HEALTH AND ENVIRONMENTAL HEALTH: EXPANDING THE MOVEMENT

[Rachel's summary: We "environmental health" activists could appeal to a larger segment of the public if we based our work on all three environments that determine human health: the natural, the built, and the social.]

By Peter Montague

Since 1970, we "environmental health" activists have tiptoed onto territory occupied by public health professionals. But so far we have not fully embraced the public health approach -- defining and conducting our "environmental health" work as a branch of public health -- and as a result, our work does not yet appeal to large segments of the public. We are seen as elitists worried about irrelevant problems. We could easily change this.

Health and "environmental health"

The U.S. Institute of Medicine (IOM) defines the mission of public health this way: "to fulfill society's interest in assuring conditions in which people can be healthy."

The preamble to the constitution of the World Health Organization (WHO, July 22, 1946), defines health as "a state of complete well-being, physical, social, and mental, and not merely the absence of disease or infirmity."

The WHO constitution also defines health as a basic human right: "The enjoyment of the highest standard of health is one of the fundamental rights of every human being without distinction of race, religion, political belief, economic or social condition." This is consistent with Article 25 of the Universal Declaration of Human Rights of 1948, which says, "Everyone has the right to a standard of living adequate for the health and well-being of himself and his/her family, including food, clothing, housing, and medical care."

The World Health Organization defines "environmental health" as "those aspects of human health, including quality of life, that are determined by physical, chemical, biological, social, and psychosocial factors in the environment. It also refers to the theory and practice of assessing, correcting, controlling, and preventing those factors in the environment that can potentially affect adversely the health of present and future generations."

The Pew Commission on Environmental Health defined environmental health this way: "Environmental health comprises those aspects of human health, including quality of life, that are determined by interactions with physical, chemical, biological and social factors in the environment. It also refers to the theory and practice of assessing, correcting, controlling and preventing those factors in the environment that may adversely affect the health of present and future generations."

In the U.S., 3000 county and municipal health officials are represented by the National Association of County and City Health Officials (NACCHO). In Resolution 99-13, NACCHO specifically recognizes that "environment and health are intimately related, and environmental health is a public health activity..." So public health workers seem to be inviting "environmental health" activists onto their turf. What is holding us back?

The crux of the matter is that there are three "environments" that affect human health: the natural environment, the built environment, and the social environment. Each is crucial to public health. We "environmental health" activists have embraced the first two, but not all of us have yet embraced the third. Getting to that third "environment" is crucial for expanding our appeal to a broader audience.

The natural environment

For eons people have recognized that human health depends on the natural world. To thrive, we need clean water, clean air, and good food. However, there are two other "environments" upon which our health also depends -- the built environment and the social environment.

The built environment

We've known about the importance of the built environment for more than 2000 years. As humans began to crowd into small cities and disease rates soared, Hippocrates in Greece and later Vitruvius in Rome realized that positive
steps must be taken to maintain conditions in which people can be healthy.

In Greece and Rome, buildings were oriented to take advantage of fresh air and sunlight. The Roman aqueducts brought in fresh water, which was then distributed city-wide. The Roman baths made cleanliness possible for everyone. Certain occupations, such as silver mining, were known to cause illness and death. These early understandings formed the basis for what in the late 19th century became known as "public health[9]."

In England during the early, awful days of industrialization, which Charles Dickens[10] described so convincingly in the 1840s, the government recognized that disease arose from both the built and the social environments. Disease was caused by fetid air and improper waste disposal, but it was also caused by poverty and human degradation. In 1845 Friedrich Engels[11] published The Condition of the Working Class in England in 1844[12], cataloging what Charles Dickens had portrayed in novels -- unspeakable conditions of filth, poverty, and degradation giving rise to disease and death.

The social environment and the social determinants of health

The German physician Rudolf Virchow[13] is known as the "father of modern pathology" but he also pioneered our understanding of how social conditions foster health or disease. In 1848 the German government sent Virchow to Upper Silesia to investigate an epidemic of typhus. In his report, he said the epidemic was attributable to miserable living conditions, inadequate diet, and poor hygiene -- and, he said, these conditions were, in turn, attributable to feudalism{14}, lack of democracy, and unfair tax policies. Thus Virchow identified the social environment as an important factor in human disease.

From these roots grew the modern public health perspective on human well-being: society must create the conditions in which people can be healthy, and disease must be prevented whenever possible, rather than relying only on the curative powers of the physician. To this day, prevention is the keystone idea of public health practice. The medical model (one doctor, one patient) can only go so far. The public health model (preventive intervention by proper authorities at the level of the entire population) is essential for community health.

Providing clean water, offering vaccinations, establishing fire codes and structural requirements for buildings, requiring landlords to provide a modicum of sunlight and fresh air in rental properties -- these are population-wide public health interventions needed to prevent disease and injury.

Public health professionals fully understand that the natural and built environments are important for maintaining human health. The National Association of County and City Health Officials (NACCHO) has passed resolutions urging governments and the public to pay attention to both the natural and built environments, in order to maintain the health of populations. (See for example, NACCHO resolution 03-02[15] to support land use planning and design, and NACCHO resolution 98-06[16] on brownfields. And see NACCHO's statement[17], "Integrating Public Health into Land Use Decision-Making."

Activists in the "environmental health" movement also understand the importance of the natural and built environments to human health. Highways, sprawl, "brownfields," poisoned land, unsafe food additives, pesticides, contaminated drinking water -- these are all things that "environmental health" activists routinely tackle.

But in one respect, public health professionals are somewhat ahead of environmental health activists -- in recognizing how the social environment affects health. For example, NACCHO Resolution 02-04[18] acknowledges that "a significant body of research in the last fifteen years documents clearly that socioeconomic inequality, poor quality of life, and low socioeconomic status are principal causes of morbidity [sickness] and mortality [death]." We have put together a bibliography highlighting some of that research[19].

NACCHO Resolution 02-04 goes on to say, "We embrace social justice as the cornerstone of our work, recognizing that equity, shared decision making and attention to the social determinants of disease are central to promoting healthy communities."

In testimony before the National Institute of Medicine March 20, 2002, Dr. Adewale Troutman, representing NACCHO, described the "root causes of the current picture of ill health of large segments of our population: social injustice, economic inequality, and racism."

The World Health Organization (WHO) accepts that the "social determinants of health" must be addressed if we are to protect and maintain public health. In 1998{20}, WHO said, "Policy and action for health need to be geared towards addressing the social determinants of health in order to attack the causes of ill health before they can lead to problems." Notice the preventive approach. Attack the causes BEFORE they can lead to problems.

In a 2003 booklet, called The Social Determinants of Health: The Solid Facts[21], WHO discussed 10 aspects of "the social determinants of health." Here are a few excerpts from that booklet:

1. THE SOCIAL GRADIENT

Poor social and economic circumstances affect health throughout life. People further down the social ladder usually run at least twice the risk of serious illness and premature death as those near the top. Nor are the effects confined to the poor: the social gradient in health runs right across society, so that even among middle-class office workers, lower ranking staff suffer much more disease and earlier death than higher
ranking staff. Both material and psychosocial causes contribute to these differences and their effects extend to most diseases and causes of death.

2. STRESS

Stressful circumstances, making people feel worried, anxious and unable to cope, are damaging to health and may lead to premature death.

Social and psychological circumstances can cause long-term stress. Continuing anxiety, insecurity, low self-esteem, social isolation and lack of control over work and home life, have powerful effects on health. Such psychosocial risks accumulate during life and increase the chances of poor mental health and premature death.

In schools, workplaces and other institutions, the quality of the social environment and material security are often as important to health as the physical environment.

3. EARLY LIFE

A good start in life means supporting mothers and young children: the health impact of early development and education lasts a lifetime.

...[T]he foundations of adult health are laid in early childhood and before birth. Slow growth and poor emotional support raise the lifetime risk of poor physical health and reduce physical, cognitive and emotional functioning in adulthood.

Slow or retarded physical growth in infancy is associated with reduced cardiovascular, respiratory, pancreatic and kidney development and function, which increase the risk of illness in adulthood.

4. SOCIAL EXCLUSION

Life is short where its quality is poor. By causing hardship and resentment, poverty, social exclusion and discrimination cost lives.

Relative poverty means being much poorer than most people in society and is often defined as living on less than 60% of the national median income. It denies people access to decent housing, education, transport and other factors vital to full participation in life. Being excluded from the life of society and treated as less than equal leads to worse health and greater risks of premature death. The stresses of living in poverty are particularly harmful during pregnancy, to babies, children and old people. In some countries, as much as one quarter of the total population - and a higher proportion of children - live in relative poverty.

Social exclusion also results from racism, discrimination, stigmatization, hostility and unemployment. These processes prevent people from participating in education or training, and gaining access to services and citizenship activities. They are socially and psychologically damaging, materially costly, and harmful to health.

5. WORK

Stress in the workplace increases the risk of disease. People who have more control over their work have better health.

In general, having a job is better for health than having no job. But the social organization of work, management styles and social relationships in the workplace all matter for health. Evidence shows that stress at work plays an important role in contributing to the large social status differences in health, sickness absence and premature death.

Having little control over one's work is particularly strongly related to an increased risk of low back pain, sickness absence and cardiovascular disease.

6. UNEMPLOYMENT

Job security increases health, well-being and job satisfaction. Higher rates of unemployment cause more illness and premature death.

The health effects of unemployment are linked to both its psychological consequences and the financial problems it brings - especially debt.

Unemployment puts health at risk, and the risk is higher in regions where unemployment is widespread. Evidence from a number of countries shows that, even after allowing for other factors, unemployed people and their families suffer a substantially increased risk of premature death.

Policy should have three goals: to prevent unemployment and job insecurity; to reduce the hardship suffered by the unemployed; and to restore people to secure jobs.

7. SOCIAL SUPPORT

Friendship, good social relations and strong supportive networks improve health at home, at work and in the community.

Social support and good social relations make an important contribution to health. Social support helps give people the emotional and practical resources they need. Belonging to a social network of communication and mutual obligation makes people feel cared for, loved, esteemed and valued. This has a powerful protective effect on health.

8. ADDICTION

Individuals turn to alcohol, drugs and tobacco and suffer from their use, but use is influenced by the wider social setting.

Drug use is both a response to social breakdown and an important factor in worsening the resulting inequalities in
health. It offers users a mirage of escape from adversity and stress, but only makes their problems worse.

The use of alcohol, tobacco and illicit drugs is fostered by aggressive marketing and promotion by major transnational companies and by organized crime. Their activities are a major barrier to policy initiatives to reduce use among young people.

9. FOOD

Because global market forces control the food supply, healthy food is a political issue.

A shortage of food and lack of variety cause malnutrition and deficiency diseases. Excess intake (also a form of malnutrition) contributes to cardiovascular diseases, diabetes, cancer, degenerative eye diseases, obesity and dental caries. Food poverty exists side by side with food plenty. The important public health issue is the availability and cost of healthy, nutritious food. Access to good, affordable food makes more difference to what people eat than health education.

10. TRANSPORT

Healthy transport means less driving and more walking and cycling, backed up by better public transport.

Cycling, walking and the use of public transport promote health in four ways. They provide exercise, reduce fatal accidents, increase social contact and reduce air pollution.

Because mechanization has reduced the exercise involved in jobs and house work and added to the growing epidemic of obesity, people need to find new ways of building exercise into their lives. Transport policy can play a key role in combating sedentary lifestyles by reducing reliance on cars, increasing walking and cycling, and expanding public transport.

[End of excerpts.]

Summary

So there you have it. By embracing all three environments -- natural, built, and social -- environmental health activists could broaden their appeal to segments of the public who now think of “environment” as irrelevant, divorced from the problems of real life. Embracing that third environment -- the social determinants of health -- would help us develop an effective, lasting movement for change. Another world IS possible.


URL: http://www.precaution.org/lib/06/diane_wilson_released_from_jail.060221.htm

From: Pesticide Action Network Updates Service (PANUPS), Feb. 21, 2006

DIANE WILSON RELEASED FROM JAIL

[Rachel's introduction: Diane Wilson{1}, indomitable activist from Seadrift, Texas, and author of An Unreasonable Woman{2} was released from jail Feb. 17. Now she can continue her quest to bring Dow Chemical to justice.]

"Unjust laws exist; shall we be content to obey them... or shall we transgress them at once?" wrote Henry David Thoreau in his famed essay, "Civil Disobedience{3}." On Friday, February 17 another inspiring American activist, Diane Wilson{4}, was released after 74 days in a cold and crowded Texas jail cell. She had been arrested in Houston on December 5th for speaking out during a fundraiser for recently-indicted U.S. Representative Tom Delay{5}, then jailed under an existing warrant for protesting at the Dow Chemical plant in her hometown of Seadrift, Texas. Diane Wilson went to prison for making the point that the world's worst chemical disaster could well be repeated in her backyard.

Take action now{6} to insist that Texas governor Rick Perry enforce laws against toxic Texas polluters.

In 2002, Wilson climbed a chemical tower at the Dow plant in her hometown of Seadrift, Texas, and dropped a banner declaring, "Dow- Responsible for Bhopal." Dow is the sole owner of the chemical company Union Carlsbe, which
operated a pesticide plant in Bhopal, India[7]. In 1984 the poorly maintained factory exploded, filling the streets of the city with toxic clouds of methyl isocyanate gas. The Indian government charged Union Carbide and its former CEO Warren Anderson with manslaughter for killing 15,000 people--although the real figure may well be over 20,000--and claimed damages for injuries to 100,000 more.

Wilson's imprisonment raises the question of just who our justice system is protecting us from. Twenty one years after the explosion, Anderson has yet to appear for his criminal trial in India. Meanwhile, the citizens of Bhopal who survived that ghoulsh night continue to suffer and die not only from the long-term effects of continuing contamination, but also from the poverty that comes from being too sick to support a family. Survivors of the Bhopal gas leak are demanding that Anderson and Dow face trial, clean up the toxic site, pay for medical treatment and compensation for illnesses, and provide economic rehabilitation for those whose ability to work has been affected.

On February 20th, 150 survivors of the Union Carbide plant explosion and victims of the resulting groundwater contamination have set off on foot to New Delhi demanding a meeting with the Prime Minister. Depending on the response of the central government, the marchers may decide to go on an indefinite fast at the end of their 900 kilometer long march. Read a daily blog on the march at http://www.bhopal.net/ march/[8].

Those who suffer from Dow's pollution in the United States are recognizing that they have a tangible common bond with the Bhopal survivors. Wilson, a mother of five, captained a shrimp boat off the coast of Seadrift, Texas for years until she noticed that her friends were getting cancer and the shrimp she depended on were dying. When she found out that Dow and other chemical plants were dumping lethal ethylene dichloride and vinyl chloride into her beloved bays, Wilson launched herself on a mission to stop the pollution. She hatched a plan to sink her shrimp boat on top of a dioxin flume, and left her livelihood behind to fight full time against the corporation. Recognizing her community's bond with others harmed by the chemical industry, Wilson forged an alliance with the survivors in Bhopal, resulting in her action at Dow's Seadrift plant.

A Texas court charged Wilson with a minor misdemeanor for trespassing, but instead of showing up for her sentence immediately, she took off in search of fellow fugitive Warren Anderson. "This company has warrants after their arrest, and they can be defiant and not show up, but let a little woman with a banner drop it... and I'm a dangerous woman, and I have to be thrown in jail," Wilson decried.

Wilson's stay in jail was not a comfortable experience. She spent her first several days sleeping huddled on the floor without even a blanket or a toothbrush, in a cell where the one tiny window was papered over. "It feels incredible, just incredible to be out," she stated Friday a few hours after being released. "I've had a lot of people, especially the girls inside who know what it's like to sit on the floor of a crowded cell every day, tell me, 'I guess you won't do this again.'"

Yet her spirit has only been strengthened. "I told them I don't regret it, and I would do it again. We have to take our issues as seriously as the corporations and administration do. We need to be as committed to our issues as we can be; we need to draw a line and hold it."

Shocked by the conditions she found in the Victoria County Jail, Wilson drafted a letter to the local sheriff deploring the worst abuses. "The women in this jail are predominantly African American or Hispanic and very poor. Most of their offenses are minor, for things like traffic tickets or soliciting or violating probation--all non-violent, yet they are forced to remain in the cell without counsel for long periods of time," she wrote. Wilson's letter also described how lack of health care in the jail resulted in cases of a ruptured gallbladder, kidney failure, and even the tragic death of a newborn baby whose inmate mother was placed in solitary confinement when her water broke, leaving her to face a breech birth on her own.

"Under a government which imprisons any unjustly, the true place for a just man is also a prison," Thoreau declared after spending one night in jail in 1849 for refusing to pay taxes to a government that supported slavery. Thoreau's teachings that individuals should follow their own moral compass when the laws of their country are unjust provided the philosophical base for the actions of Gandhi and Martin Luther King, Jr. Today, Diane Wilson uses her moral compass to draw the lines of right and wrong, to speak out that polluting her community and taking the lives of 15,000 people and injuring 100,000 more in India is a much greater crime than unfurling a banner from a tower, or the minor transgressions of her cell mates.

A government that allows corporations to commit crimes with impunity becomes implicit in these crimes itself. A freedom of information act request in 2004 revealed that the U.S. State Department denied India's extradition order for Warren Anderson after the U.S. Department of Commerce joined Union Carbide in pleading on Anderson's behalf.

Thoreau described the act of civil disobedience as asserting personal freedom--freeing oneself from the fear of state retribution for non-cooperation with injustice. "I saw that, if there was a wall of stone between me and my townsmen, there was a still more difficult one to climb or break through, before they could get to be as free as I was," he observed from his jail cell. We curtail our own freedom with fear of speaking out. Yet there is a Diane Wilson in each of us, a core of courage to honor our own moral compass, to stride past fear toward the freedom to act on our convictions, to be as committed to our issues as we can be.

Take action! [9]

The Texas Commission on Environmental Quality (TCEQ) is currently revising its penalty policy, providing an opportunity...
to push for greater accountability against polluters that break the law and put public health at risk. Call Governor Perry to support stricter enforcement and penalties against corporate polluters. {10}

Sources:

For more information on the struggle of the Bhopal survivors, visit the Students for Bhopal web site:
http://www.studentsforbhopal.org/MarchToDelhi.htm#March {11}

Wilson, Diane. 2006. Letter from Jail.
http://www.chelseagreen.com/2005/items/unreasonablewoman/fromjail {12}

http://www.panna.org/resources/panups/panup_20051202.dv.html {13}


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(PANNA {17}).

{1} http://www.chelseagreen.com/2005/items/unreasonablewoman/fromjail
{2} http://www.precaution.org/lib/06/molly_ivins_unreasonable_woman.051025.htm
{3} http://eserver.org/thoreau/civil.html
{4} http://www.chelseagreen.com/2005/items/unreasonablewoman/fromjail
{6} http://ga4.org/campaign/texasLaws
{7} http://panna.org/resources/panups/panup_20051202.dv.html
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THE WEINBERG PROPOSAL

[T Rachel's introduction: Following up on last week's Rachel's #842, about corporate hired guns faking scientific studies -- here's a long expose by the American Chemical Society describing a hired-gun consulting firm called The Weinberg Group.]

A scientific consulting firm says that it aids companies in trouble, but critics say that it manufactures uncertainty and undermines science.

By Paul D. Thacker {2}

Tucked away inside the U.S. EPA's docket on PFOA {3}, a chemical manufactured by DuPont, is a 5-page letter written in April 2003 by the Weinberg Group {4}, an international scientific consulting firm based in Washington, D.C. The letter is addressed to DuPont's vice president of special initiatives, Jane Brooks {5}, and lays out a proposal for how the Weinberg Group can help the company deal with a growing regulatory and legal crisis over PFOA (perfluorooctanoic acid). PFOA is a common building block of the perfluorocarbon family of chemicals, which are renowned for their water and stain resistance. PFOA is the compound used to make Teflon and was once used in other products such as Scotchgard, Stainmaster, and Gore-Tex.

Critics say that the tactics detailed in the Weinberg proposal are commonly used by chemical and pharmaceutical companies trying to combat lawsuits and regulations against their products. View the proposal [354KB PDF]"The constant theme which permeates our recommendations on the issues faced by DuPont is that DUPONT MUST SHAPE THE DEBATE AT ALL LEVELS," states the letter (emphasis in original). For 23 years, the letter continues, the Weinberg Group "has helped numerous companies manage issues allegedly related to environmental exposures. Beginning with Agent Orange in 1983, we have successfully guided clients through myriad regulatory, litigation and public relations challenges posed by those whose agenda is to grossly over regulate, extract settlements from, or otherwise damage the chemical manufacturing industry."
Although a DuPont spokesperson confirmed that they had hired the Weinberg Group, no evidence exist that they followed through with all the items outlined in the plan. Nevertheless, experts contend that the document provides one of the clearest examples they have seen to illustrate how consulting firms help industries deal with scientific questions about the safety or health consequences of their products. These firms develop legal defense campaigns, ostensibly based on science, to sway juries during trials, to counteract potential regulatory oversight, and to influence the public's view about the health effects of products. Critics such as David Michaels, chair of the Project on Scientific Knowledge and Public Policy at George Washington University, charge that these groups "manufacture uncertainty" -- a term Michaels coined -- in order to prevent or delay regulations and civil lawsuits.

SIDEBAR

Critics say that the tactics detailed in the Weinberg proposal are commonly used by chemical and pharmaceutical companies trying to combat lawsuits and regulations against their products.

View the Weinberg Group's proposal [345 L PDF]

The 2003 letter from the Weinberg Group arrived at DuPont as EPA was finishing up a draft risk assessment on the possible health effects of PFOA. The company was also facing a civil-action lawsuit in West Virginia with plaintiffs alleging that they suffered deleterious health effects from PFOA in their drinking water. In 2004 and 2005, JP Morgan Worldwide Securities Services released reports for DuPont investors predicting that the company faced potential EPA fines of more than $300 million and a total liability of $150-$800 million. DuPont also faced risks to its fluoropolymers and telomers business, which the report pegged at about $1.23 billion (4% of total sales), with $100 million in after-tax profits, in 2004. In fact, DuPont settled the class-action lawsuit with residents around a manufacturing plant in March 2005 for $107 million. And in December 2005, DuPont agreed to spend $16.5 million to settle allegations that it withheld from EPA the results of a 1981 study that showed PFOA can cross the placental barrier in humans.

SIDEBAR: Transcript

The following is a transcript of an interview with Mr. Matthew Weinberg, who is the CEO of the Weinberg Group. ES&T has gone to the offices of the Weinberg Group and has offered Matthew Weinberg a chance to review and comment on a letter from the Weinberg Group that was found in the US EPA docket on PFOA. The letter is addressed to DuPont and is signed by Mr. Terrence Gaffney, Vice President of product defense for the Weinberg Group.

ES&T later contacted DuPont and the company confirmed that the Weinberg Group had "assisted us in identifying scientific third party experts on an issue involving the company." The DuPont spokesperson later stated that this issue was "probably PFOA."

A: A paper ostensibly from the Weinberg group. Okay, I can see what it says.

Q: Terrence Gaffney is no longer working with you, is he?

A: That's correct.

Q: Okay. Um, did you guys, uh, ever end up taking this account?

A: I'm not at liberty to discuss our clients.

Q: Um, okay. If I called DuPont, would they tell me that you had worked for them or that you had not worked for them?

A: I have no idea what DuPont would tell you.

Q: Okay.

A: You'd have to ask DuPont.

Q: Is this a typical sort of contract that, um, that you send out?

A: No. That is not a contract.

Q: Well, I mean a typical sort of a sales pitch. Is this typical of the type of work that Weinberg does, or...?

A: I don't know. The Weinberg group is a scientific consulting firm.

Q: Okay.

A: We assist companies in putting forth the right data about their products, as one part of our business.

Q: So you're not certain if this document is, is...you don't know if this is an actual document or not? Are you uncertain?

A: No. It would appear it's a document that left our office.

Q: Okay.

Q: I could find no evidence you guys had ended up working for DuPont. Um, none that I could find, and none that, uh, when I was banging around looking for other people...EPA could find no evidence either, although there might be more documents, um, in the discovery process which would come out, um, and maybe at that time I might have to come back
and talk to you again. I don't know specifically whether I will or not.

A: I'll be happy to take your call any time you call me.

Q: Um, who are you guys representing on phthalates?

A: I'm not at liberty to discuss any of our clients.

Q: Okay.

Q: But Mr. Lamb is working for you? Is that correct?

A: Dr. Lamb is working...Dr. Lamb is an employee of the Weinberg group.

Q: Okay. Um, in the, uh, tobacco legacy documents, when it refers to a Mr. Weinberg, that was your father? Is that your father, Myron?

A: Well, I believe it refers to Dr. Weinberg, then that would be my father, Myron.

Q: Your father Myron?

A: That's correct.

Q: Your father, is he still an employee?

A: Well...

Q: Is he retired?

A: Which question do you want me to answer?

Q: Well, is he...?

A: He is no longer an employee of the Weinberg group.

Q: Okay. Do you know where Mr. Gaffney is?

A: I have no idea where Mr. Gaffney is.

Q: Okay.

A: I'm not being coy. Dr. Weinberg is still working.

Q: Oh, he is? Okay.

A: And still does work for the Weinberg group.

Q: Okay.

A: I answered your questions accurately.

Q: No. That's fine. It doesn't really matter.

A: But I realize you walked away with an...with the wrong impression.

Q: Okay.

A: He's simply no longer an employee.

Q: Okay. Um, so anything else you'd like to say, after reading this? Do you have any comment on...

A: Do you have any questions?

Q: Well, one of the things I was interested in was, um...I don't know. I just wanted to give you a chance to read it and see if you....

A: I've perused it.

Q: Okay. Um, I was wondering what specifically...there was one particular passage in here that I thought was kind of interesting. Um, "reshape the debate by identifying the likely known health benefits of PFOA exposure by analyzing existing data and/or constructing a study to establish not only that PFOA is safe over a range of serum concentrations levels but it offers real health benefits." In parentheses it says "oxygen carrying capacity and prevention of CAD." Which is cardiovascular...oh I'm sorry. Wait. cardiovascular disease. Um, but cardiovascular disease....

Did you find any, has there been any, um, anything published in the peer-reviewed literature that would lead one to believe that?

A: I have no...I am not an expert on PFOA and I couldn't tell you what's been published or what hasn't been.

Q: Okay. Alright. I just wanted to give you a chance....Do you have anything else to say?

A: I guess I have a question for you. I don't understand what you see in that document that's worthy of a conversation between us.

Q: Well, it was very interesting, is when I showed this passage, that passage, particularly to David Ozonoff[11]. I don't know if you know who he is.

A: I've heard the name, but I can't place him.

Q: Um, he's at BU. He was on the SAB panel for PFOA and he, uh, called that particular passage sort of, uh, "fantasy thinking."

A: Okay. Uh, uh, I would...would suggest strongly that the letter you are looking at appears to have been a marketing document.

Q: Okay.

A: I do not think that it is a document that in any way, shape, or form, makes claims, nor is it intended to represent a specific point of view. It is a marketing document telling them things we maybe think...are possible. But I believe it
clearly states...you just read me a part that says "study and analysis are needed." I don't believe the document purports to say that that's been done.

Q: Okay.

A: It may have been done. It may have been done by others. I don't believe this document makes this claim that we had done that work at this point or that we were ever going to do that work.

Q: Okay.

A: My only suggestion would be that you stick to what the document says and not attempt to expand beyond what it doesn't say.

Q: Okay.

A: Oh, I'm not expanding anything. I'm just passing it to other people and having them look at it and giving you what their opinion is.

A: Well, then their opinion of what we wrote, would be their opinion.

Q: Right.

A: It wouldn't necessarily be fact. Because they didn't write it.

Q: Right. Exactly. That's why it's their opinion. That's understood.

A: But in science, there's fact.

Q: Right.

A: Not opinion.

Q: Right.

A: I grant you that people can interpret scientific data differently based on various rationale. But truth in science is what I believe all reputable scientists seek.

Q: Okay. I think that's it. Do you have anything else to say?

A: I'll give you my card.

Q: Okay.

ES&T confirmed the letter's authenticity with Matthew Weinberg, CEO of the Weinberg Group, and a spokesperson for DuPont told ES&T that the Weinberg Group did work for the company several years ago: "They assisted us in identifying scientific third-party experts on an issue involving the company." However, when asked to describe the work, the spokesperson would only say, "Probably PFOA. I think the letter was written three years ago."

In an interview, Weinberg described the proposal as a "marketing document". Later, he added, "My only suggestion would be that you stick to what the document says and not attempt to expand beyond [to] what it doesn't say."

The sales pitch Passages from the letter describe how the firm will develop a defense strategy based on science. "[W]e will harness, focus and involve the scientific and intellectual capital of our company with one goal in mind -- creating the outcome our client desires." Another sentence reads, "This would include facilitating the publication of papers and articles dispelling the alleged nexus between PFOA and teratogenicity as well as other claimed harm."

Michaels agrees with Weinberg that the letter is a sales pitch, but he adds that it originates from a "product defense firm" and is not about science. "What is doesn't say here is, 'We'll get the science right,’” he points out. "What it says is, 'We'll make sure the science comes out in a way you want it.’” Michaels calls the letter one of the best examples he has seen of what he calls a common business strategy: to create scientific doubt in order to stave off lawsuits and regulatory action.

"They have experts and put papers in the scientific literature because they know regulatory agencies like to see peer review," he says. But these studies, he adds, are published in "vanity journals -- journals that publish studies with minimal peer review."

Most scientists are completely in the dark when it comes to understanding how corporations manipulate science, says David Ozonoff, chair of the department of environmental health at Boston University. Ozonoff, who spent years studying the asbestos industry, recalls, "I went into [studying the asbestos issue] really thinking that industry can have its own interpretation of the scientific findings. It was the sociology of science and the social construction of knowledge, and they would naturally tend to emphasize certain things while workers would look at the same things differently." But as he sifted through letters and documents that came to light during court cases, Ozonoff found evidence that corporate executives had not only known for decades that asbestos was dangerous but they had outlined and put into practice a defense strategy to protect their product and company profits. "It was planned out in the documents in black and white," he says. "They thought nobody would ever see it."

"I have somewhat the same reaction to this letter,” he said about the Weinberg memo to DuPont. "These are things that we know are going on."

For example, the Weinberg letter lists a series of proposed tasks designed to limit liability, including the recruitment of scientific experts on PFOA "so as to develop a premium expert panel and concurrently conflict out experts from consulting with plaintiffs.” Experts who worked for DuPont
through the Weinberg Group would have been unable to testify for plaintiffs.

"They're offering to get rid of inconvenient witnesses for the other side," says Ozonoff. He adds that he has received similar requests in the past from lawyers asking him to consult on cases. "I wouldn't have to testify," he says, "but I knew right away what they were doing was trying to conflict me out of a case."

Ozonoff, who sat on EPA's Science Advisory Board review panel for PFOA[12], points to a passage in the memo that details how to identify the likely health benefits of the chemical "by analyzing existing data, and/or constructing a study to establish" that PFOA is safe and "offers real health benefits." The next sentence mentions the oxygen-carrying capacity of blood and the prevention of coronary artery disease.

"That blew me away," says Ozonoff, adding that data on PFOA seem to show an effect on lipid metabolism; this raises concerns that the chemical may actually increase the risk of cardiovascular disease. "This [proposal] is a 'manufacturing doubt' strategy. If you say, 'Gee, this might cause heart disease,' then they'll come back with another story that says it's good for your heart." Constructing this sort of narrative, he says, sets a research agenda that any independent scientist wandering into the field must address.

However, Weinberg maintains that science is open to interpretation. "I grant you that people can interpret scientific data differently based on various rationales. But truth in science is what I believe all reputable scientists seek."

Actions from the Weinberg Group

Weinberg says that he is not at liberty to discuss his clients, but ES&T discovered that his company did product defense work for NVE Pharmaceuticals in 2004, when the U.S. Food and Drug Administration (FDA) was seeking to ban the diet drug ephedra. "In our perspective, the government's decision relied on unreliable data relating to misuse of the product, and that when used as directed, not only is ephedra safe, but it has an exemplary safety profile, over an extended period of time," said Terrence Gaffney, a vice president with the Weinberg Group. "We believe we win on the science hands down," he added.

The following month, FDA banned ephedra[13], citing numerous studies that found that the herbal supplement raised blood pressure and stressed the circulatory system. A review sponsored by the National Institutes of Health concluded that ephedra use is associated with, among other things, increased risk of heart palpitations and psychiatric and upper gastrointestinal effects.

The Weinberg Group also wrote the American Chemistry Council's (ACC) 2005 position paper on endocrine disrupters [448KB PDF][14]. ACC is the lobbying group for chemical manufacturers. One of the two coauthors of the report is James Lamb, who is an employee of the Weinberg Group[15] and has also worked for industry on other chemicals such as perchlorate. In January, when the state of California held hearings to debate the health risks and possible use restrictions for six phthalates and bisphenol-A -- suspected endocrine disrupters -- in baby toys, Lamb testified[16] that the chemicals were safe.

"This is something that's been looked at for years... with the conclusion that the phthalates are safe," he told a Sacramento, Calif., news station at the time. In 2005, Europeans permanently banned six phthalates from baby toys, and the California legislation was an attempt to replicate this ban.

"Wherever I am, [Lamb] is always there," says Frederick vom Saal[17], a professor of biology at the University of Missouri and an expert on endocrine disrupters. "He's probably heard so many of my lectures that it must make him sick."

Vom Saal has been under attack for his work that finds that bisphenol- A poses endocrine-disrupting health risks to humans. In January 2005, the journal Environmental Health Perspectives published a letter criticizing vom Saal's recent research[18] on bisphenol-A (Environ. Health Perspect. 2006, 114 [1], A16-A17). The letter was signed by Joseph Politch[19], a research associate in the department of obstetrics and gynecology at Boston University. Because of the journal's conflict-of-interest policy, Politch's letter noted that he was a consultant for the Weinberg Group.

Politch told ES&T that he neither conducts research on bisphenol-A nor plans any future studies on the chemical, but he did admit that he has done consulting for the Weinberg Group. Politch refused to answer more questions about the exact nature of his consulting work, other than confirming that he had written the letter. "You should contact the Weinberg Group," he told ES&T and then ended the conversation.

Vom Saal says that hiring scientists to send letters to scientific journals is just one tactic that industry uses to create the illusion of a scientific controversy. Many of these strategies were pioneered by the tobacco companies. "There's not one strategy that is new or creative," he says.

Smoking gun?

Stanton Glantz[20], a professor of medicine at the University of California, San Francisco, and a documenter of the scientific battles over tobacco smoking, backs up vom Saal's assertion that this is an old approach. Glantz is a coauthor of the book The Cigarette Papers[21] and has written numerous peer-reviewed studies on the tobacco industry, which are based on documents contained in the Legacy Tobacco Documents Library[22]. This library is an online database of internal company papers obtained as part of the final U.S. court settlement with Big Tobacco in the 1990s.

"Basically, the tobacco companies set up this huge sub rosa network of scientists and experts around the world who were
paid through the tobacco lawyers to give lectures contesting
the evidence on secondhand smoke -- to show up at hearings;
to, in some cases, lobby; to publish articles," he says.
Although the effort was meant to undermine the science
labeling passive smoke a health risk, he says the tactics were
very similar to what is contained in the Weinberg proposal.

"It was very effective for [tobacco companies] for years, and
[the] Weinberg [Group] did a lot of the recruiting for them.
They were the recruiting agency that helped to get the whole
thing up and running," he says. Glantz's latest paper on this
recruitment cites a Philip Morris action plan detailing what
the company expected during 1989-1992 from scientists
hired as consultants. "They should be appropriately
couraged to prepare papers, participate in scientific
societies with relevant areas of interest, and take active roles
in scientific conferences," reads the document. "Where
possible, without compromising a scientist's effectiveness,
they should be encouraged to provide statements or testimony
for use before government commissions and information to

"People in the scientific community don't want to hear about
this," says vom Saal. "When you point out corruption, it
makes scientists uncomfortable."

But Glantz has studied Big Tobacco's impact on his
profession for more than a decade, and he sees a much bigger
problem looming for science. As the federal government cuts
back on funding for research, scientists are now forced to rely
more and more on financial assistance from corporations; this
raises troubling questions about whether the results from
these studies will be impartial and objective or favorable to
the companies that paid for them.

"The whole scientific enterprise is being distorted by these
corporate interests," Glantz says. "That's why it is so
important that we have a healthy academic community, to be
a voice that isn't being controlled.

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From: Environmental Health Perspectives, Feb. 1, 2006

NEW THINKING ON NEURODEVELOPMENT

[Rachel's summary: About 17% of school-age children in the
United States suffer from a disability that affects their
behavior, memory, or ability to learn. And the incidence of
these disorders is rising. What's that about? Why can't we do
better by our kids?]

By Michael Szpir

The notion that some substances in the environment can
damage the nervous system has an ancient history. The
neurotoxicity of lead was recognized more than 2,000 years
ago by the Greek physician Dioscerides, who wrote, "Lead
makes the mind give way." In the intervening millennia many
other substances have been added to the list of known or
suspected neurotoxicants. Despite this accumulation of
knowledge, there is still much that isn't understood about how
neurotoxicants affect the developing brain, especially the
effects of low-dose exposures. Today researchers are taking a
hard look at low-dose exposures in utero and during
childhood to unravel some of the mysteries of impaired
neurodevelopment.

About 17% of school-age children in the United States suffer
from a disability that affects their behavior, memory, or
ability to learn, according to a study published in the March
1994 issue of Pediatrics by a team from the Centers for
Disease Control and Prevention (CDC). The list of maladies
includes attention deficit/hyperactivity disorder (ADHD),
autistic spectrum disorders, epilepsy, Tourette syndrome, and
less specific conditions such as mental retardation and
cerebral palsy. All are believed to be the outcome of some
abnormal process that unfolded as the brain was developing
in utero or in the young child.
These disorders have an enormous impact on families and society. According to the 1996 book Learning Disabilities: Lifelong Issues, children with these disorders have higher rates of mental illness and suicide, and are more likely to engage in substance abuse and to commit crimes as adults. The overall economic cost of neurodevelopmental disorders in the United States is estimated to be $81.5-167 billion per year, according to a report published in the December 2001 issue of EHP Supplements{1}.

Potentially even more disturbing is that a number of epidemiologic studies suggest that the incidence of certain disorders is on the rise. In the United States, the diagnosis of autistic spectrum disorders increased from 4-5 per 10,000 children in the 1980s to 30-60 per 10,000 children in the 1990s, according to a report in the August 2003 Journal of Autism and Developmental Disorders. Similarly, notes a report in the February 2002 issue of CNS Drugs, the diagnosis of ADHD grew 250% between 1990 and 1998. The number of children in special education programs classified with learning disabilities increased 191% between 1977 and 1994, according to an article in Advances in Learning and Behavioral Disabilities, Volume 12, published in 1998.

So what is going on? The short answer is that no one really knows. There's not even consensus on what the soaring rates actually mean. Heightened public awareness could account for the surge in the numbers, or it may be that physicians are getting better at diagnosing the conditions. Some autism researchers believe the rise in that condition's prevalence simply reflects changes in diagnostic criteria over the last 25 years. On the other hand, some scientists believe that the rates of neurodevelopmental disease are truly increasing, and that the growing burden of chemicals in the environment may play a role.

With that in mind, investigators are considering the effects of gene-environment interactions. A child with a mild genetic tendency toward a neurodevelopmental disorder might develop without clinically measurable abnormalities in the absence of environmental "hits." However, children in industrialized nations develop and grow up in a veritable sea of xenobiotic chemicals, says Isaac Pessah, director of the Environmental Health Laboratory, stated, "There is no safe blood [lead] level in children." He says, "most of us have a host of defense mechanisms that protect us from adverse outcomes. However, genetic polymorphisms, complex epistasis, and cytogenetic abnormalities could weaken these defenses and amplify chemical damage, initiating a freefall into a clinical syndrome."

Pessah cites the example of autism. He says susceptibility for autism is likely conferred by several defective genes, no one of which can account for all the core symptoms of social disinterest, repetitive and overly focused behaviors, and problems in communication. Could multiple genetic liabilities and exposure to a chemically complex environment act in concert to increase the incidence and severity of the condition?

Despite the uncertainties, many scientists believe it would be wise to err on the side of caution when it comes to a research agenda. As Martha Herbert, a pediatric neurologist at Harvard Medical School, puts it, "Even though we may have neither consensus nor certainty about an autism epidemic, there are enough studies coming in with higher numbers that we should take it seriously. Environmental hypotheses ought to be central to research now. The physiological systems that have been harmed by environmental factors may also point to treatment targets, and this might be a great way to help the children."

The Parade of Neurotoxicants

Among the most intensely studied neurotoxicants are metals (lead, mercury, and manganese), pesticides, polychlorinated biphenyls (PCBs), and polybrominated diphenyl ethers (PBDEs). A number of these compounds were identified as neurotoxicants when individuals were exposed to high doses during occupational accidents or childhood poisonings. Scientists are now exploring the potential consequences of low-dose exposures, especially to children and fetuses. Epidemiologic studies play a central role, and these are often complemented by experimental work on animals and cell cultures. These days, researchers are looking not only at associations between toxicants and disease, but also at the underlying cellular and molecular mechanisms.

Lead. Studies dating to the 1970s show that children exposed to lead have deficits in IQ, attention, and language. In response, the CDC revised its limits for acceptable blood levels of the metal in several steps, from 60 micrograms per deciliter (micrograms/deciliter) in the 1960s to the current level of 10 micrograms/deciliter, set in 1991. But many scientists think that limit is still too high. A study reported in the September 2005 issue of EHP found that there were significant effects on a child's IQ even when blood lead concentrations were below 10 micrograms/deciliter. Upon the July 2005 release of the Third National Report on Human Exposure to Environmental Chemicals by the CDC, Jim Pirkle, deputy director for science at the CDC's Environmental Health Laboratory, stated, "There is no safe blood [lead] level in children."

Several groups have also found evidence that lead exposure may shape a child's social behavior. An article in the May 2000 issue of Environmental Research reports a strong correlation, dating back to 1900, between violent crime and the use of lead-based paint and leaded gasoline. The research complements studies by Herbert Needleman, a professor of psychiatry and pediatrics at the University of Pittsburgh School of Medicine, who found that bone lead levels in young males were correlated with aggression and criminality. "Lead is significantly associated with a risk for delinquency," says Needleman. His research appeared in the November-December 2002 issue of Neurotoxicology and Teratology and the 7 February 1996 issue of JAMA.
Another new area of research links early lead exposure to changes in the aging brain. Nasser Zawia, an associate professor of pharmacology and toxicology at the University of Rhode Island, Kingston, and his colleagues found increased expression of amyloid precursor protein (APP) and its product, β-amyloid (which is a hallmark of Alzheimer disease), in aging rats that were exposed to lead shortly after birth. In contrast, old rats that were exposed to lead did not show an increased expression of APP and β-amyloid. The work, published in the 26 January 2005 issue of The Journal of Neuroscience, suggests that early exposure to lead can "reprogram" gene expression and regulation later in life. According to Zawia, preliminary research also shows that "monkeys exposed to lead as infants exhibit similar molecular changes as well as exaggerated Alzheimer's pathology."

Mercury. The current Environmental Protection Agency (EPA) reference dose for methylmercury (an organic, toxic form of mercury) is 0.1 micrograms per kilogram per day (micrograms/kg/day). Humans are exposed to methylmercury primarily through consumption of contaminated fish; a good 70% of this contamination comes from anthropogenic sources such as emissions from coal-fired power plants. High-level exposure to methylmercury in the womb is linked to a number of impairments, including mental retardation, cerebral palsy, seizures, deafness, blindness, and speech difficulties. An article in the May 2005 issue of EHP\textsuperscript{2} puts the economic cost to the United States of methylmercury-induced toxicity (in terms of lost productivity) at $8.7 billion annually.

The effects of low-dose exposures are not so apparent. Two large epidemiologic studies of fishing populations in the Faroe Islands and the Seychelles have produced conflicting results regarding low-dose effects. Both studies sought to examine the association between methylmercury exposure and neurodevelopment in children whose mothers ate contaminated seafood during pregnancy.

The leader of the Faroe Islands study, Philippe Grandjean, an adjunct professor of environmental health at the Harvard School of Public Health, and his colleagues reported in the November 1997 issue of Neurotoxicology and Teratology that 7-year-old Faroese children had significant cognitive deficits and neurological changes after prenatal exposure to methylmercury. Grandjean's team followed up on the children at age 14. According to a report in the February 2004 issue of The Journal of Pediatrics, the children continued to have problems, including neurological changes and decreased nervous control of the heart.

In contrast, the authors of the Seychelles study found little evidence of lasting harm on a cohort of 66-month-old children, according to their report in the 26 August 1998 issue of JAMA. A follow-up study, published in the 17 May 2003 issue of The Lancet, similarly found no lasting effects on language, memory, motor skills, or behavioral function when the children were 9 years old.

The different outcomes of the two studies are puzzling because the children of both populations appeared to be exposed to similar amounts of methylmercury. Several explanations have been proposed, including the possibility that genetic differences between the populations may alter their relative predispositions to harm from mercury exposure. The source of methylmercury is also different in the two populations. The Faroese are exposed primarily through the consumption of pilot whale meat, whereas the Seychelles population relies heavily on ocean fish. According to Gary Myers, a professor of neurology and pediatrics at the University of Rochester Medical Center and one of the principal investigators of the Seychelles study, whale meat contains many other contaminants (including PCBs) besides methylmercury. "There is also evidence," he says, "that the effects of concomitant PCB and mercury exposure are synergistic."

Researchers continue to look at whether there is a danger from methylmercury at the levels of exposure achieved by fish consumption. Another layer of uncertainty was added with findings published in the October 2005 issue of EHP\textsuperscript{3} showing that fish consumption during pregnancy appeared to boost infant cognition—but only as long as mercury intake, as measured in maternal hair, wasn't too high.

The question of whether low levels of mercury are harmful has also manifested itself in a controversy over the use of vaccines containing thimerosal, a preservative. Although thimerosal was removed from many of these vaccines in 2001, children that were immunized before that date could have received a cumulative dose of more than 200 micrograms/kg of mercury with the routine complement of childhood vaccinations, according to a study in the May 2001 issue of Pediatrics. Thimerosal is nearly half ethylmercury by weight. Because ethylmercury is an organic form of mercury, there is some suspicion that it acts like methylmercury in the brain, although research published in the August 2005 issue of EHP\textsuperscript{4} suggests that the two forms differ greatly in how they are distributed through and eliminated from the brain. Developing countries continue to use pediatric vaccines that contain thimerosal. In the United States, thimerosal is still present in influenza vaccines, which the CDC recommends be given to pregnant women and children aged 6-23 months.

Advocacy groups, such as SafeMinds, have suggested that the decades-long rise in the diagnosis of autism is related to the presence of thimerosal in vaccines. In May 2004, however, the Institute of Medicine (IOM) issued a report, Immunization Safety Review: Vaccines and Autism, stating that several epidemiological studies published since 2001 "consistently provided evidence of no association" between thimerosal-containing vaccines and autism. However, the IOM's report has been severely criticized by a number of advocacy groups, including the National Autism Association, for relying too heavily on a specific set of epidemiologic data while dismissing clinical evidence and other epidemiologic studies that showed evidence of a link.
Despite the assurances of the IOM, some scientists continue to explore the mechanisms underlying the potential neurotoxic effects of thimerosal. In the January 2005 issue of NeuroToxicology, S. Jill James, a professor of pediatrics at the University of Arkansas for Medical Sciences, and her colleagues report that the neuronal and glial cell toxicity of methylmercury and ethylmercury (as dosed via thimerosal) are both mediated by the depletion of the antioxidant peptide glutathione. Of the two cell types, neurons were found to be particularly susceptible to ethylmercury-induced glutathione depletion and cell death, according to James, and pretreatment of the cells with glutathione reduced these effects. Other studies by James and her colleagues, reported in the December 2004 issue of the American Journal of Clinical Nutrition, showed that autistic children had lower levels of glutathione compared to normal controls, and may therefore have had a significant reduction in the ability to detoxify reactive oxygen species.

James says the abnormal profile "suggests that these children may have an increased vulnerability to pro-oxidant environmental exposures and a lower threshold for oxidative neurotoxicity and immunotoxicity." Speaking at the XXII International Neurotoxicology Conference in September 2005, she presented evidence that multiple genetic polymorphisms affecting glutathione pathways may interact to produce a chronic metabolic imbalance that could contribute to the development and clinical symptoms of autism. Her paper in the American Journal of Clinical Nutrition reported that low glutathione levels in many autistic children were reversible with targeted nutritional intervention, but the ramifications of this finding are still unclear.

Manganese. As an essential nutrient, manganese is required for normal development; the reference dose for manganese is 0.14 mg/kg/day. Chronic occupational exposure to high levels of this metal is associated with manganism, a condition reminiscent of Parkinson disease that is characterized by tremors, rigidity, and psychosis. The illness is seem primarily among miners.

Animal studies published in the August 2005 issue of Neurotoxicology by David Dorman, director of the division of biological sciences at the CIIT Centers for Health Research in Research Triangle Park, North Carolina, suggest that the fetus is protected to a certain extent from maternally inhaled manganese. According to Dorman, children are exposed to manganese primarily by ingesting it, but he knows of no link between childhood exposure to manganese and later Parkinson disease.

Nevertheless, because manganese affects the adult brain, people suspect that the developing brain may be even more susceptible to harm from this metal, and recent research has unveiled a new cause for concern: In the January 2006 issue of EHP[5], child psychiatry professor Gail Wasserman and colleagues from Columbia University reported that Bangladeshi children who drank well water with high concentrations of naturally occurring manganese had diminished intellectual function. The researchers noted that the bioavailability of manganese in water is higher than that of manganese in food. They also pointed out that about 6% of U.S. wells have a high enough manganese content to potentially put some children at risk for diminished intellectual function.

The cellular and molecular mechanisms of manganese neurotoxicity are not well understood. The dopaminergic system in the basal ganglia, which is affected in Parkinson disease, may be involved, but this hypothesis is controversial. Tomas Guilarte, a professor of molecular neurotoxicology at the Johns Hopkins Bloomberg School of Public Health, described research on these systems in nonhuman primates at the XXII International Neurotoxicology Conference. According to Guilarte, unpublished positron-emission tomography studies of the basal ganglia show that "manganese does appear to have an effect on dopaminergic neurons." Guilarte found that the more manganese the animals received, the less dopamine was released through the actions of amphetamine (which is used to induce the release of the neurotransmitter). "This does not mean that manganese causes Parkinson's disease, merely that it has an effect on those neurons," he says. This is the first report of an in vivo effect on dopamine release by manganese.

PCBs, PBDEs, and pesticides. Many chemicals raise concerns because of their persistence in the environment and their tendency to bioaccumulate in animal tissues. They are typically synthetic molecules that were designed for use in everyday products, such as electrical equipment, computers, furniture, and pesticides.

PCBs appear to be present in all parts of the food chain, and humans are exposed to these molecules primarily through the ingestion of animal fat. The toxicity of these chemicals was first recognized after mass poisonings in Japan in 1968 and Taiwan in 1979. Children born to women who had ingested contaminated cooking oil in Taiwan had a number of developmental abnormalities, including psychomotor delay and lower scores on cognitive tests, according to a report in the 15 July 1988 issue of Science.

Since those earlier observations, several studies have described a connection between prenatal exposure to PCBs and delayed cognitive development and lower IQ. For example, a study in the 10 November 2001 Lancet reports those infants and young children exposed to PCBs through breast milk scored lower on tests of psychomotor and mental development. The mothers were exposed to normal background levels of PCBs in Europe. In response to such studies, the U.S. Food and Drug Administration set tolerance levels for PCBs in a number of consumer products, such as milk and manufactured dairy products (1.5 parts per million), poultry (3.0 parts per million), and baby food (0.2 part per million).

PBDEs are widely used as flame retardants in consumer products. The effects of PBDEs on humans is not clear, but animal toxicity studies described in volume 183 (2004) of...
Reviews of Environmental Contaminants and Toxicology show that PBDEs can cause permanent learning and memory impairments, hearing deficits, and behavioral changes. There is a growing concern about PBDEs because they appear to be accumulating in human tissues. Andreas Sjodin, a toxicologist at the CDC, and colleagues found a trend toward increasing concentrations of PBDEs in human serum taken from sample populations in the southeastern United States from 1985 through 2002, and in Seattle, Washington, from 1999 through 2002. This report appears in the May 2004 EHP[6]. Several studies have also discovered PBDEs in human breast milk. The current EPA reference dose for PBDEs is 2 mg/kg/day.

As for pesticides, it's been suggested by zoologist Theo Colborn of the University of California that every child conceived today in the Northern Hemisphere is exposed to these chemicals from conception through gestation and beyond. Some pesticides appear to be more harmful than others, and so the reference dose varies somewhat from one compound to another.

The effects of pesticides on the developing brain have been investigated in human epidemiologic studies and in laboratory experiments with animals. Vincent Garry, a professor of environmental medicine at the University of Minnesota, and his colleagues found that children born to applicators of the fumigant phosphine were more likely to display adverse neurological and neurobehavioral developmental effects. The herbicide glyphosate was also linked to neurobehavioral effects, according to the same report, which appeared in the June 2002 issue of EHP Supplements[7]. Another epidemiologic study, reported in the March 2005 issue of NeuroToxicology, showed that women who were exposed to organophosphate pesticides in an agricultural community in California had children who displayed adverse neurodevelopmental effects, and that higher levels of pesticide metabolites in maternal urine were associated with abnormal reflexes in the women's newborn children.

Many PCBs, PBDEs, and pesticides are the subject of the 2001 Stockholm Convention on Persistent Organic Pollutants, which became international law in May 2004. The goal of the treaty is to "rid the world of PCBs, dioxins and furans, and nine highly dangerous pesticides," according to the United Nations Environment Programme. Implementation of the treaty has significant practical challenges, however, including the difficulty of eliminating one persistent pollutant without creating another (for example, when burning PCBs yields by-products such as dioxins and furans). Not Immune to Harm

Exposure to a neurotoxicant may not be the only way to disrupt the natural growth of the brain. Scientists are now looking at the subtle physiological effects of immunotoxicants and infectious agents on biological events during development.

It turns out that mothers who experience an infection during pregnancy are at a greater risk of having a child with a neurodevelopmental disorder such as autism or schizophrenia. For example, prenatal exposure to the rubella virus is associated with neuromotor and behavioral abnormalities in childhood and an increased risk of schizophrenia spectrum disorders in adulthood, according to an article in the March 2001 issue of Biological Psychiatry. Rubella has also been linked to autism: some 8-13% of children born during the 1964 rubella pandemic developed the disorder, according to a report in the March 1967 Journal of Pediatrics. The same study also noted a connection between the rubella virus and mental retardation.

Some epidemiologic studies have found an increased risk of schizophrenia among the children of women who were exposed to the influenza virus during the second trimester of pregnancy, according to a report in the February 2002 Current Opinion in Neurobiology. In the August 2004 Archives of General Psychiatry, Ezra Susser, head of epidemiology at Columbia University's Mailman School of Public Health, and his colleagues reported that the risk of the mental disorder was increased sevenfold if the schizophrenic patient's mother had influenza during her first trimester of pregnancy. A prospective birth cohort study in the April 2001 Schizophrenia Bulletin found that second trimester exposure to the diphtheria bacterium also significantly increased the risk of schizophrenia.

How might infectious agents cause these disorders? According to John Gilmore, a professor of psychiatry at the University of North Carolina at Chapel Hill, maternal infections during pregnancy can alter the development of fetal neurons in the cerebral cortex of rats. The mechanism is far from clear, but signaling molecules in the mother's immune system, called cytokines, have been implicated. Speaking at the XXII International Neurotoxicology Conference, Gilmore described in vitro experiments showing that elevated levels of certain cytokines-- interleukin-1?, interleukin-6 and tumor necrosis factor-alpha (TNF- alpha)-- reduce the survival of cortical neurons and decrease the complexity of neuronal dendrites in the cerebral cortex. "I believe that the weight of the data to date indicates [that the maternal immune response] can have harmful effects," says Gilmore.

Inflammatory responses in the mother may not be the only route to modifying the fetal brain. The University of California, Davis, Center for Children's Environmental Health and Disease Prevention is conducting a large study of autistic children in California called CHARGE (Childhood Autism Risks from Genetics and the Environment), which suggests that the child's immune system may also be involved. According to Pessah, the study principal investigator, children with autism appear to have a unique immune system. "Autistic children have a significant reduction in plasma immunoglobulins and a skewed profile of plasma cytokines compared to other children," he says. "We think that an immune system dysfunction may be one of the etiological cores of autism."
He continues, "We know that many of the things that kids are exposed to these days are immunotoxicants... We have evidence that ethylmercury and thimerosal alter the signaling properties of antigen-presenting cells, known as dendritic cells, at nanomolar levels." Since each dendritic cell can activate 250 T cells, any dysregulation will be magnified, he says. "Add to that a genetic abnormality in processing immune information, and there could be a problem."

Such problems might extend to the central nervous system. The brains of individuals who have a neurodevelopmental disorder also show evidence of inflammation. In the January 2005 issue of the Annals of Neurology, Carlos Pardo, an assistant professor of neurology and pathology at the Johns Hopkins University School of Medicine, and his colleagues report finding high levels of inflammatory cytokines (interleukin-6, interleukin-8, and interferon-gamma) in the cerebrospinal fluid of autistic patients. Glial cells, which serve as the brain's innate immune system, are the primary sources of cytokines in the central nervous system. So it may not be surprising that Pardo's team also discovered that glia are activated—showing both morphological and physiological changes—in postmortem brains of autistic patients.

The recognition that the immune system is involved in neurodevelopmental disorders is changing people's perceptions of these conditions. "Historically, scientists have focused on the role of neurons in all kinds of neurological diseases," Pardo says, "but they have generally been ignoring the [glia]." He adds, "In autism, it could be that the [glia] are responding to some external insult, such as an infection, an intrauterine injury, or a neurotoxicant."

According to Pardo, it's still not clear whether the neuroimmune responses associated with autism contribute to the dysfunction of the brain or whether they are secondary reactions to some neural abnormality. "John Gilmore's work [showing that cytokines can be harmful to brain cells] is quite interesting and important," he says. "However, in vitro studies may produce results that don't reflect what occurs under in vivo conditions. Cytokines like TNF-alpha may be beneficial for some neurobiological functions at low concentrations, but may be extremely neurotoxic at high concentrations." Lending Brain Power to Exposure Assessment

The medical and scientific communities recognize the colossal challenges involved in identifying the ultimate causes of neurodevelopmental disorders. This is complicated by the sheer numbers of potential exposures involved. More than 67% of the nearly 3,000 chemical compounds produced or imported in amounts exceeding 1 million pounds per year have not been examined with even basic tests for neurotoxicity, according to Toxic Ignorance, a 1997 analysis by Environmental Defense.

In the past few years, several large projects have been proposed, and funding by the NIH has been increased. For example, the NIH boosted its support for autism research from $22 million in 1997 to $100 million in 2004. In 2001, the NIEHS and the EPA jointly announced the creation of four new children's environmental health research centers (including the one at the University of California, Davis), which focus primarily on neurodevelopmental disorders. More recently, the proposed multibillion-dollar National Children's Study, which is cosponsored by the Department of Health and Human Services and the EPA, has been designed to follow nearly 100,000 children over the course of 21 years. The investigators plan to study the effects of environmental factors on children's growth and development, including impacts on learning, behavior, and mental health. Study investigators hope to enroll the first participants in early 2007.

Scientists also see the need for designing better studies. In neurodevelopmental studies, as in any other field, the quality of a study is only as good as all of its parts. Jean Harry, head of the NIEHS Neurotoxicology Group, says, "You can have a valid assessment of behavior, but in the absence of good exposure data, a causative association with environmental factors will be compromised."

In a bid to address the difficulties faced by epidemiologic studies that look for neurodevelopmental effects from in utero chemical exposure, a working group of 20 experts gathered in September 2005 under the auspices of the Penn State Hershey Medical Center, coincident with the XXII International Neurotoxicology Conference. The goal of their day-long session was to develop a scheme of best practices for the design, conduct, and interpretation of future investigations, as well as the practical inclusion of new technologies, such as imaging.

At one point in the dialogue, the group recognized that perhaps the greatest challenge in these studies was determining how to evaluate in utero exposures to environmental chemicals. "Quite often the very nature of epidemiological studies limits the ability to perform accurate exposure assessments," says Harry, who was part of the expert group. "Such exposures may have occurred in the distant past, they may have been unknown, or they may have been in conjunction with many other compounds."

The group therefore recommended that actual measurements, even if indirect, are better than methods based on subject recall. It also recommended that a well-defined hypothesis should form the foundation of in utero studies for assessing neurodevelopmental outcomes. "[These and other] conclusions will move the science forward by describing methods that should improve interstudy comparisons, and they offer ways in which research results should be reported to the scientific and medical communities," says Judy LaKind, an adjunct associate professor of pediatrics at the Hershey Medical Center and a member of the workshop steering committee. The complete workshop report will be published in an upcoming issue of NeuroToxicology. Imagining the Big Picture
The challenges of addressing neurodevelopmental disorders are more than scientific. The difficulties come together at a crossroads where the communication of knowledge, the treatment of patients, and the regulation of potentially toxic chemicals meet. Says Herbert, "Evidence-based medicine has not yet developed standards for assessing, or practices for treating, the impacts of chronic, multiple low-dose exposures." Rather than waiting, she says, patients and parents of patients are turning to alternative medicine to address their concerns.

That's not always a good thing, especially when patients and parents may be misinformed. Kathy Lawson, director of the Healthy Children Project at the Learning Disabilities Association of America, says there is a disconnect between scientific knowledge and the public's awareness of ways to reduce the incidence of some disorders. "In my visits to various organizations, I've discovered that people are completely unaware that there is a connection between environmental toxicants and their health," she says. "Even pediatricians often don't know about these things," she adds.

Educating the public is only part of the solution. Elise Miller, executive director of the nonprofit Institute for Children's Environmental Health, thinks that federal regulatory agencies do not adequately protect children's health. "The Toxic Substances Control Act, which was passed thirty years ago, needs a major overhaul to ensure neurotoxicants and other chemicals are prioritized, screened, and tested properly," she says. "Currently, there are too many chemicals on the market and in the products we use every day for which there is no toxicity data."

Some politicians agree with these sentiments. In July 2005, Senator Frank R. Lautenberg (D-NJ) introduced the Child, Worker, and Consumer Safe Chemicals Act, which initially calls for chemical manufacturers to provide health and safety information on the chemicals used in certain consumer products, among them baby bottles, water bottles, and food packaging. If passed into law, the bill, coauthored by Senator James Jeffords (I-VT), would require all commercially distributed chemicals to meet the new safety measures by 2020.

The human brain is often touted as the most complex structure in the known universe. The developmental process that produces this remarkable entity may also be among the most delicate in nature. As one scientist put it, "The brain doesn't like to be jerked around." That kind of fragility makes it difficult for scientists to untangle genetic influences from what often may be subtle environmental assaults. Even so, the catalogue of harmful environmental agents will undoubtedly continue to grow as scientists learn more about the interactions between the developing brain and its environment. The hope is that enough good minds will use that catalogue to create a future with healthier brains and more peace of mind for parents and society alike.

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