4.0, or 6.0 g/kg on shaved abraded and shaved unabraded skin patches. The primary dermal irritation index was 5.4 for rabbits when applied undiluted at 0.5 mL per area to clipped abraded and clipped nonabraded skin patches. The primary eye irritation scores for rabbits were 4.7, 1.3, 0.0, 0.0, and 0.0 1 hr, 24 hr, 48 hr, 72 hr, and 7 days, respectively, after exposure to one 0.1 mL dose of undiluted 83-06 in eyes that were then immediately flushed with water. The corresponding scores for unflushed eyes were 4.3, 0.7, 0.3, 0.0, and 0.0. API 83-06 was not a skin sensitizer in guinea pigs tested by the closed patch technique. Tables and appendix, 12 p.
■ *API Health Environ. Sci. Dep. Rep.* (Dec. 1985) (49 p.).
Source: API HESD Information Specialist

33-30051
Job code classification system--2. Production operations and marketing/transportation operations have been added to the original job code classification system for petroleum refineries and petrochemical operations. The purpose is to provide a standardized method for identifying and classifying jobs for use in inter- and intracompany occupational health studies, including morbidity and mortality studies, qualitative and quantitative exposure evaluations, and medical surveillance scheduling and evaluations. A 44 p. appendix includes standardized task and process lists and an index for the latter. Also see Abstract No. 27-32280.
■ *API Publication* (Sept. 1985) (59 p.).
Source: API Publications Order Desk (Order No. I80012)

33-30162
Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-07, light catalytically cracked distillate (CAS 64741-59-9). Studies by Hazleton Laboratories America Inc. on API 83-07, which consists mainly of ~ 150-400 °C C_{9-25} hydrocarbons with a relatively large portion of bicyclic aromatic hydrocarbons, showed LD_{50} values of

} 4.66 and ~ 3.20 g/kg body weight for male and female Sprague--Dawley albino rats, respectively, when given undiluted by oral gavage. The dermal LD_{50} value for male and female New Zealand White rabbits was > 2.0 g/kg of body weight when applied undiluted at a single dose of 2.0 g/kg to shaved, abraded skin patches. The primary dermal irritation index was 5.6 when applied undiluted at 0.5 mL/area to clipped abraded or unabraded skin patches. The primary eye irritation scores for unwashed (and washed) eyes after exposure to 0.1 mL 83-07 were 4.3 (2.7), 1.7 (2.0), 1.0 (0.7), 0.0 (0.0), and 0.0 (0.0) at 1, 24, 48, and 72 hr, and 7 days, respectively. API 83-07 was not a skin sensitizer in Hartley male albino guinea pigs when tested by the closed patch technique. Tables and appendix, 12 p.
■ *API Health Environ. Sci. Dep. Rep.* (Dec. 1985) (49 p.).
Source: API HESD Information Specialist

33-30442
Four-week dermal range-finding toxicity study in rats: API 81-15, catalytically cracked clarified oil (CAS 64741-62-4). Final report. API 81-15, a dark brown liquid boiling at > 350 °C and consisting mainly of > C_{20} hydrocarbons, was applied at 0 (group 1), 400 (group 2), 2000 (group 3), 4000 (group 4), and 1000 (group 5) mg/kg/day five times a week for 4 wk on the clipped intact skin of groups of five male and five female Fischer rats to set the dosages for a 13 wk dermal toxicity study. Treatment-related findings include deaths of a male rat in group 2, of four females in group 5, of two males in group 3, and of two males and three females in group 4; no evidence of edema in all groups; statistically significantly lower mean body weights at day 8 through termination for groups 2 through 4 compared with group 1, while group 5 rats showed similar results from day 15 to termination; statistically significant increases in the relative liver weights of group 2 males and females and group 5 males; and minimal-to-slight epidermal hyperplasia and hyperkeratosis in group 2 rats. Tables, appendixes, and addendum, 36 p. See also Abstract No. 32-32743.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (54 p.).
Source: API HESD Information Specialist

33-30443
28-day dermal toxicity study in the rabbit: API 83-09 straight run kerosine (CAS 8008-20-6). Final report. A Tegeris Laboratories Inc. study on New Zealand White male and female rabbits, which were dermally treated three times a week for 28 days with 200, 1000, or 2000 mg/kg of API 83-09, which consists mainly of C_9-C_{16} hydrocarbons boiling at ~ 150-290 °C, showed that API 83-09 is a minimal-to-slight irritant at 200 mg/kg, and a moderate irritant at 1000 and 2000 mg/kg. Compared with findings from the untreated control group, those that were treatment-related include the death of a male and a female at 2000 mg/kg; erythema and edema; cracked, flaky, and/or leathery skin; and statistically significantly higher mean relative heart weights at 1000 and 2000 mg/kg. Statistically significant lower-than-control mean body weights and mean body weight gains were observed for the highest dose animals. At 2000 mg/kg, the increased granulopoiesis of the bone marrow that accompanied inflammatory dermal changes may be related to stress or other factors; and the multifocal seminiferous tubular hypoplasia of the testes, and the granulomatous or pyogranulomatous liver lesions were considered secondary and incidental to the treatment, respectively. Tables, addenda, and appendixes, 59 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (131 p.).
Source: API HESD Information Specialist

33-30444
Acute inhalation toxicity evaluation in rats: API 83-08, light catalytically cracked distillate (CAS 64741-59-9). Final report. In the first phase of a Hazleton Laboratories America Inc. study, a 4 hr exposure by aerosol of five male and five female Charles River Strain rats to 5 mg/L of API 83-08, which consists mainly of C_9-C_{25} hydrocarbons boiling at ~ 150-400 °C, three males and one female died during the 14-day observation period. In the second phase, the exposure of five additional groups of five male and five female rats to 2.34, 3.47, 6.34, and 7.29 mg/L of API 83-08 for 4 hr showed an LC_{50} of 4.65 mg/L for male rats and the combined sexes, with 95% confidence limits of 3.89-5.55 mg/L for the combined sexes. None of the untreated control rats died, but several rats died 1-2 days after exposure to the lower concentrations, and all of those exposed to the highest dose died. Dose-related body weight gain depressions, pharmacotoxic signs, and necropsy and histopathologic findings related to the lung tissues were most severe in the animals that died and were considered to be compatible with hydrocarbon toxicity. Tables and appendixes, 55 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (77 p.).
Source: API HESD Information Specialist

33-30445
Sister chromatid exchange [(SCE)] assay in Chinese hamster ovary (CHO) cells: API RO-1, raw shale oil. Final report. A second SCE assay for mutagenesis by Microbiological Associates Inc., which was based on the results of an initial toxicity test and a first SCE assay, showed positive results for API RO-1. The CHO cell cultures treated with 25-750 and with 5-250 µg/mL of API RO-1 in the presence and absence, respectively, of induced rat liver S-9 activation showed significantly higher SCE frequencies than those treated with the solvent control (acetone). The positive controls, i.e., 0.025 µg/mL of triethylenemelamine in the non-activated system and 2.5 µg/mL of cyclophosphamide in the activated system, produced significant numbers of SCEs. Tables and appendixes, 46 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (51 p.).
Source: API HESD Information Specialist

33-30493
Activity of API 83-08 (light catalytic cracked distillate) in the acute in-vivo cytogenetics assay in male and female rats. Final report. A Microbiological Associates Inc. mutagenic potential study on 13 groups of five male and five female Sprague-Dawley rats, which were given a single intraperitoneal injection of 0.3, 1, or 3 g/kg of API 83-08 (consisting mainly of C_9-C_{25} hydrocarbons boiling at ~ 150-400 °C) in corn oil and which were sacrificed 6, 24, or 48 hr after treatment, showed that API 83-08 does not induce chromosomal aberrations in rat bone marrow cells. The findings include a 9-11% weight loss in the highest-dose rats at the 48 hr sacrifice; no apparent change in ploidy or in the mitotic index; and, compared with negative controls, no significant increase in the incidence of cells with aberrations, or in the number of aberrations per cell regardless of dose or sacrifice time. The positive controls that received a single treatment of 0.5 mg/kg of aqueous triethylenemelamine showed an average of 2.942 aberrations per cell, with 39.6% of all cells analyzed containing one or more aberrations. Tables, 8 p.
■ *API Health Environ. Sci. Dep. Rep.* (May 1985) (23 p.).
Source: API HESD Information Specialist

33-30494
Activity of API 83-06 (heavy catalytic reformed naphtha) in the acute in vivo cytogenetics assay in male and female rats. Final report. A mutagenic potential study by Microbiological Associates Inc. on 13 groups of five male and five female Sprague-Dawley rats, which were injected intraperitoneally once with 0.3, 1, or 3 g/kg of API 83-06 (consisting mainly of C_7-C_{12} hydrocarbons boiling at ~ 90-230 °C) in corn oil and which were sacrificed 6, 24, or 48 hr after treatment, showed that API 83-06 does not induce chromosomal aberrations in rat bone marrow cells. The findings include no apparent change in ploidy, or effect on the mitotic index; no significant increase in the incidence of cells with aberrations, or in the number of aberrations per cell compared with the vehicle controls, despite a statistically significant rise in the highest dose group at 48 hr, since this was within the normal range of spontaneous aberrations, and the vehicle controls for this group had no aberrations; and an average of 3.234 aberrations per cell in positive controls dosed once with 0.5 mg/kg of aqueous triethylenemelamine, with 44% of cells analyzed having one or more aberrations. tables and appendix, 16 p.
■ *API Health Environ. Sci. Dep. Rep.* (May 1985) (32 p.).
Source: API HESD Information Specialist

33-30495
Dermal sensitization study in guinea pigs [by Hazleton Laboratories America Inc.]: API 81-04 light catalytically cracked naphtha (CAS

64741-55-5). **Final report.** A study on API 81-04, consisting mainly of C_4-C_{11} hydrocarbons boiling at ~ -20 to +190 °C, showed that API 81-04 is not a skin sensitizer in male albino guinea pigs tested by the closed patch technique. Two of the 10 animals dosed with 0.4 mL of 75 and 25 vol/vol % mixtures of API 81-04 in paraffin oil (PO) once a week for 3 wk in the sensitizing and challenge phases, respectively, showed slight body weight losses in the first phase and very slight erythema reactions in the latter phase, which did not exceed the reaction of 1 of the 10 similarly challenged naive controls. One of the 10 vehicle controls sensitized and challenged with 0.4 mL of undiluted PO had very slight erythema reactions. All 20 positive controls dosed in the challenge (and sensitizing) phase with 0.4 mL of a 0.1 (or 0.3) wt/vol % suspension of 2,4-dinitrochlorobenzene in acetone (or 80% aqueous ethanol) had very slight-to-moderate irritation in the challenge phase; 10 of the 20 naive positive controls challenged similarly showed very slight erythema reactions. Tables, 4 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (16 p.).
Source: API HESD Information Specialist

33-30496
Dermal sensitization study in guinea pigs [by Hazleton Laboratories America Inc.]: API 83-04 light catalytically reformed naphtha (CAS 64741-63-5). Final report. A test by the closed patch technique on API 83-04, consisting mainly of C_5-C_{11} hydrocarbons boiling at ~ 35-190 °C, showed that API 83-04 is not a skin sensitizer in male albino guinea pigs. Three of the 10 animals dosed once a week for 3 wk with 0.4 mL of 75 and 25 vol/vol % mixtures of API 83-04 in paraffin oil (PO) in the sensitizing and challenge phases, respectively, showed very slight erythema reactions in the challenge phase that did not exceed the highest reaction of 2 of the 10 similarly challenged naive controls. All 10 vehicle controls dosed with 0.4 mL of undiluted PO in both phases showed no dermal reaction in the challenge phase. All 20 positive controls dosed with 0.4 mL of 0.3 and 0.1 wt/vol % suspensions of 2,4-dinitrochlorobenzene in 80% aqueous ethanol and in acetone, respectively, in the sensitizing and challenge phases, respectively, showed very slight-to--moderate irritation in the latter phase. The reaction of 16 animals exceeded that shown by 10 of the 20 similarly challenged naive positive controls. Tables, 4 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (16 p.).
Source: API HESD Information Specialist

33-30497
Dermal sensitization study in guinea pigs [by Hazleton Laboratories America Inc.]: API 83-05 full-range catalytically cracked reformed naphtha (CAS 68955-35-1). Final report. An experimental study on API 83-05, comprising mainly C_4-C_{12} hydrocarbons boiling at ~ 30-220 °C, showed that API 83-05 is not a skin sensitizer to male albino guinea pigs tested by the closed patch technique. All 10 animals dosed with 0.4 mL of 50 and 25 vol/vol % mixtures of API 83-05 in paraffin oil (PO) once a week for 3 wk in the sensitizing and challenge phases, respectively, showed no dermal reactions. Two of the 10 similarly challenged naive controls showed very slight erythema reactions. All 10 vehicle controls dosed with 0.4 mL of undiluted PO in both phases showed no dermal reaction. All 20 positive controls treated with 0.4 mL of 0.3 and 0.1 wt/vol % suspensions of 2,4-dinitrochlorobenzene in 80% aqueous ethanol and in acetone, respectively, in the sensitizing and challenge phases, respectively, had very slight-to-moderate irritation in the challenge phase. The reaction of 16 animals exceeded the highest reaction of 10 of the 20 similarly challenged naive positive controls. Tables, 4 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (16 p.).
Source: API HESD Information Specialist

33-30498
28-day dermal toxicity study in the rabbit: API 83-19, light alkylate naphtha (CAS 64741-66-8). Final report. A Tegeris Laboratories Inc. study on groups of five male and five female New Zealand White rabbits, which were dermally treated with 200, 1000, or 2000 mg/kg of API 83-19 (consisting mainly of C_7-C_{10} hydrocarbons boiling at ~ 90-160 °C) three times a week for 28 days, showed no deaths among the rabbits. Treatment-related findings include erythema and edema in all groups, which increased in frequency and severity with increasing doses; flaking and/or cracked skin, and leathery skin texture; gross pathologic signs (dry, scaly, rough, fissured, reddened, crusted, and/or thickened skin) in the treated skin only; and statistically significantly lower over-all mean body weight gain compared with the untreated negative controls. Stress or other factors may be related to the increased granulopoiesis of the bone marrow that accompanied the slight-to-moderate proliferative and minimal-to-moderately severe inflammatory changes of the skin of the high-dose rats. Tables, appendixes, and addenda, 90 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (113 p.).
Source: API HESD Information Specialist

33-30499
28-day dermal toxicity study in the rabbit: API 83-12 hydrotreated light naphthenic distillate (CAS 64742-53-6). Final report. A Tegeris Laboratories Inc. study on groups of five male and five female New Zealand White rabbits, which were dermally treated three times a week for 28 days with 200, 1000, or 2000 mg/kg of API 83-12 (mainly C_{15}-C_{30} hydrocarbons) showed no deaths and minimal-to-moderate irritations for all groups. Compared with the untreated controls, the treated rabbits showed such treatment-related signs as erythema and flaking skin in all groups; edema in the mid- and high-dose groups; dry, scaly, rough, fissured, crusted, and/or thickened skin in the treated area; statistically significantly lower over-all body weight gains for mid- and high-dose females and high-dose males; and statistically-significant lower absolute left and right testis weights and relative right testis weights, and numerically lower relative left testis weights at 2000 mg/kg. Other findings among the highest dose groups include slight-to-moderate proliferative changes of the skin accompanied by an increased granulopoiesis of the bone marrow; and atrophy of the testes accompanied by aspermatogenesis and atrophy of the accessory sex organs. Tables, appendixes, and addenda, 94 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (118 p.).
Source: API HESD Information Specialist

33-30549
Acute inhalation toxicity evaluation in rats: API 83-07, light catalytically cracked distillate (CAS 64741-59-9). Final report. A Hazleton Laboratories America Inc. study on five groups of five male and five female rats, which were exposed for 4 hr to, 1.56, 2.83, 4.26, and 8.13 mg/L of API 83-07 (consisting mainly of C_9-C_{25} hydrocarbons boiling at ~ 150-400 °C) as an aerosol, showed an LC_{50} of 4.8 mg/L for the combined males and females. The LC_{50} for the males only was 3.35 mg/L, with 95% confidence limits of 2.5-4.5 mg/L. There were no deaths among the air exposed-controls or among those exposed to 1.56 mg/L, but all rats exposed to 8.13 mg/L died 1 to 2 days after exposure. This part of the study followed a first phase, wherein 2 females died after exposure to 5.38 mg/L of API 83-07 for 4 hr. Body weight gain losses, pharmacotoxic signs (e.g., hair coat and skin abnormalities), and gross necropsy findings and histopathologic changes in the lungs were all dose-related and considered to be compatible with acute hydrocarbon toxicity. Tables, graphs, and appendixes, 69 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (82 p.).
Source: API HESD Information Specialist

33-30589
[A literature] review of published odor [threshold values (OTVs)] and taste threshold values [(TTVs)] of [the 276] soluble gasoline components and of methanol, ethanol, isopropanol, tert.-butanol, propanol, and MTBE in this three-task study by TRC Environmental Consultants Inc., showed OTVs for 51 compounds and TTVs for five compounds. Of the 51 published OTVs, 21 met TRC's three criteria for a valid OTV determination procedure, with a maximum 300-fold difference between the lowest and highest concentrations. For the unacceptable OTVs, the maximum difference was 312,500 mg/cu m. Two of the five published TTVs met TRCs two criteria, with ethanol having a detection threshold of 79 mg/L and MTBE having detection and recognition thresholds of 0.7 and 5 mg/L, respectively. The use of even basic criteria could reduce the variability in published OTVs and TTVs 1000-fold in range to establish useful values with an acceptable range. TRCs original 13 and 12 criteria for the acceptability of OTV and TTV

measurement methods, respectively, and a recommended experimental protocol to determine these compounds' OTVs and TTVs are discussed. Tables and 98 references.
■ *API Publication* #4419 (Dec. 1985) (52 p.).
Source: API Publications Order Desk (Order No. I44190)

33-30592
Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-12, hydrotreated light naphthenic distillate (CAS 64742-53-6). Final report. Studies by Hazleton Laboratories America Inc. on API 83-12, which consists mainly of C_{15}-C_{30} hydrocarbons and produces a finished oil with a viscosity of < 100 SUS at 100 °F, showed an oral LD_{50} value of > 5 g/kg of body weight for male and female Sprague--Dawley albino rats when given undiluted once at 5 g/kg. The dermal LD_{50} value for male and female New Zealand White rabbits was > 2 g/kg of body weight when applied undiluted at a single dose of 2 g/kg to intact or abraded skin patches. The primary dermal irritation index for male rabbits was 5.4 when applied undiluted at 0.5 mL/area to abraded or intact skin patches. The primary eye irritation scores for unwashed (and washed) eyes of rabbits after treatment with 0.1 mL were 2.7 (2), 0.3 (0.0), 0.0 (0.0), 0.0 (0.0), and 0.0 (0.0) at 1, 24, 48, and 72 hr, and 7 days, respectively. API 83-12 was not a skin sensitizer in Hartley male albino guinea pigs when tested by the closed patch technique. Tables and appendix, 16 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (38 p.).
Source: API HESD Information Specialist

33-30593
Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-18, heavy catalytically cracked naphtha (CAS 64741-54-4). Final report. Studies by Hazleton Laboratories America Inc. on API 83-18, which consists mainly of C_6-C_{12} hydrocarbons boiling at ~ 65-230 °C, showed an oral LD_{50} value of > 5 g/kg of body weight for male and female Sprague-Dawley albino rats when given undiluted at 5 g/kg. The dermal LD_{50} value for male and female New Zealand White rabbits was > 2 g/kg of body weight when applied undiluted to abraded and intact skin patches at a single dose of 2 g/kg. The primary dermal irritation index for male rabbits was 6.9 for when applied undiluted at 0.5 mL/area to clipped abraded or intact skin patches. The primary eye irritation scores for unwashed (and washed) eyes of rabbits treated with 0.1 mL of API 83-18 were 3.7 (2.7), 0.0 (0.0), 0.0 (0.0), 0.0 (0.0), and 0.0 (0.0) at 1, 24, 48, and 72 hr, and 7 days, respectively. API 83-18 was not a skin sensitizer in Hartley male albino guinea pigs when tested by the closed patch technique. Tables and appendix, 17 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (41 p.).
Source: API HESD Information Specialist

33-30594
Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-19, light alkylate naphtha (CAS 64741-66-8). Final report. In these studies by Hazleton Laboratories America Inc. on API 83-19, which consists mainly of C_7-C_{10} branched alkanes boiling at ~ 90-160 °C, male and female Sprague-Dawley albino rats showed an LD_{50} value of > 7 g/kg of body weight when given a single undiluted dose of 5 or 7 g/kg of API 83-19 by oral gavage. The dermal LD_{50} value was > 2 g/kg of body weight for male and female New Zealand White rabbits when applied undiluted at a single dose of 2 g/kg to clipped abraded or intact skin patches. The primary dermal irritation index was 3.9 when applied undiluted at 0.5 mL/area to male rabbits' clipped abraded or unabraded skin patches. The primary eye irritation scores for rabbits' unwashed (and washed) eyes after treatment with 0.1 mL of API 83-19 were 0.3 (0.7); 0.0 (0.0), 0.0 (0.0), 0.0 (0.0), and 0.0 (0.0) at 1, 24, 48, and 72 hr, and 7 days, respectively. API 83-19 was not a skin sensitizer in Hartley male albino guinea rigs when tested by the closed patch technique. Tables and appendix, 16 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1986) (38 p.).
Source: API HESD Information Specialist

33-30595
Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 84-01, light paraffinic distillate (CAS 64741-50-0). Final report. These Hazleton Laboratories America Inc. studies on API 84-01, which consists mainly of C_{15}-C_{30} hydrocarbons and which produces a finished oil with a viscosity of < 100 SUS at 100 °F, showed an oral LD_{50} value of > 5 g/kg of body weight for male and female Sprague-Dawley albino rats when given undiluted at a single dose of 5 g/kg. The dermal LD_{50} value was > 2 g/kg of body weight for male and female New Zealand White rabbits when applied undiluted at a single dose of 2 g/kg to clipped abraded or intact skin patches. The primary dermal irritation index was 4.3 when applied undiluted at 0.5 mL/area to shaved abraded or intact skin patches. The primary eye irritation scores for unwashed (and washed) eyes after treatment with 0.1 mL of API 84-01 were 3 (4), 1.7 (0.0), 0.0 (0.0), 0.0 (0.0), and 0.0 (0.0) at 1, 24, 48, and 72 hr, and 7 days, respectively. API 84-01 was not a skin sensitizer in Hartley male albino guinea rigs when tested by the closed patch technique. Tables and appendix, 16 p.
■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1986) (39 p.).
Source: API HESD Information Specialist

33-30596
Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 84-02, heavy thermally cracked naphtha (CAS 64741-83-9). Final report. Studies by Hazleton Laboratories America Inc. on API 84-02, which consists mainly of unsaturated C_6-C_{12} hydrocarbons boiling at ~ 65-270 °C, showed an oral LD_{50} value of > 5 g/kg of body weight for male and female Sprague-Dawley albino rats when given undiluted at a single dose of 5 g/kg. The dermal LD_{50} value was > 2 g/kg of body weight for male and female New Zealand White rabbits when applied undiluted at a single dose of 2 g/kg to abraded or intact skin patches. The primary dermal irritation index for male rabbits was 4.9 when applied undiluted at 0.5 mL/area to abraded or intact skin patches. The primary eye irritation scores for unwashed (and washed) eyes after treatment with 0.1 mL of API 84-02 were 3 (2.7), 0.0 (0.0), 0.0 (0.0), 0.0 (0.0), and 0.0 (0.0) at 1, 24, 48, and 72 hr, and 7 days, respectively. API 84-02 was not a skin sensitizer in Hartley male albino guinea pigs when tested by the closed patch technique. Tables and appendix, 16 p.
■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1986) (40 p.).
Source: API HESD Information Specialist

33-30597
28-Day dermal toxicity study in the rabbit [by Tegeris Laboratories Inc.]. API 83-04, light catalytically reformed naphtha (CAS 64741-63-5). Final report. A study on groups of five male and five female New Zealand White rabbits showed moderate-to-severe skin irritations after API 83-04 (consisting mainly of C_5-C_{11} hydrocarbons boiling at ~ 35-190 °C) was applied to clipped intact skin patches at 200, 1000, or 2000 mg/kg three times a week for 28 days. All groups had treatment-related erythema, edema, atonicity, and cracked, flaking, dry, and/or leather-like skin at or around the test site, which, with other findings (e.g., fissured skin), were also seen in a gross pathological study. Also treatment-related were the statistically significantly lower than control mean body weights among the 2000 mg/kg groups; the statistically significantly lower than control mean weight gains among the 1000 mg/kg females and the 2000 mg/kg groups; and the slight-to-moderate proliferative and inflammatory changes that were accompanied by an increased granulopoiesis of the bone marrow, which was probably related to stress or other factors. Two male deaths, one each at 1000 and 2000 mg/kg, were considered incidental, spontaneous deaths. Tables and addenda, 21 p.
■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1986) (107 p.).
Source: API HESD Information Specialist

33-30598

[A Tegeris Laboratories Inc.] 28-day dermal toxicity study in the rabbit. API 83-05 full range catalytically reformed naphtha (CAS 68955-35-1). [This] final report shows that the application of API 83-05 (consisting mainly of C_4-C_{12} hydrocarbons boiling at ~ 30-220 °C) to the clipped intact skin of groups of five male and five female New Zealand White rabbits at 200, 1000, or 2000 mg/kg three times a week for 28 days, produced moderate-to-severe skin irritations. All groups showed treatment-related erythema, edema, atonicity, and cracked, dry, leather-like, and/or flaking skin at or around the test site. The highest dose groups showed statistically significantly lower than control body weight gains. Gross pathology treatment-related findings showed thickened, reddened, flaky, fissured, cracked, and/or dry skin on the treated site, which were microscopically described as proliferative and inflammatory changes. Such changes were accompanied by an increased granulopoiesis of the bone marrow that was probably related to stress or other factors. The deaths of two males at 2000 mg/kg and one at 1000 mg/kg were considered treatment-related. Tables and addenda, 55 p.
■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1986) (110 p.).
Source: API HESD Information Specialist

33-30599

Salmonella/mammalian-microsome plate incorporation mutagenicity assay (Ames test). Final report. This second phase of a two-phase study by Microbiological Associates Inc. indicates that the increased S-9 homogenate and co-factor concentrations in the modified Ames test enhance its ability to detect the mutagenic activity in complex hydrocarbon (HC) mixtures. Two tests in triplicate on each of the 12 HC samples on TA98 strain at 1000, 5000, 10,000, 25,000, and 50,0000 µg/plate have shown that 4 (XXX, 81-15, PS-8-76D5-ARO, and PS-8-76C5-ARO) of the 12 samples, or 33%, showed a positive response (reproducible, greater than two-fold, dose-responsive increase in revertants per plate) by the modified Ames test in the presence of an 80% S-9 mix of rat liver homogenate and nicotinamide adenine dinucleotide vs. one sample, 81-15, which showed a positive response, or 8%, by the standard Ames test in the presence of a 10% S-9 mix. Other findings include a 2.3- and 2.7-fold increase in revertants per plate in two independent modified Ames tests for sample PS-8-76D4-ARO, whose unclear dose response may prove positive at a lower dose range. Tables and graphs, 63 p. See also Abstract No. 32-32856.
■ *API Health Environ. Sci. Dep. Rep.* (Mar. 1986) (80 p.).
Source: API HESD Information Specialist

33-30601

The effect of Prudhoe Bay crude [(PBC)] oil on the homing [success] of coho salmon in marine waters was nil as shown by a study on 314 coho salmon *Oncorhynchus kisutch*, 233 of which were exposed at the School of Fisheries' Big Beef Creek Research Station on Hood Canal, Wash. and released at King Spit, Hood Canal. The fish were exposed to 913 ppm PBC (consisting mainly of soluble C_1-C_{10} hydrocarbons (HC) with concentrations ranging from 35 ppb 10 min after oil addition to 90 ppb after 1 hr), 105 ppm dispersed oil (the C_1-C_{10} HC were 1040 ppb after 10 min and 758 ppb after 1 hr), or 10.5 ppm salt-water dispersant for 1 hr before release. Of the exposed fish and the 81 untreated controls released, only 62 (19.7%) returned; the average was 15.8 ± 12 days. The speed of return, as measured by the number of days out (from time of release to the time of recapture), was the same for the controls and the treated fish. There were no treatment-related differences in latent differential mortalities in the exposed but unreleased fish (held in net pens), but there was a significant difference in the mean days alive due to age, the 2 and 3 yr olds lived 26.7 ± 19.9 and 10.3 ± 6.2 days, respectively. Tables, maps, block diagram, and 14 references.
■ R. E. Nakatani; E. O. Salo; A. E. Nevissi; R. P. Whitman; B. P. Snyder; S. P. Kaluzny (Fisheries Res. Inst., Univ. Wash.), *API Publication* #4411 (Sept. 1985) (62 p.).
Source: API Publications Order Desk (Order No. I44110)

33-30665

Oil effects on spawning behavior and reproduction in Pacific herring (*Clupea harengus pallasi*). Final Report Experimental laboratory exposure of Pacific herring eggs, sperm, and larvae to seawater containing Prudhoe Bay crude oil in 1982-83 showed that total saturate concentration is the single best predictor of abnormal larvae frequency. Exposure of the unfertilized eggs and sperm for two hours to oil-contaminated seawater had no effect on fertilization rate, and exposure of the attached eggs to contaminated seawater for 24 hours right after fertilization had no effect on the total percentage of eggs hatching successfully. Oil exposure significantly increased the frequency of abnormal larvae, causing abnormalities that would impair larval survival. The unfiltered, undispersed fresh oil treatments, in which substantial oil was present as droplets that adhered to the attached eggs, were the most severe, and the most likely to hinder herring reproductive success. Without adherence, even 47 ppm of toxic monoaromatic hydrocarbon concentrations had no effect. Map, diagrams, tables, and 62 references.
■ W. H. Pearson; D. L. Woodruff; S. L. Kiesser; G. W. Fellingham; R. A. Elston (Battelle Mar. Res. Lab.), *API Publication* #4412 (Oct. 1985) (123 p.).
Source: API Publications Order Desk (Order No. I44120)

33-30747

28-Day dermal toxicity study in the rabbit [by Tegeris Laboratories Inc.]. API 81-04, light catalytically cracked naphtha (CAS 64741-55-5). Final report. A study on API 81-04, which consists mainly of C_4-C_{11} hydrocarbons boiling at ~ -20 to +190 °C and which was applied to the clipped intact skin of groups of five male and five female New Zealand White rabbits at 200, 1000 (group 3), and 2000 (group 4) mg/kg three times a week for 28 days, showed that API 81-04 is a moderate-to-severe irritant to the rabbit skin. Treatment-related findings are erythema, edema, cracked skin, atonicity, and dry, leather-like, and flaking skin at or around the test site for all groups; the statistically significantly lower than control mean body weights for females in groups 3 and 4 and for group 4 males; the statistically significantly lower than control mean weight gains for group 3 males and group 4 females; and the slight-to-moderately severe proliferative and inflammatory changes on the treated skin. The increased granulopoiesis of the bone marrow accompanying the inflammatory changes is probably related to stress or other factors. The three male and two female deaths are not considered treatment-related. Tables, appendixes, and addenda, 84 p.
■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1986) (113 p.).
Source: API HESD Information Specialist

33-30929

Mutagenicity evaluation in the rat bone marrow cytogenetic assay. API 83-07, light catalytically cracked distillate (CAS 64741-59-9). Final report. A Litton Bionetics Inc. study on API 83-07, which consists mainly of C_9-C_{25} hydrocarbons boiling at ~ 150-400 °C and which was administered undiluted and intraperitoneally at 0.2, 0.67, or 2 g/kg to groups of five female and five male Sprague-Dawley rats, showed that API 83-07 is negative under the test conditions in the chromosomal aberration test in rat bone marrow cells. There was no significant increase in the percentage of aberrant cells above the negative controls for either sex at any of the doses or the 6, 24, or 48 hr kill times. The mitotic index of the bone marrow was not affected. A treatment of 1 mg/kg triethylenemelamine (TEM) given to positive controls induced significant increases in the percentage of cells with structural chromosomal aberrations in the males (29%) and females (59.9%). The data on the female positive controls could not be statistically analyzed, since the TEM-induced bone marrow toxicity generated insufficient analyzable metaphase cells. Tables, diagrams, and appendix, 17 p.
■ *API Health Environ. Sci. Dep. Rep.* (Mar. 1986) (30 p.).
Source: API HESD Information Specialist

33-30930

Mutagenicity evaluation in the rat bone marrow cytogenetic assay. API 83-11, straight run middle distillate (CAS 64741-44-2). Final report. A study by Litton Bionetics Inc. on groups of five male and five female Sprague-Dawley rats, which received 0.5, 1.7, or 5 g/kg of API 83-11 (consisting mainly of C_{11}-C_{20} hydrocarbons boiling at 205-345 °C) intraperitoneally and which were killed 6, 24, or 48 hr after dosing, showed that API-83-11 is negative under the test conditions for inducing chromosomal aberrations in the bone marrow cells of either male or female rats. There was neither a significant increase in the percentage of

aberrant cells above the deionized water-treated negative controls at any of the doses or kill times, nor any effect on the mitotic index. The positive vehicle of 1 mg/kg triethylenemelamine given to controls induced significant increases in the percentage of cells with structural aberrations in the males (29.3%) and females (57.6%). Tables, diagrams, and appendix, 16 p.
■ *API Health Environ. Sci. Dep. Rep.* (Mar. 1986) (29 p.).
Source: API HESD Information Specialist

33-30931
Sister chromatid exchange [(SCE)] assay in Chinese hamster ovary (CHO) cells. API PS-8-76D5 aromatics. Final report [by Microbiological Associates Inc.]. SCE assays on PS-8-76D5 aromatics (boiling at 700-1070 °F, with a total PAH fraction of 120.9 ppm, of which 48.6 ppm are active) in CHO cells showed the mutagenic potential of PS-8-76D5 in the presence or absence of Aroclor-induced rat liver S-9 activation, since it induced a dose-responsive and statistically significant increase in SCEs per second-division metaphase cell. After a 24 hr treatment of CHO cells with 0.3, 0.6, and 1.3 mg/mL PS-8-76D5 in dimethyl sulfoxide (DMSO) in the absence of activation, the SCE levels were significantly increased and appeared to be dose-dependent. A 2 hr treatment with 0.3, 0.6, 1.3, and 2.5 mg/mL in the presence of activation significantly increased the SCE/cell levels at all doses; the SCE levels were dose responsive for the three higher doses. The SCE/cell levels for the controls in the absence (and presence) of activation were: untreated, 11.04 (12.92); DMSO-treated 10.74 (14.78); and treated with 0.025 µg/mL triethylenemelamine with no activation, 46.36, (treated with cyclophosphamide with activation, 49.72). Because 5 mg/ml PS-8-76D5 floated on the growth medium, the SCE levels were not evaluated at this dosage. Tables, 6 p.
■ *API Health Environ. Sci. Dep. Rep.* (Mar. 1986) (25 p.).
Source: API HESD Information Specialist

33-30932
In-vivo sister chromatid exchange [(SCE)] assay. API PS8-76D5 saturates. Final report. A study by Microbiological Associates Inc. on PS8-76D5 saturates, which were dissolved in corn oil and injected intraperitoneally into male and female B6C3F1 mice at 1, 2, 4, 6, or 8 g/kg body weight of the test article, showed no mortality among the females at all dose levels, and a 20% mortality among males at 8 g/kg only. After this dose-range finding study, the assay was cancelled because of the uncertainty of the correct identity of the test material. The study was originally intended to measure the mutagenic potential of PS8-76D5 saturates by measuring their ability to induce SCEs in male and female mice. Table, 1 p.
■ *API Health Environ. Sci. Dep. Rep.* (Mar. 1986) (12 p.).
Source: API HESD Information Specialist

33-31034
Absorption of petroleum products across the skin of the monkey and miniature pig. Annual report (Mar. 15, 1979-Mar. 14, 1980). In-vivo studies on three male and three female adult rhesus (Macaca mullata) monkeys and on two miniature pigs of each sex, whose shaved backs were treated with ~ 0.5 mL each of carbon-14-labeled benzene, toluene, and o- and m-xylenes; and an in-vitro study on similarly treated skin obtained from adult Macaca Nemestrina monkeys, showed a rapid percutaneous absorption of these materials. The amounts of the absorbed radioactive materials recovered in the urine of monkeys (and pigs, as percentage of the applied dose) were: benzene, 0.076 ± 0.051 (0.042); toluene, 0.28 ± 0.04 (0.15); o-xylene, 0.46 ± 0.29; and m-xylene, 0.72 ± 0.20 (0.51). In-vitro absorption of the labeled compounds through the skin of Macaca Nemestrina monkeys was: benzene, 1.2 µL/sq cm; toluene, 0.15% dose/hr; o-xylene, 0.38% dose/hr; and m-xylene, 0.28% dose/hr. In-vivo percutaneous absorption by rhesus monkeys of carbon--14-labeled naphthalene dissolved in 95% ethanol was 3-10 times greater than that of BTX; in-vitro absorption was 2.39% for naphthalene dissolved in 95% ethanol and 1.42% for naphthalene dissolved in 95% toluene; in-vitro absorption of tritiated BaP dissolved in 95% ethanol, API crude oil C, or API crude oil D was < 1% of the applied dose. Charts, 7 p.
■ T. J. Franz (Univ. Wash. Sch. Med.), *API Health Environ. Sci. Dep. Rep.* (1986) (25 p.).
Source: API HESD Information Specialist

33-31092
Mutagenicity evaluation in the rat bone marrow cytogenetic assay. API 83-04, light catalytically reformed naphtha (CAS 64741-63-5). Final report [by Litton Bionetics Inc.]. For groups of five male and five female Sprague-Dawley rats, which received a single intraperitoneal injection of 0.25, 0.83, or 2.5 g/kg of API 83-04 (mainly C_5-C_{11} hydrocarbons boiling at ~ 35-190 °C) and were killed 6, 24, or 48 hr after treatment, API 83-04 is negative for inducing chromosomal aberrations in the bone marrow cells under the test conditions. API 83-04 neither induced significant increases in the percentage of chromosomally aberrant cells, nor affected the mitotic index of the bone marrow in either sex at any dose or kill time. The negative male and female controls, killed after respective doses of 0.76 and 0.57 mL of corn oil, showed a frequency of bone marrow aberrations within the test facility's historical range of control values. The positive controls killed after a 0.8 mg/kg dose of triethylenemelamine had increased frequencies of aberrations, but with no dose-related effects on the mitotic index to suggest bone marrow cytotoxicity. Tables, 9 p.
■ *API Health Environ. Sci. Dep. Rep.* (Mar. 1986) (25 p.).
Source: API HESD Information Specialist

33-31093
Mutagenicity evaluation in the rat bone marrow cytogenetic assay. API 81-08, sweetened naphtha (CAS 64741-87-3). Final report [by Litton Bionetics Inc.]. A repeat study on bone marrow cells from male and female Sprague-Dawley rats which inhaled API 81-08 (consisting mainly of C_4-C_{12} hydrocarbons boiling at ~ -10 - +230 °C) at intended (and actual) doses of 65 (69 ± 18), 300 (293 ± 42), and 2050 (2012 ± 16) ppm, showed that API 81-08 is negative in inducing chromosomal aberrations in rat bone marrow cells under the test conditions. There was no significant increase in chromosomal aberration compared with the negative controls, which after inhaling conditioned and filtered air, showed frequencies of aberrations of 0.016 in the males and 0.008 in the females. The positive controls that received a single intraperitoneal injection of 0.8 mg/kg of triethylenemelamine showed frequencies of aberrations of 0.708 in the males and 0.970 in the females. The repetition of this study is due to an uncertainty of the composition of exposure atmosphere in the original study. Tables and diagrams, 7 p.
■ *API Health Environ. Sci. Dep. Rep.* (Apr. 1986) (26 p.).
Source: API HESD Information Specialist

33-31094
Evidence for hematotoxicity and tumorigenesis in rats exposed to 100 ppm benzene was obtained by exposing a group of 40 Sprague-Dawley rats to the benzene via inhalation for 6 hr/day, 5 days/wk for life, i.e, 565 exposures over 863 calendar days, and comparing the response to that of a control group exposed only to air. During the exposure, the treated rats exhibited continuously depressed peripheral erythrocyte and lymphocyte counts; the incidences of these depressions were highly statistically significant, but their magnitudes were not. Histopathologic evaluation revealed that splenic hemosiderin pigments were much more prevalent in exposed rats than in controls, indicating either red cell hemolysis or ineffective erythropoieses. The mortality-corrected tumor incidences for exposed and control mice were not statistically different. Several exposed rats died with tumors that are most likely treatment-related owing to their rare spontaneous incidence. Thus, four rats died with liver tumors, two with zymbal gland carcinoma, and one with chronic myelogenous leukemia. Graphs, table, and 14 references.
■ C. A. Snyder; B. D. Goldstein; A. R. Sellakumar; R. E. Albert (N.Y.U. Med. Cent./Rutgers Med. Sch.), *American Journal of Industrial Medicine* 5:429-34 (1984; 1986) (6 p.).
Source: Not available from API

33-31095
A teratology evaluation of methyl tertiary butyl ether in rats and mice involved exposing mated female CD Sprague-Dawley rats and CD-1 mice to MTBE by inhalation of 0, 250, 1000, and 2500 ppm target concentrations in air for 6 hr/day during days 6-15 of gestation, and

showed that MTBE was not maternally toxic, embryotoxic, or teratogenic. None of the control or test group animals died during the treatment or posttreatment periods. No adverse effects of treatment were reflected in maternal parameters of body weight, water consumption, or liver weight or in physical examination data for both species. In rats, no treatment-related changes were recorded in the uterine implantation data, fetal size parameters, or fetal distribution data. No external abnormalities, skeletal malformations, or ossification variations were seen in mice or rats. The incidence of fused sternebrae in the mouse high-concentration group increased slightly, and might be attributed to fetotoxicity. (sponsored by Arco Chem. Co., Exxon Corp., Chem. Werke Huels A.G., Petrotex Chem. Corp., Phillips Pet. Co., Shell Oil Co., and Texaco Chem. Co. Tables and 17 references.

■ f C. C. Conaway (Texaco Inc.); R. E. Schroeder (Biodyn. Inc.); N. K. Snyder (API), *Journal of Toxicology and Environmental Health* 16:797-809 (1985).

Source: Not available from API

33-31096

The hematoxicity of inhaled benzene to Sprague-Dawley rats and AKR mice at 300 ppm in air was studied by exposing the animals for 6 hr/day, 5 days/week for life, which was a maximum of 99 weeks for the rats and 28 weeks for the mice. The calculated mean life span was 51 weeks for the rats, vs. 65 weeks for the controls, and 11 weeks for the mice, vs. 39 weeks for the controls. The rats exhibited lymphocytopenia, mild anemia, and moderately decreased survival compared with the controls. The mice showed severe lymphocytopenia and anemia accompanied by granulocytosis and reticulocytosis. The treated mice also showed significantly decreased survival and weight gain compared with the controls. Neither species showed indications of a leukemic or preleukemic response. Graphs, tables, photomicrograph, and 35 references.

■ C. A. Snyder (N.Y. Univ. Inst. Environ. Med.); S. R. Wolman (N.Y. Univ. Sch. Med.); B. D. Goldstein; A. Sellakumar; I. Bromberg; M. N. Erlichman; S. Laskin, *Journal of Toxicology and Environmental Health* 4:605-18 (1978).

Source: Not available from API

33-31097

Final Report. A four-week oral nephrotoxicity screening study in male F344 rats involved administering API PS-6 Fuel and 11 other hydrocarbon materials by gavage at a 2.0 g/kg dosage five days per week for four weeks, and saline by gavage at 2.0 g/kg to a negative control group. The nephropathy effect was severe for 2,2,4,4-tetramethyloctane, moderately severe for API PS-6 Fuel, moderate for 2,2,3-trimethyloctane, and slight for 2,2,4,4-tetramethyldecane. No nephrotoxic effect was found for 2,3-dimethyloctane, 2-methyldecane, decane, 3-methyloctane, octane, 2,2,3-trimethyldecane, 2,3-dimethyldecane, and 2,2,4,4,5,5,7,7-octamethyloctane. No deaths occurred in the saline control or the API PS-6 groups, but one rat in the 3-methyloctane group, and three rats in the octane group died. The most pronounced decrease in body weight at terminal sacrifice was seen in the API PS-6 and 2,2,3-trimethyloctane groups. No treatment-related effect on kidney weights was observed. The kidney lesions consisted of redness, congestion, mottling, paleness, dilated renal pelvis, and white precipitate in the renal pelvis. Tables and appendixes, 44 p.

■ *API Health Environ. Sci. Dep. Rep.* (Apr. 1986) (61 p.).

Source: API HESD Information Specialist

33-31098

Hydrocarbon nephropathy in male rats: Identification of the nephrotoxic components of unleaded gasoline as primarily the alkane constituents, with a positive structure-activity response relating the degree of alkane branching to the potency of the nephrotoxic response, was effected by a series of screening studies in which 0.5 or 2.0 g/kg/day of the test materials were administered via gastric lavage once daily, 5 days per week, for four weeks to groups of male F-344 rats vs. negative controls receiving isotonic saline and positive controls receiving reference unleaded gasoline. The materials tested were nine C_5-C_9 linear and branched alkanes, two alkenes, three aromatic hydrocarbons, methylcyclopentane, a light (A) and a heavy (B) catalytically cracked naphtha, a light catalytically reformed naphtha, a light alkylate naphtha, and the 145-220 °, 220-280 °, and 280 °F-to-ebp distillation fractions of the reference unleaded gasoline blend. The nephrotoxic activity observed for the naphtha streams and the distillation fractions correlated well with the proportion of branched alkanes contained in each. The largely unidentified alkenes contained in the A and B naphthas may also contribute to the nephrotoxicity of these streams. Graphs, tables, and 19 references, 9 p.

■ C. A. Halder (Amoco Corp); C. E. Holdsworth (API); B. Y. Cockrell (Exp. Pathol. Lab.); V. J. Piccirillo (Tegeris Lab. Inc.), *Toxicology and Industrial Health* 1(3):67-87 (1985; 1986) (21 p.).

Source: Not available from API

33-31163

Statistical approaches for assessing exposures to chemicals. A new, limiting distribution strategy is suggested for evaluating occupational exposures to chemicals (e.g., benzene), in lieu of the current U.S. Occupational Safety & Health Administration's permissible exposure limits (PELs), or a strategy that defines acceptable exposure distributions in terms of the fractions of eight-hour time-weighted averages which exceed the PEL. The PEL strategy, which does not allow exceedances, is flawed because compliance outcomes depend on the number of exposure measurements; the exceedance-based strategy cannot identify specific exposure distributions. The new strategy constrains both the mean and the variance of the exposure distribution, is appropriate for both acute and chronic hazard assessment, and links the assessment of acceptable exposure with the risks of disease. One method for deriving a limiting distribution and for testing exposures with it uses the elimination half-time of the chemical. Graphs, tables, flow chart, appendixes, and 48 references, 105 p.

■ *API Health Environ. Sci. Dep. Rep.* (Mar. 1985) (187 p.).

Source: API HESD Information Specialist

33-31223

Hydrocarbon nephropathy in male rats. Temporal and morphologic characterization of the renal lesions. In a study sponsored by the American Petroleum Institute, groups of male and female Fischer-344 rats were exposed by gavage to 0.5 and 2.0 g/kg of neat unleaded gasoline daily for 28 days, and compared with controls receiving 2.0 g/kg of isotonic saline. The apparent development of hydrocarbon nephropathy in the male rats was very rapid, since hyaline droplet accumulations in the epithelial cells of the proximal tubule could be seen one day after the start of the treatment. Increased foci of regenerative epithelium and parallel increased presence of dilated tubules at the junction of the inner and outer stripe of the medulla, with occlusion of the lumen by granular material were next observed, and required a minimum of ~ 2 weeks to develop. No significant involvement of the glomerulus was observed. The female rats were apparently resistant to the toxic effects of the gasoline on the kidney. Graph.

■ F. B. Thomas (Shell Oil Co.); C. A. Halder (Amoco Oil Corp.); C. E. Holdsworth (API); B. Y. Cockrell (Exper. Pathol. Lab. Inc.), *Monogr. Appl. Toxicol. [(Renal Heterogeneity Target Cell Toxicity Proc.]*, 2nd Nephrotoxicity Int. Symp. (Univ. Surrey, U.K. 8/6-9/84); #2:477-80 (1985) (6 p.).

Source: Not available from API

33-31224

Mutagenicity in a mouse lymphoma mutation assay. API 81-10, hydrodesulfurized middle distillate (CAS 64742-80-9). Final report. In-vitro treatments of the mouse lymphoma cell line L5178Y with API 81-10, consisting of C_{11}-C_{25} hydrocarbons boiling at 205-400 °C, showed that the test material was weakly active in the mouse lymphoma forward mutation assay, causing significant increases in the mutant frequency at the thymidine kinase locus in the presence and absence of metabolic activation by rat liver S9 homogenate. The cells were exposed to API 81-10 in absolute ethanol for four hours; the test material was assayed at 25-400 nL/mL under nonactivation conditions and at 12.5-300 nL/mL under activation conditions. Both series of tests showed repeatable increases in mutant frequency, but no clear-cut dose-response. Tables, 4p.

■ *API Health Environ. Sci. Dep. Rep.* (Apr. 1986) (27 p.).

Source: API HESD Information Specialist

33-31225

[In-vitro measurements of the] penetration of benzene through human [abdominal] skin from benzene solutions in water, PS-6 standardized reference gasoline (A, 2% benzene), hexadecane (B), and isooctane (C) yielded averaged permeability constants of 0.111, 0.0014, 0.0009, and 0.0037 cm/hr, respectively. The measured stratum corneum/water partition coefficient for benzene was 30.0. The partition coefficients for the other vehicles were very low and unmeasurable by the same method, but they were calculated by a new method as 0.11, 0.14, 0.17, and 0.19 for A, hexane, C, And D. The benzene flux through epidermis in vitro from air saturated with benzene at 31 °C averaged 1.0 µL/sq cm/hr. Calculations based on new and available data suggest that an adult working in ambient air containing 10 ppm of benzene with 100 sq cm of glabrous skin in contact with gasoline containing 5% benzene and the entire skin in contact with the air will absorb in 1 hr: 7.5 µL of benzene from inhalation, 7.0 from contact with gasoline, and 1.5 from body exposure. Graphs, tables, and 11 references. (supported in part by API)

■ I. H. Blank; D. J. McAuliffe (Harv. Med. Sch./Mass. Gen. Hosp.), *Journal of Investigative Dermatology* 85(6):522-26 (1985; 1986) (5 p.).
Source: Not available from API

33-31226

Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-16, light paraffinic distillate solvent extract (CAS 64742-05-8). API 83-16, consisting mainly of C_{15}-C_{30} aromatic hydrocarbons and likely to contain 5 wt% or more of 4- to 6-membered condensed ring aromatic hydrocarbons, had an estimated oral LD value of > 5 g/kg of body weight for male and female rats when administered undiluted by gavage. The dermal LD 50 value for 83-16 was > 3.0 g/kg of body weight for male and female rabbits when applied at 2.0 or 3.0 g/kg on shaved abraded and shaved nonabraded skin patches. The primary dermal irritation index was 5.4 for rabbits when applied undiluted at 0.5 mL per area to clipped abraded and clipped nonabraded skin patches. The primary eye irritation scores for rabbits were 3.3, 0.0, 0.0, 0.0, and 0.0 1 hr, 24 hr, 48 hr, 72 hr, and 7 days, respectively, after exposure to one 0.1 mL dose of undiluted 83-16 in eyes that were then immediately flushed with water. The corresponding scores for unflushed eyes were 3.7, 1.3, 0.0, 0.0, and 0.0. API 83-16 was not a skin sensitizer in guinea pigs tested by the closed patch technique. Tables and appendix, 15 p.
■ *API Health Environ. Sci. Dep. Rep.* (Apr. 1986) (41 p.).
Source: API HESD Information Specialist

33-31451

Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Twelve-month toxicity evaluation report. Final report. [Vol. 1-3]. Measurement of body weight changes, clinical observations, and tumor incidence data during the application twice a week of 50 µL of nine neat liquid and three toluene-diluted semisolid refinery streams to shaved skin patches of 16 groups of 400 male and 400 female mice, and gross pathological and histopathologic examinations on sacrificed mice after 3 and 12 mo, showed that API 81-15 at 10 and 1 wt % in toluene was a potent skin carcinogen and API 81-07, 81-09, and 81-10 liquids also were skin carcinogens. API 81-03, 81-08, 81-24, 83-01, 83-02, 83-03, and 81-13 and 81-14 at 50 wt % in toluene are locally dermatotoxic. No test materials caused any definitive chronic toxicity after 12 mo treatment. Other findings include test material-related deaths of 13 males and 13 females of 30 mice treated with API 81-15 at 10 wt % in toluene; similar dermal lesions in males and females treated with several of the test materials and with toluene only (controls); and statistically significant increases in group mean body weights vs. the controls. Tables and appendix, 430 p.
■ *API Health Environ. Sci. Dep. Rep.* (May 1986) (446 p.).
Source: API HESD Information Specialist

33-31598

Exposure data on C_7 and C_8 aromatics during handling and production of motor gasolines. A total of 1136 toluene measurements and 1062 measurements of o-, m-, and p-xylene and ethylbenzene were made in gas stations, loading facilities for trucks, rail cars, and tankers, at tank truck drivers and tanker crews, and in refineries at gasoline storage facilities, reforming units, facilities for production and transfer of benzene and xylene, and at maintenance and other personnel. The geometric mean values were < 8 mg/cu m (< 2 mL/cu m) of toluene and < 9 mg/cu m (< 2 mL/cu m) of C_8 aromatics. The data were collected by 10 West German oil companies. The analyses were by the flash and elution methods. The data are documented in great detail in order to make possible a mathematical-statistical analysis in the future. Tables.
■ H. Jungen; D. Mandak, *DGMK Ber.; API Publication* 250-1; #4439 (June 1985; June 1986) (132; 132 p.).
Source: API Publications Order Desk (Order No. I44390)

33-31641

L5178Y TK+/- Mouse Lymphoma Mutagenesis Assay. API 83-06, heavy catalytically reformed naphtha (CAS 64741-68-0). Final report [by Microbiological Associates Inc.]. This assay indicates that API 83-06, comprising mainly C_7-C_{12} aromatic hydrocarbons boiling at ~ 90-230 °C, produced dose-dependent but equivocal mutant frequency (MF) and total growth (TG) responses with or without Aroclor-induced rat liver S-9 activation. Of the 12 non-activated cultures (NACs) cloned and treated with 0.018-0.075 µL/mL API 83-06, two treated with 0.075 µL/mL and one treated with 0.056 µL/mL had MFs that were 31, 3.6, and 2 times, respectively, the ethanol controls' mean MF, with respective TGs of 1, 2, and 10%. The other NACs had MFs nonsignificantly greater than the control MFs, with a 31-115% TG. Of the six activated cultures (ACs) cloned and treated with 0.067, 0.089, or 0.12 µL/mL, two treated with 0.12 and 0.089 µL/mL had MFs that were 2.1 and 2.5 times, respectively, the control's MF, with respective TGs of 4 and 7%. The other ACs had non-significantly greater than control MFs, with a TG of 11-27%. Retesting at up to 0.22 µL/mL with activation showed a 10-91% TG. Tables, charts, diagrams, and appendix, 49 p.
■ *API Health Environ. Sci. Dep. Rep.* (May 1986) (67 p.).
Source: API HESD Information Specialist

33-31642

Twenty-eight-day dermal toxicity study in the rabbit. API 84-01, light paraffinic distillate (CAS 64741-50-0). Final report [by Tegeris Laboratories Inc.]. A study on groups of five male and five female New Zealand White rabbits, whose shaved intact backs were treated with 200, 1000, or 2000 mg/kg of API 84-01 (comprising mainly C_{15}-C_{30} hydrocarbons and producing a finished oil with a viscosity of < 1000 SUS at 100 °F) once a day, three times a week for 28 days, showed that API 84-01 is a minimal-to-moderate skin irritant to all of the rabbits. Treatment-related findings include erythema and edema in the 1000 and 2000 mg/kg groups that increased in frequency and severity with increasing dose; erythema only in the 200 mg/kg group; dry, scaly, thickened, reddened, and/or rough skin in the treatment area; and, as a marginal effect, body weight losses in a male and three females at 2000 mg/kg. Experimental Pathology Laboratories Inc.'s (EPL) microscopic data include slight-to-moderate proliferative changes in the high-dose males and females' skin. No treatment-related findings were seen in clinical hematology, chemistry, organ weights, or deaths of a low-dose female, a high-dose male, and a sham-treated male control. Tables, appendixes, and addenda, including EPL's pathology report, 91 p.
■ *API Health Environ. Sci. Dep. Rep.* (May 1986) (112 p.).
Source: API HESD Information Specialist

33-31695

28-Day dermal toxicity study in the rabbit. API 83-16, light paraffinic distillate solvent extract (CAS 64742-05-8). Final report [by Tegeris Laboratories Inc.]. A study on three groups of five male and five female New Zealand White rabbits, whose shaved intact backs were treated with 250, 500, or 1000 mg/kg of API 83-16 (comprising mainly C_{15}-C_{30} aromatic hydrocarbons) once a day, three times a week for 28 days, showed that API 83-16 was a minimal-to-moderate skin irritant to all of the rabbits. Treatment-related findings and microscopic pathology findings by Experimental Pathology Laboratories Inc. (EPLI) include very slight to slight edema in all groups; erythema in the high-dose groups only; leathery, cracked, and/or flaking skin; dry, scaly, crusted, red, fissured, and/or rough skin, and thickened dermis in the treatment

area; and slight-to-moderately severe proliferative changes of the skin in the 1000 mg/kg groups. Tables, appendixes, and addenda, including EPLI's report, 93p.
■ *API Health Environ. Sci. Dep. Rep.* (May 1986) (114 p.).
Source: API HESD Information Specialist

33-31696
28-day dermal toxicity in the rabbit. API 84-02, heavy thermally cracked naphtha (CAS 64741-83-9). Final report [by Tegeris Laboratories Inc.]. The application of 200, 1000, or 2000 mg/kg of API 84-02 (comprising mainly C_6-C_{12} unsaturated hydrocarbons boiling at ~ 65-270 °C) to the shaved intact backs of groups of five male and five female New Zealand White rabbits once a day, three times a week for 28 days, showed dose-dependent, slight-to-moderate skin irritation in all of the rabbits. The color of API 84-02 and the resultant staining of the treated site precluded erythema scoring. Treatment-related findings include statistically significantly lower than control mean body weights of some males in the 1000 and 2000 mg/kg groups; flaking and/or leathery skin at the treatment sites in all groups; flaky, crusted, red, dry, scaly, rough, and/or fissured skin, scabs, and thickened dermis on the treated skin; and, according to Experimental Pathology Laboratories Inc. (EPLI), slight-to-moderate proliferative changes accompanied by slight-to-moderately severe inflammatory changes in the skin of all the highest-dose rabbits. Tables, appendixes, and addenda, including the EPLI report, 99 p.
■ *API Health Environ. Sci. Dep. Rep.* (May 1986) (120 p.).
Source: API HESD Information Specialist

33-31750
Immunotoxicology of benzopyrenes in Fisher 344 rats. Final report. Studies showed that the Fisher 344 rat, like B6C3F1 female mice, can be used for immunotoxicology assays. In male Fisher rats which received subcutaneous injections of 2.5, 10, or 20 mg/kg BaP in corn oil and 20 mg/kg benzo(e)pyrene (BeP) in corn oil daily for 14 days, humoral immunity was decreased by BaP but not by BeP. In both species, BaP and BeP did not affect cell mediated immunity, and 20 mg/kg BaP significantly decreased the antibody forming cell response. Other data include a dose-dependent decrease in weight gain in BaP-treated rats; non-statistically significant increases in liver weights of BaP-exposed rats at 10 and 20 mg/kg and of BeP-exposed rats; slight but significant dose-dependent increases in spleen weights of rats exposed to BaP, but not to BeP; significant dose-dependent increase in lung weights of rats at 20 mg/kg BaP, but not among BeP-exposed rats; and reduced number of leukocytes in the 20 mg/kg BeP- and CPS-exposed rats. Tables, charts, and appendix, 27 p.
■ A. E. Munson; K. L. White (Med. Coll. Va.), *API Health Environ. Sci. Dep. Rep.* (4/1/86) (41 p.).
Source: API HESD Information Specialist

33-31751
Evaluation of the potential of PS-8-76D5-ARO to induce unscheduled DNA synthesis in primary rat hepatocyte cultures. Final report [by SRI International]. Results from an in vitro rat hepatocyte DNA repair assay have shown that PS8-76D5-ARO (boiling at 700-1070 °F and having a total of 120.9 ppm PAH) diluted in dimethylsulfoxide (DMSO) is genotoxic. The test material is cytotoxic to the liver cultures of two Fischer 344 rats at 5000 µg/mL in medium in the preliminary assay. The net grain counts in cells treated with 50, 100, and 500 µg/mL PS8-76D5-ARO in the preliminary (and replicate) assays are 4.6 (1.6), 18.2 (5), and 39 (19.9), respectively, vs. - 14.1 (- 13.2) for the negative control treated with 1% DMSO, 34.3 (42.7) for the positive control treated with 0.5 µg/mL 2-acetylaminofluorene, and - 9.2 (- 10.7) for the untreated medium control. The respective percentage of cells in repair are 44% (42), 86% (52), and 97% (85) vs. 1% (2), 70% (85), and 4% (2). Tables and appendix, 38 p.
■ *API Health Environ. Sci. Dep. Rep.* (June 1986) (53 p.).
Source: API HESD Information Specialist

33-31826
Evaluation of the potential of PS-8-76D5-ARO to induce unscheduled DNA synthesis [(UDS)] in the in-vivo/in-vitro hepatocyte DNA repair assay. Final report [by SRI International]. Results from in-vivo/in-vitro hepatocyte DNA repair assays suggest that PS-8-76D5-ARO (a honey-colored viscous liquid) is not genotoxic in the Fischer 344 male rat liver. The liver cell cultures obtained from rats treated by oral gavage with 50, 200, or 1000 mg/kg of PS-8-76D5-ARO in corn oil 2 and 12 hr prior to sacrifice showed mean net grains/nucleus of -6.5 and -4.1, -4.7 and -3.7, and -4.8 and -3.9, respectively, vs. 15.1 for the positive control animals treated with 50 mg/kg of 2-acetylaminofluorene in corn oil 12 prior to sacrifice and -5.5 for the negative controls treated with corn oil 12 hr prior to sacrifice. PS-8-76D5-ARO did not induce any increase in UDS, since the mean percentages of cells in repair for all the treated rats were 0-2%, vs. 78% for the positive controls and 1% for the negative controls. The low animal-to-animal variation in the results for all treatment groups indicates a high degree of reproducibility. Tables, 10 references, and appendix, 17 p.
■ *API Health Environ. Sci. Dep. Rep.* (June 1986) (32 p.).
Source: API HESD Information Specialist

33-31827
LC_{50} acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 81-08, sweetened naphtha (CAS 64741-87-3). Final report [by International Research & Development Corp.]. A study on two groups of five male and five female Sprague-Dawley rats exposed for 4 hr to 5.2 mg/L of API 81-08 (comprising mainly C_4-C_{12} hydrocarbons boiling at ~ -10 to +230 °C) in air, and on a control group exposed to air only, showed no unusual pharmacotoxic signs or behavior among the first group of rats exposed to API 81-08. The second API 81-08-exposed group had a slight incidence of nasal discharge during exposure, but the incidence of various signs after exposure was not considered significant. The treated males in the second group showed normal body weight gains, but the females in this group had slightly lower than expected weight gains. There were no deaths, no neoplastic changes, and no toxicologically significant macroscopic or microscopic changes that were exposure-related or that could be correlated with observed pharmacotoxic signs or weight gains. The observed changes were not considered unusual in type, frequency, or severity for this rat strain. Tables, charts, and diagram, 13 p.
■ *API Health Environ. Sci. Dep. Rep.* (June 1986) (36 p.).
Source: API HESD Information Specialist

33-31902
LC_{50} acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 81-03, light catalytically cracked naphtha (CAS 64741-55-5). Final report. An International Research & Development Corp. study on two groups of five male and five female Sprague-Dawley rats, which were exposed to 5.2 mg/L of API 81-03 (comprising mainly C_4-C_{11} hydrocarbons boiling at ~ -20 to +190 °C) in air for 4 hr, and on a similar group of controls exposed to air only, showed no notable incidence of pharmacotoxic signs during or after exposure to API 81-03, with only the females showing nasal discharge during exposure. There were no changes in body weights, no toxicologically significant macroscopic or microscopic changes, no deaths, and no neoplastic changes that were related to exposure. The changes that were observed were not unusual in type, frequency, or severity for this rat strain. Tables, charts, and diagram, 15 p.
■ *API Health Environ. Sci. Dep. Rep.* (June 1986) (38 p.).
Source: API HESD Information Specialist

33-32009
Lifetime dermal carcinogenesis bioassay of shale oil-derived streams in C3H/HeJ mice. Final interim report. The application of 50 µL each of 30 shale oil-derived streams to the clipped backs of 34 groups of 50 mice each twice a week until API requested termination after 30 wk, showed cutaneous tumors in weeks 20-30 in 19 mice treated with API-SFR-1 (raw shale oil No. 1), in 1 and 14 mice treated with API-SFR-1 cuts boiling at 550-700 °C and at 700-1070 °C, respectively; in 5 mice treated with API-SFR-2 (raw shale oil No. 2), and in 3 and 4 mice treated with API-SFR-2 cuts boiling at 550-700 °C and at 700-1070 °C, respectively. Some of the mice with tumors also had papillomas and/or advanced tumors. There were subcutaneous tumors in a negative control mouse treated with toluene, in a positive control mouse treated

with 0.05% BaP in toluene, and in a mouse treated with a cut of 0.06% N, hydrotreated shale oil (HSO) No. 2 boiling at 700-1070 °C. Other findings include 22 deaths from 19 treatment groups, with internal pathologic changes in kidneys, lungs, livers, lymph nodes, spleens, and urogenital systems that were typical of this mice strain. Test materials also included 0.05 and 0.19% N, HSO No. 1 and high-nitrogen crude oil. Tables and appendixes, 27 p.
■ J. H. Coleman (Bushy Run Res. Cent.), *API Health Environ. Sci. Dep. Rep.* (July 1986) (40 p.).
Source: API HESD Information Specialist

33-32010
Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Three-month toxicity evaluation report. Final report. Body weight changes, clinical observations, and tumor incidence data during the application twice a week of 50 µL of 11 neat liquid or semisolid petroleum refinery streams to the clipped backs of 1400 male and female mice, and gross pathological and histopathologic examinations on sacrificed mice after 3 or 12 mo, showed that none of the test materials was carcinogenic, although all were locally dermatotoxic. No treatment-related systemic toxicity was apparent. A dermal papilloma and a malignant bronchiolar/alveolar adenoma in two females treated with API 83-16 were the only tumors seen and were expected in this strain of mice. Other findings include dermal lesions, irritation, scabbing, erythema, desquamation, and/or alopecia in all treated mice, which were more severe in mice treated with API 83-07 and API 83-16 than in the control mice treated with 50 µL of neat toluene; more prominent and consistent body weight gains in mice treated with API 83-07 than in all other treated mice; and statistically significantly greater than control group mean liver and kidney weights in all groups. Vol. 1, tables and appendix, 187 p; Vol. 2, all appendix, 260 p.
■ *API Health Environ. Sci. Dep. Rep.* 1-2 (July 1986) (467 p.).
Source: API HESD Information Specialist

33-32622
The Role of Chemical Dispersants in Oil Spill Control. Recent studies on dispersant effectiveness in treating test spills of Murban and Prudhoe Bay crudes off the New Jersey and Southern California coasts showed accelerated removal of the toxic C_1-C_{10} hydrocarbons from the dispersed oil compared with untreated oil slicks. Other tests on intertidal release at Searsport, Maine, of nearshore subtidal release at Baffin Island, Can., and of release to a Panamanian mangrove forest showed that chemically dispersed oil, even in nearshore waters, caused fewer adverse effects on marine organisms than untreated oil stranded onshore. The dispersants caused temporary higher local concentrations of dispersants and dispersed oil in the water column, and thus increased short-term adverse effects on water column organisms, but the long-term effects of untreated, concentrated oil were much more severe. An oil spill decision diagram suggests a spill control strategy for small and large oil spills, depending on the sea state and the availability of equipment for mechanical cleanup or spray application. Charts, diagrams, maps, tables, and 62 references.
■ *API Publication* #4425 (Jan. 1986) (45 p.).
Source: API Publications Order Desk (Order No. I44250)

33-32638
Morphological transformation of BALB/3T3 mouse embryo cells. API 81-15, catalytically cracked clarified oil (CAS 64741-62-4). Final report [by Microbiological Associates Inc.]. An assay on mouse embryo cells treated with 1, 3, 6, or 9 µg/mL API 81-15 (comprising mainly greater than C_{20} hydrocarbons boiling at ~ 350 °C) for 3 days without Aroclor-induced rat liver S-9 activation showed that API 81-15 is negative, since no significant increases in morphological transformation frequency were observed. API 81-15 is suspect at 10, 30, 100, or 300 µg/mL API 81-15 with activation for 4 hr, since there was a significant increase in transformation at 100 µg/mL. Compared with the negative control cells treated with 2 µL/mL acetone, the survival of cells treated with 1, 3, 6, and 9 µg/mL API 81-15 without activation was } 96, 91, 85, and 66%, respectively, vs. ~ 69, 38, 21, and 18% for cells treated with 10, 30, 100, and 300 µg/mL API 81-15 with activation, respectively. Positive control cells treated with 0.5 µg/mL N-methyl-N'-nitro-N-nitrosoguanidine in the nonactivated study (and 12.5 µg/mL Bap in the activated study) induced 18 (6) Type II and 15 (8) Type III foci. Tables.
■ *API Health Environ. Sci. Dep. Rep.* (Aug. 1986) (20 p.).
Source: API HESD Information Specialist

33-32639
Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. API 83-15, hydrotreated heavy naphthenic distillate (CAS 64742-52-5). Final report [by Hazleton Laboratories America Inc.]. Findings from studies on API 83-15 (mainly C_{20}-C_{50} hydrocarbons that produce a finished oil of at least 100 SUS at 100 °F) include oral LD_{50}s > 5.0 g/kg of body weight for male and female Sprague-Dawley rats after a dose of 5.0 g/kg API 83-15; dermal LD_{50}s > 2.0 g/kg of body weight for male and female New Zealand White rabbits whose shaved or intact backs were treated once with 2.0 g/kg API 83-15; a primary dermal irritation index of 1.3 for rabbits whose shaved or intact backs and flanks were treated with 0.5 mL API 83-15; primary eye irritation scores 1, 24, 48, and 72 hr, and 7 days after a 0.1 mL API 83-15 treatment were 4.5, 1.3, 0.0, 0.0, and 0.0, respectively, for rabbits whose eyes remained unwashed vs. 4. 7, 0.7, 0.0, 0.0, and 0.0, respectively, for those whose eyes were later washed; pure API 83-15 is not considered to be a skin sensitizer in male Dunkin Hartley albino guinea pigs by the closed patch technique at 25, 50, and 75 vol/vol % in mineral oil. Tables and appendix.
■ *API Health Environ. Sci. Dep. Rep.* (Aug. 1986) (41 p.).
Source: API HESD Information Specialist

33-32722
Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. Final report. API 83-20, light catalytic cracked naphtha (petroleum). (CAS 64741-55-5). API 83-20, consisting mainly of C_4-C_{11} hydrocarbons and having a high olefins content, had an estimated oral LD_{50} value of > 5 g/kg of body weight for male and female rats when administered undiluted by gavage. The dermal LD_{50} value for 83-20 was > 3 g/kg for male and female rabbits when applied at 2.0 or 3.0 g/kg on shaved abraded and shaved nonabraded skin patches. The primary dermal irritation index was 3.7 for rabbits when applied undiluted at 0.5 mL per area to clipped abraded and clipped nonabraded skin patches. The primary eye irritation scores for rabbits were 3.3, 0.0, 0.0, 0.0, and 0.0 1 hr, 24 hr, 48 hr, 72 hr, and 7 days after exposure to one 0.1 mL dose of undiluted 83-20 in eyes that were then flushed immediately with water. The corresponding scores for unflushed eyes were 1.0, 0.0, 0.0, 0.0, and 0.0. API 83-20 was not a skin sensitizer in guinea pigs tested by the closed patch technique. Table and appendixes, 15 p.
■ *API Health Environ. Sci. Dep. Rep.* (Aug. 1986) (42 p.).
Source: API HESD Information Specialist

33-32723
Acute oral toxicity study in rats. Acute dermal toxicity study in rabbits. Primary dermal irritation study in rabbits. Primary eye irritation study in rabbits. Dermal sensitization study in guinea pigs. Final report. API 85-01, Stoddard solvent. (CAS 8052-41-3). API 85-01, a petroleum distillate boiling at 300-400 °F, had an estimated oral LD_{50} value of > 5.0 g/kg of body weight for male and female rats when administered undiluted by gavage. The dermal LD_{50} value for 85-01 was > 3.0 g/kg of body weight for male and female rabbits when applied at 2.0 or 3.0 g/kg to shaved abraded and shave nonabraded skin patches. The primary dermal irritation index was 4.5 for rabbits when applied indiluted at 0.5 mL per area to clipped abraded and clipped nonabraded skin patches. The primary eye irritation scores for rabbits were 4.7, 0.0, 0.0, 0.0, and 0.0 1 hr, 24 hr, 48 hr, 72 hr, and 7 days after exposure to one 0.1 mL dose of 85-01 in eyes that were then immediately flushed with water. The corresponding scores for unflushed eyes were 1.0, 0.0, 0.0, 0.0, and 0.0. API 85-01 was not a skin sensitizer for guinea pigs tested by the closed patch technique. Tables, and appendix, 13 p.
■ *API Health Environ. Sci. Dep. Rep.* (Aug. 1986) (41 p.).
Source: API HESD Information Specialist

33-32724

Four-week subchronic inhalation toxicity study in rats. Final report. API 81-07, hydrodesulfurized kerosine (petroleum) (CAS 64742-81-0). API 81-09, hydrodesulfurized middle distillate (petroleum) (CAS 64742-80-9). API 81-10, hydrodesulfurized middle distillate (petroleum) (CAS 64742-80-9). One group of 20 female and 20 male Sprague-Dawley-derived rats was exposed to a 24 mg/cu m of API 81-07 vapor atmosphere, another to 23 mg/cu m of 81-09, another to 24 mg/cu m of 81-10, and another to filtered air only for ~ 6 hr/day, 5 days/wk for four consecutive weeks. No animal died and there were no exposure-related clinical signs, body weight effects, serum biochemical changes, or hematologic changes, except for moderately increased leukocyte counts in animals exposed to 81-10, which contained high nitrogen and low aromatics and had a wide boiling range. There were no test material-related macroscopic findings in any of the groups. The only noteworthy microscopic changes were in the nasal tissues of some male and female rats exposed to 81-09, which contained low nitrogen and high aromatics and had a narrow boiling range. These changes were subacute inflammation of the respiratory mucosa lining, with the severity of the inflammation ranging from trace to mild in both sexes. Tables.
■ *API Health Environ. Sci. Dep. Rep.* (Sept. 1986) (18 p.).
Source: API HESD Information Specialist

33-32725

Characterization of the carcinogenic nature of selected low boiling [petroleum] fractions. Final report. The tests on crude C fraction C2, which contained 16.9% C_6-C_{12} paraffins, 51.7% monocycloparaffins, and 23.8% dicycloparaffins, and on crude D fraction D2, which contained 69.2, 18.7 and 0.6% of the respective compound classes, involved the classical mouse skin painting bioassay on groups of 50 male C3H/HeJ mice for up to 122 weeks. The groups received 50 µL doses (usually in acetone) of C2, D2, acetone along, the carcinogenic initiator 7,12-dimethylbenz[a]anthracene (DMBA), and the promoter 12-O-tetradecanoylphorbyl-13-acetate (TPA) in various sequences. Except for acetone, all the test materials showed carcinogenic activity, which was greatest for DMBA, less for TPA, and lowest for C2 and D2. C2 and D2 did not act as promoters after initiation by DMBA. The oils showed no more than slight carcinogenic activity when applied with DMBA, and may have acted as inhibitors when applied with TPA. Acanthosis and dermal fibrosis, both non-neoplastic changes, as well as dysplasia, were observed in all test groups. Diagrams, graphs, tables, and appendixes, 212 p.
■ *API Health Environ. Sci. Dep. Rep.* (Aug. 1986) (243 p.).
Source: API HESD Information Specialist

33-32726

An update of a mortality study of workers in petroleum refineries begun in 1972 under API sponsorship covers 19,991 workers with long duration of employment (equivalent to 297,591.9 person-years) and extends to 12/31/80. For all causes, the number of observed deaths is less than four-fifths of the number expected, with a standard mortality ratio (SMR) of 78, due in part to the healthy-worker effect. The SMRs are even lower for many specific causes, including cancer of the kidney (68), emphysema (63), and genitourinary diseases (63). Because of the large cohort, some SMRs are significantly low even though they are close to 100, including all malignant neoplasms and lung cancer. The SMR is 73 for chronic nephritis and 87 for all cancers. The SMRs for specific neoplasms are: digestive system, 90; lung, 85; brain, 89; leukemia, 101; multiple myeloma, 123; unspecified lymphoma, 112; polycythemia vera (4 deaths), 455; myelofibrosis (3 deaths), 201; and benign and unspecified brain neoplasms, 108. There were nine deaths from mesothelioma, SMR 241. Tables.
■ S. D. Kaplan (SRI Int.), *Journal of Occupational Medicine* 28(7):514--16 (July 1986).
Source: Not available from API

33-32727

Benzene levels in ambient air and breath of smokers and nonsmokers in urban and pristine environments were measured by GC/MS in urban San Francisco (SF) and in coastal, pristine Stinson Beach (SB), Calif. Ambient benzene was 2.6 ± 1.3 ppb in SF vs. 0.38 ± 0.39 ppb in SB; the highest levels in SF air were inland near heavy traffic. In both SF and SB, the benzene in smokers' breath was higher than in nonsmokers' breath and in ambient air. Benzene in SF nonsmokers' breath was greater than in SB nonsmokers' breath. There was little correlation between benzene in breath and number of cigarettes smoked, or with other benzene exposures such as diet. Benzene was 2.5 ± 8 ppb in the breath of SF nonsmokers vs. 1.4 ± 0.1 ppb in nonsmokers' ambient air; benzene was also greater in the breath of SB nonsmokers than in SB nonsmokers' ambient air. This finding suggests an additional source of benzene other than outdoor air. No correlation was found between benzene breath levels and exposure to benzene in the home or workplace as given on questionnaires filled out by each subject, suggesting a possible in vivo source of benzene production. Tables and 10 references.
■ R. C. Wester; H. I. Maibach; L. D. Gruenke; J. C. Craig (Univ. Calif., Sch. Pharmacy, Sch. Med.), *Journal of Toxicology and Environmental Health* 18:567-73 (1986).
Source: Not available from API

33-32800

Mutagenicity of API 83-15, hydrotreated heavy naphthenic distillate, (CAS 64742-52-5), in a mouse lymphoma mutation assay. Final report. In the mouse lymphoma forward mutation assay, in-vitro treatments of the mouse lymphoma cell line L5178Y with 83-15, consisting mostly of C_{20}-C_{50} hydrocarbons and containing relatively few n-paraffins, induced no significant increases in the mutation frequency at the thymidine kinase locus. The cells were exposed to 83-15 dissolved in dimethyl sulfoxide for four hours in the presence and absence of rat liver S9 metabolic activation; the test materials appeared insoluble at concentrations above 500 nL/mL. Under nonactivation conditions, the test material was assayed at 400-1000 nL/mL, and no significant increases above the average solvent control mutant frequency were induced. Little or no toxicity was induced. In the presence of metabolic activation, treatments of 200-1000 nL/mL were assayed for mutant induction, and no significant increases in the mutant frequency were induced; low to moderate toxicities were observed. API 83-15 was thus evaluated as inactive in the mouse lymphoma cell system.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1986) (18 p.).
Source: API HESD Information Specialist

33-32801

Mutagenicity of API 84-01, light paraffinic distillate (CAS 64741-50-0), in a mouse lymphoma mutation assay. Final report. In the mouse lymphoma forward mutation assay, in-vitro treatments of the mouse lymphoma cell line L5178Y with API 84-01 dissolved in acetone induced significant increases in the mutant frequency at the thymidine kinase locus only in the presence of metabolic activation. The cells were exposed to API 84-01 for four hours with and without metabolic activation with S9 rat liver homogenate. The test material was visibly insoluble above 100 nL/mL. Under nonactivation conditions, the test material was assayed from 400 to 1000 nL/mL, and little or no toxicity was observed well into the insoluble range. No significant increases in the mutant frequency were observed. In the presence of metabolic activation, the test material was converted to a more active form or forms. Treatments at 50-1000 nL/mL induced significant increases in the mutant frequency that were 2.1- to 7.3-fold above the background mutant frequency. API 84-01 was therefore evaluated as active in the mouse lymphoma cell system only in the presence of metabolic activation. Tables, 4 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1986) (25 p.).
Source: API HESD Information Specialist

33-32802

Mutagenicity of API 83-12, hydrotreated light naphthenic distillate (CAS 64742-53-6) in a mouse lymphoma mutation assay. Final report. In the mouse lymphoma forward assay, in-vitro treatments of the mouse lymphoma cell line L5178Y with API 83-12, consisting mainly of C_{15}-C_{30} hydrocarbons with relatively few n-paraffins, induced significant increases in the mutant frequency at the thymidine kinase locus in the presence and absence of metabolic activation with S9 rat liver homogenate. The cells were exposed to 83-12 in absolute ethanol for four hours. The test material was soluble at all concentrations assayed. Under nonactivation conditions, the test material was assayed at

250-1200 nL/mL, and a wide range of toxicities was induced. Repeatable increases in the mutant frequency were induced primarily at high toxicities. In the presence of metabolic activation, the test material was assayed at 400-1600 nL/mL. Treatments at and above 1000nL/mL induced repeatable increases in the mutant frequency that were 2.5- to 8.8-fold above the background mutant frequency. API 83-12 was therefore evaluated as active in the mouse lymphoma cell system with and without metabolic activation. Tables, 5 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1986) (26 p.).
Source: API HESD Information Specialist

33-32803
Mutagenicity of API 83-16, light paraffinic distillate solvent extract (petroleum) (CAS 64742-05-8), in a mouse lymphoma mutation assay. Final report. In the mouse lymphoma forward mutation assay, in-vitro treatments of the mouse lymphoma cell line L5178Y with API 83-16, consisting mainly of C_{15}-C_{30} aromatic hydrocarbons, induced significant increases in the mutant frequency at the thymidine kinase locus with and without metabolic activation by S9 rat liver homogenate. Exposure was for four hours in both cases. The test material was insoluble at and above 100 nL/mL. Under nonactivation conditions, the test material was assayed at 25-200 nL/mL, and a wide range of toxicities was induced. Significant increases in mutant frequency were induced at high toxicities. In the presence of metabolic activation, the test material was assayed at 12.5-150 nL/mL. Treatments at 25-150 nL/mL induced significant increases in the mutant frequency that were 1.9- to 3.2-fold above the background mutant frequency. API was thus active in the mouse lymphoma cell system in the presence and absence of metabolic activation. Tables, 5 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1986) (25 p.).
Source: API HESD Information Specialist

33-32804
Mutagenicity of API 83-18, heavy catalytic cracked naphtha (petroleum) (CAS 64741-54-4), in a mouse lymphoma mutation assay. Final report. In the mouse lymphoma forward mutation assay, in-vitro treatments of the mouse lymphoma cell line L5178Y with API 83-18, consisting mainly of C_6-C_{12} hydrocarbons and having a relatively large proportion of unsaturated hydrocarbons, induced significant increases in the mutant frequency at the thymidine kinase locus with and without metabolic activation by S9 rat liver homogenate. The cells were exposed to 83-18 dissolved in ethanol for four hours. The test material was insoluble at and above 250 nL/mL. Under nonactivation conditions, the test material was assayed for mutant induction at 25-80 nL/mL and a wide range of toxicities was induced. Small but significant increases in the mutant frequencies were induced at high toxicities. In the presence of metabolic activation, the test material was converted to a more active form or forms. Treatments at 25-100 nL/mL induced significant increases in the mutant frequency that were 1.9- to 9.4-fold above the background mutant frequency. API 83-18 was thus evaluated as active in the mouse lymphoma cell system with and without metabolic activation.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1986) (25 p.).
Source: API HESD Information Specialist

33-32864
A single-generation inhalation reproduction/fertility study on a commercial hexane containing also considerable amounts of 2- and 3-methylpentane and methylcyclopentane was made by whole-body inhalation exposure of male and female rats to 100, 500, and 1500 ppm of the test article for 6 hr/day, 7 days/wk during a 100 day premating period and a 15 day mating period, and of females also on days 1-20 of lactation and days 5-21 of lactation. Male and female groups were also treated at 1500 ppm and bred to untreated counterparts. A teratologic evaluation was conducted on selected fetuses. No unequivocal test article effects were detected in parental or pup parameters in any tested group during the study. For the treated groups, the appearance and behavior, body weight, food consumption, Cesarean section observations, and reproductive parameters of the adults, and the fetal morphological observations and litter parameter values, did not fluctuate relative to corresponding control group values. There were also no test article-related macroscopic, microscopic, or organ weight changes among the animals evaluated. The no-observable-effect level for this study was established at 1500 ppm.
■ *API Health Environ. Sci. Dep. Rep.* 1 & 2 (Sept. 1986) (392 p.).
Source: API HESD Information Specialist

33-32871
The toxicity of dispersed and undispersed Prudhoe Bay crude [(PBC)] oil fractions to shrimp, fish, and their larvae was tested with fresh PBC, a Stage I fraction containing diaromatics but no significant amounts of monoaromatics, and A Stage II fraction containing only aromatics with three or more rings but no saturated hydrocarbons of corresponding boiling points. Bioassays on adult shrimp showed that the removal of monoaromatics reduced toxicity about seven-fold. Shrimp larvae were about four times more sensitive to the water soluble fraction (WSF) of fresh PBC oil than the adults. Adult sand lance fish mortality did not correlate with the aromatic content of the oils, but appeared to be affected by dispersed droplets of the three oils to the same degree. Larval herring were not much more sensitive to dispersed oils than adult sand lance, but mortality is not a good measure of impacts on these larvae because they exhibit a long moribund period. Haddock larvae were about five to seven times more sensitive to dispersions of fresh PBC than to dispersed Stage I oil, and were more sensitive to both dispersions than were herring larvae. The effects on haddock are in an appended report by R. S. Carr and J. M. Neff. Graphs, tables, and 23 references, 33 p.
■ J. W. Anderson (Battelle/Mar. Res. Lab.); R. S. Carr; J. M. Neff (N. Engl. Mar. Res. Lab.), *API Publication* #4441 (Aug. 1985) (82 p.).
Source: API Publications Order Desk (Order No. I44410)

33-32910
Quantitative analysis of benzene by selected ion monitoring/gas chromatography/mass spectrometry. The method developed for benzene utilized a headspace assay with benzene-1,3,5-d_3 as the internal standard and permitted detection limits of 2 ng/mL in blood and 0.1 ppb in a 5 L sample of air or breath. The effects of contamination by background benzene and of losses owing to volatilization and leakage were carefully studied and, when background contamination could not be readily controlled, the assay was modified for the quantitative determination of labelled benzenes six mass units heavier, i.e., benzene-d_6 or benzene-carbon-13_6. The method was illustrated by measuring the benzene background levels in smokers and nonsmokers. Graphs, tables, and 34 references.
■ L. D. Gruenke (Univ. Calif., Sch. Pharmacy); R. C. Wester (Univ. Calif., Sch. Med.); J. C. Craig; H. I. Maibach, *Journal of Analytical Toxicology* 10:225-32 (Nov.-Dec. 1986).
Source: Not available from API

34-30329
Mutagenicity of API 85-01, Stoddard solvent (CAS 8052-41-3), in a mouse lymphoma mutation assay. Final report [by Litton Bionetics Inc.]. In-vitro treatments of mouse lymphoma cells, L5178Y, with API 85-01 (a colorless, refined petroleum distillate boiling at ~ 300-400 °F) in ethanol solvent for 4 hr showed that the test material induced significant increases in the mutant frequency at the thymidine kinase locus and is thus mutagenic with or without rat liver S9 metabolic activation. Compared with the positive controls treated with 0.25-0.5 µL/mL ethyl methanesulfonate in the absence of activation, 12.5-50 nL/mL API 85-01 induced, at most, moderate toxicities in the first trial; but in the second trial, mutant frequencies that exceeded the minimum criterion were induced at 50-60 nL/mL. Compared with positive controls treated with 1.0-4.0 µg/mL 3-methylcholanthrene in the presence of activation, 12.5-50 nL/mL API 85-01 induced low toxicities in the first trial, with the highest dose inducing a mutant frequency that just exceeded the minimum criterion. At 30-100 nL/mL, a wide range of toxicities and mutant frequencies that exceeded the minimum criterion were induced. Tables, 2 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1987) (26 p.).
Source: API HESD Information Specialist

34-30330
Crude petroleum and selected fractions. Skin cancer bioassays. A study on groups of 10-30 male CH3 mice, whose clipped skins were

treated for 18 mo (or until a grossly observable cancer was found) with 50 or 100 mg residuum fractions boiling at < 750 ° and 750-925 °F, which were obtained from noncatalytic cracking oils and which contained 15-380 µg/wk BaP, showed 67-100 tumors and a mean latency of 15-53 wk. Mice treated with distillate fractions (and residuum fractions, in parentheses) from catalytically cracked oils, which contained 3-80 µg/wk BaP (90-1890 µg/wk) showed 89-100 (83-100) tumors and a 13-24 wk (8-36 wk) latency. Unlike the noncatalytically cracked oil fractions, the lower boiling distillate and higher boiling residuum fractions from catalytically cracked oils showed significant correlations between tumor yield and BaP dose and between BaP dose and tumor latency, respectively. Groups of 50 mice treated with 50 mg of whole crude oils C and D containing 1.0 and 1.8 wt % PAHs, respectively, showed 20 and 38 tumors, respectively, with respective latencies of 69 and 64 wk. The tumor yields and latencies for mice treated with distillate fractions of crudes C (and D, in parentheses) were: < 120 °F, 0, 0 wk (0, > 110 wk); 120-350 °F, 16, 85 wk (22, 85 wk); 350-550 °F, 19, 70 wk (8, 62 wk); 550-700 °F, 4, 60 wk (2, 40 wk); 700-1070 °F, 62, 50 wk (62, 34 wk); and > 1070 °F, 0, > 110 wk (2, 70 wk). Tables, appendixes, and diagram, 7 p.

■ S. C. Lewis (Exxon Corp.), *Progress in Experimental Tumor Research* 26:68-84 (1983; 1987) (17 p.).

Source: Not available from API

34-30590

Influence of crude oil and dispersant on the ability of coho salmon to differentiate home water [(HW)] from non-home water [(NHW)]. A study on 334 male coho salmon *Oncorhynchus kisutch*, which were placed in Y-mazes at the University of Washington's School of Fisheries in Seattle and which were exposed to HW (holding pond water) vs. NHW (dechlorinated city water), HW plus 10 ppm dispersant oil (a mixture of Prudhoe Bay crude oil and chemical dispersant) vs. NHW, or HW plus soluble oil fraction (mainly C_1-C_8 hydrocarbons) vs. NHW, has shown a strong preference by the salmon for HW over NHW. The presence of crude oil or dispersed oil in the HW at the time the salmon were making a choice between HW and NHW masked the odor of HW, since positive rheotaxis was reduced, thus significantly altering their ability to make a choice. In the presence of oil and dispersant, there were choice ratios between HW and NHW that were not significantly different from random. If an oil spill occurred in an area where salmon depended on home odor cues for orientation prior to spawning in their natal stream, the salmon might stray to other streams, where the deposited eggs would have less chance of survival. Tables, graphs, appendixes, and 19 references, 66 p. See also Abstract No. 33-30601.

■ *API Publication* #4446 (Dec. 1986) (107 p.).

Source: API Publications Order Desk (Order No. I44460)

34-30632

Mutagenicity of API 84-02, heavy thermal cracked naphtha (CAS 64741-83-9) in a mouse lymphoma mutation assay. Final report. API 84-02, consisting mostly of unsaturated C_6-C_{12} unsaturated hydrocarbons and boiling at about 65-270 °C, was active in the mouse lymphoma cell system, since it induced significant increases in the mutant frequency at the thymidine kinase locus in vitro with and without metabolic activation by rat liver S9 homogenate. Under nonactivation conditions, the cells were exposed for four hours to 12.5-100 nL/mL concentrations of the test material in ethanol, and a wide range of toxicities was induced. A 3.9-fold increase in the mutant frequency was induced at the highest concentration, and relative growth was 16.2%. In similar tests in the presence of activation, treatments of 25-125 nL/mL induced significant, 1.9-fold to 6.7-fold, increases in the mutant frequency. The positive controls used were ethylmethane sulfonate for the nonactivation studies and 3-methyl cholanthrene for the activation studies. Tables, 5 p.

■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1987) (26 p.).

Source: API HESD Information Specialist

34-30633

Mutagenicity of API 83-20, light catalytic cracked naphtha (CAS 64741-55-5) in a mouse lymphoma mutation assay. Final report. API 83-20, consisting mostly of C_4-C_{11} hydrocarbons and boiling at about -20 to +190 °C, was inactive in the mouse lymphoma cell system, since it induced no reliable increases in the mutation frequency at the thymidine kinase locus in vitro with and without metabolic activation by rat liver S9 homogenate. Under nonactivation conditions, the cells were exposed for four hours to 50-250 nL/mL concentrations of the test material in ethanol, and nondetectable to moderate toxicities were induced. The test article induced a sharp toxicity curve, and high toxicities could not be assayed. No significant increases in the mutant frequency were induced by any of the assayed treatments. In similar tests in the presence of activation, treatments of 75-400 nL/mL were assayed for mutant induction, and a wide range of toxicities was induced. Sporadic increases in the mutant frequency were induced, but they were borderline and occurred at very high toxicities where less than 10% relative growth was observed. Tables, 5 p.

■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1987) (27 p.).

Source: API HESD Information Specialist

34-30634

Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats, API 83-09, straight-run kerosine (CAS 8008-20-6). Final report. When a group of 5 male and 5 female Sprague-Dawley rats was exposed to an atmosphere containing a mean analytical concentration of 5.28 ± 0.42 mg/L API 83-09 vapor during a single, four-hour exposure, all animals survived the study. Decreased activity was exhibited during exposure. Physical examination results were considered toxicologically unremarkable for the two-week postexposure period. Except for one male which exhibited a transient loss of body weight, the body weight results were unremarkable. Gross post mortem and histopathological results, only on the lungs, were unremarkable. No macroscopic lesions were observed on any animal. API 83-09 consists of mostly C_9-C_{16} hydrocarbons boiling at about 150-290 °C. Diagram and tables, 4 p.

■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1987) (18 p.).

Source: API HESD Information Specialist

34-30635

Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats, API 83-11, straight-run middle distillate (CAS 64741-44-2). Final report. When 5 male and 5 female young adult rats were exposed once for four hours to an actual aerosol concentration of 5.39 mg/L API 83-11, consisting mainly of C_{11}-C_{20} hydrocarbons and boiling at 205-345 °C, all ten animals died one to two days post exposure. In a second experiment, five additional groups of 5 male and 5 female rats each were exposed once to time-weighted-average aerosol concentrations of 0.01 (air-only control), 1.05, 1.60, 2.25, and 3.22 mg/L. No animals died at the control and two lower test article concentrations, but 9 of 10 animals receiving the two higher concentrations died within two days post exposure. Following exposure, individual and mean body weight gains were depressed in dose-related fashion. Histopathological examination of the lung tissues yielded dose-related acute morphological changes consistent with hydrocarbon toxicity. Based on the resultant mortality, the LC_{50} values were 1.72 mg/L for male rats, 1.82 mg/L for female rats, and 1.78 mg/L for the combined sexes. Tables and appendix, 45 p.

■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1987) (62 p.).

Source: API HESD Information Specialist

34-30636

Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats, API 83-19, light alkylate naphtha (CAS 64741-66-8). Final report. When a group of five male and five female Sprague--Dawley rats was exposed to an atmosphere containing a mean analytical concentration of 5.04 ± 0.74 mg/L API 83-19 vapor during a single, four-hour test, all animals survived the study, although languid behavior and a hunched appearance were exhibited during exposure. Physical examination results were considered toxicologically unremarkable for the two-week post exposure period. Female body weights were decreased at test day 15 compared with test day 8, a result attributed to prenecropsy fasting. Gross postmorten and histopathological results, on the lungs only, were considered unremarkable. API 83-19 consists mainly of C_7-C_{10} branched chain hydrocarbons and boils at about 90-160 °C. Diagram and tables, 4 p.

■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1987) (18 p.).
Source: API HESD Information Specialist

34-30857
The absence of hydrocarbon-induced nephropathy [HIN] in rats exposed subchronically to volatile hydrocarbon mixtures pertinent to gasoline suggests that exposures to gasoline vapors under occupational or consumer settings would not present a nephrotoxicity hazard, particularly since the male rat appears to be highly sensitive, if not uniquely predisposed, to HIN. In the studies, groups of male and female F-344 rats were exposed for 6 hr/day, 5 days/week, for 13 weeks to 4500 and 1000 ppm target concentrations of mixtures of 50:50 wt % n-butane/n-pentane and isobutane/isopentane and to 5200 and 1200 ppm target concentrations of an unleaded gasoline distillation cut boiling below 145 °F. The butanes/pentanes mixtures were chosen because the four hydrocarbons are the most prevalent gasoline vapor components met under typical occupational exposures. The rats were not significantly affected by the exposures, and there was no evidence of HIN in either sex at the termination of each study. At the interim 28 day sacrifice period for both butane/pentane mixtures, mild transient treatment-related, but not exposure-related kidney effects were observed in the male rats; these effects were not observed in rats exposed to the gasoline distillation fraction. Tables and 17 references.
■ C. Aranyi (IIT Res. Inst.); C. A. Halder (Amoco Corp.); C. E. Holdsworth (API); B. Y. Cockrell (Exper. Pathol. Lab. Inc.); W. J. O'Shea, *Toxicology and Industrial Health* 2(1):85-98 (1986; 1987).
Source: Not available from API

34-30858
Morphological transformation of BALB/3T3 mouse embryo cells. API PS-8-76D5 ARO. Final report. API PS-8-76D5-ARO [aromatic subfraction of the 700-1070 °F distillate fraction of crude oil D] was negative in the BALB/3T3 cell transformation assay when tested in the presence and absence of activation by an Aroclor-induced rat liver S-9 reaction mixture. The assay was conducted with a three day exposure in the nonactivated study at 1000, 800, 600, and 400 µg/mL dose levels of the test material, and with a four hour exposure in the presence of 100 µL/mL of S-9 treatment medium at 2000, 1000, 500, and 250 µg/mL dose levels of the test material. Survival at the highest dose tested was 98% in the nonactivated study and 90% in the S-9-activated study. No significant increases in transformation frequency were observed in either the nonactivated or activated study, although toxic levels could not be reached. The evaluation of transforming potential cannot be considered definitive because of the lack of interaction of the test article and the BALB/3T3 cells as evidenced by insolubility. Tables and appendix, 15 p.
■ *API Health Environ. Sci. Dep. Rep.* (Mar. 1987) (29 p.).
Source: API HESD Information Specialist

34-30859
Morphological transformation of BALB/3T3 mouse embryo cells. API PS-8-76C2. Final report. API PS-8-76C2 was negative in the BALB/3T3 cell transformation assay when tested in the absence and presence of activation by an Aroclor-induced rat liver S-9 reaction mixture. The assay was conducted with a three day exposure at 1, 0.1, 0.01, and 0.001 µL/mL dose levels of the test material in the nonactivated study, and with a four hour exposure at 3, 1.5, 0.8, and 0.4 µL/mL dose levels in the presence of 100 µL/mL S-9 treatment medium. Survival at the highest dose tested was 81% in the nonactivated study and 88% in the S-9 activated study. No significant increases in transformation frequency were observed in the nonactivated or the S-9-activated study, although toxic levels could not be reached. Tables and appendix, 17 p.
■ *API Health Environ. Sci. Dep. Rep.* (Mar. 1987) (31 p.).
Source: API HESD Information Specialist

34-32600
A two-year inhalation study of petroleum coke in [150 male and 150 female Sprague-Dawley] rats and [four male and four female Cynomolgus] monkeys, which were exposed to 0, 10.2, and 30.7 mg/cu m of micronized delayed-process petroleum coke dust for 6 hr/day, 5 days/wk, has shown that both species suffered from exposure-related pulmonary effects. Both species showed increased absolute and relative lung weights at terminal sacrifice, which, in the rats, were due to pulmonary deposition of the coke dust (as shown by gray-to-black discoloration of the lungs) and concurrent chronic inflammation and focal areas of fibrosis, bronchiolization, sclerosis, squamous alveolar metaplasia, and keratin cyst formation. Coke deposits and pulmonary lymph nodes attributed only to phagocytosis by pulmonary macrophages, but no inflammatory or metaplastic changes, were observed in monkey lungs. There were no spontaneous deaths among the exposed monkeys, no difference in the mortality rate between the treated and control rats, and no significant increases in chromosomal aberrations in the treated rats after 5 days, 12 mo, and 22 mo of exposure. Tables, graphs, photomicrographs, and 18 references. See also Abstract No. 30-31598 and 30-32002.
■ D. R. Klonne (Int. Res. Dev. Corp.); J. M. Burns (Hazleton Lab. Am.); C. A. Halder (Standard Oil Co. Indiana); C. E. Holdsworth (API); C. E. Ulrich, *American Journal of Industrial Medicine* 11:375-89 (4/8/87).
Source: Not available from API

34-32643
Mutagenicity of API 81-10, hydrodesulfurized middle distillate (CAS 64742-80-9) (coded as API 86-10) in a mouse lymphoma mutation assay. Final report. In-vitro treatments of the mouse lymphoma cell line L5178Y with API 86-10, which consists predominantly of C_{11}-C_{25} hydrocarbons boiling at ~ 205-400 °C, induced significant increases in the mutant frequency at the thymidine kinase locus only in the presence of rat liver S9 metabolic activation, and 86-10 is thus considered mutagenic under such conditions. The cells were exposed to API 86-10 in ethanol for 4 hr with and without activation, and the test material appeared insoluble above 125 nL/mL. In the presence of activation, 75-400 nL/mL of API 86-10 induced a wide range of toxicities, with a trend toward higher mutant frequencies at higher concentrations. Without activation, API 86-10 induced nondetectable-to-very-high toxicities at 25-600 nL/mL without inducing significant increases above the background mutant frequency. Tables, 4 p.
■ *API Health Environ. Sci. Dep. Rep.* (Apr. 1987) (36 p.).
Source: API HESD Information Specialist

34-32644
Mutagenicity of API 81-10 ARO, aromatic fraction of hydrodesulfurized middle distillate (CAS 64742-80-9) (coded as API 86-10 ARO) in a mouse lymphoma mutation assay. Final report. In-vitro treatments of the mouse lymphoma cell line L5178Y with API 86-10 ARO, consisting predominantly of C_{11}-C_{25} hydrocarbons boiling at ~ 205-400 °C, showed that the test material was nonmutagenic with or without the presence of rat liver S9 metabolic activation, since no significant increases in the mutant frequency were induced at the thymidine kinase locus. The cells were exposed to API 86-10 ARO in ethanol for 4 hr in the presence or absence of activation and the test material appeared insoluble above 60 nL/mL. Under activation and nonactivation conditions, 10-40 nL/mL of API 86-10 ARO induced a wide range of toxicities without inducing significant increases above the background mutant frequencies. Tables, 4 p.
■ *API Health Environ. Sci. Dep. Rep.* (Apr. 1987) (36 p.).
Source: API HESD Information Specialist

34-32645
Mutagenicity of API 81-10 SAT, saturated fraction of hydrodesulfurized middle distillate (CAS 64742-80-9) (coded as API 86-10 SAT) in a mouse lymphoma mutation assay. Final report. In-vitro treatments of the mouse lymphoma cell line L5178Y with API 861-10 SAT, consisting predominantly of C_{11}-C_{25} hydrocarbons boiling at ~ 205-400 °C, showed that the test material was nonmutagenic in the presence or absence of rat liver S9 metabolic activation, since no significant increases in the mutant frequency were induced at the thymidine kinase locus. The cells were exposed to API 86-10 SAT in ethanol for 4 hr. API 86-10 SAT induced nondetectable-to-moderate toxicities at 500-2500 nL/mL in the absence of activation without inducing significant increases above the background mutant frequency. Higher concentrations of API 86-10 SAT were not analyzed, since the test material was insoluble at and above

2500 nL/mL. With activation, 375-2000 nL/mL of API 86-10 SAT induced a wide range of toxicities without significant increases in the background mutant frequency. Tables, 5 p.
■ *API Health Environ. Sci. Dep. Rep.* (Apr. 1987) (37 p.).
Source: API HESD Information Specialist

34-32774
Teratogenicity testing in vitro: Status of validation studies. Fourteen alternative teratogenicity test systems for which some kind of validation study had been performed were identified in a literature search. The validation performance of 13 of these systems, which classify a test chemical as positive or negative, was analyzed for three groups of compounds. One group gives performance data as reported in the original publication. A second group gives data for 59 teratogens (BaP, ethanol, etc.) and 22 non-teratogens (e.g., phenol). A third group gives data for 47 chemicals which were selected by a panel of teratologists and which are all developmental hazards. The analysis shows the inadequacy of all the current validation studies, i.e., the dichotomous classification approach, the potency correlation approach, and an approach that compares developmental hazard estimates from in vivo and in vitro tests. The major improvements suggested are better selection of test compounds, and standardized definitions of their teratogenic potential. Tables and 25 references.
■ N. A. Brown (MRC Exp. Embryology Teratology Unit, Med. Res. Counc. Lab., U.K.), *Mech. Models Toxicology Arch. Toxicol., Suppl. 11*, 105-14 (1987) (10 p.).
Source: Not available from API

34-32775
Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 83-12: Hydrotreated light naphthenic distillate (CAS 64742-53-6). All five male and all five female rats tested died after whole-body exposure to 5.05 mg/L of API 83-12 (comprising C_{15}-C_{30} hydrocarbons) as an aerosol for 4 hr in the first phase of this study. In the second phase, five groups of five male and five female rats each were exposed to air only or to 1.04, 2.37, 1.51, or 3.49 mg/L of API 83-12 for 4 hr. There were no deaths at the control and at the 1.51 mg/L levels, but all rats died at the highest dose. The deaths were considered related to exposure concentration. The LC_{50} value was 2.18 mg/L each for the combined sexes, males alone, and females alone, with respective 95% confidence limits of 1.80-2.55, 1.64-2.71, and 1.64-2.71 mg/L. Pharmacotoxic signs, gross necropsy findings, and acute histopathological changes in the lung were all considered treatment-related and were most severe in rats that died two to four days after exposure. Few morphological abnormalities were seen in the lungs of 14-day survivors. Tables and appendixes, 48 p.
■ *API Health Environ. Sci. Dep. Rep.* (Aug. 1987) (61 p.).
Source: API HESD Information Specialist

34-32776
Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 83-18: Heavy catalytic cracked naphtha (CAS 64741-54-4). Five male and five female Sprague-Dawley rats were exposed once for 4 hr to 5.74 ± 0.76 mg/L of API 83-18, which consists of C_6-C_{12} hydrocarbons boiling at ~ 65-230 °C. All the rats survived and showed languid behavior 2 hr after exposure and when they were removed from the chamber. Squinted eyes were also seen upon removal from the chamber. Other observations were crust around the eyes, red crust around the nose, wet hair, and rough hair coat 1 hr after exposure; and crust around the nose and/or eye, and alopecia during the first, and in some cases, second post-exposure week. In general, body weight values, gross postmortem for all tissues, and histopathological results for the lungs were considered unremarkable. Tables and diagram, 7 p.
■ *API Health Environ. Sci. Dep. Rep.* (July 1987) (19 p.).
Source: API HESD Information Specialist

34-32777
Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 83-20: Light catalytic cracked naphtha (CAS 64741-55-55). All five male and five female Sprague-Dawley rats survived after a 4 hr exposure to 5.28 ± 0.55 mg/L of API 83-20, which consists of C_4-C_{11} hydrocarbons boiling at ~ - 20 to + 190 °C. Most of the rats showed languid behavior and squinted eyes by the second hour of exposure. All showed polypnea when removed from the chamber and 1 hr after exposure. Other observations include rhinorrhea in two rats on day-2 test; unremarkable body weight values; no apparent treatment--related microscopic changes in the lungs, or neoplastic changes, in rats exposed to the highest dose; and no deaths before the scheduled termination of the rats. Tables and diagram, 6 p.
■ *API Health Environ. Sci. Dep. Rep.* (July 1987) (17 p.).
Source: API HESD Information Specialist

34-32778
Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 84-02: Heavy thermal cracked naphtha (CAS 64741-83-9). All five male and all five female Sprague-Dawley rats survived a single 4 hr exposure to a vapor atmosphere containing 5.24 ± 0.42 mg/L of API 84-02, which consists mainly of C_6-C_{12} unsaturated hydrocarbons boiling at ~ 65-270 °C. No relationship was established between the treatment and such findings as languid behavior, squinted eyes, respiratory distress, rhinorrhea, salivation, urine-stained fur, and body weight loss among some rats at different post-exposure periods. No definitive microscopic observations were considered clearly related to the treatment. Although lung sections were examined microscopically, no concurrent ones were available for examination and comparison. Tables and diagram, 6 p.
■ *API Health Environ. Sci. Dep. Rep.* (Aug. 1987) (17 p.).
Source: API HESD Information Specialist

34-32779
Acute inhalation toxicity evaluation of a petroleum-derived hydrocarbon in rats. API 85-01: Stoddard solvent (CAS 8052-41-3). Five male and five female Sprague-Dawley rats were exposed for 4 hr to a vapor atmosphere containing 5.50 ± 0.43 mg/L of API 85-01, a refined petroleum distillate boiling at ~ 300-400 °F. The rats, all of which survived, showed languid behavior and squinted eyes during exposure, polypnea upon removal from the chamber, sporadic incidences of rhinorrhea, unremarkable body weights on day-15 test, no apparent treatment-related microscopic changes in the lungs, and no neoplastic changes. On day-8 test, three females showed less than or about the same body weights as their pretest body weights, but no relationship to the treatment was established. Tables and diagram, 6 p.
■ *API Health Environ. Sci. Dep. Rep.* (Aug. 1987) (17 p.).
Source: API HESD Information Specialist

34-32865
Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of [11] refinery streams in C3H/HeJ mice. Twelve-month toxicity evaluation. A 12 mo chronic toxicity study of 325 male and 325 female C3H/HeJ mice, whose clipped backs were treated twice a week with 50 µL each of the 11 refinery streams, showed that all the test materials were locally dermatotoxic. Dermal neoplasms were caused by API 84-01 (light paraffinic distillate), API 83-07 (light catalytic cracked distillate), API 83-09 (straight-run kerosine), API 83-11 (straight-run middle distillate), and API 83-16 (light paraffinic distillate solvent extract). API 83-19 (light alkylate naphtha) caused one papilloma. Survivability was reduced by API 83-08 (light catalytic cracked distillate) and API 83-16. API 84-01 and API 83-16 caused liver toxicity (hyperplastic nodules and centrilobular hypertrophy). API 83-07 and API 83-16 caused prominent increases in liver weights and liver-to-body weight ratios compared with those in the untreated and toluene controls. Most of the deaths and systemic toxic responses were not considered treatment-related. The other test materials were API 83-06 (heavy catalytic reformed naphtha), API 83-12 (hydrotreated light naphthenic distillate), API 83-18 (heavy catalytic cracked naphtha), and API 84-02 (heavy thermal cracked naphtha). Tables, graphs, appendix, and addendum, 578 p.
■ *API Health Environ. Sci. Dep. Rep.* 1-2 (July 1987) (595 p.).
Source: API HESD Information Specialist

34-32923
Further development and application of an improved population exposure model. SYSAPP-86/061. A Systems Applications Inc. study

for the API program of reviewing ambient standard setting by the EPA further extended the development of, and applied, EPA NAAQS Exposure Model (NEM) enhancements in order to improve NEMs capability for human exposure modeling. The tasks included a critical review of, and recommendations on, some limited aspects of the dose-response relationships that may be used to quantify adverse human health effects from the exposure estimates of NEM, and the development of a Personal Air Quality Model (PAQM), which is a personal exposure modeling enhancement of NEM that provides for direct calculation of dose as a function of activity pattern, including any indoor protection that might be present. The development of the PAQM involved one case study of the long-term exposure of a population to refuse recovery facility particulates and another of the exposure of a population to high episodic ozone concentrations over a 24 hr period. Diagrams, graphs, tables, 18 references, and appendixes, 149 p.
■ B. S. Austin; G. E. Anderson; B. R. Weir; C. Seigneur (Syst. Appl. Inc.), *API Publication* (4/25/86) (204 p.).
Source: API HESD Information Specialist

34-32982
Effects of crude oil and chemically dispersed oil on chemoreception and homing in Pacific salmon. A total of 215 2-yr-old, male chinook salmon that had returned to the University of Washington School of Fisheries' home pond in Seattle were placed in tanks containing filtered Lake Washington water and 910 mL of undispersed Prudhoe Bay crude (PBC) oil (702 ppm if uniformly mixed; C_1-C_{10} dissolved hydrocarbons, 0.25 ppm-hr), or 137 mL of PBC mixed with 13.7 mL of a freshwater chemical dispersant (105 ppm of oil and 10.5 ppm of dispersant if uniformly mixed; C_1-C_{10} hydrocarbons, 1.5 ppm-hr), or 13.7 mL of freshwater chemical dispersant (10.5 ppm), or untreated water alone (control). The salmon were released ~ 7 km downstream from the home pond after 1 hr of exposure. The returns totaled 154 (71.6%), of which 74 (48%) were within 2 days of release. There were no statistical differences in the proportion of returns among the treatment and control groups, indicating that exposure to PBC or chemically dispersed (CD) PBC did not diminish the success or speed of return. A longevity test on 57 similarly exposed chinook salmon showed that those exposed to CDPBC died a few days earlier than the controls, but their homing ability was unaffected. Tables, graphs, diagram, block diagram, map, and 40 references. See also Abstract No. 34-30590.
■ R. E. Nakatani; E. L. Brannon; A. E. Nevissi; R. P. Whitman; S. P. Kaluzny; T. P. Quinn (Univ. Wash. Fisheries Res. Inst.), *API Publication* #4445 (June 1987) (64 p.).
Source: API Publications Order Desk (Order No. I44450)

34-33031
[A review of] short-term toxicity tests for [atmospheric and aqueous] environmental carcinogens and mutagens covers current theories of chemical mutagenesis and carcinogenesis; general factors to consider in the application of short-term bioassays, e.g., test end points, in-vivo and in-vitro testing, test system selection, and metabolic activation; environmental sample collection and preparation; the nine most commonly used tests, i.e., the Ames test, mammalian point mutation bioassay, chromosome aberration bioassay, sister chromatid exchange, mouse micronucleus bioassay, unscheduled DNA synthesis, mammalian cell transformation, mouse lung adenoma, mouse skin papilloma bioassay, and case studies, including the use of Chinese hamster ovary cells to examine the genotoxicity of diesel exhaust particles and spark ignition particles from three different fuels; tiered testing, including examples, factors in program design, and test interpretation; and the application of short-term bioassays in public health assessments and environmental regulations; and includes a glossary and list of acronyms. Diagrams, graph, tables, and 172 references.
■ *API Publication* #4462 (Sept. 1987) (147 p.).
Source: API Library

34-33036
Studies on the absorption of inhaled hydrocarbon [(HC)] vapors. Final report. Studies on male F344/N rats that were exposed one by one to 1-5000 ppm (10,000 ppm for 2,3-dimethylpentane) of 20 gasoline-related HC vapors for 80-100 min indicate that concentration has little effect on the fractional uptake, and that important differences in dose rates occur among these vapors when inhaled at similar concentrations. Dual-column GC showed that highly volatile and branched HCs are less well absorbed than less volatile and unbranched HCs, unsaturated compounds are better absorbed than saturated ones, and inhalation of some HCs is accompanied by a decrease in respiratory minute volume. The HC vapors studied had volatilities going from propylene, boiling at -47.7 °C, to 1,2,4-trimethylbenzene, boiling at +169.2 °C, and included alkenes, alkynes, linear and branched alkanes, cycloalkanes, and aromatic hydrocarbons such as benzene. Tables, diagrams, block diagrams, graphs, and 20 references, 153 p.
■ A. R. Dahl; E. G. Damon; J. L. Mauderly; S. J. Rothenberg; F. A. Seiler; R. O. McClellan (Inhalation Toxicol. Res. Inst., Lovelace Biomed. Environ. Res. Inst.), *API Health Environ. Sci. Dep. Rep.* (Oct. 1987) (182 p.).
Source: API HESD Information Specialist

34-33107
The L5178Y TK+/- mouse lymphoma mutagenesis assay with 2,2,4-trimethylpentane. Final report. Results from these assays indicate that, under the test conditions, the test material produced a negative response in the presence or absence of Aroclor-induced rat liver S9. The first assay was repeated because the nonactivated cultures were not in the 10-50% total growth range, and several critical S9-activated cultures were contaminated. The second assay was also repeated, since the nonactivated and activated cultures produced unacceptably high background mutant frequencies, and the S9-activated cultures were too toxic to clone. In the third assay, the nonactivated and activated cultures that were cloned were treated with 3.0-0.5 and with 2.5-0.5 µL/mL, respectively, and produced respective suspension growths of 18-62 and 40-99%. Two of the nonactivated cultures, but none of the activated cultures that were cloned, had mutant frequencies that were at least twice the mean mutant frequency of the solvent controls. The results for the nonactivated cultures, which produced total growths of 22 and 8%, were not considered significant because of low cloning efficiencies. No dose-dependent response was noted in any of the treated cultures. Tables, graphs, and diagram, 10 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1987) (53 p.).
Source: API HESD Information Specialist

34-33108
[This final report on] the L5178Y TK+/- mouse lymphoma mutagenesis assay with API 220-280 °F distillate fraction of unleaded gasoline showed that under the test conditions, the test material (MBA #T5318) produced a negative response in the presence or absence of Aroclor--induced rat liver S9 metabolic activation. Cultures that were treated with 0.047-0.008 µL/mL of the test material and cloned produced a total growth of 17-107%. The activated cultures that were treated with 1.0-0.34 µL/mL and cloned produced a total growth of 32-84%. Neither the activated nor the nonactivated cultures that were cloned showed mutant frequencies that were significantly greater than the mean mutant frequency of the controls, which were treated with 1.0 and 0.5 µL/mL of ethyl methanesulfonate or with 7.5 and 5.0 µL/mL of 7,12-dimethylbenz[a]anthracene. No dose-dependent response was noted in any of the treated cultures. Tables, graphs, diagram, and appendix, 29 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1987) (52 p.).
Source: API HESD Information Specialist

34-33109
The L5178Y TK+/- mouse lymphoma mutagenesis assay with 2,3-dimethylbutane. Final report. The results of these assays showed that under the test conditions, 2,3-dimethylbutane (MBA #T5317) produced a negative response in the presence or absence of Aroclor-induced rat liver S9 metabolic activation. In the first assay, the nonactivated cultures that were treated with the test material and cloned showed a 16-69% total growth. The test material showed a precipitous dose--response for toxicity in the presence of activation; the first and second assays for the activated cultures that were cloned were repeated because they did not produce a 10-50% total growth. In the third assay, the activated cultures that were treated with 10.9, 9.9, or 8.9 µL/mL of the

test material and cloned produced a total growth of 13-91%. None of the cultures that were treated and cloned showed mutant frequencies that were at least twice the mean mutant frequency of the solvent controls, and no dose-dependent response was noted. Tables, graphs, diagram, and appendix, 35 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1987) (59 p.).
Source: API HESD Information Specialist

34-33173
Thirteen-week subchronic inhalation toxicity study in rats with API 81-03: Light catalytic cracked naphtha (CAS 64741-55-5). A study on three groups of 20 male and 20 female Sprague-Dawley albino rats, which were exposed to vapors of 1510, 2610, or 4520 ppm of API 81-03 for 6 hr/day, 5 days/wk for 13 wk, and on a group of control rats exposed to clean air, showed that, at up to 2610 ppm, API 81-03 did not cause apparent toxic effects. The only treatment-related pharmacotoxic effect was a red tinged nasal discharge in 50% of the females and in 65% of the males exposed to 4520 ppm. The high-dose males showed depressed body weights and a typical hydrocarbon-induced nephropathy. The elevated kidney weights reflect a mild male kidney pathology and were also seen in males in the lower dose groups. Body weights of males in the lower dose groups and of females in all dose groups were similar to those of the controls. The treatment-related liver weight increases in some of the high-dose males and females are associated with a trace severity of cellular hypertrophy in 50% of the males and 25% of the females, and probably reflect a non-specific biochemical/physiologic response to API 81-03. Hematologic, serum biochemical, or urinalysis tests showed no treatment-related effects. Tables, graphs, and 23 references, 310 p.
■ *API Health Environ. Sci. Dep. Rep.* (Dec. 1987) (346 p.).
Source: API HESD Information Specialist

35-30687
Methyl tertiary butyl ether inhalation in rats: A single generation reproduction study. A study on four groups of 15 male and 30 female rats, which were exposed to 0, 300, 1300, and 3400 ppm of MTBE for 6 hr/day, 5 days/wk for 12 wk (males) and 3 wk (females) during premating, mating, and mating rest intervals, and during gestation and from days 5-21 of lactation for the females, indicated that MTBE has little adverse reproductive potential in rats under the test conditions. The only notable post-mortem finding was an increased incidence of dilated renal pelves in the low- and high-dose females. Pups from the low- and mid-dose groups had a slight, but not statistically significant, increase in dilated renal pelvis incidence than pups from the control and high-dose groups in both litter intervals (LIs). There were no treatment-related effects evident in male and female body and reproductive organ weights, or in male fertility, and no statistically significant difference in mating indexes between treated and control groups. Pregnancy rates were comparable among the control, low-, and high-dose females, and slightly lower than control, but not statistically significant, in the mid-dose group in the first litter interval and in all treated groups in the second litter interval (SLI), with no clear dose relationship evident in either LI. Tables and 12 references, 9 p.
■ R. W. Biles (Exxon Biomed. Sci. Inc./Exxon Res. Eng. Co.); R. E. Schroeder (Bio/dynamics Inc.); C. E. Holdsworth (API), *Toxicol. Ind. Health* 3(4):519-34 (1987; 1987) (16 p.).
Source: Not available from API

35-30932
Mutagenicity test on ASTM D-3734 Type I C_9 in the Ames *Salmonella* /microsome reverse mutation assay. Final report. Assays on C_9 aromatic naphtha dissolved in dimethyl sulfoxide on *S. typhimurium* strains TA-1535, TA-1538, TA-98, and TA-100 at eight doses of 0.0025-0.50 µL/plate (three plates per dose level) with or without Aroclor 1254-induced rat liver S9 metabolic activation, showed that the test material did not exhibit genetic activity and was not mutagenic to all the strains under the test conditions. There were no increased numbers of histidine-independent revertant colonies. The assays on strain TA-1537 were repeated because the strain used in the initial tests was contaminated. Compared with the negative or solvent controls, the positive control treatments with and without activation exhibited large increases in the revertant numbers with all the indicator strains, thus showing the effectiveness of the S9 activation system and the ability of the test system to detect known mutagens. Tables, 7 p.
■ D. R. Jagannath (Hazleton Lab. Am. Inc.), *API Health Environ. Sci. Dep. Rep.* (Oct. 1987) (30 p.).
Source: API HESD Information Specialist

35-30933
Mutagenicity test on ASTM D-3734 Type I C_9 in an in vitro cytogenetic assay measuring sister chromatid exchange [(SCE)] frequencies in Chinese hamster ovary (CHO) cells. Final report. C_9 aromatic naphtha dissolved in dimethyl sulfoxide (DMSO) and used at doses of 0.0667-2000 µg/mL in a half-log test series was considered negative for inducing SCEs in CHO cells with or without Aroclor 1254-induced rat liver S9 metabolic activation. Findings from the nonactivation (and activation, in parentheses, with all doses in micrograms per milliliter) tests are: complete toxicity in the first trial at 200, 667, and 2000 (66.7, 200, 667, and 2000); no discernible toxicity at 66.7 (20.0); no increase in SCE and no cell cycle delay at 2.0, 6.67, 20.0, and 66.7 (0.667, 2.0, 6.67, and 20.0); complete toxicity in the second trial at 90.1, 120, 150, and 200 (66.7); an unhealthy cell monolayer with a 38% reduction in monolayer confluence at 66.7 (66.7), and a reduction in visible mitotic cells, and cell cycle delay at 66.7; and no significant increase in SCE at 35.0, 50.1, and 66.7 (15.0, 20.0, 35.0, and 50.1). The negative controls were treated with DMSO; the positive controls were dosed with mitomycin C for the nonactivation test and with cyclophosphamide for the activation test. Tables, 4 p.
■ J. L. Ivett (Hazleton Lab. Am. Inc.), *API Health Environ. Sci. Dep. Rep.* (Oct. 1987) (24 p.).
Source: API HESD Information Specialist

35-30934
Mutagenicity test on ASTM D-3734 Type I C_9 in an in vitro cytogenetic assay measuring chromosomal aberration frequencies in Chinese hamster ovary (CHO) cells. Final report. This 10 hr assay, which is based on toxicity and cell cycle kinetics data from a concurrent in vitro sister chromatid exchange assay, indicates that C_9 aromatic naphtha dissolved in dimethyl sulfoxide is negative for inducing chromosomal aberrations in CHO cells from each duplicate culture with or without Aroclor 1254-induced rat liver S9 metabolic activation. The first trials were repeated because there was no toxicity at up to 90.0 (all doses are in micrograms per milliliter) without activation and at up to 70.0 with activation. Compared with the negative and solvent controls, and with positive controls dosed with mitomycin C or cyclophosphamide in the second trials, the cultures treated with the test material showed no significant increase in chromosomal aberrant cells at up to 60.1 without activation, and at up to 120 with activation. Complete toxicity occurred at 90.2, 120.0, and 150.0 without activation, with 14% reduction in monolayer confluence at 60.1, and at 60.1, 100.0, and 120.0 with activation, with a 14% reduction in relative monolayer confluence at 40.1 and a nearly total cellular toxicity at 60.1. Tables and appendixes, 16 p. See also Abstract No. 35-30933.
■ J. L. Ivett (Hazleton Lab. Am. Inc.), *API Health Environ. Sci. Dep. Rep.* (Oct. 1987) (37 p.).
Source: API HESD Information Specialist

35-30935
Mutagenicity test on ASTM D-3734 Type I C_9 in the CHO/HGPRT [(Chinese hamster ovary/hypoxanthine-guanine phosphoribosyl transferase)] forward mutation suspension assay. Final Report. This in vitro assay indicates that C_9 aromatic naphtha dissolved in dimethyl sulfoxide is negative for inducing forward mutation at the HGPRT locus in CHO cells with or without Aroclor 1254-induced rat liver S9 metabolic activation. There were no observed dose- or toxicity-related increases in mutant frequency, and no statistically significantly higher than vehicle control mutant frequencies. A preliminary rangefinding test showed that C_9 was nontoxic from 0.001 to 0.05 (all doses are in microliters per milliliter) with and without activation, with respective relative survival of 38.1 and 1.0% at 0.1 and total cell killing at 0.2 and higher. Without activation (and with activation, in parentheses), C_9 was nontoxic from 0.01 to 0.06 (0.02-0.06), but was increasingly toxic at 0.07

and 0.08 (0.08), with the higher dose giving a relative clonal survival at 2.9% (1.6%) and a relative population growth of 10.7% (4.0%). Tests at 0.1 and higher were terminated, since such doses were completely toxic. Positive controls treated with 3-methylcholanthrene with activation or with methyl methanesulfonate without activation induced large, significant increases in mutant frequencies. Tables, graph, and appendixes, 7 p.
■ R. R. Young (Hazleton Lab. Am. Inc.), *API Health Environ. Sci. Dep. Rep.* (Oct. 1987) (34 p.).
Source: API HESD Information Specialist

35-30936
Evaluation of C_9 aromatic hydrocarbons [(C_9AHC)] for mutagenic potential...Bone marrow cytogenetics test in rats. Final report. A study on three groups of 15 male and 15 female Sprague-Dawley rats, which were exposed to 153, 471, or 1540 ppm of C_9AHC vapors for 6 hr/day for five consecutive days, indicates that the test material is non-mutagenic, since it did not induce chromatid or chromosome aberrations at any exposure level compared with the clean air-exposed negative controls. Post-exposure observations showed no signs of toxicity in any of the rats, and statistically significantly lower (11-12%) than negative control absolute body weights for the males, but less so for the females, in the 1540 ppm group at the 24 and 48 hr intervals. Combined data from necropsy on five males and five females from each group support the non-mutagenicity conclusion. Eight of a total of ten positive controls, which were injected intraperitoneally with 40 mg/kg of cyclophosphamide, showed chromatid and chromosome aberrations and chromosome aberrations in excess of the negative controls. Tables, diagram, and appendixes, 26 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1988) (52 p.).
Source: API HESD Information Specialist

35-30937
Range-finding inhalation toxicity study in mice with C_9 aromatic hydrocarbons [(C_9AHC)]. Final report. A study on six groups of five mated female Charles River CD-1 mice each, which were exposed to filtered air only, and to 100, 250, 500, 1000, and 1500 ppm of C_9AHC by whole body inhalation for 6 hr/day on days 6-15 of gestation, showed that C_9AHC was maternally toxic at 1500 ppm and less so at 1000 ppm. Uterine examination showed a slight reduction in mean fetal body weights, and thus, probable fetotoxicity at 500, 1000, and 1500 ppm, and no evident signs of treatment-related abnormal fetal development. Two 1500 ppm dams were sacrificed in extremis on day 6 of gestation when labored breathing, laterally rotated hindlimbs, and other clinical observations were noted, although no gross lesions were seen at necropsy. Increased lacrimation in mice at 1000 and 1500 ppm was considered treatment-related. Cystic ovarian bursae in one control and in a 250 ppm mouse were the only necropsy findings noted. The developmental toxicity study on mice will thus be conducted at 100, 500, and 1500 ppm. Tables, diagram, and appendixes, 34 p.
■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1988) (64 p.).
Source: API HESD Information Specialist

35-31189
Indoor vs. outdoor ambient benzene concentrations: Results of the EPA Total Exposure Assessment [Methodology] (TEAM) study. A simulation model was used to analyze the EPA TEAM findings from monitoring the personal air of several participants in Elizabeth and Bayonne, N.J; Los Angeles, Calif. (two measurement segments); and Antioch and Pittsburg, northern Calif.; indoor air; and outdoor air in the participants' backyards over two consecutive 12 hr periods for the presence of 20 compounds, including benzene, at various times during 1981, 1982, and 1984. There was a direct and consistent relationship between the use of tobacco products and the levels of ambient benzene found in the indoor environment. A thermal inversion that covered the Los Angeles area during the Jan.-Feb. 1984 measurement segment also significantly increased benzene exposure. The Antioch and Pittsburg data showed very low benzene concentrations because of high coastal winds. Both EPA and API data showed minimal direct correlation between indoor and outdoor benzene levels. A reduction in the ambient benzene exposure levels would reduce the TWA personal exposure levels, but a cost-benefit analysis would be needed to justify this approach. Tables and appendix, 5 p.
■ C. J. Sample, *API Health Environ. Sci. Dep. Rep.* (Nov. 1986) (18 p.).
Source: API HESD Information Specialist

35-31365
The L5178Y TK +/- mouse lymphoma mutagenesis assay with octane. Final report. This study showed that, in general, octane produced a negative response in the presence or absence of Aroclor-induced rat liver S9 metabolic activation under the test conditions. In the first assay, the nonactivated cultures that were cloned and treated with 2.2-0.7 µL/mL of octane solubilized in ethylene glycol dimethyl ether had total growths (TGs) of 0-99% of the controls. Six of the cultures showed mutant frequencies (MFs) that were at least twice the mean MF of the solvent controls and that were not considered biologically significant, since the TGs were 0-3% of the controls. The activated cultures that were cloned and treated with 1.5-0.3 µL/mL of octane had TGs of 0-132% of the solvent controls, and did not show MFs that were significantly greater than the mean MF of the solvent controls or that were dose-dependent. The activated portion of the assay was repeated to generate cultures with 10-50% TGs, which were achieved in the seventh assay using 1.3-1.1 µL/mL of octane. These cultures gave TGs of 6-82% of the controls, with one culture showing a MF that was significantly above the mean of the solvent controls. This one culture's response was not considered biologically significant because of unacceptably high toxicity (8% TG) and no dose-dependence. Assays 2-6 did not meet acceptance criteria or were lost due to technical problems. Tables, graphs, and diagram, 13 p.
■ *API Health Environ. Sci. Dep. Rep.* (Jan. 1988) (58 p.).
Source: API HESD Information Specialist

35-31367
Inhalation reproduction range-finding study in mated rats with C_9 aromatic hydrocarbons. Final report. Whole-body inhalation exposure of five groups of five male and five female Charles River COBS and CD rats each to 100, 250, 500, 1000, and 1500 ppm of the test material for 5 days/wk, 6 hr/day, 14 days prior to mating until day of sacrifice (the females were not exposed from day 21 of gestation to day 4 of lactation), showed that C_9 induced slight maternal toxicity and reductions in paternal body weights at 1500 ppm, as indicated by body weight losses among females during lactation and reduced food consumption among both sexes. Reduced pup body weights on lactation days 0 and 4 suggest that C_9 may have inhibited the development of male and female pups of the high-dose rats. Similar effects were not noted among rats and their pups in the other dose groups and the filtered air-exposed control group. The only abnormal postmortem findings were a focus on the stomach of one high-dose female and a focus on the liver of one control female. The exposure levels chosen for a two-generation reproduction study were 100, 500, and 1500 ppm. Tables, diagram, and appendixes, 52 p.
■ *API Health Environ. Sci. Dep. Rep.* (Apr. 1988) (79 p.).
Source: API HESD Information Specialist

35-31368
Inhalation developmental toxicity study in mice with C_9 aromatic hydrocarbons. Final report. A study on four groups of 30 mated Charles River CD-1 female mice each, which were exposed to filtered air (control) and to 100, 500, and 1500 ppm of the test material by whole-body inhalation once daily for 6 hr from day 6 to day 15 of gestation, showed that C_9 induced fetal developmental toxicity at 500 and 1500 ppm, and maternal toxicity at 1500 ppm. Compared with the controls, the treated mice showed a significant increase in mean post-implantation loss at 1500 ppm, and their fetuses showed significant decreases in mean body weights at 500 and 1500 ppm, and increased incidence of unossified sternebrae and reduced skull ossification at 1500 ppm. Maternal toxicity was indicated by a near 50% mortality, reduced food intake, and inhibited body weight gain during exposure and over-all gestation, significant decreases in mean hematocrit and mean corpuscular volume, a significant increase in mean corpuscular hemoglobin concentration, and an increase in the incidence of cleft palate that is indirectly related to treatment due to maternal stress. The no observable effect level of C_9 was 100 ppm under the test conditions. Tables, diagram, 11 references, and appendixes, 67 p.

■ *API Health Environ. Sci. Dep. Rep.* (Apr. 1988) (101 p.).
Source: API HESD Information Specialist

35-31936
Evaluation of API 86-02 (solvent refined coal distillate) in the in-vitro assay for chemical teratogenic potential: Inhibition of proteoglycan synthesis. Final report. Evaluation of API 86-02 in an in-vitro system which detected teratogenicity potential through inhibition of DNA or proteoglycan synthesis in mouse limb bud cultures showed that it was inactive in this assay at 0.0001-5.0 µL/mL of the test material (in acetone solution per milliliter of culture medium). There was no significant response in the DNA precursor/proteoglycan precursor uptake ratio. Tables.
■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1988) (15 p.).
Source: API HESD Information Specialist

35-31937
Evaluation of benzo(a)pyrene in the in-vitro assay for chemical teratogenic potential: Inhibition of proteoglycan synthesis. Final report. Evaluation of BaP in an in-vitro system which detected teratogenicity potential through inhibition of DNA or proteoglycan synthesis in mouse limb bud cultures showed that BaP was active in this assay, producing significant inhibition of DNA synthesis relative to proteoglycan synthesis. BaP in acetone was tested at 0.00001-1.0 mg/mL of culture medium; it showed a toxicity of 50-80% at 1.0 mg/mL. BaP was not highly toxic at 0.1 mg/mL, and induced a 46% and 32% decrease in DNA synthesis and proteoglycan synthesis at this concentration. There was a lower synthesis suppression at lower benzo(a)pyrene dosages. Tables.
■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1988) (17 p.).
Source: API HESD Information Specialist

35-31938
Evaluation of API 81-15 (catalytic cracked clarified oil) in the in-vitro assay for chemical teratogenic potential: Inhibition of proteoglycan synthesis. Final report. An evaluation of API 81-15 in an in-vitro system which detected teratogenicity potential through inhibition of DNA or proteoglycan synthesis in mouse limb bud cultures showed that the test material was inactive up to a maximum testable concentration of 5 µL/mL. API 81-15 in acetone was tested at 0.0001-5.0 µL/mL in the culture medium. Proteoglycan synthesis and DNA synthesis inhibitions were 87 and 84%, respectively, and toxicity, were observed at 0.1 µL/mL. Inhibitions at lower doses were not statistically significant. Tables, 5 p.
■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1988) (17 p.).
Source: API HESD Information Specialist

35-31939
Evaluation of toluene in the in-vitro assay for chemical teratogenic potential: Inhibition of proteoglycan synthesis. Final report. An evaluation of toluene in an in-vitro system which detected teratogenicity potential through inhibition of DNA or proteoglycan synthesis in mouse limb bud cultures showed that toluene was inactive up to a maximum testable concentration of 5 µL/mL. Toluene in acetone was tested at 0.0001-5.0 µL/mL of culture medium, which included toxic and nontoxic levels. Tables, 6 p.
■ *API Health Environ. Sci. Dep. Rep.* (Feb. 1988) (19 p.).
Source: API HESD Information Specialist

35-32013
An in-vivo sister chromatid exchange [(SCE)] assay of API PS-8-76D5-ARO, aromatic subtraction of 700-1070 °F boiling range fraction of a crude oil, Tests on five groups of five male and five female B6C3F1 mice, each of which received a single intraperitoneal injection of the test material at 10 mL/kg of body weight, distributed at 0.5, 1.7, and 5.0 g/kg, indicated that API PS-8-76D5-ARO is negative under the test conditions. The frequency of SCEs per cell per mouse was not significantly increased for any of the groups compared with the negative control values. A repeat SCE assay yielded the same results. Tables and appendixes, 15 p.
■ *API Health Environ. Sci. Dep. Rep.* (Sept. 1987) (32 p.).
Source: API HESD Information Specialist

35-32077
The generation and analytical methods development work for the C_9 aromatic hydrocarbons program. Final report. Preparatory to a multi-tasked bioassay program that will include a two-generation reproduction study on relatively large numbers of laboratory animals, a test mixture generation system was developed that was capable of generating about 2400 ppm and suitable for use with 16 cu m exposure chambers. The results of previous studies indicated that the high exposure level to the C_9 aromatic naphtha test material would be at least 1500 ppm; exposure atmospheres would have to be monitored for total C_9 concentration to control exposure levels. Since the maximum obtainable vapor concentration with no aerosol contamination decreased with decreasing temperature, operation at 20 °C would permit the desired high concentration level to be 1500 ppm, but would cause aerosol development at 1725 ppm. GC data for C_9 composition (i.e., o-xylene, cumene, n-propylbenzene, 2-, 3-, and 4-ethyltoluene, and 1,3,5-, 1,2,4-, and 1,2,3-trimethylbenzene) at 250-1500 ppm chamber atmospheres showed that the generated vapor phase composition was about the same as for the neat liquid. Tables, diagrams, and graphs, 21 p.
■ *API Health Environ. Sci. Dep. Rep.* (Apr. 1987) (49 p.).
Source: API HESD Information Specialist

35-32084
Inhalation neurotoxicity study in rats with C_9 aromatic hydrocarbons [(AHC)]. Tests on four groups of 20 male Sprague-Dawley rats each, which were exposed to clean air, or 101, 452, or 1320 ppm of C_9 AHC (C_9 aromatic naphtha) for 6 hr/day, 5 days/wk for 13 consecutive weeks, showed no toxicologically significant pharmacotoxic signs during exposure or post-exposure under the test conditions. A 13% decrease in body weights at 1320 ppm was not considered statistically significant, since the depression was reversible and was reduced to 3.7% at the end of the post-exposure recovery period. There were no treatment-related effects noted on motor activity, startle response latency, forelimb and hindlimb grip strength, hindlimb splay distance, and thermal response time. Necropsy on one group showed no exposure-related neuropathologic lesions in sections of the brain, spinal cord, L_3 and L_4 dorsal root ganglia, and sciatic and tibial peripheral nerves, and no degenerative changes in teased nerve fibers from the lower tibial and sural nerves. Tables, graphs, drawings, diagram, and appendixes, including a pathology report, 56 p.
■ *API Health Environ. Sci. Dep. Rep.* (June 1988) (125 p.).
Source: API HESD Information Specialist

35-32430
28-Day dermal toxicity study of API 83-15, hydrotreated heavy naphthenic distillate (CAS 64742-52-5), in the rabbit. Final report. A study on groups of five male and five female New Zealand white rabbits, whose shaved, intact backs were treated with 0, 200, 1000, or 2000 mg/kg of API 83-15 (mainly C_{20}-C_{50} hydrocarbons that produce a finished oil of at least 100 SUS at 100 °F) 3 times/wk for 28 days, indicated that API 83-15 was slightly irritating to males and females at 2000 and 1000 mg/kg and minimally irritating at 200 mg/kg. There was moderate erythema at 2000 and 1000 mg/kg; one male and one female showed slight edema at 2000 mg/kg. The sham-treated controls showed no irritation. Treatment-related findings include statistically significantly lower than control mean body weight and mean terminal body weight for high-dose males and females; statistically significantly higher than control mean relative liver weight, and numerically higher than control mean absolute liver weight for high-dose females; and liver changes ranging from yellow discolorations at 200 mg/kg to a prominent lobular pattern for females at 2000 mg/kg. Microscopic pathology showed changes in the livers of high-dose males and females characterized by hepatocytomegaly accompanied by subacute hepatitis. Tables, addenda, and appendixes, 82 p.
■ *API Health Environ. Sci. Dep. Rep.* (Apr. 1987) (114 p.).
Source: API HESD Information Specialist

35-32431
Unscheduled DNA synthesis ((UDS)) in rat primary hepatocytes with

PS-6 unleaded gasoline, its evaporation residue, and a DMSO (dimethylsulfoxide) extract. A preliminary cytotoxicity test was made on PS-6 unleaded gasoline dissolved in ethanol and on PS-6 extract dissolved in DMSO at 10 doses from 0.0003 to 10 µL/mL, and on PS-6 residue dissolved in ethanol at 12 doses from 0.03 to 10,128 µL/mL to establish appropriate doses for the UDS assays. Under the test conditions, PS-6 and PS-6 extract are considered negative in the UDS assay at 0.1, 0.3, 1.0, 3.0, and 10 µL/mL and at 0.01, 0.03, 0.1, 0.3, and 1.0 µL/mL, respectively, since there was no significant increase in the mean number of net nuclear grain counts at any of the doses. PS-6 residue is considered positive in the UDS assay because it caused a significant increase in the mean number of net nuclear grain counts. Tables and appendix, 22 p.
■ R. D. Curren (Microbiol. Assoc. Inc.), *API Health Environ Sci. Dep. Rep.* (Oct. 1988) (38 p.).
Source: API HESD Information Specialist

35-32432
Sister chromatid exchange [(SCE)] assay in Chinese hamster ovary (CHO) cells with API 83-07: Light catalytic cracked distillate. CHO cells treated with 10, 20, 40, or 80 µg/mL of API 83-07 dissolved in acetone showed statistically significant increases in the frequency of SCEs relative to the solvent controls at 10 and 20 µg/mL in the absence of Aroclor 1254-induced rat liver S-9 activation, and at 10, 40, and 80 µg/mL in the presence of activation. The nonactivated test was repeated because of cell cycle delay at 40 and 80 µg/mL. The repeat study at 5, 10, 20, and 30 µg/mL showed statistically significant increases in the frequency of SCEs relative to the solvent control at 30 µg/mL. API 83-07 was considered to be equivocal under the test conditions because of the increase in SCEs above the spontaneous background level with no clear dose response. Tables and appendix, 14 p.
■ D. L. Putman (Microbiol. Assoc. Inc.), *API Health Environ. Sci. Dep. Rep.* (Oct. 1988) (31 p.).
Source: API HESD Information Specialist

35-32433
Sister chromatid exchange ((SCE)) assay in Chinese hamster ovary (CHO) cells with API 81-10: Hydrodesulfurized middle distillate. Final report. An SCE assay on CHO cells treated with 0.008, 0.016, 0.03, and 0.06 µL/mL of API 81-10 in the absence of an Aroclor 1254-induced rat liver S-9 activation showed a non-significantly elevated frequency of SCEs relative to the solvent control at any of the doses. API 81-10 was thus considered to be equivocal when tested at 0.13, 0.25, 0.5, and 1 µL/mL in the presence of activation, since the CHO cells showed a statistically significant increase in the frequency of SCEs relative to the solvent control at 0.13 and 0.25 µL/mL. A statistically significant loss of activity observed at 0.5 and 1 µL/mL could be related to toxicity or to the availability of chemical to target cells, since insolubility was a problem at all doses. Tables and appendix, 13 p.
■ D. L. Putman (Microbiol. Assoc. Inc.), *API Health Environ. Sci. Dep. Rep.* (Oct. 1988) (31 p.).
Source: API HESD Information Specialist

35-32479
In-vivo sister chromatid exchange [(SCE)] assay with API 81-10: Hydrodesulfurized middle distillate. Final report. A study on four groups of five male and five female B6C3F1 mice each, which received a single intraperitoneal (IP) injection of 10 mL/kg of corn oil, or 5, 2.5, or 0.5 g/kg of API 81-10 dissolved in corn oil 4 hr after subcutaneous implantation of a 50 mg agar-coated BrdUrd (sensitizer) pellet, showed that API 81-10 did not induce a significant increase in bone marrow SCEs under the test conditions. API 81-10 consists mainly of C_{11}-C_{25} hydrocarbons boiling at ~ 205-400 °C. Tests on two groups of five male and five female mice each that received a single IP injection of 4 g/kg of API 81-15 (an API-designated control) or 10 mg/kg of cyclophosphamide, a positive control, showed that these materials induced significant increases in SCEs/cell/mouse relative to the corn oil controls in both males and females. Tables and appendix, 9 p.
■ D. L. Putman (Microbiol. Assoc. Inc.), *API Health Environ. Sci. Dep. Rep.* (Oct. 1988) (26 p.).
Source: API HESD Information Specialist

35-32480
Mouse ovarian tumor (MOT) cell attachment assay with API 81-15: Catalytic cracked clarified oil. Final report. A study on (3H)-thymidine labeled MOT cells, which were treated with 100, 50, 25, 12.5, 6.3, 3.1, 1.6, or 0.8 µg/mL of API 81-15 dissolved in acetone, indicates that API 81-15 should be considered a suspect teratogen (rating of 3) under the test conditions. The assessment is based on the ability of the test article to inhibit MOT cell attachment to Concanavalin A-coated disks. API 81-15 consists mainly of greater than C_{20} hydrocarbons which are likely to contain 5 wt % or more of 4- to 6-member condensed ring aromatic hydrocarbons boiling at higher than 350 °C. Tables, 5 p.
■ L. L. Yang (Microbiol. Assoc. Inc.), *API Health Environ. Sci. Dep. Rep.* (Oct. 1988) (23 p.).
Source: API HESD Information Specialist

35-32481
Mouse ovarian tumor (MOT) cell attachment assay with API 86-02: Solvent refined coal distillate. Final report. The results of a study involving the treatment of [3H] -thymidine-labeled MOT cells with 100, 50, 25, 12.5, 6.3, or 3.1 µg/mL of API 86-02 dissolved in dimethyl sulfoxide indicate that API 86-10 should be considered a potential teratogen (rating of 4) under the test conditions. The assessment is based on the ability of the test article to inhibit MOT cell attachment to Concanavalin A-coated disks. Tables, 4 p.
■ L. L. Yang (Microbiol. Assoc. Inc.), *API Health Environ. Sci. Dep. Rep.* (Oct. 1988) (20 p.).
Source: API HESD Information Specialist

35-32482
Sister chromatid exchange [(SCE)] assay in Chinese hamster ovary (CHO) cell with API 81-07: Hydrodesulfurized kerosine. Final report. An SCE assay on CHO cells treated with 0.007, 0.013, 0.025, or 0.05 µL/mL of API 81-07 dissolved in acetone in the absence of an Aroclor-induced rat liver S-9 activation, and with 0.05, 0.1, 0.2, or 0.4 µL/mL of API 81-07 dissolved in acetone in the absence of activation, indicates that the test material is negative under the test conditions. API 81-07 did not induce an increase in SCEs in the absence of activation. The small but statistically significant increases in SCEs at 0.05 and 0.4 µL/mL in the presence of activation were not dose-responsive, seemed to be random, and were not biologically significant. Cells treated with cyclophosphamide, a positive control, showed significantly increased SCEs relative to the untreated control. Tables and appendix, 13 p.
■ D. L. Putman (Microbiol. Assoc. Inc.), *API Health Environ. Sci. Dep. Rep.* (Oct. 1988) (31 p.).
Source: Hydrodesulfurized kerosine

36-30038
Evaluation of appropriate methodologies for measuring PNA exposures in the petroleum coking environment. Because of the high humidity and temperatures involved in the petroleum coking environment, there was considerable loss from filter to sorbent of 2-, 3-, and some 4-ring PAH as vapors or along with water vapor. Polyurethane foam (PUF) was more effective at collecting and storing samples of most PAH. XAD-2 resin was superior in collecting 2- and some 3-ring PAH, including acenaphthylene and acenaphthene. Commercial XAD-2 sampling tubes were prone to contamination which made quantification of naphthalene, acenaphthylene, and acenaphthene difficult. Both field and laboratory testing showed the Teflon(TM)/PUF combination, followed by HPLC analysis, to be far superior to the NIOSH glass-fiber/silver membrane filter technique, as glass fiber promoted the reactions of PAH. Methylene chloride extracted more of the PAH than either benzene or cyclohexane. Although BaP is the most commonly measured PAH, BaP was not an adequate indicator of exposure in a petroleum coking environment since, in a number of cases, no BaP was found while there were high concentrations of other PAH. Samples should be collected at as many cokers as close to the drum head as possible. Graphs, tables, diagrams, photographs, and 109 references, 239 p.
■ *API Health Environ. Sci. Dep. Rep.* (11/3/83) (268 p.).
Source: API HESD Information Specialist

36-30043
In vivo sister chromatid exchange [(SCE)] assay with API 81-07, hydrodesulfurized kerosine. Final report. Groups of five male or female B6C3F1 mice were injected once intraperitoneally either with API 81-07 in corn oil, at dose levels of 400, 2000, and 4000 mg/kg body weight 4 hr after subcutaneous implantation of a 50 mg agar-coated BrdUrd sensitizer pellet, or with corn oil (negative control), or cyclophosphamide (positive control), and API 81-15 in corn oil, 400 mg/kg (additional positive control). The bone marrow cells, arrested in metaphase with colchicine and collected 24-26 hr after pellet implantation, were examined microscopically for SCEs. Under the test conditions, API 81-07 induced a statistically significant increase in bone marrow SCEs, and toxic effects, in male B6C3F1 mice only. Slight weight loss was observed in high-dosed males and females and in mid-dosed males at, and after, treatment with colchicine. API 81-07 consists mainly of C_9-C_{16} hydrocarbons boiling at 150-290 °C. Tables and appendixes, 9 p.
■ D. L. Putman, *API Health Environ. Sci. Dep. Rep.* (Oct. 1988) (26 p.).
Source: API HESD Information Specialist

36-30044
In vivo sister chromatid exchange [(SCE)] assay with API 81-03 (light catalytically cracked naphtha). Final report. Groups of five male or female B6C3F1 mice were injected intraperitoneally once either with API 81-03 in corn oil, at 200, 1200, and 2400 mg/kg body weight 4 hr after subcutaneous implantation of a 50 mg agar-coated BrdUrd sensitizer pellet, or with corn oil, solvent vehicle (negative control), or with cyclophosphamide (positive control), or API 81-15 in corn oil at 400 mg/kg (additional positive control). The bone marrow cells, arrested in metaphase and collected 24-26 hr after pellet implantation were examined microscopically for SCE. Under the test conditions, API 81-03 induced a statistically significant increase in bone marrow SCE of male and female B6C3F1 mice. No toxic effects, body weight loss, or mitotic delay were observed. API 81-03 consists mainly of C_4-C_{11} hydrocarbons and a large proportion of unsaturated hydrocarbons and boils at -20 to +190 °C. Tables and appendixes, 9 p.
■ D. L. Putman, *API Health Environ. Sci. Dep. Rep.* (Oct. 1988) (26 p.).
Source: API HESD Information Specialist

36-30045
Sister chromatid exchange [(SCE)] assay in Chinese hamster ovary (CHO) cells with API 81-03 [(light catalytically cracked naphtha)]. Final report. The mutagenic potential of API 81-03 was measured by the induction of SCE in CHO cell chromosomes, in the presence and absence of Aroclor-induced rat liver S-9 activation, with cyclophosphamide and triethylenemelamine as the respective positive controls. In the presence of S-9 activation, API 81-03, tested at 0.03, 0.05, 0.1, and 0.2 µL/mL doses in acetone, showed a small but statistically significant and dose-responsive increase in SCE frequency relative to the solvent control, at the 0.05 and 0.1 µL/mL dose levels, and was thus equivocal in the SCE assay. In the absence of S-9 activation, API 81-13 at all dose levels tested (0.05, 0.1, 0.2, and 0.3 µL/mL) showed no statistically significant SCE frequency increases relative to the controls, and was thus negative in the SCE assay. API 81-03 consists mainly of C_4-C_{11} hydrocarbons, with relatively high olefinic content (15-70%), boiling at -20 to +190 °C. Tables and appendixes, 12 p.
■ *API Health Environ. Sci. Dep. Rep.* (Oct. 1988) (28 p.).
Source: API HESD Information Specialist

36-31364
Lifetime dermal carcinogenesis bioassay of refinery streams in C3H/HeJ mice (AP-135R). The carcinogenic and chronic toxicities of twelve petroleum refinery streams were tested, using 1700 C3H/HeJ mice randomly divided into 2 positive control groups (0.01% and 0.05% benzo[a]pyrene in toluene), 2 negative control groups (a vehicle control and a sham control), and 14 test material groups (9 test materials dosed neat, 2 diluted to 50% in toluene, and 1 at 10, 1, and 0.1% in toluene). The mice were dosed twice per week with 50 µL of test or control material applied dermally to the clipped intrascapular region of each animal's back. All the test materials were locally dermatotoxic, to greatly varying degrees. Skin tumors occurred in all exposed groups including toluene-treated controls, but incidence varied from 4 to 100%. The cracked clarified oil was the most potent skin carcinogen. Hydrodesulfurized kerosine and hydrodesulfurized middle distillates had intermediate skin carcinogenicities. Light catalytically cracked naphtha was a weak dermal carcinogen. Sweetened naphtha and vacuum residues did not produce statistically significant increases in tumor incidence. Tables.
■ *API Health Environ. Sci. Dep. Rep.* (3/1/89) (37 p.).
Source: API HESD Information Specialist

36-31429
In vivo sister chromatid exchange assay with API 83-07 (light catalytic cracked distillate). Male and female B6C3F1 mice were exposed to API 83-07 light catalytic cracked distillate at dose levels of 3400, 1700, or 340 mg/kg, which was administered as a single intraperitoneal injection 4 hr after subcutaneous implantation of a 50 mg agar-coated BrdUrd pellet. Bone marrow cells, arrested in metaphase and collected 24-26 hr after pellet implantation were examined microscopically for sister chromatid exchanges (SCE). A dose-responsive increase in SCE was observed in male and female B6C3F1 mice receiving API 83-07 when compared with the vehicle control group. The results of the assay indicate that under the conditions described, the test article API 83-07 does induce a significant increase in bone marrow SCE in B6C3F1 mice. The distillate consisted predominantly of C_9-C_{25} hydrocarbons at 150-400 °C bp. It contained a relatively large portion of bicyclic aromatic hydrocarbons. Tables.
■ D. L. Putman, *API Health Environ. Sci. Dep. Rep.* (3/17/89) (25 p.).
Source: API HESD Information Specialist

36-31430
Studies on the absorption, tissue equilibria and excretion routes of inhaled hydrocarbon vapors and their metabolites involved exposing rats to 1 or 500 ppm of carbon-14 labeled octane or isooctane for 5, 25, and 120 min. Regarding inhaled gasoline components, metabolite excretion of the branched, nephrotoxic hydrocarbon, isooctane, after inhalation was virtually from rat urine, but the unbranched, non-nephrotoxic isomer, octane was metabolized and eliminated largely as CO_2. Urinary excretion of isooctane metabolites was protracted vs. relatively prompt urinary octane metabolite excretion. The route of octane excretion, but not for isooctane, was affected by inhaled vapor concentration. The CO_2-14/carbon-14 urine ratio was 5:1 after inhaling radiolabeled octane at ~ 1 ppm, but 1:1 after inhaling at ~ 500 ppm. Different excretion patterns of isooctane metabolites vs. octane may affect differences in their nephrotoxicity. Blood concentration comparisons of carbon-14 introduced by inhaling octane or isooctane showed that volatile blood isooctane metabolites such as 2,4-trimethyl-2-octanol were present after isooctane inhalation at ~ 1 or ~ 500 ppm. Diagrams, tables, graphs, and 34 references.
■ *API Health Environ. Sci. Dep. Rep.* (3/1/89) (69 p.).
Source: API HESD Information Specialist

36-31431
Benzene pharmacokinetics and pharmacodynamics. A model has been developed that can predict venous blood, exhaled air, and tissue concentrations of benzene vs. time in animal species after benzene exposure. This pharmacokinetic model for benzene can successfully replicate benzene concentration in mouse and rat blood, expired air, bone marrow, and intraperitoneal administration. The model predicts benzene pharmokinetics in humans after inhalation exposure of 5 ppm for 6 hr to 99 ppm for 1 hr. Benzene body burden after filling a gas tank is > 4 times that of ambient. Smoking 20 cigarettes/day increases body burden by 250-300%. It is suggested that under ambient conditions, benzene binds considerably to plasma proteins, which could substantially affect estimates of risk of benzene exposure, the regulation of which costs industries more than $1 billion/yr. A computer software program for modeling the pharmokinetics of benzene has been developed. It can be run on any IBM compatible computer. Diagrams, tables, graphs, and 109 references.
■ C. C. Travis; A. D. Arms; J. C. Bowers; J. L. Quillen (Oak Ridge Natl. Lab.), *API Health Environ. Sci. Dep. Rep.* (Jan.-Dec. 1988) (125 p.).
Source: API HESD Information Specialist

36-31909
The human embryonic palatal mesenchyme (HEPM) cell growth inhibition assay [of API-81-15 (catalytic cracked clarified oil)] without activation showed API-81-15 was a suspect teratogen (rating of 3). In a study sponsored by API, Microbiological Associates Inc. plated exponentially growing HEPM cells for 23 ± 2 hr. Following this attachment period, the remaining cells were exposed in triplicate to API-81-15 at concentrations of 500, 250, 125, 62.5, 31.3, 15.6, 7.8, and 3.9 µg/mL for 72 ± 2hr at 37 °C. After the treatment period, the cells were counted. The data were analyzed to determine the cell number per dish, the average cell number for each test article concentration, and the percent growth in each test article concentration relative to that observed in the solvent control. The concentration of API-81-15 required to produce a 50% inhibition (ID50) in the growth of the number of cells was 73.2 and 60.5 µg/mL for the first and second routine assay, respectively. The average ID50 of 66.9 µg/mL corresponded to a suspect teratogen. Tables.
■ J. W. Harbell C. I. Sigler, *API Health Environ. Sci. Dep. Rep.* (30 p.).
Source: API HESD Information Specialist

36-31910
Human embryonic palatal mesenchyme (HEPM) cell growth inhibition assay [of API-81-15 (catalytic cracked clarified oil)]...S-9 mediated assay showed API-81-15 was a suspect teratogen (rating of 3). In a study sponsored by API, Microbiological Associates Inc. plated exponentially growing HEPM cells for 23 ± 2 hr. Following the attachment period, the remaining cells were exposed in triplicate to API-81-15 at concentrations of 50, 25, 12.5, 6.23, 3.1, 1.6, 0.8, and 0.4 µg/mL in the presence of S-9 and cofactors for 4 hr to check for possible activation of API-81-15 by S-9. After rinsing and addition of fresh culture medium, the cultures were allowed to grow for 72 ± 2 hr. After the growth period, the cells were counted. The data were analyzed to determine the cell numbers per dish, the average cell number of each test article concentration, and the per cent growth in each test article concentration relative to that observed in the solvent control. The concentration of API-81-15 required to produce a 50% inhibition in the growth of the cell numbers (ID50) was 42.9 and 65.7 µg/mL for the first and second routine assay, respectively. The average ID50 of 54.3 µg/mL for API-81-15 corresponded to a suspect teratogen. Tables.
■ J. W. Harbell C. I. Sigler, *API Health Environ. Sci. Dep. Rep.* (31 p.).
Source: API HESD Information Specialist

36-31911
Human embryonic palatal mesenchyme (HEPM) cell growth inhibition assay [of API-86-02 (a dark viscous petroleum liquid)] showed API-86-02 was a potential teratogen (rating of 4) without activation. In a study sponsored by API, Microbiological Associates Inc. plated exponentially growing HEPM cells for 24 hr. Following the attachment period, the remaining cells were exposed in triplicate to API-86-02 at concentrations of 50, 25, 12.5, 6.3, 3.1, 1.6, 0.8, and 0.4 µg/mL for 72 hr at 37 °C. After the treatment period, the cells were counted. The data were analyzed to determine the cell number per dish, the average cell number for each concentration, the per cent growth in each test concentration relative to that observed in the solvent control. The concentration of API-86-02 required to produce a 50% inhibition in the growth in cell numbers (ID50) was 15.9 and 14.4 ηm/mL for the first and second routine assays, respectively. The average of 15.2 for API-86-02 corresponded to a potential teratogen. Tables.
■ J. W. Harbell C. I. Sigler, *API Health Environ. Sci. Dep. Rep.* (40 p.).
Source: API HESD Information Specialist

36-31912
Human embryonic palatal mesenchyme (HEPM) cell growth inhibition assay [of API-86-02 (a dark viscous petroleum liquid)]...S-9 mediated assay showed API-86-02 was a potential teratogen (rating of 4). In a study sponsored by API, Microbiological Associates Inc. plated exponentially growing HEPM cells for 23 hr. Following the attachment period, the remaining cells were exposed in triplicate to API-86-02 at 50, 25, 12.5, 6.3, 3.1, 1.6, 0.8, and 0.4 η/mL in the presence of S-9 and cofactors for 4 hr to check for possible activation of API-86-02 by S-9. After rinsing and addition of fresh medium the cultures were allowed to grow for 72 hr. After the growth period, the cells were counted. The data were analyzed to determine the cell numbers per dish, the average cell number for each API-86-02 concentration and the per cent growth at each concentration relative to that observed in the solvent control. The concentration of API-86-02 required to produce a 50% inhibition in the growth of cell numbers (ID50) was 23.3 and 27.4 µg/mL for the first and second routine assay, respectively. The average ID50 of 25.4 µg/mL for API-86-02 corresponded to a potential teratogen. Tables.
■ J. W. Harbell C. I. Sigler, *API Health Environ. Sci. Dep. Rep.* (31 p.).
Source: API HESD Information Specialist

36-31913
Human embryonic palatal mesenchyme (HEPM) cell growth inhibition assay [of BaP] without activation showed BaP was not a teratogen (rating of 0). In a study sponsored by API, Microbiological Associates Inc. plated exponentially growing HEPM cells for 23 hr. Following the attachment period, the remaining cells were exposed in triplicate to BaP at concentrations of 27, 13.5, 6.8, 3.4, 1.7, 0.8, and 0.4 µg/mL. After the treatment period, all media were aspirated, and the cells were washed counted. The data were analyzed to determine the cell number per dish, the average cell number for each BaP concentration, and the per cent growth in each BaP concentration relative to that observed in the solvent control. BaP was noninhibitory at the highest soluble concentration (27 µg/mL) and was not a teratogen. Tables.
■ J. W. Harbell (Microbiol. Assoc. Inc.), *API Health Environ. Sci. Dep. Rep.* (26 p.).
Source: API HESD Information Specialist

36-31914
Acute exposure to methanol in fuels: A prediction of ingestion incidence and toxicity. A discussion covers statistical data on the incidence of acute exposure to gasoline (13,028 cases) and methanol (1601 cases), reported to the American Association of Poison Control Centers (AAPCC); analysis of these data which shows, inter alia, that gasoline ingestion by small children and ingestion during siphoning of fuel are the dominant routes of accidental acute poisoning by gasoline and that methanol poisoning has a 25-fold greater fatality rate than gasoline; clinical manifestations and treatment of methanol poisoning; predictions of incidence, toxicity, and health effects of methanol-based fuels, which show that the widespread use of such fuels would sharply increase the number of methanol-caused fatalities, blindness, and neurological impairment cases, with corresponding additional national expenditures for acute health care only of more than $50-100 million/yr; and concludes that the acute public health hazard posed by conversion to methanol fuels is unacceptable, and that prior to such conversion, innovative closure and packaging techniques must be developed to prevent siphoning and limit access by small children. Tables and 60 references.
■ T. Litovitz (Natl. Capital Poison Cent., Georgetown Univ. Hosp.), *API Publication* #4477 (Oct. 1988) (34 p.).
Source: API Publications Order Desk (Order No. I44770)

36-32640
A 28-day dermal toxicity study of API 85-01 in the rabbit (Stoddard solvent). API 85-01, a selective petroleum-derived hydrocarbon, at 200, 1000, or 2000 mg/kg was applied to the clipped intact skin of five male and five female rabbits three times a week for a total of 12 applications over a 28 day period with five male and five female rabbits as sham-treated controls. One female in the 2000 mg/kg had marked weight loss and was killed in a moribund condition on day 14; mucoid enteritis was the probable cause. Mean weight gains of control males and males in the 200 and 1000 mg/kg group were similar whereas weight gains in the 2000 mg/kg groups was less than controls. In females, mean body weight gains in the 1000 and 2000 mg/kg groups were less than controls. Repeated dermal application of API 85-01 produced marked to severe erythema and edema at all dose levels associated with cracked skin, leathery texture, and flaking. Treatment-related lesions consisted of thickening and downgrowth of the epidermis, hyperkeratosis, and fibrosis, frequently accompanied by inflammatory cell infiltrates. Except for the dermal lesions, gross lesions were seen only in an occasional rabbit, or in near equal incidence in control and treated groups. Tables.

■ *API Health Environ. Sci. Dep. Rep.* (June 1989) (136 p.).
Source: API HESD Information Specialist

36-32641
***Salmonella*/mammalian-microsome mutagenicity assay of the vapor phase of commercial hexane using the desiccator methodology** did not indicate any toxic or mutagenic activity from hexane vapor. The evaluation was based on hexane vapor's inability to induce back mutations at selected loci of several strains of *Salmonella typhimurium* in the presence and absence of exogenous metabolic activation. One strain contained a deletion in the {uvrB} gene, resulting in a deficient DNA excision-repair system and a greatly enhanced sensitivity to some mutagens. At exposure to hexane vapor for 7 hr in the presence or absence of exogenous metabolic activation, no appreciable toxicity or mutagenicity was seen when tester strain TA1000 was exposed to 300-9000 ppm of hexane or when tester strains TA98, TA100, TA1535, TA1537, and TA1538 were exposed to 600-9000 ppm. Tables.
■ R. H. C. San K. A. Springfield, *API Health Environ. Sci. Dep. Rep.* (July 1989) (70 p.).
Source: API HESD Information Specialist

36-32642
Three generation [(F_0, F_1, and F_2)] reproduction/fertility study in rats with C\9 aromatic hydrocarbons. C_9 aromatic hydrocarbons at 0, 100, 500, or 1500 ppm were administered by inhalation to 30 male and 30 female rats per group in the F_0 (parents) and F_1 (offspring of parents) generations for 10 wk prior to mating, and to 40 male and 40 female old rats per group in the F_2 (offspring of the F_1 generation) generation for 10-12 wk prior to mating. Clinical signs of toxicity seen at 1500 ppm in the F_0 generation included increased salivation, unkempt appearance, body staining, hunched posture, high carriage, aggressive behavior, and increased hair loss. To these were added reduced motor activity and ataxia in the F_1 generation. Increased mortality occurred among the females in both the F_0 and F_1 generations, and the death rate early in the F_2 generation was excessive. Macroscopic changes in the lungs of the 1500 ppm groups in the F_0 and F_1 generations correlated with C_9 aromatic related increase in the incidence of foci of pulmonary macrophages in the lungs. The no observable effect level was 500 ppm in the F_0 and F_1 generations, and 100 ppm in the F_2 generation for reproductive effects. Weight loss was seen at 500 and 1500 ppm in the F_0 and F_1 generations and all doses in the F_2 generation. Tables.
■ *API Health Environ. Sci. Dep. Rep.* (July 1989) (53 p.).
Source: API HESD Information Specialist

36-32643
Short-term dermal tumorigenesis study of selected petroleum hydrocarbon [processing fractions] in male CD-1 mice...Initiation and promotion phases [of dermal tumorigenesis]. Mice dosed (initiated) with petroleum fractions API 81-15 (catalytic cracked clarified oil, 1%) or API 84-01 (light paraffinic distillate) followed by phorbol-12--myristate-13-acetate in acetone as the promoter had a significant increase in the incidence of skin neoplasia at the application site and decrease in latency time compared with the control group. For the mice initiated with API 83-07 (light catalytic cracked distillate), the latency period decreased and tumor incidence increased. API 79-01 (naphthenic lubricating oil base stock), API 81-07 (hydrodesulfurized kerosine), API 81-08 (sweetened naphtha), API 81-10 (hydrodesulfurized middle distillate), API 83-03 (middle distillate high catalytic cracked stock, 50%), and API-81-13 (vacuum residue) were not tumor initiators. Rats initiated with 9,10-dimethyl-1,2-benzanthracene and promoted with either API 81-07, API 81-10, API 83-03, API 83-07, or API 84-01 had a statistically significant increase in skin neoplasms. The promoting activities of API 81-07, API 81-10, API 83-03, and API 83-07 were reasonably comparable in promoting tumorigenicity, with API 84-01 as a less active promoter. API 81-15 significantly shortened the latency period and increased the number of mice with clinically observed masses, but not histologically confirmed tumors. Tables.
■ *API Health Environ. Sci. Dep. Rep.* (July 1989) (234 p.).
Source: API HESD Information Specialist

36-32657
Monitoring near refineries for airborne chemical on the SARA [(Superfund Amendments and Reauthorization Act of 1986)] Title III Section 313 list. Volume I...Validated ambient air concentrations around three refineries. As part of a project carried out for the API by Radian Corp., ground-level ambient air concentrations of 25 pollutants on the SARA Title III Section 313 list, as well as 11 non-SARA chemicals (selected as indicators of refinery emissions), were measured in Apr.-June 1988 at or near the perimeters of the 230,000 bbl/day Convent, LA, refinery, the 213,000 bbl/day Catlettsburg, KY, refinery, and the 124,000 bbl/day Benicia, CA, refinery, located in a rural, industrial, and suburban setting, respectively. The measurements were made at nine sites for each refinery, during three consecutive 24 hr periods, while the refineries were operating under normal and nominally full-capacity conditions. The SARA chemicals measured included 14 hydrocarbons (mainly 1-3 ring aromatics); the non-SARA compounds were C_5-C_8 alkanes, methylcyclohexane, tert.-butylbenzene, and tri- and tetrachloroethylene. The measurement results and their interpretations are given, together with details of the sampling and analysis methods and Quality Assurance/Quality Control procedures used for data validation. Maps, diagrams, tables, graphs.
■ *API Publication* #4484 (Jan. 1989) (407 p.).
Source: API Publications Order Desk (Order No. I44840)

36-33132
The mutagenic potential of high flash aromatic naphtha. Specific tests, including the *Salmonella*/mammalian microsome mutagenicity assay, the hypoxanthine-guanine phosphoribosyl transferase (HGPRT) forward mutation assay in Chinese hamster embryo (CHO) cells, in-vitro chromosome aberration and sister chromatid exchange (SCE) assays in CHO cells, and an in-vivo chromosome aberration assay in rat bone marrow were performed to assess the toxicological properties of high-flash aromatic naphtha with emphasis on mutagenic activity and carcinogenic potential. The lack of mutagenic potential as shown by the tests suggests that the naphtha is unlikely to be a genotoxic carcinogen. The naphtha did not induce gene mutation in either the *Salmonella*/mammalian microsome mutagenicity test or the HGPRT forward mutation assay in CHO cells, or DNA perturbation as assessed by an SCE assay in CHO cells, and did not produce chromatid or chromosomal abnormalities in vitro (CHO cells). There was no evidence of chromosomal abnormalities in the rat bone marrow either following exposure for five days to the maximally attainable vapor concentration. Tables and 23 references.
■ C. A. Schreiner (Mobil Oil Corp.); R. H. McKee (Exxon Biomed. Sci. Inc.); M. Swanson (API); Z. A. Wong (Chevron Environ. Health Cent.); S. Schmitt (Amoco Corp.); P. Beatty (Shell Oil Co.); D. A. Edwards, *Cell Biology and Toxicology* 5(2):169-88 (1989).
Source: Not available from API

36-33219
Polynuclear aromatic analyses and modified Ames tests on base lube [lubricating] stocks. Paraffinic lubricating oil base stocks of different viscosities were analyzed for PaH and tested for mutagenic activity using the Modified Ames Assay. The tests were performed at the Mobil Oil Corp. Environmental Health & Safety Laboratory on API samples of base stocks. Both chemical and microbial tests indicated that these materials had negligible mutagenic and carcinogenic potencies.
■ *API Health Environ. Sci. Dep. Rep.* (Sept. 1989) (327 p.).
Source: API HESD Information Specialist

36-33220
Twenty-four month dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice (AP-190r). Final Report. The carcinogenic and chronic toxicity potential of 11 petroleum refinery streams were tested at New Mexico State University using 1400 C3H/HeJ mice with, as primary intent, determination of dermal carcinogenicity. The carcinogenicity and chronic toxicity screening studies were conducted concurrently. All test materials were locally dermatotoxic. Skin tumors occurred in most treatment groups. The degree of dermal toxicity and carcinogenicity varied greatly. There were virtually no dermal lesions in untreated mice. Toluene treated mice had a very high

incidence of mild desquamation. Dermal lesions in mice treated with 0.01% BaP were similar to toluene controls. However, after 85-104 wk, there was abdominal distention and head tilt in 5-30% of the mice. Dermal lesions in mice treated with 0.05% BaP were initially similar to toluene controls, but the incidence of irritation increased to 75-100% after 62-85 wk.

■ *API Health Environ. Sci. Dep. Rep.* 1-2 (Oct. 1989) (1166 p.).
Source: API HESD Information Specialist

36-33318

Developmental toxicity study of commercial hexane vapor in CD(TM)-1 mice. Developmental toxicity and teratogenicity of maternally inhaled commercial hexane vapor in the pregnant CD-1 mouse was studied. The potential of the test chemical to produce maternal toxicity was also determined. Exposure to commercial hexane vapor by inhalation during organogenesis in CD-1 mice resulted in slight maternal toxicity at 3000 and 9000 ppm and developmental toxicity (in the absence of malformations) at 9000 ppm. The "no observable effect levels" (NOEL) for maternal toxicity was 900 ppm and for developmental toxicity the NOEL was 3000 ppm. The CD-1 mice (30/group) were exposed to the commercial hexane vapor for 6 hr/day on gestational days (gd) 6 through 15 at concentrations of zero, 900, 3000, or 9000 ppm. Maternal clinical signs were taken daily, and body weights were measured on gd zero, 6, 9, 12, 15, and 18. Maternal food and water consumption was measured during gestation, gd zero-18. Sacrifice was on gd 18. Tables, graphs, and 21 references.

■ T. L. Neeper-Bradley (Mellon Inst.-Union Carbide Corp., Bushy Run Res. Cent.), *API Health Environ. Sci. Dep. Rep.* (10/27/89) (350 p.).
Source: API HESD Information Specialist

36-33319

Developmental toxicity study of commercial hexane vapor in CD(TM) (Sprague-Dawley) rats. A study was made to evaluate the developmental toxicity and teratogenicity of maternally inhaled commercial hexane vapor in the pregnant CD (Sprague-Dawley) rat. The potential of the test chemical to produce maternal toxicity was also determined. Inhalation of the hexane vapor resulted in maternal toxicity at 3000 and 9000 ppm with no apparent developmental toxicity including malformations at any exposure level. The "no observable effect level" (NOEL) for maternal toxicity was 900 ppm and for developmental toxicity NOEL was ≤ 9000 ppm. The rats (25/group) were exposed to hexane vapor 6 hr/day on gestational days (gd) 6 through 15 at target concentrations of zero, 900, 3000, or 9000 ppm. Sacrifice was made on gd 21. The fetuses were examined for deformations. The study was performed in response to the EPA Final Test Rule on commercial hexane (EPA, 1988a) and in accordance with EPA testing standards (EPA, 1983 and EPA, 1986). Tables, graphs, and 17 references.

■ T. L. Neeper-Bradley (Mellon Inst.-Union Carbide Corp., Bushy Run Res. Cent.), *API Health Environ. Sci. Dep. Rep.* (10/27/89) (325 p.).
Source: API HESD Information Specialist

37-30618

Human embryonic palatal mesenchyme (HEPM) cell growth inhibition assay [with toluene]...Final report. The teratogenic potential of toluene, based on its ability to inhibit HEPM cell growth, was assessed. Toluene was noninhibitory at the highest soluble concentration of 2150 µg/cc examined. Under the conditions of the test assay, toluene was not a teratogen (rating of zero) in the HEPM cell growth inhibition assay without activation. Toluene was tested in the HEPM cell growth inhibition assay in the absence of metabolic activation. Exponentially grown HEPM cells were plated at a density of ~ 35,000 cells/35 mm dishes and were incubated at 37 ± 1 °C in a humidified atmosphere of 5 ± 1% CO_2 in air for 23 ± 2 hr. Three dishes were counted to estimate the number of cells on each dish to establish the background count. The cells were exposed to toluene at 2150.0, 1075.0, 537.5, 268.8, 134.4, 67.2, 33.6, and 16.8 µg/cc for 72 ± 2 hr at 37 ± 1 °C. The media were aspirated, cells washed, trypsinized, and counted. Tables.

■ J. W. Harbell C. I. Sigler, *API Health & Environmental Sciences Department Report* (January 1990) (35 p.).
Source: API HESD Information Specialist

37-30619

Human embryonic palatal mesenchyme (HEPM) cell growth inhibition assay...S-9 mediated assay [with benzo(a)pyrene (BaP)]...Final report. The teratogenic potential of BaP was assessed, based on its ability to inhibit human HEPM cell growth. The concentration that would inhibit 50% of the cell growth (ID50) for the first assay was 3.3 mg/cc. The ID50 for the second assay was 8.9 µg/cc. The ID50 for the third assay was 1.7 µg/cc. Average ID50 from the three assays was 4.6 µg/cc, corresponding to a rating of 4. Under assay conditions, BaP was a potential teratogen (rating of 4). The inhibition assay was made in the presence of metabolic activation. Exponentially growing HEPM cells were plated at ~ 35,000 cells/35 mm dishes and were incubated at 37 ± 1 °C in a humidified atmosphere of 5 ± 1% CO_2 in air for 23 ± 2 hr. After the attachment period, the remaining cells were exposed to BaP concentrations of 27.0, 13.5, 6.8, 3.4, 1.7, 0.84, 0.43, and 0.22 µg/cc 4 hr at 37 ± 1 °C. After another 72 ± 2 hr of incubation, all growth media were aspirated, cells washed with phosphate buffered saline, trypsinized, and counted. Tables.

■ J. W. Harbell C. I. Sigler, *API Health & Environmental Sciences Department Report* (January 1990) (35 p.).
Source: API HESD Information Specialist

37-30620

Human embryonic palatal mesenchyme (HEPM) cell growth inhibition assay...S-9 mediated assay [with toluene]...Final report. The teratogenic potential of toluene was assessed based on its ability to inhibit HEPM cell growth in the presence of metabolic activation. Toluene was noninhibitory at the highest soluble concentration (2150 µg/cc) examined. Under assay conditions, toluene was not a teratogen (rating of 0) in the HEPM cell growth inhibition assay with activation. HEPM cells growing exponentially were plated at ~ 35,000 cells/35 mm dishes and were incubated at 37 ± 1 °C in a humidified atmosphere of 5 ± 1% CO_2 in air for 23 ± 2 hr. Following the attachment period, the remaining cells were exposed to toluene at 2150.0, 1075.0, 537.5, 268.8, 134.4, 67.2, 33.6, and 16.8 µg/cc in the presence of S-9 and cofactors for 4 hr at 37 ± 1 °C. Treatment involved 2 cc of activation medium containing various toluene concentrations. After rinsing and adding fresh medium, cultures grew for 72 ± 2 hr at 37 ± 1 °C. The medium was aspirated, cells washed with phosphate buffered saline, trypsinized, and counted. Tables.

■ J. W. Harbell C. I. Sigler, *API Health & Environmental Sciences Department Report* (January 1990) (35 p.).
Source: API HESD Information Specialist

37-31148

Special pathology report...A thirteen-week inhalation toxicity study of commercial hexane in the rat and mouse. Histopathologic examination of kidneys from FG344 rats exposed to 9000 ppm commercial hexane revealed a hydrocarbon nephropathy in 10/10 males and in 0/10 females when compared with 10 controls of both sexes. When kidneys from the lower dose male rats were examined, there appeared to be slightly increased quantities of hyaline droplet in Groups II and III male rats, but no other lesions characteristic of hydrocarbon nephropathy. Calculation of nephrotoxicity scores for the male rats indicated that only the Group IV male rats exposed to 9000 ppm commercial hexane had renal lesions characteristic of hydrocarbon nephropathy. Histopathologic examination of kidneys from male and female $B_6C_3F_1$ mice failed to reveal any treatment related lesions characteristic of hydrocarbon nephropathy. Tables.

■ B. Y. Cockrell, *API Health & Environmental Sciences Department Report* (February 1990) (36 p.).
Source: API HESD Information Specialist

37-31149

Mouse ovarian tumor (MOT) cell attachment assay with benzo(a)pyrene. The teratogenic potential of BaP, based on its ability to inhibit mouse ovarian MOT cell attachment to Concanavalin A-coated disks was assessed. The MOT cell attachment assay correctly assigned 79% of the 102 tested agents into teratogenic or nonteratogenic categories. The ID50 of the initial and confirmatory assays were greater than the highest soluble concentration in CMF-PBS, corresponding to a rating

of zero. Under the conditions of assay, BaP was considered noninhibitory (zero rating) in the MOT cell attachment assay. The human embryonic palatal mesenchyme (HEPM) cell growth inhibition assay complemented the MOT cell attachment inhibition assay in predicting teratogenicity of environmental agents. BaP was also tested in the HEPM cell growth inhibition assay, both in the presence and absence of exogenous metabolic activation. Tables.
■ C. I. Sigler D. Jacobson-Kram, *API Health & Environmental Sciences Department Report* (January 1990) (35 p.).
Source: API HESD Information Specialist

37-31150
Mouse ovarian tumor (MOT) cell attachment assay with toluene. The teratogenic potential of toluene was assessed based on its ability to inhibit MOT cell attachment to Concanavalin A-coated disks. The MOT cell attachment assay correctly assigned 79% of the 102 tested agents into teratogenic or nonteratogenic categories. The ID50 of the initial routine assay was 1560 µg/mL. The ID50 of the confirmatory assay was greater than the highest concentration which consistently produced > 85% viability (1075 µg/mL). Under the given assay conditions, toluene was considered as not likely to be a teratogen (rating of 1) in the MOT cell attachment assay. The human embryonic palatal mesenchyme (HEPM) cell growth inhibition assay complemented the MOT cell attachment inhibition assay in predicting teratogenicity of environmental agents. Toluene was also tested in the HEPM cell growth inhibition assay, both in the presence and absence of exogenous metabolic activation. Tables.
■ C. I. Sigler D. Jacobson-Kram, *API Health & Environmental Sciences Department Report* (January 1990) (37 p.).
Source: API HESD Information Specialist

37-31151
A thirteen week inhalation toxicity study of commercial hexane in the rat and mouse was conducted with Fischer 344 rats and B6C3F1 mice at vapor exposure levels of 0, 904, 2984, and 8992 ppm. There was no commercial hexane present as an aerosol. The only clinical observation which appeared treatment and dose related was a transient excess lacrimation in both sexes of mice and female rats. No signs of exposure related ocular disease were observed. Body weights and food consumption were unaffected by these exposures. Changes in the male rats in the high exposure group were increased platelets, creatinine, total protein and albumin, and a decrease in chloride. These small changes were inconsistent over sex and species and their significance was uncertain. Absolute or relative liver weights were also increased in both species in the high level group. Because of lack of a clear microscopic correlation in both species, the changes may be due to enzyme induction. There was only liver hemorrhage and acute/subacute liver and kidney inflammation in male rats.
■ P. E. Newton I. W. Daly, *API Health & Environmental Sciences Department Report* (January 1990) (887 p.).
Source: API HESD Information Specialist

37-31152
Chromosome aberrations in Chinese hamster ovary (CHO) cells exposed to commercial hexane were tested. The assay was made in the absence and presence of an Aroclor induced S-9 activation system at dose levels 0.015, 0.034, 0.074, 0.123, and 0.416 µL/mL for the nonactivated system and 0.014, 0.022, 0.056, 0.118, and 0.251 µL/mL for the S-9 activated system. Metaphase cells were collected for microscopic evaluation at 14 hr after treatment for the nonactivated study and 20 hr after treatment for the S-9 activated study. These harvest times were selected as optimum for analysis of first post-treatment metaphase cells based on the cell cycle delay observed in the preliminary toxicity study. The four highest test concentrations yielding sufficient metaphase cells were analyzed for chromosome aberrations. Toxicity was a limiting factor in the analysis of test concentrations in both the nonactivated and S-9 activated studies. No increase in chromosome aberrations was observed in either the nonactivated or S-9 activated test system. Thus, commercial hexane was negative in the CHO cytogenetics assay.
■ D. L. Putman M. J. Morris, *API Health & Environmental Sciences Department Report* (February 1990) (40 p.).
Source: API HESD Information Specialist

37-31153
An acute operant behavior study of inhaled commercial hexane in the Albino rat. Four groups of six male and six female Sprague-Dawley rats, maintained on a fixed interval schedule by food reinforcement, were treated by nose-only inhalation for 6 hr with commercial hexane at mean (standard deviations) concentrations of 0, 873 (36.4), 2974 (39.6), or 9187 (171.8) ppm. The vapor generated contained 15.8-16.6% methylcyclopentane and 51.6-52.5% n-hexane. The control group was treated similarly in all respects except it received air only. The animal response on the fixed interval 60 sec schedule was tested in a 30 min session immediately after exposure and on postexposure days one and two. No animals died or were sacrificed due to treatment. There were no toxicologically significant findings attributed to treatment with commercial hexane. The body weights and food intakes of the commercial hexane treated rats were unaffected by treatment. The response data revealed no significant differences between the control and treated groups.
■ K. Robinson,P. Beyrouty,G. Washer,B. E. Osborne, *API Health & Environmental Sciences Department Report* (February 1990) (200 p.).
Source: API HESD Information Specialist

37-31154
A 13 week inhalation study of potential effects of commercial hexane on behavior and neuromorphology in rats. Four groups of 12 male and 12 female Sprague-Dawley rats were treated by whole body inhalation exposure for 6 hr/day, 5 day/wk for 13 wk with commercial hexane at mean (S.D.) concentrations of 0, 896 (44.6), 2996 (193.5), or 9006 (386.0) ppm. No animals died or were sacrificed due to treatment. One female rat, in the air control group (No. 156) was killed on day 22 due to a fracture of the bone of the nasal cavity. There were no toxicologically significant clinical findings attributed to treatment with commercial hexane. Among animals in the commercial hexane treated groups muzzle/cranial/periorbital staining tended to be more frequent. The body weights and food intakes of commercial hexane treated rats were unaffected. The functional observational battery assessments revealed no treatment related pattern of findings indicative of a neurotoxic effect. The motor activity test revealed no significant differences between the control and treated groups.
■ K. Robinson G. Washer G. Lulham, *API Health & Environmental Sciences Department Report* 1-2 (January 1990) (800 p.).
Source: API HESD Information Specialist

37-31299
Dioxins and furans...A primer: What they are and how to measure them. This API primer provides basic information about chlorinated dioxins and furans (D&F, e.g., pollutants recently found in certain petroleum refinery wastes) to persons in the refining industry. It is directed toward environmental coordinators who must address regulatory issues, and field personnel who are responsible for obtaining analyses to fulfill regulatory requirements. The subjects covered include recent experience with D&F in the US and Canadian refining industries, EPA's regulations concerning D&F, the sources and formation of D&F, their physicochemical properties, their toxicities, environmental transport and fate of D&F, and sampling and analyses methods. Tables and 19 references.
■ *API Health & Environmental Sciences Department Report* #4506 (January 1990) (54 p.).
Source: API Publications Order Desk (Order No. I45060)

37-31336
A case-control study of kidney cancer among petroleum refinery workers. Final report. A case-control study was conducted with a cohort of ~ 100,000 male refinery workers from five petroleum companies to study increased kidney cancer risk after exposure to hydrocarbons, especially those in unleaded gasoline. A review of 18,323 death certificates identified 102 kidney cancer cases, to each of whom four control were matched. Three major hydrocarbon categories were identified, including nonaromatic liquid gasoline distillates (NLGD), aromatic hydrocarbons, and the more volatile hydrocarbons. All jobs were assessed by semiquantitative ratings for the intensity and frequency of the exposures of primary interest, and dichotomous ratings for the

secondary exposures. Each exposure had no association, or a weak association with kidney cancer in these data. The estimated relative risk for any above refinery background exposure to NLGD was 1.0 (95% confidence interval 0.5-1.9). Compared with a reference group, three groups appeared to be at increased risk: workers in receipt, storage, and movements; laborers; and unit cleaners, with relative risk of 2.5, 1.9, and 2.3, respectively. Tables, graphs, and 51 references.

■ C. Poole; M. H. Satterfield; L. Levine; K. J. Rothman; N. A. Dreyer (Epidemiology Resources Inc), *API Health & Environmental Sciences Department Report* #4504 (January 1990) (97 p.).

Source: API Publications Order Desk (Order No. I45050)

37-31487

CHO [(Chinese hamster ovary)]/HGPRT [(hypoxanthine-guanine phosphoribosyl transferase)] mutation assay of commercial hexane, Final Report. The mutagenic potential of commercial hexane or its metabolites based on its ability to induce forward mutations at the HGPRT locus of CHO cells was studied. The API's test article, commercial hexane (MBA No. T8497) was tested in the CHO/HGPRT mutation assay in the presence and absence of metabolic activation with Aroclor-induced rat liver S-9. The assay was conducted at dose levels of 0.132, 0.098, 0.063, 0.0362, and 0.0122 µL/mL in both the nonactivated study and in the presence of S-9. These doses induced a range in toxicity, as measured by relative clonal growth (cloning efficiency) 24 hr after exposure, of 2-124% without activation and 4-99% with activation (highest to lowest dose). Under the conditions of these mutagenicity tests, test article commercial hexane was negative in both the presence and absence of exogenous metabolic metabolism.

■ J. W. Harbell C. I. Sigler, *API Health & Environmental Sciences Department Report* (April 1990) (44 p.).

Source: API HESD Information Specialist

37-31827

Absorption of petroleum products across the skin of monkey and man. Final report. A systematic investigation of the percutaneous absorption of petroleum derived chemicals was conducted using animals and human subjects. After a 1 min exposure, total absorption through human palm skin was 0.022 ± 0.008 µL/sq cm for toluene and 0.035 ± 0.016 µL/sq cm for benzene. The values were close enough to justify the use of toluene as a substitute for the known carcinogen benzene in future studies in man. The in-vitro steady-state rate of benzene absorption through human skin from the vapor phase at saturation was 0.017 µL/sq cm/hr at 40% relative humidity. This yielded a permeability coefficient of 0.025 cm/hr which is about one order of magnitude lower than that found by I. Blank in an API supported project. Permeation rates of benzene vapor as determined in vitro in man and in vivo in monkey were greater when the skin was hydrated. Total absorption could be 2.5-7.5 times greater from a 100% relative humidity environment than from a 40% relative humidity environment. Diagram, tables, and graphs.

■ T. J. Franz, *API Health & Environmental Sciences Department Report* (August 1987) (21 p.).

Source: API HESD Information Specialist

37-31830

Subchronic in-vivo cytogenetics assay in rats using nose-only inhalation exposure to commercial hexane. Final report. Male and female Sprague-Dawley rats were exposed nose-only for 6 hr/day for five consecutive days to commercial hexane at 876, 3249, and 8715 ppm. Bone marrow cells, arrested in metaphase by colcemid treatment, were collected at 6 and 24 hr from the midpoint of the last exposure. Metaphase cells were examined microscopically for chromosome aberrations. No statistically significant increases in percentage of aberrant cells were observed in the commercial hexane exposed animals, regardless of exposure concentration or bone marrow harvest time. Under the conditions described, commercial hexane did not induce chromosomal aberrations in bone marrow cells of male or female rats following inhalation exposure for five consecutive days.

■ D. L. Putman (Microbiological Associates Inc), *API Health & Environmental Sciences Department Report* (May 1990) (54 p.).

Source: API HESD Information Specialist

37-31958

A 28-Day dermal toxicity study of API 83-20 [clear liquid test article] in the rabbit was made. The test article, API 83-20 (a petroleum-derived hydrocarbon), was classified as a moderate irritant when administered at dose levels of 2000, 1080, and 540 mg/kg to male and female rabbits. There was minimal irritation in both males and females at 108 mg/kg dosage. The control rabbits were not irritated. Other treatment-related dermal findings in both males and females were flaking and/or cracked skin, leathery skin texture, and scabbing. There were no treatment-related findings in the hematology or clinical chemistry parameters. Increases or decreases in organ weights were non-treatment-related. Treatment-related gross findings were confined to the rabbits' treated skin and consisted of flaking, dryness, redness, and thickened dermis. There was a slight to moderately severe proliferative change with minimal to moderate degree of inflammatory reaction in all male and four female rabbits treated topically with 1080 mg/kg of API 83-20, and mild to marked changes at 2000 mg/kg. API 83-20 had no direct effect on the non-dermal tissues of clipped intact skin of albino rabbits.

■ V. J. Piccirillo; E. M. Dauvin; T. Brewer; B. Quinn; L. Plankenhorn (Tegeris Laboratories Inc), *API Health & Environmental Sciences Department Report* (July 1990) (185 p.).

Source: API HESD Information Specialist

37-32484

In vivo sister chromatid exchange assay API RO-1 raw shale oil. Male and female B6C3F1 mice were exposed to 2.0, 0.75, and 0.2 gm/kg of API RO-1 raw shale oil which was administered as a single intraperitoneal injection 4 hr after subcutaneous implantation of a 50 mg BRdU pellet. Bone marrow cells, arrested in metaphase and collected 24-26 hr after pellet implantation, were examined microscopically for sister chromatid exchanges. Under the conditions of the test, the test article, RO-1 raw shale oil, did not induce sister chromatid exchange in bone marrow cells of B6C3F1 mice. At the time of use the raw shale oil was a dark brown, viscous liquid, and was dissolved in corn oil. The RO-1 raw shale oil was administered to the mice at five dose levels: 6, 4, 2, 1, and zero gm/kg of body weight.

■ *API Health & Environmental Sciences Department Report* (November 1985) (16 p.).

Source: API HESD Information Specialist

37-32485

A 14 day palatability and stability study in rats with benzene administered in drinking water. A study was made to assess the feasibility of administering benzene in drinking water to 30 Fischer 344 rats (5/sex/group) at dose levels of 101, 305, and 700 mg/L for 15 days. Control animals (5/sex) received unadulterated drinking water. Body weight and water consumption measurements were performed on all animals for 1 wk pretest and daily during the treatment period. After 15 days of treatment, all survivors were sacrificed and discarded. All animals had survived the 15 day treatment period and appeared healthy throughout the study. The effect on their body weight suggested that the benzene was consumed. The administration of benzene in drinking water to rats is feasible, provided that a watering system is used that will permit reliable measurement of water consumption.

■ *API Health & Environmental Sciences Department Report* (7/3/90) (210 p.).

Source: API HESD Information Specialist

37-32486

A 14 day palatability and stability study in mice with benzene administered in drinking water. A study was designed to assess the feasibility of administering benzene in drinking water to $B_6C_3F_1$ mice (5/sex/group) at dose levels of 101, 305, and 700 mg/L for 14 days. Control animals (5/sex) received unadulterated drinking water. After 14 days of administration, the animals underwent a 7 day recovery period. Body weight and water consumption measurements were performed on all animals for 1 wk pretest and daily during the treatment and recovery period. After 14 days of treatment and 1 wk of recovery, all survivors were sacrificed and discarded. Based on the tendency for lower body weights in Group IV females it appeared that $B_6C_3F_1$ mice did receive benzene from the drinking water. These results suggested that administration of benzene in

drinking water to mice is feasible, provided a watering system is used which will permit reliable measurement of water consumption.
■ *API Health & Environmental Sciences Department Report* (7/3/90) (285 p.).
Source: API HESD Information Specialist

38-31216
Final Report, April 17, 1991. Commercial hexane test rule, 40 CFR 799.2155...Two-generation reproduction study of inhaled commercial hexane in CD(TM) (Sprague-Dawley) rats was made to evaluate the potential of commercial hexane vapor in CD rats to produce alterations in parental fertility, maternal pregnancy and lactation, and the growth and development of the offspring for two generations, one litter per generation. The study was made in response to the EPA Final Test Rule on commercial hexane (40 CFR 799.2155) and in accordance with Health Effects Testing Guidelines 40 CFR 798.4700, and Good Laboratory Practices 40 CFR 792. CD rat exposure to commercial hexane vapor for two generations resulted in parental toxicity at the target dosage level of 9000 ppm. Perinatal toxicity was concomitant with parental toxicity, being well defined at 9000 ppm. There were no treatment related reproductive effects. The "no observable effect level" for general toxicity in adult animals was 3000 ppm and for offspring was also 3000 ppm, indicating no increased risk to the offspring in absence of indications of adult toxicity. Additional appendices are contained in Volume 2
■ T. L. Neeper-Bradley (Bushy Run Research Center), *API Health & Environmental Sciences Department Report* 1 (4/17/91) (108 p.).
Source: API HESD Information Specialist

38-31217
Final Report, April 17, 1991. Commercial hexane test rule, 40 CFR 799.2155...Two-Generation reproduction study of inhaled commercial hexane in CD(TM) (Sprague-Dawley) rats. This volume supplements Volume 1 [38-31216], and contains appendices.
■ T. L. Neeper-Bradley (Bushy Run Research Center), *API Health & Environmental Sciences Department Report* 2 (4/17/91) (180 p.).
Source: API HESD Information Specialist

39-30623
Neurotoxicity of organic solvents with special reference to the neurobehavioral effects: A [world] literature review through December 1989 covers neurotoxic effects that may occur with short and long term exposure to selected organic solvents, i.e., alcohols, aliphatic hydrocarbons, aromatic hydrocarbons (toluene, benzene, xylene, and styrene), chlorinated hydrocarbons (trichloroethylene, perchloroethylene, methylene chloride, 1,1,1-trichloroethane), and H_2S and CS_2; low-level, chronic exposure to these substances in the workplace, and the specific condition known as the Psycho-Organic Syndrome; identification of neurotoxic illness, especially when organic solvents are involved; solvents with proven human neurotoxicity, such as methanol, n-hexane, and methyl butyl ketone (with or without MEK), toluene (with inhalant abuse only), impure trichloroethylene, and CS_2; and the fact that no solvent has been shown convincingly to produce neurobehavioral effects at the low level exposure generally seen in the workplace. 267 references.
■ N. L. Rosenberg (University of Colorado, School of Medicine); H. H. Schaumburg (Albert Einstein College of Medicine), *API Health & Environmental Sciences Department Report* (February 1992) (68 p.).
Source: API HESD Information Specialist

39-31089
Review of natural resource damage assessments in freshwater environments. Task 2. Effects of oil releases into freshwater habitats. Eighty published studies of known oil spills into the freshwater habitats of rivers, streams, lakes, and ponds documented in oil spill reports and published literature were scrutinized as to oil spill effects on wildlife. Mortalities were frequently high, and in some cases, oil persistence was surprisingly long. In many cases, ecological impacts were often undetectable within the same growing season and impacts appeared to be transient. The faster flowing waters of rivers and creeks tended to readily self-cleanse. Freshwater lakes and other slow-moving waters experienced longer lasting effects, often still evident into the following growing season. The published literature on the occurrence, effects, and chemical characterization of oil spills into fresh waters was very limited, making impact evaluation difficult. For example, there were no published data on the effects of oil spills on aquatic mammals such as muskrat and beaver. Tables and 141 references.
■ J. H. Vandermeulen (Bedford Institute of Oceanography/P. Lane & Associates Ltd), *API Publication* #4514 (January 1992) (125 p.).
Source: API Publications Order Desk (Order No. I45140)

39-31093
Clinical toxicology of the acute ingestion of methanol/hydrocarbon blends. A state-of-the-art review. A review covers the accepted ingestion toxicology of methanol, gasoline, other aliphatic hydrocarbons, and methanol/gasoline blends; the fact that even severely poisoned methanol-intoxicated patients could be expected to survive if they presented themselves within a few hours of ingestion; the major cause of methanol fatalities being the physician's failure to recognize severe poisoning despite the latent period, failure to diagnose methanol intoxication at all, and delayed patient presentation; gastric emptying not being recommended for hydrocarbon ingestions of minimal, or nonsystemic toxicity (kerosine, naphtha, gasoline, mineral spirits, mineral seal oil) even if a large amount is ingested; gastric emptying being recommended only for ingested hydrocarbons known to cause systemic toxicity, and when the amount ingested is possibly sufficient to induce toxicity; and a need to individually evaluate ingestions of hydrocarbon/methanol mixtures. Table and 112 references.
■ T. Litovitz (Georgetown University Hospital), *API Publication* #4524 (July 1991) (55 p.).
Source: API Publications Order Desk (Order No. I45240)

39-31094
Produced water radionuclide hazard/risk assessment. Phase I. A study was made to estimate the risk to human health and the environment from radium isotopes discharged in produced water which, in turn, was discharged into freshwater streams, estuaries, coastal, and outer continental shelf waters. The study included a screening level analysis to determine whether radium discharged to coastal Louisiana in produced waters presented a potential health or environmental risk requiring further study. There was no detectable impact on fish, mollusc, or crustacean populations from radium discharged in produced waters. However, there was a potential risk if a person ingested a large amount of seafood harvested near a produced water point over a lifetime. The number of excess cancers predicted per year under a conservative scenario was comparable to those expected to result from background radium concentrations. Maps, flow diagrams, diagram, tables, graphs, and 100 references.
■ L. D. Hamilton; A. F. Meinhold; J. Nagy (Brookhaven National Laboratory), *API Publication* #4532 (June 1991) (181 p.).
Source: API Publications Order Desk (Order No. I45320)

39-31095
Methanol health effects. Epidemiology literature review and search for study population--1. Critical review of the literature. 2. Search for study population. A review covered identification, retrieval, and evaluation of the available epidemiological literature on studies of individuals exposed to methanol, basically through their occupations, with a focus on describing inhalation and percutaneous absorption exposures rather than on acute ingestion exposure. A study population was suggested for design of an epidemiological study to answer questions posed by the literature review on potential health effects of inhalation and percutaneous exposures to methanol. Epidemiologically, a cohort study would appear to be the study design of choice for a population occupationally exposed to methanol. Teacher aides may be an appropriate study cohort because some of them work with direct-process spirit duplicating machines. The study population is proposed to be Instructional Aides in San Diego County school districts, some exposed to methanol, some to ethanol, and some to neither agent. Map, diagrams, tables, and 96 references.
■ L. K. Hofherr; A. S. Benenson; L. Chan; J. Conway; A. Depeyster; D. Slymen (San Diego State University), *API Publication* #4536 (July 1991) (98 p.).
Source: API Publications Order Desk (Order No. I45360)

39-31651

API Mineral Oil Review. In February 1989, the UK Ministry of Agriculture, Fisheries & Food recommended banning almost all direct food uses of mineral hydrocarbons including white mineral oil. This decision was partly based on results of two 90-day toxicity studies in rats by Shell Research Ltd using white oil made by hydrotreatment and oleum treatment. Oil accumulated in mesenteric lymph nodes and livers, with micro granuloma forming at highest dose levels. Other white mineral oil animal studies did not reveal such effects. Published data showed that white mineral oil absorption has been cited often in animals and humans. Such absorption has apparently not had any adverse effect on humans despite the many years that white mineral oil has been used in pharmaceutical preparations and food. According to the API White Oil Workgroup, most mineral oil data generated so far indicate that current uses can be continued safely in direct and indirect food uses.

■ M. Hulse (Shell Oil Co); M. J. Klan (Amoco Corp); J. M. Noreyko (Lyondell Petrochemical Co); F. A. Reitman (Texaco Inc); C. M. Skisak (Pennzoil Co); J. H. Smith (Exxon Biochemical Sciences Inc); P. G. Tietze (Witco Corp); E. H. Vernot (API), *API Publication* DR 21 (January 1992) (212 p.).

Source: API Publications Order Desk (Order No. I00021)

39-32387

A 90-day feeding study in the rat with six different white mineral oils (N15 (H), N70 (H), N70 (A), P15 (H), N10 (A), and P100 (H)), three different mineral waxes (a low-melting-point wax, a high-melting-point wax, and a high-sulphur wax) and coconut oil. July 1992. This study, sponsored by CONCAWE, was designed to investigate the biological effects of mineral oils and petroleum waxes, representative of those used in food processing and food contact applications. The oil samples were chosen to reflect differences in crude oil source (naphthenic or paraffinic origin), manufacturing method (acid treatment or hyrogenation), and viscosity. The waxes were selected based on the manufacturing method and melting point. Groups of male and female Fischer 344 rats were fed diets containing one of the oils or waxes at dose levels of 0.002-2.0% for 90 days. No treatment related effects were seen with P100 (H) oil, the high-melting-point wax, or the high-sulfur wax. For the other materials, biological effects were observed in liver and mesenteric lymph node. With high doses of some materials, increased kidney and spleen weights and hematological changes were seen. Generally, the N10 (A), P15 (H), and N15 (H) oils and the low-melting-point wax had the greatest effects. Tables.

■ N. R. Worrell (BIBRA Toxicology International), *API Publication* #4560 (July 1992) (443 p.).

Source: API Publications Order Desk (Order No. I45600)

39-33107

Disposition and pharmacokinetics of commercial hexane following IV [(intravenous)] bolus, dermal absorption or nose-only inhalation...Final report. Studies in four volumes were conducted by Research Triangle Institute on male and female F344 rats following IV bolus of radiolabeled n-hexane and methylcyclopentane (the major components of commercial hexane) at 10 mg/kg, single inhalation at 900 and 9000 ppm for 6 hr, 900 ppm exposures for 6 hr/day for eight days, and dermal application for 6 hr at 1.1 and 11 mg/cc. Both were metabolized and excreted within 168 hr of all treatments, mainly through exhaled breath and urine, with metabolites also excreted in the urine. Both were widely distributed to, but were not concentrated significantly by, the body tissues. n-Hexane and its metabolites disappeared from the rats' blood with a half-life of ~ 9-10 hr. No great differences between males and females were noted in the rates and routes of metabolism and excretion of both compounds. Repeated inhalation had no apparent effect on the rates and routes of excretion of the compounds or their metabolites. Chromatograms and tables.

■ R. W. Slauter; A. R. Jeffcoat; G. M. Digsby (Research Triangle Institute), *API Health & Environmental Sciences Department Report* (8/11/92) (1371 p.).

Source: API HESD Information Specialist

39-33438

A mortality study of marketing and marine distribution workers with potential exposure to gasoline in the petroleum industry. This study was conducted to determine the relationship between gasoline exposure and death from kidney cancer or leukemia. There were 9026 workers in the land-based terminal cohort and 9109 in the marine cohort, with potential exposure for at least 1 yr in 1946-85, with respective deaths of 2066 and 2694. A total of 2294 (86%) death certificates were acquired. The land-based cohort had a low overall mortality, with a standardized mortality ratio (SMR) of 51.3:1 vs. 78.6:1 for the marine cohort. There was no increased mortality from kidney cancer or leukemia, with respective SMR of 65.4:1 (12 deaths) and 89.1:1 (27 deaths) among the land-based cohort vs. 83.7:1 (12 deaths) and 70:1 (16 deaths), respectively, among the marine cohort. There was no relationship between mortality from kidney cancer or leukemia and various exposure indices, nor between exposure and mortality from multiple myeloma or heart diseases. Tables. See also Abstract No. 41-31765 and 41-32613.

■ *API Publication* #4555 (October 1992) (151 p.).

Source: API Publications Order Desk (Order No. I45550)

39-33456

Petroleum in the freshwater environment...An annotated bibliography [covering] 1946-1983. This bibliography, derived from a literature review completed for API in 1984 by the Academy of Natural Sciences of Philadelphia, cites literature on the impact of specific petroleum products (e.g., crude oil, fuel oils, kerosine, and gasoline) and oil spill cleanup agents on the freshwater ecosystems, the chemistry and fate of these products and cleanup agents in freshwater, and cleanup methods. This review was prompted by a growing concern over petroleum contamination in freshwater ecosystems. More attention has traditionally been given to contamination of marine and/or brackish water aquatic habitats than to inland waterways because crude oil and refined products transportation has been primarily over open seas, and terminals of origin or destination are usually coastal. However, many ports are located in freshwater or near freshwater locations, and accidents involving inland transport (e.g., inland barges) can result in releases to freshwater systems.

■ *API Publication* #4508 (October 1992) (66 p.).

Source: API Publications Order Desk (Order No. I45080)

40-06424

Odor and taste threshold studies performed with tertiary-amyl methyl ether (TAME). As part of a research program initiated by API to gain basic information on the properties of TAME (an oxygenate component for gasoline alternatives to MTBE), a study was conducted to evaluate the odor detection and recognition thresholds of TAME in air and water, as well as its taste threshold in water. The odor detection threshold is the lowest concentration at which an odor can be noticed and the recognition threshold is the lowest concentration at which an odor can be rated in intensity. The thresholds were determined by a panel of six adult volunteers who "passed" a screening study with butyl alcohol as the odorant. The average odor detection and recognition threshold values for TAME were determined to be, respectively, 0.027 and 0.047 ppm in air and 0.194 and 0.443 ppm in water. The panelists described TAME's odor as sweet, rubbery, fruity, ether-like, and paint-like. The average TAME taste detection threshold value was determined to be 0.128 ppm. The taste of TAME was highly objectionable. Diagrams, tables, and data sheets.

■ K. Vetrano (TRC Environmental Corp), *API Publication* #4591 (September 1993) (31 p.).

Source: API Publications Order Desk (Order No. I45910)

40-30571

API Exposure Classification Scheme for collection of industrial hygiene monitoring data. The API Exposure Classification Scheme was developed to allow API members to have a uniform method for recording industrial hygiene data, and to facilitate API-sponsored data collection study among its members. Data shared among companies may be used for evaluating the impact of proposed regulations or the significance of new health findings, as input for epidemiological studies, and in other areas of study. The scheme's design allows existing systems to be modified to accept the new terms. The scheme addresses only those parameters that are likely to be of greatest importance in interpreting an aggregate grouping of exposure data. The standard terms and their

abbreviations are defined and classified under sampling period, sample type, sampling strategy, operations criteria, sample media; location, job, and task information; analytical method; and other information. Table.
■ *API Publication* #4519 (October 1990) (8 p.).
Source: API Publications Order Desk (Order No. I45190)

40-30722
Monitoring near refineries for airborne chemicals on the SARA [(Superfund Amendments and Reauthorization Act)] Title III Section 313 List--2. A Generic Study Design Protocol. As part of a project conducted by Radian Corp for API to provide an ambient air concentration perspective to the engineering estimates of petroleum refineries required under SARA Title III Section 313, a generic study design protocol was written to provide a step-by-step procedure that could be used by a refinery technical staff person to design an ambient air monitoring project similar to the one conducted by Radian. The protocol focuses on measuring ambient air concentrations of selected target SARA Title III chemicals and other indicator/tracer chemicals at ground level near a refinery fenceline in a single episode representative of that refinery's normal operating conditions, and estimates the extent to which the concentrations of airborne target chemicals are due to fugitive emissions from the refinery. Diagrams, flow diagrams, graphs, tables, maps, and 62 references.
■ *API Publication* #4494 (June 1989) (127 p.).
Source: API Publications Order Desk (Order No. I44940)

40-30723
Monitoring near refineries for airborne chemicals on the SARA [(Superfund Amendments and Reauthorization Act)] Title III Section 313 List--3. Literature survey/Monitoring data for selected chemicals in airborne emissions. A literature survey was conducted to supplement the data generated in an ambient air monitoring program conducted by Radian Corp under contract to API. The survey was to identify publications and data bases which address ambient air concentrations measured near petroleum refineries and community inhalation exposure data for the 25 SARA Title III chemicals targeted in the air monitoring program. A total of 21 relevant documents and citations were identified after review of ~ 1000 citations produced by several successive on-line searches of the scientific and technical literature. Two other potential sources of relevant ambient air data were identified outside of the literature survey. Flow diagrams, tables, and 23 references.
■ *API Publication* #4495 (December 1988) (46 p.).
Source: API Publications Order Desk (Order No. I44950)

40-30724
Monitoring near refineries for airborne chemicals on the SARA [(Superfund Amendments and Reauthorization Act)] Title III Section 313 List--4. A sampling and analytical protocol for selected chemicals in airborne emissions. As part of a project conducted by Radian Corp for API, detailed procedures were identified and recommended to measure selected petroleum refinery emissions in ambient air. For this project, API targeted 25 chemicals from the SARA Title III Section 313 list to be measured at the part-per-billion volume level in ambient air. As no single method could be applied to all the chemicals, they were arranged into four groups of compounds, which could be measured by a single, validated sampling and analytical method. The four groups included aromatic and oxygenated hydrocarbons, halogenated and other electronegative compounds, inorganic acids and ammonia, and metals. In addition, Radian Corp adapted EPA's Method T014, using a passivated stainless steel sampling canister, SUMMA, and GC analysis, for ambient measurement of C_1-C_{10} VOC. Published methods for measuring 1,3-butadiene and MEK in ambient air are also discussed. Diagrams, tables, chromatograms, data sheets, and 35 references.
■ *API Publication* #4496 (December 1988) (163 p.).
Source: API Publications Order Desk (Order No. I44960)

40-30740
Field studies on the reproductive effects of oil and emulsion on marine birds. A 3 yr field study was conducted at a breeding colony in Newfoundland, Canada, where adult Leach's Storm-petrels, *Oceanodroma leucorhoa*, captured during prelaying, prehatching, and posthatching, were either internally dosed via intubation, or externally treated with unweathered Prudhoe Bay crude oil (PBCO) alone, PBCO-dispersant emulsion (a mixture of nonionic and anionic surfactant esters in a glycol-ether solvent), or Hibernia crude oil alone. The birds were then released back into their permanently marked study burrows. Relatively high sublethal doses of crude oils or emulsion significantly reduced hatching and fledging success in a dose-dependent manner. Compared with the untreated controls, the exposed adult petrels were most sensitive to exposure late in incubation and early during posthatching. Treated adults generally showed normal return rates and breeding performance in the second season after exposure. Tables and 108 references.
■ R. G. Butler (Duquesne University), *API Publication* #4466 (October 1988) (103 p.).
Source: API Publications Order Desk (Order No. I44660)

40-30741
Bioaccumulation of polycyclic aromatic hydrocarbons and metals in estuarine organisms. Under laboratory conditions, blue mussels, blue crabs, striped bass, and hogchokers were exposed to various concentrations of phenanthrene (PHE), fluoranthene (FLU), cadmium (Cd), and vanadium for ≤ 96 hr, followed by 96 hr depuration periods. None of the species accumulated metals from the sediments. Blue mussels and hogchokers accumulated both PHE and FLU, with the mussels eliminating PHE more rapidly than FLU, while hogchokers eliminated both at similar rates. The bass rapidly accumulated PHE and FLU, but rapid elimination prevented accumulation over time. Assimilation and metabolism of PHE in blue crabs were rapid, with PHE metabolites accumulating in the muscle of male crabs and hepatopancreas of female crabs. Differences in PAH accumulation and metabolism in different marine species indicate that care must be exercised when extrapolating between species and among PAH compounds when predicting PAH burdens from field data. Tables, graphs, diagram, and 76 references.
■ J. M. O'Connor; K. S. Squibb (New York University Medical Center), *API Publication* #4473 (May 1989) (82 p.).
Source: API Publications Order Desk (Order No. I44730)

40-30807
A survey and analysis of liquid gasoline released to the environment during vehicle refueling at service stations. Data on liquid fuel releases were collected during 10/3-19/88 from 20 Stage 2 and 20 conventional service stations in the Washington, DC, and Baltimore, MD, metropolitan areas, respectively, before, during, and after refueling. Stage 2 facilities are those that use equipment to capture displaced hydrocarbon vapors at the point of vehicle refueling. Of all refueling events, 33 and 37% of the events at Stage 2 and conventional facilities, respectively, were completed without liquid release. Any release consisted of a very small amount (≤ 0.1 g), which could be described as drips. Larger releases (≥ 0.1 g) were more frequent at Stage 2 facilities. When release is expressed in terms of the amount of fuel dispensed, the resultant emission factor is significantly larger at Stage 2 facilities. The statistical significance of this conclusion depends on the larger average amount of fuel dispensed per refueling event at conventional stations. This, in turn, is due in part to the characteristics of dispensing equipment when topping-off is practiced at conventional stations. Tables, graphs, and chart.
■ *API Publication* #4498 (June 1989) (141 p.).
Source: API Publications Order Desk (Order No. I44980)

40-31953
Gasoline vapor exposure assessment at service stations. Clayton Environmental Consultants conducted this assessment for API to characterize the exposure of consumers to airborne gasoline components during self-serve fillup operations. Clayton also collected air samples at the station fenceline, measured local meteorological data, recorded numerous operational parameters, and collected 18 bulk samples of the various grades of gasoline at two service stations each in the Cincinnati, OH; Phoenix, AZ; and Los Angeles, CA, areas in October-November 1990. A total of 120 "integrated" exposure samples representing 1013 customers and 72 fenceline samples were collected, most of which were analyzed for the gasoline constituents n-hexane, BTX, MTBE, and total hydrocarbons, or Option A constituents. Other samples were analyzed for 58 Option B components, e.g., propane, isobutane, n-butane, and

isobutene. Statistical results from the analyses are provided. Tables and diagrams. This supersedes a previous report.
■ *API Publication* #4553 (July 1991) (233 p.).
Source: API Publications Order Desk (Order No. I45530)

40-32424
Analysis of foods for benzene. This Departmental Report presents results of a study, in which a wide variety of foods were analyzed for benzene to verify previous findings of benzene in a number of raw and cooked food materials, and to estimate the significance of food as a source of benzene exposure. The foods were selected to be representative of the fruits, vegetables, meat, fish, and dairy products consumed in the US. They were analyzed for benzene using a modification of EPA's "Purgeable Aromatics, Test Method 602" and GC/photoionization detection. Chromatographic peaks with retention times similar to that of benzene were noted only in red wine, roasted peanuts, fried eggs, baked potatoes, and ripe olives. GC/MS confirmed the presence of benzene in fried eggs and roasted peanuts at very low levels (~ 30 ppm), but not in red wine or baked potatoes. The peak in the ripe olives chromatogram was too small to be checked by GC/MS. Tables, chromatograms, and 16 references.
■ W. Rose (National Food Laboratory Inc), *API Publication* DR 16 (January 1992) (29 p.).
Source: API Publications Order Desk (Order No. I00016)

40-32425
A preliminary study of the effect of toluene on pregnancy [and in utero development] of the rat. (Inhalation exposure). In this assessment of the effects of toluene on the pregnancy and in-utero development of the rat, toluene dosages of 0, 500, 1000, 2000, 3500, and 5000 ppm were administered by inhalation to rat females for 6 hr/day during days 6-15 of pregnancy, inclusive. On day 20, surviving females were sacrificed, subjected to post mortem examination, with liver weights recorded, litter values determined, and fetuses examined externally. Exposure to toluene was associated with clear signs of maternal toxicity at ≥ 2000 ppm. Signs of embryofetal toxicity were observed at 5000 ppm and, to a lesser extent, at 3500 ppm. It is concluded that a suitable high dosage for an ensuing embryofetal toxicity study should not exceed 3500 ppm. Diagrams, tables, and graphs.
■ A. J. Brooker; C. Brennan; D. M. John; D. W. Coombs (Huntington Research Centre Ltd), *API Publication* TR400 (June 1993) (100 p.).
Source: API Publications Order Desk (Order No. I00400)

40-32426
Toluene...The effect on pregnancy [and in utero development] of the rat. (Inhalation exposure). In this assessment of the effects of toluene on pregnancy and in-utero development of the rat, toluene dosages of 0, 250, 1500, and 3000 ppm were administered by inhalation for 6 hr/day during days 6-15 of pregnancy inclusive. On day 20, surviving females were sacrificed and subject to post-mortem examination. Litter values were determined and fetuses were examined for skeletal and visceral changes. Exposure to toluene was associated with a dosage-related maternal response at ≥ 750 ppm. Treatment-related effects of maternal exposure on embryofetal developed were observed at 1500 and 3000 ppm and included exposure-related, but minimal reduction in litter and mean fetal weights, and an increase in fetuses with reduced or unossified sternebrae. It is concluded that exposure to toluene via inhalation does not show any selective effects on embryofetal development. Tables, graphs, and diagrams.
■ A. J. Brooker; C. Brennan; D. M. John; P. C. Kieran; T. J. Kenny; D. W. Combs (Huntington Research Centre Ltd), *API Publication* TR401 (June 1993) (215 p.).
Source: API Publications Order Desk (Order No. I00401)

40-32427
Use of biological monitoring and biomarkers. State-of-the-art review. This Departmental Report was prepared to evaluate the current and potential usefulness of these surveillance methods in assessing exposures and health effects in the petroleum industry. In this review, biological monitoring was defined as measuring internal doses of a xenobiotic substance, and biomarker monitoring was defined as measurement of biologically effective dose through monitoring of DNA and protein adducts. The strengths and limitations of the biological and biomarker monitoring approaches were examined for seven substances encountered in petrochemical operations, i.e., benzene, toluene, xylene, styrene, n-hexane, acrylonitrile, and benzo(a)pyrene. Tables, diagram, graph, and 84 references.
■ J. D. Rench (Battelle Washington Environmental Program Office), *API Publication* DR 349 (August 1990) (56 p.).
Source: API Publications Order Desk (Order No. I00349)

40-32761
Estimation of incremental benzene exposure associated with seven bulk gasoline storage facilities in North Carolina. In 1989, the North Carolina Environmental Management Commission adopted air toxics regulations which require that benzene emissions associated with the operation of a bulk gasoline storage facility shall not result in an incremental increase of > 37 ppt in the ambient benzene concentration at the facility's fenceline. This concentration is equivalent to a one-in-a-million risk of developing leukemia over a 70 yr lifetime, using the interim EPA unit risk (potency) factor. To evaluate the effect that implementation of the standard would have on the health risk of persons living near bulk gasoline storage facilities, an analysis of incremental benzene exposure and potential health risks to populations that reside near seven bulk gasoline storage facilities in North Carolina was performed by using a population exposure model developed by IT Air Quality Services Inc under contract to API. The results of applying the model to all seven facilities are given. Tables and 13 references.
■ T. R. Johnson; R. A. Paul; J. E. Capel (IT Air Quality Services Inc), *API Publication* DR111 (September 1991) (40 p.).
Source: API Publications Order Desk (Order No. I00111)

40-33133
Literature review on the effects of oil and oil dispersants on fishes. This review, intended to contribute to the understanding of the ecological impact of oil spills, provides background information on the effects of crude oil and petroleum products on fish and detailed information on fish chemoreception, including most recent information on crude oil impact on salmonid fish. The review is built on both a developmental and taxonomic basis, including petroleum effects on fish eggs, larvae, juveniles, and adult fish of a variety of species (a total of > 80 different fish). Details of the experimental methodologies and techniques used in biological studies on the effects of oil on fish are also examined. 163 references. (Reprinted in 1993.)
■ R. P. Whitman; E. L. Brannon; R. E. Nakatani (University of Washington, Seattle), *API Publication* DR194 (April 1984) (104 p.).
Source: API Publications Order Desk (Order No. I00194)

41-01257
Odor threshold studies performed with gasoline and gasoline combined with MTBE, ETBE, and TAME. Three blends of gasoline (summer, winter, and a "composite") were evaluated for their odor detection threshold (ODT) and odor recognition threshold (ORT) in air. ODT and ORT, in air, were then determined for mixtures of the summer gasoline with 3, 11, or 15% MTBE, 15% ETBE, or 15% TAME, and of winter or composite gasoline with 15% MTBE. Also determined were ODT and ORT values for neat commercial-grade MTBE (97% pure), both in air and in water, and MTBE taste detection threshold in water. Based on panel evaluation data, the esters are powerful odorants that are significantly reducing both ODT and ORT values of gasoline. The odor thresholds of the three gasolines were similar, 0.474-0.576 ppm. These thresholds were reduced by 54-82% on addition of 15% MTBE. The addition of 15% ETBE or TAME to the summer gasoline reduced its ODT value by 89 and 80%, respectively. The ODT values of summer gasoline mixtures with 15% oxygenates increased in the order ETBE (0.064 ppm), TAME (0.114 ppm), MTBE (0.264 ppm). MTBE alone had ODT of 0.053 ppm in air and 0.045 ppm in water. Tables.
■ *API Publication* #4592 (January 1994) (74 p.).
Source: API Publications Order Desk (Order No. I45920)

41-05416
Closed-patch repeated insult dermal sensitization study of tertiary

amyl methyl ether (TAME) in guinea pigs (Buehler method). This study was conducted for API by Pharmaco LSR Inc to evaluate the allergic contact sensitization potential of TAME in guinea pigs. TAME was administered to 20 Dunkin Harley guinea pigs (10 each of males and females), once a week, for 3 wk (for a total of three induction exposures), by application to saturation (~ 0.3 mL) beneath a Hilltop Chamber(TM). The chamber was occluded and left in place for 6 hr. A total of 20 control animals were similarly treated with light mineral oil (control) or dinitrochlorobenzene (DNCB, positive control). Challenge treatments followed the same administration procedure as the induction phase, but at naive sites. Dermal evaluations were made ~ 24 and 48 hr after the first induction exposure and 24 and 48 hr after the challenge exposure. Under conditions of this study, TAME did not exhibit any potential to produce dermal sensitization in guinea pigs. By contrast, all ten positive control animals treated with 0.3% DNCB exhibited clear dermal responses. Tables.

■ *API Publication* TR403 (10/8/93) (27 p.).
Source: API Publications Order Desk (Order No. I00403)

41-30101

Study of the relationship between folate status and methanol toxicity. A literature review was performed by SRI International for API to evaluate the potential public health impact of increased public exposures to methanol, used as an alternative motor fuel. The delayed phase of methanol toxicity, characterized by metabolic acidosis and ocular damage, was attributed to the accumulation of formate (product of methanol metabolism), and the extent of this toxicity depended on the rate of formate oxidation to CO_2 and water, a reaction mediated by folate (an enzyme present in blood serum). Folate levels vary from one individual to another, and "folate status" has been identified as a key factor in determining individual sensitivity to methanol toxicity. The present study assesses the effect of folate status on methanol toxicity and laboratory animals and humans; the folate status in the US (and Canadian) population; the potential for exacerbation or induction of folate deficiency due to methanol exposure; and identifies research needs for assessing the risk of methanol exposure to sensitive individuals in the US population. Table and 138 references.

■ J. T. MacGregor; C. C. Christensen (SRI International), *API Publication* #4554 (1993) (60 p.).
Source: API Publications Order Desk (Order No. I45540)

41-30735

Gasoline: Insights into the etiology of the development of hepatocellular carcinoma in the mouse. Unleaded gasoline causes an increase in hepatocellular tumors in female mice exposed chronically for a lifetime to gasoline vapors. Current evidence suggests that the induction of female mouse liver tumors may be associated with a decrease in severity of uterine cystic hyperplasia. Liver weights of female mice were slightly increased as early as 3 mo after the inhalation of 2056 ppm gasoline was initiated and remained increased throughout the 24 mo study. The early increase in liver weight was not associated with any pathological changes. The livers of female mice were more consistently affected than those of male mice or of female or male rats. Monkeys exposed for 3 mo were not affected. A total of 17 chemicals (including hexane, MTBE, hydroquinone, chlorodibromomethane, etc.) that cause liver tumors only in female mice were identified, and six of these chemicals were selected for pathological review. A mechanism is postulated that these compounds may be altering the hormonal status of female mice at high doses, affecting the incidence of liver tumors. Tables, graph, and 42 references.

■ J. McGregor (Toxicology Consulting Services), *API Publication* #4598 (February 1994) (84 p.).
Source: API Publications Order Desk (Order No. I45980)

41-31765

An exposure assessment for marketing and marine distribution workers in the petroleum industry with potential exposure to gasoline was conducted as part of an epidemiological study designed to determine if there is a quantitative relationship between long-term exposure to gasoline and risk of cancer. A methodology to estimate historic marketing and marine distribution worker exposures to gasoline was developed and applied to an exposure database for a cohort of ~ 18,000 workers, collected by four participating companies in 1975-85. The cohort was classified into several generic job groups with different histories of gasoline exposure. Exposure intensities associated with specific combinations of job tasks, types of worksites and products were identified, and a full-shift, time-weighted-average exposure was estimated for each job title and worksite type. The frequencies of peak exposures (> 500 ppm for a 15-90 min period) were estimated for different tasks and jobs by worksite types. Cumulative exposure and annual frequency of peak exposures were then derived as dose indexes for epidemiological analysis. Tables, diagrams, chromatograms, and 32 references. See also Abstract No. 39-33438 and 41-32613.

■ T. J. Smith; S. K. Hammond; M. Hallock (University of Massachusetts, Medical Center), *API Publication* #45552 (April 1994) (147 p.).
Source: API Publications Order Desk (Order No. I45552)

41-32131

Dealing with uncertainties in a biologically based risk assessment model of cyclophosphamide-induced leukemogenesis. A biologically-based risk assessment (BBRA) methodology was developed and used to study two key dose-response issues, i.e., the adequacy of cumulative exposure as a dose surrogate and the plausibility of low-dose linearity for dose-response models in the presence of realistic uncertainties. A case study was made of the applicability of BBRA to secondary leukemia induced by the cancer chemotherapeutic agent cyclophospamide (CP). It was shown that BBRA can be used to reject the hypotheses of AUC (area under curve) adequacy and dose-response linearity for CP-induced leukemogenesis, even though data values and knowledge of mechanisms are not fully adequate to determine a unique conventional ("consolidative") risk model. Diagrams, graph, and 23 references.

■ *API Publication* DR70 (May 1994) (93 p.).
Source: API Publications Order Desk (Order No. I00070)

41-32613

A nested case-control study of kidney cancer, leukemia and multiple myeloma in a cohort of land-based terminal workers exposed to gasoline in the petroleum industry. This nested case-control study used data from a cohort mortality study of marketing and marine distribution workers in the petroleum industry with potential exposure to gasoline in 1946-85. The original cohort consisted of 18,135 employees from four petroleum companies. Following an initial cohort study, additional analyses were performed on the mortality (through 6/30/89) among land-based workers of the cohort caused by four diseases, i.e., leukemia (all cell types), acute myeloid leukemia, kidney cancer, and multiple myeloma, with quantitative exposure data available for 35, 13, 13, and 11 cases of the respective diseases. In addition to a generic job classification used in the initial study for exposure assessment, three quantitative exposure indices were used, i.e., length of exposure, cumulative exposure, and frequency of peak exposure. Regression analyses identified no statistically significant excessive mortality from any of the four diseases in any job category, which could be related to gasoline exposure. Tables and 31 references. See also Abstract No. 39-33438 and 41-31765.

■ O. Wong (Applied Health Sciences); L. Trent (ENSR Health Sciences), *API Publication* #45551 (August 1994) (68 p.).
Source: API Publications Order Desk (Order No. I45551)

41-33231

An inhalation oncogenicity study of commercial hexane in rats and mice--1. Rats. Abridged Final Report. During this study, conducted for API, 400 Fisher 344 rats (200 males, 200 females; ~ 8 wk old at initiation of exposure) were exposed to vapors of commercial hexane, administered by whole-body inhalation for 6 hr/day, 5 days/wk, for ~ 24 mo, at target concentrations of 0 (control groups), 900, 3000, or 9016 ppm in air. The commercial hexane contained 51.5% n-hexane, ~ 16% of each methylcyclopentane and 3-methylpentane, ~ 13% 2-methylpentane, and ~ 3% cyclohexane. A decrease in body weight gain and excess lacrimation was found in the 3000 and 9016 ppm exposure groups. Survivorship at 24 mo in the control group was 67% in the males and 76% in the females. Survivorship was not affected by hexane exposure. Localized responses indicative of irritation were seen in the 9000 ppm group in the nasoturbinal tissues and the larynx. The exposures did not produce an

oncogenic effect. In conclusion, under the exposure conditions of this study, commercial hexane is not an oncogen in rats. Tables.
■ *API Publication* TR 404 (4/14/93) (164 p.).
Source: API Publications Order Desk (Order No. I00404)

41-33232
An inhalation oncogenicity study of commercial hexane in rats and mice--2. Mice. Abridged Final Report. During this part of the study, a total of 400 B6C3F1 mice (200 males, 200 females; ~ 8 wk old at initiation of exposure) were exposed for 6 hr/day, 5 days/wk, for 24 mo to 0, 900, 3000, or 9018 ppm of commercial hexane in air. These exposures produced an effect in the females at 9018 ppm, causing an increase in liver mass and nodules and an increase in hepatocellular neoplasm (adenoma and carcinoma) in female. A similar change was not observed among males. There was an increase in the incidence of pituitary proliferative changes (hyperplasia, adenoma, and adenocarcinoma) among all treated groups (≥ 900 ppm) of females, but not among males. The exposure to 9018 ppm hexane reduced mean feed consumption values and significantly lowered the body weight gain after ≥ 29 wk. Survivorship at 24 mo in the control group was 85% in the males and 80% in the females. Survivorship was not affected by the commercial hexane exposure. In conclusion, under the conditions of this study, commercial hexane is an oncogen in mice. See also Abstract No. 41-33231.
■ *API Publication* TR 405 (6/1/93) (114 p.).
Source: API Publications Order Desk (Order No. I00405)

American Petroleum Institute
1220 L Street, Northwest
Washington, D.C. 20005

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