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The susceptibility of *Tatera indica*, *Nesokia indica* and *Bandicota bengalensis* to three anticoagulant rodenticides

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SUMMARY

Three South-Asian rodent pest species were tested for susceptibility to anticoagulant rodenticides. Wheat flour containing 0·025% warfarin, 0·0375% coumatetralyl or 0·005% difenacoum was fed to 260 *Tatera indica*, 140 *Nesokia indica* and 81 *Bandicota bengalensis* for 1–56 days. *Tatera* was about as susceptible to anticoagulants as *Rattus rattus* has been reported to be. *Nesokia* and *Bandicota* were extremely variable: though the majority were highly susceptible, the slopes of the dose-mortality curves were close to zero. The difenacoum diet appeared to be more toxic than the warfarin diet to all three species, but less toxic than the coumatetralyl diet to *Tatera* and *Nesokia*. All of the anticoagulants were eventually lethal to all of the animals tested.

INTRODUCTION

The literature on rodent control is full of statements as to the seriousness of the rodent problem in South Asia (see, for example, Patnaik, 1969). Three of the common pest species are the Lesser Bandicoot rat (*Bandicota bengalensis*, Gray), the Short-tailed Bandicoot rat (*Nesokia indica* (Gray), and the Indian gerbil (*Tatera indica*, Hardwicke). *B. bengalensis* is a serious agricultural and commensal pest whose range extends eastwards from Pakistan to Indonesia. The closely related *N. indica* is more specialized than *Bandicota* for a fossorial existence and is an agricultural and occasional commensal pest through much of Northern India, Turkestan, Pakistan and westwards to Egypt. *T. indica* is widely distributed as an agricultural and occasional commensal pest tending to favour the more arid tracts of India and Pakistan, and westwards to Syria and Iraq.

Despite the undisputed pest status of these rodents, very little has been published on their susceptibility to rodenticides; indeed, for *Nesokia* we have been unable to trace a reference to a toxicity test of any kind. Information on the toxicity of

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rodenticides to the pest species is part of the logical basis for deciding which toxicant to use in rodent control programmes and for establishing an appropriate method of use. Baseline rodenticide toxicity data for normally susceptible rodent populations are also useful in establishing whether failures of field treatments can be attributed to the development of resistance to the rodenticide (WHO, 1970). We decided therefore, as a first step to conduct the tests described below with anticoagulant rodenticides.

METHODS

Tatera indica were trapped in the Thole Produce Yard, Karachi, a major transit area for export shipments of rice. *Nesokia indica* and *Bandicota bengalensis* were dug out and caught by hand from burrows in rice fields in Lower Sind. Voucher specimens of the three species tested are deposited in the taxonomic collections at The Vertebrate Pest Control Centre, Karachi, and the British Museum (Natural History), London. None of the populations sampled had previously been exposed to anticoagulant rodenticides. The animals were caged singly and supplied *ad libitum* with water and a laboratory animal diet formulated by Lever Brothers (Pakistan) Ltd. After a minimum of 3 weeks, during which sick, injured and pregnant specimens were excluded, the animals were weighed and the laboratory diet was replaced with a local grade of wheat flour (known as *atta*) for a few days, until feeding had stabilized. Next, the animals were allowed to feed freely on wheat flour containing the rodenticide for a fixed number of days, and were then returned to the laboratory diet for 21 days, during which mortality was recorded. Finally, the laboratory diet was replaced with the rodenticidal bait again, until all the surviving animals died. Consumption of the rodenticidal bait was measured, usually each day, and animals that died were autopsied to confirm the presence of signs of anticoagulant poisoning.

The rodenticides tested were warfarin (3-(α-acetonylbenzyl)-4-hydroxycoumarin), coumatetralyl (4-hydroxy-3-(1,2,3,4-tetrahydro-1-naphthyl) coumarin) and difenacoum (3-(3-*p*-diphenyl-1,2,3,4-tetrahydronaphth-1-yl)-4-hydroxycoumarin). Warfarin was obtained as a 0·5% commercial premix in a World Health Organization test kit (WHO, 1970) and coumatetralyl as a 0·75% commercial premix manufactured by Farbenfabriken Bayer AG, Leverkusen, West Germany. The premixes were blended with wheat flour in a 1:19 ratio to give finished baits containing either 0·025% warfarin or 0·0375% coumatetralyl. Difenacoum was supplied by Ward, Blenkinsop and Co. Ltd., Widnes, England; the technical compound was dissolved in acetone, slurried with wheat flour and the air-dried slurry was mixed with wheat flour at 1:19 to give a finished bait containing 0·005% difenacoum. The concentrations of the rodenticides are those normally used in the field.

The test procedure is close to that provisionally recommended by WHO (1970) as a method for determining the susceptibility of rodents to anticoagulant rodenticides. The data were examined in terms of log days feeding and probit mortality by the methods of Finney (1971).

RESULTS

Tatera indica

The results of the feeding experiments with warfarin are summarized in Table 1. The sex difference in mortality is insignificant. Analysis of the combined data indicated the median lethal feeding period (LFP 50) and 95 % fiducial limits to be 5·8 days (4·8–6·8 days). The LFP 95 and 95 % limits are 13·5 days (10·6–20·7 days). The slope of the probit regression line is 4·4 ± S.E. 0·7.

The coumatetralyl diet (Table 2) appeared to be more toxic, producing a complete kill of animals given a 6-day feeding period, a result not equalled by the warfarin diet with a 14-day feeding period. A pronounced sex difference in mortality was evident with coumatetralyl. Females were more susceptible than males, the LFP 50's and 95 % limits being 1·7 days (0·9–2·5 days) for females and 3·6 days (2·7–4·9 days) for males. The respective LFP 95's and limits were for females, 6·4 days (3·9–29·0 days) and for males, 8·0 days (5·7–21.5 days).

The difenacoum diet was intermediate between the warfarin and coumatetralyl diets in the mortality produced by a 1-day exposure and in the duration of unrestricted feeding required to produce 100 % mortality (Table 6).

Nesokia indica

The results of the tests with warfarin and coumatetralyl, summarized in Tables 3 and 4, are characterized, firstly, by the high mortality that occurred after a 1-day exposure (as much as 90 % in males with warfarin), indicating a high mean susceptibility to anticoagulants; secondly, by the uniformly flat slopes of the four probit regression lines (for males and females, each with warfarin and coumatetralyl), usually not differing significantly from zero (this indicates great variability in response). Females given coumatetralyl were a partial exception, but even here the slope was too flat (2·6 ± S.E. 1·1) to give a useful degree of precision in estimating the feeding periods corresponding to particular mortality percentiles. Owing to the variation it is not possible to differentiate between results with the two compounds, except by making the possibly trivial observation that warfarin, though giving consistently greater mortality with the fixed feeding periods, took longer than coumatetralyl to produce 100 % mortality (56 days as against 26 days) with an unrestricted feeding period.

Difenacoum was again intermediate in the mortality resulting from a 1-day exposure and in the time required to produce 100 % mortality on unrestricted feeding (Table 6).

Bandicota bengalensis

Results with the warfarin diet are given in Table 5, and are broadly similar to those obtained with *Nesokia* in that the slope of the probit regression line for males did not differ significantly from zero, while for females it was also very flat (2·8 ± 1·1). Again, substantial mortality occurred after a single day's feeding on the warfarin diet, while complete mortality did not occur until after an exposure of 41 days. The difenacoum diet (Table 6) gave a greater kill than warfarin after

Table 1. *Mortality in* Tatera indica *after feeding on wheat flour containing* 0·025% *warfarin for a fixed number of days or until death*

Feeding period (days)	Sex	Weight, mean ± S.E. (g)	Toxic diet eaten (g), mean ± S.E.		Warfarin consumed (mg/kg), mean ± S.E.		Mortality	Days to death	
			Died	Survived	Died	Survived		Mean	Range
1	M	120·9 ± 14·4	—	8·5 ± 0·9	—	21·2 ± 3·9	0/10	—	—
	F	90·0 ± 7·9	—	7·8 ± 0·4	—	23·6 ± 3·1	0/10	—	—
2	M	140·0 ± 15·4	22·7	15·0 ± 2·0	44·3	32·5 ± 6·2	1/10	4	—
	F	102·8 ± 10·7	—	13·4 ± 2·1	—	38·7 ± 7·7	0/10	—	—
4	M	158·2 ± 10·3	—	24·4 ± 1·8	—	38·8 ± 2·5	0/10	—	—
	F	110·1 ± 12·2	18·8 ± 0·7	21·6 ± 1·9	39·6 ± 6·5	63·0 ± 15·6	2/10	4·0	—
6	M	147·2 ± 12·4	31·0 ± 3·9	33·2 ± 5·2	54·9 ± 5·5	53·3 ± 14·0	8/10	7·3	6–9
	F	121·5 ± 7·9	28·9 ± 3·9	34·7 ± 4·9	62·2 ± 5·9	68·0 ± 8·8	5/10	6·2	2–8
10	M	123·8 ± 11·8	31·3 ± 3·8	89·1 ± 17·1	71·2 ± 7·1	130·4 ± 30·4	8/10	7·0	5–10
	F	96·2 ± 8·9	26·5 ± 3·3	53·2	70·8 ± 7·2	142·2	9/10	7·6	5–11
14	M	125·3 ± 16·0	55·3 ± 11·1	35·3	121·7 ± 34·2	64·0	9/10	8·4	6–12
	F	84·0 ± 8·0	41·5 ± 8·1	—	142·1 ± 36.6	—	10/10	8·7	4–13
Unrestricted	M	175·4 ± 7·8	51·3 ± 6·3	—	78·9 ± 15·6	—	15/15	10·2	5–23
	F	130·5 ± 7·5	39·8 ± 3·1	—	78·4 ± 6·3	—	13/13	13·3	6–46

Table 2. *Mortality in* Tatera indica *after free feeding on wheat flour containing* 0·0375 % *coumatetralyl for a fixed number of days or until death*

Feeding period (days)	Sex	Weight, mean ± S.E. (g)	Toxic diet eaten (g), mean ± S.E.		Coumatetralyl consumed (mg/kg) mean ± S.E.		Mortality	Days to death	
			Died	Survived	Died	Survived		Mean	Range
1	M	191·2 ± 13·3	—	6·8 ± 0·9	—	13·7 ± 2·0	0/10	—	—
	F	155·3 ± 15·0	7·7 ± 2·3	6·5 ± 1·2	22·9 ± 0·7	14·4 ± 1·8	3/10	4·3	3–6
2	M	176·4 ± 10·4	18·1 ± 2·6	14·3 ± 2·9	41·6 ± 2·9	31·2 ± 3·5	2/10	8·0	4–12
	F	139·6 ± 7·5	11·7 ± 3·0	14·2 ± 2·2	32·8 ± 9·8	38·9 ± 5·8	5/10	6·8	2–10
4	M	174·6 ± 13·3	31·0 ± 7·6	29·5 ± 3·4	75·9 ± 15·7	61·2 ± 6·6	3/10	8·3	7–10
	F	145·7 ± 12·8	18·1 ± 1·9	19·9 ± 9·1	55·4 ± 11·3	40·0 ± 17·4	8/10	6·1	2–13
6	M	205·6 ± 14·1	36·4 ± 1·8	—	68·2 ± 4·3	—	10/10	8·7	7–11
	F	90·5 ± 7·5	19·1 ± 2·7	—	86·8 ± 15·0	—	10/10	4·8	2–8
10	M	193·2 ± 12·6	40·4 ± 5·8	—	82·7 ± 14·6	—	10/10	11·2	7–14
	F	118·2 ± 8·6	36·8 ± 3·9	—	128·4 ± 22·9	—	10/10	8·3	5–14
14	M	201·4 ± 16·5	28·4 ± 4·0	—	56·9 ± 9·9	—	10/10	8·5	7–10
	F	127·8 ± 9·5	37·1 ± 6·3	—	123·6 ± 30·4	—	10/10	8·0	5–14
Unrestricted	M	184·5 ± 8·7	44·2 ± 2·9	—	90·3 ± 4·3	—	23/23	9·4	6–15
	F	142·7 ± 6·2	35·2 ± 2·5	—	93·2 ± 8·1	—	17/17	8·9	5–14

Table 3. *Mortality in* Nesokia indica *after free feeding on wheat flour containing* 0·025% *warfarin for a fixed number of days or until death.*

Feeding period (days)	Sex	Weight, mean ± S.E. (g)	Toxic diet eaten (g), mean ± S.E.		Warfarin consumed (mg/kg), mean ± S.E.		Mortality	Days to death	
			Died	Survived	Died	Survived		Mean	Range
1	M	108·2 ± 9·2	8·0 ± 0·9	9·4	19·9 ± 3·2	25·9	9/10	7·9	4–15
	F	116·7 ± 12·4	7·7 ± 0·9	7·1 ± 0·6	15·6 ± 1·2	18·7 ± 1·8	7/10	9·4	5–14
2	M	114·9 ± 11·0	18·3 ± 2·0	—	42·2 ± 5·1	—	10/10	7·8	4–11
	F	112·4 ± 9·1	16·3 ± 1·4	17·4	39·4 ± 4·8	31·7	9/10	7·7	4–10
4	M	113·6 ± 12·8	40·7 ± 3·3	36·8	91·7 ± 8·4	131·4	9/10	8·8	4–14
	F	116·6 ± 3·5	32·8 ± 5·9	38·2 ± 1·7	72·4 ± 13·2	79·5 ± 12·5	8/10	9·8	6–18
	M	85·6 ± 7·4	79·4 ± 29·4	—	241·0 ± 107·0	—	2/2	12·5	7–18
Unrestricted	F	108·3 ± 10·3	147·5 ± 67·7	—	317·0 ± 127·0	—	6/6	20·3	9–56

Table 4. *Mortality in* Nesokia indica *after free feeding on wheat flour containing* 0·375% *coumatetralyl for a fixed number of days or until death*

Feeding period (days)	Sex	Weight, mean ± S.E. (g)	Toxic diet eaten (g), mean ± S.E.		Coumatetralyl consumed (mg/kg), mean ± S.E.		Mortality	Days to death	
			Died	Survived	Died	Survived		Mean	Range
1	M	99·3 ± 13·1	12·9 ± 0·8	6·4 ± 0·2	51·6 ± 4·2	37·0 ± 17·9	8/10	8·3	4–12
	F	122·2 ± 7·8	12·3	8·8 ± 1·0	37·3	27·9 ± 3·4	1/10	9·0	—
2	M	157·7 ± 8·0	22·4 ± 1·0	20·1 ± 1·3	55·6 ± 5·4	47·5 ± 6·5	8/10	11·3	5–17
	F	99·7 ± 2·3	12·9 ± 2·3	15·0 ± 1·1	51·3 ± 10·5	58·3 ± 5·6	3/10	13·3	8–20
4	M	134·0 ± 15·3	48·4 ± 7·5	40·3 ± 6·5	122·8 ± 21·4	127·3 ± 35·2	7/10	6·6	5–8
	F	120·0 ± 12·8	34·4 ± 5·1	34·6 ± 2·9	106·5 ± 17·0	133·5 ± 19·9	6/10	9·7	6–15
Unrestricted	M	146·3 ± 13·0	71·9 ± 9·3	—	190·5 ± 27·3	—	8/8	12·8	4–26
	F	113·3 ± 6·6	81·3 ± 6·5	—	274·2 ± 19·7	—	20/20	12·4	4–25

Table 5. *Mortality in* Bandicota bengalensis *after free feeding on wheat flour containing* 0·025 % *warfarin for a fixed number of days or until death*

Feeding period (days)	Sex	Weight, mean ± S.E. (g)	Toxic diet eaten (g), mean ± S.E.		Warfarin consumed (mg/kg), mean ± S.E.		Mortality	Days to death	
			Died	Survived	Died	Survived		Mean	Range
1	M	215·8 ± 26·5	12·1 ± 1·7	14·7 ± 2·0	15·9 ± 2·8	19·4 ± 4·3	7/10	7·7	3–10
	F	131·6 ± 24·4	9·9 ± 1·7	9·7 ± 1·4	17·7 ± 6·2	22·4 ± 1·8	3/10	9·7	5–13
2	M	250·4 ± 37·5	24·0 ± 3·4	20·3 ± 3·2	27·2 ± 7·6	25·5 ± 4·9	5/10	10·8	10–11
	F	194·1 ± 29·4	28·0 ± 2·2	16·3 ± 2·3	28·6 ± 8·1	27·1 ± 3·4	3/10	9·3	8–10
4	M	204·4 ± 17·8	40·4 ± 5·9	54·9	49·3 ± 7·8	97·3	9/10	9·9	7–12
	F	171·0 ± 18·9	38·1 ± 5·4	45·3	63·1 ± 10·3	87·1	9/10	10·0	7–13
Unrestricted	M	267·6 ± 19·1	117·7 ± 13·5	—	109·8 ± 10·3	—	9/9	12·9	7–20
	F	172·5 ± 13·4	126·4 ± 28·9	—	182·9 ± 37·1	—	15/15	17·5	7–41

Table 6. *Mortality in three species of rodents after free feeding on wheat flour containing* 0·005 % *difenacoum for one day or until death*

Species	Feeding period (days)	Sex	Weight, mean ± S.E. (g)	Toxic diet eaten (g), Mean ± S.E –		Difenacoum consumed (mg/kg), mean ± S.E.		Mortality	Days to death	
				Died	Survived	Died	Survived		Mean	Range
Tatera indica	1	M	173·9 ± 9·4	—	10·7 ± 1·0	—	3·1 ± 0·3	0/10	—	—
		F	151·5 ± 12·3	12·5	10·4 ± 0·7	4·6	3·4 ± 0·4	1/10	20	—
	Unrestricted	M	168·6 ± 8·6	73·1 ± 8·7	—	22·6 ± 3·2	—	10/10	11·3	7–14
		F	147·0 ± 17·4	70·9 ± 5·4	—	28·0 ± 4·5	—	9/9	10·7	7–16
Nesokia indica	1	M	116·9 ± 10·0	8·5 ± 0·5	12·8	4·0 ± 0·5	3·6	9/10	10·0	7–15
		F	113·2 ± 10·3	8·3 ± 0·5	7·6 ± 1·7	4·5 ± 0·8	3·0 ± 0·4	6/10	10·0	4–16
	Unrestricted	M	206·8	199·4	—	48·2	—	1/1	27	—
		F	117·0 ± 16·1	87·0 ± 32·6	—	41·0 ± 18·7	—	4/4	13·3	6–27
Bandicota bengalensis	1	M	265·0 ± 23·4	13·3 ± 1·3	6·8	2·5 ± 0·3	1·9	10/11	10·3	7–15
		F	210·2 ± 18·0	10·5 ± 0·7	6·9 ± 2·2	2·6 ± 0·4	1·5 ± 0·5	8/10	13·3	8–20
	Unrestricted	M	156·9	57·6	—	18·4	—	1/1	13·0	—
		F	192·0 ± 6·0	58·8 ± 4·3	—	15·5 ± 0·5	—	2/2	9·0	8–10

a 1-day exposure (18/21 as against 10/20) and produced complete mortality on the 13th day of unrestricted feeding.

DISCUSSION

As far as we know, no data on the toxicity of anticoagulants to *Tatera indica* have been published before. However, a comparison of our results for *Tatera* with those of similar experiments with warfarin against *Rattus rattus* made by Krishnamurthy, Uniyal & Pingale (1968) suggests that warfarin at 0·025% and coumatetralyl at 0·0375% are respectively slightly less toxic and slightly more toxic to *Tatera* than is warfarin at 0·025% to *R. rattus*. It may be expected therefore that these anticoagulants would give similar results when used against either species in the field. Again by analogy with *R. rattus*, it is probable that *Tatera* has the potential to develop a significant degree of resistance to anticoagulants (Greaves, Rennison & Redfern, 1976).

The results with *Nesokia* are remarkable in two respects. Firstly, the high mortality after a 1-day exposure (altogether 40/60) closely approaches that obtained in similar tests with *Rattus norvegicus*, which has the highest susceptibility to anticoagulants of any known rodent. For example, Bentley & Larthe (1959) and Greaves & Ayres (1969) obtained kills of 9/12 and 7/10 respectively in 1-day feeding tests using 0·025% warfarin and 0·05% coumatetralyl. Secondly, the time taken to reach 100% mortality was longer for *Nesokia* than for either *Tatera* or *Bandicota* with all three anticoagulants. The exceptional variation shown by *Nesokia* is illustrated by the near-zero slopes of the probit regression lines. We do not think *Nesokia* is polymorphic in its response to anticoagulants, since the longer-surviving animals were obviously affected early in the tests, intermittently losing and regaining appetite until they finally succumbed; this is unlike the finding in *R. norvegicus* of a polymorphism expressed as a clear discontinuity in tolerance between susceptible and highly resistant segments of the population (Drummond & Wilson, 1968). We judge that in anticoagulant treatments in the field against *Nesokia*, around 90% of the population could be readily controlled, but that with continued use a substantial loss of effectiveness could be expected after a few years.

The striking feature of the warfarin data for *Bandicota*, as for *Nesokia*, is the relatively flat slope of the probit regression lines indicating considerable variation in susceptibility. Three papers by Deoras (1964, 1965, 1967) report upon the susceptibility of *B. bengalensis* to warfarin, and seem to indicate that 100% mortality occurred in more than 100 animals fed with various anticoagulants for 1–12 days, while 59 out of a further 60 animals died when given 0·005% warfarin for 6 days, the mean time to death being 5–6 days. However, parts of these papers are impossible to understand, owing to the omission of important details of methods and results. A further paper (Deoras, Chaturvedi, Vad & Renapurkar, 1972) agrees with the earlier work in showing complete mortality in 20 *Bandicota* fed for 2–8 days on 0·025% warfarin. Against this evidence that *B. bengalensis* is highly susceptible to warfarin, Renapurkar, Menon, Bhat & Sant (1973) report that 7/50 and 20/100 animals survived after feeding on 0·025% warfarin for 6 days and

10 days respectively, indicating a considerably higher tolerance; however, the mean daily warfarin intake of their animals was unduly low (9·8 and 4·1 mg/kg/day in 6 and 20 days respectively against 19·0, 13·5 and 15·0 mg/kg/day in 1, 2 and 4 days with our rats), which suggests that the low kills reported were due to low warfarin dosage rather than to physiological tolerance. Nevertheless, our own results do indicate, by the flatness of the dose–response curve and the survival of animals for up to 41 days of unrestricted feeding, that *Bandicota* is less susceptible to warfarin than suggested by earlier reports.

On the basis of the results reported here it would be expected that anticoagulants, would give useful results against *Bandicota* in the field but that as with *Nesokia* after prolonged use its efficiency would decrease owing to selection for tolerance. It is of interest in this connexion that resistance of *Bandicota bengalensis* (synonym *Gunomys gracilis*) to anticoagulants has been mentioned by Fernando, Kawamoto & Perera (1967), on the basis of a gradual decrease that was noticed in the effectiveness of warfarin against rice-field rats over a period of years on an estate in Sri Lanka (H. E. Fernando, personal communication).

We conclude that anticoagulant rodenticides used at conventional concentrations can play a valuable role in the control of the three species tested. A wider range of anticoagulants needs to be examined and, in particular, the possibility that higher concentrations might be more effective should be studied. The work should also be extended to include other rodent pests, such as various species of the genera *Rattus*, *Mus*, *Millardia*, *Vandeluria*, *Gerbillus* and *Meriones* that, in different areas, may commonly occur together with the species reported upon here. On present evidence it would be incautious to base major long-term programmes for the control of the three species *solely* on the use of anticoagulants, and future work should therefore include studies of other classes of rodenticides. The possibility that, with intensive use, resistance to anticoagulants may eventually develop must be balanced against the problems of poison shyness and acute toxicity hazards that occur with alternative rodenticides. We think that the present advantages of the anticoagulants in safety, efficacy and ease of use weigh heavily in their favour.

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