

MIND, DISRUPTED

How toxic chemicals change how we think and who we are



Lead

What Is Lead?

Lead is a naturally occurring heavy metal found in small quantities in the earth's crust. Lead has been recognized for its neurotoxic effects on humans for over 2,000 years.^{1,2} It is ubiquitous in the environment, mainly due to the wide-spread use of lead in manufacturing and as an additive in paint, gasoline, and other products.³

Lead was used as an additive for paint intended for buildings until it was phased out in the United States by the Lead Paint Poison Prevention Act of 1978.⁴ Lead continues to be used in coatings on ceramics and on other imported products.^{5,6} Lead has also been found in consumer items, such as toys and other children's products, jewelry, handbags, animal accessories, and other products.^{7,8,9,10}

The combustion of leaded gasoline contributed significantly to the amount of lead in air, soil and water. It is estimated that 4 million metric tons of lead were dispersed into the atmosphere in the U.S. by the combustion of leaded gasoline from 1923–1986.¹¹ These lead emissions decreased significantly with the introduction of the Clean Air Act in the 1970s and also by the use of catalytic converters on automobiles.¹² Leaded gasoline was banned in the U.S. in 1986, but lead additives continue to be used in racing fuels, boating fuels and fuels for farm equipment.¹¹

Even though lead has been banned in paint and gasoline, it continues to be used in the production of batteries, ammunition, metal products (solder and pipes), and in shields for X-rays.¹ Lead may leach out of some PVC plastic products, such as pipes, where it is used as a metal stabilizer.¹³

How Are We Exposed?

While lead is no longer used in gasoline and most paints, exposure may still occur through ingestion or inhalation of particles containing lead that may be present in dust or chips of lead paints (used in homes built before 1978) or in soils.³ The main processes that introduce lead paint into soil and air are deterioration, paint scraping and power-sanding.¹⁴ These actions release particles that may then be inhaled or ingested from hand to mouth contact with dust.

Even though leaded gasoline was banned in 1986, past emissions from leaded gasoline may still contribute significantly to soil lead levels and human exposure to lead in areas where there has been heavy traffic. In a 1997 study that compared soil lead levels in rural and urban areas with pre-1940s housing, urban areas were found to have higher soil lead levels.¹⁵ Researchers also found a strong association between high soil lead levels and high blood lead levels of urban residents.¹¹

Seasonal variations of blood lead levels have also been found in urban areas with lead contaminated soil. A study of children living in several cities found that increases in blood lead levels were more likely during hot summer months when low soil moisture results in higher dust levels.¹⁶ Researchers concluded that, "Children and adults living in urban areas where surface soils are contaminated with Pb (lead) may become exposed through indoor and outdoor inhalation of Pb dust and ingestion of Pb deposited within homes and outdoor surfaces."

According to the U.S. Centers for Disease Control and Prevention (CDC), the most common non-occupational adult lead exposures occur from shooting firearms, eating contaminated food, home renovation or painting, and gunshot wounds.⁶ Exposure may also occur by ingestion or inhalation of contaminated water or air.³ Water may become contaminated from lead pipes used in some municipalities or from lead contaminated groundwater.³ In 2004, an estimated 18% of residences in Washington, DC had lead pipes.¹⁷ A majority of the residences with lead pipes that were tested had water lead levels that were greater than the Environmental Protection Agency's (EPA) action level of 15 parts per billion (ppb)—the level at which the EPA enforces regulations and takes action in order to reduce lead contamination.¹⁷

Occupational exposure is most likely to occur from manufacturing of storage batteries, mining of lead and zinc ores, painting, and paper hanging.⁶ Children may also be exposed to lead dust that family members working in these industries carry home on their clothes.¹⁸

The most bioavailable form of lead, lead acetate, is found in some eye makeup, lipstick and hair dye.^{3,19} Eye makeup containing kohl, kajal, or surma is especially likely to contain lead.⁵ Lead

acetate dissolves easily and can be absorbed through the skin.³

Even though lead has been banned in food packaging produced in the U.S., it has been found in the cans of imported canned food and on candy wrappers.^{20,21} Containers for food and beverages such as crystal glassware and ceramic items may contain lead.¹¹ Storing acidic items such as vinegar, orange juice and coffee in lead-coated ceramics increases the leaching of lead from these items.¹¹

Lead has been also been found in some herbal supplements manufactured in the U.S., China, and India.^{22,23}

In 2007, several children's products were recalled for lead paint contamination, affecting nearly 14.5 million items.²⁴ A majority of these products had been manufactured in China, although products from Korea, India, Peru, Taiwan, and Vietnam were also recalled. A 2008 study found concentrations of lead above the regulatory limit on seasonal items manufactured in China that are intended for children, including fake teeth, Santa Claus pens, and sippy cups.²⁵ High lead concentrations have been found in inexpensive jewelry marketed to children. In some cases jewelry items were more than 80% lead.²⁶

Lead in Our Bodies

Despite the progress that has been made in decreasing childhood lead exposure, it remains a critical environmental health concern. Data from the 2003-2004 National Health and Nutritional Examination Survey (NHANES) reveals that 2.3% of 1-5 year olds have blood lead levels that are higher than 10 µg/dL, the level at which the CDC recommends intervention.²⁷ Over 50% of 1-5 year olds in the 2003-2004 NHANES had blood lead levels at least 2 µg/dL, which is above levels that have been associated with declines in IQ and school performance.²⁷

According to the CDC's state-based Adult Blood Lead Epidemiology and Surveillance (ABLES) program that tracks elevated blood lead levels in adults, a majority of adults with elevated blood lead levels are occupationally exposed to lead in manufacturing, construction or mining.⁶ Elevated blood lead levels were associated with non-occupational exposures in less than 5% of adults tracked by the National Institute for Occupational Safety and Health's (NIOSH) ABLES program.⁶

Reducing Your Exposure

You can prevent or minimize exposure to lead in the following ways:

- If you live in a house that was built before 1978, it is possible that it was painted with lead-based paint.⁶⁴ Many homes built before 1960 were painted with heavily leaded paint.⁶⁴ You can find out about lead levels in your house paint and house dust by hiring an EPA certified Lead Inspector or Lead Risk Assessor to test your paint.⁶⁵ Home testing kits are also available.⁶⁶
- If lead-based paint is in good condition, leave it undisturbed.⁶⁴ Do not sand or burn wood with lead-based paint because lead may be released into the air.⁶⁴
- It is dangerous to remove lead-based paint yourself because of the dust that it generates.⁶⁴ Hire a professional with experience in lead abatement and removal of lead-based paint.⁶⁴ Occupants should leave the house until all work is finished and clean-up is done.⁶⁴ Furnishings and carpet should be removed from the work area.⁶⁹
- If your occupation or hobby exposes you to lead, take safety precautions such as wearing protective clothing and respirators, and talk to your employer about compliance with regulations on lead exposure. Take steps to reduce the lead dust that you bring home on your body or clothes.⁶⁴ Cases of childhood lead poisoning have been caused by exposure to lead dust in cars or on car seats.¹⁸ If possible, wash your hands and change your clothing and shoes after work to reduce lead dust in your car and home.⁶⁴ Wash these clothes separately.⁶⁴
- Reduce your exposure to dust because household dust may contain lead.⁶⁴ Use a vacuum cleaner with a HEPA filter on carpeted areas.⁶⁹ Mop floors and dust with a wet rag.⁶⁴ Wash your hands often and avoid wearing shoes in the house.⁶⁴ Washing children's toys also reduces dust exposure.⁶⁴ Keep window sills coated with lead-based paint clean because opening and closing the window can create a lot of dust.⁶⁷
- Avoid using ceramics with lead-based glaze or leaded crystalware as containers for food or beverages.¹¹ Acidic food and beverages such as orange juice and coffee may accelerate the leaching of lead from these containers.¹¹
- Use cosmetics and personal care products that do not contain lead or lead acetate. Avoid eye-makeup that contains kohl.⁵ Find out about the ingredients in your personal care products by using the Skin Deep Database: www.cosmeticsdatabase.com.
- Make sure that your child is not playing with toys or jewelry that contain lead.⁶⁷ Check the lead recalls list: www.cdc.gov/nceh/lead/Recalls/default.htm
- The only way to know if your water contains lead is to have it tested.⁶⁷
- Exposure to lead in water is most likely to be caused by lead plumbing.⁶⁷ To reduce your exposure to lead from lead pipes, replace the plumbing with lead-free pipes, adjust the chemistry of your water so it is less corrosive to pipes, and run water for 5 minutes every morning to flush out the pipes.⁶⁸

What Does Exposure to Lead Mean for Our Health?

The presence of environmental chemicals in the human body does not necessarily imply that they are causing adverse health effects; however, environmental chemical exposures can and do affect human health. It is important to note that both the dosage and the timing of exposure have significant effect on any potential health outcome. There is significant evidence that lead causes health effects at much lower doses than previously assumed. The following information is intended to inform the reader about the current state of knowledge on the health effects of lead, including both human and animals studies.

Lead exposure can adversely impact health through a variety of mechanisms. It can imitate other biologically active ions such as calcium, zinc, and iron to gain access to tissues where it can cause damage.²⁸ Lead levels of 50-90 µg/dL have been associated with increased risk of death from all causes, including cardiovascular disease and cancer.²⁹

Neurological Toxicity:

Lead has been associated with neurological effects in children at levels far below the CDC's level of concern of 10 µg/dL.³⁰ One study suggests that there might be no safe threshold for lead toxicity in children, and provides evidence that cognitive function in 3-year old boys is inversely associated with prenatal very low level (<5 µg/dL) lead exposure.³¹ Prenatal exposure to lead can lead to problems including premature births, reduced growth, learning difficulties and decreased IQ.^{27,32}

The neurological toxicity of lead is not limited to children. In adults, lead exposure may reduce memory function,^{33,34} and the effects of childhood lead exposure may persist into adulthood.

Increases in lifetime average blood lead levels (BLL) by 1 µg/dL result in a decrease of an average of 0.87 IQ points.³⁵ There is evidence that intellectual impairment is steeper at BLL of *less than* 10 µg/dL, indicating that incremental changes at lower levels may be more damaging than a similar change at a higher level.³¹ For children with BLL less than 10 µg/dL, an increase in 1 µg/dL lifetime average BLL (while still remaining below 10 µg/dL) was found to result in an IQ decrease of 1.37 points.³¹ Although it may seem counterintuitive that changes at lower lead levels result in greater effect than the same change at a higher level, researchers explain that "there is evidence that high concentrations of heavy metals may enhance cellular defense mechanisms and thereby lessen the rate at which additional damage occurs."³¹

In children from North Carolina who participated in the 2003–2004 NHANES, blood lead levels as low as 2 µg/dL were associated with decreased performance in reading and mathematics.²⁷ Over 50% of children tested between the ages of 1–5 years old had blood lead levels equal to or greater than 2 µg/dL.

Increased exposure to lead is also associated with neuropsychiatric disorders such as attention deficit hyperactivity disorder (ADHD) and antisocial behavior.³⁶ In a 2002 case control study,

juvenile delinquents had higher bone lead levels, indicating a higher lifetime exposure to lead, than the control group.³⁷ No level of lead exposure appears to be 'safe' and even the current 'low' levels of exposure in children are associated with neurodevelopmental deficits.³⁵ There is evidence that childhood lead exposure promotes antisocial and criminal behavior in adulthood.³⁸

Lead also damages peripheral neurons which may lead to prolonged nerve conduction, decreased manual dexterity, weakness, and pain in the limbs.³⁹ Some studies identify lead as a risk factor for amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig's disease, a neurodegenerative disease caused by the degeneration of motor neurons.⁴⁰ Lifetime lead exposures are also associated with the risk of cognitive decline, dementia, and Alzheimer's disease.⁴¹ Lead is also associated with physical damage to brain tissue. An ongoing study has found that adults with childhood lead exposure have reduced brain volume in specific areas of the brain responsible for cognition and emotional responses.⁴²

Cancer

Lead has been determined to be a probable human carcinogen by the Environmental Protection Agency.⁴³

Reproductive Health and Endocrine Disruption

In experiments on female rodents, lead has been shown to alter estrogen receptors in the uterus and ovaries and has been associated with reductions in embryo implantations, late puberty, decreased birth weight, and delays in sexual maturity of offspring.^{44,45,46,47,48}

In a study of the effects of lead exposure on human pregnancy, the mean blood lead level of women who had spontaneous abortion was significantly higher than the group without spontaneous abortion.⁴⁹ In another study, lead was found to be associated with a lower likelihood of having reached menarche and delayed menarche in adolescent girls,⁵⁰ and delayed puberty in boys.⁵¹

Exposure of male animals to lead has been associated with significant reductions in testosterone and sperm production.⁵²

Studies of men who are occupationally exposed to lead have reported reproductive system changes that have the potential to impact fertility. In a cross-sectional study of 503 occupationally exposed men, a median sperm reduction of 49% was reported for men with a blood lead concentration greater than 50 µg/dL.⁵³ Decreases in sperm motility have also been reported for occupationally exposed men.⁵⁴ For men wanting to father a child, occupational lead exposure is associated with delayed time to pregnancy in their partners.⁵⁵

Cardiovascular Toxicity

Elevated blood lead levels have been associated with increased incidences of hypertension, coronary artery disease, cerebrovascular accidents (stroke), and peripheral vascular disease in humans.^{56,57,58}

In experimental animals, chronic lead exposure has been shown to promote atherosclerosis.⁵⁹

Hematological (Blood) Toxicity

Lead has multiple toxic effects on the blood, including interference with the formation of hemoglobin.²⁸ Hemoglobin carries oxygen, which is needed for cellular survival.

Kidney Toxicity

Acute and chronic occupational exposure to lead has been shown to impair kidney function.⁶⁰ High blood lead and bone lead levels (which represents the cumulative lifetime exposure of lead) were associated with decreases in creatinine clearance and increases in serum creatinine which indicate that kidney function is impaired by lead exposure.⁶⁰

In participants in the 1999-2006 NHANES survey, elevated blood lead levels were linked to reduced glomerular filtration rates (an indicator of reduced kidney function).⁶¹ In a longitudinal study of people with diabetes and hypertension, participants with high blood and bone lead levels were more likely to experience a decline in renal function.⁶²

Regulation of Lead

Lead-based paint was widely used in the U.S. and Europe due

to its protective qualities and durability.² Despite reports of childhood lead poisoning related to the consumption of lead based paint from as early as 1904, the United States did not begin its phase-out of lead in paint until 1971 when the Lead-Based Paint Poisoning Prevention Act was passed.² Lead-based paint was phased out of inventories, although houses painted before 1978 may be coated with lead-based paint.²

Leaded gasoline accounted for a majority of the gasoline sold in the U.S. until the Environmental Protection Agency (EPA) began a phase-out of leaded gasoline in 1972 due to its interference with catalytic converter function.² This primary phase-out was completed in 1986, but leaded gasoline remained available in selected markets until the early 1990s.² The phase-out of lead from gasoline contributed to the reduction in blood lead levels in U.S. children. "With the removal of lead from gasoline, average childhood blood lead levels in the U.S. plummeted from approximately 16 µg/dL in 1976 to 3.2 µg/dL in 1994."²

After the recall of lead-contaminated toys in 2007, several states adopted policies to protect children from exposure to lead and other toxic chemicals in toys. Maine, Washington, Connecticut, Delaware, Maryland, Michigan, and Vermont have enacted policies that eliminate or reduce lead in toys.⁶³

Endnotes

- 1 US Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Lead. Available: www.atsdr.cdc.gov/tfacts13.pdf.
- 2 Gilbert SG, Weiss B. 2006. A rationale for lowering the blood lead action level from 10 to 2 micrograms/dL. *Neurotoxicology* 27(5): 693-701.
- 3 Etzel RA, ed. 2003. *Pediatric Environmental Health*. 2nd ed. Elk Grove Village, IL: American Academy of Pediatrics.
- 4 Tong S. 1990. Roadside dust and soil contamination in Cincinnati, Ohio, USA. *Environmental Management* 14:167-113.
- 5 US Food and Drug Administration (FDA). 2009. Kohl, kahal, al-kahal, or surma: By any name, a source of lead poisoning. Available: www.fda.gov/Cosmetics/ProductandIngredientSafety/ProductInformation/ucm137250.htm.
- 6 US Centers for Disease Control and Prevention(CDC). 2009. Adult blood lead epidemiology and surveillance, United States 2005-2007. *Morbidity and Mortality Weekly* 58(14): 365-369. Available: www.cdc.gov/mmwr/preview/mmwrhtml/mm5814a3.htm.
- 7 Cox C. 2009. Pretty but Poisonous: Lead in Handbags and Wallets. Report for the Center for Environmental Health. Available at: www.ceh.org/storage/cehca/documents/accessory_bag_report.pdf
- 8 Cox C. 2008. Illegal and Unhealthy: Lead in California Jewelry. Report for the Center for Environmental Health. Available at: www.ceh.org/storage/cehca/documents/jtf_report_12_8_2008_revised.pdf
- 9 Cox C. 2008. Not for our Best Friends! Lead in Dog Accessories. Report for the Center for Environmental Health. Available at: http://cehca.nonprofitsoapbox.com/storage/cehca/documents/dog_stuff.pdf
- 10 Ecology Center. 2009. HealthyStuff.org. Available at: www.healthystuff.org/.
- 11 Mielke H. 1999. Lead in Inner cities. *American Scientist*. 87: 62– 73.
- 12 Bernard SM, Samet JM, Grambsch A, Ebi KL, Romieu I. 2001. The potential impacts of climate variability and change on air pollution related health effects in the United States. *Environmental Health Perspectives* 109 (Suppl. 2): 199– 209.
- 13 Al-Malack MH. 2001. Migration of lead from unplasticized polyvinyl chloride pipes. *Journal of Hazardous Materials B* 82:263-274
- 14 Mielke HW, Powell ET, Shah A, Gonzales CR, Mielke PW Jr. 2001. Multiple metal contamination from house paints: consequences of power sanding and paint scraping in New Orleans. *Environmental Health Perspectives* 109:973-978.
- 15 Mielke HW, Dugas D, Milke PW, Smith KS, Smith SL, Gonzales CR. 1997. Associations between soil lead and childhood blood lead levels in urban New Orleans and rural Lafourche Parish of Louisiana. *Environmental Health Perspectives* 105: 950-954.
- 16 Laidlaw MAS, Mielke HW, Filippelli GM, Johnson DL, Gonzales CR. 2005. Seasonal and children's blood lead levels: Developing a predictive model using climatic variables and blood lead data from Indianapolis, Indiana, Syracuse, NY, and New Orleans, Louisiana (USA). *Environmental Health Perspectives* 113(6): 793-800.
- 17 US Centers for Disease Control and prevention (CDC). 2004. Blood lead levels in residents of homes with elevated lead in tap water— District of Columbia, 2004. *Morbidity and Mortality Weekly*. 53: 268– 270. Available at: www.cdc.gov/mmwr/preview/mmwrhtml/mm5312a6.htm.

- 18 US Centers for Disease Control and Prevention (CDC). 2009. Childhood lead poisoning associated with lead dust contamination of family vehicles and child safety seats - Maine, 2008. *Morbidity and Mortality Weekly*. 2009 Aug 21;58(32):890-3.
- 19 Hepp NM, Mindak WR, Cheng J. 2009. Determination of total lead in lipstick: Development and single lab validation of a microwave-assisted digestion, inductively coupled plasma-mass spectrometric method. *Journal of Cosmetic Science*, 60(4), July/August, 2009.
- 20 US Food and Drug Administration (FDA). 1998. Dangers of lead still linger. Available: <http://vm.cfsan.fda.gov/~dms/fdalead.html>.
- 21 US Food and Drug Administration (FDA). 2006. Lead in candy likely to be consumed by small children. Available: www.fda.gov/Food/GuidanceComplianceRegulatoryInformation/GuidanceDocuments/ChemicalContaminantsandPesticides/ucm077904.htm.
- 22 Ernst E. 2004. Risks of herbal medicinal products. *Pharmacoepidemiology Drug Safety* 13(11):767-771.
- 23 Garvey GJ, Hahn G, Lee RV, Harbison RD. 2001. Heavy metal hazards of Asian traditional remedies. *International Journal Environmental Health Research* 11(1):63-71.
- 24 United States Consumer Product Safety Commission. 2009. Find recalled products (recalled after 10/01/01) by hazard type. Available: www.cpsc.gov/cgi-bin/haz.aspx.
- 25 Weidenhamer JD. 2009. Lead contamination of inexpensive seasonal and holiday products. *Science of the Total Environment* 407: 2447-2450.
- 26 Weidenhamer JD, Clement ML. 2007. Lead contamination of imported low-cost jewelry in the US. *Chemosphere*. 67:961-965
- 27 Needleman HL, Gunnoe CG, Leviton A, Reed RR, Peresic H, Maher C and Barrett P. 1979. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *NEJM* 300: 684-495.
- 28 Flora SJ, Mittal M, Mehta A. 2008. Heavy metal induced oxidative stress & its possible reversal by chelation therapy. *The Indian Journal of Medical Research* 128 (4): 501-23.
- 29 Schober SE, Mirel LB, Graubard BI, Brody DJ, Flegal KM. 2006. Blood Lead Levels and Death from All Causes, Cardiovascular Disease, and Cancer: Results from the NHANES III Mortality Study. *Environmental Health Perspectives* 114(10):1538-1541
- 30 Gilbert SG, Weiss B. 2006. A rationale for lowering the blood lead action level from 10 to 2 micrograms/dL. *Neurotoxicology* 27(5): 693-701.
- 31 Jedrychowski W, Perera F, Jankowski J, Mrozek-Budzyn D, Mroz E, Flak E, Edwards S, Skarupa A, Lisowska-Miszczuk I. 2009. Gender specific differences in neurodevelopmental effects of prenatal exposure to very low-lead levels: The prospective cohort study in three-year olds. *Early Human Development* 85(8):503-510.
- 32 Schnaas L, Rothenberg S, Flores MF, Martinez S, Hernandez C, Osorio E, Velasco SR, Perroni E. 2006. Reduced Intellectual Development in Children with Prenatal Lead Exposure. *Environmental Health Perspectives* 114(5):791-797.
- 33 Kunert HJ, Wiesmuller GA, Schulze-Robbecke R, Ebel H, Muller-Kupperts M, Podoll K. 2004. Working memory deficiencies in adults associated with low-level lead exposure: implications of neuropsychological test results. *Int J Environ Health* 207:521-530
- 34 Weisskopf MG, Proctor SP, Wright RO, Schwartz J. 2007. Cumulative Lead Exposure and Cognitive Performance Among Elderly Men. *Epidemiology* 18(1):59-66
- 35 Canfield RL, Henderson CR Jr, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. 2003. Intellectual impairment in children with blood lead concentrations below 10 µg/dL. *N Engl J Med* 348:1517-26.
- 36 Bellinger, D.C. 2008. Very low lead exposures and children's neurodevelopment. *Current Opinion in Pediatrics*. 20(2):172-7.
- 37 Needleman HL, McFarland C, Ness RB, Fienberg SE, Tobin MJ. 2002. Bone lead levels in adjudicated delinquents. A case control study. *Neurotoxicol Teratol* 24:711-7.
- 38 Nevin R. 2000. How Lead Exposure Relates to Temporal Changes in IQ, Violent Crime, and Unwed Pregnancy. *Environmental Research Section A* 83(1):1-22
- 39 Nora DB, Gomes I, Said G, Carvalho FM, Melo A. 2007. Modifications of the sympathetic skin response in workers chronically exposed to lead. *Braz J Med Biol Res* 40(1): 81-87.
- 40 Kamel F, Umbrach DM, Hu H, Munsat TL, Shefner JM, Taylor JA, Sandler DP. 2005. Lead Exposure as a Risk Factor for Amyotrophic Lateral Sclerosis. *Neurodegenerative Disease* 2:195-201
- 41 Weuve J, Korrick SA, Weisskopf MG, Ryan LM, Schwartz J, Nie H, Grodstein F, Hu H. 2009. Cumulative exposure to lead in relation to cognitive function in older women. *Environmental Health Perspectives* 117(4):574-80.
- 42 Cecil KM, Brubaker CJ, Adler CM, Dietrich KN, Altaye M, Egelhoff JC, Wessel Stephanie, Elangovan I, Hornung R, Jarvis K, Lanphear BP. 2008. Decreased brain volume in adults with childhood lead exposure. *PLoS Med* 5(5):e112.
- 43 US EPA. 2007. Lead compounds. Available: www.epa.gov/ttn/uatw/hlthef/lead.html
- 44 Wide M. 1980. Interference of lead with implantation in the mouse: effect of exogenous oestradiol and progesterone. *Teratology* 21: 187-191.
- 45 Wiebe JP, Barr KJ. 1988. Effect of prenatal and neonatal exposure to lead on the affinity and number of estradiol receptors in the uterus. *J Toxicol Environ Health* 24: 451-476.
- 46 Dearth RK, Hiney JK, Srivastava V, Burdick SB, Bratton GR, Dees WL. 2002. Effects of lead (Pb) exposure during gestation and lactation on female pubertal development in the rat. *Reprod Toxicol* 16: 343-352.
- 47 Ronis MJ, Badger TM, Shema SJ, Roberson PK, Shaikh F. 1996. Reproductive toxicity and growth effects in rats exposed to lead at different periods during development. *Toxicol Appl Pharmacol* 136: 361-371.
- 48 Ronis MJ, Gandy J, Badger T. 1998. Endocrine mechanisms underlying reproductive toxicity in the developing rat chronically exposed to dietary lead. *J Toxicol Environ Health A* 54: 77-99.
- 49 Borja-Aburto VH, Hertz-Picciotto I, Rojas Lopez M, Farias P, Rios C, Bianco J. 1999. Blood lead levels measured prospectively and risk of spontaneous abortion. *Am J Epidemiol* 150: 590-597.
- 50 Denham M, Schell L, Deane G, Gallo MV, Ravenscroft J, Decaprio AP, the Akwesasne Task Force on the Environment. 2005. Relationship of Lead, Mercury, Mirex, Dichlorodiphenyldichloroethylene, Hexachlorobenzene, and Polychlorinated Biphenyls to Timing of Menarche Among Akwesasne Mohawk Girls. *Pediatrics* 115(2):e127-e134.
- 51 Hauser R, Sergeev O, Korrick S, Lee MM, Revich B, Gitin E, Burns JS, Williams PL. 2008 Association of Blood Lead Levels with Onset of Puberty in Russian Boys. *Environmental Health Perspectives* 116(7):976-980
- 52 Sokol RZ, Berman N. 1991. The effect of age of exposure on lead-induced testicular toxicity. *Toxicology* 69: 269-278.
- 53 Bonde JP, Joffe M, Apostoli P, Dale A, Kiss P, Spano M, Caruso F, Giwercman A, Bisanti L, Porru S, Vanhoorne M, Comhaire F, Zshiesche W. 2002. Sperm count and chromatin structure in men exposed to inorganic lead: Lowest adverse effect levels. *Occup Environ Med* 59: 234-242.
- 54 Kasperczyk A, Kasperczyk S, Horak S, Ostalowska A, Grucka-Mamczar E, Romuk E, Olejek A, Birkner E. 2008. Assessment of semen function and lipid peroxidation among lead exposed men. *Toxicol Appl Pharmacol* 228: 378-384.

- 55 Joffe M, Bisanti L, Apostoli P, Kiss P, Dale A, Roeleveld N, Lindbohm ML, Sallmen M, Vanhoorne M, Bonde JP. 2003. Time to exposure. *Occup Environ Med* 60:742-758
- 56 Pirkle JL, Schwartz J, Landis JR, Harlan WR. 1985. The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. *Am J Epidemiol* 121 : 246-58.
- 57 Staessen J. 1995. Low-level lead exposure, renal function and blood pressure. *Verh K Acad Geneesk Belg*, 57 : 527-74.
- 58 Schwartz J. 1991. Lead, blood pressure, and cardiovascular disease in men and women. *Environ Health Perspect* 91 : 71-5.
- 59 Revis NW, Zinsmeister AR, Bull R. 1981. Atherosclerosis and hypertension induction by lead and cadmium ions: an effect prevented by calcium ion. *Proc Natl Acad Sci USA* 78 : 6494-8.
- 60 Weaver VM, Griswold M, Todd AC, Jaar BG, Ahn KD, Thompson C, Lee B. 2008. Longitudinal associations between lead dose and renal function in lead workers. *Environmental Research* 109: 101-107.
- 61 Navas-Acien A, Tellez-Plaza M, Guallar E, Muntner P, Silbergeld E, Jaar B, Weaver V. 2009. Blood cadmium and lead and chronic kidney disease in US adults: A joint analysis. *American Journal of Epidemiology* doi:10.1093/aje/kwp248
- 62 Tsaih SW, Korrick S, Schwartz J, Amarasiriwardena C, Aro A, Sparrow D, Hu H. 2004. Lead, diabetes, hypertension, and renal function: the Normative Aging Study. *Environ Health Perspect* 112(11):1178-1182.
- 63 Lowell Center for Sustainable Development. 2009. US state level chemicals policy database. Available: www.chemicalspolicy.org
- 64 US EPA. 2008. An introduction to indoor air quality: Lead. Available: www.epa.gov/iaq/lead.html
- 65 US EPA. 2000. Testing your home for lead in paint, dust, and soil. Available: www.epa.gov/lead/pubs/leadtest.pdf
- 66 Consumer Reports. 2008. Lead-paint kits. Available: www.consumerreports.org/health/healthy-living/health-safety/lead-radon-test-kits/lead-paint-kits/lead-radon-tests-kits-lead-paint-kits.htm
- 67 US Centers for Disease Control (CDC). 2009. Lead: Prevention tips. Available: www.cdc.gov/nceh/lead/tips.htm
- 68 Payne M. 2008. Lead in drinking water. *Canadian Medical Association Journal* 179(3): 253-254.
- 69 New York Department of Health. 2001. True/false questions about lead. Available: www.health.state.ny.us/environmental/lead/exposure/childhood/leadq.htm



COMMONWEAL

Fact sheets on toxic chemicals for the *Mind, Disrupted* Biomonitoring Project provided by the Alaska Community Action on Toxics (www.akaction.net) and Commonweal (www.commonweal.org). For more information, please visit the *Mind, Disrupted* website at www.minddisrupted.org, or contact Pam Miller at pkmiller@akaction.net or Sharyle Patton at spatton@igc.org.