June 9, 2004

William V. Kennedy
Executive Director
Commission for Environmental Cooperation
393, rue St-Jacques Ouest
Bureau 200
Montréal (Québec)
H2Y 1N9 Canada

Re: Taking Stock: A Special Report on Toxic Chemicals and Children's Health in North America (draft 13 April 2004)

Dear Mr. Kennedy:

The American Chemistry Council (ACC) is pleased to offer these comments regarding the April 13 draft *Taking Stock: A Special Report on Toxic Chemicals and Children's Health in North America* (draft report) released by the Secretariat of the North American Commission on Environmental Cooperation (NACEC).

ACC represents the leading U.S. companies engaged in the business of chemistry. Council members apply the science of chemistry to make innovative products and services that make people's lives better, healthier and safer. The Council is committed to improved environmental, health and safety performance through Responsible Care®; common sense advocacy designed to address major public policy issues; and health and environmental research and product testing. The business of chemistry is a $454 billion enterprise and a key element of the nation's economy. It is the nation's largest exporter, accounting for ten cents out of every dollar in U.S. exports.

Protecting the health and well-being of children is a fundamental value the chemical industry shares with society. Children live safer, healthier lives thanks to the development of chemical products and technologies that improve public health and safety. Children also benefit from the chemical industry's enduring commitment to health and environmental research. The ACC's Responsible Care® initiative, a condition of membership, represents a commitment by our members and partners to make continuous progress toward a shared vision of no accidents, injuries or harm to the environment.

The attached comments on the draft report are lengthy and detailed. We believe that they will assist the Secretariat in its discussions and revisions of the draft report.

ACC has also submitted to U.S. EPA several names of qualified scientists to participate in the Expert Review that the Secretariat has planned for this document.

If you have any questions regarding these comments, please contact Lee Salamone of my staff at (703) 741-5212.

Sincerely,

Sarah H. Brozena

Sarah H. Brozena
Assistant General Counsel
Health, Product and Science Policy Team

cc: J. Clifford, Deputy AA, OIA, U.S. EPA
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COMMENTS OF THE
AMERICAN CHEMISTRY COUNCIL
On

Taking Stock: A Special Report on Toxic Chemicals and Children's Health in North America (draft of 13 April 2004)

June 9, 2004
American Chemistry Council
1300 Wilson Boulevard
Arlington, VA 22209
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Table of Contents

Executive Summary

The American Chemistry Council (ACC) finds the draft report to be extremely problematic because it relies on selective citations, presents incomplete analyses of complex scientific issues, and is alarmist in tone. We believe its publication serves more to alarm the public and cause distrust of the three government agencies charged with protecting public health than to inform the public of trilateral efforts to protect children's health.

ACC supports the appropriate use of emissions reporting programs such as the U.S. Toxic Release Inventory (TRI) and the Canadian National Pollutant Release Inventory (NPRI). We believe such programs can provide valuable information that stimulates emission reductions, communicates important environmental information to the public, and documents significant environmental improvements. ACC believes that enhancing regional transparency in emission reporting is a laudable goal. However, the approach taken in the recent *Taking Stock* draft report goes well beyond the previous reports and purports to establish a direct link between TRI/NPRI emissions and health effects. We believe this approach is fundamentally flawed.

The draft report casts aside basic principles of information stewardship. For instance, the draft report does not make use of the full range of up-to-date scientific and epidemiologic literature on the topics contained in the report. It also relies upon methods, lists and analyses that have not been subjected to independent scientific peer review. It is ACC's belief that the authors seek more to influence the reader through the use of emotion, than to inform the reader through evidence –based science.

It is well-documented by U.S. EPA and others that the kind of data used in TRI and NPRI reports only portray a partial picture of environmental emissions. These databases only cover the substances listed, not the entire suite of substances potentially released or naturally occurring in the environment. Even when a substance is released into the environment, it does not mean that such a substance is transported in a manner that leads to human contact or exposure. Further, according to EPA, these types of data cannot be used to identify exposures and risks for specific individuals, groups or populations.

In addition to misusing the TRI and NPRI data, the authors of the draft report have implied that any exposure to a substance on the TRI or NPRI list is a risk. They have also implied that children, because of their size and rapid development, are always at a heightened risk. These two implications are contrary to the scientific evidence published in peer-reviewed journals. The science of toxicology has a much greater understanding of the effects of chemicals on human health than the draft report describes.

The general discussion regarding children's health raises questions as well. The authors of the draft report have described disease rates and disease causation without following a consistent process for a comprehensive and unbiased review of current statistics and

literature. The alarmist tone regarding rates of childhood diseases is inappropriate in a report funded by government agencies.

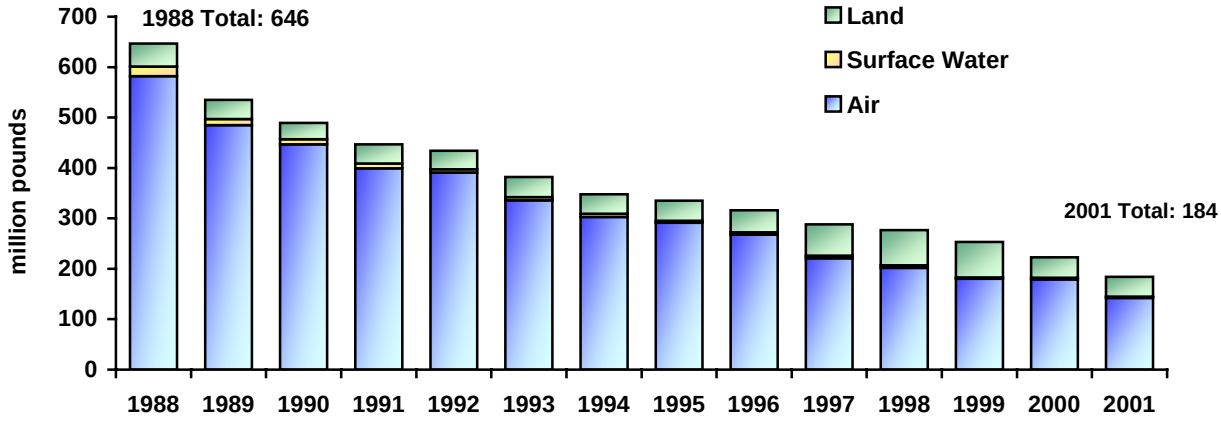
In our comments that follow, we suggest that the NACEC abandon the draft report in its current form. If the NACEC decides to publish the report in final form, however, we suggest additional data that should be included and we have indicated sections in the draft report where errors should be corrected. The scientific review of this report that was announced by the NACEC is one opportunity to fix the errors, eliminate the emotion-laden language, and balance the discussion of children's health protection.

Comments

I. Introduction

As an industry based on science, the business of chemistry appreciates the value of reliable, accurate and relevant information for making decisions. The American Chemistry Council (ACC) has supported the U.S. Toxic Release Inventory (TRI) since its inception. The industry has used the information garnered from that program to develop and measure emission reduction programs at individual facilities, across industrysectors, and overall. Since EPA began tracking TRI releases in 1988, chemical makers have reduced emissions by 71 percent, contributing significantly to today's cleaner environment. More specifically, chemical companies have reduced emissions to air by 75 percent, emissions to land by 14 percent and emissions to water by 82 percent. Chemical production rose 26 percent during the same period.

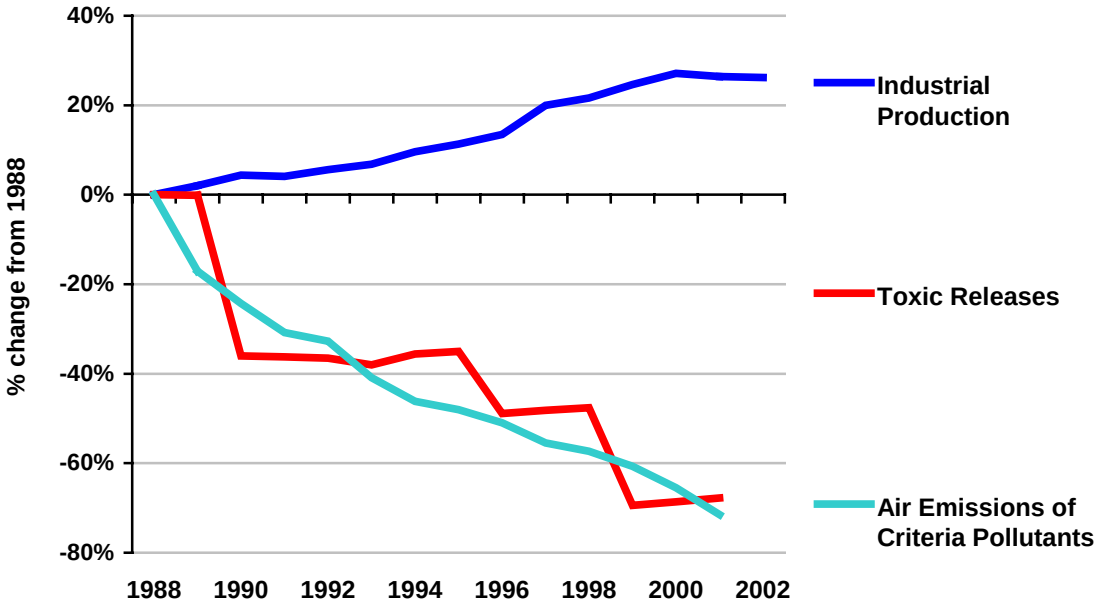
TRI Releases by Media: Business of Chemistry



Source: Environmental Protection Agency

Source: Environmental Protection Agency - 2001 TRI Public Data Releases via TRI Explorer (www.epa.gov/triexplorer), June 2003
**The American Chemistry Council defines Releases to be Total Air+Surface Water+Land. Underground injections are not included in releases to the environment. Includes 1988 core chemicals only (doesn't include delisted chemicals, chemicals added in 1990, 1991, 1994, 1995, and aluminum oxide, ammonia, hydrochloric acid, or sulfuric acid.)*
***Off-site releases include metals and metal compounds transferred off-site for solidification/stabilization and for waste water treatment, including to publicly owned treatment works (POTWs)*

Environmental Progress and Economic Growth in the Business of Chemistry



Sources: Environmental Protection Agency, Federal Reserve Board

We conclude that there are many troubling aspects of the draft report, most notably its strong implication that chemical releases, including US TRI data from specific facilities, are indicative of children's exposures and adverse health effects. In previous comments to the NACEC, ACC, along with the Canadian Chemical Producers Association (CCPA) and the Asociación Nacional de la Industria Química (ANIQ), cautioned against drawing simplistic connections or implying correlations between emissions and children's health threats (Attachment 1). We also recommended that the NACEC submit its plan to create a special report of *Taking Stock* to an expert panel for scientific peer review to assure the information is relevant and consistent with science. We are heartened to see that an expert panel is currently being assembled to review this draft report, and trust that it will be a rigorous, balanced and well-thought out review. Nevertheless, we are disappointed that time and resources have been expended to create this draft report that ignores basic tenets of risk assessment, and relies on emotional statements and implications of disease causation not grounded in a balanced view of peer reviewed science. In addition, we have noted that the draft report makes no mention of the identity of the authors or their affiliations. We believe the NACEC should be more transparent in this respect.

We believe that CEC should withdraw the draft report and re-issue it as a regular *Taking Stock* report focused only on emissions data. If children's health is discussed in a report by the CEC, the discussion should be based in science, free of emotion, include relevant up-to-date references and a balanced discussion of the current literature.

Our comments below serve as a critique of the draft report and offer additional references and information to allow the authors and expert reviewers to better judge whether the information and analysis used in the report is scientifically valid.

II. Comments on Chapter 1: Demographics and Children's Health

The draft report correctly identifies the "environment" as a confluence of factors. When assessing the effect of environment on health, one must include all environmental factors (other than genetics): physical, chemical, biological and social. The report discusses this broad definition of environment, but details only the influence of chemicals on children's health. This provides the reader with an incomplete picture of child health overall.

In addition, while the data in Table 1 (Annual Mortality Rates for Specific Causes of Death for Children in North America) reports important information regarding the health of children, it does not give an accurate picture overall regarding the health and well-being of children. We comment below on the overall health status of children only in the U.S.

America's Children: Key National Indicators of Well-Being, 2003[1] notes that the health and well-being of children in the U.S. is improving overall with 83% of children under the age of 18 in very good or excellent health. In this annual report, the U.S. Federal Interagency Forum on Child and Family Statistics reports on nine contextual measures describing the changing population, family, and environmental context in which children are living, and 25 indicators that depict the well-being of children in the areas of economic security, health, behavior and social environment, and education. The most recent edition of the report contains U.S. federal government data describing the declining infant mortality rate, declining adolescent mortality rate, declining birth rate for adolescents, the declining rate of cigarette usage among teenagers. The report also notes an increase in low-birthweight babies and an increase in childhood obesity.

Any report on children's health should strive to provide a better overall picture of child health in North America by providing some summary trend data regarding all aspects of the child's environment.

III. Comments on Chapter 2: Toxic Chemicals and Children's Health in North America

A. Chemicals and Chemical Regulation in the U.S.

The brief summary of the U.S. regulatory system (draft report, p. 12) incorrectly implies: 1) that the vast majority of chemicals in commerce are essentially unregulated; and 2) that children face significant exposures to these chemicals. Although over 89,000

[1] Federal Interagency Forum on Child and Family Statistics (FIRCFS). 2003. America's Children: Key Indicators of Well-Being. Washington, DC: National Center for Health Statistics.

chemicals are currently on the U.S. Toxic Substances Control Act (TSCA) inventory, the vast majority of those chemicals are not in commerce. The Inventory is updated every four years; the last update (2002) indicated that there are approximately 9,000 chemicals on the U.S. market in amounts greater than 10 tons per year. Of that figure, High Production Volume (HPV) chemicals (production or import greater than 1 million pounds per year) – some 3,000 chemicals -- represent more than 90% of the chemicals in commerce by volume.

The draft report discussion of the HPV assessment programs (draft report, p. 14) relies largely on information from 1998. The effect is to suggest inappropriately that little information continues to be known about HPV chemicals, and further incorrectly implies that industry has done little to address the situation. The "sidebar" on page 13 of the draft report provides some minimal detail on the HPV programs, but that information may be of little consequence given the description on page 14. Since 1998, industry volunteers in the US HPV Chemical Challenge Program and the International Council of Chemical Associations (ICCA) HPV Initiative have been working to complete their commitments in full and on time. Together, the programs will result in sets of "hazard effects" data being publicly available on 2,150 chemicals by 2005 – information produced on a far larger group of chemicals, in far less time, than ever before.

ACC strongly recommends that the report language referencing the HPV programs be revised to focus not on the situation as of 1998, but on the situation as of 2004.

B. Types of Chemicals

The draft report makes misleading, over simplified statements in implying that chemicals can be grouped as either "toxic" or "non-toxic" or "harmful" or "not-harmful." First and foremost, all substances can induce some form of toxicity at high enough dose levels. The premise -- that chemicals can be presumed to be either "toxic" or "non-toxic," "harmful" or "non-harmful," irrespective of the degree of exposure - - is fundamentally flawed. The scientific facts are exactly the opposite – the potential for developing toxicity cannot be evaluated without comparing the predicted or measured exposure to a health reference dose.

Science has established that for any given chemical, natural or synthetic, the substance can have no effect, adverse health effects or, at times, with certain substances, beneficial effects, dependant upon the dose. Many substances can produce toxic or harmful effects at high doses and not produce any effects at lower doses.

Furthermore, it is a widely held tenet in the science of toxicology that there are thresholds of exposures — threshold doses below which no effects are evident or likely. The draft report fails to acknowledge that for any and all chemical substances, low exposure levels, those below a reference dose or level of concern, are expected to <u>not pose</u> any significant degree of health risk whatsoever to any individual, including sensitive subpopulations.

The draft report fails to communicate that pesticides and other chemicals are indeed tested and evaluated by manufacturers and are subject to review by regulatory authorities. It is incorrect to presume or imply that pesticides pose a widespread hazard or risk to human health. When these substances are used in accordance with scientifically derived label instructions prescribed by regulation, they have been determined by the regulatory agencies in North America to be safe to humans and to wildlife. In particular, the U.S. EPA's pesticide evaluation and registration program is "designed to ensure that these products [pesticides] can be used with a reasonable certainty that they will pose no harm to infants, children, and adults."[2]

The authors imply that children are at increased risk from over exposures and poisoning from pesticide exposures. In fact, the available data do not support such a contention. In California, an agricultural producing region and the most populous State in the U.S. (> 34 million inhabitants), only 99 cases of reported childhood (ages 0-9 years) illnesses were associated with pesticides in 2002[3]. This out of a population of > 5 million children ages 0-9 residing in California (based on 2000 US census data[4]). Clearly, the risk is very small. Further, of these 99 cases in children ages 0 to 9, greater than 1/3 were associated with improper handling and/or storage of antimicrobials such as bleach, quaternary ammonia, pine oil, etc.

C. Comments on Pathways of Chemicals

The language used in the draft report implies that children are always "more vulnerable" than adults to potential exposures of chemical agents (draft report at p. 16). This section appears to mix the concept of vulnerability (risk) and exposure. Risk (and vulnerability) is a function not only of exposure, but also inherent hazard of the substance and susceptibility of the individual.

D. Comments on "Vulnerability"

Any categorical assertion that children are consistently more susceptible to any or all environmental agents simply because they are smaller or are undergoing periods of rapid development cannot be substantiated. The facts are that children may be more or less sensitive, dependent upon the class of compounds and/or endpoint under consideration. Scientific reviews have shown that evaluation of the sensitivity of a child requires a case-by-case, substance-specific approach. This case-by-case evaluation would consider the relevance and quality of all available data – particularly any data relating to differences (or otherwise) in responses between adults and young in both animal and human studies. The primacy of human data over animal studies would be consistent with established practice.

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[2] http://www.epa.gov/pesticides/food/risks.htm

[3] California Department of Pesticide Regulation Summary of Results from the California Pesticide Illness Surveillance Program, 2002. http://www.cdpr.ca.gov/docs/whs/2002pisp.htm

[4] http://factfinder.census.gov/servlet/QTTable?_bm=y&-geo_id=04000US06&-qr_name=DEC_2000_SF1_U_DP1&-ds_name=DEC_2000_SF1_U

> "The newborn's metabolic capacity rapidly matures and, by about 6 months of age, children are usually not more sensitive to chemical toxicity than adults. By then, most metabolic systems are reasonably mature, becoming almost completely capable by one year of age. Children over 6 months of age can be more sensitive to chemical toxicity than adults but they usually are not. In many cases children are less sensitive than adults."[5]

The claim in the draft report that children are more susceptible is linked in the report to the assertion that the vulnerability of children has not been considered to date in toxicological assessments and children are therefore not protected. These assertions are incorrect. Laboratory animal models are considered to be reliable and predictive of reproductive and developmental effects in humans, and these animal models have been used extensively -- for more than 3 decades -- in industry, government and research institutions. The predictive nature of such models has proved invaluable, and they are relied on as important elements of safety evaluation programs for pharmaceuticals, medical devices, biologics, food additives, pesticides and industrial chemicals. The use of such models requires both species extrapolation (from the test species to humans) and dose extrapolation (from the high doses used in the lab study to low, environmentally relevant human exposure levels). Further, risk-based decision making, founded on laboratory animal tests, is an effective process for prevention of exposures at levels posing a potential developmental or reproductive concern.

It has been clearly established that susceptibility depends on the substance and the exposure situation for any subpopulation, and that children may be more or less susceptible than other age groups and subpopulations to certain environmental agents[6]. There is a long-standing consensus to support this:

> "*An analysis based on available data…….suggests that susceptibility clearly depends on the substance and the exposure situation.*"[7]

> "*Differences in toxicity between young and mature animals may be in either direction but are generally modest. The younger animal may be more sensitive or may be less sensitive than the older animal to comparable levels of exposure to toxic agents.*"[8]

Windows of vulnerability during fetal development and sexual maturation *are not* a new concept, as these have been incorporated into research, testing and safety assessments for more than 35 years.[9] It has been said that any agent administered at an appropriate dose

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[5] Schuplein, R. *et al*, *Regulatory Toxicology and Pharmacology*, 2002, 35:429-447.

[6] Dourson M, Charnley G, Scheuplein R (2002) Differential sensitivity of children and adults to chemical toxicity. *Regulatory Toxicology and Pharmacology* 35, 448-467.

[7] Guzelian, PS and Henry CJ (eds). *Similarities and Differences Between Children and Adults: Implications for Risk Assessment.* (Washington, DC: International Life Sciences Institute, 1992).

[8] National Research Council. *Pesticides in the Diets of Infants and Children* (Washington, DC: National Academy of Sciences, 1993).

[9] Wilson, J.G., *Teratology Principles and Techniques*, (Chicago, IL: Univ. of Chicago Press, 1965).

and at an appropriate time of development can cause some type of disturbance in development.[10] Thus, the magnitude, frequency, duration and timing of exposure play a critical role in studies of the potential for agents to adversely affect development and reproduction.

E. Exposure

The draft report also implies that children are at risk of experiencing harm from normal uses of products in and around the home. This statement is not supported by the available evidence. It is important to include in such statements the concept that excessive exposure – exposure to levels which exceed the safety benchmarks – are of concern, rather than implying that any exposure is automatically harmful. Manufacturers go to great lengths to determine that there are no significant health threats from the normal and intended use and foreseeable misuse of products. Extensive government regulations with which manufacturers must comply address safety in and around the home. These can include product testing and evaluation, product labeling, and use of product safety devices (child-proof closures) to prevent inadvertent misuse and inadvertent overexposures.[11]

Although it is true that awareness is increasing regarding issues related to *in utero* exposures (draft report at p. 16), it is incorrect to assert that *any* chemical exposure, irrespective of the substance or magnitude, can have significant, long-term and lasting effects. Such a gross generalization is not supported by the science. Scientific studies for over the last 50 years have clearly shown that the developing organism can be more, less or equally sensitive as compared to the adult.

F. Threshold Dose

A fundamental principle of toxicology is that of the threshold dose, which holds that for all agents, there are doses below which no harm is expected. EPA's *Guidelines for Developmental Toxicity Risk Assessment* clearly state that the concept of a threshold applies to both developmental and reproductive toxicity.

> *"In general, a threshold is assumed for the dose-response curve for agents that produce developmental toxicity. This is based on the known capacity of the developing organism to compensate for or to repair a certain amount of damage at the cellular, tissue, or organ level. In addition, because of the multipotency of*

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[10] Manson and Kang 1989. Test Methods for Assessing Female Reproductive and Developmental Toxicity in Principles and Methods of Toxicology (2nd Edition) (New York: Raven Press Ltd., 1989).

[11] Products made using chemicals are subject to a myriad of U.S. laws including, Federal Food, Drug and Cosmetic Act, Federal Hazardous Substances Act, Federal Insecticide, Fungicide and Rodenticide Act, the Consumer Product Safety Act, etc.

cells at certain stages of development, multiple insults at the molecular or cellular level may be required to produce an effect on the whole organism."[12]

In a quantitative dose-response analysis, mode of action, pharmacokinetic, and pharmacodynamic information should be used to predict the shape of the dose-response curve when sufficient information of that nature is available. When that information is insufficient, it has generally been assumed that there is a nonlinear dose-response for reproductive toxicity. This is based on known homeostatic, compensatory, or adaptive mechanisms that must be overcome before a toxic endpoint is manifested and on the rationale that cells and organs of the reproductive system and the developing organism are known to have some capacity for repair of damage. … The shift to the term nonlinear does not change the RfD/RfC methodology for reproductive system health effects, including the use of uncertainty factors.[13]

In addition, some regulatory agencies have made policy decisions to approach the risk estimation for carcinogens by assuming a linear dose response. Such policy decisions are derived from the policy choice of an agency not to underestimate potential risk and, as such, these defaults necessarily overestimate risks. The resulting evaluations do not represent the actual likelihood of developing cancer, but instead are a "worst-case" or an "upper end" estimate of risk, and are not a true estimate of an individual's or population's risk. Such agencies have proposed and used linear extrapolation of risk in the low dose region as a "reasonable upper-bound on risk", and in doing so have acknowledged that using such an approach was designed specifically so as not to underestimate potential risk, and have included statements such as the "true risk may even be zero." [14,15]

However, more recently, agencies such as US EPA have proposed guidance that recognizes non-linear or threshold approaches are indeed scientifically supported for assessing human health risks from exposures to certain substances which produce cancer in laboratory animal studies.[16]

The draft report (p. 17) fails to clearly communicate these important principles, and incorrectly and unscientifically implies that any degree of exposure poses some magnitude of potential risk.

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[12] Guidelines for Developmental Toxicity Risk Assessment. U.S. Environmental Protection Agency. Risk Assessment Forum, Washington, DC, EPA/600/FR-91/001, 1991http://www.epa.gov/ncea/raf/pdfs/devtox.pdf

[13] U.S. Environmental Protection Agency, *Guidelines for Reproductive Toxicity Risk Assessment*. (Washington, DC: EPA Risk Assessment Forum, 1996) EPA Doc. 630/R-96/009. http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=2838

[14] http://potency.berkeley.edu/text/Gaylor1998.pdf

[15] http://www.epa.gov/ttn/atw/toxsource/carcinogens.html

[16] EPA/630/P-03/001ANCEA-F-0644AFebruary 2003Draft Final www.epa.gov/ncea/raf/cancer2003.htm

G. Breastfeeding

The authors claim that increased concentrations of contaminants in breast milk can increase the risk of infant infections (draft report at p. 16). There is a considerable body of evidence to show that it is far from established scientific evidence to conclude that infections among nursing infants have increased. In fact, the American Academy of Pediatrics Policy Statement on Breastfeeding[17] states that breastfeeding decreases the incidence/severity of infections:

> *Research in the United States, Canada, Europe, and other developed countries, among predominantly middle-class populations, provides strong evidence that human milk feeding decreases the incidence and/or severity of diarrhea, lower respiratory infection, otitis media, bacteremia, bacterial meningitis, botulism, urinary tract infection, and necrotizing enterocolitis. There are a number of studies that show a possible protective effect of human milk feeding against sudden infant death syndrome, insulin-dependent diabetes mellitus, Crohn's disease, ulcerative colitis, lymphoma, allergic diseases, and other chronic digestive diseases. Breastfeeding has also been related to possible enhancement of cognitive development.*

H. Cancer and Cancer Rates

The discussion of cancer and cancer rates in the draft report is too brief and selective to be complete. Its presentation in the context of the TRI and NPRI discussion implies, despite text to the contrary, that chemicals are the major cause of cancer in children in North America. We disagree with the author's presentation of the information and believe it is misleading.

For example:

The U.S.National Cancer Institute (NCI) states "childhood leukemias appeared to increase in incidence in the early 1980s, with rates in the preceding years at fewer than 4 cases per 100,000. Rates in the succeeding years have shown no consistent upward or downward trend and have ranged from 3.8 to 4.8 cases per 100,000." [18]

The NCI also states "For childhood brain tumors, the overall incidence rose from 1975 through 1998 (from 2.3 to 3.0 per 100,000), with the greatest increase occurring from l983 through l986. An article in the September 2, 1998, issue of the *Journal of the National Cancer Institute* suggests that the rise in incidence from 1983 through 1986 may not have represented a true increase in the number of cases, but may have reflected new forms of imaging equipment (magnetic resonance imaging or MRI) that enabled visualization of brain tumors that could not be easily visualized with older equipment. Other important developments during the 1983–86 period included the changing classification of brain tumors that resulted in tumors previously designated as 'benign'

[17] http://aappolicy.aappublications.org/cgi/content/full/pediatrics%3b100/6/1035
[18] http://cis.nci.nih.gov/fact/6_40.htm

being reclassified as 'malignant,' and improvements in neurosurgical techniques for biopsying brain tumors." [19]

By implication, the draft report seeks to associate chemicals reported in TRI and NPRI databases with the induction of cancer in children. This is highly misleading and not consistent with recognized authoritative sources. NCI states[20] that the causes of childhood cancers are largely unknown, and cites the following key findings from recent studies of causes of cancer in children:

- High levels of ionizing radiation from accidents or from radiotherapy have been linked with increased risk of some childhood cancers;
- Children treated with chemotherapy and radiation therapy for certain forms of childhood and adolescent cancers, such as Hodgkin's disease, brain tumors, sarcomas, and others, may develop a second primary malignancy;
- Low levels of radiation exposure from radon were not significantly associated with childhood leukemias;
- Ultrasound use during pregnancy has not been linked with childhood cancer in numerous large studies;
- Residential magnetic field exposure from power lines was not significantly associated with childhood leukemias;
- Certain types of chemotherapy drugs, including drugs that are alkylating agents or topoisomerase II inhibitors (e.g., epipodophyllotoxins), may cause increased risk of leukemia;
- Pesticides have been suspected to be involved in the development of certain forms of childhood cancer based on interview data. However, interview results have been somewhat inconsistent, and have not yet been validated by physical evidence of pesticides in the child's body or environment;
- No consistent findings have been observed linking specific occupational exposures of parents to the development of childhood cancers;
- Several studies have found no link between maternal cigarette smoking before pregnancy and childhood cancers, but increased risks were related to the father's prenatal smoking habits in studies in the United Kingdom and China;

[19] ibid
[20] http://cis.nci.nih.gov/fact/6_40.htm

- Little evidence has been found to link specific viruses or other infectious agents to the development of most types of childhood cancers, though investigators worldwide are exploring the role of exposure of very young children to some common infectious agents that may protect children from, or put them at risk for, developing certain leukemias;

- Recent research has shown that children with AIDS, like AIDS-stricken adults, have an increased risk of developing certain cancers, predominantly non-Hodgkin's lymphoma and Kaposi's sarcoma. These children also have an additional risk of developing leiomyosarcoma (a type of muscle cancer);

- Specific genetic syndromes, such as the Li-Fraumeni syndrome, neurofibromatosis, and several others, have been linked to an increased risk of specific childhood cancers.

It is noteworthy that, contrary to the implication of the draft report, the summary of scientific evidence cited by NCI does not implicate <u>any</u> of the substances addressed in this draft report as suspected of being a causative agent of cancer in children.

I. Learning Disabilities

This section similarly reflects the bias of the authors. The discussion implies that exposures to chemicals are "causing" or contributing to learning disabilities. Such implication is contrary to current scientific consensus, which holds that the causes and prevalence of learning disabilities (LD) are not known with any degree of certainty, due to the varied operational definitions, the diverse constellations of symptoms, and limited relevant research. Further, the authors fail to communicate that it is difficult to determine the prevalence rate for LD and whether or not the rates are increasing over time because of the variations in the definition of LD and the ever-evolving approaches to diagnosis and estimation of prevalence.

There is a wide range of prevalence rates for LD reported in the scientific and medical literature – rates ranging from 1% to 30% of the general population -- which is the result of variations in the definition of LD and the source of case ascertainment. The estimates for children are similarly variable. The National Institutes of Health (NIH) estimated for 1993 nearly four million school-age children in the US with LD, while the CDC estimated 1.4 million in 1991-1992. The reason the CDC reports a much lower number of children affected by LD may be due to a more restrictive definition. Finally, based on special education services of students 6 to 21 years of age, the U.S. Department of Education reported that nearly 4% of students in 1999 were learning disabled.

The authors of the draft report fail to communicate the known risk factors for cognitive deficits and other neurological problems in children: prenatal exposures to legal and illegal drugs, tobacco and alcohol, untreated Rh incompatibility during pregnancy, premature or prolonged labor, lack of oxygen during labor, and low birth weight.

Neurodevelopmental effects such as delayed speech, cognitive and attention deficit disorders, hyperactivity, and lowered IQ have been associated with poverty, social disadvantage, child abuse and neglect, malnutrition, and parental disinterest. Because many of these outcomes are included among LD functional deficits, they are considered by some to be possible risk factors or contributory factors for LD itself. However, epidemiological studies that include specific cases of LD children are rare.

The varied definitions of LD and the diverse constellation of symptoms it encompasses make understanding the causes of the disease very difficult. Where it was once thought that LD was caused by a single neurological problem, it is now recognized that it involves difficulty in bringing together information from various brain regions. Damage to the brain resulting in learning impairment may occur at any time in a person's life; however, it is much more likely to occur at certain crucial points during prenatal development or before the child is three years old, when the brain is still rapidly developing. The reverse is also true in that the developing brain has much greater plasticity, so the damage may be more likely to be reversible.

There are a number of chemical compounds (lead, mercury, ethanol, cocaine) that are known or suspected developmental neurotoxicants in humans under conditions of sustained overexposure, some of which cause cognitive deficits. The effects of relatively high exposure levels of lead and mercury on learning impairment are well established. While some have postulated that other chemicals, such as pesticides, PCBs, solvents and hormonally active agents may cause neurodevelopmental effects in children, these hypotheses are far from being proven. Several epidemiological studies of neurodevelopmental effects in children have focused on PCB exposure and have reported evidence of a relationship to child development or learning. Reviewers have noted, however, that these studies have numerous methodological problems, particularly, limitations in estimating PCB exposure and sample selection, thereby reducing confidence in the results. Scientists have also noted that because learning and development are influenced by many factors, it is not possible to conclude with any degree of certainty that exposure to PCBs is one of those factors.[21]

J. Autism

The discussion of autism is so brief that it is particularly misleading. Although the draft report notes on p. 19 that there is uncertainty about the rates of autism, the context implies that autism is on the rise. The authors neglect to inform the reader that CDC has reported that there is great uncertainty about the true incidence and prevalence of autism in the U.S., both overall and in different geographic regions and different subpopulations.[22] For example, a 1998 CDC and ATSDR investigation to determine the prevalence of these disorders in Brick Township, New Jersey, due to a perceived increased incidence, reported that the prevalence rates of autism and autism spectrum

[21] Schantz, S.L. 1996. Developmental neurotoxicity of PCBs in humans: What do we know and where do we go from here? *Neurotoxicology and Teratology*, 18(3):217-227.
[22] Centers for Disease Control (CDC). 2001. Autism among Children. NCEH Pub No. 01-084. Website: http://www.cdc.gov/ncbddd/dd/ddautism.htm.

disorder (ASD) were 4 per 1,000 and 6.7 per 1,000 children, respectively. Similar rates were found in several non-US based studies. The CDC/ATSDR report concluded that in order to properly interpret these results, improved prevalence data would be needed from large, diverse populations.[23]

The prevalence of autism and the question of whether it is actually increasing over time have generated considerable debate in the scientific and public health communities. The authors of the draft report fail to objectively communicate this. Some studies have attempted to determine whether the reported increase is real or an artifact of misdiagnosis or other factors.

In a particularly egregious statement, the authors claim that the autism rate in California increased 2.5 fold from 1987 to 1994 (draft report at p. 19). The claim that the rate of autism is increasing is based in part on a report to the California legislature.[24] The authors fail to disclose that the California report addresses only utilization of services by a State agency, not actual rates of the illness. There are many factors that could account for changes in utilization of services, including changes in diagnostic criteria, issues pertaining to insurance and access to medical care as well as a greater acceptance by parents of a diagnosis of autism in lieu of a diagnosis of mental retardation.

The peer-reviewed study of Croen *et al*.[25] reported a dramatic decrease in the prevalence in mental retardation in the California database during the same time period as a reported increase in autism (9.3/10,000 decrease in mental retardation vs. 9.1/10,000 increase in autism). This finding suggested that what has been observed in the California report could be the result of diagnostic substitution – where cases that in the past would have been diagnosed as mentally retarded have more recently been diagnosed as autistic.

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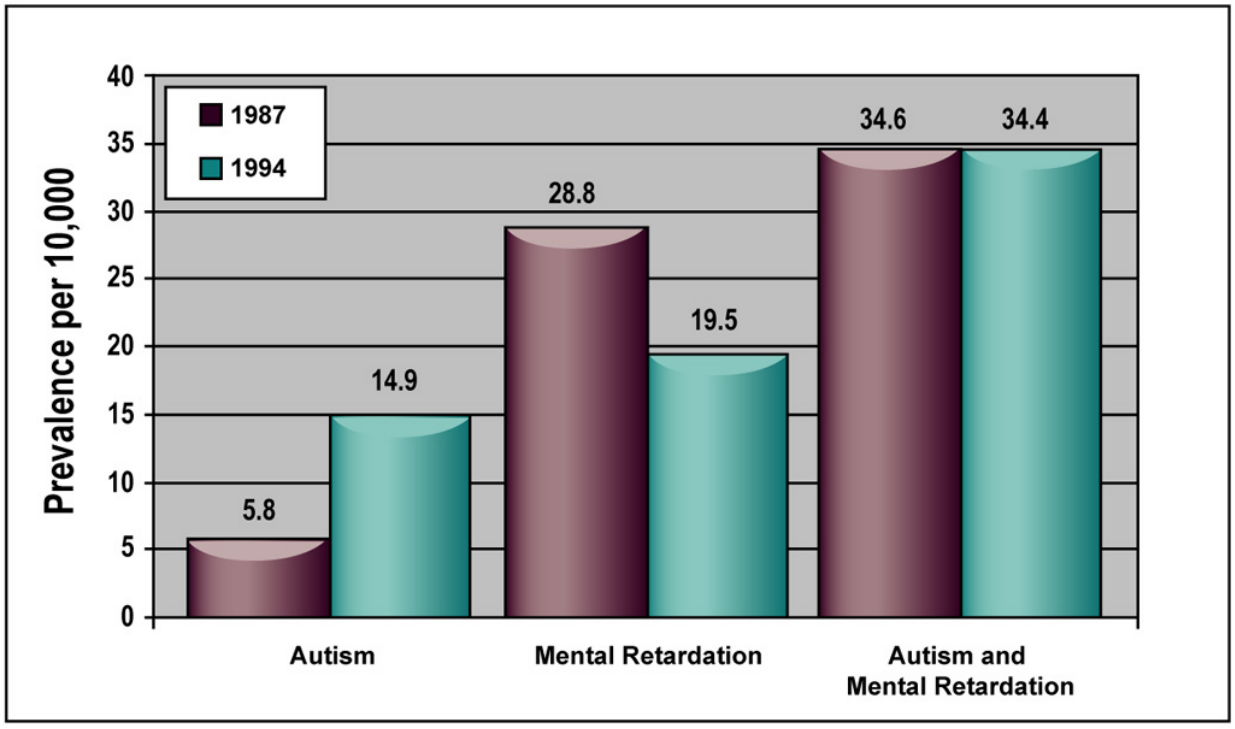
[23] Agency for Toxic Substances and Disease Registry (ATSDR). 2000a. Brick Township Investigation. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
Agency for Toxic Substances and Disease Registry (ATSDR). 2000b. Results of Brick Township Investigation of Environmental Pathways. Fact Sheet #4. Atlanta, GA: Agency for Toxic Substances and Disease Registry.

[24] California Health and Human Services, Department of Development Services, Changes in the Population of Persons with Autism and Pervasive Developmental Disorders: 1987 through 1998, A Report to the Legislature, March 1999.) http://www.autism.com/ari/dds/dds/html.

[25] Croen, L.A., J.K. Grether, J. Hoogstrate, S. Selvin. 2002. The changing prevalence of autism in California. J Autism Dev Disord, 32(3):207-215.

Figure 1. Changing Prevalence in Autism and Mental Retardation in California from 1987-1994



Source: Croen *et al*., 2002

Taylor and colleagues have reported[26] that a purported rise in prevalence of autism may not be real at all, but was likely due to factors such as increased recognition, a greater willingness on the part of educators and families to accept the diagnostic label, and better recording systems.

With respect to risk factors and the development of autism, it is generally agreed among medical experts that there is an unknown genetic component to ASD. Hypothesized contributory nongenetic risk factors, such as vaccines, diet, drugs, infections, and chemicals in the environment remain unproven. Investigations to date have not established a cause and effect relationship between exposure to substances in the environment and autism; the CDC has concluded that the current scientific evidence does not support the hypothesis that vaccines cause autism. [27]

Regardless of the lack of understanding about the causes and even the rates of autism and ASD, the authors of the draft report nevertheless chose to include it, and thereby giving

[26] *Archives of Disease in Childhood* (2003);88:666-670.
[27] Centers for Disease Control (CDC). 2001. Autism among Children. NCEH Pub No. 01-084. Website: http://www.cdc.gov/ncbddd/dd/ddautism.htm

the reader the false impression that the chemical emissions described in the draft report are somehow related to autism.

K. Birth Defects

Again, the authors of the draft report selectively cite one or a few publications in the literature to imply association between chemicals and a health problem. For example, the authors cite a single study of hypospadia rates (Paulozzi *et al* 1997) to support their contention that some birth defects are becoming more common.

In fact, hypospadia rates are not to be rising in all regions of the U.S. and a global trend has clearly not been demonstrated. Hypospadia rates have been reported to have risen in Atlanta and Italy, declined in Spain and Canada, and remained the same in New York and in California. In fact, researchers have suggested that reports of rising incidence rates may be due to changes over time of diagnostic criteria or even the age of parents at conception. Fisch, *et al*, authors of an epidemiological investigation of hypospadia rates, has stated, "Maternal age may be the single most important factor besides the genetic profile of the father and mother."[28]

As an illustration of the bias of the draft report authors, it is noteworthy that they seem to have purposefully cited one study of Paulozzi, but selectively ignored a more recent publication of Paulozzi,[29]in which the investigator's conclusions contradict the assertion of the CEC authors. Paulozzi, 1999 concluded (emphasis added):

- Nationwide data from the Birth Defects Monitoring Program of the Centers for Disease Control and Prevention (CDC) showed an upward trend in hypospadias beginning in 1970. A more discontinuous upward trend began in 1968 in the CDC's Atlanta, Georgia, surveillance system. Severe hypospadias in the Atlanta system increased from 1982 to 1985 and then leveled off. Rates from the California Birth Defects Monitoring Program for severe hypospadias showed no upward trend.

- Review of data from 29 registries that monitor a total of 4 million births per year around the world reveals wide intercountry variation in rates of hypospadias and cryptorchidism.

- The absence of an increase is perhaps most notable in Canada, whose society is similar to that of the United States.

- Among all systems showing an increase, rates tended to level off after 1985.

- A number of factors may account for reported changes in these rates. Chief among them are artifacts. In other words, the main reason for reports of increase

[28] *Journal of Urology* (2001). Volume165: pages 934-936.
[29] *Environ Health Perspecives.* (1999) Vol. 4:297-302.

in hypospadia incidences could be due to inaccurate observations or results, resulting from the methods used in the scientific investigations.

- There is conflicting evidence on whether the case definition of hypospadias has indeed loosened to include more of the milder, first-degree cases. Previously published data from the Atlanta registry indicated that the percent of first-degree cases did not increase over time In contrast, the Finnish registry communicated that the percent of more serious degrees of hypospadias declined as overall rates increased. Moreover, the California and the northeast Italy programs have shown no increase in rates of severe hypospadias. Severe hypospadias is much less likely to be affected by changes in definition because it has clearer anatomical boundaries.

The authors of the draft report imply that there is a scientific consensus that certain birth defects have been associated with exposure to endocrine active persistent organic pollutants. However, the authors do not give sufficient weight to the authoritative conclusions of the U.S. National Academy of Sciences (NAS) and the International Programme on Chemical Safety (IPCS). These independent expert panels concluded that the currently available human data are inadequate to support a conclusion that human reproductive health has been adversely affected by exposure to endocrine active substances:

- Given the evidence to date, increases in the incidence of male reproductive disorders in humans, such as testicles that have not descended to the scrotum, cannot be linked to exposure to hormonally active agents found in the environment."[30]

- "Known risk factors associated with failure of the testis to descend into the scrotal sac (cryptorchidism) include ethnicity, a family history of cryptorchidism, use of analgesics during pregnancy, birth order, and maternal obesity. Several of these are also risk factors for hypospadias, a developmental abnormality in which the urethra opens on the underside of the penis or the perineum. Evidence of seasonal effects with peaks for cryptorchidism occurring at different times of the year in various studies has been reported, although the significance of this finding has yet to be determined."[31]

- "The major limiting factor in drawing any conclusions about human reproductive health effects and putative links to EDCs is the absence of exposure data. Exposure data are very limited, if available at all, and in many studies exposure has only been inferred and not actually measured. Another major problem common to many of the human studies is that sample sizes are often too small to permit detection of an effect, even if one was present. Thus, the currently

[30] *Hormonally Active Agents in the Environment* (Washington, DC: National Research Council, 1999).
[31] *Global Assessment of Endocrine Disrupting Chemicals*, International Programme for Chemical Safety (2002) available at: http://ehp.niehs.nih.gov/who/

available human data are inadequate to support a conclusion that human reproductive health has been adversely affected by exposure to EDCs."[32]

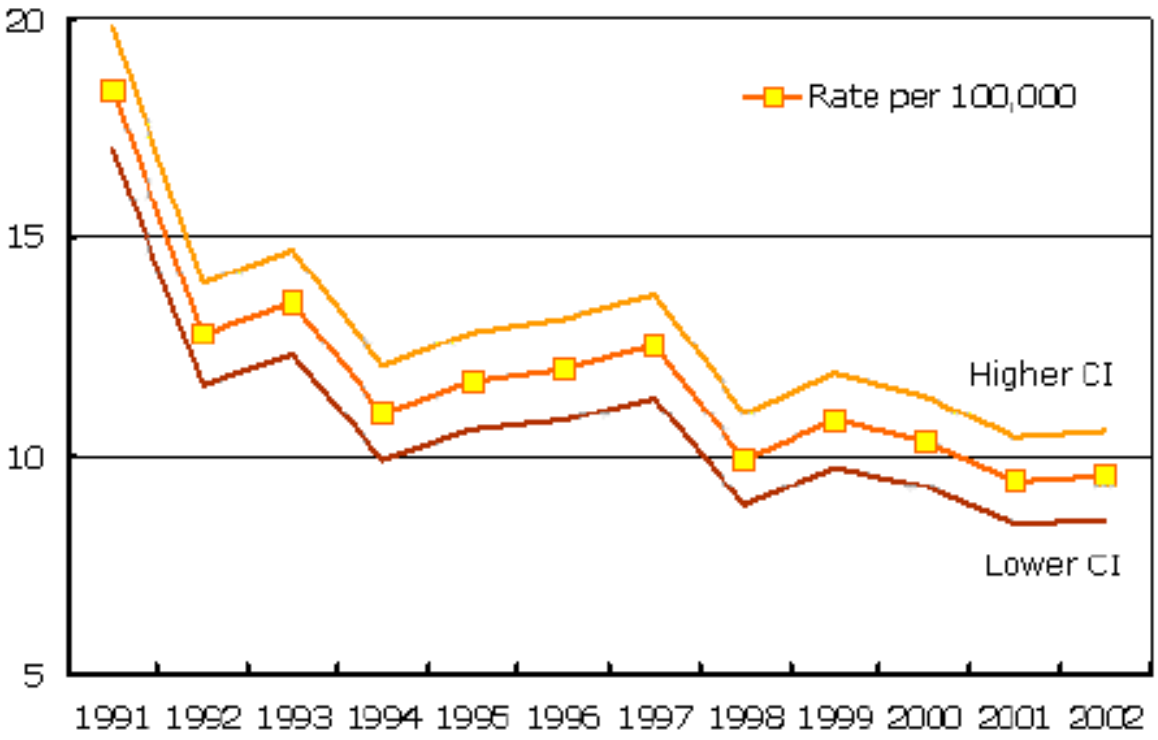
The discussion by the authors of draft report of anencephaly (draft report at p. 19) is also incomplete and without context. The authors fail to note several important facts that are apparent from an objective evaluation of the data:

- The authors neglect to inform readers that both clinical trials and observational studies have shown that 50% or more of neural tube defects can be prevented if women consume a folic acid supplement before and during the early weeks of pregnancy.[3334] This suggests that a vitamin insufficiency, not purported exposure to ambient levels of chemical substances, is a major cause of this important type of birth defect in humans. By failing to note this fact, the authors again reveal their lack of objectivity and their inherent predilection to try to link chemical exposures to health outcomes irrespective of the actual data.

- In the United States, health statistics show that the rate of anencephalus has declined over time, commensurate with actions by the US health agencies (U.S. Public Health Service, FDA and others) to recommend women of childbearing age increase consumption of the folic acid and to ensure that all enriched cereal grain products be fortified with folic acid. The rate of anencephalus in 2002 was 9.55 per 100,000 live births, significantly lower than in 1997. [35]

[32] ibid.
[33] Surveillance for anencephaly and spina bifida and the impact of prenatal diagnosis. United States, 1985-1994. Centers for Disease Control and Prevention. MMWR 1996; 44(SS-4):1-13.
[34] MRC Vitamin Study Research Group. Prevention of neural tube defects. *Lancet*. 1991 (338:131-137).
[35] http://www.cdc.gov/nchs/products/pubs/pubd/hestats/spine_anen.htm

Figure 2. Anencephalus rates in the US, 1991-2002



NOTES: Excludes data for Maryland, New Mexico, and New York, which did not require reporting for anencephalus for some years. CI is 95% confidence interval.

SOURCE: National Vital Statistics System, NCHS, CDC.

L. Endocrine Disruption

The authors assert that endocrine disruption is a new form of toxicity. In fact, endocrine disruption is not new in toxicology. Endocrine disruption describes a mechanism of action by which exposure to a substance induces an adverse effect, such as birth or developmental defects, adverse neurological effects, cancer, or reproductive dysfunctions. Chemicals have long been assessed for these adverse effects through traditional (or readily modified) testing methods, and, where they have shown to cause such adverse effects, those chemicals have been classified and managed accordingly pursuant to existing hazard classification standards on the basis of normal endpoints of concern and pursuant to available chemical risk management programs.

The assertion that endocrine disruptors have been associated with a myriad of human health effects is misleading and not a full picture of the weight of the scientific evidence (draft report, p. 20). Both the Global Assessment of the State-of-the-Science of Endocrine Disruptors[36] prepared by the IPCS the World Health Organization, and

[36] http://ehp.niehs.nih.gov/who/

Implications of Endocrine Active Substances for Human Health and Wildlife:[37] Executive Summary, prepared by the Scientific Committee on Problems in the Environment (SCOPE) and the International Union of Pure and Applied Chemistry (IUPAC) conclude that to date there is no firm evidence that exposures to endocrine active substances at levels measured in the general population are affecting human health. At the same time, they acknowledge the potential that such effects could be occurring and that further investigation is warranted. Those reports also find some clear instances of adverse effects occurring in wildlife, but, for the most part, these appear to be only at elevated exposures.

The assertion that endocrine disruptors are active at low doses seems to imply that there is widespread scientific consensus on this point. However, the authors choose to ignore a wide and growing body of evidence that shows that the low-dose hypothesis is not supported because:

- The low-dose findings have not been demonstrated consistently across different studies of the same substance in independent laboratories;

- The findings are not consistent for all substances with similar mechanisms of action; and,

- The biological significance of the reported low-dose effects is scientifically uncertain, in particular with respect to relevance of such effects, if any, to adverse effects upon health of the organism.

Further, the authors fail to report that the U.S. EPA has determined, after reviewing the hypothesis, that there is no need for the Agency to initiate across-the-board modifications to testing or evaluation approaches at the present time because the evidence for such purported low dose effects is insufficient.[38]

M. Asthma

While it is true that the incidence of childhood asthma has increased in the U.S. and other areas of the industrialized world over the last two decades, evidence suggests that the rates have now leveled off in the U.S. Some portion of the increase may be due to improved recognition and diagnosis, but not all. This rise in asthma has been particularly prominent in children and even more so in those living in urban environments, and has occurred in all developed countries around the world.

The reasons for this dramatic increase in asthma are not known and there are likely to be multiple contributing factors (draft report, p.20). There is clear evidence that both

[37] Special Topic Issue on the Implications of Endocrine Active Substances for Humans and Wildlife, Scientific Committee on Problems of the Environment and International Union of Pure and Applied Chemistry, *Pure and Applied Chemistry*, Vol. 75, Issues 11-12.

[38] EPA Statement Regarding Endocrine Disruptor Low-Dose Hypothesis (March 26, 2002) available at: http://www.epa.gov/scipoly/oscpendo/docs/edmvs/lowdosepolicy.pdf

genetics and the environment can be important factors in asthma. With regard to specific environmental exposures, there is evidence that exposure early in life to both allergens and irritants plays a pivotal role in the development of allergy and asthma.

The draft report should cite the findings of the expert panel convened by the National Academy of Sciences that concluded:

> There is inadequate or insufficient information to determine whether or not exacerbations of asthma result from nonacute, nonoccupational exposures to cow, horse, and rodent allergens; endotoxins; houseplants or cut flowers; the bacterial agent *Chlamydia trachomatis*; pesticides; plasticizers; and volatile organic compounds (VOCs) other than formaldehyde. [39]

Data from CDC do not support the authors' contention that death rates in children due to asthma have tripled. In fact, CDC has stated that among children, asthma deaths are rare.[40] In 2000, 223 children aged 0-17 years died from asthma, or 0.3 deaths per 100,000 children compared to 2.1 deaths per 100,000 adults aged 18 and over. CDC further states that "the number of deaths and death rates from asthma increased gradually during 1980—1995 and that, although a determination with certainty cannot be made, data for 1996--1998 indicate that mortality rates are starting to plateau or decrease." [41]

TABLE 11. Annual rate* of deaths with asthma as the underlying cause of death diagnosis, by race, sex, and age group, Underlying Cause of Death data set — United States, 1980–1999[†]

	1980[§]	1985	1990	1995	1996	1997	1998	1999[¶]
Race[**]								
White	12.9	15.6	17.5	18.8	18.1	17.4	17.0	14.2
Black	27.6	34.8	40.9	46.2	46.0	42.5	44.7	38.7
Other	13.5	16.9	23.6	23.3	27.6	26.6	22.7	20.4
Sex[**]								
Male	14.7	15.9	17.8	17.9	17.7	16.6	16.5	13.1
Female	14.4	19.2	22.1	25.1	25.0	23.7	23.3	20.4
Age group (yrs)								
0–4	1.8	1.5	2.0	1.8	2.3	1.9	2.1	1.7
5–14	1.9	2.9	3.2	4.0	4.6	3.4	3.8	3.6
15–34	3.0	4.2	5.0	6.7	6.5	6.1	6.4	5.9
35–64	14.0	17.7	18.8	20.6	20.3	19.0	17.6	15.6
≥65	61.8	72.5	87.0	90.6	90.3	86.7	86.9	69.9
Total[**]	14.4	17.7	20.2	21.9	21.8	20.6	20.3	17.2

* Per 1,000,000 population.
† All relative standard errors are <30%.
§ Code 493 from World Health Organization. Manual of the international statistical classification of diseases, injuries, and causes of death. 9th revision. Geneva, Switzerland: World Health Organization, 1977.
¶ Codes J45–J46 from World Health Organization. Manual of the international statistical classification of diseases, injuries, and causes of death. 10th revision. Geneva, Switzerland: World Health Organization, 1999.
** Age-adjusted to the 2000 U.S. population.

[39] National Academy of Sciences. *Clearing the Air* (Washington, DC: National Academy Press, 2000) available at: http://books.nap.edu/books/0309064961/html/1.html
[40] http://www.cdc.gov/nchs/products/pubs/pubd/hestats/asthma/asthma.htm
[41] http://www.cdc.gov/mmwr/preview/mmwrhtml/ss5101a1.htm

IV. Chapter 3: Releases of Chemicals: Data from Industrial Pollutant Release and Transfer

ACC has long supported the U.S. Toxic Release Inventory (TRI) program, believing that emissions reporting programs like the TRI can provide valuable information that stimulates emissions reductions, communicates important environmental information to the public, and documents significant environmental improvements. ACC believes that enhancing regional transparency in emissions reporting is an admirable goal despite the well-documented difficulties of attempting to compare the vastly different emissions reporting programs in the U.S., Canada, and Mexico. However, the approach taken in the draft 2004 *Taking Stock* report goes well beyond the reports of prior years and purports to establish a direct link between children's health and reported emissions. We believe this undertaking is not grounded in science and this approach should be abandoned.

ACC is particularly concerned that the draft report implies a link between reported emissions and children's health effects. As the CEC is well aware, emissions data constitute core information to document emissions reductions and communicate environmental performance. To maximize such use of the data, however, it is essential that the data be placed in a context describing the proper interpretation of the information.

The portrayal of TRI data in the draft report is inconsistent with the U.S. government positions regarding the use of TRI data. Most importantly, the draft report has three fundamental faults that contribute to the mishandling of the emissions data:

1. The draft report erroneously implies that releases to the environment can be equated to exposures to children.

2. The draft report incorrectly implies, contrary to scientific evidence, any degree of exposure results in some risk to human health.

3. The draft report relies on lists of substances developed by activist organizations rather than by an objective authoritative body.

First, with respect to the erroneous assumption that release is equated to exposure, EPA has clearly stated (emphasis added) - "While TRI provides federal, state and local governments, the public, and industry with key environmental data, it has some limitations that must be considered; TRI data reflect releases and other waste management of chemicals, <u>not exposures of the public to those chemicals</u> [emphasis added]."[42]

[42] http://www.epa.gov/tri/2002_tri_brochure.pdf

EPA's position cautioning against implying that such national-scale releases are equated to exposures or risks is also clearly stated:

> "In our judgment, it would be inappropriate to use these results alone to draw conclusions about local concentrations and risk. The results are most meaningful when viewed at the State or national level. There are important limitations that affect how the data should be used and interpreted. … But they also are based on assumptions and methods that limit the range of questions that can be answered reliably. They cannot be used to identify exposures and risks for specific individuals, or even to identify exposures and risks in small geographic regions such as a specific census tract. These limitations, or caveats, must always be kept in mind when interpreting the results, and the results should be used only to address questions for which the assessment methods are suited [emphasis added]."[43]

Second, the potential for developing adverse effects cannot be evaluated without comparing the predicted or measured exposure to a health reference dose. Science has established that for any given chemical, natural or synthetic, the substance can have no effect, adverse health effects or at times, for certain substances, beneficial effects, depending upon the dose. The report fails to acknowledge that for any and all chemical substances, there are likely to be low exposure levels, below a reference dose or level of concern, and that such exposures are not expected to pose any significant degree of health risk to any individual, including sensitive subpopulations.

Third, the draft report is fundamentally flawed because it relies on lists of substances developed by an activist organization rather than on information from an authoritative scientific body. The lists of purported neurotoxicants and developmental toxicants in the draft report were developed by Environmental Defense (ED), an environmental activist organization. These lists have not been verified by any scientific body.

On principle, NACEC should not adopt or use any materials or methods developed by any organizations, without at least taking the necessary steps to independently verify that the information is accurate, up to date and meets rigorous standards of independent scientific peer review. As it stands, the authors have ignored an important responsibility. Readers of the draft report could be misled into concluding that NACEC has assured the lists developed by ED are scientifically accurate, comprehensive and unbiased.

There is no scientific basis for using a simplified approach based on observations from toxicity tests alone to categorize an agent as "toxic" or non-toxic," as "a developmental toxicant" or "not a developmental toxicant," as "a reproductive toxicant" or "not a reproductive toxicant" or as a "neurotoxicant" or "not a neurotoxicant" because the potential toxicity must be considered in the context of exposure -- the amount, route, duration and timing of exposure (NAS 2001).[44] Therefore, it is critical that the process

[43] http://www.epa.gov/ttn/atw/nata/natsa3.html

[44] National Academy of Sciences. *Evaluating Chemical and Other Agent Exposures for Reproductive and Developmental Toxicity.* (Washington, DC: National Academy Press, 2001).

employed to assess the potential effects of agents on the neurological system or on development and reproduction must follow a deliberative evaluative framework, such as that described in Moore et al., 1995[45] and NAS 2001. This deliberative approach, which is widely employed by objective, scientific bodies assures scientific rigor and objectivity. It is our conclusion that the ED list generation method does not meet these standards, and therefore should not be relied on by CEC.

A deliberative evaluative framework needed for scientific rigor and credibility must include:

- Utilization of an expert review team of scientists with appropriate training and experience;
- Consideration of all available and relevant data in a systematic fashion to identify dose-response relationships for both reproductive and developmental effects and also general toxicology processes;
- Utilization of a weight-of evidence approach which considers both the sufficiencies or limitations of the available studies and the quality of the data;
- Integration of toxicity potential and exposure potential to derive a scientifically justified evaluation of potential human health risk;
- Consideration of the potential for neurotoxicity, developmental toxicity and/or reproductive toxicity risk in the context of dose-response for the other types of toxicity observed for an agent; and
- Clear communication about the scientific data, assumptions, limitations and importantly both the confidence and uncertainties.

Compilations of the types of lists generated by ED have long been recognized by the mainstream scientific community as fundamentally flawed. For example, Dr. Lorenzo Tomatis, the architect of the IARC monographs, has stated that IARC quickly abandoned the notion of simply collecting scientific literature and compiling lists because "simple lists would be too rigid to take into account the multiple aspects involved in a proper evaluation."[46] Subsequently, IARC initiated a deliberative process that included assembling panels of recognized experts to conduct comprehensive reviews of scientific studies, including evaluations of study design, quality, and dose-response considerations, and decision-making using a clearly agreed upon evaluative framework.

A. Comments on Body Burden Sidebar

The draft report describes the use of biomonitoring technology (draft report at p. 55). ACC supports the use of biomonitoring as a tool to help better understand human exposure to environmental chemicals – both natural and man-made. Biomonitoring identifies certain substances in the body at the time of measurement. If gathered from a representative sample of a population – for instance, children or adults in a particular area

[45] Moore et al, 1995. An Evaluative Process for Assessing Human Reproductive and Developmental Toxicity of Agents, Reprod. Toxicol. 9(1):61-95.

[46] International Journal of Occupational and Environmental Health 8:144-152, 2002.

– biomonitoring can be used to document whether that subgroup as a whole has been exposed to some chemicals. As stated by the CDC, "Just because people have an environmental chemical in their blood or urine does not mean that the chemical causes disease."[47]

Biomonitoring does *not* provide information about (1) where the exposure came from, (2) how long a substance has been in the body or (3) what effect, if any, that substance may have on the body. The answers to these questions must come from related and relevant research. The authors should take care to clarify that evaluating exposure and evaluating risks to human health are different activities. While it's true that evaluating exposure is part of assessing risk, biomonitoring data only provide a small part of the exposure picture.

B. Comments on Specific Chemicals

1. "Health Effects of PCBs Linger Long After Exposure" and "PCBs"

The highlighted box on page 60, which purports to describe the effects of PCB exposure on children presents a one-sided view of the literature. The studies cited in the box, all by "Jacobsen and Jacobsen" [sic] suffer from numerous methodological deficiencies and, in many cases, have been directly contradicted by the work of other research groups. Schell *et al.* reviewed the exposure and dose characterization of the Jacobsons' studies and concluded, "Failure to adequately characterize the PCB exposure of these mothers, or their children, precludes any causal association between *in utero* exposure to PCBs and neurodevelopmental deficits."[48]

The issue of alleged effects of PCBs on birth weight has been thoroughly reviewed by Kimbrough and Krouskas. In contrast to the report cited in the draft report, these reviewers concluded, "Thus, correlations between PCB exposure or polluted fish ingestion and birth weight were inconsistent."[49]

Performance on visual recognition memory tests and other tests of memory have resulted in inconsistent results among the various cohorts of children tested. The results of testing of six different cohorts, including that studied by the Jacobsons, have been reviewed a number of times. All reviewers note the inconsistencies among the various study groups. For example, Kimbrough *et al.*, undertaking a comprehensive review, concluded, "In the aggregate, the studies reviewed here do not provide solid conclusive evidence that environmental exposure to PCBs and related chemicals affect the neurobehavioral development of infants and children."[50]

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[47] CDC, Second National Report on Human Exposure to Environmental Chemicals, Jan. 2003, p. 2.
[48] Schell, J.D., *et al., Regulatory Toxicology and Pharmacology*, 2001, 33:300-312.
[49] Kimbrough, R. D., and C. A. Krouskas, *Regulatory Toxicology and Pharmacology*, 2001, 34:42-52.
[50] Kimbrough, R.D., *et al., Veterinary and Human Toxicology*, 2001, 43(4):220-228.

While the highlighted section on page 60 in the draft report notes that the Jacobsons report effects lasting until age 11, the section ignores studies that have reached diametrically opposite conclusions. Gladen and Rogan, reporting on the North Carolina cohort at ages 3,4, or 5 concluded, "The deficits seen in these children on the Bayley Scales of Infant Development through 2 years of age are no longer apparent."[51] And Stewart *et al.*, recently reporting on the Oswego project observed, "Although the current data partially replicate the findings of Jacobson *et al.*, … results reported here suggest that functional recovery may occur."[52]

The last paragraph of the box on page 60 relies on the Jacobsons' descriptions of the findings in North Carolina, but, as noted above, the authors of the North Carolina study reported that early indications of potential problems in that group of children disappeared as they aged. The omission of this important piece of information by the authors of the draft report is inappropriate and misleading.

In summary, every statement in the highlighted box, relying on the Jacobsens [sic] is either directly contradicted by findings in other studies or is inconsistent with a broad review of the relevant literature. The authors of the draft report, by their selective reliance on the publications of only one set of authors, have not only skewed the presentation of the alleged health effects of PCBs, but have done the readers of the document a great disservice. Such selective reporting of the literature in a document funded by the governments of Canada, Mexico, and the United States is unacceptable.

The rest of the section on PCBs (draft report, p. 60-61) also has numerous errors. While the PCBs in some electrical transformers did have some heat transfer properties, their primary function in some transformers (only about 5% of transformers contained PCBs) was as a fire-retardant dielectric (electrical insulating) fluid.

The short section on "Health effects of PCBs" (draft report, p. 60) repeats many of the inconsistent reports noted above. The authors use the word "subtle" to describe the purported effects. Indeed, the children in these studies typically have test findings in the normal ranges for the tests. There are no reports of frank mental deficits or outcomes such as ADD or ADHD. The section also states that effects are more pronounced when exposure takes place at younger ages. This statement completely misstates the literature, in which only maternal exposures had any reported effects, with only minor exceptions. Direct exposures to children, whether through breast-feeding or diet, have not been reported to have any effects. This point should have also been noted in the "PCB levels and exposure in North America" section.

Finally, the use of PCBs as an example of the "utility of bans" is misguided and fails to recognize the efforts of the chemical and electrical industries over the past 30 years to use PCBs safely in closed electrical systems. It also fails to recognize the efforts of industry over that time to develop new and more effective ways to test the potential environmental

[51] Gladen, B.C., and W. J. Rogan, *The Journal of Pediatrics.* 1991, 119(1):58-63.
[52] Stewart, P.W., *et al.*, *Neurotoxcology and Teratology*. 2003, 25:11-22.

impact of products and to prevent the entry into the environment of potentially persistent materials. Responsible management of chemicals, like any product, provides a more sensible approach to improved environmental quality.

2. Comments on Dioxins and Related Compounds

The discussions of dioxins throughout the draft report do not present an accurate, full picture of the sources of dioxins and related compounds in the environment. In addition, the draft report does not fully utilize government data to provide the current status of dioxin emissions. Finally, data showing declining levels of dioxins and related compounds in human tissues in the U.S. contradicts the alarmist tone of the discussions about these substances.

a. Many sources contribute to environmental levels of dioxins. Dioxin is an unwanted byproduct of various industrial, societal, and natural processes.

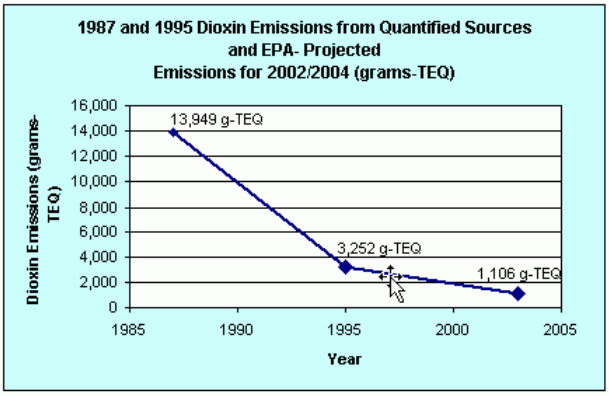
The draft report correctly reports that dioxins are unintended byproducts formed during combustion and other similar processes (draft report at p.15). However, the draft report fails to completely reveal the many varied sources of these byproducts, including industrial, societal and natural processes. For example, natural sources include forest fires and other biomass burning; societal sources include open burning, diesel emissions, coal-fired utilities, and even small quantities from cigarette smoke; and industrial sources include combustion and certain manufacturing processes. In fact, low levels of dioxins will remain, even if all man-made dioxins could be eliminated.

The authors should use more of the currently available government data and report the sources of dioxins in relative order to the current source inventories. On page 15, the text might read:

> "*Chemicals can also be changed when heated or processed. For example, dioxins and furans are not intentionally manufactured, but can be* created through a variety of processes *included during incineration, backyard burning, iron sintering*, residential wood burning, utilities burning coal utilities, and trucks using diesel fuel." ~~pesticide manufacture, etc.,~~

Note that pesticide manufacture is not a source identified by EPA and should be deleted from this sentence. (See: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=20797).

For the last several years, EPA has reported that the identified industrial sources are mostly controlled and have resulted in a 92% decline in industrial dioxin emissions over the last three decades. EPA now reports that backyard trash burning has become the largest uncontrolled, man-made source of dioxin air emissions and is turning its efforts to addressing these sources. Unfortunately, the draft report does not accurately reflect the current priorities for the U.S. When revising this report, the authors should consider more fully characterizing the multitude of societal sources, most of which play more important roles in childhood exposures.



The U.S./Canada BiNational Toxics Strategy, a collaborative effort established among environmental, industry, governmental and educational organizations, is working to reduce the reliance on backyard burning.[53] As part of its role as a facilitator for trilateral cooperation on environmental matters, the authors should add a note about the ongoing partnerships that are working to communicate the risks associated with these uncontrolled combustion sources and to move to less polluting, alternative practices.

b. U.S. dioxin emissions from man-made sources have declined over 92 percent since 1987.

Trend data from the EPA dioxin source inventory show successes from man-made sources: U.S. emissions have fallen by 92% since 1987.[54] Emissions from municipal solid waste incineration, historically, the largest industrial source of dioxin, declined more than 99 percent since 1987. Likewise, Canada reports significant progress achieving a 79 percent reduction, relative to the 1988 Canadian baseline.[55] One reason for these drastic reductions is technology. Modern incinerators are engineered to burn wastes efficiently at high temperatures and to minimize the conditions known to promote the formation of dioxin and other unwanted byproducts. One benefit of including these U.S. and Canadian dioxin inventory data in a *Taking Stock* report would be to foster a conversation about the benefits of these types of inventories and to encourage technology transfer among U.S., Canada, and Mexico.

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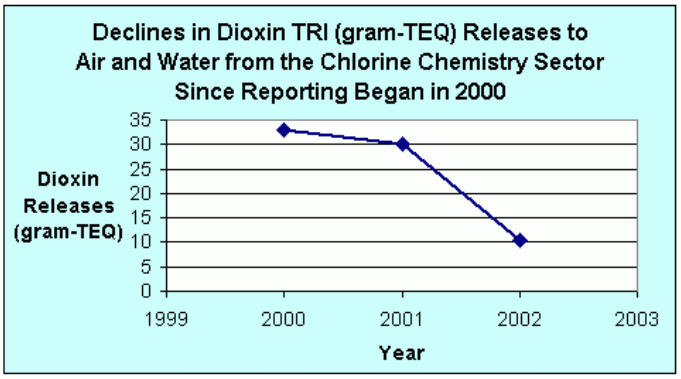
[53] See: http://www.c2p2online.com/main.php3?doc_id=282&section=138 and http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=20797

[54] EPA, Database of Sources of Environmental Releases of Dioxin-Like Compounds in the United States, available at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=20797

[55] Great Lakes BiNational Toxics Strategy 2002 Report, Chapter 3.0 Dioxins/Furans, available at: http://binational.net/bns/2002/english/2002-GLBTS_03_dioxin.pdf

Unfortunately, the draft report did not report the dioxin TRI data for 2001, which was available to the authors mid year 2002. If they had included two years of data, 2000 and 2001, CEC could have reported declining releases.

In fact, preliminary 2002 TRI data for facilities in the chlorine chemical sector shows a further decline in dioxin releases to air.[56] These preliminary data indicate acceleration in the progress by the chlorine manufacturing sector in reducing dioxin in 2002 -- it achieved a 68 percent reduction in dioxin releases to the environment since 2000.



The draft report characterizes releases of dioxins and furans in the TRI and NPRI data sets as "environmentally significant" (draft report, p. 25). However, it comments these releases are 'not matched' resulting in incomparability between TRI and NPRI and therefore are not included as part of this report. Even if these data were fully comparable, they do not capture important societal and natural contributions of dioxins to the environment. This is an artifact of pollutant release and transfer reporting procedures. The governments of the U.S. and Canada have invested considerable effort in augmenting pollutant release and transfer data with robust source inventories. By referring to these dioxin source inventories, the authors can accurately reflect the dramatic decreases in dioxins emissions since the late 1980's due to governmental controls and voluntary industrial efforts.

c. Current levels of dioxin in our bodies are so low that a 2003 study by the CDC reported dioxin levels in the blood of the average U.S. resident were below limits of detection reported by CDC.

Dioxin levels in the average resident of the U.S. are generally below the detection level for the analytical method used by the CDC and reported in its 2003 National Exposure Report.[57] Over time, these data will establish a national baseline. Other studies have investigated concentrations of dioxins in human tissues and EPA scientists have reported a sharp decline from the 1970s.[58] The draft report also expresses a concern about human milk concentrations of dioxins. Government scientists have found that levels of dioxins in the body correspond to the date of birth. People born during the 1970s have higher

[56] Preliminary TRI data from the chlorine sector, http://dioxinfacts.org/sources_trends/treands_04_10_04.html

[57] CDC, 2003, Second National Report on Human Exposure to Environmental Chemicals, Polychlorinated Dibenzo-*p*-dioxins, Polychlorinated Dibenzofurans, and Coplanar Polychlorinated Biphenyls. pp. 97-118. available on-line at: http://www.cdc.gov/exposurereport/dioxinfuran/default.htm

[58] Lorber, M. (2002). A pharmacokinetic model for estimating exposure of Americans to dioxin-like compounds in the past, present, and future. *Sci. Tot. Environ.* 288, 81-95.

levels than children born today will ever experience.[59] Clinicians have asserted that human milk is the best form of nutrition for a human infant. In fact, some have reported that breastfed infants suffer fewer diseases and chronic illnesses and reduced mortality than infants who have not been breastfed. With efforts to apply controls to uncontrolled sources, we can optimistically look to a future where these levels are further reduced.

Today's levels are dropping to what some might characterize as nearly background levels. It is critical to reflect upon the statement of the U.S. government, "Even if all human-generated dioxins could somehow be eliminated, low levels of naturally produced dioxins will remain, as will reservoirs."[60] Therefore, the authors should revise the tone of some statements to reflect an understanding that complete elimination of all dioxin releases is an unrealistic goal. Dioxins and related compounds will continue to be found in human tissues given our advanced analytical capabilities to detect smaller and smaller amounts.

For this draft report, the key point to consider is whether the authors are accurately using all available government data to represent a realistic characterization of the wide variety and number of sources of dioxins and related compounds or whether it is choosing to focus on reporting data that continues to point the finger only to industrial sources and cause unwarranted alarm in the public.

Below we provide valuable links to resources not yet covered in the draft report. With the draft report's overwhelming reliance on websites sponsored by special interest groups, we request that the authors include links to industry sites. Specifically, we request that under Step One (draft report, p.75-76), the report include the following links:

- Seek information about sources of dioxins and related compounds from Canada at http://binational.net/bns/2002/english/2002-GLBTS_03_dioxin.pdf
- Understand the variety of sources in the U.S. at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=20797
- Explore industrial dioxin activities and track their progress in achieving emission reductions at: http://dioxinfacts.org/index.html and http://trifacts.org/index.html
- Learn more about why government, industry, environmental, and educational groups are working together to reduce reliance on backyard trash burning at http://www.c2p2online.com/main.php3?doc_id=282&section=138 and http://dioxinfacts.org/sources_trends/trash_burning.html

[59] Pinsky, PF, Lorber, MN, 1998. A model to evaluate past exposure to 2,3,7,8-TCDD, Journal of Exposure Analysis & Environmental Epidemiology, Vol. 8(2): 187-206

[60] Interagency Working Group (IWG) on Dioxins, 2003. Questions and Answers About Dioxin, Question 1, available at http://www.cfsan.fda.gov/~lrd/dioxinqa.html#g7, accessed 5 May 2004. The Dioxin IWG represents the key U.S. agencies, including Department of Health and Human Services, Department of Agriculture, Department of Veteran's Affairs, Environmental Protection Agency, Department of Defense, Department of State, Executive Office of the President, and the National Science and Technology Council.

3. Comments on Metals

The draft report mischaracterizes metals by using TRI data for metals and metal compounds and combining it with the ED Scorecard's listing for a particular metal. This blending of data points misconstrues the Scorecard's listings, which are already problematic. In addition, the draft report's mischaracterization may prove detrimental to children's health by unnecessarily alarming parents about metals and thus cause parents to neglect to ensure their children receive the recommended allowances of essential metals such as chromium, copper, manganese, nickel, and zinc. Reading the draft report, parents may conclude that any exposure to these essential minerals will harm their children, when the opposite may be true.[61] The TRI data do not correspond to exposure data. These data must be presented with a balanced discussion of children's health issues.

a. EPA's TRI Data Do Not Correlate with Information in ED's Scorecard

The TRI data available regarding metal compounds do not correlate with the metal compounds included in ED's Scorecard. EPA's TRI data do not include release information specific to copper arsenate or zinc sulfate. Rather, the TRI data include only copper compounds and zinc compounds. The draft report should be corrected to exclude copper compounds and zinc compounds.

A similar problem exists regarding the inclusion of zinc and its compounds. As noted above, the TRI data do not include specific zinc compounds, but only total releases for all zinc compounds. The draft report should be corrected to exclude zinc compounds.

Given the significant weaknesses of the references used by ED's Scorecard (*i.e.*, that the listings have not been peer-reviewed, have not been the subject to a public notice and comment, and are not in all cases reliable or accurate) the draft report should not include them.

b. Metals Are Essential To Children's Health

We are concerned that the draft report fails to include information regarding the necessity of certain metals to children's health, and instead presents metals only in a negative, detrimental light. The draft report, for example, lists chromium, nickel, and their compounds as carcinogens (draft report, p. 28); copper, zinc, and their compounds as developmental toxicants (draft report, p 35); and zinc, manganese, and their compounds

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[61] For example, in addition to being an essential mineral, zinc may be beneficial as a supplementary medication in the treatment of children with attention deficit hyperactivity disorder. National Electronic Library for Health, "Zinc Supplements and Hyperactivity in Children," available at http://www.nelh.nhs.uk/hth/zinc.asp (last updated May 5, 2004).

as neurotoxicants (draft report, p. 45). Chromium, copper, manganese, and zinc are among the nine trace minerals required by humans.[62] Chromium is important in the metabolism of fats and carbohydrates, and stimulates fatty acid and cholesterol synthesis, which are important for brain function and other body processes.[63] Copper helps in the formation of red blood cells and in keeping the blood vessels, nerves, immune system, and bones healthy.[64] Manganese is used in the formation of bone and in enzymes involved in amino acid, cholesterol, and carbohydrate metabolism.[65] Although the draft report lists zinc as a developmental toxicant, maternal zinc deficiency can slow fetal growth.[66]

Since metal elements play a critical role in children's health and since TRI data cannot be used as a surrogate for exposure data, this report must be clear regarding its discussions of metals.

V. Comments on Chapter 4: What's Being Done to Protect Children's Health

"Chapter 4: What is Being Done to Protect Children's Health From Toxic Chemicals" omits a number of important national and international programs that are worthy of mention, and inappropriately highlights some untested approaches at the state and local level.

The report lists and emphasizes municipal and state bans on pesticides as a means to protect children (draft report at p. 68). Unquestionably, reasonable, risk-based regulation on the use of pesticides benefits all, and careful and judicious use of those products where children might be exposed is the goal of pesticide regulatory and labeling programs. There is also a clear continuing need to educate consumers, parents and industrial applicators on proper use. On the other hand, it is not clear whether bans, even those limited to where children might be exposed, will actually produce net public health benefits. The scientific community has not assessed the full impacts of a number of foreseeable unintended consequences of bans, such as increases in vermin, cockroaches (asthma) bees, wasps, and ticks (lyme disease) and uncertain health impacts from

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[62] *The Merck Manual of Diagnosis and Therapy* at Chapter 4, "Mineral Deficiency and Toxicity," available at http://www.merck.com/mrkshared/mmanual/section1/chapter4/ 4a.jsp.

[63] National Library of Medicine, "Medical Encyclopedia: Chromium in Diet" (Feb. 26, 2003), available at http://www.nlm.nih.gov/medlineplus/ency/article/002418.htm.

[64] National Library of Medicine, "Medical Encyclopedia: Copper in Diet" (Oct. 17, 2003), available at http://www.nlm.nih.gov/medlineplus/ency/article/002419.htm.

[65] Institute of Medicine of the National Academies, "Dietary Reference Intakes: Elements," available at http://www.iom.edu/Object.File/Master/7/294/0.pdf.

[66] NIH, "Facts About Dietary Supplements -- Zinc," available at http://www.cc.nih.gov/ccc/supplements/zinc.html (last updated Dec. 9, 2002).

substitutes. Further study is needed on the benefits of this approach for children's health. Until that is done, it is injudicious to tout it so highly in the draft report.

In listing national activities, the draft report appropriately includes the general environmental health and safety regulatory programs of the three countries. Children have always been in the ambit of those protected by our environmental regulatory schemes. What is missing is a recognition of the ongoing inquiry as to whether and where children are more susceptible or more exposed so that we can be sure those existing regulatory programs adequately take children into account. Here the report should note in this section: government funded research, such as that conducted by NIEHS (some of which is jointly sponsored by industry); the EPA Voluntary Children's Chemical Evaluation Program, piloting a scheme for evaluating chemicals to assess their potential effects on children; and the proposed U.S. National Children's Study, a planned longitudinal cohort health study of 100,000 children that will look at the effects of the chemical, biological, physical and social environmental factors on children.

Likewise, the discussion of international initiatives fails to include two significant efforts – the Healthy Environments for Children Alliance (HECA) (http://www.who.int/heca/en/) and the recommendations and agreed actions from Forum IV of the Intergovernmental Forum on Chemical Safety (IFCS) (http://www.who.int/ifcs/Documents/Forum/ForumIV/FIVreport/FIVreport_en.doc). HECA is an alliance of governments, intergovernmental organizations and non-governmental organizations inaugurated at the Johannesburg World Summit on Sustainable Development in 2002 to address global environmental risks to children's health. The outcome report of IFCS Forum IV in Bangkok included such recommendations and agreed actions as calling on governments and others: to consider exposures during preconception, gestation, infancy, childhood, and adolescence when assessing risks; to prepare national assessments of priority concerns for children; and to promote education and training on children's environmental health and the development indicators.

VI. Comments on Chapter 5: What Needs to be Done to Protect Children's Health

Society has made great progress in improving the lives and well being of children over the last century. We believe that advances in chemistry and allied technologies have played a critical role in making that possible by contributing to the development of products and services that have improved medical care, nutrition, safety and other determinants of a child's quality of life. However, there is still much to be done to help ensure that the trend of improvement continues and that all children benefit. There are legitimate concerns about the potential health effects on children low-level environmental exposures arising from products and the environment. We believe that industry has a responsibility to seek answers to these questions and to respond to concerns at the same time it continues to pursue chemical and technological innovation to improve children's lives.

The business of chemistry is built on science, and we believe that decisions made with the soundest scientific basis provide the best protection for human health and the environment. Recommendations to protect children's health from chemicals should be made from a scientific, not an emotional, basis. In the concluding chapter of the draft report, the authors again allege that increases in childhood disease rates are related to exposure to chemicals and make recommendations to reduce exposure that is based on their faulty analysis. Related to the suggestions made in this chapter, ACC supports the following:

- Ongoing efforts at pollution prevention and reductions in emissions. Declining levels of TRI data in the chemical sector reflect the industry's commitment to reducing emissions at the same time production levels increase.
- Reliance upon a scientific foundation for risk-based decision-making by government, industry and other stakeholders. The draft report's flawed analysis of health effects and their relationships to chemicals on the TRI is not an example of risk-based information.
- Continued research into the effects of chemicals on human health and the environment. ACC member companies participate in the EPA Voluntary Children's Chemical Evaluation Program, a pilot program to explore more efficient and targeted information gathering for assessing the potential risk of chemicals to children's health (for more information, see http://www.epa.gov/chemrtk/vccep/index.htm). In addition, the ACC Long-Range Research Initiative sponsors independent research that is aligned with health and environmental issues of highest priority to society, as well as the chemical industry (for more information, see http://www.uslri.org/).
- Improved monitoring and surveillance of health information. ACC supports the use of biomonitoring to provide unique exposure information to physicians, scientists, and health officials to help improve our understanding of the relationship between exposure to chemicals and human health. The limitations of such surveillance data, however, should be carefully described to the public and no implications should be made about its relationship to health status. Rather, this information should be used in the context of risk assessment and risk management.
- ACC supports the planned National Children's Study. We believe the Study, which is intended to delve into the interrelationships among genetics and the physical, chemical, biological and social environments of children will create better data for better decisions. The NACEC can have a constructive role in coordinating trilateral research related to the U.S. NCS.
- Providing useful and science-based information to policy makers, physicians and parents. We believe the resources listed beginning on page 76 of the draft report do not provide such information. The links and references provided will take readers to web pages (and the websites that house them) which are alarmist in tone, based on selective and outdated science, and clearly partisan in view. The promotion of these websites and the groups that create them in a document sponsored by and paid for by the three contributing governments constitutes an

implicit endorsement of their products. Since several of these web sites are critical of government activities (particularly of the U.S. EPA), we find their inclusion to be inappropriate.

VII. Conclusion

ACC appreciates the opportunity to review this report and supports the expert review process that NACEC announced in the cover letter released with the draft report. We hope that the expert review will rely upon the principles of peer review used by the U.S. EPA[67] and that participants in the review will be qualified experts with no previous involvement in the drafting of the report. The transparency of the peer review process is important (not only to the three governments and the interested public, but the industrial providers of the emissions data discussed in the report) so that the NACEC and the three governments can stand behind the final report if one is issued.

[67] http://www.epa.gov/osa/spc/htm/peercov.htm

Attachment 1

March 22, 2002

By E-Mail and Post

Erica Phipps
Program Manager
Pollutants and Health
Commission for Environmental Cooperation
393 rue St-Jacques ouest, bureau 200
Montreal, Quebec, Canada H2Y 1N9

RE: Comments of the Asociácion Nacional de la Industria Quimica, the Canadian Chemical Producers Association and the American Chemistry Council on North American Commission for Environmental Cooperation's Draft "Cooperative Agenda for Children's Health and the Environment in North America"

Dear Ms. Phipps:

The Asociácion Nacional de la Industria Quimica (ANIQ), the Canadian Chemical Producers Association (CCPA) and the American Chemistry Council (ACC)* appreciate the opportunity to comment on the Commission for Environmental Cooperation of North America's Draft Cooperative Agenda for Children's Health and the Environment in North America. We commend the CEC's commitment to this trilateral effort to improve children's health and its thoughtful proposals aimed at achieving that objective.

Society has made great progress in improving the lives and well being of children over the last century. We believe that advances in chemistry and allied technologies have played a critical role in making that possible by contributing to the development of products and services that have improved medical care, nutrition, safety and other determinants of a child's quality of life generally. However, there is still much to be done to help ensure that the trend continues and that all children of the world benefit. There are questions and concerns about the effect on children of chemicals in products and the environment. We believe that industry has a responsibility seek answers to these questions and to respond to concerns at the same time as it continues to pursue chemical and technological innovation to improve children's lives.

ANIQ, CCPA and ACC agree with the basic thrust of CEC's proposed agenda to address some of these environmental concerns -- encouraging intergovernmental programs that will: 1) improve the use of analytical tools to better evaluate risks to children's health; 2)

expand our knowledge base about the status of children's health and the impacts of the environment on their health; 3) provide relevant information to health professionals, parents and the public; and 4) address priority problems under existing CEC programs.

Our industry is one built on science, and we fundamentally believe that decisions made with the soundest scientific basis provide the best protection for the human health and the environment. Consequently, we particularly support those aspects of the Cooperative Agenda that help build the factual and analytical foundation for better decisions:

- **4.1 and 4.2**, advancing the understanding and use of risk assessment, in particular promoting consistent approaches among the three countries and providing training for risk assessors;
- **4.3 and 5.3**, developing the tools to better use economic valuation to support and improve decisions aimed at protecting children;
- **5.1 and 5.2**, developing appropriate indicators of children's health so that progress can be measured and the success of prevention strategies can be assessed and encouraging collaboration on longitudinal cohort studies; and
- **6.2,** working with health care professionals.

Specific Areas of Comment

Asthma.

We note only that asthma is a complex condition with a multiplicity of factors contributing to its cause and to the severity of its effects. Looking narrowly just at diesel exhaust at the border, as suggested in 1.1, may not be productive by itself. A variety of studies are already underway exploring this debilitating disease and CEC should take cognizance of those efforts and design its activities to appropriately supplement and enhance them. Additionally, CEC's efforts should focus on areas that have the potential to lead to feasible solutions to alleviate the causes or incidence of asthma. In that regard, items 1.2 and 1.3 suggest the most promise.

Toxic Substances.

We concur with CEC's approach of integrating its children's health activities relating to toxic substances into the existing Sound Management of Chemicals Program. On the other hand, with respect to the *Taking Stock* report we have some concerns about what kind of information about children's environmental health CEC intends to include. ANIQ, CCPA and ACC support providing accurate, relevant and understandable information to policy makers, parents and concerned citizens. There are many opportunities for miscommunication, however, when trying to connect emissions information with health status, as would appear to be the intention here.

We caution against drawing overly simplistic connections or implying unambiguous correlations between emissions and children's health threats. Because the causal connection between emissions and health outcomes may not be well understood, particularly with respect to effects on children, we recommend that CEC submit its plan for providing such information in *Taking Stock* to an expert panel for scientific peer review to assure the information is relevant and consistent with the science. In addition, we recommend that CEC include background information and caveats, where

appropriate, to place the *Taking Stock* information in context and to provide useful information to parents, policy makers and the public.

Risk Assessment and Economic Evaluation

We strongly support strengthening the use of the tools of risk assessment and economic evaluation for better information-based decision making. The suggested workshop to share principles and methodologies for conducting risk assessments and the risk assessment training would be particularly beneficial, fostering consistent risk assessment approaches across the three governments and leveraging limited resources. With respect to the training, CEC may want to work with or build off of the work already being done by the International Programme on Chemical Safety along similar lines.

One note of caution, however, CEC should be aware that there exists some scientific and policy debate about how best to evaluate the effects of substances on children, particularly about appropriate defaults and assumptions necessary to account for uncertainties regarding potentially differential exposure or sensitivities of children. A useful article by Dr. Gail Charnley, discussing some of the areas of controversy, can be found at http://www.rppi.org/ps283central.html. Consequently, it would be inappropriate to suggest in risk assessment training that there is a formulaic, one-size-fits-all risk assessment approach for children. If done correctly, both the trilateral workshop and training for assessors on children's health risk assessment should improve understanding of the complexities of the issues involved. ANIQ, CCPA and ACC are very interested in providing support for these efforts.

Likewise, we support the proposal on integration of risk assessment and economic valuation. As a starting point, CEC should be aware that OECD has already done some work in this area. As the output of a joint workshop between risk assessors and economists, a set of recommendations for integrating risk assessment and economic information to facilitate decision-making was issued. Although these recommendations do not specifically address children, we urge CEC to build upon this seminal work, which is available at http://www.olis.oecd.org/olis/2000doc.nsf/LinkTo/env-jm-mono(2000)5. At a minimum, appropriate use of economic information can help identify cost-beneficial risk management strategies to protect and improve children's health.

Strengthening the Knowledge Base.

First, when gathering data about children's health and the environment, we urge CEC to take a broad view of "environment, " one that includes the physical, chemical, biological and psychosocial environmental influences on a child's well being, which is the approach taken by the US National Children's Study. If the Commission looks too narrowly, it may miss critical factors or combinations of factors that are major contributors to children's health problems, thus missing important opportunities to affect positively children's health. Second, we recommend that CEC use the information on the status of the children's health to also evaluate what are the most significant problems, so that resources can be allocated appropriately.

We support efforts to facilitate cooperation among the three countries on implementing any longitudinal cohort study. At a minimum, we agree with the proposal to facilitate participation by Canadian and Mexican officials and researchers in the development of

the US National Children's Study, as suggested by 5.1. Those individuals who participate in the design and implementation planning meetings for that Study will be able to take the lessons learned back to apply to development of similar programs in their countries as those are adopted.

As 5.2 notes, indicators can be valuable tools to identify priority problems, target action, set goals and measure results toward those goals. For those reasons, we support the development of appropriate indicators of children's health. That said, as with providing children's health information in *Taking Stock*, discussed above, we recognize there is the potential for misinformation if not executed with care.

A critical determinant of the utility of a particular indicator is whether it measures the right factors and does so accurately. There must be a strong, demonstrable correlation between the indicator and the underlying condition being measured. Typically, environmental indicators measure the presence or absence of contaminants in the environment or, perhaps, the health of an ecosystem as indicated by vibrancy of important species in the ecosystem. Health indicators usually measure health outcomes, such as reported disease incidence, or indirect evidence of those outcomes, such as frequency of hospital stays or doctor visits, etc. "Environmental health indicators," on the other hand, are not well defined. Presumptively, they imply a strong relationship between certain environmental conditions and health status, such that measuring one says something about the other.

We support the development of a system for measuring the health and well being of children, and doing so broadly will help spot trends or acute problems that might require the intervention of the public health system in the three countries. We urge CEC to consider carefully the validity of any environmental health indicators it proposes. Preferably, the indicators should look more broadly at measuring children's health status, taking a comprehensive view of environment (physical, chemical, biological and psychosocial environmental influences on a child's well being) as it may impact health status. Finally, we again urge CEC to involve experts in developing and peer reviewing its indicator plan.

<u>Public Information and Outreach.</u>

ANIQ, CCPA and ACC strongly support programs that encourage cooperative efforts among those who play critical roles in child health and safety protection by providing relevant and understandable information to policy makers, health care provides, parents, industry and concerned citizens. In particular we are enthusiastic about the initiative to facilitate greater cooperation and communication among health professionals in the three countries and urge you to work with the mainstream medical professional societies, such as those of pediatricians or of family practitioners who have "on the ground" experience treating children.

Policy makers, industry, children's advocates, the medical and scientific communities, and the public in our three countries need to work in partnership to ensure that the greatest threats to children's health and safety are identified and addressed in a timely fashion. The ANIQ, CCPA and ACC are committed to making positive contributions to this effort.

Again, ANIQ, CCPA and ACC thank CEC for providing the opportunity to comment on its Draft Cooperative Agenda for Children's Health and hope that CEC finds our input helpful. If you have any additional questions, please do not hesitate to contact either of us. We look forward to working with CEC in implementing its agenda, bringing the scientific expertise, experience and resources of the industry to help address the issues as appropriate

Regards,

/s/

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Vice-president, Environment, Health & Safety
Asociácion Nacional de la Industria Quimica
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/s/

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Ph: 613-237-6215

/s/

Sarah Brozena,
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703-741-5159

cc:Dr. Carlos Santos Burgoa, Director General de Salud Ambiental, Mexico
Judith E. Ayres, Assistant Administrator International Affairs, USEPA
E. Ramona Trovato, Director, Office of Children's Health Protection, USEPA
Tony Myres, Coordinator, Special Projects, Environmental Substances Div, Health Canada
Julie Charbonneau, Manager Environment and Human Health, Environment Canada

[*] The Asociácion Nacional de la Industria Quimica, the Canadian Chemical Producers Association and the American Chemistry Council represent the represent the leading companies in Mexico, Canada and the United States, respectively, engaged in the business of chemistry. Our members apply the science of chemistry to make innovative products and services that make people's lives better, healthier and safer. The chemical industry is committed to protecting children. This commitment is personal as well as professional; the industry is made up of parents, grandparents, aunts and uncles who value children and appreciate the need to safeguard their well-being. We do so through our Responsible Care® program, through our long-range research initiative and through our products. We are committed to improved environmental, health and safety performance through Responsible Care®, common sense advocacy designed to address major public policy issues, and health and environmental research and product testing.