

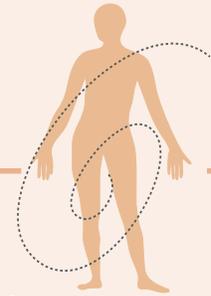


DEPARTMENT OF ENVIRONMENTAL HEALTH

# ENVIRONMENTAL HEALTH

# *N e w s*

SCHOOL OF PUBLIC HEALTH AND COMMUNITY MEDICINE ■ UNIVERSITY OF WASHINGTON ■ AUTUMN 1998



## EXPOSURE ASSESSMENT

*What is exposure assessment?*

- 1) *Surveying your sunburn after a day at the beach?*
- 2) *Deciding whether a photographic print has remained in the developing bath long enough?*
- 3) *Determining how an environmental pollutant comes into contact with the human body?*

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### ATTENTION READERS!

WE ARE UPDATING OUR MAILING LIST. PLEASE RETURN THE ENCLOSED PREPAID CARD BY JANUARY 15, 1999, TO CONTINUE TO RECEIVE ENVIRONMENTAL HEALTH NEWS.

PLEASE NOTE THAT YOU MUST REPLY TO REMAIN ON THE MAILING LIST. THANKS!

Of course all of the above definitions are usable, and have, or could have, some connection with environmental health. But, as you may have guessed, we're interested in the third definition, a theme for this issue of *Environmental Health News*.

To help us understand exposure assessment, DEH's Dr. Sally Liu shared the text of a talk given by Dr. Wayne Ott of Stanford University in 1995. In elaborating on the third definition, Dr. Ott stressed that "the key word in the definition is 'contact'—the occurrence of two events" coming together. Rather than focusing on, say, how much of a certain pollutant is coming out of a factory over a 24-hour period, or what a measuring instrument is showing concerning carbon monoxide in the air next to a freeway, exposure assessment is concerned with whether and how a pollutant is actually reaching and affecting a particular person.

The phrase which Dr. Ott uses to sum this up is "pollutants-making-contact."

Exposure assessment, he notes, "makes us consider new measurement techniques, such as personal monitors that are worn by human beings to assess the concentrations making contact with their bodies. It makes us

consider new concepts that place human beings at the center of our focus, such as human activity pattern research [having people keep journals or logs], which examines what people do in time and space. It makes us incorporate probabilistic sampling designs into our monitoring field studies. It makes us consider new kinds of mathematical models, such as total exposure models designed to predict the exposure distributions of large populations."

Implied in the assessment concept is "the important idea that exposures actually differ from person to person," for a great number of reasons. The research of DEH's Dr. Crispin Pierce, described in this issue, illustrates how two people exposed to the same degree of a toxicant may be affected quite differently. And exposure differences have to do not just with differences in human physiology, but with differences in human understanding and behavior as well.

In our last (Spring/Summer 1998) issue of *Environmental Health News*, we described how DEH scientists are studying the pathways by which organophosphate pesticides reach children—pathways which include diet residues in food, pesticide residues in dirt in agricultural

*continued on page 2*

## EXPOSURE ASSESSMENT

*continued from page 1*

areas, and others. To further understand exposure assessment, let's take a look at the difficulties pointed out by PhD student Ed Doran in a talk he gave on orchard worker exposure to pesticides.

Knowing that a certain orchard was sprayed with a certain pesticide doesn't tell us very much. First of all, Doran explained that large orchards are sprayed in blocks, on different schedules, so you have to know when a certain block was sprayed. Next, before a worker went to work, how much pesticide was still on the leaves of the trees? Did it, for example, rain overnight before a worker began work next morning? Next, what is the "transfer factor" from the leaves to the worker: What exactly is the worker doing? Did the person wear gloves or other protective gear? Next, how long did the worker actually work—how long was the length of his or her shift, and was the person in the orchard the whole time? Next, what was the degree of skin absorption of the pesticide? This involves, for one thing, how soon the person washed after finishing work. Scientists refer to the "post-shift dose," which is the time before the person washed and during which the person's skin is *still* absorbing residues from any pesticides that are on the skin. Washing does not remove all residues, but this aspect of exposure points out the importance of washing facilities for agricultural workers and their families.

In this issue, we focus particularly on exposure assessment and air pollution. We also feature articles on exposures to arsenic. And we give you a profile of DEH's new interim chair, Dr. David Kalman, who has conducted research on the use of urinary biomarkers to assess arsenic exposure. ■

Many drivers know that certain gasoline additives—such as ethanol or ethers—are sometimes added to car fuel to reduce environmental health dangers. This may be done on a seasonal basis or year-round, depending upon one's city or state. Unfortunately, some ethers are themselves under suspicion as possible causes of health problems.

Some studies have found increased tumors in rodents following very high ether exposures. One former oil company toxicologist (Dr. Myron Mehlman, subsequently an adjunct professor at Robert Woods Johnson Medical School) has called one ether, MTBE, a serious threat to public health. His assessment derives from a survey of oil refinery workers involved with MTBE; the workers complained of shortness of breath, fatigue, headaches, and even some seizures.

There have also been health and odor complaints from people more casually involved with MTBE—car drivers and citizens in its vicinity who are bothered by its smell. However, the number and type of complaints have varied greatly from place to place, and shown contradictions. To further complicate the picture, it is possible that the addition of MTBE to gasoline may actually lower health risk by reducing exposure to other constituents, such as benzene, a known human carcinogen. Since millions of people are now being exposed to MTBE, more research is clearly needed.

The Department of Environmental Health's Dr. Crispin Pierce has received a three-year grant from the National Institute for Occupational Safety and Health to do pioneering research on the toxicokinetics of fuel ethers. Toxicokinetics traces the fate of chemicals once they are taken into the body; it is the study of how the body absorbs, distributes, metabolizes, and excretes substances. Both short- and long-term biological responses are greatly influenced by factors specific to a particular person.

In previous work that laid a groundwork for this new project, Dr. Pierce and colleagues Dr. David Kalman, Dr. Michael Morgan, and Dr. Russell Dills have worked with toluene, another volatile substance contained in gasoline. They have developed computer models that probe why individuals exposed to the same amount of a toxicant end up with different body burdens.

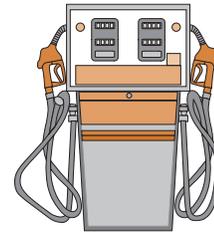
In studying toluene, Dr. Pierce's research team found that a person's age, weight, rate



Dr. Crispin Pierce

Photo: G. Paul Bishop

# THAT GAS TANK



of breathing, and amount of fat tissue all play a role in determining how much toluene is absorbed, where it goes, and how quickly it is eliminated in the case of a particular person. Also relevant is the ease with which a toxicant, once breathed into the lungs, passes into the blood. This factor, known as the “blood/air partition coefficient,” varies, and is a useful predictor of pharmacological and toxicological effects. Later study by one of Dr. Pierce’s graduate students, Therese Mar, revealed a gender difference: women tended to retain toluene in the blood a little longer than did men.

To study ethers, methods similar to those used with toluene will be employed by the research team. Male and female volunteers will inhale ethers at levels they might be exposed to while pumping gas, while the volunteer’s blood will be sampled before, during, and after inhalation. During exposure, alternating periods of stationary bicycling and rest will be used, since it is known that breathing rate affects toxicant uptake. Particular attention will be paid to gender differences. Research using ethers will evaluate whether, as with toluene, gender and other person-specific factors are predictive of amounts in the body.

In working with toluene, DEH researchers used various tools that included isotope tracers and measurements for toluene metabolites (conversion products) in urine. It turned out that toluene levels in the breath proved to be the most accurate indicator of absorbed dose, an important practical finding. Since laboratory sampling techniques are rather complex (and sometimes not particularly pleasant), it is highly desirable to have a simple, noninvasive way to measure an individual’s absorption of a toxicant. The finding that toluene levels in the breath are an accurate indicator of absorbed dose has led Dr. Pierce, in work with toluene, to use breath bags, which can be used anywhere. A breath bag is a plastic, rectangular bag that can be inflated in about two minutes by breathing into an attached tube; the bag is then taken to a lab for content analysis.

Measurement techniques have become so sophisticated that substances can be measured in molecular parts per trillion. And through the physiologically based computer models that Dr. Pierce has developed, it is possible to do “back-calculation”—that is, to measure the amount of toluene in breath samples and thereby estimate the amount in the body and the concentration in ambient air that led to this amount. The ability to back-calculate means that measuring environmental exposures to toxicants need not be limited to direct measurements in air, food, or water.

Understanding the toxicokinetics of substances like toluene and ethers enables us to also better understand the kinetics of more toxic substances, like benzene and PCBs, which are too hazardous for human volunteers to sample. So the research has implications that go beyond understanding what people around the country are breathing when they stop to pump gas. At the same time, the work should help us learn more about the health effects of two common gasoline additives, MTBE and also ETBE. ■■■

## OXYGENATED FUELS

This winter, motorists in the Puget Sound region will not be pumping oxygenated gasoline into their cars and trucks, as during some years in the recent past.

From November to February, in the years between 1993 and 1996, ethanol, rich in oxygen, was added to local gasoline in order to promote better combustion in cold engines, thus reducing carbon monoxide in car exhaust. Federal regulations, implemented by state and local bodies, required improvements in air quality, and the region was able to come into compliance with carbon monoxide standards fairly easily. A variety of factors were responsible, including the fact that newer cars no longer emit as much carbon monoxide as older models. Currently, the Puget Sound Air Pollution Control Agency projects no need for winter-oxygenated fuel within its jurisdiction before the year 2010.

Other places in the country have not been able to deal so easily with carbon monoxide. In 35 metropolitan areas and the entire states of California, Massachusetts, Connecticut, Rhode Island, and Delaware, substances called ethers (rather than ethanol) are used to add oxygen and reduce carbon monoxide and hydrocarbons in tailpipe emissions.

# FINE PARTICLE AIR POLLUTION

Fine particle air pollution is a research area that illustrates well the difficulties involved in exposure assessment. As *Environmental Health News* noted last winter, strong evidence indicates that, especially in sensitive populations, small inhalable particles such as those put out by wood burning and automobiles can impair lung functioning, aggravate chronic lung conditions such as asthma, lead to increased lung cancer, and cause premature death.

However, scientists do not agree that particles or particle size (for soot, smoke, or dust) are necessarily the main cause of the illnesses and deaths noted above. Gases such as carbon monoxide, nitrogen oxides, sulfur dioxide, ozone, or organic vapors may be involved. As DEH's Dr. David Eaton has commented: "A big issue is whether the particles themselves are responsible for ill effects or whether something carried along with the particles is the cause. If particle size and, say, the amount of harmful gases vary in parallel, regulating particle size could take care of the issue. But if they don't vary together, or if particle size is *not* the cause," we could end up spending millions of dollars without solving health problems.

The Department of Environmental Health has several faculty members who are working on air pollution issues. To gain more insight into the fine particle problem, *Environmental Health News* contacted four faculty to learn about current research on this issue—Dr. Eaton, Dr. Jane Koenig, Dr. Sally

Liu, and Dr. David Kalman.

Dr. Koenig explained the historical origins of concern with fine particles. Although the US Environmental Protection Agency (EPA) began setting standards for particulate matter in air as long ago as 1971, it was in the early 1990s that research on fine particles done in Philadelphia found that an increase in  $PM_{10}$  particles in air correlated with increased deaths in that city. (A  $PM_{10}$  particle is 10 micrometers [microns] or less—in diameter. A micrometer is one millionth of a meter.) *The New York Times* picked up "the Philadelphia story" and projected as many as 60,000 US deaths annually from  $PM_{10}$  pollution. The American Lung Association decided to sue EPA for new particle standards. The eventual outcome was that, in the summer of 1997, EPA issued new standards for even smaller particles (equal to or less than 2.5 micrometers), while retaining, with one minor adjustment, the standards for  $PM_{10}$  particles.

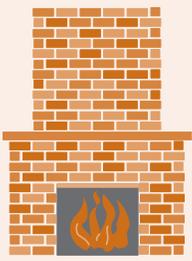
Dr. Kalman pointed out that EPA's new standards for  $PM_{2.5}$  particles are based on a number of epidemiological investigations (which found mortality rates more significantly affected by  $PM_{2.5}$  than  $PM_{10}$ ), not on any knowledge of the way in which air particles contribute to health problems. Additionally, patterns of illness may vary in different parts of the country. Both Dr. Kalman and Dr. Koenig noted that, in the East, the burning of coal to produce electricity releases sulfates that contribute to acid haze. In

Washington State, our primary power source is much cleaner hydropower.

In our Northwest region, Dr. Koenig pointed out, the burning of organic materials (from woodstoves, field burning to clear wheat stubble, grass field burning, and forest fires—whether deliberately set and controlled or accidental) needs to be considered in looking at particulate matter pollution. This September, according to press reports, farm field burning in Eastern Washington and northern Idaho resulted in smoke detectors going off inside at least one home; it also dumped ash on cars and homes in Spokane and Pullman and triggered angry calls to air pollution agencies. The Washington State Department of Ecology called off wheat-stubble burning in Whitman County as Spokane's air quality deteriorated.

Dr. Sally Liu has received a three-year grant from EPA to sort out some of the issues involved in fine particle pollution. She will study how particles are affecting local high-risk subpopulations. EPA's new standards for  $PM_{2.5}$  are set to protect the most vulnerable, who are generally the sick, older people, and the very young.

As Dr. Liu commented, the fact that air monitoring equipment is collecting particles at a certain site in a Seattle neighborhood does not take into account the fact that "people have different activities." Such factors as smoking, having a woodstove or a fireplace, living close to a road, or even using a backyard barbecue grill or poorly equipped



"A big issue is whether the particles themselves are responsible for ill effects or whether something carried along with the particles is the cause."

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vacuum cleaner could generate small particles that add to “average” background levels.

Next summer, Dr. Liu will be recruiting 96 people older than 65 who have “moderate” pulmonary or cardiovascular problems. If you were one of her volunteers, what would you be doing? For 14 days you would wear three small plastic badges that will separately measure exposure to carbon monoxide, nitrogen dioxide, and sulfur dioxide (but not ozone, since ozone is not a problem in Seattle proper). You would also wear a monitor for particulate matter. Air monitoring equipment would be placed at your house, both indoors and in either the front or back yard. You would respond to questionnaires that will track daily activities. And you would contribute a daily morning urine sample, to be analyzed using a technique Dr. Kalman has developed for detecting wood smoke in urine. In addition, Dr. Koenig would measure your pulmonary air flow, using a device called an “air watch” (a digitalized peak flow meter) and Dr. Joel Kaufman would measure your pulse rate, to check on cardiovascular functioning. The suspicion that air pollution, a known problem for the respiratory system, may also be associated with cardiopulmonary deaths, “really ups the ante,” as Dr. Koenig put it.

EPA’s new standards for  $PM_{2.5}$  will not be enforced for three years because of the complications involved in measuring fine particle pollution. This does not prevent the states from taking actions in the

interim to help the most vulnerable, while more research is being done. Since wood smoke pollution definitely causes problems for some people in the Puget Sound region during the winter, the Puget Sound Air Pollution Control Agency has been authorized to put burning bans in place at a lower trigger than in the past. Dr. Koenig estimated that 70 percent of the wood smoke particles going up a woodstove chimney are probably ending up in someone’s home. Therefore wood smoke becomes an indoor air pollutant even for those who do not burn wood, and has been associated with aggravation of asthma.

Recognizing that exposure

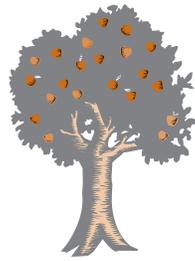
assessment in particle research is complicated, EPA plans to fund up to five research centers across the country to study particulate pollution. The centers will focus on new approaches to individual exposure assessment; new dosimetry models to reduce uncertainty in our knowledge of how the lungs and cells receive specific doses; better approaches to extrapolate data from animals to humans; greater understanding of toxicological effects; and further epidemiological studies, particularly with respect to chronic, long-term exposures. With more research, fine particle air pollution may become much better understood. ■■■



## WHAT IS FINE PARTICLE AIR POLLUTION?

Particles in air less than or equal to 2.5 micrometers in diameter are called fine particles. Particulate matter ranging from 2.5 to 10 micrometers is defined as coarse. Particles less than about 0.3 micrometers are called ultrafine. Coarse particles come from sources such as windblown dust from agricultural fields or dust kicked up on roads by vehicle traffic. Fine particles are generally emitted from vehicle exhaust and residential and industrial combustion as well as vegetative burning.

## DETECTING LEAD



Prior to the introduction of organophosphate pesticides just after World War II, lead arsenate—a compound containing both lead and arsenic—was the primary weapon to control the codling moth in thousands of acres of the orchards of eastern Washington. Because lead and arsenic do not “go away,” but persist in soils, residues from decades of past use can be found in current and former orchards.

When the State Department of Health sampled children’s blood lead levels in selected Washington cities in 1994 and 1995, fewer than 5 percent of the children sampled had blood lead levels that would be considered elevated. (This may reflect great efforts made in the US to phase out lead from many products, including paints and gasoline). However, of the small number of kids at risk, most came from the Yakima area, were of Hispanic descent, and had parents who worked in farming. This raised the possibility that orchard soils are an important source of lead exposure.

Children may absorb lead from soils through hand-to-mouth contact, or by inhalation of dust. Lead exposure may also occur from other sources such as glazes on ceramic plates and dishes, or paint chips. Identification of specific sources of exposure is necessary to enable intervention.

Department of Environmental Health researchers are doing extensive work on the exposure pathways of organophosphate pesticides in eastern Washington, including taking urine samples from children. Since arsenic also turns up in urine as a biomarker of exposure, the department’s Dr. John Kissel proposed an additional research project. Surplus urine from pesticide sampling can be tested for arsenic. And if arsenic does turn up at elevated levels in urine samples, it will provide evidence of exposure from agricultural soils and permit evaluation of lead exposures from the same source.

Dr. Kissel’s work—not yet underway—is to be funded through the new DEH Center for Child Environmental Health Risks Research as well as by the Pacific Northwest Agricultural Safety and Health Center.

## Arsenic is puzzling.

Arsenic doesn’t cause cancer in lab animals, yet it causes cancer in humans. (For some substances, the reverse is true.) Because we can’t give arsenic directly to humans, arsenic’s “mechanism of action” (the way it produces its effects) is very hard to study. We in fact don’t know how arsenic causes human cancers.

Arsenic is an element, and arsenic in very tiny amounts may be an essential nutrient for humans (some lab animals have appeared healthier when given trace amounts). However, as far as we know there is no enzyme in our bodies that requires arsenic in order to function. All other essential trace elements do relate to some enzyme’s functioning. Thus scientists can only say that arsenic is “more probably than not” an essential trace element. We ingest arsenic when we eat some foods, especially seafoods.

All of these interesting facts concerning arsenic emerged from an interview with DEH’s Dr. David Eaton, who is currently serving on an oversight board for a National Academy of Sciences-National Research Council study of arsenic in the US water supply. The study, funded by the Environmental Protection Agency (EPA), is being conducted by a committee of the world’s most knowledgeable arsenic experts.

For many years now—since 1942, in fact—the allowable standard for arsenic in US drinking water has been set at 50 parts per billion (50 micrograms per liter). According to Dr. Eaton, the standard was initially set at this level “for practical, rather than risk-based, reasons,” having to do with detectability and the fact that very few water sources in the US contain arsenic above this level. However, in amending the Safe Drinking Water Act in 1986, Congress required EPA to revisit the issue, and to set a new standard by January of the year 2000.

There has been a good deal of controversy over the current standard. While there are some who believe that the standard overestimates the arsenic risk to public health, the standard does not meet EPA’s present range of “acceptable” additional lifetime cancer risk, which is between 1 and 100 additional cancers per million people exposed for a lifetime. We also have some new data suggesting that routinely ingesting arsenic in drinking water at above 50 parts per billion is not a good idea, and may be implicated in not only skin cancers, but possibly cancers of the lung, bladder, liver, and kidneys.

At very high levels of ingestion, no one questions that arsenic causes skin cancers, and probably also lung cancer. In Taiwan, for example, research was undertaken on 40,000 people who were

drinking water containing arsenic at the level of 600 parts per billion. Over 400 of these people developed skin cancer—one out of every 100 persons. If left untreated, skin cancers (such as squamous-cell carcinomas) may spread to other body organs, causing premature death.

In parts of India and Bangladesh, thousands and thousands of people show the symptoms of chronic arsenic poisoning (“arsenicosis”), including skin discoloration, skin sores large and small, inflamed eyes (conjunctivitis), gangrene, and loss of sensory perception. Children as young as eight and ten show these symptoms because poisoning is cumulative, building up from birth over the years as people drink water that contains naturally occurring arsenic. The world seems hardly to have noticed the health crisis in West Bengal and nearby parts of Bangladesh from drinking water with high arsenic levels.

Dr. Eaton pointed out that the US arsenic standard could eventually be set by EPA at anything from 2.5 parts per billion (not likely for technical reasons) to the current 50, but at every step downwards in parts per billion, the monetary cost of meeting the standard will go up.

Meanwhile, we don’t know whether there is a threshold below which one’s dose of arsenic from water doesn’t really matter. Nor do scientists fully understand the shape of the arsenic dose-response curve. Understanding the shape of the dose-response curve is important, because all of the bad health effects from arsenic that we know about have occurred at relatively “high” doses—well above the current 50 parts per billion doses from the current drinking water standard. Scientists and risk assessors must extrapolate risk information obtained at high doses to try and predict what the risks will be at much lower doses.

Dr. Eaton stressed that there are two critical questions that EPA must ultimately deal with: 1) It must decide on a reasonable estimate of the health risks—especially from cancers—of setting the arsenic standard at a level ranging from 2.5 to 50 parts per billion; 2) It must look at the fraction of community water supplies whose arsenic content exceeds any new level proposed. The economics of “fixing” the nation’s water supply change dramatically if, say, the standard is set low enough to involve 80 percent of all potable water, as opposed to a standard that involves only 10 percent. The reason economics matters is that resources spent on one problem cannot be used on another. ■



## NAPOLEON'S WALLPAPER

Napoleon Bonaparte, France’s military genius, emperor, reformer, and dictatorial personality, was banished by the British to a remote island in the South Atlantic, in 1815. He lived there until he died, six years later, in his early fifties. A postmortem produced the medical opinion that a stomach ulcer possibly turning cancerous was the primary cause of death. Some of the people who accompanied Napoleon into exile kept locks of his hair as remembrance tokens.

In the 1960s, some of Napoleon’s hair samples were scientifically analyzed and found to contain above-normal levels of arsenic. As any reader of detective mysteries knows, poisoning people with arsenic doses has long been a well-known murder method. (In Wakayama, Japan, in September 1998, dozens fell ill after eating arsenic-laced beef curry at a local festival—four died, including two children.) A debate began over the cause of Napoleon’s death.

From here, fast forward to the spring of 1980, when Dr. David Jones, a chemist who is still doing research at the University of Newcastle upon Tyne, UK, was preparing a talk on vapor biochemistry for BBC Radio Scotland. As a minor part of his talk, Dr. Jones asked if there were any historians listening who knew if Napoleon’s rooms on St. Helena were decorated with green paint or green wallpaper.

Why did Dr. Jones ask this question? Because a color pigment called “Scheele’s green” was widely used in Europe, by 1800, in fabrics, paints, and wallpapers. As the century progressed, it became evident that “Scheele’s green” was a health hazard, for the pigment contained copper arsenite. As Dr. Jones tells the story in an article in *New Scientist*, 14 October 1982: “Hundreds of luckless householders developed the various symptoms of arsenic poisoning, and quite a number died.”

It turned out that Dr. Jones’s talk for the BBC produced what he called “the most astonishing piece of scientific good fortune of my life.” He received a letter from a woman who said she had inherited, in a scrapbook, a piece of wallpaper

*continued on page 12*

# CLIMATE CHANGE AND EMERGING PATHOGENS

## *Editor's Choice*

Environmental changes are affecting health problems worldwide. That was the message brought by Colonel Ernest Takafuji, MD, MPH, one of the US Army's top medical research commanders, to 300 attendees at a short course on "Occupational Aspects of Emerging Pathogens" held on October 7 in conjunction with the 1998 Northwest Occupational Health Conference.

Dr. Takafuji suggested to his audience that it is time for new thinking to bring together climate scientists and public health providers. Such phenomena as population growth; continuing world urbanization; water and air pollution; resource depletion; ozone depletion and increases in ultraviolet radiation; and the "recognized reality" of global warming have the potential to greatly increase acute and chronic diseases, suppression of the immune system, malnutrition, and mortality.

Dr. Takafuji presented examples of how environmental change affects health. Climate warming, for example, will have an important impact on diseases transmitted by mosquitoes [and other climate-sensitive vectors of disease like flies and ticks]. The world's most widespread mosquito-borne viral disease is dengue, now present on all continents except Europe. Different strains of the disease carry the risk of a serious complication, dengue hemorrhagic fever, with no vaccine available. Dengue has not yet appeared in the US, but Dr. Takafuji predicted that "dengue is coming," from south of the US border.

Dr. Takafuji listed as some of the other diseases that may be affected by global warming as cholera, malaria, viral encephalitis, yellow fever, filariasis, leishmaniasis, onchocerciasis, fungal infections, hanta virus, diseases resulting from malnutrition and lowered immunity, and heat-related and ultraviolet-associated illnesses.

As a US Army medical researcher (former head of the US Army Medical Research Institute of Infectious Diseases, for example), Dr. Takafuji has had unusual opportunity to see what is happening around the world, since, as he noted, there are US troops in 81 countries. He stated that "the diseases of other countries are becoming ours," and are showing up in strange places, thanks to ever-increasing international air travel and the recirculated air in planes. Many US doctors and hospitals, he feared, will not recognize diseases they may face,

because at the moment many of these diseases are still unusual. However, the motto for viruses, he joked, is "Be a virus, see the world."

Illnesses to which he particularly drew attention included a new strain of influenza Type A; Japanese encephalitis; and viral hemorrhagic fevers such as yellow fever, smallpox, Marburg disease, Ebola disease, Lassa fever, and eight other viral hemorrhagic fevers. These last eight originate in particular spots of the world, some coming from South America and others from Africa, Korea, and Russia. Some of these illnesses are more of a problem for travellers than for local inhabitants, he noted, because inhabitants may have developed antibodies to the disease that a traveller will not have. Such is the case for Japanese encephalitis, for example; travellers visiting large rice-growing areas (with water and mosquitoes) are probably at greatest risk, but there is a vaccine available. Japanese encephalitis can kill; recovery is slow and may involve personality changes.

Sponsors of the 1998 Northwest Occupational Health Conference were the PNW Industrial Hygiene Association; the Columbia River and the Washington State Associations of Occupational Health Nurses; and the Northwest Association and the American College of Occupational and Environmental Medicine.

Dr. Takafuji will be returning to Seattle next autumn to speak in a program sponsored by the UW School of Medicine's Travel and Tropical Medicine Service.

—PC

*"...it will be difficult to educate people worldwide about the climate-change issue because of its complexity. First, it is difficult to convey the anticipated size and scale of the problem, and individuals often are left feeling powerless. The subject is both technical and complex, and the media focus on conflicting scientific opinions leads to much confusion.*

*The cause—fossil-fuel consumption—is not a relatively simple and controllable issue....Health must be at the center of the climate-change debate to counteract the perceived disconnection between individuals and their environments."*

—from the authors of  
"Global Climate Change and Environmental Health"

See reading list, next page

"Climate change is likely to have wide-ranging and mostly adverse impacts on human health, with significant loss of life."

—from the Second Reassessment Report, Intergovernmental Panel on Climate Change (IPCC)



## CONTINUING EDUCATION



Photo: P. Coburn

Central America has dengue.

### For Further Reading

Patz, JA, et al. 1996. Global climate change and emerging infectious diseases. *Journal of the American Medical Association* 275: 217-23.

Kovats, Sari, et al. 1998. Global climate change and environmental health. *International Journal of Occupational and Environmental Health* Volume 4, Number 1, January-March: 41-49.

Henderson, D.A. 1998. Bioterrorism as a public health threat. *Emerging Infectious Diseases* Volume 4, Number 3, July-September: 1-4. Centers for Disease Control and Prevention, National Center for Infectious Diseases. URL: <http://www.cdc.gov/ncidod/EID/vol4no3/hendrsn.htm>

### NW OCCUPATIONAL HEALTH CONFERENCE

7-8 OCTOBER 1998, SEATTLE, WA

#### UW CONTRIBUTORS

- N. Beaudet Physicians and Industrial Hygienists Working Together
- J. Camp Introductions, Short Course on Emerging Pathogens
- B. Daniell Noise, Hearing Loss, and Hearing Conservation in WA
- M. Flanagan Nitrous Oxide Exposures in a Pediatric Dental Clinic
- M. Keifer Pacific NW Agricultural Safety and Health Center
- I. Moate Biomarkers for Insecticides in Children and Non-Occupationally Exposed Populations
- L. Monteith Introduction, Industrial Hygiene
- P. Moore Aluminum Potroom Exposures Assessed through the Use of Continuous Reading Monitors
- R. Neitzel An Assessment of Occupational Noise Exposure For Construction Trades
- C. Pierce Biological Monitoring of Controlled Toluene Exposures
- M. Wessels Quantitation of Nitrous Oxide in Urine Using Headspace Analysis

For course details, call (206) 543-1069, or visit the departmental home page on the World Wide Web at [www.sphcm.washington.edu/DEHWeb/](http://www.sphcm.washington.edu/DEHWeb/).

#### Upcoming courses:

#### NW CENTER FOR OCCUPATIONAL HEALTH & SAFETY

- Jan 13 Occupational Noise Update
- Jan 25 Annual Hazardous Waste Refresher
- Jan 26 Annual Hazardous Waste Refresher
- Jan 27 Annual Hazardous Waste Refresher
- Feb 4 Indoor Air Quality: Children's Exposure at School and Home
- Feb 19 Workers' Compensation: Return to Work
- Mar TBA Managing Hazardous Materials Incidents
- Mar 4 Fishing Safety and Health
- Mar 30-31 Safety and Health in the Forest Products Industry

#### OSHA TRAINING INSTITUTE EDUCATIONAL CENTER

- Jan 11-14 OSHA 510: OSHA Standards for the Construction Industry
- Jan 15 OSHA S: Scaffolding Users Course
- Jan 19-22 OSHA 500: Trainer Course in Occupational Safety and Health Standards for the Construction Industry
- Jan 25-28 OSHA 501: Trainer Course in OSHA Standards for General Industry (Portland)
- Feb 1-4 OSHA 309A: Electric Standards
- Feb 15-17 OSHA 225: Principles of Ergonomics
- Mar 1-4 OSHA 510: OSHA Standards for the Construction Industry (Portland)
- Mar 8-11 OSHA 204A: Machinery and Machine Guarding Standards
- Mar 15-18 OSHA 600: Collateral Duty Course for Other Federal Agencies

# DAVID KALMAN

## DEH'S INTERIM CHAIR

“My leadership style is to facilitate group decision making,” said Dr. David Kalman, new Interim Chair of the Department of Environmental Health. “It’s the faculty who will decide our course during the coming year.” Dr. Kalman previously served as associate chair, working with Dr. Gerald van Belle, who is newly retired as department head.

“My training as a chemist is broadly applicable to most of environmental health,” explained Dr. Kalman. “Not unlike Gerald’s discipline of biostatistics, chemistry contributes to what we do without running the whole show.” He added that his experience as director of the Environmental Health Laboratory prepared him for a collaborative role and helped him appreciate the service mission of the department.

Dr. Kalman received a PhD in chemistry from the UW in 1978, and came directly to Environmental Health when he was recruited to a research faculty position. In an interview with *Environmental Health News*, he commented that he feels a “strong affinity” with the department, and, in his new role, wants to avoid either running a purely caretaker operation (until a new permanent chair is selected) or setting many long-term goals and initiatives, as a permanent chair would likely do. With the help of department faculty, Dr. Kalman hopes to set some focused, achievable, short-term goals that may include finding ways to enhance the running of the department and help prepare the way for the new chair.

Dr. Kalman’s scientific interests lie in the areas of the transport and fate of chemicals in the environment; hazard management; and human exposure assessment, especially using biomarkers as a way to assess, and ultimately prevent, environmental exposures that pose risks to health. Since much of his work is laboratory based, he collaborates with others who are doing field research. Several members of the DEH faculty are collaborators (including Drs. Mike Morgan, Noah Seixas, Richard Fenske, John Kissel, and Jane Koenig). Other collaborators are Dr. Timothy Larson of UW Civil Engineering and epidemiologist Dr. Allan Smith of the University of California at Berkeley.

Dr. Kalman is involved in projects concerning arsenic in drinking water in India (West Bengal), Chile,



Photo: David Kalman

Dr. David Kalman

and Argentina. Arsenic in urine is a good biomarker of exposure to inorganic arsenic, which occurs both naturally and as a pollutant in water and soils. While high arsenic exposure is clearly associated with skin diseases and cancers, low-level exposure (including that which meets current U.S. and international safety standards) may take a very long time to show negative health effects. In parts of India and Bangladesh, tens of thousands of people are drinking water from wells naturally containing extremely high levels of arsenic; their villages typically have no central water system nearby that might serve as an alternate source, a likelihood in more developed countries. Dr. Kalman noted that getting rid of even 10 percent of the worst wells in West Bengal and Bangladesh would be a significant contribution to public health.

Studies either completed or currently underway in Chile and Argentina aim to examine the health effects of high arsenic levels in drinking water. Dr. Kalman’s work in characterizing levels of arsenic exposure will link with studies of frequency of biological changes and disease to provide clues to how arsenic produces illness.

In addition to arsenic, Dr. Kalman has studied pesticides (arsenic for pesticide use, incidentally, has been banned in the US, but remains in the environment for many years); solvents; mercury; dust emissions; and wood smoke exposure. He and other departmental and UW researchers are also trying to figure out just why it is that air containing very fine soot and dust particles appears linked to increases in disease and even death, particularly among sensitive individuals. ■

“My leadership style is to facilitate group decision making. . . it’s the faculty who will decide our course during the coming year.”



**Michael Yost** was an invited speaker at an international workshop on electromagnetic field exposure assessment methods held in Oxford, UK, in September. Sponsors were the World Health Organization and the UK National Radiation Protection Board. In June, Yost served as a member of the NIEHS working group reviewing the possible health effects of electromagnetic fields.

**Gerald van Belle** gave invited papers at the Joint Statistical Meetings, in Dallas, and at the International Society for Clinical Biostatistics, in Dundee, Scotland, in August. He also contributed to the Biomedical Research Integrity series at the UW School of Medicine with a talk on the ethical context and content of statistical design, conduct, and analysis of experiments.

**Janice Camp** has been elected president of the Pacific Northwest Section of the American Industrial Hygiene Association.

**David Kalman** travelled to Calcutta in June of 1998 as part of a new EPA study of arsenic-related disease in West Bengal and Bangladesh. Earlier, he gave a paper at the annual conference of the state Environmental Health Association on community exposure to fine atmospheric particulates.

**Sally Liu** has been awarded a two-year grant by the Health Effects Institute to study aldehyde exposures in the general population. During the summer she presented her work on bacterial exposures and health effects among South Carolina school children at professional meetings and conferences in Boston and San Diego.

**Alexandra Tres** traveled to Nicaragua this summer to share the results of a study that analyzed the knowledge, skills, and abilities required of environmental health practitioners. She was also part of a US delegation that looked at health, environment, and labor practices in that country.

**Rolf Hahne** has written a section on "Chromatographic Techniques in Industrial Hygiene" for the *Encyclopedia of Analytical Chemistry*, to be published by John Wiley & Sons in the year 2000.

**Lucio Costa** has been reappointed a member of the Pesticide Advisory Board of the WA State Department of Agriculture. The board advises the director on problems relating to the use and application of pesticides in the state. Costa presented invited talks at the International Society for Biomedical Research on Alcoholism, held in Copenhagen in June, and at the International Union of Toxicology, held in Paris in July.

**David Eaton** has been elected Vice President of the Toxicology Education Foundation. In September, he co-chaired (with **Lucio Costa**) a session on genetic predisposition and individual sensitivity to pesticides at the International Neurotoxicology Meeting, held in Little Rock, Arkansas. Eaton spoke on genetic polymor-

phisms and Costa spoke on the emerging field of ecogenetics.

**Elaine Faustman** attended a reception at the White House in honor of scientists who will head new Centers for Child Environmental Health Risks Research. She also co-taught, in Sweden, a summer course on risk assessment sponsored by the International Union of Toxicology. Back in the US, Faustman and **Richard Fenske** participated in a workshop on cumulative risk sponsored by the International Life Sciences Institute for the EPA's Office of Pesticide Programs.

**Deirdre Grace** presented a talk in Tempe, AZ, in October, on stakeholders' involvement in research conducted by CRESP, the Consortium for Risk Evaluation with Stakeholder Participation. In other CRESP matters, the second annual "Health of the Hanford Site" conference was held in November. Two CRESP reports have recently been published, one on the Hanford Openness Workshops (dealing with information access and declassification issues) and the other on using risk assessment in a tribal context, the result of a four-day conference involving 75 tribal representatives held early in 1998 in Pendleton, OR.

**Marina Cofer-Wildsmith** has been selected as a new member of the Puget Sound Air Pollution Control Agency advisory committee. She has also accepted a Board position with the Environmental Education Association of Washington.

**Ruth Sechena** will be representing the Center for Ecogenetics and Environmental Health on a new panel formed to review government educational tools dealing with household toxins. Sponsors of the panel are the WA State Department of Ecology, the King County Department of Natural Resources, and the Household and Institution Products Information Council. Sechena also gave two presentations at the Washington Public Health Association Meeting held in Yakima in October.

**Lee Monteith** was the local coordinator and an instructor for a professional development course sponsored by the Gas and Vapor Detection Systems Committee of the American Industrial Hygiene Association. The course was held in conjunction with the October Professional Conference on Industrial Hygiene held in Seattle.

**Doug Johns**, a student in the MS program in Industrial Hygiene, has been awarded a \$1000 scholarship by the Pacific Northwest Section of the American Industrial Hygiene Association.

With sorrow we note that **Paul Hammond**, a retired affiliate faculty member of DEH from 1994-97, was killed (along with his wife) in the crash of Swissair flight 111 off Nova Scotia on September 2. He was one of the leading lead toxicologists in the United States. ■■■■



*continued from page 7*

allegedly ripped and stolen from the room in which Napoleon died. While on St. Helena, Napoleon suffered from symptoms of gaseous arsenic poisoning, including shivering, swelling of the limbs, and gastric upsets. Members of his retinue also felt unwell.

How arsenical wallpaper worked its damage to health was deciphered by an Italian biochemist, E. Gosio, in 1893. Dry wallpaper was harmless. But wallpaper that became damp—from climate or other cause—could become moldy (Napoleon's residence on the island was chronically damp). Molds, not unlike people, need to get rid of most arsenic to survive. Many molds convert arsenic into the vapor called arsenic trimethyl, and discharge it into the air to be breathed as indoor air pollution. (It seems that Napoleon, on St. Helena, gradually spent more and more of his time indoors.)

It would take us too far afield to describe the elaborate detective work required for Dr. Jones to draw his conclusion that arsenical wallpaper was likely a contributing (but not the sole) factor in Napoleon's death (see the article in *New Scientist* for details of the scientific analysis).

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