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James C. Munger
*Boise State University, Boise, Idaho*

Todd A. Slichter
*Boise State University, Boise, Idaho*

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WHIPWORM (*TRICHURIS DIPODOMYS*) INFECTION IN KANGAROO RATS (*DIPODOMYS* SPP): EFFECTS ON DIGESTIVE EFFICIENCY

James C. Munger and Todd A. Slichter

**ABSTRACT.**—To determine whether infections by whipworms (*Trichuris dipodomys* [Nematoda: Trichurata: Trichuridae]) might affect digestive efficiency and therefore energy budgets of two species of kangaroo rats (*Dipodomys microps* and *Dipodomys ordii* [Rodentia: Heteromyidae]), we compared the apparent dry matter digestibility of three groups of hosts: those naturally infected with whipworms, those naturally uninfected with whipworms, and those originally naturally infected but later disinfected by treatment with the anthelminthic Ivermectin. Prevalence of *T. dipodomys* was higher in *D. microps* (53%) than in *D. ordii* (14%). Apparent dry matter digestibility was reduced by whipworm infection in *D. microps* but not in *D. ordii*. Although a statistically significant effect was shown, its small magnitude indicates that whipworm infection is unlikely to have a biologically significant impact on the energy budgets of host kangaroo rats.

**Key words:** parasite, digestive efficiency, whipworm, kangaroo rat, *Trichuris*, *Dipodomys*, energy budget.

Parasites inhabiting the gastrointestinal tract of a host may reduce the efficiency of the organs they inhabit either through direct competition for nutrients or through damage to absorptive surfaces. Because decreased digestive efficiency may reduce the rate of energy input into a host, gastrointestinal parasites have the potential to cause a change in host energy allocation (e.g., reduced activity or reduced reproduction), and thereby impact the ecology of the host (Munger and Karasov 1989).

Tapeworm infections have a measurable effect on digestive efficiency, but a biologically unimportant effect on the energy budget of host white-footed mice (*Peromyscus leucopus*; Munger and Karasov 1989). The present study was designed to determine if infection by a nematode, the whipworm *Trichuris dipodomys*, has a substantial effect on one aspect of the energy budget, digestive efficiency, of host kangaroo rats (*Dipodomys microps* and *D. ordii*).

**MATERIALS AND METHODS**

Our study site, located 2 km N of Murphy, Owyhee County, ID, is in desertscrub habitat with sandy loam substrate. Primary shrub species of the study area are *Artemisia spinescens*, *Artemisia tridentata*, *Atriplex canescens*, *Atriplex confertifolia*, *Atriplex spinosa*, and *Chrysothamnus nauseosus*. Six rodent species were captured at the site, *Ammospermophilus leucurus*, *Neotoma lepida*, *Perognathus flavus*, *Peromyscus maniculatus*, and two species of kangaroo rats, *Dipodomys ordii* and *Dipodomys microps*. *Dipodomys ordii* ranges from 42 to 72 g and consumes a diet consisting primarily of seeds (Zeveloff 1988). *Dipodomys microps* is larger, 72–91 g, and is unique among kangaroo rats in that it relies heavily on leaves of *Atriplex confertifolia* for forage (Kenagy 1972, Zeveloff 1988). Both species are liable to infection by the whipworm *Trichuris dipodomys*, a nematode that inhabits the cecum of infected hosts (Grundmann 1957, Whitaker et al. 1993). On the study site we established a 13 × 13 grid of 169 Sherman live traps baited with millet and placed at 15 m intervals. During two trapping sessions, 14–22 June and 15–18 August 1990, kangaroo rats (30 individuals of *D. microps* and 85 of *D. ordii*) were captured and brought into the laboratory. Fecal specimens from each animal were analyzed for the presence of parasite eggs by standard centrifugal flotation techniques using saturated sucrose solution (Pritchard and Kruse 1982). Six infected but untreated animals from the June experiment were included in the pool of animals used in the August experiment. The few animals that failed to thrive in the lab were removed from the experiment; data from a
total of 29 D. microps individuals and 56 D. ordii were analyzed.

Each month’s set of captures was subjected to the following protocol:

1. Kangaroo rats were acclimated to a diet of millet seed for 3–11 d.
2. A pretreatment feeding trial was performed: Animals were placed in wire-bottomed cages with a measured amount of whole millet seed. At the end of 5 d, fecal pellets were separated from spilled food and dried >24 h at 50°C. Initial digestive efficiency of each animal was measured as apparent dry matter digestibility (i.e., the proportion of mass consumed but not lost as waste), which was calculated as \( \frac{(M_{FO} - M_{FE})}{M_{FO}} \), where \( M_{FO} \) and \( M_{FE} \) are the mass of food consumed and feces produced, respectively.
3. Half of the infected animals were then injected subcutaneously with a solution of Ivermectin (a systemic anthelmintic; Ivomec brand, from MSD AGVET, Rahway, NJ). Figure 1 gives sample sizes of treatment groups. June captures received, on each of two consecutive days, a 0.2-cc injection of lvermectin in 40% glycerol formal and 60% propylene glycol; each injection delivered ca 350 \( \mu \)g Ivermectin/kg body mass. Controls received equal-volume injections of the glycerol formal–propylene glycol carrier. This dosage had little effect on the presence of whipworm eggs in feces of injected animals. Therefore animals received 8 d later a second set of two injections, each of 0.15 cc and delivering ca 2 mg Ivermectin/kg body mass; control animals received the carrier. August captures received, on each of two consecutive days, an injection of 0.15 cc volume delivering ca 2 mg Ivermectin/kg body mass. Control animals received the carrier. To control for possible side effects of Ivermectin, half of the uninfect ed animals captured in August were also injected with a solution of Ivermectin.
4. Two days after each set of injections a posttreatment feeding trial was conducted using techniques in (2) above. Only results of the pretreatment feeding trials and feeding trials following the 2-mg Ivermectin/kg body mass injection will be presented below.

**RESULTS AND DISCUSSION**

Adult worms (seven of each gender) taken from a Dipodomys microps at our site were identified as Trichurus dipodomys. Although some minor morphological differences from the original species description (Read 1956) do exist, perhaps as a result of geographical variation, the specimens most closely match Read’s description of T. dipodomys (A. Shostak personal communication). Measurements of several key morphological characters are as follows (\( \bar{X} \pm SD \)): total length: \( \bar{\varnothing} 25.6 \pm 0.8 \) mm, \( \varnothing 41.3 \pm 2.9 \) mm; hindbody length: \( \bar{\varnothing} 12.7 \pm 0.4 \) mm, \( \varnothing 23.7 \pm 1.9 \) mm; spicule length: 850 ± 85.1 \( \mu \)m; egg length: 64.8 ± 5.0 \( \mu \)m; egg width: 33.5 ± 1.0 \( \mu \)m. Voucher specimens were deposited with the University of Alberta Parasite Collection (#’s UAPC11464 and UAPC11465). Although we did not identify whipworms from D. ordii, we are confident they are T. dipodomys; the type host for T. dipodomys is D. ordii, and T. dipodomys is known only from D. ordii and D. microps (Whitaker et al. 1993).

Prevalence in Host Species.

Trichurus dipodomys occurred at substantially higher prevalence in D. microps than in D. ordii (Table 1), a result similar to that of Grundmann (1957). We can speculate as to three possible explanations for this pattern. The first is that eggs produced by adult worms in D. microps may become embryonated more easily than those in D. ordii. Freshly produced fecal pellets of D. microps appear moister than those of D. ordii (Munger personal observation), probably because of the higher amount of green or leafy vegetation in the diet of D. microps. If moisture is necessary for embryonation of the eggs (as is implied by Parry 1968),
moister feces may lead to higher embryonation rates and therefore higher prevalence among *D. microps*. The second explanation is that social behavior may differ between these species. For example, perhaps *D. microps* individuals visit one another’s burrows (and thereby become exposed to parasite eggs) at a substantially higher frequency than do *D. ordii*. Also, *D. microps* inhabits a mound up to 2 m in diameter while *D. ordii* inhabits less substantial individual holes. Studies of another system of two species of kangaroo rats has shown that the larger, mound-inhabiting *D. spectabilis* uses its burrow system for prolonged periods, while the smaller *D. merriami* rotates among several burrows (Jones 1989). This latter behavior would tend to reduce reinfection of individuals; it would be interesting to see if behaviors differ similarly between *D. microps* and *D. ordii*. The third explanation is that resistance to infection may differ between these two host species.

**Effects on Digestive Efficiency**

Apparent dry matter digestibility (ADMD) of millet seed was quite high, >95% on average (Table 2), a figure comparable to that found by Schriber (1979) for granivorous rodents. Injection of Ivermectin did not appear to affect ADMD of animals uninfected with whipworms, an effect that might occur through the removal of other symbionts, or through some direct effect (proportional change in ADMD, $\bar{X} \pm SE$: untreated: $-0.0043 \pm 0.0035$, treated: $-0.0058 \pm 0.0037$). Therefore, in the following analyses all naturally uninfected animals are combined into one class.

The effect of whipworm removal on ADMD was analyzed with a two-way analysis of variance (ANOVA). One factor analyzed was the treatment: deinfected (naturally infected but treated with Ivermectin) vs. infected (naturally infected but not treated with Ivermectin) vs. naturally uninfected. The other factor was species. Experimental period (July vs. August) was included as a blocking factor. The dependent variable in the analysis was proportional change between pretreatment and posttreatment ADMD ([(post-pre)/pre]): this measure should be more sensitive than posttreatment ADMD in expressing treatment effects because it takes account of initial differences in ADMD among hosts.

Although there were no statistically significant main effects of treatment or species on ADMD, there was a significant interaction between these factors (Table 3), indicating that the two host species differ in their response to treatment. This difference between species was explored using a separate ANOVA for each species, which revealed that treatment with Ivermectin had a significant effect on change in ADMD in *D. microps*, but not in *D. ordii* (Table 4, Fig. 1). A Tukey's *a posteriori* multiple sample test revealed that, within *D. microps*, the change in ADMD of the deinfected group differed significantly from the change in ADMD of both the infected group and the uninfected group. These results can be interpreted as showing that the deinfected group had 1.9% higher ADMD than the other two groups.

Of interest is the lack of effect *Trichuris* causes in *D. ordii*. This may be due to what appears to be a higher intensity of infection (more parasites per infected host) in *D. microps*: fecal floats of *D. microps* in general contained more eggs than did floats of *D. ordii* (*D. microps* $\bar{X} = 254$, SE = 115.2; *D. ordii* $\bar{X} = 63.5$, SE = 21.0; Mann-Whitney U-test, $U = 79$, $P = .1$). If fewer worms were present in *D.
TABLE 3. F values and probability values (P) from three-way analyses of variance on effects of species, month, and treatment (deinfected, infected, or uninfected) on apparent dry matter digestibility (ADMD).

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
<th>P</th>
<th>F</th>
<th>P</th>
<th>F</th>
<th>P</th>
</tr>
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<td>Treatment</td>
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<td>.86</td>
<td>.72</td>
<td>.49</td>
<td>.47</td>
<td>.63</td>
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<tr>
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<td>.46</td>
<td>.50</td>
<td>.82</td>
<td>.37</td>
<td>.83</td>
<td>.57</td>
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<tr>
<td>Treatment * Species</td>
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<td>.27</td>
<td>1.78</td>
<td>.18</td>
<td>4.74</td>
<td>.012</td>
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<tr>
<td>Block (= Month)</td>
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<td>9.11</td>
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<td>.00</td>
<td>.95</td>
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<tr>
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</tbody>
</table>

TABLE 4. Results from one-way analyses of variance on the effect of treatment (deinfected, infected, and uninfected) on % change in dry matter digestibility in D. microps and D. ordii.

<table>
<thead>
<tr>
<th>Species</th>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
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<td>D. microps</td>
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<td>.019</td>
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<tr>
<td></td>
<td>Error 27</td>
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</tr>
<tr>
<td></td>
<td>Error 52</td>
<td>.01442</td>
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</tr>
</tbody>
</table>

ordii, the effect of eradicating those worms would have been less apparent.

One might question the biological importance of the slight, albeit statistically significant, decrease in ADMD caused by *Trichuris* infection. Munger and Karasov (1989) showed an effect of similar magnitude resulting from tapeworm infection (*Hymenolepis citelli*) in white-footed mice (*Peromyscus leucopus*). They argued that hosts can easily compensate for such small effects by slight increases in food consumption or decreases in expenditures, or by changes in gut morphology (Mettrick 1980), and concluded that such effects on ADMD are unlikely to affect host energy budgets or to translate through to population-level effects. The same conclusion is likely to apply to the kangaroo rat–whipworm system.

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**LITERATURE CITED**


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