



# Hazardous Effects of Endocrine Disrupting Compounds in Aquatic Organisms

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## Editorial

Volume 4 Issue 1

Received Date: January 04, 2020

Published Date: January 12, 2021

DOI: 10.23880/izab-16000267

## Editorial

### Hazardous Effects of Endocrine Disrupting Compounds in Aquatic Organisms

The widespread use of different chemicals in various sectors like industry, agriculture, medical treatment, and even in common domestic conveniences, has posed a potential threat to ecology and human health. Chemical released by different anthropogenic activities has become one of the important environmental pollutants due to their massive production, extensive use, persistent and accumulative in nature [1]. Among these chemicals the presence of endocrine disrupting compounds (EDCs) is of great concern as they invade the ecosystem and aquatic environment by various routes e.g. household and Industrial waste water, wastewater discharged from treatment plants, effluents, agricultural run offs etc [2-4]. These endocrine disrupting compounds include varieties of chemicals like pesticides (polychlorinated biphenyls, DDT), fungicides (tebuconazole and Hexaconazole), xenoestrogens (bisphenol A, 17 $\beta$ -estradiol and 17 $\alpha$ -ethynylestradiol), metformin, synthetic musk etc. As aquatic animals remain in exposure of environmental pollutants in water, they may be regarded as important indicators for the toxicity assessment of endocrine disrupting chemicals [5]. EDCs are great threat to the aquatic life which in turn might also affect the human health as aquatic animals particularly fish are being consumed at large scale as rich source of nutrients. These EDCs enter into or fish body via skin, gills or through contaminated foods. These endocrine disrupting compounds are well known for exerting deleterious effects on the endocrine system [6,7] by impairing hormonal homeostasis either by interfering the synthesis, secretion, transport, metabolism or excretion or by imitating the effects of endogenous hormones that results in adverse impact on the development, reproduction and

performance of living species [8,9]. Studies have revealed that the adverse effects of EDCs are often permanent and can be considered as epigenetic modulators that can lead to likely transgenerational effects [10,11]. Hazardous effects of EDCs in aquatic organisms especially in fishes have been reported earlier in various studies [12-14]. Ye T, et al. [15] reported reduced fertilization rate of oocytes, decreased the egg production and increased plasma 17 $\beta$  estradiol (E2) in fishes exposed to a plastic softener i.e., di-(2-ethylhexyl)-phthalate (DEHP), used in manufacturing industry. One of the world's well-known anti-diabetic drugs metformin has been found to increase regulation of mRNA that encode vitellogenin egg protein present in male fish resulting in unfavourable effects on fertility and reproduction [16]. Dércia Santos, et al. [17] reported malformations and altered physiology in the fish larvae, exposed to 17 $\alpha$ -ethynylestradiol and genistein which was attributed to up regulation of ER and AR signalling pathways (gene *esr1*) and apoptotic pathways (*c-jun* genes) by these chemicals. Exposure to EDC's also resulted in development of intersex or ovotestes condition in gonochoristic species of fish and reduced sperm quality [18,19]. Jie Hou, et al. [20] reported adverse effects in ovaries following long-term exposure to Microcystin-LR which included decreased ovary weight, growth inhibition, decreased ovarian testosterone levels and ovarian ultra-pathological lesions. Several triazoles are also known to act as EDC's and has been described to change the transcription of genes which is involved in homeostasis of steroid like thyroid hormone, corticotrophin-releasing hormone and thyronine deiodinase (*Dio1* and *Dio2*) [21]. Also Liang Yu, et al. [12] found significant increase in triiodothyronine (T3) concentrations and decrease in thyroxine (T4) levels in zebra fish larvae following exposure to hexaconazole and tebuconazole representing endocrine disruption

which was attributed to induction of Dio2 transcription. Jyotshna Kanungo, et al. [22] reported effects of nicotine on neuronal development and estrogen levels in zebra fish and found its antiestrogenic activity, altered the expression of important biomarkers such as vtg 1 and vtg 2 and cytochrome p450 aromatase (cyp19a1a and cyp19a1b) at the level of transcription. Zhang Y, et al. [23] reported that bisphenol A (BPA) can induce change in ovarian morphology that can lead to higher proportion of premature oocytes and many atretic follicles in *Gobius rarus*. Many deformed follicles and degenerating vitellogenic oocytes suggested a defect in follicle recruitment have been observed in female gonads of *Gobiocypris rarus* after exposed to 2, 4-Dichloro-6-nitrophenol Chen R, et al. [24]. Sridevi P, et al. [14] reported that 17 $\alpha$ -ethynylestradiol (EE2) and diethylstilbestrol (DES) can significantly altered transcript level and the activity of aromatase (important enzyme for biosynthesis of estrogen) in catfish *Clarias gariepinus*. Fitzgerald AC, et al. [13] demonstrated in zebrafish model and observed that three alkylphenols, e.g. 4-nonylphenol, tetrabromobisphenol A, and tetrachlorobisphenol A (5–100 nM), and low BPA concentrations (10–200 nM) have ability to disrupt oocyte maturation via a non-genomic mechanism of action that involve the activation of the Gper/Egfr/Mapk3/1 pathway. In marine medaka, *Oryzias melastima*, an antifouling agent 3, 3'' diindolylmethane (DIM) at relatively low doses (0 and 8.5  $\mu$ g/l) can produce abnormal eggshell proteins and vitellogenin in the testis that undoubtedly highlight its estrogenic potency [25,26]. Wang P, et al. [27] reported that flutamide (FLU), DES and their combination can induce decrease in sperm concentration, loss of spermatogenesis and also can affect both meiotic and apoptotic processes. Exposure of triclocarban and inorganic mercury in zebrafish caused alterations in testis morphology due to variations in steroidogenic genes, such as cyp17, cyp19a, 3 $\beta$ -HSD and 17 $\beta$ -HSD [27].

### Concluding Remarks

EDCs are great concern as they invade the ecosystem and aquatic environments by various routes and have adverse effects on aquatic organisms. EDCs induced alteration in physiopathology leading to hormonal disturbance in aquatic organisms might also show negative impacts on human being through food chain. Therefore, more studies are needed to be carried out in order to understand the mechanism of EDCs based on the principle of endocrinology so that proper risk assessment of EDCs could be evaluated.

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