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Perspective

Drinking water safety: the overlooked endocrine-disrupting effects of disinfection byproducts

Zhihan Liu and Qiang Zeng*

*E-mail: zengqiang506@hust.edu.cn

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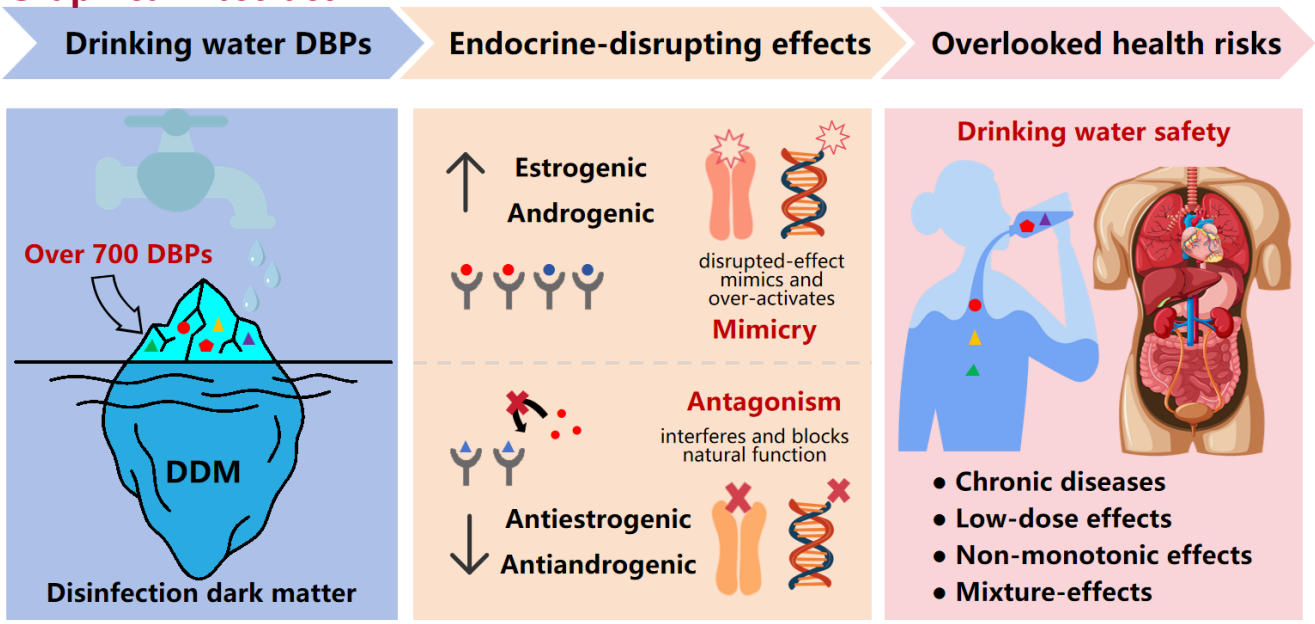
Graphical Abstract



Highlights:

- Accumulating evidence suggests that DBPs exert potential endocrine-disrupting effects.
- Potential contribution of DBP exposures to chronic diseases warrant investigation.
- Drinking water safety should consider the endocrine-disrupting potential of DBPs.

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Drinking water safety: the overlooked endocrine-disrupting effects of disinfection byproducts

Zhihan Liu[1,2] and Qiang Zeng[1,2,*]

[1] Department of Occupational and Environmental Health, School of Public Health, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

[2] Key Laboratory of Environment and Health, Ministry of Education & Ministry of Environmental Protection, and State Key Laboratory of Environmental Health (incubating), School of Public Health, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

***E-mail:** zengqiang506@hust.edu.cn.

Abstract: Disinfection byproducts (DBPs), as ubiquitous chemical contaminants in drinking water, pose a significant threat to public health owing to their documented genotoxicity, carcinogenicity, and reproductive and developmental toxicity. However, the endocrine-disrupting effects of DBPs have long remained overlooked. Recently, accumulating evidence from both experimental and human studies has suggested that certain DBPs can exert estrogenic, androgenic, antiestrogenic, and antiandrogenic activities, implicating that DBPs elicit potential endocrine-disrupting effects. Given that endocrine-disrupting properties represent key biological mechanisms contributing to a broad spectrum of adverse health outcomes, this perspective contends that exposure to drinking water DBPs may underpin overlooked health risks including chronic diseases, highlighting an urgent paradigm shift in how we evaluate drinking water safety. Knowledge gaps and future directions on the endocrine-disrupting effects of DBPs are also proposed.

Keywords: disinfection byproducts; drinking water safety; endocrine-disrupting effects; health risks

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1. Introduction

Disinfection of drinking water can ensure drinking water safety by virtually eliminating the spread of waterborne diseases, which has been hailed as one of the greatest public health achievements of the 20th century [1]. However, a myriad of unintended chemicals are formed from the reaction between disinfectants and natural organic matter or inorganic substance in water, named as disinfection byproducts (DBPs). To date, more than 700 DBPs have been identified in disinfected drinking water [1], yet these represent merely the "tip of the iceberg" [2]. Beneath this visible surface lies a vast and largely uncharted terrain, what we firstly term disinfection dark matter (DDM)—comprising unknown or structurally uncharacterized DBPs whose presence, fate, and health risks remain fully unexplored.

DBPs have been documented to possess multiple health risks including genotoxicity, carcinogenicity, and reproductive and developmental toxicity. Importantly, epidemiological evidence has fairly shown that exposure to DBPs is associated with elevated risk of bladder cancer [3–5], and emerging human studies relying on measures of blood and urinary biomarkers have consistently suggest that exposure to DBPs is associated with reduced fertility and adverse birth outcomes [6,7]. To mitigate the potentially health risks linked with daily exposure to drinking water DBPs, certain prevalent DBPs such as trihalomethanes (THMs) and haloacetic acids (HAAs) have been regulated by the World Health Organization, the United States Environmental Protection Agency, the European Union, and China.

However, a more insidious dimension of DBPs with their capacities to disrupt the endocrine system has remained largely outside the research spotlight and regulatory. Increasing experimental and human studies have documented that certain DBPs can exert potential endocrine-disrupting effects, including estrogenic, androgenic, antiestrogenic, and antiandrogenic activities, as well as altered hormone levels. This perspective contends that the endocrine-disrupting potential of DBPs may pose new threats or challenges to the drinking water safety and underscore urgent scientific scrutiny and regulatory reconsideration. Furthermore, knowledge gaps and future directions on the endocrine-disrupting effects of DBPs are also proposed.

2. Beyond carcinogenicity and genotoxicity: the overlooked endocrine-disrupting effects

Traditional health risk assessment of DBPs have largely focused on carcinogenicity and genotoxicity. Indeed, a larger number of epidemiological studies have reported that exposure to DBPs is linked with bladder and colorectal cancers, while toxicological studies have documented

DNA damage and oxidative stress as main modes of action. However, these fail to explain a growing body of human evidence on associations between exposure to DBPs and adverse reproductive and developmental outcomes, including declined semen quality, impaired ovarian function, and adverse birth outcomes such as spontaneous abortion, low birth weight, and fetal growth retardation. These endpoints that are classically hallmarks of endocrine disruption rather than carcinogenicity and genotoxicity.

The endocrine hormones regulate various physiological processes including reproduction, growth, metabolism, immune function, and neurodevelopment. Endocrine-disrupting chemicals (EDCs) can disrupt the endocrine system by interfering with hormone synthesis, transport, receptor binding, and metabolism, leading to a wide range of adverse health outcomes, not limited to reproductive and developmental problems. Recently, there has been growing concern on the endocrine-disrupting effects of DBPs [8]. Experimental studies have documented that certain DBPs elicit endocrine-disrupting potential (Table 1). Specifically, in mice models dichloroacetic acid (DCAA) and trichloroacetic acid (TCAA), the two most abundant HAAs, were found to reduce the levels of testicular androgen receptor (AR) and serum testosterone in male mice, as well as reduce the levels of ovarian, uterus, and placenta estrogen receptors (ERα and ERβ) and serum estradiol and progesterone in pregnant mice [9]. In *in vitro* of human breast cancer cells, a comprehensive evaluation was conducted on estro-androgenic disrupting effects of 43 DBPs, and the results indicated that 16 DBPs induced estrogenic, androgenic, antiestrogenic, and antiandrogenic effects and these effects were even observable at environmentally relevant concentrations commonly detected in finished drinking water [10]. Moreover, gestational and lactational exposure to iodoacetic acid (IAA) exerted endocrine-disrupting effects on F1 female offspring, as evidenced by elevated testosterone and suppressed progesterone and follicle-stimulating hormone (FSH) levels at postnatal day (PND) 21 [11].

Table 1. Selected experimental evidence on endocrine-disrupting potential of DBPs.

Author (Year)	Experimental models	Exposure patterns	Main findings
Chen *et al.* (2024) [9]	*In vivo*: C57BL/6J male mice and pregnant female mice	Chemical: DCAA and TCAA Route: Oral gavage Dose: 0, 1, 10^2, 10^3, 10^4, 5 × 10^4-fold MCL (DCAA: up to 1.32 g/kg/d; TCAA: Up to 2.64 g/kg/d) Duration: 14 consecutive days	In males, DCAA (≥ 26.4 mg/kg/d) and TCAA (≥ 52.7 mg/kg/d) decreased serum testosterone and testicular AR protein levels. In pregnant females, DCAA (≥ 264 mg/kg/d) and TCAA (≥ 52.7 mg/kg/d) increased miscarriage rates. At ≥ 10^3-fold MCL, DCAA/TCAA elevated serum FSH and decreased serum estradiol and progesterone. Ovarian ERα and ERβ protein levels were downregulated at ≥ 10^2-fold MCL (DCAA ≥ 2.64 mg/kg/d; TCAA ≥ 5.27 mg/kg/d), with tissue-dependent ER reduction in uterus and placenta.
Chen *et al.* (2024) [10]	*In vitro* bioassays (human breast cancer cell lines)	Chemical: 43 halogenated DBPs (HAAs, HANs, HAcAms, halophenols and other cyclic DBPs) Dose: 10^{-15}–10^{-3} M (up to non-cytotoxic < EC_{10} for each DBP) Duration: 24 h	Among the 43 tested DBPs, 16 exhibited estrogenic, anti-estrogenic, androgenic, or anti-androgenic activities. Endocrine-disrupting potencies followed the rank order of I-DBPs > Br-DBPs > Cl-DBPs, with polyhalogenated and halophenolic DBPs identified as potential EDCs, and certain I-DBPs (2,4,6-triiodophenol) exhibiting dual agonistic/antagonistic activities.
Gonsioroski *et al.* (2022) [11]	*In vivo*: CD-1 mice (F0 dams and F1 female offspring)	Chemical: IAA Route: Maternal drinking water Dose: 0, 10, 100, 500 mg/L (40–1500 μg/d) Duration: Premating through gestation and lactation (PND 21)	Maternal IAA exposure (10 and 100 mg/L) delayed vaginal opening and increased anogenital distance index (500 mg/L) in F1 female pups. At PND 21, IAA exposure (10 and 100 mg/L) elevated serum testosterone, and showed a borderline decrease in progesterone (10 mg/L).
Goldman & Murr (2003) [12]	*In vivo*: Female SD rats (intact & ovariectomized/ steroid-primed)	Chemical: DBAA Route: Oral gavage Dose: 0, 30, 60, 90, 120, 270 mg/kg/d Duration: 14 consecutive days	In intact cycling rats, DBAA at 270 mg/kg/d disrupted estrous cyclicity (persistent estrus/diestrus) and dose-dependently elevated circulating estradiol on the day of estrus. In ovariectomized, estradiol-implanted rats, DBAA dose-dependently elevated circulating estradiol.
Carr *et al.* (2011) [13]	*In vivo* (male SD rats) & *In vitro* (primary rat Leydig cells)	Chemical: DBAA Route: Oral gavage (rats)/Medium (cells) Dose: *In vivo*: 250 mg/kg/d; *In vitro*: 10, 100 μM Duration: *In vivo*: 4 d; *In vitro*: 2 h hCG-stimulated	*In vivo*, oral exposure to DBAA decreased testicular testosterone levels. *In vitro*, DBAA at 100 μM inhibited hCG-stimulated testosterone production in primary Leydig cells.
Gonsioroski *et al.* (2021) [14]	*In vivo*: Adult female CD-1 mice	Chemical: IAA Route: Drinking water Dose: 0, 0.5, 10, 100, 500 mg/L Duration: 35–40 consecutive days	Subchronic IAA exposure at 500 mg/L significantly shortened proestrus duration, reduced serum estradiol and androstenedione levels, and upregulated ovarian Esr1 mRNA expression.
Tian *et al.* (2024) [15]	*In vitro*: Mouse Leydig cells (MLTC-1) and human HepG2 cells	Chemical: DCAA and TCAA Dose: 12, 60, 300 nM Duration: 48 h	In MLTC-1 cells, low-dose DCAA/TCAA (12 and 60 nM) induced testosterone/androstenedione production, while high-dose TCAA (300 nM) inhibited steroidogenesis.

Abbreviations: Br-DBPs, brominated disinfection byproducts; Cl-DBPs, chlorinated disinfection byproducts; DBAA, dibromoacetic acid; ER, estrogen receptor; Esr1, estrogen receptor 1; HAcAms, haloacetamides; HANs, haloacetonitriles; hCG, human chorionic gonadotropin; I-DBPs, iodinated disinfection byproducts; MCL, maximum contaminant level.

Epidemiological studies have also suggested that exposure to DBPs is associated with altered hormone levels (Table 2). For example, a cross-sectional study among men attending an infertility clinic for semen analysis revealed positive associations between urinary DCAA and TCAA and urinary testosterone, progesterone, and estradiol [15]. A case-control study among healthy women and diminished ovarian reserve, serum concentrations of all 6 DBPs [dibromoacetic acid (DBAA), monochloroacetic acid (MCAA), DCAA, TCAA, chlorate, perchlorate] were found to be negatively associated with serum anti-Mullerian hormone (AMH) and positively associated with basal FSH [16]. In our previous study among men attending an infertility clinic for semen analysis, blood dibromochloromethane (DBCM) showed a suggestive inverse dose-response relationship with serum total testosterone [17]. Our recent human studies among women undergoing assisted reproductive technology suggested that urinary TCAA was associated with decreased serum AMH levels [18], while urinary DCAA showed U-shaped associations with serum progesterone and prolactin levels [19]. These findings from both experimental and epidemiological evidence, implicate selected DBPs as potential EDCs and call for more toxicological and epidemiological evaluations.

Table 2. Selected epidemiological evidence on endocrine-disrupting potential of DBPs.

Author (Year)	Study design	Study population	Exposure assessment	Main findings
Tian *et al.* (2024) [15]	Cross-sectional study	Adult men attending an infertility clinic in Wuhan, China (N = 449)	Creatinine-adjusted urinary DCAA and TCAA concentrations from repeated spot urine samples	Urinary DCAA and TCAA were positively associated with urinary testosterone, progesterone, and estradiol.
Tian *et al.* (2025) [16]	Case-control study	Women undergoing IVF/ICSI treatment in Beijing, China (N = 182; 91 DOR cases and 91 age-matched controls)	Serum concentrations of 6 DBPs (MCAA, DCAA, TCAA, DBAA, chlorate, perchlorate)	Serum concentrations of all 6 DBPs were negatively associated with serum AMH, and were positively associated with basal FSH.
Zeng *et al.* (2013) [17]	Cross-sectional study	Men attending an infertility clinic for semen analysis in China (N = 401)	Baseline whole-blood concentrations of 4 THMs (TCM, BDCM, DBCM, TBM)	Blood DBCM showed a suggestive inverse dose-response relationship with serum total testosterone.
Deng *et al.* (2022) [18]	Cross-sectional study	Women seeking assisted reproductive technology at a reproductive center in China (N = 956)	Specific gravity-adjusted urinary DCAA and TCAA concentrations	Elevated urinary TCAA levels were monotonically associated with decreased AMH.
Liu *et al.* (2022) [19]	Cross-sectional study	Women undergoing assisted reproductive technology in China (N = 725)	Creatinine-adjusted urinary DCAA and TCAA concentrations	Urinary DCAA was positively associated with serum progesterone. Non-linear U-shaped associations were observed between urinary DCAA and both serum progesterone and prolactin.

Abbreviation: AMH, anti-Müllerian hormone; BDCM, bromodichloromethane; DOR, diminished ovarian reserve; ICSI, intracytoplasmic sperm injection; IVF, *in vitro* fertilization; N, sample size; TBM, tribromomethane (bromoform); TCM, trichloromethane (chloroform).

3. Knowledge gaps and future directions

Endocrine disruption has been recognized as a key biological pathway linking environmental chemical exposures to multiple chronic diseases, including metabolic disorders, cardiovascular diseases, immune dysfunction, type 2 diabetes, obesity, and neurodevelopmental disorders [20]. Despite this recognition, the potential contribution of DBP exposures to these disease endpoints remains strikingly understudied [21,22]. Given the ubiquitous nature of daily DBP exposures across populations relying on disinfected drinking water, future studies should incorporate endocrine-sensitive endpoints into the health risk assessments of DBP exposures across life stages (adulthood, pregnancy, and early life), including outcomes related to metabolism, cardiovascular function, immune function, and neurodevelopment. Specifically, because endocrine disruptors are known to exert their most consequential effects during critical developmental windows, the potential effects of DBP exposures during early childhood warrant investigation. Furthermore, given that certain DBPs have been found to elicit biological effects even at environmentally relevant concentrations, future investigations should place greater emphasis on elucidating their health implications under realistic and long-term exposure scenarios. Mechanistic toxicology is also warranted to distinguish direct endocrine effects from secondary and non-specific toxicity [23,24].

A fundamental obstacle to understanding the endocrine-disrupting effects of DBPs is the sheer complexity of real-world exposure scenarios. Drinking water is not a matrix of isolated chemical entities but rather a dynamic mixture of thousands of compounds. The additive, synergistic, or antagonistic effects of these mixtures in

modulating endocrine pathways remain largely unknown [25]. This mixture conundrum poses profound challenges for both toxicity testing and epidemiological investigation, necessitating the development of integrated analytical and bioanalytical approaches to capture the aggregate endocrine activity of whole water samples. Both targeted and non-targeted screening approaches can be leveraged to establish valid internal exposure biomarkers for low-dose chronic DBP mixture exposures. To this end, future investigations should comprehensively evaluate the health effects associated with such combined exposures.

The recognition of DBPs with endocrine-disrupting potential has profound implications for drinking water regulation. Current regulatory frameworks focus primarily on a handful of regulated DBPs and rely predominantly on risk assessments of carcinogenicity or reproductive toxicity. This traditional paradigm overlooks the unique endocrine-disrupting properties of DBPs, which may inadequately protect public health. To bridge this regulatory gap, systematic screening for endocrine-disrupting activities should be extended across the broader DBP inventory, with prioritized attention to emerging iodinated, nitrogenous, and aromatic DBPs, given their heightened toxicological potential and largely uncharacterized endocrine effects.

Moreover, regulatory standards should consider the potential for effects at low, environmentally relevant exposure levels, recognizing that DBPs with endocrine-disrupting potential likely follow non-monotonic dose-response relationships. This means that conventional margin-of-exposure calculations and safety factors, which assume that lower exposures are proportionally safer, may be fundamentally invalid for assessing endocrine-mediated risks from DBP exposures. Consequently, a precautionary yet evidence-based revision of the current regulatory philosophy is warranted—one that aligns the assessment of drinking water safety with the understanding of endocrine disruption as a significant and historically overlooked dimension of DBP toxicity.

4. Conclusion

Emerging evidence that DBPs possess endocrine-disrupting potential fundamentally redefines our understanding of what constitutes "safe" drinking water. These compounds are not merely carcinogens with a safe threshold; they are hormonal modulators that can perturb physiological systems at levels commonly found in drinking water. The potential implications for chronic disease burden spanning reproductive disorders, metabolic diseases, immune dysfunction, and neurodevelopmental disorders, which demand a concerted and interdisciplinary research effort and a regulatory response commensurate with the magnitude of the threat.

Declaration of generative AI and AI-assisted technologies

The authors used DeepSeek for polishing the language during the manuscript draft. The authors take full responsibility for the content of the manuscript.

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Conflicts of interest

Qiang Zeng holds the position of Associate Editor for *International Journal of Environmental Epidemiology* and has not peer reviewed or made any editorial decisions for this paper. The authors declare no other competing financial interests or personal relationships.

Authors' contribution

Z.L.: data curation, visualization and writing—review and editing; Q.Z.: conceptualization, methodology, supervision, project administration, writing—original draft and writing—review and editing. All authors have read and agreed to the published version of the manuscript.

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