

Testimony to Support S1115 - An Act to Protect Child Development

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May 23, 2001

My name is Jill Stein. I am here today in support of An Act to Protect Child Development, S1115 (see attached summary for background information on the bill and rationale for it). I am representing Greater Boston Physicians for Social Responsibility and speaking as a physician in clinical practice caring for adolescents. I'm also speaking as a physician involved in medical education - as an Instructor at Harvard Medical School, and as co-author of curricula designed to update physicians on new developments in environmental health, particularly with regard to child development and chemical exposures. Towards this end I have recently co-authored a report entitled *In Harm's Way: Toxic Threats to Child Development*. The report examines a large body of medical literature indicating that common household and environmental chemicals contribute to learning and behavioral problems in children. I'd like to briefly summarize the major findings of the report, since they illustrate the urgent need for a more pro-active, flexible regulatory framework, a goal this bill begins to explore. I'd also like to provide a physician's perspective on the bill as an important step towards incorporating principles of sound medical decision-making into environmental public health decisions.

FINDINGS OF *IN HARM'S WAY: TOXIC THREATS TO CHILD DEVELOPMENT*

Developmental disabilities are widespread, and result from complex interactions of genetic, nutritional, social and toxicologic (chemical) factors that impact children during vulnerable early periods of development. Among these various factors, chemical exposures deserve special scrutiny because they are readily preventable. (See Figure 1: Framework for Understanding.) In reviewing the effects of these chemical exposures on child development, it is clear that children have been and continue to be harmed by toxic exposures, and that the current regulatory framework has failed to protect children.

HEALTH CONSEQUENCES OF REGULATORY INERTIA

Lead: A Case Study In Neglected Early Warnings

The harmful effects of lead on children, for example, were evident 100 years ago. In spite of clear-cut early warnings, however, the regulatory system was unable to respond until rigorous, detailed criteria for "proof" were fully met. The process of accumulating data sufficient to meet these standards took over 70 years, by which time generations of children had been injured, and lead had become the entrenched, global contaminant that it is today.

Shortly after lead was added to paint, more than 100 years ago, it became clear that children who ate these paint chips developed coma, paralysis and often died. It wasn't until 40 years later, with the publication of longer follow-up observations, that the permanent effects of lead on child development were noted. For the first time children who were lucky enough to survive lead poisoning were noted to have persistent

impairments in intellect, behavior, and sensory-motor function.ⁱ Subsequent studies began to suggest that lower levels of lead exposure might be associated with neurological damage as well.ⁱⁱ

A specific toxic threshold for lead was established for the first time in the 1960s. (See Figure 2: Declining Threshold of Harm: Lead.) At that time a toxic blood lead level was set at 60 µg/dl, only modestly below the level at which coma and paralysis occur (80 µg/dl).ⁱⁱⁱ Improvements in study design over the next 30 years led to the discovery of harmful effects at progressively lower levels of exposure.^{iv} After repeated downward revisions, the toxic threshold for lead was most recently set at 10 µg/dl, the 1990 standard that still holds today. Recent epidemiologic studies, however, indicate that lead exposure is harmful even below the current 10 µg/dl standard.^{v vi vii viii ix} Several studies suggest that there is no threshold below which lead exposure is without adverse effects,^{x xi} and, importantly, that for a given increment in blood lead, the associated impact on IQ is greater *below* a blood level of 10µg/dl than above.^{xii xiii} Recent studies also suggest that low-level lead exposure is also associated with impaired attention, hyperactivity, and antisocial and aggressive behavior.^{xiv xv xvi xvii xviii xix} Some of these effects have been observed at very low levels of exposure, and may occur in the absence of detectable IQ effects.^{xx xxi xxii xxiii}

While the effects of low-level lead exposure on the average child may be subtle, these effects are profound when applied over large populations. As shown in Figures 3 and 4, (Population Effects of A Small Shift in Average IQ), a downward shift of a mere 5 points in the average IQ results in a 50% increase in the numbers of functionally mentally retarded, and a comparable decrease in the numbers of gifted individuals in the population. This small shift in average IQ therefore has enormous implications for society, translating, for example, into increased needs for special education and services as well as a significantly diminished intellectual capacity within the population as a whole.

The growing profile of developmental impacts from low-level lead exposure is of substantial concern, considering that 4% of all American children^{xxiv} and as many as 36% of inner city African American children^{xxv} exceed even the current, increasingly obsolete toxic threshold of 10 µg/dl.

Mercury and PCBs: Widespread, Persistent Harm From Delayed Regulatory Response

Like lead, there were also early warnings for mercury and PCBs. The effects of these compounds on children were first noted in catastrophic epidemics in which exposed children were born with severe disabilities such as mental retardation and cerebral palsy. As in the case with lead, more recent research has revealed toxicity at progressively lower exposures, illustrating the same "declining threshold of harm" commonly observed with improved understanding of developmental toxicants. (See Figure 5. Mercury: Declining Threshold of Harm.) For mercury and PCBs, as with lead, recent studies demonstrate adverse effects at or near background population exposures. The problems associated with mercury and PCBs include impairments in attention, memory, learning, social behavior and IQ.^{xxvi xxvii xxviii xxix xxx xxxi xxxii xxxiii xxxiv xxxv xxxvi}

The High Price of Waiting for “Incontrovertible” Proof

The understanding of these few well-studied chemicals has been hard won, and predicated on tragic "body counts" of childhood injury and death. For example, it is estimated that over 68 million children were exposed to toxic levels of lead *from leaded gasoline alone* over the decades required to accumulate proof sufficient to motivate a regulatory response.^{xxxvii} In the 50 years since the first warnings about mercury toxicity, mercury contamination of fish has progressed to the point that over 10% of women now exceed the safe limit for mercury exposure,^{xxxviii} putting their future children at risk for learning disabilities. The National Academy of Sciences has estimated that over 60,000 children are born each year at risk for learning problems because of this exposure to mercury resulting from maternal consumption of contaminated fish.^{xxxix}

A tragic price is also being paid for delayed response to the dangers of PCBs. Although the dangers of PCBs were evident for decades,^{xl xli xlii xliii} PCB production continued until 1977, by which time PCBs had become ubiquitous global contaminants. Recent studies have revealed that pervasive impairments in learning, behavior and memory are occurring in children throughout industrialized societies at current background exposures.^{xliv xlv xlvi}
^{xlvii xlviii} (See Figure 6. PCBs: Inadequate Margin of Safety) It is estimated that the amount of PCBs still in use in older products or in landfills is greater than the quantity that has been released as of yet. These unreleased PCBs remain a potential reservoir for ongoing and future contamination of the environment, our food and ultimately ourselves.^{xliv}

Regulatory Inertia and the Magnitude of the Current Chemical Threat

It is important to put these findings into context in the modern chemical landscape. Lead, mercury and PCBs are among the very few substances whose effects on child development are well-understood. Unfortunately, for most of the 80,000 chemicals licensed for manufacture, there is little data with which to evaluate potential risks to children. Among the 3,000 chemicals produced in highest volume (over one million pounds per year), three-fourths have had no testing for effects on children,^{li} and only 12 have been adequately tested (according to EPA guidelines) for their effects on the developing brain.^{lii} This is a matter of concern since the fetus and child are already exposed to untold numbers, quantities and combinations of substances whose safety has not been established.

Clearly there are enormous limits to "currently available knowledge". While it has required 50-100 years of intensive study to understand each of a handful of well-characterized toxicants, new chemicals are rolled-out at a rate of 2000 per year.^{liii} Slow progress in understanding results partly from ethical constraints in testing: We don't test suspected toxicants on pregnant women and infants for obvious ethical reasons. In contrast, the manufacture and emission of such untested, potential toxicants is not subject to ethical constraint. As a result, the public - including the vulnerable fetus and child - is widely exposed to untested chemicals, and has become the unwitting subject of "a vast toxicologic experiment that will affect generations to come."^{liiv} Because this experiment is unmonitored and uncontrolled, the results will remain largely unknown.

ADVANCES IN DECISION-MAKING IN CLINICAL MEDICINE:
AN EVIDENCE-BASED, VALUE-DRIVEN, PARTICIPATORY
DECISION-MAKING MODEL
Implications For Decision-Making In Environmental Public Health

The complexities of environmental health policy and regulation are not unlike dilemmas encountered in clinical medicine. In the day-to-day practice of health care, clinicians and patients likewise confront urgent, complex problems in the face of incomplete and often inadequate information - - whether to treat for possible meningococcal meningitis in the absence of confirming evidence, (an infection that is often fatal within hours if not treated); when to operate for possible appendicitis; when to perform expensive and potentially dangerous diagnostic procedures (ie brain biopsies); whether to treat a breast cancer with mastectomy or chemotherapy; whether to screen for diseases (such as early prostate cancer) in which early treatment may be harmful and is as yet without proven benefit. When facing such questions, it is standard practice in medical decision-making to raise and lower the standard of proof necessary for taking action, and to adjust the bar of proof in response to a variety of considerations. In suspected appendicitis, for example, where untreated disease carries a high risk of death and the treatment itself is relatively benign, there is a low threshold for action. Traditionally, we have expected, in fact desired, a relatively large number (~ 15%) of "false positive" appendectomies (appendectomies performed when there actually was no appendicitis, as determined by pathology reports after surgery), in order to assure that this highly treatable, and otherwise very dangerous disease is not missed. In the case of breast cancer, however, where untreated disease is usually fatal but the treatment is highly adverse, the standard of proof is much higher, and a "false-positive" rate of 15% would be appalling.

Rigid adherence to an inflexible standard of scientific certainty before taking action would be inappropriate (not to mention unethical) in clinical medicine. It is also inappropriate in the realm of public health. The current regulatory requirement for incontrovertible evidence, coupled with the slow rate at which evidence emerges, limits the ability of the regulatory system to respond in a time frame compatible with public health protection. This puts generations at risk of harm from pervasive and often irrevocable threats to human health.

Critical Elements of Sound Decision-Making

Patient values and preferences, informed by best available scientific information, are an essential component of decision-making in clinical medicine.^{lv lvi lvii lviii lix lx} Likewise, informed public input is a much needed, critical component of sound environmental regulatory decisions. Critical elements of sound decision-making - as specified in the current medical model - are listed below. These critical elements can be readily applied to decisions in environmental public health, as illustrated by simply changing a word or phrase within each of the critical elements listed below.

1. Meaningful communication of scientific information to the patient/public;
2. Flexible standards of scientific certainty when facing potential health/ environmental health impacts of different magnitude, urgency and permanence;
3. Participatory decision-making, in which the values of the patient/community become an integral part of therapeutic/policy deliberations and decisions;
4. Routine consideration of a variety of alternative therapeutic strategies/policy options.

The chart below illustrates in greater detail how these critical elements are currently applied in clinical medicine, and how they might be applied in environmental public health decisions.

Models For Sound Decision-Making
in Clinical Medicine and in Environmental Public Health:
A Comparison of Critical Elements

COMPONENT	DECISION-MAKING IN CLINICAL MEDICINE	DECISION-MAKING IN ENVIRONMENTAL PUBLIC HEALTH (as proposed in Act to Protect Child Health and Development)
Process Characteristics		
Communication of technical information	Requirement of “informed consent” assures relevant scientific information is communicated to patient in lay terms;	Citizen panels draw on wide variety of technical consultants to communicate relevant scientific information in terms that can be understood by the public;
Standards of scientific certainty and thresholds for taking action	Flexible threshold of scientific certainty as a basis for taking action;	Flexible threshold of scientific certainty as a basis for taking action;
Decision-making process	Provider and patient jointly evaluate options in context of patient values and concerns;	Consensus panels, assisted by technical consultants, evaluate policy options in context of community values and concerns;
Decision-making authority	Joint decision-making: health care provider advises on medically appropriate options; patient makes ultimate decision on course of treatment;	Consensus panels review policy options, engaging & educating the wider public in the process. Under the proposed bill, however, decision-making authority is retained by regulatory and legislative bodies as currently construed.
Content Characteristics		
Evidence	Evidence based decision-making: Review all relevant, available scientific evidence;	Evidence based decision-making: Review all relevant, available scientific evidence;
Uncertainty	Consider areas of uncertainty & their potential implications;	Consider areas of uncertainty and their potential implications;
Risks and Benefits of Alternatives	Review potential risks and benefits of proposed treatment, treatment alternatives, and the option of no treatment;	Review potential risks and benefits of proposed regulation/policy, policy alternatives, and the option of no policy/regulation;

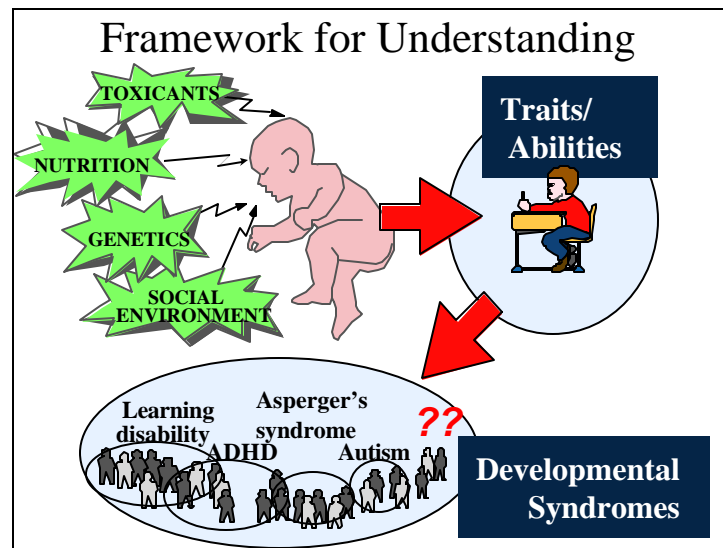
CONCLUSION

An Act to Promote Child Health and Development establishes an improved environmental public health decision-making process. Specifically, the bill promotes the application of principles of medical decision-making to environmental public health decisions. This model of decision-making is best characterized as evidence based, value-

driven, and participatory, engaging above all, those whose lives may be affected by the decisions at hand. In addition, this decision-making process considers the implication of uncertainties, and systematically evaluates a variety of alternative solutions.

By establishing citizen panels to review environmental threats to child health, the bill facilitates better communication of technical information from a wide variety of consultants to a broad spectrum of the community. At the same time, the panels also facilitate an important process of exploring and articulating community values and priorities. In so doing, this bill will help generate more informed public debate, dialogue, and participation in decisions that affect our lives. In the same way that physician-patient collaboration improves decision-making in clinical medicine, facilitating informed participation of the public in public health decisions is likely to improve regulatory decision-making as well as public confidence in those decisions. Better decisions in the public arena, as in the private, will ultimately benefit the health of our children now and in generations to come.

Figure 1

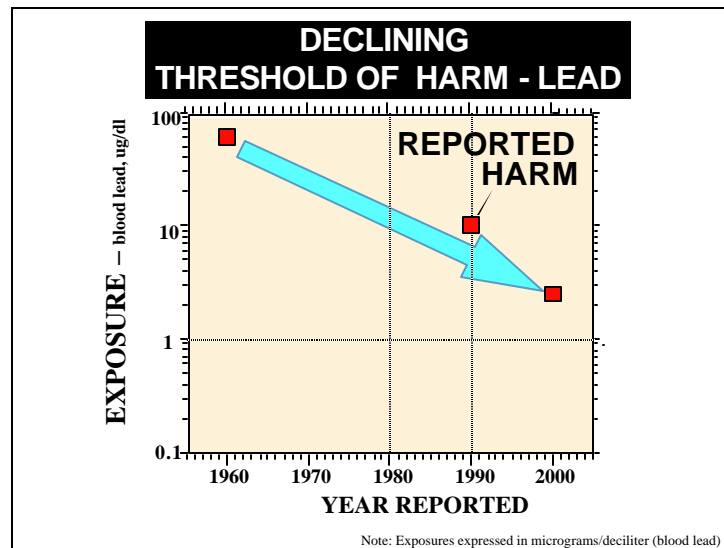


- **Multiple factors interact in complex ways during windows of vulnerability to affect child development.**
- **These factors include nutrition and chemical exposures during vulnerable periods in early life, genetic factors (which should not be viewed in isolation), and the social environment.**
- While research typically focuses on one domain at time, it is increasingly recognized that complex interactions are most important. For example, several genes have been identified that influence susceptibility to environmental chemicals, including genes that affect lead absorption and metabolism (such as the vitamin D receptor and delta aminolevulinic acid dehydratase genes) (1-9), and genes that affect the metabolism of and susceptibility to organophosphate (OP) pesticides (such as the paroxonase and acetylcholinesterase genes). (10-12)

- Among the multiple causes of disability, chemical exposures deserve special scrutiny because they are *preventable* causes of harm.
- The developmental outcomes – which result from interacting genetic, toxicologic, nutritional, and social influences - can be viewed as continuous traits, or as categorical diagnoses.

1. Smith CM, Wang X, Hu H, et al. A polymorphism in the delta-aminolevulinic acid dehydratase gene may modify the pharmacokinetics and toxicity of lead. *Environ Health Perspect* 103(3):248-253, 1995.
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Figure 2



Lead is the first, and perhaps best-understood example of a common chemical that harms learning and behavior. Because lead was one of the first pollutants recognized as harmful to a child's brain, its well-documented, long history provides a useful case study in the science of toxicology, and in the political context of regulation. This history of lead tragically illustrates that the scientific understanding of neurodevelopmental toxicity emerges very slowly.

The understanding of lead has advanced over the period of a full century, during which time the recognized "toxic threshold," (the lowest exposure thought to be harmful), has relentlessly declined. While very high lead exposures were recognized to cause encephalopathy, coma, and death in children as early as 1900,(1,2) residual effects in survivors of childhood lead poisoning went unrecognized for decades.

The enduring effects of lead poisoning on child development became apparent only with the publication of longer follow-up observations in the 1940s, which noted persistent impairments in intellect, behavior, and sensory-motor function. (3) Subsequent studies began to suggest that lower levels of lead exposure might be associated with neurological damage as well.(4)

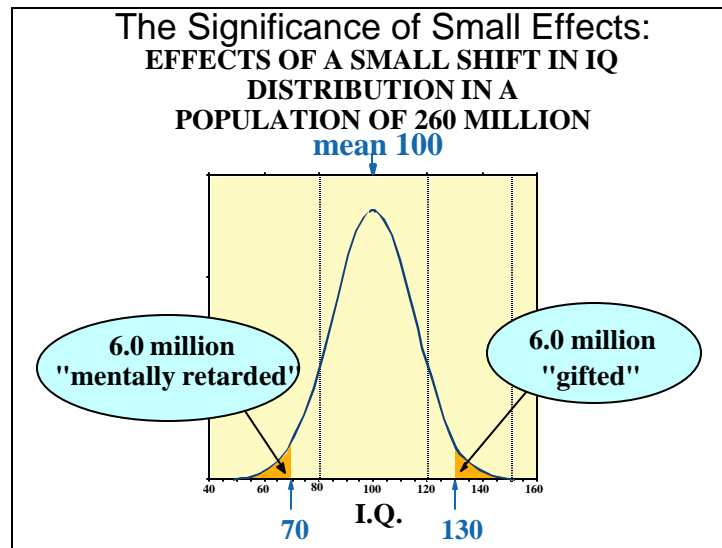
A specific toxic threshold for lead was established for the first time in the 1960s. At that time a toxic blood lead level was set at 60 micrograms/dl, only modestly below the level at which encephalopathy occurs (80 micrograms/dl).(5) With improvements in study design over the next 30 years, research revealed effects of lead on IQ at progressively lower levels of exposure.(6) After repeated downward revisions, the toxic threshold for lead was most recently set at 10 micrograms /dl, the "official" 1990 standard that still holds today. Subsequent analyses have quantified the risk of low-level lead exposure, estimating that an increase in blood lead from 10 to 20 micrograms /dl is associated with an average IQ loss of 2-3 points.(7,8)

Recent epidemiologic studies suggest that there is no threshold below which lead exposure is without harmful effects, (9,10,11,12) and that for a given increment in blood lead, the associated impact on IQ is greater below a blood level of 10 micrograms/dl than above.(13) The fact that adverse effects from lead exposure are apparent well below the toxic threshold of 10 micrograms/dl is no surprise, considering that this currently recognized limit of "safe" exposure is nearly 13% of the blood lead concentration associated with encephalopathy, and 8% of the lethal blood lead level. Indeed, it would be surprising if *some* neurological harm were not occurring a mere one order of magnitude below the *lethal* exposure level.

The growing profile of developmental impacts from low-level lead exposure is of substantial concern, considering that 4% of all American children (14) and as many as 36% of inner city African American children (15) exceed even the current, increasingly obsolete toxic threshold of 10 micrograms/dl.

1. Needleman HL. The future challenge of lead toxicity. *Environ Health Perspect* Nov 89:85-89, 1990.
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4. WHO, Ibid. pp 153-154
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Figure 3

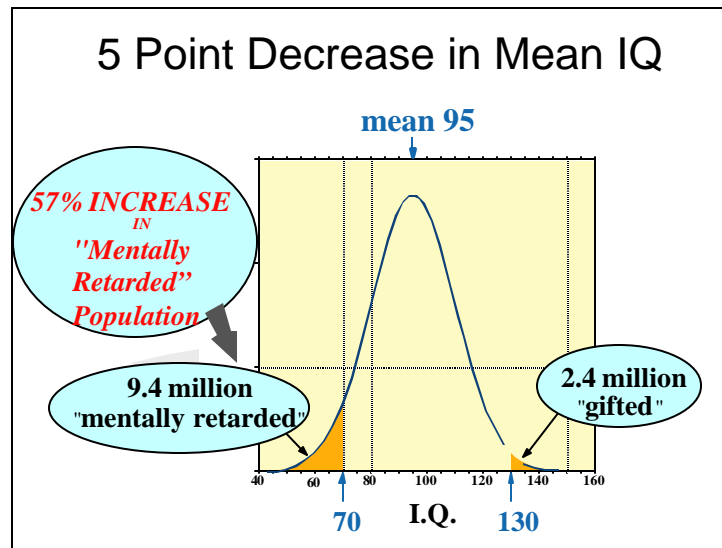


The effect of low lead exposures on IQ is relatively small for the average individual, reducing IQ by only a few points. It's important to remember, however, that impacts that are marginal for the average individual can have profound effects when applied over large populations,^[1] in effect, shifting the population distribution curve for the parameter of concern. That parameter might be IQ, as in this case, or any other cognitive or behavioral function such as memory or attention. Such shifts have dramatic effects at the high and low ends of the distribution curve, often referred to as the "tails." This is illustrated in a hypothetical population of 260 million with an average IQ of 100. The area under the left "tail" of the curve represents the 2.3% of the population with an IQ <70, the score used to define mental retardation. In a population of 260 million, about 6 million people would fall below this line.

Source: Weiss, B. Endocrine disruptors and sexually dimorphic behaviors; a question of heads and tails. *Neurotox* 18:581- 586, 1997.

Graphics: adapted from above reference

Figure 4

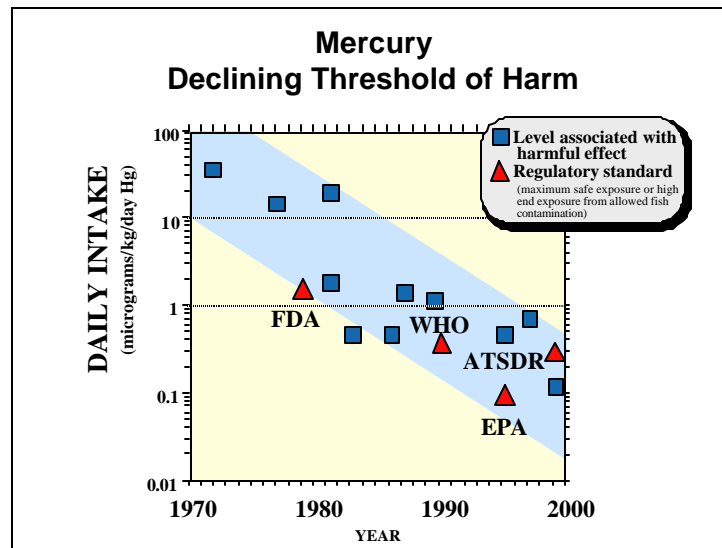


This chart shows what happens when the average IQ is shifted by 5 points from 100 to 95. Now, 3.2% of the population, or 9.4 million people have an IQ below 70. This represents more than a 50% increase in the numbers of mentally retarded. The numbers of gifted, defined as those with IQ's greater than 130, have declined by more than 50% from 6 million to 2.4 million. Thus a small shift in average IQ results in greatly increased need for special education and services, as well as diminished intellectual capacity within the population as a whole.

Source: Weiss, B. Endocrine disruptors and sexually dimorphic behaviors; a question of heads and tails. *Neurotox* 18:581-586, 1997.

Graphics: adapted from above reference

Figure 5



This graph displays the apparent toxic threshold for mercury as it was identified at various points in time over the past three decades. It illustrates the tendency for apparent toxic thresholds to decline with advancing knowledge. Exposure to mercury is shown in micrograms per kilogram per day on the vertical axis, and year is shown on the horizontal axis. The initial Iraqi toxic threshold is shown as the upper left-hand point on the graph. Within a few years of this observation, it became apparent that many children exposed prenatally to lower levels of mercury were delayed in learning to walk and talk, in spite of apparently “normal” development in infancy. (1) Subsequently, a variety of studies on diverse populations have established progressively lower thresholds for mercury effects by using increasingly sensitive exposure and outcome measures, and better statistical methods of analysis.(2-11)

Recently, a large study in the Faroe islands has identified deficits in language, memory, and attention that occur at prenatal mercury exposures *under* 0.85 micrograms per kilogram per day. (This is the second square from the right-hand edge of the figure.) This exposure is less than 3% of the toxic threshold identified in the initial observations from the Iraqi epidemic. The presence of mercury effects below this level of 0.85 micrograms/kg/day implies that the actual threshold, if one exists, is lower.

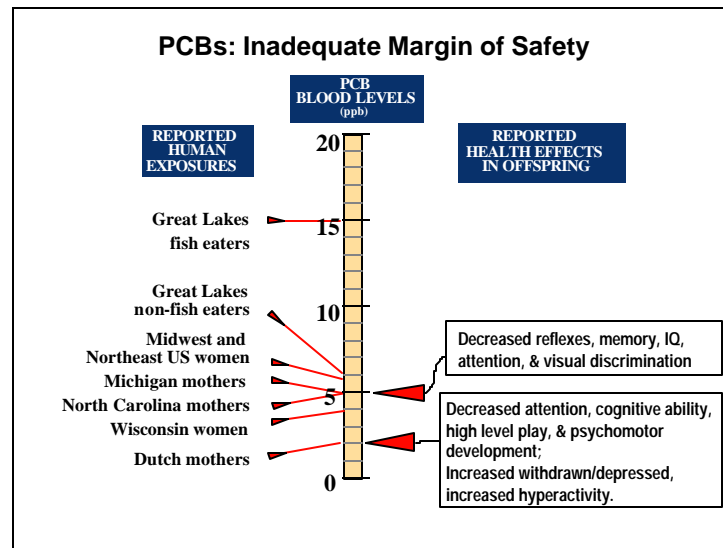
The black squares on the graph represent prenatal mercury exposures associated with adverse neurodevelopmental outcomes. The gray triangles represent World Health Organization (WHO), EPA, and Agency for Toxic Substances and Disease Registry (ATSDR) recommended limits for human mercury exposure. The standard issued by the FDA, it should be noted, regulates the level of mercury in fish, rather than in people. As a result, a wide variety of exposures may occur within the FDA regulatory limit, depending on how much and how often one eats fish, and the mercury level of the fish consumed. The indicated exposure is that of a 60 kg woman eating at the high end of fish consumption (100grams/day, the 95-97th percentile), eating fish that are contaminated at the FDA permitted limit. In this worst-case scenario, the woman is exposed to 1.65 µg/kilogram/day, or about 16.5 times EPA’s recommended safe limit.

Notes:

[1.] Studies of the neurodevelopmental effects of mercury generally use hair or blood levels as markers of exposure, since these are more accurate indicators of exposure than dietary surveys. Health-based guidelines, however, are expressed as recommended limits of dietary exposure. For the purpose of comparing data between studies, and for comparing effects levels with regulatory guidelines, exposures as indicated by hair and blood levels of mercury have been converted to approximate equivalent dietary exposures. The quantitative relationships between food intake hair and blood levels of mercury are described in the ATSDR Toxicological Profile for Mercury. (12)[2.] Study results that identified a range of exposures within which an effect was observed have been shown at the mid-point of that range. Due to differences in study methods, results are not strictly comparable between studies, and are shown here mainly to indicate general trends over time.

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3. Marsh DO, Myers GJ, Clarkson TW, et al. Dose-response relationship for human fetal exposure to methylmercury. *Clinical Toxicology*, 18(11): 1311-1318, 1981.
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Figure 6



In infancy and early childhood, prenatal PCB exposure is associated with a variety of cognitive impairments (reduced memory and attention, decreased verbal ability, impaired information processing), developmental delays (reduced psychomotor development), and adverse behavioral and emotional effects (decreased sustained activity, decreased high-level play, increased withdrawn and depressed behavior, and increased activity level).(1-7) In preteen years, prenatal PCB exposure is associated with decreased word and reading comprehension, decreased full-scale and verbal IQ, and reduced memory and attention.(9)

This chart illustrates the exposure levels (expressed as maternal blood PCB levels) at which these effects in children have been demonstrated. The left side of the figure shows the PCB blood levels in various populations, in which Great Lakes fish-eaters had the highest exposures. The exposures at which adverse effects have been demonstrated are indicated on the right side of the diagram, at the mid to low end of the exposure spectrum. (10-21)

Note: All health effects shown are associated with prenatal PCB exposure except hyperactivity which is associated with blood levels at 42 months of age.

1. Jacobson SW, Fein GG, Jacobson JL, et al. The effect of intrauterine PCB exposure on visual recognition memory. *Child Dev* Aug;56(4):853-860, 1985.
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