Environmental Endocrine-Disrupting Chemicals and Uterine Leiomyoma Development: A Narrative Review

Beredugo Sylvanus[1]* Oboma, Yibala Ibor[2] and Tatfeng, Youtchou Mirabeau.[3]
[1,2, &3] Department of Medical Laboratory Science,
Faculty of Basic Medical Sciences,
College of Health Sciences, Niger Delta University,
Wilberforce Island, Bayelsa State, Nigeria.
Corresponding Author: Beredugo Sylvanus,
Department of Medical Laboratory Science,
Faculty of Basic Medical Sciences,
College of Health Sciences, Niger Delta University,
Wilberforce Island, Bayelsa State, Nigeria
Bayelsa State, Nigeria. beredugosylvanus@ndu.ed. ng
+2348067520637.
ORCID ID:https://orcid.org/0000-0002-4334-3439.

https://doi.org/10.56201/ijhpr.vol.11.no10.2026.pg53.64

Abstract
Environmental endocrine-disrupting chemicals (EDCs) are increasingly being investigated for their potential role in female reproductive disorders, including uterine leiomyomas, commonly known as fibroids. These benign, hormone-responsive tumours are influenced by estrogen and progesterone, raising concerns that exposure to chemicals capable of interfering with hormonal regulation may contribute to their development or progression. This review examines the sources of human exposure to EDCs and explores the potential mechanisms through which selected chemicals may influence uterine leiomyoma development. Relevant literature on phthalates, bisphenol A (BPA), diethylstilbestrol (DES), and genistein was considered, with particular attention to human exposure, reproductive health outcomes, and experimental evidence. The reviewed literature identifies food, drinking water, plastics, medical devices, household materials, and personal-care products as important potential sources of exposure. Proposed mechanisms linking EDCs to leiomyomas include disruption of estrogen and progesterone signalling, epigenetic reprogramming, altered gene expression, changes in cellular signalling pathways, and impaired DNA repair following early-life exposure. Experimental studies suggest that some of these chemicals may promote leiomyoma-cell proliferation, and changes in hormone-receptor activity. However, findings from human studies remain inconsistent, and evidence from laboratory and animal studies cannot independently establish causation in humans. Overall, EDCs represent a plausible environmental factor in uterine leiomyoma development, but their precise contribution remains uncertain. Further well-designed epidemiological and mechanistic studies are needed to clarify exposure-related risks, identify vulnerable periods of development, and establish whether reducing exposure to specific EDCs could help prevent or limit fibroid growth.

Keywords: *Endocrine-disrupting chemicals; uterine leiomyoma; phthalates; bisphenol A; estrogen; epigenetic reprogramming.*

Introduction

Environmental endocrine-disrupting chemicals (EDCs) are widespread contaminants implicated in adverse effects on the endocrine system, in part because they can mimic endogenous hormones [1,2,3]. Because uterine leiomyomas are hormone-responsive benign neoplasms, they may be susceptible to the influence of these chemicals. Research has increasingly investigated the possible contribution of EDCs to leiomyoma pathogenesis [1,4,5].

The Endocrine Society has issued scientific statements on environmental EDCs. Its 2015 statement reviewed effects across metabolic regulation, female and male reproductive health, hormone-responsive neoplasms in females, prostatic function, thyroid homeostasis, and brain systems, including neurodevelopment and neuroendocrine regulation [3]. EDCs are exogenous or human-made substances that can disrupt endocrine function by altering hormone biosynthesis, secretion, transport, metabolism, or action [6]. More than 1,480 EDCs have been identified in products such as plastics, cleaning agents, flame retardants, agricultural chemicals, and personal-care products [7,8].

Types and Sources of Endocrine-Disrupting Chemicals

EDCs can be grouped into plant-derived compounds, industrial chemicals, household and consumer products, medical devices, and pharmaceutical agents.

1. **Phytoestrogens**. These compounds occur in plant-derived foods, including flaxseed, nuts, soy products, grains, and pulses. Genistein, daidzein, and resveratrol can have estrogenic or antiestrogenic activity through binding to estrogen receptors and may also interact with other nuclear hormone receptors [9].

2. **Industrial chemicals**. This category includes pesticides, flame retardants, and combustion by-products including dioxins, perchlorate, and dichlorodiphenyltrichloroethane (DDT). Although DDT was globally banned in 2001, it remains present in the environment [9].

3. **Household and consumer products**. Potential sources include cosmetics, recreational products, food and beverage packaging, contaminated food, and polluted groundwater. Bisphenol A (BPA) and phthalates have been widely used in consumer products. BPA is increasingly being replaced by bisphenol F (BPF) and bisphenol S (BPS); although these substitutes show lower potency in estrogen-receptor activation, BPF has demonstrated an ability comparable to BPA to induce estrogen receptor-alpha transcriptional activity [10].

4. **Medical devices and pharmaceutical agents**. BPA and phthalates are among the common EDCs found in medical devices and associated consumables [9]. Natural or synthetic steroid compounds in pharmaceutical agents may also act as EDCs if released into drinking water or the terrestrial environment. Diethylstilbestrol (DES) and endogenous estrogenic compounds have been detected at concentrations capable of producing physiological effects in animal studies, with possible relevance to humans [11,12].

Human Exposure and Vulnerability

EDCs enter the body through food, contaminated dust and water, inhaled gases and particles, dermal absorption, transplacental transfer, and breast milk. They have been detected throughout the human lifespan in blood, sweat, urine, breast milk, and hair [13].

Exposure to bisphenol A (BPA) during early development remains a significant public health concern because of its potential endocrine-disrupting and developmental effects. Although regulatory restrictions have reduced its use in products intended for infants and young children, BPA exposure may still occur through contaminated food and beverages, food-packaging materials, plastics and other environmental sources [14]. In the European Union, BPA has been prohibited in infant feeding bottles since 2011 and in plastic bottles and food packaging intended for babies and children below three years since 2018. Furthermore, the European Food Safety Authority (EFSA) reassessed BPA toxicity in 2023, highlighting concerns about its potential adverse effects on the immune system. In December 2024, the European Commission adopted a broader prohibition on BPA in food-contact materials, with transitional periods for affected products. These developments underscore the importance of minimizing exposure during critical periods of fetal and childhood development [15,16]. In adult female rodents, BPA exposure at levels comparable to human exposure has been associated with adverse reproductive outcomes, including ovarian cysts, cervical sarcoma, uterine polyps, and mammary adenocarcinoma [9].

Timing of exposure is important because prenatal and neonatal periods are windows of vulnerability during which even low exposure may have effects that persist after the chemical has left the body. These effects may extend into adulthood and potentially across generations [17]. This is consistent with the Developmental Origins of Health and Disease (DOHaD) concept, which proposes that exposures during pregnancy and around birth can influence later disease risk, including through epigenetic mechanisms [18].

EDCs may also act at low doses; below levels commonly assessed in conventional toxicology, within the range of usual human exposure, or at animal doses corresponding to concentrations measured in human blood. Their effects may reflect strong hormone-receptor binding and enhancement of endogenous hormone activity. Combined exposure to multiple chemicals may be harmful even when individual exposures fall below regulatory limits [19,20]. Unlike the often-linear dose-response pattern of endogenous hormones, EDCs may show non-monotonic, U-shaped, or inverted-U-shaped responses. BPA, for example, may produce different effects on cell growth depending on dose and cell type [21].

EDCs and Reproductive Disorders

EDCs have been associated with a range of female reproductive and uterine disorders, including endometrial hyperplasia, uterine leiomyomas, endometriosis, impaired fertility, and spontaneous pregnancy loss [6]. Evidence relating EDC exposure specifically to uterine fibroids includes findings concerning prenatal DES exposure. The Nurses' Health Study II reported a 13% increase in fibroid risk among women older than 35 years who had prenatal DES exposure [22]. In the NIEHS Uterine Fibroid Study of 1,364 women aged 35–49 years, the likelihood ratio for large tumours was 2.4 among Caucasian women with documented fetal DES exposure. Literature also describes experimental findings that in-utero exposure to DES or genistein alters histone methylation in estrogen-responsive genes, increasing hormonal sensitivity and accelerating fibroid development in adult uterine muscle [9].

Phthalates: Exposure, Uses, and Reproductive Health

Phthalates are ubiquitous synthetic diesters of benzene dicarboxylic acid, commonly used as plasticizers or solvents to increase flexibility and alter the properties of commercial materials. They occur in personal-care products, medical devices, food-contact materials, and building components [23,24]. Di-(2-ethylhexyl) phthalate (DEHP) is identified as a prominent environmental pollutant [25,26].

Phthalates enter humans through direct skin contact with consumer products and indirect environmental exposure. Products may release them into soil, groundwater, and aquatic environments after use or disposal [27,28]. Although phthalates are metabolized and eliminated through urine and faeces, their widespread presence in food, beverages, personal-care products, and household environments can lead to repeated exposure [29,30,31]. Exposure is of particular concern during pregnancy. Studies using NHANES data have reported that pregnant individuals may exceed exposure benchmarks measured by the Hazard Index (HI). An HI above one indicates that combined exposure to several phthalates may present a potential health concern [32,33,34]. Personal-care products are an important potential exposure source. An analysis of 252 products sold in Canada detected diethyl phthalate (DEP) across nearly all product categories, with concentrations reaching 2.6% in fragranced products. Dibutyl phthalate (DBP) was detected particularly in nail polish, at levels up to 2.4%, and in smaller quantities in hair sprays, mousses, cleansers, and infant shampoos [35]. DEP is used in fragranced products to stabilize aromatic components, while DBP has been used to increase flexibility in products such as nail polish. Studies have also found phthalates in shampoos, soaps, deodorants, and perfumes, with DEP especially prominent in fragrance products. Monoethyl phthalate (MEP), a DEP metabolite, has been associated with use of fragranced products [36,26].

Other studies have associated hair oils with higher urinary MEP, shampoo use with increased mono-n-butyl phthalate (MBP) and mono-isobutyl phthalate (MIBP), and bath oils, gels, bubble baths, hair products, and sunscreens with higher monomethyl phthalate (MMP). DEHP metabolites have also been linked to cologne or perfume use, with an inverse association reported for fragrance-free products [37,24].

Hair relaxers, lotions, root stimulators, and oils used in some Black communities have been associated with elevated phthalate exposure. Products applied directly to the scalp and left in place for long periods may contribute to sustained exposure [38,39]. Black women have been reported to experience disproportionate exposure to DEP and other EDCs from hair emollients, chemical relaxers, and oils [40]. Some hair preparations show hormone-mimicking activity, and hair emollients with estrogenic activity may remain on the scalp long enough to allow continued dermal absorption [41,34]. Greater use of hormone-mimicking hair products among African-American and African-Caribbean women, alongside use of vaginal douches and scented feminine-hygiene products, has also been associated with increased DEP exposure [42,26].

Phthalates are recognized for antiandrogenic effects that can interfere with male reproductive development [28]. Evidence on female reproductive outcomes is less extensive, but higher urinary concentrations of phthalate metabolites have been associated with fewer oocytes retrieved and fewer viable ova during in-vitro fertilization, as well as lower probabilities of clinical pregnancy and live birth [43]. A Danish cohort study also reported an association between higher urinary phthalate concentrations and spontaneous abortion; reduced progesterone levels have been proposed as one possible pathway [39].

Potential Mechanisms Linking Phthalates and Uterine Leiomyomas

Phthalates may contribute to leiomyoma development or progression through endocrine disruption, epigenetic alterations, and changes in genes and signalling pathways. However, a causal relationship has not been established [26,44].

1. Hormonal disruption. Leiomyomas respond to estrogen and progesterone. Studies have examined whether phthalates disturb these hormonal pathways, but findings in humans have been inconsistent. In women undergoing fibroid surgery, most urinary phthalate indicators were positively associated with uterine size, although the associations were not statistically significant [45]. Earlier studies have reported positive, null, or apparently protective associations between certain phthalate metabolites and fibroids [46]. More recent population-level findings have linked urinary DEHP and DBP metabolites with self-reported fibroids, particularly among premenopausal women. Other research has associated phthalate metabolites with fibroid occurrence and higher circulating estrogen and progesterone. Experimental findings have also suggested that phthalates may increase human leiomyoma-cell proliferation by increasing estrogen receptor-alpha and progesterone-receptor expression [39,47,48].

Phthalates may act as xenoestrogens by binding to estrogen receptors and altering gene expression through genomic and non-genomic pathways. They may affect hormone-receptor abundance and activity, including increasing estrogen receptor-alpha expression and sensitivity to estrogen. Altered estrogen and progesterone signaling may promote cell proliferation, survival, and tissue changes favourable to fibroid growth [49].

2. Epigenetic reprogramming. Exposure during key developmental periods may cause persistent epigenomic changes in uterine mesenchymal stem cells. Changes in DNA methylation and histone modifications may alter the sensitivity of estrogen-responsive genes and promote dysregulated proliferation in leiomyomas. Perinatal exposure to EDCs, including phthalates, has been proposed to re-pattern the epigenetic landscape of these cells and increase susceptibility to leiomyoma development later in life [49,26].

3. Gene mutations and signalling pathways. Phthalate exposure has been discussed in relation to genetic alterations involving *MED12*, a gene reported to be mutated in a substantial proportion of leiomyomas, with estimates reaching 80%. *MED12*-related changes are associated with signaling pathways including WNT/β-catenin, which can promote cellular proliferation and tumour-tissue expansion. The proposed contribution of phthalates to these changes remains a potential mechanism rather than an established causal pathway [49].

Bisphenol A and Other Bisphenols

Evidence has suggested an association between EDCs, particularly BPA, and uterine leiomyomas. A study of Han Chinese women reported higher urinary concentrations of BPA, nonylphenol, and octylphenol among women with leiomyomas than among controls [50]. A later study reported similar findings [51]. In women undergoing hysterectomy, urinary DEHP metabolites—including MEHHP, MEOHP, and MECPP—were reported to be directly associated with fibroid volume [45].

In cultured human leiomyoma cells, exposure to estrogen, BPA, nonylphenol, or octylphenol increased cell proliferation and reduced activity in the TGF-β-Smad signaling pathway compared with untreated controls. BPA was also associated with increased expression of estrogen receptor, insulin-like growth factor 1 (IGF-1), and vascular endothelial growth factor (VEGF) [50]. Research using mesenchymal stem cells isolated from leiomyomas found that BPA increased cyclooxygenase-2 (COX-2) expression and promoted cell proliferation and migration [52]. In vitro exposure to DEHP was associated with increased leiomyoma-cell viability, reduced apoptosis, and increased COX-2 expression [53,54].

BPA has also been investigated in relation to endometrial decidualization and pregnancy loss. Decidualization is necessary for embryo implantation, and impaired endometrial receptivity is implicated in implantation failure [55]. In vitro research found that BPA altered histone methylation marks H3K4me3 and H3K27me3 in endometrial stromal cells, alongside increased EZH2 and reduced MLL1 expression. Changes in the promoter regions of HOXA10, PRL, and IGFBP-1 corresponded with these expression patterns, and the effects were described as estrogen-receptor-mediated, although the precise intracellular pathways remain unclear [56].

Diethylstilbestrol, Genistein, and Early-Life Exposure

Exposure to estrogenic EDCs such as DES and genistein during sensitive stages of uterine development may activate estrogen-receptor signaling and induce epigenetic remodeling. The literature describes activation of phosphoinositide 3-kinase/Akt signaling, altered Enhancer of Zeste Homolog 2 phosphorylation, and changes in H3K27 trimethylation, with effects on estrogen-sensitive gene expression and myometrial structure [55].

In hamsters, neonatal DES exposure was associated with uterine dysplasia and neoplasia and with changes in microRNA expression. During disease onset, expression increased for miR-21, miR-200a, miR-200b, miR-200c, miR-29a, miR-29b, miR-429, and miR-141, while miR-181a decreased. During progression, miR-133a expression decreased [57].

DES, a synthetic estrogen prescribed from the 1940s to the 1970s to prevent pregnancy loss, has been linked to higher leiomyoma occurrence and larger benign uterine tumours among women reporting prenatal exposure. In rat ELT-3 cells, DES increased cell replication in a concentration-dependent manner. The plant-derived estrogens; coumestrol and naringenin also stimulated ELT-3 cell growth. Prenatal or neonatal exposure to DES or genistein was associated with greater neoplasm frequency and size in Eker rats [58]. Early DES exposure has also been investigated for its effects on genomic integrity. Myometrial progenitor cells from adult Eker rats exposed prenatally to DES showed more genomic lesions and a reduced ability to repair DNA double-strand breaks than cells from unexposed controls [59]. A subsequent study investigated vitamin D3 as a means of mitigating this impairment. DES exposure suppressed key DNA-repair proteins in rat myometrial progenitor cells, whereas vitamin D3 intervention restored aspects of the genomic-integrity maintenance response [60].

Conclusion

The reviewed literatures describe widespread human exposure to EDCs through food, water, air, consumer goods, medical devices, and personal-care products. Evidence discussed in this review links EDC exposure-particularly phthalates, BPA, DES, and genistein-with hormone-related reproductive outcomes and possible pathways involved in uterine leiomyoma development. Proposed mechanisms include altered estrogen and progesterone signaling, epigenetic reprogramming, changes in gene expression and signaling pathways, and impaired DNA repair following early-life exposure. Findings from human studies are not uniform, and the causal relationship between phthalate exposure and leiomyoma development remains unresolved. Further epidemiological and mechanistic investigation is needed to clarify these associations.

References

1. Amir, S., Shah, S. T., Mamoulakis, C., Docea, A. O., Kalantzi, O. I., Zachariou, A., Calina, D., Carvalho, F., Sofikitis, N., Makrigiannakis, A., & Tsatsakis, A. (2021). Endocrine disruptors acting on estrogen and androgen pathways cause reproductive disorders through multiple mechanisms: A review. *International Journal of Environmental Research and Public Health, 18*(3), 1464.
2. Fucic, A., Galea, K. S., Duca, R. C., El Yamani, M., Frery, N., Godderis, L., …& Moshammer, H. (2018). Potential health risk of endocrine disruptors in the construction sector and plastics industry: A new paradigm in occupational health. *International Journal of Environmental Research and Public Health, 15*(6), 1229.
3. Gore, A. C., Chappell, V. A., Fenton, S. E., Flaws, J. A., Nadal, A., Prins, G. S., *et al.* (2015). Executive summary to EDC-2: The Endocrine Society's second scientific statement on endocrine-disrupting chemicals. *Endocrine Reviews, 36*(6), 593–602.
4. Canipari, R., De Santis, L., & Cecconi, S. (2020). Female fertility and environmental pollution. *International Journal of Environmental Research and Public Health, 17*(24), 8802.
5. Street, M. E., Angelini, S., Bernasconi, S., Burgio, E., Cassio, A., Catellani, C., ... & Amarri, S. (2018). Current knowledge on endocrine disrupting chemicals (EDCs) from animal biology to humans, from pregnancy to adulthood: highlights from a national Italian meeting. *International journal of molecular sciences*, *19*(6), 1647.
6. Liang, Y., Lu, Q., Chen, M., Zhao, X., Chu, C., Zhang, C., Yuan, J., *et al.* (2025). Impact of endocrine-disrupting chemicals on epigenetic regulation in the uterus: A narrative review. *Reproductive Biology and Endocrinology, 23*, 80.
7. Bolden, A. L., Schultz, K., Pelch, K. E., & Kwiatkowski, C. F. (2018). Endocrine activity of air pollutants associated with oil and gas extraction. *Environmental Health, 17*(1), 26.
8. Green, M. P., Harvey, A. J., Finger, B. J., & Tarulli, G. A. (2021). Endocrine-disrupting chemicals and impacts on human fertility. *Environmental Research, 194*, 110694.
9. Hall, J. M., & Greco, C. W. (2020). Perturbation of nuclear hormone receptors by endocrine-disrupting chemicals: Mechanisms and pathological consequences of exposure. *Cells, 9*(1), 13.
10. Pelch, K. E., Li, Y., Perera, L., Thayer, K. A., & Korach, K. S. (2019). Characterization of estrogenic and androgenic activities for bisphenol A-like chemicals (BPs): In vitro estrogen and androgen receptors transcriptional activation, gene regulation, and binding profiles. *Toxicological Sciences, 172*, 23–37.
11. Lei, B., Peng, W., Li, W., Yu, Y., Xu, J., & Wang, Y. (2016). Diethylstilbestrol at environmental levels affects the development of early life stage and target gene expression in Japanese medaka (*Oryzias latipes*). *Ecotoxicology, 25*, 563–573.
12. Adeel, M., Song, X., Wang, Y., Francis, D., & Yang, Y. (2017). Environmental impact of estrogens on human, animal, and plant life: A critical review. *Environment International, 99*, 107–119.
13. World Health Organization. (2015). *Human biomonitoring: Facts and Figures*. WHO Regional Office for Europe.
14. Manzoor, M. F., Tariq, T., Fatima, B., Sahar, A., Tariq, F., Munir, S., Khan, S., Ranjha, M. M. A. N., Sameen, A., Zeng, X.-A., & Ibrahim, S. A. (2022). An insight into bisphenol A, food exposure and its adverse effects on health: A review. Frontiers in Nutrition, 9, Article 1047827. https://doi.org/10.3389/fnut.2022.1047827 IF: 5.5 Q1 B2

15. European Food Safety Authority (EFSA). (2023). Re-evaluation of the risks to public health related to the presence of bisphenol A (BPA) in foodstuffs. *EFSA Journal*, 21(4), e06857.
16. European Commission. (2024, December 19). Commission adopts ban of bisphenol A in food contact materials. https://food.ec.europa.eu/food-safety-news/commission-adopts-ban-bisphenol-food-contact-materials-2024-12-19_en
17. Onuzulu, C. D., Rotimi, O. A., & Rotimi, S. O. (2019). Epigenetic modifications from in utero exposure to endocrine-disrupting chemicals. *Reviews on Environmental Health, 34*(4), 309–325.
18. Ghassabian, A., Vandenberg, L., Kannan, K., & Trasande, L. (2022). Endocrine-disrupting chemicals and child health. *Annual Review of Pharmacology and Toxicology, 62*, 573–594.
19. Hamid, N., Junaid, M., & Pei, D.-S. (2021). Combined toxicity of endocrine-disrupting chemicals. *Ecotoxicology and Environmental Safety, 215*, 112136.
20. Caporale, N., Leemans, M., Birgersson, L., Germain, P.-L., Cheroni, C., Borbély, G., Engdahl, E., Lindh, C. H., Bressan, R. A., Cavallo, F., & others. (2022). Adverse impacts of endocrine-disrupting mixtures. *Science, 375*(6582).
21. Kim, H., Park, H., Hwang, B., Kim, S., Choi, Y.-H., Kim, W.-J., *et al.* (2023). Bisphenol A inhibits vascular smooth muscle cell responses. *Environmental Toxicology and Pharmacology, 98*, 104060.
22. Mahalingaiah, S., Hart, J. E., Wise, L. A., Terry, K. L., Boynton-Jarrett, R., & Missmer, S. A. (2014). Prenatal diethylstilbestrol exposure and risk of uterine leiomyomata in the Nurses' Health Study II. *American Journal of Epidemiology, 179*, 186–191.
23. Radke, E. G., Braun, J. M., Meeker, J. D., & Ferguson, K. K. (2020). Phthalate exposure and cumulative risk assessment: Lessons from biomonitoring data. *Environmental Research, 184*, 109325.
24. Kareem, F. B., & Emokpae, M. A. (2022). Association between the concentrations of some toxic metals and the risk of uterine fibroids among Nigerian women. *Environmental Disease, 7*, 96–101.
25. Bamai, Y. A., Araki, A., Kawai, T., Tsuboi, T., Saito, I., Yoshioka, E., Kanazawa, A., Cong, S., Kishi, R., & others. (2023). Human exposure to phthalates and substitutes: Global biomonitoring evidence. *Science of the Total Environment, 857*, 159385.
26. Yang, S., Yang, S., & Luo, A. (2025). Phthalates and uterine disorders. *Reviews on Environmental Health, 40*(1), 97–114.
27. U.S Environmental Protection Agency (EPA). (2021). *Phthalates action plan and cumulative risk assessment update*.
28. Mondal, T., Mondal, S., Ghosh, S. K., Pal, P., Soren, T., Pandey, S., & Maiti, T. K. (2022). Phthalates: A family of plasticizers, their health risks, phytotoxic effects, and microbial bioaugmentation approaches. *Environmental Research, 214* (3), 114059.
29. Zota, A. R., Phillips, C. A., & Mitro, S. D. (2020). Recent fast food consumption and exposure to phthalates and replacement plasticizers. *Environmental Health Perspectives, 128*(6), 067004.
30. Johns, L. E., Ferguson, K. K., McElrath, T. F., Mukherjee, B., Meeker, J. D., & Cantonwine, D. E. (2022). Urinary biomarkers of cumulative phthalate exposure in U.S. women: NHANES 2013–2018. *Journal of Exposure Science & Environmental Epidemiology, 32*, 643–652.

31. Welch, B. M., Keil, A. P., Buckley, J. P., Engel, S. M., James-Todd, T., Zota, A. R., ... & Ferguson, K. K. (2023). Racial and ethnic disparities in phthalate exposure and preterm birth: a pooled study of sixteen US cohorts. *Environmental health perspectives*, *131*(12), 127015.
32. Trasande, L., Ghassabian, A., & Petrik, J. (2021). Phthalates and reproductive health: A review of the human epidemiological evidence. *Environmental Research, 202*, 111722. https://doi.org/10.1016/j.envres.2021.111722.
33. Kortenkamp, A., & Faust, M. (2022). Regulatory characterization of chemical mixtures: Phthalates as a case study. *Environmental Sciences Europe, 34*(1), 1–13.
34. Varshavsky, J. R., Zota, A. R., & Woodruff, T. J. (2016). A novel method for calculating potency-weighted cumulative phthalates exposure with implications for identifying racial/ethnic disparities among U.S. reproductive-aged women in NHANES 2001–2012. *Environmental Science & Technology, 50*(19), 10616–10624.
35. Koniecki, D., Wang, R., Moody, R. P., & Zhu, J. (2011). Phthalates in cosmetic and personal care products: Concentrations and possible dermal exposure. *Environmental Research, 111*(3), 329–336.
36. Dodson, R. E., Nishioka, M., Standley, L. J., Perovich, L. J., Brody, J. G., & Rudel, R. A. (2012). Endocrine disruptors and asthma-associated chemicals in consumer products. *Environmental Health Perspectives*, 120(7), 935–943.
37. Parlett, L. E., Calafat, A. M., & Swan, S. H. (2013). Women's exposure to phthalates in relation to use of personal care products. *Journal of Exposure Science and Environmental Epidemiology, 23*(2), 197–206.
38. Zota, A. R., & Shamasunder, B. (2017). The environmental injustice of beauty. *American Journal of Obstetrics and Gynecology, 217*(4), 418.
39. Rosen, E. M., Ferguson, K. K., Mukherjee, B., McElrath, T. F., Cantonwine, D. E., & Meeker, J. D. (2024). Personal care product use patterns in association with phthalate biomarkers across pregnancy. *Journal of Exposure Science & Environmental Epidemiology*, 34(4), 591–600.
40. Helm, J. S., Nishioka, M., Brody, J. G., Rudel, R. A., & Dodson, R. E. (2018). Measurement of endocrine-disrupting and asthma-associated chemicals in hair products used by Black women. *Environmental Research, 165*, 448–458.
41. James-Todd, T. M., Meeker, J. D., Huang, T., Hauser, R., & Ferguson, K. K. (2016). Racial/ethnic disparities in endocrine-disrupting chemicals. *Current Epidemiology Reports, 3*(2), 161–180.
42. McDonald, J. A., Watkins, D. J., Vélez-Vega, C., Ferguson, K. K., Meeker, J. D., & Cordero, J. F. (2022). Maternal food and beverage consumption behaviours and discrepant phthalate exposure by race. *International Journal of Environmental Research and Public Health, 19*(4), 2273.
43. Hauser, R., Gaskins, A. J., Souter, I., Smith, K. W., Dodge, L. E., Ehrlich, S., …& Williams, P. L. (2016). Urinary phthalate metabolite concentrations and reproductive outcomes among women undergoing in vitro fertilization: Results from the EARTH Study. *Environmental Health Perspectives, 124*(6), 831–839.
44. Zhang, H., Zhou, H., Chen, X., Guo, H., Lin, Q., & Chen, X. (2024). Phthalate exposure as a hidden risk factor for uterine leiomyoma in adult women: Accumulated evidence from observational studies. *Ecotoxicology and Environmental Safety*, 285, Article 117069.

45. Zota, A. R., Geller, R. J., Calafat, A. M., Marfori, C. Q., Baccarelli, A. A., & Moawad, G. N. (2019). Phthalates exposure and uterine fibroid burden among women undergoing surgical treatment for fibroids: A preliminary study. *Fertility and Sterility, 111*(1), 112-121.
46. Dubey, P., Gaskins, A. J., Louis, G. M. B., Chen, Z., McElrath, T. F., & Meeker, J. D. (2022). Association of exposure to phthalate metabolites with sex hormones, obesity, and metabolic syndrome in U.S. women. *JAMA Network Open, 5*(9), e2233088.
47. Yang, Q., Ciebiera, M., Bariani, M. V., Ali, M., Al-Hendy, O., ...Al-Hendy, A. *et al.*, (2022). Comprehensive Review of Uterine fibroids: Developmental Origin, Pathogenesis and Treatment. *Endocrine Reviews,* 43(4):678 719.
48. Huang, Y. Y., Chen, L., Ma, G. X., Xu, X. D., Jia, X. G., Deng, F. S., ... & Yuan, J. Q. (2021). A review on phytochemicals of the genus Maytenus and their bioactive studies. *Molecules*, *26*(15), 4563.
49. Katz, T. A., Yang, Q., Treviño, L. S., Walker, C. L., & Al-Hendy, A. (2016). Endocrine-disrupting chemicals and uterine fibroids. *Fertility and Sterility, 106*, 967–977.
50. Shen, Y., Dong, Y. M., Lu, Q., Xu, J., Wu, Y. T., Yun, S. S., & Ren, M. L. (2016). Phenolic environmental estrogens in urine and blood plasma from women with uterine leiomyoma: Epidemiological survey. *Journal of Obstetrics and Gynaecology Research, 42*(4), 440-445.
51. Wesselink, A. K., Weuve, J., Fruh, V., Bethea, T. N., Henn, B. C., Harmon, Q. E., Hauser, R., Williams, P. L., Calafat, A. M., McClean, M., Baird, D. D., & Wise, L. A. (2021). Urinary concentrations of phenols, parabens, and triclocarban in relation to uterine leiomyomata incidence and growth. *Fertility and Sterility, 116*(6), 1590–1600.
52. He, C., Nelson, W., Li, H., Xu, Y. D., Dai, X. J., Wang, Y. X., ... & Li, T. (2022). Frequency of MED12 mutation in relation to tumour and patient's clinical characteristics: a meta-analysis. *Reproductive Sciences*, *29*(2), 357-365.
53. Kim, H. J., Kim, S. H., Oh, Y. S., Heo, S. H., Kim, K. H., Kim, D. Y., … & Chae, H. D. (2021). Effects of phthalate esters on human myometrial and fibroid cells: Cell culture and NOD-SCID mouse data. *Reproductive Sciences, 28*(2), 479-487.
54. Chou, W.-C., Lee, P.-H., Tan, Y.-Y., Lin, H.-C., Yang, C.-W., Chen, K.-H., & others. (2017). BPA-induced microRNA dysregulation in endometrial cells. *Toxicology in Vitro, 41*, 133–142.
55. Gupta, M. K., Pattnaik, S., & Sudandiradoss, C. (2025). Artificial intelligence driven based diagnostics with combined surgical and non-surgical management of uterine fibroids: A narrative review. *Middle East Fertility Society Journal*, 30 (64).
56. Xiong, Y., Wen, X., Liu, H., Zhang, M., & Zhang, Y. (2020). Bisphenol A alters decidualization via epigenetic regulation. *Journal of Steroid Biochemistry and Molecular Biology, 200*, 105640.
57. Padmanabhan, R., Hendry, I. R., Knapp, J. R., Shuai, B., & Hendry, W. J. (2017). Altered microRNA expression patterns during the initiation and promotion stages of neonatal diethylstilbestrol-induced dysplasia/neoplasia in the hamster (Mesocricetus auratus) uterus. Cell Biology and Toxicology, 33(5), 483–500.
58. Stewart, E. A., & Nowak, R. A. (2022). Uterine fibroids: Hiding in plain sight. *Physiology, 37*, 16–27.
59. Prusinski Fernung, L. E., Yang, Q., Sakamuro, D., Kumari, A., Mas, A., & Al-Hendy, A. (2018). Endocrine disruptor exposure during development increases incidence of uterine

fibroids by altering DNA repair in myometrial stem cells. *Biology of Reproduction, 99*(4), 735–748.

60. Elkafas, H., Ali, M., Elmorsy, E., Kamel, R., Thompson, W. E., Badary, O., Al-Hendy, A., & Yang, Q. (2020). Vitamin D3 ameliorates DNA damage caused by developmental exposure to endocrine disruptors in the uterine myometrial stem cells of Eker rats. *Cells, 9*(6), 1459.

IJHPR