INFLUENCE OF AN EXPERIMENTAL INFECTION OF SWINE KIDNEYWORM (STEPHANURUS DENTATUS) ON PERFORMANCE OF PIGS

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Summary
Forty pigs (average 25.2 kg live weight) were individually housed and fed to study the effect of a single infection of Stephanurus dentatus (either 0, 72, 457 or 842 S. dentatus larvae/kg body weight) on performance of growing-finishing pigs. Final weight and average daily gain were depressed (P<.05) by increasing levels of kidneyworm infections. Average daily gain of pigs not infected was 69% greater (P<.05) than that of pigs given 842 S. dentatus larvae/kg body weight. Feed to gain ratios of pigs were increased linearly (P<.05) with increasing levels of kidneyworm larvae. Feed to gain ratio for pigs not infected was 24% less (P<.05) than that for pigs given 842 kidneyworm larvae/kg body weight. In each of two trials, eight cross-bred barrows (average 26.0 kg in trial 1 and 22.6 kg body weight in trial 2) were examined for the effects of two levels of kidneyworm infections (0 and 457 larvae/kg body weight) on digestion and absorption of nutrients and on N balance. Digestion coefficients for dry matter, crude protein and energy for pigs not infected and for those experimentally infected were similar (P>.05). Pigs not infected had higher (P<.01) N intakes, excreted more (P<.05) N in feces and urine and had a higher (P<.01) N balance than pigs infected with kidneyworms, due largely to difference in feed intake.

Introduction
The swine kidneyworm (Stephanurus dentatus) is an economically important nematode parasite and occurs in Asia, Africa, North America, Central America, South America, Spain and Australia (Spindler and Andrews, 1954; Dykova, 1977). The kidneyworm has been reported from 19 states in the United States, but it is most prevalent in the Southeast where its incidence has ranged from 78 to 94% (Stewart et al., 1964). Most of the annual loss caused by the swine kidneyworm (which was estimated to be nearly $73 million in the United States) results from condemnation of liver, kidneys and other edible parts that have been invaded by the migrating larvae (Stewart et al., 1964). Although several anthelmintics have been reported to be effective in removing kidneyworms from swine (Stewart et al., 1977, 1981a,b), nearly all sows from South Georgia presently have some kidneyworms and 90% of the livers from these sows are condemned (J. Prophater, personal communication). Economic losses from condemnations of portions of the swine carcass due to kidneyworm infections are well documented, but little is known about the losses that might occur in growing-finishing swine from decreased growth rate and feed efficiency.

This study was conducted to determine the effect of different levels of experimental infections of kidneyworms on performance of growing-finishing swine and on digestion and absorption of nutrients by the growing pig.

Experimental Procedure
Exp. 1, Growing-Finishing Trials. Forty pigs (average 25.2 kg body weight and 81 d of age) were divided into four comparable groups of

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2Present address: Southern Grain Insect Res. Lab., USDA/ARS, Tifton, GA 31793.
3Reference to a company or product name does not imply approval or recommendation of the product by the USDA to the exclusion of others that may be suitable.
4Supported by State and Hatch funds allocated to the Georgia Agr. Exp. Sta.
10 based on sex and initial weight. Each group was assigned to one of four treatments: Group 1, 0 kidneyworm larvae/kg body weight; Group 2, 72 larvae/kg body weight; Group 3, 457 larvae/kg body weight and Group 4, 842 larvae/kg body weight.

Composition of diets fed during the growing-finishing experiment and the digestion-absorption experiment is shown in Table 1. Proximate analysis of the diet was conducted by AOAC (1980) methods, except for N, which was determined with a Technicon Autoanalyzer II. Pigs were housed and fed individually in concrete-floored pens (1.22 x 3.66 m), with feed and water supplied ad libitum. Pens were located under an open shed (eaves 2.1 m high) and oriented north and south in two rows separated by a 1.83-m alley so that two-thirds of each pen was under the roof.

Urine from a kidneyworm infected sow was collected in a plastic bucket and the eggs were allowed to sediment for approximately 2 h at room temperature. Supernatant urine was discarded and the eggs were concentrated and washed in tap water by repeated centrifugation at 1,000 x g for 10 min. The washed egg suspension (10 to 20 ml) was mixed into a plastic bucket containing 1 liter of moist sphagnum moss and coarse vermiculite (5:1) to which 25 ml of adult swine fecal extract (Moncol and Triantaphyllou, 1978) had been added. Several cultures were prepared to ensure recovery of sufficient larvae. Cultures were covered with aluminum foil and incubated at 25 C for 10 to 14 d. Larvae were collected from cultures by Baermannization for 4 to 6 h in tap water (15 C). Larvae were diluted and counted on a counting slide and the concentration of larvae/volume of water was adjusted as needed for infection of pigs.

Pigs were restrained with their mouths held open and the appropriate dose of larvae in a plastic tube was poured slowly into the open mouth. The tube was then rinsed with several milliliters of water, which were also given by mouth to each pig.

Pigs were infected on d 1 of the experiment and were maintained on test for 64 d. The animals were removed from test and slaughtered at a local meat processing plant, except for five pigs that failed to complete the test. Pigs were stunned with a captive-bolt pistol, bled and eviscerated. Vital organs were removed, weighed and examined for parasite damage.

Data were analyzed by appropriate least-squares analysis of variance and covariance.

Exp. 2, Digestion and N Balance Trials. In each of two trials, eight crossbred barrows (average 26.0 kg body weight in trial 1 and 22.6 kg in trial 2) were examined for the effect of an experimental infection of kidneyworms (457 larvae/kg body weight) on digestion and absorption of nutrients and on N balance. The pigs were infected in the same manner as in Exp. 1. Infected and control pigs were transferred to individual metal cages for collection and separation of feces and urine on d 17 after infection. Pigs were maintained in the metabolism cages for a 9-d adjustment period and a 5-d total collection period that began on d 26 postinfection.

During the digestion trials, pigs were given 1.6 kg feed daily in small self feeders. Any feed spilled from or left in the self feeders was recorded daily and subtracted from the total amount supplied. Most feeders for the control pigs were empty at feeding time. However,
TABLE 2. ADJUSTED LEAST-SQUARES MEANS FOR PERFORMANCE OF PIGS NOT INFECTED AND PIGS EXPERIMENTALLY INFECTED WITH KIDNEYWORMS\textsuperscript{a,b}

<table>
<thead>
<tr>
<th>Item</th>
<th>0</th>
<th>72</th>
<th>457</th>
<th>842</th>
<th>SE\textsuperscript{c}</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of pigs</td>
<td>10</td>
<td>10</td>
<td>9\textsuperscript{d}</td>
<td>6\textsuperscript{e}</td>
<td></td>
</tr>
<tr>
<td>Final wt, kg</td>
<td>92.9\textsuperscript{f}</td>
<td>80.0\textsuperscript{g}</td>
<td>76.1\textsuperscript{h}</td>
<td>65.3\textsuperscript{h}</td>
<td>2.02</td>
</tr>
<tr>
<td>ADG, kg</td>
<td>.81\textsuperscript{f}</td>
<td>.65\textsuperscript{g}</td>
<td>.60\textsuperscript{g}</td>
<td>.48\textsuperscript{h}</td>
<td>.02</td>
</tr>
<tr>
<td>Feed consumed, kg</td>
<td>202\textsuperscript{f}</td>
<td>163\textsuperscript{g}</td>
<td>158\textsuperscript{h}</td>
<td>141\textsuperscript{h}</td>
<td>5.60</td>
</tr>
<tr>
<td>Feed:gain ratio</td>
<td>2.97\textsuperscript{f}</td>
<td>3.05\textsuperscript{f}</td>
<td>3.16\textsuperscript{f}</td>
<td>3.67\textsuperscript{g}</td>
<td>.07</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Means adjusted to a common initial pig weight of 25.2 kg.
\textsuperscript{b}Pigs were housed and fed individually for an 84-d test period.
\textsuperscript{c}Standard error of the mean.
\textsuperscript{d}One pig died before completing test and was omitted from the analysis.
\textsuperscript{e}Four pigs were removed before completing test and were omitted from the analysis.
\textsuperscript{f,g,h}Means in same row with different superscripts are different (P<.05).

...variably amounts of feed were left in the feeders of the infected pigs.

Total fecal output was collected daily during each 5-d collection period, frozen and later thawed and composited for analysis. The urine was collected in plastic pans containing 25 ml of 50% concentrated sulfuric acid plus 200 ml of water. The daily collection of acidified urine was diluted to constant volume, sampled and composited in containers maintained under refrigeration and then frozen until analyzed.

Proximate analyses of diets and feces and N content of diets, feces and urine were performed by the same methods used for the diet in Exp. 1. The data were analyzed for statistical significances using a general least-squares program.

Results and Discussion

Exp. 1, Growing-Finishing Trials. Adjusted least-squares means for performance by pigs not infected and by those experimentally infected with kidneyworms are shown in table 2. Because initial weight affected (P<.01) rate of gain and feed efficiency, means for performance were adjusted to a common initial pig weight of 25.2 kg.

Final weight and average daily gain were altered (P<.05) by increasing levels of kidneyworm infection. Pigs in Group 1 (no infection) gained weight about 25% faster (P<.05) than pigs in Group 4 (842 larvae/kg body weight). A similar trend was obtained in feed intakes because pigs in Group 1 consumed 24, 28 and 43% more (P<.05) feed than did pigs in Groups 2, 3 and 4, respectively. Feed to gain ratios for pigs in Groups 1, 2 and 3 were not significantly different (P<.05), but were less (P<.05) than for pigs in Group 4.

During the growing-finishing trial, one infected pig from Group 3 died and four infected pigs from Group 4 became moribund and were removed from the study and necropsied. All five pigs had histories of weight loss, bloody feces and slight to severe vomiting of blood. All had extensive fibrosis and hypertrophy of the liver, massive occlusion of the hepatic portal veins by thrombi containing larval kidneyworms and large ulcerations of the esophagogastric region of the stomach. Child (1954) noted that portal hypertension in humans can develop in response to obstruction to portal blood flow and that the primary manifestation of portal hypertension is development of esophagogastric ulcer and splenomegaly. In addition, development of fibrous tissue and regeneration of liver lobules can impede blood flow at the level of the hepatic venules. The coronary vein (left gastric vein) is particularly susceptible to portal hypertension (Child, 1954) and it is this vein that serves the area of the stomach in which ulcers developed after S. dentatus infection.

Esophagogastric ulcers in swine were produced by two infections of pigs with eggs of
Ascaris suum (Gaafar and Keittevuti, 1972). They suggested that immunological phenomena were responsible for development of ulcers because a second dose of Ascaris eggs were required. However, Lopez and Gaafar (1980) proposed that portal hypertension was responsible for ulcer development. Ascaris and Stephanurus infections are similar in that both produce fibrosis of liver tissue and thus, impede blood flow through hepatic venules. Stephanurus also produces occlusion of hepatic portal veins.

Exp. 2, Digestion and N Balance Trials. Digestion coefficients for dry matter, crude protein, energy and N intake, excretion and balance for pigs not infected and for those experimentally infected with kidneyworms are shown in table 3. Digestion coefficients for dry matter, crude protein and energy for pigs not infected and for those experimentally infected with kidneyworms were not different (P>.05); however, pigs not infected had 2.5% greater digestion coefficients for dry matter (P<.06).

This is our third experience with a nematode parasite that did not depress digestion coefficients for ether extract. In fact, in one study (Hale and Stewart, 1979), we found that pigs experimentally infected with Trichuris suis (swine whipworm) actually had higher digestion coefficients for ether extract (P<.05) than pigs not infected.

The collection period for the digestion trial was scheduled to coincide with the peak circulating eosinophils, which occurs during d 28 to 32 postinfection (Batte et al., 1960, 1966). The rise in eosinophils indicates stress in the animal that could affect performance. In our trials, feed intake of infected pigs during the collection period was depressed because feed intake (dry matter basis) of pigs not infected averaged 1.41 kg daily and only .94 kg daily for infected pigs. Thus, pigs not infected had higher (P<.01) N intakes, excreted more (P<.05) N in feces and urine and had a higher (P<.01) N balance than pigs infected with kidneyworms. The differences in N excretion and balance were probably due to the differences in N intake because percentage N retained by both groups of pigs was similar (P>.05).

Weights of certain internal organs and girth of pigs in relation to slaughter weight are shown in table 4. Generally, relative organ weights tended to increase with increasing infection levels of kidneyworm larvae. The largest increase in weight of organ was obtained in liver because pigs infected with 842 kidneyworm larvae/kg body weight had greater than a threefold increase in liver weights over those of pigs not infected.

Because the liver is the normal site of infection during most of the larval phase of S. dentatus (Waddell, 1969), the increases in liver weight were both absolute increases caused by fibrosis in response to the presence of larval kidneyworms as well as relative increases caused by failure of infected pigs to make body weight gains comparable with pigs not infected. Relatively few kidneyworms were noted in the lungs.

No kidneyworms were found in hearts and
TABLE 4. SIZE OF CERTAIN ORGANS AND GIRTH IN RELATION TO SLAUGHTER WEIGHT OF PIGS NOT INFECTED AND EXPERIMENTALLY INFECTED WITH KIDNEYWORMS

<table>
<thead>
<tr>
<th>Treatment, larvae/kg body wt</th>
<th>Organ weight (g/kg slaughter wt)</th>
<th>Girth(a), cm/kg slaughter wt</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Liver</td>
<td>Lungs</td>
</tr>
</tbody>
</table>
| 0                           | 16.7\(d\) | 9.1\(d\) | 3.3\(d\) | 1.6\(d\) | 1.2\(d\) 
| 72                          | 36.1\(e\) | 10.5\(de\) | 3.4\(de\) | 2.0\(de\) | 1.4\(e\) 
| 457\(b\)                   | 53.0\(f\) | 12.5\(e\) | 3.5\(de\) | 2.4\(e\) | 1.4\(e\) 
| 842\(c\)                   | 54.5\(f\) | 13.4\(f\) | 3.8\(e\) | 2.3\(e\) | 1.6\(f\) 
| SE                          | 1.26   | .38    | .08    | .08    | .02   

\(a\) Girth was determined at the navel of each pig. 
\(b\) One pig died before completing test and was omitted from analysis. 
\(c\) Four pigs were removed before completing test and were omitted from analysis. 
\(d,e,f\) Means in the same column with different superscripts are different (P<.05).

Spleens of pigs. In contrast to livers, hearts and spleens of infected pigs showed relative increases in weight produced, not by fibrosis in response to presence of larvae, but by failure of infected pigs to make body weight gains comparable with uninfected pigs.

Girth size, determined at the navel, followed a similar trend, in that pigs infected with 842 kidneyworm larvae/kg body weight had a 38% greater (P<.05) circumference than pigs not infected. The enlarged livers of the heavily infected pigs probably accounted for the increased girth size.

Swine kidneyworms are insidious parasites, and when present in sufficient numbers, cause marked reductions in growth rate and feed efficiency and increased condemnations of edible tissues and death of the host. Subclinical effects caused by this parasite in terms of reduced growth and feed utilization must be considered as well as losses due to condemnations of edible parts of the carcass when assessing its economic importance.

Literature Cited