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Acute Toxicity of Castor Oil Bean Extract and Tolerance Level of Raw Castor Oil Bean (*Ricinus communis* L) By Broilers

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Target Audience: Ingredient processors, Feed millers, Poultry farmers, Nutritionists, Animal Scientists.

Abstract

The experiment was carried out to determine the acute toxicity of raw castor oil bean (Ricinus communis) extract and the tolerance level of raw castor oil bean by broilers. The seeds were ground, defatted with petroleum ether and the residue was subjected to extraction with phosphate-buffered saline. The extract volume equivalents of six doses (0 g/kg, 4 g/kg, 9 g/kg, 14 g/kg, 19 g/kg, and 24 g/kg) were determined and then given by oral drenching to thirty 6-week-old broilers, with liveweight of 500-600 g. The doses were designated treatments 1, 2, 3, 4, 5 and 6, respectively. There were 5 birds per treatment. The drenched birds were left in their respective pens with full access to feed and water and observed for 48 hours. Few minutes after the administration of the extracts, dizziness was observed, followed by diarrhoea. The intensity increased with increase in the doses. At the end of 48 hours, the total percent mortality was 60% for treatment 2, and 100% for treatments 3, 4, 5 and 6. In conclusion, Ricinus communis is acutely toxic, especially when defatted; and the tolerance level of broiler birds for raw Ricinus communis is about 4000 parts per million or 4 g of seed per kg body weight.

Keywords: Raw castor oil bean, acute toxicity, lethal dose, tolerance level, broiler.

Description of problem

Unconventional ingredients such as castor oil bean has been shown to have some amount of nutrients and good chemical composition and may serve as a protein source to animals (1, 2). However, the presence of anti-nutritional factors and toxic substances which may equally be proteins poses a serious limitation on its utilization (3). Such constituent proteins are potent poisons and highly toxic to animals and even to man, especially when improperly processed (4). The anti-nutritional factors and toxins have adverse effect on the physiological functions of the animal system. The presence of these toxins and

anti-nutritional factors inhibits protein synthesis, and this has been the major limitation to its effective use in animal production in high quantity (5, 6). The principal toxin of castor bean is ricin. Others include lectins, *Ricinus communis* agglutinin, hydrogen cyanide and oxalates, ricinine and castor allergen. Typically, 2.5-20 raw seeds can kill an adult human; 4 a rabbit, 5 a sheep, 6 an ox, 6 a horse, 7 a dog, but 80 for cocks and ducks. As little as one castor oil bean, about 0.5 grams, may be fatal to a child (7). Its incorporation in rations of animals has posed a great challenge to most researchers and producers.

There is an emerging situation in Cross River State of Nigeria, where the State Government has gone into a project of producing castor oil seed for its oil. The residue after oil extraction (castor oil cake/meal) is being advocated for use as livestock/poultry feed ingredient. There is, therefore, the need to evaluate this product for its safe use in livestock/poultry feeding. The aim of this research was to determine the severity of the toxic effect of defatted castor oil bean cake/meal on broilers and thereby establish a tolerance level at which the defatted raw seed may be included in poultry diets without lethal consequence.

Materials and methods

The experiment was carried out in the Poultry Unit of the Teaching and Research Farms of Michael Okpara University of Agriculture, Umudike. Thirty 6-week-old Anak broiler chicks ranging from 500 to 600 g body weight were used to study the effect of defatted raw *Ricinus communis* extract. The seeds were obtained from Cross River State of Nigeria. The birds were raised on deep litter system of management. They were given commercial broiler diet *ad libitum* and water was offered without restriction. Routine medications and vaccinations were carried out.

A known quantity of finely ground sample of *Ricinus communis* was defatted using petroleum spirit (40-60^{o}C) in a Soxhlet extraction apparatus for about 2 hours. The resulting defatted residue was then used in preparing the extract. A known volume of universal solvent, phosphate-buffered saline of pH 7.0 was added to the known quantity of the fat-free residue and stirred very well. The set-up was allowed to stand for 1 hour, but with stirring for 2 minutes at interval of ten minutes. At the end, the solvent was strained out using a clean muslin cloth. The filtrate was used as the extract in accordance with the procedure of (4). Also, determination of seed equivalent and volumes of the extract to be given to each bird was according to the procedure of (4), which is as follows:

(a) Seq = Vex/Wus
Where:
Seq = Seed equivalent of extract
Vex = Volume of extract;
Wus = Weight of undefatted seed

(b) Vad = Seq x Dosage
Where:
Vad = Volume of extract to be administered

Dosage = calculated based on recommended dose, e.g. given a dose of 14 g/kg (i.e. weight of seed per body weight), dosage for 0.5 kg bird will be (14/1)*0.5 = 7 g.

Thereafter, extract volume equivalents (Vad) of six doses of 0 g/kg, 4 g/kg, 9 g/kg, 14 g/kg, 19 g/kg, and 24 g/kg were administered by oral drenching to thirty 6-week-old broiler chicks averaging 500-600 g at 5 birds per dose (or treatment) (Table 1). The oral drenching was achieved by the use of force-feeding technique as described by Sibbald (8). The drenched birds were left in their respective pens with access to feed and water, and observed for 48 hours.

Table 1: Volumes of *Ricinus communis* extract given to broiler birds according to their body weights and dosage

Repl	Control		4000mg		9000mg		14000mg		19000mg		24000mg	
	BdWt	ExtV	BdWt	ExtV	BdWt	ExtV	BdWt	ExtV	BdWt	ExtV	BdWt	ExtV
1	500	-	575	4.4	575	9.8	530	14.1	527	18.9	500	22.6
2	600	-	550	4.2	500	8.5	575	15.2	600	21.5	575	26.0
3	525	-	500	3.8	525	9.0	600	15.9	550	19.7	525	23.8
4	500	-	500	3.8	600	10.2	500	13.2	525	18.9	575	26.0
5	525	-	500	3.8	500	8.5	525	13.9	500	18.0	600	27.2

BdWt – Body weight; ExtV – Extract volume; Repl – Replicate mg – milligramme.

Results and discussion

After the administration of the castor oil bean extract, some general effects were observed on the chicks. There was an immediate observation of dizziness in all the chicks that received the extracts, except the control (0 g/kg). The birds stood with droopy heads and closed eyes and staggered on trying to walk. The dizziness effect was more pronounced with the higher dosage groups. Dizziness as observed in the birds could have been as a result of the presence of ricin found in castor oil bean. Ricin, as stated by (9, 10), is one of the major principles or toxins in castor oil bean. It is extremely toxic when introduced into the nervous system and is transported into the nerve cell bodies by axons (11, 12), and it is evident that ricin can alter "blood-nerve barrier" (13, 12). Symptoms of ricin toxicity may include weakness, tachycardia, muscle cramps, dysponea and lethargy (14). Richard and Anthony (15) had noted that experimental evidence exists for a central nervous system mechanism for ricin toxicity. Clinically, there is substantial distribution of ricin to muscle tissues after administration regardless of the route (16), which is responsible for muscle cramps and weakness commonly experienced in animals (17, 18). Hydrogen cyanide, another toxin in

castor oil bean (19, 20), may have contributed to the dizziness effect. Exposure to hydrogen cyanide can cause unconsciousness, weakness, vertigo as well as severe nose irritation (21, 22, 23).

All the birds in all the dosage groups other than the control (0 g/kg) had diarrhoea. Again, the intensity of the diarrhoea increased with increase in doses. This suggests that there were increased motility and irritations of the gastro-intestinal tract, which may be responsible for the diarrhoea. Franz and Jaax (24) had reported that serious cases of castor oil bean ingestion resulted in nausea, vomiting, and abdominal pains. Oxalate is another anti–nutritional factor in castor oil bean. Its poisoning includes oral irritation when ingested (25).

Another general observation was in response of the chicks to feed and water offered to them after the administration. The birds that received the raw castor oil bean extract tended to stay off or consume less feed and water. Again, the high-dose (14 g/kg to 24 g/kg) treatment groups exhibited this effect more than the low-dose (4 g/kg and 9 g/kg) treatment groups. The motility and irritation of the gastro-intestinal tract could impair the feed consumption. It has also been noted earlier that the identified toxins in castor oil bean produce weakness and feeling of vertigo when ingested.

Sequential occurrence of death started within the first 12 hours after administration of the extract (Table 2). All the birds in treatment 4 (14 g/kg), 5 (19 g/kg) and 6 (24 g/kg), which were the high-dose treatments died within 12 hours (that is 100% mortality). Other birds in treatments 2 (4 g/kg) and 3 (9 g/kg) had 40% and 80% mortality, respectively within the first 12 hours after administration. Forty-eight hours after the administration, the remaining bird in treatment 3 (9 g/kg) died, while in treatment 2 (4 g/kg) only one more bird died leaving two birds alive. No death was recorded in treatment 1 (0 g/kg), which was the control group. The death presentation in each case was due to seizure, and it was followed by death.

Table 2: Death pattern of 3-week old broilers after the administration of raw castor bean extract

	Treatment					
Time (h)	0 g/kg	4 g/kg	9 g/kg	14 g/kg	19 g/kg	24 g/kg
0	0	0	0	0	0	0
12	0	2	4	5	5	5
24	0	-	-	-	-	-
36	0	-	-	-	-	-
48	0	1	1	-	-	-
Total	0	3	5	5	5	5
% Mortality	0	60	100	100	100	100

The presence of ricin as the most potent toxin in castor oil bean may have been the cause of the death. According to (26) and (27), a single molecule of ricin in a cell can cause the cell to die. In fact, they further noted that the intoxication of ricin may induce "programmed cell death" (apoptosis) in pulmonary endothelial cells and in lymphatic tissues of poisoned rats (24). In humans, evidence of cardiac injury with ricin includes a case of heart block (18). All these point to the fact that the lethal target tissue was probably the central nervous system. Ricin toxicity is less potent when administered orally as compared with other routes due to poor absorption in the body (18, 24). Also, onset of symptoms can range from hours to several days (14). The results of the present study showed that the onset of symptoms and mortality was earlier than previously reported. According to (28), a single lethal dose of the seeds (g) per kg body weight for birds is 14 g/kg and the latent period between ingestion of the seed and the onset of symptom is about 48 hours. The observed difference in this study may be associated with absence of attenuation effect because the seed was defatted and the residue extracted leaving only the filtrate, which was used. Oil is an anti-oxidant, which is capable of exerting an attenuation effect on the toxins (29, 30, 31). Therefore, defatting of the bean contributed to the hastened death of the birds. Castor oil bean contains a lot of other anti-nutritional factors and toxins which are equally deadly, though contained in smaller amounts (1, 5, 6, 19, 32, 33, 34). It is possible that these factors exerted synergistic effects on the birds. The difference may also be due to climatic and edaphic factors, which have been shown to affect composition and concentration of toxins and anti-nutritional factors in the castor oil bean (35, 36).

At 48 hours (and even weeks) after administration, the treatment group that received 4 g/kg had 60% mortality (Table 2 and Figure 1). This shows that the single lethal dose of the raw *Ricinus communis* seeds in g/kg falls below 4 g/kg of broiler. The reported single lethal dose of the seed in g/kg of hen is 14.0 kg of the seed (28). The lower dose reported in this experiment could also be explained by the absence of attenuation effect of oil as observed earlier. Also, species effect could be another reason, since it is broiler, and not hen, that was used in this study. From the above results, it can be concluded that the tolerance of broiler chicks to defatted castor oil bean is about 4,000 parts per million.

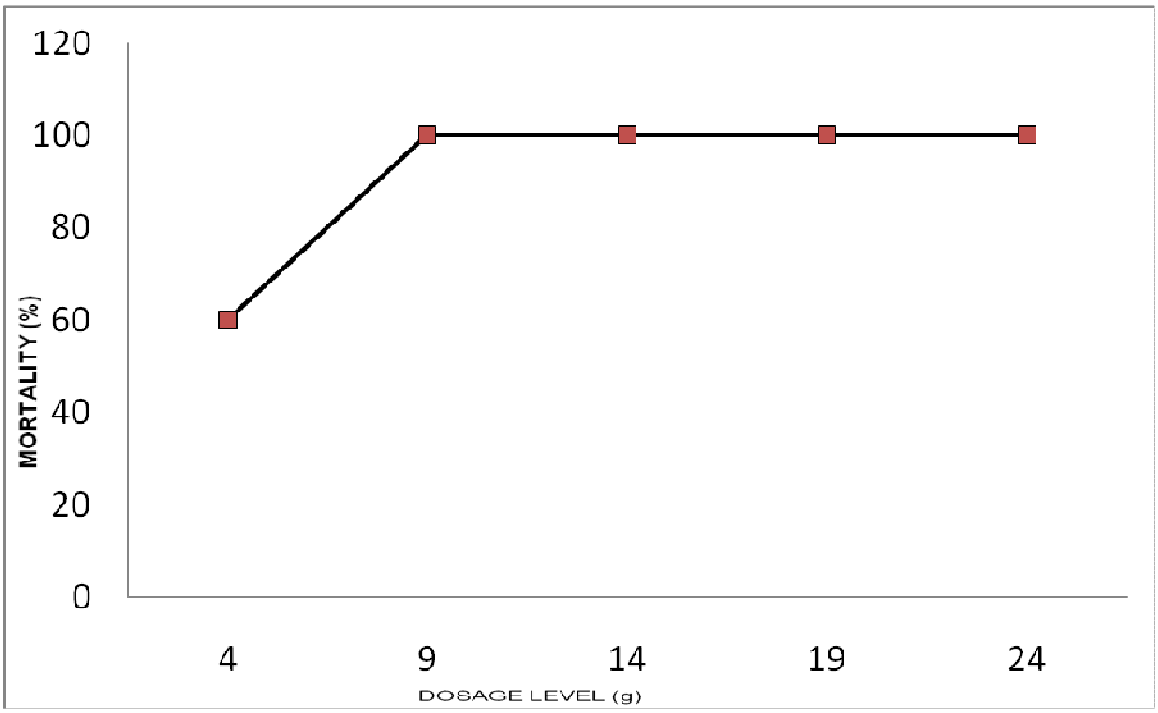

Figure 1: Mortality (%) against Dosage levels (g) of raw castor bean extract administered to 3-week old broilers.

Conclusion and Applications

1. The result of the experiment showed that the single lethal dose of the defatted raw *Ricinus communis* seeds in g/kg after 48 hours of oral administration was below 4 g/kg. This indicates that raw *Ricinus communis*, after oil extraction, is acutely toxic even at 4 g of seed per kg of body weight of chicks when administered orally. Hence, birds can rarely tolerate it at levels above 4,000 ppm.

2 An implication of the above is that oil extraction does not make castor oil bean residue less toxic than the non-defatted seed, rather it increases its toxicity. This is possible because of the absence of the attenuating effect of the extracted oil. Also, the extraction of the oil leads to concentration of its constituent toxin and anti-nutritional factors, which synergize to bring about quicker and greater toxic effects.

3. For effective and optimum use of defatted castor oil bean (*Ricinus communis*), it is recommended that: further experiments be carried out at levels below 4 g/kg body weight to determine the actual single lethal dose and tolerance level of castor oil bean

meal. Also, various ways of processing the bean in order to remove the toxins and anti-nutritional factors, which make it acutely toxic and limits its use in animal feed as protein source should be assessed.

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