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Research Article

Toxicological Effects of Dichlorvos Treated Bean Diets on Some Biochemical Indices and Liver Histology in Albino Wistar Rats

***El-Khalid Oinonyilokwu Onche Amali[1], Goodwin O Obochi[1], Benjamin A. Anhwange, Iwuchukwu Bruno Obinna[3], and Emmanuel Tolani Oladele[4]**

[1]Department of Biochemistry, College of Health Sciences, Benue State University, Makurdi, Benue State, Nigeria
[2]Department of Chemistry, Benue State University, Makurdi, Benue State, Nigeria
[3]Department of Biochemistry, Abia State University, Uturu, Abia State, Nigeria
[4]Department of Biochemistry, Federal University of Agriculture, Abeokuta, Ogun State, Nigeria
****Corresponding Author's email*: amalielkhalid@gmail.com; Phone: +2348034591172**

ABSTRACT

Food poisoning caused by agricultural pesticides poses a significant global health risk, particularly in developing countries. This study examined biochemical and histological alterations in Wistar rats fed with diets containing dichlorvos-treated beans. Freshly harvested beans were categorized into ten different groups. Group 1 (control) and groups 2–10 (treated with dichlorvos). The beans sample in each group received different doses: high doses (8ml/4kg, groups 2, 5, 8), medium (4ml/4kg, 3, 6, 9), and low (2ml/4kg, 4, 7 and 10). Groups 2-4 were un-parboiled, while 5-7 were parboiled. Groups 8, 9, and 10 contained a 1:1 mixture of un-parboiled and parboiled beans. After six months of storage, the beans were ground to powder, mixed with rat feed, and administered for 28 days. Rats were euthanized via mild chloroform inhalation, and blood samples were collected through cardiac puncture for biochemical analysis. The liver was excised and preserved in 10% formaldehyde for histopathological assessment. Results indicated significant ($p<0.05$) increases in AST, ALT, ALP, creatinine, and urea, while albumin (ALB) and total protein levels declined in Groups 2-4 and IV compared to the control. Elevated AST, ALT, and ALP levels, severe centrilobular hepatocellular necrosis, lobular necrosis and inflammation, and mild portal congestion of the liver suggest are indices of compromised structural integrity and myocardial infarction.

Keywords: Dichlorvos; Food poisoning; Pesticide toxicity; Myocardial infarction; Ultra-structural changes

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INTRODUCTION

Plant pests and diseases destroy about 45% of world crops (Islam *et al.,* 2014). This large-scale loss is a perennial threat to meeting global food demand and is one of the bases of global food insufficiency and hunger in many parts of the world. Food crop is lost at various stages in the chain of production, from the point of keeping seeds viable for planting, to germination or sprouting, to growth, maturity, and harvesting, processing and post-harvest storage. Ensuring effective, efficient, safe and affordable protective schemes at these various stages has been the farmers' and the marketers'· nightmare. Natural protective modules have not been widely successful as they hardly can keep pace in preventing the rapidly destructive activities of these pests. Consequently, pesticides of different types, ranging from natural to synthetic formulations, organic and inorganic compounds are often employed by farmers and marketers to improve the quantity and quality of harvest and post-harvest crops. Over 3 billion kilograms of pesticides are used annually in crop protection by the agro-allied sector, which is the highest benefactor of pesticides (Barrett, 2012).

Though pesticides have played remarkable roles in improving global food availability by preventing post-harvest loses, most pesticides however cause severe health challenges to humans, fishes and other animals (Khandakar 1990). Some of these health challenges could be acute, leading to such ill conditions as burning eyes or blindness resulting from direct pesticide contact with the eyes, rashes and blisters upon contact with the skin, nausea etc. In chronic cases, food substances containing low level of pesticide residues consumed by humans over time may lead to serious health hazards such as, cancer, teratogenesis, genetic damage and suppression of immune system (Okoroiwu and Iwara, 2018), congenital malformations, neurotoxic disorders, infertility, blood dyscrasias, and many others (Ogah *et al.,* 2012).

Dichlorvos (2, 2-dichlorovinyl dimethyl phosphate – DDVP), a pesticide of the class insecticide and an organophosphate, is widely implicated in the preservation of post-harvest crops, especially in beans (Yusuf *et al.,* 2017; Karigidi, 2018). In Nigeria, it is marketed as Sniper® and known locally as *'ota-pia pia'*.

Dichlorvos has been shown to have toxic effects on organisms in the environment using indices including mortality, immobilization, and growth inhibition. It is categorized as Group 2B (the agent is possibly carcinogenic to humans) by the IARC (IARC, 1991) and has shown positive genotoxicity results in several in-vitro tests of bacterial gene mutation, DNA damage, and DNA adduct formation (CERI, 2007). In acute or chronic doses, Dichlorvos has different adverse effects on biochemical parameters in humans and animals. In an experiment on hepatic and renal toxicity of dichlorvos on rabbits, dichlorvos caused significantly higher levels of serum ALT, AST, ALP, and γ-GT in dichlorvos-treated rabbits as compared to the control (Almalihi, 2016). In another experiment investigating the hepatotoxic and clastogenic effect of dichlorvos in male rats, all the different levels of doses of DDVP used produced significantly higher levels of γ-GT, ALP, ALT & AST as well as micro-nucleated polychromatic erythrocytes (mPCEs). The toxic effect was dose-dependent for both parameters (Gbadegesin, 2018).

Dichlorvos toxicity also affects human and animal protein profiles by influencing total protein and albumin metabolism. In a study by Almalihi (2016) involving exposure of rabbits to dichlorvos, there was a significant decline in the levels of total protein, albumin and globulin. The decline in protein profile may be as a result of changes in metabolism of proteins and free amino acids and their synthesis in the liver, damaging effects of dichlorvos on liver cells, increased proteolytic or degradative activities (Almalihi, 2016).

Histological studies on experimental animal models exposed to dichlorvos have demonstrated its toxic effects on several vital organs. In mice treated with dichlorvos, significant disruptions in hepatic histo-architecture were observed compared to the control group (Anderson *et al.,* 2020a). Additionally, rats exposed to a 1:1 dilution of dichlorvos exhibited moderate hepatocytic steatosis and lymphatic infiltration in the liver and lungs, while no significant changes were detected in the kidneys (Nwankwo *et al.,* 2019). Furthermore, various histopathological alterations were identified in the brain, heart, and lungs of rats exposed to sub-lethal doses of dichlorvos, commonly referred to as 'Ota pia-pia' (Benjamin *et al.,* 2020). Despite these numerous and grievous health hazards of dichlorvos, its use in domestic applications and for post-harvest protection of stored food crops is dangerously on the increase (Okoriowu and Iwara 2018). This study, therefore, evaluated changes in biochemical and histological parameters in liver architecture in albino Wistar rats fed with bean diets, which had been treated with different doses of dichlorvos six months previously.

MATERIALS AND METHODS

Collection of beans and treatment

Forty kilograms (40 kg) of beans (*Phaseolus vulgaris*) with the herbarium index number 001-BSU24, untreated with any pesticide, were obtained directly from farmers for this research. After thorough cleaning, the beans were divided into ten airtight glass containers, each holding 4 kg, and labeled as follows: Group *1* (control), Groups 2, 3, 4, 5, 6, 7, 8, 9, and 10. A 100 mL bottle of dichlorvos, labeled *Sniper 100EC* and manufactured by Ruigreat Company, Shanghai, China, was procured from Franklen Technologies, Nigeria Limited, located on Onitsha Street, behind City Bay Event Center, Makurdi, Benue State, Nigeria. The dichlorvos was then mixed with the beans according to the specifications outlined in Table 1.

Experimental animal models

Fifty (50) albino Wistar rats were obtained and housed in the Animal House Laboratory, College of Health Sciences, Benue State University, Makurdi. They were acclimatized for 2 weeks before treatment. Water was provided *ad libitum*. Ethical approval for this research was granted by the College of Health Sciences Research and Ethics Committee, Benue State University, Makurdi (Certificate Number: CRFC/THS/007), adhering to the guidelines of the National Institutes of Health for the care and use of laboratory animals. The rats

were grouped into four different feeding groups as demonstrated in Table 1.

Reagents and Apparatus

Other general laboratory chemicals such as acetone, anhydrous sodium sulphate, ethylacetate, and hexane are of analytical grades, and standard glasswares were used for this research.

Biochemical Parameters

After four (4) weeks of treatment with the various feed compositions, the rats were euthanized by chloroform inhalation and sacrificed. A blood sample (5 mL) was collected by cardiac puncture using a Vacotena, a sterile, pyrogen-free, non-toxic Maxicom Multiple Sample Needle. Four (4 mL) out of the 5 mL of blood collected was transferred into heparinized bottles and centrifuged at 4000rpm for 5 minutes using Centrifuge Model 80-2, Lenfield Medical England. The supernatant containing the plasma was carefully siphoned into plain bottles using a 5mL syringe and kept for biochemical analysis, which was conducted at Benue State University Teaching Hospital using an automated analyzer and by the method outlined in the works of Almalihi (2016). The biochemical indices measured included aspartate amino transferase (AST), alanine amino transferase (ALT), alkaline phosphatase (ALP), albumin (ALB), total protein (TP), creatinine, and urea.

Table 1: Rats allocation, feed treatment and dichlorvos dose per group

Group	No. of Rats	Feed Treatment	Dichlorvos (sniper) dose*
I	5	Control group Rat feed + untreated bean diet	None
2	5	Rat feed + treated un-parboiled beans	High
3	5	Rat feed + treated un-parboiled beans	medium
4	5	Rat feed + treated un-parboiled beans	Low
5	5	Rat feed + dichlorvos treated and parboiled bean diets	High
6	5	Rat feed + dichlorvos treated and parboiled bean diets	Medium
7	5	Rat feed + dichlorvos treated and parboiled bean diets	Low
8	5	Rat feed + dichlorvos treated un-parboiled bean diet + Dichlorvos treated parboiled bean diet	High
9	5	Rat feed + dichlorvos treated un-parboiled bean diet + dichlorvos-treated parboiled bean diet	Medium
10`	5	Rat feed and dichlorvos-treated un-parboiled bean diet, along with dichlorvos-treated parboiled bean diet	Low

*Key: High dose = 8mL dichlorvos/4 kg beans; Medium dose = 4mL dichlorvos/4 kg beans; Low dose = 2mL dichlorvos/4 kg beans

Histological Parameter

The liver was harvested, placed in sample tubes, and preserved in 10% formaldehyde before being stored in a refrigerator for further histological analysis using the paraffin technique described by Sadeghipour and Babaheidarian (2019). The formaldehyde-fixed tissues were processed and embedded in paraffin wax to form formalin-fixed, paraffin-embedded blocks. Thin tissue sections (5–6 µm) were then cut using a microtome, stained with haematoxylin and eosin, and examined under a microscope equipped with a digital camera at a magnification of ×400. Photomicrographs of each section were taken and carefully analyzed for any histological abnormalities in the liver.

Data Analysis

Data was presented as mean value ± standard deviation. Analysis of variance (ANOVA) was used for multiple factor analysis. Duncan's multiple range test at $P < 0.05$ was adopted for analysis of differences between means that are statistically significant.

RESULTS

Biochemical parameters

The results for biochemical parameters, such as aspartate amino transferase (AST), alanine amino transferase (ALT), alkaline phosphatase (ALP), albumin (Alb), total protein, creatinine and urea obtained from the treatment of albino Wistar rats with dichlorvos are presented in Table 2.

Histopathological parameters

Photomicrographs of the liver of rats fed with dichlorvos bean diet are presented in Plates I-X.

Table 2: Biochemical parame**ters of albino rats fed with dichlorvos-treated beans Diets**

Parameters/Groups	Control	1	2	3	4	5	6	7	8	9	10
AST (IU/L)	319.64± 1.23	354.68±3.06*	342.40 ±3.06*a	335.90 ±1.12*a	324.10± 1.55a	323.74±0.7b	320.30±2.1c	337.6±1.3*ad	334.40±1.2*e	328.90± 1.36*
ALT (IU/L)	119.10± 1.52	134.30± 2.20*	129.40± 1.79*	126.68± 1.27*a	123.50± 1.76a	121.20±0.9b	120.50±1.11	127.30±1.6*	123.60±1.13	121.50± 1.25
ALP (IU/L)	163.50 ± 1.59	180.00± 1.84*	170.20 ±1.06*a	165.62± 0.77a	164.90± 1.48a	164.50± 0.28	163.80±0.45	169.10±0.9a	167.70±1.72	166.10± 1.00
ALB (g/L)	27.30 ± 1.13	18.70±0.47*	21.40 ± 1.33*	23.80± 0.60a	25.30± 0.68a	26.10± 0.61	26.90 ± 0.96	21.00± 0.71*	22.70± 1.57	24.74 ± 1.47
Total Protein (g/dL)	60.02 ±0.77	51.40±0.71*	53.10 ± 1.70*	54.80 ±1.30	57.20± 1.43	57.90± 0.86	58.68 ± 0.84	53.40± 1.87*	55.10± 1.51	55.80 ± 1.49
Reatinine (µmol/L)	47.52 ±0.77	54.30±1.37*	51.30 ± 0.61	48.00± 1.30a	48.90 ± 0.51a	48.06 ± 0.75	47.70 ± 0.24	52.10± 1.26*	50.30± 0.50	49.30 ± 0.76
Urea (mmol/L)	5.60 ±0.31	9.60±0.19*	8.40 ± 0.22*a	7.20± 0.16*ab	6.04 ± 0.17a	5.80 ± 0.11b	5.50 ± 0.15c	7.80±0.29*ad	6.20 ± 0.19b	5.90 ± 0.08c

N = 5, *= significant relative to control (1) at P < 0.05, a = significant relative to 2 at P < 0.05, b = significant relative to 3 at P < 0.05, c = significant relative to 4 at P < 0.05, d = significant relative to 5 at P < 0.05, e = significant relative to 6 at P < 0.05

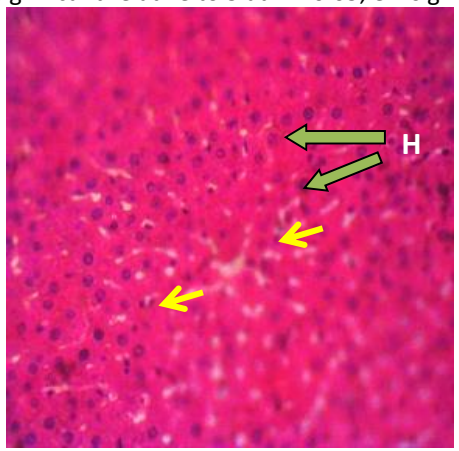

Plate I. Control (Group 1). Liver photomicrograph shows partial lobular hepatocyte (arrows). H= Hepatocytes. *Haematoxylin & eosin stain, x 400*

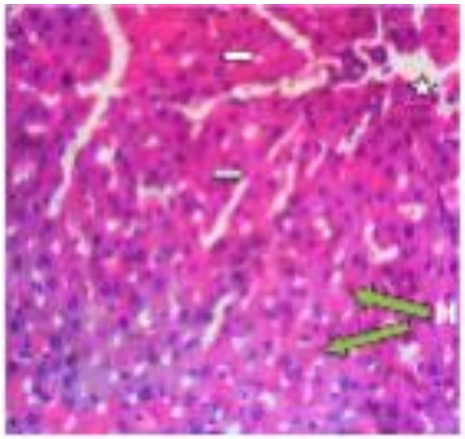

Plate II (Group 2). Severe centrilobular hepatocellular Necrosis (white arrow), more severe in the regior closer to the vein (White star).H = Hepatocytes. *Hematoxylin & eosin stain x400*

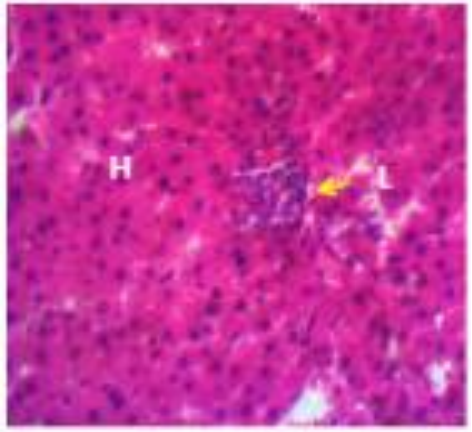

Plate III (Group-3). Lobular necrosis and inflammation is represented in this focus with hepatocyte dropout and lymphocyte aggregates (arrow). H Hepatocyte, L = Lymphocyte. *Hematoxylin & eosin stain, x 400*

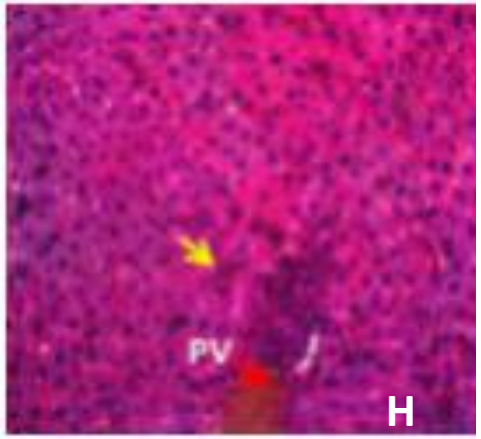

Plate IV (Group-4). Mild portal congestion (red arrow) and hepatocellular coagulation necrosis (yellow arrow). PV = Portal vein. *Hematoxylin & eosin stain x400*

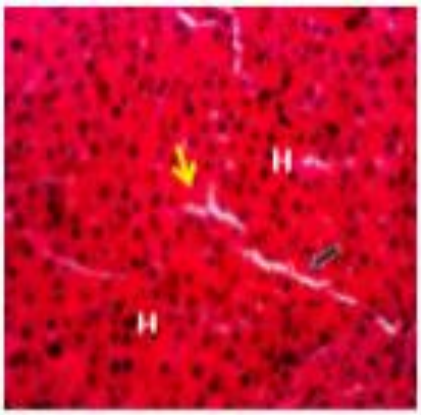

Plate V (Group-5). Partial necrosis of hepatocytes (yellow arrow) adjoining a sinusoid (black arrow). H = Hepatocyte. *Hematoxylin & eosin stain x400*

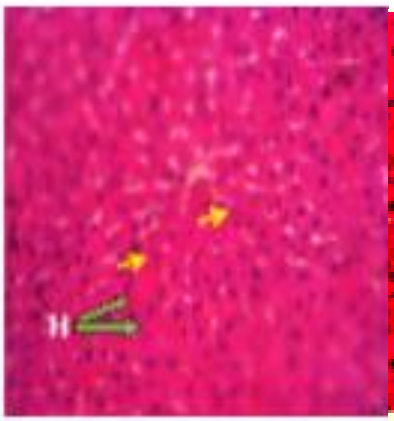

Plate VI (Group-6). Partial lobular hepatocyte necrosis (arrows). H= Hepatocytes. *Hematoxylin & eosin stain, x 400*

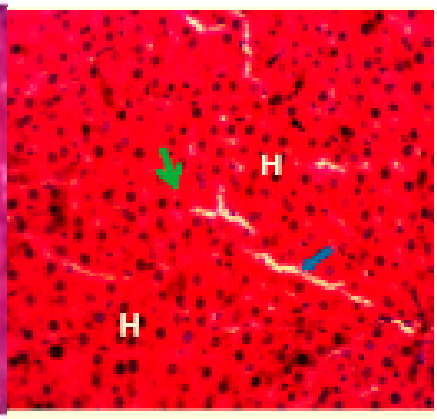

Plate VII (Group-7). Partial necrosis of hepatocytes (yellow arrow) adjoining a sinusoid (white arrow). H = Hepatocyte. Hematoxylin & eosin stain x400

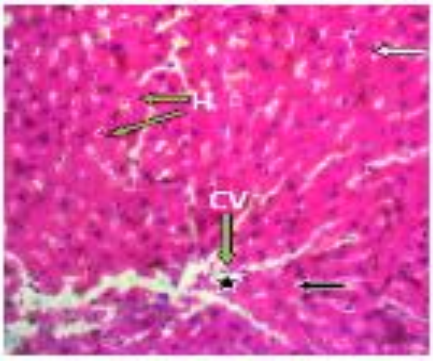

Plate VIII (Group-8). Cords of hepatocytes radiating away from a central vein (black star). Centrilobular necrosis is demonstrated here with better preservation of cellular features in the cells father away (white arrow) from the vein compared with the adjacent ones (black arrow). H = Hepatocytes, CV = Central vein. *Hematoxylin & eosin stain, x 400*

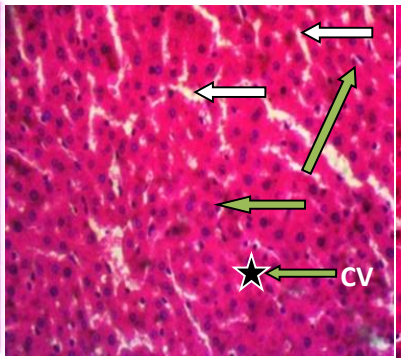

Plate IX (Group-9). Cords of hepatocytes, separated by sinusoids (black arrow), which radiate away from a central vein (black star). Centrilobular necrosis is demonstrated here with better preservation of cellular features in the cells father away (white arrow) from the vein. CV = Central vein. *Hematoxylin & eosin stain x400*

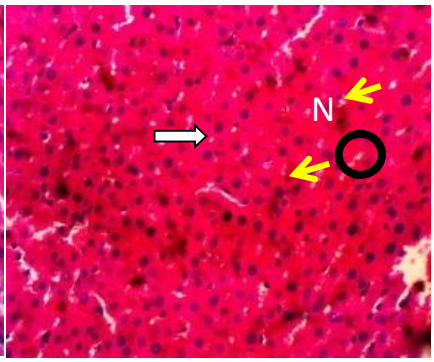

Plate X (Group-10). Hepatocytes with increased cytoplasmic eosinophilia and less distinct cell borders and some nuclear fragments consistent with hepatocellular necrosis. *Hematoxylin & eosin stain x400*

DISCUSSION

Dichlorvos has been detected in unacceptably high concentrations in bean samples six months after its application (Yusuf *et al.,* 2017). Different researchers have equally reported the effect of dichlorvos residues present in diets on the human and animal system (Almalihi, 2016). However, there is paucity of information on the effect of dichlorvos after it had been applied for six months and fed to human or animal models.

The results obtained from this research provided useful information on the health risks associated with consuming bean diets treated with dichlorvos and stored for over a supposedly 'long and safe period´ of six months

A significant ($p<0.05$) increase in the values of AST was observed in all the treated groups with the increase being significant in groups 2, 3, 4, 8, 9 and 10 when compared to the control. The result is in agreement with other research works where dichlorvos has been shown to increase AST levels in humans and other animals models (Idi-Ogede *et al.,* 2016).

The implication of this rise is an indication of a distress in the integrity of the liver. Liver disease can be caused by several causes among which is exposure to drugs that are harmful to the liver and other toxic substances. Dichlorvos is a known potent toxicant capable of increasing the flow of aspartate amir o transferase from the mitochondrion into the blood circulatory system (Almalihi, 2016).

Nwauzobilom *et al.* (2020) in their work on effect of different preparations of beans treated with dichlorvos on biochemical parameters of rats reported a dose dependent increase in liver enzymes. Their finding is corroborated by the results obtained in this research work. In groups 2-4 the result showed that even though the feed of the sub-groups were under the same treatment of not being parboiled, they differ significantly with the values of AST declining with decreasing dose level. For groups 5, 6 and 7 whose feed were treated and parboiled, the AST level was generally lower as compared to its corresponding dose level in groups 2-4 whose feed were treated but not

parboiled. This comparative reduction in AST may not be unconnected to the degrading effect of heat on the stability of dichlorvos (Milani, 2023). For groups 8, 9, 10 (mixture of parboiled and un-parboiled bean diets), there was a slight increase in the value of AST compared to both the control and groups 5, 6 and 7 (parboiled beans) but comparatively lower than that of groups 2, 3 and 4 (un-parboiled bean diets). This is so because groups 8, 9 and 10 contained components of un-parboiled beans treated with dichlorvos in a 1.1 ratio to parboiled diets, hence the dose level is expectedly higher than in groups 5, 6 and 7 but less than in groups 2, 3 and 4. These dichlorvos level differentials in the various groups is responsible for the amount of aspartate amino transferase (AST) released into the circulatory blood system by the liver, which in effect is a measure of the toxic effect of the substance on the liver. The primary function of the liver includes biotransformation of drugs and food that may include chemicals, anticancer drugs, analgesics, immunosuppressant, biological agents, radiations, heavy metals etc. (Ingawale *et al.,* 2014). Assessment of specific enzyme activities is an important tool for the diagnosis of liver disease though a drastic increase in liver enzymes may not be an outright certification of cell death. Mitochondrial enzymes released by the liver is associated with liver necrosis, elevated reactive oxidant species and lipid peroxidation (Contreras–Zentella, 2016). Intra and inter-group comparisons showed that the value of AST released increased with the dose level of dichlorvos used. This is in line with the dose dependent result obtained by Nwauzobilom *et al.* (2020) and Holy (2015a).

The elevated plasma ALT levels observed in treated rats, particularly in groups 2, 3, 4 and 8 align with findings from previous studies conducted by Almalihi (2016), Idi-Ogede *et al.* (2016), Kanu *et al.* (2016), and Ezeji and Onwurah (2017). The increase in ALT levels appeared to be dose-dependent, with group II showing the highest values compared to the control group. As shown in Table 1, the ALT values exceeded the normal range, suggesting potential liver inflammation. Alanine aminotransferase (ALT), like aspartate aminotransferase (AST), is a liver enzyme, and the liver serves as the primary detoxification site for foreign substances in the body (Holy *et al.,* 2015b). Consequently, it is highly susceptible to exposure to various toxins. In this study, dichlorvos exposure may have led to the leakage of cytosolic enzymes from the liver into the bloodstream, resulting in increased ALT levels, a finding consistent with the work of Holy *et al.* (2015a) and Nwauzobilom *et al.* (2020). A significant ($p<0.05$) decline in ALT levels was observed in groups 4 and 5 compared to group 2, while group 6 also showed a notable reduction when compared to group 3. These results further support the dose-dependent response of ALT to dichlorvos, as reported in previous studies Idi-Ogede *et al.* (2016), Kanu *et al.* (2016), Ezeji and Onwurah (2017). However, due to the non-specificity and multifunctional role of ALT in biochemical activities such as food metabolism, ALT levels may not always increase proportionally with certain liver impairments (Anderson *et al.,* 2020a).

Alkaline phosphatase increased in all the groups treated with dichlorvos as compared to the control group in agreement with the work of Jalili *et al.* (2022), which showed significantly($p<0.05$) higher values of ALP in rats exposed to dichlorvos. This increase was also noted for other liver enzymes. The result in this study showed a dose dependent increase in ALP level. This finding is also similar to other previous works (Holy *et al.* (*2015a*), Jalilli *et al.* (2022). The increase in ALP values obtained in this work gives an indication of liver disease or an abnormality of the bone. Previous work of Nwauzobilom *et al.* (2020) showed that dichlorvos has the potency to cause an upward trend in the value of alkaline phosphatase (ALP).

The result showed that plasma albumin level decreased across all the groups exposed to dichlorvos as compared to the control group. The result here is with the work of Almalihi (2016) which showed significant decline in the level of albumin in rabbits exposed to dichlorvos. Reduction in albumin concentration may be due to damaging effect of dichlorvos to the hepatocytes, changes in metabolism of proteins and free amino acids and their synthesis in the liver, increased proteolytic and catabolic activities (Almalihi 2016). The results demonstrated a consistent decline in albumin levels corresponding to increasing dichlorvos doses, with the most significant reduction observed in the group exposed to the highest dose (Group II). This dose-dependent trend aligns with the findings of Yang *et al.* (2011), who reported decreased albumin levels following 12 weeks of high-dose treatment. A reduction in total protein and albumin concentrations due to dichlorvos exposure was also documented by Oguteu *et al.* (2008), who suggested that this decline may result from disruptions in protein and albumin metabolism. Since the liver is responsible for albumin synthesis, exposure to dichlorvos can lead to liver damage, subsequently impairing albumin production. Additionally, albumin in the liver plays a crucial role in binding to and transporting drugs or chemicals (Oguteu *et al.,* 2008). The implication of the result obtained here is that the functional capability of the liver may have been compromised as a result of the deleterious activities of dichlorvos present in the

diets of the rats. This condition is known as hypo-albuminemia a situation where effective and efficient transportation of substances such as hormones, drugs and general maintenance of blood pressure has failed. This may result in health conditions such as jaundice where the white portion of the eye and the skin turn yellow (O´Carroll, 2023).

The result showed a decrease in value across all the groups exposed to dichlorvos. The decrease was dose dependent and significant when groups 2, 3 and 8 were compared to the control. These findings support the works of Almalihi (2016), Nwauzobilom *et al.* (2020) and Achikanu and Ani (2021).Reduction in concentration of total protein may be a consequence of the degradation of protein for formation of energy for the organism (Tulasi *et al.,* 2013; Revathi *et al.,* 2020). In a research investigation of effect of chlorphyrifos on protein metabolism, Reddy *et al.* (2011) reported a decline in total protein and amino acid concentration in the period of study. The decline was attributed to the conversion of protein to meet other vital prerequisites occasioned by chlorphyrifos toxicity, or due to the fact that chlorphyrifos causes vitality emergence and change protein digestion (Revathi *et al.,* 2020). A low total protein level is an indication of a liver or kidney distress or a disturbance in proper digestion and subsequent absorption of protein. The result of this work showed that dichlorvos may have caused hepato-renal toxicity thus impeding the organs from carrying out their metabolic functions properly and efficiently. A low total protein level (hypoproteinemia) can ultimately lead to anemia as a result of insufficient oxygen in the cells.

The result depicted increased creatinine values in all the groups treated with dichlorvos relative to the control group. This is in line with the work of Ojo *et al.* (2014) on dichlorvos induced nephrotoxicity in rats where increased creatinine was recorded. Different substances may exert nephrotoxic effects on the kidney thus leading to kidney dysfunction. The functional condition or status of the kidney is assessed by a constant evaluation of its biomarkers which consist of creatinine and urea amongst other more sensitive and specific biomarkers (Al-naimi *et al.,* 2019). About a quarter of the total cardiac output is directly received by the kidney and thus it is exposed to a large number of nephrotoxins leading to renal distress and eventually a possible renal failure (Shamna *et al.,* 2020).

The result of this study shows that dichlorvos acted as a nephrotoxin thus leading to a possible damage of the kidney such that there is poor clearance of creatinine by the kidney. This may be responsible for the rise in the level of creatinine in the blood. Other research works with exposure to different dose levels of dichlorvos validate increase in creatinine value (Ojo *et al.,* 2014; Almalahi, 2016).The increase in creatinine value in this research is dose dependent with the increase being significant ($p>0.05$) in groups 2 and 8 when compared to control group. For intra- and inter-group comparison only groups 4 and 5 differ significantly when compared to group 2.

The level of serum urea as displayed in Table 2 was generally elevated across all groups in response to dichlorvos presence in their diets as compared to the control. The increase was significant in groups II and 8 while non-significant in all the other remaining groups. This result is similar to previous research works that documented increased values of urea under the influence of dichlorvos (Ojo *et al.,* 2014; Almalahi, 2016; Shamna *et al.,* 2019). Formation of urea in most vertebrates is a function of the liver (Deka, 2015). Distortion in the normal value of urea depends on the pathological condition of the liver. This condition may lead to either increase or decrease in production of urea (Deka, 2015). The increase in urea concentration recorded in this study may be attributed to inadequate excretion due to kidney damage or aggravated degradation of protein due to the presence of dichlorvos in the system as evidenced in the report of Deka and Dutta (2015). Excess ammonia may also be converted to urea which is a less toxic substance by the liver in the urea – ornithine cycle (Donkor *et al.,* 2020). The major product generated from protein metabolism is urea while creatine phosphate metabolism leads to the production of creatinine. Both compounds are thus important biomarkers for the assessment of the functional status of kidney (Donkor *et al.,* 2020).

No abnormal structural distortions were identified in the photomicrograph of the liver of the control group. This is so because of the absence of the toxicant. However, in the treated groups various degrees of hepatic histoarchitectural alterations were observed. The result agrees with the work of Nwanko *et al.* (2019). Centrilobular necrosis recorded here corroborates the work of Holy *et al.* (2015a) while Anderson *et al.* (2020b) reported abnormalities of the structure of the liver. Normal liver structure was markedly affected by exposure of Wistar rats to dichlorvos in a work conducted by Ajao *et al.* (2017). The result of this work is in agreement with these reports.

CONCLUSION

This study demonstrates that chronic exposure to dichlorvos-treated bean diets, even after a six-month storage period, induces significant toxicological effects in albino Wistar rats. The

observed alterations in biochemical indices, including elevated levels of AST, ALT, ALP, creatinine, and urea, coupled with decreased levels of albumin and total protein, strongly suggest hepatic and renal dysfunction. These changes were generally dose-dependent, highlighting the cumulative toxicity of dichlorvos. Furthermore, the histological examination of the liver revealed structural abnormalities, confirming the hepatotoxic potential of dichlorvos. These findings underscore the health risks associated with consuming dichlorvos-contaminated foods and emphasize the need for stringent regulations and monitoring to minimize human and animal exposure. The results highlight that even after a seemingly safe storage period of six months, significant toxic residues of dichlorvos remain, posing a serious health hazard.

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