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SOME EFFECTS OF TICK INFESTATIONS ON JUVENILE NORTHERN BROWN BANDICOOT (*ISOODON MACROURUS*)

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ABSTRACT: The effect of tick infestations on body weight and various blood parameters was monitored in juvenile northern brown bandicoots (*Isoodon macrourus*) after release into tick-infested or tick-free enclosures. Three species of ticks were observed in the enclosures, *Haemaphysalis humerosa*, *Ixodes tasmani* and *Ixodes holocyclus*. Bandicoots released into tick-infested enclosures showed a reduced growth rate (1.8 versus 2.5 g/day increase in body weight), a reduced haematocrit value (27.4 versus 40.0%) and an increased number of white blood cells when compared with bandicoots released into tick-free enclosures. These results suggest that tick infestations may influence the health of juvenile *I. macrourus*.

Key words: Northern brown bandicoot, *Isoodon macrourus*, ticks, blood parameters, captivity, mortality, morbidity, growth rate.

INTRODUCTION

Ixodid ticks are major pests of both domestic and wild animals (Roberts, 1970). Pathogenic effects observed in domestic animals are two-fold: heavy infestations have a serious debilitating effect on the host (Francis, 1960; Little, 1963; Johnston, 1969) and they are also vectors of important haemoprotozoan and rickettsial diseases (Stewart et al., 1982). O'Kelly and Seifert (1970) examined the effect of infestation with *Boophilus microplus* on the blood composition of *Bos taurus* steers and demonstrated that the effects of tick infestation was associated with depressed food intake. They also observed that a toxin secreted by the ticks directly influenced the host's metabolism and that these effects resulted in reductions in haematocrit and haemoglobin values. To our knowledge similar effects have not been observed in native animals.

Several important parasite species including haemoprotozoans have been observed in native animals from Australia (Backhouse and Bollinger, 1957; Collins et al., 1986; Mackerras, 1958, 1959; Priestly, 1915; Weilgama, 1986). Parasites also could affect the well being of native animals in captivity.

The bandicoot is a recognised host for *Haemaphysalis humerosa*, *Ixodes tas-*

mani and *Ixodes holocyclus* (Doube, 1975, 1979; Stewart and de Vos, 1984). Marks and Cribb (1966) have suggested that native mammals and birds, when they first become independent of their parents, are particularly vulnerable to the debilitating effects of tick infestations. Mortality of young marsupials is greatest during the last phase of lactation, a period when their physiological control is not completely developed and when they are changing from a milk to an adult diet (Dunnet, 1964; Thomson and Owen, 1964; Tyndale-Biscoe and Smith, 1969; Tyndale-Biscoe, 1979, 1984). Bandicoots are especially vulnerable to infections and disease immediately after weaning in both the wild (Gordon, 1971; Stoddart and Braithwaite, 1979; Hall, 1983) and in captivity (Gemmell, 1989). In a previous study, 92 female juveniles weighing on average 600 g at 150 days post partum were released into enclosures each 900 m². Only 40% of the juveniles survived (Gemmell, 1989). Although natural predators including the carpet snake (*Python spilotes*) and the goanna (*Varanus varius*) were responsible for some deaths, we believed that other factors were involved in this juvenile mortality. In the present study, the effect of tick infestations were examined to determine whether they

contributed to the high mortality in juvenile *Isodon macrourus*.

MATERIALS AND METHODS

Methods of capture and maintenance of the northern brown bandicoot, have been published previously (Gemmell, 1982, 1989). To examine the effect of ticks on juvenile bandicoots, mothers with young 45- to 50-days-old were removed from the external breeding enclosures and housed in cages. Young were weaned at day 60 of lactation, at which time each young was individually housed and thereafter weighed weekly. Thirteen juvenile bandicoots, each approximately 600 g, were examined for 3 wk prior to release into external enclosures. Seven juveniles were released into tick-infested enclosures and six juveniles into adjacent tick-free enclosures. The latter enclosures were surrounded by a water moat to prevent the entry of ticks. The yards and bandicoots were sprayed bi-weekly with Taktic EC (Schering Pty. Ltd., 57-79 Anzac Parade, Kensington, 2033, Australia), 15 ml in 2 l of water. Both groups of bandicoots received similar food and water ad libitum (Gemmell, 1982, 1989).

Bandicoots were lightly anaesthetised with a 'Halothane' (ICI Australia Operations Pty. Ltd., Melbourne, 3000, Australia)-oxygen mixture for ease of handling, blood sampling and weighing. Blood samples were obtained weekly from the 13 juveniles by heart puncture 3 wk prior to release into the external enclosures and for 6 wk after release. Blood samples were also obtained 9 wk after release. Each bandicoot was returned to the enclosure immediately after examination.

Leukocytes were counted using an improved (Carolina Biological Supply Company, Burlington, North Carolina 27215, USA) Neubauer haematocytometer. The haematocrit value was measured using gold seal heparinized micro haematocrit tubes (Clay Adams, Beeton Dickinson and Company, Parsippany, New Jersey 07054, USA). Blood films for differential white cell counts and red cell morphology were stained with Jenner-Giemsa stain (McClung, 1939).

Differences in body weight, haematocrit value and the number of white blood cells in both groups of juveniles were analysed by ANOVA to determine whether the differences were significant. The level of α was established at $P \leq 0.05$.

Blood samples were obtained from 15 adult bandicoots trapped in the environs of Brisbane, Queensland, Australia (27°24'S, 152°45'E). On the day of capture each bandicoot was lightly anaesthetised with 'Halogen'-oxygen mixture, weighed and a blood sample obtained by cardiac puncture. To compare these samples from

wild bandicoots with those from bandicoots in enclosures, similar samples also were obtained weekly from eight adult bandicoots which had been housed in the outside tick-infested enclosures for at least 6 mo.

To determine numbers and species of ticks present on animals in enclosures the following procedure was adopted. Each week, either two or three adult bandicoots were transferred from an enclosure and placed in wire mesh cages (30 × 40 × 50 cm) surrounded by a water moat. All ticks falling from each bandicoot over a 7 day period from October 1984 and March 1986 were collected for identification.

RESULTS

Three species of ticks were obtained from adult bandicoots in the enclosures, *H. humerosa*, *I. tasmani* and *I. holocyclus*. These ticks were found throughout the year with the majority of adult stages being present in the latter one-half of the year (January through June, Table 1).

Blood parameters, including packed cell volume and white cell count of adult wild bandicoots and adults housed in the outside enclosures were similar (Table 2).

When juvenile bandicoots were transferred from cages into enclosures, body weight decreased initially irrespective of tick status but recovered to pre-enclosure levels by 3 wk (Fig. 1). Although variation in body weight was greater in bandicoots housed in tick-free enclosures, the growth rate of this group was greater than that of the juveniles housed in enclosures with ticks. Within 6 wk of release, the mean body weights of bandicoots housed in tick-free enclosures increased by 104 g, representing a mean weight gain of 2.5 g/bandicoot/day (Fig. 1). Over a similar period, seven bandicoots released into tick-infested enclosures had a total mean body weight gain of 74 g, or 1.8 g/bandicoot/day (Fig. 1). There also was variation in the white blood cell count (WBC) and haematocrit values between the two groups.

During the 3 wk in cages there was no significant difference between WBC numbers for the seven bandicoots which were to be housed in enclosures with ticks, and the six bandicoots which were to be housed

TABLE 1. The number and species of ticks present on bandicoots housed in enclosures from October 1984 to March 1986 expressed as the number of larvae, nymphs, males, and females obtained from the number of bandicoots examined per month.

	<i>Haemaphysalis humerosa</i>				<i>Ixodes tasmani</i>				<i>Ixodes holocyclus</i>				Number of bandicoots examined
	L ^a	N ^b	M ^c	F ^d	L	N	M	F	L	N	M	F	
1984													
Oct	—	109	153	901	71	32	1	68	—	1	2	27	11
Nov	18	6	39	182	120	10	—	34	—	1	—	8	8
Dec	19	25	14	27	120	23	25	28	—	—	—	—	7
1985													
Jan	—	10	16	19	26	226	—	12	—	3	—	—	10
Feb	4	9	10	3	151	30	4	4	225	14	—	—	7
Mar	6	4	—	1	145	34	—	1	313	103	—	—	8
Apr	2	33	—	6	303	123	1	2	11	474	—	—	10
May	—	—	—	—	52	49	—	5	>1,000	213	1	6	8
Jun	—	4	—	1	57	223	—	21	13	51	1	4	8
Jul	17	35	—	8	43	166	—	3	6	222	1	6	10
Aug	—	13	4	15	8	97	—	9	—	44	2	4	8
Sep	1	4	5	18	33	37	—	10	—	20	—	4	8
Oct	—	1	7	46	55	21	1	30	—	9	—	1	8
Nov	7	3	7	11	31	12	—	—	—	1	—	1	8
Dec	8	19	6	11	46	66	—	2	20	—	—	1	10
1986													
Jan	41	82	25	78	19	13	—	2	30	2	—	—	8
Feb	3	56	4	36	11	8	—	4	48	12	—	—	6
Mar	8	18	50	28	29	3	—	—	1,200	8	—	—	8

^a L, larva.

^b N, nymph.

^c M, male.

^d F, female.

in tick-free enclosures (ANOVA, $F_{(1,37)} = 0.51$, $P < 0.05$; Fig. 2). There was a significant increase in WBC numbers in the seven bandicoots housed in enclosures with ticks when compared with values obtained when these animals were housed in cages prior to their release (ANOVA, $F_{(1,61)} = 5.08$, $P < 0.05$; Fig. 2). The ratio of neutrophils

TABLE 2. Blood parameters of adult bandicoots housed in tick-infested, external enclosures (eight animals, 24 samples) and bandicoots trapped in the wild (15 animals, 15 samples).

	In enclosures		In wild	
	\bar{x} ^a	SE ^b	\bar{x}	SE
Weight (g)	1,322.3	29.1	1,272.6	118.0
Haematocrit	37.8	1.3	43.8	2.1
White blood cells (/m ³)	16,320	1,410	13,954	1,774
Neutrophils (%)	8.8	0.8	9.6	0.9
Basophils (%)	0	0	0	0
Eosinophils (%)	4.8	0.7	3.2	0.6
Lymphocytes (%)	85.3	1.2	85.7	1.5
Monocytes (%)	1.1	0.1	1.5	0.1

^a \bar{x} , mean.

^b SE, standard error.

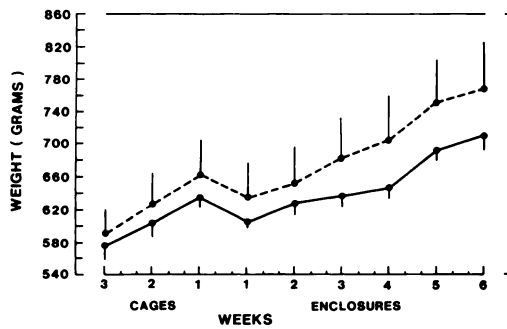


FIGURE 1. The weekly weights of seven juvenile bandicoots housed for 3 wk in cages and then placed in tick-infested, external enclosures for 6 wk (—). The weekly body weights of six juvenile bandicoots housed for 3 wk in cages and then placed in tick-free, external enclosures for 6 wk (---) ($\bar{x} \pm SE$).

to lymphocytes did not change significantly in these seven bandicoots (Table 3). There was no significant increase in WBC numbers in the six bandicoots transferred from cages to the tick free enclosure (ANOVA, $F_{(1,52)} = 1.27$, $P < 0.05$; Fig. 2).

During a 3 wk period, haematocrit values for bandicoots in cages varied from 45 to 50%. Haematocrit values decreased about 5% in the six bandicoots transferred to tick-free enclosures. A more dramatic drop in haematocrit values was found in the seven bandicoots transferred to enclosures with ticks, with a mean haematocrit

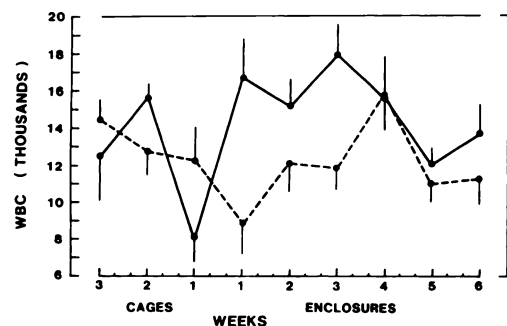


FIGURE 2. The white blood cell count of seven juvenile bandicoots housed for 3 wk in cages and then placed in tick-infested, external enclosures for 6 wk (—). The white blood cell count of six juvenile bandicoots housed for 3 wk in cages and then placed in tick-free, external enclosures for 6 wk (---) ($\bar{x} \pm SE$).

TABLE 3. Blood parameters of seven juvenile bandicoots housed in cages and then in tick-infested, external enclosures (21 samples were obtained in cages, followed by 42 samples in enclosures).

	In cages		In enclosures	
	\bar{x} ^a	SE ^b	\bar{x}	SE
Neutrophils (%)	8.5	1.9	11.1	2.0
Basophils (%)	0	0	0	0
Eosinophils (%)	3.3	0.9	1.5	0.2
Lymphocytes (%)	87.5	2.2	86.6	2.1
Monocytes (%)	0.2	0.2	0.7	0.3

^a \bar{x} , mean.

^b SE, standard error of mean.

value of 27% being observed 4 wk after release (Fig. 3).

Nine wk after release into the enclosures with ticks, the seven bandicoots weighed 900 ± 29.4 g, had a WBC count of $11,366 \pm 1,879$ cells/cm and a haematocrit value of $40.0 \pm 1.1\%$.

DISCUSSION

The results obtained in this study clearly indicated that infestation with *H. humerosa*, *I. tasmani* and *I. holocyclus* affected the health of juvenile northern brown bandicoots held in captivity. Bandicoots kept free of ticks had significantly better daily weight gains that similar bandicoots which were tick infested. This agrees with the

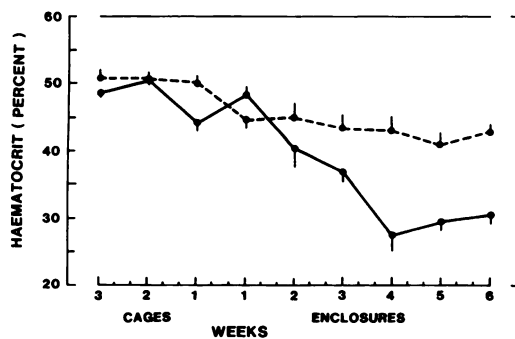


FIGURE 3. The haematocrit value of seven juvenile bandicoots housed for 3 wk in cages and then placed in tick-infested, external enclosures for 6 wk (—). The haematocrit value of six juvenile bandicoots housed for 3 wk in cages and then placed in tick-free, external enclosures for 6 wk (---) ($\bar{x} \pm SE$).

observations of Francis (1960), Little (1963), and Johnston (1969) on the debilitating effect of tick infestations. Also, tick-free bandicoots did not experience the same destruction of erythrocytes as tick infested animals. A similar effect was observed by O'Kelly and Seifert (1970) when *B. taurus* steers were exposed to *B. microplus*. In addition leucopenia was observed in tick-infested bandicoots and at this stage the reason for this is not clear. While it could be attributed directly to the presence of ticks, it is possible that the increased number of white blood cells observed in tick-infested bandicoots was due to parasitic infections or from toxins transmitted by the ticks.

Bandicoots are the principal hosts for *I. holocyclus* (Stewart and de Vos, 1984). This tick causes paralysis in domestic animals and occasionally in man (Stone et al., 1983). Albiston (1967) believed that in nature bandicoots develop an immunity to the toxin produced by *I. holocyclus* as a result of repeated infestations with larvae and nymphs.

Other ixodid ticks including those observed in the study are vectors of important haemoprotozoan parasites. Mackerras (1959) observed that *Theileria perameles* transmitted by *I. tasmani* resulted in anaemia in the bandicoot host. Priestly (1915) indicated that in *Tachyglossus aculeatus*, although the pathogenicity of the parasite is unknown, it was believed to have been associated with the deaths of 12 echidnas in the Taronga Park Zoological Garden in Sydney (Australia) (Backhouse and Bolliger, 1957). Mackerras (1959) also reported a species of *Theileria* from the blood of the platypus. However, the pathogenicity of this parasite may never be known because of the highly protective status of the host. Finally, *H. humerosa* is a likely vector of a species of *Babesia* found in *I. macrourus*. The latter parasite was found to be non-pathogenic, even in splenectomised bandicoots (N. P. Stewart, unpubl. obs.) and thus, would be an unlikely cause of anaemia observed in the present study.

In marsupials, the number of white blood cells and ratio of neutrophils to lymphocytes varies with the species. The koala, (*Phascolarctos cinereus*) has 7,000–8,000 white blood cells/mm³ and a 1:1 ratio of neutrophils to lymphocytes (Gaughin and Judson, 1980). Haematocrit values in marsupials also vary between species. The hairy-nosed wombat has a haematocrit of 39 and 43% in the wild and captive specimens, respectively (Gaughin and Judson, 1980).

Male possums have a higher haematocrit than females, 46 and 41% per male and female *Trichosurus vulpecula*, respectively (Barnett et al., 1979). The haematocrit value for the koala is 37% with a range of 28 to 45% (Canfield et al., 1989). The dasyurid *Antechinus stuartii* has a white blood cell count for 4,400 to 7,600 cells and a ratio of 16 neutrophils to 88 lymphocytes in February which changes to 68 neutrophils to 22 lymphocytes in August. The haematocrit value decreases from 45 to 33%. The haematological changes were thought to be associated with the reaction to latent disease and infection observed in *A. stuartii* after mating and the total mortality in males (Cheal et al., 1976).

Several disease syndromes associated with tick infestations include anaemia, malaise, loss of condition, allergic manifestations, paralysis, and certain local and systemic disturbances (Roberts, 1970). It has been demonstrated that only 40% of juvenile *I. macrourus* released into tick-infested enclosures survived to maturity (Gemmell, 1989). In the present study, tick-infested juveniles showed anaemia, elevated white blood cell counts and a reduction in body weight gains. Alternatively, young bandicoots without ticks maintained high haematocrit values, gained weight at a faster rate and were less subject to variations in white cell numbers.

While heavy tick infestations may be detrimental to the health of the bandicoots, it should be remembered that all species of ticks collected during this study

are capable of transmitting disease (Stewart and de Vos, 1984). Mackerras (1959) observed that of the parasites found in bandicoots at least one, *T. perameles*, is capable of producing anaemia. Therefore, bandicoots in tick-infested enclosures could be at high risk of contracting theileriosis. Weillgama (1986) examined the effect of two species of ticks, *I. tasmani* and *H. humerosa* on *I. macrourus*. *Ixodes tasmani* was suggested to be the natural vector of *T. perameles*, the most frequently encountered haemoprotozoan parasite in the short-nosed bandicoot. The prevalence and effect of *T. perameles* in juvenile *I. macrourus* needs further examination.

Estimates of the seasonal abundance of *I. holocyclus* on *I. macrourus* in south-eastern Queensland have been made previously (Doube, 1979). *Ixodes holocyclus* was abundant and at the peak of abundance, each bandicoot had 500 to 2,000 larvae, 100 to 200 nymphs and four to six engorged females. Females were most abundant in spring and early summer, larvae in summer-autumn, and nymphs in autumn-winter (Doube, 1979). Our results are in reasonable agreement with those of Doube (1979).

Arthropod parasites have been implicated in causing disease and death in several species of marsupials including *P. cinereus* and *A. stuartii* (Dickens, 1976; Cheal et al., 1976). The effects observed in this study were either due to ticks or tick-borne parasites, but this requires further investigation. It is suggested that anaemia associated with tick infestation, although not fatal may have contributed in rendering the bandicoots more susceptible to disease, and as observed in previous studies (Gordon, 1971; Stoddart and Braithwaite, 1979; Hall, 1983; Gemmell, 1989) their subsequent death.

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