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Review Article

Endocrine-disrupting chemicals and human infertility: a scoping review of daily-life exposures and clinical implications for clinical practice

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ABSTRACT

Infertility is a growing worldwide health issue that cannot be addressed only by hereditary or behavioural causes. Growing research suggests that endocrine-disrupting chemicals (EDCs), which are pervasive environmental pollutants, are substantial contributors to poor male and female reproductive health, particularly in fast industrialising nations like India. The objective was to comprehensively map and synthesise current knowledge on the origins, mechanisms, and clinical reproductive consequences of EDC exposure, with an emphasis on the implications for infertility practice and public health policy. A scoping review was conducted using the PRISMA-ScR framework. The PubMed/MEDLINE, Scopus, and Web of Science databases were searched for systematic reviews, meta-analyses, umbrella reviews, and significant human and animal research published between January 2014 and June 2025. The Endocrine Society, World Health Organisation, and United Nations Environment Programme provided authoritative position statements. Evidence was synthesised thematically based on exposure sources, molecular pathways, and clinical consequences. EDCs, which include bisphenols, phthalates, pesticides, PFAS, and heavy metals, have been linked to poor sperm quality, testosterone shortage, ovulatory dysfunction, decreased ovarian reserve, endometriosis, poor pregnancy outcomes, and possible transgenerational impacts. Disruption of the hypothalamic-pituitary-gonadal axis, interference with hormone receptors, poor steroidogenesis, oxidative stress, mitochondrial dysfunction, and epigenetic alteration were among the mechanisms implicated. Exposure reduction treatments showed short-term reversibility of biomarker load, indicating therapeutic significance. EDCs are controllable yet underappreciated risk factors for infertility. Integrating exposure assessment, preventative counselling, biomonitoring, and regulatory tightening, particularly in India, may improve reproductive outcomes and supplement assisted reproductive technology.

Keywords: Endocrine-disrupting chemicals, Infertility, Reproductive endocrinology, Environmental exposure, Preventive endocrinology, India

INTRODUCTION

Human reproductive health has been found to vulnerable to chemical agents in the twenty first century, Over 168 million individuals and 48 million couples are facing the problem of infertility worldwide, which are no longer attributable to genetic or behavioural factors alone.[1] Current evidence proves endocrine-disrupting chemicals (EDC) as a key environmental diver of this escalating trend.[2] EDCs are defined as any exogenous chemicals,

combination of chemicals that disrupt the hormonal action at any level such as synthesis, secretion, transport, binding with carriers, action at distant sites or metabolic clearance.[3] From the foetal stage to senescence, human populations are exposed to a "cocktail" of chemicals, ranging from epoxy resins inside canned goods to plasticisers in personal care products and persistent pesticides in agricultural runoff.[4] Diverse mechanism of action, including non-linear and non-monotonic dose responses (NMDR), can lead to biological activity at low levels that diverge from standard toxicological thresholds.[3] Rapid industrialisation, high population density, and distinct food patterns contribute to EDCs' clinical relevance.[5] Reports show a steady decrease in sperm quality among Indian men over the last four decades, while female reproductive problems such as PCOS have reached pandemic proportions.[1] Furthermore, the Indian population may be particularly vulnerable due to socioeconomic variables such as low healthcare access and malnutrition, which might alter the impact of environmental contaminants.[5] This review attempt to do a systematic scoping review of the most recent literature to provide medical professionals with evidence-based insights for clinical practice and public health advocacy.

METHODS

This scoping review's approach follows the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) framework to ensure transparency, completeness, and rigour. Primary evidence was gathered from high-impact, PubMed-indexed sources, such as systematic reviews, meta-analyses, and umbrella reviews published between January 2014 and June 2025.6 To build a worldwide consensus baseline, authoritative position statements from the Endocrine Society, WHO, and UNEP were given top priority. To offer regional context, studies on the Indian population, such as biomonitoring data from major metropolitan areas and national regulatory updates from the Food Safety and Standards Authority of India (FSSAI), were included.[1]

Databases and search strategy

The search strategy was executed across three major bibliographic databases: PubMed/MEDLINE, Scopus, and the Web of Science Core Collection.7 The search utilized Boolean operators to combine keywords related to endocrine disruption (e.g., "EDC", "bisphenol A", "phthalate", "PFAS", "pesticide", "organophosphate") with those related to reproductive health (e.g., "infertility", "semen quality", "ovarian reserve", "PCOS", "endometriosis", "implantation failure").Specific search strings were customized for each database to capture titles, abstracts, and MeSH terms.

Inclusion and exclusion criteria

Studies were considered if they analysed human epidemiological data or major animal models on EDC exposure and reproductive consequences, addressed the daily exposure through food, consumer product, or environmental contact and published in English within a 10-year period. Exclusion criteria included non-peer-reviewed editorials, minor case reports (n<10), and research focussing solely on non-hormonal effects.

Data charting and synthesis

Data were plotted using a standard template to extract information on chemical class, exposure route, mechanistic pathway, and clinical consequence. The synthesis took a theme approach, combining molecular causes, clinical consequences, and regulatory viewpoints to create a fluid, expert-level narrative appropriate for professional clinical practice.

EDCs

EDCs are classified functionally rather than structurally. They are a diverse category of molecules that include both natural substances (such as phytoestrogens) and manmade chemicals (such as industrial additives).[7] Categories and common sources of EDC are mentioned in Table 1.[5]

Table 1: Categories and common sources of EDC in daily life.

Major category	Common sources in daily life	Representative chemicals
Plasticizers	Personal care products, food packaging, medical tubing	Phthalates (DEHP, DBP, DEP)
Bisphenols	Can linings, thermal receipts, polycarbonates	BPA, BPS, BPF
Pesticides	Agricultural residues, groundwater, household bug sprays	Organophosphates, DDT, Neonicotinoids
PFAS	Non-stick cookware, waterproof textiles, firefighting foam	PFOA, PFOS
UV Filters	Sunscreens, anti-aging cosmetics	Benzophenones (BP-3)
Antimicrobials	Antibacterial soaps, toothpaste	Triclosan, Triclocarban
Industrial By-products	Industrial effluents, combustion of waste	Dioxins, PCBs

Persistent vs. non-persistent EDCs

EDCs are generally classified based on their environmental persistence and bioaccumulative capacity. Persistent Organic Pollutants (POPs) include substances such as polychlorinated biphenyls (PCBs), dioxins, and DDT. These chemicals are lipophilic and resistant to breakdown, allowing them to accumulate in the food chain and be stored in human adipose tissue. Non-persistent EDCs, such as bisphenols (e.g., BPA) and phthalates, have shorter half-lives but are classified as "pseudo-persistent" due to constant daily exposure.[4]

Industrial chemicals

Bisphenols, notably bisphenol A (BPA), are common EDCs found in polycarbonate plastics, epoxy resins that line food containers and thermal receipt paper.[8] Despite legal limits in certain countries, BPA analogues (BPS, BPF) with similar endocrine disruptive effects have developed as alternatives.[8] Phthalates, which are employed as plasticisers in polyvinyl chloride goods, personal care items, and medical equipment, are another prominent EDC class with high human exposure.[8] Per- and polyfluoroalkyl substances (PFAS), sometimes known as "forever chemicals" owing to their long-term environmental persistence, are used in nonstick cookware, water-repellent textiles, and firefighting foams, and may be detected in more than 95% of the US population.[9] Despite being outlawed since the 1970s and 1980s, polychlorinated biphenyls (PCBs) remain in the environment and bioaccumulate in food chains, notably in fish.[9]

Pesticides and herbicides

Organophosphate insecticides, such as chlorpyrifos and malathion, have anti-androgenic and anti-estrogenic properties.[10] Despite worldwide bans, organochlorine chemicals like as dichlorodiphenyltrichloroethane (DDT) continue to be found in the environment and human tissues, particularly in underdeveloped nations where use was more widespread.[10] Glyphosate-based herbicides, among the world's most frequently used agricultural pesticides, have endocrine-disrupting qualities that impair steroidogenesis.[11] Pyrethroids, common household insecticides, have anti-androgenic properties and alter thyroid hormone signalling.[11]

Heavy metals

Lead exposure, while falling in industrialised nations, remains an issue in poorer countries, where it occurs through polluted water, paint, and occupational exposures.[12] Cadmium, which accumulates through tobacco smoke, tainted food, and industrial pollutants, impairs steroidogenesis and has oestrogenic characteristics.[12] Mercury, particularly methylmercury from fish diet, influences reproductive hormone production and function.[12] Arsenic pollution of groundwater is a major problem in Bangladesh and portions of India, with known reproductive harm.[13]

Cosmetics, personal care products, and food-contact materials

Parabens, which are often used as preservatives in cosmetics and pharmaceuticals, have oestrogenic properties and are found in human urine, serum, and breast milk. Triclosan, an antibacterial ingredient included in personal care items, has been shown to alter thyroid hormone balance and reproductive function.[14] UV filters in sunscreens, such as benzophenones and octinoxate, have hormonal action.[14] Food packaging materials considerably contribute to EDC exposure by allowing plasticisers such as phthalates and bisphenols to migrate into food items.[15]

Occupational and environmental exposure pathways

Occupational exposures are major sources for some groups, such as agricultural workers exposed to pesticides, industrial workers handling plastics or solvents, and hospital personnel exposed to sterilisers.[30] Environmental routes include polluted drinking water, air pollution with particulate-bound EDCs, and contaminated food consumption.[16] Indoor dust is an unappreciated exposure pathway, especially for flame retardants and plasticisers.[16] Dietary chains lead to the concentration of lipophilic EDCs in fish, dairy products, and meat.[17]

PATHOGENESIS AND MECHANISMS OF REPRODUCTIVE TOXICITY

Disruption of the hypothalamic-pituitary-gonadal axis

EDCs disrupt HPG axis function on several levels, including GnRH pulsatility, LH and FSH production, and feedback processes.[18] BPA exposure affects GnRH neurone activity via kisspeptin signalling, a key regulator of reproductive neuroendocrine regulation.19 Phthalates inhibit LH secretion, which disrupts the central drive for gonadal steroidogenesis.[19] PFAS chemicals inhibit hypothalamic and pituitary hormone production and release, contributing to reproductive failure.[20]

Hormone receptor interference

EDCs interact with nuclear hormone receptors using a variety of methods, including agonism, antagonism, and altered receptor expression.[21] Although BPA binds oestrogen receptors (ERα and ERβ) with lesser affinity than oestradiol, it can have considerable effects due to non-monotonic dose-response relationships.[22] Phthalates and their metabolites have anti-androgenic effects via inhibiting androgen receptor signalling.[22] Given the importance of thyroid hormones in fertility, many EDCs have an indirect influence on reproductive function by affecting thyroid hormone receptors and transport proteins.[23] Some EDCs modulate receptors selectively, acting as agonists or antagonists in specific tissues.[23]

Altered steroidogenesis

EDCs inhibit steroidogenic enzyme expression and activity, impairing sex hormone production.[24] Phthalates, pesticides, PFAS, and heavy metals interfere with enzymes such as 3β-hydroxysteroid dehydrogenase, 17β-hydroxysteroid dehydrogenase, aromatase, and the steroidogenic acute regulatory (StAR) protein, resulting in reduced testosterone and estrogen production.[25,26]

Oxidative stress and mitochondrial dysfunction

EDC-induced oxidative stress is a prevalent mechanism for reproductive harm.[27] Excessive generation of reactive oxygen species (ROS) overwhelms the antioxidant defence system, leading to lipid peroxidation, protein oxidation, DNA damage, and mitochondrial dysfunction. BPA impairs granulosa cell and oocyte function through oxidative stress, whereas phthalates and heavy metals compromise mitochondrial integrity, ATP production, and steroidogenic capacity. Mitochondrial DNA damage further reduces oocyte developmental competence.[28,29]

CLINICAL IMPLICATIONS IN MALE INFERTILITY

Semen quality and sperm function

Epidemiological studies show a continuous relationship between EDC exposure and altered sperm parameters. BPA concentrations in the urine correlate adversely with sperm concentration, motility, and morphology in many populations. Phthalate metabolite levels are associated with decreased sperm count, altered shape, and compromised DNA integrity.[30] PFAS exposure leads to a reduction in total sperm count and progressive motility. Heavy metal biomarkers, such as lead and cadmium, have a negative correlation with a variety of semen quality measures. Pesticide exposure among agricultural workers is associated with oligozoospermia and asthenozoospermia.[30] Beyond traditional characteristics, EDCs influence sperm functional competence, such as capacitation, acrosome response, and fertilisation capacity. EDC exposure increases sperm DNA fragmentation, which is a key driver of fertility and pregnancy outcomes.[31]

Testosterone deficiency and hypogonadism

EDC exposure adds to the decline in testosterone levels reported in male populations. Cross-sectional studies show a negative connection between phthalate metabolites and serum testosterone.[32] BPA exposure is associated with lower testosterone levels in both occupationally exposed workers and the general population. PFAS chemicals are associated with reduced testosterone levels and changed LH:Testosterone ratios, implying both central and peripheral effects. Heavy metal exposure, particularly lead, reduces testosterone via several routes. These endocrine disturbances contribute to clinical hypogonadism, which affects fertility, sexual function, and general health.[33]

Male reproductive outcomes relevant to infertility practice

Couples utilising assisted reproductive technology (ART) had greater EDC biomarker levels than fertile controls.[34] In ART cycles, paternal EDC exposure had a deleterious influence on fertility rates, embryo quality, and pregnancy outcomes. Sperm from males who have been exposed to more phthalates have a lower success rate with intracytoplasmic sperm injection (ICSI). Time to pregnancy, a sensitive reproductive metric, rises with male partner EDC exposure in prospective cohort studies. Recurrent pregnancy loss is associated with a higher paternal EDC load, which may be mediated by sperm DNA damage and epigenetic changes.[34]

CLINICAL IMPLICATIONS IN FEMALE INFERTILITY

Ovulatory dysfunction and PCOS-like phenotypes

EDC exposure is linked to ovulatory dysfunction and polycystic ovary syndrome (PCOS) development Multiple studies have found a link between BPA levels and PCOS, hyperandrogenism, and insulin resistance.[35] Phthalates are associated with anovulation and irregular menstrual periods. Dioxin exposure is linked to PCOS-like endocrine characteristics. Possible causes include impaired steroidogenesis, altered insulin signalling, and inflammatory pathway activation.[35]

Diminished ovarian reserve and premature ovarian insufficiency

EDCs promote follicular depletion and lead to decreased ovarian reserve (DOR). Women with detectable PFAS had decreased anti-Müllerian hormone (AMH) levels and fewer antral follicles.[36] BPA exposure is associated with an earlier age at menopause and indicators of ovarian ageing. Phthalates have an inverse relationship with AMH in reproductive-aged women. Heavy metals, especially cadmium from smoking, hasten ovarian ageing. Direct oocyte toxicity, increased follicular atresia, and disruption of granulosa cell function are all possible mechanisms. In sensitive groups, exposure to EDC raises the incidence of premature ovarian insufficiency (POI).[36]

Menstrual irregularities and pregnancy outcomes

EDC exposure is associated with menstrual cycle anomalies, such as shorter cycle lengths and heavier menstrual flow. In prospective cohort studies, PFAS exposure is associated with a longer time to conception. Pregnancy problems such as gestational diabetes, hypertension, and premature delivery are associated with maternal EDC load.[37] Prenatal EDC exposure is associated with intrauterine growth restriction and bad birth

outcomes. The risk of pregnancy loss increases with increasing maternal concentrations of particular EDCs, such as BPA and certain phthalates. These connections continue after adjusting for confounders, implying causal links.[37]

PREVENTION AND EXPOSURE REDUCTION IN ROUTINE LIFE

Clinical and epidemiological data increasingly supports the incorporation of endocrine-disrupting chemical (EDC) exposure screening and targeted counselling into standard infertility therapy.[38] Validated exposure surveys aid in the identification of modifiable dietary, home, and occupational risk factors for reproduction.[39] Dietary interventions, notably increasing fresh food consumption and avoiding canned items, dramatically reduce exposure to bisphenol A (BPA), pesticides, and other food-contact EDCs.[40] Preference for organic products, dairy, and meat reduces consumption of persistent organic pollutants and hormonally active residues.[41] Fish eating should combine cardiometabolic advantages with lower mercury and PCB exposure, favouring low-contaminant species. Household techniques such as replacing plastic containers with glass or stainless steel, limiting microwave heating of plastics, and enhancing interior ventilation significantly reduces phthalate and BPA exposure.[42] Household techniques such as replacing plastic containers with glass or stainless steel, limiting microwave heating of plastics, and enhancing indoor ventilation can significantly minimise phthalate and BPA exposure.[43] The use of fragrance- and paraben-free personal care products reduces cutaneous absorption of hormonally active substances.[44] Occupational exposure reduction by protective equipment, hygienic procedures, and less thermal receipt handling is especially important for high-risk personnel.[45] Combined lifestyle changes have been demonstrated to reduce urine EDC biomarkers within weeks, demonstrating the reversibility of exposure load. Preconception counselling is a vital educational moment, and using the precautionary principle supports low-cost exposure-reduction measures that may enhance fertility, pregnancy outcomes, and child health.[46]

Future directions

Despite growing evidence linking endocrine disrupting chemicals (EDCs) to infertility, significant research gaps remain, particularly a lack of longitudinal studies with repeated exposure assessment and fertility outcomes, as well as a scarcity of high-quality data from Indian populations with region-specific exposure patterns. The use of established, non-invasive biomarkers in biomonitoring and exposomic techniques might allow for cumulative exposure assessment, individual susceptibility identification, and mechanistic insights pertinent to reproductive endocrinology. To provide complete, prevention-oriented treatment, infertility clinics should implement systematic EDC exposure assessment, reduce procedural and laboratory-related exposures, and interact with environmental health and public health specialists. Incorporating environmental reproductive health into professional training and clinical guidelines would promote evidence-based counselling and the translation of study findings into practice. Preventive endocrinology, which focusses on reducing preconception exposure and advocating for evidence-based policies, is a complementing method to assisted reproductive technologies that has the potential to improve fertility and child health.

CONCLUSION

Endocrine-disrupting chemicals are common environmental exposures with compelling mechanistic and clinical evidence linking them to reduced male and female fertility via disruption of the hypothalamic-pituitary-gonadal axis, steroidogenesis, oxidative stress, epigenetic modification, and direct gonadotoxicity. Human studies consistently link EDC exposure to worse semen quality, ovulatory dysfunction, decreased ovarian reserve, endometriosis, poor pregnancy outcomes, and possible transgenerational reproductive impacts. Reproductive endocrinologists should recognise EDCs as modifiable risk factors and include evidence-based exposure reduction methods into infertility treatment, while also realising that individual-level therapies are ineffective in the face of pervasive environmental pollution. To preserve reproductive health and supplement existing fertility therapies, we urgently need more regulatory control, population-specific research, particularly in India, comprehensive biomonitoring, and a preventive endocrinology framework.

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