Executive Summary - Body Burden — The Pollution in Newborns

A benchmark investigation of industrial chemicals, pollutants and pesticides in umbilical cord blood
Environmental Working Group, July 14, 2005

Summary. In the month leading up to a baby's birth, the umbilical cord pulses with the equivalent of at least 300 quarts of blood each day, pumped back and forth from the nutrient- and oxygen-rich placenta to the rapidly growing child cradled in a sac of amniotic fluid. This cord is a lifeline between mother and baby, bearing nutrients that sustain life and propel growth.

Not long ago scientists thought that the placenta shielded cord blood — and the developing baby — from most chemicals and pollutants in the environment. But now we know that at this critical time when organs, vessels, membranes and systems are knit together from single cells to finished form in a span of weeks, the umbilical cord carries not only the building blocks of life, but also a steady stream of industrial chemicals, pollutants and pesticides that cross the placenta as readily as residues from cigarettes and alcohol. This is the human "body burden" — the pollution in people that permeates everyone in the world, including babies in the womb.

In a study spearheaded by the Environmental Working Group (EWG) in collaboration with Commonweal, researchers at two major laboratories found an average of 200 industrial chemicals and pollutants in umbilical cord blood from 10 babies born in August and September of 2004 in U.S. hospitals. Tests revealed a total of 287 chemicals in the group. The umbilical cord blood of these 10 children, collected by Red Cross after the cord was cut, harbored pesticides, consumer product ingredients, and wastes from burning coal, gasoline, and garbage.

This study represents the first reported cord blood tests for 261 of the targeted chemicals and the first reported detections in cord blood for 209 compounds. Among them are eight perfluorochemicals used as stain and oil repellants in fast food packaging, clothes and textiles — including the Teflon chemical PFOA, recently characterized as a likely human carcinogen by the EPA's Science Advisory Board — dozens of widely used brominated flame retardants and their toxic by-products; and numerous pesticides.

Of the 287 chemicals we detected in umbilical cord blood, we know that 180 cause cancer in humans or Chyanimals, 217 are toxic to the brain and nervous system, and 208 cause birth defects or abnormal development in animal tests. The dangers of pre- or post-natal exposure to this complex mixture of carcinogens, developmental toxins and neurotoxins have never been studied.

Chemicals and pollutants detected in human umbilical cord blood

Mercury (Hg) - tested for 1, found 1
Pollutant from coal-fired power plants, mercury-containing products, and certain industrial processes. Accumulates in seafood. Harms brain development and function.

Polyaromatic hydrocarbons (PAHs) - tested for 18, found 9
Pollutants from burning gasoline and garbage. Linked to cancer. Accumulates in food chain.

Polybrominated dibenzodioxins and furans (PBDD/F) - tested for 12, found 7
Contaminants in brominated flame retardants. Pollutants and byproducts from plastic production and incineration. Accumulate in food chain. Toxic to developing endocrine (hormone) system

Perfluorinated chemicals (PFCs) - tested for 12, found 9
Active ingredients or breakdown products of Teflon, Scotchgard, fabric and carpet protectors, food wrap coatings. Global contaminants. Accumulate in the environment and the food chain. Linked to cancer, birth defects, and more.

Polychlorinated dibenzodioxins and furans (PCDD/F) - tested for 17, found 11
Pollutants, by-products of PVC production, industrial bleaching, and incineration. Cause cancer

in humans. Persist for decades in the environment. Very toxic to developing endocrine (hormone) system.

Organochlorine pesticides (OCs) - tested for 28, found 21
DDT, chlordane and other pesticides. Largely banned in the U.S. Persist for decades in the environment. Accumulate up the food chain, to man. Cause cancer and numerous reproductive effects.

Polybrominated diphenyl ethers (PBDEs) - tested for 46, found 32
Flame retardant in furniture foam, computers, and televisions. Accumulates in the food chain and human tissues. Adversely affects brain development and the thyroid.

Polychlorinated Naphthalenes (PCNs) - tested for 70, found 50
Wood preservatives, varnishes, machine lubricating oils, waste incineration. Common PCB contaminant. Contaminate the food chain. Cause liver and kidney damage.

Polychlorinated biphenyls (PCBs) - tested for 209, found 147
Industrial insulators and lubricants. Banned in the U.S. in 1976. Persist for decades in the environment. Accumulate up the food chain, to man. Cause cancer and nervous system problems.

Source: Chemical analyses of 10 umbilical cord blood samples were conducted by AXYS Analytical Services (Sydney, BC) and Flett Research Ltd. (Winnipeg, MB).

Chemical exposures in the womb or during infancy can be dramatically more harmful than exposures later in life. Substantial scientific evidence demonstrates that children face amplified risks from their body burden of pollution; the findings are particularly strong for many of the chemicals found in this study, including mercury, PCBs and dioxins. Children's vulnerability derives from both rapid development and incomplete defense systems:

- A developing child's chemical exposures are greater pound-for-pound than those of adults.
- An immature, porous blood-brain barrier allows greater chemical exposures to the developing brain.
- Children have lower levels of some chemical-binding proteins, allowing more of a chemical to reach "target organs."
- A baby's organs and systems are rapidly developing, and thus are often more vulnerable to damage from chemical exposure.
- Systems that detoxify and excrete industrial chemicals are not fully developed.
- The longer future life span of a child compared to an adult allows more time for adverse effects to arise.

The 10 children in this study were chosen randomly, from among 2004's summer season of live births from mothers in Red Cross' volunteer, national cord blood collection program. They were not chosen because their parents work in the chemical industry or because they were known to bear problems from chemical exposures in the womb. Nevertheless, each baby was born polluted with a broad array of contaminants.

U.S. industries manufacture and import approximately 75,000 chemicals, 3,000 of them at over a million pounds per year. Health officials do not know how many of these chemicals pollute fetal blood and what the health consequences of *in utero* exposures may be.

Had we tested for a broader array of chemicals, we would almost certainly have detected far more than 287. But testing umbilical cord blood for industrial chemicals is technically challenging. Chemical manufacturers are not required to divulge to the public or government health officials methods to detect their chemicals in humans. Few labs are equipped with the machines and expertise to run the tests or the funding to develop the methods. Laboratories have yet to develop methods to test human tissues for the vast majority of chemicals on the market, and the few tests that labs are able to conduct are expensive. Laboratory costs for the cord blood analyses reported here were $10,000 per sample.

A developing baby depends on adults for protection, nutrition, and, ultimately, survival. As a society we have a responsibility to ensure that babies do not enter this world pre-polluted, with 200 industrial chemicals in their blood. Decades-old bans on a handful of chemicals like PCBs, lead gas additives, DDT and other pesticides have led to significant declines in people's blood levels of these pollutants. But good news like this is hard to find for other chemicals.

The Toxic Substances Control Act, the 1976 federal law meant to ensure the safety of commercial chemicals, essentially deemed 63,000 existing chemicals "safe as used" the day the law was passed, through mandated, *en masse* approval for use with no safety scrutiny. It forces the government to approve new chemicals within 90 days of a company's application at an average pace of seven per day. It has not been improved for nearly 30 years — longer than any other major environmental or public health statute — and does nothing to reduce or ensure the safety of exposure to pollution in the womb.

Because the Toxic Substances Control Act fails to mandate safety studies, the government has initiated a number of voluntary programs to gather more information about chemicals, most notably the high production volume (HPV) chemical screening program. But these efforts have been largely ineffective at reducing human exposures to chemicals. They are no substitute for a clear statutory requirement to protect children from the toxic effects of chemical exposure.

In light of the findings in this study and a substantial body of supporting science on the toxicity of early life exposures to industrial chemicals, we strongly urge that federal laws and policies be reformed to ensure that children are protected from chemicals, and that to the maximum extent possible, exposures to industrial chemicals before birth be eliminated. The sooner society takes action, the sooner we can reduce or end pollution in the womb.

Tests show 287 industrial chemicals in 10 newborn babies

Pollutants include consumer product ingredients, banned industrial chemicals and pesticides, and waste byproducts

Sources and uses of chemicals in newborn blood	Chemical family name	Total number of chemicals found in 10 newborns (range in individual babies)
Common consumer product chemicals (and their breakdown products)		**47 chemicals (23 - 38)**
Pesticides, actively used in U.S.	Organochlorine pesticides (OCs)	7 chemicals (2 - 6)
Stain and grease resistant coatings for food wrap, carpet, furniture (Teflon, Scotchgard, Stainmaster...)	Perfluorochemicals (PFCs)	8 chemicals (4 - 8)
Fire retardants in TVs, computers, furniture	Polybrominated diphenyl ethers (PBDEs)	32 chemicals (13 - 29)
Chemicals banned or severely restricted in the U.S. (and their breakdown products)		**212 chemicals (111 - 185)**
Pesticides, phased out of use in U.S.	Organochlorine pesticides (OCs)	14 chemicals (7 - 14)

Stain and grease resistant coatings for food wrap, carpet, furniture (pre-2000 Scotchgard)	Perfluorochemicals (PFCs)	1 chemicals (1 - 1)
Electrical insulators	Polychlorinated biphenyls (PCBs)	147 chemicals (65 - 134)
Broad use industrial chemicals - flame retardants, pesticides, electrical insultators	Polychlorinated naphthalenes (PCNs)	50 chemicals (22 - 40)
Waste byproducts		**28 chemicals (6 - 21)**
Garbage incineration and plastic production wastes	Polychlorinated and Polybrominated dibenzo dioxins and furans (PCDD/F and PBDD/F)	18 chemicals (5 - 13)
Car emissions and other fossil fuel combustion	Polynuclear aromatic hydrocarbons (PAHs)	10 chemicals (1 - 10)
Power plants (coal burning)	Methylmercury	1 chemicals (1 - 1)
All chemicals found		**287 chemicals (154 - 231)**

Source: Environmental Working Group analysis of tests of 10 umbilical cord blood samples conducted by AXYS Analytical Services (Sydney, BC) and Flett Research Ltd. (Winnipeg, MB).

Part 2
Babies are vulnerable to chemical harm

Parents know intuitively that babies in the womb are more vulnerable to the effects of industrial chemicals than adults. A pregnant woman may avoid using hair dye and nail polish, pumping gas, or painting the nursery, for example, to protect her baby. This intuition is backed by science that has unfolded primarily over the past two decades. In 1993 the National Academy of Sciences enumerated, in a Congressionally mandated study, the primary factors that contribute to children's unique vulnerability to the harmful effects of chemicals (NAS 1993):

- A developing child's chemical exposures are greater pound-for-pound than those of adults.
- An immature, porous blood-brain barrier allows greater chemical exposures to the developing brain.
- Children have lower levels of some chemical-binding proteins, allowing more of a chemical to reach "target organs."
- A baby's organs and systems are rapidly developing, and thus are often more vulnerable to damage from chemical exposure.
- Systems that detoxify and excrete industrial chemicals are not fully developed.
- The longer future life span of a child compared to an adult allows more time for adverse effects to arise.

The pace and complexity of growth and development in the womb are unmatched later in life. Three weeks after conception, an embryo, still only 1/100th the size of a water droplet, has nevertheless grown at such

an explosive rate that were it not to slow down, it would be born literally the size of a million Earths. Over the next five weeks the baby constructs the beginnings of elbows, knees, eyelids, nipples, hair follicles on chin and upper lip, external genitals, primitive internal organs, a four-chambered heart, working fingers and toes, and even a footprint (Greene 2004). At no other time in life does a person create so much from so little in so short a time. Industrial chemicals that interrupt this intricate process can, at high levels, wreak havoc in the form of severe birth defects, or at lower levels cause subtle but important changes in development that surface later in childhood as learning or behavioral problems, or in adulthood in the form of certain cancers or perhaps neurodegenerative disease.

A recent review by government scientists of the "critical windows" of vulnerability reveals an urgent need for public health policies that recognize childhood sensitivity (Selevan et al. 2000). Many of these windows of vulnerability are found in the early months of human pregnancies, when cells are multiplying and differentiating into specific tissues and organs. Exposures during these times can lead to permanent damage. But a child's vulnerability continues long beyond early pregnancy: the central nervous system, immune, reproductive and endocrine systems, for example, continue to mature even after birth (NAS 1993, Makri et al. 2004). As a whole, these windows facilitate more pronounced risks and effects for chemical exposures in childhood than adulthood. For example, a mother's exposure to dioxins, mercury, or certain pesticides during pregnancy could measurably harm her baby, while affecting her own health perhaps not at all.

In a decades-long mercury poisoning disaster in Minamata, Japan that began in the 1950s, some babies born to women who ate mercury-polluted seafood died within days of birth, while their mothers were free of symptoms. Autopsies revealed that in adults, mercury induced lesions that were concentrated in a few areas of the brain. In the fetus, however, mercury spawned lesions over nearly the entire brain cortex.

In the decades following Minamata, scientists have developed a much fuller understanding of children's vulnerability to chemicals, discovering links between a host of health problems — including asthma, childhood cancer, and brain damage — and such common contaminants as solvents, pesticides, PCBs, and lead (Trasande and Landrigan, 2004). A recent National Academy of Sciences study suggests that environmental factors contribute to at least 28 percent of childhood developmental disabilities (NAS 2000a).

The latest research investigates not only relationships between disease and exposures, but the root causes of chemically-induced disease with *in utero* origins. This research pinpoints traits of a fetus that contribute to vulnerability: low levels of some chemical-binding proteins in the blood, immature excretion pathways, and an immature blood brain barrier, for instance, which combine to increase the transfer of chemicals from the blood to the aptly named "target organs" that may ultimately bear the harm.

The risks to a baby derive not only from his or her physical makeup, but also from the very behaviors and events that prepare the baby for life outside the womb. Beginning in the fifth month of pregnancy, babies regularly swallow and breathe, building muscles essential for survival after birth. Through these actions, the lungs and the gut are filled, again and again, with the same amniotic fluid that collects the baby's urine. Pollutants like plasticizers and pesticides excreted in urine accumulate in this fluid and are cycled right back into the baby's body through the mouth and nose. And in the third trimester the mother's body dissolves stored, maternal fat, shunting it to the baby through the blood, but with this fat the child also receives the persistent pollutants clinging to it, like PCB's, flame retardants, and dioxins. Faced with such diverse exposures and armed with a body ill-equipped to rid itself of chemicals, it is small wonder that a developing baby so often proves vulnerable to chemical exposures (Makri et al. 2004).

Some studies are beginning to measure the sensitivity of a child relative to an adult for suffering impacts from chemical exposures. For instance, studies of mutagens called polyaromatic hydrocarbons (PAHs) — target chemicals examined in this study and waste products from burning gasoline and garbage — found that even though levels of PAHs are thought to be lower in the fetus than the mother (Srivastava et al. 1986), the fetus bears more cancer-inducing DNA damage from the exposures (Whyatt et al. 2001).

But health and environmental officials have been slow to act on the wealth of studies on childhood vulnerability produced in the past 20 years. After nearly a decade of review, the Environmental Protection Agency updated its cancer risk guidelines in 2003 to explicitly acknowledge the importance of childhood exposures. The agency concluded, after a review of 23 studies of early life exposures to cancer-causing chemicals, that carcinogens average 10 times the potency for babies than adults, and that some chemicals are up to 65 times more powerful (EPA 2005a).

EPA's new policy, though, targets only cancer. It leaves EPA with no formal policy regarding childrens' vulnerability to chemicals that damage the immune system, the brain, or the hormone system, kidney, liver, lungs, thyroid or a host of other potential targets, even though plenty of evidence says that children face higher risks for harm.

Part 3
Human health problems on the rise

Over the past 50 years, as infectious childhood diseases like polio, smallpox, rheumatic fever, and diphtheria have largely been controlled, chronic conditions of less obvious origins have taken their place. Asthma, autism, attention deficit and hyperactivity disorders (ADD and ADHD), childhood brain cancer and acute lymphocytic leukemia have all increased over the past 30 years. Five to ten percent of American couples are infertile. Up to half of all pregnancies end in miscarriage. Three to five percent of babies are born with birth defects (CDC 2004, Jahnke et al. 2005, Trasande and Landrigan 2004). Scientists cannot fully explain these increases, but early life exposure to environmental pollutants is a leading suspect.

AUTISM	10X	increase early 80's-1996
MALE BIRTH DEFECTS	2X	increase hypospadias, 1970-1993
CHILDHOOD ASTHMA	2X	increase 1982-1993
ACUTE LYMPHOCYTIC LEUKEMIA	62%	increase in children, 1973-1999
CHILDHOOD BRAIN CANCER	40%	increase 1973-1994
PRETERM BIRTH	23%	increase mid 80's-2002
INFERTILITY	5-10%	of couples
BIRTH DEFECTS	3-5%	of all babies
SPERM COUNTS	1%	decrease yearly 1934-1996

Sources: Yeargin-Allsopp et al. 2003, CDC 1995, Robison et al. 1995, Schecter 1999, Ananth et al. 2001, Branum and Schoendorf 2002, Swan et al. 1998, Paulozzi et al. 1997, Dunson et al. 2004, Trasande and Landrigan 2004, Jahnke et al. 2005

Fetal exposures lead to adult disease. Some chemicals are directly toxic to an exposed child — lead and mercury, for example, which harm a developing brain — while other chemicals induce a chain of events that may culminate in a diagnosed health problem later in life. Hormone-mimicing chemicals like dioxins and furans, for example, could induce delayed cancers in hormone-sensitive tissues like the breast, testicle, or prostate gland. Chemicals like PCBs or DDT can reduce growth rates in the womb, initiating in low birthweight babies lasting, internal survival mechanisms that cascade into cardiovascular disease or diabetes later in life.

The fact is, a child can bear a lifelong imprint of risks from the countless molecules of industrial pollutants that find their way through the placenta, down the umbilical cord, and into the baby's body. The consequences — health disorders, subtle or serious — can surface not only in childhood but also in adulthood. Studies now support origins in early life exposures for a startling array of adult diseases, including Alzheimers, mental disorders, heart disease, and diabetes.

Laboratory studies show increased deposits of the Alzheimer-related protein amyloid in the brains of older animals exposed to lead as newborns, but not in animals that were exposed to an equal amount of lead as adults (Basha et al. 2005). And over the past two decades numerous studies have linked low birth weight with adult onset of coronary heart disease, diabetes, stroke, hypertension, depression and other conditions (Barker 1995, Wahlbeck et al. 2001, Thompson et al. 2001, Hales et al. 1991). Low birth weight can arise not only from poor maternal nutrition but also from a host of industrial pollutants, including arsenic, mercury, lead, organic solvents, PCBs, and pesticides, including DDT.

Recent studies shed new light on how early life chemical exposures set adult disease in motion. In laboratory studies scientists from the University of Texas found that fetal exposures to the synthetic hormone (and now-banned drug) DES permanently "reprogrammed" body tissues, dramatically raising rates of uterine cancer, in this case, in later life (Cook et al. 2005). With an estimated 75,000 chemicals registered for use in the U.S., and an average of seven new chemicals approved each day, many not

tested for safety and certaintly not tested for their ability to "reprogram" body tissues, the ramifications of this study are enormous.

Fetal exposures cause disease in future generations. Remarkably, it appears that early life exposures can lead to health problems not only in adulthood, but also down through subsequent generations. For instance, adult diseases linked to newborns' low birth weight, enumerated above, cause adverse effects not only in those babies born small, but also in their children of any birth size, through heritable changes in gene expression that result in a phenomenon known as "epigenetic inheritance." Very different from genetic mutations, which are physical changes in gene structure, epigenetic inheritance is instead characterized by certain genes being turned on or off, but near permanently in ways that can be inherited.

If a genetic mutation is like changing a light fixture, the comparable epigenitic change would involve taping the light switch on or off. Since genes are responsible for making the chemicals that build and repair the body, this unnatural forcing to a permanent on or off position can have far-reaching consequences. In humans, both kinds of genetic changes, mutations as well as epigenetic changes in gene expression, can be passed down to a baby in the womb.

Scientists have recently found heritable epigenetic changes linked to the fungicide vinclozolin and pesticide methoxychlor, which impaired sperm counts and sperm motility not only among animals exposed in utero, but also in three subsequent generations (Anway et al. 2005). In other words, what each of us was exposed to in our mother's womb might affect the health of our great-grandchildren.

Notably, both of these pesticides were recently banned under a federal law that requires pesticides to be safe for newborns and children. The government gives children no explicit protection under the federal law meant to ensure the safety of other commercial chemicals (the Toxic Substances Control Act), even though risks from childhood exposures to industrial chemicals are no lower than those from pesticides.

Cord blood pollutants in this study, linked to health problems. Scientific studies implicate some of the chemicals we detected in cord blood with serious, ongoing human health problems:

- **Dioxin** exposures during fetal development have been implicated in endocrine-related cancers in women (breast and uterine, for example) by altering hormone levels, increasing the sensitivity of children and adolescents to other carcinogens (Birnbaum and Fenton 2003). In men, tiny levels of dioxin in the range of 0.02 to 10 parts per billion alter testosterone levels and are linked with diabetes (EPA 2004a). Dioxin at 80 parts per trillion in paternal — but not maternal — serum causes a significant change in the sex ratio of children (Mocarelli, et al. 1996, Mocarelli, et al. 2000). At this tiny dose, men father nearly twice as many girls as boys. As body burdens increase within and above these ranges, the likelihood, severity, and potential spectrum of non-cancer effects increases (EPA 2004a). Fetal dioxin exposure can harm the immune system, thyroid, and brain (Van Loveren et al. 2003, Faroon et al. 2001, ten Tusscher and Koppe 2004). Dioxin from garbage incinerators is associated with increased incidence of infant death and birth defects (Tango et al. 2004).

- **Methylmercury** exposure in the womb causes measurable declines in brain function in children exposed to levels corresponding to 58 parts per billion in maternal blood (NAS 2000b). Researchers in the Netherlands found a doubling in the risk of heart attacks and death from coronary heart disease at methylmercury hair levels of 2 mg/kg, which corresponds to about one fifth the assumed safe maternal blood level (Salonen, et al. 1995). Increased diastolic and systolic blood pressure and decreased heart rate variability in developmentally exposed children have also been observed at doses below what the EPA considers a safe maternal blood level (NAS 2000b, Sorensen et al. 1999).

- **PCBs** at 9.7 ppb in maternal serum during fetal development can impair brain development, with resultant attention and IQ deficits that appear to be permanent (Jacobson and Jacobson 1996). Notably, IQ deficits are linked to the mother's PCB levels, not the PCB levels in children at 4 and 11 years of age (by which time the children's PCB levels had decreased substantially compared to levels at birth), underscoring the limitations of studies that look for correlations between current body burdens and health effects in the absence of data on *in utero* exposures. Levels of PCBs in the general population are also associated with abnormal menstrual cycles (Cooper et al. 2005).

- **DDE** above 15 ppb in maternal blood is associated with preterm birth and low birth weight, with weight corrected for gestational age (Longnecker et al. 2001). DDE is a metabolite of the banned, persistent pesticide DDT. Using the associations derived from tests of archived blood samples from a pool of 42,000 women, researchers estimated that DDT exposures in the U.S. population could have accounted for up to 15 percent of infant deaths during the 1960s. Low birth weight is recognized as a risk factor for type II diabetes, high blood pressure, and cardiovascular disease later in life (Prentice and Moore 2005, Godfrey and Barker 2001, Hales and Barker 2001). Even if these lower birth weight babies "catch up" later, the damage may have already been done. A substantial number of studies have found that low birth weight followed by an accelerated growth rate during childhood is a significant risk factor for high blood pressure, stroke, insulin resistance and glucose intolerance (Eriksson, et al. 2000a, Eriksson, et al. 2002, Eriksson et al. 2000b, Eriksson et al. 1999, Eriksson and Forsen 2002, Forsen et al. 2000, Ong and Dunger 2002, Stettler et al. 2002).

Some facts about human health trends

Cancer. Cancer incidence has steadily increased over the decades for many forms of the disease, including breast, prostate, and testicular (NCI 2005). The incidence of childhood cancer increased by 27.1 percent between 1975 and 2002, with the sharpest rise estimated for brain and other nervous system cancers (56.5 percent increase) and acute lymphocytic leukemia (68.7 percent increase). The incidence of testicular cancer also steadily rose 66 percent between 1975 and 2002 (NCI 2005). The probability that a U.S. resident will develop cancer at some point in his or her lifetime is 1 in 2 for men and 1 in 3 for women (ACS 2004). A broad array of environmental factors plays a pivotal role in the initiation and promotion of cancer. Just 5 to 10 percent of all cancers are directly linked to inherited, genetic factors (ACS 2001).

- **Breast cancer.** Among girls born today, one in seven is expected to get breast cancer and one in 30 is expected to die from it. Invasive female breast cancer increased an average of 1.5 percent per year between 1973 and 1996, for a total increase of 25.3 percent. Among those 65 and younger, breast cancer incidence rose 1.2 percent per year, corresponding to a doubling every two generations (58 years). If trends continue, the granddaughters of today's young women could face a one in four chance of developing breast cancer (NCI 1996, NCI 1997).

- **Testicular cancer.** At its current pace, the incidence of testicular cancer is doubling about every one and a half generations (39 years). In the U.S. the incidence of testicular cancer rose 41.5 percent between 1973 and 1996, an average of 1.8 percent per year (NCI 1996, NCI 1997). Testicular cancer is now the most common cancer in men age 15 to 35 (NCI 2005).

- **Prostate cancer.** Prostate cancer rates rose 4.4 percent a year between 1973 and 1992, or more than a doubling of risk in a generation. Since 1992, the incidence has declined, but it is still 2.5 times its 1973 rate. Part of this increase can be explained by better detection, but increased incidence has also been accompanied by an increase in mortality - which better detection cannot explain. Prostate cancer is now the most common cancer among U.S. men, and the second most lethal, killing an estimated 31,900 men in the year 2000 alone (NCI 1996, NCI 1997).

Major nervous system disorders. Several recent studies have determined that the reported incidence of autism is increasing, and is now almost 10 times higher than in the mid-1980's (Byrd 2002, Chakrabarti and Fombonne 2001). The number of children being diagnosed and treated for attention deficit disorder (ADD) and attention deficit hyperactivity disorder (ADHD) has also increased dramatically in the past decade (Robison et al. 1999, Robison et al. 2002, Zito et al. 2000). The causes are largely unexplained, but environmental factors, including chemical exposures, are considered a likely contributor. Environmental factors have also been increasingly linked with Parkinson's disease (Checkoway and Nelson 1999, Engel et al. 2001).

Preterm births and low birth weights. Preterm births have increased 23 percent over the past 2 decades; low-weight births have become more common (Ananth et al 2001, Branum and Schoendorf 2002). The causes are largely unknown, but environmental factors such as chemical pollutants and nutrition are thought to play a role. Low birth weight has been linked to adult obesity, diabetes, cardiovascular disease, schizophrenia, and other conditions (Barker 1995, Wahlbeck et al. 2001, Thompson et al. 2001, Hales and Ozanne 2003). It has also been linked to lower academic performance, neurosensory impairment, and lower rates of pregnancy in the offspring (Hack et al. 2002).

Defects of the reproductive system. Studies show that sperm counts in certain parts of the world are decreasing (Swan, et al. 2000, Toppari, et al. 1996). Scientists have measured significant regional differences in sperm count that cannot be explained by differences in genetic factors (Swan et al. 2003). Girls may be reaching puberty earlier, based on comparing current appearance of breast development and pubic hair growth with historical data (Herman-Giddens, et al. 1997). Rates of hypospadias, a physical deformity of the penis, have risen in recent years (Paulozzi et al. 1997). The incidence of undescended testicles (cryptorchidism) and testicular cancer also appear to be rising in certain parts of the world (Bergstrom et al. 1996, McKiernan et al. 1999, Toppari et al. 1996, Paulozzi 1999). Several studies have suggested links between developmental exposure to environmental contaminants and cryptorchidism or testicular cancer (Hardell, et al. 2003, Hosie, et al. 2000, Toppari, et al. 1996, Weidner, et al. 1998).

- **Declining sperm count.** An analysis of 101 studies (1934-1996) by Dr. Shanna Swan of the University of Missouri confirms results of previous studies: average sperm counts in industrialized countries appear to be declining at a rate of about one percent each year (Swan et al. 2000).

- **Hypospadias.** Incidence of hypospadias, a birth defect of the penis, doubled in the United States between 1970 and 1993, and is estimated to affect one of every 125 male babies born (Paulozzi et al. 1997). Data from the Centers for Disease Control and Prevention show that rates in the U.S. began climbing in about 1970, and continued this increase through the 1980s. This condition is a physical deformity of the penis in which the opening of the urethra occurs on the bottom of the penis instead of the tip.

- **Undescended testicles.** This birth defect, where testicles fail to completely descend into the scrotum during pregnancy, occurs in two to five percent of full-term boys in Western countries. Rates of the defect increased greatly in the U.S. in the 1970s and 1980s. Men born with this defect are at higher risk for testicular cancer and breast cancer (Paulozzi 1999).

Together with 287 industrial pollutants in 10 newborn babies, this body of science and the litany of serious, continuing human health concerns reveals the critical need for reform of our system of public health protections, which fails to require proof that chemicals are safe for children.

Part 4
Recommendations

U.S. industries manufacture and import approximately 75,000 chemicals, 3,000 of them at over a million pounds per year. Studies show that hundreds of industrial chemicals circulate in the blood of a baby in the womb, interacting in ways that are not fully understood. Many more pollutants are likely present in the womb, but test methods have yet to be developed that would allow health officials to comprehensively assess prenatal exposure to chemicals, or to ensure that these exposures are safe. From a regulatory perspective, fetal exposure to industrial chemicals is quite literally out of control.

The reason: the Toxic Substances Control Act (TSCA), the nation's notoriously weak chemical safety law. TSCA deprives the EPA of the most basic regulatory tools. The vast majority of chemicals in use today do not have anywhere near sufficient data needed to assess their safety, particularly their safety for the unborn baby or young child. Under TSCA, however, the EPA cannot require this data as a condition of continued chemical use. Instead, the EPA must negotiate with industry or complete a formal "test rule" for every study that it needs, for every chemical on the market. Consequently, very few high quality toxicity tests are conducted.

When industry submits results of voluntary testing to the agency, huge portions, including key health and safety findings, are routinely redacted as confidential business information, meaning that even state regulatory agencies are not allowed to review them. If risks are identified and action is contemplated, minimizing "unreasonable" costs to industry is the TSCA mandate, no matter how serious the risks and no matter the population that bears them — even unborn babies. And if there is any scientific uncertainty, as there often is, TSCA prohibits precautionary action and requires certainty of harm before actions can be taken to protect the public health. TSCA has not been improved for nearly 30 years — longer than any other major environmental or public health statute.

This study and a strong body of supporting science suggest that fetal exposure to industrial chemicals is contributing to adverse health effects in the human population. This is cause for concern.

But experience also shows us that it is never too late to take action. Blood levels of PCBs and pesticides like DDT are lower today than 30 years ago when they were banned. Since these watershed actions in the 1970s, however, few industrial chemicals have been regulated to any significant degree. The various reasons for this stagnation — the need for data on chemical toxicity and exposure, lack of ambition at the EPA, and chemical industry intransigence — all come back to one central cause: the absence of a strong federal chemical safety law that provides the EPA with unambiguous statutory authority to take the actions needed to ensure that chemicals are safe.

Because TSCA does not mandate safety studies and makes it difficult for EPA to demand them, a number of voluntary initiatives to gather more information about chemicals have been attempted, most notably the high production volume (HPV) chemical screening program. These efforts, however, have been largely ineffective at reducing exposure and are no substitute for a clear statutory requirement to protect children from the toxic effects of chemical exposure.

Federal law must be reformed to ensure that children are protected from chemical exposures, and that to the maximum extent possible exposure to industrial chemicals before birth be eliminated entirely. The nation's pesticide law was amended nearly a decade ago to require explicit protection of infants and children from pesticides. Actions taken under the 1996 Food Quality Protection Act (FQPA) have reduced or eliminated children's exposures to a number of highly hazardous pesticides, with no discernable adverse impact on the availability or price of a wholesome food supply, and without adverse impact on the agricultural or pesticide industry. We recommend a similar standard be applied to commercial chemicals.

This would mean transforming TSCA into a true public health and environmental law, with the following core provisions. A new TSCA would:

- Require chemical manufacturers to demonstrate affirmatively that the chemicals they sell are safe for the entire population exposed, including children in the womb. In the absence of information on the risks of pre-natal exposure, chemicals must be assumed to present greater risk to the developing baby *in utero*, and extra protections must be required at least as strict as the 10 fold children's safety factor in FQPA.

- Require that the safety of closely related chemicals, such as the perfluorochemicals used to make Teflon and other stain-resistant and water repellant products, be assessed as a group. The presumption would be that these chemicals have additive toxicity unless manufacturers clearly prove otherwise.

- Grant the EPA clear and unencumbered authority to demand all studies needed to make a finding of safety and to enforce clear deadlines for study completion.

- Remove from the market chemicals for which tests demonstrating safety are not conducted.

- Eliminate confidential business protection for all health, safety, and environmental information.

- Require that material safety data sheets provided to workers contain the results of studies conducted under these provisions.

- Provide strong incentives for green, safer chemicals in consumer products and industrial processes.

Test results from 10 newborn babies find 287 chemicals from a diverse range of chemical families and subclasses - table coming soon

Chemical class and subclass	Concentrations of chemicals in umbilical cord blood from 10 newborns (average and range among individual umbilical cord blood samples)		Number of newborn umbilical blood samples with detections
Metals [parts per billion wet weight]			
Methyl Mercury	0.947	(0.07 - 2.3)	10 of 10
Polybrominated dioxins and furans [parts per trillion lipid weight]			
Brominated dioxins	5.33	(0 - 53.3)	1 of 10
Brominated furans	50.5	(0 - 246)	7 of 10
Tetrabrominated dioxin	0	(0 - 0)	0 of 10
Pentabrominated dioxin	0	(0 - 0)	0 of 10
Hexabrominated dioxin	5.33	(0 - 53.3)	1 of 10
Tetrabrominated furan	1.65	(0 - 11.1)	2 of 10
Pentabrominated furan	10.7	(0 - 48.5)	6 of 10
Hexabrominated furan	12.6	(0 - 73.3)	3 of 10
Heptabrominated furan	25.6	(0 - 118)	6 of 10
Octabrominated furan	0	(0 - 0)	0 of 10
Polychlorinated dioxins and furans [parts per trillion lipid weight]			
Chlorinated dioxins	53.4	(37 - 79.6)	10 of 10
Chlorinated furans	6.04	(0.758 - 35)	10 of 10
Tetrachlorinated dioxin	0	(0 - 0)	0 of 10
Pentachlorinated dioxin	0.291	(0 - 2.910)	1 of 10
Hexachlorinated dioxin	7.1	(3.79 - 12)	10 of 10
Heptachlorinated dioxin	8.92	(5.3 - 12.6)	10 of 10
Octachlorinated dioxin	37.1	(19.9 - 55)	10 of 10
Tetrachlorinated furan	0	(0 - 0)	0 of 10
Pentachlorinated furan	1.62	(0 - 8.660)	4 of 10
Hexachlorinated furan	2.31	(0.379 - 15.4)	10 of 10
Heptachlorinated furan	2.12	(0.379 - 11)	10 of 10
Octachlorinated furan	0	(0 - 0)	0 of 10

Organochlorine Pesticide (OC) [parts per trillion lipid weight]			
Organochlorine Pesticides (OCs)	18600	(8720 - 35400)	10 of 10
Perfluorochemical (PFCs) [parts per billion wet weight]			
Perfluorochemicals (PFCs)	6.17	(3.37 - 10.6)	10 of 10
Perfluorinated sulfonate	4.25	(2.26 - 7.760)	10 of 10
Perfluorinated carboxylic acid	1.92	(1.1 - 2.870)	10 of 10
Polyaromatic hydrocarbon (PAHs) [parts per trillion lipid weight]			
Polyaromatic hydrocarbon (PAHs)	285	(217 - 384)	5 of 5
Polybrominated diphenyl ether (PBDEs) [parts per trillion lipid weight]			
Polybrominated diphenyl ether (PBDEs)	6420	(1110 - 14200)	10 of 10
Dibrominated diphenyl ether	40.4	(0 - 82.7)	7 of 10
Tribrominated diphenyl ether	160	(75.6 - 303)	10 of 10
Tetrabrominated diphenyl ether	1660	(16.6 - 3950)	10 of 10
Pentabrominated diphenyl ether	574	(0 - 1750)	9 of 10
Hexabrominated diphenyl ether	1310	(272 - 7590)	10 of 10
Heptabrominated diphenyl ether	46.6	(12.2 - 117)	10 of 10
Octabrominated diphenyl ether	74.3	(41.2 - 134)	10 of 10
Nonabrominated diphenyl ether	859	(0 - 3250)	7 of 10
Decabrominated diphenyl ether	1700	(0 - 9630)	3 of 10
Polychlorinated biphenyl (PCBs) [parts per trillion lipid weight]			
Polychlorinated biphenyls (PCBs)	7880	(2990 - 19700)	10 of 10
Mono-PCB	95.3	(44.1 - 210)	10 of 10
Di-PCB	154	(0 - 304)	9 of 10
Tri-PCB	275	(41.3 - 540)	10 of 10
Tetra-PCB	366	(140 - 873)	10 of 10
Penta-PCB	671	(304 - 1300)	10 of 10

	Hexa-PCB	2760	(766 - 6890)	10 of 10
	Hepta-PCB	2400	(435 - 6870)	10 of 10
	Octa-PCB	889	(172 - 2740)	10 of 10
	Nona-PCB	191	(10.2 - 617)	10 of 10
	Deca-PCB	75.4	(6.55 - 211)	10 of 10
Polychlorinated naphthalene (PCNs) [parts per trillion lipid weight]				
Polychlorinated naphthalenes (PCNs)		617	(295 - 964)	10 of 10
	Monochlorinated naphthalene	65.7	(3.2 - 216)	10 of 10
	Dichlorinated naphthalene	27.8	(1.1 - 79.3)	10 of 10
	Trichlorinated naphthalene	164	(104 - 315)	10 of 10
	Tetrachlorinated naphthalene	292	(127 - 409)	10 of 10
	Pentachlorinated naphthalene	30	(2.2 - 64.5)	10 of 10
	Hexachlorinated naphthalene	22.9	(2.2 - 111)	10 of 10
	Heptachlorinated naphthalene	12.4	(0 - 68.4)	3 of 10
	Octachlorinated naphthalene	2.81	(0 - 19.3)	2 of 10

Source: Chemical analyses of 10 umbilical cord blood samples conducted by AXYS Analytical Services (Sydney, BC) and Flett Research Ltd. (Winnipeg, MB).

The chemicals found in 10 newborns are linked to a number of health problems

Health Effect or Body System Affected	Number of chemicals found in 10 newborns tested that are linked to the listed health impact		
	Average number found in 10 newborns	Total found in all 10 newborns	Range (lowest and highest number found in individual newborns)
Cancer [1]	133	180 [2]	92 to 155
Birth Defects / Developmental Delays	151	208 [3]	101 to 176
Vision	1	1 [4]	0 to 1
Hormone System	153	211 [5]	104 to 179
Stomach Or Intestines	194	275 [6]	147 to 227

Kidney	128	174 [7]	84 to 149
Brain, Nervous System	157	217 [8]	108 to 183
Reproductive System	185	263 [9]	136 to 219
Lungs/breathing	144	200 [10]	93 to 170
Skin	159	226 [11]	115 to 187
Liver	40	46 [12]	30 to 45
Cardiovascular System Or Blood	162	226 [13]	117 to 190
Hearing	135	187 [14]	85 to 161
Immune System	130	177 [15]	89 to 151
Male Reproductive System	172	245 [16]	122 to 207
Female Reproductive System	142	196 [17]	92 to 168

* Some chemicals are associated with multiple health impacts, and appear in multiple categories in this table.

References for Health Effects
Footnotes

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