Rachel Carson's Legacy: Endocrine Disrupting Chemicals and Gender Concerns

In Silent Spring, *Rachel Carson noted that DDT might alter sexual development and reproduction. Yet DDT was only the first of many endocrine disrupting pesticides, pharmaceuticals, and industrial chemicals. Their impact upon nature in turn impacts upon human health.*

Nancy Langston

Rachel Carson's Legacy: Endocrine Disrupting Chemicals and Gender Concerns
GAIA 21/3 (2012): 225–229

Abstract
Rachel Carson's *Silent Spring* ignited a controversy over synthetic chemical residues, which illustrates several important elements of gender in Carson's legacy. First, Carson's approaches in *Silent Spring* challenged traditional gender stereotypes. Second, the reception to *Silent Spring* reveals assumptions about gender that influenced the ways in which Carson's critics understood human and environmental health. Finally, endocrine disrupting chemicals had the potential to disrupt sexual differentiation in exposed animals. Two of Carson's core insights – the trans-generational effects of synthetic chemicals and the ecological context of human health – have continuing relevance for understanding the environmental and human health effects of endocrine disrupting chemicals.

Keywords
endocrine disruptors, gender, Rachel Carson

Contact: *Prof. Dr. Nancy Langston* | King Carl XVI Gustaf Visiting Professor | Umeå University | Department of Historical, Philosophical and Religious Studies | 90187 Umeå | Sweden | Tel.: +46 90 7867040 | E-Mail: nelangst@gmail.com

In 1962, Rachel Carson's *Silent Spring* was published, triggering public concern over chemical residues in food and the environment. Her work helped lead to the founding of the United States Environmental Protection Agency and international agreements to ban or restrict several key synthetic chemicals (Lear 1998). Yet while Carson drew international attention to the pollution problem, the decades that followed witnessed a dramatic increase, rather than decrease, in chemical use. The modern chemical industry, now a two-trillion-dollar-a-year global enterprise, has become central to the global economy, generating millions of jobs and consuming enormous quantities of energy and raw materials. Since 1952, more than 140 000 synthetic chemical compounds have been made, while each year over 70 000 different industrial chemicals are synthesized and sold, which means that billions of pounds of chemicals make their way annually into our bodies and ecosystems (Langston 2010). More than 358 industrial chemicals and pesticides have been detected in the cord blood of minority American infants (EWG 2009).

DDT, Gender, and Responses to *Silent Spring*

The publication of Rachel Carson's *Silent Spring* ignited a controversy over DDT (dichlorodiphenyltrichloroethane) residues, which illustrates several important elements of gender in Carson's legacy. As a scientist, Carson had long worked with and been accepted by male colleagues (see figure, p. 226), but nonetheless the controversy about her book reveals assumptions about gender made by Carson's critics. Second, her approaches in *Silent Spring* challenged traditional gender stereotypes. Finally, as an estrogenic chemical, DDT itself had the potential to disrupt sexual differentiation in exposed animals.

Like many other chemicals developed during the war years, DDT was originally envisioned as a miracle chemical that would improve the quality of human lives at relatively low risk to people (Russell 2001). Researchers in the United States (U.S.) found that low doses of DDT killed mosquitoes and lice, and the Allies be- >

gan spraying DDT where soldiers were threatened by malaria and civilians by typhoid. After World War II, despite the objections of American army scientists, civilian uses and marketing of DDT were permitted. The properties that made DDT so useful in the war – persistence and a broad spectrum of activity – were the same traits that caused concern within the scientific community about its wider use (Russell 2001).

Carson's central argument against DDT in *Silent Spring* was that humans could not separate themselves from nature, and that science, while a powerful tool for understanding nature, could only offer an illusion of control over it. She argued that excessive use of DDT was indicative of a growing ideology of technological control over nature's unruliness that found expression in the wholehearted adoption of the chemical miracle: synthetic hormones in food systems, synthetic pesticides in field crops, synthetic plastics in the home. Scholars such as Hazlett (2004) and Smith (2001) argue that in stressing the ways pesticides such as DDT made their way into the home and body – spaces seen as traditionally belonging to the private, domestic sphere of women – Carson challenged contemporary gender boundaries.

Critics were quick to attack *Silent Spring* in explicitly gendered terms (Lear 1998, Sideris and Moore 2008, Smith 2001, Mauch 2012, in this issue). A letter to *The New Yorker* stated: "We can live without birds and animals, but, as the current market slump shows, we cannot live without business. As for insects, isn't it just like a woman to be scared to death of a few little bugs! As long as we have the H-bomb everything will be O.K." A review in the popular American magazine *Time* accused Carson of using "emotion-fanning words" and making an "unfair, one-sided, and hysterically overemphatic" case, based on her "mystical attachment to the balance of nature" (Smith 2001, pp. 741 f.). The industry representative Robert White Stevens said: "The crux, the fulcrum over which the argument chiefly rests, is that Miss Carson maintains that the balance of nature is a major force in the survival of man, whereas the modern chemist, the modern biologist and scientist, believes that man is steadily controlling nature" (Gore 2007, pp. 65 f.). Hazlett (2004, pp. 715 f.) argues that "in questioning the boundaries between humans and nature, Carson unintentionally had questioned the lines between humans, such as those defined by gender. (...) In response, Carson's detractors – including some conservationists – tightened the entire cultural system of oppositions. (...) Science and technology (...) kept humans and nature separate. Carson and her supporters were hysterical and over-emotional (read, feminine) for suggesting otherwise."

FIGURE: Rachel Carson and U.S. Fish & Wildlife Service artist Bob Hines conducting marine biology research in Florida in 1952. When Hines started his job in 1948, he was chagrined to find that a female biologist was his supervisor. But soon Carson and Hines developed a congenial working relationship that evolved into a loyal friendship (Juriga 2010).

Transgenerational Effects

Endocrine disrupting chemicals (EDCs) such as DDT, polychlorinated biphenyls (PCBs), diethylstilbestrol (DES), and polybrominated diphenyl ethers (PBDEs) have caused increasing concern (Vandenberg et al. 2012). EDCs can interfere with the hormonal signaling systems that control sexual and reproductive development. Sources include pesticides, pharmaceuticals, plastics, and increasingly flame retardants. Routes of human exposure include water, atmosphere, and food (McKinlay et al. 2008, Scheringer 2012, in this issue, Werner and Hitzfeld 2012, in this issue).

Carson gave voice to growing concerns about the reproductive risks posed by synthetic chemicals with hormonal actions. In particular, well before the development of epigenetics[1] as a thriving research field, Carson noted that DDT might have transgenerational effects, writing that "the insecticidal poison affects a generation once removed from initial contact with it" (Carson 1962, p. 121). Concerns about DDT's effects on sexual differentiation emerged even before the publication of *Silent Spring*. In 1950, the American biologists Burlington and Lindeman found that DDT effectively castrated male chicks: the testes of treated chicks were less than one-fifth the size of those of control chicks (Burlington and Lindeman 1950). Burlington and Lindeman urged further investigations of these estrogenic effects of DDT, and other researchers showed that DDT affected steroid hormones and reproduction (Conney et al. 1967, Welch et al. 1969). After the publication of *Silent Spring*, journalists focused on DDT's potential to affect sexual traits and the expression of gender. Common headlines, such as *Scientist Warns of DDT Peril to Sex Life, Scientist Fears DDT Can Cause Sex Change,* and *DDT Termed Peril to the Sex Organs,* foreshadowed media responses to EDC research 25 years later (Dunlap 1981). Recent research in epigenetics has shown that EDCs, particularly those that mimic the effects of estrogen, may alter DNA methylation, promoting reproductive and sexual problems across subsequent generations (Crews et al. 2007).

1 "Epigenetics" is the study of how the environment affects the genome of the individual during its development (and in some cases the development of its descendants) without changing the DNA sequence (Crews 2008).

DES, Endocrine Disruption, and Gender Concerns

The case of DES, the first synthetic chemical to be marketed as an estrogen and one of the first synthetic chemicals identified as an endocrine disruptor, illustrates some complexities of EDCs and gender. Starting in the 1940s, millions of women were prescribed DES by doctors, at first to treat the symptoms of menopause. In 1947 the Food and Drug Administration (FDA) approved DES for pregnant women with diabetes, and drug companies advertised it widely, promoting the use of DES even in healthy pregnancies to reduce the risk of miscarriage. DES was also approved in 1947 in the U.S. as a steroid to promote growth in livestock by increasing fat deposition, first in poultry, then in cattle. At the peak of its use in the 1960s, DES was given to nearly 95 percent of feedlot cattle in the U.S., which meant that millions of people consumed meat contaminated with low-level residues, while the estrogenic wastes from feedlots made their way into aquatic ecosystems. In 1971 researchers in Boston reported a cluster of formerly rare vaginal cancers in young women whose mothers had taken DES while pregnant. These problems emerged only at puberty or young adulthood, sometimes decades after fetal exposure (Langston 2010). Recent research has shown that in utero exposure to DES induces persistent epigenetic changes in the developing uterus and also increases the risk of breast cancer in adult women (Doherty et al. 2010).

Even before the FDA approved DES for human use in 1941, researchers knew that it caused problems with sexual development in laboratory animals, but these problems were not apparent until adulthood (Langston 2010). These concerns led FDA commissioner Walter Campbell to reject the drug in 1940, insisting regulators must follow the "conservative principle", essentially adopting the precautionary principle 60 years before that term came into common usage. Yet a year later, the agency abandoned its position, and by 1947 the FDA was insisting that women with concerns about DES exposure had to prove that DES had caused harm, rather than drug companies having to show that it was safe. When companies applied for approval to use DES in livestock and for pregnant women, the same pattern unfolded twice more, with the agency first refusing approval, citing the need for precaution given the known risks of the drug, but then changing its position under industry pressure, when short-term studies did not show direct effects on exposed adults (Langston 2010).

While the reasons for the regulatory failure were complex, gender assumptions were part of the picture. Many of the patterns that Carson experienced in the DDT case were true for DES as well: Regulators were far more skeptical of women's claims of harm from DES than they were of industry claims for its safety. Many regulators shared with industry staff a modernist worldview combining faith in scientific expertise with the belief that technological progress could and should control nature. Cultural assumptions about gender differences shaped the ways that scientists, regulators, medical practitioners and consumers understood chemicals and their effects on the body. For example, even while DES use in cattle was being banned due to the chemical's direct link to vaginal cancer in girls exposed in utero, its use to stunt the height of tall girls was expanding. In 1946, a research abstract had suggested that by closing growth plates in bones, DES could help limit the height of prepubescent girls who were "becoming alarmed and unhappy about the extremes to which their exuberant, albeit normal growth was carrying them" (Crawford 1978, cited in Lee and Howell 2006, p. 1036). The abstract received little attention until 1956, when the endocrinologist Goldzieher began promoting DES to treat "excessive growth in the adolescent female" (Goldzieher 1956). By 1977, when DES use had long been shown to cause cancer in women, fully half of surveyed pediatricians reported that they had treated tall girls with DES and related estrogens to prevent them from getting taller than feminine women "ought" to be (Lee and Howell 2006). As Lee and Howell (2006, p. 1039) point out, "idealized gender relations may be as important as scientific studies in determining what we will do as practicing clinicians," a lesson that also applied to DES treatment for menopause and pregnancy. While DES is no longer prescribed to pregnant women or girls who might become tall, the fetal and transgenerational effects of current fertility drugs remain understudied (Elizur and Tulandi 2008), suggesting that the lessons of DES have not yet been learned.

Sex Ratios

Carson used her examples of wildlife affected by synthetic chemicals to make an important point: chemicals that affect wildlife and laboratory animals can also affect people. However, establishing those relationships has proven difficult. Many researchers are concerned that EDCs might lead to problems with human reproductive health (Fowler et al. 2002, Caserta et al. 2011, Kortenkamp et al. 2011). Yet studies of people exposed to estrogenic chemicals have led to conflicting results, as sex ratio research illustrates.

Concern about endocrine disruptors has long been entangled with questions of gender boundaries because even low doses of hormone disrupting chemicals can have significant effects on sexual differentiation (Vandenberg et al. 2012). Sex ratio has been suggested as a potential environmental health indicator of exposure to estrogenic chemicals (Jarrell 2002). Some wildlife populations exposed to EDCs have developed high rates of intersex conditions and increased numbers of female offspring. In Britain, for example, intersex fish have been found at 86 percent of river locations sampled (Jobling et al. 1996), and estrogenic chemicals in sewage effluent have been linked to feminization of male fish (Vajda et al. 2008). Whole-lake experiments on fathead minnows in northwestern Ontario, Canada, showed that chronic, ecologically relevant levels of estrogen exposure led to intersex males, altered oogenesis in females, and resulted in the collapse and near-extinction of the fish from the lake (Kidd et al. 2007). In the Great Lakes basin of North America, several wildlife populations exposed to organochlorines, including herring gulls, Caspian terns, and bloaters, showed significant changes in sex ratio, producing more females than expected (Fox 2001). Erikstad and colleagues >

(2009) found that female (but not male) gulls with the highest loads of organochlorines produced significantly more daughters than sons. More male than female fish embryos were found near a pulp mill that discharged anti-estrogenic chemicals (Larsson et al. 2000).

In humans, the evidence is conflicting. While human sex ratios at birth do appear to be partially influenced by parental hormone levels (James 2008), Terrell and colleagues (2011) reviewed over a hundred studies on sex ratio and human exposure to EDCs and concluded that the results were too variable and inconsistent to provide a clear warning system for broader reproductive health effects, as PCB exposures illustrate.

People who eat large sport fish, which accumulate PCBs, have some of the highest body burdens of PCBs (Turyk et al. 2006). Karmaus and colleagues (2002) found that women with PCB exposure via fish from the Great Lakes gave birth to more sons than expected (sex ratio males/females: 2.29, 95 percent confidence interval: 1.11 to 4.74), but in similar studies exposed women gave birth to fewer sons than unexposed women (Weisskopf et al. 2003). Hertz-Picciotto et al. (2008) note that "maternal exposure to PCBs may be detrimental to the success of male sperm or to the survival of male embryos. Findings could be due to contaminants, metabolites or PCBs themselves." When PCBs were divided into estrogenic and anti-estrogenic congeners, Taylor and colleagues (2007) found that the odds ratio (OR) of male births was slightly elevated among women with intermediate (in the second tertile)(OR = 1.29) and high (third tertile) (OR = 1.48) concentrations of estrogenic PCBs; odds (OR = 0.70) were reduced among women in the highest tertile of anti-estrogenic PCBs.

Because PCBs can travel from industrial countries up the food chain into the blubber of large predatory fish and marine mammals, Arctic indigenous people who eat traditional diets may become exposed to high levels of PCBs. The *Arctic Monitoring and Assessment Programme* (AMAP 2004) examined indigenous women in several Arctic communities in eastern Russia and Greenland. Researchers found that women with the highest levels of PCBs in their blood (greater than four micrograms per liter) gave birth to fewer boys (sex ratio males/females: 0.41). However, women with intermediate levels (two to four micrograms per liter) gave birth to more boys (sex ratio: 1.6). A recent review found no significant differences in expected sex ratios across a wide range of Arctic communities (Bjerregaard et al. 2012). PCB blood levels were not measured, however, making it difficult to compare these results with the *AMAP* results.

Conclusion

Because fetal development is so complex and because low-dose exposures to endocrine disrupting chemicals are so hard to monitor, it can be difficult to determine exactly what exposures are likely to cause significant harm. People are exposed not to one chemical but to many chemicals, which may have effects that magnify or counteract each other.

Carson argued that "we are subjecting whole populations to exposure to chemicals which animal experiments have proved to be extremely poisonous and in many cases cumulative in their effects. These exposures now begin at or before birth and – unless we change our methods – will continue through the lifetime of those now living. No one knows what the results will be because we have no previous experience to guide us" (Carson 1963, pp. 69 f.). Five decades later, we have more experience to guide us, yet two of her critical insights still have relevance for understanding the effects of EDCs on human and environmental health.

First, Carson recognized transgenerational effects of chemical residues. With recent attention to epigenetics, this has emerged as a key focus of concern for EDCs. Age at exposure is likely to be a critical factor in understanding the outcomes. Just as DES exposure had its most profound effects in utero (Doherty et al. 2010), in utero exposure to PCBs or other hormone disruptors such as bisphenol A or the PBDEs can lead to adult changes in reproductive and environmental health (Kortenkamp et al. 2011). With PCBs, while adults often do not show measurable effects of exposure, women who were exposed in utero later developed reproductive health problems such as increased time to pregnancy (Cohn et al. 2011). People exposed to PCBs in utero have not yet been examined to see if their offspring have altered sex ratios or other impacts on reproductive health, but this would be an interesting avenue for future research.

Second, Carson recognized that scientists could not continue to consider humans in isolation from their broader ecological context. Recently, Crews and Gore (2011) and Wingfield and Mukai (2009) have called for EDC research examining the effects of populations and life history variables in changing environments. Climate change, habitat loss, and the complex effects of chemical mixtures all suggest that while mechanisms will remain important, understanding the effects of EDCs will require broader ecological studies. During a lively discussion about whether low levels of organochlorine contaminants continued to affect fish recoveries in the Great Lakes, Carpenter and colleagues (1996) called for whole-ecosystem experiments, which have used direct manipulation of entire ecosystems to unambiguously demonstrate the impacts of environmental factors such as acid rain (for ethical concerns of such experiments, see Farnsworth and Rosovsky 1993.) When whole-ecosystem experiments were performed on estrogens and fish populations (Kidd et al. 2007) they showed that low dose, environmentally relevant estradiol levels over several generations could lead a population close to extinction. Similar whole-ecosystem experiments on the environmental and health effects of emerging contaminants like PBDEs should be a high priority.

References

AMAP (Arctic Monitoring and Assessment Programme). 2004. *Persistent toxic substances, food security and indigenous peoples of the Russian North. Final report.* Oslo: AMAP Secretariat.

Bjerregaard, P., S. Chatwood, B. Denning, L. Joseph, T. K. Young. 2012. Sex ratios in the Arctic: Do man-made chemicals matter? *American Journal of Human Biology* 24/2: 165–169.

Burlington, H., V. F. Lindeman. 1950. Effect of DDT on testes and secondary sex characters of white leghorn cockerels. *Experimental Biology and Medicine* 74/1: 48–51.
Carpenter, S., L. J. Jackson, J. F. Kitchell, C. A. Stow. 1996. Organochlorine contaminants in the Great Lakes: response. *Ecological Applications* 6/3: 971–974.
Carson, R. 1962. *Silent spring.* New York: Houghton Mifflin.
Carson, R. 1963. *Speech to the Garden Club of America.* Quoted in Gore 2007: 69–70.
Caserta, D. et al. 2011. Environment and women's reproductive health. *Human Reproduction Update* 17/3: 418–433.
Cohn, B. A., P. M. Cirillo, R. I. Sholtz, A. Ferrara, J. Park, P. J. Schwingl. 2011. Polychlorinated biphenyl (PCB) exposure in mothers and time to pregnancy in daughters. *Reproductive Toxicology* 31/3: 290–296.
Conney, A. H., R. M. Welch, R. Kuntzman, J. J. Burns. 1967. Effects of pesticides on drug and steroid metabolism. *Clinical Pharmacology and Therapeutics* 8/1: 2–10.
Crawford, J. D. 1978. Treatment of tall girls with estrogen. *Pediatrics* 62/6 (Part 2): 1189–1195.
Crews, D. 2008. Epigenetics and its implications for behavioral neuroendocrinology. *Frontiers in Neuroendocrinology* 29/3: 344–357.
Crews, D., A. C. Gore. 2011. Life imprints: Living in a contaminated world. *Environmental Health Perspectives* 19/9: 1208–1210.
Crews, D. et al. 2007. Transgenerational epigenetic imprints on mate preference. *Proceedings of the National Academy of Sciences* 104/14: 5942–5946.
Doherty, L. F., J. G. Bromer, Y. Zhou, T. S. Aldad, H. S. Taylor. 2010. In utero exposure to diethylstilbestrol (DES) or bisphenol-a (BPA) increases EZH2 expression in the mammary gland: An epigenetic mechanism linking endocrine disruptors to breast cancer. *Hormonal Cancer* 1/3: 146–155.
Dunlap, T. 1981. *DDT: Scientists, citizens, and public policy.* Princeton, NJ: Princeton University Press.
Elizur, S. E., T. Tulandi. 2008. Drugs in infertility and fetal safety. *Fertility and Sterility* 89/6: 1595–1602.
Erikstad, K., J. Bustnes, S. Lorentsen, T. Reiertsen. 2009. Sex ratio in lesser black-backed gull in relation to environmental pollutants. *Behavioral Ecology and Sociobiology* 63/6: 931–938.
EWG (Environmental Working Group). 2009. *Pollution in minority newborns.* www.ewg.org/minoritycordblood/home (accessed September 14, 2012).
Farnsworth, E. J., J. Rosovsky. 1993. The ethics of ecological field experimentation. *Conservation Biology* 7/3: 463–472.
Fowler, P. A., T. Murray, D. R. Abramovich, N. Haites, R. G. Lea. 2002. Environmental chemical effects on testicular function. *Reproductive Medicine Review* 10/2: 77–100.
Fox, G. A. 2001. Wildlife as sentinels of human health effects in the Great Lakes-St. Lawrence basin. *Environmental Health Perspectives* 109/S6: 853–861.
Goldzieher, M. 1956. Treatment of excessive growth in the adolescent female. *Journal of Clinical Endocrinology & Metabolism* 16/2: 249–252.
Gore, A. 2007. Rachel Carson and *Silent Spring.* In: *Rachel Carson: Courage for the Earth.* Edited by P. Matthiessen. New York: Houghton Mifflin. 63–78.
Hazlett, M. 2004. "Woman vs. man vs. bugs": Gender and popular ecology in early reactions to *Silent Spring. Environmental History* 9/4: 701–729.
Hertz-Picciotto, I. et al. 2008. A cohort study of in utero polychlorinated biphenyl (PCB) exposures in relation to secondary sex ratio. *Environmental Health* 7/1: 37.
James, W. H. 2008. Evidence that mammalian sex ratios at birth are partially controlled by parental hormone levels around the time of conception. *Journal of Endocrinology* 198/1: 3–15.
Jarrell, J. 2002. Rationale for the study of the human sex ratio in population studies of polluted environments. *Cadernos de Saúde Pública* 18/2: 429–434.
Jobling, S. et al. 1996. Predicted exposures to steroid estrogens in U.K. rivers correlate with widespread sexual disruption in wild fish populations. *Environmental Health Perspectives* 114/S1: 32–39.
Juriga, J. D. 2010. *In memoriam – Bob Hines.* www.rachelcarson.org/memoriamBH.aspx (accessed September 10, 2012).
Karmaus, W., S. Huang, L. Cameron. 2002. Parental concentration of dichlorodiphenyl dichloroethene and polychlorinated biphenyls in michigan fish eaters and sex ratio in offspring. *Journal of Occupational Environmental Medicine* 44/1: 8–13.
Kidd, K. A. et al. 2007. Collapse of a fish population after exposure to a synthetic estrogen. *Proceedings of the National Academy of Sciences* 104/21: 8897–8901.
Kortenkamp, A. et al. 2011. *State of the art assessment of endocrine disrupters. Final report.* http://ec.europa.eu/environment/endocrine/documents/4_SOTA%20EDC%20Final%20Report%20V3%206%20Feb%2012.pdf (accessed September 14, 2012).
Langston, N. 2010. *Toxic bodies: Hormone disruptors and the legacy of DES.* New Haven: Yale University Press.
Larsson, D. G. J., H. Hällman, L. Förlin. 2000. More male fish embryos near a pulp mill. *Environmental Toxicology and Chemistry* 19/12: 2911–2917.
Lear, L. 1998. *Rachel Carson: Witness for nature.* New York: Owl Books.
Lee, J. M., J. D. Howell. 2006. Tall girls: The social shaping of a medical therapy. *Archives of Pediatrics and Adolescent Medicine* 160/10: 1035–1039.
Mauch, C. 2012. Der Mensch als Gast der Borgias. Rachel Carsons *Silent Spring* aus historischer Sicht. *GAIA* 21/3: 230–231.
McKinlay, R. J., A. Plant, J. N. Bell, N. Voulvoulis. 2008. Endocrine disrupting pesticides: Implications for risk assessment. *Environment International* 34: 168–183.
Russell, E. 2001. *War and nature: Fighting humans and insects with chemicals from World War I to Silent Spring.* New York: Cambridge University Press.
Scheringer, M. 2012. Umweltchemikalien 50 Jahre nach *Silent Spring:* ein ungelöstes Problem. *GAIA* 21/3: 210–216.
Sideris, L. H., K. D. Moore. 2008. *Rachel Carson: Legacy and challenge.* New York: State University of New York Press.
Smith, M. B. 2001. "Silence, Miss Carson!" Science, gender, and the reception of *Silent Spring. Feminist Studies* 27/3: 733–752.
Taylor, K. C., L. W. Jackson, C. D. Lynch, P. J. Kostyniak, G. M. Buck Louis. 2007. Preconception maternal polychlorinated biphenyl concentrations and the secondary sex ratio. *Environmental Research* 103: 99–105.
Terrell, M. L., K. P. Hartnett, M. Marcus. 2011. Can environmental or occupational hazards alter the sex ratio at birth? A systematic review. *Emerging Health Threats Journal* 2011/4: 7109 doi:10.3402/ehtj.v4i0.7109.
Turyk, M. et al. 2006. Relationship of serum levels of individual PCB, dioxin, and furan congeners and DDE with Great Lakes sport-caught fish consumption. *Environmental Research* 100/2: 173–183.
Vajda, A. M., L. B. Barber, J. L. Gray, E. M. Lopez, J. D. Woodling, D. O. Norris. 2008. Reproductive disruption in fish downstream from an estrogenic wastewater effluent. *Environmental Science and Technology* 42/9: 3407–3414.
Vandenberg, L. N. et al. 2012. Hormones and endocrine-disrupting chemicals: Low-dose effects and nonmonotonic dose responses. *Endocrine Reviews* 33/3: 378–455.
Weisskopf, M. G., H. A. Anderson, L. P. Hanrahan, Great Lakes Consortium. 2003. Decreased sex ratio following maternal exposure to polychlorinated biphenyls from contaminated great lakes sport-caught fish: A retrospective cohort study. *Environmental Health* 2/1: 2.
Welch, R. M., W. Levin, A. H. Conney. 1969. Estrogenic action of DDT and its analogs. *Toxicology and Applied Pharmacology* 14/2: 358–367.
Werner, I., B. Hitzfeld. 2012. 50 years of ecotoxicology since *Silent Spring* – A Review. *GAIA* 21/3: 217–224.
Wingfield, J. C., M. Mukai. 2009. Endocrine disruption in the context of life cycles: Perception and transduction of environmental cues. *General and Comparative Endocrinology* 163/1–2: 92–96.

Submitted June 29, 2012; revised version accepted September 5, 2012.

Nancy Langston

Born 1961 in Hyattsville, MD, USA. Studies in English and zoology. PhD 1994 in environmental studies. Since 2006 member of the Center for Culture, History, and Environment, since 2007 professor at the Nelson Institute for Environmental Studies and Department of Forest and Wildlife Ecology, both at the University of Wisconsin, Madison, WI, USA. 2012 to 2013 King Carl XVI Gustaf Professor of Environmental Science, Umeå University, Sweden. Research area: environmental history of endocrine disruptors.