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Bisphenol-A and disparities in birth outcomes: a review and directions for future research

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Abstract

Racial disparities in pregnancy outcome in the United States are significant, persistent and costly, but the causes are poorly understood. We propose that disproportionate exposure of African-American women to environmental endocrine disrupting compounds (EDCs) may contribute to birth outcome disparities. Marked racial segregation, as well as health behaviors associated with poverty could result in differences in exposure to particular EDCs. One EDC that has aroused concern in recent years is bisphenol-A (BPA), a widely used industrial plasticizer with known estrogenic properties. Published studies indicate that excessive BPA exposure is associated with reduced fetal survival, as well as reductions in maternal weight and fetal body weight. Related findings include adverse effects of BPA exposure on ovarian function, mammary gland development, earlier age of puberty onset, and some metabolic parameters. However, these findings are largely limited to experimental animal studies, and need to be validated in human populations. Our review supports the need to move beyond the currently dominant toxicological approach to examining the effects of BPA exposure, and rely more on observational human studies and epidemiological methods. Many of the risk factors for racial disparities in pregnancy outcome are global or difficult to modify, but exposure to BPA is a potentially malleable risk factor. If BPA contributes to racial disparities in pregnancy outcome, there are important implications for prevention. It is our hope that this review will stimulate further research in this important and neglected area.

Keywords

Bisphenol-A; endocrine disrupting compounds; birth outcomes; racial disparities; environmental exposures

Introduction

Racial/ethnic disparities in pregnancy outcome remain one of the most persistent and challenging public health problems in the United States. Consistently, infant mortality and

low birth weight rates have been at least twice as high for Black women compared with White women [1, 2]. Although numerous risk factors have been identified, the basic biological mechanisms underlying these disparities remain unknown. Social inequalities that shape environmental and social exposures may be at least partially responsible [3]. Place-based stressors, or biologically relevant components of the human environment that can function independently of individual stressors, can influence the outcome of pregnancy by affecting birth outcomes directly, by increasing exposure to environmental hazards, and by enhancing vulnerability to the toxic effects of contaminant exposures [4].

Of particular interest are environmental endocrine disrupting compounds (EDCs) that interfere with the normal functioning of the endocrine system. Exposure to EDCs continues to be a significant and controversial public health issue. Popular attention has largely focused on the anti-androgenic properties of EDCs, prompted by the increase in multiple endpoints of male dysgenesis over the past few decades, including decreasing sperm counts and quality, and increasing rates of testicular cancer, cryptorchidism and hypospadias [5–8]. Although sexual differentiation has been, and continues to be the major endpoint for the toxicological assessment of EDCs, concern with these substances also stems from their potential to affect reproductive, metabolic and immune functions, growth, behavior and memory. Increasingly, research has confirmed that EDCs work via diverse mechanisms, including estrogenic / anti-androgenic properties, anti-oxidant actions, inhibition of cell cycles and cell differentiation, modulation of angiogenesis and modulation of the activity or expression of steroidogenic enzymes [9–11], with potential to affect an array of organ systems. The developing fetus and neonates are especially susceptible to EDC exposure resulting in adaptations and organizational changes that appear to predispose them to later dysfunctions. Most effects of developmental exposure to EDCs appear to be irreversible; indeed, some of these effects may even be transgenerational, i.e., they lead to epigenetic alteration in the germ line, and thus have biological impacts on all subsequent generations [12]. Exposure to EDCs is chronic, their effects may be latent, and there may be no lowest dose that is without adverse consequences [13, 14].

In recent years, attention has focused on human exposure to bisphenol-A (BPA), a widely used industrial plasticizer with known estrogenic properties. Every year, over 6 billion pounds of BPA are used in the manufacture of epoxy resins and polycarbonate plastics, which in turn find application in a wide variety of domestic products [15–18]. BPA is present in dental fillings, plastic food and water containers, baby bottles, food wrap, as well as in the lining of beverage and food cans, presenting a large number of routes for human exposure. Numerous studies have confirmed leaching of BPA from food containers and detectable levels of BPA are present in a wide range of packaged foods [15–18]. While hydrolysis of the ester bond in BPA and the resultant food contamination is facilitated by heating [19], normal use, such as storage, brushing and dishwashing can also result in polymer degradation leading to release of BPA [20, 21]. Acidic or basic food or beverages, as well as fatty foods, increase the rate of leaching of BPA [17, 22]. Human consumption of BPA from food cans alone has been estimated to be about 6.6 μg/person/day [23]. Evidence exists that high urinary BPA levels are positively correlated with the consumption of canned foods [24], suggesting that oral exposure is likely the primary source of human exposure to BPA. Further, a recent study found that urinary concentrations of BPA collected from male

and female partners on the same day were correlated, suggesting a common source of exposure, most likely diet or common residential sources [25]. Small quantities of BPA have been detected in river water and sediments [26–28], and recently, in indoor air and dust samples [29]. BPA also accounts for most estrogenic activity that leaches from landfills into the surrounding ecosystem [30]; effluent from industrial activity, including treatment of leachate, may serve as an additional route of human exposure [31], particularly if it finds its way into aquatic species.

Given the ubiquity of BPA in human environments, it is not surprising that exposure to BPA is virtually universal. In a recent US based study of human exposure, urine samples were assessed for BPA in a subset of NHANES 2003–2004 study, constituting 2,517 US subjects aged ≥ 6 years [32]. With a detection threshold of 0.4 μg/L urine, BPA was detected in 92.6% of the samples examined. The geometric mean reported was 2.6 μg/L (2.6 μg/g creatinine) and the 95th percentile concentration 15.9 μg/L (11.2 μg/g creatinine) [32]. Total urinary BPA concentrations also differed by race/ethnicity, age, sex, and household income. The prevalence levels and ranges reported in this US study are comparable to those seen in studies in other populations [24, 33, 34]. Scientific dogma holds that BPA cannot be a biologically important pollutant since it is metabolized and excreted relatively quickly. However, studies have found parent (bioactive) BPA in blood of pregnant women and in newborns [15, 35–37], suggesting either continuous exposure of the mother and fetus to BPA, or alternatively, that only a portion of BPA is metabolized and excreted. Data also confirm passage of BPA across the placenta. BPA is found in concentrations of 1–18 ng/mL in the maternal serum, 1–10 ng/mL in amniotic fluid and cord serum taken at birth and up to 100 ng/g in placenta [35–37]. BPA accumulates in body fat [38,39], which in turn could be mobilized during pregnancy and lactation. Breast milk is an additional route of transfer of maternal BPA to offspring; one study reported BPA in more than 60% of human breast milk samples tested, with levels up to 6. 3 ng/mL [40]. A recent study reported BPA concentrations of 1–7 ng/ml (mean ± SD, 3.41±0.13 ng/ml) in the colostrum of all 101 mothers tested [41]. While the reported level of exposure to BPA in these studies is within the current `safe exposure' level established by the US Environmental Protection Agency, 50 μg/kg (50 ppb), recent studies demonstrating that BPA exhibits estrogenic activity at extremely low levels [13–15] suggest the need for revision of these reference levels. On the basis of an expert review of current scientific evidence, the National Toxicology Program has concluded that there is some concern for neural and behavioral effects in fetuses, infants and children at current human exposures [42]. Concerns also appear to relate to effects on the prostate gland, mammary gland, and earlier onset of puberty in females. In contrast, the European Food Safety Authority AFC panel had concluded that the TDI of 0.05 mg/kg body weight (based on the no-observed-adverse-effect level of 5 mg/kg body weight/day in rats and including an uncertainty factor of 100) from their previous risk assessment provides sufficient margin of safety for the protection of the consumer, including fetuses and newborns (43).

There is long-standing evidence that bisphenol A binds to the estrogen receptor and induces estrogen receptor-mediated gene expression, most commonly by mimicking estradiol [10]. *In vivo* studies have shown that prenatal exposure to BPA is associated with changes in

hypothalamic pituitary gonadal axis function, mammary development, and cognitive function as well as sex-specific behaviors in the offspring [13–15, 44]. Several studies have shown that some of these effects occur at low physiologically relevant doses [13–15]. BPA is, in addition, the first environmental chemical known to bind to non-classical estrogen receptors [45]. There is evidence that it binds to the thyroid receptor and affects thyroid hormone signaling *in vitro* [46]. Other studies show that BPA binds to plasma membrane receptor in pancreatic cells [45] and may induce insulin resistance [47]. A recent widely cited study demonstrated that BPA within the normal range of human exposure suppresses levels of adiponectin, and may thus directly increase risks of the metabolic syndrome and associated conditions [48]. BPA is particularly potent during fetal and neonatal development because the liver has limited capacity to deactivate BPA in fetuses and newborns [49, 50]. BPA does not bind to plasma estrogen binding proteins that limit the bioavailability of estradiol [51]. Accumulation of BPA also occurs in pregnant adult females [14], likely because of accumulation in fat [38, 39]. The widespread presence of BPA in human environments, the range of target organs, and the multiplicity of mechanisms all underscore the need for a more comprehensive evaluation of the health effects resulting from exposure to BPA.

Effects of BPA on birth outcomes

Much of the concern surrounding EDCs is centered on their potential links to the disruption of male reproductive function and phenotype. Prenatal or neonatal exposure to BPA has been associated with anomalies in male reproductive function, including increased anogenital distance, prostatic enlargement, decreased epididymal weight, decreased testosterone levels and epididymal sperm counts and compromised sperm quality, in a number of rodent and aquatic species [13–15, 52, 53]. While continued research efforts to elucidate the effects of BPA on male reproductive function are warranted, it is important not to overlook the potential implications of BPA for a number of other population-wide health trends. Examples of population-wide trends in specific health outcomes that may be linked to increasing EDCs in the environment include increases in the incidence of breast cancer [15, 54] and endometriosis [10, 55], population-specific trends in low birth weight [56], the falling age of puberty [57, 58], and increases in cardiovascular problems [59] and obesity [60, 61].

If we consider low birth weight as a potential outcome of exposure to EDCs, there is evidence to suggest that inappropriate exposure to sex steroids /steroid mimics could have an impact on fetal growth and organ differentiation. Fetal exposure to excess prenatal testosterone, an estrogen precursor, from days 30–90 of gestation (term 147 days), resulted in intrauterine growth restriction (IUGR) and low birth weight offspring in sheep [62]. This combined with the fact that *in utero* exposure to diethylstilbestrol (DES), an estrogenic agent, is associated with IUGR [63], supports the hypothesis that increased estrogen signaling during inopportune times of fetal development can lead to IUGR. Because BPA mimics estrogen in its actions, continued exposure to BPA during gestation is likely to have an impact on the developmental trajectory of the fetus. In the remainder of this section, we focus on BPA research that has implications for perinatal outcomes.

A growing body of evidence confirms adverse effects of prenatal BPA exposure on fetal growth and survival parameters. Early studies provided conflicting results. Intraperitoneal injection of BPA to mated female rats at dose levels of 85–125 mg/kg during gestational days 1–15 resulted in decreased fetal survival, and decreased fetal body weight among surviving fetuses [64]. In contrast, a later study, using oral administration of BPA to pregnant rats at dose levels of 160, 320 and 640 mg/kg during gestational days 6–15, did not induce either fetal toxicity or morphological alterations [65]. Significant reduction in the body weight of both male and female mice following prenatal or post-weaning exposure to BPA in varying doses have been reported in a few studies [64, 66, 67]. Administration of BPA at 300 mg/kg during the entire gestational period in Sprague-Dawley rats reduced maternal body weight and weight gain, and decreased food intake and reduced body weight of male fetuses. At 100 mg/kg or more, significant toxic effects were induced. These included increased postimplantation loss, reduced litter size and reduced fetal body weight [68]. Exposure of sheep to BPA from days 30–90 of gestation that resulted in maternal levels of 30–50 ng/mL, which approximates two times the highest level found in human maternal circulation resulted in low birth-weight offspring [69].

Reproductive tract anomalies following prenatal or neonatal BPA exposure provide another potential means to adverse perinatal outcomes. Several recent studies have reported that fetal and neonatal exposure to BPA may impact fertility, age of reproductive senescence, and onset of disease later in life [70]. Changes in uterine, ovarian, and vaginal weights of pregnant mice or their offspring following developmental exposure to BPA have been variously reported [44, 71–73], although the doses at which these effects occur vary across studies. BPA exposure has been found to be associated with timing of puberty [74]. Exposure of pregnant mice to 20 μg/kg BPA from gestational days 11 through 17 has been shown to induce both vaginal opening and first vaginal estrus at a significantly earlier age in female offspring, as well as increased body weight at the time of weaning [74, 75]. Yet another reproductive parameter in females that appears to be adversely affected by BPA exposure is mammary gland development and activity [15, 44, 54]. Alterations in FSH levels in female animals following fetal or pubertal exposure and delayed and sustained hyperprolactinemia following fetal or pubertal exposure have also been reported [75, 76]. Human studies have shown that BPA is elevated among women with ovarian dysfunction, including women with polycystic ovaries [77, 78] and endometrial hyperplasia [79].

Other findings of relevance to pregnancy maintenance include the effects of BPA on metabolic parameters. Long-term exposure to BPA significantly increased insulin secretion from rat pancreatic islets [47]. The exposure of adult mice to a single low dose (10 μg/kg) of either estradiol or BPA induced insulin resistance and compensatory increase in plasma insulin. Chronic exposures to BPA induced an increase in pancreatic beta-cell insulin content in an estrogen-receptor-dependent manner [45, 47]. There is some evidence that BPA exposure may result in lowering of total serum cholesterol [80]. Emerging evidence suggests that weight homeostasis may also be altered by BPA. A small study found alterations in leptin and ghrelin levels in field voles, following exposure to 10, 50, or 250 mg/g of BPA injected subcutaneously [81]. An *in vitro* study using cultured mouse cells reports that exposure to BPA in combination with insulin triggers the conversion of cells to

adipocytes and increases the quantity of stored fat [82]. These findings suggest that BPA can compromise the metabolic environment during pregnancy, pointing to another possible pathway to low birth weight offspring among exposed mothers.

Extending current research on BPA and perinatal health

Taken together, and in conjunction with the fact that measurable levels of BPA are present in pregnant females and in fetuses, this body of work underlines the need to extend research on the possible effects of BPA on fetal growth and survival. Such an extension would be facilitated considerably by recognizing the limitations of the existing predominant research paradigm, which is rooted in toxicology. Apart from exposure studies, the vast majority of research on BPA has tended to rely on animal (predominantly rodent) models, a mainstay of toxicology studies. Limitations of existing studies are increasingly becoming evident. Several of the studies showing negative results rely on a species of rat that appears to be insensitive to estrogen [13–15]. Species-specific effects narrow the range of useful evidence, and may even be misleading. Second, a growing body of evidence suggests that BPA may have non-monotonic effects in some cases, inducing different effects at low and high doses [13, 15]. Another inadvertent consequence of experimental studies is an emphasis on intake, rather than circulating concentrations of BPA. In light of the fact that BPA may bioaccumulate in fetuses and pregnant women, this may not be a realistic measure of the exposure.

Epidemiological observation studies with human beings represent an attractive alternative paradigm for studying the effects of BPA. It is evident that there is a paucity of research with human populations. For example, recent report on BPA released by the National Toxicology Program [42] concluded that although there is clear evidence that exposure to BPA can cause fetal death, and reduced birth weight and growth during infancy, these effects occur only at high levels of exposure, and therefore, risks are negligible. However, all but two of the studies reviewed were based on laboratory animals. It is possible that the chronic BPA exposure experienced by humans may reflect a qualitatively different exposure to that seen in controlled laboratory conditions. Biomonitoring studies on BPA exposure confirm that exposure measurement on large populations is feasible. Combining exposure measurement with measurement of specific health outcomes should be equally feasible. Human exposure data, which relies on epidemiological observations, already suggests substantial maternal and fetal exposure. Understanding the distribution of exposure across geographies, race and social classes will give us insight into who is at risk of excess exposure. The goal of epidemiological investigation would be to establish that there are systematic variations in exposure to BPA and specific health outcomes across sub-populations. We propose here that well-known, but poorly understood social disparities in a variety of specific health outcomes may be related to differences in exposure to BPA or other xenoestrogens. Below, we elaborate this hypothesis with specific reference to the well-known African-American disadvanage in perinatal outcomes, drawing upon epidemiological observations that are consistent with this hypothesis.

Are racial disparities in perinatal outcomes in the United States related to differential exposure to BPA?

African-American and/or other typically poor communities are disproportionately exposed to environmental pollutants through residential segregation, lifestyles and other cultural/structural factors, including widespread poverty [3, 83, 84]. Poor and racial/ethnic minorities tend to live in the most disadvantaged communities and hazardous environments. Mediators of the relationship between minority status and exposure to environmental hazards include sources of pollution, illegal dumping, failure to enforce environmental regulations, and failure to respond to community complaints [83]. In this context, a growing body of evidence suggests that African Americans may be more likely to be at higher risk of exposure to EDCs. As noted, BPA has been shown to account for most estrogenic activity that leaches from landfills into the surrounding ecosystem [30] i.e. communities in which African Americans are more likely than Whites to reside [85]. Furthermore, communities with higher proportions of African Americans have a disproportionate number of fast food restaurants compared to communities with lower proportions of African Americans, and fewer sources of fresh food [86, 87]. Studies of the spatial distribution of fast food restaurants and supermarkets found that all African American areas, *regardless of income*, were less likely to have access to healthy food options than predominantly White higher-income communities [87]. Specifically, stores in predominantly black areas have been found to carry less fresh produce and higher proportions of canned foods than stores in predominantly white areas [88]. African American households are overrepresented among the food insecure; in 2005, 23. 6% of African American households were food insecure, compared with only 8. 6% of White households [89]. Given these high rates of food insecurity, African American households are more likely to rely on cheap, energy dense fast food [90] and on food banks, which typically distribute canned foods [91]; as noted, a known and significant source of BPA exposure. Food pantry use is more than twice as high among blacks than among non-Hispanic whites (7.8% vs. 2.7%) [89]. It bears mentioning here that other poor minority communities are likely subject to similar risks at comparable levels.

Other factors in the African-American community may enhance vulnerability to environmental pollutants. Animal studies suggest that stress can influence response to environmental pollutants [4] and increase absorption of toxicants [92]. In the African-American community, the stresses of poverty, racism and other aspects of minority status may enhance vulnerability to exogenous EDC exposures [93, 94]. However, a recent population-representative survey found no differences in urinary concentrations of BPA between African Americans and Whites, although levels were found to be higher for participants in lower income households (32). In this context, it is interesting to note that African Americans are overrepresented among low-income households. It remains to be determined if the same trend holds for pregnant African Americans and Whites. Future research should also focus on determining if sensitivity to BPA differs between races and if the cumulative EDC burden or interaction of BPA with other EDCs differ by race.

Obesity is another confounding factor that can influence susceptibility to EDC exposure. Support for increased risk of BPA exposure with obesity comes from studies that document

higher levels of BPA in obese women [79]. A recent study using data from NHANES 2003–2004 survey reported mean urinary BPA concentrations of 3.91 ng/ml (95% CI 3.34–4.48) in participant with BMI of 18.5–24.9 and 6.93 ng/mL (95% CI 4.39–9.47) in those with BMI ≥35 (obese II category) [59]. Animal studies also suggest BPA accumulates in body fat [39]. A recent study also found higher BPA metabolites (inactive) in 6–9 year old girls with BMI <85th percentile compared to those with BMI >85th percentile [3]. Assuming environmental exposure levels are similar between these BMI groups, this would imply the reverse in circulation, namely high levels of parent (active) BPA levels in girls with BMI>85th percentile. Fat stores of BPA may be mobilized during pregnancy and lactation, pass the placental barrier, and increase the level of BPA exposure in fetuses of obese women. Obesity is highly prevalent among African American women; a recent analysis of NHANES data found 50. 3% of African American women aged 20–39 were obese, compared with 23.8% of White women [95]. Although obesity per se has a positive effect on birth weight [96], it should be recognized that obesity is also associated with many negative pregnancy outcomes including intrauterine fetal death, miscarriages, fetal anomalies, preeclampsia, gestational diabetes, cesarean delivery, and lower breastfeeding frequency [97]. A 10-year population based study in Norway found that overweight or obesity concurrent with IUGR increased risk of stillbirth [98].

Racial disparities in IUGR and low birth weight

Fetal growth restriction is the second leading cause of perinatal morbidity and mortality, followed only by prematurity. The health and social consequences of being born too small are profound. Infants who experience low birth weight/IUGR have higher perinatal morbidity and mortality. The risk of neonatal death for infants weighing 2,000–2,499 grams at birth is 4 times higher than for infants weighing 2,500–2,999 grams and 10 times higher than for infants weighing 3,000–3,499 grams. For surviving neonates, the risks of IUGR and low birth weight extend into the post-neonatal period, and include greater risk of mortality, and increased risk for a host of adverse health and developmental outcomes, including lower mean intelligence quotient scores and a higher instance of cognitive impairment [94]. Intrauterine growth restriction and low birth weight are also associated with increased risk of costly and disabling adult onset diseases, including cardiovascular disease, hypertension, type II diabetes, and obesity [99–101]. Importantly, a growing body of evidence suggests that the effects of IUGR/low birth weight are intergenerational, due to shared environmental factors, genetic factors, or both [94]. The Washington State Intergenerational Study of Birth Outcomes found that low maternal birth weight conferred a two-fold increase in risk of low birth weight of offspring and a 30% greater risk of preterm delivery among African Americans, Hispanics, and Whites [102].

Efforts at prevention of low birth weight are hampered by limited knowledge about causes. One of the more important clues about the causes of low birth weight comes from the observation that there are large ethnic group differences in these outcomes [102]. Infant mortality and low birth weight rates have been at least twice as high for African American women compared with White and Asian American women in the United States for decades [103]. In 2004, the infant mortality rate of 13.7 deaths per 1000 live births for African American infants exceeded the 5.7 rate for Whites [104], and the low birth weight rate for

African American infants amongst singleton births was 11.7 percent compared with 5.2 percent for Whites [105]. The 1985 Report of the Secretary's Task Force on Black and Minority Health noted the persistent disparity in infant mortality and low birth weight between African Americans compared with Mexican Americans and Whites, despite similar sociodemographic risk factors [106].

These observational data are consistent with a hypothesis that increased incidence of IUGR and low birthweight among African-Americans is caused, at least partially, by increased exposure to endocrine disrupting chemicals such as BPA. These effects of EDC exposure are likely exacerbated by the higher levels of stress and obesity among African-Americans.

Conclusion

It is our hope that this review will stimulate further research on the potential contribution of BPA to racial/ethnic disparities in pregnancy outcome, one of the most intractable public health problems in the United States. The risk factors identified by previous research have often been global, not readily modified, or have otherwise provided little direction for intervention. However, exposure to BPA is a potentially modifiable risk factor with many implications for prevention. Far-reaching political and socioeconomic forces, discrimination, and industrialization have segregated African Americans into communities characterized by high levels of poverty and material deprivation [84, 85]. If BPA exposure is shown to be a contributor to disparities in birth outcomes, this implies the need to intervene in those macro level systems -governmental and regulatory, business, industry, and community - that shape African American women's disproportionate exposure or enhanced susceptibility to BPA. As Gee and Payne-Sturges pointed out [84], environmental health disparities may require policies and interventions aimed at eliminating environmental toxins and developing community resources. Although micro-level approaches such as biochemical interventions or health education are useful, they require major resources to affect outcomes at the population level, and are less efficient because they must be reapplied to each successive birth cohort. However, the benefits of many endocrine disrupting compounds for industry and agriculture ensure their continued use [107]. The contentiousness of the current debate surrounding BPA [108] indicates that macro-level interventions such as requiring alternative manufacturing processes for certain human-use products will require developing effective partnerships between scientists, policymakers, business and community leaders [108]. In the long run, this may be the most cost-effective prevention strategy.

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