



International Journal of Veterinary Sciences and Animal Husbandry



ISSN: 2456-2912

NAAS Rating (2026): 4.61

VET 2026; 11(2): 618-623

© 2026 VET

www.veterinarypaper.com

Received: 12-12-2025

Accepted: 18-01-2026

Swati Koli

Department of Pharmacology
and Toxicology, College of
Veterinary Science & A.H.,
Mhow, NDVSU, Jabalpur,
Madhya Pradesh, India

Gayatri Dewangan

Department of Pharmacology
and Toxicology, College of
Veterinary Science & A.H.,
Mhow, NDVSU, Jabalpur,
Madhya Pradesh, India

Shweta Rajoriya

Department of Veterinary
Physiology and Biochemistry,
College of Veterinary Science &
A.H., Mhow, NDVSU, Jabalpur,
Madhya Pradesh, India

RS Tayde

Department of Veterinary Public
Health, College of Veterinary
Science & A.H., Mhow, NDVSU,
Jabalpur, Madhya Pradesh,
India

Rashmi Chaudhary

Department of Veterinary
Pathology, College of Veterinary
Science & A.H., Mhow, NDVSU,
Jabalpur, Madhya Pradesh,
India

Rajeev Ranjan

Department of Pharmacology
and Toxicology, College of
Veterinary Science & A.H.,
Rewa, NDVSU, Jabalpur,
Madhya Pradesh, India

Corresponding Author:

Rajeev Ranjan

Department of Pharmacology
and Toxicology, College of
Veterinary Science & A.H.,
Rewa, NDVSU, Jabalpur,
Madhya Pradesh, India

Mercury exposure as a determinant of female infertility: Mechanistic insights and reproductive outcomes

Swati Koli, Gayatri Dewangan, Shweta Rajoriya, RS Tayde, Rashmi Chaudhary and Rajeev Ranjan

DOI: <https://www.doi.org/10.22271/veterinary.2026.v11.i2g.3082>

Abstract

Mercury is the third most toxic heavy metal affecting ecosystems and human health. This review examines mercury's toxic effects on female reproductive function through acute and chronic pathways. Mercury exposure occurs through various sources and leads to infertility and reduced fertility in females. The primary mechanisms underlying mercury-induced infertility include oxidative stress, endocrine disruption, mitochondrial dysfunction, and direct effects on ovarian tissue. This review highlights recent evidence linking mercury exposure to female infertility and underscores the need for improved biomonitoring, risk assessment, and public health strategies to mitigate exposure. Understanding the molecular mechanisms involved in female infertility is essential for developing targeted therapeutic and preventive interventions to protect female reproductive health.

Keywords: Mercury, infertility, endocrine disruptor, oxidative stress, molecular mechanisms

Introduction

Mercury is the third-ranking element detrimental to human health according to the United States Agency for Toxic Substances and Disease Registry. It is characterized as a persistent, bio-accumulative and toxic contaminant [4]. It exists in various physical and chemical forms, primarily elemental mercury and cationic forms, namely mercurous and mercuric ions. Elemental mercury exists as a liquid at room temperature while the mercuric form is the most prevalent cationic form of mercury in occupational and environmental settings. Methylmercury, formed by the methylation of inorganic (mercuric) forms of mercury by microorganisms in soil and water is recognized as the most commonly encountered organic mercuric compound in the environment [55]. Mercury, when released into the environment, contaminates the air, water, and soil, affecting the global food chain [7]. It persistently bioaccumulates and biomagnifies within the ecosystem [38].

The global mercury assessment in the year 2018 revealed that mining operations are the primary contributors to atmospheric mercury emissions, followed by stationary industrial facilities fueled by fossil fuels and fixed domestic burning. Additionally, other sources of mercury include cement production, non-ferrous metal production, large-scale gold production, vinyl-chloride monomer, biomass burning, iron and steel production, chlor-alkali production, oil refining and waste incineration [46]. Mercury fumes and mercury-laden dust in electronics waste and lamp recycling sectors also present a risk of exposure to elemental mercury through inhalation by workers [59]. In addition to thermometers, thermostats, and dental amalgam, old paint supplies and certain fungicides contain mercury compounds [45]. Pharmaceuticals, such as mercurochrome and merthiolate, as well as algacides and childhood vaccines are additional potential sources of mercury toxicity [54].

Health Implications of Mercury Exposure in Females

Environmental mercury exposure has been found to have profound cellular, cardiovascular,

haematological, renal, pulmonary, immunological, neurological, reproductive, genotoxicity, embryotoxicity, endocrine disruption, cancer promotion, pregnancy and reproductive system damage in humans [53,68]. Mercury and its forms, even in small doses, affect the nervous, respiratory, and cardiovascular systems. It can cause disorders such as cancers, endothelial dysfunction, gastric and vascular disorders, hormonal imbalances, miscarriages, and reproductive disorders, skin lesions, vision damage and even death [6]. The brain and kidney are the target organs for long-term exposure to mercury [49].

Mercury has negative effects on fertility in both men and women [40]. Individuals consuming substantial amounts of fish may have low-level and long-term exposure to methylmercury (MeHg), leading to a considerable public health concern. Such chronic exposure to MeHg in utero by infants leads to the dysfunction of the central nervous system (CNS), especially the cerebral and cerebellar cortex [29]. In addition to female infertility, mercury exposure is linked with conditions like polycystic ovary syndrome, premenstrual syndrome, dysmenorrhea, amenorrhea, early menopause, endometriosis, benign breast cancers, and galactorrhea [71, 28, 47]. High mercury levels in women's blood are correlated with infertility. Similar studies have shown that 80% of infertile women and 68% of pregnant women had hair methylmercury levels above the reference dose of 1 mg/kg set by the US Environmental Protection Agency, which also correlated with their daily methylmercury exposure [24]. Women inhabiting the Gangetic plains of Bihar had blood, urine and breast milk mercury levels above the WHO permissible limits. This depicts that women infants fed via breastfeeding by such women are likely at health risk. Their infants' urine had higher mercury contamination with complete accumulation of mercury in their bodies [34].

Effect of Mercury on Fertility and Pregnancy Outcomes

In recent years, infertility has emerged as a significant medical and social concern. A variety of health conditions, including women's age, tubal disease, ovulation disorders, and uterine abnormalities, in conjunction with lifestyle factors such as the consumption of alcohol and smoking, as well as environmental stressors, have significantly increased the prevalence of infertility on a global scale [50]. Multiple toxicants contribute to the impact on fertility; however, the primary factor is widely regarded as stemming from exposure to heavy metals, particularly mercury. Elevated mercury levels have been associated with infertility and decreased fertility [22]. Mercury acts as an endocrine disruptor affecting the hypothalamus, pituitary gland, ovary, adrenal gland, thyroid gland, etc. [62, 26]. Prenatal mercury exposure may influence pregnancy or its outcome, and this effect seems to be contingent upon the dose, duration, and timing of exposure. The nutritional status of an exposed individual might also influence the impact of mercury [35]. The administration of nearly all chemical forms of mercury during and even before gestation resulted in reproductive problems, such as stillbirths, spontaneous abortion, congenital malformations, infertility, disturbances in the menstrual cycle, inhibition of ovulation and behavioural effects in the offspring [33, 66, 67]. Furthermore, occupational mercury vapour exposure is hazardous to pregnant female workers, increasing the risk of adverse reproductive outcomes, especially congenital anomalies [48]. Mercury vapours can cross the placental barrier, so pregnant women exposed to mercury vapour are at

an increased risk of adverse reproductive outcomes [16, 42, 9]. Mercury exposure causes damage to ovarian follicles and diminishes ovarian reserve due to oxidative stress. Pregnant women exposed to methylmercury (MeHg) mainly via diet and or inhalation, the absorbed MeHg crosses the placenta and accumulates in fetal tissues, causing neural tube defects, craniofacial malformations, and delayed fetal growth [73].

Effect of Mercury on Oogenesis and Ovarian Function

Mercury exposure negatively impacts reproductive health by altering oogenesis and ovarian function. In rat ovaries, mercury has been reported to be deposited particularly in macrophages of atretic young and mature ovarian follicles and corpora lutea, within granulosa cells of atretic follicles and in the lutein cells of freshly formed corpora lutea [61]. In animal models, inorganic mercury exposure causes anovulation in hamsters [11], prolonged diestrus up to 10 days in rats, with evidence of mercury accumulation in the ovaries [61]. Similarly, high doses of mercury cause adverse effects on the mouse ova [36]. Mercuric chloride (HgCl_2) has been found to delay the meiosis of mouse ova *in vitro* [27]. Female hamsters subcutaneously injected with very high doses of HgCl_2 (6.4 and 12.8 mg/kg) showed a slight increase in the incidence of chromosome aberrations in bone marrow, inhibition of ovulation, despite a low HgCl_2 concentration in the ovaries, and an increase in the number of degenerated oocytes after 12.8 mg/kg injection [65]. Additionally, mercury has been documented to alter female gonadal function in fish, mice, and hamsters. Studies on fish subjected to mercury-contaminated water have demonstrated a decline in gonadal steroidogenesis and pituitary gonadotropin-releasing levels, inhibition of ovarian maturation, a reduction in ovarian weight, and a decrease in both the quantity and size of oocytes. However, the process of meiosis in ova was impacted by mercury in both mice and hamsters [43, 51, 75].

Methylmercury chloride (MMC) markedly decreases oocyte maturation during *in vitro* cultivation of mouse oocytes. The defects in oocyte maturation induced by MMC exposure can be attributed to impaired cytoskeleton dynamics and DNA damage [14]. Additionally, *in vivo* studies in mice revealed that mercury exposure impairs oocyte maturation due to the hyperactivation of mitochondrial autophagy via the monophosphate-activated protein kinase (AMPK)/mammalian target of rapamycin (mTOR) signal pathway induced by mitochondrial dysfunction [23].

Mercury-induced Molecular Signalling Pathways in Females

Methylmercury alters intracellular calcium concentration by disrupting the regulation of calcium from the intracellular calcium pool and increasing the permeability of the plasma membrane to Ca^{2+} [2]. Mercury has also been shown to have calcium-mimetic effects [20] and to alter intracellular calcium homeostasis [3]. Mercury influences the binding of hormones by their receptors. Mercuric chloride (HgCl_2) has been shown to inhibit estradiol binding to rat uterine nuclear receptors, and the presence of divalent ions enhanced the binding [30]. Toxic effects of mercury and mercury-containing xenobiotics occur through interference with cellular and molecular mechanisms, leading to oxidative stress, DNA damage, mitochondrial damage, apoptosis, epigenetic alterations, and impaired signalling pathways [44].

Female rats exposed to mercuric chloride (HgCl_2) exhibit histological alterations in the uterine epithelium like vacuolation in uterine epithelial cells, cystic dilatation of

uterine glands, and degeneration of the epithelial lining along with ovarian changes [52]. Female Wistar rats orally exposed to mercury (5, 50 and 500 µg/L in drinking water) for 28 days have been shown to increase receptor-dependent (PGF2α-induced) and receptor-independent (calcium chloride-induced and high K⁺-depolarizing solution-induced) myometrial contractions coupled with a corresponding increase in the uterine calcium levels at 5 µg/L mercury concentration significantly. These results suggest that mercury at low levels (5 µg/L) has a detrimental effect on myometrial activity by altering calcium entry into the smooth muscle and/or the release of calcium from intracellular stores without causing any apparent systemic toxicity in rats [31]. *In vitro* exposure to HgCl₂ interacts with muscarinic receptors and activates calcium signalling cascades involving calcium channels, Rho-kinase, protein kinase-C, and phospholipase-C pathways to exert uterotonic effects in rat myometrium [32].

Endocrine Disruptive Effects of Mercury

Mercury possesses an endocrine-disruptive potential affecting the hypothalamus, pituitary, thyroid, adrenal gland and gonads (testis and ovary) in laboratory animals as well as in humans [70]. Its acute, subacute, or chronic exposure disrupts the hypothalamic-pituitary-adrenal and hypothalamic-gonadal axes, which negatively affect reproductive functions. This predominantly interferes with hormones such as FSH and LH, further altering steroids such as estrogens, progesterone, and androgens. Mercury primarily inhibits 21-alpha hydroxylase, affecting adrenal function and hormone production. It also has impacts on reproductive organs, including gonad size and gamete production [58, 56].

Chronic ingestion of low levels of mercuric chloride in female rats lowers the progesterone level, increases LH and FSH levels, indicating that mercuric chloride has a disruptive effect in the corpora lutea, which manifests itself after ovulation [21]. Methyl mercury (20 mg/l) in the drinking water of adult rats has been reported to be deposited in the pituitaries [41]. Similarly, it is found to be deposited in the hypothalamus of rats on exposure to mercury in any form [18]. Mercuric chloride is a weak estrogen mimic and depending on its concentration and exhibits estrogen receptor agonist-antagonist properties. It does not affect the binding of estradiol to estrogen receptor (ER). It exhibits estrogen-like effects through binding and activating ER rather than binding to ER by competing with estradiol [74]. However, as is the case with estradiol, mercury stimulates Erk1/2 activation that is dependent on mobilization of intracellular Ca²⁺, suggesting similar signalling mechanisms [37]. In MCF-7 cells, the estrogenicity of HgCl₂ has been examined stimulating both estrogen receptor-dependent transcription and an increase in proliferation of MCF-7 cells [8].

Molecular Mechanisms Involved in Mercury-Induced Oxidative Stress

A cell maintains redox homeostasis under physiological conditions through well-regulated systems, including enzymatic antioxidants (e.g., superoxide dismutase, SOD; catalase, CAT; glutathione peroxidase, GPx) and non-enzymatic scavengers (e.g., glutathione, vitamin E) [39]. Reactive oxygen species (ROS), such as superoxide (O₂^{•-}) and hydrogen peroxide (H₂O₂), act as signalling molecules at low levels modulating metabolic and vascular functions [60]. Mercury causes redox imbalances that lead to oxidative stress through both direct and indirect pathways. The direct pathways include direct generation of reactive oxygen species

(ROS) by altering the mitochondrial respiratory chain. The indirect pathway is characterized by the accumulation of ROS resulting from the interference of mercury in the biochemical reactions of antioxidants [63, 12, 10]. The adverse health effects of mercury exposure occur through the disruption of redox homeostasis, primarily caused by alterations in microtubule assembly. Mercury-induced toxicity associated genes/proteins expressed can be identified by several comprehensive analyses of transcriptomics, proteomics, and metabolomics [29]. Several oxidative stress-related genes are modulated by mercury-induced cytotoxicity and genotoxicity in rat lungs [13].

Adult Wistar rats treated with mercury (II) thiocyanate (Hg(SCN)₂) at a dose rate of 0.001 mg/L *ad libitum* for 65 days show altered oxidative stress biomarkers: superoxide dismutase (SOD), glutathione peroxidase (GPx), malondialdehyde (MDA), and hydrogen peroxide (H₂O₂) in blood and tissue samples with evident cellular alterations [1]. Similarly, fresh tissue homogenates and body fluids from the liver, kidney, lung, testis, and serum in rats exhibit enhanced lipid peroxidation after parenteral administration of mercuric chloride (HgCl₂) at a dose rate of 5 mg/kg body weight (subcutaneously). Elevated levels of malondialdehyde (MDA) suggest that lipid peroxidation is one of the molecular mechanisms for cell injury in acute HgCl₂ poisoning [25].

Mercury exposure affects the fertility of both sexes; however, due to the fixed and non-renewable pool of germ cells in the ovaries, it has a more severe impact on females than males [5]. The ovary is a crucial organ in the female reproductive system, as it performs both reproductive and endocrine functions. The production of excessive ROS can cause severe damage to the ovary. Mercury exposure causes oxidative stress in the ovaries through both direct and indirect mechanisms, altering signal transduction pathways. The detrimental effects of mercury on the ovaries include follicular atresia and decreased estrogen production, causing premature ovarian insufficiency in women [69]. The ROS accumulation triggers oxidative damage and apoptosis via the hypoxia-inducible factor-1α (HIF-1α) vascular endothelial growth factor (VEGF) signalling pathway leading to follicular atresia with age [72], and causing DNA damage in oocytes [64]. Methylmercury (MeHg) exhibits a high affinity for selenohydryl groups, sulfhydryl groups, and selenides, which directly activate the antioxidant Keap1/Nrf2 signalling pathway, leading to the impairment of antioxidant enzymes and proteins and ultimately disrupting the antioxidant systems [19]. In laying hens, oxidative stress and ovarian disorders are caused by impeding the Nrf2/Keap1 signalling pathway after mercury exposure [15]. Mercury diminishes the viability of the human ovarian granulosa cell line in a concentration-dependent manner. It significantly reduces cellular ATP levels, mitochondrial membrane potential, oxygen consumption rates, and lactate production, increases membrane fluidity, promotes mitochondrial swelling and enhances the permeability of the inner mitochondrial membrane. Consequently, the bioenergetic disruption may represent a key cause of mercury-induced ovarian dysfunction [17].

Conclusion

Mercury and its compounds' toxicity continues to remain a paramount global issue, impacting human and animal health. Its versatile effects interfere with multiple organ systems with profound effects on fertility and reproductive processes through several molecular pathways. Mercury exposure at low

concentrations for a longer period of time in females adversely affects fertility and pregnancy outcomes by altering oogenesis and ovarian function, as a limited non-replenishable source of ova is available. Mercury acts as an endocrine disruptor, interferes with cellular and molecular mechanisms, leading to oxidative stress, apoptosis, epigenetic alterations, and impaired signalling pathways in the female reproductive system. Thus, the toxicological impact of mercurial compounds on female reproductive systems highlights the need for preventive measures and comprehensive research to understand and mitigate the mechanisms of toxicity and associated female reproductive health risks.

Conflict of Interest

Not available

Financial Support

Not available

References

- Adeniyi T, Moronkeji A, Oluwatunase G, Adekomi A, Adunmo G. Mercury-induced oxidative stress and neuropathologic changes in the prefrontal cortex of Wistar rats. *Nig J Neurosci*. 2024;15:97-104.
- Atchison WD, Hare MF. Mechanisms of methylmercury-induced neurotoxicity. *FASEB J*. 1994;8(9):622-629.
- Binah O, Meiri U, Rahamimoff H. The effects of HgCl₂ and mersalyl on mechanisms regulating intracellular calcium and transmitter release. *Eur J Pharmacol*. 1978;51(4):453-457.
- Budnik LT, Casteleyn L. Mercury pollution in modern times and its socio-medical consequences. *Sci Total Environ*. 2019;654:720-734.
- Canipari R, de Santis L, Cecconi S. Female fertility and environmental pollution. *Int J Environ Res Public Health*. 2020;17(23):8802-8802.
- Charkiewicz AE, Omeljaniuk WJ, Garley M, Niklinski J. Mercury exposure and health effects: what do we really know? *Int J Mol Sci*. 2025;26(5):2326-2326.
- Chen JQ, Dong J, Chang JJ, Guo TT, Yang QC, Jia W, et al. Characterization of an Hg(II)-volatilizing *Pseudomonas* sp. strain DC-B1 and its potential for soil remediation when combined with biochar amendment. *Ecotoxicol Environ Saf*. 2018;163:172-179.
- Choe SY, Kim SJ, Kim HG, Lee JH, Choi Y, Lee H, et al. Evaluation of estrogenicity of major heavy metals. *Sci Total Environ*. 2003;312(1-2):15-21.
- Clarkson TW, Magos L. The toxicology of mercury and its chemical compounds. *Crit Rev Toxicol*. 2006;36(8):609-662.
- Cuyppers A, Plusquin M, Remans T, Jozefczak M, Keunen E, Gielen H, et al. Cadmium stress: an oxidative challenge. *Biometals*. 2010;23(5):927-940.
- Davis B, Price H, O'Connor R, Fernando R, Rowland A, Morgan D. Mercury vapor and female reproductive toxicity. *Toxicol Sci*. 2001;59(2):291-296.
- de Vizcaya-Ruiz A, Barbier O, Ruiz-Ramos R, Cebrian ME. Biomarkers of oxidative stress and damage in human populations exposed to arsenic. *Mutat Res*. 2009;674(1-2):85-92.
- Diako K, Rahimi S, Mirbagheri Saghaleksari SA, Mousavi Eshkelani SM, Keshavarz Taramsari N, Beigi Harchegani A, et al. N-acetylcysteine as an antioxidant and anti-inflammatory agent in decreasing histopathological damages and oxidative stress after mercury exposure in lung tissue of rats. *BMC Biotechnol*. 2025;25(1):108-108.
- Ding Z, Sun Y, Wu C, Ma C, Ruan H, Zhang Y, et al. Methylmercury chloride inhibits meiotic maturation of mouse oocytes *in vitro* by disrupting the cytoskeleton. *Food Chem Toxicol*. 2024;193:115024-115024.
- El-Badry A, Rezk M, El-Sayed H. Mercury-induced oxidative stress may adversely affect pregnancy outcome among dental staff: a cohort study. *Int J Occup Environ Med*. 2018;9(3):113-119.
- Elghany NA, Stopford W, Bunn WB, Fleming LE. Occupational exposure to inorganic mercury vapour and reproductive outcomes. *Occup Med (Lond)*. 1997;47(6):333-336.
- Elmorsy EM, Al Doghaither HA, Al-Ghafari AB, Amer S, Fawzy MS, Toraih EA. Fucosanthin mitigates mercury-induced mitochondrial toxicity in the human ovarian granulosa cell line. *Reprod Toxicol*. 2025;132:108855-108855.
- Ernst E, Christensen MK, Poulsen EH. Mercury in the rat hypothalamic arcuate nucleus and median eminence after mercury vapor exposure. *Exp Mol Pathol*. 1993;58(3):205-214.
- Fujimura M, Usuki F. Methylmercury-mediated oxidative stress and activation of the cellular protective system. *Antioxidants*. 2020;9(10):1004-1004.
- Hare MF, Rezazadeh SM, Cooper GP, Minnema DJ, Michaelson IA. Effects of inorganic mercury on [3H] dopamine release and calcium homeostasis in rat striatal synaptosomes. *Toxicol Appl Pharmacol*. 1990;102(2):316-330.
- Heath JC, Abdelmageed Y, Braden TD, Nichols AC, Steffy DA. The effects of chronic mercuric chloride ingestion in female Sprague-Dawley rats on fertility and reproduction. *Food Chem Toxicol*. 2009;47(7):1600-1605.
- Henriques MC, Loureiro S, Fardilha M, Herdeiro MT. Exposure to mercury and human reproductive health: a systematic review. *Reprod Toxicol*. 2019;85:93-103.
- Hou S, Wang C, Ma X, Zhao J, Wang J, Fang Y, et al. Methylmercury chloride exposure affects oocyte maturation through AMPK/mTOR-mediated mitochondrial autophagy. *Int J Mol Sci*. 2025;26(8):3603-3603.
- Hsi HC, Hsu YW, Chang TC, Chien LC. Methylmercury concentration in fish and risk-benefit assessment of fish intake among pregnant versus infertile women in Taiwan. *PLoS One*. 2016;11(5):e0155704-e0155704.
- Huang YL, Cheng SL, Lin TH. Lipid peroxidation in rats administered with mercuric chloride. *Biol Trace Elem Res*. 1996;52(2):193-206.
- Iavicoli I, Fontana L, Bergamaschi A. The effects of metals as endocrine disruptors. *J Toxicol Environ Health B Crit Rev*. 2009;12(3):206-223.
- Jagiello G, Lin JS. An assessment of the effects of mercury on the meiosis of mouse ova. *Mutat Res*. 1973;17:93-99.
- Jones L, Bunnell J, Stillman J. A 30-year follow-up of residual effects on New Zealand school dental nurses from occupational mercury exposure. *Hum Exp Toxicol*. 2007;26:367-374.
- Kang B, Wang J, Guo S, Yang L. Mercury-induced toxicity: mechanisms, molecular pathways, and gene regulation. *Sci Total Environ*. 2024;943:173577-173577.
- Kimura T. Effects of hypophysectomy and ACTH

- administration on the level of adrenal cholesterol side-chain desmolase. *Endocrinology*. 1969;85(3):492-499.
31. Koli S, Prakash A, Choudhury S, Mandil R, Garg SK. Mercury affects uterine myogenic activity even without producing any apparent toxicity in rats: involvement of calcium-signaling cascades. *J Trace Elem Med Biol*. 2020;57:40-47.
 32. Koli S, Prakash A, Choudhury S, Mandil R, Garg SK. Calcium channels, rho-kinase, protein kinase-C, and phospholipase-C pathways mediate mercury chloride-induced myometrial contractions in rats. *Biol Trace Elem Res*. 2019;187(2):418-424.
 33. Koos BJ, Longo LD. Mercury toxicity in the pregnant woman, fetus, and newborn infant. *Am J Obstet Gynecol*. 1975;126:390-409.
 34. Kumar A, Agarwal R, Kumar K, Chayal NK, Kumar G, Kumar R, et al. Mercury poisoning in women and infants inhabiting the Gangetic plains of Bihar: risk assessment. *BMC Public Health*. 2025;25(1):1275-1275.
 35. Kumar S, Sharma A, Sedha S. Occupational and environmental mercury exposure and human reproductive health: a review. *J Turk Ger Gynecol Assoc*. 2022;23(3):199-199.
 36. Leonard A, Jacquet P, Lauwerys RR. Mutagenicity and teratogenicity of mercury compounds. *Mutat Res*. 1983;114:1-18.
 37. Li X, Zhang S, Safe S. Activation of kinase pathways in MCF-7 cells by 17beta-estradiol and structurally diverse estrogenic compounds. *J Steroid Biochem Mol Biol*. 2006;98:122-132.
 38. Liu M, Zhang Q, Maavara T, Liu S, Wang X, Raymond PA. Rivers as the largest source of mercury to coastal oceans worldwide. *Nat Geosci*. 2021;14:672-677.
 39. Lushchak VI. Classification of oxidative stress based on its intensity. *EXCLI J*. 2014;13:922-937.
 40. Maeda E, Murata K, Kumazawa Y, Sato W, Shirasawa H, Iwasawa T, et al. Associations of environmental exposures to methylmercury and selenium with female infertility: a case-control study. *Environ Res*. 2019;168:357-363.
 41. Moller-Madsen B, Thorlacius-Ussing O. Accumulation of mercury in the anterior pituitary of rats following oral intraperitoneal administration of methyl mercury. *Virchows Arch B Cell Pathol Incl Mol Pathol*. 1986;51(1):303-311.
 42. Morgan DL, Chanda SM, Price HC, Fernando R, Liu J, Brambila E, et al. Disposition of inhaled mercury vapor in pregnant rats: maternal toxicity and effects on developmental outcome. *Toxicol Sci*. 2002;66(2):261-273.
 43. Mukherjee D, Kumar V, Chakraborti P. Effect of mercuric chloride and cadmium chloride on gonadal function and its regulation in sexually mature common carp *Cyprinus carpio*. *Biomed Environ Sci*. 1994;7(1):13-24.
 44. Naik SR, Gamare D. Toxicity of mercury and its compounds: impacts on human and animal health, ecosystems, mechanisms, and bioremediation strategies—a systematic review. *J ISSN*. 2025;2766:2276-2276.
 45. O'Brien J. Mercury amalgam toxicity. *Life Ext Mag*. 2001;7(5):43-51.
 46. Outridge PM, Mason R, Wang F, Guerrero S, Heimburger-Boavida L. Updated global and oceanic mercury budgets for the United Nations Global Mercury Assessment 2018. *Environ Sci Technol*. 2018;52(20):11466-11477.
 47. Pan J, Song H, Pan XC. Reproductive effects of occupational exposure to mercury on female workers in China: a meta-analysis. *Zhonghua Liu Xing Bing Xue Za Zhi*. 2007;28:1215-1218.
 48. Pan Y, Qu K, Li H, Song Y. Toxic effects of chronic occupational mercury vapor exposure on female workers of childbearing age. *J Occup Med Toxicol*. 2025;20(1):5-5.
 49. Park JD, Zheng W. Human exposure and health effects of inorganic and elemental mercury. *J Prev Med Public Health*. 2012;45(6):344-352.
 50. Rattan S, Zhou C, Chiang C, Mahalingam S, Brehm E, Flaws JA. Exposure to endocrine disruptors during adulthood: consequences for female fertility. *J Endocrinol*. 2017;233(3):R109-R129.
 51. Reddy PS, Tuberty SR, Fingerman M. Effects of cadmium and mercury on ovarian maturation in the red swamp crayfish, *Procambarus clarkii*. *Ecotoxicol Environ Saf*. 1997;37(1):62-65.
 52. Rhyaf AG, Chelab KG, Naji HA. Pathological effects of mercury chloride on reproductive system in white rats. *AL-Qadisiyah J Vet Med Sci*. 2018;17(1):1-?
 53. Rice KM, Walker EM Jr, Wu M, Gillette C, Blough ER. Environmental mercury and its toxic effects. *J Prev Med Public Health*. 2014;47:74-83.
 54. Roberts JR. Metal toxicity in children. In: Training manual on pediatric environmental health: putting it into practice. Emeryville (CA): Children's Environmental Health; 1999.
 55. Rooney JP. The role of thiols, dithiols, nutritional factors and interacting ligands in the toxicology of mercury. *Toxicol*. 2007;234(3):145-156.
 56. Rowland AS, Baird DD, Weinberg CR, Shore DL, Shy CM, Wilcox AJ. The effect of occupational exposure to mercury vapour on the fertility of female dental assistants. *Occup Environ Med*. 1994;51(1):28-34.
 57. Schneider H. Molecular physiology of smooth muscle cells. *Front Physiol*. 2023;14:1223278-1223278.
 58. Schrag SD, Dixon RL. Occupational exposures associated with male reproductive dysfunction. *Annu Rev Pharmacol Toxicol*. 1985;25:567-592.
 59. Shi DS. Occupational exposure to mercury at an electronics waste and lamp recycling facility—Ohio, 2023. *MMWR Morb Mortal Wkly Rep*. 2025;74:1-?
 60. Sies H, Mailloux RJ, Jakob U. Fundamentals of redox regulation in biology. *Nat Rev Mol Cell Biol*. 2024;25(9):701-719.
 61. Stadnicka A. Localization of mercury in the rat ovary after oral administration of mercuric chloride. *Acta Histochem*. 1980;67(2):227-233.
 62. Tan SW, Meiller JC, Mahaffey KR. The endocrine effects of mercury in humans and wildlife. *Crit Rev Toxicol*. 2009;39(3):228-269.
 63. Valko M, Morris H, Cronin MT. Metals, toxicity and oxidative stress. *Curr Med Chem*. 2005;12(10):1161-1208.
 64. Wang Y, Li SH, Yang SJ, Li XQ, Liu L, Ma X, et al. Exposure to phenanthrene affects oocyte meiosis by inducing mitochondrial dysfunction and endoplasmic reticulum stress. *Cell Prolif*. 2023;56(1):e13335-e13335.
 65. Watanabe T, Shimada T, Endo A. Effects of mercury compounds on ovulation and meiotic and mitotic chromosomes in female gold hamsters. *Teratology*.

- 1982;25:381-384.
66. World Health Organization. Environmental health criteria 101: methylmercury. Geneva: World Health Organization; 1990.
 67. World Health Organization. Environmental health criteria 118: inorganic mercury. Geneva: World Health Organization; 1991.
 68. Wu YS, Osman AI, Hosny M, Elgarahy AM, Eltaweil AS, Rooney DW, *et al.* The toxicity of mercury and its chemical compounds: molecular mechanisms and environmental and human health implications: a comprehensive review. *ACS Omega*. 2024;9(5):5100-5126.
 69. Xiao C, Lai D. Impact of oxidative stress induced by heavy metals on ovarian function. *J Appl Toxicol*. 2025;45(1):107-116.
 70. Xinqiang Z, Yukinori K, Kazuhiro S, Qunwei Z. The endocrine disruptive effects of mercury. *Environ Health Prev Med*. 2000;4(4):174-183.
 71. Yang JM, Chen QY, Jiang XZ. Effects of metallic mercury on the perimenstrual symptoms and menstrual outcomes of exposed workers. *Am J Ind Med*. 2002;42:403-409.
 72. Yang Z, Hong W, Zheng K, Feng J, Hu C, Tan J, *et al.* Chitosan oligosaccharides alleviate H₂O₂-stimulated granulosa cell damage via HIF-1 α signaling pathway. *Oxid Med Cell Longev*. 2022;2022:4247042-4247042.
 73. Yoshida M. Placental to fetal transfer of mercury and fetotoxicity. *Tohoku J Exp Med*. 2002;196(2):79-88.
 74. Zhang X, Wang Y, Zhao Y, Chen X. Experimental study on estrogen-like effect of mercuric chloride. *Biometals*. 2008;21(2):143-150.
 75. Zhu X, Kusaka Y, Sato K, Zhang Q. The endocrine disruptive effects of mercury. *Environ Health Prev Med*. 2000;4(4):174-183.

How to Cite This Article

Koli S, Dewangan G, Rajoriya S, Tayde RS, Chaudhary R, Ranjan R. Mercury exposure as a determinant of female infertility: Mechanistic insights and reproductive outcomes. *International Journal of Veterinary Sciences and Animal Husbandry*. 2026;11(2):618-623.

Creative Commons (CC) License

This is an open-access journal, and articles are distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 4.0 International (CC BY-NC-SA 4.0) License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.