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Original Research Article

The Toxic Effects of Cucurbitacin in Paddy Melon (*Cucumis Myriocarpus*) on Rats

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ABSTRACT

Untold losses of livestock are caused by various poisonous plant families each year globally, through death, physical malformation, abortion and lowered gain. Such families include; Solanaceae, Apocynaceae, Euphobiaceae and Cucubitaceae, where paddy melon (*Cucumis myriocarpus*) belongs. The main objective of the study was to carry out acute toxicity test of crude cucurbitacin in the ripe fruits of paddy melon (*Cucumis myriocarpus*) and determine its lethal dose (LD_{50}) on laboratory rats. The crude extract of paddy melon was highly lethal, with an LD_{50} of 0.68g/kg body weight.

Key words: Cucumis myriocarpus, Toxicity, rats, LD_{50}.

INTRODUCTION

Plant poisonings cause about 10-25 % livestock losses due to lack of knowledge on the chemical composition, medicinal and toxic effects of many pasture plants. Solanaceae family like *Daturastramonium* contain Atropine toxins which exert an antimuscarine effect blocking transmission of autonomic impulses at ganglia and neuromuscular junctions (Kurzbaum*et al*., 2001). Those of Apocynaceae like *Acokanthera* spp. contain toxic cardiac glycosides, such as quabain, that affect the cardiovascular system causing bradycardia, heart block; poison the respiratory and nervous systems; and the Gastro-Intestinal Tract (GIT). Toxic materials in the latex of Euphobiaceae irritate the eyes and mucous membrane and injure the integument. Oxalic acid in oxalics damages kidneys. Many losses occur with irregularities due to unpredictable conditions but some can be avoided by good management such as spraying the pasture with herbicides (Ashton, *et al*., 1991). Plants that contain parasympatholytic alkaloids, atropine, hyascine and hyoscyanine exert an antimuscarinic effect causing neurological disorders without pathological lesions (Matthews and Endress, 2004). Neurological disorders with distinct pathological lesions are caused by plants which produce mycotoxins that cause muscle tremors, greyish-white areas of hyaline degeneration and necrosis particularly near the insertions and origins of the larger muscle groupsCAB, 1987-1989; 1990-1991). Paralysis occurs in animals that survive. Plants that poison the GIT induce signs other than diarrhoea, cause diarrhoea and bloat, impaction or gastroenteritis due to mechanical injury. Death may occur due to asphyxia, a result of chocking on inhaled ingest a, or paralysis of the respiratory centre, exhaustion from vormition and purgation, heart failure and acute or chronic foreign body pneumonia (CAB, 1987-1989; 1990-1991).

Objectives

The objectives of the study were;

1. To assess the symptoms and side effects of crude cucurbitacin extract on the gastro-intestinal tract, lungs and liver of laboratory rats
2. To determine the lethal dose (LD_{50}) of crude Cucurbitacin extract

RESEARCH HYPOTHESIS

There will be no significant association between the concentration of crude cucurbitacin extract administered and the number of rats that die.

RESEARCH METHODOLOGY

Breeding of rats

Rats were bred and crude cucurbitacin administered at The Kenya Polytechnic (now, The Technical University of Kenya) animal house. One male rat was confined in a cage with three females and left to mate freely. After confirming that conception had taken place, the pregnant rats were separated and each put in its own cage. After parturition, the young ones were reared for two months before carrying out the tests.

Sampling of fruits and Extraction

About 95% ripe fruits ofPaddy melon (*Cucumis myriocarpus*) were sampled randomly from plants growing on free land fields and hedges at Ngecha village in Kiambu County where livestock graze freely. Extraction of crude cucurbitacin from the fruits was done at National Agricultural Research Laboratories (NARL). Before extraction, the fruits were thoroughly washed and rinsed using tap water. The fruits were then cut into small pieces, mashed and their weight taken. Fruits were then mixed with clean tap water in a big pan (sufuria) at a ratio of 1:2 (w/v). Normal boiling was done using an electric cooker for 30 minutes. The crude extract obtained after boiling was cooled using a cold water bath, filtered using a clean fine woven cloth, kept under refrigeration and administered to the test rats the following day.

Determination of Cucurbitacin concentration

After taking the weight of 10 empty Petri-dishes, a sample of 20ml of the crude cucurbitacin was measured using a 20 ml. pipette and poured in each of the dishes. The dishes were heated in an oven at 44^{o}c for 3 days to evaporate water and leave the dry crude cucurbitacin compound. The petri-dishes containing the crude compound were weighed and the average weight of the crude cucurbitacin in the sample extract calculated. The weight of dried crude cucurbitacin in the 20 ml. sample was used to calculate the weight of crude cucurbitacin in the volume of crude cucurbitacin extract given to the rats in each treatment. Weights of test rats were taken, recorded and the average weight calculated. The concentration of the crude cucurbitacin administered to the rats in each treatment was calculated using the average weight of the test rats and the crude cucurbitacin in the volume of the extract administered orally to the rats. Single doses of varying concentrations of crude cucurbitacin extract were administered to six groups of rats and a placebo (distilled water) administered to the control.

Treatment administration

The design of the experiment was a Randomised Complete Block Design (RCBD). The 7 treatments indicated in Table 1 were replicated 3 times. Each treatment had ten (10) rats. Each test rat was placed in a desicator containing diethyl ether to sleep and a catheter pushed through the mouth down the gullet. The indicated dose was then administered through the inserted catheter using a syringe. This was repeated for all the rats in all treatments. Rats were left to recover in their cages after which observations started immediately. Symptoms of toxicity were observed immediately after administration, and for a period of 2 weeks. Rats from the control, the ones that died and those that survived were dissected and macroscopic observations made on the Gastrointestinal Tract (GIT), lungs, and the liver.

Table 1: Treatments of crude cucurbitacin administered to test rats

Treatment number	1	2	3	4	5	6	7
Concentration (g/kg body weight)	(Control) 0	0.22	0.47	0.68	0.94	1.15	1.17

Statistical Analysis

Data was entered in SPSS version 18.0 then subjected to analysis of variance (ANOVA) to compare effects of the factors being studied. Significant results were further analyzed to check where the differences occurred using Fisher's least significant difference (LSD) at 5% level

RESULTS

There were very high significant differences ($p < 0.001$) between the number of live and dead rats for concentrations 1.15 and 0.47g/kg body weight. However, dead rats for concentration 1.15g/kg were significantly ($p<0.001$) more than live ones while live rats for 0.47g/kg were significantly ($p < 0.05$) more than dead ones. Concentration 1.17g/kg body weight killed all the rats (Fig. 1; Fig. 2).

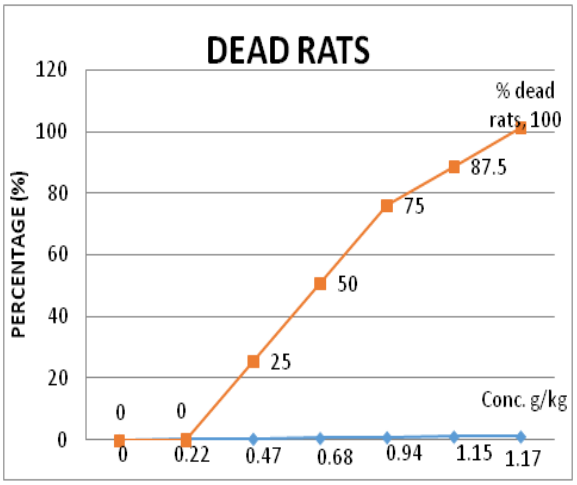

Fig.1: Dead rats versus crude cucurbitacin concentration p = 0.05

Poisoning symptoms

Symptoms of poisoning were observed, severity increasing with concentration of crude cucurbitacin. Rats given concentration 0.22g/kg and 0.47g/kg took a sitting posture immediately after administration, breathed fast, moved slowly and were dull on 1st and 2nd days. Most rats began recuperating on the 3rd day, with full recovery on the 4th and 5th day respectively. Symptoms were however, the same for higher concentrations 0.68g/kg, 0.94g/kg, 1.15g/kg and 1.17g/kg, but severity increased with concentration of Cucurbitacin. Rats were seen lying down immediately after administration (instead of sitting like in the lower doses). Rats given concentration of 0.94g/kg, 1.15g/kg and 1.17g/kg started rolling, breathed very fast, were unable to move and eat, and looked very miserable for most days. Severity of poisoning was reflected in high death rate of 50% within 6 hours for concentrations 1.15 and 1.17g/kg, and 25% within 12 hours for concentration 0.94 g/kg. Recovery time for survived rats increased with concentration of cucurbitacin.

Macroscopic observations

Rats dissected from the control and the lowest concentration 0.22g/kg showed a normal stomach, intestines, lungs and liver. Dead rats dissected from higher concentrations 0.47g/kg to 1.17g/kg had acute inflammation of intestines and the stomach that were poorly vascularized as compared to rats from the control. Lungs were swollen and had severe haemorrhage and accumulation of blood whose severity increased with concentration of cucurbitacin. Livers were swollen/ enlarged and were harder than the normal ones. Rats that survived from concentrations 0.47g/kg to 1.15g/kg showed intestines whose blood vessels had diminished and intense congestion was observed over large areas of the small intestines. Some lungs were degenerated and had haemorrhages while others had hard enlarged lobes. Enlarged livers appeared darker and degenerated.

Lethal dose (LD_{50})

The lethal dose (LD_{50}) is the concentration that kills 50% of the test animals during the time of study. Results (Figs. 1 and 2) in this study indicated that the LD_{50} of crude cucurbitacin was 0.68g/kg body weight.

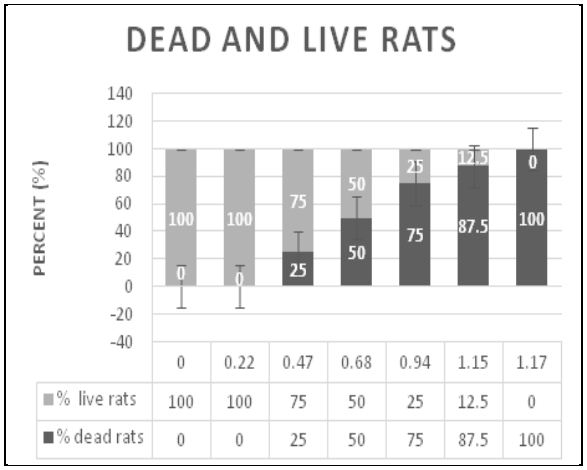

Fig.2: Dead and live rats versus crude cucurbitacin concentration

DISCUSSION

All rats (except in the control) showed signs of toxicity after ingesting crude cucurbitacin extract. Dullness, breathing fast and abhorrence of food (anorexia) due to lack of appetite observed in the test rats were signs of poisoning from the crude extracts of *Cucumis myriocarpus* (Paddy melon). In addition, slow movement observed in rats given lower doses (0.22 and 0.47g/kg) and complete inability to move for rats given higher doses (0.68 to 1.15g/kg) were signs of toxicity that caused a defect in muscle control and subsequent irregularity of movement. Nevertheless, the dog sitting posture, a sign of abdominal/ stomach pain, became severe causing rats to lie down and even roll on their stomachs. However, toxicity increased with concentration of the extract (Fig. 1; Fig. 2). This was in agreement with Van Wyk *et al*. (2002) whose research revealed that *Cucumis myriocarpus* (paddy melon) contained a toxin, cucurbitacin, which caused poisoning to the GIT, liver, lungs and the heart. Irritants induce production of Phospholipase A2 enzyme which release arachidonate (from membrane phospholipids) which is metabolize to prostaglandins and leucotrienes causing pain and acute inflammation (*Foegh and Ramwell, 2001). Test rats experienced severe stomach pains and had inflammations* in the intestines, an indication that Cucurbitacin is an irritant toxin to the GIT. Inflammation is a frequent local response to irritant substances. Severe haemorrhage and accumulation of blood observed in lungs and intestines indicated that cucurbitacin was a haemorrhagic toxin with more affinity for lungs and intestines. It hydrolyzed the intracellular sement substances of the small blood vessels, causing haemorrhages. The liver being the main organ for detoxification, cucurbitacin and its metabolites accumulated, inactivated or destroyed liver tissues and enzymes, causing it to swell.

CONCLUSION

The toxic effects of crude cucurbitacin increased with concentration, causing death due to acute inflammation and congestion in the intestines, swelling of the liver and severe haemorrhages with congestion in the lungs. It was highly lethal, with an LD_{50} of 0.68g/kg body weight. The weed, Paddy Melon, should therefore, be removed from the pasture to prevent poisoning of livestock.

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BIOGRAPHY

Violet Nakhungu Momanyi received a Diploma and Higher National Diploma in Applied Biology from The Kenya Polytechnic; a Master's degree in Public Health and Community Development (Epidemiology and Population Health) from Maseno University, and is currently pursuing a PhD in Environmental and Population Health at Kenyatta University, Kenya.

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