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Dangerous Properties of Industrial Materials Report

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Dangerous Properties of Industrial Materials Report

July/August 1983 Vol.3 No.4

FEATURES

HAZARDOUS MATERIALS

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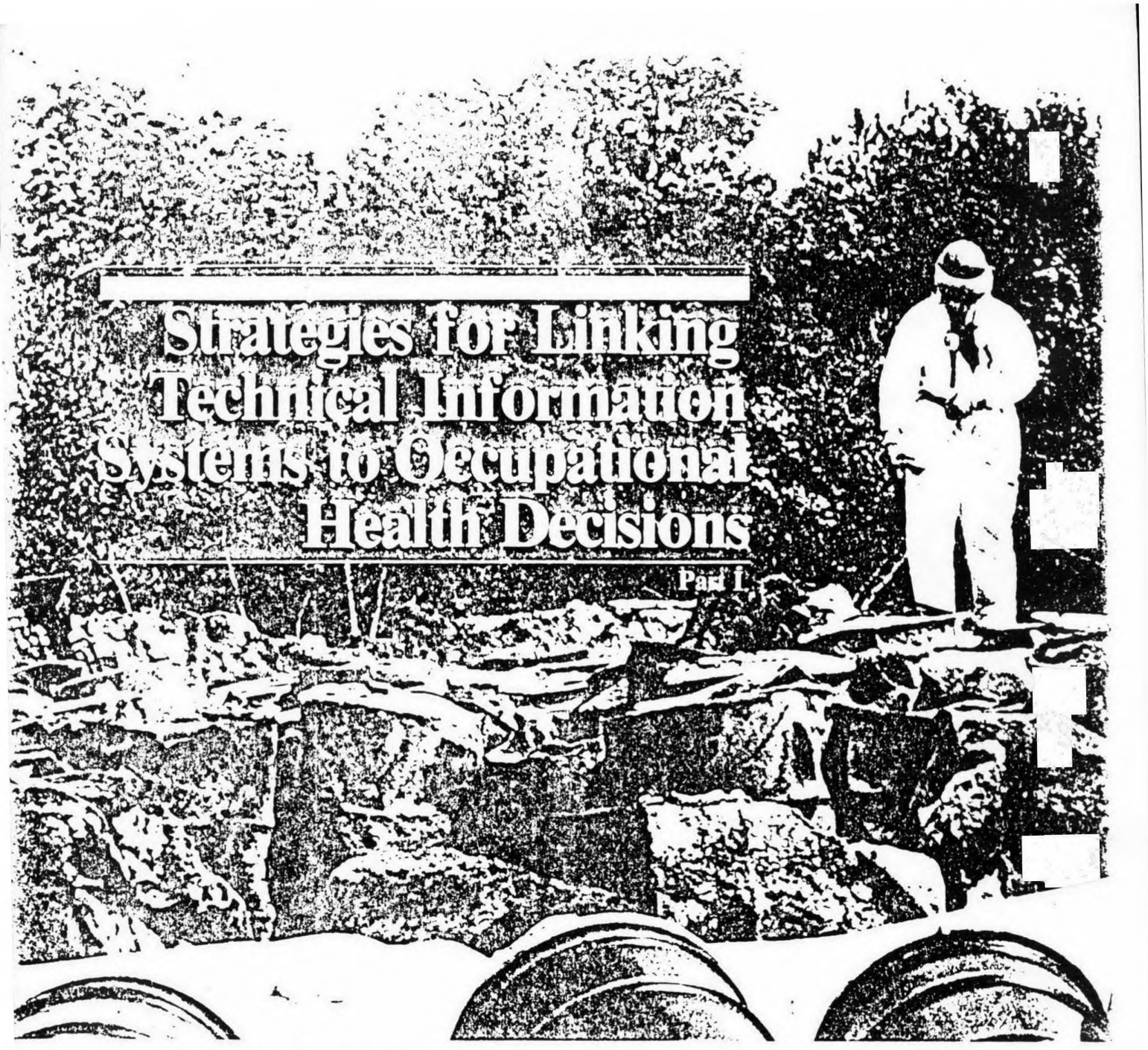
Papers are invited for review by the Editor and Editorial Board. Please address all queries and correspondences to the Editor-in-Chief.



Strategies for Linking Technical Information Systems to Occupational Health Decisions

Part I

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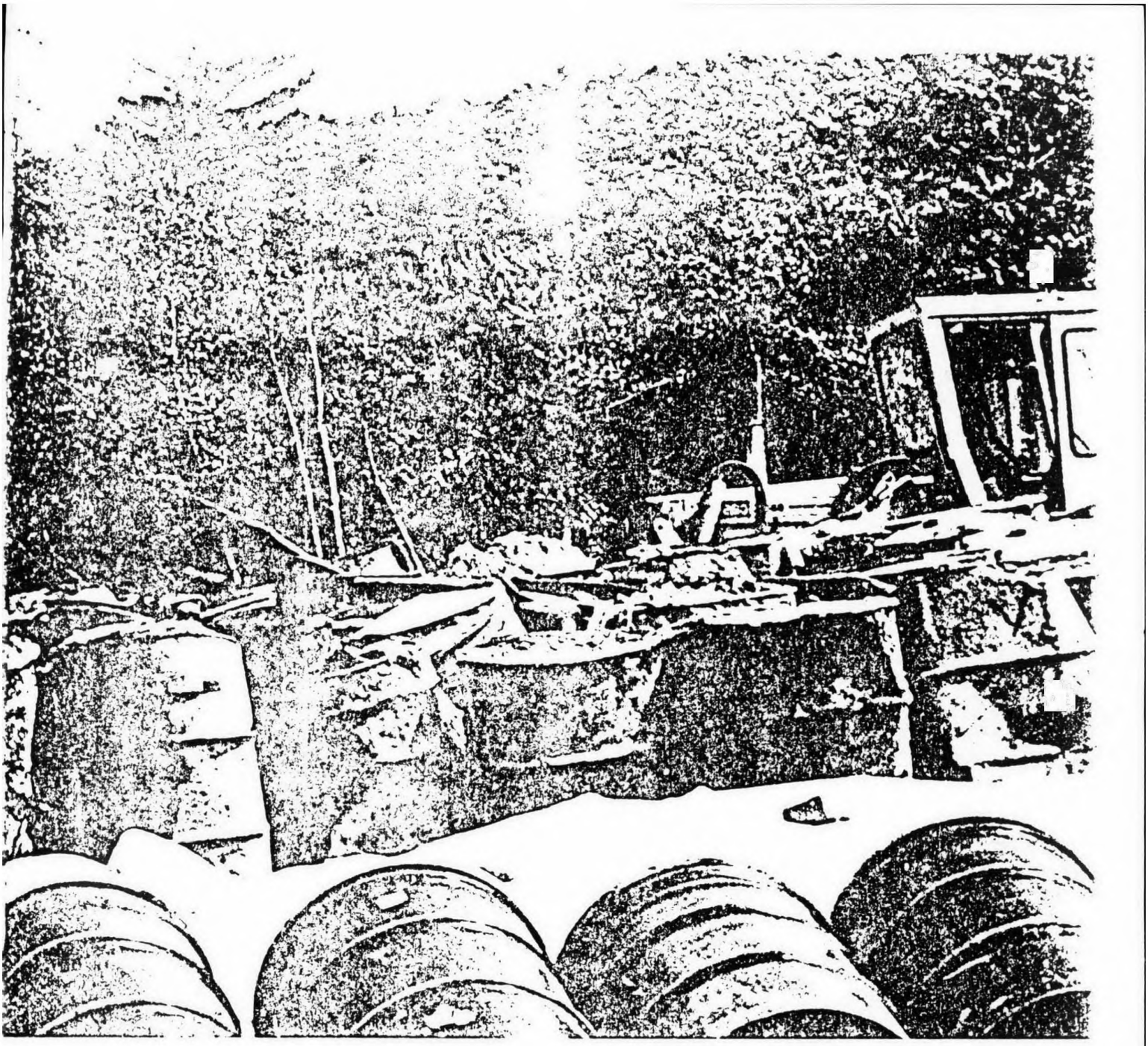
INTRODUCTION

The study of the occupational health environment requires some knowledge and understanding of a number of basic scientific disciplines including biochemistry, physical chemistry, physiology, mathematics, engineering, pathology and toxicology, as well as some comprehension of the non-scientific disciplines of law, business and socioeconomics. Occupational health problems must be counted as an important facet of public health today. Not only are there obvious problems of accident and injury, but there is also ample evidence of long term deteriorative health effects. The Second Task Force for Research Planning in Environmental Health Science (1976) noted that there is clear evidence that "occupational hazards influence the development and progression of chronic degenerative disease." For some of these degenerative diseases, the precipitation factor(s) may lie in the workplace as well. The Task Force also reported that "exposures to low levels of toxic agents may cause subclinical effects which are manifested in increased suscepti-



bility to infectious diseases, psychiatric illnesses, and other non-occupational disease problems," and concluded that the extent of occupational related illness is probably still further underestimated, since it is "known that cases of classic occupational diseases are being missed because many physicians are not relating disease states to occupational exposure."

Essentially, occupational diseases resulting from exposure to toxic chemicals may occur in any organ system, with an immediate response to substances such as carbon monoxide or hydrogen sulfide, or a delayed response, ranging from overnight response to nitric oxide or a twenty year delay in response to asbestos exposure (Christensen, 1976). Health effects may occur from a single, acute exposure, or from the cumulative effects of chronic exposure experienced over a long period of time, as observed with inorganic lead. When the rate of lead intake exceeds excretion rates, lead poisoning occurs (Christensen, 1976). Chronic effects can be expressed by changing the biological organization of an organ, such as changes in cellular structure brought about by asbestos and cotton dust. Toxic effects can also be conditioned reactions where initial exposure sensitizes the body for later, more intensive damage, for example, toluene diisocyanate (Christensen, 1976).

PRESENT ATTITUDES TOWARDS OCCUPATIONAL HEALTH

Occupational health is basically preventive medicine which characteristically is concerned with the prevention

of disease occurrence and with prevention of the consequences and the sequences of disease processes. Epidemiological studies and biophysical and/or biochemical studies have been designed to establish the mechanism(s) of biological interactions of environmental agents and to define the thresholds at which such agents interact with mammalian physiological systems. It is evident that occupational health policies should be developed and formulated in light of these studies. It is well recognized that there may be no threshold for exposure to some agents, such as ionizing radiation, whereby any level of exposure can interact with biological processes to present a health hazard. The effects of toxicant exposure upon a living organism depends upon four basic factors: toxicity, concentration, duration of exposure and individual susceptibility. Individual susceptibility can be influenced by age, nutritional status, preexisting health conditions and heredity.

The primary goal of occupational health policies is to set standards whereby the risk(s) of detrimental consequences to the worker's health as a result of exposure to xenobiotics are minimized. In formulating the policies of occupational health, issues pertaining to concepts of risks must be addressed. In this regard, there has been much debate over the role of the recognition of high risk groups in the formulation of occupational health policies and practices. The high risk group has been defined as a population or set that differs from the general population or universal set because it is composed of individuals who show an unusual frequency of a specific disease process or individuals who are exposed to an unusually high concentration of a suspected disease stimulus. Since the establishment of OSHA in 1970, and the concomitant Congressional mandate, (the Clean Air Act) for the development of criteria for Federal occupational exposure standards to protect "all" workers, the recognition of the high risk population has had a decided impact upon the development and implementation of standards which are intended to protect all workers.

Labor feels that prejob and periodic health examinations throughout the individual's employment to identify the susceptible individual would be used against, rather than for the good of the individual. They claim that for economic reasons these persons would not be relocated within the company's work force, or that they could be eliminated from a particular job because of increased health risks from exposure to hazards which could result in increased compensation costs to the corporation. The general consensus appears to be that unless there is a national policy of job security, or unless the union contract protects the hypersusceptibles, the monitoring of the health status of the worker would be used by management to place or replace the worker. Risk or safety judgement

therefore becomes a normative, subjective and socioeconomic discussion.

In order to set health standards which are effective, prudent and enforceable, it is important to differentiate clearly between risk assessment, which can be a quantifiable, scientific, impartial or data-based determination, and the risk or safety judgment which is a normative, subjective or political evaluation of how much risk or safety is acceptable to society, particularly to those facing the risks. The ultimate acceptability of the latter generally becomes a function of cost, relativity of the risks involved, and who will take the risk. What is needed is an integrative and rational approach whereby labor and management work together towards meeting a mutual goal—the protection of the safety and health of the worker.

THE ROLE OF TOXICOLOGICAL AND EPIDEMIOLOGICAL DATA IN OCCUPATIONAL HEALTH MANAGEMENT*

Classical toxicological LD50 studies, which permit nothing more than a preliminary classification of the poisonousness of a substance, are not valid for predicting the detrimental latent health effects of a toxicant, particularly if death is the only effect examined. However, if more than just the endpoint of death is examined, this

type of acute toxicity study could and should yield much more valuable information regarding the interactions of potentially toxic substances with normal biological processes. With careful clinical and laboratory observations prior to death, information regarding minimal symptomatic and toxic dose, early symptoms of intoxication, target age involvement, duration of effect(s), and reversibility of toxic insults to key physiologic processes could be obtained from these acute toxicity studies.

The LD50 type of toxicological studies employ a single dose to determine a dose which kills 50 percent of the treated animals. There is no consistent linear relationship between the effects of a given single lethal dose and the chronic administration of sublethal doses which ultimately result in death since the physiologic processes which are impaired may be quite different in the two exposure presentations (Peck, 1968).

Industrial accidents involving a single exposure to a high level of a toxic agent are less likely to occur than the chronic intermittent exposure to low levels of toxicants. Truhart (1976) described the concept of "long-term toxicity" resulting from the cumulative effects of absorption of small repeated doses of a toxicant. In identifying an occupational intoxicant it is important to determine how it might, and if it does indeed, interfere with the normal processes of a cell at "low" levels of exposure encountered during chronic intermittent exposure. Furthermore, the importance of routine physical examination of workers is indicated in order to monitor the health status of the individual, especially those with greater than two years' employment. These examinations should be made with an emphasis on detecting any predicted physiologic impairment that could be attributable to exposure to an occupational chemical(s) specific to a particular work environment.

Although the best evidence of adverse human health effects of xenobiotics is probably a well conducted epidemiological study, this should not be the only means of evaluating the health effects of these agents. The design of laboratory testing of environmental/occupational agents on biological processes should be based upon the known chemical and physical properties of the compounds in question in order to elucidate and quantify injurious biochemical and/or biophysical interactions. The key to understanding biochemical and/or biophysical interactions resulting in injury to a living organism is the development of investigative methods by which changes in cellular function and structure can be detected, and the application of these methods to elucidate the mechanism and/or factors which modulate chemical injury. Understanding the biological mechanisms for the interaction of these compounds will enable us to recognize the potential of impairment and/or injury to human health.

An example of a predictable interaction can be seen with polybrominated biphenyl (PBB). The halogenated biphenyls are relatively insoluble in water. Their high lipid volatility allows for sequestration in body fat, thereby greatly increasing their biologic half-life. A random survey of PBB levels in breast milk in Michigan revealed detectable body loading of PBB (Brilliant *et al.*, 1978)'and suggested the biostability of these compounds. The structural similarity of the halogenated biphenyls with that of thyroxine suggested that PBB's could potentially compromise thyroid function. Thyroid ultrastructural changes in the rat have been reported following PBB feeding (Kasza, 1978).

Results from a recent report (Allen-Rowlands *et al.*, 1981) clearly indicate that PBB's do extend both immediate and residual effects on thyroid function in the rat from chronic low level exposure. The degree in the reduction of plasma thyroxine (T_4) as a result of PBB ingestion was both dose and time dependent. Those animals given the lowest accumulative dose, 9 mg over 20 days, had a significant depression of plasma T_4 levels, compared to control animals, which persisted up to two months after cessation of PBB ingestion. PBB administration also resulted in a

significant reduction in the extrathyroidal incorporation of radioiodine into monoiodothyrosine, suggesting that PBB acts in some manner to inhibit the organification of iodine by peroxidase (Gilman and Marad, 1975). The effect of PBB on thyroid T_4 secretion produced the expected negative feedback stimulation of pituitary TSH secretion, thereby indicating that chronic exposure to small doses of this xenobiotic resulted in the disruption of the normal hormonal cascade pituitary-thyroid axis. The persistence of this antithyroid action by PBB was indicated by the continued depressed T_4 levels observed up to 5 months after cessation of PBB administration. These animal studies further demonstrated that even at the highest dose administered (6 mg/kg), where marked suppression in circulating T_4 occurred after only 10 days of treatment, there was no significant alteration in the body weight or the observed clinical appearance of the animal. In these same animals there was minimal alteration of behavior (Geller *et al.*, 1979), and no observed effect on adrenal or testicular function (Castracane *et al.*, 1982). These findings indicate the selective deleterious interaction of PBB with the pituitary-thyroid axis. The clinical relevance of these animal studies was indicated by the recent report of an increased incidence of hypothyroidism in a group of male workers exposed to PBB as evidence by increased pituitary TSH and decreased thyroid T_4 levels (Bahn *et al.*, 1980).

The metabolic changes of toxicants such as PBB to more aqueous soluble and readily excretable molecules are minimized due to their lipophilic nature. Therefore, sequestration in adipose tissue greatly augments the biological half-life of these xenobiotics. Mobilization of these lipid soluble compounds could occur with conditions of stress and thereby potentiate or augment the latent health effects of these xenobiotics. Another potential target of impact in health is that of the offspring of exposed mothers who are generally much more sensitive to toxic effects of xenobiotics. The above evidence suggests that without specific biological testing directed to detect predictable detrimental long-term interactions of the xenobiotics with biological processes, impairment of health could go undetected. Furthermore, impairment of key homeostatic processes could contribute to the latent development of a disease process such as cancer.

It is increasingly clear that for an effective occupational health care monitoring system to be developed, the available biochemical, biophysical and toxicological data management systems must be used, not only by researchers, but also by corporate, labor and governmental participants involved in the formulation of occupational health care policies. The NIH-EPA Chemical Information System contains spectroscopic, crystallographic, toxicological and regulatory data for more than 200,000 chemicals, with access by a local telephone call, on a fee for service basis (Milne *et al.*, 1982). The CIS has proved to be especially useful for modeling environmentally significant properties of certain chemicals, e.g., pesticides. The Oil and Hazardous Materials Technical Assistance Data System (OHMTADS), managed by EPA, assists scientific personnel in identifying toxic spills, and the Chemical Hazard Response Information System (CHRIS) makes available reference guides for hazard assessment and spill response methods. The Chemical Transport Emergency Center (CHEMTREC) of the Manufacturing Chemists Association, acts as a clearinghouse for spill information and provides immediate action response information for accidents (Cosmides, 1976b).

The President's Science Advisory Committee, in their 1966 report on the handling of toxicological information (PSAC Report) defined toxicological information as "all information descriptive of the effects of chemicals on living organisms or their component subsystems." Kissman (1980) emphasized that toxic substance control is clearly an area of growing public concern, and went on to discuss the variety of publications and services addressing toxicological information, emphasizing on-line information retrieval systems. Participants at the Symposium on the Handling of Toxicological Information also considered the availability of technical information and assessed its validity and usefulness (Cosmides, 1976a).

The Toxicology Information Program (TIP) of the National Library of Medicine was established in 1967, and a Toxicological Information Response Center (TIRC) was added in 1971. These systems were designed to create data bases with information taken from the scientific literature and from the files of cooperating governmental, academic and industrial organizations; and to provide a variety of toxicological information services for the scientific community (Cosmides, 1976b). The TIRC is located at the Oak Ridge National Laboratory, and provides a query response capability, including evaluative multimedia monographs, and state of the art reviews (Huff, 1976). TOXLINE, an on-line interactive bibliographic retrieval system based on the scientific literature, provides references to published human and animal toxicity studies, and contains full bibliographic citations. CHEMLINE, an on-line service based on chemical abstracts, and CANCERLINE, based on carcinogenesis abstracts, are also included in TIP.

The Toxicology Data Bank (TDB), designed to provide an on-line integrative computer based retrieval system, is another component of TIP. Selection of chemical data for inclusion in the TDB is based on three major considerations: (1) those toxic and potentially toxic chemicals most likely to reach a large segment of the population; (2) those agents shown to be carcinogenic, mutagenic or teratogenic; and (3) those substances identified by other governmental agencies as being important to their mission (Huff, 1976). Prior to acceptance into TDB, all records

undergo rigorous review by the National Institutes of Health's Toxicology Study Section for scientific content and merit.

Other data sources include the chemical registry services of the Chemical Abstracts Service (Tate, 1976), and the International Agency for Research on Cancer (IARC), a division of the World Health Organization. The IARC brings together experts on the carcinogenicity of various chemicals to help determine and evaluate the open literature relating to the carcinogenic hazard of chemicals to man. The IARC program represents information that has been evaluated by experts in the field (Siegel, 1976). The IARC Carcinogenesis Bioassay Program was designed to develop exposure categories for chemicals in the world around us, and to make estimates as to how these chemicals may be impacting man. The International Registry of Potentially Toxic Chemicals (IRPTC), part of the United Nations Environmental Program, provides published evaluations, data compilations and reviews of chemicals, and guidelines for developing toxic substances legislation. Another vital United Nations program, the International Referral System (IRS), links users with information by providing to interested parties a list of names and addresses of individuals with technical expertise about particular toxic substances (Cosmides, 1976b).

The Biomedical Sciences Section (BIOSCI) at the Oak Ridge National Laboratory provides reviews of the biologic effect of toxic compounds. Using a team approach, BIOSCI reviews focus on uptake and transport mechanisms, exposure potential, chronic and acute effects, and epidemiologic and clinical studies of human exposure to toxic substances in the environment (Huff, 1976). The Environmental Mutagen Information Center (EMIC) and Environmental Teratology Information Center (ETIC) are also located within the Oak Ridge National Laboratory, and are charged with monitoring the literature and responding to information requests (Wassom, 1976). Online services are also made available by Bibliographic Retrieval Service (BRS), Lockheed Information Service, and the System Development Corporation (Kissman, 1980).

Integrating the findings of biochemists, toxicologists and epidemiologists into the management decision making structure is not an easy task, but one which must be addressed. The current litigative model for resolving corporate responsibility and providing compensation for injury, although effective in instituting social policy, is expensive, time-consuming and cumbersome. On a large scale, exclusive reliance on the litigative approach toward occupational health problems does not provide a sound basis for the development of an effective rationl health care policy. Furthermore, this method of conflict resolution does not necessarily encourage preventive health care attitudes or practices on the part of management or employees. Clearly, we need a change in direction.

An effective information system for incorporating various kinds of toxicological and epidemiological data into decision making about occupational health would confer several advantages. First, better worker health would prevent the unnecessary loss of productivity due to illness, absenteeism or lowered output due to health problems. Second, fewer work related illnesses would reduce disability and compensation disbursements, as well as direct medical payments and indirect insurance costs. Third, an occupational health system which incorporates baseline health observations and routine monitoring of specific biologic systems suspect of being impaired by exposure to occupational toxicants could well act as a negative feedback system to preclude serious health problems, and to inculcate in the worker positive attitudes about the value of preventive health care. Feedback information derived from health monitoring of workers would probably reduce the risk of adverse health effects, and in doing so, provide a better self image for the worker who then feels some modicum of personal control over workplace risks.

This idea of co-responsibility of the worker and management obviously presupposes changes in the structure of contracts, the negotiation process, the parameters of management responsibilities and the patterns of information exchange. It also offers a perspective for evaluation and selection of a suitable approach toward the concept of "acceptable risk." The idea of co-responsibility represents an attempt to consider benefits and risks related to the same person. ◆

Part 2 in next issue.

POLYCHLORINATED BIPHENYLS

CAS RN: 1336-36-3
NIOSH #: TQ1350000

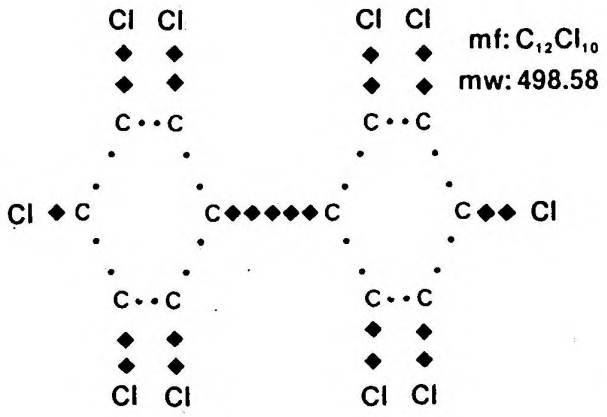
SYNS: PCB; polychlorinated diphenyls; askarels; aroclor; aroclor 1221; aroclor 1232; aroclor 1242; aroclor 1248; aroclor 1254; aroclor 1260; aroclor 1262; aroclor 1268; aroclor 2565; aroclor 4465; chlophen; chlorextol; chlorinated biphenyl; chlorinated diphenyl; chlorinated diphenylene; chloro biphenyl; chloro-1,1-biphenyl; chlorodiphenyl (42% chlorine); chlorodiphenyl (54% chlorine); dykanol; fenclor; inerteen; kanechlor; kanechlor 300; kanechlor 400; kanechlor 500; montar; noflamol; phenochlor; phenoclor; polychlorinated biphenyls; polychlorinated diphenyls; polychlorobiphenyl; pyralene; pyranol; PCB's; santotherm; santotherm FR; sovol; therminol FR-1; 1,1'-biphenyl, chlorinated; 1,1'-biphenyl, chloro derivs.

CHEMICAL FORMULA: $(C_5H_{5-x})Cl_x$

SPECIES IN MIXTURE: Technical polychlorinated biphenyls are mixtures of chlorinated biphenyls containing many kinds of isomers and species. Typical PCB mixtures may contain 3,4,5,6, and 7 chlorine atoms per biphenyl molecule in varying proportions. Technical mixtures may vary from mobile oily liquids to white crystalline solids and non-crystalline resins. Technical mixtures may contain traces of polychlorinated dibenzofurans which are considered more toxic than PCBs. (DPIMR 1(3)38,81/SAX)

COMMON USES: Coolants in transformers, fluorescent ballasts, electrical insulation plasticizer, epoxy paints, carbonless reproduction paper, lubricants; much of the total production of PCBs still in use as dielectric (insulator), heat transfer or hydraulic fluids. (DPIMR 1(3)38,81/SAX)

STRUCTURAL FORMULA:



STORAGE AND HANDLING INFORMATION:

TRANSPORT, RAIL (%): 40.3

TRANSPORT, BARGE (%): 47.8

TRANSPORT, TRUCK (%): 11.9

CONTAINERS: Former shipment containers include glass bottles (5L), earthenware (5L), plastic bottles (5L), and metal cans and drums (30 L and 250 L respectively) (85EZAO IMDG,77/IMCO) (49CFR 101.1) (RARAD5 80/IATA)

GENERAL STORAGE PROCEDURE: Store PCBs in isolated areas where the drums are not vulnerable to damage from vehicles, forklifts, or other moving equipment.

GENERAL HANDLING PROCEDURE: Slight fire hazards. Strong acute irritants via inhalation. Absorbed through skin and mucous membranes and eyes. (DPIMR 1(3)38,81/SAX)

PRODUCTION SITES: Monsanto, Anniston, AL; Sauget (E. St. Louis), IL; Not produced after 1977. Monsanto largest former producer.

HYDROLYSIS, PRODUCT OF: Not hydrolyzed under normal reaction conditions.

BINARY REACTANTS: PCBs are generally inert when placed in contact with other materials under normal conditions of temperature and pressure. However, strong sunlight conditions may cause the formation of phenolic materials and traces of polychlorinated dibenzofurans. (EVHPAZ 1,15,72/HUT) (BECTA6 10(6)372,73/CRO)

SYNERGISTIC MATERIALS: Carbon tetrachloride

DETECTION LIMIT (Lab; Techniques, Ref) (ppm): .001; gas layer chromatography; (E234)

STANDARD CODES: Superfund designated (hazardous substances) list; UN No. 2315; IMCO-UN class 9 miscellaneous dangerous substances, no label required. stow on deck or under deck for passenger and cargo ships, stow in a recoverable position separated from all foodstuffs. Packaging group II. (85EZAO IMDG,77/IMCO) NO IATA; CFR-class ORM-E, no label required, CFR packaging requirement 173.510, no limit on package size. (45FR34560, 5-22-80)

FLAMMABILITY: Nonflammable

TOXIC COMBUSTION PROD.: Emits highly toxic vapors when heated to decomposition.

FLASH POINT (C.): 200°

AUTO IGNITION POINT (C.): 240°

EXPLOSIVENESS: Stable

BOILING POINT (C.): 340°; 340° to 375°

BOILING CHARACTERISTICS: DPIMR 1(3)38,81/SAX

SPECIFIC GRAVITY: 1.182 to 1.44 (DPIMR 1(3)38,81/SAX)

PERSISTENCY: High; highly chlorinated forms of PCBs containing 5 or more chlorine atoms per biphenyl molecule are much more persistent in the environment than PCBs containing 1,2, or 3 chlorine atoms. Tetrachlorobiphenyls are considered intermediate in per-

sistence. (AWQC** PB81-117798,80/ECAO) Environmentally, approximately one chlorine atom of each chlorinated biphenyl is lost per year. (39KOAS 56,78/BUN) Microbial aerobic degradation studies using mixed cultures in water indicated.

TOXICITY:

POTENTIAL FOR ACCUMULATION: High in liver and fatty tissues. Eagle 14,000 ppm. Shrimp at 10 ppb for 48 hour had 1300 ppb. Perequin falcon 2000 ppm, oysters at 10 ppb for 96 hour had 33,000 ppb. Freshwater residue data show that PCBs accumulate to relatively high levels in invertebrate tissues and that for most species PCBs are not rapidly depleted when exposure is discontinued. Bioconcentration factors for invertebrate species range from 2700 to 108,000. Bioconcentration factors for PCB exposures of fish species range from 3000 to 274,000. (ECAO** PB81-117798,80/ECAD)

FOOD CHAIN CONTAMINATION POTENTIAL: Displays same accumulative characteristics as DDT and other chlorinated pesticides. Bioaccumulation and/or biomagnification of PCBs in the Lake Ontario ecosystem has been shown to occur via water, sediments, plankton, catfish, herring gull and eggs, and humans and human milk. (TEHEDH 4,81,80/SAF)

ETIOLOGICAL POTENTIAL: Renders mallards more susceptible to duck hepatitis. PCBs in combination with rubella virus cause an increase in cleft palate formation in offspring of pregnant mice (Teratogenic effect). (HKEGDK 7(1)21,80/WAT)

CARCINOGENICITY: Kanechlor 500 added to the dirt for 32 weeks at doses of 100 to 500 mg/kg resulted in hepatocellular carcinoma (liver cancer) in 5 of 12 male mice. (JNCIAM 51,1637,73/ITO) Chronic administration of aroclor 1260 to female rats at an average dose of 5 mg/kg/day for 21 months resulted in a 14% incidence of liver cancer and a 78% incidence of neoplastic nodules. (JNCIAM 55,1453,75/KIM) in 1979 PCBs were classified as "probably carcinogenic for humans" by a working group of the International Agency for Research on Cancer (IARC), an agency of the World Health Organization (WHO). (IMEMDT 1-20,1,79/IARC) Two retrospective mortality studies of a cohort of workers occupationally exposed to these chemicals have been conducted. In the U.S., two malignant tumors and four other cancers were diagnosed in 31 workers (or 19%) heavily exposed to aroclor 1254. In Japan, 9 of 22 deaths (or 41%) were due to malignant neoplasms (tumors in stomach, liver, lungs, and breast) after prolonged exposure to PCBs. (IMEMDT 18,78/IARC)

MUTAGENICITY: Aroclor 1221 causes mutagenic effects in salmonella typhimurium. Other aroclors including 1242, 1254, 1268 do not cause mutagenic effects. (RCOCB8 15,653,76/WYN) (IMEMDT 18,78/IARC)

TERATOGENICITY: Aroclor 1248, when fed to female rhesus monkeys, produced offspring suffering from chloracne, hyperpigmentation of skin, small head circumference and other teratogenic effects. In chickens, dogs, swine, mice, teratogenic effects include cleft palates, extra phalanges, shortened long bones, and other effects. (IMEMDT 18,78/IARC) In Japan, several teratogenic effects in offspring of patients suffering from yusho (oil) disease were noted, including skull deformations, increased melanin pigmen, small size for age, and stillborn infants. (PDTNBH 6(1)20,77/YAM)

CHRONIC AQUATIC TOXICITY LIMIT (Reference): PCB criterion to protect freshwater aquatic life is .014 .micro.g/l (parts per billion) as a 24-H average. (AWQC** PB81-117798,80/ECAO)

ACUTE WATERFOWL TOXICITY: 2000 ppm mg/kg LD50, mallards (D31)

CHRONIC WATERFOWL TOXICITY LIMIT: Causes thin eggs (E91)

MAJOR SPECIES THREATENED: Bird (egg production) aquatic life and predators.

INHALATION LIMIT (Text): Threshold limit values —skin exposure. Time-weighted averages at 42 and 54% chlorine respectively; short term exposure limits at 42 and 54% chlorine respectively. Skin exposure notation refers to the potential contribution to the overall exposure by the cutaneous route including mucous membranes and eye, either airborne, or direct contact with PCB itself. (ACGIH* TLV 80/ACGIH)

GENERAL SENSATION: Strong irritant. Can be absorbed through the skin. Causes liver damage and skin changes. The higher the chlorine content, the more toxic. Oxides are more toxic yet. Skin lesions characterized by small pimples and dark pigmentation. Later comedones and pustules develop. Systemic poisoning symptoms include nausea, vomiting, loss of weight, jaundice, edema, and abdominal pain. Can be absorbed through skin. Strong irritant.

DIRECT HUMAN INGESTION (mg/KGwt.): 500; 10 mg/m³; lowest toxic dose reported, LDLO, for humans by oral route is 500 mg/kg body weight for PCBs as a general class of compounds. Lowest toxic concentration, TCLO, for humans by inhalation route is 10 mg/m³ for aroclor 1242. (RTECS* 77/RTECS)

RECOMMENDED DRINKING WATER LIMITS (Reference): Due to the potential carcinogenic effect, the concentration of PCBs should be zero based on the non-threshold assumption. Since this level may not be attainable, levels which raise the human lifetime cancer risk 1E-5, 1E-6 and 1E-7 are allowed. These levels are .79 ppt, .079 ppt, and .0079 ppt, respectively. (AWQC** PB81-117798,80/ECAO)

PERSONAL SAFETY PRECAUTIONS: Wear personal protective clothing and use self-contained breathing apparatus.

ACUTE HAZARD LEVEL: Spill area should be considered as a source of strong inhalation irritant which is highly toxic when inhaled or absorbed by the skin. Protect

TOXICITY

FRESH WATER TOXICITY TEXT:

Conc.	Expos (Hr)	Specie	Effect	Test Environment	Reference
1.17-60	96	Trout	TLM		E91
.278	96	Bluegill	TLM		E91

SALT WATER TOXICITY TEXT:

Conc.	Expos (Hr)	Specie	Effect	Test Environment	Reference
.03	24	Saltwater Aquatic Life	Acute Toxicity		AWQC** PB81-117798, 80/ECAD

ANIMAL TOXICITY TEXT:

Value	Time	Species	Param.	Route	Refs.
500-1000		Rats			(1254) (D31)
2000-4000		Rats			(1268) (D31)
170		G. Pigs		Est.	E187
2000-3000		Mallard			E187
1000-3000		Pheasant			E187
600-3000		Bobwhite			E187
2000-5000		Coturnix			E187

fish and wildlife by containment procedures. Protect potable water supplies by containment procedures.

CHRONIC HAZARD LEVEL: Strong chronic irritant. Toxic. Skin absorption potential. Liver and skn disorders in humans. Reproduction abnormalities in humans and mammals. In birds causes thin egg shells.

DEGREE OF HAZARD TO PUBLIC HEALTH: While acute toxicity is reported low, typical contaminants of PCB are some of the most toxic materials known to man. Considered strong irritant. Highly toxic when inhaled or ingested. Chronically toxic with inhalation or skin absorption. Rapidly accumulated in food chain. Some of the hazards of the PCB's can be attributed to polychlorodibenzofuran contaminants (E190).

ENVIRONMENTAL IMPACT DUE TO A SPILL OR MASSIVE RELEASE:

AIR POLLUTION: Toxic. Volatilizes slowly from bodies of water.

ACTION LEVELS: Notify air authority. Restrict access to affected waters or land spill areas. Evacuate area if near homes.

IN SITU AMELIORATION: Seek environmental engineering assistance through EPA's Environmental Response Team (ERT), Edison, NJ, 24-hour no. 201-321-6660. Single-stage contactor dose of powdered carbon required to reduce the initial concentration (C.I.) of PCB-1232; PCB-1221 at 1.0 mg/L to a final concentration (C.F.) of .001 mg/L at neutral pH is 240;520 mg/L for C.I. .01 mg/L to C.F. .001 mg/L, use 2.2 5.7 mg carbon per liter. Granular carbon column doses for continuous operation until breakthrough (where effluent concentration equals influent concentration) are 1.6; 4.1 mg/L to treat a C.I. of 1.0 mg/l or a dose of .5; 1.1 mg/L to treat a C.I. of .01 mg/L. Multiplying the carbon dose given by 8000 will convert it to the number of pounds required per one million gallons of water. (CAITO* 80/DOB) Pump or vacuum undissolved portion from stream or pond bottom. Use carbon or peat on contaminated water. Absorb spilled portions on land with carbon or peat. Remove contaminated soil.

BEACH/SHORE RESTORATION: Absorb spilled portions with carbon or peat. Do not burn. Remove contaminated soil.

AVAL. OF COUNTERMEASURE MATERIAL: Pumps—fire department; vacuum—swimming pool suppliers; carbon—water treatment plants, sugar refineries; peat—nurseries, floral shops.

DISPOSAL METHOD: Capacitors (small and large); properly drained transformers; contaminated soil, dirt, rags, and other debris, dredge spoils; municipal sludges; and properly drained containers (drums) may be sent to EPA approved chemical waste landfill sites for burial. Liquid PCB waste must be stored and sent to incineration

facilities approved by EPA. Possible use of PCBX mobile unit from Sunohio for treatment of transformer oil containing not more than 1000 ppm arolcor. (PCBX** 81/SAX)
DISPOSAL NOTIFICATION: Contact EPA regional offices for location of EPA approved chemical waste landfills and incineration facilities.
INDUSTRIAL FOULING POT.: Not acceptable in food processing waters.
MAJOR WATER USE THREATENED: Fisheries, potable supply, recreation.
PROBABLE LOCATION AND STATE OF MATERIAL: Liquid, waxy solids, or resins. Will sink to bottom of streams or ponds and dissolve slowly.
SOIL CHEMISTRY: All absorb strongly on soils
COLOR IN WATER: Colorless

PCB
DATA REFERENCES:
TUMORIGEN
CARCINOGENIC DETERMINATION:HUMAN SUSPECTED
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
OCCUPATIONAL EXPOSURE TO POLYCHLORINATED BIPHENYLS recm std-air: TWA 1.0 ug/m'
"NIOSH MANUAL OF ANALYTICAL METHODS" VOL 1 244,253, VOL 2 S121, VOL 4 s120* NIMAM
NIOSH CURRENT INTELLIGENCE BULLETIN 7, 1975
REPORTED IN EPA TSCA INVENTORY, 1980
EPA TSCA 8E NO:07780209-FOLLOWUP REPLY RECEIVED AS OF APRIL, 1980

CODEN:
IARC 18,43,78**
EVHPAZ 1,105,72
JOCMA7 18,109,76
FEPRA7 34,1675,75
ARVPAX 14,139,74
ARPAAQ 94,125,72
CHRYAQ 49(4),14,76
STEVA8 2(4),305,74
BISNAS 20,958,70
27ZTAP 3,34,69
NTIS**

PCB (AROCLOR 1260)

MUTAGEN DATA:

CYT-RAT: ORL	1080	mg/kg/26W-C

TERATOGENIC DATA:

ORL-RAT TDLO:	1675	mg/kg (MGN)
ORL-MUS TDLO:	74	mg/kg (62D pre/1-10D preg)
SCU-MUS TDLO:	143	mg/kg (21D post)

TUMORIGENIC DATA:

ORL-RAT TDLO:	4380	mg/kg/83W-C TFX:CAR

TOXICITY DATA:

ORL-RAT LD50:	1350	mg/kg
SKN-RBT LDLO:	2000	mg/kg

DATA REFERENCES:
TUMORIGEN
MUTAGEN
TERATOGEN
CARCINOGENIC DETERMINATION: HUMAN SUSPECTED
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
OCCUPATIONAL EXPOSURE TO POLYCHLORINATED BIPHENYLS: RECM STD-AIR: TWA 1.0 ug/m3

CODEN:
APTOD9 19,A16,80

CODEN:
FCTXAV 12,63,74
ENVRAL 6,176,73
ENPBBC 5,54,75

CODEN:
JNCIAM 55,1453,75

CODEN:
FCTXAV 12,63,74
ARVPAX 14,139,74

CODEN
IARC** 18,43,78
EVHPAZ 1,105,72
ARVPAX 14,139,74
STEVA8 2(4),305,74
BISNAS 20,958,70
NTIS

PCB (AROCLOR 1254)

TERATOGENIC DATA:

ORL-RAT TDLO:	188	mg/kg (MGN)
ORL-RAT TDLO:	645	mg/kg (MGN)
ORL-RAT TDLO:	90	mg/kg (7-15D preg)
ORL-RBT TDLO:	350	mg/kg (1-28D preg)

TUMORIGENIC DATA:

ORL-RAT TDLO:	4	gm/kg/2Y-I TFX:ETA
ORL-MUS TDLO:	17	gm/kg/48W-C TFX:NEO
SKN-MUS TDLO:	4	mg/kg TFX:ETA

TOXICITY DATA:

ORL-RAT LD50:	1295	mg/kg
IVN-RAT LD50:	358	mg/kg
IPR-MUS LD50:	2840	mg/kg

CODEN:
FCTXAV 12,63,74
FCTXAV 12,63,74
FCTXAV 11,471,73
EVPHBI 1,67,71

CODEN
NCITR*
NCI-CG-TR-38,78
JNCIAM 53,547,74
BECTA6 18,552,77

CODEN:
FCTXAV 12,63,74
FCTXAV 12,63,74
BECTA6 8,245,72

DATA REFERENCES:
TUMORIGEN
TERATOGEN
CARCINOGENIC DETERMINATION: HUMAN SUSPECTED
TLV-TWA 500 ug/m3: STEL 1 mg/m3 (skin)
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
OSHA STANDARD—air: TWA 500 ug/m3 (skin)
(SCP-1)
OCCUPATIONAL EXPOSURE TO POLYCHLORINATED BIPHENYLS RECM STD-air: TWA 1.0 ug/m3
NCI CARCINOGENESIS BIOASSAY COMPLETED: RESULTS INDEFINITE: RAT
"NIOSH MANUAL OF ANALYTICAL METHODS" VOL 2 S121 NIMAM*

CODEN:
IARC** 18,43,78
DTLVS* 4,89,80
EVHPAZ 1,105,72
ARVPAX 14,139,74
RREVAH 44,1,73
STEVA8 2(4), 305,74
BISNAS 20,958,70
FEREAC 39,23540,74
NTIS**

NCITR*
NCI-CG-TR-38,78

PCB (AROCLOR 1248)

TERATOGENIC DATA:

ORL-MKY TDLO:	455	mg/kg (26W pre)
ORL-RBT TDLO:	165	mg/kg (1-31D preg)

TOXICITY DATA:

ORL-RAT LD50:	11	gm/kg
SKN-RBT LDLO:	1269	mg/kg

DATA REFERENCES:
TUMORIGEN
TERATOGEN
CARCINOGENIC DETERMINATION: HUMAN SUSPECTED
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
OCCUPATIONAL EXPOSURE TO POLYCHLORINATED BIPHENYLS: RECM STD-AIR: TWA 1.0 ug/m3
NCI CARCINOGENESIS BIOASSAY COMPLETED: RESULTS INDEFINITE: RAT
"NIOSH MANUAL OF ANALYTICAL METHODS" VOL 2 S121 NIMAM*T LD

CODEN
IARC** 18,43,78
DTLVS* 4,89,80
EVHPAZ 1,105,72
ARVPAX 14,139,72
RREVAH 44,1,73
STEVA8 2(4),305,74
BISNAS 20,958,70
FEREAC 39,23540,74
NTIS**
NCITR*
NCI-CG-TR-38,78

PCB (AROCLOR 1242)
TERATOGENIC DATA:

ORL-RAT TDLO:	945	mg/kg (36W pre)
ORL-RAT TDLO:	1890	mg/kg (36W pre)

TOXICITY DATA:

IHL-HMN TCLO:	10	mg/m3 TFX:IRR
ORL-RAT LD50:	4250	mg/kg
SCU-GPG LDLO:	345	mg/kg

DATA REFERENCES:
TUMORIGEN
TERATOGEN
CARCINOGENIC DETERMINATION: HUMAN SUSPECTED
TLV-TWA 1 mg/m3; STEL 2 mg/m3 (skin)
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
TOXICOLOGY REVIEW
OSHA STANDARD-air: TWA 1 mg/m3 (skin) (SCP-1)
OCCUPATIONAL EXPOSURE TO POLYCHLORINATED BIPHENYLS: RECM STD-AIR: TWA 1.0 ug/m3 - NTIS**
"NIOSH MANUAL OF ANALYTICAL METHODS" VOL 4 S120

CODEN:
AECTCV 3,479,75/76
AECTCV 3,479,75/6

CODEN:
85CYHB 2,153,59
TXAPA9 24,434,73
PHRPA6 59,1085,44

CODEN:
IARC** 18,43,78
DTLVS* 4,88,80
EVHPAZ 1,105,72
ARVPAX 14,139,74
RREVAH 44,1,73
STEVA8 2(4),305,74
BISNAS 20,958,70
FEREAC 39,23540,74
NTIS**

RECENT FEDERAL REGISTER CITATIONS (Polychlorinated Biphenyls))

CITATION NUMBER = 47.211.2
(47FR49471, 11-1-82)

NOTICE: National Institute of Occupational Safety & Health, Centers for Disease Control; Occupational exposure, possible adverse health effects, research project initiation. (47FR21302)

CITATION NUMBER = 47.223.58
(47FR52066ff, 11-18-82)

PROPOSED RULE: Revision of 40cFR430.174, 430.175, Environmental Protection Agency, Clean Water Act; Proposed effluent limitation guideline, pulp, paper, & paperboard point source category, New Stationary Source Performance Standard; Comments solicited by 1-17-83. (46FR1430)

CITATION NUMBER = 47.233.6
(47FR54436, 12-3-82)

FINAL RULE: Revision of 40FR761.3, Environmental Protection Agency, Office of Toxic Substances, Toxic Substances Control Act; Manufacturing, processing, distribution in commerce, use prohibitions; Use in electrical equipment. (47FR37342)

ADDITIONAL REFERENCES

CIS Sources of Information

CIS, EI Mass Spectrometry
NIOSH/CIS RTECS: TQ1360000
EPA/CIS, WaterDROP
CIS, Cambridge X-Ray Crystallography: 2051-24-3.01
CIS, CI Mass Spectrometry
EPA/CIS, OHM/TADS: 7216860

Non-CIS References

EPA, Pesticides—Analytical Ref. Stnds.: 5700E,5705
EPA, STORET: 39512,39504,39505,39506,39507
EPA, Pesticides—Registered Inert Ingredients
FDA/EPA, Pesticides Ref. Standards: 370
EPA, Effluent Guidelines Priority Pollutants
ORNL, EMIC
ORNL, ETIC
USFWS, Fish Control Lab Data Base
IARC, Bulletin on Chemicals Tested for Carcin.
EPA, TSCA Inventory List
IARC, Monographs (Carcinogenicity Reviews)
EPA, Expanded Potential Industrial Car. & Mut.
NLM, Tox Tips: 9:23,9:27,8:19,35:41,18:37
NLM, CHEMLINE: TOXLINE, TOXBACK
USAF, Defense Pest Mgt. Information Analysis Cntr.
OSHA, Air Contaminants
*NFPA *491M, Manual of Hazardous Chemical Reactions: 491M-338*
EPA, Potential Carcinogen Study, Selected Chem.
Aldrich Catalog/Handbook: 163279
USCG, CHRIS (Chemical Hazards Response Info System: PCB
EPA/IERL, Non-Criteria Pollutant Emissions
EPA/NCTR, Industrial Carcinogen and Mutagen Study
EPA, Environmental Carcinogen Assessment Program
EPA, Quality Criteria for Water
EPA, Chemical Indicators, Industrial Contamination
Michigan Critical Materials Register
DHEW/NIH, Laboratory Chemicals Monographs

or the environment, taking into account the economic, social and environmental costs and benefits of the use of any pesticide'. Whenever the Agency becomes aware of the potential for unreasonable effects, a pesticide may be subjected to an extensive scientific review and risk-benefit analysis.

Such a review was instigated for paraquat in 1978. Several potential adverse effects (or risk criteria) were identified: teratogenicity, lack of emergency treatment, chronic effects, reproductive effects. In response to the identification of these possible effects, the Agency undertook a comprehensive initial data screen involving approximately 5,000 papers, the majority of which were deemed inadequate for Agency evaluation. The Agency then concentrated its review upon selected papers germane to the evaluation of the risk criteria at issue.

With regard to the lack of emergency treatment, the Agency evaluated all paraquat poisoning incidents. It was found that nearly all fatal cases involved ingestion either as a result of suicide attempts or drinking from unlabelled storage containers. In either instance, little may be accomplished through more stringent regulation. The paraquat products implicated in poisonous incidents already bear restricted use classification. Current label precautions inform the user that the pesticide is not to be mixed or stored in any container other than the original. In addition to present precautionary labelling, the Agency believes that the Cavalli and Fletcher treatment regime offers a therapy demonstrated to be partially effective. The Agency has, therefore, adopted the position that the existing labelling, coupled with the Cavalli and Fletcher treatment regime, adequately address the issue of emergency treatment for exposure.

Available data does not allow for an assessment of the adequacy of emergency treatment for dermal absorption. Howwever, relatively few dermal exposure cases have resulted in fatalities. Incident data, principally thos obtained through the pesticide incident monitoring system (PIMS), have indicated an extremely limited number of dermal toxicity cases. The majority of these cases did not result in fatalities, but were related to dermal irritation, loss of fingernails and other subacute dermal effects. Again, paraquat products, with the exception of a homeowner use product containing a very low concentration of the active ingredient, bear restricted use classification. Applicators of such products are required to undergo training in the safe handling and use of pesticides and receive instruction in product labelling and label interpretation. Current paraquat products bear labelling instructions for mixers and applicators in exposure reduction techniques.

In relation to chronic effects, review of the available data indicated inadeqacies in the studies for nearly every chronic effect criterion. Insufficient or invalid data are available for oncogenicity, mutagenicity, and reproductive effects. The Agency has made no conclusion concerning these effects. The Agency will impose data requirements aimed at clarifying any potential risks related to these risk criteria. In the case of teratogenicity, two adequate studies have been reviewed. The Agency has concluded that paraquat does not exhibit any teratogenic effect.

The available information does not indicate conclusively the existence of adverse unreasonable effects. Therefore, no immediate regulatory action need be taken. Additional data will, however, be required of paraquat registrations, although the majority of the required testing has been either recently concluded or is in progress. The Agency anticipates the data to begin arriving within the next few months. Pending the receipt and evaluation of these data, paraquat has been returned to normal registration processing.

Source: United States Department of State, Washington, D.C., USA (February 1983).

POLYCHLORINATED BIPHENYLS (PCBs)

The Environmental Protection Agency (EPA) of the United States of America issued a final fule on 21 October 1982 amending the 31 May 1979 PCB rule which generally prohibits the manufacture, processing, distribution in commerce and use of PCBs (Bulletin, Vol. 3 No. 1 and Vol. 4 No. 2).

The rule-making exludes from the ban processes that manufacture PCBs as by-products and impurities as long as the PCBs released from the processes into the air, water, or products do not exceed the limits stated in this rule.

In the final rule: (i) EPA is setting numerical cutoffs for purposes of defining the absence of PCBs in releases from closed and controlled waste processes; (ii) EPA is adding additional disposal mechanisms to the list of acceptable mechanisms for the disposal of controlled wastes containing PCBs in concentrations between the limit of quantitation and 50 ppm; and (iii) EPA is instituting a new record-keeping requirement in addition to the record-keeping requirements listed in the proposed rule.

In this final rule, EPA is excluding from the requirements of section 6 (e) the manufacture, processing, distribution in commerce, and use of PCBs created in closed manufacturing processes and controlled waste manufacturing processes. A closed manufacturing process is defined as a manufacturing process that produces PCBs, but releases PCBs only in concentrations below the practical limits of quantitation for PCBs in air emissions, water effluents, products, and process wastes.

Similarly, a controlled waste manufacturing process is a manufacturing process that produces PCBs, but releases PCBs only in concentrations below the practical limits of quantitation for PCBs in air emissions, water effluents, and products, and all remaining PCBs are disposed of in accordance with methods for disposal specified in this rule. Controlled wastes containing PCBs in concentrations between the practical limit of quantitation and 50 ppm, must

Update / Continued

be disposed of in a qualified incinerator or in an EPA-approved PCB landfill, or be stored for incineration or landfilling.

For the purposes of this rule, the practical limit of quantitation for PCBs in any release to air is 10 micrograms per cubic meter (roughly 0.01 ppm) per resolvable gas chromatographic peak; in any release to water the limit is 100 micrograms per liter (roughly 0.1 ppm) per resolvable gas chromatographic peak; and in any product or waste, the limit is 2 micrograms per gram (2 ppm) per resolvalbe gas chromatographic peak. These PCB concentrations represent the lowest concentrations of PCBs which EPA believes can be pracitally quantified in air, water, products, and process waste streams. EPA believes that, for all practical purposes, it would be impossible to determine whether regulation of PCBs below these levels had any effect on actually reducing releases of PCBs. Consequently, EPA has concluded that there would be no measurable gain in protecting the environment or public health by attemtping to regulate PCBs at levels that are not practically measurable.

In specifying the methods for the disposal of controlled wastes containing less than 50 ppm PCBs, EPA is confident that these wastes will be disposed of in a manner that will result in little or no environmental contamination. At the same time, EPA believes that this rule will not place unreasonable burdens on existing disposal facilities or create excessive disposal costs for manufacturers disposing of wastes containing PCBs in concentrations between the practical limit of quantitation and 50 ppm.

In addition to meeting the criteria for eligibility described above, manufacturers who want to take advantage of the exclusion must fulfil certain record-keeping and reporting requirements. These include: (i) certifying that their processes qualify; (ii) notifying EPA that they have made this certification and how they have made the determination; and (iii) maintaining a record of the determination that their processes qualify for exclusion. Manufacturers are provided the option of conducting theoretical assessments to support certification or of conducting actual monitoring of PCB levels in releases. Recertification and renotification to EPA are required upon significant process changes.

In providing for theoretical assessments in lieu of actual monitoring of PCB levels, EPA has concluded that such determinations may be possible in certain process situations; therefore, it would br unreasonalbe to require actual monitoring of PCB levels in all situations. Manufacturers have the burden of making the decision on when a theoretical assessment in lieu of actual monitoring of PCB levels is appropriate. Because of the difficulty of estimating actual PCB levels, EPA recommends that a theoretical assessment be used to qualify for the exclusion only when the results of the theoretical assessment indicate that PCB concentrations in releases will be substantially below the practical limits of quantitation.

EPA is issuing some general guidelines for conducting a theoretical assessment in order to aid manufacturers in completing this assessment. Nonetheless, EPA expects that each individual manufacturer will exercise judgment in choosing the methodology to be used in conducting a theoretical assessment, and in deciding when to undertake chemical analysis of process streams to determine if a process qualifies for exclusion. EPA believes that record-keeping and reporting are necessary to ensure that only processes which meet the definitions of closed and controlled waste processes are permitted to operate under this exclusion. A reporting requirement also enables EPA to develop an effective compliance monitoring program.

Source: United States Federal Register No. 47 of 21 October 1982.

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