

Toxicological Effects Of Some Mosquito Coils Brands In Experimental Rats

M Abubakar, L Hassan

Citation

M Abubakar, L Hassan. *Toxicological Effects Of Some Mosquito Coils Brands In Experimental Rats*. The Internet Journal of Toxicology. 2006 Volume 4 Number 1.

Abstract

Biochemical parameters were used to evaluate the toxic effects of different brands of mosquito coil smoke in experimental rats. The smoke from the coils produced significant increase ($P < 0.05$) in the levels of total protein, total albumin, bilirubin and blood urea nitrogen when animals were exposed to smoke for 14 days. Similarly, the smoke from the coil also caused an elevation in the activities of aspartate aminotransferase and alanine aminotransferase. Although the smoke from the coils did not produce lesions in the hearts, lungs and liver examined, the increase in liver enzyme activities could be due to early liver damage.

INTRODUCTION

Mosquito coils are widely used as mosquito repellent. The major active ingredients of most mosquito coils are pyrethrins, accounting for about 0.3 – 0.4% of the coil mass [1]. When a Mosquito coil is burnt, the insecticides evaporate (pyrethrin, PAH, aldehyde etc.) with smoke, which prevent the mosquito from entering the room and harm those already in the room. The remaining components of mosquito coils include organic fillers, binders, dyes and other additives capable of burning well without flame. The combustion of these remaining components generates large amounts of submicrometer particles and gaseous pollutants such as acenaphthene, penanthrene, benzo(a)pyrene, etc.. [2]. These particles can reach the lower respiratory tract and may be coated with a wide range of organic compound generated through incomplete combustion of mosquito coil base materials. Mosquito coils are often used overnight in sleeping quarters; where continuous exposures may occur. Chronic exposure to coil smokes occurs during rainy periods because mosquitoes are found to be more active in the environment due to collection of water and increase in green plants [3]. This long-term exposure calls for concerns on the potential toxicological effects of the smoke on humans.

Epidemiological studies have shown that long-term exposure to mosquito coils smoke can induce asthma and persistent wheeze in children [4,5]. This study therefore aims to investigate the toxicological effects of mosquito coil smoke in rats with hope that the results would provide a guideline for proper use of these coils.

MATERIALS AND METHODS

SAMPLE TREATMENTS

Three commonly used brands of mosquito coils were purchased from sample dealers in Sokoto Central Market (Sokoto, Nigeria). These brands included Cock Mosquito coil (NNPA, China), Rambo Mosquito coil (MI, Nigeria) and Swam Mosquito coil (Malaysia).

EXPERIMENTAL ANIMALS AND DESIGN

The experiment involved four groups of Wistar albino rats, weight between 150-200g and the animals were obtained from the animal house of Biological Sciences Department, Usmanu Danfodiyo University Sokoto-Nigeria with three rats in each group. Group A served as the control group whereas groups C, R and SA were exposed to the smoke of Cock mosquito coil, Rambo mosquito coil and Swam mosquito coil, respectively. These rats were exposed to the smoke for 8 hours each day, for 14 days in a 5 m³ container. Thereafter, blood samples were taken from rats for analysis and comparison was made with those in the control group. The control group was exposed to normal room (125 m³) air for the same exposure duration.

Specimen collection: Blood samples were collected in centrifuge tubes, and were spun at 3000 rpm for 5 minutes to obtain the serum sample from each individual animal. The serum was obtained using a Pasteur pipette. The rats were exposed to chloroform for about 30 seconds, which killed them, and the tissue samples liver, kidney, lungs and the heart were immediately obtained after dissecting the rats.

Sample collected from each rat was used for quantitative determination of total protein, albumin, alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP). These were described spectrophotometrically according to the methods determined by Paul [6]. Serum Bilirubin and blood urea nitrogen were also determined spectrophotometrically according to the methods described by Baron et al. [7]. An examination of the tissues was carried out following the Teased preparation method [7]. Data were expressed as mean \pm standard error of the mean. Statistical significant was performed using the analysis of variance.

RESULTS AND DISCUSSION

During the initial period of the experiment, the rats were observed to be very active, feeding normally on the first day. However, changes were noticed on the second day. The rats in the test groups started to pass watery stool, which continued during the period of exposure to the coil smoke. The feeding habit was also observed to decrease. Sneezing was also noticed on day six which continued for the remaining period of exposure. None of these signs was observed in the control group.

As shown in table 1, the concentration of the blood urea nitrogen in the test groups was significantly higher ($p < 0.05$) than the control groups. The concentration changes observed in the Swam study group was higher than that of Rambo and Cock brands of mosquito coil.

Figure 1

Table 1: Effects of mosquito coil smoke on plasma proteins, albumin and blood urea nitrogen in rats

Group	Total protein (g/dl)	Total albumin (g/dl)	Blood urea nitrogen (mg/dl)
Control	7.50 \pm 0.30	3.00 \pm 0.45	19.77 \pm 0.23
Cock	10.57 \pm 0.37*	4.80 \pm 0.81*	24.63 \pm 0.58*
Swam	9.60 \pm 0.82*	5.20 \pm 0.57*	29.00 \pm 1.59*
Rambo	10.73 \pm 0.60*	5.93 \pm 0.33*	28.23 \pm 0.41*

* $P < 0.05$.

The results in table 2 show that the total bilirubin in the test groups were higher than that of the control group but the difference was not significant ($P > 0.05$) in all the groups.

Figure 2

Table 2: Effects of coil smoke on plasma bilirubin levels in rats

Sample	Total Bilirubin (g/dl)	Direct Bilirubin (mg/dl)
Control	0.43 \pm 0.18	0.05 \pm 0.006
Cock	1.47 \pm 0.31	0.21 \pm 0.014
Swan	0.63 \pm 0.09	0.08 \pm 0.009
Rambo	1.43 \pm 0.24	0.23 \pm 0.027

As shown in table 3, the activities of ALP and ALT were a significantly increased.

Figure 3

Table 3: Effects of coil smoke on liver enzyme activities of rats

Sample	ALP (u/L)	AST (u/L)	ALT (u/L)
Control	37.33 \pm 3.49	45.70 \pm 14.74	17.00 \pm 3.81
Cock	70.67 \pm 8.88*	48.00 \pm 2.45	29.33 \pm 0.67*
Swan	70.33 \pm 5.79*	48.00 \pm 1.73	29.67 \pm 4.34*
Rambo	78.33 \pm 15.23*	51.67 \pm 0.88	38.00 \pm 5.30*

The results in Table 1 show that the concentration of total protein, total albumin and blood urea nitrogen in the test groups are significantly higher compared to the control groups. Many factors may be responsible for the increment among which are excess breakdown of blood protein and increase in tissue protein catabolism. Similarly, high urea may be associated with low blood volume. Table 2 shows the total bilirubin in the control group, but the difference was not significant in all the groups. Here the Cock mosquito coil had higher total bilirubin than the Rambo and Swan coils though the difference was not significant. In the direct bilirubin determination assays, the results showed no significant difference between the test groups and the control group. Even within the test groups, there was no significant difference.

Oedema was noticed in various organs of the smoke-exposed rats (data not presented). Oedema could have resulted from the inflammatory processes taking place as a result of irritation of various organs by toxic chemicals from coil smoke. The reduced activity in the exposed rats could as well be due to toxic chemicals in coil smoke. Cyanide, which is released in mosquito coil smoke, is known to cause reduced oxygen carrying capacity of erythrocytes, leading to reduced metabolism and consequently result in reduced

energy output which may explain the body weakness [7]. The sneezing that resulted after 6 days exposure could be the result of irritants released in the coils smoke such as aldehydes, sulphates [8] and polycyclic aromatic hydrocarbons such as acenaphthene, penanthrene, benzo(a)pyrene, etc.[2].

An increase in total protein and total albumin could be the result of the loss of plasma fluid into tissue due to inflammation, which may be the result of exposure to irritants released from coil smoke. Aldehydes, pyrethrins and sulphates, which are present in the coil smoke, are known irritants, and they can induce inflammatory response [9]. The inflammation may cause damage to the liver cells, which are sites of the protein synthesis leading to the release of plasma protein thereby causing an increase in the amount of protein. The increase in protein levels conforms to the previous experiment, in which levels of total protein and total albumin were found to increase as a result of exposing rats to coil smoke [10]. The coil smoke elevated plasma urea levels; the elevation could probably be due to the increase in activities of urea enzymes, ornithine carbomoyl transferase and arginase can provide evidence of liver damage in many animal species, since the urea cycle is confined to the liver [11]. Elevated blood urea in this study may also indicate kidney damage however; no gross lesions were seen on the kidneys examined from the tested animals. However, chlorine is known to be hepatotoxic when inhaled in high concentration [12]. This could most likely be responsible for the defective urea cycle and the inability of the liver to transform ammonia to urea.

The smoke also elevated the liver enzymes activities. The elevation in the activities of these enzymes suggested liver tissue damage. Previous studies indicated that ALT is found in high concentrations in the hepatic tissues of dogs, cats and primates and elevation of its activity in plasma indicates hepatocellular damage [13,14]. Similarly another study by Woodman [11] indicated that the increase in plasma enzyme activities often seen following liver damage does not indicate an increase in the liver ability to synthesis that enzyme, but rather a loss of material from damaged hepatocytes. Blood urea nitrogen in the test groups were significantly higher compared to the control groups. Many factors may be responsible for the increment among which are excess breakdown of blood protein and increase in tissue protein catabolism. Similarly high urea may be associated with low blood volume. Damage to the kidney may result in reduced erythropoietin production, resulting in high urea

which may in turn be associated with low blood volume [15]. Thereby leading to an elevation in inflammatory cells types, which usually occurs during the inflammatory process [9]. Inflammation exposes the body organs to infections, leading to the release of high amount of white blood cells [9].

In conclusion, results of this study show that short term exposure of rats to mosquito coil smoke possess toxic properties. Studies aimed at producing alternative mosquito repellents with minimal toxicity affects should be an area of practical interest.

References

1. Lukwa N, Chandiwana SK. Efficacy of Mosquito coils containing 0.3% and 0.4% pyrethrins Against an *Gambiae sensu* Lola Mosquitoes center. *Afr. J. Med.* 1998; 44 (4): 1041.
2. Liu WK, Zhang J, Hashim JH, Jalaludin J, Hashim Z, Goldstein BD. Mosquito coil emissions and health implications. *Environ Health Perspect.* 2003; 111(12) : 1454 - 60.
3. Chang J-Y, Lin J-M. Aliphatic aldehydes and allethrin in mosquito-coil smoke. *Chemosphere* 1989 ; 36(3): 617 - 24.
4. Azizi BO, Henry RL. The effect of indoor environmental factor on respiratory illness. Primary school children Kaula Lumpur. *Int. J. Epidemiol.* 1991 ; 20 (1): 144 - 9.
5. Koo LC, Ho, JH-C. Mosquito coil smoke and respiratory health among Hong Kong Chinese: Results of three epidemiological studies. *Indoor and Built Environment* 1994 Sep; 3 (5): 304-10.
6. Paul R. Clinical chemistry in diagnosis and treatment. London Loyd Luke, 4th Ed. ,1988.
7. Baron DN, Wicher JT, Lee K.E. A New short textbook of chemical pathology. 5th Ed. 1992: 100 - 210
8. EPA. Pesticides evaluation scheme, Division of Control of Tropical Diseases, guideline specifications for household insecticide products. Environmental Protection Agency, USA, 1998.
9. Robb RM, Marchevsky A. A pathology of the lens in Down syndrome. *Arch Ophthalmol* 1978; 96: 1039.
10. Liu WK, Ng TB, Wong CC. Biochemical and cellular changes in bronchoalveolar lavaged samples from rats after inhalation of mosquito-coil smoke *Toxicol Lett.* 1989; 45: 121 - 32.
11. Woodman DD. Assessment of hepatic function and damage in animal species. A review of the current approach of the academic, governmental and industrial institutions represented by the Animal Clinical Chemistry Association. *J Appl Toxicol*, 1980 ;8 (4): 249 - 254.
12. Halliwell B, Gutteridge JMC. Free Radical in Biology and Medicine: Oxidative Stress. 3rd (ed.) Oxford Science Pub., 1999: 105 - 245.
13. Kaneko JK, Cornelius LE. Clinical Biochemistry of Domestic Animals New York Academic Press, 3rd Ed. Publications 1980: 5 - 6.
14. Sigma Diagnostic. Transaminase (ALT/GPT) and (AST/GOT). Quantitative colorimetric determination in serum, plasma or cerebrospinal fluid procedure 1985; No. 505.
15. Ezzati M, Kammen DM. Quantifying the Effects of Exposure to indoor air pollution from biomass combustion on acute respiratory infections in developing countries. *Environ Health Perspect.* 2001; 109: 481 - 8.

Author Information

M. G. Abubakar, Ph.D.

Department of Biochemistry, Faculty of Science, Usmanu Danfodiyo University

L. G. Hassan, Ph.D.

Reader, Department Pure and Applied Chemistry, Faculty of Science, Usmanu Danfodiyo University