Table 1: **Descriptive Statistics Protein and Some Liver Enzyme Assay of Groups**

	Group	N	Mean	Std. Deviation	Std. Error
Protein (g/dl)	Control	6	4.37	0.48	0.19
	ExpA	8	5.64	0.12	0.04
	ExpB	8	6.60	0.28	0.10
	Total	22	5.64	0.95	0.20
Albumin (g/dl)	Control	6	2.74	0.34	0.14
	ExpA	8	3.39	0.17	0.06
	ExpB	8	4.19	0.26	0.09
	Total	22	3.51	0.64	0.14
ALT (µmol/l)	Control	6	32.22	0.48	0.20
	ExpA	8	71.78	11.13	3.93
	ExpB	8	108.15	26.35	9.31
	Total	22	74.21	34.89	7.44
AST (µmol/l)	Control	6	12.13	0.59	0.24
	ExpA	8	53.03	8.66	3.06
	ExpB	8	75.49	27.25	9.63
	Total	22	50.04	30.55	6.51

Table 1 above shows the mean and standard deviation of each group for the different components. The table revealed that experimental group B (ExpB) has the highest mean for components. Total Protein, Albumin, ALT and AST. The Control group had the least mean for all the components.

Table 2: **Analysis of Variance (ANOVA) of the Three Groups**

	Source of Variation	Sum of Squares	df	Mean Square	F	Sig.
Protein	Between Groups	17.121	2	8.560	91.528	.000
	Within Groups	1.777	19	0.094		
	Total	18.898	21			
Albumin	Between Groups	7.419	2	3.710	56.341	.000
	Within Groups	1.251	19	0.066		
	Total	8.670	21			
ALT	Between Groups	19843.463	2	9921.731	32.915	.000
	Within Groups	5727.303	19	301.437		
	Total	25570.766	21			
AST	Between Groups	13873.376	2	6936.688	23.022	.000
	Within Groups	5724.897	19	301.310		
	Total	19598.273	21			

P <0.0001

Table 2 shows the analysis of variance for the three groups for the five components. The results revealed that the difference between the means of the three groups for each component is significant (*P* <0.0001).

Table 3: T-Test of the Difference Between the Means of High and Low Dosage

	Grp	N	Mean	Std. Deviation	Df	t	Sig.
Protein (g/dl)	ExpA	8	5.634	0.124	14	-9.024	0 .000
	ExpB	8	6.601	0.276			
Albumin (g/dl)	ExpA	8	3.394	0.167	14	-7.374	0 .000
	ExpB	8	4.195	0.258			
ALT (µmol/l)	ExpA	8	71.775	11.130	14	-3.597	0 .003
	ExpB	8	108.150	26.347			
AST (µmol/l)	ExpA	8	53.025	8.657	14	-2.222	0.043
	ExpB	8	75.487	27.251			

Table 3 shows the t-test of the difference between the means of the high and low dose of each component. The result revealed that there is a significant difference in the means of high dose and that of low dose for components protein and albumin ($P < 0.0001$), ALT ($P = 0.003$) and AST ($P = 0.043$).

Discussion

The results of the histochemical studies revealed that with increasing dose of monosodium glutamate consumption, there were varying degrees of dilatations of the central vein of the liver which contained lysed red blood cells in the treatment group compared to the control sections of the liver. The necrosis observed is in consonance with the findings recorded in previous work on MSG. [14, 15, 16] This suggests that the distortion of the cyto-architecture of the liver could be associated with functional changes that may be detrimental to the health of the rats. The proliferating cells of the liver, which produce red and white blood cells, are normally found between the hepatic cells and the walls of the vessels.[17] As a result of the distortion and dilatation of the hepatocytes and their central vein, the haematopoietic function of the liver may have been highly affected as a result of probable toxic effect of MSG. This was further buttressed by the increase in the liver enzymes obtained in the test group. In addition, total protein and albumin increased in this study The increase in total protein may be due to the fact that MSG was given for a short period of time. The resultant effect is acute toxicity leading to enhanced hepato-cellullar activity and increase in globulin and albumin components of the protein .However, with prolonged usage, hepatic necrosis is likely to occur with a resultant low albumin levels.

Cellular degeneration has been reported to result in cell death, which is of two types, namely apoptotic and necrotic cell death. These two types differ morphologically and biochemically.[18] Pathological or accidental cell death is regarded as necrotic and could result from extrinsic insults to the cell such as osmotic, thermal, toxic and traumatic effects.[19] Cell death in response to toxins occurs as a controlled event involving a genetic programme in which caspase enzymes are activated.[20]

As the hepatocytes swell as seen in this study the activities of cellular transporters are is approximately modified by up or down regulations as earlier reported in the case of hyponatraemia or hypernatraemia. [21] Ischaemic or pharmacologic disruption of cellular transporters can cause swelling of parenchyma of the liver cells.

MSG may have acted as toxins to the hepatocytes, thereby affecting their cellular integrity and causing defect in membrane permeability and cell volume homeostasis.

The atrophic and degenerative changes observed in this experiment may have been caused by the cytotoxic effect of MSG on the liver. This obviously will affect the normal detoxification processes and other functions of the liver.

This study had some limitations. The actual quantity of MSG consumed per day by each rat in the various group could not be actually ascertained

since the substance was mixed with their feeds. Some rats could have consumed more of the MSG than others and this could vary the pathology seen. Another factor was the duration of study (acute) as opposed to chronic which could have yielded more light on the pathology.

The results obtained in this study following the administration of 0.04g/kg and 0.08g/kg per day of MSG to adult Wistar rats affected the histology of the liver and affected liver function. Thus the ingestion of this substance by humans should be reviewed. It is recommended that further studies be carried out to corroborate these findings.

References

1 Moore KL. Congenital malformations due to environmental factors. In: Developing Humans 2nd ed. Philadelphia: W.B. Saunders Co. Ltd; 2003; 173-183.

2 Allen GH. The genetic basis of diseases In: General Pathology. New York: Churchill Livingstone Medical Division Longman; 1987; p 35056.

3 Zerasky K. Nutrition and healthy eating; monosodium glutamate: Is it harmful? [Assessed on 23/12/2010]. Available @ mayo clinic.com

4 Okwuraiwe PE. The role of food and Drug Administration and control (FDA&C) in ensuring the safety of food and food ingredients: A symposium held at Sheraton Hotel, Lagos. 1992, 1st Sept.: 6-15.

5. Rogers PP and Blundell JE. Umani and appetite: Effects of Monosodium glutamate on hunger and food intake in human subjects. Physiol Behav 1990; 486:801-804.

6. Iwase M, Yamamoto M, Iino K, Ichikawa K, Shinohara N and Yoshinari F. Obesity induced by neonatal monosodium glutamate treatment in spontaneously hypertensive rats: an animal model of multiple risk factors. Hypertens Res 1998; 43: 62-68.

7. Gobatto CA, Mello MA, Souza CT and Ribeiro IA. The monosodium glutamate (MSG); obese rat as a model for the study of exercise in obesity, Res Commun Mol Pathol Pharmacol 2002; 2: 116-128.

8. Belluardo M, Mudo G and Bindoni M. Effect of early destruction of the mouse arcuate nucleus by MSG on age dependent natural killer activity. Brain Res 1990; 534:225-333.

9. Onakewhor JUE, Oforofuo IAO and Singh SP. Chronic administration of Monosodium glutamate Induces Oligozoospermia and glycogen accumulation in Wister rat testes. Afr J Reprod Health 1998; 2(2): 190- 197.

10. Oforofuo IAO, Onakewhor JUE and Idaewor PE. The effect of chronic administration of MSG on the histology of the Adult Wister rat testes: Bios Resch Comms 1997; 9 (2):12-14.

11. Eweka AO and Adjene JO. Histological studies of the effects of monosodium Glutamate on the medial geniculate body of adult Wistar rat. Electron J Biomed 2007; 2: 9-13.

12. Samuels A. The Toxicity/Safety of MSG: A study in suppression of information. Account in Resech 1999; 6(4): 259-310.

13 Gartner LP and Hiatt JL. Color Atlas of Histology 3rd ed. New York: Lippincott Williams & Wilkins Publishers; 2000; 294-301.

14 Eweka AO and Om'Iniabohs FAE. Histological studies of the effects of monosodium glutamate on the small intestine of adult Wistar rat. Electron J Biomed 2007; 2: 14-18.

15 Eweka AO, Om'Iniabohs FAE and Adjene JO. Histological studies of the effects of monosodium glutamate on the stomach of adult Wistar rats. Ann Biomed Sci 2007; 6:45-52.

16 Eweka AO and Om'Iniabohs FAE. Histological studies of the effects of monosodium glutamate on the Liver of adult Wistar rats. The Internet Journal of Gastroenterology 2008; 6; available @ http://www.ispub.com/journal/the_internet_journal_of_gastroenterology/.

17 Singh I. Textbook of Human Histology with color atlas 3rd ed. New Delhi: Jaypee Brothers Medical Publishers Ltd; 1997; 238-244.

18 Wyllie AH. Glucocorticoid-induced thymocyte apoptosis is associated and endogenous endonuclease activation. Nature 1980; 284:555- 556.

19. Farber JL, Chein KR and Mittnacht S. The pathogenesis of irreversible cell injury in ischemia. Am J Path 1981; 102: 271-281.

20. Waters CM, Wakinshaw G, Moser B and Mitchell IJ. Death of neurons in the neonatal rodent globus pallidus occurs as a mechanism of apoptosis. Neuroscience: 1994; 63: 881-894.

21. Johnson CE. Effects of fluid imbalances In: Neurosciences in Medicine. Conn MP (Ed).New York: JB Lippincott Company; 1995; 187-189.