SWINE ASCARIASIS:
EFFECT ON PRODUCTION
AND ABATTOIR SURVEILLANCE

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ABSTRACT

An epidemiological study was carried out to determine whether ascarids had an effect on production, as measured by average daily gain (ADG), and to critically assess abattoir surveillance of liver lesions (milk spots) as a screening test for ascariasis.

Fifteen swine herds in Prince Edward Island (Canada) were purposively selected to provide a range of ascarid infection for investigation. On each farm 30 pigs were randomly selected from the first 4 or 5 litters born after March 7th, 1987. The pigs were weighed and rectal fecal samples were collected at approximately 11, 15, 19 and 22 weeks of age, and again at slaughter. At the abattoir the carcass weight and index, and levels of anteroventral pneumonia, atrophic rhinitis, and liver lesions were recorded for each hog. The number of ascarids in the small intestines were counted.

To evaluate the presence of white spots as a screening test for ascariasis, 2x2 tables were created using the presence of ascarids at slaughter and presence of ascarid eggs in feces as indicators of ascariasis. In both cases, sensitivity was high (91% and 96% respectively) and specificity was very low (22% and 24%). Negative predictive values of 82.1% and 92.5% indicate that ascariasis is unlikely in the absence of liver lesions at slaughter.

Regression analyses were used to determine associations between ADG and independent variables controlling for sex, farm, and litters nested within farm. The regression model accounted for 75.4% of the variation in ADG. The number of intestinal ascarids at slaughter did not affect ADG. However, the "lifetime burden" (a composite measure based on fecal egg counts and duration of infection) was associated with ADG ($p<0.05$) in a quadratic manner. Although heavy ascarid burdens decreased the growth rate of swine, the magnitude of the effect was minimal. The maximum improvement one could expect from reducing the ascarid burden on heavily infected farms would be less than 1%.

Severe atrophic rhinitis and the presence of anteroventral pneumonia each had a detrimental effect on ADG ($p<0.001$). The corresponding reductions in mean ADG were 7.7% and 2.8% respectively. There was significant interaction between the effects of atrophic rhinitis and anteroventral pneumonia on ADG ($p<0.05$). Hogs with both anteroventral pneumonia and severe atrophic rhinitis had a 17.6% lower ADG than hogs with neither disease. There is much greater potential for improvement in ADG through control of respiratory diseases than through control of ascariasis.

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1. GENERAL INTRODUCTION

Ascariasis is common in all swine rearing areas of the world. It results in condemnation of livers at slaughter and is suspected to inhibit growth of swine.

This study was conducted because a large proportion of Prince Edward Island swine have milk spot liver lesions at slaughter. Milk spot lesions are usually ascarid-induced foci of interstitial hepatitis, hence the concern that there was an ascarid problem in the province.

1.1 Life cycle of Ascaris suum

Pigs are infected by ingesting infective Ascaris suum eggs which liberate second-stage larvae into the small intestine (1,2). The larvae penetrate the bowel wall, enter the blood stream (mainly the hepatic portal vein), and reach the liver six to 18 hours after ingestion. Here they moult to third-stage larvae by 36 hours after ingestion. Third-stage larvae migrate via the blood to the lungs, four to six days after ingestion. They move up the bronchial escalator to the pharynx and are swallowed. By day eight to ten they arrive in the small intestine.

Ascaris suum induces a host immune reaction all along its route of migration (3). This is manifested by milk spots on the liver and by focal hemorrhages, edema and congestion in the lung (4). There are waves of larval elimination from the small intestine, starting approximately 10 (2) and 21 (5) days after ingestion, which are believed to correspond with the third and fourth molts respectively. The immune reaction increases with the size of the challenge and there is an anamnestic response with repeated exposures (3). A heavy challenge may stimulate a reaction strong enough to eliminate the infection (6). It is therefore

possible to have milk spots on the liver without an infection being established in the small intestine. Conversely, ascarids may exist in the small intestine although evidence of hepatic migration has resolved, as milk spots heal three to five weeks after their formation (3,7).

If A. suum is successful in migrating to the gut, it grows rapidly in the small intestine and females start producing eggs approximately 60 days after ingestion (1,8). Each female can produce in excess of 1.5 million eggs daily (9). Eggs are fertilized providing both sexes are present. After being passed in the feces, fertile eggs require a minimum of ten days to develop to an infective stage at an environmental temperature of 30°C (10). Ascarid eggs are resistant to environmental stress; they can survive more than four years with repeated freeze and thaw cycles (11). However, 37 days continual desiccation is lethal to ascarid eggs even at moderate temperatures (12). Development resumes when the temperature exceeds 15°C and relative humidity is greater than 80% (13). Environmental contamination serves as the main reservoir for ascarid infection; infected pigs are important only as the initial contaminator of the environment.

1.2 Effects of ascariasis

Ascarids may have a detrimental effect through various means. The migratory phase damages both liver and lungs. Apart from physical injury to these organs, livers may have to be trimmed at slaughter and the lungs may be rendered more susceptible to subsequent invading pathogens (11). Once an intestinal infection is established, production losses may arise through inefficient feed conversion or hampered growth rates. Ascarid eggs have zoonotic potential (14,15,16) and can also have serious consequences for other animal species

(17,18,19,20,21,22).

Although it is easy to postulate routes through which ascarids may affect swine production, there is no conclusive evidence that they do. Experimental studies have suffered from small sample sizes, lack of control of confounding variables, insignificant results and inconsistent results. Many experimental investigations used very large inocula to establish infections, therefore they are probably not relevant to the situation to which pigs are exposed on farms (23).

There is only one field study available which provides quantitative information on the effect of naturally acquired ascariasis on the growth rate of individual hogs (24). There was no difference in average daily gain (ADG) over the fattening period between hogs with low and intermediate ascarid burdens. The ADG of hogs with high ascarid burdens, however, was 4.8% less than that of hogs with low burdens. The difference was of a greater magnitude for females than for males. Although this study controlled for sex of pigs, it did not control for other diseases which may affect ADG and could confound the results.

The principal objective of this study was to examine the effect of Ascaris suum on swine production. To do this effectively, it was necessary to account for possible confounding variables, including demographic characteristics and other diseases which may affect production. A secondary objective was to evaluate post mortem inspection of swine livers as a screening test for ascariasis.

Chapter 2 presents the levels of production, ascarid burden and confounding variables observed in this study and examines the relationship between various estimates of ascarid burden. In Chapter 3 the abattoir surveillance of liver lesions is assessed as a screening test for ascariasis. The relationships among ascarid burden and the control variables, and their effects on production are explored through path analysis in Chapter 4.

2. DESCRIPTIVE STATISTICS

2.1 Introduction

It has been traditionally accepted that the presence of intestinal nematodes results in a reduction of growth rate. However, little quantitative information is available on the effect of naturally acquired ascariasis on the growth rate of individual hogs under field conditions (24). To determine the effect of ascarids on growth rates it is necessary to measure those growth rates, ascarid burden and potential confounding variables.

In previous studies, ascarid infection has commonly been measured by the presence of ascarids in the small intestine. Surveys of gastrointestinal parasitism in the province of Quebec (25) and in Britain (26) reported the prevalence of ascarids in the intestines of market hogs to be 38.8% and 16.3% respectively. Levels of infection were less than 15 ascarids per hog in the first study and averaged 5 ascarids per infected hog in the latter. Ascarid burden has also been measured by the presence of ascarid eggs in the feces (24,27) and by the presence of milk spots on the liver (foci of ascarid-induced interstitial hepatitis) (24,28). In a study of 69 bacon pigs (29) Bindseil found that there was poor correlation between fecal egg counts and the number of intestinal ascarids. He concluded that ascarid burden should not be judged by egg counts except at very low levels of eggs. These three measures of ascarid burden (intestinal ascarids, eggs and milk spots) are all measures of prevalence. They reflect the situation at a point in time and do not account for the duration of the infection.

Average daily gain is commonly used as a measure of growth rate because it incorporates both days to market and weight at slaughter. When studying growth

rate reduction due to ascariasis, confounding variables that could be controlled include farm of origin, litter and sex. The analysis would be strengthened by the inclusion of other measurable variables which may affect production. The choice of variables for this study is discussed in depth in Chapter Four.

The objective of this chapter is to present descriptive statistics, including farm to farm variation, on the levels of production, ascarid burden and confounding variables observed in this study. The relationships among various estimates of ascarid burden will be described, including a measure which accounts for the duration of infection.

2.2 Materials and Methods

Fifteen swine herds from across Prince Edward Island were purposively selected to provide a range of levels of ascarid infection for investigation. Farm selection was based on previously recorded levels of liver lesions when this information was available. Otherwise, farms were selected based on a willingness to participate and estimates of likelihood of ascarid infection provided by Prince Edward Island Department of Agriculture swine specialists. Sow identification and date of birth were recorded for the first four or five litters until at least 30 piglets were born on each farm after March 7th, 1987. Personnel from the PEI Department of Agriculture visited all farms after all the litters were born. Birth data were verified and all piglets individually identified by ear notches. Although many producers treat with anthelmintics at weaning, anthelmintics were withheld from all piglets in these litters.

Regular follow-up farm visits commenced May 1987. Thirty pigs from the identified litters were randomly selected on each farm using previously prepared

computer generated random number tables, and these pigs were further identified by ear tags. The pigs were weighed with a portable hog scale and rectal fecal samples were collected at mean ages (±SD) of 78±5, 107±7, 133±5 and 154±6 days. Fecal samples were also collected at slaughter.

Quantitative fecal analyses were performed using the McMaster technique as described by Thienpont et al (30). Due to its speed and accuracy, this technique has been recommended as a standard egg-counting technique for valid comparisons between different operators and laboratories (31). A concentrated sucrose solution (32) was substituted for sodium chloride as sucrose solution has been reported to recover the highest number of ascarid eggs (33) and does not readily crystallize or cause distortion of parasite eggs (31,32). Results were expressed as estimates of eggs per gram of feces (epg).

Slaughter data were recorded at Garden Province Meats Inc., the only federally inspected abattoir in PEI. Date of slaughter, dressed carcass weight, index, and levels of anteroventral pneumonia, atrophic rhinitis and liver lesions were recorded for each test hog. The primary product inspectors were provided with guidelines for estimating the percentage of affected lung tissue (34). They scored lungs for anteroventral pneumonia as follows:

negative - no anteroventral pneumonia;

mild - 1% to 9% of the lung affected;

moderate - 10% to 25% of the lung affected; and

severe - greater than 25% of the lung affected.

Similarly, livers were scored according to the number of milk spots as follows:

negative - no milk spots;

mild - one to six milk spots; and

severe - greater than six milk spots.

In addition, the author conducted a systematic random sample, scoring viscera from every fifth test hog as above and recording the estimated percent of lung affected by anteroventral pneumonia.

Samples of milk spot lesions from 16 livers were submitted for histopathology.

Scores for atrophic rhinitis were assigned according to a system commonly used in North America (35,36). The snouts were sectioned at the level of the second upper premolar tooth. The space between the ventral turbinate and the floor of the nasal cavity was measured on both sides. The total amount of space (mm) was converted to a scale from zero to five:

0-2 mm = 1 (less than 3 mm is considered abnormal);

3-6 mm = 0;

7-9 mm = 1;

10-12 mm = 2;

13-16 mm = 3;

17-20 mm = 4; and

>20 mm = 5.

An extra one half point was added to the score if septal deviation or turbinate asymmetry were present. The maximum score was five.

The entire small intestine of each slaughtered test pig was tied off and transported from the abattoir to the Atlantic Veterinary College. There it was separated from its mesentery and drawn through thumb and finger to expel all contents which were strained through a ten mesh sieve. The number of macroscopic ascarids was determined. Samples of intestinal nematodes collected from hogs from five participating farms were submitted to parasitology.

Identification of Ascaris suum was confirmed by scanning electron microscopy.

The data were entered on a microcomputer database management system. Fecal egg counts and duration of infection were combined to calculate a composite measure of ascarid burden called "lifetime burden". The average egg count, measured as eggs per gram of feces, for consecutive visits was multiplied by the number of days elapsed between these visits; the sum of these figures until the fourth visit (mean age 154 days) constitutes that hog's raw lifetime burden. These high numeric values were standardized by dividing by a constant. The constant selected, the average lifetime burden for hogs whose lifetime burden was greater than zero, was chosen to facilitate interpretation of the results. A hog with a lifetime burden of one has an average lifetime burden for an infected hog. It follows that a hog with a lifetime burden of two has twice the average lifetime burden etc. Further detail of the method of calculation is provided in Appendix A.

The time between the onset of fecal shedding of ascarid eggs and slaughter was calculated in days. The onset of fecal shedding was determined as the midpoint between the first positive fecal analysis and the previous negative one. If the hog had a positive result on the first fecal analysis, the midpoint between the end of the prepatent period (approximately 60 days (1,8)) and the first fecal analysis was used.

Average daily gain was determined by dividing live weight at slaughter by days to market. The abattoir recorded only dressed carcass weights so it was necessary to convert them to live weights. Multiple linear regression was used to determine the relationship between dressed carcass weight and dressing percentage based on data from 80,000 hogs shipped in Ontario during one week in July 1986 (Dr. Gordon Bowman, University of Guelph, written communication). Dressed

carcass weight divided by dressing percentage provided an estimate of live weight at slaughter. Further detail of the method used is presented in Appendix B.

Descriptive statistics, linear regressions and analyses of variance were calculated using Statgraphics (37). Regression analyses were used to determine the associations between continuous measures of ascarid burden. One-way analyses of variance were performed to see how measures of ascarid burden varied with the categories of liver lesions.

2.3 Results

The average farm size was 77 sows (SD=32), with a range from 30 to 140 sows. Means and standard deviations of production indices are presented in Table I. Results are presented for 386 of the 450 hogs initially included in the study. Six hogs that lost weight either between visits or between the fourth visit and slaughter (if the interval was greater than two weeks), were assumed to have clinical illness and were removed from the analysis. Data from the remaining 58 missing hogs were not recorded at Garden Province Meats Inc. At least 12 of these hogs were shipped to another abattoir.

Average days to market ranged from 160 days on farm 15 to 218 days on farm 13. However, a direct comparison of days to market cannot be made since the hogs were shipped at different weights. The overall average dressed carcass weight of 77.0 kg, when used to calculate the average live weight at slaughter, gives an estimate of 96.5 kg.

Mean average daily gain ranged from 0.440 kg/day on farm 12 to 0.601 kg/day on farm 3. If a hog gained 0.440 kg/day it would take 219 days to reach the average live weight of 96.5 kg, whereas a hog gaining 0.601 kg/day would

Table I. Means and Standard Deviations of Production Indices for 15 Prince Edward Island Swine Herds, 1987

Farm number	No. hogs marketed[a]	Days to market	Dressed carcass wt (kg)	Average daily gain (kg/day)
1	29	183 (12)	81.9 (5.6)	0.563 (0.040)
2	24	171 (14)	79.4 (3.3)	0.590 (0.044)
3	29	166 (11)	79.1 (4.0)	0.601 (0.051)
4	28	207 (19)	74.8 (5.3)	0.460 (0.043)
5	24	178 (8)	76.7 (4.4)	0.543 (0.043)
6	24	199 (13)	78.6 (4.5)	0.496 (0.044)
7	26	182 (15)	81.0 (4.1)	0.560 (0.048)
8	29	203 (22)	74.8 (8.0)	0.475 (0.085)
9	29	179 (9)	72.2 (5.6)	0.514 (0.046)
10	18	194 (12)	73.4 (4.1)	0.480 (0.044)
11	28	204 (13)	75.2 (2.7)	0.464 (0.028)
12	18	209 (20)	71.2 (5.0)	0.440 (0.048)
13	26	218 (22)	80.3 (6.0)	0.464 (0.042)
14	25	199 (9)	79.6 (5.3)	0.502 (0.040)
15	29	160 (12)	74.6 (4.2)	0.589 (0.053)
Total[b]	386	189 (22)	77.0 (5.9)	0.519 (0.071)

[a] No. hogs marketed: number of study hogs for which sufficient data was recovered

[b] Total: calculations based on all hogs marketed

reach the same weight in 160 days. Thus the first hog would take 37% longer to reach market weight.

The results from the series of fecal examinations are presented in Table II. Only two farms had hogs shedding ascarid eggs at the time of the first post-weaning visit (when the animals were approximately 78 days of age). On subsequent visits this increased to 4, 8, and 10 farms, and when the hogs were marketed 13 farms shipped hogs that were shedding ascarid eggs in their feces. In general, the percentage of hogs shedding eggs on a given farm increased as their age increased. In total 32.1% or 124 of the 386 hogs shed ascarid eggs at some point in their lifetime.

Table III presents summary data for the three measures of ascarid burden used in this study: 1) number of intestinal ascarids at slaughter; 2) lifetime burden based on egg counts; 3) liver score for milk spots. There was a wide range in the percent of hogs on each farm that had ascarids present in the small intestine at slaughter: 0.0% on farm 11 to 96.2% on farm 7. For the 133 hogs (34.6%) that had intestinal ascarids, the average number of ascarids was 12.3 and the median was 4 ascarids. The highest ascarid count was 146.

On an individual basis 286 hogs had a lifetime burden of 0; that is to say they did not have any ascarid eggs in the four fecal samples taken up until the time they reached approximately 154 days of age. The other 100 hogs had an average lifetime burden of one, with values ranging from 0.012 to 9.453. All farms had milk spot lesions in at least 25% of the hogs. Overall, only 17.6% of the hogs were free of milk spot lesions. It was possible to have extensive liver lesions in the absence of patent infections. On farm 11 no ascarids were found and none of the hogs ever shed ascarid eggs, yet 89.3% of the hogs had milk spot lesions on their livers. The 16 milk spot lesion specimens submitted for

Table II. Percentage of Hogs Shedding Ascarid Eggs at Various Times for 15 Prince Edward Island Swine Herds, 1987

Farm number	% of hogs[a] with +ve fecal exams at approximately:					% of hogs that ever had a +ve fecal exam
	78 days (26 kg)	107 days (43 kg)	133 days (60 kg)	154 days (74 kg)	market (97 kg)	
1	0.0	0.0	0.0	7.1	6.9	10.3
2	0.0	0.0	12.5	25.0	37.5	41.7
3	0.0	0.0	10.3	7.7	10.7	17.2
4	0.0	0.0	0.0	0.0	7.1	7.1
5	21.7	8.3	37.5	50.0	30.4	66.7
6	0.0	0.0	4.2	4.2	8.3	12.5
7	3.9	69.2	96.1	100.0	96.1	100.0
8	0.0	0.0	0.0	24.1	44.8	55.2
9	0.0	0.0	0.0	0.0	0.0	0.0
10	0.0	0.0	11.1	33.3	16.7	44.4
11	0.0	0.0	0.0	0.0	0.0	0.0
12	0.0	5.6	0.0	0.0	5.9	11.1
13	0.0	0.0	3.9	4.0	7.7	7.7
14	0.0	0.0	0.0	0.0	8.0	8.0
15	0.0	69.0	86.2	92.9	100.0	100.0
Total[b]	1.6	10.7	17.9	23.4	26.1	32.1

[a] The denominator is the number of hogs examined (approximately 30) at each time period.

[b] Total: % of all hogs that had positive fecal exams at each measurement

Table III. Indices which reflect Ascarid Burden for 15 Prince Edward Island Swine Herds, 1987

Farm number	% hogs with ascarids	No. of ascarids in hogs that have ascarids: mean (sd)	Lifetime burden[a] : mean (sd)	% of hogs with the following number of milk spots: 0	1 - 6	>6
1	20.7	1.7 (0.8)	0.003 (0.013)	31.0	55.2	13.8
2	41.7	14.8 (22.4)	0.179 (0.558)	8.3	54.2	37.5
3	41.4	2.9 (3.3)	0.065 (0.206)	13.8	55.2	31.0
4	14.3	1.0 (0.0)	0.000 (0.000)	7.1	39.3	53.6
5	39.1	8.1 (3.5)	0.216 (0.320)	0.0	26.1	73.9
6	20.8	1.6 (0.5)	0.003 (0.013)	54.2	45.8	0.0
7	96.2	14.8 (15.2)	1.831 (2.082)	3.8	19.2	76.9
8	51.7	4.6 (2.9)	0.045 (0.147)	3.4	72.4	24.1
9	6.9	1.5 (0.7)	0.000 (0.000)	17.9	57.1	25.0
10	5.6	2.0 (---)	0.051 (0.115)	5.6	27.8	66.7
11	0.0	--- (---)	0.000 (0.000)	10.7	39.3	50.0
12	5.6	1.0 (---)	0.002 (0.009)	29.4	52.9	17.6
13	15.4	7.3 (11.2)	0.025 (0.126)	73.1	19.2	7.7
14	48.0	1.9 (1.1)	0.000 (0.000)	8.0	24.0	68.0
15	93.1	31.9 (30.7)	1.309 (1.292)	0.0	34.6	65.4
Total[b]	34.6	12.3 (19.8)	0.259 (0.853)	17.6	42.1	40.3

[a] Lifetime burden: for detailed calculation see Appendix A

[b] Total: calculations based on all hogs slaughtered

histopathology were all diagnosed as being consistent with ascarid migration.

Regression of the number of ascarids on the egg count at slaughter yielded a coefficient of determination (r^2) of 0.505. The inclusion of a squared term of egg count increased the r^2 to 0.735.

One-way analysis of variance showed that the number of intestinal ascarids varied significantly ($p=0.01$) with the category of liver lesions. Hogs with severe liver lesions (greater than six milk spots) had an average of 6.7 ± 1.4 intestinal ascarids versus 2.7 ± 0.6 for those with mild liver lesions and 2.5 ± 1.2 for hogs with no liver lesions. Mean lifetime burden rose with increasing levels of severity of liver lesions ($p<0.01$). The average time from the onset of fecal shedding until slaughter was directly related to the severity of the liver score ($p<0.01$), being longest for hogs with severe liver lesions. The averages were 3.1 ± 1.6 days for hogs with no liver lesions, 15.9 ± 2.3 days for hogs with mild liver lesions, and 26.1 ± 2.9 days for hogs with severe liver lesions. The proportion of hogs that never shed eggs in each category was 93%, 68% and 59% respectively.

Farm level data for anteroventral pneumonia and atrophic rhinitis are presented in Table IV. Although 45.2% of the hogs showed no evidence of anteroventral pneumonia, there was a wide variation in the percentage of hogs per farm affected. On farm 6 only 4.2% of the hogs were free of anteroventral pneumonia upon inspection, whereas 82.8% of the hogs on farm 3 had no evidence of anteroventral pneumonia. Nine percent of the hogs had an atrophic rhinitis score of five, but this ranged from 0% to 57% on individual farms.

Table IV. Levels of Anteroventral Pneumonia and Atrophic Rhinitis in 15 Prince Edward Island Swine Herds, 1987

Farm number	% of hogs with the following % of total lung volume affected by anteroventral pneumonia:				Atrophic rhinitis[a] :	
	0	1 - 9	10 - 25	>25	score from 0 to 5 mean (SD)	% hogs scored five
1	13.8	51.7	20.7	13.8	2.0 (1.1)	0
2	29.2	37.5	25.0	8.3	1.1 (1.0)	0
3	82.8	10.3	3.4	3.4	1.6 (1.4)	3
4	51.9	44.4	0.0	3.7	1.7 (1.4)	10
5	30.4	34.8	13.0	21.7	1.3 (1.1)	0
6	4.2	70.8	20.8	4.2	1.7 (0.7)	0
7	50.0	42.3	7.7	0.0	0.6 (0.6)	0
8	27.6	51.7	10.3	10.3	3.8 (1.6)	57
9	51.7	44.8	0.0	3.4	0.8 (0.7)	0
10	44.4	33.3	11.1	11.1	2.0 (1.6)	17
11	40.7	55.6	3.7	0.0	1.0 (0.5)	0
12	35.3	58.8	0.0	5.9	2.1 (1.7)	18
13	80.0	16.0	4.0	0.0	1.2 (0.7)	0
14	56.0	40.0	4.0	0.0	2.5 (1.6)	24
15	76.0	4.0	16.0	4.0	1.0 (1.2)	4
Total[b]	45.2	39.6	9.3	5.9	1.6 (1.4)	9

[a] Atrophic rhinitis: for method of scoring see text

[b] Total: calculations based on all hogs marketed

2.4 Discussion

2.4.1 Measures of ascarid burden

This study used purposive as opposed to random sampling and thus results of measures of prevalence should not be extrapolated to all herds in PEI. However, it is of interest that an abattoir survey of Saskatchewan pigs (28) found intestinal ascarids in 36.7% of 2500 market hogs sampled, similar to 34.6% recorded in the present study. In contrast, the prevalence of livers with milk spots (46%) was almost half of the 82.4% observed in this study. In a study conducted in Sweden (24), 46.5% of 489 pigs were shedding ascarid eggs at the beginning of the fattening period (approximately 26 kg) compared to 1.6% for pigs of the same weight in this study. At slaughter 66.5% of the Swedish pigs had intestinal ascarids. Although this would seem to indicate a higher level of infection, the percentage of hogs with milk spots, 72.1% of 818 hogs examined, was less than the 82.4% recorded in the present study. In the Swedish investigation 201 pigs were treated with a single dose of anthelmintic (7.5 mg/kg of levamisole) at the start of the fattening period, then were mixed with 196 untreated controls. The proportion of hogs with intestinal ascarids at slaughter was 54.1% and 60.7% respectively. The mean number of intestinal ascarids per infected hog was 9.5 and 8.4 for the two groups, which is in general accordance with the mean of 12.3 ascarids in this study and the low numbers reported above.

2.4.2 The relationship between intestinal ascarids and fecal egg counts

In examining the association between the measures of ascarid burden it was noted that the proportion of hogs with ascarids (34.6%) was higher than the proportion of hogs that were shedding ascarid eggs at slaughter (26.1%). The major reason for this difference was that 44 hogs had intestinal ascarids, but

never shed eggs. This scenario would be expected since 39% of the hogs that had ascarids had only one or two ascarids, and these may have been male ascarids only. However, the difference was actually greater than it appeared as there were 16 hogs that shed ascarid eggs at slaughter, but did not have intestinal ascarids. The phenomenon of fecal shedding of eggs in the absence of intestinal ascarids has been reported previously (29). It is possible that these were uninfective eggs (either unfertilized or not sufficiently developed) which the hogs ingested and passed. After experimental inoculation of three weaner pigs, Schwartz (5) found that most, if not all, of the nonembryonated eggs were eliminated by two of the pigs within two days.

The egg count at slaughter accounted for approximately 50% (r^2=0.505) of the variation in the number of ascarids in hogs. Bindseil (29) found the correlation coefficient (r) between the number of ascarids and egg count at slaughter to be 0.44 (equivalent to an r^2 of 0.19). The much lower value he obtained could be due to either lack of precision or a consistent bias in his egg-counting technique. The r^2 was raised to almost 75% by the addition of a quadratic term (egg count squared). The strength of this association would suggest that lifetime burden, which is comprised of a series of fecal egg counts, would reflect the ascarid burden over time.

2.4.3 The relationship between intestinal ascarids and milk spots

It was not anticipated that such a large discrepancy between the proportion of hogs with ascarids (34.6%) and the proportion of hogs with milk spot lesions (82.4%) would be found.

A total of 12 hogs on seven different farms had ascarids, but no milk spots. This is easily explained, as milk spot lesions do resolve (3,7,38,39). Copeman and Gaafar (7) found that milk spots were no longer visible 35 days after primary

inoculation with ascarid eggs, although greater persistence was observed after secondary inoculation. Eriksen (3) found that healing was almost complete 21 days after primary or secondary inoculation. In the Saskatchewan survey (28), of 917 hogs found to have intestinal ascarids, 348 (38%) did not have milk spot lesions.

Since milk spot lesions resolve, the presence of milk spots at slaughter indicates a recent uptake of eggs and larval migration (3,40,41). This does not necessarily mean that hogs with milk spots at slaughter were the most recently infected.

Current knowledge of the dynamics of ascarid infection and host resistance helps to elucidate the distribution of milk spot lesions. Host defence plays an important role in the pathogenesis of the lesions including the formation of milk spots in the liver. Resistance mechanisms of the host work at all sites of ascarid migration towards elimination of the parasite (3). Ascarid larvae are eliminated during milk spot formation in the liver and during migration in the lungs (42). There is also a massive expulsion of larvae from the small intestine during the early prepatent period (3,41,43). There is an inverse relationship between the number of ascarid eggs ingested and the number of ascarids established in the small intestine (23,40,43,44). The expulsion of larvae from the gut does not seem to take place following primary inoculation with a small number of eggs (23,43,44), possibly because there is insufficient antigenic material released to elicit a response (3). After repeated exposure to ascarid antigens the lesions are similar to those which follow the initial exposure, however, an anamnestic response occurs (45,46,47). The acquired immunity is manifested by an earlier and more intense reaction against the parasite over the whole migratory route of the larvae (3). The occurrence of milk spots in the liver is augmented in pigs that

have become resistant after repeated inoculations (3,40).

In this study, hogs with severe liver lesions were the earliest to be infected. On average they started shedding ascarid eggs 26 days before slaughter. Since the prepatent period is approximately 60 days, these hogs were infected approximately 86 days before slaughter.

Hogs with severe liver lesions had the highest mean number of ascarids. Eriksen et al (40) also found that intestinal burdens were highest in pigs where inoculations started early in life. The early ascarid burden in these hogs may cause considerable environmental contamination. This in turn may promote frequent egg consumption and larval migration with resultant liver lesions. The tendency of lifetime burden to increase with the severity of liver lesions supports this hypothesis.

Milk spots occurred in the absence of intestinal ascarids in 51.3% of the hogs. This difference was also pronounced on a farm basis. On farms 10, 11, and 12, which had a maximum of one hog with two ascarids, there were milk spot lesions on the livers of 94.5%, 89.3% and 70.5% of the hogs respectively. Two possible explanations for this phenomenon are: 1) there had been a recent uptake of eggs and larval migration, but not enough time had elapsed to allow development of macroscopically detectable ascarids; and 2) the infection had been cleared, but the lesions had not yet resolved.

Late third-stage larvae arrive in the small intestine eight to ten days after ingestion of eggs (2). Some larvae are spontaneously eliminated from the small intestine, concomitant with the third ecdysis, from days 10 to 15 after ingestion. Early 4th stage larvae are only about 2mm in length (2) and thus could easily go undetected. The fourth and final moult occurs approximately 21-25 days after ingestion which marks the onset of another period of larval expulsion (5). At 24

days post infection larvae are approximately 9 mm long. It seems reasonable that for hogs infected in the 3 weeks previous to slaughter, ascarids would probably not be detected in the small intestine and secondly, that hogs could clear their infection before liver lesions were completely resolved.

In this study it was found that milk spots were more than twice as prevalent as ascarids in the small intestine at slaughter. There was a high correlation between counts of intestinal ascarids and concomitant fecal egg counts. This lends credence to the calculated variable lifetime burden as a reflection of ascarid burden over time. The ability to predict ascariasis according to the presence or absence of milk spots will be explored in the next chapter. In chapter four, the variables described here will be used to ascertain the effects of ascarid burden on swine production.

3. ABATTOIR SURVEILLANCE

3.1 Introduction

The migration of ascarid larvae through swine livers stimulates a host immune response which has been well documented (3,7,42). The resultant focal areas of interstitial hepatitis are commonly referred to as milk spots or white spots.

Helminths other than ascarids which induce hepatic lesions in swine are Fasciola hepatica, Cysticercus tenuicollis (48), and Stephanurus dentatus (49). However, none of these parasites are indigenous to PEI where this study took place. Other ascarids, Toxocara canis, Toxocara cati and Parascaris equorum, can cause milk spots in pigs under experimental conditions (42). A serological survey of Toxocara- and Ascaris- infection in Britain (50) concluded that species of Toxocara were unlikely to be an important cause of milk spot lesions under field conditions.

Although the presence of milk spots is routinely recorded at federally inspected abattoirs in Canada and is commonly used as a measure of ascariasis (24,28,51), this technique has not been formally evaluated as a screening test for ascariasis.

Sensitivity, specificity and predictive values are useful attributes when evaluating a test (52,53). Sensitivity is the ability of a test to correctly detect diseased animals, whereas specificity is the ability of a test to correctly detect non-diseased animals. Predictive values are affected by sensitivity, specificity and the true prevalence of disease. The positive predictive value is the proportion of animals with a positive test result that actually have the disease. The negative

predictive value is the proportion of animals with a negative test result that do not have the disease.

The objective of this chapter is to critically assess the abattoir surveillance of liver lesions as a screening test for ascariasis. This study will determine whether routine inspection of swine livers reflects ascarid exposure in individual hogs. It will also assess the ability of routine inspections to identify farms with ascarid problems.

3.2 Materials and Methods

Information pertinent to this investigation was gathered according to procedures described in Chapter 2. The following data were collected on an individual basis for 380 hogs marketed between August 5th and November 25th, 1987:

1) primary product inspectors' scores for the number of milk spots on the liver;
2) the author's count of milk spots on every fifth of the above livers examined by the inspectors;
3) the number of intestinal ascarids present at slaughter; and
4) a series of fecal ascarid egg counts (taken at approximately 78,107, 133 and 154 days of age and again at slaughter).

It was desirable to establish the accuracy with which liver scores for milk spot lesions could be assigned during rapid post mortem inspections by Agriculture Canada primary product inspectors. The statistic kappa was used to assess the agreement between the inspectors' and the author's scores for liver lesions.

The ability to predict ascariasis in individual hogs, based on the presence of milk spots on livers, was assessed by calculating sensitivity, specificity, and negative and positive predictive values. The presence of milk spots was determined by the inspectors' scores. Ascariasis was measured in two ways: 1) by the presence of intestinal ascarids; and 2) one or more positive fecal egg counts during the hog's life.

The following farm data from Chapter 2 were used to assess liver scores as a screening test for farms with ascarid problems:

1) the percentage of hogs per farm with milk spot lesions on their livers;
2) the percentage of hogs per farm with intestinal ascarids at slaughter; and
3) the average lifetime burden of ascarids for each farm.

Two 2x2 tables were created in order to calculate sensitivity, specificity and predictive values of the farm level observations. In the first table (Table VIII), points of division were chosen to ensure approximately equal groups of farms in each category. Farms were considered to have a low level of ascariasis if fewer than 35% of the hogs had intestinal ascarids at slaughter. The remaining farms were considered to have a high level of infection. Similarly, farms were considered to have a low level of liver lesions if fewer than 90% of the study hogs had liver lesions. The remaining farms constituted the group with a high level of liver lesions.

In the second 2x2 table (Table IX), farms were considered to have a low level of ascariasis infection if their average lifetime burden was less than one. (A lifetime burden of one is equal to the average lifetime burden for hogs that had intestinal ascarids.) The group of farms considered to have a low level of

liver lesions included farms where fewer than 95% of the study hogs had liver lesions. The 95% cut-off point was chosen to eliminate false negative results.

3.3 Results

Kappa was equal to 0.783 for liver scores from the inspectors as compared to those of the author (Table V). The calculations are included in Appendix C. Only one of the 79 livers was scored negative by the inspector, but was found to have greater than six spots by the author.

The presence of milk spots indicated the presence of intestinal ascarids with a sensitivity and specificity of 90.8% and 22.0% respectively. The 34% prevalence of intestinal ascarids in this group of hogs gave rise to positive and negative predictive values of 37.7% and 82.1% respectively. These calculations were based on the presence or absence of milk spots and the concurrent presence or absence of intestinal ascarids as presented in Table VI.

The sensitivity and specificity for milk spots as an indicator of ever having a positive fecal egg count were 95.8% and 23.8%. The positive and negative predictive values were 36.7% and 92.5% respectively (Table VII).

The sensitivity and specificity for the percentage of hogs per farm with liver lesions as an indicator of the percentage of hogs per farm with intestinal ascarids were 85.7% and 75.0% respectively (Table VIII). The positive predictive value was 75.0% and the negative predictive value was 85.7%.

Table IX shows that the percentage of hogs per farm with liver lesions was a very sensitive (100%) indicator of farms with high average lifetime burdens, but was less specific (85%). The positive and negative predictive values were 40% and 100% respectively.

Table V. Agreement Between Inspectors' Estimate and Investigator's Count for the Number of Milk Spots on the Livers of 79 Market Hogs

Number of livers with the following number of milk spots:

		Inspectors' Estimate			
		0	1 - 6	> 6	Total
Investigator's Count	0	5	0	0	5
	1-6	5	27	0	32
	> 6	1	4	37	42
		11	31	37	79

Table VI. Crosstabulation of the Presence or Absence of Ascarids in the Intestines and Milk Spots on the Livers of 380 Market Hogs

		Intestinal Ascarids		
		+	-	
Milk Spots	+	118	195	313
	-	12	55	67
		130	250	380

Sensitivity = 90.8%

Specificity = 22.0%

Positive Predictive Value = 37.7%

Negative Predictive Value = 82.1%

Table VII. Crosstabulation of Having One or More Positive Fecal Egg Counts and the Presence or Absence of Milk Spots on the Livers for 380 Market Hogs

		Fecal Egg Count Ever Positive		
		+	-	
Milk Spots	+	115	198	313
	-	5	62	67
		120	260	380

Sensitivity = 95.8%

Specificity = 23.8%

Positive Predictive Value = 36.7%

Negative Predictive Value = 92.5%

Table VIII. Crosstabulation of the Percentage of Hogs with Intestinal Ascarids on the Farm versus the Percentage of Hogs Per Farm with Milk Spot Lesions for 15 PEI Swine Herds

		% of Hogs/Farm with Intestinal Ascarids		
		≥ 35%	< 35%	
% of Hogs/Farm with Milk Spot Lesions	≥ 90%	6	2	8
	< 90%	1	6	7
		7	8	15

Sensitivity = 85.7%

Specificity = 75.0%

Positive Predictive Value = 75.0%

Negative Predictive Value = 85.7%

Table IX. Crosstabulation of the Average Farm Lifetime Burden versus the Percentage of Hogs Per Farm with Milk Spot Lesions for 15 PEI Swine Herds

		Number of Farms with an Average Lifetime Burden		
		≥ 1	< 1	
% of Hogs/Farm with Milk Spot Lesions	≥ 95%	2	2	4
	< 95%	0	11	11
		2	13	15

Sensitivity = 100%

Specificity = 85%

Positive Predictive Value = 40%

Negative Predictive Value = 100%

3.4 Discussion

Kappa values greater than 0.75 represent excellent agreement beyond chance (54). The author counted more milk spots than the inspectors did. This difference may be attributed to increased time allotted to the author for examination of the livers. However, it can be concluded that rapid post mortem inspection of livers is a valid technique for assessing the severity of milk spots.

Using milk spots as a screening test for hogs with ascariasis yielded similar results for both measures of ascariasis. The low specificities and positive predictive values are related to the high number of false positive results. As was evident from Chapter 2, there are many hogs with liver lesions that do not have intestinal ascarids at slaughter and never shed ascarid eggs in their feces. The best explanation for this is that the host immune response was able to overcome the infection before an adult population was established in the intestine. Although the presence of milk spots does not necessarily coincide with an ascarid infection, the high negative predictive values indicate that ascariasis is unlikely in the absence of liver lesions at slaughter.

Predictive values will vary with the true prevalence of ascariasis. The hogs in this study were not dewormed, so the observed prevalence of ascariasis may be an overestimate of the true prevalence on these farms. As the true prevalence of disease decreases, the negative predictive value increases, so the absence of liver lesions would still be a reliable indicator of the absence of infection.

There is a higher prevalence of livers with milk spots lesions during the summer (55,56) when temperatures are more favorable for the development of ascarid eggs (55,57). It is not known whether the reduction in the number of hogs with milk spots in the winter would be matched by the reduction in the

number of hogs with intestinal ascarids. Although there would be fewer new infections, the existing intestinal ascarids may persist after the accompanying liver lesions had resolved. Therefore it is difficult to predict how sensitivity, specificity and predictive values would differ during the winter months.

The results of the farm level analysis must be interpreted with caution due to the small sample size. For Table VIII, the points of division for farms into high and low categories for ascarid infection and liver lesions were chosen to ensure approximately equal groups in each category. Using these criteria, 86% of the farms with a high percentage of infected hogs were identified by having high levels of liver lesions.

It will be shown in Chapter 4 that a lifetime burden greater than two has a detrimental effect on growth rates of swine. Although only 18 hogs had a lifetime burden exceeding two, 17 of these hogs were from two farms which had average lifetime burdens of 1.3 and 1.8. An average farm lifetime burden of one was chosen as the division point to see if abattoir data could facilitate detection of these farms. The 95% cut-off point (for the percentage of hogs per farm with liver lesions) was selected to ensure that there were no false negatives. This resulted in 100% sensitivity and negative predictive value. Although it would appear that this test would detect farms with serious ascarid problems it must be remembered that the divisions were specific for the data from this study and consequently may not have a broad application.

Chapter 2 showed that mean lifetime burden rose with increasing levels of severity of liver lesions. However, only 1.2% of the hogs had a lifetime burden exceeding two, while 82.4% of the hogs had milk spots on their livers with 40.3% in the category with greater than six spots. Therefore, inspection of swine livers would not selectively detect individual hogs whose lifetime burden was greater

than two.

In summary, the routine abattoir surveillance of swine livers is a sensitive, but not a specific test for ascariasis in individual hogs. However, ascariasis is highly unlikely in the absence of liver lesions. It was possible, with the results from this study, to construct divisions which allowed the detection of farms with high levels of ascarid infection based on the percentage of hogs with liver lesions at slaughter. It would not be possible to identify individual hogs with heavy ascarid burdens based on abattoir surveillance of liver lesions.

4. A PATH ANALYSIS OF FACTORS AFFECTING AVERAGE DAILY GAIN IN SWINE

4.1 Introduction

Ascariasis, anteroventral pneumonia and atrophic rhinitis are common in all swine rearing areas of the world (11,58). Although it has been traditionally accepted that these diseases are of economic importance, the literature contains conflicting reports as to their effects on production and their interrelationships (36,58,59,60,61).

The pathological effects of ascarids in swine consist of damage inflicted during larval migration and the effects of adult ascarids in the small intestine. Ascariasis has also been targeted as an etiologic factor in the development of esophagogastric ulcers in swine (62,63,64). Although ulcers have been induced experimentally in swine by sequential inoculation with large doses of ascarid eggs or larvae (62,63), there is strong evidence that ascariasis is not a factor in the development of gastric lesions under field conditions where the size of the challenge would probably be much smaller (65).

Larval migration through the liver and lungs may precipitate losses through condemnation of livers at slaughter and a possible enhanced susceptibility to pneumonic pathogens (11,59,66,67). The pathogenicity of adult ascarids is less well defined (4). Many of the experimental investigations used very large inocula to establish infections, therefore they are probably not relevant to the situation to which pigs are exposed on farms (23). There is little quantitative information available on the effect of naturally acquired ascariasis on the growth rate of individual hogs under field conditions (24).

A comprehensive review of the association between anteroventral pneumonia and performance (59) tallied ten studies which reported no significant effect and 13 which reported decreased ADG or an increased feed to gain ratio. The authors stated that either enzootic pneumonia was inconsistent in its effect on performance parameters, or more likely, differences in study design led to variation in the results. Furthermore, the authors recognized that resolution of pneumonic lesions and the presence of other diseases which may influence performance (ascariasis, atrophic rhinitis and diarrhoea) would account for some of the inconsistencies.

The association between pneumonia and atrophic rhinitis is controversial, as are the alleged effects of atrophic rhinitis on production. Of 19 studies reviewed (59), 11 reported a decrease in ADG due to atrophic rhinitis, while the remaining eight found no significant effects.

The relationship between these disease variables and production may be further elucidated through the use of path analysis. Path analysis entails an investigation of variables which have been assigned a weak causal ordering based on temporal, plausibility or biological associations. It is generally more powerful than an analysis of data in the absence of a postulated causal structure (68). Another advantage of path analysis is that the effects of variables, other than the one of immediate interest, are controlled through their inclusion in the analysis.

Path analysis allows identification of direct, indirect and total effects which are presumed to be causal. The total effect is the sum of the direct and indirect effects. Direct effects are those effects which are not mediated through an intervening variable. In contrast, indirect effects are mediated through one or more intervening variables. For example, if atrophic rhinitis influences ADG

directly, that would be a direct effect. If atrophic rhinitis affects pneumonia which in turn affects ADG, atrophic rhinitis would have an indirect effect on ADG. The total effect of a variable predicts the magnitude and direction of change in the dependent variable (outcome) which would result from a change of one unit in that variable, all other variables being held constant.

The primary objective of this study was to examine the effect of ascarids on swine production. This investigation is strengthened by controlling for potential confounding variables including demographic factors and other variables which may affect production. An advantage of using analytic control is that the effects of control variables can also be assessed (69). This chapter examines the relationships among the variables and their effects on production in a path model.

4.2 Materials and Methods

Complete data for use in regression analyses were available for 352 hogs which were born between March 8th and March 28th, 1987 and marketed between August 5th and November 26th, 1987. The measures of growth rate, ascarid burden and confounding variables have been described in Chapter Two. A summary of the hog data is presented in Table X. Growth rates were measured as average daily gain from birth to slaughter. Measures of ascarid burden included the number of ascarids in the small intestine at slaughter and the lifetime burden. Lifetime burden is a composite measure of ascarid burden based on the magnitude of a series of fecal egg counts and the duration of infection.

Data for putative confounding variables included demographic characteristics, the presence or absence of anteroventral pneumonia and the scores for atrophic rhinitis.

Table X. Summary of Data Used in a Path Model to Study the Effects of Ascarids, Anteroventral Pneumonia and Atrophic Rhinitis on Growth of Swine

Variable	Lowest farm[a]	Highest farm[a]	All Hogs
average days to market[b]	160±12	218±22	189±22
average dressed carcass weight (kg)	71.2±5.0	81.9±5.6	77.0±5.9
average ADG (kg/day)	0.440±0.048	0.601±0.051	0.519±0.071
% fecals ever positive	0.0	100.0	32.1
% hogs with intestinal ascarids	0.0	96.2	34.6
average no. ascarids in hogs that had intestinal ascarids	0	31.9±30.7	12.3±19.8
average lifetime burden	0	1.83±2.08	0.26±0.85
average lifetime burden for hogs whose lifetime burden > 0	0	1.83±2.08	1.00±1.44
% hogs with anteroventral pneumonia	17	95	54
% hogs with atrophic rhinitis score 5	0	57	9
average atrophic rhinitis score (0-5)	0.6±0.6	3.8±1.6	1.6±1.4

[a] for each variable

[b] mean ± standard deviation

The effects of the farm of origin, litter of birth (nested within farm) and sex of the hogs were removed from the model by forcing appropriate dummy variables into each regression equation. Variation in these predetermined variables, called exogenous variables, is not of concern to the investigation so their effects have not been reported for each regression. ADG was regressed on the exogenous variables in order to identify their contribution to variation in ADG.

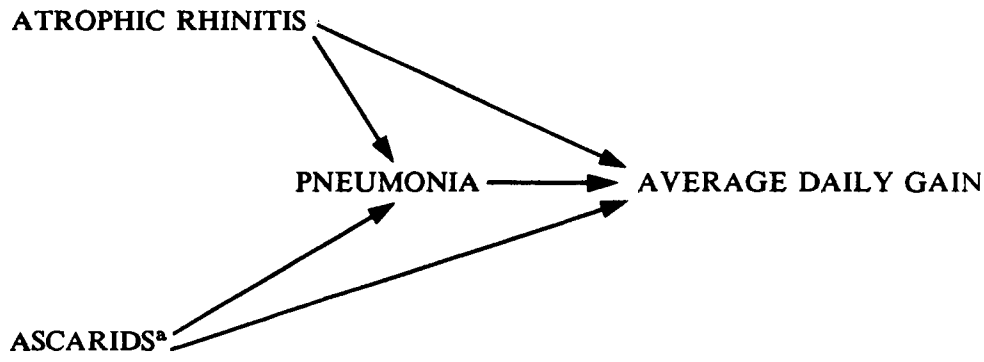
A causal model of factors was postulated and is contained in Fig. 1; the description of the variables is contained in Table XI. In a weak causal model, all causal arrows must flow from left to right. This is also a recursive causal model in that variables are postulated to affect all variables situated to their right, based on temporal or plausibility associations. For example, atrophic rhinitis and measures of ascarid burden were both placed to the left of anteroventral pneumonia as it is plausible that they could influence its occurrence, but not vice versa. The path analysis was executed with each of the two measure of ascarid burden: 1) the number of intestinal ascarids; and 2) lifetime burden.

The regression analyses were performed using the general linear models procedure in the Statistical Analysis System for Personal Computers (70). The principal analysis examined the effects of the variables on ADG for individual hogs, using lifetime burden as the estimate of ascarid burden.

The direct effects of each variable on ADG (the outcome or dependent variable) were obtained from the following regression analyses. ADG was regressed on atrophic rhinitis, pneumonia and lifetime burden. The regression was repeated including a squared term for lifetime burden.

The direct effects of atrophic rhinitis and lifetime burden on pneumonia

Fig. 1. A Postulated Causal Path Model of Factors Affecting Average Daily Gain in Swine



[a] ASCARIDS as measured by:1) number of intestinal ascarids; and
2) lifetime burden

Table XI. Variables Used in a Path Model to Study Their Effect on Average Daily Gain in Swine

Variable	Description
AVERAGE DAILY GAIN	continuous variable - average daily gain from birth to slaughter (range from 0.317 - 0.707 kg/day)
FARM	14 dummy variables
LITTER(FARM)	63 dummy variables for litters nested within farms ie. farm 1 litter 1, farm 1 litter 2, etc.
SEX	2 dummy variables: - females - castrated males - others (includes hermaphrodites, cryptorchids and boars)
PNEUMONIA	1 dummy variable: 1 = presence of anteroventral pneumonia 0 = no anteroventral pneumonia
ATROPHIC RHINITIS (categorical)	10 dummy variables: - atrophic rhinitis scores (scale from 0 - 5 with increments of 0.5)
ATROPHIC RHINITIS (dichotomous)	1 dummy variable: 1 = score of 5 0 = scores from 0 to 4.5 inclusive
ASCARIDS	continuous variable - number of macroscopic ascarids at slaughter (range from 0 - 146)
LIFETIME BURDEN	continuous variable - estimate of lifetime ascarid burden (range from 0 - 36.5)

were obtained from a regression with pneumonia as the dependent variable.

Atrophic rhinitis was considered both as an 11 category variable and as a two category variable.

The coefficients which corresponded to total effects provided the amount of change in ADG anticipated if that variable changed by one unit. These estimates were used to express the magnitude of the effect that variables had on ADG.

The following secondary analyses were performed:

1) the regression was repeated substituting number of intestinal ascarids as the measure of ascarids in order to obtain its direct effect on ADG;

2) a temporally restricted model was examined in which lifetime burden and growth rate were calculated up to the time of the third farm visit (average age 133 days, average weight 60 kg) to see if the effects on ADG up to the third farm visit were the same as those observed over the lifespan of the hogs;

4) the analyses were performed on a farm by farm basis; and

5) the regression was performed with an additional variable, the product of atrophic rhinitis and pneumonia, to check for interaction between these two variables.

4.3 Results

The regression of ADG on the exogenous variables farm, litter and sex yielded a coefficient of variation (r^2) of 0.700.

As an 11 category variable, atrophic rhinitis did not significantly affect pneumonia, but had a significant detrimental affect on ADG ($p<0.001$). The coefficients for atrophic rhinitis scores from 0 to 4.5 did not differ significantly from each other, but were different than the coefficient for a score of five. For

this reason, the variable atrophic rhinitis was dichotomized into scores of five and scores less than five.

The final model used in the path analysis accounted for 75.4% of the variation in ADG. It included the variables farm, litter nested within farm, sex, atrophic rhinitis (dichotomous), pneumonia, lifetime burden, lifetime burden squared and an interaction term for atrophic rhinitis and pneumonia. There were no indirect effects, therefore the direct effects equal total effects. The path model in Fig. 2 presents the direct effects of disease variables on ADG.

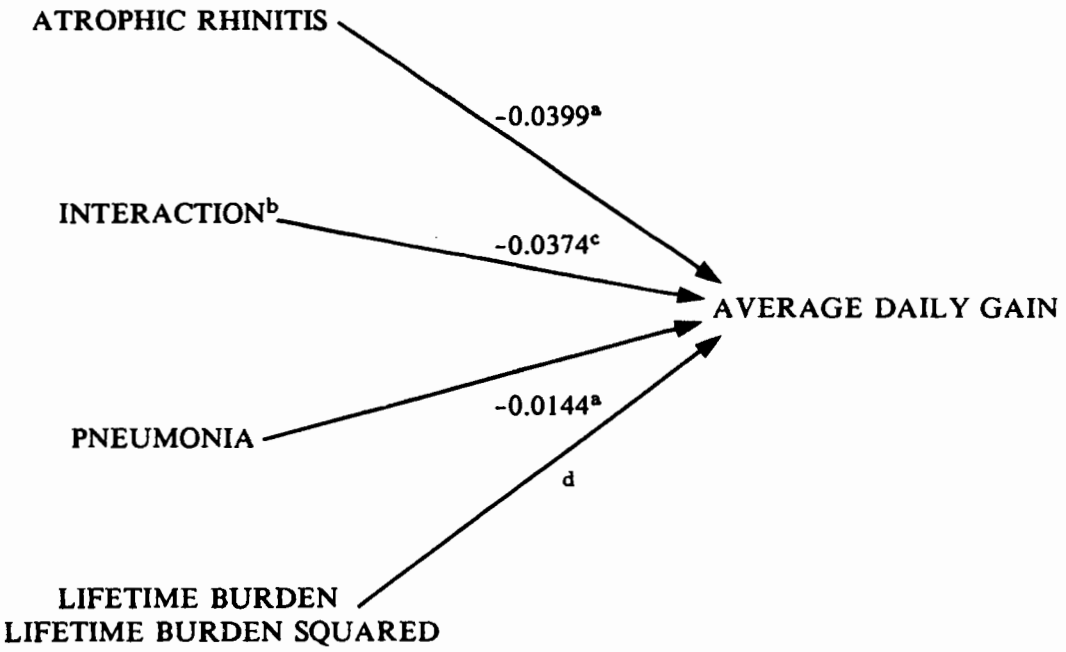
Anteroventral pneumonia and atrophic rhinitis each had a significant detrimental direct effect on ADG ($p<0.001$). The presence of anteroventral pneumonia corresponded with a 0.0144 kg/day decrease in ADG (2.8% of the average ADG). An atrophic rhinitis score of five corresponded with a 0.0399 kg/day decrease in ADG (7.7% of the average ADG) when compared to scores less than five.

The interaction term for anteroventral pneumonia and atrophic rhinitis had a significant detrimental effect on ADG ($p<0.05$). Concurrent pneumonia and an atrophic rhinitis score of five would result in a decrease in ADG of 0.0374 kg/day (7.2% of the average ADG) in addition to the individual effects of these two variables.

Lifetime burden had a detrimental direct effect on ADG ($p= 0.11$). The addition of a squared term for lifetime burden significantly improved the model ($p<0.05$) and lifetime burden was rendered insignificant due to the strong collinearity with its squared term. The relationship between lifetime burden and ADG is depicted in Fig. 3. ADG started to decrease once lifetime burden exceeded a value of two.

When the number of intestinal ascarids was used as the measure of ascarid

Fig. 2. The Direct Effects of Disease Variables on Average Daily Gain in Swine from a Proposed Path Model



[a] $p<0.001$

[b] Interaction between atrophic rhinitis and pneumonia

[c] $p<0.05$

[d] Average daily gain varies with lifetime burden (LB) as follows: $0.0083(LB) - 0.0020(LB^2)$

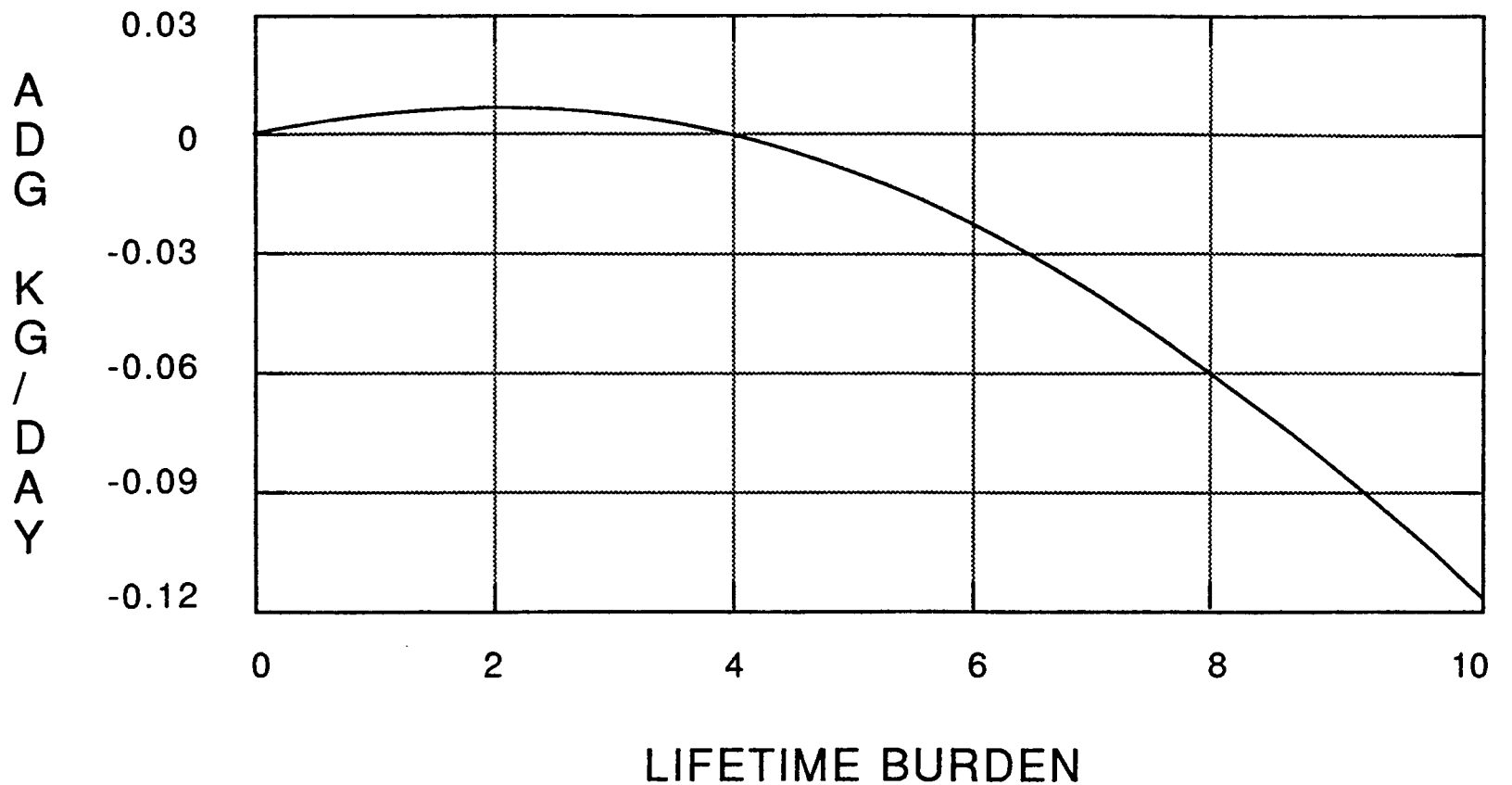


Fig. 3. The relationship between lifetime burden and average daily gain in swine: ADG = 0.0083 (LB) - 0.0020 (LB) ^{2}.

burden, it was not significantly associated with ADG.

The temporally restricted model examined the effects of variables on growth rate until the hogs reached approximately 133 days of age (average 60 kg). The resulting equation was ADG = 0.0368 (LB) - 0.0110 $(LB)^2$. ADG started to decrease once lifetime burden exceeded 1.6 and dropped more rapidly than for the general model which was shown in Fig. 3.

On each of the two farms with the greatest ascarid burden (highest average lifetime burden, greatest percentage of hogs infected and largest number of intestinal ascarids per infected hog) there was a quadratic relationship between lifetime burden and ADG similar to that in Fig. 3. On these farms over 90% of the hogs had intestinal ascarids and average lifetime burdens were 1.3 and 1.8. Lifetime burden did not have a significant effect on ADG for the 13 farms with lower levels of ascariasis.

4.4 Discussion

Performance of hogs is commonly measured by rate of growth (ADG) or the efficiency of feed utilization (amount of feed/kg of gain). It was impracticable to obtain feed conversion data for individual hogs in an observational field study of this size, therefore ADG was chosen as the measure of production.

As would be expected, much of the variation in ADG could be attributed to differences in management between farms, variations in genetic potential between litters, and differences in growth rate due to gender. The variable litter within farm controlled for variation in ADG due to the mothering ability of the sow while the piglets were suckling. This study differs from its predecessors in the number of potentially confounding variables which were controlled through

analysis.

Spindler (71), in 1947, was the first to conduct experiments to evaluate the effects of ascarid infection on weight gain. Although he was able to demonstrate an inverse relationship between ADG and the number of intestinal ascarids, the small sample size and lack of control of confounding variables (such as different diets) invalidates any conclusions.

An initial study by Zimmerman et al (72) looked at the effect of feeding pyrantel, which inhibits hatching of ascarids in the intestine, on both rate of gain and feed efficiency. In a series of experiments they compared performance in pigs fed different levels of pyrantel and high or low protein diets. They found that pyrantel decreased the number of milk spots, larval migration through the lungs and establishment of ascarids in the intestine. Although significant effects were reported on both ADG and feed/gain during the 35 day treatment period, these effects were not consistent across experiments and feeding pyrantel had no significant effects over the entire feeding period. However, one of their experiments demonstrated that pigs raised on concrete had a significantly higher ADG ($p<0.01$) and improved feed conversion ($p<0.10$) compared to those that had access to ascarid contaminated soil.

In a follow-up study (73) there was a significant improvement in both ADG and feed/gain for both the initial 28 day treatment period and the entire feeding period. Pigs that were treated with pyrantel exhibited a 9.1% improvement in ADG over the feeding period (average 18 kg to 98 kg body weight). There was also a significant interaction ($p<0.05$) between not feeding pyrantel and challenge with mycoplasma: no pyrantel increased feed/gain more in mycoplasma-challenged than in unchallenged pigs.

In egg infections, the pathological effects of larval migration through the

liver and lung complicate experiments that are intended to study the effects of the late larval and adult stages in the small intestine (74). In order to study the effects of ascarids in the intestine independent of the effects due to larval migration, Stephenson et al (75) infected young pigs with 200 15-day-old larvae which had undergone migration in rabbits. After eight weeks there were no significant differences in ADG, feed consumption, efficiency of feed utilization, or nutrient absorption between infected and noninfected pigs when diet was controlled.

Forsum et al (76) found that in pigs on a low protein diet, there were no differences in weight gain between uninfected pigs and pigs with an average intestinal burden of 31 ± 25 ascarids. However, pigs with an average burden of 65 ± 11 ascarids gained significantly less weight ($p<.05$) than the uninfected controls regardless of protein levels in the diet. These differences were not observed until about 43 days post infection which corresponds with the period when ascarids are maturing in the small intestine. The authors noted that the reduction in food intake was probably the main contributing factor to growth reduction in these pigs.

In a more recent study by Hale et al (77), pigs infected with 0, 600, 6,000 or 60,000 ascarid eggs had means of 3.2, 11.5, 16.3 and 18.5 intestinal ascarids respectively at slaughter. Regression analyses were performed controlling for initial body weight and sex of the pigs. Feed/gain increased linearly with increasing egg doses ($p<0.01$). There was a linear ($p<0.07$) and a quadratic ($p<0.09$) effect of increasing levels of ascarid egg infection on ADG. The group of pigs with the lightest infection were 13% more efficient at feed conversion and had a 10% higher ADG than the group with the heaviest infection. This study also found that ascarids did not have a significant effect on digestion and

absorption until the ascarids approached maturity.

The only previous observational study of ascariasis in hogs was a field study by Nilsson (24). The performances of hogs with various ascarid burdens were compared. There was no difference in ADG between the hogs with the lowest ascarid burden (negative fecal count at the start of the fattening period and no intestinal ascarids at slaughter) and those with an intermediate burden (maximum fecal count of 10,000 eggs per gram at the start of the fattening period and fewer than 20 ascarids at slaughter). However, the 83 hogs with the lowest ascarid burden had a 4.8% higher ADG over the feeding period than the 75 hogs with the highest ascarid burden (fecal count > 10,000 eggs per gram at the start of the period or > 20 intestinal ascarids at slaughter). The difference in ADG between hogs with high and low ascarid burdens was of a greater magnitude for females than for males. A comparison of pigs gaining less than 0.6 kg/day to those gaining more than 0.7 kg/day showed that there was no significant difference between these groups in the percentage of hogs with intestinal ascarids at slaughter or the number of ascarids they harboured.

The present study agrees with Nilsson's results in that the number of ascarids at slaughter was not associated with ADG.

The general model illustrated that only heavy ascarid burdens decreased growth rates of swine. This is also in agreement with Nilsson. Although performance of hogs may only be affected by heavy ascarid burdens, this threshold was exceeded on individual farms. Only 18 hogs had a lifetime burden greater than two, but 17 of these hogs were from the two farms with the highest average lifetime burdens.

From the analysis of the temporally restricted model (up to an average hog weight of 60 kg), the difference between a lifetime burden of two and three was

associated with a 0.019 kg/day decrease in ADG. The same lifetime burden from the general model was associated with a decrease of only 0.002 kg/day in ADG. These figures represent a 4.2% and 0.4% decrease in ADG respectively. This may indicate that ascarid infection has a greater deleterious effect on young growing pigs and that compensatory growth occurs later on. It is also possible that the damage incurred during the migratory phase contributes to this lag in growth. The two farms with the highest average lifetime burdens had a high percentage of hogs infected at an early age.

Both atrophic rhinitis and the presence of anteroventral pneumonia had a negative influence on ADG. Eleven authors have reported that atrophic rhinitis decreases ADG by as much as 5% (59). In this study there was no significant difference in ADG among hogs with atrophic rhinitis scores from 0 to 4.5. However, hogs with an atrophic rhinitis score of five had a 7.7% lower ADG than hogs with scores below five.

Atrophic rhinitis scores were assigned according to a system which increases incrementally for each three to four mm increase in the total space between the ventral turbinates and the floor of the nasal cavity. If the space exceeds 20 mm (and it can exceed twice that) it is assigned the maximum score of five. There is a much greater range between scores of five and scores below five than there is among scores below five.

The following example illustrates the magnitude of the effect of atrophic rhinitis on ADG. The highest percentage of hogs per farm with snout scores of five was 55%. Reducing the level of atrophic rhinitis on this farm to the point where all scores were below five would increase the ADG by approximately 4.2% (55% of 7.7%).

In the 13 studies which reported that anteroventral pneumonia had a

significant effect on ADG, estimates of the magnitude of effect ranged from 4.6% - 25% (59). For an individual hog in this study, ADG was 2.8% higher if the hog was free of anteroventral pneumonia at slaughter. The difference between the highest (95.8%) and lowest (17.2%) percentage of hogs affected per farm coincided with approximately a 2.2% improvement in ADG.

The significant interaction term for atrophic rhinitis and anteroventral pneumonia indicates that the presence of both these factors had a greater detrimental effect on ADG than the sum of their individual effects. The effect of atrophic rhinitis on ADG varied with the presence of pneumonia and vice versa. The combination of these two factors resulted in a total decrease in ADG of 0.0917 kg/day, or 17.6% of the average ADG.

This study took an analytical approach to try to shed new light on the old controversies regarding the relationships of pneumonia, atrophic rhinitis and ascarids to ADG and to each other. Once it is established which relationships exist, it is necessary to evaluate whether attempts to control for the disease(s) in question would be of economic benefit. This depends on the cost of intervention, the degree of improvement that is achievable, and production costs.

Individual farms with heavy ascarid burdens could improve their ADG by up to 1% through ascarid control. This would only be economically advantageous if the benefits exceeded the cost of the control measures (anthelmintics, improved sanitation etc.) and if the control measures were effective. Depending on which control measures were instituted, benefits in addition to a slight improvement in ADG may arise through concurrent reduction of other parasites and diseases.

This study indicates that the greatest degree of improvement in ADG could be realized by reducing the severity of atrophic rhinitis and that this may have additional merit through a concomitant reduction in anteroventral pneumonia. The

cost of reducing the level of atrophic rhinitis may differ for farms with different starting points. For example, a simple improvement in ventilation might be cost effective for a farm with a severe level of atrophic rhinitis, while a farm with low levels of the disease might have to resort to major changes (eg. disease-free status) to further diminish the level of disease. The ability to make decisions in this regard would be greatly enhanced by the availability of appropriate records, particularly records of disease levels present in the herd and of input costs of production.

5. SUMMARY OF RESULTS AND CONCLUSIONS

5.1 Chapter 2. Descriptive Statistics

Study hogs were marketed at an average of 189±22 days. The average dressed carcass weight was 77.0±5.9 kg and the mean ADG was 0.519±0.071 kg/day.

Only two of the 15 study farms had hogs shedding ascarid eggs at approximately 78 days of age, but this increased to 13 farms by the time hogs were marketed. The percent of hogs with ascariasis varied widely between farms, no matter what measure of ascariasis was used: the percent with intestinal ascarids at slaughter ranged from 0% to 96%; the percent that shed ascarid eggs during their lifetime ranged from 0% to 100%; and the range for hogs with liver lesions ranged from 27% to 100%.

Of all the study hogs slaughtered, 35% had intestinal ascarids, 32% shed ascarid eggs during their lifetime and 82% had milk spot lesions. The average number of intestinal ascarids was 12, and the median was four, for those hogs that had intestinal ascarids at slaughter.

The egg count at slaughter accounted for approximately 50% (r^2=0.505) of the variation in the number of ascarids in hogs. The r^2 was raised to 0.735 by the addition of a quadratic term (egg count squared). The strength of this association suggested that lifetime burden, which was comprised of a series of fecal egg counts, would reflect the ascarid burden over time.

There was a large discrepancy between the proportion of hogs with ascarids (35%) and the proportion of hogs with milk spot lesions (82%). Resistance mechanisms of the host work at all sites of ascarid migration towards elimination of the parasite (3). Ascarid larvae are eliminated through milk spot formation in

the liver and during migration in the lungs (42). There is also a massive expulsion of larvae from the small intestine during the early prepatent period (3,41,43). If the host immune response overcomes the challenge before a patent ascarid infection is established, the liver may have milk spots although there are no ascarids in the intestine.

Milk spot lesions do resolve (3,7,38,39), which explains why a few hogs had intestinal ascarids in the absence of liver lesions.

The average number of intestinal ascarids per hog and the mean lifetime burden increased with the severity of liver lesions ($p<0.01$). The average time from the onset of fecal shedding until slaughter was directly related to the severity of the liver score ($p<0.01$), being longest for hogs with severe liver lesions.

At slaughter, 55% of the hogs had anteroventral pneumonia and 9% had atrophic rhinitis scores of five. The percent of hogs per farm with pneumonia ranged from 17% to 96%. The percent of hogs per farm with atrophic rhinitis scores of five ranged from 0% to 57%.

5.2 Chapter 3. Abattoir Surveillance

There was excellent agreement, beyond chance, between the inspectors' estimate and the author's count of milk spot lesions. It can be concluded that rapid post mortem examination of livers provides a valid assessment of the severity of milk spot lesions.

The presence of milk spots had a high sensitivity, very low specificity, and a high negative predictive value as a screening test for ascariasis in individual hogs. Results were consistent whether ascariasis was measured as the presence of

intestinal ascarids at slaughter, or by a positive fecal egg count during the hog's lifetime. The presence of milk spots does not necessarily indicate that an ascarid infection has been established in the small intestine. The absence of milk spots, however, is a reliable indicator of the absence of an established ascarid infection, provided that the prevalence of ascariasis is equal to or less than that observed in this study.

The severity of the ascarid infection in an individual hog could not be ascertained by the number of milk spot lesions on the liver.

It was possible to select divisions which enabled detection of most study farms that had a high level of ascarid infection. Detection was based on the percentage of hogs per farm with liver lesions at slaughter. Either farms with a high average lifetime burden, or farms with a high percentage of hogs affected, could be identified in this manner. The divisions selected were specific for the distribution of ascariasis and liver lesions on the farms in this study.

5.3 Chapter 4. A Path Analysis of Factors Affecting ADG in Swine

Most of the variation in ADG was due to the exogenous variables farm, litter and sex. This emphasizes the importance of controlling for confounding variables which may distort the association of interest.

The variables atrophic rhinitis, anteroventral pneumonia and lifetime burden each had a significant direct effect on ADG in swine. Neither atrophic rhinitis nor lifetime burden had a direct effect on pneumonia, therefore neither of these variables had an indirect effect on ADG. There was significant interaction between the effects of atrophic rhinitis and anteroventral pneumonia on ADG.

ADG was 2.8% lower in hogs with anteroventral pneumonia than in those

without. There was no significant difference in ADG among hogs with atrophic rhinitis scores from 0 to 4.5. However, hogs with an atrophic rhinitis score of five had a 7.7% lower ADG than hogs with scores below five. Hogs with both anteroventral pneumonia and atrophic rhinitis had a 17.6% lower ADG than hogs with neither disease.

ADG was not significantly affected by the number of intestinal ascarids at slaughter. However, there was a quadratic relationship between lifetime burden and ADG. ADG began to decrease once lifetime burden exceeded a value of two (twice the average lifetime burden for infected hogs).

Although heavy ascarid burdens decreased the growth rate of swine, the magnitude of the effect was minimal. Only 5% of the study hogs had a lifetime burden greater than two. These hogs constituted approximately one third of the hogs on the two farms with the heaviest ascarid infections. Regardless, the maximum improvement one could expect from reducing the ascarid burden on these farms would be less than 1%.

The effect of a high lifetime burden on growth rate was of a greater magnitude in the early part of the hogs' lives. This may indicate that ascarid infection has a greater deleterious effect on young growing pigs and that compensatory growth occurs later on.

There is much greater potential for improvement in ADG through the control of respiratory diseases than through the control of ascariasis.

6. APPENDIX A - Lifetime Burden

The average fecal ascarid-egg count, up to the fourth farm visit (mean age 154 days), was calculated for each hog in the following manner: the average egg count, measured as eggs per gram of feces (epg), for consecutive visits (column c) was multiplied by the number of days elapsed between these visits (column e); the sum of these figures until the fourth visit constitutes that hog's raw lifetime burden. It was assumed that counts would be 0 eggs per gram until the end of the prepatent period, which is approximately 60 days (1,8).

For cases where there was a missing value for one of the egg counts, the preceding and following counts were averaged and multiplied by the total days between them.

For example:

a visit	b epg	c average epg	d age (days)	e days in period	c x e
0	0		60		
		50		10	500
1	100		70		
		50		27	1350
2	0		97		
		100		28	2800
3	200		125		
		500		20	10000
4	800		145		
					14650 = raw lifetime burden

These high numeric values were standardized by dividing by the average raw lifetime burden for hogs whose lifetime burden was greater than zero (32,068). Therefore, a hog with a lifetime burden of one has an average lifetime burden for an infected hog. In the above example the lifetime burden would be 0.46.

Lifetime burden = raw lifetime burden/32068

7. APPENDIX B - Average daily gain

Average daily gain for the hogs' lifetime was calculated by dividing live weight by days to market. In this study the abattoir recorded only dressed carcass weights, so it was necessary to obtain appropriate dressing percentages in order to convert dressed carcass weights to live weights. The following data provide average dressing percentage per live weight class, based on 80,000 hogs shipped in Ontario during one week in July 1986 (Dr. Gordon Bowman, University of Guelph, written communication).

live wt (kg)	dressing %	dressed carcass wt (kg)	midpoint
77.6 - 82.5	75.5	58.6 - 62.3	60.4
82.6 - 87.5	77.25	63.8 - 67.5	65.6
87.6 - 92.5	79.0	69.2 - 73.1	71.1
92.6 - 97.5	79.5	73.6 - 77.5	75.5
97.6 - 102.5	80.0	78.1 - 82.0	80.0
102.6 - 107.5	80.0	82.1 - 86.0	84.0
107.6 - 112.5	80.0	86.1 - 90.0	88.0

Using these data, live weights could be estimated from dressed carcass weights. However, this would result in some gaps in the range of dressed carcass weights. For example, for a dressed carcass weight of 62.5 kg it is not clear whether a dressing percentage of 75.5% or 77.25% should be used. In order to circumvent this problem and to be able to include hogs whose dressed carcass weight was outside the range for which conversion factors were provided, multiple regression was used to determine the relationship between dressing percentage and the seven midpoints for dressed carcass weight. It yielded the following formula:

$$\text{dressing \%} = 19.21 + 1.461437(\text{dressed carcass wt}) - 0.008767(\text{dressed carcass wt})^2$$

$$\text{adjusted } r^2 = 0.9938$$

The adjusted r^2 is high because data from a large number of hog carcasses contributed to each point. Therefore the equation yields a very precise estimate of dressing percentage.

It follows that:

$$\text{live weight} = \frac{\text{dressed carcass wt} \times 100}{\text{dressing percentage}}$$

The resulting live weights were used to calculate average daily gain.

8. APPENDIX C - Kappa

The statistic kappa was used to assess the agreement, beyond that which would be expected due to chance, between the inspectors' and the author's scores for liver lesions.

Number of livers with the following number of milk spots:

		Inspectors' Scores			
		0	1 - 6	> 6	Total
Author's Scores	0	5	0	0	5
	1-6	5	27	0	32
	> 6	1	4	37	42
		11	31	37	79

observed % agreement = $\frac{(5 + 27 + 37)}{79} = 87.34$

expected % agreement = $\frac{(11 \times 5) + (31 \times 32) + (37 \times 42)}{79 \times 79} = 41.68$

kappa = $\frac{\text{observed \% agreement - expected \% agreement}}{100 - \text{expected \% agreement}} = 0.783$

9. REFERENCES

1. SOULSBY EJL. Nematodes of the small intestine. In: Textbook of veterinary clinical parasitology. Oxford: Blackwell Scientific Publications, 1965: 184-216.

2. DOUVRES FW, TROMBA FG, MALAKATIS GM. Morphogenesis and migration of Ascaris suum larvae developing to fourth stage in swine. J Parasitol 1969; 55: 689-712.

3. ERIKSEN L. Host parasite relations in Ascaris suum infection in pigs and mice. Thesis, Royal Veterinary and Agricultural University, Copenhagen. 1981: 1-193.

4. BARKER IK, VAN DREUMEL AA. The alimentary system. In: Jubb KVF, Kennedy PC, Palmer N, eds. Pathology of Domestic Animals. Volume 2. 3rd ed. Orlando: Academic Press, Inc., 1985: 1-203.

5. SCHWARTZ B. Experimental infection of pigs with Ascaris Suum. Am J Vet Res 1959; 20: 7-13.

6. TAFFS LF. Immunological studies on experimental infection of pigs with Ascaris suum Goeze, 1782. VI. The histopathology of the liver and lung. J of Helminthol 1968; 42: 157-172.

7. COPEMAN DB, GAAFAR SM. Sequential development of hepatic lesions of ascaridosis in colostrum-deprived pigs. Aust Vet J 1972; 48: 263-268.

8. MARQUARDT WC, DEMAREE RS. Parasitology. New York: Macmillan Publishing Company, 1985.

9. KELLEY GW, SMITH LJ. The daily egg production of Ascaris suum and the inability of low levels of aureomycin to affect egg production and embryonation. J Parasitol 1956; 42: 587.

10. BROWN HW. A quantitative study of the influence of oxygen and temperature on the embryonic development of the eggs of the pig ascarid (Ascaris suum Goeze). J Parasitol 1928; 14: 141-160.

11. CORWIN RM, DiMARCO NK, McDOWELL AE, PRATT SE. Internal parasites. In: Leman AD, et al., eds. Diseases of Swine. 6th edition. Ames, Iowa: Iowa State University Press, 1986: 646-664.

12. BROWN HW. Further studies on the longevity of the eggs of Ascaris lumbricoides and A. suum. J Parasitol 1929; 15: 14-22.

13. THOMAS RJ, FERBER M. The epidemiology of helminthiasis in pigs. The Pig Veterinary Society Proceedings 1985; 12: 50-55.

14. JASKOSKI BJ. An apparent swine ascaris infection of man. J Am Vet Med Assoc 1961; 138: 504-505.

15. PHILLS JA, HARROLD AJ, GABRIEL MD, WHITEMAN V, PERELMUTTER L. Pulmonary infiltrates, asthma and eosinophilia due to Ascaris suum infestation in man. N Engl J Med 1972; 286: 965-970.

16. PHILLIPSON RF, RACE JW. Human infection with porcine ascaris. Br Med J 1967; 3: 865.

17. McLENNAN MW, HUMPHRIS RB, RAC R. Ascaris suum pneumonia in cattle. Aust Vet J 1974; 50: 266-268.

18. MITCHELL GBB, LINKLATER KA. Condemnation of sheep livers due to ascariasis. Vet Rec 1980; 107: 70.

19. GREENWAY JA, McCRAW BM. Ascaris suum Infection in Calves. I. Clinical Signs. Can J Comp Med 1970; 34: 227-237.

20. KENNEDY PC. The migrations of the larvae of Ascaris lumbricoides in cattle and their relation to eosinophilic granulomas. Cornell Vet 1954; 44: 531-565.

21. McCRAW BM, LAUTENSLAGER JP. Pneumonia in calves associated with migrating Ascaris suum larvae. Can Vet J 1971; 12: 87-90.

22. MORROW DA. Pneumonia in cattle due to migrating Ascaris lumbricoides larvae. J Am Vet Med Assoc 1968; 153: 184-189.

23. ANDERSEN S, JORGENSEN RJ, NANSEN P, NIELSEN K. Experimental Ascaris suum infection in piglets. Acta Pathol Microbiol Scand 1973; 81: 650-656.

24. NILSSON O. Ascariasis in the pig. An epizootiological and clinical study. Acta Vet Scand [Suppl] 1982; 79: 1-108.

25. MARTIN LJ, GIBBS HC, PULLIN JW. Gastrointestinal parasites of swine in Quebec. I. An incidence survey. Can Vet J 1974; 15: 72-76.

26. PATTISON HD, THOMAS RJ, SMITH WC. A survey of gastrointestinal parasitism in pigs. Vet Rec 1980; 107: 415-418.

27. LAUTENSLAGER JP, MARTIN SJ, OLIVER PG. A field survey of internal parasites of swine from pure bred herds in southern Ontario. Final report to research committee of the Veterinary Services Branch, 1976.

28. POLLEY LR, MOSTERT PE. Ascaris suum in Saskatchewan pigs: An abattoir survey of prevalence and intensity of infection. Can Vet J 1980; 21: 307-309.

29. BINDSEIL E. Observations on the relationship between Ascaris suum burdens in pigs and faecal egg counts. Acta Pathol Microbiol Scand 1974; 82: 879-884.

30. THIENPONT D, ROCHETTE F, VANPARIJS OFJ. Diagnosing helminthiasis through coprological examination. Beerse, Belgium: Janssen Research Foundation, 1979.

31. LEVINE ND, MEHRA KN, CLARK DT, AVES IJ. A comparison of nematode egg counting techniques for cattle and sheep feces. Am J Vet Res 1960; 21: 511-515.

32. KENNEDY MJ. Manual of veterinary parasitology laboratory techniques. Alberta Agriculture Animal Health Division, 1982.

33. BELLO TR. Comparison of the flotation of metastrongylus and ascaris eggs in three different levitation solutions. Am J Vet Res 1961; 22: 597-600.

34. HILLEY H, STRAW B, SCHWOCHERT M. Conducting slaughter checks. Proceedings of the American Association of Swine Practitioners. 1982: 220-230.

35. ELANCO PRODUCTS COMPANY. Trac clinics: total respiratory analysis and control. Indianapolis, Indiana, 1984.

36. STRAW E, BURGI EJ, HILLEY HD, LEMAN AD. Pneumonia and atrophic rhinitis in pigs from a test station. J Am Vet Med Assoc 1983; 182: 607-611.

37. STSC, INC. Statgraphics. Maryland: STSC, Inc., 1987.

38. RONEUS O, SWAHN O. Liver lesions in young pigs caused by ascarids. Nord Vet Med 1969; 21: 324-326.

39. TSVETAEVA NP. Pathogenic importance of helminths in early stages of their development. Proceedings of the XVII (17th) International Veterinary Congress 1963; 1: 813-814.

40. ERIKSEN L, ANDERSEN S, NIELSEN K, PEDERSEN A, NIELSEN J. Experimental Ascaris suum infection in pigs. Serological response, eosinophilia in peripheral blood, occurrence of white spots in the liver and worm recovery from the intestine. Nord Vet Med 1980; 32: 233-242.

41. ERIKSEN L. Experimentally induced resistance to Ascaris suum in pigs. Nord Vet Med 1982; 34: 177-187.

42. RONEUS O. Studies on the aetiology and pathogenesis of white spots in the liver of pigs. Acta Vet Scand [Suppl] 16 1966; 7: 1-112.

43. JORGENSEN RJ, NANSEN P, NIELSEN K, ERIKSEN L, ANDERSEN S. Experimental Ascaris suum infection in the pig. Population kinetics following low and high levels of primary infection in piglets. Vet Parasitol 1975; 1: 151-157.

44. RONEUS O. Studies on the inter-relationship between the number of orally administered A. suum eggs, blood eosinophilia and the number of adult intestinal ascarids. In: Gaafar SM, ed. Pathology of parasitic diseases. Lafayette, Indiana: University Studies, 1971: 339-343.

45. TAFFS LF. Immunological studies on experimental infection of pigs with Ascaris suum Goeze, 1782. III. - The antibody response and acquired immunity. J Helminthol 1964; 38: 129-150.

46. KELLY GW, NAYAK DP. Aquired Immunity to Migrating Larvae of Ascaris suum induced in pigs by repeated oral inoculations of infective eggs. J Parasitol 1964; 50: 499-503.

47. KELLEY GW, NAYAK DP. Passive immunity to migrating Ascaris suum transmitted by parenterally administered immune serum or immune globulins. Cornell Vet 1965; 55: 607-612.

48. WHITE EG. Chronic focal interstitial hepatitis in the pig. Vet J 1941; 97: 155-172.

49. BATTE EG, HARKEMA R, OSBORNE JC. Swine kidney worm. J Am Vet Med Assoc 1960; 136: 622-625.

50. STEVENSON P. Toxocara and ascaris infection in British pigs: A serological survey. Vet Rec 1979; 104: 526-528.

51. FLESJA KI, ULVSAETER HO. Pathological lesions in swine at slaughter. I. Baconers. Acta Vet Scand 1979; 20: 498-514.

52. MARTIN SW. The evaluation of tests. Can J Comp Med 1977; 41: 19-25.

53. MARTIN SW. Estimating disease prevalence and the interpretation of screening test results. Prev Vet Med 1984; 2: 463-472.

54. FLEISS JL. Statistical methods for rates and proportions. 2nd ed. Toronto: J Wiley and Sons, 1981.

55. STEVENSON P. The influence of environmental temperature on the rate of development of Ascaris suum eggs in Great Britain. Res Vet Sci 1979; 27: 193-196.

56. STRAW BE, BACKSTROM L, LEMAN AD. Examination of swine at slaughter. Part I. The mechanics of slaughter examination and epidemiologic considerations. Comp Cont Ed 1986; 8: S41-S47.

57. CONNAN RM. Ascariasis: The development of eggs of Ascaris suum under the conditions prevailing in a pig house. Vet Rec 1977; 100: 421-422.

58. WILSON MR, TAKOV R, FRIENDSHIP RM, MARTIN SW, MCMILLAN I, HACKER RR, SWAMINATHAN S. Prevalence of respiratory diseases and their association with growth rate and space in randomly selected swine herds. Can J Vet Res 1986; 50: 209-216.

59. MORRISON RB, PIJOAN C, LEMAN AD. Association between enzootic pneumonia and performance. Pig News and Information 1986; 7: 23-31.

60. MORRISON RB, HILLEY HD, LEMAN AD. The association between pneumonia and atrophic rhinitis in slaughter weight swine. Can Vet J 1985; 26: 95-97.

61. CONNAN RM. The effect of management systems on helminth parasitism of pigs. The Pig Veterinary Society Proceedings 1986; 15: 29-35.

62. GAAFAR SM, KEITTEVUTI B. Experimental induction of esophagogastric ulcers with inoculations of Ascaris suum eggs in swine. Gastroenterology 1972; 63: 423-426.

63. RIVERA MA, GAAFAR SM. Sequential development of esophagogastric ulcers induced in swine by infections with Ascaris suum Goeze, 1782. Vet Parasitol 1976; 2: 341-353.

64. QURESHI SR, OLANDER HJ, GAAFAR SM. Esophagogastric ulcers associated with Ascaris suum infestation in swine. Vet Pathol 1978; 15: 353-357.

65. HANI H, INDERMUHLE NA. Esophagogastric ulcers in swine infected with Ascaris suum. Vet Pathol 1979; 16: 617-618.

66. UNDERDAHL NR, KELLEY GW. The enhancement of virus pneumonia of pigs by the migration of Ascaris suum larvae. J Am Vet Med Assoc 1957; 130: 173-176.

67. UNDERDAHL NR. The affect of Ascaris suum migration on the severity of swine infuenza. J Am Vet Med Assoc 1958; 133: 380-383.

68. MARTIN SW, MEEK AH. A path model of factors influencing morbidity and mortality in Ontario feedlot calves. Can J Vet Res 1986; 50: 15-22.

69. MARTIN SW, MEEK AH, WILLEBERG P. Veterinary epidemiology principles and methods. 1st ed. Iowa: Iowa State University Press, 1987.

70. JOYNER SP, ed. SAS/STAT Guide for personal computers, version 6 edition. Cary, North Carolina: SAS Institute Inc., 1985.

71. SPINDLER LA. The effect of experimental infections with ascarids on the growth of pigs. Proceedings of the Helminthology Society 1947; 14: 58-63.

72. ZIMMERMAN DR, SPEER VC, ZIMMERMAN W, SWITZER WP. Effect of pyrantel salts on Ascaris suum infection in growing pigs. J Anim Sci 1971; 32: 874-878.

73. ZIMMERMAN DR, SPEAR ML SWITZER WP. Effect of Mycoplasma hypopneumoniae infection, pyrantel treatment and protein nutrition on performance of pigs exposed to soil containing Ascaris suum ova. J Anim Sci 1973; 36: 894-897.

74. STEPHENSON LS, GEORGI JR, CLEVELAND DJ. Infection of weanling pigs with known numbers of Ascaris suum fourth stage larvae. Cornell Vet 1977; 67: 92-102.

75. STEPHENSON LS, POND WG, NESHEIM MC, KROOK LP, CROMPTON DWT. Ascaris suum: nutrient absorption, growth, and intestinal pathology in young pigs experimentally infected with 15-day-old larvae. Exp Parasitol 1980; 49: 15-25.

76. FORSUM E, NESHEIM MC, CROMPTON DWT. Nutritional aspects of Ascaris infection in young protein-deficient pigs. Parasitology 1981; 83: 497-512.

77. HALE OM, STEWART TB, MARTI OG. Influence of an experimental infection of Ascaris suum on performance of pigs. J Anim Sci 1985; 60: 220-225.