International Journal of Advanced Research in Biological Sciences
ISSN : 2348-8069
www.ijarbs.com

Research Article

Acute Toxicity of the Non-Selective Herbicide "Uproot"® (Isopropylamine Salt) on The Survival of Juveniles of *Clarias gariepinus* (Burchel, 1882)

Alagoa, K.J[1*]; Eremasi, Y.B[2];Ipeteikemoh, B[3]

[1]Department of Agric Education, Isaac Jasper Boro College of Education, Sagbama, Bayelsa state, Nigeria.
[2]Department of Biological Sciences, Baayelsa State College of Arts and Science, Elebele, Bayelsa State, Nigeria.
[3]Department of Mathematics, Isaac Jasper Boro College of Education, Sagbama, Bayelsa State, Nigeria.
*Corresponding author

Abstract

The acute toxicity of the herbicide uproot®(Isopropylamine salt) on the survival of juveniles of *Clarias gariepinus* was conducted in order to determine the possible effect of these group of chemicals and the possible fate on fishes in the aquatic ecosystem. Fish were exposed to 10ppm, 12ppm, 14ppm, 16ppm and 18ppm of Uproot®(Isopropylamine) in replicates of ten fish per aquaria tank. The control tank had no toxicant (0ppm). Exposure concentrations were determined after a range-finder test prior to the definitive test. The highest concentration that caused death was used as the lowest concentration in the definitive test. Fish were exposed for a total of 72hrs. Mortality was recorded every 24hours, 48hours, 72hours and the percentage mortality noted. A Probit analysis was conducted and the linear relation and the LC50 determined. Results from the test indicate that the LC50 were 16, 15, and 12 ppm respectively for 24hrs, 48hrs and 72hrs respectively. Analysis of Variance revealed a significant difference between control and treatment levels throughout the entire test period. This indicates a high toxicity of the herbicide. Care should therefore be taken in the use of herbicide especially in the rainy season because of the risk of intrusion into inland waters.

Keywords: Acute, Toxicity, Herbicide, Survival, Juveniles, *Clarias gariepinus.* Uproot®

Introduction

Herbicides are simply known as weed killers. They are employed for various purposes such as to clear waste grounds, industrial sites, railway embankment forest management and weed control in commercial agriculture. Weeds constitute perhaps the most important natural barrier to commercial agricultural crop production. All over the world, the menace of weed leads to poor yields and the loss of enumerable output. Fortunately, herbicides provide an effective and economic weed control mechanism in terms of reduced labour (Akobundu, 1987). The importance of herbicide in weed control cannot be overemphasized.
Sadly, herbicides even when applied in restricted areas are washed and carried away by rains and floods to large water bodies like ponds and rivers and alter the physicochemical properties of water (Behalchandra et al, 2001). Residues of herbicides have been detected in phyto-toxic concentrations in ground water, lakes and streams as a result of run-off from treated fields.

More disturbing is the fact that herbicides are also widely used for the control of aquatic and terrestrial plants under the formulation of different trade mark names (Okomada and Alaguba, 2011).Toxicants often contaminate fresh water bodies and affect non-target organisms including fish. The effect of some known herbicides such as Roundup®, Rramoxone® and Rodeo® on aquatic life especially fish, is well documented (Servisi et al, 1989, Henry et al, 1994; Kolo et al, 2008) while research in other combination such as Uproot® (Isopropylamine Salt) is scarce.

Fishes are by far the most useful and indeed the most priced aquatic biota to man, as they serve mostly for food among other uses.

The necessity of determining the toxicity of substances to commercially important aquatic forms at the lower levels of the food chain has been useful and accepted for water quality management (Nikam et al, 2011)
As *Clarias gariepinus* represents one of the popular fresh water fish delicacies in Nigeria and occupy flood plains, swamps and pools, it is necessary to measure their response on exposure to this widely used herbicide. In view of the poor knowledge of the aquatic side-effects of this agrochemical, the result will serve useful purposes for the management of our fisheries and the protection of the environment.

Materials and Methods

Toxicant

The general herbicide uproot® (Isopropylamine) was procured from the open market chemical store at Ekeki, Yenagoa, Bayelsa State, Nigeria. The chemical composition of the toxicant was noted from the manufacturer's instruction manual leaflet and this information cross checked against the information provided in the referral book Rhone-poutenc Laboratory chemicals and reagents. This chemical is classified in the W.H.O III classification as slightly toxic but highly inflammatory.

Test Organism

Juveniles of *Clariasgariepinus* of mean length 14.0cm±1.2cm and mean weight 8.0g0±0.3g were procured from Ellah Lakes Obrikom, Rivers State, Nigeria. They were transported in plastic containers under cool condition to the Agricultural Science Laboratory of Isaac Jasper Boro College of Education, Sagbama, Bayelsa State, Nigeria. Juveniles were chosen due to the more sensitive nature of juveniles than adult for toxicity test (Reish andOshida, 1987; Solbe, 1995; Odiette, 1999).

Acclamatization.

The fish were acclimated in big plastic basins for 7 days and fed pelleted diets at an estimated 3% body weight. Mortality during acclamation did not exceed 2% of total fish population. Therefore the fish stock was assumed to be fit and disease free.

Range Finder Test

A range finder test was conducted prior to the definitive test. This was done to determine the suitable range of concentration for the experimental test. During the range finder test, the fish were exposed to different concentrations of the toxicant in increasing log series. The highest concentration of toxicant that resulted in dead of the fish in the range-finder test was taken as the least concentration in the definitive test.

Definitive Test (Acute Toxicity Test)

Ten (10) fish were put in each aquaria tank containing 30Lt of water. A total of six aquaria tanks were used for the experiment. Exposure concentration of 10ppm, 12ppm, 14ppm, 16ppm and 18ppm of the toxicant were added to each of the aquaria tank. The toxicant concentrations decided were measured into each plastic tank using a calibrated measuring cylinder and marked accordingly. The control tank had no toxicant added to it. Proper mixing of the toxicant with the water was ensured by steering each water tank vigorously for 5minutes with a glass rod. During the test, each tank was also steered every 12hours to ensure proper mixing and proper circulation of oxygen.

Each tank was checked for mortality every 24hours. Fish were considered dead when they were non-responsive to gentle probing with a wooden stick and also when there was no opercula movement. Dead fish were carefully removed from the tanks and the numerical count noted. Fish were exposed for a total of 72 hours. Fish were fed continuously during the definitive test.

Data Analysis

Data from this investigation were analysed for percentage mortalities and transformed into their respective probits (Finney, 1952). Logs of the concentrations were plotted against the probits and the LC50 (concentration that results in 50% mortality) determined from the plot.Probit Analysis is a specialized regression model of binomial response variables.

Analysis of variance at the 95% confidence limit was conducted using the SPSS statistics tool in order to determine the relation between observed variable from the different exposure tanks.

Results

The result for the study is represented below in Tables 1 – 4 and figures 1 – 3.

The mortalities of fish for the exposure period of 72hours are reflected in Tables 1 – 4 while the graph of the linear relation between probits and concentration is shown in Figures 1-3. The fish exhibited erratic swimming immediately on exposure to the varying levels of the toxicant. There was also noticeable rapid opercula movement as the fish were seen gasping for air. Mortality occurred mainly during the first 24hours of exposure to the toxicant and later reduced in all concentration levels as time progressed. Fish were seen swimming very slowly shortly before death occurred. Twitching of fish also occurred before death.

The control tank (0ppm) fish showed no noticeable signs of stress or disturbance. There was absence of mortality in the control tank. LC50 for 24 hours was 16 ppm (16mg/L). The LC50 for 48 hours is 15 ppm while the 72 hours LC50 was recorded as 12 ppm.

Table 1: Acute mortality of *Clariasgariepinus* on exposure to Uproot®

Conc. (ppm)	Log conc.	Fish exposed	Mortality			% total mortality
			24hrs	48hrs	72hrs	
0	0	10	-	-	-	0
10	1.0	10	1	2	3	30
12	1.08	10	2	3	5	50
14	1.15	10	2	4	7	70
16	1.20	10	5	6	7	70
18	1.26	10	6	8	10	100

Table 2: Probits and % mortality of Uproot® at 24 hours

S/N	Conc.	Log conc.	Probits*	% mortality
1	0	0	0	0^{a}
2	10	1.0	3.72	10^{b}
3	12	1.08	4.16	20^{b}
4	14	1.15	4.16	20^{b}
5	16	1.20	5.0	50^{c}
6	18	1.26	5.25	60^{c}

*Transformation of percentages to Probits (Finney, 1952)
Means with the same letter superscripts are not significantly different.

Table 3**:** Probits and % mortality of Uproot® at 48 hours

S/N	Conc.	Log conc.	Probits*	% mortality
1	0	0	0	0^{a}
2	10	1.0	4.16	20^{b}
3	12	1.08	4.48	30^{bc}
4	14	1.15	4.75	40^{c}
5	16	1.20	5.25	60^{d}
6	18	1.26	5.84	80^{e}

*Transformation of percentages to Probits (Finney, 1952)
Means with the same letter superscripts are not significantly different.

Table 4**:** Probits and % mortality of Uproot® at 72 hours

S/N	Conc.	Log conc.	Probits*	% mortality
1	0	0	0	0[a]
2	10	1.0	3.72	30[b]
3	12	1.08	4.16	50[c]
4	14	1.15	4.16	70[d]
5	16	1.20	5.0	70[d]
6	18	1.26	5.25	100[e]

*Transformation of percentages to Probits (Finney, 1952).
Means with the same letter superscripts are not significantly different.

Figure 1 linear relationship between concentrations and probits(24 hrs)

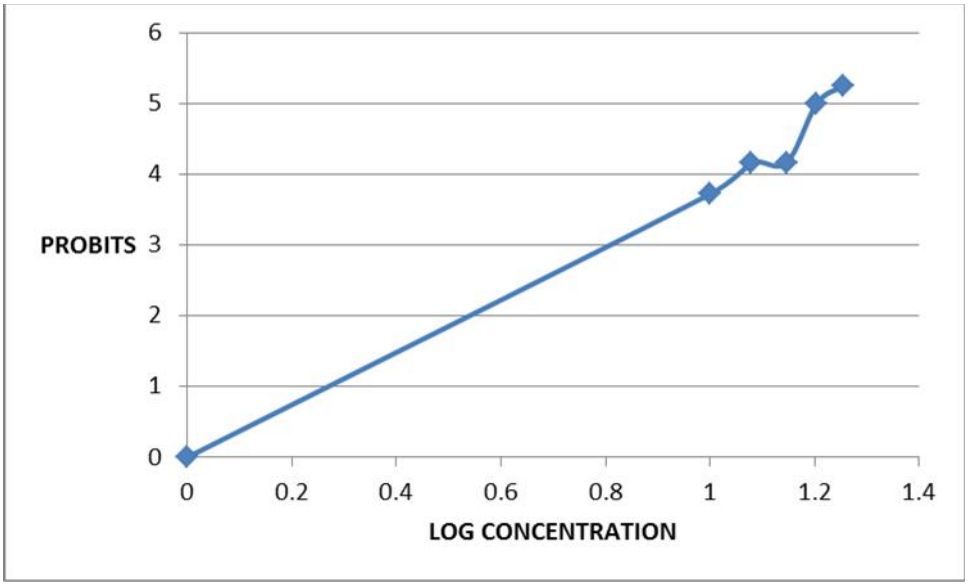


Figure 2: Linear relationship between concentration and probits (48 hrs)

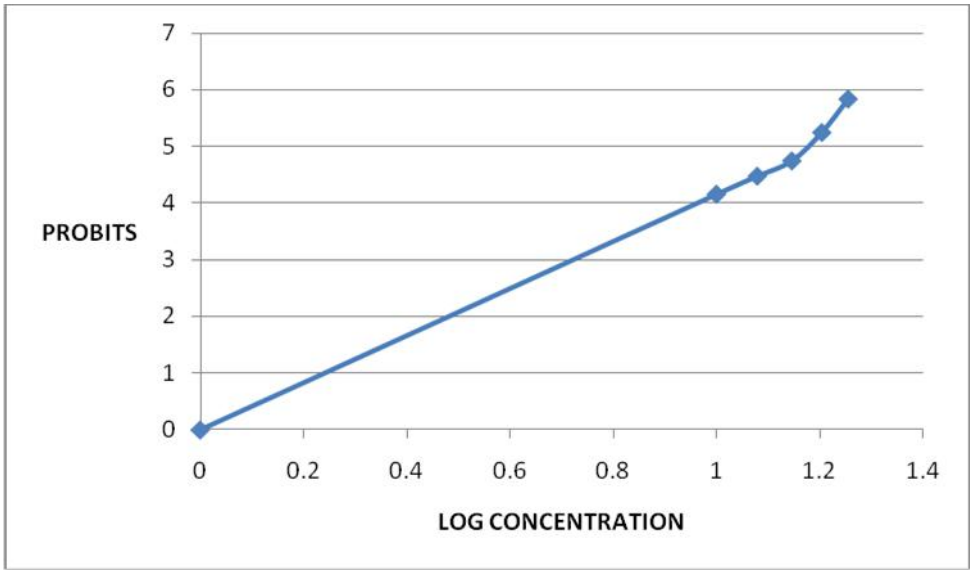
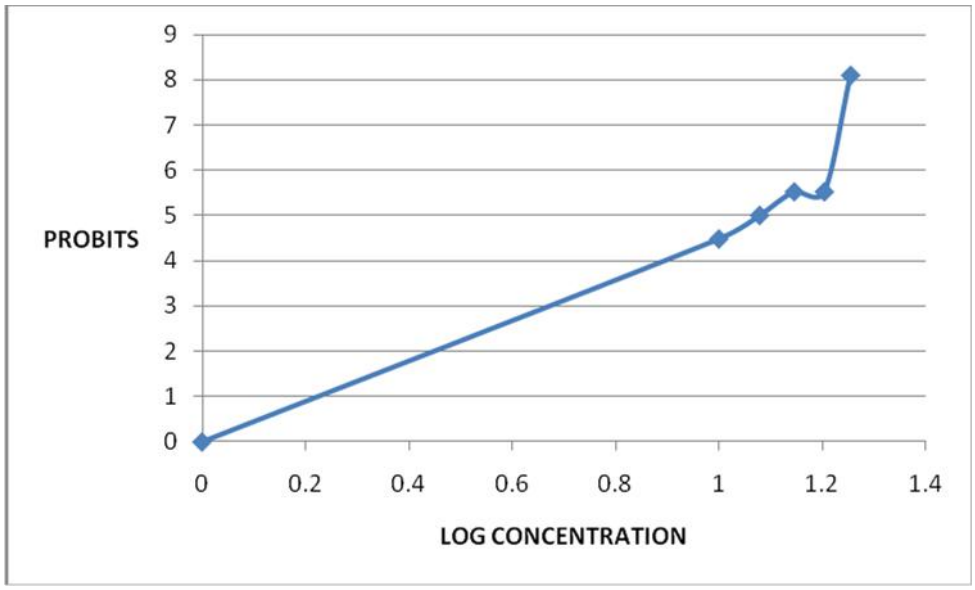


Figure 3: Linear relationship between concentration and probits (72hrs)



Discussion

Fish exposed to the different levels of toxicant exhibited erratic swimming, increase opercula movement and gasped for air shortly on exposure to the toxicant. Nikam et al, (2011) also observed similar trends when he exposed the fresh water fish *Nemacheilus botia to* acute concentrations of the organophosphate pesticide Metasystox. One reason for this kind of behavior may be connected to the fact that Herbicides react with the water rapidly to exclude oxygen from it, and the reaction of the fish is therefore a primary reaction to the need to get increased oxygen demand. Warren (1977) had earlier reported that the introduction of a toxicant into an aquatic system might decrease the dissolved oxygen concentration, which will impair respiration leading to asphyxiation. Erratic behaviour of fish in the present study indicated respiratory impairment, probably due to the effect of the glyphosate herbicide on the gills.T he fish became inactive at higher concentrations with increased time of exposure to toxicant and this is a normal observation in acute and chronictoxicity test (Kulakkattolickal and Kramer, 1997).

Toxicity increased with increased concentration. This observation is in consonance with earlier reports (GESAMP, 1991; WHO, 1994; Cox, 1998).The observed increasing state of inactivity with both increasing concentrations and exposure period agree with the report of Ayoola (2008).The level of toxicity of any toxicant depends on its bioaccumulation, the different chemistries of the compound forming the pesticide and the reactions of the organisms receiving the toxicant (Neibor and Richardso, 1980)

Also, mortality rates during the first 24 hours were highest at all concentration levels but declined as exposure times increased. This trend may be as a result of the fact that the fish tend to have obtained homeostasis as time progressed. Alagoa and Ekweozor (2009) noted that the cat fish *Clarias gariepinus* showed no noticeable differentials in blood parameters after two weeks of exposure to the dispersant Goldcrew. This demonstrates the observation of Fryer (1977) who found that in all toxicant; a threshold is reached above which there is no drastic survival of animal. Below the threshold, animal is in a tolerance zone, above the tolerance zone is the zone of resistance. Finally, Okomoda and Ataguba (2011) reported LC50 of 17.5mg/L for African catfish exposed to acute concentrations of Sunsate®. This result is consistent with the result of this study where LC50 ranged between 16ppm to 12ppm.

The result from this study demonstrates that the herbicide is toxic to fish and therefore care should be taken in its usage because of the probability of its intrusion into the natural environment.

References

Akobundu, I.O., 1987. Weed Science in the Tropics, Principle and Practice. John Wiley and Sons.ISBN 471915440.522 pages.

Alagoa, K.J.; Ekweozor, I.K.E. 2009.Toxicological & Environmental ChemistryVol.91, No. 2, March 2009, 339–343.

Ayoola, S.O., 2002. Acute toxicity, Behavioural changes and Histopathological effect of Glyphosate on tissues(gill, liver and kidney) of Nile tilapia (*Orechromisniloticus*) Juvenile. *Obeche Journal*, 20, 96-108.

Behalchandra, B.B., Wayker, I and Lornte, V.S., 2001.Acute toxicity of Pesticides Carbaryl and Endosulfan to fresh water bivalve (*Perreysiacydindrica*).Pollut. Res. 20: 25 – 29.

Cox C., 1998. Glyphosate (roundup). J. Pestic Reform, 18: 3-17.

Finney, D. J., Ed. 1952. Probit Analysis.Cambridge, England, Cambridge University Press.

Fryer JD. 1977. Weed control handbook Vol.1 Edited by Make Peace,pp. 384-389.

GESAMP (INO/FAO/UNESCO/WHO/IAEA/UN/UNDP 1991. Jointgroup of experts on the scientific aspects of marine pollution. Reviewof potential harmful substance carcinogen. *Report study*.GESAMP,40: 56pp.

Henry, C.J., HigginsK. F., and BuhlK. J. 1994. Acute toxicity and hazard assessment of RodeoR, X-77 SpreaderR, and Chem-TrolR to aquatic invertebrates. Arch. Environ. Contam.Toxicol. 27: 392-399.

Kolo, R.J.; Yisa, T.A. and Esogban, S.A., 2009. Acutetoxicity of Round up (Glyphosate) on JuvenileTilapia zilli. Journal of Applied AgriculturalResearch
1(1):105-109.

Kulakkattolickal AT, Kramer DI. 1997. The Role of Air Breathings in theResistance of Bimodally Respiring Fish to Water. J. Fish Biol. 32:119-127.

Neibor E, Richardson DH. 1980. Replacement of non-descript term heavy metal by a biological and chemically significant classification of metal ions. Environmental pollution series. 3(1): 24-45.

Nikam SM, Shejule KB, Patil RB. 2011. *Biology and Medicine, 3 (4): 13-17.*

Odiette WO 1999 Environmental physiology of Animal and pollution. 1st Ed. Diversified Resources Ltd. ISBN 978-028-957-7; Lagos. 360 pp.

Okomoda V.T. and Ataguba G.A. 2011. Blood glucose response of *Clariasgariepinus* exposed to acute concentrations of glyphosate-isopropylammonium (Sunsate®).Journal of Agricultural and VeterinarySciences. 3(6): 69-75.

Reish DL, Oshida OS., 1987. Manual of Methods in aquaticenvironment research.Part 10. Short- term static Bioassays. FAOFisheries Technical Paper No. 247. Rome 62pp.

Rhone-Poulenc. 1992. Laboratory Chemicals and Reagents. Prolabo, 12 Rue Pelee-75011 Paris. 562pp.

Servizi JA, Gordon RW, Martens DW. 1989 Acute toxicity of Garlon 4and round up herbicides to salmon, daphnia and trout. Bull. Environ.Contam.Toxicol. 39: 15-22.

Solbe JF 1995. Fresh water in: Handbook of Ecotoxicology (Edited byPeter Collins) Black Well Science Ltd. Osneymeed OX 20EL. 683pp.

Warren CE 1977.Biology and water pollution. W.B. Sanders andCompany Philadelphia, USA, 434pp.

World Health Organization (WHO) 1994: Glyphosate. EnvironmentalHealth Criteria, Publication NO 159, Geneva, Switzerland.