"The study carried out by Environmental Health Associates on behalf of the U.S. Chemical Manufacturers Association is the largest and most informative investigation thus far undertaken."

Sir Richard Doll
Scand J Work Enviorn Health 1988;
14:61-78

GGB 3/15/94

BENEFITS OF UPDATING INTER-INDUSTRY STUDY OF VINYL CHLORIDE WORKERS

- Product Stewardship
  - Demonstrates to workers, community residents, customers, and other audiences our collective commitment to characterize cancer risks associated with employment in VCM/PVC processes.

- Scientific Knowledge
  - Refines the estimate of the number of cases of human angiosarcoma of the liver (ASL) associated with operating VCM/PVC plants in the pre-1972 era in North America.
  - Provides basis for refining human cancer risk assessments which are used by government agencies for permitting facilities, etc.
  - Contributes ASL cases to the international registry.
  - Helps to resolve unanswered questions about alleged links to cancers of the brain, lung, and hematopoeitic system as well as address non-cancer causes of death such as emphysema.

- Litigation Defense
  - VCM/PVC manufacturers continue to face toxic tort litigation alleging that numerous other types (non-ASL) of cancer are related to VCM/PVC employment. This type of research is useful in defending such litigation.

GGB 3/15/94

BENEFITS OF UPDATING INTER-INDUSTRY STUDY OF VINYL CHLORIDE WORKERS

(cont.)

- Chlorine Issue
  - VCM/PVC continue to be a part of the general debate on health and environmental impacts of chlorinated organics. This type of research serves a valuable role in helping to debate the issues on the basis of good science.

CHEMICAL MANUFACTURERS ASSOCIATION

Vinyl Chloride Panel

Vinyl Chloride Research Coordinators

Tentative Agenda

DATE: May 19, 1994

TIME: 10:00 a.m. - 3:00 p.m., EDT

PLACE: CMA Offices
2501 M Street, NW
Washington, D.C.

1.0 Approval of February 14, 1994 Record of Meeting

2.0 Discussion of Scope of Work of the Epidemiology Study

3.0 Discussion of Potential Contractors for the Epidemiology Study

4.0 Discussion of Cost Sharing Formula for the Epidemiology Study

5.0 Presentation by Richard Reitz on Predicting Cancer Risk from Vinyl Chloride Exposure with a Physiologically-Based Pharmacokinetic Model - TENTATIVE

6.0 Discussion of Course of Action with EPA on Vinyl Chloride Risk Assessment

7.0 Discussion of ATSDR Research Data Needs for Vinyl Chloride

8.0 Discussion of Course of Action with ATSDR on Its Priority Data Needs

9.0 Financial Statement

10.0 Schedule Date for Next Meeting or Conference Call

Has Shah

Hasmukh C. Shah, Ph.D.
Manager, Vinyl Chloride Panel

Subject to Approval

ANTITRUST CHECKLIST FOR CMA MEETINGS

This antitrust checklist is for use by CMA staff and member representatives in the conduct of CMA-sponsored meetings. *Prohibited discussion topics apply equally to social gatherings incidental to CMA-sponsored meetings.* The Checklist is not exhaustive and does not address antitrust issues relating to activities other than CMA meetings. Participants in CMA meetings also should be thoroughly familiar with: (1) "Antitrust Guide for CMA Committee Members;" and, (2) "General Principles Applicable to the Structure and Operations of Committees." Both of these documents may be found in the CMA Directory.

DO

Ensure strict performance in areas of:

OVERSIGHT/SUPERVISION:

- Have a CMA staff representative at each CMA-sponsored meeting (unless an exception has been authorized by the appropriate CMA vice-president);
- *consult with an attorney of the CMA Office of General Counsel on all antitrust questions relating to CMA-sponsored meetings;*
- limit meeting discussions to agenda topics (unless additional topics have been approved by the appropriate CMA staff representative); and
- provide each member company representative and CMA staff representative attending a CMA-sponsored meeting with a copy of this checklist, and have a copy available for reference at all CMA-sponsored meetings.

RECORDKEEPING:

- Have an agenda and minutes which accurately reflect the matters which occur;
- provide agendas and minutes to the CMA Office of General Counsel for review and approval in advance of distribution; and,
- fully describe the purposes and authorities of all task groups, work groups, ad hoc or other standing committee subgroups in the minutes of the appropriate parent committee.

VIGILANCE:

- Protest against any discussion or meeting activities which appear to violate this checklist; disassociate yourself from any such discussion or activities and leave any meeting in which they continue.

DON'T

Do not, in fact or appearance, discuss or exchange information on:

PRICES, INCLUDING:

- Individual company prices, price changes, price differentials, markups, discounts, allowances, credit terms, etc.;
- individual company data on costs, production, capacity, inventories, sales, etc.; and,
- industry pricing policies, price levels, price changes, differentials, etc.

PRODUCTION, INCLUDING:

- Plans of individual companies concerning the design, production, distribution or marketing of particular products, including proposed territories or customers; and,
- changes in industry production, capacity or inventories.

TRANSPORTATION RATES:

- Rates or rate policies for individual shipments, including basing point systems, zone prices, freight equalization, etc.

MARKET PROCEDURES, INCLUDING:

- Company bids on contracts for particular products; company procedures for responding to bid invitations; and,
- matters relating to actual or potential individual suppliers or customers that might have the effect of excluding them from any market or influencing the business conduct of firms toward them.

CONSENT DECREE SUBSTANCE:

- Any matter relating to trisodium phosphate (a restriction required by a 1962 consent decree to which CMA is a party).

Revised 3/80 (single page version)
Reformatted 1/89 MDB

CHEMICAL MANUFACTURERS ASSOCIATION

April 15, 1994

Dear Vinyl Chloride Research Coordinators:

The next VCRC meeting is scheduled for May 19, 1994. The agenda for the meeting is enclosed. Also enclosed are: 1) a draft scope of work for an update of the mortality among vinyl chloride workers; 2) an historical perspective on inter-industry vinyl chloride study; and, 3) the benefits of updating the inter-industry study of vinyl chloride workers. Please review these documents in conjunction with Dr. Richard Reitz's abstract on physiologically-based pharmacokinetics model for vinyl chloride and the ATSDR Federal Register notice on vinyl chloride research data needs that were sent to you with my March 30 letter.

I am looking forward to seeing you on May 19.

Sincerely,

Has Shah

Hasmukh C. Shah, Ph.D.
Manager, Vinyl Chloride Panel

Enclosures

2501 M Street, NW, Washington, DC 20037 Telephone 202-887-1100 Fax 202-887-1237

HISTORICAL PERSPECTIVE ON INTER-INDUSTRY VINYL CHLORIDE STUDY

1960-1963	Report published documenting anesthetic effects in animals and humans and liver injury in animals from chronic exposure.
1967	Acroosteolysis reported in humans exposed to high levels of VCM.
1971	Carcinogenicity of VCM discovered in animals, including angiosarcoma of the liver.
1973	CMA sponsors Tabershaw-Cooper Associates to conduct an epidemiologic study of 8,384 vinyl chloride workers from 34 plants.
1974	First reports of angiosarcoma of the liver cases in VCM workers.
1974	Tabershaw-Cooper report preliminary results confirming high risk of angiosarcoma of the liver and suggesting excess cancers of respiratory system, brain and lymphoma.

HISTORICAL PERSPECTIVE ON INTER-INDUSTRY VINYL CHLORIDE STUDY

(cont.)

1974	OSHA holds hearings and revises PEL down to 1 ppm.
1981	Cooper expands original epidemiology study to include 10,173 workers from 37 plants--reports show angiosarcoma and brain cancer to be in excess through 1972.
1986	EHA updates inter-industry study with follow-up through 1982--reports angiosarcoma, brain cancer and emphysema to be in excess--no excess respiratory cancer or lymphoma/leukemia.
1991	EHA study is published.
1993	CMA letter to the editor and EHA response are published clarifying the brain cancer and emphysema findings.
1994	CMA Vinyl Chloride Research Coordinators meet to discuss updating study.

GGB 3/15/94

DIALOG File 159: CANCERLIT(R)_1963-1994/Apr (c) format only 1994 Dialog Info.Svcs.

Enzyme No.: EC 2.4.2.8 .(Hypoxanthine Phosphoribosyltransferase)
Gene Symbol: hprt

01080254 94142776 MEDL/94142776
Detection of 1,2,4-benzenetriol induced aneuploidy and microtubule disruption by fluorescence in situ hybridization and immunocytochemistry.
Zhang L; Venkatesh P; Creek ML; Smith MT
Department of Biomedical and Environmental Health Sciences, School of Public Health, University of California, Berkeley 94720.
Mutat Res; 320(4):315-27 1994 ISSN 0165-1110
Journal Code: NNA
Contract/Grant No.: P42-ES04705, ES, NIEHS; P30-ES01896, ES, NIEHS
Languages: ENGLISH
Document Type: JOURNAL ARTICLE
Journal Announcement: 9404
Subfile: L; M
Fluorescence in situ hybridization (FISH) is becoming increasingly used to detect chromosomal changes in cancer cytogenetics. Here, we report its use in human HL60 cells to detect aneuploidy induced by the benzene metabolite, 1,2,4-benzenetriol (BT). Human centromeric probes specific for chromosomes 9 and 7 were used. Untreated HL60 cells were 0.72 +/- 0.29% hyperdiploid for chromosome 9. Treatment with 5 microM BT increased this level 3-fold to 2.20 +/- 0.87% and 50 microM increased it 4-fold to 2.96 +/- 0.74%. Similar results were obtained with the chromosome 7 probe. The induction of aneuploidy by BT is therefore not chromosome-specific nor is it artifactual. Immunocytochemical staining with anti-tubulin antibodies also showed that BT disrupted microtubule organization at these concentrations. Thus, mitotic spindle disruption probably plays an important role in BT-induced aneuploidy. Trisomy and not tetrasomy accounted for the majority of the hyperdiploidy induced by BT in the two C-group chromosomes 7 and 9. Since trisomy of C-group chromosomes is commonly observed in leukemia, BT-induced aneuploidy may be involved in benzene-induced leukemia.
Tags: Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Major Descriptors: *Aneuploidy; *Chromosomes, Human, Pair 7; *Chromosomes, Human, Pair 9; *Hydroquinones--Toxicity--TO; *Microtubules--Drug Effects--DE
Minor Descriptors: Chromosome Aberrations; Colchicine --Toxicity--TO; Immunohistochemistry; In Situ Hybridization, Fluorescence; Leukemia, Myelocytic, Acute--Genetics--GE; Leukemia, Myelocytic, Acute--Pathology--PA; Tumor Cells, Cultured
CAS Registry No.: 0 .(Hydroquinones); 533-73-3 .(hydroxyhydroquinone); 64-86-8 .(Colchicine)

01080252 94142774 MEDL/94142774
Evaluation of the yeast DEL assay with 10 compounds selected by the International Program on Chemical Safety for the evaluation of short-term tests for carcinogens.
Carls N; Schiestl RH
Department of Molecular and Cellular Toxicology, Harvard School of Public Health, Boston, MA 02115.
Mutat Res; 320(4):293-303 1994 ISSN 0165-1110
Journal Code: NNA
Languages: ENGLISH
Document Type: JOURNAL ARTICLE
Journal Announcement: 9404
Subfile: L; M
The Saccharomyces cerevisiae DEL assay detects a wide variety of nonmutagenic carcinogens (Schiestl et al. (1989) Carcinogenesis, 10, 1445-1455). This study shows the effect on DEL recombination of 8 carcinogenic compounds (o-toluidine, hexamethylphosphoramide, safrole, acrylonitrile, benzene, diethylhexylphthalate, phenobarbital and diethylstilbestrol) and 2 noncarcinogenic compounds (caprolactam and benzoin). These chemicals have been selected by the Program on Chemical Safety for the evaluation of short-term tests for carcinogens, because sufficient carcinogenicity data for these compounds exist, and because they are difficult to detect with the Salmonella assay. 5 of 8 carcinogens reproducibly gave a strong positive response and the noncarcinogen benzoin was negative. Thus, 60% of the chemicals tested in this study have been correctly identified with the DEL assay compared to only 20% with the Salmonella assay.
Tags: Support, U.S. Gov't, Non-P.H.S.
Major Descriptors: *Carcinogens--Toxicity--TO; *Mutagenicity Tests--Methods--MT; *Saccharomyces cerevisiae--Drug Effects --DE
Minor Descriptors: Acrylonitrile--Toxicity--TO; Benzene --Toxicity--TO; Chromosome Deletion; Diethylhexyl Phthalate --Toxicity--TO; Diethylstilbestrol--Toxicity--TO; Hempa --Toxicity--TO; Phenobarbital--Toxicity--TO; Saccharomyces cerevisiae--Genetics--GE; Safrole--Toxicity--TO; Toluidines --Toxicity--TO
CAS Registry No.: 0 .(Carcinogens); 0 .(Toluidines); 107-13-1 .(Acrylonitrile); 117-81-7 .(Diethylhexyl Phthalate); 50-06-6 .(Phenobarbital); 56-53-1 .(Diethylstilbestrol); 680-31-9 .(Hempa); 71-43-2 .(Benzene); 94-59-7 .(Safrole); 95-53-4 .(2-toluidine)

01079808 94140140 MEDL/94140140
[Genetic effects of lead nitrate on seeds of chronically irradiated populations of Arabidopsis thaliana]
Geneticheskie posledstviiu deistviia nitrata svintsa na semena khronicheski obluchaemykh populiatsii Arabidopsis thaliana.
Dineva SB; Abramov VI; Shevchenko VA
Genetika; 29(11):1914-20 1993 ISSN 0016-6758
Journal Code: FNN

(cont. next page)

DIALOG File 159: CANCERLIT(R)_1963-1994/Apr (c) format only 1994 Dialog Info.Svcs.

Tags: Human
Major Descriptors: *Breast Neoplasms--Chemistry--CH; *Proto-Oncogene Proteins--Analysis--AN; *Receptors, Epidermal Growth Factor-Urogastrone--Analysis--AN
Minor Descriptors: Breast Neoplasms--Etiology--ET; Breast Neoplasms--Metabolism--ME; Prognosis; Proto-Oncogene Proteins --Metabolism--ME; Proto-Oncogene Proteins--Physiology--PH; Receptors, Epidermal Growth Factor-Urogastrone--Metabolism--ME ; Receptors, Epidermal Growth Factor-Urogastrone--Physiology --PH
CAS Registry No.: inase); O .(proto-oncogene protein erbB-2) ; O .(Proto-Oncogene Proteins); O .(Receptors, Epidermal Growth Factor-Urogastrone)
Enzyme No.: EC 2.7.1.- .(Epidermal Growth Factor Receptor Protein-Tyrosine K
Gene Symbol: EGFR; neu

01077006 94126838 MEDL/94126838
Carcinogenicity of TCDD in laboratory animals: implications for risk assessment. (177 Refs)
Lucier G; Clark G; Hiermath C; Tritscher A; Sewall C; Huff J
Laboratory of Biochemical Risk Analysis, N.I.E.H.S., Research Triangle Park, NC 27709.
Toxicol Ind Health; 9(4):631-68 1993 ISSN 0748-2337
Journal Code: VWS
Languages: ENGLISH
Document Type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
Journal Announcement: 9404
Subfile: L; M
Tags: Animal; Female; Human; Male
Major Descriptors: *Tetrachlorodibenzodioxin--Toxicity--TO
Minor Descriptors: Biological Assay; Carcinogenicity Tests; Hamsters; Mice; Rats; Risk Factors
CAS Registry No.: 1746-01-6 .(Tetrachlorodibenzodioxin)

01077005 94126837 MEDL/94126837
Effects of acute and subchronic exposure of topically applied fullerene extracts on the mouse skin.
Nelson MA; Domann FE; Bowden GT; Hooser SB; Fernando Q; Carter DE
Pathology Department, University of Arizona, Tucson 85724.
Toxicol Ind Health; 9(4):623-30 1993 ISSN 0748-2337
Journal Code: VWS
Contract/Grant No.: ES05533-02, ES, NIEHS; CA-40584, CA, NCI
Languages: ENGLISH
Document Type: JOURNAL ARTICLE
Journal Announcement: 9404
Subfile: L; M
The recent discovery that fullerenes (C60) can be produced in macroscopic quantities has sparked much interest in the chemistry of this unusual molecule. Concerns have also arisen about the potential carcinogenic effects of this molecule. We have addressed the potential acute and subchronic toxic effec' of fullerenes applied in benzene on the mouse skin. The e toxic effects measured in this study included epidermal DNA synthesis and the induction of ornithine decarboxylase activity in the epidermis. At the topical dose of fullerenes used in these studies (i.e., 200 micrograms), we found no effect on either DNA synthesis or ornithine decarboxylase activity over a 72 hour time course after treatment. The subchronic effects of the fullerenes as a mouse skin tumor promoter was assessed by repeatedly applying the chemical to the skin after initiation with the polycyclic aromatic hydrocarbon, 7,12-dimethylbenzanthracene (DMBA). Repeated administration of the fullerenes for up to 24 weeks post-initiation did not result in either benign or malignant skin tumor formation, whereas promotion with the phorbol ester, 12-O-tetradecanoyl-phorbol- 13-acetate (TPA) resulted in the formation of benign skin tumors. Our data indicate that fullerenes applied in benzene at a likely industrial exposure level do not cause acute toxic effects on the mouse skin epidermis.
Tags: Animal; Female; Support, U.S. Gov't, P.H.S.
Major Descriptors: *Carbon--Toxicity--TO; *Skin --Drug Effects--DE
Minor Descriptors: Administration, Cutaneous; Carbon --Administration and Dosage--AD; Carbon--Pharmacokinetics--PK ; Carcinogenicity Tests; DNA--Biosynthesis--BI; DNA--Drug Effects--DE; Enzyme Induction--Drug Effects--DE; Hyperplasia --Chemically Induced--CI; Mice; Ornithine Decarboxylase --Biosynthesis--BI; Ornithine Decarboxylase--Drug Effects--DE ; Skin Neoplasms--Chemically Induced--CI; Time Factors; 9,10-Dimethyl-1,2-benzanthracene
CAS Registry No.: 115383-22-7 .(fullerene C70); 57-97-6 .(9,10-Dimethyl-1,2-benzanthracene); 7440-44-0 .(Carbon); 9007-49-2 .(DNA); 99685-96-8 .(fullerene C60)
Enzyme No.: EC 4.1.1.17 .(Ornithine Decarboxylase)

01076864 94126081 MEDL/94126081
Review of the genotoxicity of nitrogen oxides.
Victorin K
Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden.
Mutat Res; 317(1):43-55 1994 ISSN 0165-1110
Journal Code: NNA
Languages: ENGLISH
Document Type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
Journal Announcement: 9404
Subfile: L; M
Nitrogen oxides (NOx) are formed in combustion processes and are major pollutants in urban air. Relatively few studies on the genotoxicity of NO2 and NO have been performed. These studies indicate that NO2 is genotoxic in vitro, but the effect of NO seems to be very slight. One in vivo study showed chromosome aberrations and mutations in lung cells after inhalation of NO2 (and NO), but tests for chromosome aberrations in lymphocytes and spermatocytes or micronuclei in bone marrow were negative after inhalation of NO2. Based on present studies, there is no clear evidence of a carcinogenic

(cont. next page)

DIALOG File 40: Enviroline(R)_1970-1993/Jan (c) 1994 CIS, Inc. Information Service.)

SPECIAL FEATURES: 5 graph(s); 14 reference(s); 1 table(s)
MAJOR DESCRIPTORS: CRUSTACEANS; BIOLOGICAL INDICATORS, WATER ; AGE COMPARISONS; DIAZINON; SURFACTANTS; COPPER; CADMIUM; ZINC; SEDIMENT;
REVIEW CLASSIFICATION: O2

00255504 ENVIROLINE NUMBER: 94-00829
Effects of Contaminants from Chromated Copper Arsenate-Treated Lumber on Benthos
Weis, J. S., (Rutgers University, Newark, NJ); Weis, P., (University of Medicine and Dentistry of New Jersey, Newark)
JOURNAL: Arch Environ Contam Toxicol v26, n1, p103(7)
PUBLICATION DATE: Jan 94
DOCUMENT TYPE: research article LANGUAGE: English

ABSTRACT: Toxic chemicals are known to leach from chromated copper arsenate (CCA) -treated bulkheads and adsorb onto fine-grained sediment. Sediment samples were collected in the vicinity of two CCA-treated bulkheads in Florida, and the effects of Cu, chromium, and arsenic at varying distances were investigated on benthic community structure and sediment toxicity. Except for Cu, concentrations of the contaminants were highest immediately adjacent to each bulkhead, and they decreased with distance. Copper and As concentrations were elevated in Neanthes succinea found nearest the CCA-treated wood. In worms living at 1, 3, and 10 m from the bulkheads, Cu and As concentrations decreased with distance. Mortality of amphipods was highest in sediment collected nearest the bulkheads. Species diversity was reduced significantly nearest the bulkheads compared to controls. While the contamination was very localized, the high numbers of CCA-treated bulkheads in coastal waters makes the contamination problem significant. (Full text available from Congressional Information Service.)

SPECIAL FEATURES: 8 graph(s); 1 map(s); 23 reference(s); 1 table(s)
MAJOR DESCRIPTORS: SEDIMENT; COPPER; ARSENIC; CHROMIUM; BENTHIC COMMUNITIES; BIOACCUMULATION, ANIMAL;
REVIEW CLASSIFICATION: O2

00255494 ENVIROLINE NUMBER: 94-00819
Congener-Specific Analysis of Polychlorinated Biphenyls in White-Tailed Sea Eagles Haliaeetus albicilla Collected in Poland
Falandysz, J., University of Gdansk, Poland; Yamashita, N.; Tanabe, S.; Tatsukawa, R.; Rucinska, L.; Mizera, T.; Jakuczun, B.
JOURNAL: Arch Environ Contam Toxicol v26, n1, p13(10)
PUBLICATION DATE: Jan 94
DOCUMENT TYPE: research article LANGUAGE: English

ABSTRACT: PCB congeners were determined in white-tailed sea eagles collected in Poland over the period 1982-90. All samples were analyzed by gas chromatography/mass spectrometry. In breast muscles, the most persistent PCBs were found to be the congeners 153, 138, 180 118, 187, 99, and an unidentified congener. The extremely high PCB congener concentrations in the sea eagles collected from the Baltic coast appeared to be related to relatively high contamination levels in animals of the lower food chain in the area. Apart from PCBs, the sea eagles were also probably highly contaminated with other persistent organochlorines, including highly toxic dioxins and furans, as well as dieldrin. The dioxin toxic equivalents of the coplanar PCBs are presented. The high PCB concentrations found in females were correlated with the high contamination of eggs, and may be associated with eggshell thinning and the low reproduction success of these birds. (Full text available from Congressional Information Service.)

SPECIAL FEATURES: 14 graph(s); 30 reference(s); 3 table(s)
MAJOR DESCRIPTORS: POLAND; POLYCHLORINATED BIPHENYLS; BIOACCUMULATION, BIRD; EAGLES;
REVIEW CLASSIFICATION: O2

00255477 ENVIROLINE NUMBER: 94-00802
Toxic Effects of Pollutants on the Mineralization of Acetate in Methanogenic River Sediment
van Vlaardingen, Peter L. A.; van Beelen, Patrick, National Institute of Public Health and Environ Protection, Bilthoven, Netherlands
JOURNAL: Bull Environ Contam Toxicol v52, n1, p46(8)
PUBLICATION DATE: Jan 94
DOCUMENT TYPE: research article LANGUAGE: English

ABSTRACT: The sediments of the Rhine River, the Netherlands, are highly polluted with organic compounds and heavy metals. The organic matter is converted to acetate by acetogenic bacteria. The mineralization of acetate under methanogenic conditions is then performed by a few specialized methanogenic bacteria. Results are presented from a study on the effect of six toxicants on the anaerobic mineralization of acetate. The toxicants include: benzene, chloroform, 1,2-dichloroethane, pentachlorophenol, mercury, and zinc. Sediment microcosms were prepared using sediment samples from the Rhine River. Highest inhibition of acetate mineralization was found for chloroform, 1,2-dichloroethane, and pentachlorophenol. Benzene was the least toxic of the organic compounds for acetate mineralization. Even at their highest concentrations, Hg and Zn had no observable effects on the anaerobic acetate mineralization. (Full text available from Congressional Information Service.)

SPECIAL FEATURES: 3 graph(s); 22 reference(s); 3 table(s)
MAJOR DESCRIPTORS: SEDIMENT; RHINE RIVER; BENZENE; PENTACHLOROPHENOL; CHLOROFORM; MERCURY; ZINC; BACTERIA; WATER POLLUTION EFFECTS;

(cont. next page)

DIALOG File 40: Enviroline(R)_1970-1993/Jan (c) 1994 CIS, Inc.
SPECIAL FEATURES: 1 diagram(s); 6 reference(s); 8 table(s)
MAJOR DESCRIPTORS: FERTILIZERS, ORGANIC; SEWAGE APPLICATION, LAND; HEAVY METALS; CADMIUM; COPPER; LEAD; BIOACCUMULATION, PLANT;
REVIEW CLASSIFICATION: 02

00255264 ENVIROLINE NUMBER: 94-00525
Increased Risk of Proteinuria Among a Cohort of Lead-Exposed Pregnant Women
Factor-Litvak, Pam, Columbia School of Public Health, New York, NY; Stein, Zena; Graziano, Joseph
JOURNAL: Environ Health Perspec v101, n5, p418(4)
PUBLICATION DATE: Oct 93
DOCUMENT TYPE: research article LANGUAGE: English

ABSTRACT: Exposure to environmental lead and pregnancy outcomes were studied in two Yugoslavian towns to determine risks of proteinuria. Blood lead levels were 17.1 and 5.1 (gr)mg/dl in a smelter town and nonexposed town, respectively. The adjusted odds ratio for proteinuria was 4.5, and for trace proteinuria was 2.3. These results support other studies that found an association between chronic exposure to lead and renal dysfunction manifested as proteinuria. (Full text available from Congressional Information Service.)

SPECIAL FEATURES: 2 graph(s); 41 reference(s); 4 table(s)
MAJOR DESCRIPTORS: TOXICOLOGY; PREGNANCY; BLOOD LEAD LEVEL; KIDNEY DISEASE; SYNERGISTIC EFFECTS;
REVIEW CLASSIFICATION: 02

00255240 ENVIROLINE NUMBER: 94-00499
Identification of 6-Hydroxy-trans,trans-2,4-hexadienoic Acid, a Novel Ring-Opened Urinary Metabolite of Benzene
Kline, Stanley A., University of Medicine and Dentistry, Piscataway, NJ; Robertson, J. Forbes; Grotz, V. Lee; Goldstein, Bernard D.; Witz, Gisela
JOURNAL: Environ Health Perspec v101, n4, p310(3)
PUBLICATION DATE: Sep 93
DOCUMENT TYPE: research article LANGUAGE: English

ABSTRACT: Research has shown that benzene toxicity arises from the formation of metabolites in the liver. One of these metabolites is the open-ringed intermediate trans,trans-muconic acid (MA). One such compound has been identified in the laboratory as the dialdehyde trans,trans-muconaldehyde (MU), which as been shown to be hematotoxic when injected into mice. Two of the endproducts of MUC are MA and 6-hydroxy-trans,trans-hexadienoic acid (HH). Using CD-1 mice treated with MUC, the identification of HHA as a urinary metabolite of benzene is confirmed, showing the intermediacy of MUC in benzene metabolism. (Full text available from Congressional Information Service.)

SPECIAL FEATURES: 3 diagram(s); 14 reference(s); 2 table(s)
MAJOR DESCRIPTORS: BENZENE; BIOLOGICAL INDICATORS; METABOLIC ACTIVATION; CHEMICAL ANALYSIS; BIOACCUMULATION, ANIMAL;
REVIEW CLASSIFICATION: 02

00255071 ENVIROLINE NUMBER: 94-00295
A Survey of Some Waste-to-Energy (W-E) Issues: Ash Residues and Mercury
Shaub, Walter A.
JOURNAL: Solid Waste Assoc of North Am 30th Annu Int Expo, Orlando, FL p395(36)
PUBLICATION DATE: Aug 3-6, 92
DOCUMENT TYPE: conf paper LANGUAGE: English

ABSTRACT: Waste-to-Energy (W-E) facilities are gaining in popularity among communities seeking to reduce landfill utilization while saving energy. A number of issues that must be considered when evaluating designs for W-E facilities are examined, including the need to effectively manage ash and other residues, and the need to manage mercury compounds. The proposed techniques were developed for the express purpose of protecting the environment from undue impacts. To prevent the buildup of contaminants in ashes and residues, it will probably be necessary to screen wastes being introduced into the combustors to weed out potentially dangerous items, such as fluorescent lights and lead batteries. (Full text available from Congressional Information Service.)

MAJOR DESCRIPTORS: SOLID WASTE ENERGY; ENV CONSTRAINTS, SOLID WASTE ENERGY; INCINERATORS; TOXIC SUBSTANCES; MERCURY ; AIR POLLUTION CONTROL; EMISSION CONTROL PROGRAMS; ASH;
REVIEW CLASSIFICATION: 17

00254863 ENVIROLINE NUMBER: 94-00081
Monitoring Heavy Metals in the Gulf of Thailand Using Mussel Watch Approach
Sukasem, Phaka; Tabucanon, Monthip S., Environ Research and Training Center, Bangkok, Thailand
JOURNAL: Sci Total Environ v139-140, p297(9)
PUBLICATION DATE: Nov 1, 93
DOCUMENT TYPE: conf paper LANGUAGE: English

ABSTRACT: Due to rapid industrial development along the major rivers in Thailand that drain into the Gulf of Thailand, marine water concentrations of zinc, manganese, copper, chromium, nickel, and cadmium have increased. Green mussels Perna viridis were collected from ten locations along the gulf coast and analyzed for concentrations of the toxic metals. All mussel samples were analyzed by spectrophotometry. Differences in metal concentrations in mussels from different areas were relatively large for Zn, Mn, Cr, and Cd, but less for Cu and Ni. Highest metal concentrations were found in the regions adjacent to river discharges. When compared to similar data compiled in 1986, metal concentrations had remained relatively unchanged, with the exception of Mn and Ni, which had

(cont. next page)

DIALOG File 40: Enviroline(R)_1970-1993/Jan (c) 1994 CIS, Inc.
increased significantly.

SPECIAL FEATURES: 6 graph(s); 1 map(s); 11 reference(s); 3 table(s)
MAJOR DESCRIPTORS: THAILAND; MUSSELS; HEAVY METALS; BIOACCUMULATION, ANIMAL; BIOLOGICAL INDICATORS, MARINE;
REVIEW CLASSIFICATION: O2

00254860 ENVIROLINE NUMBER: 94-00078
Utilization of Vegetation Sample Bank as an Indicator of Environmental State in the Areas of Industrial Pollution
Kovnatsky, Evgeny F.; Surnin, Valery A., Institute of Experimental Meteorology, Obninsk, Russia
JOURNAL: Sci Total Environ v139-140, p271(7)
PUBLICATION DATE: Nov 1, 93
DOCUMENT TYPE: conf paper LANGUAGE: English

ABSTRACT: In the villages of Ust-Kamenogorsk and Glubokoje in Russia, poplar leaves were studied for their use as biological indicators of industrial pollution. Circular zones were established around the industrial town centers. Multi-element analysis was used to separate the elements according to purely technogenic origin and those derived from soil dust. It was found that calcium, potassium, strontium, lead, iron, and manganese were natural pollutants caused by transport and sedimentation of soil dust, which enters leaf surfaces through the root system. Zinc, Pb, copper, and arsenic were found to be technogenic elements, since their concentration decreased with distance from the industrial center. Besides these toxic elements, it is suggested that mercury, antimony, cobalt, and chromium be included for chemical analysis in the leaves. Overall results indicated that poplar leaves are a good biological indicator of industrial pollution.

SPECIAL FEATURES: 2 reference(s); 3 table(s)
MAJOR DESCRIPTORS: BIOLOGICAL INDICATORS, AIR; BIOACCUMULATION, PLANT; METAL CONTAMINATION; ELEMENTAL ANALYSIS;
REVIEW CLASSIFICATION: O2

00254805 ENVIROLINE NUMBER: 94-00023
Protecting Reproductive Health and the Environment: Toxics Use Reduction
Geiser, Kenneth, Univ of Massachusetts Lowell
JOURNAL: Environ Health Perspec v101, supplement 2, p221(5)
PUBLICATION DATE: Jul 93
DOCUMENT TYPE: conf paper LANGUAGE: English

ABSTRACT: Several states have passed laws that mandate reductions in the use of toxic chemicals as a method of managing hazardous materials. Since some of the regulated compounds are reproductive and developmental toxicants, reductions in use should improve pregnancy outcome. These include lead, mercury, solvents, pesticides, ethylene oxide, PCBs, ethylene glycol ethers, for which use reduction schemes are provided. Such toxicants should be considered priority compounds for use reduction, should be excluded from use as substitutes for other toxic chemicals, and should be phased out. Compensation for workers displaced by plant closings should be considered. Coordination of efforts among environmentalists, business, and government is required. (Full text available from Congressional Information Service.)

SPECIAL FEATURES: 11 reference(s); 2 table(s)
MAJOR DESCRIPTORS: REPRODUCTION, HUMAN; CHEMICAL CONTAMINATION; CHEMICAL REDUCTION; LEGISLATION, STATE AND LOCAL; POLICY-PLANNING, STATE AND LOCAL;
REVIEW CLASSIFICATION: O2

DIALOG File 159: CANCERLIT(R)_1963-1994/Mar (c) format only 1994 Dialog Info.Svcs.

01075247 94120233 MEDL/94120233

[H2 antagonists as inhibitors of cytochrome P-450 in rat liver: in vitro and in vivo effects]

Antagonistas H2, como inhibidores del citocromo P-450 en higado de ratas: efecto in vitro e in vivo.

Carrasco M; Gaule C; Vega P; del Villar E

Servicio de Medicina Interna, Hospital Regional de Copiapo.

Rev Med Chil; 120(5):539-44 1992 ISSN 0034-9887

Journal Code: SHD

Languages: SPANISH

Document Type: JOURNAL ARTICLE English Abstract

Journal Announcement: 9403

Subfile: L

Cytochrome P-50 is a well known participant in the metabolism of xenobiotics as well as an activator or inactivator of hepatotoxic substances and carcinogenic agents. H2 antagonists, cimetidine, famotidine and ranitidine were used to inhibit cytochrome P-450 in rat liver. After 200 mg cimetidine, 85% inhibition of cytochrome P-450 in vitro and 50% in vivo were demonstrated through demethylation of aminopyrine. Inhibition was further confirmed by differential absorption spectra (Type II). The percentage inhibition obtained with famotidine or ranitidine were lower than those obtained with cimetidine. Inhibition of the microsomal oxidative system by cimetidine could lead to decreased production of superoxide radicals and protection against damage induced by toxic agents activated in the liver.

Tags: Animal; Comparative Study; Male

Major Descriptors: *Cytochrome P-450 --Antagonists and Inhibitors--AI; *Histamine H2 Receptor Blockaders --Pharmacology--PD; *Microsomes, Liver--Drug Effects--DE

Minor Descriptors: Administration, Oral; Aminopyrine --Metabolism--ME; Biotransformation--Drug Effects--DE; Cimetidine--Pharmacology--PD; Enzyme Induction--Drug Effects --DE; Famotidine--Pharmacology--PD; Hemeproteins--Metabolism --ME; Methylation--Drug Effects--DE; Microsomes, Liver --Enzymology--EN; Mixed Function Oxidases --Antagonists and Inhibitors--AI; Oxidation-Reduction; Ranitidine--Pharmacology --PD; Rats; Rats, Wistar; Superoxides--Metabolism--ME

CAS Registry No.: 0 .(Hemeproteins); 0 .(Histamine H2 Receptor Blockaders); 11062-77-4 .(Superoxides); 51481-61-9 .(Cimetidine); 58-15-1 .(Aminopyrine); 66357-35-5 .(Ranitidine); 76824-35-6 .(Famotidine); 9035-51-2 .(Cytochrome P-450)

Enzyme No.: EC 1.13.12. .(Mixed Function Oxidases)

01075018 94119131 MEDL/94119131

Evaluation of the Allium anaphase-telophase test in relation to genotoxicity screening of industrial wastewater.

Rank J; Nielsen MH

Department of Environment, Technology and Social Studies, Roskilde University, Denmark.

Mutat Res; 312(1):17-24 1994 ISSN 0165-1110

Journal Code: NNA

Languages: ENGLISH

Document Type: JOURNAL ARTICLE

Journal Announcement: 9403

Subfile: L; M

The Allium anaphase-telophase test was evaluated to find out if it could be recommended in the screening of wastewater for genotoxicity. Five mutagenic or carcinogenic chemicals usually found in wastewater were tested in the Allium anaphase-telophase test. Sodium dichromate (25 microM), benzene (100 microM), dichloromethane (175 microM) and 1,1,1-trichloromethane (175 microM) increased the frequency of chromosome aberrations in the root cells, whereas formaldehyde (1 mM) was found to be non-mutagenic in this test system. Other studies where chemicals were tested in the Allium test were reviewed. For 15 chemicals the results were compared with results from the Ames test, the Microscreen assay, and carcinogenicity tests in rodents. The sensitivity of the Allium test was calculated to be 82%. In conclusion the Allium test is recommended for the screening of wastewater because it has a high sensitivity, is cheap, rapid, easy to handle, and because it can be used on wastewater without pretreatment of the sample.

Major Descriptors: *Allium--Drug Effects--DE; *Industrial Waste--Adverse Effects--AE; *Mutagenicity Tests--Methods--MT; *Mutagens--Toxicity--TO; *Water Pollutants, Chemical--Toxicity --TO

Minor Descriptors: Allium--Genetics--GE; Benzene--Toxicity --TO; Chromates--Toxicity--TO; Chromosome Aberrations; Formaldehyde--Toxicity--TO; Genes, Plant--Drug Effects--DE; Methylene Chloride--Toxicity--TO; Trichloroethanes--Toxicity --TO

CAS Registry No.: 0 .(Chromates); 0 .(Industrial Waste); 0 .(Mutagens); 0 .(Trichloroethanes); 0 .(Water Pollutants, Chemical); 10588-01-9 .(sodium bichromate); 50-00-0 .(Formaldehyde); 71-43-2 .(Benzene); 71-55-6 .(1,1,1-trichloroethane); 75-09-2 .(Methylene Chloride)

01074863 94118541 MEDL/94118541

A randomized phase II study of low-dose cytosine arabinoside (LD-AraC) plus granulocyte-macrophage colony-stimulating factor (rhGM-CSF) in myelodysplastic syndromes (MDS) with a high risk of developing leukemia. EORTC Leukemia Cooperative Group.

Gerhartz HH; Marcus R; Delmer A; Zwierzina H; Suciu S; Dardenne M; Solbu G; de Witte T; Jacobs A; Visani G; et al

Medical Dept. III, Klinikum Grosshadern, Munich, Germany.

Leukemia; 8(1):16-23 1994 ISSN 0887-6924 Journal Code: LEU

Contract/Grant No.: 5U10-C111488-18; 5U10-C111488-19; 5U10-C111488-20; +

Languages: ENGLISH

Document Type: CLINICAL TRIAL; CLINICAL TRIAL, PHASE II; JOURNAL ARTICLE; MULTICENTER STUDY; RANDOMIZED CONTROLLED TRIAL

Journal Announcement: 9403

Subfile: X; L; M

(cont. next page)

DIALOG File 159: CANCERLIT(R)_1963-1994/Mar (c) format only 1994 Dialog Info.Svcs.

showed a similar requirement for MgATP and were equally enhanced by protein kinase C activator 12-O-tetradecanoylphorbol 13-acetate (TPA) but not by kinase A activator 8-bromoadenosine 3',5'-cyclic monophosphate free base. The protein kinase C inhibitor staurosporine blocked the TPA-stimulated component of secretion but had no effect on the NE release in the absence of TPA. Calmidazolium, an inhibitor of calmodulin, inhibited the secretion evoked by both metals to similar extent. Agents interacting with microtubules (colchicine and vinblastine) or microfilaments (cytochalasin B and phalloidin) had no effect on secretion induced by either metal cation. These observations indicate that both Pb2+ and Ca2+ act at a common site and activate the exocytotic release of NE by an analogous mechanism.

Tags: Animal; Comparative Study; Support, U.S. Gov't, P.H.S.

Major Descriptors: *Adrenal Medulla--Physiology--PH; *Calcium--Pharmacology--PD; *Lead--Toxicity--TO; *Norepinephrine--Metabolism--ME

Minor Descriptors: Adenosine Triphosphate--Pharmacology--PD; Adrenal Medulla--Drug Effects--DE; Alkaloids--Pharmacology--PD; Calcium--Metabolism--ME; Calmodulin --Antagonists and Inhibitors--AI; Cattle; Cell Membrane Permeability; Cells, Cultured; Cyclic AMP-Dependent Protein Kinases--Metabolism--ME; Dose-Response Relationship, Drug; Egtazic Acid--Pharmacology--PD; Enzyme Activation; Imidazoles--Pharmacology--PD; Kinetics; Lactate Dehydrogenase--Analysis--AN; Protein Kinase C--Antagonists and Inhibitors--AI; Protein Kinase C--Metabolism--ME; Spectrophotometry, Atomic Absorption; Tetradecanoylphorbol Acetate--Pharmacology--PD; 8-Bromo Cyclic Adenosine Monophosphate--Pharmacology--PD

CAS Registry No.: 0 .(Alkaloids); 0 .(Calmodulin); 0 .(Imidazoles); 16561-29-8 .(Tetradecanoylphorbol Acetate); 23583-48-4 .(8-Bromo Cyclic Adenosine Monophosphate); 51-41-2 .(Norepinephrine); 56-65-5 .(Adenosine Triphosphate); 57265-65-3 .(R 24571); 62996-74-1 .(staurosporine); 67-42-5 .(Egtazic Acid); 7439-92-1 .(Lead); 7440-70-2 .(Calcium)

Enzyme No.: EC 1.1.1.27 .(Lactate Dehydrogenase); EC 2.7.1.- .(Protein Kinase C); EC 2.7.10.- .(Cyclic AMP-Dependent Protein Kinases)

01072196 94105459 MEDL/94105459

Comparing the results of a Monte Carlo analysis with EPA's reasonable maximum exposed individual (RMEI): a case study of a former wood treatment site.

Copeland TL; Paustenbach DJ; Harris MA; Otani J

ChemRisk, a Division of McLaren/Hart, Irvine, California 92714.

Regul Toxicol Pharmacol; 18(2):275-312 1993 ISSN 0273-2300

Journal Code: RBH

Languages: ENGLISH

Document Type: JOURNAL ARTICLE

Journal Announcement: 9403

Subfile: L; M

In the United States, there are about 250 former sites that treated wood with preservatives that are now in need of some degree of remediation. The soil at many of these sites is contaminated with creosote, polycyclic aromatic hydrocarbons (PAHs), polychlorinated dibenzo-p-dioxins (PCDDs), and polychlorinated dibenzofurans (PCDFs). This paper compares the results of the current USEPA point estimate (deterministic) approach for predicting the health risks associated with exposure to PCDDs/PCDFs in soil with the results of a probabilistic approach which uses a Monte Carlo analysis. At many of these wood treatment sites the hazard posed by the PAHs, and especially pentachlorophenol, can be much greater than that due to PCDD and PCDFs; however, because at this site the health risk associated with PAHs was deemed negligible by ATSDR, only PCDDs/PCDFs were evaluated. Octachlorodibenzo-p-dioxin (OCDD) and octachlorodibenzofuran (OCDF) congeners were evaluated independently from the other congeners due to their prevalence in the environment and the availability of congener-specific data. The results of the reevaluation of the rodent bioassay data for 2,3,7,8-TCDD were considered in the probability distribution for the cancer potency factor. The authors' analyses indicate that when assessing exposure to soil via inhalation, ingestion, and dermal contact, the current regulatory approach used to estimate the reasonable maximally exposed individual (RMEI) (USEPA, Risk Assessment Guidance for Superfund, Vol. 1, Part A, 1989) can predict risks which are 10- to 100-fold greater than the 95th percentile risk predicted by a Monte Carlo analysis.

Tags: Animal; Case Report; Comparative Study; Female; Human

Major Descriptors: *Environmental Pollution--Statistical and Numerical Data--SN; *Industry; *Soil Pollutants--Analysis--AN; *Wood

Minor Descriptors: Abnormalities, Drug-Induced--Epidemiology--EP; Algorithms; Benzofurans--Analysis--AN; Benzofurans--Pharmacokinetics--PK; Benzofurans--Toxicity--TO; Dose-Response Relationship, Drug; Environmental Pollution --Adverse Effects--AE; Mice; Monte Carlo Method; Pregnancy; Probability; Risk; Soil Pollutants--Toxicity--TO; Structure-Activity Relationship; Tetrachlorodibenzodioxin --Analogs and Derivatives--AA; Tetrachlorodibenzodioxin--Analysis--AN; Tetrachlorodibenzodioxin--Pharmacokinetics--PK; Tetrachlorodibenzodioxin--Toxicity--TO; United States; United States Environmental Protection Agency

CAS Registry No.: 0 .(chlorinated dibenzofurans); 0 .(polychlorodibenzo-4-dioxin); 0 .(Benzofurans); 0 .(Soil Pollutants); 1746-01-6 .(Tetrachlorodibenzodioxin)

01072032 94104627 MEDL/94104627

Base incorporation and extension at a site-specific ethenocytosine by Escherichia coli DNA polymerase I Klenow fragment.

Simha D; Yadav D; Rzepka RW; Palejwala VA; Humayun MZ

Department of Microbiology and Molecular Genetics, UMD-New Jersey Medical School, Newark 07103-2714.

Mutat Res; 304(2):265-9 1994 ISSN 0165-1110

Journal Code: NNA

Contract/Grant No.: CA47234, CA, NCI

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DIALOG File 159: CANCERLIT(R)_1963-1994/Mar (c) format only 1994 Dialog Info.Svcs.
Languages: ENGLISH
Document Type: JOURNAL ARTICLE
Journal Announcement: 9403
Subfile: L; M

Ethenocytosine (epsilon C) is a highly mutagenic exocyclic DNA lesion induced by carcinogens vinyl chloride and urethane. We have examined base incorporation and extension at a site-specific epsilon C residue by a quantitative gel electrophoretic assay using an exonuclease-deficient version of Escherichia coli DNA polymerase I (Klenow fragment) as the model enzyme. The data show that the KM for incorporation of adenine or thymine opposite epsilon C by is about 5 orders of magnitude higher than that for the incorporation of guanine opposite normal cytosine. The KM for base extension past epsilon C:A and epsilon C:T pairs is 1-2 orders of magnitude higher than that observed for a C:G pair. Although adenine misinsertion is favored over that of thymine, base extension occurs more readily when the base incorporated opposite epsilon C is thymine.

Tags: Support, U.S. Gov't, P.H.S.

Major Descriptors: *Cytosine--Analogs and Derivatives--AA; *DNA--Metabolism--ME; *DNA Damage; *DNA Polymerase I--Genetics --GE; *Mutagenesis, Site-Directed; *Mutagens--Metabolism--ME

Minor Descriptors: Alkylating Agents--Metabolism--ME; Alkylating Agents--Toxicity--TO; Base Composition; Base Sequence; Cytosine--Chemistry--CH; Cytosine--Metabolism--ME; Cytosine--Toxicity--TO; DNA--Chemistry--CH; DNA Polymerase I --Metabolism--ME; Escherichia coli--Enzymology--EN; Escherich ia coli--Genetics--GE; Molecular Sequence Data; Mutagens --Toxicity--TO; Oligodeoxyribonucleotides--Metabolism--ME; Templates

CAS Registry No.: 0 .(Alkylating Agents); 0 .(Mutagens); 0 .(Oligodeoxyribonucleotides); 0 .(3,N(4)-ethenocytosine); 71-30-7 .(Cytosine); 9007-49-2 .(DNA)

Enzyme No.: EC 2.7.7.- .(DNA Polymerase I)

01071935 94104039 MEDL/94104039

Tobacco-specific N-nitrosamines and Areca-derived N-nitrosamines: chemistry, biochemistry, carcinogenicity, and relevance to humans.

Hoffmann D; Brunnemann KD; Prokopczyk B; Djordjevic MV

American Health Foundation, Valhalla, New York 10595.

J Toxicol Environ Health; 41(1):1-52 1994 ISSN 0098-4108 Journal Code: KAA

Contract/Grant No.: CA-29580, CA, NCI; CA-17613, CA, NCI

Languages: ENGLISH

Document Type: JOURNAL ARTICLE; REVIEW; REVIEW, ACADEMIC

Journal Announcement: 9403

Subfile: X; L; M

Nicotine and the minor tobacco alkaloids give rise to tobacco-specific N-nitrosamines (TSNA) during tobacco processing and during smoking. Chemical-analytical studies led to the identification of seven TSNA in smokeless tobacco (< or = 25 micrograms/g) and in mainstream smoke of cigarettes (1.3 micrograms TSNA/cigarette). Indoor air polluted by tobacco smoke may contain up to 24 pg/L of TSNA. In mice, rats, and hamsters, three TSNA, N'-nitrosonornicotine (NNN), 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL), are powerful carcinogens; two TSNA are moderately active as carcinogens; and two TSNA appear not to be carcinogenic. The TSNA are procarcinogens, agents that require metabolic activation. The active forms of the carcinogenic TSNA react with cellular components, including DNA, and with hemoglobin (Hb). The Hb adducts in chewers and smokers serve as biomarkers for the uptake and metabolic activation of carcinogenic TSNA and the urinary excretion of NNAL as free alcohol and as glucuronide for the uptake of TSNA. The review presents evidence that strongly supports the concept that TSNA contribute to the increased risk for cancer of the upper digestive tract in tobacco chewers and for the increased risk of lung cancer, especially pulmonary adenocarcinoma, in smokers. The high incidence of cancer of the upper digestive tract especially among men on the Indian subcontinent has been causally associated with chewing of betel quid mixed with tobacco. In addition to the TSNA, the betel quid chewers are exposed to four N-nitrosamines that are formed during chewing from the Areca alkaloids, two of these N-nitrosamines are carcinogens. The article also reviews approaches toward the reduction of the carcinogenic potency of smokeless tobacco, betel quid-tobacco mixtures, and cigarette smoke. Although the safest way to reduce the risk for tobacco-related cancers is to refrain from chewing and smoking, modifications of smokeless tobacco and of cigarettes are indicated to lead to less toxic products. Another more recent approach for reducing the carcinogenic effect of tobacco products is the application of chemopreventive agents, primarily of micronutrients. Future aspects in tobacco carcinogenesis, especially as it relates to TSNA, are expected in the field of molecular biochemistry and in biomarker studies, with the goal of identifying those tobacco and betel quid chewers and tobacco smokers who are at especially high risk for cancer. (250 Refs)

Tags: Animal; Human; Support, U.S. Gov't, P.H.S.

Major Descriptors: *Areca--Chemistry--CH; *Carcinogens --Toxicity--TO; *Neoplasms--Chemically Induced--CI; *Nitrosami nes--Toxicity--TO; *Tobacco--Chemistry--CH

Minor Descriptors: Carcinogens--Analysis--AN; Carcinogens --Chemistry--CH; Hamsters; Mice; Neoplasms --Prevention and Control--PC; Nitrosamines--Analysis--AN; Nitrosamines --Chemistry--CH; Rats; Tobacco Smoke Pollution --Adverse Effects--AE; Tobacco Smoke Pollution--Analysis--AN; Tobacco, Smokeless--Adverse Effects--AE; Tobacco, Smokeless--Chemistry --CH

CAS Registry No.: 0 .(Carcinogens); 0 .(Nitrosamines); 0 .(Tobacco, Smokeless)

01069997 94094449 MEDL/94094449

The intensity of vanadium(V)-induced cytotoxicity and morphological transformation in BALB/3T3 cells is dependent on glutathione-mediated bioreduction to vanadium(IV).

(cont. next page)

DIALOG File 159: CANCERLIT(R)_1963-1994/Mar (c) format only 1994 Dialog Info.Svcs.

Sabbioni E; Pozzi G; Devos S; Pintar A; Casella L; Fischbach M

Commission of the European Communities, Environment Institute, Joint Research Centre-Ispra Site, Varese, Italy.

Carcinogenesis; 14(12):2565-8 1993 ISSN 0143-3334

Journal Code: C9T

Languages: ENGLISH

Document Type: JOURNAL ARTICLE

Journal Announcement: 9403

Subfile: X; L; M

Cytotoxicity and morphological transformation has been studied in BALB/3T3 Cl A31-1-1 mouse embryo cells for ammonium vanadate [vanadium(V)] and vanadyl sulphate [vanadium(IV)] alone or in combination with diethylmaleate (DEM), a cellular glutathione (GSH)-depleting agent. Cells exposed for 24 h to 10(-5) M vanadium(V) alone or in combination with 3 x 10(-6) M DEM showed the characteristic hyperfine EPR signal of vanadium(IV), which was more obvious in the case of exposure to vanadium(V) alone. This suggests that the amount of vanadium(V) reduced to vanadium(IV) decreased in GSH-depleted cells. While vanadium(IV) at concentrations of 3 x 10(-6) M and 10(-5) M was not transforming in the cells, vanadium(V) showed neoplastic transforming activity ($P < 0.025$ and $P < 0.001$ for the two doses, respectively) in comparison to controls (vanadium unexposed cells). Cytotoxicity and morphological transformation in cells exposed to vanadium(V) in combination with 3 x 10(-6) M DEM were significantly more intensive ($P < 0.005$ and $P < 0.01$ for the two doses of vanadate tested) compared to the corresponding values observed in cells exposed to vanadium(V) alone. This suggests that the final transforming activity response is dependent on the intracellular GSH-mediated mechanism of reduction of vanadium(V) to vanadium(IV): (i) the extent to which vanadium(V) should be bioreduced to less toxic vanadium(IV) via intracellular GSH is a key point in determining the intensity of the observed neoplastic action; (ii) the carcinogenic potential of vanadium(V) should be strictly dependent on its intracellular persistence which could lead to changes in normal metabolic patterns of vanadium(V) in the oxidized form due to lack of GSH-mediated reduction.

Tags: Animal

Major Descriptors: *Cell Transformation, Neoplastic--Drug Effects--DE; *Glutathione--Metabolism--ME; *Vanadium Compounds --Toxicity--TO

Minor Descriptors: Biotransformation; Cell Survival--Drug Effects--DE; Electron Spin Resonance Spectroscopy; Maleates --Pharmacology--PD; Mice; Mice, Inbred BALB C; Oxidation-Reduction; Vanadium Compounds--Metabolism--ME; 3T3 Cells

CAS Registry No.: 0 .(Maleates); 0 .(Vanadium Compounds); 141-05-9 .(diethyl maleate); 27774-13-6 .(vanadyl sulfate); 70-18-8 .(Glutathione)

01069982 94094434 MEDL/94094434

Benzene and phenol metabolism by mouse and rat liver microsomes.

Schlosser PM; Bond JA; Medinsky MA

Chemical Industry Institute of Toxicology, Research Triangle Park, NC 27709.

Carcinogenesis; 14(12):2477-86 1993 ISSN 0143-3334

Journal Code: C9T

Languages: ENGLISH

Document Type: JOURNAL ARTICLE

Journal Announcement: 9403

Subfile: X; L; M

Benzene, an important industrial solvent and constituent of unleaded gasoline, causes leukemia and aplastic anemia in humans. Mice are more sensitive than rats to benzene toxicity, though neither species has been shown to respond consistently with benzene-induced leukemia. Benzene biotransformation in liver to phenol, hydroquinone, catechol and/or muconaldehyde is thought to be necessary for its hematotoxicity and/or genotoxicity. Our goal is to develop a mathematical simulation model capable of describing the pathways and kinetics of benzene metabolism by rat and mouse liver microsomes and to assess the role of species metabolic differences in species sensitivity. Microsomes were incubated with 4 microM [U-14C]-benzene or 4 microM [U-14C]phenol. Metabolite production was quantified by extraction into ethyl acetate, HPLC separation and liquid scintillation spectroscopy. After 45 min, mouse liver microsomes converted 20% of the benzene to phenol, 31% to hydroquinone and 2% to catechol. Rat liver microsomes converted 23% of benzene to phenol, 8% to hydroquinone and 0.5% to catechol. Production of hydroquinone and catechol continued for 90 min for mouse liver microsomes, while production by rat liver microsomes had virtually ceased by 90 min. Muconic acid production by mouse liver microsomes was < 0.2% and < 0.04% from benzene and phenol respectively after 90 min. A quantitative simulation model was constructed to describe the in vitro metabolism of benzene, incorporating the reaction sequences: benzene-->phenol-->catechol-->trihydroxybenzene and phenol-->hydroquinone-->trihydroxybenzene. In the model, all of the reaction steps are assumed to be catalyzed by the same enzyme(s), cytochrome(s) P450, and benzene, phenol, hydroquinone and catechol in solution are all assumed to compete, through reversible binding, for the same reaction site(s) on cytochrome(s) P450. The simulation model accurately described both the benzene and phenol kinetic data, supporting this proposed mechanism. In particular, this model suggests that the observed inhibition of benzene on phenol metabolism, and of phenol on benzene metabolism, occurs through competition for a common reaction site, which can also bind catechol and hydroquinone.

Tags: Animal; In Vitro; Male; Support, Non-U.S. Gov't

Major Descriptors: *Benzene--Metabolism--ME; *Microsomes, Liver--Metabolism--ME; *Phenols--Metabolism--ME

Minor Descriptors: Biotransformation; Mice; Models, Biological; Rats; Rats, Inbred F344

(cont. next page)

DIALOG File 159: CANCERLIT(R)_1963-1994/Mar (c) format only 1994 Dialog Info.Svcs.

CAS Registry No.: 0 .(Phenols); 108-95-2 .(phenol); 71-43-2 .(Benzene)

01069781 94093257 MEDL/94093257

[Handling of red silica gravel on sports, play and resort surfaces with special reference to new toxicologic results]

Umgang mit Kieselrot auf Sport-, Spiel- und Freizeitflachen unter Berucksichtigung neuer toxikologischer Untersuchungserge bnisse.

Heudorf U

Gesundheitsamt der Stadt Frankfurt am Main.

Gesundheitswesen; 55(10):521-6 1993 ISSN 0941-3790 Journal Code: BFD

Languages: GERMAN

Document Type: JOURNAL ARTICLE English Abstract

Journal Announcement: 9403

Subfile: L

In 1991 "Kieselrot", a material with extremely high PCDD/PCDF-contamination up to about 200,000 ng TEQ/kg was detected on many leisure centres and sports fields in Germany. The contaminated grounds were closed, and covered up. The following examinations to prepare the planned decontamination are described here. The need for decontamination of those "Kieselrot"-areas is discussed with special reference to its relevance on behalf of environmental toxicology and preventive medicine. Risk estimates, based on mathematical models had shown a considerable health and cancer risk for the users of such play- and sports grounds. However, recent studies conducted with sportsmen, groundsmen, and residents after many years of contact with "Kieselrot"-covered grounds did not reveal any additional PCDD/PCDF blood fat burden. But raised TEQ-levels and a typical congeneric pattern pointing to a "Kieselrot"-load could be seen in some of the examined children. Following these results it was concluded that children play grounds contaminated with "Kieselrot" should remain closed and should be decontaminated, whereas sports fields could be used again. Another possibility could be the adaption of the decontamination concepts and methods to the proven low bioavailability of PCDD/PCDF from "Kieselrot".

Tags: Female; Human; Male

Major Descriptors: *Benzofurans--Analysis--AN; *Environmental Pollutants--Analysis--AN; *Polymers--Analysis--AN; *Soil Pollutants--Analysis--AN; *Tetrachlorodibenzodioxin --Analogs and Derivatives--AA

Minor Descriptors: Adolescence; Adult; Aged; Aged, 80 and over; Benzofurans--Toxicity--TO; Body Burden; Child; Child, Preschool; Infant; Leisure Activities; Maximum Permissible Exposure Level; Middle Age; Play and Playthings; Polymers --Toxicity--TO; Sports; Tetrachlorodibenzodioxin--Analysis--AN; Tetrachlorodibenzodioxin--Toxicity--TO

CAS Registry No.: 0 .(polychlorodibenzo-4-dioxin); 0 .(polychlorodibenzofuran); 0 .(Benzofurans); 0 .(Environmental Pollutants); 0 .(Polymers); 0 .(Soil Pollutants); 1746-01-6 .(Tetrachlorodibenzodioxin)

01069668 94092366 MEDL/94092366

Methods development toward the measurement of polyaromatic hydrocarbon-DNA adducts by mass spectrometry.

Giese RW; Vouros P

Department of Medicinal Chemistry, Northeastern University, Boston, MA 02115.

Res Rep Health Eff Inst; (61):1-25; discussion 27-36 1993 ISSN 1041-5505 Journal Code: AH8

Languages: ENGLISH

Document Type: JOURNAL ARTICLE

Journal Announcement: 9403

Subfile: L; M

The measurement of DNA adducts in human samples is at an early stage. The accuracy of some of the current measurements is not defined, the structures are unknown for a significant number of the adducts that have been detected, and there is little information about how many adducts remain to be discovered. This is due largely to the trace amounts of human DNA adducts in any sample. A consequence of this is that the true potential of DNA adducts as indicators of exposure and risk in human toxicology is far from realized. Mass spectrometry, a powerful technique for organic analysis, is the key to exploiting fully the usefulness of human DNA adducts as biomarkers of human exposure and risk. Mass spectrometry can make accurate measurements, discover unknown compounds, and determine the structures of these unknown compounds. However, the trace (very small) amounts of human DNA adducts have limited mass spectrometry's usefulness in analyzing such samples. This project focused on increasing the sensitivity of mass spectrometry for measuring human DNA adducts. Advances in sensitivity have been achieved for two modes of mass spectrometry applied to standards related to DNA adducts: gas chromatography with electron-capture negative ion mass spectrometry, and fast-atom-bombardment mass spectrometry. These advances involve both sample preparation and instrument conditions.

Tags: Animal; Human; Support, U.S. Gov't, Non-P.H.S.

Major Descriptors: *DNA--Analysis--AN; *DNA--Chemistry--CH; *DNA Damage; *Polycyclic Hydrocarbons--Analysis--AN; *Polycyclic Hydrocarbons--Chemistry--CH; *Spectrum Analysis, Mass

Minor Descriptors: Benzene--Chemistry--CH; Benzo(a)pyrene --Analogs and Derivatives--AA; Benzo(a)pyrene--Chemistry--CH; Carcinogens, Environmental--Chemistry--CH; Chromatography, Gas --Methods--MT; Chromatography, High Pressure Liquid; Deoxyadenosines--Chemistry--CH; Deoxyguanosine --Analogs and Derivatives--AA; Deoxyguanosine--Chemistry--CH; Electrophoresis--Methods--MT; Fluorenes--Chemistry--CH; Hydrazines --Chemistry--CH; Hydrogenation; Oxidation-Reduction; Pyrenes --Chemistry--CH; Spectrometry, Mass, Fast Atom Bombardment; Spectrum Analysis, Mass--Methods--MT; Superoxides--Chemistry --CH; 2-Acetylaminofluorene--Chemistry--CH

CAS Registry No.: 0 .(benzo(a)pyrene-DNA adduct); 0 .(Carcinogens, Environmental); 0 .(Deoxyadenosines); 0 .(Fluorenes); 0 .(Hydrazines); 0 .(Polycyclic Hydrocarbons);

(cont. next page)

DIALOG File 55: BIOSIS PREVIEWS(R)_1985-1994/MAR ISS 12 (c) 1994 BIOSIS

Concept Codes:
*00512 . General Biology-Conservation, Resource Management
*22506 . Toxicology-Environmental and Industrial Toxicology
*37015 . Public Health: Environmental Health-Air, Water and Soil Pollution
10059 . Biochemical Methods-Minerals

10932578 BIOSIS Number: 97132578
Modeling the concentrations of gas-phase toxic organic air pollutants: Direct emissions and atmospheric formation
Harley R A; Cass G R
Environ. Eng. Sci. Dep., Calif. Inst. Technology, Pasadena, CA 91125, USA
Environmental Science & Technology 28 (1). 1994. 88-98.
Full Journal Title: Environmental Science & Technology
ISSN: 0013-936X
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 006 Ref. 082471

An Eulerian photochemical air quality model is described for the prediction of the atmospheric transport and chemical reactions of gas-phase toxic organic air pollutants. Model performance was examined in the Los Angeles, CA (USA), area over the period August 27-28, 1987. The organic compounds were drawn from a list of 189 species selected for control as hazardous air pollutants in the Clean Air Act amendments of 1990. The species considered include benzene, various alkylbenzenes, phenol, cresols, 1,3-butadiene, acrolein, formaldehyde, acetaldehyde, and perchloroethylene among others. It is found that photochemical generation contributes significantly to formaldehyde, acetaldehyde, acetone, and acrolein concentrations for the 2-day period studied. Phenol concentrations are dominated by direct emissions, despite the existence of a pathway for atmospheric formation from benzene oxidation. The finding that photochemical production can be a major contributor to the total concentrations of some toxic organic species implies that control programs for those species must consider more than just direct emissions.
Descriptors/Keywords: RESEARCH ARTICLE; AIR QUALITY MODEL; PHENOL; 1,3-BUTADIENE; ACROLEIN; FORMALDEHYDE; ACETALDEHYDE; PERCHLOROETHYLENE; ACETONE
Concept Codes:
*07504 . Ecology; Environmental Biology-Bioclimatology and Biometeorology
*22506 . Toxicology-Environmental and Industrial Toxicology
*37015 . Public Health: Environmental Health-Air, Water and Soil Pollution
10050 . Biochemical Methods-General

10932564 BIOSIS Number: 97132564
Toxicity, Bioavailability and metal speciation
Jonnalagaddá S B; Rao P V V P
Dep. Chemistry, University Zimbabwe, Box MP 167, Mt. Pleasant, Harare, RHO
Comparative Biochemistry and Physiology C Comparative Pharmacology and Toxicology 106 (3). 1993. 585-595.
Full Journal Title: Comparative Biochemistry and Physiology C Comparative Pharmacology and Toxicology
ISSN: 0742-8413
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 006 Ref. 082440

1. Environmental toxicology emphasizes the difference from traditional toxicology in which pure compounds of interest are added to purified diets, or injected into the test animals. When the objective is to study the fate and effects of trace elements in the environment, knowledge of the speciation of the elements and their physico-chemical forms is important. 2. Cadmium salts such as the sulfides, carbonates or oxides, are practically insoluble in water. However, these can be converted to water-soluble salts in nature under the influence of oxygen and acids. Chronic exposure to Cd is associated with renal toxicity in humans once a critical body burden is reached. 3. The solubility of As(III) oxide in water is fairly low, but high in either acid or alkali. In water, arsenic is usually in the form of the arsenate or arsenite. As(III) is systemically more poisonous than the As(V), and As(V) is reduced to the As(III) form before exerting any toxic effects. Organic arsenicals also exert their toxic effects in vivo in animals by first metabolizing to the trivalent arsenoxide form. Some methyl arsenic compounds, such as di- and trimethylarsines, occur naturally as a consequence of biological activity. The toxic effect of arsenite can be potentiated by dithiols, while As has a protective effect against the toxicity of a variety of forms of Se in several species. 4. Selenium occurs in several oxidation states and many selenium analogues of organic sulfur compounds exist in nature. Selenium in selenate form occurs in alkaline soils, where it is soluble and easily available to plants. Selenite binds tightly to iron and aluminum oxides and thus is quite insoluble in soils. Hydrogen selenide is a very toxic gas at room temperature. The methylated forms of Se are much less toxic for the organism than selenite. However, the methylated Se derivatives have strong synergistic toxicity with other minerals such as arsenic. 5. Aquatic organisms absorb and retain Hg in the tissues, as methylmercury, although most of the environmental Hg to which they are exposed is inorganic. The methylmercury in fish arises from the bacterial methylation of inorganic Hg. Methylmercury in the human diet is almost completely absorbed into the bloodstream. The nervous system is the principal target tissue affected by methylmercury in adult human beings, while kidney is the critical organ following the ingestion of Hg(II) salts.
Descriptors/Keywords: LITERATURE REVIEW; HUMAN; PLANT; FISH; CADMIUM; ARSENIC; SELENIUM; METHYLMERCURY; ENVIRONMENTAL EXPOSURE; RENAL; NERVOUS SYSTEM TOXICITY
Concept Codes:
*13010 . Metabolism-Minerals
*15506 . Urinary System and External Secretions-Pathology
*20506 . Nervous System-Pathology

(cont. next page)

DIALOG File 55: BIOSIS PREVIEWS(R)_1985-1994/MAR ISS 12 (c) 1994 BIOSIS
86190 . Primates-Unspecified
86375 . Muridae
Super Taxa:
Animals; Chordates; Vertebrates; Nonhuman Vertebrates; Mammals; Nonhuman Mammals; Primates; Nonhuman Primates; Rodents

10926075 BIOSIS Number: 97126075
Structure-activity studies of human tumour necrosis factors
Van Ostade X; Tavernier J; Fiers W
Inst. Med. Vet. Sci., Div. Human Immunol., PO Box 14 Rundle Mall Post Office, Adelaide 5000, South Australia, AUL
Protein Engineering 7 (1). 1994. 5-22.
Full Journal Title: Protein Engineering
ISSN: 0269-2139
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 006 Ref. 075947

The mechanism by which tumour necrosis factors (TNF and lymphotoxin, also called TNF-alpha and TNF-beta respectively) exert their cytotoxic activity on many malignant cells, remains largely unknown. Furthermore, the broad array of differentiation (gene induction) and mitogenic activities towards many primary cells is still a subject of intensive investigation. TNF is an important mediator inflammation, immune responses and infection-related phenomena and these activities contribute to the severe toxicity seen when TNF is used as an anticancer agent. The first step in the mechanism of action is the specific binding of the ligand to its receptors and dissection of the molecular mechanism involved in this interaction is the subject of this review. The reasons for the interest in this aspect are obvious: first, the development of strong antagonistic TNF analogues can be useful in dampening the potentially lethal or debilitating effects of an overproduction of the cytokine (as in septic shock or rheumatoid arthritis). Secondly, since two distinct TNF receptors exist, construction of TNF muteins that distinguish between both types may lead to derivatives of this pleiotropic agent with a more restricted biological activity pattern. Ideally, one would like to develop a TNF mutant that has retained its cytotoxic action on tumour cells without inducing the deleterious systemic toxicity. Such an optimized TNF molecule could become a potent anticancer agent.

Descriptors/Keywords: LITERATURE REVIEW; MOUSE; TUMOR NECROSIS FACTOR; HORMONE-DRUG; ANTINEOPLASTIC-DRUG; PHARMACODYNAMICS; TUMOR NECROSIS FACTORS; LYMPHOTOXIN; INFLAMMATION; ACTION MECHANISM; MOLECULAR SEQUENCE DATA; AMINO ACID SEQUENCE; PROTEIN ENGINEERING

Concept Codes:
*10010 . Comparative Biochemistry, General
*10064 . Biochemical Studies-Proteins, Peptides and Amino Acids
*10506 . Biophysics-Molecular Properties and Macromolecules
*12508 . Pathology, General and Miscellaneous-Inflammation and Inflammatory Disease
*12512 . Pathology, General and Miscellaneous-Therapy (1971-)
*15008 . Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
*17002 . Endocrine System-General
*22005 . Pharmacology-Clinical Pharmacology (1972-)
*22016 . Pharmacology-Endocrine System
*24008 . Neoplasms and Neoplastic Agents-Therapeutic Agents; Therapy
10068 . Biochemical Studies-Carbohydrates

Biosystematic Codes:
86215 . Hominidae
86375 . Muridae
Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans; Nonhuman Vertebrates; Nonhuman Mammals; Rodents

10925475 BIOSIS Number: 97125475
Benzene and phenol metabolism by mouse and rat liver microsomes
Schlosser P M; Bond J A; Medinsky M A
Chemical Industry Inst. Toxicol., 6 Davis Dr., PO Box 12137, Research Triangle Park, NC 27709, USA
Carcinogenesis (Oxford) 14 (12). 1993. 2477-2486.
Full Journal Title: Carcinogenesis (Oxford)
ISSN: 0143-3334
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 006 Ref. 075344

Benzene, an important industrial solvent and constituent of unleaded gasoline, causes leukemia and aplastic anemia in humans. Mice are more sensitive than rats to benzene toxicity, though neither species has been shown to respond consistently with benzene-induced leukemia. Benzene biotransformation in liver to phenol, hydroquinone, catechol and/or muconaldehyde is thought to be necessary for its hematoxicity and/or genotoxicity. Our goal is to develop a mathematical simulation model capable of describing the pathways and kinetics of benzene metabolism by rat and mouse liver microsomes and to assess the role of species metabolic differences in species sensitivity. Microsomes were incubated with 4-mu-M (U-14C)-benzene or 4-mu-M (U-14C)phenol. Metabolite production was quantified by extraction into ethyl acetate, HPLC separation and liquid scintillation spectroscopy. After 45 min, mouse liver microsomes converted 20% of the benzene to phenol, 31% to hydroquinone and 2% to catechol. Rat liver microsomes converted 23% of benzene to phenol, 8% to hydroquinone and 0.5% to catechol. Production of hydroquinone and catechol continued for 90 min for mouse liver microsomes, while production by rat liver microsomes had virtually ceased by 90 min. Muconic acid production by mouse liver microsomes was lt 0.2% and lt 0.04% from benzene and phenol respectively after 90 min. A quantitative simulation model was constructed to describe the in vitro metabolism of benzene, incorporating

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DIALOG File 55: BIOSIS PREVIEWS(R)_1985-1994/MAR ISS 12 (c) 1994 BIOSIS

the reaction sequences: benzene fwdarw phenol fwdarw catechol fwdarw trihydroxybenzene and phenol fwdarw hydroquinone fwdarw trihydroxybenzene. In the model, all of the reaction steps are assumed to be catalyzed by the same enzyme(s), cytochrome(s) P450, and benzene, phenol, hydroquinone and catechol in solution are all assumed to compete, through reversible binding, for the same reaction site(s) on cytochrome(s) P450. The simulation model accurately described both the benzene and phenol kinetic data, supporting this proposed mechanism. In particular, this model suggests that the observed inhibition of benzene on phenol metabolism, and of phenol on benzene metabolism, occurs through competition for a common reaction site, which can also bind catechol and hydroquinone.

Descriptors/Keywords: RESEARCH ARTICLE; HUMAN; CARCINOGENS; LEUKEMIA

Concept Codes:
*02506 . Cytology and Cytochemistry-Animal
*13002 . Metabolism-General Metabolism; Metabolic Pathways
*14004 . Digestive System-Physiology and Biochemistry
*15006 . Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and Reticuloendothelial Pathologies
*15008 . Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
*22501 . Toxicology-General; Methods and Experimental
*24006 . Neoplasms and Neoplastic Agents-Biochemistry
*24007 . Neoplasms and Neoplastic Agents-Carcinogens and Carcinogenesis
*24010 . Neoplasms and Neoplastic Agents-Blood and Reticuloendothelial Neoplasms
10060 . Biochemical Studies-General
32600 . In Vitro Studies, Cellular and Subcellular

Biosystematic Codes:
86215 . Hominidae
86375 . Muridae

Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans; Nonhuman Vertebrates; Nonhuman Mammals; Rodents

10925453 BIOSIS Number: 97125453

The intensity of vanadium-(V)-induced cytotoxicity and morphological transformation in BALB-3T3 cells is dependent on glutathione-mediated bioreduction to vanadium-(IV)

Sabbioni E; Pozzi G; Devos S; Pintar A; Casella L; Fischbach M

Commission European Communities, Environment Inst., Joint Res. Centre Ispra Site, 21020 Ispra, ITL

Carcinogenesis (Oxford) 14 (12). 1993. 2565-2568.
Full Journal Title: Carcinogenesis (Oxford)
ISSN: 0143-3334
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 006 Ref. 075348

Cytotoxicity and morphological transformation has been studied in BALB/3T3 Cl A31-1-1 mouse embryo cells for ammonium vanadate (vanadium(V)) and vanadyl sulphate (vanadium (IV)) alone or in combination with diethylmaleate (DEM), a cellular glutathione (GSH)-depleting agent. Cells exposed for 24 h to 10-5 M vanadium(V) alone or in combination with 3 times 10-6 M DEM showed the characteristic hyperfine EPR signal of vanadium (IV), which was more obvious in the case of exposure to vanadium(V) alone. This suggests that the amount of vanadium(V) reduced to vanadium(IV) decreased in GSH-depleted cells. While vanadium(IV) at concentrations of 3 times 10-6M and 10-5M was not transforming in the cells, vanadium(V) showed neoplastic transforming activity (P lt 0.025 and P lt 0.001 for the two doses, respectively) in comparison to controls (vanadium unexposed cells). Cytotoxicity and morphological transformation in cells exposed to vanadium (V) in combination with 3 times 10-6 M DEM were significantly more intensive (P lt 0.005 and P lt 0.01 for the two doses of vanadate tested) compared to the corresponding values observed in cells exposed to vanadium(V) alone. This suggests that the final transforming activity response is dependent on the intracellular GSH-mediated mechanism of reduction of vanadium(V) to vanadium(IV): (i) the extent to which vanadium(V) should be bioreduced to less toxic vanadium(IV) via intracellular GSH is a key point in determining the intensity of the observed neoplastic action; (ii) the carcinogenic potential of vanadium(V) should be strictly dependent on its intracellular persistence which could lead to changes in normal metabolic patterns of vanadium(V) in the oxidized form due to lack of GSH-mediated reduction.

Descriptors/Keywords: RESEARCH ARTICLE; MOUSE FIBROBLASTS; AMMONIUM VANADATE; VANADYL SULFATE; CARCINOGENS; DIETHYLMALEATE; METABOLIC-DRUG

Concept Codes:
*02506 . Cytology and Cytochemistry-Animal
*13010 . Metabolism-Minerals
*13012 . Metabolism-Proteins, Peptides and Amino Acids
*22003 . Pharmacology-Drug Metabolism; Metabolic Stimulators
*22501 . Toxicology-General; Methods and Experimental
*24005 . Neoplasms and Neoplastic Agents-Neoplastic Cell Lines
*24006 . Neoplasms and Neoplastic Agents-Biochemistry
*24007 . Neoplasms and Neoplastic Agents-Carcinogens and Carcinogenesis
10060 . Biochemical Studies-General
10064 . Biochemical Studies-Proteins, Peptides and Amino Acids
10069 . Biochemical Studies-Minerals
18004 . Bones, Joints, Fasciae, Connective and Adipose Tissue-Physiology and Biochemistry
32500 . Tissue Culture, Apparatus, Methods and Media

Biosystematic Codes:
86375 . Muridae

Super Taxa:
Animals; Chordates; Vertebrates; Nonhuman Vertebrates; Mammals; Nonhuman Mammals; Rodents

DIALOG File 55: BIOSIS PREVIEWS(R)_1985-1994/MAR ISS 12 (c) 1994 BIOSIS
Super Taxa:
Animals; Chordates; Vertebrates; Nonhuman Vertebrates; Mammals; Nonhuman Mammals; Carnivores; Primates; Humans

10917672 BIOSIS Number: 97117672
Sinus histiocytosis of pelvic lymph nodes after hip replacement: A histiocytic proliferation induced by cobalt-chromium and titanium
Albores-Saavedra J; Vuitch F; Delgado R; Wiley E; Hagler H
Div. Anat. Pathol., Dep. Pathol., UT-Southwestern Med. Cent., 5323 Harry Hines Blvd., Dallas, TX 75235-9072, USA
American Journal of Surgical Pathology 18 (1). 1994. 83-90.
Full Journal Title: American Journal of Surgical Pathology
ISSN: 0147-5185
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 006 Ref. 067550
Descriptors/Keywords: RESEARCH ARTICLE; HUMAN; TOXICITY; IMMUNOHISTOCHEMISTRY
Concept Codes:
*02508 . Cytology and Cytochemistry-Human
*10511 . Biophysics-Bioengineering
*15006 . Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and Reticuloendothelial Pathologies
*15008 . Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
*18006 . Bones, Joints, Fasciae, Connective and Adipose Tissue-Pathology
01056 . Microscopy Techniques-Histology and Histochemistry
10069 . Biochemical Studies-Minerals
11316 . Chordate Body Regions-Pelvis (1970-)
Biosystematic Codes:
86215 . Hominidae
Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans

10915693 BIOSIS Number: 97115693
Twenty-eight-day repeated dose toxicity tests of O-nitrotoluene in Wistar rats
Kaneko T; Shimo T; Yasuhara K; Hirose A; Ogawa Y; Suzuki S; Nakaji Y; Kurokawa Y
Div. Toxicol., Biol. Safety Res. Cent., Natl. Inst. Health Sci., JAP
Journal of Toxicological Sciences 18 (4). 1993. 422.
Full Journal Title: 20th Annual Meeting of the Japanese Society of Toxicological Sciences, Chiba, Japan, July 29-30, 1993. Journal of Toxicological Sciences
ISSN: 0388-1350
Language: ENGLISH
Document Type: CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 003 Ref. 044586
Descriptors/Keywords: MEETING PAPER; LIVER; SPLEEN; HEMOLYTIC ANEMIA; NITROBENZENE TOXICITY
Concept Codes:
*14006 . Digestive System-Pathology
*15006 . Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and Reticuloendothelial Pathologies
*15008 . Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
*22501 . Toxicology-General; Methods and Experimental
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10060 . Biochemical Studies-General
Biosystematic Codes:
86375 . Muridae
Super Taxa:
Animals; Chordates; Vertebrates; Nonhuman Vertebrates; Mammals; Nonhuman Mammals; Rodents

10914606 BIOSIS Number: 97114606
Density, diversity and incidence of deformities of benthic invertebrates near a vinyl chloride discharge point source in the Niagara River watershed
Dickman M D; Rygiel G
Biol. Sci. Dep., Brock Univ., St. Catharines, ON L2S 3A1, CAN
0 (0). 1993. 663-680.
Full Journal Title: Gorsuch, J. W., et al. (Ed.). ASTM (American Society for Testing and Materials) Special Technical Publication, 1216. Environmental toxicology and risk assessment, 2nd volume; Symposium on Environmental Toxicology and Risk Assessment: Aquatic, Plant, and Terrestrial, Pittsburgh, Pennsylvania, USA, April 26-30, 1992. xii+730p. American Society for Testing and Materials (ASTM): Philadelphia, Pennsylvania, USA. ISBN 0-8031-1485-0.
ISSN: 0066-0558
Language: ENGLISH
Document Type: BOOK; CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 003 Ref. 043499
Descriptors/Keywords: BOOK CHAPTER; MEETING PAPER; CHIRONOMID; ENVIRONMENTAL TOXICITY; TERATOGENICITY
Concept Codes:
*07508 . Ecology; Environmental Biology-Animal
*07514 . Ecology; Environmental Biology-Limnology
*22506 . Toxicology-Environmental and Industrial Toxicology
*25503 . Developmental Biology-Embryology-Pathological
*25552 . Developmental Biology-Embryology-Descriptive Teratology and Teratogenesis
*64078 . Invertebrata, Comparative and Experimental Morphology, Physiology and Pathology-Insecta-Pathology
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals

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DIALOG File 55: BIOSIS PREVIEWS(R)_1985-1994/MAR ISS 12 (c) 1994 BIOSIS
10060 . Biochemical Studies-General
Biosystematic Codes:
75314 . Diptera
Super Taxa:
Animals; Invertebrates; Arthropods; Insects

10914597 BIOSIS Number: 97114597
Metal accumulation in blood and milk of dairy cows grazed or fed by fodder grown on a sewage water disposal site
Varadarajan K; Paliwal K; Rajamanickam C
Sch. Biol. Sci., Madurai Kamaraj Univ., Madurai 625 021, IND
0 (0). 1993. 510-520.
Full Journal Title: Gorsuch, J. W., et al. (Ed.). ASTM (American Society for Testing and Materials) Special Technical Publication, 1216. Environmental toxicology and risk assessment, 2nd volume; Symposium on Environmental Toxicology and Risk Assessment: Aquatic, Plant, and Terrestrial, Pittsburgh, Pennsylvania, USA, April 26-30, 1992. xii+730p. American Society for Testing and Materials (ASTM): Philadelphia, Pennsylvania, USA. ISBN 0-8031-1485-0.
ISSN: 0066-0558
Language: ENGLISH
Document Type: BOOK; CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 003
Ref. 043490
Descriptors/Keywords: BOOK CHAPTER; MEETING PAPER; LEAD; IRON; ZINC; ENVIRONMENTAL TOXICITY
Concept Codes:
*13010 . Metabolism-Minerals
*15002 . Blood, Blood-Forming Organs and Body Fluids-Blood and Lymph Studies
*16506 . Reproductive System-Pathology
*22506 . Toxicology-Environmental and Industrial Toxicology
*25502 . Developmental Biology-Embryology-General and Descriptive
*26504 . Animal Production-Feeds and Feeding
*37014 . Public Health: Environmental Health-Sewage Disposal and Sanitary Measures
*38004 . Veterinary Science-Pathology
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10069 . Biochemical Studies-Minerals
13518 . Food Technology-Dairy Products
Biosystematic Codes:
85715 . Bovidae
Super Taxa:
Animals; Chordates; Vertebrates; Nonhuman Vertebrates; Mammals; Nonhuman Mammals; Artiodactyls

10914575 BIOSIS Number: 97114575
A short-term test for dioxin teratogenicity using chicken embryos
Henshel D S; Hehn B M; Vo M T; Steeves J D
Indiana Univ. Sch. Public Environ. Affairs, Bloomington, IN 47405, USA
0 (0). 1993. 159-174.
Full Journal Title: Gorsuch, J. W., et al. (Ed.). ASTM (American Society for Testing and Materials) Special Technical Publication, 1216. Environmental toxicology and risk assessment, 2nd volume; Symposium on Environmental Toxicology and Risk Assessment: Aquatic, Plant, and Terrestrial, Pittsburgh, Pennsylvania, USA, April 26-30, 1992. xii+730p. American Society for Testing and Materials (ASTM): Philadelphia, Pennsylvania, USA. ISBN 0-8031-1485-0.
ISSN: 0066-0558
Language: ENGLISH
Document Type: BOOK; CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 003
Ref. 043468
Descriptors/Keywords: BOOK CHAPTER; MEETING RAPER; ENVIRONMENTAL TOXICITY TESTING METHOD
Concept Codes:
*22501 . Toxicology-General; Methods and Experimental
*22506 . Toxicology-Environmental and Industrial Toxicology
*25503 . Developmental Biology-Embryology-Pathological
*25552 . Developmental Biology-Embryology-Descriptive Teratology and Teratogenesis
*37015 . Public Health: Environmental Health-Air, Water and Soil Pollution
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10060 . Biochemical Studies-General
Biosystematic Codes:
85536 . Galliformes
Super Taxa:
Animals; Chordates; Vertebrates; Nonhuman Vertebrates; Birds

10911371 BIOSIS Number: 97111371
Enhanced fatty-acid synthase (FAS) activity in hypertrophic alveolar type II cells from silica-treated rats: Studies on function and messenger RNA levels
Rami J; Stenzel W; Puel-M'Rini C; Besombes J P; Rooney S A
INSERM CJF 9107, 31054 Toulouse, FRA
Molecular Biology of the Cell 4 (SUPPL.). 1993. 335A.
Full Journal Title: Thirty-third Annual Meeting of the American Society for Cell Biology, New Orleans, Louisiana, USA, December 11-15, 1993. Molecular Biology of the Cell
ISSN: 1059-1524
Language: ENGLISH
Document Type: CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 003
Ref. 040264
Descriptors/Keywords: MEETING ABSTRACT; MEETING POSTER; TOXICITY; CHOLINE PHOSPHATE CYTIDYLYLTRANSFERASE
Concept Codes:
*02506 . Cytology and Cytochemistry-Animal

(cont. next page)

DIALOG File 55: BIOSIS PREVIEWS(R)_1985-1994/MAR ISS 12 (c) 1994 BIOSIS

10908624 BIOSIS Number: 97108624
Volatilizing toxic metals from soil
Clifford D A; Chen S-S; Reznik C; Hampton M
Dep. Civil and Environ. Eng., Univ. Houston, Houton, TX 77204-4791, USA
Waste Management 13 (5-7). 1993. 520.
Full Journal Title: Symposium on Emerging Technologies: Metals, Oxidation, and Separation, Belmont, Texas, USA, February 25-26, 1993. Waste Management
ISSN: 0956-053X
Language: ENGLISH
Document Type: CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 003 Ref. 037517
Descriptors/Keywords: MEETING ABSTRACT; LEAD; LEAD SULFIDE; LEAD SULFATE; LEAD NITRATE; LEAD CARBONATE; LEAD OXIDE; CADMIUM; MERCURY; ZINC; ARSENIC; SELENIUM
Concept Codes:
*22506 . Toxicology-Environmental and Industrial Toxicology
*37014 . Public Health: Environmental Health-Sewage Disposal and Sanitary Measures
*37015 . Public Health: Environmental Health-Air, Water and Soil Pollution
*52805 . Soil Science-Physics and Chemistry (1970-)
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10059 . Biochemical Methods-Minerals

10908606 BIOSIS Number: 97108606
Volatilizing toxic metals from soil
Clifford D A; Chen S-S; Reznik C
Dep. Civil and Environ. Eng., Univ. Houston, Houston, TX 77204-4791, USA
Waste Management 13 (5-7). 1993. 467-479.
Full Journal Title: Symposium on Emerging Technologies: Metals, Oxidation, and Separation, Belmont, Texas, USA, February 25-26, 1993. Waste Management
ISSN: 0956-053X
Language: ENGLISH
Document Type: CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 003 Ref. 037499
Descriptors/Keywords: MEETING PAPER; LEAD SULFATE; LEAD CARBONATE; LEAD NITRATE; LEAD; CADMIUM; MERCURY; ZINC; ARSENIC; SELENIUM; BATTERY WASTE SITE
Concept Codes:
*22506 . Toxicology-Environmental and Industrial Toxicology
*37014 . Public Health: Environmental Health-Sewage Disposal and Sanitary Measures
*37015 . Public Health: Environmental Health-Air, Water and Soil Pollution
*52805 . Soil Science-Physics and Chemistry (1970-)
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10059 . Biochemical Methods-Minerals

10905778 BIOSIS Number: 97105778
Generation of reactive oxygen from the copper-mediated oxidation of the benzene metabolite, 1,4-hydroquinone: Role in DNA damage
Li Y; Kuppusamy P; Zweier J L; Trush M A
Johns Hopkins Med. Inst., Baltimore, MD, USA
Free Radical Biology & Medicine 15 (5). 1993. 540.
Full Journal Title: 1st Annual Meeting of the Oxygen Society on Oxygen, Charleston, South Carolina, USA, November 12-17, 1993. Free Radical Biology & Medicine
ISSN: 0891-5849
Language: ENGLISH
Document Type: CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 003 Ref. 034671
Descriptors/Keywords: MEETING ABSTRACT; TOXICITY
Concept Codes:
*13002 . Metabolism-General Metabolism; Metabolic Pathways
*13010 . Metabolism-Minerals
*22501 . Toxicology-General; Methods and Experimental
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10012 . Biochemistry-Gases (1970-)
10060 . Biochemical Studies-General
10062 . Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10069 . Biochemical Studies-Minerals
Biosystematic Codes:
33000 . Animalia-Unspecified
Super Taxa:
Animals

10904133 BIOSIS Number: 97104133
Leukemia induced by chemicals
Snyder R
Joint Graduate Program Toxicol., Environmental Occupational Health Sci. Inst., Rutgers State Univ. New Jersey/UMDNJ-Robert Wood Johnson Med. Sch., 681 Frelinghuysen Road, Piscataway, NJ 08855-1179, USA
Annals of Hematology 67 (SUPPL.). 1993. A119.
Full Journal Title: Annual Meeting of the German and the Austrian Society of Hematology and Oncology, Essen, Germany, October 10-13, 1993. Annals of Hematology
ISSN: 0939-5555
Language: ENGLISH
Document Type: CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 003 Ref. 033026
Descriptors/Keywords: MEETING ABSTRACT; HUMAN; BENZENE; ENVIRONMENTAL TOXICITY; APLASTIC ANEMIA; ANALYTICAL METHOD

(cont. next page)

DIALOG File 55: BIOSIS PREVIEWS(R)_1985-1994/MAR ISS 12 (c) 1994 BIOSIS
Concept Codes:
*15001 . Blood, Blood-Forming Organs and Body Fluids-General; Methods
*15006 . Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and Reticuloendothelial Pathologies
*15008 . Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
*22506 . Toxicology-Environmental and Industrial Toxicology
*24007 . Neoplasms and Neoplastic Agents-Carcinogens and Carcinogenesis
*24010 . Neoplasms and Neoplastic Agents-Blood and Reticuloendothelial Neoplasms
*37015 . Public Health: Environmental Health-Air, Water and Soil Pollution
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10050 . Biochemical Methods-General
10060 . Biochemical Studies-General
Biosystematic Codes:
86215 . Hominidae
Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans

10901738 BIOSIS Number: 97101738
Blood lead levels and neuropsychologic function in elderly women
Muldoon S B; Cauley J A; Kuller L; Scott J
Univ. Pittsburgh, Pittsburgh, PA 15261, USA
American Journal of Epidemiology 138 (8). 1993. 644-645.
Full Journal Title: Twenty-sixth Annual Meeting of the Society for Epidemiologic Research, Keystone, Colorado, USA, June 16-18, 1993. American Journal of Epidemiology
ISSN: 0002-9262
Language: ENGLISH
Document Type: CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 003
Ref. 030631
Descriptors/Keywords: MEETING ABSTRACT; HUMAN; TOXICITY; EPIDEMIOLOGY; DIAGNOSIS; PATHOLOGY; COGNITIVE FUNCTION; PSYCHOMOTOR SPEED; SENSORIMOTOR FUNCTION
Concept Codes:
*15006 . Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and Reticuloendothelial Pathologies
*20506 . Nervous System-Pathology
*21002 . Psychiatry-Psychopathology; Psychodynamics and Therapy
*22501 . Toxicology-General; Methods and Experimental
*24500 . Gerontology
*37054 . Public Health: Epidemiology-Organic Diseases and Neoplasms
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10060 . Biochemical Studies-General
10069 . Biochemical Studies-Minerals
12512 . Pathology, General and Miscellaneous-Therapy (1971-)
Biosystematic Codes:
86215 . Hominidae
Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans

10901732 BIOSIS Number: 97101732
Blood lead levels among radiator repair workers in Colorado
Dalton C B; McCammon J B; Hoffman R E
Colorado Dep. Health, Denver, CO 80222, USA
American Journal of Epidemiology 138 (8). 1993. 643.
Full Journal Title: Twenty-sixth Annual Meeting of the Society for Epidemiologic Research, Keystone, Colorado, USA, June 16-18, 1993. American Journal of Epidemiology
ISSN: 0002-9262
Language: ENGLISH
Document Type: CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 003
Ref. 030625
Descriptors/Keywords: MEETING ABSTRACT; HUMAN; EPIDEMIOLOGY; OCCUPATIONAL HEALTH; TOXICITY
Concept Codes:
*15006 . Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and Reticuloendothelial Pathologies
*22501 . Toxicology-General; Methods and Experimental
*37013 . Public Health: Environmental Health-Occupational Health
*37054 . Public Health: Epidemiology-Organic Diseases and Neoplasms
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10060 . Biochemical Studies-General
10069 . Biochemical Studies-Minerals
Biosystematic Codes:
86215 . Hominidae
Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans

DIALOG File 55: BIOSIS PREVIEWS(R)_1985-1994/Mar Iss 16 (c) 1994 BIOSIS

115697

The Province of Ontario has had a surveillance program for workers in dusty industries for almost 70 years. This paper reports the detection rates of silicosis among 68,701 silica-exposed individuals who were first exposed to dust in 1950 or later, and who were still employed in 1979 or later. The detection rate varied strongly with latency, being less than two new cases per 10,000 examinations during the first two decades from first exposure, reaching two new cases per 1,000 examinations at 27 years from first exposure, and averaging between two and four new cases per 1,000 examinations thereafter. The silicosis incidence rate among miners was only about half that among foundry workers. Cigarette smoking was also found to be a risk factor for the diagnosis of silicosis. These data were used to model the detection rate of new cases of silicosis as a function of the time interval between examinations, and results are presented for examination cycles between 2 and 10 years.

Descriptors/Keywords: RESEARCH ARTICLE; HUMAN; OCCUPATIONAL TOXICITY; DIAGNOSIS; CANADA

Concept Codes:
*12504 . Pathology, General and Miscellaneous-Diagnostic
*16006 . Respiratory System-Pathology
*22506 . Toxicology-Environmental and Industrial Toxicology
*37013 . Public Health: Environmental Health-Occupational Health
10069 . Biochemical Studies-Minerals

Biosystematic Codes:
86215 . Hominidae

Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans

10982025 BIOSIS Number: 97182025

Chewing electric wire coatings: An unusual source of lead poisoning

Franco G; Cottica D; Minoia C
Cattedra di Med. dellavoro dell'Universita di Modina, via Campi 287, I-41100 Modena, ITL

American Journal of Industrial Medicine 25 (2). 1994. 291-296.
Full Journal Title: American Journal of Industrial Medicine
ISSN: 0271-3586
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 008 Ref. 115695

This report describes a case of lead poisoning occurring in an electrician as the result of an unusual personal habit, namely, the chewing of lead-containing coatings of electric wires. A coating chewing test showed that a few minutes after beginning chewing, saliva lead concentration increased from 10 mu-g/l to several milligrams per liter. This case is an example of poisoning caused by an occupationally related source (coatings containing lead) as a consequence of a singular and unconventional worker's habit.

Descriptors/Keywords: CASE STUDY; HUMAN; EMPLOYEE BEHAVIOR; HYGI[illegible]; OCCUPATIONAL TOXICOLOGY

Concept Codes:
*21002 . Psychiatry-Psychopathology; Psychodynamics and Therapy
*22506 . Toxicology-Environmental and Industrial Toxicology
*37013 . Public Health: Environmental Health-Occupational Health
07004 . Behavioral Biology-Human Behavior
10060 . Biochemical Studies-General

Biosystematic Codes:
86215 . Hominidae

Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans

10982024 BIOSIS Number: 97182024

Muconic acid in urine: A reliable indicator of occupational exposure to benzene

Lauwerys R R; Buchet J-P; Andrien F
Industrial Toxicology Occupational Med. Unit, Fac. Med., Catholic Univ. Louvain, 30.54. Clos Chapelle-aux-Champs, 1200 Brussels, BEL

American Journal of Industrial Medicine 25 (2). 1994. 297-300.
Full Journal Title: American Journal of Industrial Medicine
ISSN: 0271-3586
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 008 Ref. 115694

In male subjects not occupationally exposed to benzene, the concentration of muconic acid (MA) in urine is usually below 0.5 mg/g creatinine. At ambient levels of benzene exposure (below 0.01 ppm), the mean MA level was greater in 21 smokers than in 14 nonsmokers. In 38 male subjects employed in garages and coke ovens, a statistically significant correlation was found between the airborne concentration of benzene measured with passive monitors and MA in postshift urine. The mean postshift MA concentrations corresponding to a benzene 8-hour time-weighted average exposure (TWA) of 0.5 and 1 ppm were 0.8 and 1.4 mg/g creatinine, respectively.

Descriptors/Keywords: RESEARCH ARTICLE; HUMAN; OCCUPATIONAL TOXICOLOGY

Concept Codes:
*15506 . Urinary System and External Secretions-Pathology
*22506 . Toxicology-Environmental and Industrial Toxicology
*37013 . Public Health: Environmental Health-Occupational Health
10060 . Biochemical Studies-General

Biosystematic Codes:
86215 . Hominidae

Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans

DIALOG File 55: BIOSIS PREVIEWS(R)_1985-1994/Mar Iss 16 (c) 1994 BIOSIS
Investigative Dermatology and the Western Student Medical Research Committee, Carmel, California, USA, February 9-12, 1994. Clinical Research
ISSN: 0009-9279
Language: ENGLISH
Document Type: CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 004
Ref. 055034
Descriptors/Keywords: MEETING ABSTRACT; RAT; LUTEINIZING HORMONE; GONADOTROPIN; GONADOLIBERIN; LHRH; TRANSCRIPTIONAL MESSENGER RNA LEVEL; MALE REPRODUCTIVE TOXICITY
Concept Codes:
*03506 . Genetics and Cytogenetics-Animal
*03510 . Genetics and Cytogenetics-Sex Differences
*10300 . Replication, Transcription, Translation
*13014 . Metabolism-Nucleic Acids, Purines and Pyrimidines
*16506 . Reproductive System-Pathology
*17006 . Endocrine System-Gonads and Placenta
*17014 . Endocrine System-Pituitary
*17020 . Endocrine System-Neuroendocrinology (1972-)
*20504 . Nervous System-Physiology and Biochemistry
*22501 . Toxicology-General; Methods and Experimental
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10062 . Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10064 . Biochemical Studies-Proteins, Peptides and Amino Acids
10068 . Biochemical Studies-Carbohydrates
10069 . Biochemical Studies-Minerals
Biosystematic Codes:
86375 . Muridae
Super Taxa:
Animals; Chordates; Vertebrates; Nonhuman Vertebrates; Mammals; Nonhuman Mammals; Rodents

10957542 BIOSIS Number: 97157542
Monitoring occupational exposure to some industrial chemicals by the determination of hemoglobin adducts
Van Sittert N J; Van Vliet E W N
Shell Internationale Petroleum Maatschappi B.V., Health Safety and Environment Div., P.O. Box 162, 2501 AN The Hague, NET
Naunyn-Schmiedeberg's Archives of Pharmacology 348 (SUPPL.). 1993. R174.
Full Journal Title: Deutsche Gesellschaft fuer Pharmakologie und Toxikologie (German Society for Pharmacology and Toxicology) Fifth Winter Meeting, Hannover, Germany, December 1-3, 1993. Naunyn-Schmiedeberg's Archives of Pharmacology
ISSN: 0028-1298
Language: ENGLISH
Document Type: CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 004
Ref. 0[illegible] 3
Descriptors/Keywords: MEETING ABSTRACT; HUMAN; ETHYLENE OXIDE; PROPYLENE OXIDE; ETHYLENE; 1,3-BUTADIENE; TOXICITY
Concept Codes:
*10006 . Clinical Biochemistry: General Methods and Applications
*10054 . Biochemical Methods-Proteins, Peptides and Amino Acids
*22506 . Toxicology-Environmental and Industrial Toxicology
*37013 . Public Health: Environmental Health-Occupational Health
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10060 . Biochemical Studies-General
10064 . Biochemical Studies-Proteins, Peptides and Amino Acids
10065 . Biochemical Studies-Porphyrins and Bile Pigments
Biosystematic Codes:
86215 . Hominidae
Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans

10957540 BIOSIS Number: 97157540
Biomonitoring and subclinical effect in tetraethyl lead exposed persons in Hubei, China
Zhang W; Golka K
Inst. Occupational Med., Tongji Med. Univ., Wuhan/Hubei, 430030, CHN
Naunyn-Schmiedeberg's Archives of Pharmacology 348 (SUPPL.). 1993. R174.
Full Journal Title: Deutsche Gesellschaft fuer Pharmakologie und Toxikologie (German Society for Pharmacology and Toxicology) Fifth Winter Meeting, Hannover, Germany, December 1-3, 1993. Naunyn-Schmiedeberg's Archives of Pharmacology
ISSN: 0028-1298
Language: ENGLISH
Document Type: CONFERENCE PROCEEDINGS
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 004
Ref. 054141
Descriptors/Keywords: MEETING ABSTRACT; TOXICOLOGY; GASOLINE ADDITIVE; OCCUPATIONAL HEALTH; URINE
Concept Codes:
*15504 . Urinary System and External Secretions-Physiology and Biochemistry
*22506 . Toxicology-Environmental and Industrial Toxicology
*37013 . Public Health: Environmental Health-Occupational Health
00520 . General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10069 . Biochemical Studies-Minerals
Biosystematic Codes:
86215 . Hominidae

(cont. next page)

DIALOG File 154: MEDLINE_1985-1994/Apr (9404W4)

dysfunctional despite continued oxygen ventilation. Spontaneous breathing and cardiovascular performance following STX-induced apnea could all be promptly restored (typically in less than a minute) by combined oxygen/antitoxin therapy. Notable also was a state of uncompensated acidemia (as revealed by changes in arterial pH and CO2 tension) which persisted throughout the course of therapeutic intervention. Notwithstanding, the ventilatory frequency continued to be low, the central respiratory activity pattern remained aberrant, and the ECoG amplitudes were still depressed. In consideration of these findings, and of the large molecular weight of alpha-STX antitoxin (> 150,000 Da) which limits its entry into the CNS, we are of the opinion that the therapeutic effects of antitoxin are probably confined primarily to the periphery.

Tags: Animal

Descriptors: *Antitoxins--Immunology--IM; *Antitoxins --Therapeutic Use--TU; *Cardiac Output, Low --Chemically Induced--CI; *Cardiac Output, Low--Therapy--TH; *Respiratory Insufficiency--Chemically Induced--CI; *Respiratory Insufficiency--Therapy--TH; *Saxitoxin--Immunology--IM; *Saxitoxin --Toxicity--TO; Adrenal Glands--Drug Effects--DE; Antibodies --Therapeutic Use--TU; Cardiac Output, Low--Physiopathology --PP; Cardiovascular System--Drug Effects--DE; Electrocardiography; Electrophysiology; Guinea Pigs; Oxygen Inhalation Therapy; Perissodactyla; Respiratory Insufficiency --Physiopathology--PP

CAS Registry No.: 0 .(Antibodies); 0 .(Antitoxins); 35523-89-8 .(Saxitoxin)

08805516 94120516

Pharmacokinetic interaction between benzene metabolites, phenol and hydroquinone, in B6C3F1 mice.

Legathe A; Hoener BA; Tozer TN

Department of Pharmacy, School of Pharmacy, University of California at San Francisco 94143-0446.

Toxicol Appl Pharmacol (UNITED STATES) Jan 1994, 124 (1) p131-8, ISSN 0041-008X Journal Code: VWO

Contract/Grant No.: ES 04705, ES, NIEHS

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9404

Subfile: INDEX MEDICUS

There is strong evidence that metabolites are responsible for adverse effects of benzene. Benzene myelotoxicity, reproduced by coadministering phenol (PH) and hydroquinone (HQ) but not when these benzene metabolites were administered alone, has been postulated to be induced by PH stimulating the myeloperoxidase-mediated oxidation of HQ to the toxic 1,4-benzoquinone in bone marrow. A pharmacokinetic interaction between PH and HQ is also hypothesized to contribute to the observation. Both metabolites are sulfoconjugated and glucuronoconjugated. Sulfoconjugation of phenolic substrates has been shown to approach saturation at high concentrations in rats. Thus, more PH may be converted to HQ and HQ conjugation may be diminished. These effects would increase the amounts of PH and HQ present and result (by further oxidation) in the formation of more 1,4-benzoquinone. To test this hypothesis, we investigated the pharmacokinetics in blood and the recovery of hydroquinone and phenol in urine when the metabolites were administered intraperitoneally alone or in combination at 75 mg/kg each to B6C3F1 mice. The combination resulted in a 2.6-fold increase in the area under the blood concentration-time curve (AUC) of HQ compared to the sum of AUC values observed after administration of each compound alone. The half-life of HQ was also increased from 9 +/- 2 to 15 +/- 3 min. The AUC of PH was increased by a factor of 1.4. The clearance of phenol decreased from 89 +/- 13 ml/min per kilogram when injected alone to 62 +/- 7 ml/min per kilogram after coadministration. A decreased clearance of formation of each conjugate demonstrated that both conjugation pathways were diminished. This interaction may contribute to the observed production of myelotoxicity when these metabolites are coadministered.

Tags: Animal; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Descriptors: *Benzene--Toxicity--TO; *Hydroquinones --Pharmacokinetics--PK; *Phenols--Pharmacokinetics--PK; Benzene--Metabolism--ME; Drug Interactions; Glucuronates--Urine--UR; Hydroquinones--Blood--BL; Hydroquinones--Pharmacology--PD; Hydroquinones--Urine--UR; Mice; Mice, Inbred Strains; Models, Biological; Phenols--Blood--BL; Phenols--Pharmacology--PD; Phenols--Urine--UR; Sulfates--Urine--UR

CAS Registry No.: 0 .(Glucuronates); 0 .(Hydroquinones); 0 .(Phenols); 0 .(Sulfates); 108-95-2 .(phenol); 123-31-9 .(hydroquinone); 71-43-2 .(Benzene)

08805515 94120515

Role of quinone reductase in in vivo ethanol metabolism and toxicity.

Chung JH; Cha YN; Rubin RJ

Division of Toxicological Sciences, School of Hygiene and Public Health, Johns Hopkins University, Baltimore, Maryland 21205.

Toxicol Appl Pharmacol (UNITED STATES) Jan 1994, 124 (1) p123-30, ISSN 0041-008X Journal Code: VWO

Contract/Grant No.: RO1-AA06049, AA, NIAAA

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9404

Subfile: INDEX MEDICUS

Quinone reductase (QR), in the presence of suitable substrate, results in the regeneration of NAD+ from NADH. To test the hypothesis that QR can play a role in ethanol metabolism and toxicity, we studied the effect of a quinone as well as of induced levels of QR on ethanol administered in vivo to male rats and mice. Butylated hydroxyanisole (BHA) is known both to induce QR and to be metabolized to tert-butyl quinone (TBQ). Dietary BHA (0.75% for 10 days), followed by oral ethanol (4 g/kg, by gavage), increased the rate of

(cont. next page)

DIALOG File 154: MEDLINE_1985-1994/Apr (9404W4)
Tags: Animal
Descriptors: *Acetic Acids--Toxicity--TO; *Furans--Toxicity --TO; *Water Supply; Acetic Acids--Pharmacokinetics--PK; Carcinogenicity Tests; Furans--Pharmacokinetics--PK; Mice; Mutagenicity Tests; Research; Salmonella--Drug Effects--DE; Salmonella--Genetics--GE; Sterilization
CAS Registry No.: 0 .(Acetic Acids); 0 .(Furans)

08791828 94106828
Presence and importance of organochlorine solvents and other compounds in Germany's groundwater and drinking water.
Dieter HH; Kerndorff H
Institute for Water-, Soil- and Air Hygiene of the Federal Health Office of Germany (BGA), Berlin.
Ann Ist Super Sanita (ITALY) 1993, 29 (2) p263-77, ISSN 0021-2571 Journal Code: 5BP
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9404
Subfile: INDEX MEDICUS
Organochlorine compounds are widely used in Germany although the inland production of chlorinated solvents has greatly decreased since 1985. Data on groundwater contamination are incomplete, but there are some regional data sets from the States (Lander). Approximately 25% of the groundwater samples contain more than 1 microgram/l of a single solvent, the most prominent ones being tri- and tetrachloroethene, 1,1,1-trichloroethane and dichloromethane, but also chloroform. The most important causes for contaminations of the groundwater are unprotected storage and leaking sewage systems. Abandoned waste sites are, besides chlorinated compounds, also a source of many other contaminants. A ranking procedure according to their exposure potential (concentration, incidence, toxicology) is proposed. The compound of greatest concern is vinyl chloride, which is formed from tri- and tetrachloroethene under reducing conditions in the subsoil. The most important contaminant in drinking water is tetrachloroethene followed by 1,1,1-trichloroethane and trichloroethane. Chlorobenzene may also be present on occasion, while only about 20% of the finished drinking waters contain more chloroform after treatment than before. Only about 10% of all analyses of drinking water derived from groundwater shows the presence of organochlorine solvents and most of these show total concentrations less than 2 micrograms/l. The degradation product, vinyl chloride, was found up to now only in different groundwaters. To stabilize and to improve the situation, which still is much more favorable for drinking than for groundwater, precautions are going to be taken which should assure that these and other problematic substances which endanger water are used only in closed systems and rigid safety measures be imposed on their disposal and transport.
Descriptors: *Hydrocarbons, Chlorinated--Analysis--AN; *Solvents--Analysis--AN; *Water Pollutants, Chemical--Analysis --AN; *Water Pollution, Chemical; *Water Supply--Analysis--AN ; Germ /; Hydrocarbons, Chlorinated--Metabolism--ME; Solvents --Metabolism--ME; Water Pollutants, Chemical--Metabolism--ME
CAS Registry No.: 0 .(Hydrocarbons, Chlorinated); 0 .(Solvents); 0 .(Water Pollutants, Chemical)

08791780 94106780
Effects of alcohol, carbon tetrachloride, and choline deficiency on iron metabolism in the rat.
Batey RG; Johnston R
Department of Medicine, Westmead Hospital, New South Wales, Australia.
Alcohol Clin Exp Res (UNITED STATES) Oct 1993, 17 (5) p931-4, ISSN 0145-6008 Journal Code: 35X
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9404
Subfile: INDEX MEDICUS
The effects of alcohol on hepatic iron uptake and intestinal iron transport were studied in rats fed a nutritionally replete liquid diet containing varying quantities of ethanol. Results were compared with those from animals exposed to carbon tetrachloride (CCl4) to produce hepatocellular necrosis or a choline-deficient diet to produce steatosis and cirrhosis. A high ethanol intake for 4 or 10 weeks produced hepatic steatosis. CCl4 produced hepatocellular necrosis. Choline deficiency was associated with steatosis +/- cirrhosis. Intestinal iron transport was unaffected by ethanol, CCl4, or choline deficiency. Hepatic iron uptake was significantly depressed in rats consuming 11.7 g/kg/day ethanol ($p < 0.01$) for 4 weeks. Choline-deficient animals studied at 14 weeks also had significantly decreased hepatic iron uptake ($p < 0.01$); results were similar in the cirrhotic and noncirrhotic animals. Conversely, CCl4 exposure produced a significant 5-fold increase in hepatic iron uptake ($p < 0.001$). Results suggest that ethanol consumption, fatty liver, and cirrhosis are not responsible for any increase in iron absorption or of hepatic iron uptake in the rat model. Acute hepatocellular injury is followed by increased hepatic iron uptake.
Tags: Animal; Female; Support, Non-U.S. Gov't
Descriptors: *Alcohol, Ethyl--Toxicity--TO; *Carbon Tetrachloride--Toxicity--TO; *Choline Deficiency--Blood--BL; *Hepatitis, Toxic--Blood--BL; *Intestinal Absorption --Drug Effects--DE; *Iron--Blood--BL; *Liver--Drug Effects--DE; *Liver Diseases, Alcoholic--Blood--BL; Choline Deficiency --Pathology--PA; Fatty Liver, Alcoholic--Blood--BL; Fatty Liver, Alcoholic--Pathology--PA; Hepatitis, Toxic--Pathology --PA; Intestinal Absorption--Physiology--PH; Liver--Metabolism --ME; Liver--Pathology--PA; Liver Cirrhosis, Alcoholic--Blood --BL; Liver Cirrhosis, Alcoholic--Pathology--PA; Liver Diseases, Alcoholic--Pathology--PA; Rats; Rats, Wistar; Weight Gain--Drug Effects--DE; Weight Gain--Physiology--PH
CAS Registry No.: 56-23-5 .(Carbon Tetrachloride); 64-17-5 .(Alcohol, Ethyl); 7439-89-6 .(Iron)

DIALOG File 154: MEDLINE_1985-1994/Apr (9404W4)
08791583 94106583
Intracellular mechanism of Pb(2+)-induced norepinephrine release from bovine chromaffin cells.
Tomsig JL; Suszkiw JB
Department of Physiology and Biophysics, University of Cincinnati College of Medicine, Ohio 45267-0576.
Am J Physiol (UNITED STATES) Dec 1993, 265 (6 Pt 1) pC1630-6, ISSN 0002-9513 Journal Code: 3U8
Contract/Grant No.: ES-04090, ES, NIEHS
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9404
Subfile: INDEX MEDICUS
The intracellular mechanism of Pb(2+)-induced release of norepinephrine (NE) was investigated in comparison with Ca2+ in bovine chromaffin cells permeabilized with staphylococcal alpha-toxin. Pb2+ activated NE release at considerably lower concentrations [concentration of free metal giving half maximal metal-dependent release (K0.5) 4.6 nM] than Ca2+ (K0.5 2.4 microM). The release of NE was associated with the release of dopamine-beta-hydroxylase but not lactate dehydrogenase. The maximal secretory responses produced by Pb2+ and Ca2+ were similar and nonadditive. Pb(2+)- and Ca(2+)-dependent releases showed a similar requirement for MgATP and were equally enhanced by protein kinase C activator 12-O-tetradecanoylphorbol 13-acetate (TPA) but not by kinase A activator 8-bromoadenosine 3',5'-cyclic monophosphate free base. The protein kinase C inhibitor staurosporine blocked the TPA-stimulated component of secretion but had no effect on the NE release in the absence of TPA. Calmidazolium, an inhibitor of calmodulin, inhibited the secretion evoked by both metals to similar extent. Agents interacting with microtubules (colchicine and vinblastine) or microfilaments (cytochalasin B and phalloidin) had no effect on secretion induced by either metal cation. These observations indicate that both Pb2+ and Ca2+ act at a common site and activate the exocytotic release of NE by an analogous mechanism.
Tags: Animal; Comparative Study; Support, U.S. Gov't, P.H.S.
Descriptors: *Adrenal Medulla--Physiology--PH; *Calcium --Pharmacology--PD; *Lead--Toxicity--TO; *Norepinephrine --Metabolism--ME; Adenosine Triphosphate--Pharmacology--PD; Adrenal Medulla--Drug Effects--DE; Alkaloids--Pharmacology--PD ; Calcium--Metabolism--ME; Calmodulin --Antagonists and Inhibitors--AI; Cattle; Cell Membrane Permeability; Cells, Cultured; Cyclic AMP-Dependent Protein Kinases--Metabolism--ME ; Dose-Response Relationship, Drug; Egtazic Acid--Pharmacology --PD; Enzyme Activation; Imidazoles--Pharmacology--PD; Kinetics; Lactate Dehydrogenase--Analysis--AN; Protein Kinase C--Antagonists and Inhibitors--AI; Protein Kinase C --Metabolism--ME; Spectrophotometry, Atomic Absorption; Tetradecanoylphorbol Acetate--Pharmacology--PD; 8-Bromo Cyclic Adenosine Monophosphate--Pharmacology--PD
CAS Registry No.: 0 .(Alkaloids); 0 .(Calmodulin); 0 .(Imidazoles); 16561-29-8 .(Tetradecanoylphorbol Acetate); 23583-48-4 .(8-Bromo Cyclic Adenosine Monophosphate); 51-41-2 .(Norepinephrine); 56-65-5 .(Adenosine Triphosphate); 57265-65-3 .(R 24571); 62996-74-1 .(staurosporine); 67-42-5 .(Egtazic Acid); 7439-92-1 .(Lead); 7440-70-2 .(Calcium)
Enzyme No.: EC 1.1.1.27 .(Lactate Dehydrogenase); EC 2.7.1.- .(Protein Kinase C); EC 2.7.10.- .(Cyclic AMP-Dependent Protein Kinases)

08791132 94106132
[Correction of disorders in the monooxygenase system function with diethylnicotinamide (cordiamine) in tetrachloromethane- induced and viral hepatitis]
Korrektsiia dietilnikotinamidom (kordiaminom) narusheniia funktsii monooksigenaznoi sistemy pri tetrakhlormetanovom i virusnom gepatitakh.
Zavodnik LB; Lukienko PI; Bushma MI; Shoka AIu; Tsyrkunov VM
Vopr Med Khim (RUSSIA) Sep-Oct 1993, 39 (5) p45-7, ISSN 0042-8809 Journal Code: XIQ
Languages: RUSSIAN Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE English Abstract
JOURNAL ANNOUNCEMENT: 9404
Subfile: INDEX MEDICUS
Content of cytochromes P-450 and b5 and the rate of oxidative dealkylation in liver microsomes as well as the antipyrine pharmacokinetics were normalized in rats with acute CCl4-induced hepatitis after treatment with cordiamine (diethyl nicotinamide) at a dose of 40 mg, subcutaneously, 2 times daily within 4 days. Cordiamine (30 drops 3 times daily within 8 days) contributed to normalization of the hydroxylating reaction in liver tissue of patients with viral hepatitis A, estimated by the "antipyrine" test. The drug exhibited stabilizing effect on hydrophobic interactions in microsomal membranes; diethyl nicotinamide possessed antiradical and vitamin properties.
Tags: Animal; Female; Human; Male
Descriptors: *Carbon Tetrachloride; *Cytochrome b5 --Antagonists and Inhibitors--AI; *Cytochrome P-450 --Antagonists and Inhibitors--AI; *Hepatitis A--Drug Therapy --DT; *Hepatitis, Toxic--Drug Therapy--DT; *Nikethamide --Therapeutic Use--TU; Adolescence; Adult; Microsomes, Liver --Enzymology--EN; Rats
CAS Registry No.: 56-23-5 .(Carbon Tetrachloride); 59-26-7 .(Nikethamide); 9035-39-6 .(Cytochrome b5); 9035-51-2 .(Cytochrome P-450)

08790738 94105738
[Hematologic changes in patients chronically exposed to benzene]
Alteracoes hematologicas em pacientes expostos cronicamente ao benzeno.
Ruiz MA; Vassallo J; de Souza CA
Setor de Hematologia, Faculdade de Ciencias Medicas de Santos (FCMS), SP, Brasil.
Rev Saude Publica (BRAZIL) Apr 1993, 27 (2) p145-51, ISSN 0034-8910 Journal Code: T5X
Languages: PORTUGUESE Summary Languages: ENGLISH
(cont. next page)

DIALOG File 154: MEDLINE_1985-1994/Apr (9404W4)
Document type: JOURNAL ARTICLE English Abstract
JOURNAL ANNOUNCEMENT: 9404
Subfile: INDEX MEDICUS

A study was carried out into the hematological abnormalities of peripheral blood bone marrow in patients chronically exposed to benzene. The metabolic biotransformation and the mechanisms involved in toxicity are described. Hematological data are described and discussed. Macrocytosis and lymphopenia are the earliest hematological signs of benzene toxicity. Bone marrow abnormalities are demonstrated by the complementary methods of cytology and histology. Global hypocellularity was mainly due to the granulocytic series. Mastocytosis, eosinophilia and magakariocytic abnormalities are also presented. Inflammatory abnormalities and signs of dismyelopoiese could also be observed. The importance of peripheral blood abnormalities and the need for a critical approach to this important public health problem are emphasized.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: *Benzene--Poisoning--PO; *Hematologic Diseases --Chemically Induced--CI; *Occupational Diseases--Chemically Induced--CI; *Occupational Exposure--Adverse Effects--AE; Benzene--Metabolism--ME; Bone Marrow--Drug Effects--DE; Bone Marrow--Pathology--PA; Hematologic Diseases--Blood--BL; Occupational Diseases--Blood--BL; Risk Factors

CAS Registry No.: 71-43-2 .(Benzene)

08790460 94105460

Scientific principles for evaluating the potential for adverse effects from chlorinated organic chemicals in the environment.

Willes RF; Nestmann ER; Miller PA; Orr JC; Munro IC

CanTox Inc., Mississauga, Ontario, Canada.

Regul Toxicol Pharmacol (UNITED STATES) Oct 1993, 18 (2) p313-56, ISSN 0273-2300, Journal Code: RBH

Languages: ENGLISH

Document type: JOURNAL ARTICLE; REVIEW; REVIEW, ACADEMIC

JOURNAL ANNOUNCEMENT: 9404

Subfile: INDEX MEDICUS

The term chlorinated chemicals is used to describe diverse groups of chemicals of varying chemical structure, including those used in water disinfection, as well as numerous aliphatic, aromatic, and polycyclic chlorinated substances. This report elaborates a number of scientific principles that govern the evaluation of the potential for chlorinated organic chemicals to cause adverse effects on the environment and to human health. The purpose of the report is to demonstrate the importance of applying these scientific principles in the evaluation of potential adverse effects of chlorinated organic chemicals. The four major principles upon which such a scientific analysis must be based are: (1) the fate and biological activity of a compound are determined by the chemical properties of the compound; (2) compounds do not show adverse effects below certain threshold concentrations, and the magnitude of response is related to dose; (3) inherent metabc processes allow organisms to accommodate low doses of chlorinated organic chemicals; (4) observations associated with the presence of a certain compound must be biologically plausible effects, based on the specificity of the compound's activity in experimental systems. With respect to the first of these principles, there is abundant scientific evidence that the physical and chemical properties of chlorinated organic chemicals govern their bioaccumulative potential, toxicological properties, and thus their potential behavior and effects in the environment. Chemicals that have low solubility in water are highly lipophilic and have low vapor pressure, tend to accumulate in biological systems, and degrade slowly in the environment. Chlorinated organic chemicals that possess these characteristics include those having a carbon ring structure and multiple chlorine substitution. Other chlorinated organic chemicals with lesser degrees of chlorine substitution, such as 2,4-dichlorophenoxyacetic acid (2,4-D), trichlorophenol, chloroform, and dichloroethane, do not share the physical and chemical properties of the high molecular weight, cyclic, polychlorinated compounds and, as such, do not have the same potential to bioaccumulate. These differences among chlorinated organic chemicals with respect to their physical and chemical properties and behavior in the environment preclude the generalization that all organic chemicals containing chlorine behave similarly in the environment and act as persistent, bioaccumulative chemicals. The reactivity of chlorinated organic chemicals and hence their potential to produce biological effects depends on their specific molecular features. The substitution of chlorine into an organic molecule may increase or may reduce its biological activity.(ABSTRACT TRUNCATED AT 400 WORDS) (204 Refs.)

Tags: Animal; Human; Support, Non-U.S. Gov't

Descriptors: *Environmental Pollutants--Toxicity--TO; *Hydrocarbons, Chlorinated--Toxicity--TO; Environmental Pollutants--Analysis--AN; Hydrocarbons, Chlorinated--Chemistry --CH

CAS Registry No.: 0 .(Environmental Pollutants); 0 .(Hydrocarbons, Chlorinated)

08790459 94105459

Comparing the results of a Monte Carlo analysis with EPA's reasonable maximum exposed individual (RMEI): a case study of a former wood treatment site.

Copeland TL; Paustenbach DJ; Harris MA; Otani J

ChemRisk, a Division of McLaren/Hart, Irvine, California 92714.

Regul Toxicol Pharmacol (UNITED STATES) Oct 1993, 18 (2) p275-312, ISSN 0273-2300 Journal Code: RBH

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9404

Subfile: INDEX MEDICUS

In the United States, there are about 250 former sites that treated wood with preservatives that are now in need of some

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DIALOG File 154: MEDLINE_1985-1994/Apr (9404W4)

serum, brain, liver, kidney and uterus were increased significantly by HCH and malathion exposure, irrespective of the protein content in the diet. The incorporation of [1,2-14C]acetate into the hepatic lipids was stimulated by both HCH and malathion, suggesting a higher rate of lipid synthesis in the liver of normal and protein-deficient diet fed dams. The low protein content in the diet intensified the pesticide-induced changes and more severe alterations were noticed in HCH exposed dams than in malathion exposed dams.

Tags: Animal; Female

Descriptors: *Benzene Hexachloride--Toxicity--TO; *Lipids --Metabolism--ME; *Malathion--Toxicity--TO; *Pregnancy Complications--Metabolism--ME; *Pregnancy, Animal--Metabolism --ME; *Protein-Energy Malnutrition--Metabolism--ME; Cholesterol--Metabolism--ME; Glucosephosphate Dehydrogenase--Metabolism --ME; Hydroxymethylglutaryl CoA Reductases--Metabolism--ME; Kidney--Drug Effects--DE; Kidney--Metabolism--ME; Lipolysis --Drug Effects--DE; Lipoprotein Lipase--Metabolism--ME; Liver --Drug Effects--DE; Liver--Metabolism--ME; Malate Dehydrogenase--Metabolism--ME; Phospholipids--Metabolism--ME; Pregnancy; Rats; Rats, Sprague-Dawley; Triglycerides --Metabolism--ME

CAS Registry No.: 0 .(Lipids); 0 .(Phospholipids); 0 .(Triglycerides); 121-75-5 .(Malathion); 57-88-5 .(Cholesterol); 58-89-9 .(Benzene Hexachloride)

Enzyme No.: EC 1.1.1.37 .(Malate Dehydrogenase); EC 1.1.1.49 .(Glucosephosphate Dehydrogenase); EC 1.1.1.88 .(Hydroxymethylglutaryl CoA Reductases); EC 3.1.1.34 .(Lipoprotein Lipase)

08787258 94102258

Identification of 6-hydroxy-trans,trans-2,4-hexadienoic acid, a novel ring-opened urinary metabolite of benzene.

Kline SA; Robertson JF; Grotz VL; Goldstein BD; Witz G

Department of Environmental and Community Medicine, University of Medicine and Dentistry-New Jersey, Robert Wood Johnson Medical School, Piscataway.

Environ Health Perspect (UNITED STATES) Sep 1993, 101 (4) p310-2, ISSN 0091-6765 Journal Code: EIO

Contract/Grant No.: ES02558, ES, NIEHS; ES05022, ES, NIEHS

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9404

Subfile: INDEX MEDICUS

We studied the in vivo metabolism of benzene in mice to ring-opened compounds excreted in urine. Male CD-1 mice were treated intraperitoneally with benzene (110-440 mg/kg), [14C]benzene (220 mg/kg) or trans, trans-muconaldehyde (MUC; 4 mg/kg), a microsomal, hematotoxic metabolite of benzene. Urine, collected over 24 hr, was extracted and analyzed by HPLC with a diode-array detector and by scintillation counting. In addition to trans,trans-muconic acid, previously the only known ring-opened urinary benzene metabolite, a new metabolite, 6-hydroxy-trans,trans-2,4-hexadienoic acid, was detected in urine of mice treated with either benzene or MUC. We identified the new metabolite based on coelution of metabolites and UV spectral comparison with authentic standards in unmethylated and methylated urine extracts. Results presented here are consistent with the intermediacy of MUC in the in vivo metabolism of benzene to ring-opened metabolites.

Tags: Animal; Male; Support, U.S. Gov't, P.H.S.

Descriptors: *Benzene--Metabolism--ME; *Sorbic Acid--Analogs and Derivatives--AA; Benzene--Toxicity--TO; Mice; Microsomes, Liver--Metabolism--ME; Sorbic Acid--Chemistry--CH; Sorbic Acid--Metabolism--ME

CAS Registry No.: 110-44-1 .(Sorbic Acid); 505-70-4 .(muconic acid); 71-43-2 .(Benzene); 88973-47-1 .(6-hydroxy-2,4-hexadienoic acid)

08786126 94101126

A community-based epidemiologic study of health sequelae of exposure to hydrofluoric acid.

Dayal HH; Brodwick M; Morris R; Baranowski T; Trieff N; Harrison JA; Lisse JR; Ansari GA

University of Texas Medical Branch, Galveston 77550.

Ann Epidemiol (UNITED STATES) May 1992, 2 (3) p213-30, ISSN 1047-2797 Journal Code: BX8

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9404

Subfile: INDEX MEDICUS

An accident at an oil refinery in Texas City, Texas, released around 40,000 lb of hydrogen fluoride, exposing the community to the highly toxic and corrosive substance. A population-based epidemiologic study was conducted to evaluate the impact of the accident on the health of the community. Exposure assessment was done using a multipronged approach through a door-to-door survey of 10,811 individuals. A symptom survey resulting in 1994 completed interviews was conducted with a stratified random sample selected from the exposure study database. The sampling was balanced with respect to age, gender, and predisposition across the three ordinal exposure categories. The results show a strong dose relationship ($P < 10(-4)$) between the exposure and symptoms reported following the accident and 2 years later, most notably breathing and eye symptoms. However, substantial improvement in health was reported over the 2-year period regardless of the level of exposure. Problems of recall bias and behavioral sensitization are considered and it is recognized that the study may have overestimated the effect. It is also recognized that the study may not have completely unraveled the relative importance of exposure and host response in health outcome, since the two were probably conflated in the exposure measure. Nevertheless, the independence of predisposition and reported level of exposure, the magnitude of effect and its consistency, the unmistakable dose response, the large sample size, and the mutual corroboration of various findings make it difficult to dismiss the interpretation that the hydrofluoric acid exposure indeed caused health problems in the community that continued

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DIALOG File 154: MEDLINE_1985-1994/Apr (9404W4)

vanadium(IV), which was more obvious in the case of exposure to vanadium(V) alone. This suggests that the amount of vanadium(V) reduced to vanadium(IV) decreased in GSH-depleted cells. While vanadium(IV) at concentrations of 3 x 10(-6) M and 10(-5) M was not transforming in the cells, vanadium(V) showed neoplastic transforming activity ($P < 0.025$ and $P < 0.001$ for the two doses, respectively) in comparison to controls (vanadium unexposed cells). Cytotoxicity and morphological transformation in cells exposed to vanadium(V) in combination with 3 x 10(-6) M DEM were significantly more intensive ($P < 0.005$ and $P < 0.01$ for the two doses of vanadate tested) compared to the corresponding values observed in cells exposed to vanadium(V) alone. This suggests that the final transforming activity response is dependent on the intracellular GSH-mediated mechanism of reduction of vanadium(V) to vanadium(IV): (i) the extent to which vanadium(V) should be bioreduced to less toxic vanadium(IV) via intracellular GSH is a key point in determining the intensity of the observed neoplastic action; (ii) the carcinogenic potential of vanadium(V) should be strictly dependent on its intracellular persistence which could lead to changes in normal metabolic patterns of vanadium(V) in the oxidized form due to lack of GSH-mediated reduction.

Tags: Animal

Descriptors: *Cell Transformation, Neoplastic--Drug Effects --DE; *Glutathione--Metabolism--ME; *Vanadium Compounds --Toxicity--TO; Biotransformation; Cell Survival--Drug Effects --DE; Electron Spin Resonance Spectroscopy; Maleates --Pharmacology--PD; Mice; Mice, Inbred BALB C; Oxidation-Reduction; Vanadium Compounds--Metabolism--ME; 3T3 Cells

CAS Registry No.: 0 .(Maleates); 0 .(Vanadium Compounds); 141-05-9 .(diethyl maleate); 27774-13-6 .(vanadyl sulfate); 70-18-8 .(Glutathione)

08779434 94094434

Benzene and phenol metabolism by mouse and rat liver microsomes.

Schlosser PM; Bond JA; Medinsky MA

Chemical Industry Institute of Toxicology, Research Triangle Park, NC 27709.

Carcinogenesis (ENGLAND) Dec 1993, 14 (12) p2477-86, ISSN 0143-3334 Journal Code: C9T

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9404

Subfile: INDEX MEDICUS

Benzene, an important industrial solvent and constituent of unleaded gasoline, causes leukemia and aplastic anemia in humans. Mice are more sensitive than rats to benzene toxicity, though neither species has been shown to respond consistently with benzene-induced leukemia. Benzene biotransformation in liver to phenol, hydroquinone, catechol and/or muconaldehyde is thought to be necessary for its hematotoxicity and/or genotoxicity. Our goal is to develop a mathematical simulation model capable of describing the pathways and kinetics of benzene metabolism by rat and mouse liver microsomes and to assess the role of species metabolic differences in species sensitivity. Microsomes were incubated with 4 microM [U-14C]-benzene or 4 microM [U-14C]phenol. Metabolite production was quantified by extraction into ethyl acetate, HPLC separation and liquid scintillation spectroscopy. After 45 min, mouse liver microsomes converted 20% of the benzene to phenol, 31% to hydroquinone and 2% to catechol. Rat liver microsomes converted 23% of benzene to phenol, 8% to hydroquinone and 0.5% to catechol. Production of hydroquinone and catechol continued for 90 min for mouse liver microsomes, while production by rat liver microsomes had virtually ceased by 90 min. Muconic acid production by mouse liver microsomes was < 0.2% and < 0.04% from benzene and phenol respectively after 90 min. A quantitative simulation model was constructed to describe the in vitro metabolism of benzene, incorporating the reaction sequences: benzene-->phenol-->catechol-->trihydroxybenzene and phenol-->hydroquinone-->trihydroxybenzene. In the model, all of the reaction steps are assumed to be catalyzed by the same enzyme(s), cytochrome(s) P450, and benzene, phenol, hydroquinone and catechol in solution are all assumed to compete, through reversible binding, for the same reaction site(s) on cytochrome(s) P450. The simulation model accurately described both the benzene and phenol kinetic data, supporting this proposed mechanism. In particular, this model suggests that the observed inhibition of benzene on phenol metabolism, and of phenol on benzene metabolism, occurs through competition for a common reaction site, which can also bind catechol and hydroquinone.

Tags: Animal; In Vitro; Male; Support, Non-U.S. Gov't

Descriptors: *Benzene--Metabolism--ME; *Microsomes, Liver --Metabolism--ME; *Phenols--Metabolism--ME; Biotransformation; Mice; Models, Biological; Rats; Rats, Inbred F344

CAS Registry No.: 0 .(Phenols); 108-95-2 .(phenol); 71-43-2 .(Benzene)

08779365 94094365

Liver accumulation of 2,3,7,8-tetrachloro-[3H]dibenzofuran in mice: modulation by treatments with polychlorinated biphenyls.

Darnerud PO; Tornwall U; Bergman A; Brandt I

Department of Toxicology, Uppsala University, Sweden.

Chem Biol Interact (IRELAND) Dec 1993, 89 (2-3) p89-102, ISSN 0009-2797 Journal Code: CYV

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9404

Subfile: INDEX MEDICUS

The distribution of 2,3,7,8-tetrachloro-[3H]dibenzofuran ([3H]TCDF; 40 micrograms/kg) resembled that earlier reported for 2,3,7,8-tetrachlorodibenzo-p-dioxin, with a strong accumulation in the liver and a selective uptake in the nasal olfactory mucosa of adult and fetal mice. Pretreatments with a series of selected congeners of polychlorinated biphenyls

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