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DURATION OF EXPOSURE DEPENDENT EFFECT OF CARBAMATE TREATED NET ON SERUM LIPID PROFILE OF WISTAR RATS

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ABSTRACT

The duration of exposure dependent effect of Carbamate treated net on serum lipid profile of female Wistar rats was investigated in this study. Eighteen (18) animals were randomly divided into three (3) study groups with six (6) rats each. Group 1 was designated as the control group while groups 2 and 3 constituted the experimental groups containing rats exposed to Carbamate treated net for 30 and 60 days respectively. The rats were exposed daily to Carbamate treated net by whole body and nose inhalation mode of exposure. The body weight of rats was measured at the commencement of the experiment and at the end of the experiment. The organ weight result obtained indicated that the body weight increased significantly in the rats of the experimental groups when compared to the rats in the control group at $p<0.05$. The result obtained indicated that the body weight increased significantly in the rats of the experimental groups when compared to the rats in the control group at $p<0.05$. The total cholesterol (TC), triacylglycerol (TG), high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) concentrations in the sera of the rats were determined at the end of the experiment. The result obtained showed that the TC, TG, HDL-C and LDL-C levels increased significantly in the rats of the experimental groups when compared to the rats in the control group at $p<0.05$. The observation from this study suggests that long-term exposure to Carbamate treated net in household setting may alter the level of the serum lipid profile of the experimental animals, thereby leading to various health complications.

KEYWORDS: Carrbamate**,** total cholesterol (TC), triacylglycerol (TG), high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C

INTRODUCTION

Malaria is a life-threatening disease that is caused by plasmodium parasite which is usually transmitted to humans through bites of infected female Anopheles mosquito. It remains a major cause of mortality and morbidity in the world and particularly in sub-Saharan Africa [Ekam et al., 2019 and Zain et al., 2000]. The use of insecticide treated nets has proven to be one of the most effective preventive measure of malaria during the past decade, they not only prevent entry of mosquitoes into the net but kills them as well thus reducing exposure of man to mosquito bites [Ukpanukpong et al., 2018]. Roll back malaria (RBM) has also stated that one of the strategies set to fight malaria is to increase the utilization of mosquito nets as it is also a cost-effective intervention which may also provide community protection through mass impact on vector mosquito population when used at a high coverage rate [Roll Back Malaria, 2005].

WHO had identified pyrethriods as the major insecticide recommended for treatment of mosquito net owing to their strong insecticidal activity [Diaro et al., 2015] but as a result of widespread resistance of mosquito parasite to pyrethriods, research into the use of other insecticides such as carbamates, organochlorines, organophosphates etc. have been greatly encouraged and the use of these different classes of insecticides with different modes of action in combination with pyrethriods have also been recommended especially in factory production of wash resistant insecticide treated nets also known as permanent insecticide treated nets [Pierre and Guessain, 2004]. One of the factors that determines the efficacy of these insecticide treated nets (ITNs) in preventing malaria is the concentration of the insecticides use in treating them [Jima et al., 2007], but many of the chemical constituents of these insecticides that serve as their active ingredients are considered to have the potential to affect human health as they tend to

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accumulate in the tissues of organisms that are exposed to them when they come in contact with them either directly via dermal and pulmonary absorption or indirectly via food across the gastrointestinal tract (GIT) [Malaria Consortium, 2016].This is mostly as a result of poor knowledge, ignorance, illiteracy and abuse of the use of ITNs as can be seen in cases of skin irritation or reaction which occurs when nets are used without first airing them properly and use of ITNs as hedges in farmlands especially in rural areas or communities [Ukpanukpong et al., 2019a].

Insecticide treated net and Long-lasting insecticidal net are the most commonly used malaria control tool to be developed since the advent of indoor residual spraying and chloroquine in the 1940s, and as such they have been an important component of global and national malaria control policies since the mid-1990s. Insecticide treated net is a mosquito net that is treated with an insecticide that kills mosquitoes which comes in contact with it, or repels them and Long-lasting insecticidal net is a mosquito net that has been treated with insecticide during the manufacturing process which does not require retreatment with insecticide and lasts for up to three years. The use of mosquito treated net has increased from only 3% to 60% of African children sleeping under an insecticide treated net [Jenny and Mark, 2006]. Sleeping under an insecticide treated net is the most widely adopted preventive measure against malaria because they form a protective barrier around people sleeping under them against mosquitoes and the insecticides that are used for treating them kill and also repel mosquitoes, thus reducing the number that enter the house and attempt to feed on people inside. They have proven to be effective in many areas of malaria-endemic regions of the world [Odutuga et al., 2016].

It is estimated that the number of cases averted since 2000 and the cost associated with care in the public sector, insecticide treated net and long-lasting insecticidal nets saved US $900 million dollars between 2000 and 2014. As a result, over one billion nets have been distributed to Africa alone since 2000, between 2008 and 2010, a total of 294 million nets were distributed in sub-Saharan Africa. Of the US$1.6 billion invested in malaria commodities in 2014, 63 percent was spent on insecticide-treated nets [WHO, 2015]. Hence, insecticide treated nets and long-lasting insecticidal nets, in particular, represent a cost effective means of malaria prevention for a risk population and are currently recommended as the first choice net by the World Health Organization [WHO, 2015]. Factors that determine the efficacy of insecticide-treated nets and long-lasting insecticidal nets include the type of nets used, the class of insecticide used in treating them, frequency of retreatment, cost of the insecticide, level of pyrethroid resistance, funding for nets, lifespan of nets, awareness of its importance, proper use and maintenance of the nets etc [CDCP, 2019].

Pesticides comprise a large group of substances with the common characteristic of being effective against pests. This group include insecticides, herbicides, fungicides, nematocides etc. which are formulated to kill or inhibit the growth of plant and animal competitors which interfere with man's health, comfort or production of food [Piage et al., 2010]. Hence, pesticides are of vital importance in the fight against diseases. Notwithstanding their efficacy against pest, they can cause some adverse effect to various body organs, tissues, systems, alteration in biological parameters and human health at large on exposure to them or if abused, because they contain various chemical constituents [Murray et al., 2013]. Organophosphate and carbamate insecticides form are the groups of chemicals that are mainly used in agriculture and medicine [Jaga et al., 2003].

Carbamate is a chemical compound that is formally derived from carbamic acid (NH_2COOH). The term includes organic compounds (e.g., the ester ethyl carbamate), formally obtained by replacing one or more of the hydrogen atoms by other organic functional groups; as well as salts with the carbamate anion H_2NCOO^- (e.g. ammonium carbamate). Carbamate insecticides have the carbamate ester functional group. Included in this group are aldicarb (Temik), carbofuran (Furadan), carbaryl (Sevin), ethienocarb, fenobucarb, oxamyl, and methomyl. These insecticides kill insects by reversibly inactivating the enzyme acetylcholinesterase [Piage et al., 2010]. Carbamates represent one of the main category of synthetic organic pesticides which was introduced into the agrochemical market in the 1950s and are used annually on a large scale worldwide [Piage et al., 2010].They constitute a versatile class of compounds and due to their high range of activities they are used as insecticides, fungicides, nematocides, acaracides, molluscicides, sprout inhibitors or herbicides. Carbamates are effective insecticides by virtue of their ability to inhibit the enzyme acetylcholinesterase in the nervous system. The mechanism of carbamates poisoning, except herbicidal carbamates, involves carbamylating of the active site leading to the inactivation of this essential enzyme which has the important role of catalysing the hydrolysis of the neurotransmitter acetylcholine (ACh) to choline and acetic acid. Hence, Carbamate compounds are often called anticholinesterases because like organophosphates, they can inhibit esterases that have serine in their catalytic centre [Jokanovic and Maksimovic, 2009].

In the presence of inhibitors, acetylcholinesterase becomes progressively inhibited and is not further capable of hydrolyzing acetylcholine to choline and acetic acid [Jokanovic, and Maksimovic, 2009]. Consequently, acetylcholine accumulates at cholinergic receptor sites and produces effects equivalent to excessive stimulation of cholinergic receptors throughout the central and peripheral nervous system. The Inhibited enzyme can be spontaneously reactivated, with reversal of inhibition occurring typically with half-time of an hour or less after exposure. This fact may reduce the possible period of intoxication in situations of accidental overexposure or suicide attempts. The inhibition of other esterases may also occur [Thompson, 2000 and Jokanovic, 2008].

Thus, inhibition of AChE by carbamate esters, causes toxic effects in animals and human beings that result in a variety of poisoning symptoms and eventually culminate in respiratory failure and death. The site for carbamylation of the enzyme is the hydroxyl moiety of the serine amino acid. The rate of regeneration of the carbamylated enzyme to AChE is relatively rapid compared with that of an enzyme that has been inhibited (phosphorylated) by an organophosphorus pesticide [Reiner, 2001]. Therefore, human exposure to the

carbamate pesticides is less dangerous than exposure to organophosphorus pesticides, because the ratio between the dose required to produce mortality and the dose required to produce minimum poisoning symptoms is in general, substantially larger for carbamate compounds than for organophosphorus compounds [Ukpanukpong et al., 2017].

MATERIALS AND METHODS: Animal cages, Carbamate treated net, water bottles, feeding troughs, hand gloves, nose mask and plain vial tubes.

Chemicals and reagents: Carbamate, deionised water, buffer formalaldehyde and chloroform.

Equipment: Electronic weighing balance, chemistry analyzer, curvet, refrigerator, dessicator, homogenizer, spectrophotometer, centrifuge, dissecting set and plain sample bottles.

Animal handling design: Eighteen (18) albino wistar rats (Rattus norvegicius) weighing 140g – 180g were used for the experiment.. The rats were housed in a standard laboratory cages and kept in the animal house of the Department of Biochemistry, University of Calabar, Calabar. The cages were cleaned regularly and the animals were fed with pelletized feed and allowed access to water. The animals were randomly divided into three (3) study groups consisting of six (6) animals each. Group 1 was the control group while groups 2 to 3 were the experimental groups. Acclimatization of the animals to the environmental conditions was allowed for two (2) weeks prior to the commencement of the experimental carbamate exposure procedures.

MODE OF EXPOSURE

A modified whole body and nose-inhalation exposure method which involved placing the cage housing the animals in the test groups under carbamate treated net was used. The animals were exposed for 9 hours per day to the net throughout the respective exposure periods to established full contamination. At the end of the exposure time each day, the animals were removed from the exposed nets and transferred to carbamate free section of the animal house.

COLLECTION AND PREPARATION OF SAMPLES FOR ANALYSES

At the end of the experimental period, the rats in each study group were anaesthetized with chloroform vapour and sacrificed by dissection. Blood was collected from each rat by cardiac puncture and put into well labelled plain screw-cap sample bottles for serum separation. The blood samples collected were allowed to clot and the sera separated with Pasteur pipette, after spinning with MSE model (England) table centrifuge for 3000 rpm for 10 minutes. The separated serum samples were then used for biochemical analyses. Basically, all biochemical analyses were carried out within 24 hours of serum separation.

SERUM BIOCHEMICAL ANALYSES

Triacylglycerol (TAG), total cholesterol (TC), high density cholesterol (HDL) and low density cholesterol (LDL) concentrations were determined in serum by colorimetric methods using commercial assay kits. The assay procedures used were as contained in the kit literature.

Experimental design

Group	No. of rats	Treatment
Control group	6	Water and rat pellets
Test group 1	6	Exposure to carbamate treated net for 40days
Test group 2	6	Exposure to carbamate treated net for 60days

Lipid profile determination

Serum total cholestrol (TC) determination

Principle: Cholesterol was measured in serum, in a series of coupled reactions that hydrolyze cholesteryl esters and oxidize the 3-OH group of cholesterol. The free cholesterol was and then oxidised. One of the reactions by-products, hydrogen peroxide then combined with phenol and 4-aminoantipyrine catalysed by peroxidase to produce a red coloured quinoneimine which was quantitatively measured at absorbance of 546nm. The intensity of the colour produced is directly proportional to the total cholesterol concentration of the sample.

Cholesterol ester + H_2O Cholesterol esterase Cholesterol+Fatty acid

Cholesterol + O_2 Cholesterol oxidase Cholestene-3-one + H_2O_2

$2H_2O$ + Phenol + 4-aminoantipyrine Peroxidase Quinoneimine + $4H_2O$

Calculation: The concentration of cholesterol in the sample was calculated using the formula

Cholesterol concentration = x Concentration of standard

Where sample A represent absorbance of sample and standard A represents absorbance of standard

Serum triacylglycerol (TAG) determination

Principle:

The triacylglycerides were determined in serum using a series of coupled reaction in which triglycerides were hydrolyzed to produce glycerol and fatty acids by lipases. The resultant glycerol was then phosphorylated by ATP in the presence of the enzyme, glycerol kinase to glycerol-3-phosphate oxidase which was further oxidised to dihydroacetone phosphate by glycerol phosphate oxidase. Hydrogen peroxide was produced as a by- product. The final step was the reaction of hydrogen peroxide, 4-aminophenazone and 4-chlorophenol catalysed by peroxidase to form the

indicator, quinoneimine as well as hydrochloric acid and water. The concentration of H_2O_2 was directly proportional to the intensity of the colour and hence the triacylglycerides. Absorbance was measured at 500nm.

Triglyceride + H_2O Lipase Glycerol+Fatty acids
Glycerol + ATP Glycerokinase Glycerol-3-phosphate+ADP
Glycerol-3-phosphate + O_2 Glycerophosphate oxidase Dihydroxyacetone + Pi + H_2O_2
$2H_2O_2$ + 4-aminophenazone+4-chlorophenol Peroxidase Quinineimine+HCl+$4H_2O$

Calculation: The concentration of triacylglyceride in the sample was calculated using the

Formular: Triacylglyceride concentration =x Concentration of standard

High density lipoprotein cholesterol (HDL-C) determination

Principle:

HDL was measured directly in serum by selectively precipitating chylomicrons, very low density lipoproteins (VLDL) and low density lipoproteins (LDL) with phosphotung state and magnesium reagent .The mixture was then centrifuged, the supernatant obtained contains HDL-C which was determined according to the mono reagent enzymatic colorimetric end point method as previously described for serum cholesterol level determination.

Calculation

HDL-Cholesterol (mg/dL) in supernatant = x Conc. of standard

Low density lipoprotein cholesterol (LDL-C) determination

Principle:

Low density lipoprotein-cholesterol was determined using the method described by Friedwalds et al., (1972). According to the Friedwalds relationship, low density lipoprotein –cholesterol is derived from measured values of total cholesterol, triglycerides and HDL-C. This was due to the fact that most of the circulating cholesterol were found in the three major lipoproteins fractions i.e very low density lipoproteins (VLDL), low density lipoproteins (LDL) and high density lipoproteins (HDL).

Calculation: LDL-C = Total cholesterol – (HDL-C + TG/5)

Where TG/5 is an estimate of VLDL-C. All the values are expressed in mg/dL.

Statistical analysis: The data collected were analysed using student't' test and values expressed as Mean ± SEM at $p<0.05$ to determine if there was a significant difference between test and control group.

RESULT

Duration of exposure dependent effect of carbamate treated net on body weight changes.

The result of the duration of exposure dependent effect of carbamate treated net on the body weight of Wistar rats is given in table 1. It was observed that 30 days of exposure to carbamate showed significant increase in the body weight of rats in group 2 (175.15 ± 1.14) when compared to rats in group 1(control) (104.93 ± 1.09), and 60 days of exposure to carbamate showed significant increase in the body weight of rats in group 3 (202.29 ± 0.46) when compared to rats in group 1(control) at $p < 0.05$.

Table 1: Effect carbamate treated net on Body weight indices of albino wistar rats.

TREATMENT PERIOD	BODY WEIGHT(g)
0 Day	140.93 ± 1.09
30 Days	175.15 ± 1.14 [a]
60 Days	202.29 ± 0.46 [b]

Values are expressed as Mean ± SEM, n=6
a= significantly different from control group at $p<0.05$
b= significantly different from control group at $p<0.05$

Duration of exposure dependent effect of carbamate treated net on organs weight indices of albino wistar rats. The result of the duration of exposure dependent effect of carbamate treated net on the liver weight of albino Wistar rats is given in table 2. It was observed that 30 days of exposure to carbamate showed significant increase in the liver weight of rats in group 2 (7.77 ± 0.34) when compared to rats in group 1(control) (6.08 ± 0.26) and 60 days of exposure to carbamate showed significant increase in the liver weight of rats in group 3 (9.91 ± 0.08) when compared to rats in group 1(control) at $p < 0.05$. The result showed that 30 days of exposure to carbamate resulted in a statistically significant increase in the weight of the heart of rats in group 2 (0.56 ± 0.01) when compared to rats in group 1(control) (0.38 ± 0.01) and 60 days of exposure to carbamate also resulted in a statistically significant increase in the liver weight of rats in group 3 (0.72 ± 0.02) when compared to rats in group 1(control) at $p < 0.05$. The result also showed that 30 days of exposure to carbamate showed significant increase in the weight of kidney of rats in group 2 (0.56 ± 0.01) when compared to rats in group 1(control) (0.46 ± 0.01) and 60 days of exposure to carbamate showed significant increase in the liver weight of rats in group 3 (0.75 ± 0.01) when compared to rats in group 1(control) at $p < 0.05$.

Table 2: Effect of carbamate treated net on relative organ weight of Wistar rats

Treatment Period	Liver (G)	Heart (G)	Kidney (G)
0 Day (GROUP 1)	6.08 ± 0.26	0.38 ± 0.01	0.46 ± 0.01
30 Days (GROUP 2)	7.77 ± 0.34[a]	0.56 ± 0.01[a]	0.56 ± 0.01[a]
60 Days (GROUP 3)	9.91 ± 0.08 [b]	0.72 ± 0.02[b]	0.75 ± 0.01[b]

Values are expressed as Mean ± SEM with n=6
a= significantly different from control group at p<0.05
b= significantly different from control group at p<0.05

Duration of exposure dependent effect of carbamate treated net on serum lipid profile of albino wistar rats. The result of the duration of exposure dependent effect of carbamate on serum TC level of albino Wistar rats is presented in table 4.. A statistically significant increase in TC level was observed at P<0.05 in rats of group 2 which were exposed to carbamate for 30days (40.50 ± 0.24) when compared to rats in group 1 (control) (37.83 ± 1.43) and showed significant increase in TC level at P<0.05 in rats of group 3 which were exposed to carbamate for 60days(40.50 ± 0.25) when compared to rats in group 1 was also observed. The result of the duration of exposure dependent effect of carbamate on serum TG level of albino Wistar rats is presented in Table 3. The result showed a significant increase in TG level at P<0.05 in rats of group 2 which were exposed to carbamate for 30days (75.00 ± 0.28) when compared to rats in group 1 (control) (71.50 ± 1.43) and a significant increase in TG level at P<0.05 in rats of group 3 which were exposed to carbamate for 60days (86.50 ± 0.68) when compared to those in group 1 (control). Therefore, exposure to carbamate treated net was observed to cause a significant increase at P<0.05 in serum TG level of rats in the experimental groups.

The result of the duration of exposure dependent effect of carbamate on serum HDL-C level of albino Wistar rats is presented in table 3. The result showed a significant increase in HDL-C level at P<0.05 in rats of group 2 which were exposed to carbamate for 30days (35.00 ± 0.40) when compared to rats in group 1 (control) (29.67 ± 0.23) and also showed a significant increase in HDL-C level at P<0.05 in rats of group 3 which were exposed to carbamate for 60days (39.67 ± 0.23) when compared to those in group 1 (control). Therefore, exposure to carbamate treated net was observed to cause a significant increase at P<0.05 in serum HDL-C level of rats in the experimental groups.

The result of the duration of exposure dependent effect of carbamate on serum LDL level of albino Wistar rats is presented in table 3. A significant increase in serum LDL level was observed at P<0.05 in rats of group 2 which were exposed to carbamate for 30days (21.30 ± 0.37) when compared to rats in group 1 (control) (17.33 ± 0.23) and a significant increase in serum LDL level at P<0.05 in rats of group 3 which were exposed to carbamate for 60days (27.67 ± 0.26) when compared to those in group 1 (control) was also observed. Therefore, exposure to carbamate treated net was observed to cause a significant increase at P<0.05 in serum LDL level of rats in the experimental groups.

Table 3: effect of carbamate treated net on serum lipid profile of albino wistar rats.

Treatment Period	TC (mmol/L)	TG (mmol/L)	HDL (mmol/L)	LDL (mmol/L)
0 Day (GROUP1)	37.83 ± 1.43	71.50 ± 1.43	29.67 ± 0.23	17.33 ± 0.23
30 Days (GROUP 2)	40.50 ± 0.25	75.00 ±0.28[a]	35.00 ± 0.40[a]	21.33 ± 0.37[a]
60 Days (GROUP 3)	40.50 ± 0.25	86.50 ±0.68[b]	39.67 ± 0.23[b]	27.67 ± 0.26[b]

Values are expressed as Mean ± SEM with n=6

TC=Total Cholesterol
TG=Triglyceride
HDL=High density lipoprotein
LDL=Low density lipoprotein
a = Significantly different from control group at P <0.05
b= Significantly different from control group at P <0.05

DISCUSSION

The present study was designed to evaluate the duration of exposure dependent effect of carbamate treated net on serum lipid profile of Wistar rats. The study was carried out using eighteen (18) Wistar rats that were randomly divided into three (3) study groups consisting of one control group and two experimental

groups. The body and organ weight indices of the rats were also measured.

The result obtained from the study showed that the final body weight changes of rats in the experimental groups at the end of the experiment indicated a significant increase when compared to the initial weight of rats of the control group at $p<0.05$.This may be as a result of the effect of exposure to chemicals on the metabolic rate of the body and is in agreement with Akinola and Ukpanukpong [2015] who reported that chemicals on entry into the system of an organism alters the normal physiological state of the system as a result of the unwanted chemical reactions which they elicit. The result also showed a significant increase in the organ`s weight of rats in the experimental groups when compared to the initial weight of rats of the control group at the commencement of the experiment at $p<0.05$. This may be as a result of exposure of animals to chemicals that exert toxic effects on vital body organs like the kidney, liver, lung and brains and is in line with Ukpanukpong et al, [2019b] whose study showed that toxic chemicals affect vital body organs. Analysis of body and organ weight in toxicology studies is an important endpoint for identification of potentially harmful effects of chemicals in experimental groups and is correlated since increase in organ weight of an organism is usually accompanied by and results in increase in body weight of that organism [Jaga et al., 2003 and Ukpanukpong et al., 2016]

The serum lipid profile comprising of total cholesterol (TC), triglyceride (TG), high density lipoprotein-cholesterol (HDL-C) and low density lipoprotein-cholesterol (LDL-C) of the rats in the experimental groups, comparison with that of the rats in the control group were observed and recorded in the result. The result indicated a significant increase in the TC level of the experimental groups, when compared in to rats in the control group at $P<0.05$. This may be due to lipogenesis of the liver in response to exposure to chemicals and other xenobiotics [Murray, 2003]. This result is in accordance with the report of Uboh et al, [2007 and Aliyu et al, [2016] who noted that exposure of animals to chemicals enhances the serum TC level of that organism. Cholesterol aids in biosynthesis of hormones, bile acid and vitamin and serve as a component of cell membrane. The result obtained from the experiment also indicated a significant increase in the TG level of the experimental groups, when compared to rats in the control group at $P<0.05$. This may be due to exposure to chemicals which is known to cause alterations in lipid metabolism and oxidative stress [Aliyu et al., 2016] and is in accordance with the report of Uboh et al, [2007] who noted that exposure of animals to chemicals enhances the serum TG level of that organism. Triglyceride is a blood lipid which enables the bidirectional transference of adipose fat. The result obtained from the experiment also indicated a significant increase in the serum HDL level of the experimental groups, when compared to rats in the control group at $P<0.05$. This may be as a result of exposure to chemical which causes increased level of bio-activated compounds in an organism leading to potential adverse effects [Ukpanukpong et al., 2013]. This result is in contrast to that of Uboh et al, [2007] who noted that exposure of animals to chemicals decreases the serum HDL level of that organism, but is in accordance with Saheed et al, [2015] whose study indicated an increase in serum HDL-C level in response to exposure to chemicals. HDL-C is an essential lipoprotein which is required for transportation of fat molecules out of the walls of the artery and from macrophages and other peripheral tissues along with subsequent transfer of cholesteryl ester to plasma or hepatic acceptors. The result obtained from the result also indicated a significant increase in the LDL-C level of the experimental groups, when compared to rats in the control group at $P<0.05$. This may be due to exposure of animals to chemicals which may cause the effectiveness in regulation of lipid metabolism by the tissue to be compromised [Murray et al., 2013] and is in accordance with the report of Uboh et al,[2007] who noted that exposure of animals to chemicals enhances the LDL-C level of that organism. LDL-C is an essential lipoprotein which is required for transportation of fat molecules into the walls of the artery and as such has been associated with promoting the development of atherosclerosis. The result of the lipid profile is in agreement with Ejoba et al, [2013 and Ukpanukpong et al, [2018] who reported that changes in lipid profile have been used toxicologically in evaluating the toxicity of chemicals and other xenobiotics in the living system and that serum lipid profile serves as diagnostic biochemical index in some clinical conditions such as chronic obstructive jaundices, hepatitis and coronary artery disease..

CONCLUSION: Conclusively, this study indicates that exposure to carbamate treated net may have effect on the body weight, organ weight and serum lipid profile of albino Wistar rats depending on the duration of exposure.

CONTRIBUTION OF AUTHORS: Authors Enyievi Peter Bassey and Asuquo Dorcas Daniel carried out the research, laboratory work and wrote the first draft of the manuscript. Author Essien Nsima. Monday supervised the research work and author Ukpanukpong Richard Undigweundeye made extensive corrections to the manuscripts.

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REFERENCES

Akinola, F. F. and Ukpanukpong, R. U. 2015**.** Hepato-protective effect of artesunate on carbon tetrachloride-induced toxicity in rats. International Journal of Pharma Sciences and Research, 6(5):869-873.

Aliyu Ahmed Warra, Richard Ukpanukpong Undigweundeye and Ibrahim Garba Wawata, 2016. Physio-chemical and GC-MS Analysis of Calabash (Lageneria siceeraria) seed oil. International Journal of Biochemistry Research and Review 14(1): 1-7.

CDCP [Centre for Disease Control and Prevention], 2019. Factors that affect efficacy of mosquitoes net in treatment of malaria.

Dairo Joshua Olugbenga, Ukpanukpong Richard Undigweundeye and Uyabeme Rachael Ngozi 2015.

Phytochemical Screening, Proximate Analysis and Acute Toxicity Study of Launaea Taraxacifolia Ethanolic Extract on Albino Rats. The International Journal of Science and Technoledge (ISSN 2321 – 919X) www.theijst.com 199 Vol 3 Issue 6 June.

Ejoba, R., Utu-Baku, B. A., Ukpanukpong, R. U., Pekene, D. J. B., Dasofunjo, K., Ugwu, M. N., Abong, A, A. and Ekpo, B. O. 2013. Effect of Saccharum barberi extract on lipid profile level in guinea pigs. Australian Journal of Basic and Applied Sciences, 7 (8): 857-862.

Ekam, V. S., Ukpanukpong, R. U., Ihe, S. O., Odio, S. B. and Ekam, N. V. 2019. The assessment of serum liver enzymes in malaria induced Wistar rats treated with the crude extract of Artemisia annua and Artemisinin combination therapy. International Journal of Medicine Research, 4(3):84-86.

Jaga, K., Francis, M. and Dharmani, C. 2003. Sources of exposure to and public health implications of Organophosphosphate pesticides. Panam Salud Publication. 7(14):171-185.

Jenny, H. and Mark, R. 2006. Relevances in parasitology, 61(77–128) retrieved from https://doi.org/10.1016/30065 - 308(05) 61003-2.

Jima, D., Getachew, H. and Bilark, M. 2007. Malaria indicate survey; coverage in use of major malaria prevention and control intervention. Malaria Journal, 9(1):58-59.

Jokanovic, M. 2008. Medical treatment of acute poisoning with organophosphorus and carbamate pesticides. Toxicology letters, 11(190):107-115.

Jokanovic, M. and Maksimovic, M. 2009. Abnormal Cholinesterase activity: understanding and interpretation. European Journal of Clinical and Chemical Biochemistry, 16(35):11-16

Malaria Consortium, 2016. Treatment and prevention of malaria, London, UK.

Murray, R. 2003. Metabolism of xenobiotics. Harper's illustrated Biochemistry (26th ed). New York. McGraw-Hills companies Pp 626-632.

Murray, F., Roth, R., Nicolich, M., Gray, T. and Simpson, B. 2013. The relationship between developmental toxicity and aromatic ring class profile of a high boiling substances. Regulatory Toxicology and Pharmacology, 5(20):123-136,

Piage, P., Morias, S., Correia, M., Alves, A. and Duleru, M. 2010. Screening of carbamate and ureas in fresh and processed tomato samples. International Journal of Environmental Chemistry. 8(9):199-210.

Pierre, G. and Guessain, R. 2004. Medical and veterinary entomology, International Journal of Medicine Research, 15(1):105 -112.

Reiner, E. 2001. Spontaneous reactivation of phosphorylated and carbamylated cholinesterases. The Pharmaceutical and Chemical Journal, 44:109-112.

Roll Back Malaria, 2005. Global strategy in plan, Roll back malaria 2005–2015, Geneva and Switzerland.

R .U. Ukpanukpong, R. N. Uyabeme, B. S. Akinfesola, F. Dadebiyi, P. O. Aigbadumah, and D. I. Basiru, 2019a. Hormonal assessment of Soda rhombifolia ethanolic leaf extract on micronor induced infertility in female rats. International Journal of Current Research in Biosciences and Plant Biology, 6(4):1-8.

Saheed, S., Jayashree, M. and Singhi, S. 2015. Effect of chronic exposure to Cadmium on serum lipoproteins and oxidative stress indices in male rats. Interdisciplinary Toxicololgy, 8(3):115-154.

Thompson, H. 2000. Esterases as markers of exposure to Organophosphates and Carbamates. Journal of Ecotoxicology, (8):368-384.

Uboh, F., Akpanabiatu, M., Atangwho, I., Ebong, P. and Umoh, I. 2007. Effect of gasoline vapours on serum lipid profile and oxidative stress in hepatocyte of male and female rats. Acta Toxicology, 15(1):13-18.

Odutuga, A. A., Ukpanukpong, R. U. and Uyabeme, R .N 2016. Biochemical and histological studies on the effects of Lonart Ds on Plasmodium berghei infected Mice. JABU Journal of Science Technology, (2)4: 69-78.

Ukpanukpong, R. U., Bassey, S. O., Akindahunsi, D. O., Omang, W. A. and Ugor, J. A 2018, Antidirrheal and Antihepatic Effect of Andrographis paniculata Leaf Extract on Castor Oil Induced Diarrhea in Wistar Rats. The Pharmaceutical and Chemical Journal, 5(1):62-76.

Ukpanukpong, R. U, Bassey, S. O., Edoamodu, C. E., Ugor, J. A., Omang, W. A. and Odutuga, A. A., 2018. Haematological Effect of Ethanolic Leaves Extract of Jatropha curcas on Plasmodium Berghei Infected Mice. The Pharmaceutical and Chemical Journal, 5(1):77-83.

Ukpanukpong, R .U., Eban, L .K., Adebiyi, F. D., Aigbadumah, P. O., Yusuff, A. A. and Akpan U. E. 2019b. Hormonal and electrolyte assessment of on the effect of Garlic Vitamin C And E in tramadol induced toxicity in female wistar rats. European Journal of Biotechnology and Bioscience. (7)5:38-42.

Ukpanukpong, R .U., Eteng, M .U., Dasofunjo, K. 2013. Antioxidant Interactions of Pefloxacin, Garlic, Vitamins, C and E on lipid profile level of Albino Wistar Rats., Journal of Applied Pharmaceutical Sciences, 3(03):167-170.

Ukpanukpong, R. U., Odutuga, A . A., Berena, G .A., Olarinde, E. O. and Aileme, R. 2016, Antidiabetic and antilipidemic effects of Andrographis paniculata ethanolic root extract in alloxan induced diabetic rats. Journal of Advances in Biological and Basic Research, 2(6):24-27.

Ukpanukpong, R .U., Uyabeme, R. N, Adekunle, I. A . and Adeleke, C. O. 2017. Biochemical Assessment of Parkia biglobosa Ethanolic Root Extract in Streptozocin Induced Diabetic rats. Journal of Advances in Medical and Pharmaceutical, 13(1):1-9.

World Health Organization, 2015, World malaria report, Geneva.

Zaim, M., Pritio, A. and Nakushima, N. 2000. Malaria study. Medical and Verterinary Entomology, 14(1):1-5.