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Evaluation of Lipid Profile in Male Albino rats exposed to petrol fumes

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Abstract

Aim: The effect of petrol fumes on lipid parameters were observed in albino rats exposed to petrol fumes.
Method: Thirty five (35) albino rats divided into seven (7) groups of five (5) rats each were exposed to petrol fumes at concentrations (Parts per Minute) of 0.00, 16,737 for 5 minutes daily, 20,240 for 10 minutes daily, 23,077 for 15 minutes daily, 27,344 for 20 minutes daily, 30,920 for 25 minutes daily and 34,458 for30 minutes daily for 21 days respectively using a modified nose-inhalation exposure method. The Cholesterol, HDL Cholesterol, LDL Cholesterol and Triglycerides estimations was done by enzymatic colorimetric methods. The data were subjected to statistical analysis using statistical package for social sciences (SPSS) version 21.
Result: The result showed that there was significant difference (P<0.05) in Total cholesterol (Mmol/L) concentrations of 1.58±0.06, 1.38±0.05, 1.38±0.09, 1.65±0.08, 1.52±0.07, 1.51±0.02 and 1.67±0.06 and Triglyceride (Mmol/L) concentrations of 0.99±0.08, 0.70±0.04, 0.52±0.07, 0.56±0.07, 0.74± 0.07, 0.45±0.05 and 0.040±0.05 at petrol concentrations (ppm) of 0.00, 16,737, 20,240, 23,077, 27,344, 30,920 and 34,458 respectively. Also there was significant difference(P<0.05) in HDL Cholesterol (Mmol/L) concentrations of 0.54±0.03, 0.69±0.01, 0.58±0.04, 0.44±0.04, 0.58±0.04, 0.65±0.07 and 0.58±0.42 and LDL Cholesterol (Mmol/L) was 0.58±0.08, 0.38±0.06, 0.56±0.06, 0.95±0.13, 0.61±0.05, 0.66±0.07 and 0.92±0.23 at petrol concentrations (ppm) of 0.00, 16,737, 20,240, 23,077, 27,344, 30,920 and 34,458 respectively. Overall there was significant difference (P<0.05) in Total cholesterol (Mmol/l) of 1.58±0.06 in control and 1.52±0.03 in the petrol fumes group while Triglycerides (Mmol/l) of 0.99±0.0806 in control was significantly different (P<0.05) from 0.56±0.03 in the petrol fumes group. HDL Cholesterol (Mmol/l) of 0.54±0.03 and 0.59±0.02 and LDL Cholesterol (Mmol/l) of 0.58±0.08 and 0.68±0.05 in the control groups and petrol fumes groups respectively did not show significant difference(P>0.05).
Conclusion: The study has shown that petrol fumes caused changes in lipid profile at different doses which could cause cardiovascular disease.
Keywords: Lipids. Petrol, Hydrocarbon, inhalation.

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1. Introduction

Petrol or Gasoline, being a volatile liquid is gotten from the fractionation of petroleum. Primarily, it is used as fuel in the internal combustion engines and some electricity generating machines. Gasoline consists mostly of aliphatic hydrocarbons, cyclic hydrocarbons and aromatic hydrocarbons. Additives such as tetraethyl lead, tetra methyl lead, methylcyclopentadienyl Manganese carbonyl (MMT), and ethanol are added to petrol to improve its quality (octane rating) or to prevent engine knocking [1]. Some constituents of gasoline are toluene, pentane, octane, xylene, hexane, heptane, 2, 2, 4–trimehtylbenzene and others. Benzene and toluene (which are non-aliphatic hydrocarbons) have been reported to be carcinogenic, which may be due to the free radicals they generate [2]. Studies with rats and mice with chronic inhalation exposure to gasoline vapours have found hepatocellular tumors in female mice, and nephropathy and related renal tumors in male rats [3]. Based on these report, the potential harmful effects associated with chronic or sub-chronic exposure to gasoline vapour should be the concern of the general public and scientific community. Petrol or gasoline vapour inhalation has been reported to change blood chemistry and induce anaemia by causing bone marrow hypoplasia in experimental animal [4]. This is due to the heavy metal contained in gasoline. It has become necessary to investigate the effects of acute gasoline exposures on lipid panels due to its apparent health implications

Lipids are organic compounds that contain hydrocarbons which are the foundation for the structure and function of living cells. They are insoluble in water, but are soluble in non-polar solvents. Lipids are also used as metabolic fuels. Lipoproteins are complex particles made up of multiple proteins that transport lipids around the body [5]. Lipoproteins consist of a mixture of protein, phospholipid, cholesterol, and triacylglycerol. The major classes m of phospholipids include: triglyceride, high density lipoprotein, low density lipoprotein, very low density lipoprotein, chylomicrons.

Triglyceride is an ester derived from glycerol and three fatty acids and is the main constituents of body fats and other animals [6]. In the blood, they enable the bidirectional transference of adipose fat and blood glucose from the liver; it plays an important role in metabolism as energy sources and transporters of dietary fat [7]. It could be saturated or unsaturated.

High density lipoproteins help in removing fat molecules from cells which need to export fat molecules such as cholesterol, phospholipids and triglycerides at variable amounts. They are referred to as "good cholesterol" since they help prevent the accumulation of fat within the arterial walls, increase in their concentration is associated with decreased risk of atherosclerosis [8]. Low density lipoproteins; are lipoproteins that transport fat molecules to the cells and can drive the progression of atherosclerosis if they become oxidized within the walls of arteries [9].

Chylomicrons; are lipoprotein particles that consist of triglycerides, phospholipids, cholesterol and proteins [10]. They transport dietary lipids from the intestine to other parts of the body such as adipose, cardiac and skeletal muscle tissue where their triglyceride components are hydrolyzed by the activity of the lipoprotein lipase, allowing the released free fatty acids to be absorbed by the tissues [11].

This study is looking forward to assessing the effect of petrol exposure on levels of triglyceride, high density lipoprotein, low density lipoprotein and total cholesterol in man using wistar albino rats as a model.

2. Materials and Method

2.1 Animals

Thirty-five (35) male albino wistar rats weighing about 177.20-198.80g were obtained from animal house of Pharmacology Department of University of Port Harcourt. The rats were housed in a wire meshed cage *adlibitum* (12 hours light and 12 hours darkness cycles) at standard temperature of 35-37^{0}C and were fed rat pellets and water.

2.2 Reagents

Commercially prepared reagents for Cholesterol and Triglycerides as well as HDL Cholesterol and LDL Cholesterol precipitants were purchased from Randox Diagnostics, London.

2.3 Petrol

The petrol sample was obtained from the Nigerian National Petroleum Corporation (N.N.P.C) zonal office at Moscow road, Port Harcourt.

2.4 Method for LC_{100}

Preliminary study was done to determine the LC_{100} and LC_{50} of petrol fumes on the rats. Twenty albino rats were divided into four groups of 5 rats each and exposed to 500ml of Petrol fumes for 0 hour, 4hours, 6hours and 8hours for 14 days. Physical changes (weakness, abnormal movement, and diarrhea), Behavioral changes (aggressiveness, rotational movement, biting and hyperactivity) and death were observed. The LC_{50} was calculated based on Arithmetic method of Karber [12].

2.5 Animal studies

The 35 albino rats used were divided into 7 groups of five rats each. The animals were acclimatized for six weeks and fed *ad libitum* with normal rat food and water at 12 hours daylight and 12 hours of darkness before commencement of studies.

2.6 Experimental design

An animal model consisting of 35 rats was used; they were divided into seven (7) groups containing five (5) rats each and were exposed to petrol fumes at petrol concentrations (Parts per Minute) 0.00, 16,737, 20,240, 23,077, 27,344, 30,920 and 34,458 ppm for groups 1,2, 3, 4, 5, 6 and 7 respectively. Signs of toxicity due to petrol fumes were observed in the rats such as grooming, sniffing around the cage, and standing on their hind legs also hyperactivity and weakness. The Group 1 served as the control and was not exposed to petrol fumes, Group 2 were exposed to16,737ppm of petrol fume for 5 minutes daily, Group 3 were exposed to 20,240ppm of petrol fume for 10 minutes daily, Group 4 were exposed to 23,077ppm of petrol fume for 15 minutes daily, Group 5 were exposed to 27,344ppm of petrol fume for 20 minutes daily, Group 6 were exposed to 32,047ppm of petrol fume for 25 minutes daily and Group 7 were exposed to 34,458ppm of petrol fume for30 minutes daily for 21 days. The Exposure to petrol fumes was carried out using a modified nose-inhalation exposure method [13]. According to this modification, the cages housing the animals in the test groups were placed in respective exposure chambers (1 cage per chamber), each with one open calibrated beaker of 1000mls containing 500mls of petrol. The petrol was allowed to evaporate freely within the respective exposure chambers at ambient humidity and temperature for an hour and test animals in cages were exposed to fumes generated from direct evaporation of petrol. The animals were exposed at 5 minutes time interval for 30 minutes/day after saturation of chamber with fumes, 7 day/week to fumes for 21 days. At the end of each exposure day, the animals were transferred to petrol fumes-free section of the experimental animal house. During the exposure period, the initial and final volumes of petrol were respectively recorded before and after daily exposure. The daily differences in volume were used to estimate relative concentrations of fumes used in this exposure method.

2.7 Sample collection

After three weeks of exposure, all the rats were sacrificed by chloroform sedation. Each of the animals was slaughtered and blood samples from each animal were collected into plain containers; Blood samples collected into plain containers were allowed to stand for about 15 minutes to clot and further spun in a centrifuge. Serum was separated from the clot with Pasteur pipette into sterile sample tubes for the measurement of selected hepatic enzyme activities.

2.8 Biochemical analysis

Cholesterol was determined by enzymatic hydrolysis and oxidation. The indicator quinoneimine is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxidase [14].

$$\text{Cholesterol ester} + H_20_2 \xrightarrow[\text{Oxidase}]{\text{Cholesterol}} \text{Cholesterol} + \text{fatty acids}$$

$$\text{Cholesterol ester} + 0_2 \xrightarrow[\text{Oxidase}]{\text{Cholesterol}} \text{Cholesterol } 3 + \text{one} + H_2O$$

$$2H_20 + \text{phenol} + \text{4-Aminoantipyrine} \xrightarrow{\text{Peroxidase}} \text{Quinoneimine} + 4H_2O$$

The test tubes were labeled blank, standard, sample and control. One milliliter (1ml) of Cholesterol reagent was pipetted into each tube while 0.01ml of distilled water, standard, sample and control was pipetted into their respective tube mixed, and incubated at 37^0C for 5minutes. The absorbances were measured at 520nm against the reagent blank. The concentration of Cholesterol (mmol/l) was determined by multiplying the absorbance of test with concentration of standard and dividing by absorbance of standard.

2.8.1 HDL

Cholesterol was measured according to the method previously reported by Friedewald *et al*., (15). The method was based on precipitation. Low density lipoproteins (LDL and VLDL) and chylomicron fractions were precipitated quantitatively by the addition of phosphotungstic acid in the presence of magnesium ions. After centrifugation, the cholesterol concentration in the HDL (high density lipoprotein) fraction, which remained in the supernatant, was determined at an absorbance of 520nm.

The HDL determination was divided into 2 stages namely the precipitation stage and the cholesterol determination.

Precipitation stage: The test tubes were labeled sample and control. One millilitre (1ml) of HDL cholesterol precipitant was pipetted into test tube containing 500µl of sample and 500µl of control. It was mixed and centrifuged at 4000rpm for 10minutes. The supernatant was separated and cholesterol content determined.

Cholesterol determination: The test tubes were labeled blank, standard, sample and control. One milliliter (1ml) of cholesterol reagent was pipetted into each tube, while 0.01ml of distilled water, standard, sample and control was pipetted into their respective tube mixed, and incubated at 37^0c for 5minutes. The absorbances were measured at 520nm against the reagent blank. The concentration of HDL Cholesterol (mmol/l) was determined by multiplying the absorbance of test with concentration of standard and dividing by absorbance of standard.

LDL Cholesterol Determination was done by enzymatic (colorimetric) method.

2.8.2 LDL

Cholesterol was measured according to the method previously reported by Friedewald *et al*., [15]. Low density lipoproteins are precipitated by the addition of heparin at their isoelectric point (pH 5.04). The HDL and VLDL remain in the supernatant and can be determined by enzymatic methods. The LDL determination was divided into 2 stages namely the precipitation stage and the cholesterol determination.

Precipitation stage- The test tubes were labeled sample and control. One millilitre (1ml) of LDL cholesterol precipitant was pipetted into test tube containing 100µl of sample. It was mixed and centrifuged at 4000rpm for 10minutes.The supernatant was separated and cholesterol content determined.

Cholesterol determination: The test tubes were labeled blank, standard, sample and control. One milliliter (1ml) of Cholesterol reagent was pipetted into each tube, while 0.01ml of distilled water, standard, sample and control was pipetted into their respective tube mixed, and incubated at 37^0C for 5minutes. The absorbances were measured at 520nm against the reagent blank. The concentration of LDL Cholesterol (mmol/l) was determined by multiplying the absorbance of test with concentration of standard and dividing by absorbance of standard.

Triglyceride determination was done by Colorimetric method was used. Triglycerides are determined after enzymatic hydrolysis with lipase. The indicator is a quinoneimines formed from hydrogen peroxide. 4 – aminophenazone and 4-chlorophenol under the catalytic influence of peroxidase [16].

$$\text{Triglyceride} + H_20 \xrightarrow{\text{Lipase}} \text{Glycerol} + \text{fatty acids}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{\text{GR}} \text{Glyceol} - 3\text{- Phosphate} + \text{ADP}$$

$$\text{Glycerol} - 3\text{- phosphate} + 0_2 \xrightarrow{\text{GPO}} \text{Dihydroxyacetone} + \text{phosphate} + H_2O$$

$$2H_20_2 + 4\text{- aminophenazone} + 4 \text{ chlorophenol} \xrightarrow{POD} \text{quinoneimine} + \text{HCL} + H_2O$$

The test tubes were labeled blank, standard, sample and control. One millilitre (1ml) of triglyceride reagent was pipetted into each tube, while 0.01ml of distilled water, standard, sample and control was pipetted into their respective tube mixed, and incubated at 37^0C for 5minutes. The absorbances were measured at 540nm against the reagent blank. The concentration of Triglycerides (mmol/l) was determined by multiplying the absorbance of test with concentration of standard and dividing by absorbance of standard.

2.9 Quality control

A randox normal quality control serum was assayed with all analyses to determine the precision.

2.10 Statistical Analysis

The biochemical data were subjected to statistical analysis using tools such as analysis of variance (ANOVA) and student's t-test using statistical package for social sciences (SPSS) version 21.0 for windows 8.1. Probability values less than 0.05 was taken to be significant.

3. Result

Table 1 below shows the effect of different concentrations of petrol fumes on lipid parameters. Total cholesterol (Mmol/L) was 1.58±0.06, 1.38±0.05, 1.38±0.09, 1.65±0.08, 1.52±0.07, 1.51±0.02 and 1.67±0.06 at petrol concentrations (ppm) of 0.00, 16,737, 20,240, 23,077, 27,344, 30,920 and 34,458 respectively while Triglyceride (Mmol/L) was 0.99±0.08, 0.70±0.04, 0.52±0.07, 0.56±0.07, 0.74± 0.07, 0.45±0.05 and 0.040±0.05 at petrol concentrations (ppm) of 0.00, 16,737, 20,240, 23,077, 27,344, 30,920 and 34,458 respectively.

HDL Cholesterol (Mmol/L) was 0.54±0.03, 0.69±0.01, 0.58±0.04, 0.44±0.04, 0.58±0.04, 0.65±0.07 and 0.58±0.42 at petrol concentrations (ppm) of 0.00, 16,737, 20,240, 23,077, 27,344, 30,920 and 34,458 respectively while LDL Cholesterol (Mmol/L) was 0.58±0.08, 0.38±0.06, 0.56±0.06, 0.95±0.13, 0.61±0.05, 0.66±0.07 and 0.92±0.23 at petrol concentrations (ppm) of 0.00, 16,737, 20,240, 23,077, 27,344, 30,920 and 34,458 respectively.

Table 1: Effect of different concentrations of petrol fumes on Lipid Parameters

	Concentrations (ppm)	Total Cholesterol (Mmol/l)	Triglycerides (Mmol/l)	HDL Cholesterol (Mmol/l)	LDL Cholesterol (Mmol/l)
	0.00	1.58±0.059	0.99±0.081	0.54±0.034	0.58±0.084
	16,737	1.39±0.054	0.70±0.044	0.69±0.006	0.38±0.057
	20,240	1.38±0.093	0.52±0.077	0.58±0.042	0.56±0.055
	23,077	1.65±0.083	0.56±0.072	0.44±0.042	0.95±0.131
	27,344	1.52±0.076	0.74±0.072	0.58±0.042	0.61±0.049
	30, 920	1.51±0.028	0.45±0.049	0.65±0.065	0.66±0.071
	34,458	1.68±0.064	0.40±0.046	0.58±0.042	0.92±0.233
	F	2.913	9.948	3.453	7.588
	P	0.025	0.000	0.011	0.000
Post Hoc					
0.00	16,737	0.866	1.000	0.352	0.275
	20,240	0.924	0.984	0.506	0.233
	23,077	0.890	0.316	0.750	0.326
	27,344	0.912	0.374	0.533	0.241
	30,920	0.880	1.000	0.996	0.310
	34,458	1.000	0.996	1.000	0.596
16,737	0.00	0.866	1.000	0.352	0.275
	20,240	1.000	0.832	1.000	1.000
	23,077	1.000	0.189	0.968	1.000
	27,344	1.000	0.234	1.000	1.000
	30,920	1.000	1.000	0.771	1.000

Table 1…… Continue

	34,458	0.990	0.969	0.029	0.262
20,240	0.00	0.924	0.984	0.506	0.233
	16,737	1.000	0.832	1.000	1.000
	23,077	1.000	0.051	1.000	1.000
	27,344	1.000	0.063	1.000	1.000
	30,920	0.999	0.867	0.943	0.997
	34,458	0.999	1.000	0.152	0.062
23,077	0.00	0.890	0.316	0.750	0.326
	16,737	1.000	0.189	0.968	1.000
	20,240	1.000	0.051	1.000	1.000
	27,344	1.000	1.000	1.000	1.000
	30,920	1.000	0.845	1.000	1.000
	34,458	0.995	0.110	0.234	0.784
27,344	0.00	0.912	0.374	0.533	0.241
	16,737	1.000	0.234	1.000	1.000
	20,240	1.000	0.063	1.000	1.000
	23,077	1.000	1.000	1.000	1.000
	30,920	1.000	0.891	0.961	1.000
	34,458	0.996	0.131	0.105	0.183
30,920	0.00	0.880	1.000	0.996	0.310
	16,737	1.000	1.000	0.771	1.000
	20,240	0.999	0.867	0.943	0.997
	23,077	1.000	0.845	1.000	1.000
	27,344	1.000	0.891	0.961	1.000
	34,458	0.983	0.930	0.959	0.510
34,458	0.00	1.000	0.996	1.000	0.596
	16,737	0.990	0.969	0.029	0.262
	20,240	0.999	1.000	0.152	0.062
	23,077	0.995	0.110	0.234	0.784
	27,344	0.996	0.131	0.105	0.183
	30,920	0.983	0.930	0.959	0.510

Table 2 below shows the overall effect of petrol fumes on lipid parameters. Total cholesterol (Mmol/l) was 1.58±0.06 and 1.52±0.03 in the control group and the groups exposed to petrol fumes respectively. Triglycerides (Mmol/l) was 0.99±0.08 and 0.56±0.03 in the control group and the groups exposed to petrol fumes respectively.HDL Cholesterol (Mmol/l) was 0.54±0.03 and 0.59±0.02 in the control groups and the groups exposed to petrol fumes respectively. LDL Cholesterol (Mmol/l) was 0.58±0.08 and 0.68±0.05 in the control groups and the groups exposed to petrol fumes respectively.

Table 2: The Effect of petrol fumes on Lipid Profile

Parameter	Control	Petrol fumes	t	P value
Total Cholesterol (Mmol/l)	1.58±0.06	1.52±0.03	0.444	0.510
Triglycerides (Mmol/l)	0.99±0.08	0.56±0.03	24.840	0.000
HDL Cholesterol (Mmol/l)	0.54±0.03	0.59±0.02	0.571	0.455
LDL Cholesterol (Mmol/l)	0.58±0.08	0.68±0.05	0.686	0.413

4. Discussion

The result of the study showed dose dependent increase in total cholesterol and low density lipoprotein cholesterol (LDL-C) with dose dependent decrease in triglyceride and high density lipoprotein cholesterol (HDL-C). This is similar to work by Uboh *et al* [17]. Uboh *et al* [17] reported an increase in triglyceride & total cholesterol in rats exposed to petrol & kerosene fumes, but the decrease in triglyceride in this study is in contrast to the work by Uboh *et al*[17]. This could be as a result of a number of factors such as prolonged fasting prior to blood collection. The result is also similar to reports of previous studies by Ogbevire *et al* [18] and Egbuonu *et al* [19]. Serum cholesterol is a term that includes the total level of cholesterol that is found in the bloodstream, it includes identifying all types or classes of cholesterol that are found in the system [20]. This helpful measurement makes it possible to determine if the balance between the high density lipoprotein cholesterol (good cholesterol) and low density lipoprotein cholesterol (bad cholesterol) is within acceptable limits. While the presence of high density lipoprotein cholesterol (HDL-C) is beneficial to maintaining organ health and providing the body with necessary energy, the presence of low density lipoprotein cholesterol (LDL-C) can lead to blockages that may lead to problems with the heart and lungs. The result in this study is an indication that inhalation exposure to kerosene and petrol fumes also affect lipid metabolism.

On one hand, lipid metabolism is affected once there is liver damage since the disturbance of cell membrane integrity is likely to cause some membrane lipids to be released into circulation; while on the other hand, it causes the tissue to compromise its effectiveness in regulating lipid metabolism. There is likelihood that exposure to kerosene and petrol fumes predispose the subject to atherosclerotic condition [17]. Study by Ubani *et al* [21] established that petrol interfered with lipid metabolism resulting in high TC, LDL-C and decreased high density lipoprotein cholesterol (HDL-C). This is suggestive of the risk of developing cardiovascular disease as postulated by Mckee and Mckee [22] when crude oil or crude oil contaminated foods are ingested.

There was significant difference ($p<0.05$) in total cholesterol, low density lipoprotein cholesterol (LDL-C), triglyceride and high density lipoprotein cholesterol (HDL-C) in this study. This is similar to the study by Kapil *et al* [23]. According to the study, the significant abnormal rise in serum cholesterol in all test groups indicates the negative influence of petrol fumes on lipid metabolism. The lipid metabolism is affected as a result of liver damage and disturbance of the cell membrane integrity which releases some membrane lipids into circulation. On the other hand, petrol fumes reduce the capacity of tissues to regulate lipid metabolism. So, there is likelihood that inhalation of petrol fumes leads to atherosclerosis as observed in earlier animal studies also [13,24]. Cholesterol is an essential structural component of mammalian cell membranes and is required to establish proper membrane permeability and fluidity. Within the cell membrane, cholesterol also functions in intracellular transport, cell signaling and nerve conduction. It is synthesized in many types of tissues, but principally in the liver and intestinal wall of vertebrates. Cholesterol assays are used to screen for atherosclerotic risk and in the diagnosis and treatment of disorders involving elevated cholesterol levels as well as lipid and lipoprotein disorders [25].

The result further showed that petrol fumes caused significant decrease in triglycerides concentration but no significant changes in total cholesterol, low density lipoprotein cholesterol (LDL-C) and high density lipoprotein cholesterol (HDL-C). This decrease in TG levels is in contrast to the work by Uboh *et al* [17]. This could be due loss of weight, poor diet, exercise [26]. Exercise causes a periodic decrease in free fatty acid level [27]. Triglycerides are fatty acid esters of glycerol and represent the main lipid component of dietary fat and dietary depot of animals. During the exposure of the rats to petrol fumes, restlessness, hyperactivity and loss of appetite was observed among the animals. This could be the reason for their weight loss and decreased triglyceride level.

5. Conclusion

The results have shown that petrol fumes caused changes in total cholesterol (TC), triglyceride (TG), high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) concentrations at different doses. The study has shown that petrol fumes caused changes in lipids which could cause cardiovascular disease.

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