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I wish to express my strong support for the bill, “Toxics Disclosure and Innovation for Healthy Children” (RB 6526). Without doubt, there can be no higher priority than for the State of Connecticut to enact this forwarding looking legislation that protects the health and well-being of our young children. It does so by creating and maintaining “. . . a list of priority chemicals that are of high concern to children after considering a child’s or developing fetus’s potential for exposure to each chemical”. I make these comments from the vantage point of a biomedical scientist, who has been deeply involved in the public policy arena of environmental health sciences for the past forty-five years, both nationally and internationally. Currently, I teach a graduate level course on children’s environmental health at UConn Health Center in their MPH program.

At the outset, let me make one crucially important statement – *children are not scaled-down, younger versions of adult human beings*. Too often we tend to forget this fundamental biological reality. This profound lack of understanding happened all too often in the past when we enacted health-based policies to protect the public from unsafe products and hazardous substances found in our environment.

A young child’s health is uniquely susceptible to a number of harmful environmental factors, such as ionizing radiation, toxic chemical compounds and infectious biological agents. These adverse impacts occur not only at lower exposure levels than those that affect a fully grown person, the harms they exert can be of a *quite different nature* not observed in adult men and women. Alluding to these differences, the 1993 landmark National Academy of Sciences’ (NAS) report on pesticides in the diets of children stated that “[q]ualitative differences in toxicity are the consequence of exposures during [a young child’s] special windows of vulnerability – brief periods early in development when exposure to a toxicant can permanently alter the structure or function of an organ system.”<sup>1</sup>

To begin with, a newborn baby breathes over three times as much air, and an infant drinks two and half times more water, than an adult. A youngster less than five years old, pound for pound of body weight, consumes three to four times more food than an adult. Moreover, young children constantly place their hands in their mouths, picking up materials that may be contaminated with harmful substances found indoors or in the playground. This is also the case with infants and toddlers when they come into contact, either orally or dermally, with toys, furniture, car seats, clothing and other household products containing

unsafe ingredients. Although the child's resting metabolic rate is greater than an adult, its body's metabolism is not fully developed and therefore processes materials differently when ingested or inhaled. This also pertains to a young child's newly forming immune system, which is generally less capable of reacting against or detoxifying harmful substances in the body.

At the same time, many organs of a child's body after birth and months later are still undergoing critically important development. For instance, the child's lung undergoes increased expansion of its airway passages and in the formation of alveoli sacs through which air and other gases are exchanged with the bloodstream. This factor alone makes the young child's respiratory system especially vulnerable to infectious agents, and to a variety of harmful air pollutants and airborne allergens.

Similarly, during the first year after birth, the child's brain is rapidly developing, allowing for proper growth of its sensory and mental capacities. This includes the ability to see and to hear more acutely, to acquire speech and language skills, and to develop higher cognitive ability. Therefore, exposure of young children at this stage to neurotoxic agents, such as lead or mercury, can cause severe impairment of sensory and cognitive functions, lead to behavioral disorders, and may result in permanent brain damage or death. Because of these non-genetic, acquired neurological impacts, even relatively low levels of blood lead levels (less than 5 to 10 micrograms per deciliter) are correlated with significant IQ declines in school age children.<sup>2</sup>

Although in recent decades we have made remarkable progress in decreasing overall childhood mortality and preterm birth rates in the United States, we have seen increases in childhood diseases not known to be highly prevalent in the 1960s and 1970s. As presented in the recently released report by the US Environmental Protection Agency (USEPA) (*"America's Children and the Environment"*, January 25, 2013), this includes unprecedented increase in the past four decades in the incidence rates of childhood asthma, attention deficit hyperactivity disorder (ADHD), autism, and obesity.<sup>3</sup>

For instance, among black non-Hispanic children and adolescents of all income levels, greater than 15% of them suffer from asthma. In 2010, more than 10% of boys of all ethnic groups in the US (5 to 17 years old) were diagnosed with ADHD, while incidence rates of autism among all children in the same age group increased ten-fold between 1997 and 2010. Of great concern is the rise of obesity among young people (2 to 17 years) of all ethnic and income groups in this country, rising from 5% in the late 1970s to well over 15% in 2008.

According to the Center for Disease Control and Prevention (CDC), today one in 400 children and adolescents suffer from type I and type II diabetes in the US, with a reported 215,000 cases in 2010.<sup>4</sup> In addition, childhood cancer incidence rates for the past two decades, such acute lymphoblastic leukemia and central nervous system tumors, continue to remain unacceptably high (25 to 35 per million children, under 19 years), followed by incidences of germ cell tumors, soft tissue sarcomas and Hodgkin's lymphoma (each greater than 10 per million children).<sup>5</sup>

At present, even though federal and state regulatory framework in controlling harmful substances takes some account of children's vulnerability, we are now faced with two continuing dilemmas in the marketplace: (1) tens of thousands of existing chemical substances that were inadequately tested for their impacts on young children, and (2) myriads of new substances and consumer products being introduced

each year that still do not routinely test for their potential ill effects on children's health, such as developmental, genotoxic or neurological disorders.

In 2006, based on these enormous testing data gaps and inadequate regulatory structure in their region, the European Union (EU) enacted a far reaching set of mandatory guidelines pertaining to the production and use of potentially toxic substances in commerce. Often referred to by its catchy acronym, **REACH** (*“Regulation, Evaluation, Authorization and Restriction of Chemicals”*), it is one of the most sweeping and comprehensive regulatory approaches for controlling toxic substances in the marketplace.<sup>6</sup> These guidelines include the evaluation of *imported* industrial chemicals and manufactured products into EU countries, which falls under the overall jurisdiction of the European Chemical Agency (ECHA).<sup>7</sup> The agency's governing philosophy is the adoption of the concept of the “precautionary principle”, which places the burden of environmental and health risk evaluation of commercially used chemicals on the producer, the formulator and the product manufacturer.

In order to develop its final list of chemical compounds that are to be placed under strict regulatory oversight or marketplace restrictions, ECHA has presently prepared a preliminary dossier of 138 chemicals called the candidate list of “Substances of Very High Concern” (SVHC)(last updated in December 19, 2012).<sup>8</sup> To be placed on the SVHC list, the agency in cooperation with the European Commission (EC), receives information on the production, use and potential toxic effects of a chemical substance from both governmental and non-governmental (NGO) sources. Once placed on the SVHC list, a public consultation process is formally set into motion, whereby interested parties have up to 45 days to provide further comments on the decision.<sup>9</sup>

At the same time, under its Community Rolling Action Plan (“CoRAP”), ECHA has published its first list of 90 chemical substances that are now being evaluated in greater detail for their environmental and human health risks.<sup>10</sup> I have examined this initial list, and selected 20 chemical compounds that may pose significant human health risk because of their *widespread consumer use* (see **Table A**). These include well known toxic chemicals, such as methanol, formaldehyde and p-cresol. At the same time, there are a number of chemical compounds that pose potential health risks to young children, which have carcinogenic, mutagenic and reproductive impacts or are endocrine disrupters.

These substances should be placed on a priority list that is being considered under the proposed legislation in the State of Connecticut. The EU list should be combined with the lists of compounds obtained from other agencies at the federal level – USEPA, Center for Disease Control and Prevention (CDC), Agency for Toxic Substances and Disease Registry (ATSDR), Consumer Product Safety Commission (CPSC), Food and Drug Administration (FDA) – and at the state level (New York, New Jersey, California, Vermont, Maine, Washington).

In the intervening twenty years since the publication of the NAS's pesticide report, we have acquired considerably better knowledge of the adverse health impacts on young children – including at the prenatal and perinatal stages of growth – of a multitude of chemical compounds. These include the hitherto unknown impacts of hormone-mimicking, endocrine disrupters on reproductive organs, and the developmental and neurotoxic effects of a wide variety of commercially used and formulated chemical compounds, such as bisphenol A, phthalates and polybrominated diphenylethers (PBDE). In a critical review of its potential toxicity, phthalate compounds were amply demonstrated to cause reproductive and developmental effects in a number of chronic animal studies.<sup>11</sup> In one human-based prospective study of

460 mother-infant pairs, fetal exposure to phthalate compounds during gestation were correlated with reduced mental and psychomotor development at an early age (6 months) of the newly born children.<sup>12</sup>

Of particular concern is the wide use of different kinds of phthalate compounds – diethyl phthalate (DEP), diethylhexyl phthalate (DEHP), dibutyl phthalate (DBP), butylbenzyl phthalate (BBP), diisononyl phthalate (DIIP), to name the most prominent chemicals – as ingredients in vast variety of consumer products, such as plasticizers in plastic containers (e.g., PVC bottles) and food wraps, in cosmetic products (such as skin softeners and moisturizers) and fragrances, and in baby lotions, shampoos and powders.<sup>13 14</sup> Since they are loosely bound in these products, phthalates can easily leach out into foodstuffs or adhere to the human skin. In addition, a number of phthalate compounds can be found in certain food products, detergents, flexible toys, modeling clays, paints & inks, adhesives & glues, textiles, medical devices, electronics, building materials, etc.<sup>15</sup> In other words, phthalate compounds are ubiquitously present in many commercial and consumer products.

In addition, biomedical advances in recent years show the exquisitely sensitive nature of the embryo and the growing fetus to foreign substances that pass across the placental barrier during gestation. In particular, severe birth deformities, such as cleft lip/palate and cerebral anencephaly, are caused by exposure to toxic teratogenic substances or by lack of proper nutrients during the first several weeks after conception.<sup>16 17</sup> We now know that tobacco smoking and alcohol ingestion by a child's mother can retard overall fetal growth.<sup>18</sup> Such maternal habits can also cause attention deficit hyperactivity disorder (AHDH) to appear in sizeable number of young children.<sup>19</sup>

A mother's dietary regimen before and after conception can be a determining factor in child's health status after birth. In a study recently published in the *Journal of the American Medical Association (JAMA)*, February 13, 2013, the maternal intake of sufficient amounts of folic acid (a water-soluble vitamin B) four weeks before becoming pregnant significantly reduced (by 40%) the incidence of childhood autism.<sup>20</sup> The conclusions of this well-conducted study are fairly robust, since greater than 85,000 Norwegian women were followed over a four to ten year period.

Past studies showed that folic acid deficiencies during pregnancy are a risk factor to neural tube defects in a growing fetus.<sup>21</sup> This could lead to serious neurologic and developmental problems in a young child, such as behavioral disorders and reduced ability to acquire cognitive skills.<sup>22</sup> Unfortunately, in animal studies, it was shown that common disinfection byproducts found in drinking water, such as trihalomethanes (THMs) and haloacetic acid, could deplete the protective stores of folic acids.<sup>23</sup> In an epidemiological study of nearly 400,000 births in Taiwan, exposure to chlorinated disinfection byproducts was correlated with increased number of birth defects, such as ventricular septal defect, cleft palate and anencephalus.<sup>24</sup>

Thus, we see that in today's society, the growing fetus and the young child's ability to reach a healthy and productive adulthood is constantly under siege by the sheer abundance and intricate chain of human made products containing potentially harmful ingredients. Once again, we see the need to remain especially vigilant in developing educational programs and regulatory measures in controlling hazardous substances that impact the health of a young child. This involves a better informed public, which should also include more intensive counseling of women of child-bearing ages. Without doubt, it requires a more rapid phase-out of toxic substances presently found in child-related products, such as toys, clothing, school supplies, restraining devices, and fresh produce and prepared foods.

To replace these compounds, we should place greater emphasis in introducing safer, alternative substances in the marketplace. Starting from now on, we should adopt a societally enlightened and ambitious policy that only non-toxic and innovative “green” chemical compounds – and highly biodegradable polymers – be employed in our manufacturing processes and in the formulation of all consumer products. This should emphatically be the case with home- and school-based products, childhood playthings and various foodstuffs that new born babies, infants and young children most come into contact with. This ought to be carried out along the lines that the USEPA is currently engaged in, by its recent launch of the Phthalate Action Plan and Green Chemistry initiatives.<sup>25 26</sup>

It is no exaggeration to state that the future of our children and our society as a whole is at stake unless we give these urgent considerations our highest priority.

**TABLE A**  
**Chemical Substances in Widespread Consumer Use** – selected from the European Chemical Agency’s (ECHA) first list of 90 compounds under its Community Rolling Action Plan (“CoRAP”)

(CMR = Carcinogenic/Mutagenic/Reproductive risk; ED = Endocrine Disrupter)

Name of Compound	Potential Health Risk
Methanol	Human health/CMR
4,4'- isopropylidenediphenol (Bisphenol A)	Suspected ED
2-(4-tert-butylbenzyl) propionaldehyde	Human health/CMR
4-methylanisole	Human health/CMR
Toluene	Human health/CMR, systemic toxicity
Hydroquinone	Human health/CMR
2-ethylhexanoic acid	Human health/CMR
Dimethylphosphonate	Human health/CMR
Hexylsalicylate	Human health/suspected CMR
Formaldehyde	Human health/CMR (worker exposure)
Furfuryl alcohol	Human health/CMR
Hexamethyldisiloxane	Human health/suspected CMR (personal care)
Tetrahydrofuran	Human health/suspected CMR
1,3,5 –tris(oxianylmethyl)-1,3,5-triazine-2,4,6(1H,3H,5H)-trione	Human health/CMR
Diethyl phthalate	Suspected ED
2,4-di-tert-butylphenol	Suspected ED
Methyl 4-hydroxybenzoate	Suspected ED
2,2',2"-nitrotriethanol	Human health/suspected CMR/suspected sensitizer
p-cresol	Human health/suspected CMR; suspected ED
Titanium dioxide	Human health/CMR/suspected respiratory sensitizer

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## References:

- <sup>1</sup> National Academy of Science/National Research Council, *Pesticides in the Diets of Infants and Children*, NAS Press, Washington, DC, 1993.  
[http://www.nap.edu/openbook.php?record\\_id=2126&page=13](http://www.nap.edu/openbook.php?record_id=2126&page=13)
- <sup>2</sup> B. P. Lanphear, et al., “Low-Level Environmental Lead Exposure and Children’s Intellectual Function: An International Pooled Analysis”, *Environmental Health Perspectives*, **113**:894 – 899 (2005).  
<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1257652/>
- <sup>3</sup> US Environmental Protection Agency, *America’s Children and the Environment, 3<sup>rd</sup> Edition (ACE3)*, USEPA, Washington, DC, January 25, 2013.  
<http://www.epa.gov/ace/>
- <sup>4</sup> US Center for Disease Control and Prevention (CDC), *National Diabetes Fact Sheet, 2011*, CDC, Atlanta, GA, 2011.  
[http://www.cdc.gov/diabetes/pubs/pdf/ndfs\\_2011.pdf](http://www.cdc.gov/diabetes/pubs/pdf/ndfs_2011.pdf)
- <sup>5</sup> *Ibid*, USEPA (Ref 3). Original data obtained from the National Cancer Institute’s *Surveillance, Epidemiology and End Results (SEER)* Program.  
[http://seer.cancer.gov/csr/1975\\_2009\\_pops09/browse\\_csr.php?section=28&page=sect\\_28\\_table.02.html](http://seer.cancer.gov/csr/1975_2009_pops09/browse_csr.php?section=28&page=sect_28_table.02.html)
- <sup>6</sup> European Union (EU), *REACH Program*, (EC 1907/2006), European Commission - Environment, Brussels, Belgium.  
[http://ec.europa.eu/environment/chemicals/reach/reach\\_intro.htm](http://ec.europa.eu/environment/chemicals/reach/reach_intro.htm)
- <sup>7</sup> European Chemicals Agency (ECHA), *REACH Hazard Assessment Program*, Helsinki, Finland  
<http://echa.europa.eu/regulations>
- <sup>8</sup> European Chemicals Program (ECHA), *Candidate List of Substances of Very High Concern for Authorization*, Helsinki, Finland  
<http://echa.europa.eu/candidate-list-table>
- <sup>9</sup> European Chemicals Program (ECHA), *Substances of Very High Concern Identification*, Helsinki, Finland  
<http://echa.europa.eu/web/guest/addressing-chemicals-of-concern/authorisation/substances-of-very-high-concern-identification>
- <sup>10</sup> European Chemicals Program (ECHA), *Community Rolling Action Plan (CORAP) – First List*, Helsinki, Finland.  
[http://echa.europa.eu/documents/10162/13628/corap\\_2012\\_en.pdf](http://echa.europa.eu/documents/10162/13628/corap_2012_en.pdf)
- <sup>11</sup> J. L. Lyche, et al., “Reproductive and developmental toxicity of phthalates”, *Journal of Toxicology and Environmental Health, B, Critical Review*, **12**(4):225 – 249 (2009).  
<http://www.ncbi.nlm.nih.gov/pubmed/20183522>
- <sup>12</sup> Y. Kim, et al., “Prenatal exposure to phthalates and infant development at 6 months: prospective Mothers and Children Environmental Health (MOCEH) study”, *Environmental Health Perspectives*, **119**(10):1495 – 1500 (2011).  
<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3230435/>
- <sup>13</sup> J. J. Adibi, et al., “Prenatal exposure to phthalates among women in New York City and Krakow, Poland”, *Environmental Health Perspectives*, **111**:1719 – 1722 (2003).  
<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241713/pdf/ehp0111-001719.pdf>

- 
- <sup>14</sup> S. Sathyanarayan, et al., “Baby care products: possible source of infant phthalate exposure”, *Pediatrics*, **121**(2);e260 – e268 (2008).  
<http://pediatrics.aappublications.org/content/121/2/e260.full>
- <sup>15</sup> T. Schettler, “Human exposures to phthalates via consumer products”, *International Journal of Andrology*, **29**(1):134 – 139 (2006).  
<http://www.ncbi.nlm.nih.gov/pubmed/16466533>
- <sup>16</sup> M. J. Edwards, et al., “Case-Control study of cleft lip or palate after maternal use of topical corticosteroids during pregnancy”, *American Journal of Genetics*, **120A**:459 – 463 (2003).  
<http://onlinelibrary.wiley.com/doi/10.1002/ajmg.a.20130/abstract?deniedAccessCustomisedMessage=&userIsAuthenticated=false>
- <sup>17</sup> National Institute of Neurological Disorders and Stroke (NINDS), *Anencephaly Information Page (website)*, NIH, Bethesda, MD.  
<http://www.ninds.nih.gov/disorders/anencephaly/anencephaly.htm>
- <sup>18</sup> X. O. Shu, et al., “Maternal smoking, alcohol drinking, caffeine consumption, and fetal growth: results of a prospective study”, *Epidemiology*, **6**(2):115 – 120 (1995).  
<http://www.jstor.org/discover/10.2307/3702310?uid=3739576&uid=2&uid=4&uid=3739256&sid=21101894299437>
- <sup>19</sup> A. Thapar, et al., “Maternal smoking during pregnancy and attention deficit hyperactivity disorder symptoms in offsprings”, *The American Journal of Psychiatry*, **160**(11):1985 – 1989 (2003).  
<http://ajp.psychiatryonline.org/article.aspx?articleID=176495>
- <sup>20</sup> P. Suren, et al., “Association between maternal use of folic acid supplements and risk of autism spectrum disorders in children”, *The Journal of American Medical Association (JAMA)*, **309**(6):570 – 577 (2013).  
<http://jama.jamanetwork.com/article.aspx?articleid=1570279>
- <sup>21</sup> A. Milunsky, et al., “Multivitamin/folic acid supplements in early pregnancy reduces the prevalence of neural tube defects”, *The Journal of American Medical Association (JAMA)*, **264**(20):2842 – 2852 (1989).  
<http://jama.jamanetwork.com/article.aspx?articleid=379576>
- <sup>22</sup> M. M. Black, “Effects of Vitamin B12 and folate deficiency on brain development of children”, *Food and Nutrition Bulletin*, **29**(2 Suppl):S126 – 131 (2008).  
<http://www.ncbi.nlm.nih.gov/pmc/articles/pmc3137939/>
- <sup>23</sup> J. L. Dow and T. Green, “Trichloroethylene induced vitamin B(12) and folate deficiency leads to increased formic acid excretion in the rat”, *Toxicology*, **146**(2 – 3):123 – 136 (2000).  
<http://www.ncbi.nlm.nih.gov/pubmed/10814845?dopt=Abstract>
- <sup>24</sup> B. F. Hwang, et al., “Water disinfection by-products and the risk of specific birth defects: a population based cross-sectional study in Taiwan”, *Environmental Health*, **7**:23 (2008).  
<http://www.ehjournal.net/content/7/1/23>
- <sup>25</sup> US Environmental Protection Agency, *Phthalate Action Plan Summary*, USEPA, Washington, DC.  
<http://www.epa.gov/opptintr/existingchemicals/pubs/actionplans/phthalates.html>
- <sup>26</sup> US Environmental Protection Agency, *Green Chemistry Program*, USEPA, Washington, DC.  
<http://www.epa.gov/greenchemistry/>