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# Environmental Pollution: a Risk Factor for Female Fertility-A Letter to Editor

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## Abstract

Experimental data indicate that exposure to environmental pollution can lead to serious complications on the female reproductive system. Environmental pollution appears to affect reproductive success and outcomes by endocrine disruption and/or reactive oxygen species (ROS)-induced oxidative stress. The aim of this letter to editor is to elucidate the impact of exposures to environmental pollutants on female reproductive health and outcomes. There is a need to increase the awareness among women to avoid exposure to the reproductive risk factors.

**Keywords:** Environmental pollution, Female, Fertility

Significant evidences exist to support environmental pollution exposure as a risk factor for fertility (1-3). These studies mostly focused on certain toxicants, heavy metals (arsenic, lead, cadmium and mercury), pesticides such as bis(4-chlorophenyl)-1,1,1-trichloroethane and organic solvent such as benzene, toluene and ionizing radiation (4). There are also evidences that exposure to indoor air pollution could play a role in the pathogenesis of female infertility (5, 6). The most relevant air pollutants are in four main categories: organic compounds, gaseous pollutants (sulfur dioxide (SO2), nitrate oxide (NO2) and carbon monoxide (CO)), heavy metals and particulate matter (PM10 PM2.5-10and PM2.5) (5, 7). Environmental pollution has been suggested to be acted on one or more organs through different and/or similar mechanisms of action. Although the specific mechanism is unclear, observed evidences have shown that exposure to environmental pollution induces endocrine disruption and may lead to disrupt the dynamic hormone-dependent signaling pathways in differentiating tissues (8). Endocrine-disrupting compounds (EDCs) are able to mimic hormones and can cause reproductive problems such as miscarriages, impaired fertility, endometriosis, irregularities of the menstrual cycle, intrauterine growth restriction, polycystic ovarian syndrome and infertility (9, 10) . Bisphenol-A(BPA), phthalates, parabens, tributyltin (TBT) and triclosan (TCS), are abundant endocrine disruptor and widely spread in the environment and food chain (11). Moreover, environmental chemicals may involve reactive oxygen species (ROS)-induced oxidative stress or DNA methylation impairing fertility as well as pregnancy and its outcomes (12-14). Oxidative stress induced by ROS can lead to oxidative damage of cellular macromolecules and mitochondrial DNA (15). Therefore, mitochondria is a vital target for many environmental pollution (16). Pollutant-induced mitochondrial ROS may affect early embryogenesis and post-implantation prenatal development (17). Thus, some pregnancy-related disorders such as early spontaneous abortion, embryonic mortality, premature delivery and low birthweight are considered to related to imbalance between ROS production and antioxidant ROS detoxification pathways (2, 18, 19). Maternal exposure to heavy metals including cadmium (Cd) has been shown to be associated with preeclampsia (20). Others found association of early delivery and low birthweight with maternal cadmium exposure (2, 21, 22). There are reports on maternal exposure to lead and adverse pregnancy outcomes including delay in growth and pubertal development (23). Some evidences have been shown that exposure to heavy metal can induce the excessive production of ROS, oxidative stress and developmental toxicity (2, 14). Epidemiological studies have documented close association between working women and higher risk of undesirable reproductive outcome. As preventing the discharge of environmental pollutants is an important factor, it would seem to be crucial to increase awareness of risk factors among the population to efficiently reduce environmental pollutants.

**References**

1. Kumar S, Sharma, A, Kshetrimayum C. Environmental & occupational exposure & female reproductive dysfunction. Indian Journal of Medical Research. 2019;150(6):532-45.
2. Al-Gubory KH. Environmental pollutants and lifestyle factors induce oxidative stress and poor prenatal development. Reproductive BioMedicine Online. 2014; 29(1):17-31.
3. Nateghian Z, Aliabadi. E Aspects of Environmental Pollutants on Male Fertility and Sperm Parameters. Journal of Environmental Treatment Techniques. 2020;8(1):299-309.
4. Rzymski P, Tomczyk K, Poniedzialek BImpact of heavy metals on the female reproductive system. Annals of Agricultural and Environmental Medicine. 2015;22(2).

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5. Conforti A, Mascia M, Cioffi G, De Angelis, et al. Air pollution and female fertility: a systematic review of literature. Reproductive Biology and Endocrinology. 2018;16(1):117.
6. Vizcaíno MA, González-Comadran M, Jacquemin B. Outdoor air pollution and human infertility: a systematic review. Fertility and Sterility. 2016; 15;106(4):897-904.
7. Alaee S. Air Pollution and Infertility–A letter to Editor. Journal of Environmental Treatment Techniques. 2018;6(4):72-3.
8. Rider CV, Carlin DJ, DeVito MJ, et al. Mixtures research at NIEHS: an evolving program. Toxicology. 2013;313(2-3):94-102.
9. Balabanič D, Rupnik M, Klemenčič AK. Negative impact of endocrine-disrupting compounds on human reproductive health. Reproduction, Fertility and Development. 2011;23(3):403-16.
10. Fisher JS, Macpherson S, Marchetti N, Sharpe RM. Human 'testicular dysgenesis syndrome': a possible model using in-utero exposure of the rat to dibutyl phthalate. Human Reproduction. 2003;18(7):1383-94.
11. Klingmüller D, Alléra A. Endocrine disruptors: hormone-active chemicals from the environment: a risk to humans?. Deutsche medizinische Wochenschrift. 2011;136(18):967-72.
12. Desrosiers TA, Herring AH, Shapira SK, et al. Paternal occupation and birth defects: findings from the National Birth Defects Prevention Study. Occupational and Environmental Medicine. 2012;69(8):534-42.
13. Slama R, Cordier S. Impact of chemical and physical environmental factors on the course and outcome of pregnancy. Journal de Gynecologie, Obstetrique et biologie de la Reproduction. 2013;42(5):413.
14. Kumar, S., A. Sharma, and C. Kshetrimayum, Environmental & occupational exposure & female reproductive dysfunction. The Indian Journal of Medical Research. 2019;150(6):532.
15. Buonocore G, Perrone S, Tataranno ML. Oxygen toxicity: chemistry and biology of reactive oxygen species. In Seminars in Fetal and Neonatal Medicine 2010;15(4):186-190.
16. Meyer JN, Leung MC, Rooney JP, et al. Mitochondria as a target of environmental toxicants. Toxicological Sciences. 2013;134(1):1-7.17. Ornoy A. Embryonic oxidative stress as a mechanism of teratogenesis with special emphasis on diabetic embryopathy. Reproductive toxicology. 2007;24(1):31-41.
18. Agarwal A, Aponte-Mellado A, Premkumar BJ, Shaman A, Gupta S. The effects of oxidative stress on female reproduction: a review. Reproductive Biology and Endocrinology. 2012;10(1):1-31.
19. Al-Gubory KH, Garrel C. Antioxidative signalling pathways regulate the level of reactive oxygen species at the endometrial–extraembryonic membranes interface during early pregnancy. The International Journal of Biochemistry & Cell Biology. 2012;44(9):1511-8.
20. Pollack AZ, Ranasinghe S, Sjaarda LA, Mumford SL. Cadmium and reproductive health in women: a systematic review of the epidemiologic evidence. Current Environmental Health Reports. 2014;1(2):172-84.
21. Kippler M, Tofail F, Gardner R, et al. Maternal cadmium exposure during pregnancy and size at birth: a prospective cohort study. Environmental Health Perspectives. 2012;120(2):284-9.
22. Alaee S, Khodabandeh Z, Asadollahpour R. Subjection to Cadmium Chloride Compromises Oocyte Maturation, Fertilization Competence and Subsequent Embryo Development in Mature Female Mice. Journal of Infertility and Reproductive Biology. 2017;5(3):16-8.
23. Selevan SG, Rice DC, Hogan KA, et al. Blood lead concentration and delayed puberty in girls. New England Journal of Medicine. 2003;348(16):1527-36.