REGION/ORD WORKSHOP ON EMERGING POLLUTANTS

SUMMARY REPORT

August 11- 14, 2003
Chicago, IL

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FOREWORD

The U.S. EPA ORD/Regional Emerging Pollutants Workshop was the twelfth in a series of Regional Science Topic Workshops sponsored by the Office of Science Policy in the Office of Research and Development at the United States Environmental Protection Agency (EPA). Other workshops in this series have included the following:

Asthma: The Regional Science Issues
Communicating Science: Waves of the Future Info Fair
Fully Integrated Environmental Location Decision Support (FIELDS)
Non-Indigenous Species
Pesticides
Endocrine Disruptors
Emerging Issues Associated with Aquatic Environmental Pathogens
Aquatic Life Criteria
Critical Ecosystems
Air Toxics Exposure Assessment
Cumulative Risk Assessment

The ORD/Regional Science Topic Workshops have two complementary objectives: 1) establish a better cross-agency understanding of the science applicable to specific region-specific human health and/or ecological topics; and 2) develop a network of EPA scientists who will continue to exchange information on these science topics as the Agency moves forward in planning education, research, and risk management programs.

Each year, EPA Regions identify high priority science topics on which to conduct workshops. The workshops address the science issues of greatest interest to the regions on the selected topic areas. Each workshop is planned and conducted by a team of regional, ORD, and interested program office scientists, is led by one or more Regional Science Liaisons or ORD, and is facilitated by a regional chairperson. Participants maintain the cross-Agency science networks they establish at the workshops through planned post-workshop projects and activities such as identifying collaborative research opportunities, creating information sharing mechanisms (e.g., interactive web sites), and developing science fact sheets for regional use.

For additional information on a specific workshop or on the Regional Science Topic Workshop series in general, contact David Klauder in ORD's Office of Science Policy (202-564-6496).

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EXECUTIVE SUMMARY

The U.S. EPA ORD/Regional Emerging Pollutants Workshop was hosted by Region 5 and held August 11-14, 2003, in Chicago, Illinois.

The workshop was organized into multiple sessions covering a number of diverse topics, all of which addressed chemicals/substances considered to be either emerging pollutants, or substances for which additional research may be warranted based upon recent risk evaluation research or for other reasons. Break out and roundtable discussion sessions to explore specific topics – and the overall issue of tracking and researching emerging pollutants – were held at intervals throughout the course of the workshop. Topics discussed in the workshop included the following:

- Case Study – Triclosan: One Example of How Emerging Pollutants Come to Our Attention
- Perfluoro Octane Compounds in the Environment
- Toxic Chemicals Resulting From the Disposal of Electronic Equipment
- Brominated Flame Retardants
- Bisphenol A and Phthalate Esters
- Radium in Oil and Gas Piping and Production Facilities
- Exposures to Thallium and to Platinum Group Metals
- Asbestos and Related Durable Fibers
- Pharmaceuticals and Personal Care Products in the Environment
- Environmental Monitoring for Chemicals in Waters
- Veterinary Pharmaceuticals: Potential Environmental Impact and Treatment Strategies
- Assessing the Environmental Risk of Substances Under the US Food and Drug Act
- Endocrine Disrupting Pollutants in Effluent Dominated Streams
- Biotechnology: The Promise, Potential Risks, and How Can We Find Out
- Environmental Futures: Nano Technology and Genomics Coming Over the Horizon
- Feedback from an Environmental Journalist

Scientists from EPA (Regions; Office of Research and Development; Office of Pollution Prevention and Toxics; Great Lakes National Program Office; Office of Radiation and Indoor Air; Office of Science Policy) and invited speakers from academia, government laboratories, Environment Canada, U.S. Geological Survey, U. S. Food and Drug Administration, U.S. Department of Agriculture, and the media presented research information and discussed additional research needs and strategies for ensuring that emerging pollutants are addressed.

The diverse presentation topics covered in the workshop allowed participants to explore these topics as well as the issue of ensuring that emerging pollutants receive Agency attention and are adequately researched and monitored. The roundtable and break out sessions, which addressed the presentation topics, sought to answer the following questions:

- Are risks to human health and the environment from the substances being discussed quantifiable, and are they likely to increase over time?
- What knowledge gaps need to be filled to clarify present and projected risks? and
- Are there pollution prevention/risk reduction or other risk management options for these pollutants?

According to the workshop evaluations, most participants found the workshop very useful, and many expressed interest in making such dialogs a more regular feature of ORD activity. Planned outcomesfred

include continued communication among participants and others on the emerging pollutants discussed in the workshop.

WORKSHOP SESSION SUMMARIES

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Monday, August 11, 2003

WELCOME AND INTRODUCTORY REMARKS

Bharat Mathur, Deputy Regional Administrator
U.S. EPA Region 5

Mathur welcomed attendees and said the workshop is one of the first held by U.S. EPA's Office of Research and Development (ORD) on emerging pollutants. He added that emerging pollutants is an important topic for ORD and for EPA headquarters (e.g., as a factor in developing strategic plans). The topic is also important to the regions, because they are on the front line to respond to new pollution concerns raised by the public.

Region 5 also is taking an increasingly active role in science issues and dialogue with ORD. Mathur added that Region 5 has held other workshops on topics such as pesticides and TMDLs and has collaborated with other Regions to hold additional workshops. "It is my hope that Region 5 and other regions can influence the Agency's science interests to meet our own needs," he said.

ORD/Regional Workshops - Videotape
Paul Gilman, Assistant Administrator, Office of Research and Development (ORD), U.S. EPA Agency Science Advisor

Gilman began by saying that this is the 13th in a series of workshops on selected topics involving the Regions. ORD has 2,000 employees and a $700 million budget, including $100 million in external grants that it administers. It has 13 labs and research facilities located throughout the regions. At EPA, science is used to make good decisions. High priority research areas are human health and ecological health.

The workshops are driven and planned by the Regions to help promote cross-agency networking and to integrate EPA science into Region decision making. The next workshop will address inhalation risk. Gilman said one objective of the current workshop is to identify emerging pollutants in U.S. EPA regions.

The Agency would like to use scientific knowledge and research for the decision making needed to address regional programmatic areas. Some of the topics that have been identified in the past for research include the following: particulate matter, drinking water, global climate change, endocrine disruptors, and ecological systems. Past workshops have addressed such topics as cumulative risk assessment and asthma.

ORD's goal is to anticipate future or emerging issues, to determine whether there are human health or environmental concerns, and to develop a research agenda to quantify impacts. Most emerging pollutants are as yet unregulated, he said, and the Agency must explore whether data on these substances currently exist and what additional data may be needed to identify such

pollutants and their effects. "The public will need to be educated" about the existence of emerging pollutants and their impacts "to provide an action agenda for the future," he said.

Introduction to the Emerging Pollutant Workshop
David Macarus, U.S. EPA Region 5, Regional Science Liaison to ORD, Chair, Workshop Planning Group

Thousands of chemicals are used in products that provide beneficial uses. But along with the beneficial uses there also are wastes or negative consequences that must be addressed. Additionally, there can be delayed consequences, as in the case of PCBs, which are no longer manufactured, but are still bioaccumulating in fish.

One of the goals of the workshops is to bring EPA science into regional decision making. This particular workshop also has some industry representatives in attendance. Macarus noted that industry has more information about some substances than does EPA, so a dialogue with them is important.

An important question to ask is whether EPA's processes are adequate for dealing with all of the chemicals that come onto the market. These products are beneficial, but can we prevent negative consequences of their manufacture and use? "We must maintain the beneficial uses and keep the impacts on the environment from affecting us," he said. Macarus ended his comments by saying that it is important to identify unanswered questions – and unquestioned answers.

Case Study – Triclosan: One Example of How Emerging Pollutants Come to Our AttentionEmerging Pollutants Come to Our Attention
Steve Reimer, U.S. EPA Region 10

This case study briefly describes the serendipitous discovery of an emerging pollutant, Triclosan, while seeking to identify and quantify other suspected contaminants in the environment.

During 1997 and 1998, the Region 10 laboratory was involved in the Columbia River Intertribal Fish Commission Project (CRITFC). This was the first large-scale survey of fish consumption in the area. For the study, 300 fish samples were analyzed for organic and metal contaminants. The lab was involved in identifying the target list of chemicals. The usual pollutants were found, such as Arochlor (PCBs), DDT, mercury, and some other metals.

When analyzing for Aroclor with electron capture detectors (EDC), extra peaks were detected eluting after Aroclor 1260. These peaks didn't match anything researchers had experience analyzing. They didn't match Aroclor 1268 or a chlorinated terphenyl pattern, and analysis on the gas chromatograph (GC) with an atomic emission detector showed bromine rather than chlorine. These late eluting peaks were detected in bottom-dwelling and older fish (e.g., sturgeon and white fish).

Analysis determined that the peaks represented brominated compounds, with polybrominated diphenyl ethers (PBDE) as the probable compounds. PBDE is used as a flame retardant in

upholstery foam. Because, at the time, there were no commercially available standards, the lab produced its own homemade "Bromkal 70" to make a standard. This allowed the lab to report a value for total PBDE in the fish samples.

By 2001, standards were available commercially through Cambridge Isotopes, and the research team decided to revisit the fish samples using both the homemade and the commercial standard. At this time they also began to look for sources of PBDE in the environment. Potential environmental sources of PBDE were identified based on a literature search. They included storm water runoff, industrial effluent, and wastewater treatment plant effluent.

Samples from five wastewater treatment plants (water and sludge) were collected along with sediment from above and below outfalls from plants treating wastes for industrial (Tacoma), agricultural (Yakima), and residential (Port Orchard) areas. Because bio-solids from treatment plants can be used as a soil conditioner, samples also were collected from several farm fields. Analytical procedures included solvent extraction, florisil treatment, acid treatment, sulfur removal, and GC/ECD analysis.

Using gas chromatograph/mass spectrometer (MS) analysis, researchers found, among other compounds, 5-chloro-2-(2,4-dichlorophenoxy)phenol, or triclosan. This unexpected finding again required innovative thinking and methods development. In order to create a quick standard, they used a toothpaste (Colgate) extract, which contained 0.3% triclosan, to make a standard for the GC/MS. Triclosan was detected in wastewater treatment plant sludge in concentrations as high as 23 ppm. The compound was found in concentrations in the low parts per billion in some sediment samples. Bio-solids that had not yet been spread showed 29 ppm triclosan. Triclosan was detected in soil from one of the hop fields at 150 ppb. The compound was not detected in the control hop field at 0.04 ppb.

In summary, the laboratory was able to find the triclosan in the environment for several reasons. The analysts had time to work on the problem and the necessary equipment, including an AED and an MS to identify the unknown peaks. The lab also was able to develop and find standards for the analysis, though the PBDE standards weren't commercially available in 1997. Also credited for making the finding were "picky" analysts, who wanted to identify the initially unidentified peak, and luck.

PERFLUORO OCTANE COMPOUNDS IN THE ENVIRONMENT

PFOS Toxicity Studies
Christopher Lau, U.S. EPA/ORD/National Health and Environmental Effects Research Laboratory (NHEERL)/Research Triangle Park (RTP)

Perfluorinated organic chemicals such as perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) have been in the news over the past few years. At one point the *New York Times* reported, albeit erroneously, that some of these compounds were posing health risks to young women and girls. PFOS also is discussed on the website for the book *Our Stolen Future*. Some have commented that PFOS will be the PCB of the 21st century. Perfluorooctane sulfonate is an organic compound that does not easily break down. It is hydrophobic (does not

bond to water) and oleophobic, so it is ideal for use as a surfactant in many industrial and consumer applications. Perfluorooctane sulfonate has been mass produced for the past 50 years.

Two major processes are used to make PFOS. One involves electrochemical fluorination in the presence of hydrofluoric acid, favored by 3M, that produces a mixture of straight-chained and branched isomers. The other process is telomerization, favored by DuPont to produce straight-chained telomer alcohol. Hence, PFOS compounds exist in a mixture in the environment.

PFOS is a very useful chemical found in more than 200 products. The best known is probably 3M's Scotchguard™. It also has been used commercially in a variety of other products as: paper coatings, floor polish, alkaline cleaners, denture cleaners, shampoos, fire-fighting foams, aviation hydraulic fluid, mining/oil well surfactant, acid rust suppressant, metal plating, electronic etching bath, and ant/roach insecticides.

PFOS came to EPA's attention because of its presence in the blood serum of workers involved in the manufacture of the product (300 – 8,000 ppb. mean 2,500 ppb). Once they began bio-monitoring for the compound, they found it in non-production employees (29-96 ppb, mean 47 ppb), human serum samples (7 – 82 ppb, mean 28 ppb), blood bank pools (9 – 56 ppb, mean 30 ppb), and children (7 – 515 ppb, mean 44 ppb).

The mean concentration level in children of 44 ppb was somewhat alarming, because low levels of contaminants are typically found in this sub-population. Additionally, researchers have found the compound in the environment, in plasma and in liver samples from many types of wildlife animals – from dolphins, fish, and birds to polar bears in the arctic.

PFOS is readily absorbed orally but remains in the body for a long time; the half-life in humans was estimated at 8.7 years, compared to 200 days for monkeys and 7.5 days in rats. Exposure results in body weight loss, liver hypertrophy/lesions, and reduced serum/liver cholesterol. Such effects are pretty consistent among the species examined.

U.S. EPA is concerned about PFOS for the following reasons: the chemical is widespread in humans and wildlife; it is stable, persistent, and bio-accumulative; it is developmentally toxic in lab animals; levels in children are relatively high; modes and mechanisms of toxicity are not well defined; and fate and transport of the compound is unknown at this time.

3M voluntarily stopped its manufacture of PFOS as of December, 2002, though there are still some companies in Europe and Asia that make it. In addition, alternative fluorochemicals substituting for PFOS are under development and being marketed.

Studies of maternal toxicity, developmental toxicity (including pre- and post-natal evaluation), and mechanisms of pathophysiology were undertaken using Sprague-Dawley rats and CD-1 mice. Researchers found maternal toxicity in pregnant rats with the level of the chemical increasing over the period of gestation. The liver was found to have a PFOS level about four times higher than the blood serum. There was a weight gain deficit for the pregnant rat when given higher doses (3-10 mg/kg) due to reduced food and water intake. Reduced serum thyroid hormone levels also were observed in the rat.

In pregnant mice, PFOS at the end of gestation was four times higher in the liver compared to serum levels, and the liver was enlarged. Liver enlargement was not observed to the same extent in rats. A weight gain deficit also was observed to a lesser extent in the mice. The reductions in maternal weight gains for both rats and mice corresponded to administered doses and body burden.

In regard to developmental toxicity, PFOS exposure during pregnancy in the test animals did not cause the fetuses to die or to be low in weight, suggesting that the mother protected the fetus. *In utero* exposure did not significantly alter implantation or the viability and weight of the fetus at term. At the highest doses, some structural malformations were observed (enlarged right atrium of the heart and cleft palate).

The postnatal evaluations were a different story, however. All of the test animals gave birth live, but the pups born to mothers which had been administered high doses of PFOS began turning gray and dying shortly after birth. Other newborns in the lower dosage groups lived a while longer, but also died within the ensuing days. Thus, postnatal survival was severely compromised by exposure to PFOS, in a dose-dependent fashion, corresponding to body burden.

Pharmacokinetic findings suggested that there was no placental barrier in the mother to keep PFOS out of the fetuses; and that PFOS is found in the milk, as well, so the pups received additional doses. Moreover, at birth, the serum and liver levels of PFOS in the pups are the same, indicating a lack of hepatic accumulation at this stage. Certain developmental effects were observed, such as a small decline in growth (some stunting) in the surviving pups, and a delay in the opening of the eyes of both the mice and the rats.

Hypothyroxinemia was observed in the PFOS-exposed neonates. Liver hypertrophy was seen in the developing mice. Both the mice and the rats showed an identical pattern in regard to the postnatal effects, except that the lethal dose for the mouse was 20 mg/kg, while the lethal dose for the rat was 10 mg/kg.

At this point, little is known about how this chemical works. Researchers are now exploring several mechanisms. Putative mechanisms of PFOS toxicity include the following: interference of cholesterol and lipid synthesis/transport; impedence of cell-cell communication through gap junctions; alterations of mitochondrial bioenergetics; hepatotoxicity: peroxisome proliferation; and thyroid hormone imbalance. Yet, these mechanisms are unlikely to account for the neonatal mortality.

For an on-going study, researchers also examined whether there were critical periods of PFOS toxicity during gestation. For this portion of the study, a four-day treatment, with a high dose of 25 mg/kg, was given. Findings thus far indicate that if the exposure occurs early in gestation, deaths are attenuated; in contrast, if the exposure occurs late in gestation, all of the pups die.

Because development of the lung tissues occurs late in gestation, researchers hypothesize that the PFOS may have adversely affected the maturation of the pulmonary system.

In summary: the mechanism of developmental toxicity for PFOS is largely undefined, but is currently under active investigation, and our preliminary findings suggest that PFOS may

compromise the structures and/or functions of the developing lung and liver, potentially leading to neonatal morbidity and mortality.

Researchers are hoping to include PFOS in the Center for Disease Control's (CDC) NHANES monitoring program. Long-range research plans at NHEERL/ORD would relate body burden to reproductive and developmental toxicity, clarify mechanisms for PFOS developmental toxicity, and use computational toxicology to explore the mechanisms of toxicity for the perfluorinated organic chemicals as a class. The evaluation of toxicity using small fish and amphibian models also is planned

Studies of Human and Animal Toxicity
John Cicmanec, U.S. EPA/ORD/National Risk Management Research Laboratory (NRMRL)/Cincinnati

Ammonium Perfluorooctanate, C-8, is like a hydrocarbon with hydrogen atoms replaced with fluorine. The compound is very stable and repels water and oil and reduces surface tension dramatically. More than 100,000 pounds are produced each year. NASA is the primary user of PFOA for space applications.

The research results presented here came out of a risk assessment performed under a consent order for a site in West Virginia.

The focus of occupational studies conducted on PFOA included liver function, pancreatic function, the endocrine system, and lipid metabolism. There were "possible" endocrine-related findings in workers – increased cholecystokinin levels in two workers and elevated estrogen levels in four male workers, though these levels were correlated with body fat index ratios.

PFOA was detected in workers' blood at levels ranging from 0 –114 ppm, with a mean of 5 ppm. PFOA was present in the groundwater near the plant. Investigators believe that these concentrations were the result of an aerosol dispersion of the compound. Thus, airborne transmission appears to be important.

PFOA has a biopersistence of 94 days and a biodegradation rate of 13% at 28 days.

A quantitative non-cancer risk assessment was performed. In the case of PFOA, non-cancer risk drove the case involving PFOA more than cancer risk did. The non-cancer risk assessment involved analysis of critical studies for identification of critical effects, supporting data, and uncertainty factors, so that the reference dose (RfD/RfC) could be derived and screening values for water, soil, and air calculated. Uncertainty factors were extrapolated.

There was a desire to correlate the occupational exposures to the data gathered through animal studies. The PFOA screening levels developed were as follows: water – 150 micrograms/liter (ppb), soil – 240 milligrams/kilogram (ppm), and air – 1 microgram/cubic meter (ppb).

Despite the fact that non-cancer risk was the main driver in the PFOA case, researchers also looked at animal carcinogenicity studies that had been performed. These studies indicated that

animals exposed to PFOA developed fewer cancers than animals exposed to many other compounds.

An interesting finding of the studies indicated that there are significant differences in PFOA metabolism for humans and rats. For example, there is a marked gender difference in serum and tissue half-lives in rats that is not shared by humans. In rats, PFOA has a much shorter half-life in females than in males. In humans, the half-life is about equal in both males and females. Additionally, peroxisome proliferation is much more significant in rats than humans.

The risk characterization involved the following steps: describing the scientific data base; describing the exposure scenario; listing key assumptions that were made; describing how the process was performed, including calculations; describing other approaches and conflicting data; and considering uncertainty versus variability.

A comparison of experimental monkey blood levels to environmental human exposure levels provided the following data: a 26-week monkey study resulted in blood concentrations of 10 ppm; occupational human exposure levels - 0 to 114 ppm (mean 5 ppm); and environmental human exposure levels – 0 to 40 ppb.

In summary**,** the results of animal studies and environmental monitoring indicate that PFOA is not presently an environmental health threat. Although some endocrine effects have been observed, they are not the critical adverse effect.

Risk Information UpdateRisk Information Update: Overview of Assessment and Activities of PFOS and PFOA
Jennifer Seed, U.S. EPA/Office of Prevention, Pesticides and Toxic Substances (OPPTS)/Office of Pollution Prevention and Toxics (OPPT)

PFOS came to the attention of U.S. EPA in 1999 when 3M used blood samples of workers and animals in toxicology studies and compared the samples to controls from blood banks. Researchers found that "the blanks weren't blank." They found PFOS, suggesting that exposure is widespread. Control animals also had high background serum levels that were traced to fish meal feed. These results launched a series of studies.

PFOS was even found in polar bears. The source was likely the U.S. Navy, which has its sailors wash ships at sea with fire fighting agents. 3M showed EPA data with concentrations of the compound in blood samples, so 3M phased out PFOS in December, 2002. Although there was no need to go through a formal risk assessment, as 3M was discontinuing production, there was a concern that other manufactures would enter the PFOS market once 3M ceased production.

Thus, the Office of Pollution Prevention and Toxics (OPPT) published Significant New Use Reports (SNURs) in 2000 and 2002 to prevent other manufacturers from producing PFOS. An OECD hazard assessment was completed in 2002, and an IRIS assessment is in internal review and will be out in the next six months.

EPA is now looking at PFOA, a C-8 perfluorinated compound that might be used as a substitute for PFOS. PFOA was thought to be chemically inert, however, this is not the case. In September 2002, the director of OPPT announced a priority review process for PFOA. In November 2002, OPPT released a hazard assessment of PFOA. After the data began coming in, EPA found PFOA in humans and animals, though in lower concentrations than PFOS.

By April 2003, OPPT released a preliminary risk assessment of development toxicity for PFOA. After the data for the two-generation study came in, they decided to do a formal risk assessment to determine whether they had to act under the Toxic Substances Control Act (TSCA) against PFOA for developmental toxicity. By Spring 2004, the Science Advisory Board is expected to review a revised risk assessment covering all endpoints.

There are tremendous uncertainties on how to do a quantitative risk assessment on these compounds. The preliminary risk assessment was based on post-weaning mortality and delayed sexual maturation in a rat, using a two-generation reproductive toxicity study. The rat is not a good model for humans, however, because of the gender differences in how rats metabolize PFOA. The female rats get rid of the PFOA to which they are exposed in four hours while the males take eight days to rid themselves of the PFOA.

DuPont and 3M are currently studying the role of exposure to pups via serum and milk. They are also looking into the gender differences rats exhibit in how quickly they eliminate this compound. A two-year bioassay shows liver, pancreatic, leydig cell, and mammary tumors with uncertainty regarding human relevance. The ILSI Risk Science Institute has formed an expert workgroup to summarize the state of tumor science.

Critical reviews in toxicology will take place in November 2003 or January 2004 and an OPPTS policy paper on induced liver tumors will be issued for a joint SAP/SAB review in Dec. 2003. The paper will focus on how to look at different tumor types. U.S. EPA has nominated several perfluorinated compounds to CDC for the next NHANES, and a class study of perfluorinated compounds to the National Toxicology Program (NTP). International producers and communities also need to be aware of the problem.

PFOS and PFOA Program Office Update Program Office Update
Mary F. Dominiak, U.S. EPA/OPPTS/OPPT

Perfluoroalkyl sulfonates (PFAS) are acids, salts, halides, etc. including perfluorooctylsulfonate (PFOS), which was produced in quantity before the end of 2000 and widely used (over 300 chemicals on inventory, including polymers). The chemicals have been produced since 1950 for use in surface treatment, paper protection, and performance chemical (surfactant and insecticide) products.

Perfluorooctanoic acid (PFOA) is used only as an essential processing aid to make fluoropolymers in an enclosed process with controllable releases. PFOA is not expected to be found in final fluoropolymer products except as a trace contaminant but testing is needed to determine if this is true. Fluoropolymers are widely used in electrical, chemical, automotive,

aerospace, military, medical, and construction applications. They are also used to coat cookware and in fabric waterproofing.

U.S. EPA began investigating perfluorinated compounds in 1999 based on new data on perfluorooctyl sulfonate (PFOS). The Agency expanded the investigation in 2000 to include PFOA and fluorinated telomers. EPA published SNURs in 2000 and 2002 to close out the return of PFOS after 3M's phase-out.

The SNURs require prior notice before any new manufacture or import of 88 PFAS, including PFOS, with exceptions only for three high-tech, low-volume, low exposure/release uses for which there are no good substitutes. These uses are semiconductor manufacture, aviation hydraulics, and limited imaging. Industry began voluntary research on PFOA and telomers in 2000. This research produced additional concerns, and EPA released draft hazard assessments on PFOA in September 2002 and November 2002.

In March 2003, industry submitted letters of intent (LOIs) for voluntary research. In April 2003, EPA released a Preliminary Draft Risk Assessment on developmental concerns and solicited parties for enforceable consent agreements (ECAs). ECAs are useful because they allow EPA to move faster than under more traditional processes. The ECA focuses on understanding sources of PFOA in the environment and pathways leading to human and environmental exposure.

The ECA negotiations substitute for the Toxic Substances Control Act (TSCA) subsection 4 test rule. Industry LOIs promise data on market and use, product and article contamination analysis, monitoring/modeling for releases, test chemical, and product selection. The ECAs also seek data on incineration, degradation, article aging, additional chemicals, and additional sampling.

In June 2003 EPA held an initial public meeting with 191 attendees and 50 interested parties. The parties included: FDA, CPSC, industry, environmental groups, local and state government entities, and plaintiffs in a class action suit involving PFOA contamination of local water supplies in Ohio and West Virginia. Technical workgroups were formed to pursue possible ECAs. The next plenary public meeting will be in October 2003. Agreement on one or more ECAs should be reached during this session. The process is expected to continue into the future. Additional ECA negotiations will be pursued on remaining data issues.

The ECA process is open to public participation and comment. The on-line docket may be found at www.epa.gov/edocket/; use "Quick Search" to locate docket OPPT-2003-0012. There is an extensive data repository in file AR-226 at oppt.ncic@epa.gov.

Additionally, EPA has proposed that CDC include PFOS, PFOA, and other perfluorinated chemicals in the next NHANES study. A decision on this is expected soon. An NHANES study will provide a baseline of human exposures to compare with current data and track future trends to assess release/exposure reductions. EPA also is nominating this class of chemicals for study to the National Toxicology Program (NTP).

TOXIC CHEMICALS RESULTING FROM THE DISPOSAL OF ELECTRONIC EQUIPMENT

Examining the Pollution Potential of Discarded Electronic Equipment Disposal
Timothy Townsend, University of Florida

Researchers have been investigating leaching from discarded electronic equipment (e-waste) to determine what is released and under what types of conditions. Electronic devices such as computers, cell phones, DVD players, and other electronic equipment contain metals and metalloids as well as organic and inorganic chemicals in their circuit boards, wire boards, and plastic housings.

Lead is a major potential contaminant. Its most prominent use is in cathode ray tubes (CRTs). Lead-tin solders also are used in printed circuit boards. Other solder elements can include bismuth, copper, silver, indium, and antimony. Semiconductors contain arsenic and selenium, and batteries contain nickel and cadmium. Laptop flat screens are lit with lamps contain mercury.

Other e-waste constituents that have been identified (primarily by environmental groups), include the following: beryllium, palladium, tantalum, zinc, polychlorinated napthalene, gallium, and liquid crystals. Plastics contain brominated flame retardants. The electrical and electronics industries consume 56% of BFRs produced with building construction consuming 31%. In regard to potential exposure pathways, researchers did not look at potential exposure during use. Instead, they considered exposure after disposal, through landfilling (impact on leachate, groundwater, gas), waste-to-energy (impact on air emissions, ash quality), and demanufacturing and recycling. Human exposure can occur during dismantling of electronic equipment to sell parts. In some facilities in Europe where they grind up electronics, some workers have elevated brominated compound concentrations in their blood.

A concern exists that e-waste may leach toxic chemicals into the leachate of lined landfills or contaminate groundwater near unlined landfills There also is concern that chemicals such as mercury may escape through landfill gas. EPA Regions 4 and 5 are studying the leaching of hazardous chemicals from discarded electronic devices.

The current study is focusing on Toxicity Characteristic Leaching Procedure (TCLP) methods development for discarded electronic devices and toxicity characterization of computer CPUs. During other research funded by the Florida Center for Solid and Hazardous Waste Management, the leachability of cathode ray tubes was examined. An assessment is also being conducted into the true impacts of e-waste disposal in Florida.

TCLP is designed to represent what is leaching from a landfill under a worst-case scenario of acid leaching. In summary, a solid waste is leached in an acid solution for 18 hours and the resulting “leachate” is analyzed. If the concentration of certain pollutants in the leachate exceeds a standard in the rules, it is a TC hazardous waste, unless otherwise exempted. However, it is hard to do TCLP on e-waste, because of the way the testing is currently done (requires small particles of a representative sample).

Researchers studied the questions of how best to accomplish size reduction and how to determine a representative sample. In some cases, the researchers resorted to cutting up pieces of the individual components by hand, and in others, they resorted to modified procedures that allowed characterization of entire devices (e.g., immersing the entire device in a 55-gallon drum).

Researchers found that 21 out of 30 CRTs failed TCLP for lead when they used the standard TCLP methodology. However, when they used the modified TCLP methodology, three of three samples failed TCLP for lead. When they used the standard TCLP methodology on cell phones, five of 10 failed for lead, but when they used a modified TCLP methodology, 23 of 33 samples failed.

Researchers used three different methods for analyzing computer CPUs. When they used the standard methodology and hand cut pieces for analysis, all eight samples passed TCLP (they did not exceed the 5 mg/L standard). When they used the standard methodology and shredded the pieces for analysis, all seven samples passed TCLP. When they used the modified TCLP methodology involving immersion of the entire device, however, 9 out of 10 failed (exceeded 5 mg/L).

All of the laptops analyzed using either the standard or the modified methods failed TCLP (exceeded 5 mg/L for lead). Interestingly, the laptops failed for lead, but not for mercury. One problem is that when equipment is shredded, it exposes steel, which causes lead not to leach as much. So policy makers may need to decide if the TCLP test is appropriate for this use.

Future research should be conducted using real landfill leachate. Researchers are constructing simulated landfills with and without co-disposed e-waste devices. They are also looking for brominated flame retardants in the leachate and they are working on developing data to support keeping CRTs out of landfills. Currently, more than 25 landfills have been sampled and compliance monitoring data have been reviewed. Toxics in e-waste can be reduced by using lead-free solders and alternatives to BFRs, and initiatives have been proposed for these. Silver or copper solders may, however, be worse due to silver toxicity.

Emissions from the Incineration of Electronics Industry Waste Combustion Issues
Eric Stewart, ORD/NRMRL/RTP

At least 2.1 million tons of e-waste was generated in 2000. About 1.9 million tons entered U.S. landfills or incinerators. E-waste may contain leachable toxic metals and BFRs. In addition to disposal/recovery in the U.S., some of the remaining waste goes to developing countries for recovery operations.

Open burning releases these chemicals, which may result in exposures. For example, recovery of valuable metals from e-waste in China may involve open burning of plastic encased parts to reduce the wastes to metal. This may release the metals as well as persistent organic compounds such as polybrominated dioxins and furans.

Researchers have looked at controlled combustion to determine the emissions when e-waste was burned. They expected metals and the brominated flame retardants, as well as the potential for brominated furans and dioxins and, because chlorine also is present, bromo-chlorinated dioxins and furans.

The research involved tests conducted over a kiln temperature range of 570 – 830 degrees Centigrade. The lower temperature combustion was intended to simulate a scenario in which more of the metal was kept in the ash so it could be recovered. The higher temperatures were intended to simulate incineration in which the resulting ash is landfilled. As part of the study, the researchers varied the kiln preheating time.

Heat generated from the combustion of the waste accounted for 17% of kiln firing rate. The e-waste used in the tests was equal parts (by weight) shredded keyboards, motherboards, and cases. Flue gas measurements included the following: continuous emissions of O2, CO2, CO, NO; volatile organic compounds (VOCs) (measured with an on-line gas chromatograph); and sampling trains for modified method 5 (semi-volatiles), method 26 (halogens), method 23 (PCDD/Fs, PBDD/Fs), and method 29 (metals).

The NO concentration averaged 55 ppm. VOC emissions were independent of kiln temperature over the range tested and consisted primarily of bromobenzene and other brominated organics. No PCDD's were detected using LRMS. Emissions of PCDFs were near the detection limits using LRMS. PBDFs were detected, but due to a lack of standards, results for brominated and mixed bromochloro-DF are semi-quantitative at best. MCDF was detected during low-temperature runs at 4 ng/dscm. OCDF was detected during one of the high-temperature runs at 2 ng/dscm.

Some brominated furans appeared to be present in higher concentrations than those of MCDF. MBDF appears to be present at 5-10 times the concentration of MCDF. In general, PBDF was greater than PB-MCDF, which was greater than PB-DCDF. Metal concentrations were significant, consisting primarily of copper, lead, antimony, cadmium and manganese. TCLP results for bottom ash showed no chemicals above standards. Controlled incineration may be an alternative method of e-waste disposal.

In the future work will be done with Baptist University in Hong Kong to determine the size, type, and composition of e-waste. They are also scoping tests in an open burn facility to quantify emissions of metals, VOCs, SVOCs, and PXDDs/Fs.

Toxic Chemicals Resulting From the Disposal of Electronic Equipment
Jason Swift, U.S. EPA Region 5 RCRA Program

There is a growing waste stream of electronic equipment. Nearly 250 million computers will become obsolete in the next five years. Mobile phones will be discarded at a rate of 130 million per year by 2005, resulting in 65,000 tons of waste. Many chemicals are used in electronics, including mercury for the back lighting in flat screen displays in laptop computers, and lead in the cathode ray tubes and solder.

It is estimated that twenty-two percent of the world's consumption of mercury goes into electrical and electronic equipment. Approximately four pounds of lead can be found in each computer. Beryllium is used in computer, telecommunications, and automotive electronics industries.

Many unused computers end up in stockpiles in people's homes and offices. This will add to the e-waste streams when they are finally disposed. In California an estimated 18% of households stockpile TVs and 19.4% stockpile computer monitors. The National Safety Council (NSC) estimates that three-quarters of all computers ever sold in the U.S. remain stockpiled. In 2001, 11% of PCs retired in the U.S. were recycled.

According to the Basel Action Network and Silicon Valley Toxics Coalition, about 80% of the e-waste that is supposed to be recycled is sent overseas to China and other such places. Electronic equipment recycling processes include grinding, shredding, manual disassembly, metal reclamation, and glass cutting.

According to the Basel Action Network and Silicon Valley Toxics Coalition, approximately 100,000 migrant workers labor in Guiyu, China, breaking down imported computers. Cathode ray tubes are smashed to remove copper. The glass is dumped in irrigation canals and along rivers where it could possibly leach lead.

It is estimated that more than 3.2 million tons of electronic waste is put in landfills each year. Around 70% of heavy metals (mercury, cadmium) in landfills is thought to come from e-waste. Plastics, such as those used in electronics casings, have a high BTU value and are incinerated in cement kilns or other combustion devices for energy recovery.

Chemicals and metals can escape into the environment during disassembly. Are risks quantifiable? Inhalation of beryllium dust can cause chronic lung disease at concentrations as low as 0.01µg/m3. Beryllium exposure and dose response data are needed. Waste samples collected a few feet from the Lianjiang River near Guiyu, China, showed high levels of many metals, including cadmium, chromium, copper, lead, mercury, nickel, silver, and zinc.

Research is needed to determine where e-wastes go, and what happens to hazardous substances released from it during recycling, incineration, and landfilling. Monitoring is needed during processing. There also are insufficient data on liquid crystal toxicity, substance interaction during incineration, and effects of low dose exposure over time.

The environmental impacts of alternatives to using toxics in equipment also must be explored. There are some lawsuits that allege that chemical exposures in the semiconductor industry have caused cancer, birth defects, and miscarriages. The Common Sense Initiative proposed comparing California health records to employment records in an effort to find any increased incidence of chronic illness. The project never went further than discussions.

To manage risk, the options include disclosure of hazards, material selection, proper handling, and controlled pre-treatment.

Comments/Questions and Answers

Q – Do you have a personal preference for incineration or landfilling for disposal?

A – Unable to give an opinion in regard to incineration, the incineration emissions data have only recently become available. However, testing of leachate from lined, subtitle D landfills is not showing much leaching of lead. But you may not want to use a landfill for other reasons.

Q – Are you seeing fugitive emissions of mercury?

A – Mercury may not show in TCLP because of its interaction with other chemicals in the sample.

Q – Do you expect to see a relationship between the metals that leach with the pure metals within the waste?

A – The metals do interact, which can influence analytical results, but there is a relationship. Other metals can affect how much copper or lead can leach, for example.

Q – Is there mercury in landfill gas? What about other metals?

A – We have found mercury and also molybdenum.

A comment was made that the ORD lab in Cincinnati is working on the recycling of CRT glass to keep the lead out of landfills and incinerators. This project will expand next year.

Q – Can you relate furans and dioxins in the combustion of e-waste?

A – We can relate it for mono-BDF, but not for the others.

Q – With a mixed waste stream, would you expect dioxins and furans?

A – Yes. We don't know why we didn't see more, possibly because we had more bromine than chlorine in this case.

Q – How typical is it to have waste with five parts bromine to one part chlorine?

A – Atypical. There is more furniture and other such waste in municipal waste so the bromine is in much lower quantities relative to chlorine.

Q – Did you think that the metals reacted with the bromines to strip the bromines out of the waste itself leaving a metallic form of halide?

A – Responder did not understand the question.

Tuesday August 12, 2003

BROMINATED FLAME RETARDANTS

Welcome & Introduction
Ted Smith, U.S. EPA/Great Lakes National Program Office (GLNPO)

Brominated Flame Retardants (BFRs) have been in the news a great deal because of the penta, octa, and deca forms. The European Union is banning the penta and octa forms and California has passed a bill that is awaiting the governor's signature banning penta, octa, and deca. Region 9 has spearheaded efforts that have led to the formation of an EPA a*d hoc* work group to address the BFR issue. EPA has held two multi-stakeholder meetings in San Francisco: in 2002 on BFRs/electronics and in 2003 on BFRs in residential furniture.

Overview of Brominated Flame Retardants
Leif Magnuson, U.S. EPA, Region 9 Pollution Prevention Coordinator

Magnuson began by saying that this workshop is a great opportunity to renew a commitment to researching emerging pollutants. This is a very tough challenge because of the overwhelming number of pollutants. U.S. EPA has limited staff and resources available to address new issues. "We must raise the profile of these chemicals in the agency. They are as important as dioxin and mercury. How do we spend our research dollars and recruit involvement from participants? We need your input to determine where to spend the next available dollars," he said.

According to Magnuson, Region 9 has gotten involved in the BFR issue in California because the Region has received many comments about BFRs directly from the public. Thus, they have been pulled into the issue by their constituents. Because California has stringent flammability standards for residential furniture, the Region gets some of the more worrisome BFRs.

In the past, more products were made of wood and metal. Today, numerous products are made with plastic or other petroleum-derived materials that are quite flammable. As a result, the government – especially California – has set high standards for flame retardancy, particularly for furniture. Flame retardant additives are used to treat plastics, foams, and other materials to keep them from burning so fast. For example, if a room is filled with plastics, "flash over" can occur in which everything catches on fire at once. There is no doubt that BFRs save lives by slowing down "flash over" and giving people more time to get out of buildings during a fire.

There are dozens of chemical BFRs with different properties. These include brominated bisphenols, diphenylethers, cyclododecane, and others. When plastics catch fire, the long-chain polymers in them heat and break up very quickly, releasing flammable gases that combine with oxygen to spread the fire. When BFRs are introduced into plastics, they act by suppressing the release of free radicals, which slows the fire. The BFRs are released very quickly in a narrow temperature range as they heat up, which makes them effective flame retardants, unlike chlorine.

BFRs of recent concern include polybrominated diphenylethers (PBDE), hexabromocyclododecane (HBCD), and tetrabromobisphenol A (TBBPA).

Products in which BFRs are used are found everywhere. BFRs are used in polyurethane foam, textiles, plastics, rubber, resins, etc. Home products include couches, beds, chairs, TVs, stereos, microwave ovens, carpets, curtains, etc. Office products include computer casings and circuit boards, printers, fax machines, lighting, furniture, etc. The Americas account for 95% of the world's use of PentaBDE at 15.7 million pounds per year. PentaBDE is highly bioaccumulative. Around 86-99% of the total PBDE congeners found in human tissues are present in this product.

The basis of concern regarding BFRs arises from the increasing concentrations of BFRs (mainly PBDEs) in the environment, humans, and wildlife. The European Union has banned/is phasing out certain PBDEs. Toxicity concerns include the following: endocrine disruption (PBDE, TBBPA, HBSD), dioxin formation (PBDE, TBBPA, HBCD), altered behavior and learning (PBDE). More testing is needed to determine suspected sensitive endpoints.

Currently, it is not known how PBDEs and other BFRs are released into the environment, but they can be found in samples taken virtually anywhere (indoor and outdoor air, surface waters, sewage sludge, foods, and biota). PBDEs are related to a number of important topics warranting special attention, including children's health, endocrine disruption, persistent organic pollutants (POPs), emerging environmental challenges, and high production volume chemicals.

Regulations for BFRs are appearing along with reporting and monitoring requirements. There is interest in developing a substitute for BFRs. Europe is ahead of the U.S. on the issue of BFRs, and will ban penta and octa across the EU by August 15, 2004. They have gathered a lot of information on production and are working on plans for mitigation. The Europeans are currently conducting a risk assessment for deca and draft risk assessments for TBBPA and HBCD have been released for comment. U.S. EPA has included three BFRs in the Toxic Release Inventory, and under TSCA, the Agency can require testing of certain chemicals. Three BFRs (Deca-, Penta-, and Octa-BDE) have been submitted for voluntary testing under the Voluntary Children's Chemical Evaluation Program (VCCEP).

Another action U.S. EPA has taken is the issuance of a requirement that BFRs be evaluated under TSCA's "Significant New Use Rule" (SNUR). The SNUR was driven by the California and CPSC proposed residential furniture standards. It will likely cover only flame retardants for fabrics, but those used in foam are possible candidates as well. The Agency also could require more toxicity data and risk assessments and could restrict use.

Several European eco-labels prohibit BFRs in electronics and some American groups promote the purchase of BFR-free electronics. Some electronics manufacturers are working on alternative chemicals. In the 1990s, Japanese companies took BFRs out of many products.

Furniture manufacturers, including Ikea, Eddie Bauer, and Crate and Barrel have requested alternatives to PentaBDE, and Hickory Springs has converted a plant to produce a non-halogenated phosphorous-based compound. There have been some problems with this new process, however, as some of the treated foam comes out scorched and discolored. BFRs keep the foam white. Some buyers won't accept the discoloration. Public and manufacturer

education is needed on this topic so producers and consumers will understand that there is a health issue involved.

Environmental Presence and Known Sources

Polybrominated Diphenyl Ethers in People
Ron Hites, Indiana University

At a Stockholm, Sweden, conference in 1998 a presentation was given on the concentration of BFRs in human milk, showing a doubling in concentration, of PBDE 47 over a five-year period at the same time that DDT concentrations were going down. Hites thought the compound had a strong resemblance to PCBs. The Stockholm presentation was so shocking it is likely responsible for the focus, within the emerging pollution workshop, on BFRs.

After seeing the Stockholm presentation, Hites decided to look at PBDE concentrations in human maternal and umbilical cord blood (to represent the baby's blood). In this study, which took place at Indianapolis hospitals in 2001, paired mother-cord blood samples were taken randomly from 20 pairs with the goals of finding the range of PBDE levels and the extent of placental transfer and correlating levels with environmental factors and health indices.

The concentrations of total PBDE were about the same in both mothers and umbilical cord blood, indicating that the compound moves through the placenta. Concentrations were detected in all samples, although a couple showed very high concentrations (350 - 450 ng/g lipid). During analysis, researchers had some problems with the lab blanks, because foam – and BFRs – are everywhere.

Brominated biphenyls (BB-154) were found consistently in all the samples, though researchers didn't know why. The mothers in public hospitals were under age 31, and smoking did not appear to be responsible for incidence of this compound. All of the mothers and babies had concentrations of this compound in their blood, with an average of around one ng/g lipid, though some samples had concentrations ranging from 5 to 16 ng/g lipid. There was a problem with brominated biphenyls in Michigan cows' milk some years ago, but this problem was supposedly resolved.

After conducting the Indianapolis analysis, researchers began to pull together other available data on PBDEs. A full literature search on PBDEs in people and the environment was conducted. A tabulation of all concentration data by congener was made, omitting papers that did not report congener-specific data. One difficulty in this research was that not all papers reported or measured the same set of congeners.

Papers on human samples (blood, milk, tissue) were sorted out for additional review, noting the years the samples were taken. Ambient samples included the following: 41 data sets from Europe (mostly from Sweden); 14 data sets from Japan; and nine data sets from North America, (including 2 or 3 from Canada). Occupational samples (of people who worked as dismantlers of electronic equipment) included 12 data sets from Sweden and Norway. The data were organized by time, totals, averages, and congener distribution, and were calculated by percentage.

The data were plotted from 1970 to 2000. The data show a doubling time in detected concentrations of 4.4 years since 1970 all over the world, though Japanese levels are relatively low and North American levels are much higher than elsewhere in the world. Researchers also looked at individual PBDE congener concentration distributions in human samples. These congeners included PBDE 47, 99, 100, 153, 153, 154, 183, and 209.

Congener 47 appears in the highest concentrations throughout the world. Mean concentrations for BDE-47 in North America were 28 ng/g lipid – which was significantly higher than occupational exposures in the EU, which had a mean of 4 ng/g lipid compared to a mean of 2 ng/g lipid for non-occupational EU samples. The samples from North America also had higher mean concentrations of congeners 99, 100, 153, and 154 than found in Japan or in EU non-occupational samples.

Researchers don't know why concentrations of BDE-47 are higher than the other congeners – whether this is due to having a higher vapor pressure, which allows it to be transported more readily, or if it metabolizes differently than the other congeners.

In another plot of recently collected data (2002 and 2003), concentrations of total PBDE for North American neonates and mothers were 35 ng/g lipid in blood (and in milk taken from mothers in Texas) compared to 2 ng/g lipid for Sweden. The wide difference between the concentrations in the two areas is most likely due to the fact that Americans use 95% (or 7,100 metric tons) of the world's penta product .

Some conclusions and recommendations from this work include the following:

- PBDE levels are 10 to 20 times higher in people from North America than in Europeans (or at least in Swedes). Levels in Japan seem to be about half of those in Europe.
- This finding is consistent with the market for the Penta-BDE product, 95% of which is used in North America.
- Levels in people worldwide seem to be increasing exponentially with a doubling time of four to five years.
- The congener pattern is dominated, especially in North America, by BDE-47; occupational samples also show BDE-183 and 209.
- In the future all congeners should be measured all of the time.
- PBDEs cross the placental barrier, but are there any effects?
- The range of concentrations is wide – what accounts for high outliers?
- Begin a regular program of annual monitoring of PBDE (at least) in people, in selected food, and in Great Lakes fishes.
- Don't forget about the polybrominated biphenyls (PBBs).

Brominated Flame Retardants in the Environment
Mehran Alaee, Environment Canada

The latest issue of *Environment International* contains an article on BFRs. PBBs were introduced in the 1970s as flame retardants. PBBs were found in 1981 in samples collected along the Viskan River in Sweden. Others have found PBBs throughout the world. In 1987

Jansson and his colleagues first indicated that PBDEs are "global contaminants" by demonstrating their presence in samples collected from the Baltic Sea, North Sea, and Arctic Ocean.

U.S. researchers were aware of the presence of these compounds, as well. In 1983, Stafford reported the presence of PBDEs in eggs and tissues of fish-eating birds from the Great Lakes Region, and in 1991, researchers from EPA reported 180-220 ng/g of PBDEs in blubber from bottlenose dolphin on the south Atlantic US coast. In 1995, EPA researchers reported up to 8000 ng/g PBDEs in bottlenose dolphin from the Gulf of Mexico.

In 1997 research on PBDEs was started at the Canada Centre for Inland Waters (CCIW) and the Institute of Ocean Studies (IOS), located in Sidney B.C., but people were still skeptical until the Swedish data were released in 1998. In 1999 research was expanded to include PBBs, HBCDD, and TBBPA.

In the U.S., EPA Method 1614 is used for the determination of PBDEs. Isomer specific/isotope dilution method is used for the determination of HBCDD and the isotope dilution method for TBBPA. Researchers have made a lot of progress in identifying PBDEs; however, no method is perfect. Each BFR has its own analytical requirements. PBDEs are amenable to GC analysis, though there are 209 possible congeners (less than 45 are commercially available), and there can be co-elution among various congeners.

GC/ECD is the simplest and least selective detector used in determination of PBDEs. It requires careful sample preparation and there is the potential for interference from PCBs and other organohalogen contaminants. GC/ECN/MS (which is the ISO method) monitors bromine anion formed by dissociative capture. This method is more selective than ECD, but there is susceptibility to interference from organobromine compounds. GC/HRMs is the more selective technique (US EPA method 1614) but it is susceptible to interference from PCBs.

A new EPA method 1614, which is due to be released shortly, will use a high resolution GC. It will be based on eight BDE congeners (28, 47, 99, 100, 153, 154, 183, and 209) and is intended to reduce the variability among the results achieved by different laboratories. Alaee encouraged everyone who is involved with PBDEs analysis to conduct isomer-specific analysis so they will know what they are analyzing.

Concentrations of BDE-209 in sediment cores from the St. Louis Estuary showed that PBDEs are increasing and that PCBs were high in the 1980s but are now decreasing. TBBPA was found in sewage sludge samples from various cities in Canada. Suspended solids samples from the Detroit River showed more concentration on the western or industrial side of the river. Temporal trends in marine mammals and Great Lakes biota show increasing levels from north to south.

In testing of birds with a heavy fish diet (osprey, herring gull, bald eagle), PBDEs were found in egg specimens. Peregrine falcons, which were also tested, showed much lower levels of PBDEs, apparently because they have a primarily terrestrial diet. PBDEs have been shown to be increasing in Great Lakes walleye between 1980 and 2000. In marine seas, the levels in beluga whales from Baffin Island are increasing exponentially.

Over time, PBDE levels in Sweden and Japan rose until the mid-1980s and have since dropped. The increase and decrease correspond to bans of some of these substances. Analysis of various species occupying increasing positions up the food web indicates that PBDEs bio-magnify, and that PBDEs follow a similar increasing trend within the lake's food web as PCBs. HBCDD also biomagnifies within the food web. Unlike the other congeners, however, 60 days after exposure through spiked food, BDE 209 was no longer detectable in fish. More analyses indicate that the PBDEs are being metabolized.

In summary, PBDEs are ubiquitous, global environmental pollutants. Levels of PBDEs are rising in North America. TBBPA has been observed in suspended sediments from the Detroit River and in sewage sludge across Canada. HBCDD has been observed in fish from Lake Winnipeg and in the Lake Ontario food web. There will be more information on BFRs presented at a conference June 6-9, 2004.

Comments/Questions and Answers

Comment – One attendee took issue with the Lake Ontario food web information because data on lake trout are mixed.

Q – Were the mothers with high PBBs the same ones who had high PBDEs?

A – There is no correlation. The data on the Texas women were very limited. About one-third were Hispanic, but there was no difference in outcomes. The number of births and age was considered, but there was no relationship to compound levels. The sample size was small.

Q – Did you have information on their diets?

A – No.

Q – Were the Texas women from the Texas/Mexico border?

A – No – they were from Dallas and Austin.

Q – Is laboratory contamination an issue with the state and regional laboratories?

A – Yes, it's an issue. In one commercial lab, they went into a new area of the lab to perform the analysis and the concentrations in the blanks went out of sight. It turns out the contractor had put foam in the air ducts to cut down on noise. You need to perform some ambient air sampling to determine what type of problem you might have.

Q – In the work on the BDE-209 core samples, how certain is the data? It looks like 209 was there before it was being manufactured.

A – It may have been produced on an experimental basis at one time. There are strong indications of BFR manufacturing in the area since World War II.

Brominated Flame Retardants: Toxicology and Risk
Linda Birnbaum, U.S. EPA/ORD/National Health and Environmental Effects Research Lab (NHEERL)

Birnbaum felt that BFRs were being ignored, but now interest is growing. She believes, however, that it is important to look at all of the congeners.

Flame retardants save lives, but BFRs are a global, transboundary problem. These compounds are persistent and they have a potential for bioaccumulation. There are more than 75 different BFRs, which comprise 40% of the total usage of bromine. Worldwide, approximately 200,000 metric tons are used each year, with half of the use in the U.S. At this time, there is a limited data base on BFRs when compared with other compounds, such as PCBs and dioxins.

Br-Bisphenols, especially TBBPA, are detected in air, sediment, sludge, and biota. TBBPAhas a relatively short half-life in the environment and a low chronic toxicity. It is not teratogenic or mutagenic and, based on limited data in biota, little is retained in tissues. Limited data on TBBPA indicate that it may be immunotoxic, and it appears to be hepatotoxic. The data also suggest that it could be neurotoxic (inhibits dopamine uptake and generates free radicals). In regard to endocrine disruption, AhR effects do not appear to be relevant for commercial products though it can affect thyroid hormone. Because it also inhibits sulfotransferase (decreases estrogen clearance), it could theoretically lead to higher estrogen levels.

U.S. EPA has regulated TBBPA under TSCA. There is concern for air (long range transport), clean water (sediment), and waste (sludge). There has been a Region 9 workshop, Region 5 grants, GLNPO and cross-agency workgroup (Region 9), and an agency "what we know" statement (though it may now be out of date).

Hexabromocylododecane (HBCD) exhibits ecotoxicity to algae, daphnia, and fish. In regard to general toxicity, it is highly absorbed and is a mild irritant and skin sensitizer, with liver effects after repeated doses. More information is needed on repeated dose studies and reproductive toxicity. It may be developmentally neurotoxic.

Because it has been detected in the blood of workers, there are concerns regarding occupational settings. HBCD has been found in human breast milk and is persistent, bio-accumulative, toxic, and capable of long range transport. The Europeans have substituted HBCD for the penta group of PBEs – which may be an example of jumping from the frying pan into the fire.

Polybrominated Diphenyl Ethers (PBDEs) include DBDE (the deca mixture), OBDE (the octa mixture), and PeBDE (the penta mixture). The BDE-47 in the penta mixture appears to be bioaccumulative. The penta is more toxic to the environment than the octa, which, in turn, is more toxic to the environment than the deca. PeBDE (penta) is highly toxic to invertebrates.

Deca and octa may be of low risk to surface water organisms and top predators, but there is concern for wastewater, sediment, and soil organisms. There also are concerns about lower brominated congeners in octa for the potential for debromination and the generation of

PBDDs/PBDFs (dioxins and furans). Adult rodent studies show PBDEs to be hepatotoxic and to lower thyroid T4 levels. At high doses, deca is hepatocarcinogenic.

Some people thought that deca couldn't be absorbed, but new data shows that it can. Deca also can be metabolized into reactive intermediates and can undergo debromination. Deca can be found in human blood and milk. It may be developmentally neurotoxic and it may be breaking down in fish. Europe, which is banning penta and octa, is looking at deca, as well.

New information on penta (SOT, 2003) indicates that it can affect the endocrine and reproductive systems and have developmental neurotoxic effects. In regard to endocrine disrupting effects, there is a question as to whether AhR effects are relevant to BFRs. Thyroid homeostatis has been reported in rats and mice in many labs. There are still questions regarding estrogen homeostatis, because OH-PBDEs may be anti-estrogenic while sulfotransferase inhibition could be estrogenic.

In regard to developmental reproductive effects, you won't see them if you look only at adult animals. Pubertal exposures of DE 71 have been shown to delay puberty and to have effects on male organs. *In utero* exposure of BDE-99 has been shown to delay puberty, cause ovarian toxicity and produce male organ effects and decreased sperm levels.

In regard to developmental neurotoxicity, rats exposed perinatally to DE-71 showed deficits in sensory and cognitive function. Other studies on mice in Sweden and Italy showed that when infantile exposure to BDE-99 occurred (during a critical window of rapid brain growth), there were permanent effects on learning. Perinatal exposures appear to delay sensory-motor development. Recently, studies at other labs also are showing developmental toxicity. A Swedish study of mice exposed to BDE-99 and PCB-52, appears to suggest that effects may be more than additive, though more testing needs to be done on this.

In regard to pharmacokinetics of PBDEs, DBDE is poorly absorbed. Lipid binding is important, with accumulations in fat showing more 47 than 99, and relatively little, if any, 209, because it is a bulky molecule. Covalent binding of 99 and 209 is seen in the liver. Metabolism involves hydroxylation, debromination, and O-methylation. Feces are the major route of excretion.

Different organizations are involved in other ongoing PBDE research. Dose/response research is being conducted by NHEERL, NIEHS, USDA, and Sweden. Cell signaling *in vitro*, involving altered calcium associated with changes in learning and memory, is being conducted by NHEERL.

Developmental toxicity of PBDEs is seen in both mice and rats. Mice are very sensitive in the infantile period. Sensory and cognitive effects also are seen. The mechanism of these effects is unknown. PBDEs alter cell signaling *in vitro* by altering calcium-dependent release of arachidonic acid, which is associated with learning and memory.

Potential health risk of PBDEs can be summarized as follows:

- In a comparison of animal and human data, daily dose isn't important, while the body burden is important (body burden allows cross-species comparison for PBTs).

- The most sensitive effects to date include developmental neurotoxicity and effects on thyroid hormones (0.8 mg/kg). Preliminary reports indicate that developmental reproductive toxicity is seen at an even lower dose (0.06 mg/kg).
- The top five percent of human exposures in the U.S. result in concentrations of more than 400 ng/g lipid. If humans are 25% lipid, then their "dose" is approximately 0.1 mg/kg. A significant dose causing developmental neurotoxicity in mice is 0.8 mg/kg, thus, mouse tissue concentrations are only about 10 times higher than total PBDE concentrations in human tissues. We are not seeing the same distribution in terms of exposures as with PCBs. We don't know why we have highly exposed people, because we don't know the pathways by which they are being exposed.
- Margin of exposure for PBDEs appears low.
- Additional concern: are PBDEs interacting with other PBTs?

Comments/Questions and Answers

Q – Can the presence of PCBs indicate the presence of PBDEs?

A – There is no correlation between PCBs and PBDEs. They are from different sources.

Status of the VCCEP Consultation re: penta, octa and decabromo diphenyl ethers
Jennifer Seed, U.S. EPA/OPPTS/OPPT

The Voluntary Children's Chemical Evaluation Program (VCCEP), a pilot program to test 20 chemicals, was set up over the course of 18 months of meetings with various stakeholder groups representing children, industry, the environment, government, and others. Through these meetings, these stakeholders developed a program with which they felt they could live. In this program, a special emphasis will be placed on gathering exposure data.

Within the program, exposure and toxicity data is to be gathered in three stages. Tier 1 will involve bringing forth all of the information available to date on the 20 chemicals. Much of the data will be offered up by the industry participants. Often, much information is available on toxicity, with exposure data lagging behind. Tier 2 will involve obtaining additional information on toxicity and exposure. Tier 3 will involve elaborate studies. At this point, the industry stakeholders have only committed to provide data for Tier 1. Other stakeholders want independent evaluation of these data.

The entire assessment is focused on children. The underlying question: Are there adequate data about these chemicals to determine risk to children?

The VCCEP has a cooperative agreement with Toxicology Excellence for Risk Assessment (TERA) to perform peer evaluation of the data. Thus far, information on four chemicals, including penta, octa, and deca BDEs have been submitted. TERA will write up summaries of the information and post them on its website. These won't be EPA recommendations – EPA has agreed to wait until the peer consultation.

Issues that have thus far arisen from all three assessments of exposure studies suggest that for children, the major routes of exposure are breast milk and fish consumption. Most of the exposure appears to come from tetra BDE, although this compound is not part of the VCCEP's pilot activities. One of the important questions we must answer is, how can we relate what we have in commercial mixtures to exposures in children?

Comments/Questions and Answers

Q – We need to move upstream and look at the parents' exposures also.

A – Under EPA's definition of children, we also look at exposure of parents and prospective parents.

Q – What is the web site?

A – www.tera.org Click on peer review for peer consultations information.

Roundtable Discussion on PBDE Research Needs

Comments/Questions and Answers

Comment – There is no central repository for BFR research. There are 14 research projects with $12.5 million being spent, but these studies are not coordinated.

Comment – We need to get out in front of this issue so that the data gaps will be filled in the next few years.

Comment – Leif Magnuson asked workshop participants to complete an exercise asking how they would distribute $100 of new research money into various categories. The BFR breakout session group did this and it is included under the breakout session reports.

Comment – We haven't yet formalized a research strategy. We need to get a sense from the workshop participants of what type of research strategy we should have and then determine who should do the research.

Q – What research is being done?

A – We don't have it broken out, but a smattering of research is being done.

Comment – What would help is not just a laundry list of research needs, but to decide what are we trying to accomplish.

Comment – Wants more input into a research strategy. People who have been working on this topic have ideas, but they have a general sense of what is lacking. Want to prioritize the research. So far, the research is very ad hoc, with no strategy or prioritization. The Great Lakes

Program is involved, the pollution prevention people, and the Science to Achieve Results (STAR) grants. Different groups are involved.

Comment – We need a way of deciding how best to fund this research – not just fund projects as they come up.

Comment – The VCCEP program will come up with specific data needs. We will need coherent Agency research.

Comment – The VCCEP data will have concrete drivers. That will give reasons for what research should be done.

Comment – The VCCEP stakeholder round tables looked at research needs.

Comment – Want broader categories of research needs.

Comment – We also should add in what the strategy will be for figuring out substitutes for BFRs.

Comment – We also need a data management strategy and standardization to make sure that we are analyzing all of the congeners, for example, to make sure data are comparable.

Comment – They held a session with the Design for the Environment people on alternatives.

Q – Can we switch to a less toxic chemical? Should this be part of the study?

(A 10-minute discussion followed on how to develop a research strategy – and when a strategy is developed, how to get it funded.)

Comment – ORD has a different approach to planning for research. The ORD planning process involves getting representatives from all of the program offices and one representative from all the Regions. It involves a balancing act between research needs and capabilities. If there aren't capabilities in the Agency, they use a STAR grant to go outside. There is often some conflict in budgeting.

Comment – There are a number of cross-agency initiatives that don't necessarily come up through this process.

Comment – If this research is for the Regions, then we need to put the dollars into answering the sorts of questions people are asking them.

Q – Are they requesting proposals on BFRs for the next STAR grants?

A – The Regions have been meeting on how they can raise awareness of the science issues that need to be raised. People should go through Paul Gilman. We ought to bring forward what we have in the way of data now, what the data gaps are, and determine where we need help and what we want.

Comment – We are looking for Region support. Region 4 is the lead region for ORD .

Comment – Also looking for multimedia research.

Comment – Regions need to get into the Agency's strategic planning. Some Regional issues are being addressed.

Q – If they put together ideas, where should they take them for funding?

A – Bring them to the regional science council. There is also a national regional science council. We can use this mechanism to get the ideas up to the Deputy Regional Administrator level. If they want to move forward, then we give them more.

Comment – The pollution prevention people are interested in looking at alternatives to BFRs.

Comment – To find studies, look at the science inventory on the ORD intranet.

Comment – It is the responsibility of the project officers on these project to take the information to the BFR group. We should put all of these projects into the science inventory so people will know about them.

Comment – It is important to keep people who are working on BFRs in contact with each other.

Comment – Whoever is writing a strategy needs to make use of the questions people are raising so that other groups that are already doing other work can be involved. Put questions out so that many can answer them. It may be beneficial to make a clear matrix presenting the projects. Have a BFR conference in two years to bring all of these people together with the goal of answering these questions.

Comment – Want to mesh the strategy with what comes out of the VCCEP.

Bisphenol A and Phthalate Esters: Potential Sources of Resin Components in the Everyday Environments of Preschool Children
Marsha Morgan, U.S. EPA/ORD/RTP/National Exposure Research Laboratory (NERL)

Previous research on a small number of children suggested that ingestion (dietary and indirect) is a major route of exposure to environmental chemicals. Exposure routes differ by class of chemical:

Inhalation > indirect ingestion >> dietary ingestion for PCBs
Dietary ingestion > indirect ingestion > inhalation for B2 PAHs, phthalate esters, 2,4,D herbicide
Dietary ingestion > inhalation > indirect ingestion for Total PAHs, phenols, OP and OC pesticides.

Potential doses may be greater for children than for adults in the same households.

The Children's Total Exposure to Persistent Pesticides and Other Persistent Organic Pollutants (CTEPP) study is the first large aggregate study in the country, a pilot of approximately 260 preschool children in North Carolina and Ohio. CTEPP was a large, multimedia, and multipathway exposure study of young children in both residential and daycare settings that looked at the potential exposures that preschool children may have to common pollutants in their everyday environment. The study considered more than 50 different types of pollutants.

CTEPP is significant because it is seeking to provide a greater understanding of children's aggregate exposure to pollutants, particularly pesticides, in their homes and in daycare centers. The study seeks to identify important sources and pathways of exposure that contribute to children's exposures to pollutants and to provide improved approaches for estimating children's exposures to and potential doses of pollutants. It also is intended to fill in critical data gaps under the Food Quality Protection Act of 1996.

The objectives of CTEPP were as follows:

- Measure the aggregate exposures of a small set of preschool children in their everyday environments to persistent pesticides and other persistent organic pollutants
- Apportion the exposure pathways and identify important exposure media, and
- Identify and formulate important hypotheses to be tested in future research.

The study involved 257 preschool children and their adult caregivers. The study was conducted on subjects in six different counties in both North Carolina and Ohio. In both states, four of the counties were urban and two were rural. Half of the children were in daycare and half were in residential settings. The study also considered socioeconomic factors by selecting subjects from lower income and middle/upper-income households. Thus, the stratification of the study involved daycare versus residential, urban versus rural, and low income versus middle-/upper-middle income households.

The sampling periods were 48 hours. Care givers collected food, beverages, urine, and hand wipe samples, while the field staff collected drinking water, indoor air, outdoor air, play area soil, and indoor floor dust samples. Additionally, if pesticides were applied with seven days of, or during, the 48-hour monitoring period, other samples also were collected. These included transferable residues (collected with a polyurethane foam roller), hard floor surface wipe, food preparation surface wipe, and urine (not pooled).

Supplemental information also was collected, including food diaries, child day care menus, activity diaries, and videotapes of 10% of the children to monitor hand-to-mouth and object-to-mouth behavior. Information on aggregate exposures and absorbed doses was collected with urine as the biomarker of exposure.

Targeted pollutants for the CTEPP study include the following:

- Polycyclic aromatic hydrocarbons (e.g., benzo[a]pyrene)
- Phthalates (benzyl, butyl, dibutyl)
- Phenols (e.g., pentachlorophenol, bisphenol-A)
- Polychlorinated biphenyls (PCBs)

- Organochlorine pesticides (e.g., lindane, DDT, heptachlor)
- Organophosphorus pesticides (chlorpyrifos, diazinon)
- Acid herbicides (e.g., 2, 4 – D, dicamba)
- Triazine pesticides (atrazine)
- Pyrethroid pesticides (e.g., cyfluthrin, cis/trans-permethrin)

These compounds were selected because they are possible carcinogens, endocrine disruptors, teratogens, and/or neurotoxins, and are ubiquitous or residues are common indoors or in food or water.

Phthalate esters are used as plasticizers in polyvinyl chloride (PVC) products, such as floor tiles, children's toys, synthetic leather, carpet backings, shower curtains, and adhesives. These uses employ large molecular weight phthalates (e.g., benzyl butyl phthalate). Phthalate esters are also used as solvents and fixatives in personal care products, such as perfumes, hairsprays, and nail polishes. These uses employ the smaller molecular weight phthalates (e.g., dibutyl phthalate).

Bisphenol-A is an industrial chemical commonly used to make polycarbonate plastics and epoxy resins for use in reusable bottles (bottled water, baby bottles), tableware (plates, cups), digital media (DVDs, CDs), electronics (cell phones, computers), dental sealants, and protective liners in metal cans (food, beverages).

There are some analytical issues in looking for phthalate esters, such as background contamination of phthalates in the field and in lab blanks for all media. Possible contamination sources include gauze pads, latex gloves, solvents, nitrile gloves, filtration cartridges, and pipette holders. To minimize background contamination, researchers tried to use as much glass or Teflon as possible and to minimize the handling of samples by using cotton gloves over the plastic gloves.

In regard to the surrogate recovery standard – benzyl butyl phthalate-d4, there were major interferences in certain media (wipes, air, dust, soil). As a result, researchers changed to an alternative surrogate recovery standard (e.g., benzyl butyl phthalate-d10), which had a higher molecular weight and could reduce interferences.

There also were analytical issues in regard to Bisphenol-A. The surrogate recovery standard – bisphenol-A-d6 – also was subject to major interference in certain media (wipes, air, dust, solid food). As a result, researchers changed to an alternative surrogate recovery standard (e.g., bisphenol-A-d10) that could reduce interference. Additionally, researchers had to deal with background contamination in the field and lab blanks and in a few wipe samples.

The preliminary results revealed that dibutyl phthalate was detected at or above method quantification limits in 99% of the indoor air samples collected from homes and 100% of the indoor air samples collected from daycare centers, in 100% of the samples collected in homes and daycare centers, in 83% of the hand wipe samples collected in homes, and 84% of the hand wipe samples collected in daycare centers. Additionally, DBP was detected in 100% of the samples of floor wipes and 85% of the food preparation surface wipes collected in homes.

BBP was detected at or above the method quantification limits in 100% of the indoor floor dust samples collected from homes and daycare centers, 60% of the hand wipes collected in homes, and 96% of the floor wipes and 67% of the food preparation surface wipes collected in homes.

Bisphenol-A was detected at or above the method quantification limits in 93% of the hand wipes collected in homes and 100% of the hand wipes collected in daycare centers, 72% of solid food collected in homes and 74% of the solid food collected in daycare centers, 75% of the floor wipes from homes, and 89% of the food preparation surface wipes collected in homes.

There were no statistically significant differences between the concentrations of DBP and BBP in indoor floor dust samples at preschool children's homes and daycare centers. BBP was found in higher concentrations in homes, while DBP was higher at daycare centers. There was little difference in concentrations of BPA between homes and daycare centers. BBP was detected in much higher concentrations in floor wipes than in other samples at preschool children's homes.

The preliminary conclusions of the study are that DBP, BBP, and BPA were detected in all media; however, they were detected the most often in the following:

- DBP – indoor air, indoor floor dust and wipes
- BBP – indoor floor dust and wipes
- BPA – solid food and wipes.

Hard floors (e.g., vinyl flooring) in homes may be important sources of BBP exposure for preschool children.

The results suggest that these preschool children were potentially exposed to low levels of DBP, BBP, and BPA in their everyday environments.

Comments/Questions and Answers

Q – Are the urine data available?

A – The North Carolina data is done and will be on the Internet in a year and a half. They are still working on the Ohio data.

Q – Did you compare play area soil with indoor play areas?

A – The soil contained low concentrations of the target compounds, so researchers didn't want to make a correlation, because it wouldn't mean anything.

Q – What about sampling grass in grass play areas?

A – In such cases, we would collect surface soil samples from the closest area with bare soil.

Q – When will the results for Ohio be available? Did you adjust the data after completing the North Carolina data?

A – They made some adjustments to the data. The data will be completed in the next year.

Q – This is exposure data. Is anyone looking at toxicological effects?

A – Don't know.

Q – Did you determine the sources of pentachlorophenol?

A – Don't know about sources.

Q – Did you look at agricultural areas and urban areas?

A – This wasn't the objective of the study. Researchers looked at urban and rural areas, but didn't consider agricultural activities in the study design.

Radium in Oil and Gas Piping and Production Facilities
Loren Setlow, U.S. EPA Office of Radiation and Indoor Air (ORIA)

Radium is toxic and a potent carcinogen. Health problems associated with it were first discovered among the women who used radium to paint watch dials and faces. A high incidence of cancer of the mouth was found among these women, who would place paint brushes in their mouths to create a point in the bristles between applications of radium paint. Radium degrades to cause radon and other radionuclides and is present naturally in soil and water throughout the U.S.

The presentation covered the problem of radiation in oil and gas production facilities and in other industrial processes. Radiation at oil and gas facilities is not normally evaluated during environmental site investigations and cleanups. Such sources of radiation may pose potential health and safety hazards to inspection personnel and to the public. Because of recycling of radioactive piping and other materials from oil and gas production facilities, radiation is present in many unexpected residential and industrial settings.

Technologically Enhanced Naturally Occurring Radioactive Materials (TENORM) are materials containing radionuclides that are present naturally in rocks, soils, water, and minerals and whose radioactivity has become concentrated and/or exposed to the accessible environment as a result of human activities.

Because of concerns about TENORM, a Regional EPA Guidance, "Potential for Radiation Contamination Associated with Resource Extraction Industries," was issued on April 15, 2003 to EPA Regional radiation personnel, regional Superfund staff, the National Hardrock Mining Team, and On-scene Coordinators. This guidance informs EPA staff of radiation associated with specific minerals resource extraction, processing, or manufacturing industries. The guidance will help EPA field staff to contact key EPA regional radiation staff personnel to implement radiation safety measures and conduct radiation surveys, as appropriate. The guidance can be found at www.epa.gov/radiation/tenorm/about.htm.

Some industries with TENORM contamination include the following: aluminum (bauxite), copper, fluorspar, gypsum, molybdenum, phosphate, phosphorus, potassium (potash), precious metals (gold, silver), rare earths including monazite, tin, titanium (leucoxene, ilmenite, and rutile), tungsten, vanadium, zircon, coal (and coal ash), oil and gas, and geothermal energy. Water and sewage treatment facilities also can be contaminated with TENORM.

In regard to oil and gas facility TENORM, radium levels can range from non-detectable to more than 100,000 picoCuries/gram. Five picoCuries is the allowable limit for radium in pipe scale. It should be noted that radium is much more dangerous than uranium or thorium ore. In 1996, the American Petroleum Institute (API) estimated that the equivalent of 30,000 m^3 of TENORM was generated in 1993. This same study, using radiation survey data, estimated that there is the equivalent of 3.4 million m^3 in total legacy inventory. In some cases, TENORM has gotten into facilities that recycle steel. Such facilities have had to install radiation monitors to reject radioactive materials. Some contaminated pipe has been used in homes and businesses.

In regard to TENORM in Region 5 oil and gas facilities, oil field equipment with 5,000 microR/hr was found at one Michigan pipe yard. An API study found that 92% of production facilities in Illinois had radiation contamination.

Radium is selectively soluble in underground formations and is mobilized in production waters in preference to uranium and thorium. The radium precipitates out at the surface because of changes in temperature and pressures compared to underground conditions and it plates the interior of pipes, separators, storage tanks, gas lines, etc. On the basis of a 1989 API report and other studies, it is believed that one-third of all producing U.S. oil and gas wells have elevated radiation.

The first oil wells were drilled in the 1860s. According to the Illinois State Geological Survey, 155,000 wells have been drilled in the Illinois basin alone. A hot trend now is to drill natural gas wells into coal beds to recover coal bed methane. Because coal contains radioactivity, TENORM can be generated. Contaminated water also is a problem that must be managed. There is a problem managing TENORM contaminated water in the Powder River Basin coal fields because a lot of water is produced.

By anecdote, Setlow has heard that scrap yards have been under orders to not accept oil field pipe, and there have been some problems in the automotive industry from radioactive metal used in cars. Contaminated metal made from scrap has been used in many places, including playgrounds. Exposure to TENORM in pipe scale has been a problem for pipeyard workers. Until the 1990s, workers at pipeyards used to knock the scale out using hammers; now, chemical and hydraulic pressure means are used.

Exposure to the scale and disposal of the TENORM-contaminated scale is an issue. When radium is in the mineral scale, it is locked up in the mineral matrix and not readily leachable. But when it is broken out of the pipe or tanks or other vessels and ground up, it becomes an inhalation hazard and a toxin that gets into the ground.

TENORM also can contaminate soil. The MCL for Radium-226 in soil is 5 picoCuries/gram. At one site in Michigan, soil samples contained up to 2700 picoCuries/gram.

Oil and gas TENORM waste disposal methods include injecting wastes into the original producing formation, deep injection well disposal, disposal at licensed facilities, land and road spreading, and re-use and recycling of equipment. TENORM cannot be sent to RCRA Subtitle C or D landfills (except under some special cases). It usually has to be disposed of at a facility for low level radioactive wastes.

There have been more than 5 million oil wells drilled in the U.S. and any of them may have TENORM that may never have been dealt with or even recognized. Modern environmental protection methods didn't commence until the 1970s. Pre-1970s legacy wastes may remain at sites that could be converted to home and building sites. As residential communities move into former oil and gas production areas, the need to identify and address TENORM becomes increasingly important.

As already mentioned, potential hazards exist from TENORM contaminated recycled metal scrap, mineral scale, and pipes. In one case in New Orleans, 26 acres of land was leased to a pipe yard. The pipeyard company did not clean up the radium scale on the property, and the owner of the land sued the pipeyard company and the oil companies which supplied the pipe to be cleaned. In 2001, a $1 billion judgment was handed down in the case for both compensatory and punitive damages. The workers at the pipe yard were exposed to radiation while they broke the scale out of the pipes and several of them subsequently sued their employer saying they were never told of the risks associated with this activity.

In regard to TENORM contamination associated with current production sites, RCRA may not apply, but other U.S. EPA authorities are available (CERCLA, CWA, SDWA, and TSCA). Petroleum industry associations, individual companies, and some states have developed guidances for handling TENORM. EPA ORIA is investigating ways to partner with industry and other stakeholders to address this issue.

More research is needed on the occurrence of legacy oil and gas radium wastes and current operational and disposal practices. This research is needed to reduce potential for public, professional, and worker radiation exposures.

Comments/Questions and Answers

Q – What levels of radiation can be found in the pipe scale?

A – The scale can have levels of hundreds of picoCuries/gram. Scrap metal companies are supposed to have zero tolerance for radioactivity.

Q – Do the automobile manufacturers look for radioactivity in the metal they receive?

A – Don't know.

Q – Why don't scrap companies use Geiger counters?

A – It depends on the company. Not all train their workers on how to deal with pipe scale.

Q – What is the level of exposure to radiation to workers in oil fields? Are there any effects?

A – Don't have data on this. OSHA has standards. OSHA and the Nuclear Regulatory Commission say occupational exposures can be five times the level of exposure allowed for the public.

Q – Is radon a degradation product of radium?

A – Yes. In many cases radon emissions become the driver of risk assessments that are performed. Radium dust contaminates soil. It is a problem if contaminated soil is used for a playground. Radiation also can occur if a house is built over a contaminated site.

Q – Are there any pollution prevention remedies to this problem, like using water softeners?

A – Some producers use scale inhibitors, but this just moves the problem further down the line toward the refineries.

Comment – Institutional controls could limit it.

A – At this point, 37 states have oil or gas wells, but only 10 states have regulations for TENORM. The issue is whether there are controls built into the production process.

Q – Is radium leachable into the new steel in which the scrap is recycled?

A – Some of the radium goes into the slag, some of it goes into the steel. The percentages depend on the smelting temperature.

Q – Have you found radioactivity in the natural gas used to burn in our homes? Is it then air borne?

A – Some has been found in gas lines, but it isn't a risk in most homes. The radium goes into the air when the gas is burned.

Q – Is lead 210 a scale that develops?

A – Yes, it is deposited over time.

Q – Is radium a problem in drilling mud?

A – It can be. It can also be a problem in produced water. Producers try to recycle the drilling muds. There are pits (including dried out pits) containing produced muds and water all over the country.

Balanced Perspectives Regarding Environmental Levels of Thallium and Platinum Group Metals

Frank Anscombe, U.S. EPA Region 5

(This talk is available in full from the presenter (anscombe.frank@epa.gov) and represents his views, which may in places depart from those of EPA. This summary is provided for the Proceedings.)

This talk discusses environmental exposures to two metals, thallium and platinum. To have perspectives on their risks, it seems useful to place them within the broad context of trace environmental exposures to chemical compounds.

Trace Exposure Risk Primer

Our dominating doses are to the chemicals that comprise foods that we eat and medicines that we take. Approaching 1,000 different chemical compounds have been analytically distinguished in a cup of coffee. A National Research Council study has affirmed it plausible that our exposure to carcinogens naturally contained within foods dwarfs risks presented by trace residues of synthetic chemicals on these foods. There is no comprehensive program to evaluate the carcinogenic potency of the natural ingredients within foods.

The gentlest substances are regarded by pharmacology as "practically non-toxic." If one drinks enough water, this will dilute your electrolytes to a lethal degree. Thus water can be a toxicant. So is oxygen. Long-term respiration generates free radical oxidants thought to be the most important cause of age-related diseases. We require oxygen minute-to-minute, yet in the long run it kills us. We are the physical manifestations of many chemicals. All chemicals can be at least somewhat toxic. Toxicants R Us.

It is comforting to think of certain chemicals as "bad." Some are indeed virulent poisons, devoid of biological value. It is comforting to think that other chemicals are safe. The truth is murkier. A large dose of a practically safe substance can be just as deadly as a tiny dose of a deadly poison. We learn of chemical harm to humans owing to high-dose tragedies. It is hard to assess what risks may be presented by low doses of these same chemicals, given myriad possible biological outcomes without acute symptoms.

With sufficiently sensitive and selective analytic technique, with available reference standards, and with funding, an analytical chemist could distinguish thousands of synthetic chemicals, at trace levels, in us. What risks to these trace exposures present, individually or collectively? How can we evaluate this situation prudently and thoughtfully, ranking one risk against another, and set logical priorities? Low analytic detection capabilities create a frustrating problem. Chemists can detect myriad chemicals at levels lower than our capacity to estimate robustly what risks might be entailed. We can find a part per billion, but does it threaten us? We can make estimates, but they must be predicated on assumptions.

Should we fully inform citizens that they are exposed to myriad trace chemicals, predominantly natural, yet many others synthetic in origin? Or should we narrowly inform the public of just one trace chemical exposure at a time? Generally, the latter choice seems to be made.

Simplicity is a mighty virtue in messages to the public. Yet is it to some degree misleading not to mention the full range of chemical exposures? Is this ethical?

If the honest situation is that we genuinely do not know how to compare all the risks presented by a host of trace chemical exposures, would acknowledgment of this be worthwhile? Are we best served to avert our eyes from the complexity of the real world's trace exposures? Or should we acknowledge reality, however inconveniently perplexing and troubling?

An intrinsic mission of EPA is to be skeptical about chemical exposures, especially ones involuntarily presented via food, water, or air. It seems a responsible idea to hold this workshop, regardless of profound uncertainties. But it would be irresponsible not to be mindful of these same uncertainties.

EPA-derived food consumption advisories tend not to factor in the health benefits of all the chemicals that are natural constituents within food. Does such narrow framing beget advice that is defensible on medically holistic grounds? Do advisories depend on a premise that there are alternative healthy foods readily available? If so, does this premise deserve to be expressly acknowledged?

Thallium

On the periodic table, thallium lies between lead and mercury, two other potent neurotoxins. Owing to the observed symptoms, thallium was the first suspect for the neurological poisoning at Minamata, Japan. Within our bodies, thallium mimics an essential metal, potassium. It affects potassium-activated enzymes in the brain, muscles, and skin. Symptoms of thallium poisoning include slurred speech, numbness, and hair loss. A fatal dose for an adult is about one gram. This qualifies as extremely toxic. Thallium's abundance in the earth's crust is 59th among elements, ten times more common than silver, in contrast. EPA canceled all pesticide uses of thallium in 1972, partly owing to a survey that found 24 percent of sick or dead bald eagles had been poisoned by thallium. Global air emissions of thallium from coal combustion have been estimated at 650 to 1,600 tons per year. Cement production may yield much more: 2,700 to 5,300 tons. Plants absorb thallium through their roots.

Thallium has long been undetectable by conventional analytic means. This is an important factor in explaining why this dangerous metal is virtually ignored. Lin and Nriagu have found that the average concentration of thallium in 37 lake trout taken from Lake Michigan during 1994 was 140 ng/g. In 1992, EPA suggested a reference dose for thallium of 5 ug/70kg/day. High consumption fish consumers exceed this safe dose by several fold.[i] The margin of safety for thallium in fish seems modest, as it does for methyl-mercury. The two substances together may pose additive risk greater than either alone. Recent advances in analytic methods can empower more understanding of thallium exposures.

Platinum Group Metals

The platinum group metals (PGM) Platinum, Rhodium, and Palladium have been used in catalytic converters installed on U.S. automobiles since 1975. These convert NOx, hydrocarbons, and CO to CO2, nitrogen, and water, improving air quality. These metals are in

75th, 76th and 79th place in abundance among the earth's elements. This rareness brings two implications. Use of these metals in a hot gas stream produces new vapor exposures. Ambient PGM levels near roads have risen, viewed from the perspective of ultra-low natural background. The second implication is that there is not a lot of knowledge about inhaling platinum group metals.

The good news is that the three metals are of low toxicity. Yet, a study of vegetation and soils beside roads in South Bend, Indiana reports abundance is comparable to many European studies and approaching levels that would be economically viable to recover.[ii] A study from Rome showed a six-fold increase in platinum levels in top soils between 1992 and 2001.[iii] However, air concentrations are still three orders of magnitude below levels for which occupational health limits have been set. The known health risk is sensitization of the airways by soluble platinum compounds, known as platinum salt hypersensitivity. This has been suffered by platinum workers, who develop runny noses and asthma. Given that present exposure limits offer a 1000-fold safety margin in relation to this condition, inhalation of platinum group metals does not seem to pose a health risk to the general population, based on existing understanding.

Yet neither should we assume that we understand all human or ecosystem outcomes for these metals. Our experience with them is not extensive. I have tried to make the general case that all substances, in sufficient dose, can create harm. We should always be wary of chemical exposures.

Closing

We humans are made up of many chemicals and we are exposed to many more, both natural and man-made. When you next hear of a synthetic chemical being banned, you may wonder: what is the public health gain, in a holistic sense? Does anyone know of the totality of the risks that will be presented by its replacement? How does the risk of this one chemical exposure compare to the many others?

In writing these remarks, I chanced on a paper regarding caffeine in some lakes in Switzerland.[iv] Caffeine is contained in coffee, tea, soft drinks, chocolate, and pharmaceuticals, from cough syrups to No Doz. Caffeine is likely the most consumed drug in the world. It is found in municipal wastewater effluents, and in surface and ground waters worldwide.

I looked at the National Toxic Pollutant repository of the National Institutes of Health. Under caffeine, I read "at higher doses, symptoms include convulsions, Cheyne-Stokes respiration, apnea of preterm infants, arrhythmias….Overdoses may cause death, convulsions…Continued excessive use may lead to depressed mental states. Symptoms may include pulmonary edema, myocardial infarction."[v] A report from the Massachusetts Poison Center by a doctor at the Boston Children's Hospital: "The issue of fetal toxicity of caffeine remains controversial. The drug has been demonstrated to induce chromosomal mutations in vitro, but not in vivo. Clinical trials in humans have shown heavy coffee drinkers to have an increased incidence of miscarriages and premature birth, however these differences lose significance when adjusted for other factors such as smoking. Further study is needed.[vi]

What would happen if it were reported that one chemical, yet only one, deemed toxic by the respected National Institutes of Health were present in our drinking water supplies? , Many people have to have caffeine. Yet what if there were another chemical and it had an unappealing name? What if someone invokes the precautionary principle?

Conclusions

- Thallium is intrinsically unhealthful. The same seems true for lead, mercury, cadmium, and beryllium. This is an evolutionary perspective that living things have not incorporated and made use of these metals. This natural avoidance seems a useful insight about them. Many other metals are used gainfully by the human body. I do not claim that thallium exposures constitute a problem worthy of action. Yet more studies seem warranted.
- As regards all chemicals, dose (including pathway, quantity, chemical forms, timing, and the vulnerability of an exposed person) determines harm. I am skeptical of all chemical exposures, from a holistic perspective and bearing in mind uncertainties.
- The world abounds in risks from chemical exposures. It seems reasonable to focus on ones that seem most important. Chlorides may be a long-term threat to the Great Lakes, for instance. We and our planet contain ingredients that can be regarded as good or ill, depending on dose. Health consists of striking the best balance.

Break Out Sessions

Break out sessions were held to discuss the following topics:

- BFRs and Electronic Equipment
- Radium, Thallium, Platinum, and other metals, and
- PFOS/PFOA.

Flip chart and discussion notes from these break out sessions can be found in Appendix D of this document.

Wednesday, August 13, 2003

Asbestos and Related Durable Fibers: Too Ubiquitous, Too Persistent, Too Complex to Put Health Risks to Rest?
Phillip Cook, U.S. EPA/ORD/NHEERL/Duluth, MN

Health effects from exposures to microscopic durable fibers is not an old issue – it is a continuing issue. An extensive history of research, discussion, and debate, which focused on occupational exposures to a few types of asbestos fibers, has not lead to an understanding of all risks. "Asbestos" is more a slowly expanding pollutant problem than a re-emerging one. It hasn't gone away, even if it no longer commands a lot of attention. A hallmark complication for risk assessment in the case of asbestos is the very long lag time between exposure and effects. It may take 30 to 50 years from exposure to the development of pulmonary disease.

Objectives of the presentation are to provide an overview of asbestos health risks, to give examples of EPA's experience with asbestos-like fibers, and to describe why we need and how we can develop a relative potency model to assess risks from complex mixtures of different mineral fibers and new synthetic fibers. Asbestos is a persistent, bioaccumulative toxin, much like PCBs. We need to look at asbestos the same way we have looked at PCBs. Some synthetic nanofibers that are being developed are similar to asbestos. We should take what we know about asbestos risk to look at nanofibers so we can avoid developing substances that would pose asbestos-like risks.

Chrysotile asbestos is the main type of asbestos used in the U.S. Some say it is not a carcinogen. Cook believes that it is a carcinogen. Amphibole crystals are present in taconite (iron ore) from Northeast Minnesota – ferroactinolite replaces hornblende in some rocks with cummingtonite-grunerite being the most common amphibole overall. Ferroactinolite appears to be the amphibole which is most consistently fibrous. Amphibole can't be extracted from the rock for use, but it has fiber-like characteristics and can be released in dust when the rock is crushed. Particles of chrysotile or amphibole asbestos occur as thin fibers or in bundles that can enter the lungs and cause asbestosis. However, short fibers that can break off of larger rodlike bundles can be respirable, as well. In the past, scientists have tended not to consider the risk from the short fibers, and science today still rests on dose-response relationships based on light microscopy, which was inadequate for detecting short and thin fibers. The assumption was that only the longer and thicker fibers pose a health risk.

Once the transmission electron microscope (TEM) became available, it enabled researchers to see very small particles and to even analyze the chemical composition of individual fibers. It is still difficult to tell whether individual fibers grew as a single crystal or were fragments of single crystals. Some researchers believe that cleavage fragments aren't a problem and should not be counted. Cook believes that the cleavage surfaces would be no different than the natural fiber surfaces and that they also can cause disease if sizes and shapes are similar to asbestos fibers which cause disease.

Cancers in the respiratory tract develop when the fibers reach the alveoli and are retained for many years. The fibers can also cause fibrosis and the lung changes responsible for asbestosis, a

fatal condition. There also is a significant incidence of gastrointestinal cancers in highly exposed occupational settings, but researchers don't know whether the cause is inhalation or ingestion of the fibers.

Previously, health officials thought asbestosis was associated with high exposure levels, and that cancer resulted from lower exposures of harmful fibers. The highest rates of asbestosis in the 1990s were clustered around seaports or industrial sites where occupational exposures were high in the 1940s and 50s. There also were some hot spots in less industrial areas, such as Libby, Montana, where vermiculite mining was associated with amphibole fiber exposures. Cancer loci associated with asbestos fibers exposures include the lung (with smoking a strong co-factor), the pleura and peritoneum (mesothelioma), gastrointestinal tract and kidney.

A conceptual model is needed for development of methods for prospective assessment of health risks associated with exposures to mineral fibers. Researchers need to know what dose in the tissues/lung should not be exceeded and to explore temporal exposure issues, such as the effects of life-time, short-term, or early life exposures (from environmental exposures). Existing data on the dose-response in tissues is based only on long fibers and light microscopy. Data is needed on relative potencies, respiration response, retention/clearance, and fate/transport issues.

Vermiculite mining in Montana started in the 1920s about six miles from the town of Libby. About 80% of the world's vermiculite was produced there, involving the processing of more than 300,000 pounds a day. Around 5,000 lbs/day of amphibole fibers were released into the Libby airshed. W.R. Grace bought the mine in 1963 and closed it in 1990. Products made from the fiber included construction aggregate, fireproof coatings, insulation, and soil additive and fertilizer. EPA's primary focus is on insulation, because it is present in thousands of homes across the U.S. Exposures weren't just limited to Libby, however, because approximately 300 plants across the U.S. and in Canada processed the vermiculite ore. Many workers were affected and some children who played in the waste piles from the processing activities got mesothelioma.

Iron formations are found in the Lake Superior region. Amphibole, a potentially fibrous material, exists in the taconite rock at the eastern end of the Mesabi Iron formation. Iron ore from the Peter Mitchell Pit was processed at Silver Bay, Minnesota. Taconite tailings from processing 67,000 tons of fine crushed rock were deposited into the lake each day. Not all of the fine tailings went to the bottom of the lake. The transport of fine tailing particles caused noticeable increases in turbidity in western Lake Superior, leading to ecological concerns in the 1970s. When particles in water samples were viewed with an electron microscope, the resemblance of amphibole particles to amphibole asbestos fibers in these tailing particles was noted.

Tailings particles were tracked through the lake and uses of water from the lake. Researchers found amphibole in toilet bowls and in the drinking water of Duluth, Minnesota in 1973. The connection to asbestos first emerged through a citizen's comments made in opposing the tailings discharge into the lake. Later, Cook examined the literature and found that indeed there was a report of high incidence of gastrointestinal cancers in Japan in association with ingestion of similar amphibole fibers. Thus, what was first thought to be a case of ecological concern turned into a case of human health risk after the fibers were found in the drinking water. EPA moved

quickly and people were given the option of filtered water or well water. Risk from ingestion of fiber in water is still considered controversial as the evidence remains largely circumstantial.

With litigation pending, EPA took action immediately on human health concerns. The Minnesota Pollution Control Agency was involved in issues associated with fibers in the air. Cook found that people who ingested unfiltered water uniquely had amphibole fibers in their urine, probably as a consequence of penetration of the gastrointestinal tract by a small fraction of ingested fibers. Tailings discharges to the Lake were stopped and air emissions regulated. There is, however, a proposal to use the dry cob tailings, which contain amphibole fibers, as aggregate for road construction. This raises a new issue in regard to possible future exposures and health risks.

Concerns about risks associated with non-occupational exposures to mineral fibers (e.g., Reserve Mining Case) and interest in the effects of synthetic fibers led to EPA research on the effects associated with a wide variety of durable fibers during the period of 1978-85. A major objective was to determine the carcinogenic potencies relative to known asbestos materials. The Duluth EPA lab provided electron microscopic characterizations of samples used in biological tests, quantitative measurements of fiber doses in test animals, and determinations of dose-response relationships. Researchers determined that amphibole fibers from taconite could cause cancer.

In the intratracheal and intrapleural exposures of Fischer-344 rats study, the primary objective was to determine relative potencies of different fiber types for carcinogenesis. The studies included two samples of amphibole from taconite at the Peter Mitchell Pit – ferroactinolite (fibrous) and grunerite (non-fibrous). For details, see Coffin et al. Toxicology Letters, 1982, and Cook et al. Toxicology Letters, 1982.

Results showed that ferroactinolite fibers were dissolving and splitting longitudinally while residing in rat lung tissues over time. The dose was changing because fibers were splitting, producing more and thinner fibers. In a hypothetical relative potency model proposed by Pott in 1978, long fibers were assumed to be most potent, but short fibers were also assigned finite potencies which could be significant when many more short fibers were in an exposure.

In fiber carcinogenicity equivalence dose (CED) studies comparing relative carcinogenicity factors (RCFs) based on Pott's hypothesis, data show that RCFs for short and thin fibers greater than those proposed by Pott should be investigated and considered.

Conclusions from rat studies include:

- Fiber splitting in vivo greatly enhanced the potency of ferroactinolite in rat studies.
- Short and thin amphibole fibers appear to affect toxicity. If they do not, long ferroactinolite fibers would have to be regarded as many times more potent than long amosite or crocidolite fibers.
- Because risk is a function of cumulative fiber dose, exposures should be measured on the basis of all fiber sizes with consideration of relative carcinogenicity and fibrogenicity of different size and shape categories.
- Similarly, exposure predictions should be based on all fiber sizes so that relative potencies can be included in risk assessments.

Narrow definitions of hazardous fibers perpetuate risk assessment limitations:

- No methodology for assessing risks from short fibers – we need a relative potency model.
- Weak links to mechanism of action data.
- We are unable to provide precise definition of undesirable synthetic fibers so that safe alternatives can be developed (we need to define risk in a generic way so we can predict risk from new products, such as nanofibers).
- Human dose-response relationships are very uncertain and may be inaccurate.

Properties of microscopic fibers that indicate the potential for causing asbestos-like pathologies include the following:

- Size and shape that allows respiration, retention in lungs, and translocation to the pleura.
- Durable, persistent in tissues.
- Reactive surfaces, ability to induce ROS (reactive oxygen species).
- High collective surface area.
- Propensity to split into thin fibers in vivo.

An example of how our general knowledge of effects caused by different durable fibers can be helpful in predicting potential emerging health risks is provided by carbon nanofibers which are being used to produce zeolite tubes resembling the most potent asbestos fibers. These fibers are proposed for use in monitoring and filtering applications, but should not be produced in sizes that allow them to be respired and retained in human lungs. We should use our knowledge of asbestos to work with industry to develop fibers that aren't respirable. Effective environmental protection requires that each new generation advances the knowledge passed on by the previous generation.

Comments/Questions and Answers

Q – Is the relative potency based on fiber size or weight basis?

A – Cook believes relative potency should be measured on a per particle basis.

Q – Would the zeolite fibers that are manufactured split?

A – They have a high tensile strength, which is why they are so attractive. Like the carbon nanofibers they are produced from, they would not have an internal structure that would promote splitting.

PHARMACEUTICALS AND PERSONAL CARE PRODUCTS

Pharmaceuticals and Personal Care Products (PPCPs) As Environmental Pollutants (Pollution From Personal Action)

Christian G. Daughton, U.S. EPA/ORD/NERL-Las Vegas

The focus of this research, funded by U.S. EPA, is primarily on Pharmaceuticals and Personal Care Products (PPCPs) in the environment, originating from end-use rather than from manufacturing. The emphasis is on the use/disposal of PPCPs primarily from activities/actions of individuals and to a lesser degree from hospitals and industry – not from the PPCP manufacturing sector, whose waste streams are much better defined, confined, and controlled/controllable.

Another class of potential pollutants, the Endocrine Disrupting Compounds (EDCs) are also a concern. A plethora of terms have often been used interchangeably with EDCs (rightly and wrongly): environmental estrogens, endocrine-disruptors, endocrine-modulators, estrogenic mimics, eco-estrogens, environmental hormones, xenoestrogens, hormone-related toxicants, hormonally active agents (HAAs), endocrine-active chemicals (EACs), endocrine active substances (EASs), and phytoestrogens (a naturally occurring subset). Estrogens represent but one mode of action. Others include androgens.

PPCPs and EDCs are not synonymous. They are intersecting sets, and confusion regarding their relationship must be avoided. Only a small subset of PPCPs are known/suspected of being direct-acting EDCs (e.g., synthetic steroids); toxicological concerns usually differ. EDCs comprise members from many disparate chemical classes.

PPCP-relevant goals for the U.S. EPA's ORD include the following:

- Identification of potential environmental concerns: anticipatory research, and emerging issues. Identifying pivotal sources of uncertainty that affect risk estimates.
- Proactive vs. Reactive – pollution prevention vs. remediation/restoration: identifying and fostering investigation of "hidden" or potential environmental issues/concerns before they become critical ecological or human health problems.
- Foster interdisciplinary research and collaboration: catalyze research by academe, private sector, government. Attempt to determine whether certain classes of chemicals deserve more attention in rule-making.
- Ruling-in/out vs. Uninformed rules: Provide bases for informed decisions. Ensure that science leads to eventual decisions for guidance or to regulate/not regulate.

Since the 1970s, the impact of chemical pollution has focused almost exclusively on conventional "priority pollutants," especially on those collectively referred to as "persistent, bio-accumulative, toxic" (PBT) pollutants, "persistent organic pollutants" (POPs), or" bio-accumulative chemicals of concern" (BCCs). The "dirty dozen" is a ubiquitous, notorious subset of these comprising highly halogenated organics (e.g., DDT, PCBs). The conventional priority pollutants, however, are only one piece of the larger risk puzzle. More attention must be devoted to the unregulated chemicals.

PPCPs come from industry, agriculture, and household maintenance activities. Chemicals are overlooked because there is no analytical framework to define them. The set of neglected or omitted/overlooked chemicals is constantly expanding with many new chemicals being introduced to the market, and with the poorly characterized influence of environmental weathering and transformation thattakes place.

Regarding the prevalence of xenobiotic occurrence, some possible generalizations may be made about its ubiquity:

- The lower the concentration, the higher the probability of larger numbers of distinct chemicals occurring.
- Exponentially more types of chemicals occur at exponentially lower concentrations.
- At the very lowest concentrations (septomolar to yoctomolar (zM-yM), the off-the-cuff truism may apply that, "Everything can be found everywhere."

According to Einstein, "Not everything that can be counted counts, and not everything that counts can be counted." Daughton reflects on the state of the art of environmental monitoring by saying, "Not everything that can be measured is worth measuring, and not everything worth measuring is measurable."

Further truisms regarding environmental monitoring hold: what one finds usually depends on what one aims to search for; only those compounds targeted for monitoring have the potential for being identified and quantified; those compounds not targeted will elude detection; and the spectrum of pollutants identified in a sample represent but a portion of those present and they are of unknown overall risk significance.

There is a major unanswered question regarding the toxicity of complex environmental mixtures. Is the overall toxicity (or the spectrum of unique toxic effects) of a complex mixture caused by few or many chemical constituents? And do these constituents comprise a small or large fraction of the total dissolved mass? PPCPs in waters is likely not a new phenomenon, but has become widely evident in the last decade because continually improving chemical analysis methodologies have lowered the limits of detection for a wide array of xenobiotics in environmental matrices. There is no reason to believe that PPCPs have not existed in the environment for as long as they have been used commercially.

PPCPs are a diverse group of chemicals comprising all human and veterinary drugs available by prescription or over-the-counter. People are largely not aware that their daily actions affect the environment in this way. Certain pharmaceutically active compounds (e.g., caffeine, aspirin, nicotine) have been known for over 20 years to occur in the environment, primarily from treated and untreated sewage effluent. Now we know that numerous PPCPs can occur at very low concentrations. Prior discovery was delayed by limitations in analytical environmental chemistry (ultra-trace enrichment and detection).

Confined animal feeding operations (CAFOs) are a major source of antibiotics.

Portions of most ingested drugs are excreted in varying unmetabolized amounts, primarily via

urine and feces. Other portions sometimes yield metabolites that are still bio-active or are excreted as conjugates. Free excreted drugs and derivatives can escape degradation in municipal sewage treatment facilities; the conjugates can be hydrolized back to the free parent drug. Undegraded molecules are then discharged to receiving surface waters or find their way to groundwaters through leaching and recharge.

Continual input of PPCPs to the aquatic environment can lend persistence to compounds that otherwise possess no inherent environmental stability. The full extent of their impacts is unknown. Most ecological monitoring studies to date have been performed in Europe (except for USGS). The use/release of antibiotics and natural/synthetic steroids to the environment has generated most of the controversy to date, but a plethora of other PPCPs have yet to be examined. The scope of the overall issue is ill-defined.

Regarding pharmaceuticals in the environment, toxicological significance for both humans and ecological exposure to multiple chemicals at trace concentrations (ppb-ppt) for long durations is poorly understood. If PPCPs eventually prove to be an environmental concern, it is unknown whether sewage treatment facilities could be cost-effectively modified to reduce emissions. Source control aimed at both disposal and medical practices ("environmental stewardship" programs) may prove more effective. The focus should be on proper and sufficient science for establishing occurrence, exposure, and susceptibility/effects, so that sound decisions can be made regarding human and ecological health.

Other potential routes to the environment include leaching from landfills, runoff from CAFOs and medicated pet excreta, loss from aquaculture, spray-drift from agriculture, direct discharge of raw sewage (from overflow events and residential "straight piping"), sewage discharge from cruise ships (millions of passengers per year), oral contraceptives used as soil amendment and plant growth tonic (urban legend), and transgenic reduction of proteinaceous therapeutics by genetically altered plants (aka "molecular farming" – "biopharming"). Discharge to the environment also occurs via dislodgement/washing of externally applied PPCPs to the skin.

Unanticipated routes of inadvertent ecological and human exposure to drugs is likely via water (and secondarily, sludges and sediments). In certain localized situations, however, inhalation exposure through sorption of drugs to respirable particles could occur despite the low volatility of these compounds. . Dust from confined animal feeding operations where drugs are used to supplement feed and given directly to animals also could contaminate the environment. Pig house dust in samples collected over 20 years contained five antibiotics in concentrations up to 12.3 mg/kg.

In 1999-2000, USGS implemented the first ever U.S. national reconnaissance of "emerging pollutants" in waters. In 2001 the first ever study was published on geographic variation across the U.S. of prescription drug use. These occurrence data demonstrate the potential for ANY consumer-use chemicals to enter the environment and warn us to be watchful for drugs with new mechanisms of action and ever-increasing biochemical potencies.

Some drugs have double uses as medicines and pest controls. The potential significance of these alternative uses as sources of environmental release has never been explored. Examples include 4-aminopyridine, an experimental multiple sclerosis drug and an avicide, or warfarin, an

anticoagulant and a rat poison, among others. In September 2001, U.S. EPA allowed an exemption from FIFRA for caffeine use to control the reproduction of coqui frogs in Hawaii, which were out-competing birds by massive consumption of insects and producing a piercing and annoying chirp.

Acetaminophen as been used on Guam to control brown tree snakes, which were reproducing in great numbers without a natural predator and decimating bird, bat, and reptile populations and causing economic losses to agriculture. Acute effects of larger doses of acetaminophen on local non-target species have not been detected. Vultures (Gyps bengalensis) in Pakistan and India may have declined in the 1990s due to pesticide use, diclofenac poisoning, or from pathogens. The pesticide is used for cattle in India, and carcasses are a major food source of vultures.

All municipal sewage contains PPCPs. Local doctors' prescribing customs and cultural habits vary the types of PPCPs found in receiving waters. Illicit and abused drugs also may be found. Groundwaters may be affected. Sources of raw sewage in the U.S. include combined sewer overflows, sanitary sewer overflows, leakage from sewage transport infrastructures, failing septic systems, unpermitted privies, and straight piping. The American Society of Civil Engineers (ASCE) gave a grade of D in 2001 to U.S. drinking water and wastewater infrastructures. More than $20 billion may be needed to repair them to a suitable level.

Subtle effects of PPCPs emanate from their numerous receptors, with response being exquisitely specific and sensitive. Receptors of exposure and resulting effects can differ greatly from those of currently regulated pollutants. Receptors in non-human species could differ from those in humans. Researchers believe that acute effects may be less important than chronic effects to low concentrations over time. Receptors could be different in aquatic environments, but this is difficult to predict.

Subtle effects also include impacts on development, spawning, and other behaviors in shellfish, ciliates, and other aquatic organisms. Calcium-channel blockers can inhibit sperm activity. Antiepileptic drugs could trigger apoptosis in the developing brain. Very small amounts of NSAIDS, glucocorticoids, and anti-fibrotics affect collagen metabolism in teleost fish, leading to defective fin regeneration. Multi-drug transporters (efflux pumps) are common defense strategies for aquatic biota, but may compromise aquatic health.

Latent damage in humans could cause subtle shifts in behavior or intelligence. Advances are required in developing and implementing new aquatic toxicity tests to better ensure that such effects can be detected. Slow poisoning of a victim with low doses administered over a long time period was a popular practice in Europe in the 1600s and in the 1800s, akin to what may be going on in the environment today with PPCPs.

Aquatic organisms are captive to continual, life-cycle, and multi-generational exposures of chemicals via sewage released to surface waters. The supply of chemical contaminants is also continually replenished. The risk cup body burden can be great due to additive effects from multiple agents sharing common mechanisms of action (MOAs).

There may be possible interactive effects, especially synergism, which is difficult to demonstrate. Non-target species receptor repertoires are not well characterized. Hormesis, or

unknown effects, can accrue from doses below the therapeutic level. There has been little research done at extremely low concentration levels. There may be susceptible genetic outliers within species. MOAs are not fully understood.

The major collateral benefits of research and development efforts with PPCPs include optimizing or improving existing wastewater/sludge treatment technologies (primarily for POTWs) and developing or implementing new, cost-effective technologies for further lowering the trace levels of PPCPs. Any improvements to remove trace levels of PPCPs also will likely remove numerous other unregulated pollutants from water.

Pollution prevention efforts like source reduction could have significant consequences for improved consumer health and the economy. These "cradle-to-cradle" stewardship efforts could originate from the healthcare industry or from the individual consumer. Voluntary compliance with a life-cycle stewardship approach also could improve medical healthcare outcomes and consumer safety as well as reduce healthcare costs. (See Environmental Healthcare Perspectives 111, 2003.)

There is a wealth of materials and links addressing environmental stewardship of PPCPs at the U.S. EPA's web page on the Green Pharmacy: http://www.epa.gov/nerlesd1/chemistry/ppcp/greenpharmacy.htm. In communicating risk more effectively, perhaps there will be growing public acceptance of wastewater re-use for human consumption ("toilet to tap" re-use programs). It also is important to forge collaborations between scientists and the medical community. (See "Environmental stewardship and drugs as pollutants," C.G. Daughton, The Lancet, 2002, 360:1035-1036).

In the future, the safety of drinking water supplies must be ensured by removing PPCPs and other unregulated pollutants from treated wastewater. The decaying water/sewage infrastructure must be improved and cost-effective technologies developed for optimal removal of PPCPs. Concerns regarding the introduction of PPCPs to the environment from molecular farming must be investigated. Integrated industry-consumer stewardship programs for minimizing the introduction of PPCPs to the environment must be developed. A cohesive national or international guidance must be developed for disposal/recycling of PPCPs.

Comments/Questions and Answers

Q – Confined animal feeding operations (CAFOs) use lots of antibiotics. Is there any information on altered microbiological communities downstream of hog farms and resistance to antibiotics?

A – U.S.D.A. has done a lot of research into this. There is no doubt some resistance – but are they pathogens that could affect people? There are some people who worked in animal operations who have died of resistant pathogens.

Q – Have we engaged Proctor & Gamble or the pharmaceutical industry, such as Johnson & Johnson, in cradle-to-grave stewardship programs?

A – The trade associations look at this. Most companies have their own stewardship program relative to production processes, but not the introduction of drugs into the environment by consumers. They may look at this somewhat, because they are concerned for other reasons.

Environmental Monitoring for Chemicals in Waters
Michael J. Focazio, U.S. Geological Survey

Since 1998, the U.S. Geological Survey (USGS) has been developing analytical capabilities to measure pharmaceuticals and other organic wastewater contaminants (OWCs) in a variety of environmental matrices. USGS' objectives were to develop sensitive and specific methods to measure trace organics at low levels and to evaluate environmental occurrence in "susceptible" waters.

Initial efforts to perform national reconnaissance studies for OWCs in water resources addressed streams (1999-2000, 139 sites in 30 states); drinking water sources (2001, 76 sites in 27 states, U.S. EPA); and groundwater (2000, 56 sites in 17 states). Data can't be compared except for specific compounds among the reconnaissance studies, however, because researchers developed analytical methods and added compounds as they moved forward. Ninety-five chemicals were initially targeted, including drugs, antibiotics, lotions, detergents, antioxidants, plastics, PAHs, fragrances, pesticides, fumigants, disinfectants, and fire retardants.

The stream monitoring studies focused on susceptible settings, including downstream of wastewater treatment plants (WWTPs), confined animal feeding operations (CAFOs), and a small number of undeveloped or natural settings. About half the sites were urban and half were rural, with 17 sites having mixed use and eight being minimally developed. The most commonly detected compounds were the naturally occurring sterols coprostanol, which was detected in 85.7% of the samples, and cholesterol, detected in 84.3% of the samples; N-N-diethyltoluamide or DEET, in 74.1% of the samples; and caffeine, in 70.6% of the samples. The chemicals found in the highest median concentrations weren't the most frequently detected chemicals. Triclosan, which was found in 57.6% of the samples, had a median concentration of 0.14 ppb, (the highest median concentration of any of the compounds), and the detergent 4-nonylphenol, which was detected in 50.6% of the samples, had a median concentration of 0.8% ppb (the next highest median concentration for non-naturally occurring chemicals).

For drinking water sources reconnaissance, similar chemicals were targeted, 124 pharmaceuticals and other OWCs, plus sterols (but no synthetic hormones). A total of 49 surface sites and 25 groundwater sites serving both large (e.g., 8 million) and small (e.g., 1,000) populations were sampled in 25 states and Puerto Rico. Results of the study are not yet published. Many of the chemicals were not detected and when they were detected, the frequency of detection was low due to source strength and the effects of treatment.

The most commonly detected chemicals were caffeine, at a frequency of 54% of all samples, and the herbicide metolachlor, in 43% of the samples. The insecticide diazinon was detected in 19% of the samples. An anti-convulsant drug, carbamazepine, was detected in 22% of the samples. There was a higher frequency of detections in surface water, including caffeine at 69%, metolachlor at 53%, and diazinon at 27%. The most frequently detected compounds were the

same as those in the combined surface water and groundwater results, though the order of frequency varied somewhat.

The frequency of detections in groundwater was significantly lower than in surface water. For example, caffeine was detected in only 24% of the samples and 1,7 dimethylxanthine (a caffeine degradate) in 16% of samples. Lower frequencies may be due to source strength or to absorption. Chemicals that appeared among the top 10 in frequency in the groundwater that differed from the top 10 detected in surface waters included the solvent tetrachloroethylene, in 24% of the samples, the plasticizer bisphenol A, in 20% of the samples, and the antibiotic sulfamethoxazole, in 20% of the samples. Besides diazinon, several other OWCs found are suspected endocrine disrupters, including ethanol 2-butoxy-phosphate and bisphenol A.

Preliminary results of source-water reconnaissance included the following findings:

- At least one OWC in 55% of samples
- Mixtures of OWCs common
- Concentrations generally low (<1 g/L)
- Comparison between groundwater and surface water sites showed fewer detections, different compounds detected, and lower concentrations in groundwater.

In 2000, a non-drinking water groundwater reconnaissance was performed at 47 sites in 18 states: 42 wells ranging from 2.4 to 310.9 meters deep (median depth 19.2 meters); 3 springs; and 2 sumps as part of a seepage monitoring system. Drainage water also was collected from buried perimeter tile lines surrounding earthen basins containing livestock waste.

Preliminary results showed OWCs detected at 98% of sites. Forty-five of the 82 OWCs were found at least once. The top four chemicals found were bisphenol A, N,N,-diethyltoluamide (DEET), Tri(2-chloroethyl) phosphate, and para-nonylphenol. Regarding detection of multiple OWCs, there was a median of 2 and as many as 19 found in a given sample.

Where are we going from here? Researchers must continue methods development, including for additional parents and degradates. Research is needed on the fate, transport, and effects of agricultural and human sources. The study of microbial aspects must be expanded. Input from stakeholders also helps to target sources.

In 2001, Iowa streams were sampled 3 times at 23 sites up stream and downstream of 10 cities. Four small towns were added to the third round (low-flow). At high flows fewer compounds were detected than at low flow, possibly due to a dilution effect.

Comments/Questions and Answers

Q – Can you determine sources by concentrating on the few areas that have many contaminants?

A – Missed answer.

Q – Did you look at sediments in streams or bio-solids in WWTPs?

A – We just did stream bed sediments. As expected, some compounds were found in high concentrations.

Q – Do you get sediments in your water samples because of unfavorable partitioning?

A – We tried to minimize those effects through the sampling methods we used.

Q – Can you identify sources when you publish your reports?

A – When we prepare such reports, we will say that there was a wastewater treatment plant nearby, but we don't name it.

Q – Did you look for any hormones that are used in the veterinary industry for animal production?

A – Yes. We looked specifically for some, but we looked primarily for a broad spectrum of compounds.

Q – Did you look at microbial breakdown of compounds?

A – At WWTP outfalls, but we did not look at this extensively. It may explain the white or nondetection areas on graphs.

Comment – If you took data on the frequency of detections plotted against the proximity to WWTP outfalls, maybe you would know if some plants are better at removing chemicals or dilution effects.

A – Iowa and Colorado are looking at this.

Comment – Look at the sources of wastewater going to the WWTPs.

Q – Could you look at the input into the POTWs and calculate what you'd expect to see in the outfall?

A – Yes, we're heading toward this. We have a list of compounds based on prescriptions and pharmaceuticals produced.

Veterinary Pharmaceuticals: Potential Environmental Impact and Treatment Strategies
John L. Cicmanec, U.S. EPA/ORD/NRMRL/Cincinnati

In the late 1970s, the increasing occurrence of antibiotic-resistant organisms was recognized in Europe and the U.S. The source was most likely the large-scale treatment of animals for infectious disease as well as for growth enhancement. More than 3,200 tons of antibiotics were used in 1998 for humans and animals in Europe and the U.S.

More concern arose when the topic of endocrine disrupters was introduced. In many farm operations, long-acting drugs such as trenbolone acetate and melengestrol acetate are routinely used to enhance weight gain in beef cattle and, unfortunately, these compounds persist in the environment. The public also is concerned about the use of bovine somatotropin to increase milk production in dairy cattle.

Data gathered in watersheds with large, confined animal feeding operations (CAFOs) showed that this way of doing business in American agriculture was having a significant environmental impact, and that new practices need to be developed. This presentation will define the problem and present some treatment or remedial strategies.

The veterinary group of compounds includes the following categories: antibiotics, anthelmintics (worming agents), hormones, steroids and bovine ST, pesticides, vaccines, and x-ray contrast media - iodine and barium-based. Potential endocrine disrupting chemicals include the following: trenbolone acetate, melengestrol acetate, estradiol benzoate, ractopamine, testosterone propionate, human chorionic gonadotropin, and pregnant mare serum.

Little is known about the fate of trenbolone and melengestrol after excretion by animals. The half-life for trenbolone when stored as liquid manure is 267 days. In farm field studies, trenbolone was detected at the end of the fertilization period, and melengestrol, which is even more persistent, was detected at harvest. Ractopamine is a beta adrenergic agonist. It partitions energy from fat growth to lean growth, increases protein accretion and muscle growth, and increases muscle fiber diameter.

Swine are confined in gestational buildings and in growth/finishing buildings throughout their lives, and related steroid hormones have been found in a study of human and farm animal waste. Swine exceeded human sources for estradiol five-fold (swine 152 ppb, compared to municipal sources at 31 ppb). For testosterone, municipal sources showed 29 ppb, compared to 21 ppb for swine. Estradiol was measured in cattle waste at 19 ppb.

While the antibiotic tylosin is used most in swine, others include: sulfonamides – sulfamethoxazole, etc.; quinolones – ciprofloxacin; beta-lactams – penicillins and cephalosporins; aminoglycosides – streptomycin and gentamycin; and macrolides – erythromycin and tylosin.

Europe uses more antibiotics for humans, 7,000 tons in 1998, compared to the U.S., 4,800 tons in 1997, but Europe's population is 390 million compared to 270 million in the U.S. Europe uses one-third as much antibiotic in farm animals, but has a much lower population than the U.S. – 19.8 million cattle in Europe (3,902 tons of antibiotic) compared to 97 million in the U.S. (15,100 tons of antibiotic).

In Europe the ratio is 7:4 in favor of humans because of relatively low animal populations. In the U.S. the ratio is 15:5 in favor of animals because of larger animal numbers. An adjustment for all animal populations was made for body size: four pigs = one cow; 100 chickens = 1 cow. In 1997, before controls, the annual use of antibiotics as growth promoters, not as treatment for microbial diseases, was 1599 tons in Europe. In 1999, after controls were introduced, the annual use dropped to 786 tons.

In Europe, only four antibiotics – monensin, avilamycin, flavomycin, and salinomycin are presently approved for animal use. Primarily the macrolides and sufonamides persist in the environment. Tetracyclines, penicillins, and fluoroquinolones rapidly degrade. Concentrations found were usually 1-5 ng/L but ranged up to 300 ng/L.

Tylosin is used as a feed-additive for swine. It is a macrolide antibiotic called Tylan made by Eli Lilly & Company, and is administered at 20 g/ton dry feed. Its mechanism and resistance is similar to lincosamide and streptogramin B type. One lagoon screening study showed tylosin tartrate at 10 mg/L. This compound is used to reduce the amount of methane, but methane could be harvested for electricity generation.

A study of resistance in E. coli in different species showed that swine operations had much more antibiotic resistence relative to humans and cattle. Geese also showed more antibiotic resistance than humans or cattle, and bacterial samples were positive from animal, bedding, and transport cages. Sixty percent of chickens have streptogramin resistant E.faecium (SREF). There is some geographical variation, but it was present at all sites tested.

For both Europe and the U.S., a study of resistance in retail meat showed that 30 to 60% of samples had streptogramin resistant SREF. This also was evident in pork and beef products. Among humans, 0.4 to 1% of people in the U.S. have SREF. Up to approximately 10% SREF was reported in humans in Europe.

Linking animal and human use- The animal drug virginiamycin, streptogramin (Group B), has been used for more than 26 years as a growth promoter at sub-therapeutic levels. In humans synercid, streptogramin – quinupristin/dalfopristin combination – was approved for therapy in the U.S. in 1999 as a "drug of last resort" to save lives when resistance has been developed to other drugs. This is a family of drugs that should work on resistant strains, but now we find that there are bugs that are resistant to it.

Antibiotics can have an adverse effect on some field crops. Chlorotetracycline, metronidazole, and sulfamethoxine are toxic to plants such as soybeans, poinsettia, and corn at concentrations of 10-160 ppm.

There are a number of processes to remove pharmaceuticals from water. The common ones include: sorption to particles, ultraviolet light, bacterial degradation, and reverse osmosis. WWTPs were not designed, however, to sterilize wastes or to remove pharmaceuticals. In some industries, such as printing, waste must be pre-treated, but hospitals are not required to do so. Pre-treatment also should be considered for large CAFOs.

Pharmaceuticals occur in U.S. wastewaters at levels of 61 g/L for acetominophen; 37 g/L for ibuprofen; 27 for g/L amoxicillin; 14 g/L for cephalexin. These compounds are used in both animals and humans. Sorption data for selected veterinary pharmaceuticals include tetracycline at –1.19 log Kow; cipro at 0.4 log Kow; avermectin at 3.19 log Kow; tylosin at 3.5 log Kow; sulfamethazine at 0.89 log Kow; metronidazole at 0.02 log Kow; and chloramphenicol at 1.14 log K_{ow}.

Ceftiofur (Naxcel R) is a new drug approved by the FDA for treating respiratory disease in cattle, with no withholding time for milk required. However, a bacillus stearothermophilus test will detect levels of 50 ppb in milk. But when used according to directions, none is detected in the milk. Flunixin (banamine) is used in horses to treat inflammation, pyrexia, and colic. It is a non-steroidal anti-inflammatory analgesic agent, and is approved for use in cattle to treat bovine respiratory disorder.

What can we learn from studies of human pharmaceuticals? The most persistent drugs are diazepam, carbamazepine, bezafibrate, and lopromide. Elimination of these drugs from wastewater depends primarily upon sludge retention time. For activated sludge systems, 4 –5 days are needed; for nitrogen removal, 8 – 20 days are needed.

Presently, antibiotic resistance and endocrine disrupting chemicals have been shown to have the greatest potential for causing environmental damage. The vast quantities of compounds used and their widespread distribution make veterinary pharmaceuticals a significant environmental challenge. Product control and distribution is more complex than for human pharmaceuticals.

Comments/Questions and Answers

Q – What is the likelihood that CAFOs can bear the cost of adding wastewater pretreatment?

A – We looked at the cost in Europe. It would cost more than twice the profits and put operators out of business. However, we came up with six wastewater treatment processes that, if scaled down, could be used. But there is no really cost effective method yet. In the Chesapeake Bay area (Maryland, Delaware, and Virginia), there are heavy concentrations of waste and not enough land for disposal, so wastewater pretreatment of animal waste is necessary. For poultry, composting and treatment is more cost effective.

Q – Are human and farm wastes sources of steroid hormones?

A – From raw waste, levels were found at 140 mg for cattle and 20 mg for humans. High doses cause death in humans.

Q – There are so many different chemicals. Should we focus on one group of pharmaceuticals over others?

A – Antibiotic resistance and endocrine disruptors should be the first emphasis, but there is danger in generalization, because pharmaceuticals can have different effects on different species. We have to balance against the quantities and where they are being used.

Q – Wouldn't it cost more to build a wastewater pretreatment plant for CAFOs than just not using antibiotics?

A – An economist would have to answer that.

Q – Regarding antibiotics in wastewater – what about the biosolids? Should we look at the effects of spreading them on the land? Are they looking for these types of compounds in biosolids?

A – Work is being done to look at sites where biosolids are being spread. Is there aerosol spreading? USGS has started studies, and these compounds should be included, but priorities may need to be set.

Q – Is anyone trying to balance the tons of antibiotics being fed to animals, metabolized, and released to the environment?

A – Someone must be looking at this. For example, sulfas are given in concentrations to saturate the body, so a lot would be excreted. Many medications get bound to molecules in the body. Efficient use in animals and shedding to the environment has not been studied yet.

Q – Are veterinary pharmaceuticals lower grade than human products? Is there a disposal protocol for ear implants?

A – You must dispose of the ears that have held implants very carefully, but there are no regulations over disposing of the waste – just the implants.

Comment – Although there are only a few drugs approved for use in Europe, a lot of other ones, bought on the black market, are also being used. The presence of these drugs comes up when testing is performed.

Assessing the Environmental Risk of Substances Under the US Food and Drug Act
Charles Eirkson, U.S. Food and Drug Administration (FDA)

In addition to looking at effects of substances on humans and animals, the FDA has been performing environmental assessment since 1975. The Food and Drug Administration's main authority comes from the Federal Food, Drug, and Cosmetics Act (FFDCA), which provides the legal authority for assuring the safety and effectiveness of drugs, food/feed additives, devices, biologics, and cosmetics.

The FDA, like all government agencies, also must comply with the National Environmental Policy Act (NEPA) and assess the environmental impacts of their "actions." Under this statute, the FDA performs reviews for assuring the safety of drugs and food additives to the environment. FDA regulations implementing NEPA appear in 21 CFR 25. These regulations were revised in 1997 under the ReGO Initiative.

Although the EPA's Toxic Substances Control Act (TSCA) doesn't look at environmental effects – and drugs are specifically exempted from pre-market approval under TSCA – the FDA does work on issues involving aquaculture and drugs used in aquaculture.

Under NEPA, the FDA considers the environmental impacts (use and disposal) from the federal "action" of approving a drug or food/feed application. In regard to veterinary products and

drugs, veterinarians do a lot of the review. The environmental review under NEPA incorporates environmental issues into FDA decision making.

It is important to note that NEPA does not require that the most environmentally beneficial course of action be taken. Instead, the FDA looks at alternatives, and what looks best for what one wants to do. Additionally, the FDA is required to comply with FFDCA requirements before NEPA – and if there is a conflict, FFDCA prevails. Thus, the FDA can't refuse to approve something just because of environmental impacts if it meets all other requirements.

There are some "categorical exclusions" under NEPA. Classes of actions that individually or cumulatively do not significantly affect the quality of the human environment are ordinarily excluded from the requirement to prepare an environmental assessment (EA) or an environmental impact statement (EIS). Categorical exclusions include investigational new (animal) drug applications (IND/INAD). These are excluded based on the small amount of the drug being produced for investigational purposes.

Another categorical exemption is approval of natural substances when approval does not significantly alter the distribution of the substance, its metabolites, or degradation products in the environment. It should be noted that in this case, the "natural substance" has to be natural for the area in which it is distributed (e.g., something that exists in Africa but not in the U.S. is not "natural" to the U.S.).

Examples of categorical exclusions include the following: approval of a drug when the approval will not increase the use of the drug; drugs for use in nonfood animals (because of the low volume of use); drugs for minor species, including wildlife and endangered species, when previously approved under similar animal management practices (e.g., drug used on cattle then used on deer); and therapeutics, under veterinarian order or prescription.

An environmental assessment is required for an excludable action if extraordinary circumstances indicate that the specific proposed action may significantly affect the quality of the environment. Examples include actions for which available data establish that, at the expected level of exposure, there is the potential for serious harm to the environment. FDA has used this on some parasiticides. They also may go back and take a new look at products if the company decides to make changes or a new application. EAs also are done if there are adverse effects to species/critical habitat/endangered species.

Non-excluded applications include the following: therapeutic drugs used to treat whole herds/flocks (e.g., anthelmintics: ivermectin); production drugs (e.g., subtherapeutic antibiotics/synthetic hormonal growth promotants); coccidiostats (anticoccidials and growth promotants: e.g., arsanilate); aquaculture drugs (FDA routinely assesses these); biotech products (including genetically modified organisms (GMOs): e.g., transgenic salmon) and use of wild plants/animals (harvesting). FDA frequently performs assessments on these last two categories.

The focus of the environmental assessments FDA performs is on environmental fate and ecotoxicology based on drug use pattern. The EAs are publicly available documents, however, they only become available after the product is approved under FFDCA. The FDA has also

prepared Environmental Impact Statements on selenium in animal feed; subtherapeutic antibacterial agents in animal feeds; taxol; and chlorofluorocarbon propellants.

The FDA uses basically the same standard approaches as EPA for its EAs; however, they have, in the past, sometimes included mitigations, such as labeling requirements or reducing dosages, within the EAs rather going to an EIS to do this. Putting mitigations in the EA can save them from having to do an EIS, but the FDA's General Counsel is disputing whether this is appropriate.

When the FDA looks at effects, the agency uses a tiered approach – acute toxicity testing is conducted first and then chronic toxicity if the data so indicate. Researchers also perform chronic toxicity testing if they see that a compound is particularly persistent. When testing human drugs, the FDA typically runs from 2 to 4 effects studies. They also look at introduction into the environment through wastewater. When testing animal drugs, the FDA typically performs 4 to 5 effects studies, including terrestrial studies because of the dung that animals generate. Special studies the FDA may undertake include antibiotic minimum inhibition concentrations (MIC), dung beetles, sediment toxity, avian toxicity, wash-off studies, and gene transfer.

The main emphasis in testing animal drug effects under NEPA is on ecology. Issues having to do with public health would be handled under FFDCA. Animal drug effects testing may involve the aquatic environment, using daphnia, algae, and fish; the terrestrial environment, involving plants (typically five species), microbial function, and earthworms; and pasture parasiticides, including dung insects (flies and beetles). EAs for human drugs normally focus on the aquatic environment.

Under the regulations, human drugs are categorically excluded if the expected introductory concentration (EIC) in the aquatic environment is less than 1 ppb (absent extraordinary circumstances). The data on human drugs routinely demonstrated, based on data for EAs done between 1987 – 1996, no effects at concentrations of less than 1 ppb. Approximately 90% of the toxicity effects occurred at levels at or above 1 ppm, while approximately 10% of the toxicity results were reported for levels between 1 ppb and 1 ppm.

Of those, approximately one-third were antibiotics and one-third were central nervous system drugs. Animal drugs do not have a categorical exclusion based on an EEC; however, less information can be provided if expected environmental concentrations (EECs) are less than 1 ppb at release from an aquaculture facility or less than 100 ppb in soil. One hundred ppb is below the level shown to have effects in studies conducted with earthworms, microorganisms, and plants.

Various sources of information are available through FDA's website. EAs on human drugs are available through FOIA requests. FOIA summaries also are available about how FDA made decisions on the safety of a product, how it is used, and its efficacy. Information about veterinary drugs is available through the FDA website, as well.

Two issues that FDA needs to look at are endocrine disrupting compounds and microbial interaction. Trenbolone and its metabolites are under investigation.

Comments/Questions and Answers

Q – If you have multiple sites using these drugs in water, do you look at cumulative risk?

A – Yes. Though if you have multiple sites using a chemical, it will still dilute to the same amount.

Q – What about additive effects?

A – These are almost impossible to consider. It's hard to look at effects in a single species and then extrapolate to the environment.

ENDOCRINE DISRUPTING POLLUTANTS IN EFFLUENT DOMINATED STREAMS

A Regional Case Study of Alkyl Phenol and Ethoxylates
Peter Howe, U.S. EPA Region 5

Approximately 500,000,000 pounds of alkylphenol is used each year in the U.S. These compounds are used in industrial, institutional, and domestic surfactants, as antioxidants in plastics, PVC stabilizers, oil additives, in oil field recovery, and in metal extractants. Alkylphenolpolyethoxylates (APEs) have degradation products that are extremely toxic to aquatic organisms (will probably be the regulatory end point), though not to humans or other animals. The degradation products from APEs also are responsible for endocrine disruption to aquatic organisms, though to date, the water quality standards have not addressed this endpoint.

The common environmental metabolites of nonylphenol ethoxylates include short chain ethoxylates that are very toxic and also are endocrine disruptors. The European Parliament has banned sale of nonylphenol (NP) and nonylphenol ethoxylates (NPE) to the general public and restricted industrial use to specific, authorized purposes based on toxicity, not endocrine disruption. Environment Canada has proposed that NP and ethoxylates be considered "toxic" under the Canadian Environmental Protection Act.

NP bioconcentration factors are: bluegill – 220; fathead minnow 271 and 293 as reported by Keith et al (2001). NP is not highly bioaccumulative, but is, however, very toxic. Depuration half-life (hours) are: gammerus pulex – 38; killifish – 9; mussels – 7; salmon – 96 as summarized in Gross-Sorokin et al (2003). The NP aquatic criteria document (draft March 2003) provides a freshwater chronic criterion of 5.9 ppb, and an acute criterion – 27.9 ppb, while the saltwater chronic criterion is 1.4 ppband the acute criterion is 6.7 ppb. These values do not, however, include toxicity of ethoxylates nor dothey address endocrine disruption. A study using Japanese killifish indicates that as ethylene oxide chains increase in length, toxicity decreases.

A Canadian risk assessment was based on the cumulative toxicity of all APEs and estimates the toxicity of NP1EO and NP2EO at half that of NP. Although this risk assessment recognized endocrine disruption, it was not the major regulatory end point. Apparently APEs are to be controlled through mandatory pollution prevention.

ORD has provided funds for the Region 5 Central Regional Laboratory to conduct acute toxicity testing on NP1EO and NP2EO to develop a TEQ for aquatic toxicity. At this time, the QAPP for this work is under development.

A study of British streams hypothesized endocrine disruption effects in fish outside the immediate discharge zone due primarily to 17 a-ethinylestradiol (EE2), a synthetic estrogen used in birth control, and alkylphenols. A major issue in regard to EE2 is how far downstream it persists.

Biomarkers for endocrine disruption in fish include decrease or cessation of egg production, imposex (oocytes in testes), and vitellogenin (VTG) – egg yoke protein formed primarily in female fish produced by the liver in response to estrogen or produced by male fish in response to exposure to estrogen or estrogen mimics. (If researchers find VTG in males, they know they are being exposed to estrogen). NP exposure is known to cause all of these effects, though researchers do not yet know the relevance of the effects on fish populations.

Findings of NP endocrine disruption in fathead minnows (Miles-Richardson et al [1999]) are that the NOAEL estimated from effects on endocrine disruption is six-fold less than standard chronic toxicity for the early life stage of the fathead minnow. There is a question as to biological relevance of endocrine disruption to fish populations, though, and this risk assessment does not include APEs other than NP.

Hormones are another major factor in endocrine disruption in effluent dominated streams. In one study, exposure to 17 B-estradiol (E2) to male fathead minnows showed a threshold for histologic changes in the testes equal to approximately 0.04 ppt. Additionally, the threshold for VTG induction also was equal to approximately 0.04 ppt. Testing of effluent concentrations of E2 at four Michigan sewage treatment plants showed an average concentration of 1.5 ppt (range = nd – 3.7 ppt). VTG induction in 14-day exposures to juvenile female rainbow trout indicate that EE2 is more potent than E2, which, in turn, is more potent than E1 (estrone).

Studies of male fish exposed to effluent from the Minneapolis Metro Plant show inconsistencies. Carp and walleye collected in the effluent showed VTG increase and testosterone decrease; however, goldfish exposed to effluent samples for 10 weeks exhibited no change in testosterone.

Walleye collected from the Metro Minneapolis effluent showed total APE concentrations in fish tissue at 6.2 ppm. Data gathered in 1995 on APE concentrations in the effluent of 15 paper mills along the Fox River (Wisconsin) ranged from a low of 0.03 ug/L to a high of 26.60 ug/L for nonylphenol and from a low of 1.28 ug/L to a high of 712.0 ug/L for total NPE.

Another study conducted in 1995 of concentrations of APEs from Publicly Owned Treatment Water (POTW) along the Fox River (Wisconsin) yielded a range from a low of 0.18 ug/L to a high of 15.9 ug/L for nonylphenol and a low of 8.8 ug/l to a high of 78.8 ug/l for total NPE. Fox River APE concentrations (ug/L) upstream of the Green Bay wastewater treatment plant showed NP at 0.58; NPE at 2.78; NP1EO 1.70; NP2EO 11.8 for a total concentration of 16.88 ug/L.

Because of concerns about the APE levels in the effluent of wastewater treatment plants, researchers looked at eight other large POTWs. They found little removal of alkylphenols in plants with only primary treatment (less than 10% removal), while plants with secondary treatment removed from 30% to almost 100%, and plants with tertiary displayed treatment removal rates were in the above 90%.

Effluent streams are very important in the Chicago area because the combined effluent from the major Chicago wastewater treatment plants is greater than the flow of most of the rivers in the region. Eighty percent of the water that flows through the Lockport lock and dam, for example, is effluent. The amount of effluent is influenced significantly by precipitation in the area, because much of the area is served by a combined sewer system (stormwater runs through the wastewater treatment system).

The Region 5 Central Regional Laboratory, working with ORD, developed a methods initiative proposal for APEs for water and sediment. Concentrations of NP and NPEs in carp from the North Branch of the Chicago River also have been tested.

Although water quality based effluent limits (WQBEL) control traditional toxics, there is a question of whether they facilitate recovery of large effluent-dominated streams. Cooperative whole effluent toxicity (WET) testing at the three major Metropolitan Water Reclamation District of Greater Chicago (MWRDGC) plants show no chronic toxicity (though all effluents were toxic 10 years ago). Toxicity testing of water collected at the Lockport lock and dam, which also used to be toxic, also shows no chronic toxicity. Despite these decreases in toxicity, these waterways still don't have good fish populations. Some of the factors that may contribute to the poor fish population include: combined sewer overflows; contaminated sediments; barge traffic; and habitat limitations.

Conclusions include the following: there is widespread APE occurrence in Region 5 water, sediments, and fish tissue; there appear to be some effluents that may exceed the proposed water quality criteria for NP; the Region 5 Central Research Laboratory has an SOP for NP, NP1EO, and NP2EO; Region 5 is developing TEQs for NP, NP1EO, and NP2EO to account for the toxicity of ethyxolates; consideration should be given to phasing out APEs in the U.S. due to the increased costs associated with removing them from POTW effluents.

Comments/Questions and Answers

Q – What about the Canadian study on nonylphenol in Atlantic Salmon?

A – Didn't have time to get into that.

Q – Have you heard about a plan to use NP as an emulsifier as a petroleum substitute?

A – Haven't heard of this.

Field Observations of the Contributions of Alkyl Phenols on Fish Endocrine Disruption

Cliff Rice, U.S. Department of Agriculture
Dr. Carys Mitchelmore, University of Maryland

Cliff Rice gave the presentation. Many chemicals mimic natural estrogen. For this study, they used nonylphenol ethoxylates (NPEs) as the token endocrine disrupting chemical (EDC). Numerous effects can occur that are detrimental to normal endocrine functioning, such as disrupting sex ratios, impairing fertility, infertility, and even changing behavior. There are often high levels of EDCs in rivers, due to industry, agriculture, and urbanization, though the EDCs resulting from agricultural pesticidal applications as extenders/wetting agents are unlikely point source problems in this river.

There are especially high levels of EDCs found in the discharges of some wastewater treatment plants, though some other plants provide treatment that removes or reduces many of these compounds. For the study, researchers used biomarkers to determine exposure to and effects of these chemicals, such as the presence of the female yolk protein, vitellogenin (VTG), in male fish, and changes in normal steroid profiles (estrogen (E2) and 11-ketotestosterone (11-KT).

The study site was the Cuyahoga River, which is famous for an oil slick that caught on fire in 1969. This incident played a critical role in the crafting of the 1972 Clean Water Act. The species sampled for the study was the common carp, a benthic bottom feeders, because the top predator fish haven't re-established in the river. The researchers looked for NPEs, since these are especially prevalent in wastewater treatment plant (WWTP) discharges and are known to have estrogenic effects on resident wildlife.

The aims and objectives of the study included the following: determining levels of nonylphenol ethoxylates in sediment, water, and carp tissues, including nonylphenol (NP), NP1EO, NP2EO, and total NPEs (tNPEs) (this was actually part of a larger study by the Ohio Environmental Protection Agency to determining whether these compounds were there); determining if there is a difference between levels of NPEs in pristine (headwaters) versus possibly affected sites (downstream); determining whether there is a correlation between levels of NPE and possible WWTP discharge sites; and determining whether there is a correlation between biological parameters and levels of NPEs, in particular in regard to measures of VTG, E2, and 11-KT.

There were seven sampling sites along a 74-mile stretch of the Cuyahoga River. Site 4 was near the confluence of the Little Cuyahoga River and several abandoned hazardous waste sites. For the study, 12 fish, half male and half female, were sampled at each site for levels of NPEs in their tissue. Additionally, general biological and health measures were determined for each fish, including length, weight (whole fish and gonad weight), gonadal somatic index, condition factor (weight divided by length), and percentage lipid. Endocrinological parameters measured included circulating steroid levels and VTG levels in the blood.

The lowest levels of total NPEs in the fish were found at site 1, which was the uppermost site along the river. Peak total NPE concentrations were found just downstream from the Akron WWTP (site 6). High levels were also found at sites 7 and 8, the farthest downstream of the

sampling locations. In all locations, similar levels of total NPEs were found in males and females.

Similar results were found for NP, NP1EO, and NP2EO, with the following differences: NP levels were highest in fish from sites 6 and 8, downstream from WWTPs, but there were highly variable concentration levels; NP1EO provided the greatest contribution to total NPE, though NP2EO was detected in the greatest concentration at site 6, with high levels also shown at sites 7 and 8.

In regard to biological parameters, a reduction of weight and length in the sampled fish, both male and female, was noted in progressing down the river. Although there was no difference in gonad weight in females as they moved downstream, there was a difference in males (bigger upstream). There were, however, no differences in GSI or percentage of lipid. Because NP, particularly, is lipophilic, researchers thought there would be a correlation, but there was not.

For males, there was no difference between the sites in levels of E2 or 11-KT, though there is a suggested difference in levels of VTG. Higher levels of VTG at site 6 correlates with the highest total NPE, NP1EO, and NP2EO downstream of the Akron WWTP, but NP was highest at site 8, rather than site 6, although this was not significant at $P > 0.05$, due to the low number of samples and variability. Overall, there were very poor correlations between NPEs and male VTG levels, and total NPEs, NP1EO, and NP2EO. There is also a poor correlation between NP and VTG levels.

To increase the sample size, researchers grouped the sampling sites into five zones. When they did this, they found that higher levels of VTG correlated with high NPEs downstream of the Akron WWTP.

In regard to female fish, there were no differences among the sites in levels of VTG or E2. There were significant differences in levels of 11-KT and the ratio of E2 /11-KT between sites, though there was no correlation/patterns with levels of NPEs, leading researchers to wonder about the role of other chemicals or factors.

The summary and conclusions include the following observations: Levels of NPEs are comparable to those reported in other studies for a moderately contaminated river; the highest NPEs are downstream of the Akron WWTP, except for NP – the highest levels of NP were at site 8; NPEs especially NP have been shown to affect fish endocrine parameters (e.g., elevated VTG in males – though this study showed a weak correlation – and changes in steroid hormones for males and females – though this wasn't seen in the study.); WWTPs also have been shown in other studies to affect the fish endocrine parameters; there was a clear estrogenic impact along the whole length of the river as all male fish have some VTG present; there was no obvious impact of NPEs on females; and more data are required, suggestions include increasing the sample size and sampling during a different season.

The percentage of contribution of plant discharge from the Akron WWTP to the Cuyahoga River for the sampling period was anywhere from 40 to 60% of the river flow at that location.

In another study of female carp in the Des Plaines River (Illinois), a reduction in VTG did appear to correlate with an increase in NP.

Comments/Questions and Answers

Q – In the last graph, it looks like all of the data is driven by one point.

A – Maybe.

Comment – You should call that point an outlier.

Q – Were the fish collected in the spring?

A – No. They were collected in July.

Comment – Female weights maybe affected by the discharge of eggs.

Q – Given that you were looking at effluent dominated streams and we know that NP is 100,000 times less potent than natural estrogens, did you look for natural hormones?

A – No. We didn't have the data or the funding for that.

Effects of Wastewater Treatment Effluents on Fish Endocrine Disruption
Larry Barber, U.S. Geological Survey

Determining chemical impacts in the environment requires an interdisciplinary approach involving hydrology, chemistry, and biology. Chemicals can enter waterways in either urban or rural settings through industrial discharges, publicly owned treatment works (POTW), combined sewer outfalls, landfills, agricultural activities, and animal production activities. Rural areas have sources of chemical pollution, including landfills, manufacturing, and agricultural activities. One confined animal feeding operation (CAFO) may produce the equivalent amount of waste as one to two million people. In areas where water is reused, we have to think about wastewater effluent.

The effects of endocrine disrupting chemicals can include ecosystem changes: population effects as reproductive output falls below the critical level; individual reproductive effects, as reproduction is impaired in individual animals; secondary behavioral and morphological effects (changes in characteristics affecting mating); primary molecular and biochemical responses (changes blood hormone levels, effects on the neuroendocrine system); and exposure to potential environmental agents. Researchers are just learning about secondary behavioral effects because it is hard to see changes in behavior.

Hormones are chemical regulators secreted by glands to the blood that effect a change at a target site. It is difficult to identify hormones in the environment – and even more difficult when dealing with congeners.

In regard to the aerobic degradation of surfactants (linear alkylbenzene sulfonate (LAS) and alkyl phenol ethoxylates (APEOs), as they degrade, they become less toxic and have a shorter half-life. These are very high production volume chemicals, however. Interestingly, researchers often find that concentrations of nonylphenols are higher in river water than in effluent.

The researchers conducted a study of the effects of environmental estrogens on reproduction in fathead minnows. Fathead minnows were a good subject for this test because paternal nest care is crucial for reproductive success; fatheads are a widely used fish model for toxicological studies; and they readily reproduce in the laboratory year round. Researchers performed a "flow through" experiment at the St. Paul, MN wastewater treatment plant (WWTP) in which 100% effluent was pumped through 15 tanks containing 100 fish for four weeks. One of the things they were looking for was competitive spawning behavior between exposed fish and control fish to see effects on population.

The control exposures were conducted at a separate facility in St. Cloud. The negative controls involved the use of old groundwater in some tanks and groundwater with XAD-8 treatment. The positive controls involved 17-b-estradiol and a mixture of AP compounds. The experiment was performed in July and replicated in October.

The XAD-8 effectively removes hydrophobic compounds and has a high affinity for phenolic moieties. The columns used were suitable for high flow rates and removed 50 to 95% of AP compounds. The researchers collected seven XAD-8 fractions, 100% water and 100% acetonitrile. Characterization was done with the GC/MS/, LC/MS/, FTIR, and NMR.

Researchers removed the gonads upon completion of competitive spawning. Different species have shown different effects as indicated by gonadal abnormalities reported in fish from many sites contaminated with environmental estrogens, including the Metro sewage treatment plant. Histopathology of reproductive tissues documents long-term ontogenetic disturbances in the endocrinology of the animal (in contrast to vitellogenin analysis).

A portion of the study considered competitive spawning. Only the nest-holding male will reproduce successfully (confirmed through DNA fingerprinting). Failure to defend the nest site for the entire egg development period will result in loss of the brood. The endpoint was the number of observations (two per day, AM and PM) during which the nest was held.

The preliminary results of the study are as follows: no intersex development was found in any fathead minnow exposed in the study. VTG induction was observed in positive controls and effluent. GSI and HIS appear to be regulated by temperature. There are seasonal effects of VTG induction, probably because of changes in the wastewater treatment regime. XAD-8 partially removes estrogenic effect.

Comments/Questions and Answers

Q – Which male fish won the competitive spawning?

A – The "control" (non-exposed) fish had an advantage and typically won.

Aquatic Toxicity, Estrogenicity and Treatability of Nonyl Phenol and Ethoxylates in Waste Water Effluents

Charles Staples, Assessment Technologies, Inc.

Nonylphenol ethoxylate (NPE) surfactants are widely used in applications that are sent to sewage treatment plants when spent. While NPEs are generally easily treatable in well-run treatment plants, residues from biodegradation are often detectable in effluents. It is of interest to examine the potential risks to the environment from these residues. Risk assessment is the preferred means of addressing these compounds.

Risk assessment requires exposure and ecotoxicity data. The results of risk assessment are used by risk managers to control and manage potential unwanted risks. The purpose of the researchers' study was to examine the ecotoxicity database, including endocrine-modulating effects, for the NPEs, NPEC and NP, that are most common in effluents as shown in wastewater treatability studies.

In an example of a treatability study for NPE surfactants, the effluent from two Canadian sewage treatment plants, both of which receive municipal and industrial wastes was analyzed. Plant A used a process involving activated sludge, nitrification, tertiary treatment, and UV disinfection. Effluent from this plant showed a removal efficiency for NPE of 97%. Plant B had a non-nitrifying activated sludge process and chlorine disinfection. The removal efficiency for NPEs was 86%.

According to earlier studies, the main NPE related products in raw sewage are commercial NPE, while the main components in final effluents were NPEC with some NPE1,2 and traces of NP. Risk assessments of NPE residues from sewage treatment plants in surface water should therefore encompass NPEs, NPEC, and NP.

The researchers performed a critical review of chronic aquatic toxicity data for NP and NPE. This included dozens of studies, endpoints, LOEC, and NOEC and considered survival, growth, and reproduction. The species sensitivity analysis shows that the data support the draft U.S. EPA WQC for NP of approximately 5.9 ug/L. In addition to these studies, the researchers looked at a study in which medaka fish were exposed from 1 to 100 days post-hatch to NP, NPE1, NPE4, NPE9, and NPEC1 and examined for sex ratio, secondary sex characteristics, liver VTG, and testis-ova. This study showed effects in the Testis-ova and in the secondary sex characteristics in the fish exposed to NP and effects in secondary sex characteristics in the fish exposed to NP1EO.
The conclusions from the medaka study are that NP induced gonadal intersex and mixed secondary characteristics in male medaka at 20 and 100 ug/L. Mixed secondary sex characteristics were induced in males at 300 ug/L NPE1. However, there was no evidence of estrogenicity with NPE4, NPE9, or NPEC up to 1000 or 3000 ug/L and no effect on sex ratio for any compound.

The ecotoxicity studies show increasing toxicity with decreasing ethoxylation in both chronic and acute studies. Ecotoxicity studies of NPECs show them to be relatively non-toxic. Only NP showed estrogenic potential in a long-term developmental study with medaka. Thus, the

available database of conventional chronic studies should be adequate or nearly adequate to address the ecotoxicity of the various biodegradation intermediates of NPE surfactants.

Treatability studies show that well-run sewage treatment plants are effective at removing NPE surfactants, but residues do enter receiving waters. Risk assessments of surface waters receiving sewage treatment plant effluents should address all biodegradation intermediates. Risk management to reduce risk from natural hormones in human waste simultaneously addresses NPE, NP, and NPEC. Risk management to reduce risk from industrial sources should include best management practices.

Comments/Questions and Answers

Comment – It's not a matter of how well run a wastewater treatment plant is if it doesn't have the equipment to remove NPEs. A plant may be well run, but if it doesn't have the equipment, it can only do so much.

A – Agreed.

Q – Some data suggest that NPE becomes NP downstream, so we need to test for that.

A – They don't break down to NP except in anaerobic conditions.

Q – What is the toxicity of ether carboxylates?

A – They aren't particularly toxic.

Q – Is NPE still being considered for use in petroleum applications?

A – The University of Massachusetts did some research in this area a few years ago. Also, there was an application to the State of Florida at that time but I believe that project was abandoned.

Break Out Sessions

Break out sessions were held to discuss the following topics:

- EDC and Effluent Dominated Streams
- Asbestos and Durable Fibers
- Pharmaceuticals and Personal Care Products

Flip chart and discussion notes from these break out sessions can be found in Appendix D of this document.

Thursday, August 14, 2003

Objectives of the Final Session
Dan Hopkins, Toxics Reduction Manager, U.S. EPA Region 5

There are still many unanswered questions in regard to emerging pollutants. Some of the topics we will be discussing today aren't just emerging pollutants or issues, they are emerging science. How do we identify emerging chemicals and how do we get them to "stick" on our radar screen? We need to identify these issues, but we also have to make sure they are pursued.

Biotechnology: A New Frontier, The Promise, Potential Risks, and How Can We Find Out?
John A. Glaser, U.S. EPA/ORD/NRMRL – Cincinnati

Biotechnology has much to contribute to a sustainable environment. There has, however, been significant suspicion regarding biotechnology, most recently in Europe. EPA and other agencies in the U.S. have the mission and the responsibility to assess correctly biotechnological inventions for their suitability and acceptability for a sustainable environment and to understand how these inventions fit into our commerce and our environment.

The Promise

Biotechnology promises new products and useful processes that may permit the use of biomass to serve the demands of society. These include the development of agronomic traits, one of the first of these type of products to be developed, as well as products for the feed processing, chemical, and health care industries. We still rely very heavily on microbial activity to make the products we are interested in or can use. Now we are looking at using enzymes engineered from microorganisms found in nature to make a wide variety of products, from pharmaceuticals to chemicals. These advances hold much promise for the future.

Products made using biotechnologies include Nitto's polymer-grade acrylamide, Lonza's carnitine, and Du Pont's key monomer for it's highest performing polyester. The entire high fructose corn syrup industry was created by enzymes. Hercules' G3 is a product of biotransformation. Researchers are studying a polymer referred to as biosteel that is made from the filaments of spiders. The promise of future useful technology lies in areas such as phytoremediation, biobased conversion technology, agricultural biotech, molecular farming, and analytical biotech.

With phytoremediation, plants offer unique means to move chemicals in the contaminated soil phase. Plants also offer a unique means to sequester organic and inorganic pollutants. And plants offer unique opportunities to degrade organic pollutants in cooperation with soil microbiota. Through the transpiration of liquids, plants can be used for their ability to pump subsurface contaminated water. The contaminants in such a scenario can be volatilized from water and possibly metabolized by the plant. Biodegradation products in the root zone of trees

could be utilized, as well as the oxygen that trees give off in the root zone. Planted areas could be useful in wetland filtering.

With biobased conversion technology, biofuels, such as bioethanol and biodiesel fuels, can be made by the microbial fermentation of sugars from a variety of natural sources and crops. Large-scale development is underway, with batch conversion using one-million-gallon reactors making the process commercially viable. This should lead to the use of bioethanol as a major fuel source. With increasing production capacity of reactors, however, waste stream management also will be important and challenging.

Bioethanol conversion is mainly based on cellulose hydrolysis for more complete utilization of the available carbon in the source material. Hemicellulose and cellulose hydrolysis require different microbial action. Bioethanol has been used as a replacement for MTBE. Two million gallons were produced in 1998, and this amount is expected to increase to 4 billion gallons in 2010 and 9.5 billion gallons in 2020. Current production is around 1 billion gallons.

For biodiesel, rapeseed oil is used due to its high quality. Triglycerides are broken up to release fatty acids, which are separated and converted to esters. U.S. production of biodiesel in 2002 was 15 million gallons. In February 2003, the largest biodiesel plant came on line in California with a production capacity of 35 million gallons per year. A cooking-oil-to-diesel recycle/reuse program is being evaluated in several locations.

Cargill-Dow is using corn for starch and sugar fermentation to produce lactic acid, which is polymerized to polylactic acid (PLA) at a production target of about 300 million pounds per year. About 40,000 bushels of corn per day will be used in this process. Further use of agricultural biotech lies in molecular farming for commodity and specialty chemicals and pharmaceuticals. An application for crop protection includes herbicidal control and Plant Incorporated Protectants (PIP).

Why should we use plant biotechnology? Traditional breeding depends on inherent genetic traits that occur within an interbreeding population. Specific desired traits, such as those that enhance production, may not exist within a particular population. Mutation breeding is inefficient and cannot be targeted to specific traits. Novel traits can be introduced from other species to produce new or improved end products and can enable environmentally beneficial agricultural practices. The ability to use environmentally beneficial agricultural practices is an objective for these new biotechnology ventures, though it is not achieved in all cases.

Crop engineering might take a desired biological pesticide, for example, and work it into a corn cell yielding a newly transformed corn plant. DNA from microbes in the soil expressing useful pesticide traits might be used in the crop itself to give protection and this is the case with Bacillus thuriengensis. Selection of potential crop traits focuses on those that affect yield or efficiency, or that lower production costs, rather than the properties of the commodity produced. Output traits are modifications that affect the composition, quality, or value of the commodity rather than the ability to produce the crop.

There are a variety of ways to put modifications into plants, such as using male sterility or, for example, putting modifications into plants such as trees with long life spans so that the trait isn't

exchanged in nature. Other inducible or delayed-action modifications can be made in seed composition, the timing of trait expression, tissue localization of expression, or the removal of unwanted genes.

Molecular farming involves crops that aren't used for food, feed, or fiber but for plant-made chemicals and pharmaceuticals. Modified plants offer new and possibly more useful ways to produce valuable molecules and therapeutic agents. Plants make proteins in the same way that humans do, so we can use plants to put proteins together.

The development of human protein therapies has significantly increased from 1982 to the present. The driving force is the ability to produce crops for pharmaceutical uses that don't require fermentation for processing. Pharmaceutical crop developers include Dow, Monsanto, and Prodigene. Plants figure very strongly in these production systems. The focus of work today for modifications in the pharma crop area is corn, other monocot and dicot grains, forage crops, and tobacco.

Herbicide tolerance can be built into plants, such as in Monsanto's Roundup Ready Crops. Herbicides target specific enzymes or processes in the plant metabolism. Engineering herbicidal resistance into plants involves modifying the target enzyme or introducing an enzyme that detoxifies the herbicide. The introduced enzyme can be obtained from bacteria, fungi, or other plants. Rather than using a broad-spectrum insecticide that would kill all insects, including those that are beneficial, the DNA coding for crystalline bodies (Cry) protein from bacteria can be installed in a plant to engineer crop resistance to specific insects that affect the crop yield and health.

Analytical biotech can be used to detect pathogens and toxin expression in Plant Incorporated Protectants (PIPs) crops, and offer opportunities to replace some animal testing with genomic and proteomic approaches. Structural genomics looks at information at the DNA level. Protein engineering can be used with metabolic engineering and other forms of functional genomics to enhance understanding of biological activities at the functional level. Using simple techniques, we can amplify DNA in samples to get to useable material in a shorter time frame for analysis that will lead to more information on microbial species.

Potential Risks

The agency plays a strong role in PIP Bt crops to manage the potential risks associated with them. Not all PIP crops will be required to implement integrated resistance management (IRM) requirements. Bt crops are selected for IRM when there is a compelling need to preserve the toxin, because other growers may be using it in spray form, and there is a need to protect its efficacy against pests.

Around 140 million acres of genetically modified (GM) crops are produced annually worldwide. EPA registers all pesticides, including new substances and DNA in the plant when it is pesticidal in nature, under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). EPA must ensure that pesticides will not pose unreasonable risk to human health or the environment. For PIPs, EPA considers risk to human health, risks to nontarget organisms and the environment, the potential for gene flow, and impact to the Insect Resistance/Management Plan.

Major recommendations for continued research from consultative bodies such as the National Academy of Sciences include assessment of the following: allergenicity from GM Foods; the possibility for gene transfer; the ecological risks associated with GM crops; gene transfer; and resistance management. EPA has initiated a Biotechnology Program through ORD, which received funding in 2003 and is strongly supported by and coordinated with the Office of Pesticide Programs.

Food allergies occur in about 5% of children and about 1-2% of adults. Novel proteins introduced into food could be allergens. EPA's ORD research program is pursuing a rodent model that is needed to evaluate the allergenicity of GM crops. Researchers also must address the potential ecological risks related to GM crops regarding the likelihood and impacts of gene transfer, impacts on non-target species, such as the monarch butterfly, and determine/manage pesticide resistance.

Because resistance evolves in insects, EPA requires resistance management plans to prolong the usefulness of Bt crops and avoid the need for broad-spectrum pesticides. Bt spray used in organic farming may be rendered ineffective if insect resistance becomes significant. EPA requires implementation of a "high dose, structured refuge" strategy for resistance management.

How Can We Find Out?
Remote Sensing in Agriculture

One example of research leading to new information is the use of crop spectral features to discern crop conditions as related to pest infestation. This research involves use the remote sensing of bioengineered crops to monitor crop status, nutrient and pesticide management applications, and landscape characteristics. The current Bt corn crop is about 20 million acres. There is no simple and economic means to gain access to sufficient discrete samples that would inform us about the development of pest resistance for Bt corn on a statistical basis. Remote sensing has been used to show different irrigation applications and levels of stress. This same technology could be applied to developing monitoring information about pest resistance. When considering monitoring of pests to determine whether any have developed resistance, it is necessary to assay baseline susceptibility levels for resistance monitoring, changes in the frequencies of resistance alleles, the presence of any dominant alleles related to resistance, and effects of resistant insects. Such information is important to dealing with resistance failures.

The current monitoring strategies' use of limited sections of a corn crop as proxies for the entire crop may not provide adequate information for resistance detection. Infestations of corn pests are expected to begin as local phenomena and may not be adequately identified. Remote sensing can be used to check grower compliance with EPA's resistance management requirements. As an early warning system to strengthen resistance monitoring strategies, remote sensing is based on stress inference, and for detection of pest infestation as part of resistance monitoring of the corn.

Airborne and satellite imagery can detect plant pigments and plant stress, and can stratify images and field surface conditions. Infrared imagery may be able to distinguish between Bt corn and conventional corn crops. EPA can use this information to organize data for different fields and

determine whether management plans are being carried out. For ground truthing in Nebraska and Pennsylvania, remote sensing has been used to monitor the activities of insects that are released to determine the efficacy of the Bt corn.

Infrared spectral wavelengths are potentially useful for detecting a bioengineered crop and obtaining the spectral signature crops, but because the signal is a composite of many factors, it needs to be deconvoluted to make the information useful. The signal-to-noise ratio is important to define detection constraints. Information needs to assist the use of remote sensing to transgenic crop monitoring include compliance, inference of resistance, and resistance detection.

Comments/Questions and Answers

Comment – The use of new genomic technology won't replace animal testing, but could reduce it.

Environmental Futures: Nano Technology and Genomics Coming Over the Horizon
Gerardo (Pasky) Pascual, ORD/Office of Science Policy, Environmental Futures Workgroup

Under Goal 5 of U.S. EPA's ORD Research and Strategy Plan, the agency will evaluate opportunities for and, as appropriate, conduct research to anticipate and assess future environmental stressors before they adversely affect people or the environment. In 1995, EPA's Science Advisory Board called for an EPA-led effort to anticipate and respond to environmental change and to provide the public with an understanding of risks and opportunities, both environmental and economic, and to better define actions needed today to reduce risks and preserve opportunities.

EPA is building the capacity to develop a "foresight" effort focused on potential issues 10 to 20 years into the future. Terrorist attacks in recent years have added urgency to this effort so that the hostile use or release of hazardous chemicals or the disruption of environmental systems can be avoided. EPA endeavors to interpret the future by scanning weak signals, scoping strong signals and developing foresight tools.

By reading scientific journals, researchers can scan weak signals to identify leads in the domains of conflict and governance, science and technology, agriculture and food security, natural resources and environment, and energy use. Experts then rank the leads by their novelty, scope and severity.

An example of looking at a "weak signal" and using the information to anticipate or prevent future problems involves work done by Massoud Amin of the Electric Power Research Institute, in which he examined the cause of a four-state power outage that occurred in 1996. Amin determined that the outage could have been prevented by shedding 0.4% of the load for 30 minutes.

He then designed a ubiquitous, real-time sensor system that would detect potential problems with the transmission system before a major outage so it could be prevented. According to Wired, 2001, Amin's design would have the following effects: "Every node in the power network of the future will be awake, responsive, adaptive, price-smart, eco-sensitive, real-time, flexible, humming – and interconnected with everything else."

The Woodrow Wilson Foresight and Governance Project brings in experts to run symposia on emerging and future issues. In one symposium, participants considered the issue of toxicogenomic testing. A presenter at the symposium made the points that if individuals become capable of detecting their own genetic susceptibilities, this may be the lowest cost risk avoidance mechanism, because it would allow individualized decisions on whether a person would use specific products rather than relying on government regulations designed to protect the entire population when only a few individuals are susceptible.

The concluding question by this symposium presenter was what criteria should be used to shift primary responsibility for avoiding/preventing risks from the manufacturer to individuals? At this point, some substances, for example, are simply labeled so that people who have certain sensitivities will know to avoid them (or can make their own choice about avoiding them).

At this point, there are two impediments to using toxicogenomic testing. One is technological – results of testing may differ depending on how the lab performs the test, so there are efforts afoot to standardize toxicogenomic testing. The second is who will drive the demand for this technology? Pharma companies have basically withdrawn at this point because the small number of people who would be served may not make it economically attractive.

Some demand may be driven by lawsuits. In one case, employees of a company are suing, saying that the company should have screened them for susceptibility to certain substances.

Nanotechnology, a setting in which materials are manipulated at the molecular level, is already here, though some researchers weren't initially thinking in terms of potential ecological or health effects of using this technology. Some products have already been introduced. ORD has given out research grants supporting "green" applications of nanotechnology. For example, Clemson University is working on improving the wavelength to which photocatalysts respond.

The University of Miami is developing nanoscale monitoring of marine toxins through artificial receptor sites that glow when toxins are present. Rice University, which has been very involved in nanotechnology research, is focusing on nanotechnology and its effects on the environment. A recently released request for proposal is soliciting projects into the human effects of nanoparticles.

The state of Pennsylvania is developing foresight tools to determine where future vulnerabilities lie – and the extent to which an ecosystem might be harmed by stressors. Researchers are looking into a system's sensitivity and adaptability, its exposure to stress, and society's valuation of the system. Because vulnerability is something that varies over time and space, the model used to make such assessments must be dynamic.

For more information, the web site address is www.epa.gov/osp/efuture.htm or contact Anita Street at EPA, phone 202/564-3626.

Comments/Questions and Answers

Q – How much money is involved in the STAR program for emerging issues?

A – It varies from year to year. It started with an open-ended RFP for $7 million, but it wasn't successful. Last year the focus was on 13 nanotech studies for $6 million. This year it is down to $5 million. Contact Barbara Carn to find out more.

Q – Regarding how to track Bt resistance and how it spreads to other plants, would biofluorescence placed on gene trackers work?

A – We are looking for signals that are not affected by background noise. We need to distinguish between a Bt crop and a conventional crop. Once we develop a signature for pest infestation near a Bt crop, then we could go to the field for more information. Because we look at 20 million acres a year, we can look at the entire crop.

Pest infestation usually occurs where there is stress, which may indicate where to focus sampling. We use a hierarchical approach with remote sensing. We are willing to have a fraction of false positives and we can still address the problem early. What we don't want are false negatives. What we are looking for is to address problems early on.

Q – Is there anything in the current futures program to bridge the gap between the goals of identifying future problems and focusing the agency on preventive actions?

A – Some of the actions I have described are preventive in nature, such as toxicogenomics. Also, at Rice University in the fall 2001 a researcher spoke on developing greener ways of doing things using nanotechnology.

She was then working with engineers and not with public health officials, so we asked her to build bridges between the two in order to identify human and environmental effects. This spring her presentation was on looking at human health effects, so awareness is building.

Q – Regarding the workers who wanted a company to do gene testing. Years ago enzyme testing for coal miners was proposed but unions were against it because they wanted management to take the responsibility for providing protection for workers, so jobs wouldn't be endangered. Women also don't want to be denied jobs because of concerns about being exposed to chemicals known for fetal toxicity. How would that play out in this situation?

A – Lawsuits are voluntary acts of employees. The employees have taken it on themselves to ask for genetic testing. Do we slip down that slope where employers use or require tests to screen people out? A percentage of the funds in the Human Genome Project is being used to look at such ethical, legal, and socio-economic issues.

Wrap-Up Roundtable of Speakers
Dan Hopkins, Toxic Reduction Team Manager, U.S. EPA Region 5

The session was devoted to discussion of how EPA can improve its focus on emerging pollutants and how researchers can put these issues on the Agency's radar screen and keep them there to ensure that they are resolved. EPA's charge is to protect the environment and human health, so it must respond to threats in a timely fashion. The session is intended to explore ways of bringing good ideas to EPA decision-makers and making sure they are addressed. Questions posed to the workshop participants included the following:

Question 1 – Processes for identifying chemicals for focus. Why is it important to address emerging chemical issues?

Question 2 –Are there realistic opportunities to address the data gaps?

Question 3 –What would you suggest to improve the way we identify chemicals that might pose threats?

Flip chart and discussion notes from this session can be found in Appendix D of this document.

Feedback From Environmental Journalist – An Outsider's Perceptions
Janet Raloff, Science News

Raloff is familiar with all of the topics that were discussed and has covered all of them except for thallium. Asbestos is not an emerging pollutant, but a resurfacing one, she said. Pollutants have been building up for decades, so Raloff would call them newly recognized concerns, quietly coming up on the radar screen

Raloff commented that she likes to cover newly emerging issues, with risks that are still fairly indeterminate, because she can extrapolate to everything else – can say the issue is a potential problem for all of mankind and that it deserves everyone's attention. Once more data is available, the issue becomes a lot more circumscribed.

She doesn't dwell on what we don't know. She focuses on the known and asks players in the field to speculate on what new data might be and what concerns it might raise. Raloff says science goes at a glacial pace. She can't write on scientists "mulling over possible findings." It takes five years to get a paper out on new data, she added. Her readers want her to cut to the chase, so she takes a snapshot of what's happening now. She uses lots of qualifiers, such as "suggests," or "hints at." This allows her to avoid caveats.

News isn't what's expected. It's what's new, different, unexpected, or superlative. Most science stories don't have this, so you have to find an angle for them. What is newsworthy to her is what is newsworthy to politicians and bosses.

We worry about pollutants because they pose risk. You have to show why that is important.

Raloff advised that to elevate managers' interest in issues, EPA staff must make thumbnail descriptions or concerns to grab attention and compel action. Raloff had the following advice on how one can compel people:

- Tell people what the risk is and say attention to the problem can't wait. "If you're on the fence, no one will listen."
- Project authority and confidence to sell an idea. Say it's important with conviction. Raloff said most federal agency people don't do this – they say they have data. She wants – and other people want – information. Informed, knowledgeable analysis. Not just data.
- Don't worry about data gaps. Focus on information with knowledge applied to gain perspective and analysis.
- Be proactive by publishing a lot. This increases visibility and credibility. She advises researchers to try to publish in first-tier, peer-reviewed journals that will be seen by the right people.
- Contact the media to raise the issue. "We need to know about it before a paper comes out. You can even do this for papers that aren't your own if you believe the media should know about an issue."
- Tell reporters in one paragraph why it's important.

Raloff says she can give play and exposure to an issue in the100 countries in which the magazine circulates. Go to Newsweek and Time magazines, "because you are fighting against entrenched bureaucrats for a share of money. Enlist grassroots support."

To raise concern about estrogenic effects in 1993, Theo Colborn pulled together all the research on the topic and went on the lecture circuit. Then Theo Colborn, John Peterson Myers, and Dianne Dumanoski wrote Our Stolen Future. This got the issue out to the public and the media. This caused enough people to be worried that they wanted research done. EPA started throwing money at the issue because pressure to do so came from Congress.

In less than eight years, the people who were championing this issue created a whole new area of toxicology, even though they originally had no money. "It's a savvy approach to enlist the media to carry an issue. You use people outside the Agency to affect the way things are done inside the Agency," she said. Sometimes the new papers on emerging issues are coming only from Europe.

"I can talk about the issues agency people have been told not to talk about," Raloff said. "I am very disappointed in EPA in Washington, D.C. The gatekeepers try to keep us from talking with researchers. It's not as big a problem outside Washington, but people are scared to talk with the media."

Endnotes

[i] T. S. Lin et al. 2001. "Thallium Concentration in Lake Trout from Lake Michigan." *Bull. Environ. Contam. Toxicology*, 67, 921-925.

[ii] James C. Ely et al. 2001. "Implications of Platinum-Group Element Accumulation along U.S. Roads from Catalytic-Converter Attrition." *Environ. Sci. Technol.*, 35, 3816-3822

[iii] D. Cinti et al. 2002. "Platinum levels in natural and urban soils from Rome and Latium (italy): significance for pollution by automobile catalytic converter." *Sci. of the Total Env.,* 293, 47-57

[iv] Ignza J. Buerge et al. 2003. "Caffeine, an anthropogenic marker of wastewater contamination of surface waters." *Environ. Sci. Technol.*, 37, 691-700

[v] Available at: http://ntp-server.niehs.nig.gov/htdocs/Chem_H&S/NTP_Chem5/Radian58-08-2.html.

[vi] Available at: http://www.maripoinsoncenter.com/ctr/9411caffeine.html.

APPENDIX A: AGENDA

DATE: AUGUST 11 – 14, 2003
LOCATION: REGION 5 OFFICES, CHICAGO, IL

Monday, August 11, 2003

10:00 am ***Registration Opens***

1:00 pm **Welcome:** Bharat Mathur, Deputy Regional Administrator
U.S. EPA Region 5

ORD/Regional Workshops:
Paul Gilman, Assistant Administrator
Office of Research and Development
U.S. EPA Agency Science Advisor

1:20 pm **Introduction to this Emerging Pollutants Workshop:**
David Macarus, U.S. EPA Region 5
Regional Science Liaison to ORD
Chair, Workshop Planning Group

Moderator for the Day: Brad Schultz, U.S. EPA Region 5

1:30 pm **Case Study – Triclosan: One Example of How Emerging Pollutants Come to Our Attention:** Steve Reimer, U.S. EPA Region 10

Perfluoro Octane Compounds in the Environment:

1:45 pm	PFOS Toxicity Studies	Christopher Lau, U.S. EPA/ORD/RTP
2:15 pm	PFOA Toxicity Studies	John Cicmanec, U.S. EPA/ORD/NRMRL- Cincinnati
2:40 pm	Risk Information Update	Jennifer Seed, U.S. EPA/OPPTS
2:55 pm	Program Office Update	Mary F. Dominiak, U.S. EPA/OPPTS

Questions from audience

3:10 pm ***Break***

Toxic Chemicals Resulting From the Disposal of Electronic Equipment:

3:20 pm	Potential to Leach	Timothy Townsend, University of Florida
3:50 pm	Combustion Issues	Eric Stewart, ORD/NRMRL/RTP

4:20 pm **Round Table:** Timothy Townsend, Eric Stewart, Jason Swift
U.S. EPA, Region 5 RCRA Program

1. Risks quantifiable?
2. Research needs?
3. Risk management options?

4:40 pm ***Review of Tuesday's Program***

4:45 pm ***Adjourn for the day***

Tuesday August 12, 2003

Brominated Flame Retardants:

8:30 am **Welcome and Introduction:**
Edwin (Ted) Smith, U.S. EPA/GLNPO

8:45 am **Overview of BFRs:**
Leif Magnuson, P2 Coordinator, U.S. EPA Region 9

Environmental Presence and Known Sources:

9:15 am Human Health
Ron Hites, Indiana University

9:45 am Environmental
Mehran Alaee, Environment Canada

10:15 am ***Break***

10:30 am **Toxicology/Risk Analysis Status:**
Linda Birnbaum, U.S. EPA/ORD/NHEERL

11:00 am **Status of the VCCEP: Consultation re penta, octa, and decabromo diphenyl ethers:**
Jennifer Seed, U.S. EPA/OPPT

11:05 am **Introduction to Afternoon Roundtable Discussion:**
Leif Magnuson, U.S. EPA Region 9

11:15 am **Round Table Discussion with BFR Panel**

12:15 pm ***Lunch***

Afternoon Moderator: Michael Mikulka, U.S. EPA Region 5

1:30 pm **Bisphenol A and Phthalate Esters: Potential Sources of Resin Compounds in the Everyday Environments of Children:**
Marsha Morgan, U.S. EPA/ORD/RTP/NERL

2:15 pm **Radium in Oil and Gas Piping and Production Facilities:**
Loren Setlow, U.S. EPA Office of Air and Radiation

2:45 pm ***Break***

3:00 pm **Exposures to Thallium and to Platinum Group Metals:**
Frank Anscombe, U.S. EPA Region 5

3:20 pm **Break Out Groups Meet:** Additional Suggestions on Where We Go From Here
Group A – PFOS/PFOA and BFRs
Group B – Bis-A and Phthalates
Group C – Electronics Disposal
Group D – Radium, Platinum, and Thallium

2:15 pm **Break Out Groups Report Out**

4:45 pm ***Adjourn for the day***

6:00 pm *Meet at local restaurant for refreshments, dinner, networking & relaxed conversation.*

Wednesday August 13, 2003

Moderator: Robert Pepin, U.S. EPA Region 5

8:30 am **Asbestos and Related Durable Fibers: Too Ubiquitous, Too Persistent, Too Complex to Put Health Risks to Rest?**
Phillip Cook, U.S. EPA/ORD/NHEERL/Duluth, MN

9:30 am **Pharmaceuticals and Personal Care Products in the Environment, Introduction and Overview:**
Christian Daughton, U.S. EPA/ORD/NERL/Las Vegas

10:15 am ***Break***

10:30 am **Environmental Monitoring for Chemicals in Waters:**
Michael J. Focazio, U.S. Geological Survey

11:15 am **Veterinary Pharmaceuticals: Potential Environmental Impact and Treatment Strategies:**
John Cicmanec, U.S. EPA/ORD/NRML/Cincinnati

12:00 pm ***Lunch***

1:00 pm **Assessing the Environmental Risk of Substances Under the US Food and Drug Act:**
Charles Eirkson, U.S. FDA

Endocrine Disrupting Pollutants in Effluent Dominated Streams:

Moderator: Dennis Weslowski, U.S. EPA Region 5

1:45 pm **A Regional Case Study of Alkyl Phenol and Ethoxylates:**
Peter Howe, U.S. EPA Region 5

2:10 pm **Field Observations of the Contributions of Alkyl Phenols on Fish Endocrine Disruption:**
Cliff Rice, U.S. Department of Agriculture
Dr. Carys Mitchelmore, University of Maryland

2:40 pm **Effects of Waste Water Treatment Effluents on Fish Endocrine Disruption:**
Larry Barber, U.S. Geological Survey

3:10 pm ***Break***

3:25 pm **Aquatic Toxicity, Estrogenicity and Treatability of Nonyl Phenol and Ethoxylates in Waste Water Effluents:**
Charles Staples, Assessment Technologies, Inc.

3:45 pm **Break Out Groups Meet:**
A. Pharm and Personal Care Products
B. EDCs in streams
C. Asbestos, et. al

1. Is it reasonable to expect increased risks over time for human health and the environment?
2. What knowledge gaps need to be filled to clarify the present and projected risks?
3. Pollution prevention/risk reduction options

4:30 pm **Break Out Groups Report Out**

4:45 pm ***Adjourn for the day*** *– Prearranged entertainment/social event*

Thursday August 14, 2003

8:30 am **Objectives of the Final Session:**
Dan Hopkins, Toxic Reduction Team Manager, U.S. EPA Region 5

8:40 am **Biotechnology: The Promise, Potential Risks, and How Can We Find Out!**
John Glaser, U.S. EPA/ORD/NRMRL – Cincinnati

9:25 am **Environmental Futures: Nano Technology and Genomics Coming Over the Horizon:**
Gerardo (Pasky) Pascual, ORD/OSP Environmental Future Workgroup

10:10 am ***Break***

10:25 am **Wrap up Round Table of Speakers to Address:**
Processes for identifying chemicals for focus
Are there realistic opportunities to address the data gaps?
Do we still need champions to move issues forward?
Are outside pressures necessary to move issues forward?

11:30 am **Feed Back from Environmental Journalist:**
Janet Raloff, Science News

How an outsider perceives these efforts:
This workshop
Action within the government agencies
A personal perspective

12:00 am ***Conference Closes***

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U.S. EPA Region/ORD Emerging Pollutants Workshop

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APPENDIX C: SLIDES FROM PRESENTATIONS

These slides can be found at: www.epa.gov/osp/regions/emerpoll.htm

Triclosan: One Example of How Emerging Pollutants Come to Our Attention	Steve Reimer
Perfluoro Octane Compounds in the Environment	
PFOS Toxicity Studies	Christopher Lau
Studies of Human and Animal Toxicity	John Cicmanec
Risk Information Update: Overview of Assessment and Activities of PFOS and PFOA	Jennifer Seed
PFOS and PFOA Program Office Update	Mary F. Dominiak
Toxic Chemicals Resulting From the Disposal of Electronic Equipment	
Examining the Pollution Potential of Discarded Electronic Equipment Disposal	Timothy Townsend
Emissions from the Incineration of Electronics Industry Waste	Eric Stewart
Toxic Chemicals Resulting From the Disposal of Electronic Equipment	Jason Swift
Brominated Flame Retardants	
Overview of Brominated Flame Retardants	Leif Magnuson
Polybrominated Diphenyl Ethers in People	Ron Hites
Brominated Flame Retardants in the Environment	Mehran Alaee
Brominated Flame Retardants: Toxicology and Risk	Linda Birnbaum
Bisphenol A and Phthalate Esters: Potential Sources of Resin Components in the Everyday Environments of Preschool Children	Marsha Morgan
Radium in Oil and Gas Piping and Production Facilities	Loren Setlow
Balanced Perspectives Regarding Environmental Levels of Thallium and Platinum Group Metals	Frank Anscombe
Asbestos and Related Durable Fibers: Too Ubiquitous, Too Persistent, Too Complex to Put Health Risks to Rest?	Phillip Cook

Pharmaceuticals and Personal Care Products in the Environment

Pharmaceuticals and Personal Care Products as Environmental Pollutants: Pollution from Personal Actions	Christian Daughton
Veterinary Pharmaceuticals: Potential Environmental Impact and Treatment Strategies	John Cicmanec
Environmental Assessments – Human and Animal Drugs	Charles E. Eirkson III

Endocrine Disrupting Pollutants in Effluent Dominated Streams

A Regional Case Study of Alkylphenol and Ethoxylates	Peter Howe
Field Observations of the Contributions of Alkyphenols on Fish Endocrine Disruption	Cliff Rice Carys Mitchelmore
Effects of Waste Water Treatment Effluents on Fish Endocrine Disruption	Larry Barber
Aquatic Toxicity, Estrogenicity, and Treatability of Nonyl Phenol and Ethoxylates in Waste Water Effluents	Charles Staples
Biotechnology: A New Frontier The Promise, Potential Risks, and How Can We Find Out?	John A. Glaser
Environmental Futures: Nano Technology and Genomics Coming Over the Horizon	Gerardo (Pasky) Pascual

APPENDIX D: FLIP CHART NOTES

The following information reflects two types of information developed during breakout sessions: flip chart notes – which, in some cases, were rather abbreviated; and other points made by discussion participants regarding the topics in question.

As a general matter, the breakout group participants were tasked with answering the following questions about each emerging pollutant/issue being discussed:

1. Is it reasonable to expect increased risks over time for human health and the environment?
2. What knowledge gaps need to be filled to clarify the present and projected risks?
3. Are there pollution prevention/risk reduction options?

Break Out Sessions – Tuesday, August 12

BFRs and Electronic Equipment

Participants of this break out session gave a run down on how they would apportion $100 if this amount were to be spent on researching these two topic areas:

	Responses
SOURCES Where the chemical is manufactured	$7
FATE Where it is found in the environment	$11
TRANSPORT How it is getting from the manufacturer, products and wastes to the environment and, movement through the environment	$13
EXPOSURE PATHWAYS How it is getting from product or the environment into the biota	$23
EFFECTS What are the effects on humans, animals	$30
RISK MANAGEMENT How to prevent release and exposure	$15

Other comments:

The above priorities from the Regions will help the Agency begin setting agendas for research, if not specific funding levels. It is important also that EPA not do all the research or fund all of it, but the

Agency should know Regional priorities. Regions represented at the workshop include Regions 2, 4, 5, and 9.

Regions can get involved in the ORD process and help raise ORD's voice. However, dollars for research at U.S. EPA will have to be taken from other programs as there is little new money available. A number of channels already exist for possible funding for research on BFRs and electronic devices:

RARE program funded at $200,000/year for each region or a total of $2 million annually
STAR program funded at $5 million/year
NHANES – Add BFRs by December 2004
Pollution Prevention Programs alternatives analysis, funded at $140,000/year per region
GLNPO emerging issues program funded at $150,000/year
GLAD (ARD R5) funded at $1.2 million/year for air deposition studies
Program Offices funding and priorities
Office of Solid Waste product stewardship for electronics
Office of Children's Health programs
Champions in program areas like TSCA under OPPT (BFRs not listed as PBT or BNS substance)
Water Division's IRIS program
Research Coordination Teams: Endocrine Disruption Team, Multimedia Team, B. Walls R4 Paper
ORD Multi-Year Plans (for 2006 budget cycle); recognize regional priorities
Agency Strategic Plan – all regions work together to identify top three common research priorities
"Short-Circuit" Process: Get priorities to Deputy Regional Administrator level, then to Gilman
Health Canada 2-gen study
Partner with CDC, OSHA, NIHS, other outside agencies
USDA dietary surveys; market basket surveys to get data on food other than fish.

More research is needed on indoor air quality in houses and house dust. The VCCEP documents food and dust, but there is no consideration of effects. There are people with high-end concentrations, but an exposure study is needed to determine why; perhaps the source is fatty animal food.

BFR composition in foam is 1-10%, but it is not known if the BFRs crumble out of the foam. Research is needed on the BFR content in foam and how much is released. There isn't much released from chemical manufacturers, but what about foam and furniture manufacturers? Priorities must be expressed to the Deputy Regional Administrator. Non-governmental organizations are good allies.

States need to know what to communicate to the public about this issue as a health risk. Is BFR in fish the number one route of exposure? States need to move forward and look at BFR prevalence and what risk that may pose to the population. However, it is hard to assess risks without much information. There are no human studies, only animal studies to date.

States also would like to have fish advisories for BFRs. If states push their needs, EPA might fund the research. Analytical methods for emerging pollutants must be standardized and cost-effectiveness examined. Health Canada is screening 23,000 substances, including BFRs.

Radium, Thallium, Platinum, and Other Metals

Question 1 – Is it reasonable to expect increased risks over time for human health and the environment?

Yes. We expect the risk from TENORM to go up because of the recycling of property and materials and making new products out of materials that have radium in them (e.g., coal ash used to make wall board). Also, as oil wells age, they have lower yields of oil and more water is generated. This can produce more radium in the pipe scale. In regard to platinum, because its use is increasing, there may be more human and environmental exposures – though we don't know the effects. Unsure as to whether thallium will pose more of a risk – but we wonder if some concerns about mercury poisoning actually involve thallium. Additionally, there are new metals being developed through nanotechnology involving combining rare earth metals with organic molecules, some of which can cross the blood-brain barrier. This could lead to additional risk. More research is needed to better quantify future risk.

Question 2 – What knowledge gaps need to be filled to clarify the present and projected risks?

Need to know more about "legacy" TENORM sites.

What is the fate of the radium in oil when descaling products are used? Is it simply moving the TENORM problem down the pipe to the refineries?

What is the chronic toxicity of some of these metals?

Also need to work on method development for laboratories. The levels of thallium and platinum are so low good laboratory analysis needs to be done.

Question 3 – Pollution prevention/risk reduction options?

In regard to TENORM, have to get information out to EPA people who are working at legacy sites (on-site people), on how you measure it and what you need to do if you find it.

Get information out to "ma and pa" oil and natural gas businesses to make them aware of the TENORM problem. Put information on radium in oil in the Oil Pollution Act of 1990 web site so that others in the industry will also know about it (including the Coast Guard).

Develop a GIS of legacy sites so that developers can be made aware of potential problems before they build houses on old, contaminated sites.

PFOS/PFOA

Question 1 – Is it reasonable to expect increased risks over time for human health and the environment?

We know that PFOS/PFOA is in the blood, so there is a concern about risk. We also know it is in children, and that it lasts in the body for a long time. But since PFOS is no longer being made in the U.S., don't know about future risk.

Question 2 – What knowledge gaps need to be filled to clarify the present and projected risks?

Need to figure out how the timing of the dose may determines effects. What is the mechanism that makes it lethal?

Need to look more closely at the effects data so they can extrapolate better.

Because effects on the lungs of mice were seen, is there a link between PFOS exposure and childhood asthma?

Have CDC include PFOS/PFOA in NHANES to monitor what is happening with this chemical.

Question 3 – Pollution prevention/risk reduction options?

More research to understand mechanisms by which it causes effects.

Bisphenol A and Phthalate Esters

Check current data on web site – synthesize data to find data gaps.

After review of data, determine chronic and acute toxicity.

Emerging data.

CDC review.

Recommend further study on exposure of humans and the environment and on endocrine disruption.

Break Out Sessions – Wednesday August 13

EDC and Effluent Dominated Streams

Question 1 – Is it reasonable to expect increased risks over time for human health and the environment?

Looked at NP and hormones. Is risk increasing? APEs production is stable and/or is being phased out.

Also looked at whether WWTP performance will increase or decrease in effectiveness over time. Believe that removals will be going down as excess capacity is removed, because the water will be retained for shorter periods of time. Additionally, a large number of modern plants are 20 to 30 years old and are coming to the end of their lives.

Question 2 – What knowledge gaps need to be filled to clarify the present and projected risks?

APEs and hormones – need to understand fate of hormones when they leave WWTP. Are effects significant?

Some hormones enter WWTPs as conjugated, others do not. When they are deconjugated, they become biologically active. What happens?

What happens to NP and carboxylated compounds when they degrade? Do carboxylated compounds go into anaerobic zones where they can become NP?

Need to understand the effects of combined sewage overflows on effluent dominated streams.

What is the long-term fate of NPs and hormones in the sediments near WWTP outfalls?

Did not address pollution prevention for this topic.

Asbestos and Durable Fibers

Question 1 – Is it reasonable to expect increased risks over time for human health and the environment?

Scope of what is asbestos and asbestos-like fibers?

Risk could increase because:

Long latency period. Disease doesn't appear until 30, 40, or 50 years after exposure.

Incidence of disease is increasing.

What exposures are going on now?

Products are still out there.

Natural asbestos in the soils means that exposure to the general population may increase.

Risk may not increase because:

In the 40s, working conditions were different. Different products, vacuums, etc. Great reductions of exposure in occupational settings.

Other observations:

Different analytical schemes for Libby, Montana and the World Trade Center.

Do not rule out certain fiber sizes. Short, thin fibers are strongly connected to tumors. Will not agree that different types of fibers are different in regard to effects.

Question 2 – What knowledge gaps need to be filled to clarify the present and projected risks?

Premise of risk – where risk comes from. Ideas about asbestos are based on old data. Need to reassess the role of fiber size.

Measure relative potency of different sizes and times. Refine methods for evaluating different fibers (relative potency factor).

Model what a person would accumulate in the lungs and then base evaluation on that.

Exposures are always a mixture.

Question 3 – Pollution prevention/risk reduction options?

What characteristics of fibers make them hazardous? We don't want to replicate the hazard of asbestos with other materials. Use our knowledge of which fibers cause disease to point out sizes and shapes that would not mimic asbestos.

Nanofibers are not into full-scale production yet. This is the optimum chance to prevent production of nanofibers than could be harmful.

Where we have mineral fiber dust that bears similarities to asbestos fibers, we should be very careful.

Pharmaceuticals

These contaminants could be broken up into categories: hormonal, antibiotics, and highly chemically reactive for discussion purposes. However, audience members decided that their answers to all three questions applied equally to all three categories.

Question 1 – Is it reasonable to expect increased risks over time for human health and the environment?

Yes, risks will increase due to the aging population's needs for medications, and perhaps to the growing uses of growth hormones in children. There are no controls on disposal for existing drugs. People are told to flush old medications down the toilet. Doctors and pharmacists must be educated and regulations established to prohibit this practice, which immediately transports myriad compounds to wastewater treatment plants.

Other choices for disposal include solid waste landfills, incineration, or recycling. Health care workers, industry, and the public must also be made aware of disposal concerns. The FDA does not regulate pharmaceuticals after their manufacture, but they do perform environmental impact analysis for animal drugs.

Question 2 – What knowledge gaps need to be filled to clarify the present and projected risks?

There are many gaps in data and knowledge for pharmaceuticals. Researchers need to determine what the degradation products are, and all of them must be part of any analysis. It must be determined whether there are additive compounds being formed, and the risk of all possible chemical interactions must be assessed. There is a data gap in regard to exposure data, i.e., what are the relevant exposures?

There is a need to define what constitutes a harmful amount of each compound as well as for all of them together. Detailed information on product constituents is needed. Some components of products may not be "inert" as listed on product labels. All applications of a product must be determined as well as whether anti-microbial resistance in the field translates to humans.

Personal Care Products

Time was limited for this discussion. Researchers have hardly begun to address the problem. Terminology can be a problem. Personal care products are those that are placed in contact with or on the human body, according to industry representatives. Lotions, cosmetics, and perfumes fit into this category. Home care products include soaps and detergents.

Effects of personal care products may be greater because they are placed directly on the body. Some are meant to be chemically interactive. There is a chemical ingredient review process by the FDA under the Food, Drug and Cosmetics Act, which regulates products. A toxics profile is produced by the FDA. For fragrances there is a toxics profile developed by RIFM, an industry group, with the FDA having oversight.

Final Group Discussion – Thursday August 14

This final group discussion was held at the end of the scientific presentations. The following information was recorded on flip charts and through other note-taking of participants' comments. Questions posed to participants were as follows:

1. Processes for identifying chemicals for focus. Why is it important to address emerging chemical issues?

2. Are there realistic opportunities to address the data gaps?

3. What would you suggest to improve the way we identify chemicals that might pose threats?

Question 1 – Processes for identifying chemicals for focus. Why is it important to address emerging chemical issues?

There is always competition for the interest and resources necessary to get emerging issues addressed.

Preventive action is important to prevent human and environmental impact and is always better than contingent action. It is faster, cheaper, and smarter. Unfortunately, it often takes a crisis to generate the political will to dedicate resources to a problem.

The Agency's future lies in getting ahead of problems rather than merely reacting to them.

Rachel Carson's *Silent Spring* proposed alternatives to pesticides. This is important – you don't just want to criticize. EPA needs to also pose alternatives. Several of the presentations that were given during the workshop mentioned alternatives. This is good.

Agrees that it is important to be proactive. The radium issue is already spreading and the levels are the highest seen in 20 years. This is a big problem. The Superfund group does inspections and was asked to come to the workshop, but no one attended.

Asbestos is an example of an issue we thought was closed, but it isn't. It was addressed when the issue was hot, and then EPA moved on to other things. It's still an issue.

Nanotechnology brings a ray of hope as a model for preventive action. We need to look at asbestos and make sure that with nanotechnology, we are creating products that will prevent the problems we've had with asbestos. Legacy pollutants have a long latency period and are problems for decades. You want to prevent them.

EPA's futures group has only five staff members. More are needed to raise emerging pollutant issues. We also need to look at more networking and look to ORD to help with future issues. Extrapolate from other information to move issues forward. Policy staff should be linked with research staff.

Scientists don't want to be too speculative, but they need to do something to be able to exchange information on things they have found that need to be looked at further, without looking like alarmists.

ORD should be proactive in helping EPA staff network and share ideas and solutions. A cross-department committee could assist in looking at possibilities without seeming alarmist.

An issue that is frustrating is the reluctance of EPA to apply the "precautionary principle" appropriately. Don't just ban chemicals when little is known about them, but if you do know something and it's negative – maybe this is a road we don't want to go down. Look at the signals. Europe and Canada have moved ahead of the U.S. in this regard. If we look at a chemical and see it's persistent, then maybe this should be a warning. We aren't listening to the signals.

We haven't applied what we've learned. Issues aren't resolved. PCBs, asbestos, mercury – we haven't completed these things. We need a straight-forward approach that carries through to an endpoint. We found that when we began looking for PCBs and mercury that they were all over the place. We need to push the knowledge forward, finish projects, and get the information out.

Smart people have to stay on jobs and finish them. We should not get distracted with the "crisis chemical" of the moment. Research was done on asbestos in the 1980s and preserved on magnetic tape, but then abandoned and never used. If we have to address something else in the short term, "park" the data and come back to it. Don't just abandon it.

There is a need to have a structure for addressing emerging pollutants. Europe and Canada both have structures. They looked cumbersome initially, but they work. They set priorities, achieve their goals, and move on. There are a lot of stakeholders involved in Canada and Europe.

The Europeans are six years ahead of us in regard to brominated flame retardants.

Partner with Europe and Canada to combine processes. This would put us much further down the road.

There is a need to link the policy folks with the research folks. Need to enlist OPPT in this.

EPA should function as one Agency, not groups of individuals competing with each other for money.

We had a national lake study going on and we looked at sites that we thought were important, but the studies were designed by people who didn't know about the work and included testing for pollutants we decided weren't there. OPPT required that we test for these things that they knew weren't there. We didn't take advantage of the knowledge we have.

Researchers need to know ecological thresholds and whether pollutants are heading toward concentrations that would cause an ecological collapse and not just rely on old linear trends.

Care must be taken when talking about thresholds so people won't think that there is a point below which there is no problem. Lead in humans is a classic example. We have to look at distributions within populations, not just thresholds.

An internal EPA forum or seminar could be designed to engage outside researchers and industry representatives to broaden the knowledge base. They would not have the burden of program ownership and could take information to EPA managers who aren't involved in specific programs.

Enlisting assistance and information from outsiders is important, but care must be taken not to disenfranchise EPA staff who need to be empowered, supported, and involved.

(Comment by an industry representative) When working on an issue with EPA, she ends up talking with people in the agency about others in the Agency who may have information they didn't know about. EPA needs a network of some sort for passing information around internally.

Question 2: Are there realistic opportunities to address the data gaps?

There may be realistic authorities and programs that could be tapped for resources to do research, such as the following:

TSCA Section 5: Chemicals to be introduced into commerce.

TSCA Section 8: Chemicals already in commerce, but industry must notice a problem

HPVC Program: High production volume chemicals – screening data.

VCCEP: Chemicals that may pose risk to children – 35 companies, 10 consortia sponsor 20 chemicals.

Monitoring Programs: State and federal levels.

Academic Studies

Other Federal Agency/Department Efforts

How can EPA start looking ahead to identify emerging areas of pollution and science and then get them to "stick" to the radar screen so they don't fall off?

Start a valid, objective, well-defined monitoring program and devote about 15% of the Agency's budget to it. EPA has been driven out of monitoring by politicians so we don't know what some conditions really are.

Cut back on spending time researching pollutants we already know are there. Look for unidentified peaks in analytical results and put more effort into identifying emerging pollution.

Labs should be involved from the beginning when developing testing programs. We need to have methodologies and know what we are looking for and how to quantify the results. The chemists need to be involved so they know what methods need to be developed.

Have tried to put together an emerging pollutants monitoring program, but haven't had any luck.

Have had the experience of finding negative data that weren't used.

TSCA can be used to ban chemicals, but some chemicals, such as dioxins, aren't manufactured as a product. They are by-products.

Have any chemicals gone through TSCA and been approved, then found later to be harmful, or was most of the bad stuff we are concerned with made before TSCA?

Believes that TSCA has cut down on the manufacture of harmful chemicals because companies know they will never be allowed under TSCA, so they don't develop them further.

Under section 8 of TSCA, EPA has gone after certain chemicals. At that time, they didn't realize that BFRs were an environmental problem.

There is a trend away from Section 8 to voluntary actions. Industry likes to work in dialogue groups and work with the Agency so research is done, but industry provides funds to accomplish it.

Question 3 – What would you suggest to improve the way we identify chemicals that might pose threats?

Champions may need to promote issues to gain EPA attention, but that may not be the most proactive role for the Agency to identify concerns.

The multimedia pollution prevention forum is one way to bring an emerging issue to management's attention, but they tried to do this with BFRs and the managers weren't interested.

OEI could catalogue information management issues, but may need outside people in an ombudsman role to assure the Agency that there is a constituency that cares about an issue. Bosses often want to know "who cares?" Have people from outside come in with issues and show why there is interest.

OEI could help develop a computer list serve in the Agency to help scientists and policy makers share information on emerging issues. Every six months or so, OEI could ask participants what they believe the priorities should be. This will help get around some of the isolation of researchers so they can see that others may be working on similar projects.

"Who cares" is an important issue. Multimedia issues are a problem because the program offices have louder voices. The Regions can have a lot of issues but they don't bubble up to ORD planning. We need to get more people involved from the Regions in planning. At this point, only one representative from the Regions participates, so not all interests can possibly be represented.

(Comment from an Environment Canada participant) NAFTA may provide a mechanism for dialogue that EPA could use.

The International Transport of Air Pollutants (ITAP) gets top management to call people together on issues.

Someone needs to develop a structure that can be used to raise issues. Challenges anyone who has good ideas to put them out there and get others involved. Get support and get it going.

Interest generated across the Agency will generate funding.

Some emerging issues are not associated with chemicals, e.g., wetlands quality or legal issues. We're not just dealing with scientific issues.

APPENDIX E: PARTICIPANT EVALUATION SUMMARY

Twenty-one written workshop evaluation forms were received at the close of the workshop, containing variable amounts of feedback. Meeting participants agreed that the Emerging Pollutants Workshop was a valuable opportunity to gain new information and insights about emerging issues, make valuable contacts, and exchange perspectives.

All responding participants rated the workshop either as "good" or "excellent" and most offered similar ratings for the major workshop components (Opening Welcome, Roundtable Discussions, and Closing Session), though about half (9 of 20) rated the Breakout Sessions as only "fair" and two rated them "poor." Accompanying written comments suggested that more structure (e.g., a designated moderator) and a more precise discussion question would have improved the effectiveness of these sessions. Five participants similarly rated the Roundtable Discussions either as "fair" or "poor." Regarding workshop accommodations and logistics, responding participants rated all categories (Meeting Materials, Registration Process, Hotel Accommodations, Helpfulness of Meeting Staff, and Meeting Room) as "good" or "excellent" with the exception of three individual "fair"ratings. Ratings were generally split fairly evenly between "good" and "excellent" for all five categories. Comments suggested that a more moderate room temperature and more legible handouts would improve the quality of future workshops.

Substantively, the information provided on brominated flame retardants (BFRs) was viewed by many (9 of 21) participants as the most valuable, and a number of respondents stated that EPA needs to develop a strategy for addressing concerns raised by BFR production and use. Several others made favorable comments about the value provided by the presentation on asbestos. Many other topics were singled out as being of particular value by individual workshop participants. Some respondents made particular note of the value provided by being able to better understand the state of existing Agency science regarding emerging pollutant issues, and the process by which specific issues are elevated within EPA. Others expressed the view that emerging pollutants should be a top Agency priority, and be examined on a continuing basis.

Many participants expressed appreciation for the opportunity to engage in dialog with Agency peers and counterparts on important emerging pollutant issues, and numerous suggestions were offered that this type of dialog be either continued or even instituted as a formal ORD process. Several respondents also appreciated the participation of people from outside EPA, and suggested that external stakeholders be included in future Agency dialog on emerging pollutant issues. Many respondents also suggested one or more mechanisms by which ongoing communication could/should be fostered among workshop participants and their respective organizations. Specific examples included the following: annual or semi-annual conferences and/or teleconferences, maintaining list-serves, publishing a newsletter, and supporting various approaches to less formal networking.