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Hormonal Injustice Environmental Toxicants as Drivers of Endocrine Health Disparities

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INTRODUCTION

A wide spectrum of endocrine disorders are characterized by health disparities, including metabolic,[1] thyroid,[2] and reproductive disorders.[3] Although metabolic disease prevalence has increased across sociodemographic groups, minority racial/ethnic groups carry a greater disease burden.[4] This difference is exacerbated by educational and socioeconomic disparities,[5] reduced access to resources, and systemic oppression.[6] Evidence indicates that vulnerable populations are disproportionately exposed to various environmental toxicants, including multiple endocrine-disrupting chemicals (EDCs).[7] An analysis of EDC exposures and disease burden across racial/ethnic groups noted higher exposures among non-Hispanic Blacks and Mexican Americans relative to non-Hispanic Whites.[8] Furthermore, per capita EDC-associated health care costs are higher among people of color. Of $340 billion in

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DISCLOSURE

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US health care costs associated with EDCs, Black and Mexican American communities accounted for 31.4% of total expenditures while only comprising 26.1% of the population.[8] The disproportionate burden of hazardous chemical exposures borne by vulnerable communities is an environmental justice crisis with multigenerational consequences.[9] This review highlights key data linking EDC exposures to endocrine health disparities.

HEALTH DISPARITIES IN ENDOCRINOLOGY

Metabolic Disorders

Projected to afflict 783 million adults by 2045, global diabetes prevalence has increased dramatically over recent decades.[10] In the United States alone, 37.3 million people have diabetes; however, the disease burden is not uniform.[11] Diabetes prevalence is markedly lower among non-Hispanic Whites compared with all other minority racial/ethnic groups. Critically, racial/ethnic minorities also suffer from higher diabetes-associated morbidity and mortality.[12] Marked diabetes disparities are also noted by socioeconomic status (SES).[13]

In 2017, US adult obesity prevalence reached an astonishing 41.9%.[14] Prevalence was highest among Hispanics/Latinos and non-Hispanic Blacks. Across all groups, prevalence increases with lower educational attainment. Although associations between obesity rates and SES remain somewhat murky, SES seems to predict body fat distribution, with those of lower SES accumulating fat more centrally, a characteristic associated with various metabolic comorbidities.[13] Drivers of obesity disparities include differences in food availability arising from variations in the retail food environment that concentrate poor-quality resources in more disadvantaged areas,[15] and neighborhood insecurity and a lack of green spaces[16]; more recently, chemical exposures have been implicated.[17]

Nonalcoholic fatty liver disease (NAFLD) is a metabolic disorder characterized by alcohol-independent hepatic fat accumulation associated with inflammation, fibrosis, cirrhosis, liver failure, and hepatocellular carcinoma. By 2030, approximately 100.9 million US adults are projected to be afflicted by NAFLD.[18] Importantly, NAFLD prevalence and complications exhibit notable disparities, with the greatest disease burden among Hispanics/Latinos.[19] Although men are disproportionately affected, evidence indicates that women may have higher rates of NAFLD-related complications.[20] NAFLD disparities are also noted based on SES, with low-income populations experiencing greater prevalence, disease progression, and complications.[21]

Thyroid Disorders

Globally, 200 million people are affected by thyroid diseases,[22] with incidence increasing with age, iodine deficiency, and radiation exposure.[23] Thyroid nodules are more prevalent in women than men, with White individuals disproportionately affected.[24] Importantly, epidemiologic evidence links thyroid nodules with the metabolic syndrome. While this association is observed in both sexes, women are at greater risk of developing thyroid nodules than men with the same metabolic disturbances.[25] Although diagnostic bias may confound the associations, thyroid cancer is more prevalent among Whites, with socioeconomic factors predicting survival.[25,26] Despite this, evidence suggests that Black

patients have lower survival rates regardless of screening or SES.[27] In addition to disparities in thyroid cancer, Blacks are more likely to develop thyrotoxicosis than Whites,[28] with disparities also present for hypothyroidism. Among those with subclinical hypothyroidism and comorbid congestive heart failure, Blacks have higher mortality rates than non-Blacks.[29]

Reproductive Disorders

Racial/ethnic and socioeconomic disparities characterize women's health from menarche to menopause. For example, Hispanic/Latina and Black individuals reach menarche at younger ages than their White counterparts.[30] Furthermore, early onset of menarche is associated with fibroid tumors and increased risk for breast and ovarian cancer.[31] Among women with fibroids, racial minorities with lower SES had greater fibroid severity and decreased health-related quality of life.[32] In addition to earlier menarche, Black women reach menopause earlier than White women, and they experience longer menopausal transitions.[33] Polycystic ovarian syndrome (PCOS) is an endocrine disorder characterized by ovarian cysts, menstrual irregularities, hyperandrogenism, infertility, and metabolic dysfunction. Women of lower SES are at greater risk of developing PCOS, and Black women with PCOS are more likely to develop metabolic complications.[34]

Among men, reproductive disparities are also notable. Compared with Whites, Black individuals were significantly more likely to have lower sperm volume and concentration with fewer motile sperm.[35] These issues are likely exacerbated by the fact that Black populations are disproportionately burdened by metabolic disorders, which also negatively affect spermatogenic activity and semen quality.[36]

Adrenal Disorders

Disruptions in adrenal function adversely affect health and development in myriad ways. Compared with White men, Black men are more likely to exhibit dysregulated cortisol secretion patterns, which are linked to multiple adverse health outcomes.[37] Furthermore, conditions of excess cortisol production are more prevalent among women.[38] Primary hyperaldosteronism, the most common cause of secondary hypertension, is more common in Black populations.[39] Androgen excess is linked to PCOS and other reproductive disorders in men and women, with prevalence varying by race/ethnicity and SES. Collectively, the available evidence points to race/ethnicity-, gender-, and income-based disparities across a spectrum of endocrine disorders; achieving health equity requires identifying and addressing the drivers of these differences.

ENDOCRINE-DISRUPTING CHEMICAL EXPOSURE DISPARITIES

Although various social and structural determinants of health contribute to disparities in endocrine health, less appreciated are the contributions of differential exposures to EDCs. The Endocrine Society defines EDCs as "an exogenous chemical, or mixture of chemicals, that can interfere with any aspect of hormone action." More than 500 EDCs have been identified, including polychlorinated biphenyls (PCBs), bisphenols, phthalates, organochlorine (OC) pesticides, per/polyfluoroalkyl substances (PFASs), metals/metalloids, flame retardants, and air pollutants, among others. Critically, exposure to these chemicals

is not uniform across populations. Rather certain groups are disproportionately exposed, including various minority racial/ethnic groups, with noted gender-based disparities.[8,40,41] Those with low incomes or of lower SES are also disproportionately exposed.[42] These exposure disparities are driven by multiple social factors, including dietary patterns, consumer product usage, living conditions, labor practices, and geography,[41] all of which are driven by current and historical policies influenced by racist and classist power structures. Thus, environmental injustice drives exposure disparities. The following highlights key literature regarding notable EDC exposure disparities and adverse endocrine health effects (Fig. 1, Table 1).

Polychlorinated Biphenyls

PCBs are a class of persistent organic pollutants (POPs) historically used for a multitude of industrial applications. Although PCB production was banned in the US in 1979, they persist in the environment because of their long half-lives and continued leaching from older consumer products and industrial waste facilities. Their hydrophobicity and chemical stability results in accumulation in the fatty tissue of animals over time through consumption of contaminated food or prey, resulting in biomagnification at higher trophic levels of the food system. Consequently, human exposure continues through air, soil, food, and water sources. Following exposure, PCBs are associated with multiple adverse endocrine health effects. PCBs mimic the structure of thyroxine and interfere with thyroid hormone homeostasis.[43] PCBs are also associated with metabolic dysfunction in human studies.[44,45] Specifically, a 2014 meta-analysis revealed that individuals exposed to the highest levels of PCBs had a two-fold higher risk of diabetes.[46] In cellular and animal models, PCBs alter insulin release and insulin sensitivity, among other effects, resulting in a diabetic phenotype.[47] Both epidemiologic and preclinical data also link PCBs with NAFLD.[48]

PCB exposure is not homogenous across racial/ethnic groups. Among US adults, Black individuals have significantly higher blood PCB levels than all other racial/ethnic groups, whereas among adolescents and young adults, Pacific Islanders and Native Americans have the highest levels.[49] This disparity may be particularly important in the United States where PCB levels are generally higher in US-born individuals than immigrants.[50] It is postulated that these PCB exposure patterns are primarily driven by dietary consumption and past exposures, but more evidence is needed.[49] Lastly, occupation and housing are associated with higher PCB levels, and there are noted income-based disparities as well.[51,52]

Phthalates

Phthalates are a family of nonpersistent phthalic acid esters with short half-lives but numerous exposure sources. Often classified by their molecular weight, low-molecular-weight phthalates are commonly used in personal care products and solvents, whereas high-molecular-weight phthalates serve as plasticizers in medical equipment and food packaging.[53] These compounds leach from products, leading to exposure via ingestion, dermal contact, or inhalation.

Although phthalates are nonpersistent pollutants, continuous and repeated exposure can promote adverse health effects. In women, phthalates are associated with ovarian and uterine

dysfunction, contributing to the development of PCOS, uterine fibroids, and endometriosis through direct estrogenic effects.[54] During pregnancy, maternal phthalate exposure is associated with placental disruption, pregnancy loss, and greater risk of preterm birth.[55] Phthalates also disrupt male sex steroid signaling, including antagonism of androgen receptor signaling. Consequently, phthalates are linked to hypospadias and male infertility.[56] Moreover, via interactions with various nuclear receptors, phthalates promote diabetogenic and obesogenic effects.[43,44] Indeed, numerous epidemiologic studies associate phthalates and phthalate metabolites with increased diabetes risk.[57] Lastly, phthalates disrupt enzymes involved in adrenal hormone production.[58]

Women are disproportionately expose to phthalates, likely a consequence of their greater use of personal care products. Among women, Black and Mexican American women have higher levels of almost all phthalates and phthalate metabolites.[59] This may be the case because beauty products directly marketed to women of color contain higher levels of phthalates and other chemicals.[60] Marketing strategies perpetuate European beauty standards among minority populations, leading to disproportionate use of skin lighteners, hair relaxers/straighteners, and odor reduction products known to contain EDCs.[60] Similarly, inexpensive building materials, fast food, and consumer products are more likely to contain a variety of EDCs, including phthalates, placing those of low SES at greater risk of exposure.[61,62]

Bisphenols

Bisphenols, such as bisphenol A (BPA), are found in numerous consumer products, including the linings of food containers, plastics, thermal paper, and preservatives.[63] Like phthalates, bisphenols are nonpersistent pollutants; however, humans are continuously exposed. BPA is a well-studied EDC that disrupts multiple signaling cascades, including estrogen receptors, growth factor receptors, and other pathways implicated in diabetes, cardiovascular disease, and cancer.[64] As a xenoestrogen, BPA is linked to multiple reproductive disorders, including PCOS, cryptorchidism, and male infertility.[56,65] Because of BPA's disruption of metabolic pathways, human and preclinical studies have linked BPA exposure with increased metabolic disease risk, including obesity and diabetes.[44,66] The latter findings are supported by robust in vivo and in vitro data demonstrating BPA's capacity to perturb metabolic homeostasis.[64] Additionally, BPA is implicated in adrenal dysfunction through targeting of enzymes involved in the synthesis of glucocorticoids, mineralocorticoids, and androgens.[58]

Across the general population, women and those with lower incomes have greater BPA exposure.[67] Women of color, especially Black women in the United States, have higher BPA levels.[68,69] Furthermore, low-income families and those that received emergency food assistance have higher levels of BPA independent of other sociodemographic factors.[70,71]

Organochlorine Pesticides

Historically used throughout the world, OC pesticides are POPs that were banned in the United States in the 1970s. Despite their toxicity, several OC pesticides are still used in developing countries, including dichlorodiphenyltrichloroethane (DDT),

hexachlorocyclohexane, aldrin, and dieldrin.[72] Because of their persistence and biomagnification, OC pesticides continue to contaminate soil, water, air, and fatty meat and fish.[73] US biomonitoring data indicate near universal exposure to OC pesticides, such as DDT and its metabolites.[74]

Many studies have linked OC pesticides with endocrine dysfunction, including disruptions in thyroid hormone function and an increased risk of thyroid cancer.[75–77] OC pesticides are also associated with other endocrine-related cancers, including prostate and breast.[78,79] Levels of multiple OC pesticides are associated with diabetes and obesity.[46,80–82] Lastly, because of its lipophilicity, DDT accumulates in adrenal glands.[58]

Prospective studies show that OC pesticide levels are higher among Black and Asian women compared with White women.[83,84] Furthermore, recently immigrated Hispanics/Latinos have higher levels of OC pesticides than longer-term US residents of the same race/ethnicity, suggesting important international exposure disparities.[7,50] Importantly, OC pesticide exposures vary based on occupation and income, with agricultural work an important exposure source.[85]

Air Pollutants

Air pollution includes atmospheric contamination by a wide array of chemical, biologic, and physical agents. The most common air pollutants include fine particulate matter less than 10 μm and less than 2.5 μm in size (PM_{10} and $PM_{2.5}$, respectively), ozone, and nitrogen dioxide (NO_2), among others. Although outdoor air pollution is more widely appreciated, indoor air pollution is increasingly recognized to adversely impact health. Many studies have linked various air pollutants to endocrine disorders. Indeed, elevated PM and NO_2 exposures are associated with dysregulated glucose homeostasis and increased diabetes risk.[86,87] Additionally, air pollution is implicated in the progression of childhood and adult obesity.[88,89] This metabolic dysfunction is likely further exacerbated by associations between PM and other conditions, such as chronic liver disease.[90] Lastly, although more evidence is needed, air pollution has been linked to male infertility, including reduced sperm motility and impaired gametogenesis.[91,92]

Critically, air quality varies geographically. Minority communities are disproportionately situated in areas with higher levels of multiple air pollutants, with exposures amplified by low-income status.[93,94] Additionally, minority communities are more likely to live in geographic areas with higher levels of traffic-related air pollution, which contributes significantly to NO_2 exposure and more toxic forms of PM.[95,96] Black and Hispanic/Latino communities have been noted to have some of the highest air pollution exposures.[97] With the implementation of federal environmental policies, absolute air quality in the US has improved since 1990; however, racial/ethnic exposure disparities persist under the influence of income inequality and historical discriminatory policies.[7,98] Moreover, lower income status is consistently shown to predict exposure to higher air pollution levels independent of race, ethnicity, or sex.[99]

Per- and Polyfluoroalkyl Substances

PFASs encompass a diverse family of synthetic chemicals widely used in manufacturing and consumer products, including food packaging, cookware, and outerwear among others. Considered “forever chemicals,” PFASs are another class of POPs with long half-lives that contaminate food, water, soil, and air. Various PFASs are associated with multiple adverse endocrine effects. These include links to diabetes, obesity, NAFLD, and reproductive dysfunction.[100,101]

Importantly, PFAS exposures vary across populations. Black and Hispanic/Latino populations have been noted to have significantly higher PFAS levels compared with Whites.[102,103] Although some studies suggest that PFAS levels have begun to decline, this is not true across all racial/ethnic groups. For example, individuals of Chinese descent were found to have almost no decline in PFAS levels between 1999 and 2011.[104] Beyond race/ethnicity, lower SES and food insecurity are also linked to PFAS exposures, potentially as a consequence of food packaging and chemicals in less expensive personal care products.[103,105] A recent report by the National Academies recommends PFAS testing in those likely to have a history of elevated exposures, including those based on occupation and place of residence.[106] Such expanded testing may further illuminate PFAS exposure disparities.

Toxic Metals/Metalloids

Toxic metal/metalloid exposures include organic and inorganic forms of several elements, including arsenic, cadmium, and lead. These elements leach naturally into groundwater via geochemical processes; however, environmental exposures are enhanced via historical and current anthropogenic activities. Climate change also increases exposure to toxic metals/metalloids. Arsenic exposure threatens the health of millions of people worldwide, with exposure occurring through contaminated food and water, tobacco use, and industrial activities among other processes.[107] Cadmium contaminates food, water, and tobacco; it is also found in plastics, dyes, and fertilizers.[108] In addition to occupational contact, lead exposure occurs through lead-containing products, including some cosmetics and jewelry, and via inhalation and ingestion from contaminated foods, lead plumbing, lead-based paints, and the largely historical use of leaded gasoline.[109]

Toxic metals/metalloids are linked to multiple acute and chronic adverse health effects through a variety of mechanisms, including oxidative stress, inflammation, and endocrine disruption.[110] Despite similar mechanisms of toxicity, metals/metalloids may uniquely target certain organ systems. With respect to metabolic disease, arsenic and cadmium are noted to disrupt metabolic physiology, promoting the development of insulin resistance and diabetes, with somewhat conflicting evidence regarding their obesogenic properties.[111] Both alone and in combination with other toxic metals, lead is associated with diabetes.[112] These metals are also implicated in other endocrine conditions including thyroid disruption, infertility, and other reproductive disorders.[113–115]

There are important racial disparities in toxic metal/metalloid exposures. For example, lead is a major public health threat in the US, and there is extensive evidence that Black

and Hispanic/Latinx families are more highly exposed, especially those who live in low-income areas.[116] Additionally, in utero and postnatal lead biomarkers are higher in Black children.[117] Regions with higher groundwater arsenic levels tend to be home to greater minority populations, especially Hispanics/Latinxs.[118] Exposures also vary across countries as more recently immigrated individuals have higher toxic metal/metalloid levels than individuals who have lived in the US for longer.[119] Lastly, regardless of race/ethnicity, lower SES is associated with increased risk of heavy metal exposures, including lead, cadmium, and arsenic.[120]

Brominated Flame Retardants

Added to consumer products, such as furniture, insulation, plastics, and electronics, flame retardants are synthetic chemicals used to prevent the initiation or spread of fires.[121] The most widely used brominated flame retardants (BFRs) are polybrominated diphenyl ethers (PBDEs) and polybrominated biphenyls. BFRs are persistent pollutants that accumulate in the environment and in human tissue, with contaminated dust the major source of BFR exposures, especially in North America.[122] BFRs are associated with various adverse endocrine health effects, including diabetes, obesity, altered thyroid function, cancer, and reproductive dysfunction.[123]

Studies have shown that lower income individuals and low-income housing residents had higher exposures to PBDEs, especially children.[124,125] There are also significant gender disparities in BFR exposures. Among young adults in China, women had PBDE levels three-times higher than those of men.[122] Racial/ethnic disparities in BFR exposures are also noted, with Black and Hispanic/Latinx populations having the highest PBDE exposures.[126]

SUMMARY

Multiple social and structural determinants of health undoubtedly contribute to the marked racial/ethnic-, gender-, and socioeconomic-based disparities in endocrine health; however, the contribution of environmental injustice is vastly underappreciated. Indeed, those groups disproportionately burdened by endocrine disorders are often exposed to higher levels of various EDCs, including PCBs, phthalates, bisphenols, OC pesticides, air pollutants, PFASs, toxic metals/metalloids, and BFRs. Furthermore, the contribution of disparate exposures to health disparities is likely underestimated because of a paucity of data examining the adverse effects of combined EDC exposures. As such, health equity requires interventions to address environmental injustice. Such approaches must include a complementary array of individual action and public policy,[127] the latter of which is inadequately used.[128] Based on the Developmental Origins of Health and Disease hypothesis that posits long-term health risks imposed by stressors during sensitive developmental windows (including EDC exposures), it is essential that interventions be targeted to those most vulnerable, including pregnant mothers and infants, among others. However, it is also critical to recognize that EDC exposures and their disproportionate burden on low income and communities of color is a systemic problem for which individual action is insufficient. Rather, policy interventions are required, including robust efforts to identify EDCs before they enter commerce, eliminate EDCs already in use, mitigate contaminated sites, and develop socially just policies that

end the discriminatory siting of polluting industries. Vigorously pursued, such efforts have the potential to improve endocrine health equity while reducing the burden of disease for everyone.

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KEY POINTS

- Vulnerable populations carry a disproportionate burden of multiple endocrine disorders and their associated comorbidities and medical costs.
- Racial/ethnic minorities, those with low incomes, and other disadvantaged groups are disproportionately exposed to various endocrine-disrupting chemicals (EDCs) linked to adverse endocrine health effects.
- Achieving endocrine health equity requires comprehensive efforts to eliminate environmental injustice.

CLINICS CARE POINTS

- Health care providers should recognize that EDC exposures may amplify endocrine disease risk and incorporate occupational and environmental history-taking into their practices to identify modifiable patient-specific environmental risk factors.
- Patients should be offered strategies to reduce their exposures to EDCs (Table 2).
- Although not yet broadly endorsed, clinicians should follow recommendations of professional organizations and consider measuring EDCs in patients likely to have a history of high-level exposures, as recently recommended for PFASs.
- Health care providers should advocate for incorporation of environmental health into clinical practice guidelines, public policies that promote environmental health, and improved environmental health literacy among the health care workforce and patients.

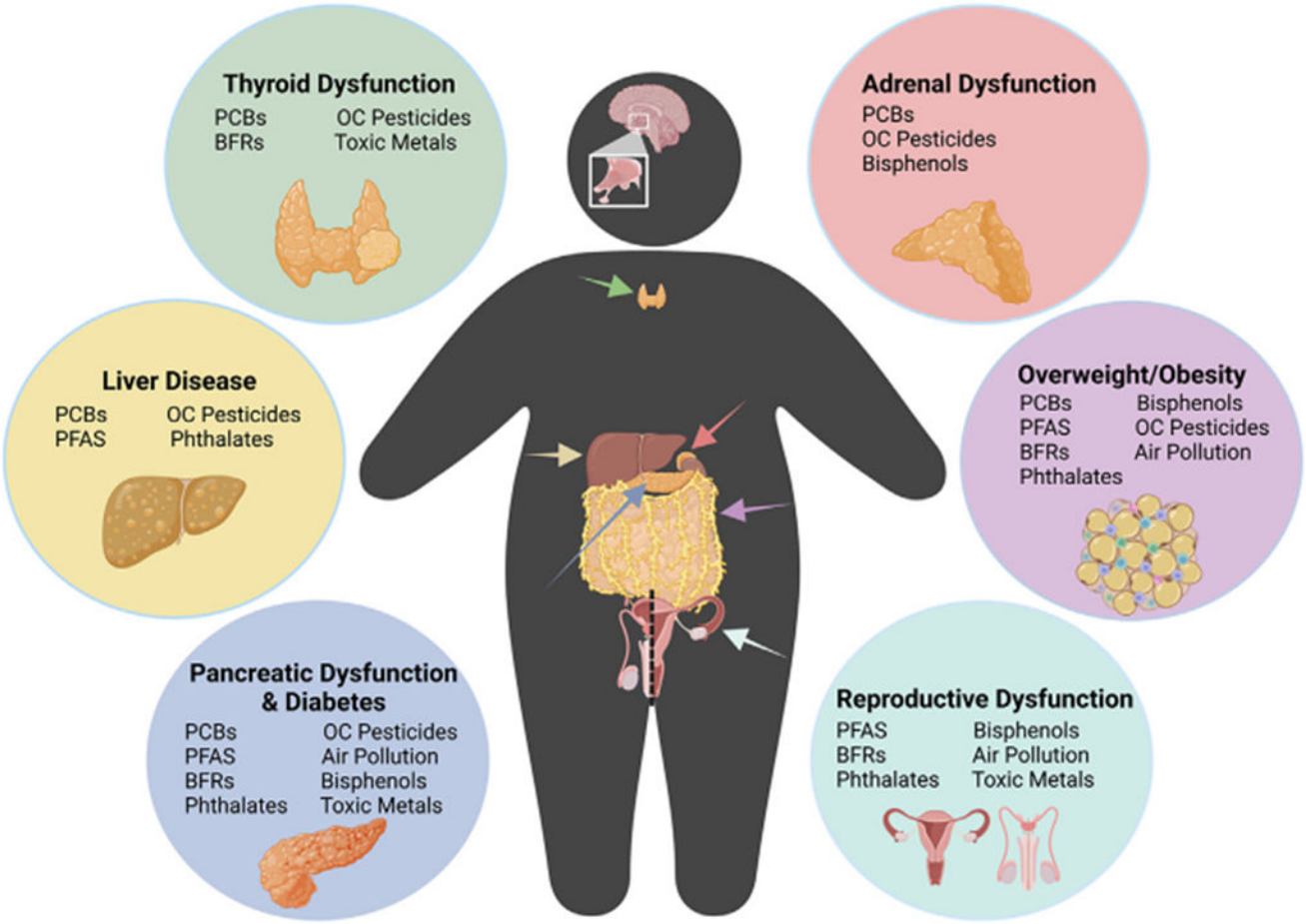

Fig. 1.

Summary of endocrine-disrupting chemicals with known exposure disparities and their effects on the endocrine system. These manifestations may be direct effects or downstream consequences of hypothalamic and/or pituitary dysfunction. BFRs, brominated flame retardants; OC Pesticides, organochlorine pesticides; PCBs, polychlorinated biphenyls; PFAS, per/polyfluoroalkyl substances. (*Created with* BioRender.com.)

Table 1

Endocrine-disrupting chemical exposure disparities

Endocrine Disruptor	More Highly Exposed Groups	Endocrine Impacts
Polychlorinated biphenyls	Non-Hispanic Black populations[49] Pacific Islanders and Native Americans[49] US-born individuals[50] Immigrant women[119] Low income[51]	Thyroid dysfunction[43] Obesity[44] Diabetes[45,47] Adrenal dysfunction[10,58] NAFLD[48]
Phthalates	Women[60] Non-Hispanic Black populations[59] Mexican Americans[59] Low income[61]	Male infertility[56] Diabetes[43,44] Obesity[129,130]
Bisphenols	Women[67] Low income[67,70,71] Non-Hispanic Black populations[68,69]	Polycystic ovarian syndrome[65] Male infertility[56] Obesity[44] Diabetes[66] Adrenal dysfunction[58]
Organochlorine pesticides	Non-Hispanic Black populations[83] Asian populations[84] Women[84] Immigrants[7,50] Low income[85]	Thyroid dysfunction[75–77] Diabetes[46,80,81] Obesity[81,82] Adrenal dysfunction[58] NAFLD[131]
Air pollution	Non-Hispanic Black populations[93,94] Hispanic/Latinx Americans[97] Low income[93,94,98,99]	Diabetes[86,87] Obesity[88,89] Infertility[91,92]
Per- and polyfluoroalkyl substances	Women[103] Chinese populations[104] Black populations[102] Hispanic/Latinx populations[102] Low income[103,105]	Diabetes[100] Obesity[100] Reproductive dysfunction[100] NAFLD[101]
Toxic metals	Black populations[116,117] Hispanic/Latinx Americans[116,118] Low income[116,120] Immigrants[119]	Diabetes[111,132] Reproductive dysfunction[113] Infertility[114] Thyroid dysfunction[115]
Brominated flame retardants	Low income[124,125] Women[122] Non-Hispanic Black populations[126] Hispanic/Latinx populations[126]	Diabetes[123] Obesity[123] Thyroid dysfunction[123] Reproductive dysfunction[123]

Table 2

Proposed strategies to reduce EDC exposures

Exposure Source	Interventions
Personal and home care	1. Wash hands regularly using fragrance- and antibiotic-free soaps. 2. Regularly clean floors and remove dust using a damp cloth. 3. Eliminate or drastically reduce use of household chemicals, including cleaning supplies, pesticides, and solvents. 4. Choose electrical appliances and lawncare equipment. 5. Forbid smoking indoors. 6. Do not burn trash or yard waste. 7. Read product labels and avoid items containing parabens, bisphenols, and phthalates. 8. Minimize use of products packaged or stored in plastics. 9. Avoid cosmetics with synthetic fragrances, phthalates, or toxic metals. Choose instead those labeled as “no synthetic fragrance,” “scented with essential oils,” or “phthalate-free.” 10. Minimize handling of receipts.
Food and beverages	1. Prioritize eating locally grown fresh or frozen foods. 2. Ensure adequate intake of calcium, iron, and iodine and other essential vitamins and minerals. 3. Consume a diversified diet that is high in fiber. 4. Consult local guidance regarding safe sport fish consumption. 5. Trim fat from meat and skin from fish. Cook meat and fish on a rack to allow fat to drain. 6. Avoid canned, processed, and fast foods. 7. Store food in glass, stainless steel, or porcelain containers. 8. Avoid heating foods in plastic containers. 9. Consider testing your water and using a water filter. 10. Determine whether a lead service line provides water to your home and pursue local programs to remove and replace it.
Travel and transportation	1. Schedule outdoor activities, including exercise, at low traffic times and away from busy roads. 2. Check local air quality and avoid outdoor activities when air pollution levels are high. 3. Pick efficient travel routes that limit time in traffic. 4. Substitute driving with active transportation (walking, cycling, and public transportation). 5. Encourage local school councils to reduce school bus emissions, including “No Idling Zones.”
Industrial activities and urban design	1. Advocate for sustainable development, including renewable energy, walkability, bike lanes, and public transit. 2. Promote the expansion of green spaces and tree planting and the elimination of synthetic turf fields and the use of pesticides. 3. Demand transparency in product labeling and access to real-time data on local industrial emissions to empower individual actions.

Adapted from Sargis RM, Heindel JJ, Padmanabhan V. Interventions to Address Environmental Metabolism-Disrupting Chemicals: Changing the Narrative to Empower Action to Restore Metabolic Health. Front Endocrinol (Lausanne). 2019;10:33. Published 2019 Feb 4.