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Indicators of Glucose and Glycogen Levels in Acute Nurel Poisoning

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Abstract The article is devoted to the study of the effect of organophosphorus insecticides, in particular the pesticide Nurell, on carbohydrate metabolism in animals. In experiments using rabbits, changes in blood glucose levels and liver glycogen content after aeroallergenic intoxication with the pesticide were studied. The results showed that during the first days, hyperglycemia is observed due to disruption of metabolic enzymes, which is confirmed by an increase in blood glucose levels by 25-30% compared to the control group. On the 15th day, the glucose level decreased, indicating normalization of the condition. The glycogen content in the liver initially decreased, but then significantly increased, which may be associated with compensatory reactions of the body. These studies confirm that pesticide exposure has a significant effect on carbohydrate metabolism and emphasize the need to control the use of pesticides to minimize the risk of metabolic disorders in humans and animals.

Keywords Pesticides, Glucose, Glycogen, Liver, Rabbit

1. Introduction

Pesticides are regularly used in integrated agricultural practices to protect crops and livestock from pests, weeds, and diseases.** Intensive use of pesticides at various stages of cultivation, from seed treatment to the storage of agricultural products, poses a serious threat to human health due to their intense interaction with the body. It is well-established that pesticides used in pest control programs induce numerous physiological and biochemical changes in the human body, influencing the activity of various enzymes, which is reflected in general and biochemical blood tests.

Organophosphorus insecticides (OPIs) represent one of the leading groups of compounds employed to control pest populations in diverse agricultural and domestic settings. The primary mechanism of OPIs lies in the inhibition of the enzyme acetylcholinesterase, which plays a critical role in neurotransmission by hydrolyzing acetylcholine, a neurotransmitter involved in nerve impulse transmission. The disruption of acetylcholinesterase activity leads to the accumulation of acetylcholine and excessive stimulation of acetylcholine receptors, considered the principal mechanism of acute toxicity for these compounds.

Numerous studies have demonstrated that pesticide exposure, particularly organophosphorus compounds, can alter blood glucose levels and liver glycogen content. For instance, acute exposure to acephate, one of the OPIs, causes a transient increase in blood glucose levels (hyperglycemia), followed by a normalization phase. Notably, this effect is reversible and is accompanied by changes in the activity of gluconeogenesis enzymes, such as glucose-6-phosphatase and tyrosine aminotransferase, indicating stimulation of hepatic gluconeogenesis [1].

Another example is a study analyzing blood enzyme activity in tobacco farmers exposed to pesticides. The activities of AST, ALT, CK, and LDH were significantly elevated ($p < 0.001$) in farmers compared to the control group. These findings indicate cytotoxic disturbances in the kidneys and liver [2].

Furthermore, oral intoxication with chlorpyrifos in male Sprague-Dawley rats revealed significant decreases in succinate dehydrogenase and lactate dehydrogenase activities in blood biochemical analyses. Both enzymes are crucial for energy metabolism, and their reduction impacts cellular energy exchange [3].

Subsequent studies observed a reduction in hemoglobin and hematocrit levels, alongside an increase in leukocyte count due to lymphocytes and monocytes in the general blood analysis of Sprague-Dawley rats [4].

Thus, studying changes in glucose and glycogen levels under pesticide exposure is a vital task for assessing their toxicological potential, developing methods to protect public health, and maintaining ecological sustainability.

2. Materials and Methods

As an experimental model, sexually mature male New Zealand rabbits weighing 2.4–2.7 kg were used. The animals

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were kept under standard vivarium conditions with a natural light cycle (12 hours light/12 hours dark) and ad libitum access to food and water. Before the experiments, the animals underwent a 7-day adaptation period.

To study the effects of pesticides on carbohydrate metabolism, the organophosphorus insecticide chlorpyrifos was used. Chlorpyrifos was dissolved in saline and sprayed as a mixture twice daily every 3 days.

The animals were divided into five groups:

1. Control group (n=5): Not subjected to aeroallergen intoxication.
2. 24-hour pesticide exposure group (n=5).
3. 48-hour pesticide exposure group (n=5).
4. 7-day pesticide exposure group (n=5).
5. 15-day pesticide exposure group (n=5).

Blood glucose levels were measured using a glucometer with capillary blood samples taken from the central ear vein at various time intervals after aeroallergen intoxication: 0 hours, 24 hours, 48 hours, 7 days, and 15 days. All measurements were performed after fasting.

At the end of the experiment, the animals were euthanized by decapitation under mild ether anesthesia. The liver was excised and immediately frozen in liquid nitrogen for subsequent biochemical analysis. Liver glycogen content was determined using the Morris method with spectrophotometric analysis at a wavelength of 490 nm. Results were expressed in micromoles of glucose per gram of tissue.

3. Research Results

This study examined the changes in blood glucose levels and liver glycogen content in rabbits under the influence of the pesticide Nurelle. During the experiment, blood glucose levels and liver glycogen content were evaluated at 24 and 48 hours, as well as on days 7 and 15 after aeroallergen intoxication.

The results of blood glucose measurements are presented in Table 1.

The most significant increase in blood glucose levels was observed in the group exposed to Nurelle for 24 and 48 hours, and on day 7 (4.77±0.26 mmol/L, 4.60±0.33 mmol/L, 4.77±0.21 mmol/L, respectively), corresponding to increases of 30%, 25%, and 30% compared to the control group (87±4 mg/dL). By day 15, blood glucose levels decreased to 3.53±0.37 mmol/L, indicating a reduction compared to the control group (see Figure 1).

Table 1. Status of Glucose and Glycogen Indicators during Acute Nurelle Poisoning

Indicator	unit of measurement	Control	24 hours	48 hours	Day 7	Day 15
Glucose (mmol/L)	M ± m в %	3,68±0,24	4,77±0,26*** 129,6	4,60±0,33*** 125,0	4,77±0,21*** 129,6	3,53±0,37 95,9
Glycogen (mg/g)	M ± m в %	8,52±0,66	6,87±0,62* 80,6	9,23±0,4 108,3	9,62±0,82 113,0	9,95±0,75 115,6

Statistical significance compared to the control group is denoted as *p < 0.05; **p < 0.01; ***p < 0.001.

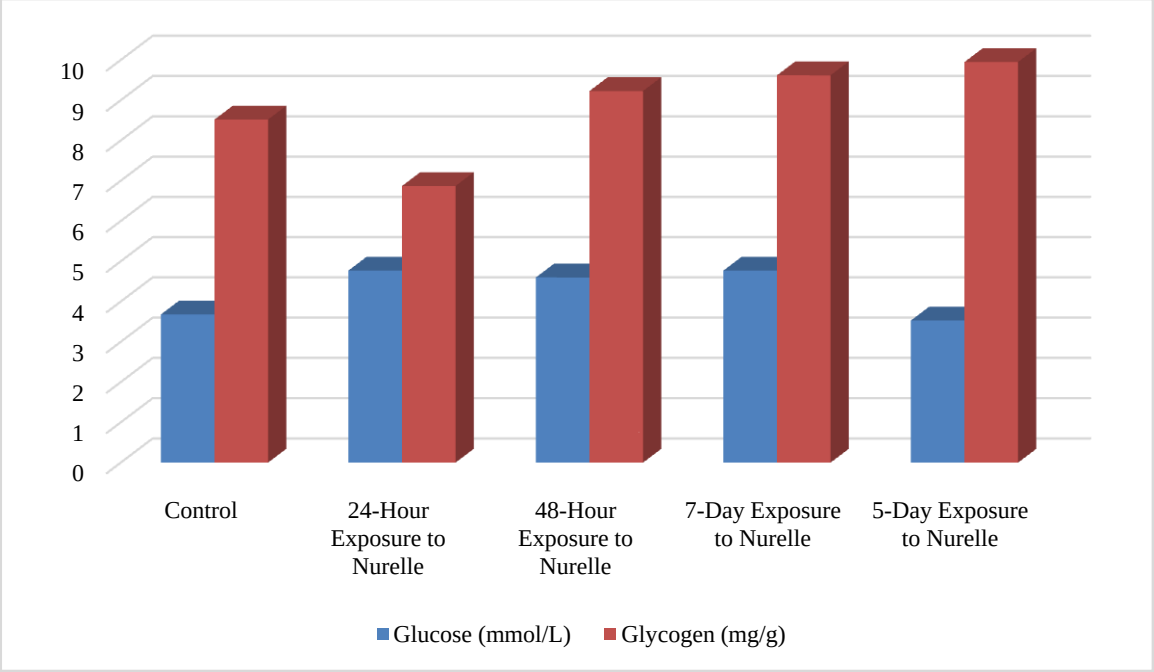

Figure 1. Status of Glucose and Glycogen Indicators During Acute Nurelle Poisoning

The liver glycogen content was determined using Morris's method and is presented in Table 1.

The glycogen level in the liver of the control group was 8.52±0.66 mg/g. After 24 hours of Nurelle exposure, glycogen content decreased to 6.87±0.62 mg/g, which slightly differed from the control value. However, after 48 hours, as well as on days 7 and 15 following pesticide intoxication, glycogen levels significantly increased to 9.23±0.4 mg/g, 9.62±0.82 mg/g, and 9.95±0.75 mg/g, representing increases of 8.3%, 12.9%, and 16.7% compared to the control group, with significant differences from the control values.

4. Discussion

The results of this study demonstrate significant changes in glucose and glycogen levels in rabbits exposed to the pesticide Nurelle. These findings align with conclusions from other researchers, confirming the impact of organophosphorus pesticides on carbohydrate metabolism.

Our results indicate that at 24 and 48 hours and on day 7 following exposure to Nurelle, blood glucose levels increased to 4.77±0.26, 4.60±0.33, and 4.77±0.21 mmol/L, exceeding control group levels by 25–30%. These changes support findings by Mbah Ntepe Leonel Javeres et al., who reported hyperglycemia, dyslipidemia, and arterial hypertension in individuals with prolonged organophosphorus compound intoxication. Their study also observed elevated insulin and blood sugar levels in people exposed to organophosphorus pesticides, particularly those with metabolic syndrome. Chronic exposure to such pesticides can lead to endocrine disruptions that affect enzymes involved in multiple metabolic pathways [5].

The observed increase in glucose levels also aligns with findings by Mathuramat Seesen et al. [6], who noted that organophosphorus pesticides could reduce tissue insulin sensitivity, impairing glucose utilization and resulting in hyperglycemia. Interestingly, by day 15, glucose levels decreased to 3.53±0.37 mmol/L, representing 95.9% of the control value.

The liver glycogen level, as shown in our data, decreased to 6.87±0.62 mg/g (80.6% of the control value) at 24 hours. This reduction may be attributed to active glycogen mobilization to maintain elevated glucose levels during intoxication. Similar conclusions were presented by Somayyeh Karami-Mohajeri et al. [7], who described that pesticides trigger glycogen breakdown in the liver in response to metabolic stress.

However, by 48 hours and at later stages, glycogen levels significantly increased, reaching 9.95±0.75 mg/g by day 15. This trend corresponds to findings by Raja Rezg et al. [8], who observed glycogen accumulation following the cessation of acute toxic effects. In our case, this may be associated with the activation of overcompensation mechanisms or disrupted glycogen metabolism under prolonged toxin exposure.

5. Conclusions

The study confirms that exposure to organophosphorus pesticides, such as Nurelle, leads to significant alterations in carbohydrate metabolism, particularly fluctuations in blood glucose levels and liver glycogen content. In the initial days following intoxication, hyperglycemia occurs due to disruptions in metabolic enzyme function, consistent with other research findings. Subsequently, glucose levels normalize, and glycogen content increases, potentially due to compensatory mechanisms in response to toxic exposure.

These results underscore the necessity of controlling and limiting pesticide use to reduce the risk of metabolic disorders in humans and animals. Additionally, further research is crucial to develop preventive measures and health protection strategies.

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