INTRODUCTION

Fisheries and aquatic systems has been accompanied by increasing concern about the effects of growing human populations and human activity on aquatic life and water quality. Pesticides are one group of toxic compounds linked to human use that have a profound effect on aquatic life and water quality. Among the aquatic life Fishes are the victims of pesticide poisoning. Pesticide use is one of many factors contributing to the decline of fish and other aquatic species. Exposure of fish and other aquatic animals to a pesticide depends on its biological availability (bioavailability), bioconcentration, biomagnification, and persistence in the environment. Toxic effects of pollutants in aquatic ecosystem can be well-studied by using fish as a bio-indicator since it is constantly exposed to unfavourable environmental conditions, including chemical contaminants and potential pathogens, thus fishes are regarded as one of the primary risk groups among the aquatic organisms (Kime, 1999). Fish and aquatic animals are exposed to pesticides in three primary ways (1) dermally, direct absorption through the skin by swimming in pesticide-contaminated waters, (2) breathing, by direct uptake of pesticides through the gills during respiration, and (3) orally, by drinking pesticide-contaminated water or feeding on pesticide-contaminated prey. Poisoning by consuming another animal that has been poisoned by a pesticide is termed “secondary poisoning.” Hormones are important to both vertebrates and invertebrates. They are essential for controlling a large number of processes in the body from early processes such as cell differentiation during embryonic development and organ formation, to the control of tissue and organ function in adulthood (Melmed & Williams, 2011). Many chemicals that have been identified as endocrine disruptors are pesticides (Wissem Mnif et al., 2011). Endocrine disrupting chemicals (EDCs) were defined as an exogenous agent that interferes with synthesis, secretion, metabolism, binding action, or elimination of hormones responsible for homeostasis, reproduction and developmental process. EDCs act mainly by interfering with natural hormones because of their strong potential to bind to estrogen or androgen receptors (Tabb, 2006). Thus, the present study was aimed to evaluate
the sublethal toxic effects of malathion on various hormones such as thyroid stimulating hormone (TSH), follicle stimulating hormone (FSH) and serum vitellogenin levels in the circulating blood.

MATERIAL AND METHODS:

Experimental fish: *Glossogobius giuris* (length: 12 ± 1 cm; weight: 125 ± 5 g [mean ± SD]) were collected using net (mesh: 10 mm) from the cauvery River, fishes were transported in a container filled with river water and then acclimatized under laboratory conditions for a week period (7 days) prior to the commencement of the experiment. During the acclimatization period, the fish were fed 4% of their body weight with earthworms daily once and the water was renewed every other day. The mortality throughout the period of acclimatization was less than 10%. The test substance- malathion was dissolved in acetone and was added to test water to obtain the desired concentration. The stock solution of 1 mg/L was prepared separately and the dispersed degrees of concentration were prepared by adopting the dilution techniques as outlined in APHA (1971). Toxicity tests: Experiments were carried out in 50 L aquarium. Tests were carried out in three batches: (i) Control-10 fishes were maintained in water without malathion; (ii) and (iii) Experimental-10 fishes were kept in each of the aquarium with different sub-lethal concentration of malathion (0.25 and 0.5 ppm) for a period of 5, 10 and 15 days. Environmental conditions were similar in the entire three aquarium.

Sample Collection: On the termination of exposure period of 5, 10 and 15 days blood sample of both control and experimental group were collected in a heparinized glass culture tube eppendorf by sterile syringe from caudal vein. The serum was separated by centrifuging blood at 5000 rev. /min for 10 min at 4°C and stored at −20°C for further hormone assessment of TSH, FSH and Vitellogenin were done as per standard protocol- Micro plate immune enzymometric Assay for all the control and treated fish *Glossogobius giuris*.

RESULT

*Glossogobius giuris* at the concentration of 0.25ppm malathion exposure, the serum Thyroid Stimulating Hormone (TSH) level showed increased level from the 2.8 µIU (control value) to 3.34+ 0.14 µIU (5days malathion exposure), then decreased to 0.616
+0.01 µIU (10days malathion exposure) following an increase to 3.17+0.067 µIU (15days malathion exposure) while at the concentration of 0.5ppm malathion exposure, the serum Thyroid Stimulating Hormone (TSH) level showed increased level from the 2.8 µIU (control value) to 3.53+ 0.04 µIU (5days malathion exposure), then decreased to 0.952 +0.018 µIU (10days malathion exposure) following an increase to 3.612+0.115 µIU (15days malathion exposure).

*Glossogobius giuris* at the concentration of 0.25ppm malathion exposure, the serum Follicle Stimulating Hormone (FSH) level showed increased level from the 105.0 µIU (control value) to 125.6+ 1.64 µIU (5days malathion exposure), following an increase to 130.9 +3.92 µIU (10days malathion exposure) then decreased to 110.0+3.62µIU (15days malathion exposure) while at the concentration of 0.5ppm malathion exposure fishes , the serum Follicle Stimulating Hormone (FSH) level showed increased level from the 105.0 µIU (control value) to 114.9+ 2.68 µIU (5days malathion exposure), following an increase to 120.6 +3.53 µIU (10days malathion exposure) then decrease to 103.9+2.33µIU (15days malathion exposure) much lower than the untreated control fishes.

*Glossogobius giuris* at the concentration of 0.25ppm malathion exposure, the serum Vitellogenin level showed increased level from the 2.5 µIU (control value) to 2.613+ 0.04 µIU (5days malathion exposure), following a decrease to 2.586+0.066 µIU (10days malathion exposure) and 2.011+0.122µIU (15days malathion exposure) while at the concentration of 0.5ppm malathion exposure fishes , the serum Vitellogenin level showed decrease level from the 2.5 µIU (control value) to 2.341+ 0.136 µIU (5days malathion exposure), following an increase to 2.831+0.141 µIU (10days malathion exposure) then decrease to 1.676+0.165µIU (15days malathion exposure) much lower than the untreated control fishes.
DISCUSSION:

Endocrine-disrupting chemicals (EDCs) act by blocking, mimicking, or synergizing endogenous hormones (31–34). Additionally, these compounds also interfere with immune response, growth and development, as well as increasing osmotic stress, protein degradation, and altering numerous metabolic processes (1,34). The pesticide in study, malathion is either known or suspected to be an Endocrine-disrupting chemicals (EDCs), they are know to produce an endocrine disrupting effect by inhibiting the catecholamine secretion, by binding to the thyroid hormone receptors (Cocco, 2002 and Ishihara et al., 2003). Fishes occupy the highest tropic level of the aquatic ecosystem, and they are the most sufferers due to the pesticide exposure that gain entrance from agricultural or storm water runoff to waterbodies. Out of the different systems, reproductive system is one of the most important organ which gets affected to a great extent. Effects linked to endocrine disruption have been largely noted in invertebrates including fishes (Munkittrick et al., 1991 and Purdom, et al., 1994). Thyroid-stimulating hormone (also known as thyrotropin, thyrotropic hormone, or abbreviated TSH) is a pituitary hormone that stimulates the thyroid gland. In the study an Increased serum TSH level and simultaneously decrease in the serum FSH level after fifth day treatment of malathion coincides with the report of Hulburt (1977), who showed that the thyroid hormone act synergistically with gonadotropin and also Prakriti Verma et al., (2014) while Saravanan et al. (2010) showed reduction in serum TSH level in endosulfan induced fish, Labeo rohita. The level of TSH in our study was very low as compared to control group in 10days malathion exposure in both 0.25ppm and 0.5ppm concentration, resulting in reproduction and developmental impairment in the fishes. It is well documented that functions of thyroid system may be affected by organophosphate pesticides (Zhang et al., 2014), this correlates with our study since malathion is also an organophosphate. Follicle-stimulating hormone (FSH) is
a gonadotropin, a glycoprotein polypeptide hormone, is synthesized and secreted by the gonadotropic cells of the anterior pituitary gland and regulates the development, growth, pubertal maturation, and reproductive processes of the body. Pesticides may also affect the Hypothalamus-Pituitary Gonadal (HPG) axis causing low level of FSH production as seen in 0.5 ppm malathion exposed fish for 15 days. It was reported earlier that pesticide exposure altered sex hormone level by disrupting the transcription of related genes in the Hypothalamic-Pituitary-Gonadal axis (Sharma et al., 2014; Sun et al., 2015; Zhang et al., 2016 and Shadab Ahmad et al., 2018. The altered level of FSH may be due to the altered regulatory mechanism of HPG axis. Female-specific proteins expressed in the blood (body fluid) during oogenesis were named vitellogenin (Vtg)’ (vitelline + genin, meaning source of egg yolk) (Pan et al., 1969), Vtgs are specifically found in females and induced by estrogen, and function as precursors of egg yolk proteins. In our study the serum vitellogenin level showed increasing level, but in 0.5ppm malathion treated fish for 15 days the value was too less when compared to the control.

REFERENCES:


