



Smart Guide

Hormones in the Food System

Hormones, functioning in balance, are essential for people to grow and develop. Hormones coordinate early brain growth, sexual development and other important sequencing that takes places in our bodies.

The body makes its own hormones. But exogenous hormones, those originating outside the body—birth control, estrogen replacement therapy, pharmaceuticals, pollutants, and so on—clearly can disrupt our own hormone function, sometimes causing grievous harm, as with diethylstilbestrol (DES).¹

Some exogenous hormones are intentionally added to the food system. Others accumulate in food as unintentional (though foreseeable) contaminants. Common practices of our industrialized food system—such as the “recycling” of rendered animal fats back into feed for other animals—can exacerbate contamination.

Hormones in the food system include:

- Hormone growth promoters given to food animals
- Hormone-active pesticides sprayed on food crops
- Hormone plastic additives in baby bottles, infant formula cans or other food packaging
- Hormone disruptors that build up in the food chain, e.g., brominated flame retardants (PBDEs)

Today, many hormone-related chronic diseases are common and/or on the rise.² We know these unhealthy trends likely have multiple social and environmental causes (they're too recent to be attributable to a change in genetics!). The fact that we know this is a problem of multiple causes—and the severe limitations of our existing scientific tools to actually be able to examine multiple factors simultaneously—is why journalists will almost never hear responsible scientists claiming that factor X causes disease Y, or even that we know the three or four separate factors working together to cause disease Y. For one thing, those factors likely vary from person to person.

Nonetheless, ever-strengthening science links exposure to many individual hormone disruptors—pesticides, Teflon chemicals, plasticizers and food contaminants—with these common or rising chronic conditions,³ including:

- Breast and prostate cancer^{4, 5, 6, 7}
- Thyroid disease^{8, 9, 10}
- Obesity and diabetes^{11, 12, 13, 14, 15}
- Endometriosis,¹⁶ uterine fibroids¹⁷ and infertility^{18, 19}
- Immune-related disease, such as asthma or allergies^{20, 21}

Increasingly, exposure in the womb to these same chemicals is implicated in serious problems found in newborns such as birth defects and low birth weight, as well as reduced odds of having a boy child.^{22, 23, 24, 25}

A recent study links a mother's high beef consumption while pregnant (steroid growth promoter use is widespread in beef production) with lower sperm counts in her son.²⁶



Hormone growth promoters given to food animals

Steroid growth promoters. Hormones routinely given to U.S. beef cattle to spur faster growth end up in the meat, and ultimately, our bodies. The Food and Drug Administration (FDA) banned one synthetic estrogen, DES, as an animal growth promoter in 1979. But at least three natural steroids and three synthetic surrogates remain in widespread use as growth hormones by U.S. and Canadian beef cattle producers.²⁷ One of them, trenbolone acetate, is thought to have 8–10 times greater anabolic activity than testosterone.²⁸ A 2004 congressional investigation also revealed that the U.S. veal industry had been giving trenbolone implants to more than 90 percent of veal calves, an illegal practice the industry admitted had been commonplace for decades.²⁹

Though illegal in Europe since 1988, the U.S. government's position is that hormone residues in beef from adult cattle pose no threat to human health.^{30, 31} This safety presumption, however, rests mostly on outdated research concerning the ability of estrogen (estradiol) to mutate genes. The latest research suggests instead that harm from early life exposure to hormones and hormone-disrupting chemicals could stem not from their ability to change the genes, but rather their ability to change the crucial protein

environment surrounding the genes, called the epigenome. It is this protein environment that determines, in part, at which points in one's life particular genes will be turned on and off. By changing this environment, hormone exposure early in life may basically re-program the body's resilience, reproduction and metabolism later in life.

Arsenic growth promoters. Inorganic arsenic causes cancer. Adult cancers may form decades after in-womb exposure to arsenic because it re-programs some genes responsible for proper hormone function. Recent research shows arsenic affects at least 187 different genes, about a quarter of which impact how estrogen or other steroid hormones work in the body.³² Arsenic now appears to also interfere with thyroid function, essential for normal brain development as well as adult function.³³ Researchers see arsenic-related hormone effects even at exposures below 1 parts per billion (ppb), or more than 10 times lower than the legal limit for arsenic in drinking water.³⁴ Americans drinking water containing greater than 10 ppb of arsenic number 13 million.

Since 1946, an arsenic compound has been fed to American poultry to spur growth, feed efficiency and to pigment meat. Adding arsenic to animal feed was never approved as safe in the European Union. The FDA fails to test chicken meat for arsenic, although retail chicken meat appears to commonly contain arsenic residues. A 2005 IATP study consistently detected arsenic in retail chicken, much more often in "conventional" brands than in certified organic or other premium brands.³⁵ Roxarsone, the most common U.S. arsenic feed additive, promotes the growth of blood vessels in chickens to produce pinker meat. However, a 2008 study also found that roxarsone promotes human blood vessel growth. This process, called angiogenesis, is one that occurs in cancers and other diseases.³⁶

rbGH (recombinant bovine growth hormone, also known as rBST) is a genetically engineered growth hormone injected into dairy cows to increase milk production. rbGH is unnecessary to produce milk. Though declared "safe" by the FDA, food safety officials in many other countries—including Canada, Japan, Australia, New Zealand and all 25 nations of the European Union—have refused to approve its use. Concerns with use of rbGH revolve around its known adverse impacts on dairy cows (including increased mastitis infections needing antibiotic use) and the potential harm to humans.³⁷ Increased antibiotic use in food animals contributes to antibiotic resistance transmitted to humans.

rbGH use also increases levels of a hormonal growth factor called IGF-1 in cows and in cow's milk. Increased IGF-1 levels in humans have been implicated in higher rates of colon, breast and prostate cancer.³⁸ As yet, the science is insufficient to assure the safety of drinking milk from cows given rbGH because it is unknown whether doing so will also increase IGF-1 levels in the human bloodstream.

Hormone-disrupting pesticides used in food production

Farmers use synthetic pesticides for the economic purpose of increasing production. Pesticides are inherently

A Food Tale of Two Estrogens

DES. Diethylstilbestrol (DES) was the first identified hormone-disrupting chemical.³⁹ It is a synthetic estrogen created in the 1930s and then used for more than three decades to prevent miscarriage. In 1971, the FDA banned DES use in pregnant women after the first science appeared showing higher cancer risks to their daughters. These "DES daughters," we now know, are *at least* 40 times more likely than the general population to have certain "clear cell" cancers of the vagina or cervix—cancers striking young women in their teens or twenties.⁴⁰ Up to a quarter of DES daughters may be infertile.⁴¹ At birth, DES daughters commonly have defective reproductive organs—one-third of them with an abnormal cervix and more than two-thirds with an abnormal uterus or vaginal tumors. As women, these daughters face a two-in-three chance of failing to produce a live baby *with each pregnancy*.⁴²

Practically since its first manufacture, DES was known to cause cancer.⁴³ Yet for decades, DES was added to animal feed to promote growth in chickens, sheep and beef cattle, alone or in combination with antibiotics.⁴⁴ Congress first passed the Delaney Clause barring the use of cancer-causing food additives in 1958, but language inserted into Congress' Animal Drug Amendments of 1968 allowed continued DES use in food animals, so long as no residues were left in meat. The FDA first tried, unsuccessfully, to ban DES from animal feeds in 1972, after new detection methods found that DES residues were indeed in retail beef and poultry meat. DES use in meat production continued until 1979. The phased removal of DES spurred development and marketing of the several other hormone growth promoters for cattle, still in use today.⁴⁵

Bisphenol A (BPA). BPA is another synthetic estrogen, structurally and functionally similar to DES. In the 1930s, Sir Charles Edward Dodds considered using BPA as an estrogenic human drug before developing the more potent estrogen, DES, for that purpose instead. Only in the 1950s was it discovered that BPA molecules could be linked together to create polycarbonate plastic and epoxy resins.⁴⁶ Despite this history, neither the manufacturers nor end users of BPA ever studied the chemical for its potential to cause cancer or disrupt estrogen function before putting it on the market. In the U.S., BPA use amounts to 2 billion pounds per year, much of it in clear plastic water and baby bottles, food containers, and linings for metal food and infant formula cans. Today, nearly 93 percent of Americans tested carry detectable (ppb) levels of BPA in their urine according to the CDC. Levels are higher in females than males and higher in children than adults.⁴⁷



toxic to pests (through the same mechanisms that cause toxicity in humans). We now suspect or know that dozens of pesticides also disrupt normal hormone function in humans.

Seventy-three suspect pesticides and related chemicals are being reviewed for endocrine-disrupting effects by the U.S. EPA;⁴⁸ another 27 pesticides are defined as known or suspected hormone disruptors by the European Union⁴⁹ or noted expert Dr. Theo Colborn.⁵⁰ Beyond Pesticides has compiled a comprehensive list of these chemicals.⁵¹

These lists include some of the most persistent, chlorine-containing pesticides, like the now-banned DDT as well as lindane (used in food production and to treat children's lice), endosulfan, common corn herbicides like 2,4-D and atrazine, fungicides like maneb and vinclozolin, and organophosphate insecticides like malathion.



Synthetic hormones put into food packaging

Plastic additives are of concern because many plastics are incorporated into food packaging, and have been shown to leach into food or beverages.

PVC and plasticizers. The plastic packaging in some cling wraps, squeeze bottles, cooking oil and peanut butter jars is made of polyvinyl chloride (PVC) or #3 plastic. In addition to the risks PVC itself poses to human and environmental health (see IATP's Smart Plastics Guide), chemical plasticizers often are added to PVC products to increase flexibility or plasticity, and are suspected of interfering with estrogen and thyroid hormones (Ghisari M. et al., 2009). Vinyl lunch boxes can contain hormone-disrupting phthalates, for example. Moreover, PVC manufacture and incineration produces dioxins, another hormone-disrupting family of chemicals and a human carcinogen.

Bisphenol A. Bisphenol A has been found to leach from plastic polycarbonate containers into infant formula and other foods at ppb levels known to cause harm in animals.^{52, 53} Scientists reviewing hundreds of studies of low-dose BPA exposures reached a consensus "great cause for concern" for this exposure. Canadian health officials have determined that BPA is a "toxic substance," supporting Canada's decision earlier in 2008 to ban BPA use in baby bottles.⁵⁴ In 2009, Minnesota and Connecticut joined Canada in banning BPA from some baby products. In light of extensive BPA use and exposure, parallel trends in hormone-related human disease are especially concerning,

including: increasing prostate and breast cancer; urogenital abnormalities in male babies; declining semen quality; early puberty onset in girls; increasing metabolic disorders such as type 2 diabetes and obesity; and disorders of learning and behavior such as ADHD.⁵⁵

Unknown plastic hormones. Concerns about PVC and polycarbonate plastics have focused scrutiny on alternative plastic food packaging as well. Though PET (polyethylene tetrachloride) has been considered a somewhat safer alternative, a recent German study found that mineral water stored in PET bottles included traces of an as-yet-unidentified estrogenic substance.⁵⁶

Fat-friendly, hormone-disrupting industrial contaminants in the food chain

Many industrial chemicals or byproducts of industrial processes also disrupt hormone function while polluting and accumulating in the food chain. Like many pesticide hormone disruptors, dioxins, PCBs (polychlorinated biphenyls) and perchlorate are all persistent, chlorine-containing chemicals that build up in fatty foods.

Dioxins are created when chlorine-containing products, such as vinyl, are manufactured or burned. Once used as industrial lubricants and insulators, PCBs were banned in 1979 due to their high toxicity, including evidence they harm brain development in children, and persistence—they remain in the food chain today. Perchlorate, a rocket fuel ingredient, and brominated flame retardants also make their way into the food chain.

Dioxins and PCBs. Animal forage and feed are one primary route by which people are exposed to hormone-busting dioxins and closely related chemicals, like PCBs. Airborne dioxins, for example, accumulate in dairy fats from animals eating contaminated grasses. Each year, several billion pounds of rendered animal fat is recycled into feed for other animals and fish, along with the dioxins and related toxins found in that fat. In 2003, the Institute of Medicine highly recommended intervention to stop or reduce this recycling of toxics in animal fat in animal feed.⁵⁷

PBDE flame retardants. Polybrominated diphenyl ethers (PBDEs), chemically, are close cousins to PCBs. Like PCBs, these bromine-containing flame retardants disrupt hormone function, especially estrogen and thyroid hormones.⁵⁸ They are also toxic to the brain, reproductive system and liver, and "possibly" cause cancer in people.⁵⁹

People ingest PBDEs in contaminated food and house dust.⁶⁰ Since PBDEs are persistent pollutants, they build up in the environment and food chain, resulting in significant levels in common fatty foods like fish, ground beef, butter and cheese. PBDE accumulates in human fat tissue as well, including breast milk.^{61, 62} High PBDE levels also are found in sewage sludge.

Perchlorate widely contaminates drinking water supplies and common foods; it also disrupts thyroid function. Perchlorate actually blocks the body's ability to move iodine to where it is most needed to make the thyroid hormone.⁶³

Our thyroid glands need iodine to make hormones essential for our bodies' function, as well as for early brain and nervous system development in fetuses and children.^{64, 65} Nursing infants must get essential iodine from mothers' milk; babies lacking sufficient iodine may become mentally impaired.

In women, levels of thyroid hormone change inversely to the level of their exposure to perchlorate. Perchlorate-exposed moms actively concentrate perchlorate in their breast milk,⁶⁶ passing it directly to their nursing infants—plus, iodine levels in their milk are diminished. That's a double whammy for nursing infants who receive less iodine in milk, as well as more perchlorate, which blocks their ability to use the iodine.

Three-quarters of 285 commonly consumed foods and beverages are contaminated with perchlorate, including lettuce, milk and produce, according to FDA data. Some fruits can contain enough perchlorate to exceed the National Academy of Sciences' safe daily dose by more than 25 percent;⁶⁷ 250,000 one-year-olds have perchlorate exposure above the government's safe dose, from these 285 food sources alone.⁶⁸

Perchlorate also contaminates drinking water in 28 states, including Minnesota, at concentrations from 4 to 420 ppb.⁶⁹

Conclusion

Worrisome science around individual hormone-like chemicals continues to build. Meanwhile, our daily cumulative exposure to all these hormone disruptors (also known as "endocrine disruptors") remains unknown, and is virtually unmonitored. This should concern anyone with breast or prostate cancer, thyroid disease, infertility or other hormone-related diseases or conditions, as well as policymakers alarmed about the rising costs of treating these diseases. What can you do?

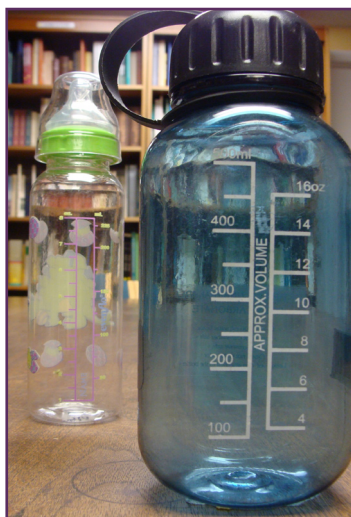
Reduce your total hormone exposure in food and drink

1. **Eat low-fat meats and dairy products.** Steroids and related hormones are fat friendly—they concentrate in whole milk and fatty tissue in meats. Serve low-fat milk to adults and children age two and older. Choose other low-fat dairy products such as cheese, yogurt and cottage cheese. Use lower-fat cooking methods: broiling, grilling, roasting or pressure-cooking, and avoid frying in lard, grease or butter. Cut off visible fats before cooking, and avoid using cooking fats for gravies. This can reduce hormone-busting dioxin levels by half.⁷⁰
2. **Eat "certified" organic when possible.** Organic meat or dairy comes from animals never given hormones (or growth promoting antibiotics or arsenic), and only fed organic grains. Organic grains or produce are those raised without synthetic pesticides, which may disrupt

hormones. Organic production also cannot involve sewage sludge, which may contain oral contraceptives, cadmium and heavy metals, and other industrial chemicals that disrupt hormones and are absorbed into plants and grasses eaten by pastured animals.

3. **Avoid pesticide hormones.** Peel your fruits and vegetables, especially if they have been waxed, or wash them with a vegetable wash or diluted vinegar to remove surface pesticide residues.

4. **Use hormone-free cans and bottles.** Minimize exposure to hormone-busting BPA and other plastics additives by using glass, ceramic, or stainless steel food and beverage containers. Avoid microwaving plastic food containers. Call food companies and tell them you want



baby bottles, food liners and other food packaging made without hormone-disrupting plastic additives.

5. **Demand that your elected officials support stronger efforts to keep synthetic hormones out of our food supply:**

- Stop the feeding of dioxins and PCBs in rendered fats back to food animals.

- Require that safer, available alternatives for toxic flame retardants and hormone-busting additives (like BPA and phthalates) be used in food packaging and other products.

- Require the EPA to regulate perchlorate so as to keep it out of food and drinking water.

6. **Read** the "Smart Plastics Guide," the "Smart Guide on Sludge Use in Food Production," and IATP's other Smart Guides at healthobservatory.org.

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References:

1. Colborn T, Dumanoski D, Myes JP. *Our Stolen Future*. Dutton: Penguin Books; 1996; Chlebowski RT, et al. Breast cancer after use of estrogen plus progestin in postmenopausal women. *NEJM*. 2002;360:573-587; Swan SH. Intrauterine exposure to diethylstilbestrol: long-term effects in humans. *APMIS*. 2000;108(12):793-804; Reigart R, Cummins S. Limit hormone-disrupting chemical exposure. *AAP News*. 1996;12:17.
2. McLachlan J. Environmental signaling: what embryos and evolution teach us about endocrine disrupting chemicals. *Endocr Rev* 2001; 22(5):319-341; Diamanti-Kandaraki E, et al. Endocrine-disrupting chemicals: An endocrine society scientific statement. *Endocr Rev*. 2009;30(4):293-342.
3. Diamanti-Kandaraki E, et al. Endocrine-disrupting chemicals: An endocrine society scientific statement. *Endocr Rev*. 2009;30(4):293-342.
4. Grandjean P, Bellinger D, Bergman A, et al. The faroes statement: human health effects of developmental exposure to toxins in our environment. *Basic Clin Pharmacol Toxicol*. 2008; 102:73-75.
5. Cowin PA, Foster P, Pedersen J, Hedwards S, McPherson SJ, Risbridger GP. Early onset endocrine disruptor induced prostatitis in the rat. *Environ Health Perspect*. 2008; 116:923-929.
6. Gray LE, Wilson VS, Stoker TS, et al. Adverse effects of environmental antiandrogens and androgens on reproductive development in mammals. *Int J Androl*. 2006; 29:96-104.
7. vom Saal FS, Belcher SM, Guillette LJ, et al. Chapel Hill bisphenol A expert panel consensus statement: integration of mechanisms, effects in animals and potential impact to human health at current exposure levels. *Reprod Toxicol*. 2007; 24:131-138.
8. Meeker JD, Calafat AM, Hauser R, Di(2-ethylhexyl) phthalate metabolites may alter thyroid hormone levels in men. *Environ Health Perspect*. 2007; 115(7):1029-34.
9. Crofton KE, Craft ES, Hedge JM, et al. Thyroid hormone disrupting chemicals: evidence for dose-dependent additivity or synergism. *Environ Health Perspect*. 2005; 113(11):1549-54.
10. Davey JC, Nomikos AP, Wungjiranirun M, et al. Arsenic as an endocrine disruptor: arsenic disrupts retinoic acid receptor- and thyroid hormone receptor-mediated gene regulation and thyroid hormone-mediated amphibian tail metamorphosis. *Environ Health Perspect*. 2008; 116(2):165-72.
11. Grandjean P, et al. 2008.
12. Ruhlen RL, Howdeshell KL, Mao J, et al. Low phytoestrogen levels in feed increase fetal serum estradiol resulting in the "fetal estrogenization syndrome" and obesity in CD-1 mice. *Environ Health Perspect*. 2008; 16:322-328.
13. Smink A, Ribas-Fito N, Torrent M, et al. Exposure to hexachlorobenzene during pregnancy increases the risk of overweight in children aged 6 years. *Acta Paediatrica*. 2008; 97(10):1465-9.
14. Hugo ER, Brandebourg TD, Woo JG, Loftus J, Alexander JW, Ben-Jonathan N. 2008. Bisphenol A at environmentally relevant doses inhibits adiponectin release from human adipose tissue explants and adipocytes. *Environ Health Perspect*. doi:10.1289/ehp.11537 available at: <http://dx.doi.org/> Accessed August 15, 2008.
15. Lang I, Galloway T, Scarlett A, et al. Association of urinary bisphenol A concentration with medical disorders and laboratory abnormalities in adults. *JAMA*. 2008; 300(11):1303-1310.
16. Rier S, Foster WG. Environmental dioxins and endometriosis. *J. Toxicol. Sci.* 2002; 70:161-170.
17. Newbold RR, Jefferson WR, Banks EP. Long-term adverse effects of neonatal exposure to bisphenol A on the murine female reproductive tract. *Reprod Toxicol*. 2007; 24:253-258.
18. Hauser R, Meeker JD, Duty S, Silva MJ, Calafat AM. 2006. Altered semen quality in relation to urinary concentrations of phthalate monoester and oxidative metabolites. *Am J Epidemiol*. 2006; 17:682-691.
19. Swan SH, Kruse RL, Fan L, et al. Semen quality in relation to biomarkers of pesticide exposure. *Environ Health Perspect*. 2003; 111:1478-1484.
20. Grandjean P, et al. 2008.
21. Takano H, Yanagisawa R, Inoue K, Ichinose T, Sadakane K, Yoshikawa T. Di-(2-ethylhexyl) phthalate enhances atopic dermatitis-like skin lesions in mice. *Environ Health Perspect*. 2006; 114(8):1266-9.
22. Hertz-Picciotto I, Jusko TA, Willman EJ, et al. A cohort study of in utero polychlorinated biphenyl (PCB) exposures in relation to secondary sex ratio. *Environ Health Perspect*. 2008; 7:37.
23. Apelberg BJ, Witter FR, Herbstman JB, et al. Cord serum concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) in relation to weight and size at birth. *Environ Health Perspect*. 2007; 115(11):1670-6.
24. Brucker-Davis F, Wagner-Mahler K, Delattre L, et al. Cryptorchidism at birth in Nice area (France) is associated with higher prenatal exposure to PCBs and DDE, as assessed by colostrums concentrations. *Hum Reprod*. 2008; 23:1708-1718.
25. Developmental origins of environmentally induced disease and dysfunction. Proceedings of the International Conference on Foetal Programming and Developmental Toxicity. Tórshavn, Faroe Islands. May, 20-24, 2007. *Basic Clin Pharmacol Toxicol*. 2008; 102(2):71-273.
26. Swan SH, Liu F, Overstreet JW, Brazil C, Skakkebaek NE. Semen quality of fertile US males in relation to their mothers' beef consumption during pregnancy. *Hum Reprod*. 2007; 22(6):1497-1502.
27. Lange IG, Daxenberger A, Schiffer B, Witters H, Ibarreta D, Meyer HHD. Sex hormones originating from different livestock production systems: fate and potential disrupting activity in the environment. *Anal Chim Acta*. 2002; 473:27-37.
28. Henricks DM, Brandt, Jr, RT, Titgemeyer TC, Milton CT. Serum concentrations of trenbolone-17b and estradiol-17b and performance of heifers treated with trenbolone acetate, melengestrol acetate, or estradiol-17b. *J. Anim. Sci.* 1997; 75:2627-2633.
29. Weise E. Growth hormones in veal spark debate. *USA Today*. 2004.
30. vom Saal F, et al. 2007.
31. Swan SH, et al. 2007.
32. Liu J, Xie Y, Cooper R, et al. Transplacental exposure to inorganic arsenic at a hepatocarcinogenic dose induces fetal gene expression changes in mice indicative of aberrant estrogen signaling and disrupted steroid metabolism. *Toxicol Appl Pharmacol*. Published online Feb 5, 2007. doi:10.1016/j.taap.2007.01.018.
33. Davey JC, et al. 2008.
34. Bodwell JE, Gosse JA, Nomikos AP, Hamilton JW. Arsenic disruption of steroid receptor gene activation: Complex dose-response effects are shared by several steroid receptors. *Chem Res Toxicol*. 2006; 19(12):1619-29.
35. Wallinga D. Playing Chicken: Avoiding arsenic in your meat. Institute for Agriculture and Trade Policy: Minneapolis, MN. Available at: <http://www.iatp.org/iatp/publications.cfm?accountID=421&refID=80529>. Accessed October 3, 2008.
36. Basu P, Ghosh RN, Grove LE, Klei L, Barchowsky A. Angiogenic potential of 3-nitro-4-hydroxy benzene arsonic acid (roxarsone). *Environ Health Perspect*. 2008; 116:520-523.
37. Health Care Without Harm. Position Statement on rBGH. Available at: www.NoHarm.org.
38. For example, S. E. Hankinson, et al. Circulating concentrations of insulin-like growth factor I and risk of breast cancer. *Lancet*. 1998; 351 (9113): 1393-1396; June M. Chan, et al. Plasma insulin-like growth factor-1 [IGF-1] and prostate cancer risk: a prospective study. *Science*. 1998; 279:563-566; LeRoith D, et al. The role of the insulin-like growth factor-I receptor in cancer. *Ann NY Acad Sci*. 1995; 766:402-08; Holmes M, et al. Lifestyle correlates of plasma insulin-like growth factor I and insulin-like growth factor binding protein 3 concentrations. *Cancer Epidemiol Biomarkers Prevent* 2002; 11:862-867; Holmes M, et al. Dietary correlates of plasma insulin-like growth factor I and insulin-like growth factor binding protein 3 concentrations. *Cancer Epidemiol Biomarkers Prevent*. 2002; 11:852-861.
39. Swan SH. Intrauterine exposure to diethylstilbestrol: long-term effects in humans. *APMIS*. 2000; 108(12):793-804.
40. Centers for Disease Control and Prevention. Health Risks and Related Concerns for DES Daughters. Available at http://www.cdc.gov/DES/hcp/information/daughters/risks_daughters.html. Accessed March 11, 2008.
41. Centers for Disease Control and Prevention. Health Risks and Related Concerns for DES Daughters. Available at http://www.cdc.gov/DES/hcp/information/daughters/risks_daughters.html. Accessed March 11, 2008.
42. Swan SH. 2000.
43. Center for Disease Control and Prevention. Current Trends Report of the Recommendations of the 1985 DES Task Force of the U.S. Department of Health and Human Services. *MMWR* 35(10); 155-6,161-2. Publication date: 03/14/1986. Available at <http://wonder.cdc.gov/wonder/prevguid/m0000700/m0000700.asp>. Accessed September 26, 2008.
44. IARC. 1979. Sex Hormones (II). IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans, vol. 21. Lyon, France: International Agency for Research on Cancer. 583 pp.
45. Office of Technology Assessment. Drugs in Livestock Feed (NTIS order #PB-298450). Available at <http://www.princeton.edu/~ota/disk3/1979/7905/790504.PDF>. Accessed October 6, 2008.
46. vom Saal F, et al. 2007.
47. Calafat AM, Ye X, Wong L-Y, Reidy JA, Needham LL. Exposure of the US population to bisphenol A and 4-tertiary-octylphenol: 2003-2004. *Environ Health Perspect*. 2008; 116(1):39-44.
48. Federal Register Notice, Draft List of Initial Pesticide Active Ingredients and Pesticide Inerts to be Considered for Screening under the Federal Food, Drug, and Cosmetic Act, in [EPA-HQ-OPP-2004-0109]. 2007, U.S. EPA.
49. European Commission. Endocrine Disruptor Research in the European Union. [cited 2008 Jan 11]; Available at: http://ec.europa.eu/research/endocrine/index_en.html.
50. Colborn T, Dumanowski D, Myers JP. *Our Stolen Future*. New York, NY: Plume/Penguin Books; 1997.
51. Harriott N, Feldman J. Pesticides that disrupt endocrine system still unregulated by EPA. *Pesticides and You* [Serial online]. 2008; 28(1):11-14. Available at: www.BeyondPesticides.org. Accessed July 31, 2008.
52. Lopez-Cervantes J, Pasiro-Losada P. Determination of bisphenol A in, and its migration from, PVC stretch film used for food packaging. *Food Addit Contam*. 2003; 20(6):596-606.
53. Work Group for Safe Markets. Baby's Toxic Bottle: Bisphenol A Leaching from Popular Baby Bottles. Available at <http://www.chej.org/documents/BabysToxicBottleFinal.pdf>. Accessed March 10, 2008.
54. Canada Gazette. Government notices: department of the environment: significant new activity notice no. 15290. 2008;142(42). Available at <http://canadagazette.gc.ca/part1/2008/20081018/html/notice-c.html>. Accessed November 7, 2008.
55. vom Saal F, et al. 2007.
56. Lubick N. More bad news about plastic containers. *Environ. Sci. Technol*. 2009; 43(10):3406. Available at <http://pubs.acs.org/doi/full/10.1021/es900885t>. Accessed June 25, 2009; Wagner M, Oehlmann J. Endocrine disruptors in bottled mineral water: total estrogenic burden and migration from plastic bottles. *Environ Sci Pollut Res* 2009;16:278-286. Available at <http://www.ncbi.nlm.nih.gov/pubmed/19274472>. Accessed June 25, 2009.
57. Institute of Medicine. Dioxins and dioxin-like compounds in the food supply: Strategies to decrease exposure. Washington, D.C.: National Academies Press; 2003; 9.
58. Legler J, Brouwer A. Are brominated flame retardants endocrine disruptors? *Environ Int*. 2003; 29(6):879-885.
59. National Toxicology Program. 1986. Toxicology and carcinogenesis studies of decabromodiphenyl oxide (CAS No. 1163-19-5) in F344/N rats and B6C3F1 mice (feed studies). TR-309. Research Triangle Park, NC: NTP.
60. Njodin A, Papke O, McGahee E, et al. Concentration of polybrominated diphenyl ethers (PBDEs) in household dust from various countries. *Chemosphere*. 2008 May 22. [Epub ahead of print.]
61. Hites RA. Polybrominated diphenyl ethers in the environment and in people: a meta-analysis of concentrations. *Environ Sci & Technol*. 2004; 38(4):945-56.
62. Schecter A, Pavuk M, Papke O, et al. Polybrominated diphenyl ethers (PBDEs) in U.S. mother's milk. *Environ Health Perspect*. 2003; 111(14):1723-1729.
63. The NIS protein in our body that moves iodide from the bloodstream into breast milk (so that young children can access it) and into the thyroid gland (so the latter can make thyroid hormone) actually prefers perchlorate to iodide. (Dohan et al. *PNAS*. 2007.) In fact, mammals exposed to perchlorate had breast milk perchlorate levels 6-fold higher than in their bloodstream. (Blount BC, et al. 2006).
64. Zoeller RT, Dowling A, Hertzig C, Iannacone EA, Gauger KJ, Bansal R. Thyroid hormone, brain development, and the environment. *Environ Health Perspect*. 2002; 110(suppl 3):355-361.62. Ginsberg GL, Hattis DB, Zoeller RT, Rice DC. Evaluation of the U.S. EPA/OSWER preliminary remediation goal for perchlorate in groundwater: focus on exposure to nursing infants. *Environ Health Perspect*. 2007; 115(3):361-369.
65. Ginsberg GL, Hattis DB, Zoeller RT, Rice DC. Evaluation of the U.S. EPA/OSWER preliminary remediation goal for perchlorate in groundwater: focus on exposure to nursing infants. *Environ Health Perspect*. 2007; 115 (3): 361-369.
66. Dohan Orlaya, Portulano C, Basquin C, Reyna-Neyra A, Amzel LM, Carrasco N. The Na⁺/I⁻ symporter (NIS) mediates electroneutral active transport of the environmental pollutant perchlorate. *Proc Natl Acad Sci*. 2007; 104(51):20250-5.
67. El Aribi H, Le Blanc Y, Antonsen S, Sakuma T. Analysis of perchlorate in foods and beverages by ion chromatography coupled with tandem mass spectrometry (IC-ESI-MS/MS). *Analytica Chimica Acta*. 2006; 567(1):39-47.
68. Murray CW, Egan S, Henry K, Beru N, Bolger PM. US Food and drug administration's total diet study: dietary intake of perchlorate and iodine. *J Exposure Sci Environ Epidemiol*. 2008; 1-10.
69. GAO (U.S. Government Accountability Office). Perchlorate: A system to tract sampling and cleanup results is needed. GAO-05-462. 20 May 2005.
70. Osman D, Wallinga D, Smart Meat and Dairy Guide for Parents and Children. Institute for Agriculture and Trade Policy: Minneapolis, MN. Available at: <http://www.agobservatory.org/library.cfm?refID=72846>.