

ACUTE TOXICITY AND EFFECT OF SOME PETROLEUM HYDROCARBON ON THE METABOLIC INDEX IN *ETROPLUS SURATENSIS*

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Acute toxicity (LC_{50}) and effect of some petroleum hydrocarbons (Toluene, Quinoline, Pyridine & Naphthalene) on the metabolic index (oxygen consumption rate) of an estuarine fish, *Etroplus suratensis* is reported. The LC_{50} values were lowest for naphthalene, suggesting that this hydrocarbon is most toxic. The oxygen consumption reduced significantly after 6 hours in all the cases. The reduction in oxygen consumption was maximum in naphthalene, reaffirming its high toxic nature. Initially the oxygen consumption rate was high in the petroleum contaminated tanks than in the control. However, it slowly decreased afterwards with slowing down in fish movement.

INTRODUCTION

Petroleum is the most critical source of energy. There is an ever increasing demand of petroleum products, world over. The increased exploration of crude oil has enhanced the possibilities of oil contamination and the problem of oil pollution in the marine environment.

Most of the crude oil differ in physical and chemical properties and therefore the study of individual component of crude oil become equally important. Such studies will offer better explanation for the differences in the effect of refined products on biota and will help in the comparison of data.

Respiratory is valuable in detecting signs of stress and metabolic involvement in fishes. The advantage of measuring oxygen consumption in fishes shows the suitability of the environment and the requirement of optimum oxygen for their survival. Therefore the oxygen uptake as an index of metabolic activity in *Etroplus suratensis* was studied using refined petroleum products.

MATERIALS AND METHODS

The experimental fish specimens were collected from the Mandovi estuary and acclimatized in the laboratory conditions for one week prior to the bioassay test. During this period the fishes were maintained at room temperature in large aquaria having seawater of 30 ± 1 ppt salinity. They were fed macroalgae. The fishes used for experiment had relatively uniform size (length 6.8 ± 1 cm, weight 10-12g).

Acute Toxicity: ANL grade toluene, pyridine, quinoline and naphthalene were used as test material for petroleum hydrocarbon. Stock solutions were prepared by dissolving a known quantity of the hydrocarbon in 100 ml of acetone. The concentration of hydrocarbon was estimated spectrophotometrically at 360 nm. From the stock solution a known amount was taken and diluted in seawater to

get the desired concentration of petroleum hydrocarbon. Test concentrations of petroleum hydrocarbon. Test concentrations were decided from the preliminary tests.

Glass aquaria with a capacity of 15 l were used for the bioassay. Fishes were randomly selected for the test. The static bioassay procedure (APHA, 1978) was followed to determine the acute toxicity value (LC_{50}) of hydrocarbon. In most cases the acute toxicity was determined by experimenting with increasing narrower ranges of hydrocarbon. In each concentration 10 test animals were introduced and the test media were renewed every 24 hours. All experiments were carried out in triplicate. Mortality in the experimental tanks were recorded at 24, 48, 72 and 96 hours. The LC_{50} values were calculated by the method of Hamilton *et al.* (1977).

Oxygen Consumption : The respiratory chambers used in this study were closed type. Experiments were carried out in 6 litre capacity cylindrical fibreglass tanks with fitted outlet. The tanks were filled with 5 l of seawater of 24 ppt salinity and subjected to aeration for 15 minutes. The dissolved oxygen level in each tank was determined by an oxygen probe. The sublethal concentrations (30 ppm toluene, 27 ppm pyridine, 14 ppm quinoline and 8 ppm naphthalene) was selected, based on LC_{50} values (96 hours) and added to the tanks. The water of each tank was stirred for 5 minutes and one fish was introduced in each tank. The water surface of respirometer was sealed with liquid paraffin to prevent interaction with atmospheric oxygen. Water samples from each tank was collected at hourly interval for 6 hours with the help of an outlet. Three samples were taken at each sampling time and the average of three reading was considered for the calculation of oxygen uptake. At the end of the experiment the wet weight of the fishes was recorded. The oxygen concentration was expressed as mg/g body weight/hour.

RESULT

The 96 hours acute toxicity experiment with different petroleum hydrocarbon on fish *E. suratensis* and the LC_{50} values thus derived are presented in Table 1. The values suggest that the fish was most sensitive to naphthalene. In other words the naphthalene was most toxic to the fish. Naphthalene was about three times more toxic than the other hydrocarbons used.

The rate of oxygen consumption in different hydrocarbons is illustrated in Fig. 1. In general the oxygen consumption rate decreased significantly after 2nd hour in all respiratory chambers. In some cases initially there was an abrupt increase in the consumption of oxygen by the fishes kept in different concentrations of petroleum hydrocarbon, as compared to the values of control. There after each hydrocarbon recorded different response of the fish. The reduction in rate of oxygen consumption was maximum in naphthalene which reaffirms its high toxic nature. The reduction in oxygen consumption was in the range of 3.4% to 57% during the 6 hours observation period. It suggest that hydrocarbon can affect the metabolic activity of the fish adversely even in low concentration. A change in the visual behaviour of the fishes was also noticed.

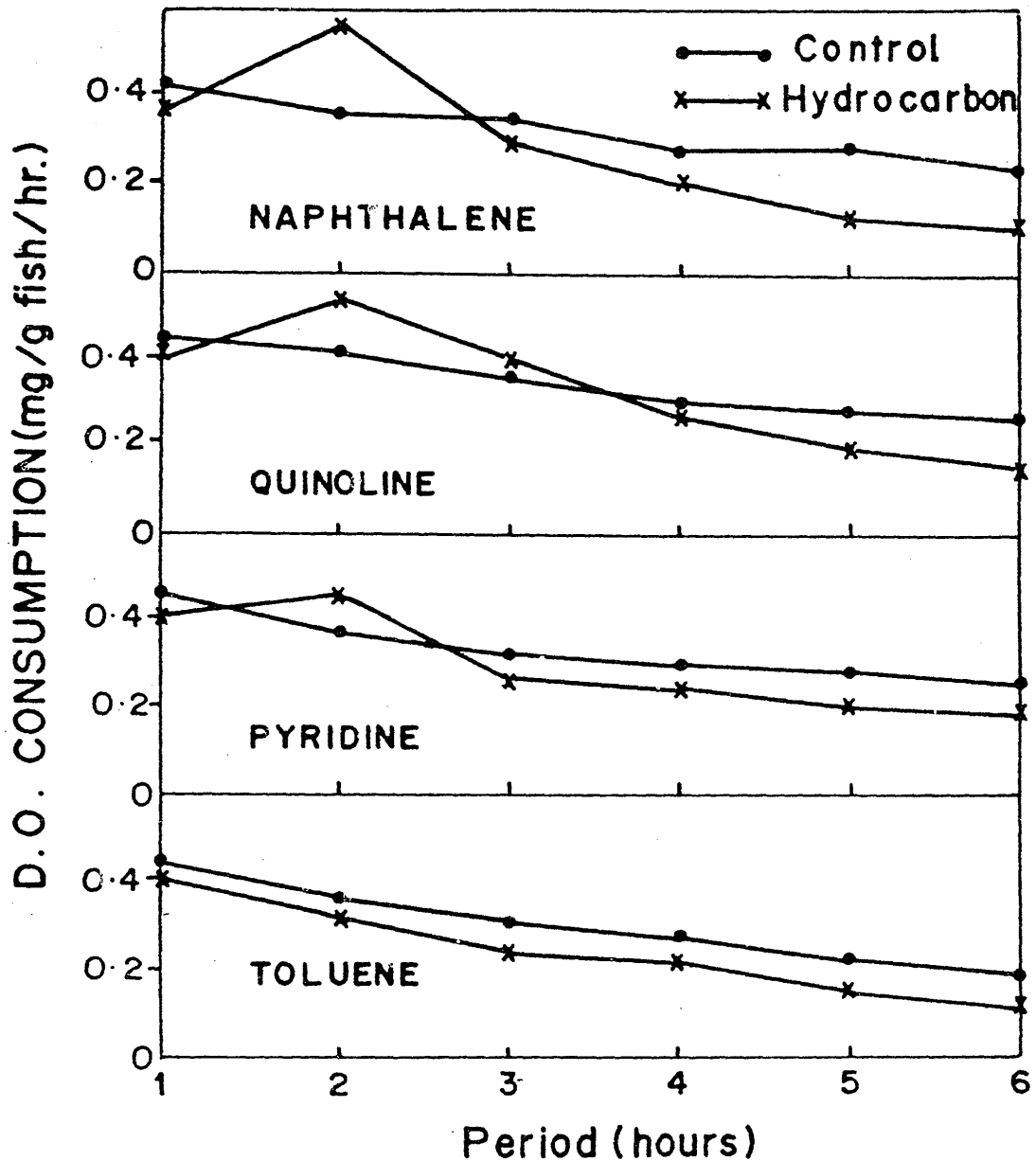


Fig. 1: Effect of hydrocarbon on oxygen consumption in *Etropolis suratensis*

DISCUSSION

The difference in the acute toxicity of hydrocarbon is related to the physico-chemical properties of the refined products. According to Nelson-Smith (1972) the wide range of degree of toxicity of various oils reported may also be related to the production of lower aromatics in these oils. The fish was unable to avoid the contact with the dissolved hydrocarbon which can easily enter the body tissue through gill. The more soluble, lower boiling aromatic hydrocarbons have also been

Table 1: Acute toxicity (C_{50} value in ppm) of petroleum hydrocarbon to *Etroplus suratensis*.

Hydrocarbon	24 hour	48 hour	72 hour	96 hour
Toluene	122	116	101	98
Pyridine	102	98	89	82
Quinoline	72	61	48	32
Naphthalene	56	42	33	23

implicated as the most toxic components of oil (Anderson *et al.*, 1974). The wide range of degree of toxicity of various hydrocarbon may also be related to the proportion of lower aromatics in the oil. It is possible that due to greater solubility and lesser degradability of these aromatic hydrocarbons, they are more persistence and hence, may be absorbed and accumulated in the tissue. The LC_{50} values calculated in the present study were similar to those reported by Dange and Masurekar (1985) for *Therapon jarbua*. These authors also reported gill hyperplasia and lesions on the body of the fish as a result of toxic effect of the hydrocarbon.

The oxygen consumption is frequently used to assess the extent of stress caused due to the pollutants. Although the oxygen consumption in fishes is complicated and depend on several factors, this study showed that the addition of petroleum hydrocarbon in the marine environment can affect the metabolic index and the status of the fish even in low concentration. Though slightly elevated values were noticed in some cases, the oxygen consumption rate was depressed in varying magnitude in general.

The initial oxygen consumption during the first hour can assigned to the excitability of the fish in the new environment. Smith (1965) has demonstrated differences in oxygen consumption between short and long term exercises for a given swimming speed which display a decreasing trend in oxygen consumption with time. Anderson *et al.* (1974) have also reported reduction in oxygen consumption in marine animals on exposure to No. 2 fuel oil.

The oxygen consumption was found to decrease with the decline in the dissolved oxygen content. This leads to a greater restriction of animal oxygen consumption. Progressive depletion of oxygen in the water breathed by a fish produces no change, but eventually leads to a greater restriction of the animals oxygen consumption. when oxygen uptake falls at low oxygen levels in water, there is an increase in the production of lactate by the fish (Holeton and Randal, 1967). An increase in ventilation volume associated with decrease heart rate appears to be general response of fish to hypoxia. Verheyen *et al.* (1985) have observed a decrease in oxygen consumption with the increased carbon dioxide concentration in water. However, no attempt was made to analyse the carbon dioxide in the water of the present study.

The overall decrease in the oxygen uptake might be associated with the lesser activity of the fish in the polluted water. Anderson *et al.* (1974) suggested a close relationship between oxygen consumption and accumulation of aromatic hydrocarbon in the tissue of the marine animals. Deshmukh and Masurekar (1983) have also reported initial increase in respiratory rate, followed by some degree of

reduction in the hydrocarbon exposed penaeid prawns. The increased activity initially will increase the ventilation leading to more uptake of pollutants in the tissue. This accumulated pollutants would have caused narcosis to sluggishness in the fishes. This might have reduced the oxygen consumption, as observed in the present study. Therefore those areas continuously receiving large quantities of toxic effluents from petroleum refineries and other industries will have larger damaging effect on the whole ecosystem.

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