

TOXICITY OF PROMETHAZINE HYDROCHLORIDE (PHENERGAN) TO *Clarias gariepinus* FINGERLINGS

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ABSTRACT

The acute toxicity of promethazine hydrochloride (phenergan) to *Clarias gariepinus* fingerlings was conducted using static bioassay under laboratory conditions. The 96h LC₅₀ was determined as 172.5 mg/l. Mean mortality was 5, 10, 20, 50, 100% in the concentration of 69, 103.5, 138.0, 172.5 and 207 mg/l respectively, while there was no mortality in the control treatment. There were significant differences ($P < 0.05$) on the effect of concentration, the higher the mortality of *C. gariepinus* fingerlings while toxicity of phenergan increased with duration of exposure. *C. gariepinus* showed increased hyperactivities, cell deformation, lesions and necrosis during the period of exposure. The physicochemical parameters also showed a slight increase as the concentration increased.

INTRODUCTION

The introduction of most chemicals into the aquatic environment has been to control fish populations, fish disease and fish parasites. Promethazine hydrochloride (phenergan) is an antihistamine, which is a potent sedative and is generally used for this purpose (Browman et al., 1980). Generally, synthetic chemicals cause death of the fish due to destruction of gill epithelia (Slabber and Morgan, 1982). At very high dose, it induces symptoms of slow suffocation, dizziness, paralysis, destruction of gill lamellae and mucus cell depletion in several freshwater fishes. Toxicants contaminate freshwater bodies and affect non-target organisms. Various researchers have reported on the effects of chemicals on aquatic organisms (Jiraunghkoorskul et al., 2002). Environmental factors such as pH, turbidity, alkalinity, dissolved oxygen, temperature and conductivity influence the rate of reaction of the pollutants entering the water or the lethal effects on the aquatic organisms (Fagbenro, 2002). This study determines the 96h LC₅₀ of phenergan to *C. gariepinus* and reports the effects on fish gills and liver.

MATERIALS AND METHODS

Clarias gariepinus fingerlings (mean wt., 9.6g) were acclimatized in glass tanks for 24 hours prior to the commencement of the study. Phenergan tablets were milled and were sieved using a 0.1mm sieve. The fine powder was dissolved in water at room temperature (23°C) for 24 hours. The clear solution was filtered to remove the particles. A stock solution of 750 mg/l was made from which 69.0, 103.5, 138.0, 172.5, and 207 mg/l solutions were prepared and introduced into each of the tanks in duplicate treatments and 0.0 mg/l (control). The fish were distributed randomly in duplicate treatments into 12 glass tanks (10 fish/tank). The pH of the solutions was measured with the pH meter, temperature with mercury-in-glass thermometer, dissolved oxygen with a digital DO₂/CO₂ meter and total alkalinity was measured by titrating 0.1N of HCL against 25ml of the test solution using two drops of methyl orange indicator. The exposure lasted for 96h. Water temperature, pH, dissolved oxygen, and total alkalinity were determined every six hours daily of the experiment. Gills and liver specimens were excised from fish and preserved in 10% formal saline and processed for histological examination using standard histological techniques. The LC₅₀ was determined graphically using logarithm transformation at the end of the exposure period.

All data obtained were subjected to Analysis of variance (ANOVA) and multiple range test using the MINITAB Statistical package (V.10.51 Xtra, Minitab Inc. PA, USA).

RESULTS AND DISCUSSION

The fish in the phenergan solutions showed erratic swimming, loss of reflex, peeling of the skin, discolouration, behavioural changes and increasing opercula ventilation and movement (Table 1). Varied morphological changes occurred in the gill (degeneration and necrosis) and liver of *C. gariepinus* fingerlings exposed to phenergan which was more evident at higher concentrations and exposure time. At higher concentrations (172.5 and 207 mg/l) the gill exhibited marked alterations in the epithelial and severe epithelial disintegration occurred at lower concentrations (69.0 and 103.5 mg/l) (Table 2). Phenergan was highly toxic to *C. gariepinus*. The toxicity rate of each

organism is dosage dependent. The observation is confirmed by the significantly different mortality rates obtained due to dosage, according to Ufodike and Omoregie (1994) and Aguigwo (1998). The abnormal rapid movements of the fish subjected to high concentrations of phenergan suggest that it acted on the nerves of the fish similarly reported by Okon (1991) of pesticides.

Table 1: Behaviour features and mortality of *C. gariepinus* during 96h exposure to phenergan.

Behaviour	Concentration (mg/l)					
	Control	69.0	103.5	138.0	172.5	207.0
Erratic Swimming	-	-	+	+	+	+
Loss of Reflex	-	-	+	+	+	+
Hyper-ventilation	-	-	+	+	+	+
Changes in behaviour	-	-	+	+	+	+
Discolouration	-	-	+	+	+	+
Mortality (%)	0	5	10	20	50	100

Key:

+ Indicate Present

- Indicate Absent

Histopathological examinations of *C. gariepinus* gave significant indications of toxicity of phenergan (Table 2). The effects include gill alterations such as epithelial hyperplasia, vacuolation, cellular infiltration, degeneration of the filaments, necrosis and fusion which denotes gill functional disorders which may affect the fish physiology or cause death of the fish, while hyperplasia, hypertrophy and excessive mucous secretion are associated with asphyxiation, loss of O₂-CO₂ exchange. The gill tissue damages were similar to Annune et al.(1994) findings, leading to damage to the respiratory and osmoregulatory activities of fish.

Table 2: Histopathological changes in gills and liver of *Clarias gariepinus* exposed to phenergan.

Conc. (mg/l)	Gills	Liver
69.0	No vacuolation. Very little infiltration was observed. Lamellae were intact.	No severe cellular changes. Vacuolation within the cells.
103.5	Low level of degeneration, slightly inflammation, little cellular infiltration.	Vacuolation in the tissue parenchyma.
138.0	There was high level of vacuolation	
172.5	Cellular infiltration and level of filament degeneration .	Thickening of the nucleus (pyknosis)
207.0	Necrotic process, cellular infiltration and complete degeneration of the gill filaments were observed	Disorientation of cellular structure, fatty degeneration of hypotocytes i.e. pyknosis
Control	Normal gill filaments, normal lamellae.	No pathological features observed.

There were changes in the gills of fish species particularly the formation of vacuoles and sloughing of the epithelia layer. Oronsaye (1997) further reported that toxicants in the secondary lamellae and the enlarged sub-epithelial spaces reduced the amount of oxygen diffusing into the fish by increasing the water to blood distance similar types of gill lesions were observed. The liver of *C. gariepinus* showed disorientation of cellular structure, damages such as fatty infiltration, fatty degeneration of the hepatocytes, fatty necrosis and severe oedema. Similar effects were reported by Chinabut et al.(1978) using dipterex on freshwater fishes. Increasing mortality with increasing phenergan concentrations showed that phenergan is toxic to *C. gariepinus* and more control on the use is advocated.

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