Biochemical Evaluations of Male Wistar Rats Exposed to Pesticides

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ABSTRACT

The aim of the research work was based on the finding of the implications and possible toxicity of preservation of food crops in different pesticides. A total of 120 male wistar rats were purchased from Chris Animal house situated at Awka. The breakdown was as follows; 30 male wistar rats were for the toxicity study, the remaining ones were grouped into A, B, C, and D groups. Group A had 15 male wistar rats and served as control while B, C and D groups were made of 25 male wistar rats each fed with different growers rat feeds treated with pesticides (Aluminium phosphide, chloropyrifos and Dichlorvos). The animals were sacrificed after a period of 60 days, blood samples were collected for the evaluations of liver function test, kidney function test and lipid profile, using a standard biochemistry techniques. The pesticides were used to make different dilutions of 1/10, 1/50 and 1/100. The result obtained in the three pesticides treated growers feeds followed the same pattern but in a few cases of dilution with a statistical significance, it was apparent that the pesticides could not elicit any deleterious effect to the male wistar rats. From the result of the research work, farmers could be encouraged to use these pesticides for the preservation of their food crops since food preservation and curing are important tools in long term food storage, and reducing spoilage rates and thus increased productivity for the nation. Conclusively, Scientists in Nigeria can explore further research works on other pesticides since many advanced countries have long banned the use of pesticides based on deleterious effects reported in the use of pesticides in their various countries.

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1. INTRODUCTION

The environmental pollution is one of the most serious problems that face mankind in this century. There are many types of pollutants that interfere with our life both directly and indirectly (Zaahkoku *et al.*, 2000). The authors also reported that potential hazards to human health and wildlife can be created by residues from some long-lived pesticides that may build up in food chain and cause widespread contamination of the food crops. When a crop is treated with a pesticide, a very small amount of the pesticide or its metabolites or degradation products, can remain in the crop after it has been harvested. This is known as residual pesticides (Michael *et al.*, 2005). Pesticides are substances or mixture of substances intended for preventing, destroying,

repelling or mitigating any pest. A pesticide may be a chemical substance, biological agent, antimicrobial, disinfectant or device used against any pest. Pests include insects, plant pathogens, weeds, molluscs, birds, mammals, fish, nematodes (roundworms), and microbes that destroy property, spread disease or are vectors for diseases.

Pesticide residues may be present in fresh fruits and vegetables or processed food and drinks made from the crops or freshly processed animal products (Kasozi *et al.*, 2006). Over 98% of sprayed insecticides and 95% herbicides reach a destination other than their target species, because they are sprayed across entire agricultural fields (George, 2004). Runoff can carry pesticides into aquatic environments while wind can carry them to other fields, grazing areas, human settlements and undeveloped areas, potentially affecting other species. Over

time, repeated application increases pest resistance, while its effects on other species can facilitate the pest's resurgence (Damalas and Eleftherohorinos, 2011). Large worker populations in the third world are exposed to increasing amount of pesticides, including pesticides severely restricted and banned in industrial countries. Studies on knowledge, attitudes, and practises indicate that unsafe use of pesticides is the rule in the third world countries (Cartharina *et al.*, 1997). Organophosphate pesticides are esters derived from phosphoric acid. In man it acts on the central nervous system by inhibiting acetyl cholinesterase, an enzyme that modulates the amounts and levels of neurotransmitter acetylcholine, disrupting the nerve impulse by serine phosphorylation of hydroxyl group in the active site of the enzyme, (Francisco *et al.*, 2012). Osibanjo and Adeyeye, (1995) reported that high efficacy and low cost of organochlorine pesticide compared with alternative pesticide (such as chlorpyrifos and dichlorvos) is the reason for their continued use in developing countries. The US *Environmental Protection Agency* (EPA) classifies aldrin, lindane and dieldrin as probable human carcinogens, where increased rates of liver tumors were found in both rats and mice that ingested low levels of them in the diet (EPA, 2005). In addition, exposure to large amount of aldrin, lindane and dieldrin, can harm the nervous system, producing a range of symptoms from headache and dizziness to seizures, convulsions and more rarely death (EPA, 2005). The vital importance is well recognized, as is the usefulness of pesticides in increasing agricultural yields and reducing spoilage rates. The usefulness of pesticides in mitigating disease carrying pest (e.g. mosquitoes) is also known. However, there are also risks associated with pesticide use. In addition to causing acute poisoning, (Calvert *et al.*, 2008), and they are also associated with increased cancer risk, (Alanvaja *et al.*, 2013). Endocrine disrupting chemical (EDCs) are common place in the manufacturing of consumer products especially plastics, some pesticides as well act as EDCs. The polychlorinated pesticides (DDT) is one of the first recognized EDCs; although DDT was banned in many countries in the 1970s, it remains widespread in the environment and food supply and exposure to the pesticide DDT can raise cardiovascular risks among premenopausal women, (Teixeira *et al.*, 2015). They primarily affect the nervous system of the exposed organisms by inhibiting acetylcholinesterase (AChE) and raising acetylcholine levels in the cholinergic synapse. Beside cholinergic effects, organophosphates (Ops) induce oxidative stress (Soltaninejad and Abdollahi, 2009 and Dettbern *et al.*, 2006), affect metabolic pathways (Karami-Mohajeri and Abdollahi, 2011), and cause multiple organ dysfunctions such as hypoxia and inadequate tissue perfusion of the liver and heart (Kumar *et al.*, 2010). In the liver, they cause ultrastructural, biochemical, metabolic, and mitochondrial damage, evidenced by changes in hepatic biomarkers such as serum aminotransferase and direct and indirect bilirubin (Hoekstra *et al.*, 2013). Impaired bile flow and biliary excretion could also serve as indirect indicators of liver damage. Goel and Dhawan (2001), for example, suggested that poor biliary excretion and longer half-life of ^{99m}Tc -mebrofenin in chlorpyrifos-treated rats reflected impaired hepatobiliary function. As impaired liver function affects the metabolism, consequently it also affects the concentrations of waste products. Several animal studies have pointed to the indirect effects of sub-lethal doses of phorate,

fenitrothion, and dimethoate on urea and bilirubin (Farghaly *et al.*, 2007). In humans occupationally exposed to a wide range of OPs, blood urea nitrogen and albumin along with serum are frequently elevated (Malekirad *et al.*, 2013). Since, there are limited data about the hepatotoxicity of OPs in humans, it is not certain whether changes in biochemical parameters indicate actual liver damage.

2. MATERIALS AND METHODS

2.1 MATERIALS

Commercially available standard poultry growers mash containing 21% protein, 4.4% fibre, 7.2% fat and 5.8% ash was procured from Chris Animal farms, Awka.

Pesticides used for the research were:

Aluminium phosphide (trade name: fumigation tablet)

Chlorpyrifos (trade name: Termox)

Dichlorvos (trade name: DAKSH)

Chemicals and kits used in biochemical analysis were of analytical grade purchased from reputable companies.

Sample (Animal model)

120 apparently healthy adult male wistar rats weighing $200 \pm 20\text{g}$ were obtained from Chris Animal farms, Awka.

2.1.1 Research Design

Dose Administration for acute toxicity study

Male wistar rats were fasted for 18 hours prior to dosing. The compound was administered once orally to the rats, using 22-gauge oral feeding needle. The volume of the dose was dependent on the weight of the animal.

LD_{50} was determined by up and down method using two animals and increasing the doses of individual pesticide, (Mohineesh *et al.*, 2014). For each of the pesticide, five doses was administered in the proportion (0, 3, 5, 10, 15mg) making a total of 10 wistar rats. Those on zero level were regarded as control for that particular pesticide while the graded doses were used for the calculation of LD_{50} for a particular pesticide. 30 male wistar rats were used for the determination of LD_{50} orally to five groups of rats.

The wistar rats were observed for 2 hours, 4 hours, 6 hours and 24 hours for any toxic signs and symptoms. After 24 hours, the number of diseased rats in each group was counted, and percentage of mortality was calculated using the graphical method of Miller and Tainter (Mohineesh *et al.*, 2014).

2.1.2 Biochemical analysis

Male wistar rats were fed with only the standard 1100g growers mash and clean water. The wistar rats were allowed one week to acclimatize within the environment while on

normal growers mash and water. These served as control animals. Other male wistar rats were fed with mixture of grower's mash feeds and their respective pesticides (Aluminium phosphide, Chlorpyrifos and Dichlorvos). A total of 90 wistar rats were recruited into this study and divided into four groups: Group A had 15 male wistar rats and served as control animals while Group B, C, D contained 25 male wistar rats each and they were fed with a mixture of grower's feed and pesticides respectively. After 60 days the male wistar rats were sacrificed and blood serum samples collected for the following analysis:

Liver function tests (AST, ALT, and ALP)

Kidney function tests (Urea and Creatinine)

Lipid profile tests (Cholesterol, Triglyceride, HDL, and LDL)

2.2 METHODS OF DATA COLLECTION

Blood samples were left to clot and centrifuged at 5,000 rpm for 10 minutes to separate sera from the clotted blood.

Biochemical analysis were performed as shown below:

Serum aspartate transaminase (AST) and serum alanine transaminase (ALT) activity was carried out according to the colorimetric method, (Schmidt and Schmidt, 1963).

Serum alanine phosphatase (ALP) activity was determined using colorimetric method, (King and Jegathesan, 1953).

Blood urea was estimated by enzymatic method, (Pattern and Crouch, 1977). Serum creatinine was also determined according to the method of Faulkner and King (1976).

Plasma triglyceride was determined by enzymatic method (Mc Gowan *et al.*, 1983).

Plasma total cholesterol and HDL were determined enzymatically by Allain *et al* (1974).

LDL concentration was calculated from total cholesterol, HDL and triglyceride concentrations according to equation by (Friedewald *et al.*, 1972).

2.3.3 STATISTICAL ANALYSIS

The result was expressed as mean $\pm$ SD of five determinations for each group. The statistical package for the social science (SPSS) software version 16.01 was used for the data analysis. Analysis of variance (ANOVA) was used to test whether or not significant differences existed between groups. Pair-wise comparisons were made using the Post hoc test. Test probability value of $P < 0.05$ was considered significant.

3. RESULT

The result of the biochemical evaluation of male albino rat exposed to mashed grower's feed treated with different pesticides used in the research are presented as Tables 1(a, b and c), 2 (a, b and c) and 3 (a, b and c). The explanation of the result is thus as shown in the legend below.

Table 1a: Lipid Profile test (LP) on wistar rats administered with Dichlorvos pesticides

	Total Cholesterol (mmol/L)	Triglyceride (mmol/L)	HDL Cholesterol (mmol/L)	LDL Cholesterol (mmol/L)	VLDL Cholesterol (mmol/L)
1/10 dilution N=5	2.98 $\pm$ 0.52	1.10 $\pm$ 0.18 ^a	0.93 $\pm$ 0.14	0.93 $\pm$ 0.14	0.50 $\pm$ 0.08
1/50 dilution N=5	2.78 $\pm$ 0.41 ^a	1.35 $\pm$ 0.42 ^a	0.81 $\pm$ 0.08	0.81 $\pm$ 0.08 ^a	0.61 $\pm$ 0.19
1/100 dilution N=5	3.54 $\pm$ 0.66	1.15 $\pm$ 0.18 ^a	0.85 $\pm$ 0.06	0.85 $\pm$ 0.63	0.52 $\pm$ 0.89
Control N=5	3.74 $\pm$ 0.31	2.50 $\pm$ 0.32	0.89 $\pm$ 0.14	0.89 $\pm$ 0.14	0.60 $\pm$ 166
F-value	4.25	24.85	1.10	5.17	0.88
P-value	0.22	0.00	0.38	0.11	0.48

Key: **a** showed significant when compared to control; **b** showed significant when compared to 1/10 dilution; **c** showed significant when compared to 1/50 dilution; **d** showed significant when compared to 1/100 dilution.

Table 1b: Lipid Profile test (LP) on wistar rats administered with Aluminum Phosphide pesticides

	Total Cholesterol (mmol/L)	Triglyceride (mmol/L)	HDL Cholesterol (mmol/L)	LDL Cholesterol (mmol/L)	VLDL Cholesterol (mmol/L)
1/10 dilution N=5	3.28 $\pm$ 0.67	1.34 $\pm$ 0.14 ^a	0.79 $\pm$ 0.08	1.88 $\pm$ 0.65	0.61 $\pm$ 0.06
1/50 dilution N=5	3.040 $\pm$ 0.51	1.37 $\pm$ 0.12 ^a	0.92 $\pm$ 0.17	1.50 $\pm$ 0.45	0.62 $\pm$ 0.06
1/100 dilution N=5	2.840 $\pm$ 0.47	1.30 $\pm$ 0.12 ^a	0.86 $\pm$ 0.00	1.39 $\pm$ 0.47	0.59 $\pm$ 0.05
Control N=5	3.740 $\pm$ 0.31	2.50 $\pm$ 0.32	0.89 $\pm$ 0.14	2.24 $\pm$ 0.19	0.60 $\pm$ 0.16
F-value	2.9	44.46	1.07	3.45	0.09
P-value	0.07	0.00	0.39	0.04	0.97

Key: **a** showed significant when compared to control; **b** showed significant when compared to 1/10 dilution; **c** showed significant when compared to 1/50 dilution; **d** showed significant when compared to 1/100 dilution.

Table 1c: Lipid Profile test (LP) on wistar rats administered with Chloropyriphos pesticides

	Total Cholesterol (mmol/L)	Triglyceride (mmol/L)	HDL Cholesterol (mmol/L)	LDL Cholesterol (mmol/L)	VLDL Cholesterol (mmol/L)
1/10 dilution N=5	2.50±0.10 a	1.76 ± 0.03 a	0.67 ± 0.01	1.02 ± 0.10 a,d	0.80 ± 0.01 d
1/50 dilution N=5	2.70± 0.26 a	1.57 ± 0.25 a	0.89 ± 0.08	1.09 ± 0.12 a,d	0.71 ± 0.14 d
1/100 dilution N=5	3.03± 0.32 a	2.05± 0.15	0.86 ± 0.05	1.82 ± 0.20 a	0.35 ± 0.11
Control N=5	3.74± 0.31	2.50 ± 0.32	0.89 ± 0.14	2.24 ± 0.19	0.60 ± 0.16
F-value	15.91	11.08	3.57	49.95	6.97
P-value	0.00	0.00	0.06	0.00	0.01

Key: **a** showed significant when compared to control; **b** showed significant when compared to 1/10 dilution; **c** showed significant when compared to 1/50 dilution; **d** showed significant when compared to 1/100 dilution.

Table 2a: Kidney function test on wistar rats administered with Aluminum Phosphide pesticides

	Na (mmol/L)	K (mmol/L)	CL (mmol/L)	HCO ₃ (mmol/L)	Urea (mmol/L)	Creatinine (μmol/L)
1/10 dilution N=5	133.20 ± 2.17	11.34 ± 1.15	104.40 ± 3.05	16.60 ± 1.34 c	8.88 ± 3.65	91.80± 17.29
1/50 dilution N=5	133.60 ± 2.51	11.40 ± 1.14	103.60 ± 5.27	12.20 ± 1.64	9.44 ± 1.26	85.80 ±16.54
1/100 dilution N=5	133.60 ± 3.0	11.54 ± 1.92	105.00 ± 4.53	13.00 ± 3.39	8.28 ± 1.44	90.80 ± 15.73
Control N=5	135.80 ± 5.72	10.20 ± 0.73	104.40 ± 2.07	13.60 ± 1.34	9.44 ± 0.76	65.00 ±22.76
F-value	0.53	1.12	0.10	4.14	0.35	2.34
P-value	0.67	0.37	0.96	0.24	0.79	0.11

Key: **a** showed significant when compared to control; **b** showed significant when compared to 1/10 dilution; **c** showed significant when compared to 1/50 dilution; **d** showed significant when compared to 1/100 dilution.

Table 2b: Kidney function test on wistar rats administered with Chloropyriphos pesticides.

	Na (mmol/L)	K (mmol/L)	CL (mmol/L)	HCO ₃ (mmol/L)	Urea (mmol/L)	Creatinine (μmol/L)
1/10 dilution N=5	134.33±0.58	10.63± 0.15 c	108.00± 1.00	15.67± 0.58	8.63±0.321 c	187.67±0.577 a,c,d
1/50 dilution N=5	129.33±4.93	14.66± 1.95 a,d	110.67± 3.06 a	11.67±5.69	16.53±4.981 a	103.00±31.60
1/100 dilution N=5	134.33±2.08	11.70±1.15	108.00±3.00	15.00±3.00	12.60±.529	89.33±16.25
Control N=5	135.80±5.72	10.20±0.73	104.40±2.07	13.60±1.34	9.44±.764	65.00±22.76
F-value	1.44	10.91	4.71	1.05	7.93	21.20
P-value	0.29	0.00	0.23	0.41	0.01	0.00

Key: **a** showed significant when compared to control; **b** showed significant when compared to 1/10 dilution; **c** showed significant when compared to 1/50 dilution; **d** showed significant when compared to 1/100 dilution.

Table 2c: Kidney function test on wistar rats administered with Dichlorvos pesticides

	Na (mmol/L)	K (mmol/L)	CL (mmol/L)	HCO ₃ (mmol/L)	Urea (mmol/)	Creatinine (μmol/L)
1/10 dilution N=5	134.20 ± 1.10	11.58 ±2.10	107.80 ±5.26	15.00 ±3.94	10.48 ± 2.57	89.00 ±24.77
1/50 dilution N=5	127.20 ±5.85 a	15.20 ±2.95 a	110.80 ±2.17	10.00 ±2.74 a,b,d	9.04 ±3.73	97.40 ±33.69
1/100 dilution N=5	133.80 ±5.85	11.92± 1.72	106.80 ±4.21	15.60 ±1.14	7.96 ±0.91	73.00 ±15.60
Control N=5	135.80 ±5.85	10.20 ±.735	104.40 ±2.07	13.60 ±1.34	9.44 ±0.76	65.00 ±22.76
F-value	3.62	5.40	2.57	4.83	0.99	1.73
P-value	0.36	0.01	0.90	0.14	0.42	0.20

Key: **a** showed significant when compared to control; **b** showed significant when compared to 1/10 dilution; **c** showed significant when compared to 1/50 dilution; **d** showed significant when compared to 1/100 dilution.

Table 3a: Liver function tests (LFT) on wistar rats administered with Aluminum Phosphide pesticides

	AST (IU/L)	ALT (IU/L)	ALP (IU/L)	TOTAL BILIRUBIN (μmol/L)	CONJU. BILIRUBIN (μmol/L)
1/10 dilution N=5	51.40 ± 9.9	27.00 ± 4.47 a	65.60 ± 14.84 a	6.48 ± 1.24 a	1.92 ± 0.25
1/50 dilution N=5	53.40 ± 20.70	32.00 ± 8.71	80.00 ± 8.63	7.70 ± 1.30	2.28 ± 0.11
1/100 dilution N=5	53.20 ± 18.36	39.80 ± 14.37	81.00 ± 10.30	7.50 ± 0.62	2.32 ± 0.25
Control N=5	73.00 ± 15.86	50.60 ± 10.35	97.80 ± 11.78	8.66 ± 0.54	7.04 ± 10.04
F-value	1.87	5.16	6.43	4.11	1.18
P-value	0.18	0.01	0.00	0.02	0.35

Key: **a** showed significant when compared to control; **b** showed significant when compared to 1/10 dilution; **c** showed significant when compared to 1/50 dilution; **d** showed significant when compared to 1/100 dilution.

Table 3b: Liver function tests (LFT) on wistar rats administered with Dichlorvos pesticides

	AST (IU/L)	ALT (IU/L)	ALP (IU/L)	TOTAL BILIRUBIN (μmol/L)	CONJU. BILIRUBIN (μmol/L)
1/10 dilution N=5	63.80 ± 23.85	28.20 ± 5.12 a	93.20 ± 14.39	2.24 ± 0.30	2.24 ± 0.30
1/50 dilution N=5	75.40 ± 15.45	38.60 ± 12.40	74.40 ± 11.55 a	2.00 ± 0.12 a	2.00 ± 0.12
1/100 dilution N=5	75.40 ± 22.36	44.60 ± 5.68	90.00 ± 9.35	2.40 ± 0.42	2.40 ± 0.42
Control N=5	73.00 ± 15.85	50.60 ± 10.36	97.80 ± 11.78	2.55 ± 0.17	2.55 ± 0.17
F-value	0.39	5.71	3.64	3.12	4.35
P-value	0.76	0.01	0.04	0.06	0.02

Key: **a** showed significant when compared to control; **b** showed significant when compared to 1/10 dilution; **c** showed significant when compared to 1/50 dilution; **d** showed significant when compared to 1/100 dilution.

Table 3c: Liver function tests (LFT) on wistar rats administered with Chlorpyrifos pesticides

	AST (IU/L)	ALT (IU/L)	ALP (IU/L)	TOTAL BILIRUBIN (μmol/L)	CONJU. BILIRUBIN (μmol/L)
1/10 dilution N=5	41.00 ± 0.00 a,c	34.00 ± 0.00	60.33 ± 0.58 a	6.33 ± 0.05 a	2.00 ± 0.00
1/50 dilution N=5	76.00 ± 12.16	38.67 ± 0.57	63.33 ± 15.27 a	6.26 ± 1.32 a	2.20 ± 0.43
1/100 dilution N=5	48.33 ± 9.23	35.00 ± 6.93	87.67 ± 8.32	7.90 ± 0.43	2.43 ± 0.41
Control N=5	73.00 ± 15.85	50.60 ± 10.35	97.80 ± 11.77	8.66 ± 0.53	7.04 ± 10.04
F-value	6.96	4.64	10.78	10.52	0.62
P-value	0.01	0.03	0.00	0.00	0.62

Key: **a** showed significant when compared to control; **b** showed significant when compared to 1/10 dilution; **c** showed significant when compared to 1/50 dilution; **d** showed significant when compared to 1/100 dilution.

4. DISCUSSION

The result of our research findings showed that preservation of food crops with organophosphate pesticides could elicit minor deleterious effects to the organs of the consumers, some of the effects were shown in our biochemical parameters. The result of kidney function test such as electrolyte, urea and creatinine (EUC) for the group fed with food crops preserved in Dichlorvos showed that the values of sodium, potassium and bicarbonate 127.20 ± 5.85, 15.20 ± 2.95 and 10.00 ± 2.74 mmol/L respectively were significant as compared with their control values. The sodium and bicarbonate values were lower than control values while the potassium exhibited a higher value. The result of EUC of

chlorpyrifos pesticides treated male wistar rats followed the same trend. Whereas the electrolyte result of the Aluminium phosphide pesticide treated male wistar rats never followed the same pattern. It is possible that certain chemical in Dichlorvos and chlorpyrifos are responsible for the electrolyte imbalance in the male wistar rats. The US Environmental Agency (EPA) reported that pesticides are portable human carcinogens and produce a range of symptoms from headache and dizziness to seizures, convulsions and more rarely death (EPA, 2005).

The result of triglyceride of different dilutions (1/10, 1/50 and 1/100) were 1.34 ± 0.14, 1.37 ± 0.12 and 1.30 ± 0.12 mmol/L were lower than the control value of 2.50 ± 0.32 mmol/L of

male wistar rats administered with Aluminium phosphide pesticide treated. While the triglyceride, total cholesterol and LDL cholesterol followed the same pattern as the above. The results of the lipid profile on male wistar rats administered with chlorpyrifos pesticide treated showed LDL cholesterol and VLDL cholesterol values exhibited a statistical significant result as compared with the 1/100 dilutions with the LDL cholesterol values, it is probable that chlorpyrifos cannot elicit cardiovascular diseases since it can suppress high values of the LDL. Other parameters of the lipid profile followed the same trend as Dichlorvos and Aluminium phosphide. Teixeira *et al.*, (2015) reported that long exposure to polychlorinated pesticides DDT can raise cardiovascular risks among premenopausal women. Our research findings revealed that liver function test parameters for the male wistar rats exposed to Dichlorvos and Aluminum phosphide pesticides never distorted the normal functions of the liver since the parameters were intact with various parameters. It is probable that these pesticides are liver friendly, while male wistar rats fed with chlorpyrifos pesticides deviated slightly from above, on the value of AST at 1/10 dilution was significant when compared with the value at 1/50 dilution. While other parameters are within the normal value which signifies that there was no hepatotoxicity observed from the result of the parameters of the liver function test. They primarily affect the nervous system of the exposed organisms by inhibiting acetylcholinesterase (AChE) and raising acetylcholine levels (Soltaninejad and Abdollahi, 2009 and Dettbern *et al.*, 2006). Karami-Mohajeri and Abdollahi, (2011) reported that organophosphate pesticides induces oxidative stress that affect metabolic pathways, and cause multiple organ dysfunctions. Kumar *et al* (2010) reported similar observation. In human occupationally exposed to a wide range of organo phosphates (Ops), blood urea nitrogen and albumin along with serum aminotransferases are frequently elevated (Malekirad *et al.*, 2013). Since, there are limited data about the hepatotoxicity of OPs in humans, it is not certain whether changes in biochemical parameters can indicate actual liver damage. Conclusively this work is opened for further research investigations since there are several pesticides that are availably intended for storage and in the reduction of food spoilage rates. There is need therefore to investigate further on other pesticides with a view to determining their status vis-à-vis achieve a better agricultural yield with less deleterious effect in human.

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