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Distribution and Elimination of Lindane in Goats

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Man is often exposed to residues of chlorinated insecticides when consuming products from animals or plants previously treated with these insecticides. Such residues may be unsafe even if the daily intake is low, as they accumulate in the body and may induce changes in biotransformation of endogenous and exogenous compounds (Buck et al. 1976). There is therefore a need to ensure that food products are free from insecticide residues. The present study centres on the distribution and elimination of lindane (an organochlorine insecticide) in the goat, which is a source of meat and milk in Tanzania.

MATERIALS AND METHODS

Nine adult female goats with body weight between 19 and 30 kg were used in the study. Eight of the goats were in milk with an average yield over the observation period of 120 mL per day. These 8 goats were administered lindane while the remaining one served as a control.

The goats were confined in a pen where hay and water were supplied ad libitum. The goats were not dipped in any acaricide for at least one week before the commencement of the experiment and during the whole experimental period. Lindane was given in peanut oil by stomach tube at the same time daily at the dose of 6 mg/kg body weight orally for five consecutive days.

Blood and milk sampling was done prior to drug administration and on day 1,3,5,7 for all goats, and where applicable on day 10,15, 20,30,45 and 60 post-exposure. A pair of goats was killed on each of days 7,15,30 and 60 post-exposure and liver, kidney, skeletal muscle (longissimus dorsi), fat and brain samples collected. Similar tissues were collected from the control. All samples were stored at -20°C before lindane was extracted. The method used for extraction of lindane from blood was that of Dale et al. (1970) while for milk that of Norèn and Westöö (1968) was applied.

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The extraction of lindane from tissues was done using the method of Kraul (personal communication 1983). A known weight of each tissue in homogenised form was thoroughly mixed with activated florisil and eluted in a glass column (2.2 x 40 cm) using 6% ether in petroleum ether at a speed of less than 5 mL/min. The eluates from the fat samples were evaporated to dryness and the residues cleaned up as described by Norèn and Westöö (1968). All extracts were filtered through silicone filter paper.

A Varian 1400 gas chromatograph equipped with a ^{3}H- electron capture detector was used for quantification of lindane. The all-glass column (6' x 1/8") was packed with 4% SE-96 as the liquid phase coated on the solid support chromasorb - W, aw, DMSC. The carrier gas was nitrogen and the temperature of the column was around 180°C. The detection limit for lindane was 0.5 µg/kg (ppb).

RESULTS AND DISCUSSION

There was no clinical evidence of toxicity shown by the goats during the experimental period. This was expected as the dose administered was lower than the minimum toxic dose of 25 mg/kg in cattle, sheep and presumably goats (Buck et al. 1976).

The lindane levels in blood were approximately 0.1 ppm during the dosing period, but soon after the last dose they fell rapidly (table 1). Between day 10 and 20 the average levels were very low and after day 20 lindane was no longer detected in the blood.

On average the levels in milk were much higher than those in the blood; however the rate with which the milk levels declined between day 5 and 10 was similar to that of blood during the same period. The amount of lindane excreted through milk per goat per day in the dosing period was about 0.1 mg and decreased rapidly thereafter (table 1).

This rapid fall in lindane levels in both blood and milk is in accordance with the fact that lindane is generally eliminated rapidly in mammals (Ulman 1972). Mice, for example, given an oral dose of 5 mg/kg eliminate about 80% of the absorbed lindane in 3 - 6 hours (WHO 1972). However, some species variation must exist as in a similar experiment on rabbits, Mosha (1984) found lindane to be eliminated much more slowly from blood as well as tissues than in goats (table 2).

The lindane levels in fat on day 7 averaged 1.4 ppm; the rest of the tissue samples including fat collected after day 7 had levels below 0.1 ppm. That much more lindane was found in fat than in other tissues is probably due to the lipophilic nature of lindane (Ulman 1972). A similar distribution has been recorded in rabbits (table 2, Mosha 1984), cattle (McParland et al. 1973), dogs and cats (Ulman 1972) and buffaloes, goats and chicken (Kaphalia and Seth 1981).

Table 1. Lindane levels in blood and milk (ppb) from goats treated with 6 mg/kg for 5 days (mean ± S.E.M.)

	Days after initial dosing										
	0	1	3	5	7	10	15	20	30	45	60
Blood	n.d.	117 ± 18	112 ± 16	119 ± 16	9.7 ±4.8	2.1 ±0.7	1.1 ±0.5	0.5 ±0.3	n.d.	n.d.	n.d.
Milk	n.d.	941 ±129	958 ±113	883 ±142	72 ±25	38 ±22	17 ±13	3.4 ±1.9	1.0 ±0.6	n.d.	n.d.
No. of goats	9	8	8	8	8	6	6	4	4	2	2

n.d. = not detectable

Table 2. Lindane levels in blood and tissues (ppb) from rabbits treated with 4 mg/kg for 5 days

	Days after initial dosing								
	0	1	3	5	7	10	15	20	30
Blood (mean ± S.E.M.)	n.d.	36.1 ±4.1	92.6 ±8.2	112 ± 5	68.5 ±6.1	35.6 ±4.7	33.5 ±3.5	28.2	11.1
Liver	n.d.	-	-	-	875	-	175	-	190
Muscle	n.d.	-	-	-	470	-	105	-	100
Fat	n.d.	-	-	-	42,000	-	15,000	-	5,750
No. of rabbits	7	6	6	6	6	4	4	2	2

n.d. = not detectable
\- = tissue not collected

The levels of lindane in tissues were generally low. However male rabbits similarly dosed with lindane at 4 mg/kg and killed on coresponding days showed much higher tissue levels than goats (table 2, Mosha 1984). The difference and the faster decline of lindane in blood of goats are not sufficiently explained by mammary excretion since for a total of 720 mg given to a goat of the average weight of 24 kg over a period of 5 days less than 1.0 mg was excreted in the milk. It appears that a higher metabolic rate in goats is the most likely explanation. Variation in accumulation of the same chlorinated pesticide among different species of domestic animals has previously been demonstrated by Kaphalia and Seth (1981) and Hashemy-Tonkabony et al. (1981).

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