Chulabhorn Research Institute

INTERNATIONAL CENTRE FOR ENVIRONMENTAL AND INDUSTRIAL TOXICOLOGY (ICEIT)

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Migration From Food-Contact Materials

A recently published progress report by the U.K. Working Party on Chemical Contaminants from Food Contact Materials* details a major programme on paper and board. The programme included the analysis of 33 samples, which proved to contain 11 volatile substances at concentrations above 1 mg/kg. One of these was the carcinogen Michler's ketone, found in unbleached paper used for dry goods and paper bags. Cadmium, chromium and lead were found in some paper and board samples, but the levels were not of toxicological concern even if total extraction was assumed.

A detailed study of the migration of mineral hydrocarbons into food revealed levels of up to 100 mg/kg in individual portion packs of UHT milk, cream and butter, 105 mg/kg in skinless sausages, 550 mg/kg in wrapped bread, biscuits and breakfast cereals, 1300 mg/kg in confectionary products wrapped in waxed paper, and 1700 mg/kg in sandwiches and microwave-reheated foods encased in waxed paper bags. No mineral hydrocarbons could be detected in dairy products wrapped in polystyrene, acrylonitrile-butadiene-styrene or waxed board, beverages in polystyrene containers, or food simulants in contact with printed polypropylene and aluminium foil.

Studies on polymeric plasticizers showed that poly (isobutylene) migrated from polyethylene cling film into microwaved soup, baby food and macaroni cheese, and into sandwiches refrigerated overnight and then left at room temperature for 5 hr. Poly (butylene adipate) also migrated into foods under conditions of simulated home use, the lowest molecular weight fraction being most mobile. Neither poly (butylene adipate) nor poly (propylene adipate) was hydrolysed to monomers after 4 hr at 37°C in simulated gastric fluid, and only the latter showed some minor evidence of hydrolysis after the same treatment with simulated intestinal fluid.

Benzene in thermoset polyester cookware was found to result from *tert*-butyl perbenzoate used as a catalyst, and UK manufacturers obviated the problem by switching to alternative catalysts. Benzene was also identified in polystyrene packaging and non-stick cookware, but no migration could be detected into food cooked in the latter items, or on microwave susceptors. Further studies on benzene levels in a range of different food-contact materials, and on its migration into food, are now in progress.

Analysis of dual-ovenable cookware made of thermoset polyester revealed a large variety of potential migrants, of which three-ethylbenzene, benzaldehyde and styrene – were studied in detail. All three migrated into olive oil at 175°C, and styrene was also detected at concentrations in the range 1 to 2 mg/kg in pork belly after cooking for 1 hr at the same temperature. Several migrants, including antioxidants, were found in the aqueous extracts from plastic flasks and kettles, and these are being further examined. Preliminary studies have also been conducted on the transformation products of some major antioxidants during plastics processing, and work to identify such products is continuing.

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* MAFF Food Surveillance Paper No. 26. HMSO, London, 1989.

Source: Fd Chem. Toxic. Vol. 32, No. 4, 1994.

Epidemic Poisonings Caused by Pesticides: Safety Measures

Pesticides are biocidal agents used to kill organisms that are unwanted because of their effects on crops or because they are vectors of infectious diseases.

Pesticide poisoning in the human population has occurred via oral, respiratory and cutaneous routes of contact. The most important sources of collective pesticide poisoning have been accidental contamination of foods, contamination of clothes and other vehicles for cutaneous contact and occupational exposure.

The latter affects groups of workers in three typical situations: manufacture of pesticides, pesticide application, and crop management.

Many episodes have occurred among plantation workers and farmers spraying pesticides. The largest sprayer episodes, affecting 7,500 field workers in the Pakistan Malaria control program, occurred in 1976. In the peak month of the epidemic, it was estimated that there were about 2,800 cases. The poisoning probably led to the death of five workers (two mixers and three spraymen). Symptoms consistent with organophosphate intoxication were associated with a low level of cholinesterase activity in blood. This particular epidemic was linked with poor work practices that had originally been developed when DDT was the insecticide used for malaria control.

Toxic epidemics caused by pesticides can be prevented by the following measures:

- Discerning, selective use of chemicals in alternation with other methods of controlling pests.
- Bans on the manufacture of the pesticides that are the most dangerous as judged by their high acute toxicity or by their ability to persist and accumulate in biological entities, resulting in long-term risk; or, failing that, at least prohibition of their use on products that may be consumed by man or animals.
- Alteration of the organoleptic properties of toxic chemicals that are used in the domestic environment, to permit their differentiation from alimentary substances. In the case of treatment of seed, it is important that dyes or unpleasant admixture compounds should not be water soluble.
- Labelling to include: product composition; clear indications, written and drawn in a language or form understood by the user, concerning the toxicity of the product, safe method for its use and disposal, and the symptoms and treatment of poisoning; and advice to avoid change or subsequent use of pesticide containers as well as advice to store the product away from food and out of the reach of children.
- Training of health personnel and of populations at risk.
- Implementation of safe work practices in manufacture, spraying and harvesting operations, including individual safety measures and protective equipment, safety re-entry delays in treated fields, and bans on the agricultural use of most toxic products.

Source: Afr Newslett on Occup. Health and Safety 1994; 4:33-35.

Health Risks of Dioxins: EPA's Draft Report

The United States' Environmental Protection Agency (EPA) agreed in 1991 to review the health risks of dioxin. Recently, their draft report has been released for comment to specialist panels sponsored by industry and environmental groups.

The draft report concludes that dioxin, which is a byproduct of paper bleaching, incineration and other industrial processes, is more dangerous than has previously been claimed and that dioxin's current status as a "probable" human carcinogen at levels found in the environment should be retained.

As evidence, the agency cites animal studies and epidemiological data that tentatively link some forms of cancer to people exposed to large amounts of dioxin. Because it had no direct evidence that environmental levels cause cancer, however, EPA decided against elevating dioxin to a "known" human carcinogen.

But the report does suggest a link between minute quantities of dioxin and noncancer effects in humans. Fueled mainly by studies that have appeared in the last decade, the report asserts that dioxin – in levels found in the food supply – may trigger problems such as endometriosis in women and decreased sperm counts in men.

In its indictment of dioxin, EPA is including many similar compounds, which agency scientists believe exert similar effects. The underlying assumption is that the dioxin of utmost concern – 2, 3, 7, 8 TCDD – exerts its harmful effects by binding to the aryl hydrocarbon (Ah) receptor on the cell surface. Because some PCBs, furans, and other dioxins bind to the same receptor, EPA appraises the risk of exposure to these compounds based on their binding affinities.

Some critics of the report argue that this assumption is unwarranted because it fails to take into account how such chemicals might compete for binding sites in some cases or exert a synergistic effect in others.

EPA scientists, while defending the study, acknowledge the difficulty of writing a balanced summary of dioxin's health risks.

Source: Science. Vol. 265, 16 Sept. 1994.

Respiratory Sensitisation by Chemical Agents in the Workplace

Respiratory sensitisation occurs when there is an immunological reaction to an inhaled sensitising agent. Repeated exposure causes immunocompetent cells to produce antibodies – known as specific IgE antibodies against the sensitising agent. Subsequent exposures to the same agent result in the production of large quantities of the specific IgE. This IgE acts as a mediator in a complex system of cellular and bronchial wall inflammation. Respiratory sensitisers are divided into those of high and low molecular weight. Agents with high molecular weight are usually proteins including occupationally encountered agents such as flour-associated proteins, various wood dusts, as well as general environmental agents such as house dust mite proteins or grass pollens. Respiratory sensitisation to agents with low molecular weight usually occurs in the workplace. More than 150 such agents have been shown to be respiratory sensitisers.

The highest rate of sensitisation for many low molecular weight agents occurs in the first 18 months on exposure. Atopic people (defined as those with raised levels of specific IgE to common inhalant allergens) are at greater risk of sensitisation to some agents e.g. acid-anhydrides. This is the basis for the exclusion of atopic subjects from certain types of employment such as platinum refining.

Recently, attention has been paid to the examination of genetic susceptibility with the analysis of immune response genes which are thought to be responsible for the recognition of particular allergens. These genes form part of the human leucocyte antigen system.

Of particular importance is the observation that concurrent exposure to non-sensitising, respiratory irritants increases the risk of sensitisation to inhaled allergens. Cigarette smoking, for example, is associated with a higher incidence of specific IgE development in platinum refinery workers. The same may also be true for other irritants such as solvent fumes or chlorine gas inhaled in the workplace.

Source: Chemistry & Industry, No. 17, 5 Sep. 1994.

EPA Campaigns for Safer Chemicals

EPA has just unveiled a new program called Designing Safer Chemicals. A budget has been requested to promote research into the mechanisms of toxicity with a view to redesigning compounds to make them less hazardous. The program is predicated on the belief that a better understanding of what happens to industrial chemicals inside the body will make it possible to reduce or eliminate toxic and carcinogenic agents in dyes, paints, solvents, pesticides, weed killers, and other chemicals.

The first step is understanding how a particular toxin is metabolised. Once the mechanism of toxicity is known, it is often possible to remove the harmful agent with the simplest sort of substitution.

At the Center for Drug Design at the University of Florida, this technique has been employed to prevent drug side effects by creating an inactive relative of the drug that is activated by enzymes present at the site where it is needed, for example the eye or the skin. In principle, the same technique could lead to the production of safer pesticides by creating inactive compounds that become an active poison only on contact with chemicals unique to the biochemistry of a particular pest.

In promoting research of this kind in its new program, EPA will no longer be viewed by industry merely as a watch-dog. However, the redesign work will require a new breed of chemist: one who understands both synthesis and toxicology so as to be able to recognise trouble within a chemical structure. It will take time to train such skills.

Source: Science, Vol. 265, 9 Sep. 1994.

Atmospheric Pollution Due to Mobile Sources and Effects on Human Health in Japan

Air pollution caused by automobile exhaust and the effects on people living in areas where there is heavy traffic has become a major environmental health issue in Japan.

Since 1969, environmental quality standards have been set up (and revised) for sulfur dioxide (SO_2), carbon monoxide (CO), suspended particulate matter (SPM), nitrogen dioxide (NO_2) and photochemical oxidants (O_x). Emission and effluent regulations have been introduced to ensure compliance with these standards. The Environment Agency's 1990 report on the quality of the environment in Japan gives the following environmental quality standards:

Substance	Standard values	
Sulfur dioxide	1-hr	0.1 ppm
	24-hr	0.04 ppm
Carbon monoxide	8-hr	20 ppm
	24-hr	10 ppm
Suspended particulate matter	1-hr	0.20 mg/m^3
	24-hr	0.10 mg/m^3
Nitrogen dioxide	24-hr	0.04-0.06 ppm
Photochemical oxidants	1-hr	0.06 ppm

In the report of the Japanese Expert Committee for evaluating the relationship between air pollution and health hazards, published by the Environment Agency in 1986, the following conclusions were drawn, based on comprehensive assessment of evidence from animal experiments, experimental human exposure studies, epidemiological studies and clinical findings.

1) As was true of air pollution in the past, present-day air pollution is mostly attributable to the combustion of fossil-derived fuels. Therefore, it would not be a significant error if the present air pollution in Japan is dealt with in terms of three major air contaminants, SO_2, NO_2, and SPM. Of these three, the latter two seem to warrant particular attention, considering the current status of fuel consumption, countermeasures against air pollution and changes in the source of pollutants (especially changes in the structure of traffic systems).

2) It cannot be denied that air pollution as a whole has had some influence on the natural history of chronic obstructive pulmonary disease (COPD). From 1955 to 1965, COPD in individuals living in certain areas of Japan was chiefly attributable to air pollution, on the grounds that its prevalence was considerably higher in districts with high air pollution levels. However, it appears to be impossible to attribute COPD to air pollution alone.

3) Based on an assessment of the present level of air pollution in Japan in relation to COPD, we would like to call attention to the following points: What we studied was primarily the effects of environment air pollution on the population. Therefore, the effects of air pollution in particular areas, especially those with higher air pollution levels than the average, must be considered separately. Also, the existence of a group of people with increased sensitivity to air pollution has been attracting attention. If the number of individuals belonging to this group is not large enough, this group may be overlooked during epidemiological studies on the general population. This possibility must be borne in mind.

The Japanese Expert Committee affirmed the possibility that air pollution in Japan is affecting the natural history of COPD. In its opinion, the levels of air pollution in Japan are not high enough to clearly demonstrate a cause – effect relationship between air pollution and COPD, but they also are not low enough to rule out a causal relationship. The committee added that the influence of automobile exhaust on residents living along roadsides requires special consideration.

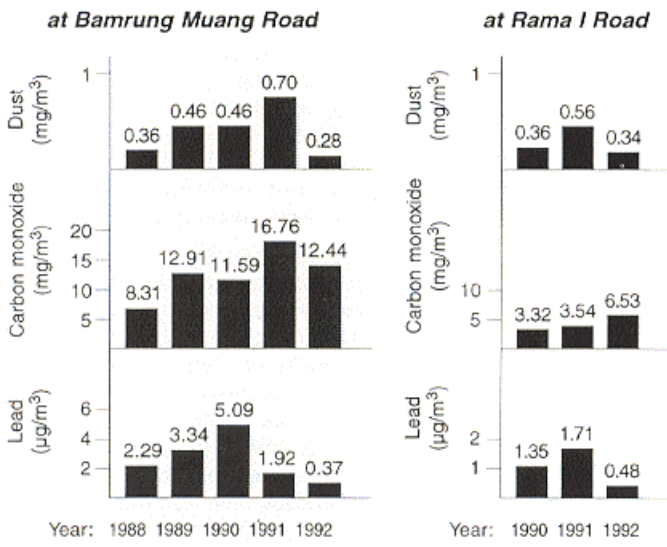
To evaluate the contribution of automobile exhaust to health hazards, it is necessary to evaluate the percentage of the total amount of air pollution to which individuals are exposed that is attributable to automobile exhaust. The effect of a given level of automobile exhaust exposure differs in individuals who are also exposed to air pollutants from other sources and in individuals who are not exposed to air pollutants from any other source. When city dwellers are exposed to air contaminated with pollutants from fixed and mobile sources to a degree close to the minimal exposure level presenting a health hazard, health effects appear likely to become manifest as they are exposed to indoor air pollutants or to automobile exhaust by living along roads with heavy traffic.

More research, including assessment of individual exposure to auto exhaust and assessment of health effects among roadside residents more highly exposed to auto exhaust, is necessary before any definitive conclusion concerning a causal association between exposure to auto exhaust and increased risk of respiratory symptoms and diseases can be drawn.

Source: Environmental Health Perspective, Vol. 102, Supplement 4, October 1994.

Air Quality in Bangkok

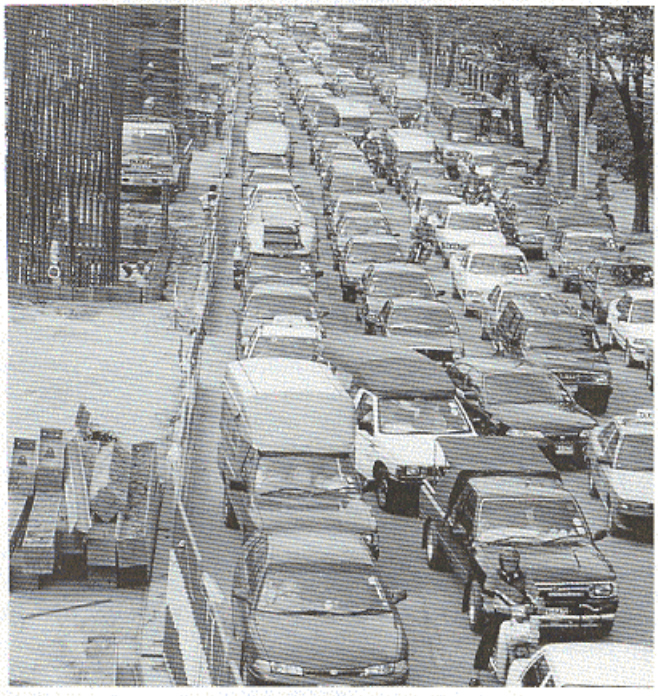
in areas of heavy traffic congestion



The tables for two areas in central Bangkok which experience heavy traffic congestion were given in the 1992 Report on Air Pollution in Thailand, published by the Pollution Control Department of the Ministry of Science, Technology and Environment.

The same report gives the following table of lead levels in the air in the central metropolitan areas of Pratunam, Yaowaraj, Silom, Sipraya, Patumwan, Huamark and Ramkamhaeng for 1992.

Lead	1991	1992	standard value
24 hr. average	0.6-2.3 µg/m³	0.2-1.5 µg/m³	10 µg/m³



Traffic congestion during the construction of an elevated expressway in the central metropolitan area of Bangkok.

Heavy metals in food

Area	Cadmium levels shop food	Cadmium levels street food	Lead levels shop food	Lead levels street food
Din Daeng-Suthisarn	0.02	0.04	0.30	0.11
Maensri-Bamrung Muang	0.03	0.03	0.10	0.11
Pratunam	0.02	0.07	0.31	0.28
Saphan Khwai	0.05	0.05	0.30	0.16
Wong Wien Yai	0.02	0.02	0.16	0.11
Yaowaraj	0.05	0.05	0.11	0.12
SAFETY STANDARD	***0.05 mg/kg***		***1.0 mg/kg***	

(***Source:*** 1994 survey by Mahidol University's Institute of Nutrition)

The Institute of Nutrition, Mahidol University, has carried out a study on the amount of lead and cadmium contamination in food sold by street vendors.

In Bangkok, there are over 300 pavements where vendors are allowed to operate and a total of some 17,000 vendors sell different types of food popular with residents and workers who need to eat quickly and cheaply.

Assistant Professor Songsak Srianuchart at the Institute of Nutrition says that according to recent surveys 51 per cent of people in Bangkok buy their lunch from street vendors.

Due to the volume of traffic in the metropolitan area, toxic substances in exhaust emissions as well as dust and carcinogens in the atmosphere could lead to health risks to consumers of street food.

For the Institute's study, samples of street food were collected from heavily polluted areas in Yaowaraj, Pratunam, Saphan Khwai, Din Daeng-Suthisarn, Maensri-Bamrung Muang, and Wong Wian Yai.

For purposes of comparison, tests were made on food shops located approximately 200 meters from main streets in the same areas.

The table gives readings for both lead and cadmium levels in shop and in street food in the six central areas. Importantly, lead level is well below the safety standard, and surprisingly, in some areas, shop food has a higher level of lead contamination than street food.

Assistant Professor Srianuchart observed that food in the Saphan Khwai and Din Daeng-Suthisarn areas was mainly composed of meat, which probably contributed to the high reading for shop food. Previous studies have shown that over 83 per cent of raw meat in Bangkok markets contains higher lead levels than those specified by existing health regulations.

Both shop and street food shows a higher level of contamination from cadmium than from lead, although this is still below the accepted safety standard. It is estimated that eating one dish of street food will contaminate consumers with 14.53 µg of lead and 4.29 µg of cadmium. This is below the standard set by the World Health Organization which allows for a daily lead intake not exceeding 43.6 µg and a daily cadmium intake not exceeding 71 µg.

The main source of cadmium contamination is from seafood. The Institute's study showed that seafood can be contaminated with cadmium levels as high as 0.18 to 3.15 mg/kg. However, rinsing with fresh water can help to reduce the amount of cadmium by up to 80 per cent.

The study recommends that government agencies should provide information on nutrition to vendors and consumers as well as advice to vendors on efficient methods to clean raw materials and food utensils and containers with small amounts of water.

Alzheimer's Disease – Understanding Its Causes

A group of researchers at Harvard University's Massachusetts General Hospital have reported biochemical data suggestive of a Zinc-Alzheimer's connection. Their research has shown that zinc ions can cause one form of the Aß protein (Aß) to form clumps resembling the amyloid plaques found in the brains of Alzheimer's patients.

The current work began in 1992 under the direction of Rudolph Tanzi. Scientists had known for some time that Aß was the main constituent of Alzheimer's plaques, and had recently shown that Aß is present in a soluble form in cerebrospinal fluid. The question was, what made the protein clump together in plaques only in the brain?

Tanzi's team decided to see how readily zinc and other transition metals could bind to Aß and prompt formation of amyloid clumps. Zinc, they found, had by far the most significant effect.

At low concentrations such as those found in cerebrospinal fluid, zinc bound to Aß without causing it to clump and precipitate out of solution. But when zinc was present at a concentration just above that, the peptide suddenly clumped. The clumps were similar in size to naturally occurring amyloid plaques, and they looked the same when stained and viewed with polarized light. Zinc did not have this effect on Aß produced by rats, which do not develop amyloid plaques as they age – and whose Aß differs from the human version by three amino acids. In the brain, Tanzi says it's quite plausible that if the strict regulation of zinc broke down, sufficient concentrations of zinc could contact Aß and form it into clumps.

Other researchers, though they praise the intriguing nature of this work, stress that the findings are very preliminary – and only from the test tube. Experiments are now being carried out with solutions that more closely mimic the environment of the brain.

Source: Science. Vol. 265. 2 Sept. 1994.

Aluminium in Food

In the United Kingdom, the Steering Group on Chemical Aspects of Food Surveillance has re-investigated levels of aluminium in food, which it last considered in 1985.[1] From the 1988 total diet study, the average dietary intake of aluminium in the UK was estimated to be 27 mg/week which is well below the benchmark provisional tolerable weekly intake (PTWI) of 7 mg/kg body weight for an adult.

Some concern was expressed in 1989 about the relatively high levels of aluminium then found in infant formulae.[2] From a 1990 survey, daily intakes have now been estimated in the range of 0.27 to 0.53 mg from soya-based formulae and 0.03 to 0.05 mg from cows' milk based formulae. These were well below the levels previously reported, and indicated the success of an industry programme to reduce aluminium content. For young Caucasian and Asian vegetarian and non-vegetarian children, daily intakes were in the range 1.5 to 4.2 mg, levels well within the PTWI of 21-42 mg for a 3 to 6 kg infant and 70-140 mg for a 10 to 20 kg child. A review of recent studies on aluminium toxicity in the present report[3] indicates that the benchmark PWTI remains appropriate. There is no evidence that the occurrence of Alzheimer's disease is affected by aluminium in food, and several epidemiological studies suggesting an association with aluminium in drinking water are difficult to interpret, unless aluminium is more readily absorbed from water than from food.

① Survey of Aluminium, Antimony, Chromium, Cobalt, Indium, Nickel, Thallium and Tin in Food. Fifteenth Report of the Steering Group on Food Surveillance, the Working Party on the Monitoring of Foodstuffs for Heavy Metals, Food Surveillance paper No. 15. HMSO, London 1985

② MAFF Food Facts. 3 89.20 April 1989

③ Aluminium in food. The thirty-ninth report of the Steering Group on Chemical Aspects of Food Surveillance. MAFF Food Surveillance Paper No. 39. HMSO, London, 1993

Source: Fd Chem. Toxic. Vol. 32, No. 4, 1994.

PCB'S AS ENVIRONMENTAL ESTROGENS

Polychorinated biphenyls (PCBs) are widespread, low-level environmental pollutants associated with adverse health effects such as immune suppression and teratogenicity. There is increasing evidence that some PCB compounds are capable of disrupting reproductive and endocrine function in fish, birds and mammals, including man, particularly during development.

Reproductive disorders resulting from exposure to xenobiotic estrogens may include reductions in fertility, lower hatch rates in fish and birds, and decreased viability of offspring, as well as alterations in hormone levels or adult sexual behaviors. There is increasing suspicion that effects of estrogenic compounds are correlated to disorders of the male reproductive system, including increased occurrence of prostatic and testicular cancers. There is a need for a sensitive bioassay with which the developmental effects of environmental estrogens can be determined.

Researchers from the Department of Zoology, Institute of Reproductive Biology, University of Texas at Austin and the National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina have conducted a study to determine whether reptiles with temperature dependent sex determination (TSD), in which the incubation temperature of the egg determines the sex of the individual, may provide such a bioassay.

Their findings from experiments on eggs of the red-eared slider turtle, *Trachemys scripta,* indicate that the nature of TSD in this and other species provides a useful system in which to assay the extent of estrogenic activity found in xenobiotic compounds. The laboratory evidence that their research provides emphasizes the usefulness of a TSD species as a biomarker to assess environmental contamination and serve as a warning of conditions threatening wildlife populations.

The study also supports the contention that environmental estrogens have the potential to alter wildlife populations as well as to contribute to reproductive dysfunction in humans. By using the TSD model the researchers demonstrate that some PCBs have an estrogenic effect. Future studies investigating the mechanisms through which these estrogenic compounds act to affect sex differentiation will continue to shed light on a human contribution to the environment.

Source: Environmental Health Perspective, Vol. 102, No. 9, Sep. 1994.

RISK ASSESSMENT OF MERCURY CONTAMINATION

The various chemical forms of mercury have intrinsic toxic properties that render them harmful to humans. Of greatest concern are the organomercury species, which are lipophilic and bioaccumulate in the food chain. In most cases the predominant exposure pathway for humans is consumption of fish containing high mercury levels. Concern for exposure via fish consumption is reflected in the U.S. Food and Drug Administration action level for mercury in fish of 1 µg/g wet weight (3.6 µg/g dry weight). Understanding the transport, transformation, and fate of mercury in the environment is critical, given the elements' potential for methylation and subsequent biomagnification in the food chain.

Unfortunately, measuring trace levels of mercury is complicated by the fact that it is ubiquitous in nature, and ultraclean sampling and analytical protocols are required to prevent accidental contamination.

It was for this reason that researchers from the Department of Environmental and Resource Sciences, University of Nevada and the Desert Research Institute, Biological Sciences Center, Reno, Nevada elected to carry out their study of mercury transport, fate, food chain contamination, and effects on the biosphere in the Carson River Drainage Basin of west-central Nevada where mercury contamination is high and the duration of exposure is long. The latter factor is important since biomagnification is time dependent, and long exposures are likely to result in mercury-specific responses in sensitive organisms. These responses include accommodation at the physiologic or biochemical level, genetic adaptation at the population level, and behavioral avoidance mechanisms at the species level.

Approximately 5.5 x 10^9 g (4.0 x 10^5 L) of mercury was discharged into the Carson River Drainage Basin during processing of the gold and silver rich Comstock ore in the late nineteenth century. For the past thirteen decades, mercury has been redistributed throughout 500 Km^2 of the basin, and concentrations are some of the highest reported values in North America.

The current study assesses and documents mercury contamination in the geosphere, hydrosphere and atmosphere within the Carson River Drainage Basin; the background mercury concentrations outside the area of contamination but within the mercuriferous belt; the total mercury and methyl mercury (CH_3Hg) in surface waters of the river basin; comparative mercury levels in multiple media in contaminated and uncontaminated areas in western Nevada with other sites in North America; and the risks to humans and other organisms in the Carson River Drainage Basin.

With regard to the issue of risk assessment for human health and ecology, the researchers emphasize that the framework for addressing at-risk populations places a premium on understanding the pathways of mercury exposure and measuring specific rates of contaminant transport to receptors.

It is generally assumed that the principal pathway for mercury exposure in humans is food consumption; however, the present study indicates that in the Carson River Drainage Basin the pathways by which humans are exposed to mercury are likely to be more complex, extending to inhalation, dermal contact, and ingestion of contaminated water. Each single pathway for mercury exposure may be individually low but significant to the total body burden of mercury.

Concern for multiple exposure pathways in the Carson River Drainage Basin is also appropriate for ecological risk assessment and, as a result of their study, the researchers propose that there is a far greater probability of biological effects occurring in nonhuman versus human species in both terrestrial and aquatic environments. This is a consequence of several factors but largely reflects the distinctive nature of the Carson River Drainage Basin riparian and wetland environment, which focuses biological activity in areas that have contained very high levels of mercury for many decades.

Source: Environmental Health Perspectives, Vol. 102, No. 9, Sep. 1994.

Mercury in the Umbilical Cord: Implications for Risk Assessment for Minamata Disease

Minamata disease, first recognized in the 1950s, derives its name from a factory of the New Japan Chisso Fertilizer Company where the discharge of mercury compounds led to contamination of fish and shellfish. It was only later that methylmercury was finally identified as the causative agent of the disease. By that time, data on individual exposures were difficult to obtain retrospectively. However, because of a local tradition of keeping dried umbilical cords, researchers were able to collect cord tissue from 12 patients who had been born with congenital Minamata disease. For comparison, similar cord specimens were obtained from 16 children with other mental disturbances and 64 other children who served as controls.[1] The analyses showed considerably higher mercury concentrations in the Minamata patients compared to the control groups, but the distributions overlapped, particularly between Minamata disease and other mental disturbances.

The data, although supporting the notion that Minamata disease was due to methylmercury poisoning, were difficult to translate into dose-response relationships. The most frequently used measures of methylmercury exposures are mercury concentrations in

(Continued on page 8)

Advance Notice

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The Third Princess Chulabhorn Science Congress

Water and Development: Water is Life
will be held from 11 – 15 December 1995
at the Shangri-la Hotel, Bangkok, Thailand.

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The Chulabhorn Research Institute will organize the Third Princess Chulabhorn Science Congress on "Water and Development: Water is Life" to commemorate the auspicious occasion of the fiftieth anniversary (Golden Jubilee) Celebrations of His Majesty King Bhumibol of Thailand's accession to the throne.

This Congress, the third in an internationally acclaimed program, will provide a forum for both experts and practitioners in the field of science and engineering and also economics, law, health, water resources management and technology, environmental, social and political sciences to exchange knowledge and experience on use of water resources for sustainable development.

The scientific program of the Congress, under the direction of Professor Dr. Her Royal Highness Princess Chulabhorn D. Mahidol, addresses research, education and training, planned and on-going implementation and regional and international co-operation within a multidisciplinary framework. The program will comprise plenary lectures, workshops, symposia, contributed papers, poster sessions and exhibitions on the following aspects of water use:

- Economics
- Legal and Institutional Aspects
- Management: Engineering and Scientific Aspects
- Water and Health
- Social and Cultural Aspects

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Mercury in the Umbilical Cord: Implications for Risk Assessment for Minamata Disease

(Continued from page 7)

hair and in blood, but no such data were available from the original Minamata incident.

However, a recent research study[2] has examined the relation between the mercury concentrations in umbilical cord tissue (VC-Hg) with that in maternal hair and in umbilical cord blood. The samples were obtained from a cohort study in the Faroe Islands where mercury exposures from seafood may approach those that occurred in Minamata.

Umbilical cord tissue was obtained from 50 births. The mercury concentration correlated significantly with the frequency of maternal whale meat dinners during pregnancy and with mercury concentrations in umbilical cord blood and in maternal hair. The results were compared with published values for mercury in umbilical cord tissue from 12 infants diagnosed with congenital methylmercury poisoning in Minamata, Japan. From the regression coefficients obtained in the Faroese samples, the median umbilical cord mercury concentration of 4.95 nmol/g dry weight in Minamata would correspond to 668 nmol/1 cord blood and 114 nmol/g maternal hair. These levels agree well with other evidence of susceptibility of the fetus to increased exposure to methylmercury.

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① Harada M. Mental deficiency due to methylmercury poisoning. [English summary]. Brain Dev 6:378-387 (1997).

② Grandjean P, Weihe P, Jorgensen PJ, Clarkson T, Cernichiari E, Videro T. Impact of maternal seafood diet on fetal exposure to mercury, selenium, and lead. Arch Environ Health 47:185-195 (1992).

Source: Environmental Health Perspectives, Vol. 102, Number 6-7, June-July 1994.

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