Effect of Premium Motor Spirit (Petrol) on Lethality of *Tympanotonus Fuscatus* after Acute Exposure

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ABSTRACT

Tympanotonus fuscatus acclimated to laboratory conditions for seven days were exposed to different concentrations (30, 60, 90, 120 and 150ml/L) of petrol and a control for 96 hours to examine the acute effect of the petrol on Tympanotonus fuscatus. The response of the Tympanotonus fuscatus in the toxicant showed that mortality was concentration and time dependent. The 48th and 96th hour LC_{50} were 127.36 and 34.12ml/L respectively. While the 48th and 96th hour LC_{99} were 396.82 and 126.64ml/L respectively. The probit equations were however significant at 48, 72 and 96th hour. The probit equations were: Y= - 1.10 + 0.1X, Y = -0.91 + 0.014X and Y = - 0.86 + 0.25X respectively. The mean lethal time (MLT_{50}) and their associated confidence limits were: 30mg/L, 77.13 (38.50-92.11), 60 mg/L, 61.64 (30.13-86.41), 90mg/L, 68.09 (29.35-75.58), 120mg/L, 44.71 (22.98-58.30) and 150 mg/L, 43.17 (16.95-56.30). The results obtained indicated that petrol is very toxic to Tympanotonus fuscatus. Therefore adequate measure should be put in place to check avoidable spill in the environment, more especially the aquatic environment which is the natural habitat of many susceptible aquatic species.

Keywords: Premium Motor Spirit, Petrol, Lethality, Tympanotonus Fuscatus

INTRODUCTION

The aquatic ecosystem is continuously subjected to changes in quality due to the introduction of substances of diverse characteristics arising from man's activities (Oluah, 2001). Oil exploration, exploitation, transportation and storage of crude oil have greatly affected the marine environment (Doertter, 1992). Petroleum hydrocarbons are the major

contaminants of the estuarine and coastal environments (Windows *et al*.,1982), and are quantitatively the most important constituents of petroleum. They arise from natural as well as anthropogenic sources (Law and Biscaya, 1994; Medeiros *et al*., 2005). The control of such pollution problems in the aquatic environment is difficult because of the large number of input sources and their geographic dispersions (Howard et al, 2009). Studies have also shown that petroleum hydrocarbons mixes with water and penetrates to the underlying sediments (Carbioch *et al*., 1977; Patin, 1999) and therefore may constitute a problem to benthic organisms which are particularly vulnerable to oil spills and forage the bottom sediments into most pollutants (Sprague *et al*., 1981).

Petroleum (crude oil) poses a great risk to aquatic organisms which are sources of protein for the coastal dwellers in the Niger Delta (Doertter, 1992). Such risk includes: mortality (Ewa-Oboh and Otogo, 2009), stress (ATSDR, 1999), changes in species composition, low abundance, loss of species and tainting (Widdows *et al*., 1982), changes in physiological and biochemical response (Lopes et al., 2001; Jee and Kang, 2005).

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Crude oil and its products vary considerably in their toxicity and sensitivity of the organism to the product (Lopes et al., 2001). Due to the problem of crude oil toxicity examination resulting from batch differences, investigations are directed towards testing the biological effects of crude oil (petroleum) fractions (Meyerhoff, 1975). Therefore this study was undertaken to study the effects of acute toxicity of premium oil (petrol) a fraction of crude oil on periwinkles (*Tympanotonus fuscatus*) an important brackish water specie in the Niger Delta, Nigeria.

MATERIALS AND METHODS

Periwinkles (*Tympanotonus fuscatus*) of size between 4.5 - 5.5cm were handpicked at the Eagle Cement area of the New Calabar River near the Ignatius Ajuru University of Education Rumuolumeni, Port Harcourt. They were transported in plastic buckets to the Chemistry Department Laboratory of the University. Two hundred apparently healthy periwinkles were acclimated to laboratory conditions in plastic tanks of six litre capacity. The tanks were half filled with brackish water and sediments collected from same source. The acclimation was done for seven days. The substrate was prepared by air drying the sediment and then macerated in a mortar and sieved in 2mm mesh.

About 250g of finely prepared sediment were put into each of the plastic tanks to serve as the substrate base. Completely randomized design (CRD) was used for the experiment. The experiment was divided into five treatment levels with three replicates. The test media (petrol) were prepared in the following concentrations: 30.00ml/L, 60.00ml/L, 90.00ml/L, 120.00ml/L, and 150.00ml/L of petrol and a control. Ten of the test animals were introduced into each of the toxicant media. Dead periwinkles were ascertained if the animal has completely retracted into the shell or if it fails to respond to prodding of a glass rod for a period of 15minutes. Mortality assessment was carried out at defined intervals of 24, 48, 72 and 96hours.

The data obtained were subjected to analysis of variance (ANOVA) to determine if significant differences existed between the means in the mortality at different levels of contamination. Where differences existed, Duncan's multiple range test (DMRT) was used to compare the means (Zar, 1984). Toxicological response data involving quantal response (mortality) was analysed using probit analysis (Finney, 1991) to determine the lethal concentrations (LCs) and median lethal times (MLTs).

RESULTS

The total mortality of the periwinkles (*Tympanotonus fuscatus*) in the petrol media indicated a concentration and time dependent death rate. In each of the concentrations, at the different time intervals, the mortality observed were slightly higher than those in the lower concentrations. In the 24^{th} hour, the death rate in 120 and 150 ml/L concentrations were twice as high the value observed in the 30 ml/L concentration. At the 96^{th} hour, the gap in mortality trend tend to close up with deaths recorded in the 30 and 150ml/L which were 22 and 30 respectively (Table 1). The mean mortality of the periwinkle showed slight differences in values between the toxicant concentrations, but significant ($P>0.05$) variations between the time intervals. However, in the 96^{th} hour, at 150ml/L concentration all the periwinkles died (Table 2).

The lethal effects of the petrol on the periwinkles were expressed as $LC_{50,}$ $LC_{90,}$ LC_{95} and LC_{99} for 48, 72 and 96 hrs. The result showed that there was great variation between the 48^{th} and the 96^{th} hour. The values of the associated lethality decreased progressively as the

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exposure duration increased. The 96hr LC_{50} was 34.12ml/L as against the 96hr LC_{99} of 126.64ml/L, while the 48hr LC_{50} was 127.36ml/L and the 48th LC_{99} was 396.82ml/L. The probit equation were Y= -1.10+0.1X, -0.91+0.014X and -0.86+0.25X for 48, 72 and 96 hrs respectively (Table 3).

The MLT_{50}, $_{90,\ 95}$ and $_{99}$ in the exposure concentrations decreased with time and varied appreciably. The probit equation for the variation were however significant. The MLT_{50} data obtained with the lower and upper limits were 77.13 (38.50-92.11), 61.64 (30.13-86.41), 68.09 (29.35-75.58), 44.71 (22.98-58.30) and 43.17 (16.95-56.30) hours for 30, 60, 90, 120 and 150ml/L respectively. The MLT_{99} varied from 164.38 (132.78-260.18) to 101.17 (84.94-122.21) hours between 30.00ml/L to 150.00ml/L of the petrol concentrations (Table 4).

Table 1: Total mortality of *Tympanotonus fuscatus* in different concentrations of petrol after acute exposure

Time Duration	*Concentration of kerosene in mg/L*				
(Minutes)	*30*	*60*	*90*	*120*	*150*
24	5^{c}	8^{d}	6^{d}	10^{d}	11^{d}
48	11^{bc}	14^{c}	12^{c}	15^{c}	14^{c}
72	15^{b}	19^{b}	19^{b}	21^{b}	24^{b}
96	22^{a}	25^{a}	24^{a}	29^{a}	30^{a}

Means with the same alphabet in the same column are not significantly different (P>0.05)

Table 2: Mean mortality of *Tympanotonus fuscatus* in different concentrations of petrol after acute exposure

Time Duration	*Concentration of kerosene in mg/L*				
(Minutes)	*30*	*60*	*90*	*120*	*150*
24	1.67 ± 0.23^{c}	2.67 ± 1.01^{c}	2.00 ± 0.00^{c}	3.33 ± 1.23^{c}	3.67 ± 1.11^{c}
48	3.67 ± 0.32^{b}	4.67 ± 1.74^{bc}	4.00 ± 1.25^{b}	5.00 ± 0.95^{b}	4.67 ± 1.24^{c}
72	4.00 ± 1.10^{b}	6.33 ± 0.55^{b}	6.33 ± 0.67^{ab}	7.00 ± 0.00^{ab}	8.00 ± 0.00^{b}
96	7.33 ± 1.32^{a}	8.33 ± 1.54^{a}	8.00 ± 0.00^{a}	9.67 ± 2.01^{a}	10.00 ± 0.00^{a}

Means with the same alphabet in the same column are not significantly different (P>0.05)

Table 3: Mean lethal concentration of petrol to *Tympanotonus fuscatus* exposed 96 hours

Exposure Duration (hrs)	*Lethal concentrations (ml/L) with associated 95% confidence interval*					
	LC_{50}	*LC_{90}*	*LC_{95}*	*LC_{99}*	*Probit equation*	*Test sig.*
48	127.36	275.80	317.88	396.82	Y= -1.10+0.1X	***
72	64.83	156.11	181.99	30.54	Y= -0.91+0.014X	***
96	34.12	85.09	99.54	126.64	Y= -0.86+0.25X	***

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Table 4: Median lethal time of petrol with associated 95% confidence interval to *Tympanotonus fuscatus* after exposure to various concentrations of petrol

Concentration of Kero petrol (ml/L)	*Median lethal time (hrs) and associated 95% confidence interval*					
	MLT_{50}	MLT_{50}	MLT_{50}	MLT_{50}	*Probit equation*	*Test sig.*
30	77.13 (38.50-92.11)	122.96 (101.70-173.90)	137.36 (113.14-203.27)	164.38 (132.78-260.18)	Y= -1.82+0.25X	***
60	61.64 (30.13-86.41)	110.82 (90.09-147.50)	123.89 (102.41-173.23)	149.68 (122.51-224.50)	Y= -1.63+0.26X	***
90	68.09 (29.35-75.58)	99.48 (85.56-123.73)	110.07 (94.65-140.81)	129.96 (110.30-174.29)	Y= -2.13+0.034X	***
120	44.71 *(22.98-58.30)*	78.02 (63.85.96.26)	87.46 (93.07-109.16)	105.17 (88.62-135.11)	Y= -1.72+0.038X	***
150	43.17 (16.95-56.30)	75.00 (60.20-93.29)	84.18 (69.60-105.59)	101.17 (84.94-122.21)	Y= -1.73+0.04X	***

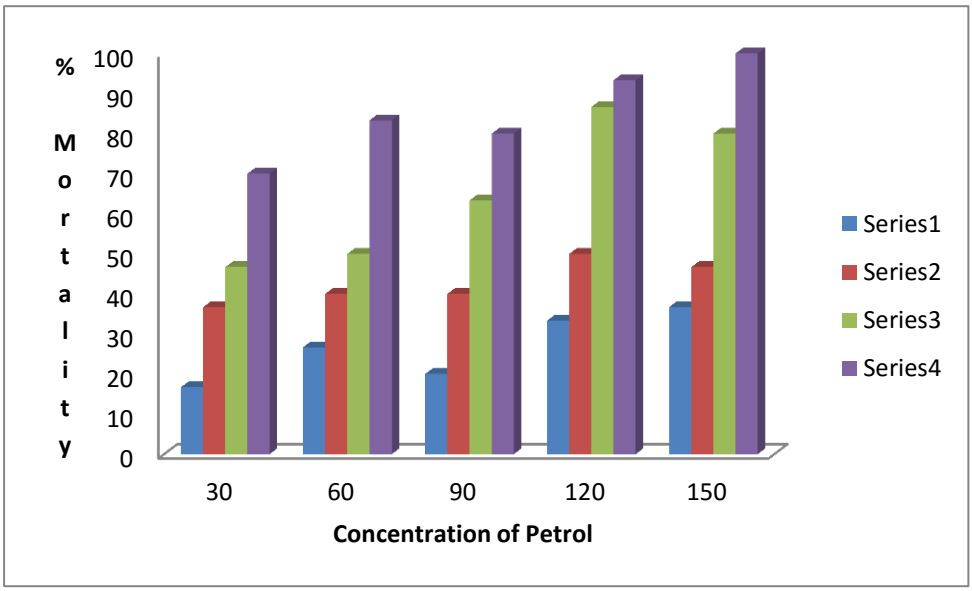

Figure 1: Percentage mortality of *Tympanotonus Fuscatus* in different concentrations of petrol. (series 1=24 hours, series 2=48 hours, series 3= 72 hours, series 4 = 96 hours).

DISCUSSION

Crude oil and petroleum fractions cause the blockade of atmospheric oxygen from dissolving in water, thereby limiting the supply of oxygen to aquatic animals, thus resulting in incidences of excretory products in the ambient water environment. The decrease in oxygen content can also explain reasons for mortality of organisms (Ugwu *et al*., 2011) which in this case is periwinkle. Chemical and physical characteristics of petroleum products dictates the penetrability of the compounds in living organisms and their metabolism and hence the toxic actions they exert on the exposed organism (Chukwu and Odunzeh, 2006). Death of organism can also result from the ability and rate at which the organism excretes transformed products

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of the chemical which may be more toxic than the parent chemical and or metabolites which may be an all important factor in determining the toxicity of chemicals (Gabriel and Edori, 2010).

Mortality of periwinkles after exposure to crude oil and other petroleum products have been reported (Renner *et al*., 2008; Ewa-Oboho and Otogo, 2009). The response to death or mortality of the periwinkles was time and concentration dependent similar to that reported in other studies with periwinkles (Chukwu and Odunzeh, 2006; Renner *et al*., 2008). The 96hr LC_{50} (34.12ml/L) observed indicated that petrol is very toxic to periwinkles. This may have resulted from the volatility of petrol and its penetrating power into the organs of the periwinkle. The interaction of the toxicant (petrol) may have altered the normal biochemistry of the organism to the extent of causing damage. The periwinkle may have adjusted its homeostatic or body defense mechanism to adapt to the new environment up to a point where it got maladjusted thereby leading to death (Gabriel and Edori, 2010).

Death of the organism may have also resulted from respiratory problems, since petrol decreases the oxygen content of aquatic environments and prevents the external organs from interacting with immediate environment by coating the organism. This situation leads to insufficient oxygen supply or utility by organism and therefore suffers from asphyxiation and the organism finally dies.

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