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THE EFFECT OF THE PESTICIDE DICHLORVOS (DDVP) ON ELECTROLYTE, METABOLITES AND ENZYME ACTIVITIES IN LIVER AND KIDNEY TISSUES OF NEW ZEALAND RABBITS

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ABSTRACT
This study assessed the biochemical, electrolyte, and enzyme parameters in liver and kidney of the New Zealand rabbits following oral exposure. This was done because the field exposure of rabbits and other animals is common occurrence due to the wide spread use of DDVP in pest control in farms. New Zealand rabbits were exposed to varying sublethal concentrations (0.00–0.03 mg/l) of Dichlorvos (DDVP) under controlled laboratory conditions. Electrolyte, metabolites and enzyme activities were measured and the data analyzed for means and standard deviations. Analyses of variance (ANOVA) was applied at the 95% confidence limit to check for variability among treatment groups. Turkey HSD Post HOC test was used to separate means where variability occurred. This was done by the aid of the SPSS® 20.0 statistical tool kit. Result indicate that Liver sodium rose to 88.33 ± 2.02 mg/L, potassium declined to 7.00 ± 0.58 mg/L, calcium from 7.27 ± 0.29 to 5.80 ± 0.17 mg/L. ALT increased to 44.33 ± 6.25 U/L, AST fell to 26.33 ± 1.76 U/L, creatinine dropped to 0.36 ± 0.06 mg/L, magnesium to 0.93 ± 0.03 mg/L, ALP to 103.67 ± 3.84 U/L, T.P to 4.90 ± 0.26 mg/L, ALB to 2.80 ± 0.17 mg/L, and urea to 4.23 ± 0.15 mg/L in comparison with the control. Kidney showed elevated sodium (up to 82.33 ± 3.84 mg/L), reduced potassium (6.33 ± 0.33 mg/L), stable calcium (~7.73 mg/L), increased AST, steady ALT, and reduced magnesium (1.00 ± 0.06 mg/L), ALP (87.33 ± 4.63 U/L), and urea (5.90 ± 0.20 mg/L) compared with the control. These findings suggest DDVP exposure disrupts electrolyte homeostasis and enzyme activities in liver and kidney tissues indicating organ-specific vulnerability to DDVP toxicity compared to the control.

KEY WORDS: *Dichlorvos, Electrolytes, Metabolites, Enzyme, Liver, Kidney, Rabbits*

1.0 INTRODUCTION
Modern crop agriculture relies heavily on pesticide use. This is because of the ability of pesticides to prevent pest infestation of crops, reduce food loses and thereby increasing yield. Pesticides are biocidal agents which alters metabolic process of target organisms. They are of different formulations which could work either as a microbial agents or disinfectant against target pests [1]. Organophosphates are among the most widely used pesticides globally due to their effectiveness in controlling a broad range of insect pests [2]. Dichlorvos (2,2-dichlorovinyl dimethyl phosphate, DDVP) is a commonly utilized organophosphate insecticide employed in agricultural, domestic, and veterinary settings.

However, despite the importance of pesticides, their application in open fields may present a plethora of environmental problems due to their toxic effect on both target and non-target organisms in the environments, as they by extension present a serious risk to humans and other bio-life forms in the environment [3]. There is a growing concern about the risk-benefit of pesticide use even in crop agriculture. Its extensive application raises environmental and public health concerns, especially in developing countries where regulatory control is limited.

Contamination of aquatic ecosystem and fresh fodder by pesticides either directly or indirectly can lead to fish kills, reduced fish productivity or elevated concentration of undesirable toxicants in fresh fodder, used for feeding herbivores animals like rabbits and this eventually affect the health of animals and humans alike consuming them [2].

Chronic exposure to DDVP has been associated with various toxicological effects, including neurotoxicity, hepatotoxicity, nephrotoxicity, and hemato-toxicity ([4], [5]).

Hence, the objective of this research is aimed at evaluating the sublethal toxicological effects of DDVP on New Zealand Rabbits.

2.0 MATERIALS AND METHOD
2.1 Source of Experimental Animals
Twenty-five (25) healthy New Zealand rabbits weighing between 1,800 to 2000g were obtained from Kester rabbit farm at Mbiama, Rivers State of Nigeria. Rabbits were handled with care using gloves. They were lifted or carried by the skin in the dorso-cervical area, a fold of muscles at the dorsum of the upper part of the neck. They were all transported individually in plastic baskets in a closed vehicle to the animal farm, Department of Livestock Production, Niger Delta University Amassoma, Bayelsa State, Nigeria.

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2.2 Acclimation

The sample rabbits were put inside rabbit hutches, one rabbit in a compartment and the remainder in a reservoir for acclimation. The acclimation period lasted for 21 days and during this period, rabbits were provided with 1.5L of tap water, held in metal containers. Rabbits were fed with 200g of feed (synthetic grower's marsh pelletized) daily with antibacterial drugs mixed at their right proportions to prevent common poultry diseases such as scabies and other related poultry infections. The feed and water were changed at 24 hour intervals, while anti-bacterial drugs were renewed on weekly basis (every seven days). The compartments were also cleaned and swept at 24 hours' intervals to maintain good hygiene during the experimental period. The acclimation procedure was stopped 24hours prior to the start of the definitive experiment.

2.3 Experimental Chemical

The insecticide, Dichlorvos also known as DDVP with trade name commonly called sniper® was purchased from a chemical store at Swali market, Yenagoa, Bayelsa State, Nigeria.

2.4 Determination of Sublethal Doses

Sublethal doses used for the definitive experiment was determined following a range verdict experiment (trial experiment). The trial test lasted for two weeks (14 days) during which four arbitral concentrations (0.00mg/l, 0.01mg/l, 0.02mg/l and 0.03mg/l) was prepared. A renewal bioassay was carried out throughout the period of the trial test and definitive experiment. The concentrations obtained during the trial test was converted to milligram per litre (mg/L), following the method and formula described by Inyang, [6] where:

$N_1 V_1 = N_2V_2$

N_1 = manufacturers concentration (770g/l)
V_1 = concentration of the test solution preferred
N_2 = quantity of the original solution added
V_2 = quantity of the test solution

2.5 Definitive Test

The experiment was divided into two main groups, referred to as treatment group and control group respectively. The control (0.00mg/l) and treatment (0.01mgl, 0.02 and 0.03) group was replicated into four (4) replicates.

A completely randomized bioassay experiment was conducted and lasted for about four weeks (30 days), after which rabbits were killed and samples collected for analysis in the laboratory. Four arbitral sub-lethal concentrations were used as obtained from the trial test, following the method described by Inyang, [6]. Experimental rabbits were randomly selected and exposed to these concentrations in the definitive test for 30 days to determine the sublethal effect of toxicant on various tissues and organs of New Zealand rabbits.

The concentrations obtained from the trial test was mixed with 1.5L of tap water in metal containers and orally served to experimental rabbits in various compartments housing experimental rabbits, except for the control group with no toxicant exposure. Exposure lasted for thirty (30) days with the water and toxicant renewed daily (24 hours). During this period, rabbits were also fed with synthetic poultry feed (grower's marsh pelletized) on daily basis (24 hours).

2.6 Collection of Organs

Liver and kidney from experimental rabbits were collected by dissecting animals using a dissecting knife. A part of these organs (liver and kidney) were collected and crushed in a ceramic mortar. Crushed organ (s) were then mixed with 0.5 ml of perchloric acid for metabolites, deionized water (distilled water) for electrolytes and physiological saline (normal saline) for enzymes as the case may be. Mixture (s) were also centrifuged for 15 minutes at 3000 rpm and the supernatant (serum) poured into well labelled sample bottles and refrigerated until analysis.

2.7 Laboratory Analysis

Biochemical (enzymes) parameters, electrolytes and metabolites in the liver and kidney of exposed Rabbits (New Zealand Rabbits) were analyzed in the chemical laboratory of the Federal Medical Centre (FMC), Yenagoa to determine the sublethal effect of the toxicant (Dichlorvos) on experimental Rabbits**.** Enzymes such as AST, ALT and ALP in the liver and kidney, electrolytes (Na^+, K^+, Cl^- and Mg^{2+}) in the liver and kidney as well as metabolites (Total protein, Albumin, Urea and Creatinine) in the liver and kidney of rabbits (New Zealand Rabbits) exposed to the toxicant were analyzed in this experiment.

3.0 RESULT

The result of the electrolytes, metabolites and enzymes in the liver and kidney of the New Zealand rabbit are presented in Tables 1 to 6.

Table 1: THE EFFECT OF DDVP ON THE ELECTROLYTES IN THE LIVER

Concentration of Dichlorvos (mg/l)	Na^+ (mmol/l)	K^+ (mmol/l)	Cl^- (mmol/l)	Mg^{2+} (mmol/l)
0.00	9.70 ± 4.15^a	10.30 ± 3.85^b	7.38 ± 4.78^a	0.73 ± 0.17^a
0.01	41.33 ± 40.44^c	17.05 ± 6.94^d	40.53 ± 36.42^c	0.41 ± 0.28^a
0.02	3.03 ± 2.18^{ab}	12.50 ± 4.88^b	6.80 ± 3.30^{ab}	0.70 ± 0.17^a
0.03	12.88 ± 6.57^b	12.78 ± 5.21^b	13.80 ± 2.45^b	0.31 ± 0.25^a

The superscript used (a, b & c) represent a significant or no significant difference among treatment groups at alpha level $p<0.05$.

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Table 2: THE EFFECT OF DDVP ON THE ELECTROLYTES IN THE KIDNEY

Concentration of Dichlorvos (mg/l)	Na^+ (mmol/l)	K^+ (mmol/l)	Cl^- (mmol/l)	Mg^{2+} (mmol/l)
0.00	6.50 ± 3.44^{ab}	5.35 ± 3.20^{ab}	10.6 ± 2.92^{b}	0.44 ± 0.06^{a}
0.01	5.75 ± 2.52^{a}	7.30 ± 2.72^{a}	8.03 ± 3.59^{a}	0.48 ± 0.07^{a}
0.02	12.85 ± 6.70^{b}	2.08 ± 1.51^{a}	7.85 ± 2.44^{ab}	0.31 ± 0.13^{a}
0.03	24.55 ± 16.25^{b}	4.28 ± 1.59^{a}	4.05 ± 2.44^{a}	0.02 ± 0.13^{a}

The superscript used (a, & b) represent a significant or no significant difference among treatment groups at alpha level $p<0.05$.

Table 3: THE EFFECT OF DDVP ON THE METABOLITES IN THE LIVER

Concentration of Dichlorvos (mg/l)	T.P (g/l)	ALB (g/l)	UREA (mmol/l)	CREAT (µmol/l)
0.00	3.75 ± 1.50^{a}	2.00 ± 0.82^{a}	0.25 ± 0.17^{a}	43.75 ± 12.18^{b}
0.01	3.25 ± 0.96^{a}	2.25 ± 0.96^{a}	0.25 ± 0.06^{a}	46.75 ± 16.26^{b}
0.02	1.25 ± 0.50^{a}	1.75 ± 0.50^{a}	0.75 ± 0.44^{a}	19.00 ± 3.65^{c}
0.03	3.50 ± 0.58^{a}	2.00 ± 0.82^{a}	1.18 ± 1.13^{a}	46.75 ± 32.68^{b}

The superscript used (a, & b) represent a significant or no significant difference among treatment groups at alpha level $p<0.05$.

Table 4: THE EFFECT OF DDVP ON THE METABOLITES IN THE KIDNEY

Concentration of Dichlorvos (mg/l)	T.P (g/l)	ALB (g/l)	UREA (mmol/l)	CREAT (µmol/l)
0.00	2.50 ± 0.58^{a}	2.25 ± 1.26^{a}	0.25 ± 0.13^{a}	45.00 ± 16.75^{b}
0.01	2.25 ± 0.50^{a}	1.75 ± 0.96^{a}	0.20 ± 0.08^{a}	33.00 ± 12.25^{b}
0.02	1.75 ± 0.96^{a}	2.00 ± 1.41^{a}	0.25 ± 0.17^{a}	48.00 ± 7.62^{b}
0.03	2.00 ± 0.82^{a}	2.00 ± 0.82^{a}	0.55 ± 0.37^{a}	36.5 ± 5.07^{b}

The superscript used (a, & b) represent a significant or no significant difference among treatment groups at alpha level $p<0.05$.

Table 5: THE EFFECT OF DDVP ON THE ENZYMES IN THE LIVER

Concentration of Dichlorvos (mg/l)	AST (µ/l)	ALT (µ/l)	ALP (µ/l)
0.00	454.25 ± 75.94^{a}	51.00 ± 15.25^{b}	87.5 ± 24.53^{b}
0.01	77.75 ± 35.32^{c}	20.75 ± 3.10^{b}	41.75 ± 11.98^{b}
0.02	30.25 ± 17.48^{b}	203.75 ± 144.77^{a}	41.75 ± 31.28^{b}
0.03	148.50 ± 142.40^{a}	75.00 ± 40.85^{c}	18.25 ± 3.59^{d}

The superscript used (a, b, c & d) represent a significant or no significant difference among treatment groups at alpha level $p<0.05$.

Table 6: THE EFFECT OF DDVP ON THE ENZYMES IN THE KIDNEY

Concentration of Dichlorvos (mg/l)	AST (µ/l)	ALT (µ/l)	ALP (µ/l)
0.00	83.75 ± 67.27^{b}	55.50 ± 36.13^{b}	140.25 ± 45.21^{d}
0.01	59.00 ± 24.51^{b}	17.25 ± 4.65^{a}	185.75 ± 153.60^{d}
0.02	192.50 ± 145.86^{d}	37.00 ± 34.07^{a}	191.00 ± 179.74^{d}
0.03	164.75 ± 117.02^{d}	65.75 ± 6.85^{b}	51.25 ± 40.57^{b}

The superscript used (a, b, c & d) represent a significant or no significant difference among treatment groups at alpha level $p<0.05$.

3.1 DISCUSSION

Effect of DDVP on the Electrolytes on Liver of New Zealand Rabbit

The data presented in the Tables 1 and 2 illustrate the significant impact of dichlorvos (DDVP) exposure on hepatic electrolyte concentrations in New Zealand rabbits, with a clear dose-dependent alteration across sodium (Na^+), potassium (K^+), chloride (Cl^-), and magnesium (Mg^{2+}) levels. The control group (T_0R_{14}) maintained normal electrolyte values, indicating stable liver function under non-toxic conditions. However, deviations observed in treated groups suggest hepatic stress and cellular disruption following DDVP exposure.

In 0.01mg/l, there was a marked increase in Na^+ (41.33±40.44 mmol/l) and Cl^- (40.53±36.42 mmol/l), indicating impaired ionic regulation, possibly due to disrupted Na^+/K^+-ATPase function or hepatocellular injury [7]. These abnormal elevations may result from ion retention due to membrane pump failure or inflammation-induced oxidative stress. Similar trends were reported by Ogbonna et al. [8], where increased sodium and

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chloride levels were associated with pesticide-induced liver damage in rodents.

Interestingly, group 0.02mg/l exhibited significantly reduced Na^+ (3.03±2.18 mmol/l), with moderate Cl^- levels (6.80±3.30 mmol/l), indicating electrolyte depletion or excessive loss, potentially through bile or urine due to hepatic inefficiency [9]. The relatively high potassium (12.50±4.88 mmol/l) in this group suggests cellular leakage of K^+ from hepatocytes undergoing necrosis or apoptosis, a finding that aligns with Arinze et al. [10], who noted similar intracellular-extracellular ion shifts in DDVP-exposed rabbits.

T_3R_{14} showed moderately elevated Na^+ (12.88±6.57 mmol/l) and the highest Cl^- concentration (13.80±2.45 mmol/l) among treated groups, implying partial restoration of electrolyte regulation possibly due to adaptive or compensatory hepatic responses after prolonged or lower-dose exposure [11]. Nonetheless, the consistent rise in K^+ across all treatment groups underscores persistent membrane permeability damage—a hallmark of hepatotoxicity.

Magnesium levels progressively declined from control (0.73±0.17 mmol/l) to 0.03mg/l (0.31±0.25 mmol/l), suggesting chronic depletion. This may be attributed to impaired absorption, increased excretion, or utilization during antioxidant defense processes [12]. Overall, the observed hepatic electrolyte imbalances highlight DDVP's toxic potential, particularly its capacity to disrupt membrane integrity and impair ion homeostasis. These findings are corroborated by earlier reports emphasizing the systemic toxicity of organophosphates, especially their effects on liver enzyme activity, oxidative stress markers, and electrolyte dynamics ([13], [14]). The data further reinforce the need for regulated pesticide use and underline the liver's vulnerability to organophosphate-induced toxicity in rabbits.

Effect of DDVP on the Electrolytes on the kidney of New Zealand Rabbit

The current study reveals substantial alterations in kidney electrolyte levels of New Zealand rabbits following DDVP exposure. The electrolytes exhibited marked variations across treatment groups, indicating DDVP's nephrotoxic potential and its influence on electrolyte regulation. Sodium levels rose progressively from the control group (6.50 ± 3.44 mmol/L) to significantly higher levels in 0.02mg/l (12.85 ± 6.70 mmol/L) and 0.03mg/l (24.55 ± 16.25 mmol/L). This sharp increase may reflect impaired sodium reabsorption or glomerular filtration dysfunction, suggesting possible tubular damage or reduced renal clearance of sodium. Similar hypernatremia was reported by El-Demerdash [15] in rats exposed to chlorpyrifos, an organophosphate, where elevated sodium levels were linked to nephrotoxicity and oxidative damage to renal tissue. These findings imply that DDVP may compromise renal sodium handling, potentially through oxidative stress and inhibition of renal Na^+/K^+ ATPase activity [16].

Potassium concentrations showed a significant decline, especially in 0.02mg/l (2.08 ± 1.51 mmol/L) and 0.03mg/l (4.28 ± 1.59 mmol/L), compared to the control (5.35 ± 3.20 mmol/L). This hypokalemia may be attributed to excessive renal potassium loss or redistribution of potassium into cells due to stress-induced hormonal responses. The observed decline contradicts some reports of hyperkalemia in acute organophosphate toxicity [17], suggesting that chronic or subacute DDVP exposure may trigger aldosterone-mediated potassium excretion or tubular dysfunction leading to impaired reabsorption.

Chloride levels followed a downward trend, with significant reductions from the control value (10.6 ± 2.92 mmol/L) to 4.05 ± 2.44 mmol/L in 0.03mg/l. Decreased chloride levels are indicative of renal handling disturbances or possible metabolic alkalosis, where chloride is lost in compensation for pH regulation. These findings align with the study by Rezg et al. [18], which demonstrated chloride depletion in rats following pesticide exposure, linked to tubular damage and compromised ion transport.

Magnesium levels also declined significantly, most notably in 0.03mg/l (0.02 ± 0.13 mmol/L), compared to the control (0.44 ± 0.06 mmol/L). Magnesium plays a vital role in kidney function, and its loss may exacerbate other electrolyte imbalances. Reduced Mg^{2+} has been associated with oxidative renal injury and increased urinary excretion in pesticide-exposed animals [19]. DDVP exposure leads to dose-dependent disruptions in kidney electrolyte concentrations in New Zealand rabbits. The elevated sodium and reduced potassium, chloride, and magnesium levels reflect impaired renal function, consistent with previous reports of pesticide-induced nephrotoxicity.

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