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The Impact of Environmental and Occupational Exposures on Reproductive Health

Stephanie Chalupka and Andrew N. Chalupka

ABSTRACT

Environmental exposures during critical periods of susceptibility in utero may result in lifelong or intergenerational adverse health effects. Most chemicals in commercial use in the United States have not been tested for possible developmental toxicity to fetuses, infants, and children. Environmental and occupational exposures can result in adverse effects on female and male reproduction. Nurses can identify at-risk patients, provide education about the impact of chemical toxicants, and empower women to take precautionary action.

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A growing body of scientific research provides disturbing evidence of the potential impact of environmental toxicants that profoundly affect human reproductive health and human development. The results of in utero exposure to environmental toxins were thrust into the public consciousness in the 1960s with events such as the poisoning of Minamata Bay in Japan with mercury dumped by a plastics and petrochemical company (Takeuchi, Morikawa, Atsumoto, & Shiraishi, 1962). Mercury poisoned thousands of people who consumed fish caught in the bay. In addition, infants born to mothers who consumed fish from contaminated waters developed blindness, diffuse central nervous system damage including cerebral palsy, and profound mental retardation (Satoh, 2003). Media coverage of chemically contaminated drinking water, hazards in the work environment, industrial accidents, poor air quality, and other environmental links to fertility, birth defects, and chronic illnesses has significantly affected the public consciousness with the potential effects of the environment on human health.

Since the end of World War II, more than 87,000 synthetic chemicals have been introduced into commercial use in the Unites States (United States Environmental Protection Agency, 2007). Many of these chemicals are widely dispersed in air, water, food crops, and consumer products providing ample opportunity for exposure in the home, workplace, school, and the community. Although exposures during critical periods of susceptibility in utero may result in lifelong or even intergenerational adverse health effects, only a few of these chemicals have been tested for their potential for human toxicity and for their possible developmental toxicity to fetuses, infants, and children (Chalupka, 2005a).

Until recently, it was believed that the low-level exposure to chemicals in everyday life did not pose a risk to fertility, reproduction, or development. However, a significant body of scientific literature suggests otherwise. Adverse reproductive outcomes exact enormous emotional, health, and economic costs for those involved. It is believed that

approximately 3% of fetal developmental defects are attributable to chemical exposures, with an additional 25% attributable to the combination of environmental and genetic factors (Weck, Paulose, & Flaws, 2008). However, these figures are based on scarce chemical testing data about neurotoxicity and therefore likely underestimate the actual prevalence of abnormalities resulting from toxicant exposures. This emerging scientific evidence, and an increasingly informed patient population, presents women's health, obstetric, and neonatal nurses with important challenges and opportunities for health promotion and prevention through education, surveillance, research, and advocacy.

This article provides an overview of selected environmental and occupational hazards and their impact on reproductive health and reproductive outcomes. It also provides guidance for women's health, obstetric, and neonatal nurses in the identification of those most susceptible to environmental health hazards and discussion of appropriate health promotion and health protection strategies for environmentally related illnesses.

Types of Reproductive Hazards

Reproductive hazards many be categorized as toxic (e.g., organic solvents, metals, pesticides, polychlorinated biphenyl [PCB]), physical (e.g., ionizing radiation), and biological (e.g., toxoplasma, Parvovirus B19) (Smedley, Dick, & Sadhra, 2007). Because there has been limited exploration of chemical exposures in the obstetric and gynecologic nursing literature, this article focuses on toxic exposures. Types of adverse reproductive and developmental outcomes associated with exposure to toxins include subfecundity (Fei, McLaughlin, Lipworth, & Olsen, 2009); infertility (Mendola, Messer, & Rappazzo, 2008); spontaneous abortion, still birth/infant death, preterm birth, congenital anomalies (Ritz, Wilheim, Hoggatt, & Ghosh, 2007); low birth weight (Bell, Ebisu, & Belanger, 2007); developmental delays (Mendola, Selevan, Gutter, & Rice, 2002); and childhood cancers (Andersen, Nielsen, & Grandjean, 2000) (see Table 1).

Routes of Exposure

The routes of exposure to environmental toxins include inhalation (e.g., particulate matter, or polycyclic aromatic hydrocarbons), ocular contact (e.g., chemical splash), ingestion/absorption through the gastrointestinal tract (e.g., drinking water contaminated with disinfection by-products or consumption of food with pesticides, plasticizers, or heavy metals), percutaneous/dermal contact (e.g., contact with pesticides, cosmetics, perfumes), transfer from mother to fetus or amniotic fluid/transplacental (e.g., carbon monoxide), and transfer of chemicals to offspring through lactation (e.g., polybrominated diphenylethers [PBDEs] used as flame retardants in furniture foam and plastics for personal computers to name a few).

Environmental toxicant exposures during critical periods of organ development can cause profound systemic damage to the developing fetus.

Transfer to the Fetus

The placenta offers protection against some unwanted chemical exposures during fetal development but not all. The teratogenic effects of selected environmental toxicants not filtered by the placenta are increasingly recognized (Anderson, Diwan, Fear, & Roman, 2000). For example, many heavy metals easily cross the placenta, and the concentration of mercury, a potent neurotoxin, in umbilical cord blood can be substantially higher than in the maternal blood supply (Sakamoto et al., 2004). Mazdai, Dodder, Abernathy, Hites, and Bigsby (2003) studied human maternal and fetal serum concentrations of PBDE in the United States and found concentrations 20- to 106-fold higher than the levels previously reported in a similar population of Swedish mothers and infants (Guvenius, Aronsson, Ekman-Ordeberg, Bergman, & Noren, 2003; Thomsen, Lundanes, & Becher, 2002).

Specific pesticides and other industrial compounds such as PCBs can accumulate in maternal adipose tissue and may even be transmitted to the infant via breast milk. Polychlorinated biphenyls were widely used as coolants and lubricant in transformers, capacitors, and other electrical equipment. Although manufacture of PCBs was stopped in the United States in 1977 because of evidence that they build up in the environment and can cause a variety of serious harmful health effects, they are a persistent organic pollutant and are still present in the environment. Today, the main routes of exposure occur through dietary sources such as fish (especially sportfish such as salmon caught in contaminated lakes or rivers), meat, and dairy products (Agency for Toxic Substances and Disease Registry, 2007). When persistent substances accumulate in maternal adipose tissue and are

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Table 1: Environmental Contaminants: Sources and Selected Health Effects From Developmental and Adult Exposures (Animal and Human Data)

Contaminant	Sources	Examples of Health Effects Associated With Exposure During Adulthood	Examples of Health Effects Associated With Exposure During Development
Air pollution	Common air pollutants include carbon monoxide, lead, ground-level ozone, particulate matter, nitrogen dioxide, and sulfur dioxide. Air pollution arises from a variety of sources, including motor vehicles, industrial production, energy (coal) production, wood burning, and small local sources such as dry cleaners.	Fetal loss[a]	Low-birth-weight preterm delivery
Bisphenol A (BPA)	Industrial chemical and building block for polycarbonate plastic and epoxy resins. Found in the lining of metal food and drink cans, plastic baby bottles, pacifiers and baby toys, dental sealants, computers, cell phones, hard plastic water bottles, paints, adhesives, enamels, varnishes, CDs and DVDs, and certain microwavable or reusable food and drink containers.	Oocyte chromosome abnormalities, recurrent miscarriage, decreased semen quality	Altered puberty onset, obesity, altered prostate development, decreased semen quality, hormonal changes
Disinfection by-products in drinking water	Over 600 compounds formed by the reaction of chemical disinfectants (most often chlorine) with natural organic matter, primarily in surface waters. Most prevalent compounds are trihalomethanes.	Menstrual irregularities[b]	Fetal growth, IUGR
Glycol ethers	Used in paints, varnishes, thinners, printing inks, electronics, semiconductor industry, leather, photographic film, varnish, enamels, cosmetics, perfumes, brake fluids, wood stains.	Longer menstrual cycles, decreased semen quality,[c] reduced fertility,[d] and fetal loss[a]	
Pesticides	Broad category that includes many classes of insecticides, fungicides, herbicides, rodenticides, and fumigants. Pesticides are used on food, in residential and industrial settings. Exposures can occur through food, drinking water, or from home use.	Menstrual irregularities,[b] reduced fertility,[d] decreased semen quality,[c] miscarriage in female partner, sperm chromosome abnormalities, hormonal changes	Altered sex ratio (H,A), altered puberty, onset malformations of reproductive tract,[a] reduced fertility and fetal growth, IUGR
Phthalates	Plasticizers added to soften plastics like PVC; also found in cosmetics, perfumes, toys, pharmaceuticals,	Altered (earlier) menarche onset, estrous cycle, ovulatory irregularities, decreased semen	Shortened anogenital distance malformations of reproductive tract, hormonal changes, decreased semen quality[c]

Table 1. Continued

Contaminant	Sources	Examples of Health Effects Associated With Exposure During Adulthood	Examples of Health Effects Associated With Exposure During Development
	medical devices, lubricants, and wood finishers.	quality,[c] reduced fertility,[d] fetal loss,[a] endometriosis	
Solvents	Benzene, toluene, xylene, styrene, 1-bromopropane, 2-bromopropane, perchloroethylene, trichloroethylene, and others. Solvents include some of the top production volume chemicals in the United States used in plastics, resins, and nylon, synthetic fibers, rubbers, lubricants, dyes, detergents, drugs, pesticides, glues, paints, paint thinners, fingernail polish, lacquers, detergents, printing and leather tanning processes, insulation, fiberglass, food containers, carpet backing, cleaning products, and a component of cigarette smoke. Exposure is primarily through breathing contaminated air.	Hormonal changes, menstrual irregularities,[b] decreased semen quality,[c] reduced fertility,[d] fetal loss,[a] miscarriage in female partner	
Metals			
Lead	Used in batteries, ammunition, metal products, X-ray shields. Reduced use in gasoline, paints, ceramic products, caulking, and pipe solder. Most common source of exposure in the United States is lead-based paint in older homes, lead-contaminated house dust, and soil and vinyl products.	Fetal loss,[a] reduced fertility[d] hormonal changes, menstrual irregularities,[b] abnormal sperm, altered puberty onset	Hormonal changes, altered puberty onset
Mercury	Used in thermometers, dental fillings, batteries, vaccines, and other industries. Air and water contaminated by industrial emissions and the combustion of coal and waste. Accumulates in food chain; most common source of exposure in United States is contaminated seafood.		
Manganese	Used in the production of batteries, in dietary supplements, and as ingredients in some ceramics, pesticides, and fertilizers. Gasoline additive.		

Table 1. Continued

Contaminant	Sources	Examples of Health Effects Associated With Exposure During Adulthood	Examples of Health Effects Associated With Exposure During Development
Chlorinated hydrocarbons			
Dioxins/furans	Byproducts of the manufacture and burning of products that contain chlorine.	Menstrual irregularities,[b] hormonal changes, reduced fertility, endometriosis, fetal loss, decreased semen quality, altered puberty onset, altered menarche onset	Malformations of the reproductive tract[a] reduced fertility, altered sex ratio, altered puberty onset, decreased semen quality, delayed time to pregnancy
Organochlorine pesticides	Class of pesticides used largely as insecticides. (ex: DDT, chlordane, HCB.) Largely banned in the United States. Persist for decades in the environment. Accumulate up the food chain.		
Penta-chlorophenol	Wood preservative for utility poles, railroad ties, and wharf pilings. Formerly used as a pesticide.		

Note: DES = diethylstilbestrol; DDT = dichlorodiphernyltrchloroethane; HCB = hexachlorobenzene; IUGR = intrauterine growth retardation

[a]Malformations of the reproductive tract: in males, could include shortened ano-genital distance in animals or hypospadias (humans), undescended testicles (cryptorchidism), small testicles (hypoplasia), and structural abnormalities of the epididymis. In females, could include small ovaries, reduced number of follicles (eggs), and structural abnormalities of the oviducts, uterus, cervix, and/or vagina.

[b]Menstrual irregularities could include short or long menstrual cycles, missed periods, abnormal bleeding, anovulation.

[c]Decreased semen quality could include low semen, abnormal sperm shapes or motility, decreased sperm counts.

[d]Reduced fertility could include infertility and increased time to pregnancy (reduced fecundity).

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transmitted to the infant via breast milk, they can result in infant exposure that may significantly exceed the mother's own exposure (Eslami, Koizumi, Ohta, & Inoue, 2006; Jensen & Slorach, 1991). Reliance on formula feeding does not guarantee an absence of contaminants because water used in the formula can also contain contaminants.

Scope of Adverse Reproductive Outcomes

Reduced Fertility and Fecundity

A significant decline in the fertility rates of people in developed countries has been reported in the past three decades. This change is largely attributable to improved contraceptive technology and social mores regarding optimal family size. However, this trend may also be attributable, in part, to reduced biological capacity to reproduce (Olsen & Rachootin, 2003). Even before conception, exposure to environmental toxins can affect fertility and the outcome of pregnancy via germ cell toxicity or hormonal disruption in either males or females.

Of the approximately 62 million women of reproductive age in 2002, about 1.2 million, or 2%, had an infertility-related medical appointment within the previous year, and 8% had an infertility-related medical visit at some point in the past (Centers for Disease Control [CDC], 2009). Infertility services include medical tests to diagnose infertility, medical advice and treatments to help a woman become pregnant, and services other than routine prenatal care to prevent miscarriage. Additionally, 7% of married couples in which the woman was of reproductive age (2.1 million couples) reported that they had not used contraception for 12 months and the woman had not become pregnant (CDC).

The consequences of diethylstilbestrol (DES) use in pregnant women between 1938 and 1971 demonstrated the capacity of synthetic chemicals to alter reproductive function. Research on DES provided evidence of the susceptibility of the female fetus to reproductive abnormalities of environmental etiology. It also illustrated that during critical fetal exposure windows gonadal organoogenisis is sensitive to synthetic hormones and that it may take decades for the results of the exposure to become manifest (Newbold, 2004).

Of particular concern today are endocrine disrupting compounds (EDCs), which are chemical compounds that may be naturally occurring or synthetic compounds capable even at low doses of interfering with endocrine-regulated events like reproduction. They act by mimicking or disrupting the action of the naturally occurring hormones. EDCs are widely used in manufacturing as emulsifiers and industrial surfactants, consumer products including paper wraps, upholstery, carpets, textiles including clothing, fire retardants, pesticides, medical applications, personal care products, and even food contain chemicals that are thought to be hormone disruptors.

Phthalates are ubiquitous, multifunctional, synthetic chemical compounds that are persistent in the environment and have been detected in wildlife and humans around the world (Apelberg et al., 2007; Calafat, Wong, Kuklenyik, Reidy, & Needham, 2007; Fei, McLaughlin, Tarone, & Olsen, 2007). They are used in a variety of consumer and personal care products and as plasticizers in the manufacture of flexible vinyl (used in medical devices, consumer products, flooring and wall coverings, and food contact applications). Manufacturers use phthalates in personal care products (e.g., perfumes, lotions, cosmetics), as solvents and plasticizers for cellulose acetate, and in making lacquers, varnishes, and coatings, including those used to provide timed release in some pharmaceuticals (Hauser, Meeker, Duty, Silva, & Calafat, 2006). Animal studies have demonstrated altered sex hormone homeostasis and have been associated with increased incidence of fetal reabsorptions and pregnancy loss (Lau et al., 2006; Wolf et al., 2007). Epidemiologic evidence suggests that the exposure to the phthalates at plasma levels commonly seen in the general population of developed countries is associated with longer time to pregnancy (TTP) and reduced fecundity (Fei et al., 2009).

Endocrine-disrupting compounds may result in a wide variety of altered cyclicity in females as well. Adult exposure to EDCs have been linked to altered cyclicity with shortening menstrual cycles and increased odds of missed periods, intermenstrual bleeding, and long cycles (Crain et al., 2008). For example, recent studies have provided evidence that women working with or applying pesticides either in horticultural (e.g., flower production and greenhouse work) or agricultural settings are at increased risk for decreased fertility and or fecundability (Abell, Juul, & Bonde, 2000; Idrovo et al., 2005; Lauria, Settimi, Spinelli, & Figa-Talamanca, 2006; Mendola et al., 2008).

The placenta offers some protection against chemical exposures during fetal development, but it does not act as an effective filter against many exogenous chemical agents.

Preconception

Parental exposures before conception can result in an array of adverse reproductive effects ranging from infertility to spontaneous abortion, as well as genetic damage that can lead to a viable, though defective, fetus. Oogonia develop completely during fetal life. Oocytes begin their first meiotic division in utero and then lie dormant, until puberty when ovulation begins. Oocytes are vulnerable to environmental insults during fetal development with resulting impaired fertility (Hage & Frazier, 1998; Woodruff & Walker, 2008). Similarly, injury to stem-cell spermatogonia can occur at any time and lead to infertility. Male reproductive biology includes numerous, narrow windows of vulnerability in parallel with the continual postpubertal production of semen and regeneration of spermatozoa. Paternal exposures might also lead to adverse reproductive outcomes by transmission of toxicants in seminal fluid (Agency for Toxic Substances and Disease Registry, 2002).

Fetal Development

Transplacental transport of toxicants encountered by the mother can have extremely adverse effects on the fetus. The fetus is affected by gestational and previous maternal exposures. During gestation, the placenta that establishes its circulation by around day 17 after fertilization, acts as the most important route of exposure. With an extremely limited ability to detoxify environmental contaminants, the placenta is capable of mitigating only very low concentrations of toxicants. The pregnant woman's past and current contaminant exposure can harm the fetus. In addition, physiologic changes during pregnancy actually mobilize stored toxicants, such as lead from bone (Hu et al., 2006) or PCBs from fat cells, resulting in fetal exposure (Agency for Toxic Substances and Disease Registry, 2002).

Environmental toxicant exposures during critical periods of organogenesis (i.e., the 6-week period that follows the establishment of the placental circulation) can cause profound systemic damage that is out of proportion with the usual dose response. The organ system's susceptibility depends on the timing of the exposure. For example, the critical period for the neural tube is 2 to 4 weeks (the tube is closed by day 28). However, the blood–brain barrier, which

Research is beginning to elucidate the link between environmental toxicants and reproductive health including precocious puberty, altered cycles, infertility, miscarriage, ovarian failure, and endometriosis.

protects the brain from many toxic chemicals, is not completely formed until about 6 months after birth. Thus the fetal brain is particularly vulnerable (Adinolfi, 1985).

In utero exposure to environmental toxicants cause more damage to the nervous system than does exposure at any other stage of development. In the fetus, neurons originate in a germinal matrix and later migrate to predetermined sites (Grandjean & Landrigan, 2006). Exposure to environmental toxicants during this stage might interrupt migration and lead to brain malformation, as is sometimes seen in more well known instances such as fetal alcohol syndrome. Although obvious functional abnormalities after high-dose exposure to environmental toxicants as in the case of heavy metals like lead and mercury has long been apparent to clinicians, lower dose exposures that happen in the home or community setting have more subtle manifestations. Landrigan et al. (1975) and Needleman et al. (1979) originated the notion of subclinical toxicity in children that causes reductions in intelligence and changes in behavior even in the absence of clinically visible symptoms of lead toxicity. Parallel findings have been reported on some other industrial chemicals (Grandjean & Landrigan).

A growing body of evidence suggests that the developing fetus may be particularly susceptible to adverse effects of urban air pollution (e.g., vehicular emissions, woodstoves, etc.). Numerous studies have linked maternal exposure to air pollution containing nitrogen dioxide (NO_2), carbon monoxide (CO), particulate matter with aerodynamic diameter 10 μm or less (PM_{10}), and particulate matter with aerodynamic diameter 2.5 μm or less ($PM_{2.5}$), to adverse birth outcomes including low birth weight (Bell et al., 2007). Other studies link CO and sulfur dioxide (SO_2), PM_{10}, and $PM_{2.5}$ with prematurity, intrauterine growth retardation, and cardiac defects (Ritz & Wilheim, 2008; Ritz et al., 2007). Other pollutants such as polycyclic aromatic hydrocarbons (PAH), which are generated through the combustion of gasoline and diesel-powered vehicles, coal-fired power plants, residential heating, and smoking, are associated with reductions in birth weight, length, and head circumference as well as intrauterine growth restriction (IUGR), fetal death, and premature birth (Weck et al., 2008). Although most attention has been given to maternal smoking as a primary source of exposure to PAHs, recent studies demonstrate that exposure from air pollution alone significantly increases the risk of adverse outcomes of fetal development (Choi, Jedrychowski, & Spengler, 2006).

Fetal exposures can also occur independently of the placenta and are of particular concern to those working in health care, including nurses. Maternal exposure to ionizing radiation can increase the likelihood of the occurrence of childhood leukemia and neurologic delays (Anderson et al., 2000; Fushiki, Matsushita, Yoshioka, & Schull, 1996; Mendola et al., 2002). Although the mechanism is uncertain, some parental exposures during gestation, including occupational exposure to antineoplastic agents, some anesthetic gases and solvents, are associated with adverse reproductive outcomes such as increased time to conception, premature delivery, and low birth weight (Fransman et al., 2007; Saurel-Cubizolles, Hays, & Estryn-Behar, 1994).

Reproductive Effects in Males

The *2002 National Survey of Family Growth* found that approximately 7.5% of men sought help for infertility some time during their lifetime (3.3-4.7 million men) (CDC, 2009). Of men who sought help, 18.1% were diagnosed with a male-related infertility problem, including sperm or semen problems (Anderson, Farr, Jamieson, Warner, & Macaluso, 2008).

In the past 50 years human sperm concentrations have significantly declined globally by approximately 1% per year. Furthermore, and perhaps most disturbingly, it is the younger respondents who demonstrate the lower sperm counts (Swan, Elkin, & Fenster, 2000). It is theorized that this phenomenon is related to increasing levels of environmental exposure to antiandrogenic, estrogenic, or antiestrogenic chemical exposure during critical phases of testicular development (Mocarelli et al., 2008).

The effects of chemicals on male reproductive capacity were first documented in the scientific literature in the late 1880s, such as lead (Reutman & LeMasters, 2007). In 1975, Lancranjan, Popescu, and Gavanescu found that moderately increased absorption of lead associated with occupational exposures resulted in teratospermia, hypospermia, and asthenospermia. Although these early reports concerned lead toxicity, subsequent studies suggested occupational and environmental exposures to other chemicals had male reproductive effects.

During the 1970s, an agricultural fumigant in the United States (1,2-dibromo-3-chloropropane [dibromochloropropane/also known as DBCP]) was used and was demonstrated to have caused sterility in males working in a chemical plant (Whorton, Krauss, Marshall, & Milby, 1977). Subsequently, several studies have documented the association between specific occupational and environmental exposures, including solvents and pesticides, and adverse effects on male endocrine function or alterations in semen quality including alterations in sperm motility, morphology, decreased sperm count and density, and genetic integrity (Jensen, Bonde, & Joffe, 2006).

Although the influence of occupational exposure on male reproductive function has been well studied, an emerging body of evidence similarly points to the potential for deleterious actions to male reproductive function from general environmental exposures. Selevan et al. (2000) provided evidence that exposure to episodes of air pollution may have adverse effects on semen quality, specifically on sperm chromatin integrity. Rubes et al. (2005) demonstrated that exposure to intermittent air pollution may result in sperm DNA damage and thereby increase the rates of male infertility, miscarriage, and other adverse reproductive outcomes. Increased rates of spontaneous abortion are theorized to be mediated through DNA fragmentation in the sperm.

Animal studies have demonstrated the adverse impact of phthalates on male reproduction through their disruption of the androgenic signaling pathways in males. The capacity of phthalates to induce a marked reduction in fetal testosterone and the resulting constellation of male reproductive abnormalities that include hypospadias, cryptorchidism, and malformations of the epididymis, seminal vesicles, prostate and shortened ano-genital distance constitutes the "phthalate syndrome" (Swan, 2008; Welsh, 2008). The impact on humans is thus far less well documented and more controversial (Fisch, Lambert, Hensle, & Hyun, 2009). However, human studies suggest the similar adverse effects on male reproduction with the consequences of phthalate exposure in utero being much more severe than human adult exposure (Lottrup et al., 2006). Guo, Hsu, Hsu, and Lambert (2000) observed alterations in sperm morphology and motility after prenatal exposure to PCBs/PCDF. In human adult males, decreases in sperm motility, concentration, and morphology; increases in sperm DNA damage; as well as decreases in free T and increased LH/free T; and decreases in FSH and LH have been reported as health outcomes associated with exposure to PCBs and phthalates (Duty, Calafat, Silva, Ryan, & Hauser, 2005; Hauser & Calafat, 2005; Hauser et al., 2006, 2007; Jonsson, 2005; Pan et al., 2006; Pant et al., 2008; Zhang, Zheng, & Chen, 2006).

Reproductive Effects in Females

The scientific literature is beginning to elucidate the connection between environmental toxicants and several reproductive health impacts in females including precocious puberty, longer or shorter cycles, missed periods, abnormal bleeding, polycystic ovary syndrome (PCOS), subfecundity, infertility, recurrent miscarriage, ovarian failure, and endometriosis (Cordain, Eades, & Eades, 2003; Drbohlav, Bencko, Masata, & Jirsova, 2004; Falsetti & Eleftheriou, 1996; Genuis, 2006; Mendola et al., 2008; Mlynarcikova, Fickova, & Scsukova, 2005; Sugiura-Ogasawara, Ozaki, Sonta, Makinom, & Suzumori, 2005; Tsutsumi, 2005).

Earlier age at puberty, including outcomes such as thelarche (the beginning of breast development at the onset of puberty), and earlier age at menarche have been observed with exposure to phthalates (Colon, Caro, Bourdony, & Rosario, 2000) and persistent organic pollutants including PCBs (Denham et al., 2005), the pesticide dichlorodiphenyltrichloroethane (DDT) (Ouyang et al., 2005), and its primary metabolite dichlorodiphenyldichloroethylene (DDE) (Krstevska-Konstantinova et al., 2001).

Consumption of drinking water disinfection byproducts (DBPs) and fish contaminated with PCBs and other pollutants have been associated with variations in menstrual and ovarian function. Rather than the development of clinical disorders, data in many studies describe functional variations (alterations in luteal or follicular phase or cycle length indicating a hormonal disruption) (Mendola et al., 2008). Tang and Zhu (2003) observed shorter cycles in women occupationally exposed to lead, as did Windham et al. (2003) in their study of women exposed to chlordibromomethane in drinking water. DDT exposure has been similarly linked to shorter cycles (Ouyang et al., 2005).

Hsieh, Wang, Cheng, and Chen (2005) observed longer cycles in women in the semiconductor industry with exposure to ethylene glycol ethers. Studies of endocrine-active compounds such as 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD/dioxin) (Eskenazi et al., 2002), serum polychlorinated biphenyls (PCBs) (Cooper, Klebanoff, Promislow, Brock, & Longnecker, 2005), and hormonally active pesticides (Farr, Cooper, Cai, Savitz, & Sandler,

2004) have also found evidence of longer cycles. In addition Windham et al. (2003), Farr et al., and Cooper et al. also found evidence of abnormal bleeding and missed periods.

Although the etiology of endometriosis is unknown, the most widely accepted theory has been that it results from the retrograde menstruation into the peritoneum of endometrial cells. Given that retrograde menstruation occurs in 90% of women whereas the actual prevalence of endometriosis is much lower (5-10%), others factors are almost certainly contributing to the development of the condition (Anger & Foster, 2008). The potential of environmental toxicants to trigger the development of endometriosis is suggested by the results of laboratory and population-based studies. Koninckx, Braet, Kennedy, and Barlow (1994) observed that Belgian women, who have the world's highest incidence of endometriosis, also had extremely high levels of dioxin (a known disruptor of estrogen metabolism) in their breast milk. Although some subsequent studies of dioxin levels comparing women with endometriosis and those without endometriosis have not supported this association and have not been consistent with Eskenazi et al. (2002), others have found increased risk (Bruner-Tran, Yeaman, Crispens, Igarashi, & Osteen, 2008; Buck-Louis, Schisterman, Dukic, & Shieve, 2005; Porpora et al., 2006; Quaranta et al., 2006; Reddy et al., 2006; Shi et al., 2006). Meat, fish, and dairy products are the major source (>90%) of human exposure to dioxins. Other pathways of exposure for the general population include inhalation of dioxins from municipal, medical, and industrial waste incinerators or other combustion processes and drinking water. Dioxin has been found in plastic packaging, clothes dryer lint, vacuum cleaner dust, room and car air filters, furnace filter dust, and bleached paper products (Department of Health and Human Services, 2005).

Role of Women's Health, Obstetric, and Neonatal Nurses

Women's health, obstetric, and neonatal nurses are challenged to deal with an ever-growing body of knowledge regarding the impact of environmental and occupational influences on reproductive health. Nurses need to be well informed about sources and effects of exposures in the environment and the workplace during periconceptional, prenatal, and critical windows of susceptibility in fetal and child development. Women's health, obstetric, and neonatal nurses are uniquely positioned to identify at-risk patients and to educate women about the impact of chemical toxicants on reproductive health and help empower women to take precautionary action to protect themselves and their children (see Table 2). Nurses can play a critical role in reproductive health promotion by helping women to sort through very complex and

Table 2: Anticipatory Guidance for Common Environmental Exposures

Potential Toxicant and Sources	Selected Potential Health Effects	Recommended Patient Guidance
Bisphenol A The primary route of exposure to BPA is through diet as BPA migrates into food from containers.	In animal studies BPA acts as a weak estrogen and can impact biological systems at low doses; low levels of exposure to BPA during development could cause changes in behavior, the brain, prostate gland, mammary gland, and the age at which the female attains maturity (Della Seta et al., 2006; Durando et al., 2007; Howdeshell, Hotchkiss, Thayer, Vandenbergh, and Vom Saal, 1999; Timms et al., 2005; Alonso-Magdalena, Morimoto, Ripoll, Fuentes, and Nadal, 2006).	Eliminate the use of containers that have the #7 on the bottom, because some of these are polycarbonate and might contain BPA, substituting glass, porcelain, stainless steel, or safer types of plastics for hot food or liquids, and BPA-free baby bottles; elimination of microwaving polycarbonate plastic food containers; and reducing use of canned food because BPA containing epoxy resins are used to coat metal cans. Promote the use of baby bottles made of glass, polypropylene, or polyethylene and BPA-free baby products.
Drinking water hazards Private wells may be contaminated by a wide range of chemicals. Household plumbing materials may	Health outcomes are toxin specific. Adverse pregnancy outcomes including spontaneous abortion, intrauterine growth retardation, and	If the source of drinking water is a private well, documentation of water quality should be sought because private wells are not regulated for water quality by the

Table 2. Continued

Potential Toxicant and Sources	Selected Potential Health Effects	Recommended Patient Guidance
contain lead and municipal water may use lead service lines.	methemoglobinemia have been attributed to contaminated well water (see also lead in this table) (Chalupka, 2005b)	EPA. All households served by private well must take special precautions to ensure the protection of drinking water. This includes pumping and inspection of septic systems at intervals recommended by the local health department and never disposing of hazardous materials in septic systems. All wells should be tested annually for pH levels, coliform bacteria such as *E. coli*, nitrates, total dissolved solids, and any contaminants of concern locally. Water testing should always be performed by the local health department or a state certified laboratory, not by commercial vendors of water filtration systems. Water testing guidance available at http://www.epa.gov/safewater/faq/pdfs/fs.home watertesting.pdf
Household exposures Residential activities including pesticide/herbicide application, hobbies using solvents (e.g., oil-based paints), paint-stripping agents (see also lead in this table).	Pesticides: altered cyclicity with organochlorine exposure, shortening menstrual cycle with hormonally active nonorganochlorine pesticides resulting in increased odds of missed periods, intermenstrual bleeding, and long cycles (Crain et al., 2008) and Misc. paint-stripping agents: many contain methylene chloride, which metabolizes to carbon monoxide and can be toxic to the fetus. Solvents: menstrual disorders, including menorrhagia, dysmenorhea (Reutman and LeMasters, 2007).	Application of any of these products should be avoided by the preconception patient as well as during pregnancy.
Lead Commonly found in lead-based paint, occupational settings, and contaminated soil. Lead exposure may also occur through hobbies (e.g., stained glass, jewelry making), use of dishes and pottery with lead glaze, occupational exposure, cosmetics from outside of the United States, cultural use of lead (e.g., azarcon, greta, bali goli, ghasard) and household plumbing materials and water service lines.	Known neurotoxin, especially for vulnerable populations such as young children and the fetus. Exposures, even early in pregnancy can pose a risk to the fetus. Lead levels of 10-15 g/dL may lead to central nervous system (CNS) damage; hydroceles; skin tags; hemangiomas, lymphangiomas, and undescended testicles in males; miscarriage; and stillbirth. Adverse effects of elevated maternal blood lead levels (BLLs) during pregnancy include spontaneous abortion, intrauterine fetal demise, premature delivery, intrauterine growth restriction, and postnatal	Education about the risk to unborn child and ways to eliminate the potential for exposure to lead including precautions to take regarding lead sources including potential occupational risks, during home renovation is recommended and information about minimizing/eliminating exposure to lead in household water supplies to improve perinatal outcomes. Use cold water for drinking, cooking, and preparing formula. Lead and other unsafe substances can build up in hot water heaters. If the water has not been turned on for 6 or more hours, let it run for a minute or more before using to make

Table 2. Continued

Potential Toxicant and Sources	Selected Potential Health Effects	Recommended Patient Guidance
	neurologic sequella (McDiarmid et al., 2008).	formula or baby foods. This will help prevent exposure to lead and other pollutants that may be in the water or the pipes. Provide information about potential health effects of lead to women who use it for cultural/cultural practices.
Mercury Fish containing methylmercury Cultural/spiritual/ritual use of mercury for example, azogue	Well-established human neurotoxin with the developing fetus is most sensitive to its adverse effects.	Women of childbearing age who may become pregnant should avoid eating shark, swordfish, king mackerel, and tile fish. Other fish consumption (e.g., tuna) should also be limited but is allowed in up to two meals of 3 ounces each per week. Consult individual state government agencies issue fish advisories and bans relating to mercury concentration in locally caught fish. (Maternal diet may be supplemented with essential fatty acids from non-seafood sources.) Note: The IOM recommendations are slightly more conservative with recommendations for pregnant women, those who could become pregnant, and those breastfeeding, stating that a "reasonable intake" of fish with lesser mercury content is two meals weekly of 3 ounces each (typical can of tuna contains 7 ounces). Provide information about potential adverse health effects of mercury to women who use it for cultural/cultural practices.
Phthalates Used in soft, flexible plastics, polyvinyl chloride (PVC) products, and are found in many consumer products (including shampoos and cosmetics), medical devices, flooring, and a variety of personal care products (such as some, hair polish, hairspray, shampoos, and cosmetics).	Animal studies: disruption of the androgenic signaling pathways in males resulting in a marked reduction in fetal-testosterone and the resulting constellation of male reproductive abnormalities which include hypospadias, cryptorchidism, and malformations of the epididymis, seminal vesicles, prostate and shortened AGD (Swan, 2008; Welsh, 2008). Human studies: suggest similar adverse effects on male reproduction and also include observed alterations in sperm morphology and motility (Guo et al., 2000) after prenatal exposure to PCBs/PCDF In human adult males, decreased	Eliminate the use of PVC or vinyl containing plastics (#3) as food or beverage containers, particularly when microwaving. Use safe alternatives such as glass or polyethylene plastic (symbol #1). Do not place plastics in the dishwasher. Avoid microwaving or heating plastic cling wraps. Promote the use of baby products with labels that say they don't contain phthalates. Opt for products labeled" phthalate free" and "fragrance-free" (not unscented) products whenever possible. "Unscented" may mean that the manufacturer has simply added a fragrance to mask the original odor.

Table 2. Continued

Potential Toxicant and Sources	Selected Potential Health Effects	Recommended Patient Guidance
	sperm motility, concentration, and morphology, increased sperm DNA damage, as well as decreased free T and increased LH/free T, decreased FSH and LH have been reported as health outcomes associated with exposure to PCBs and phthalates in adults (Duty et al., 2005; Hauser & Calafat, 2005; Hauser et al., 2006; Hauser et al., 2007; Jonsson, 2005; Pan et al., 2006; Pant et al., 2008; Zhang et al., 2006).	

technical information about reproductive health risks and offering practical and easy-to-understand guidance. This important opportunity for health promotion and prevention has the potential to prevent adverse outcomes of pregnancy that carry a lifelong burden.

Efforts to prevent adverse pregnancy outcomes can begin with a discussion of environmental hazards during preconception counseling. Educating couples and pregnant women about the preventable environmental exposures that contribute to adverse reproductive outcomes is critical to health promotion efforts. Strategies that patients can use to minimize exposure to occupational or environmental reproductive hazards include assisting patients to identify potentially hazardous exposures at work and at home and providing them with appropriate guidance and assistance in helping them in the selection of safer products (see Table 2).

Many environmental exposures are easily prevented or mitigated. The nurse can provide valuable information about preventing household exposures to solvents (e.g., metal cleaner, furniture strippers, non-latex-based paint) and pesticides, herbicides, and rodenticides that are generally the most common chemical toxicant in the home. The nurse should also discourage hobbies and activities that expose the patient to heavy metals including ceramics, painting, stained glass making (lead solder), and cultural/ritual use of mercury and lead. Similarly, the patient should be advised that home renovations may pose a risk as activities like paint stripping, using either chemicals or heat, make it possible to inhale old lead paint that may have been used previously on the home interior.

Even some nonlatex paints are solvent based and can pose a hazard as can many commercial paint strippers that contain dichloromethane which poses a particular threat to the fetus (McDiarmid & Gehle, 2006). Many excellent resources for providers to use in patient counseling are available from organizations including the March of Dimes (http://www.marchofdimes.com), Physicians for Social Responsibility (http://www.psr.org), the Endocrine Disruption Exchange (http://www.endocrinedisruption.com), the Natural Resources Defense Council (http://www.nrdc.org), and the Environmental Working Group (http://www.ewg.org).

Although the American College of Obstetrics and Gynecology (ACOG) Ante Partum Record already includes environmental history questions about smoking and alcohol use, a much more broad assessment of the home, work, and community environment is required for effective health promotion and prevention efforts (McDiarmid, Gardiner, & Jack, 2008). To prevent or minimize adverse reproductive consequences, the nurse can base anticipatory guidance, health promotion, and prevention activities on a thorough assessment of exposures in the home, community, and workplace to identify potentially harmful exposures (see Figure 1). The Environmental and Occupational Health History Profile (Figure 1) can be used in preconception counseling and at antepartum visits to better understand and prevent significant environmental and occupational exposures.

Because 50% of all pregnancies are unplanned, appropriate protective measures should be in place to ensure the reproductive health of all workers (Reutman & LeMasters, 2007). Unfortunately, that is not always the case. Many chemicals with well-documented reproductive or developmental effects

Please help us to understand your home and work environment so that we can provide better reproductive care to you. Please check the appropriate boxes below.

Present Work

Please tell us where you work.

- ☐ Agriculture
- ☐ Beauty Salon/Nail Salon
- ☐ Construction
- ☐ Dry Cleaning
- ☐ Education
- ☐ Health Care
- ☐ Hospitality (hotels, restaurants)
- ☐ Hazardous Waste
- ☐ Metalworking
- ☐ Mining
- ☐ Office/Clerical
- ☐ Printing
- ☐ Pharmaceutical Manufacture/Compounding
- ☐ Public Safety (Police/Fire/EMS)
- ☐ Veterinary Care
- ☐ Other, please specify _____

Please explain your job to us_____

Potential Workplace Exposures

In your work are you exposed to any of the following?

Biological Agents

- ☐ Animal dander
- ☐ Bacteria
- ☐ Enzymes/Proteins
- ☐ Endotoxin
- ☐ Fungi
- ☐ Protozoa
- ☐ Viruses
- ☐ Other, please specify_____

Physical/Psychological Conditions

- ☐ High Demand/Low Control
- ☐ Prolonged standing/lifting
- ☐ Rotating shift work
- ☐ Strenuous work/Musculoskeletal Strain
- ☐ Other, please specify: _____

Physical Agents

- ☐ Cold
- ☐ Heat
- ☐ Ionizing radiation (e.g., x-rays)
- ☐ Lasers
- ☐ Loud noise
- ☐ Non-ionizing radiation
  - ☐ RF radiation/microwave
  - ☐ Infrared radiation
  - ☐ UV radiation (e.g., welding, photochemical processing)
- ☐ Vibration
- ☐ Other, please specify_____

Chemical Agents

- ☐ Chemotherapeutic agents/pharmaceuticals
- ☐ Inorganic chemical
- ☐ Metals
  - ☐ arsenic ☐ lead ☐ mercury
  - ☐ cadmium ☐ beryllium ☐ chromium
  - ☐ Other, please specify_____
- ☐ Nanoparticles
- ☐ Organic Solvents and Fuels
- ☐ Pesticides/herbicides
- ☐ Oil and Petroleum
- ☐ Other, please specify: _____

Environmental History

Home

When was your home built?
What type of heating do you have?
_____
Have you recently remodeled your home?
_____
What chemicals are stored on your property? _____
Where does your drinking water come from? _____
If you have a well for drinking water, do you have it tested annually? ___ If so for what?

Neighborhood

Do you have any environmental concerns in your neighborhood?
_____
What type of industry or farm is near your home?_____
Do you live near a hazardous waste site?_____

Hobbies/Activities

What activities or hobbies do you and your family engage in?
_____
Do you burn, melt, solder any products?
Do you use pesticides/herbicides in your home or yard?____
Do you eat what you catch or grow?___
Do you use any alternative healing or cultural practices involving lead or mercury?_____

Instructions for use by the nurse: All questions answered in the affirmative should be followed up with questions to elicit specific information to quantify exposure. Quantitative information should include route of exposure (inhalation, dermal, ingestion), timing (relation of exposure to critical time windows), duration (of exposure; hours in work shift), and frequency (of exposure per shift/per week).

Figure 1. Environmental and occupational health history profile.

are in regular commercial use and thus pose a risk to women before pregnancy (McDiarmid et al., 2008). Health outcomes are dependant on the toxicant encountered in the workplace. For example, dry cleaning work with exposure to solvents is associated with menstrual disorders including menorrhagia and dysmenorhea, conception delays are found in partners of male greenhouse workers, and increased rates of aneuploidy (error in cell division that results in the "daughter" cells having the wrong number of chromosomes) is found in the sperm of organophosphate pesticide workers (Reutman & LeMasters). The nurse should elicit the specifics of the patient's employment setting, including the tasks performed, chemicals handled (e.g., metals and solvents), and potential exposures in the workplace. This more complete understanding of the potential hazards present in the patients work is necessary for the nurse to provide comprehensive patient counseling regarding appropriate measures to protect the reproductive health of the patient (see Figure 1).

The nurse should also counsel the patient that in the occupational setting employees have the right to request copies and read the Material Safety Data Sheets (MSDS) for any products to which they may be exposed. However, it is important to note that MSDSs frequently have inadequate information about reproductive or developmental toxicity. The patient should be counseled to use the occupational health nurse as a resource for protecting reproductive health as reproductive safety in the workplace may necessitate the use of engineering controls or PPE to reduce or eliminate exposure. Work areas with potential occupational exposures should be well ventilated with proper control of chemical vapors or other toxicants. If exposures cannot be controlled or eliminated, the patient should be transferred to a different job without the same risks of exposure. Whenever dermal contact with chemicals occurs the area should be thoroughly washed as soon as possible (Reutman & LeMasters, 2007). Patients should also be counseled about avoiding "take-home toxins" that result from the unwise, unknowing transfer of toxicants from the workplace that may result in contamination of the home via soiled clothes, shoes, or other items.

Some environmental or occupational exposures can be extremely complex and may require highly specialized expertise. The national network of Pediatric Environmental Health Specialty Units (PEHSUs) is a valuable resource to those providing care to women and children. PEHSUs (http://aoec.org/PEHSU/index.html) represent a collaborative effort of the U.S. Environmental Protection Agency, Agency for Toxic Substances and Disease Registry, and the Association of Occupational Environmental Health Clinics. The PEHSUs provide a network of experts for consultation and referral in pediatrics, allergy/immunology, neurodevelopment, toxicology, occupational and environmental medicine, and nursing. They were created to ensure that communities have access to, usually at no cost, specialized medical expertise and resources for children faced with a health risk due to a natural or human-made environmental hazard.

Conclusion

The protection of women and their children against environmental toxins is a major challenge in the 21st century. Although hundreds of new chemicals are developed every year and are released into the environment, most are untested for their ability to cause reproductive and developmental effects. Clearly the current chemical testing standards do not provide adequate health protection. Women's health, obstetric, and neonatal nurses, as trusted providers of care, have a special opportunity and responsibility to advocate for policies that will protect the health of women and their children now and in the future.

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Learning Objectives

After reading this article, the reader will be able to

1. Describe the routes of exposure to environmental toxins that have the potential to adversely affect reproductive health.
2. Identify selected adverse reproductive outcomes associated with exposure to environmental toxicants.
3. Employ basic prevention and control strategies in clinical practice to protect reproductive health.
4. Apply knowledge of environmental toxicants with the potential to adversely effect reproductive health to identify at-risk patients, provide guidance, and make appropriate referrals.
5. Provide patients with current and easy-to-understand information about mitigation and prevention of environmental toxicants in the home and work environment.

Post Test Questions

1. Since the end of WWII there has been a rapid proliferation of chemicals introduced into commercial usage. The approximate number is

   1. 5,000
   2. 27,000
   3. 87,000

2. It is estimated that approximately what percent of fetal developmental defects are attributable to chemical exposures or a combination of environmental and genetic factors?

   a. 15
   b. 28
   c. 45

3. Reproductive hazards like organic solvents, metals, pesticides and PCBs are classified as

   a. biological hazards
   b. physical hazards
   c. toxic hazards

4. Anticipatory guidance to prevent exposure to Bisphenol A includes which of the following?

   a. always microwave food or liquids in polycarbonate containers
   b. only use plastic containers with #7 on the bottom for storage of food and liquids
   c. use baby bottles made of glass, propylene, or polyethylene

5. Epidemiologic evidence suggests that the exposure to the phthalates at plasma levels commonly seen in the general population of

developed countries is associated with which of the following?

a. gestational diabetes
b. longer time to pregnancy (TTP)
c. stillbirth

6. Adverse outcomes associated with maternal exposure to the air pollutants carbon monoxide (CO) and sulfur dioxide (SO_2), PM_{10}, and $PM_{2.5}$ include

a. cystic fibrosis, fragile x syndrome, and spinal musculature atrophy
b. prematurity, intrauterine growth retardation, and cardiac defects
c. type I diabetes, achondroplasia, and beta thalassemia

7. In males, exposure to episodes of air pollution may have adverse effects on

a. semen quality
b. semen volume
c. testicular development

8. Exposure to phthalates, persistent organic pollutants including PCBs, and certain pesticides has been associated with

a. earlier age at puberty
b. later age at menarche
c. later thelarche

9. After completing an occupational and environmental health history, a patient is determined to have a very complex set of occupational exposures. The best resource for expert consultation is

a. American College of Obstetricians and Gynecologists
b. March of Dimes
c. Pediatric Environmental Health Specialty Unit

10. Selected health effects from developmental exposures to Bisphenol A (BPA)(found in the lining of certain metal food and drink cans, plastic baby bottles, pacifiers and baby toys, dental sealants, computers, cell phones, hard plastic water bottles, and certain microwavable or reusable food and drink containers) include which of the following:

a. decreased semen quality
b. intrauterine growth restriction (IUGR)
c. miscarriage in female partner

11. Examples of health effects associated with pesticide exposure in residential or industrial settings during adulthood include which of the following:

a. decreased semen quality
b. intrauterine growth restriction (IUGR)
c. shortened anogenital distance malformations of reproductive tract

12. "Phthalate syndrome" refers to capacity of phthalates to induce a constellation of

a. female reproductive abnormalities including precocious puberty, longer menstrual cycles, menstrual irregularities, and longer time to pregnancy
b. gestational abnormalities including recurrent miscarriage, fetal loss, and gestational diabetes
c. male reproductive abnormalities that include hypospadias, cryptorchidism, and malformations of the epididymis, seminal vesicles, prostate and shortened ano-genital distance

13. Which of the following is appropriate guidance for the patient with a private residential well as the source for drinking water?

a. Commercial vendors of water filtration systems are the best choice for routine water testing in residential wells.
b. No drinking water guidance is required as underground wells are naturally protected from environmental contamination.
c. Wells should be tested annually for pH levels, coliform bacteria such as *E. coli*, nitrates, total dissolved solids, and any contaminants of concern locally.

14. In counseling women about potential workplace exposures, the advice that the nurse provides should include which of the following:

a. Employees have the right to request copies and read the Material Safety Data Sheets (MSDS) for any products to which they may be exposed.
b. If workplace exposures cannot be controlled or eliminated, the women cannot ask to be transferred to a different job without the same risks of exposure.
c. Material Data Safety Sheets (MSDS) are required to have adequate and thorough

information about reproductive or developmental toxicity.

15. When educating women about the risk of lead to the unborn child and ways to eliminate the potential for exposure through household water supplies, the nurse should counsel the woman to
    a. always use hot water straight from the tap for cooking, and preparing formula
    b. boil water in a pot for at least one minute before making formula with it
    c. let water run for at least one minute before using it to make formula or baby food if the water hasn't been turned on for six or more hours