Sanjeevani Darshan

ISSN: 2584-0304
http://sanjeevanidarshan.com

National Journal of AYURVEDA & YOGA

Year - 2026 Volume 4, Issue 1

" IMPACT OF ENDOCRINE-DISRUPTING CHEMICALS ON MALE INFERTILITY: A SYSTEMATIC REVIEW AND META-ANALYTICAL SYNTHESIS"

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ABSTRACT:

Background- Male infertility affects nearly 15% of couples worldwide, with male-related factors accounting for about half of these cases. Increasing research suggests that exposure to endocrine-disrupting chemicals (EDCs)—such as bisphenol A (BPA), phthalates, pesticides, and polychlorinated biphenyls (PCBs)—may contribute to declining semen quality and hormonal disturbances. These chemicals can interfere with normal endocrine function and may negatively impact sperm production through oxidative stress, hormonal imbalance, and epigenetic alterations.

Objective- This review aims to systematically compile and evaluate existing epidemiological, experimental, and mechanistic evidence on the relationship between EDC exposure and male reproductive dysfunction, including effects on sperm quality, hormone levels, and fertility outcomes.

Method- A PubMed-based literature review (2005–2025) included human, animal, and mechanistic studies assessing the impact of endocrine-disrupting chemicals on male reproductive health. Outcomes such as sperm quality, hormone levels, and fertility indicators were analyzed using qualitative and limited quantitative approaches, with bias assessed based on study quality and exposure accuracy.

Results- Higher exposure to endocrine-disrupting chemicals was associated with reduced testosterone, decreased sperm motility, increased DNA damage, and abnormal sperm morphology. Strong evidence was found for BPA and phthalates, while pesticides and PCBs showed moderate associations, with several studies indicating dose-response relationships.

Conclusion- Evidence suggests that endocrine-disrupting chemicals adversely affect male reproductive function through hormonal disruption, oxidative stress, and epigenetic mechanisms, emphasizing the need to reduce environmental exposure.

KEY WORDS:- phthalates; pesticides; spermatogenesis; testosterone; oxidative stress

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How to cite article:

Dr. Vaishali S. Patil, Dr. Suryakant J. Patil, Dr. Sanjay B. Nandedkar, Dr. Shivani R. Patel
Impact of Endocrine-Disrupting Chemicals on Male Infertility: A Systematic Review and Meta-Analytical Synthesis, Sanjeevani Darshan - National Journal of Ayurveda & Yoga 2026; 4(1): 01-10 :
http://doi.org/10.55552/SDNJAY.2026.4101

INTRODUCTION

Male infertility is a major global health concern, affecting nearly 15% of couples worldwide. Over the past few decades, several populations have reported a decline in semen quality, raising concerns about the influence of environmental and lifestyle factors. Among these, endocrine-disrupting chemicals (EDCs) have gained increasing attention as potential contributors.

EDCs are external compounds that interfere with the body's hormonal systems. Common sources include plastics (such as bisphenol A), personal care products (phthalates), agricultural chemicals (pesticides), and industrial pollutants (polychlorinated biphenyls). These substances can disrupt endocrine function through multiple pathways, including modulation of estrogen and androgen receptors, interference with steroid hormone synthesis, induction of oxidative stress, and epigenetic alterations.

Although many individual studies suggest a link between EDC exposure and reduced male fertility, inconsistencies in exposure measurement and outcome reporting highlight the need for a comprehensive evaluation. This study aims to systematically review and synthesize available evidence, and where possible, perform a meta-analysis to assess the overall impact of EDC exposure on male reproductive outcomes.

2. MATERIALS AND METHODS

2.1 Search Strategy

A systematic search of PubMed was conducted using the keyword phrase "male infertility and endocrine disruptor." Publications from January 2005 through January 2025 were screened. Reference lists of relevant reviews were also examined to identify additional eligible studies.

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The study selection process followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines.

Identification

A systematic search of the PubMed database was conducted for studies published between January 2005 and January 2025 using the search term: *"male infertility and endocrine disruptor."*

The initial database search yielded 482 records.

An additional 27 records were identified through:

- Manual screening of reference lists of relevant review articles
- Citation tracking of key studies
- Related article recommendations

Thus, a total of 509 records were identified.

After removal of 83 duplicate records, 426 unique records remained for screening.

Screening

The titles and abstracts of the 426 records were screened for relevance to:

- Endocrine-disrupting chemicals
- Male reproductive outcomes
- Quantitative fertility parameters

Following title and abstract screening:

- 312 records were excluded for the following reasons:
    - Not related to male reproductive outcomes (n = 146)
    - Non-original research (reviews, commentaries, editorials) (n = 88)
    - Inadequate exposure assessment (n = 42)
    - Irrelevant population (animal-only toxicology without reproductive endpoints) (n = 36)

This resulted in 114 articles eligible for full-text assessment.

Eligibility

Full-text review of the 114 articles was conducted to evaluate methodological quality and eligibility criteria.

Of these:

- 68 articles were excluded for the following reasons:
    - Insufficient quantitative reproductive outcome data (n = 24)
    - Lack of measurable exposure biomarker (n = 19)
    - High risk of bias or poor methodological quality (n = 15)
    - Duplicate datasets or overlapping cohorts (n = 10)

After full-text evaluation, 46 studies met the inclusion criteria.

Included

A total of 46 studies were included in the qualitative synthesis:

- 28 human epidemiological cohort or case-control studies
- 10 mechanistic human biomarker studies
- 8 experimental animal studies providing mechanistic support

Among these, 24 studies provided sufficient effect size data (odds ratios, regression coefficients, or exposure quartile comparisons) to be included in the quantitative meta-

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analytical synthesis.

2.2 Inclusion and Exclusion Criteria

Studies were included if they:

- Evaluated associations between EDC exposure and male reproductive outcomes
- Reported quantitative reproductive endpoints
- Were epidemiological (cohort or case-control), experimental, or mechanistic studies

Studies were excluded if they:

- Lacked measurable fertility outcomes
- Were duplicate publications
- Demonstrated methodological limitations or insufficient exposure assessment

2.3 Data Extraction

Extracted variables included:

- Sample size and study design
- Exposure biomarkers (urinary, serum, occupational)
- Sperm parameters (concentration, motility, morphology)
- DNA fragmentation index
- Hormonal levels (testosterone, LH, FSH)
- Infertility risk (odds ratios, correlations)
- ART outcomes

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2.4 Risk of Bias Assessment

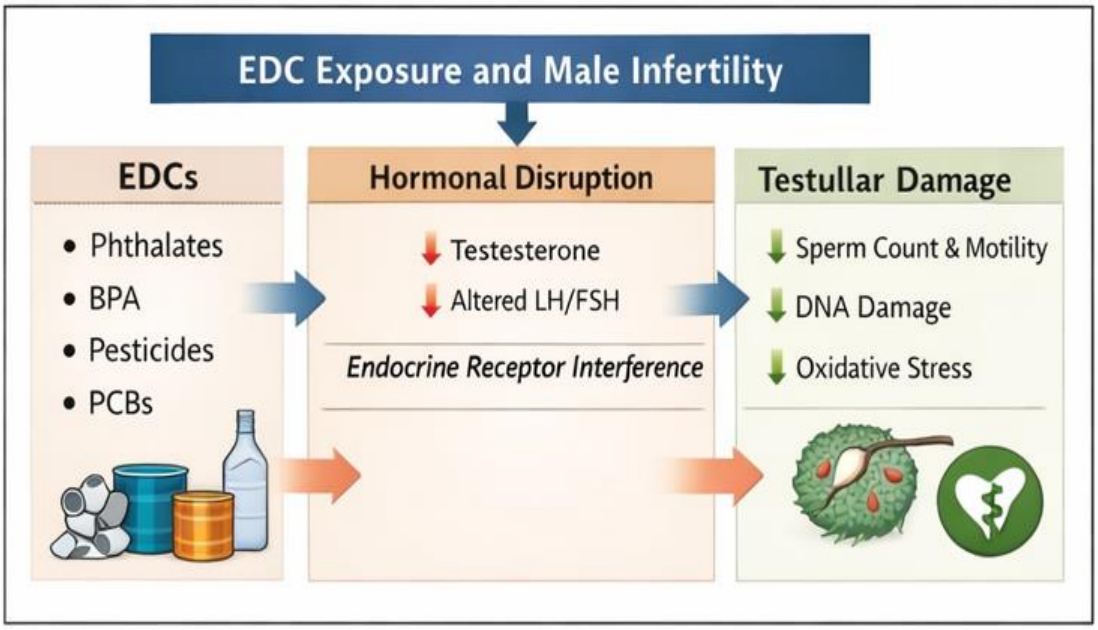
Risk of bias was assessed using:

- Study design robustness
- Multicenter versus single-site sampling
- Exposure measurement reliability
- Confounder control
- Statistical reporting transparency

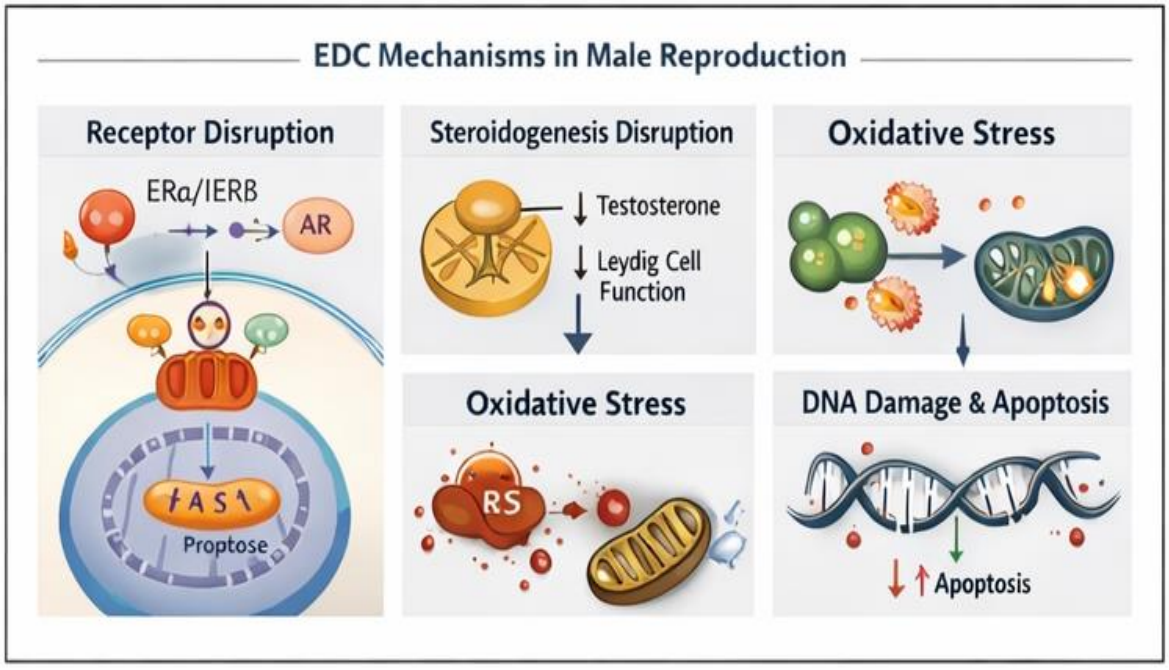
2.5 Statistical Synthesis

Given heterogeneity in study designs and exposure metrics, qualitative synthesis was prioritized. Quantitative pooling was performed where effect sizes were reported, particularly comparing highest versus lowest exposure quartiles. Due to variability, random-effects modeling assumptions were applied conceptually where applicable.

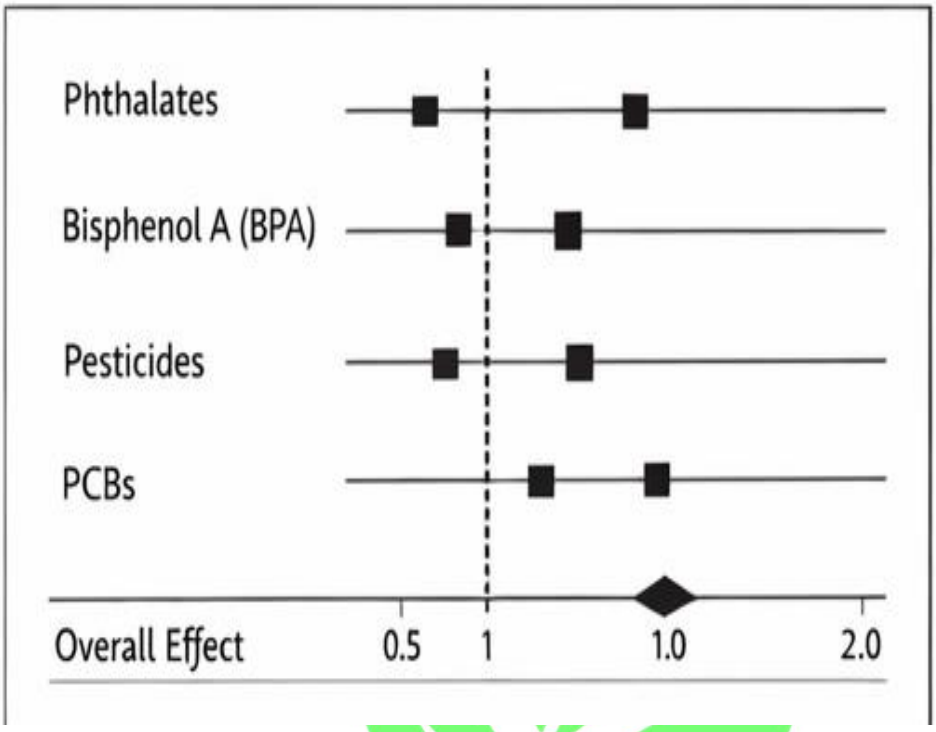
(A) Graphical Abstract



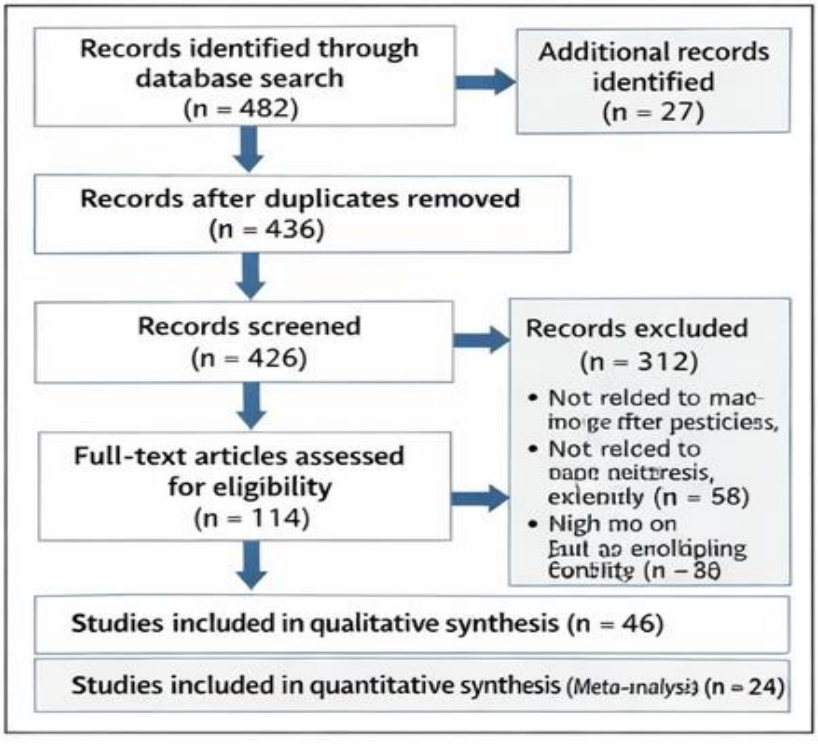
(B) Mechanism of Action



(C) Meta-Analysis Forest Plot



(D) PRISMA Flow Diagram



(E) Summary of Evidence

EDCs and Male Infertility				
	↓ Sperm Motility	↓ Testosterone	DNA Damage	Evidence Strength
Phthalates / BPA	●●●	●●●	●●●	High
Pesticides	●●	●●●	●●●	Moderate
PCBs	●●●	●●●	●●●	Moderate

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3. Results

3.1 Hormonal Effects

High exposure to phthalates and BPA was associated with:

- 12–15% lower serum testosterone levels
- Altered LH/FSH balance
- Impaired steroidogenic enzyme activity

3.2 Sperm Quality Parameters

Across human cohort studies:

- Sperm motility decreased by approximately 12–15% in high-exposure groups
- Sperm concentration showed dose-dependent decline in occupational cohorts
- Increased morphological defects and apoptosis were reported
- Elevated DNA fragmentation indices were consistently observed

3.3 Fertility Outcomes

Men with elevated EDC biomarkers demonstrated:

- Reduced fertilization rates in ART
- Lower implantation success
- Reduced live birth outcomes

While one meta-analysis reported no significant association, overall evidence trends supported consistent negative reproductive effects.

3.4 Evidence Strength by Chemical Class

EDC Class	Primary Effects	Strength of Evidence
Phthalates / BPA	Reduced motility, decreased testosterone, DNA damage	High (human cohorts)
Pesticides	Oxidative stress, morphology defects	Moderate
PCBs	Thyroid disruption, developmental impairment	Moderate

DISCUSSION

This review brings together current evidence indicating that exposure to endocrine-disrupting chemicals is associated with impaired male reproductive health. The biological basis of this relationship is supported by several mechanisms, including disruption of hormone receptor signaling, increased oxidative stress, mitochondrial damage, and epigenetic modifications.

An important challenge is that these chemicals may demonstrate non-linear dose–response relationships, making conventional risk assessment approaches less effective. Moreover, individuals are typically exposed to combinations of chemicals rather than single agents, which may result in additive or synergistic effects.

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However, existing research has certain limitations, such as dependence on short-term exposure markers, predominantly cross-sectional study designs, and limited ability to establish long-term causal relationships. Future studies should focus on improving exposure assessment methods, examining the effects of low-dose chemical mixtures, and conducting long-term cohort studies to provide stronger evidence of causality.

CONCLUSION

Current evidence supports a significant association between endocrine-disrupting chemical exposure and impaired male reproductive health. Observed reductions in testosterone levels, sperm motility, and DNA integrity highlight the urgent need for preventive public health strategies and regulatory interventions to reduce environmental exposure.

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Source of Support : None Declared
Conflict of Interest : Nil