ASCARIS SUUM: OCCURRENCE, EPIDEMIOLOGY AND CONTROL IN
SASKATCHEWAN PIGS

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ABSTRACT

_Ascaris suum_ Goeze, 1782, the large roundworm, is a common parasite of pigs (_Sus scrofa domestica_ Linnaeus, 1758) throughout the world. It is an important cause of economic loss to the pig industry and control of this parasite is difficult. In addition, _A. suum_ is of zoonotic importance and therefore raises concerns related to manure disposal.

There are reports describing the epidemiology of _A. suum_ from other areas of the world but little is known regarding the situation in Saskatchewan. This thesis project examined aspects of the occurrence, epidemiology and control of this parasite in Saskatchewan. The project comprised four inter-related studies.

First, two surveys investigating the prevalence and intensity of _A. suum_ were conducted at a Saskatchewan abattoir examining livers and intestines of market pigs. Fifty-three percent of animals examined in these surveys displayed evidence of ascarid infection (adult parasites, hepatic lesions or both).

Second, a postal survey to determine anthelmintic use in Saskatchewan during 1995 was sent to 580 pig producers selected based on annual pig production. Response rate was 33% and represented 20% of the province’s annual market pig output. Of all respondents, 76% treated some animals. Sows were treated most commonly (90%) followed by weanlings (86%), boars (75%) and growers (67%). Determination of a parasite problem was not based on quantitative measures of
infection and treatment patterns did not appear to be based on known epidemiological information.

Third, a study investigated the effects of seasonal temperature variations on the rate of development to infectivity of *A. suum* eggs in a Saskatchewan barn. Eggs from experimental egg cultures placed in the barn each month (July, 1997 to July, 1998) were monitored weekly for development to the infective larval stage assessed by bioassay. Development to the infective stage took from three to four weeks in summer to as long as 11-12 weeks in winter.

Lastly, two groups of pigs were monitored from weaning to market by regular fecal examinations for *A. suum* infection. No pigs became definitively patent during the study but 92% of animals examined *post-mortem* had liver lesions consistent with ascarid larval migration.
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<tr>
<td>PI</td>
<td>post infection</td>
</tr>
<tr>
<td>L2</td>
<td>second stage larvae</td>
</tr>
<tr>
<td>L3</td>
<td>third stage larvae</td>
</tr>
<tr>
<td>GT</td>
<td>granulation type</td>
</tr>
<tr>
<td>LT</td>
<td>lymphonodular type</td>
</tr>
<tr>
<td>DU</td>
<td>development units</td>
</tr>
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<td>epg</td>
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1.0 LITERATURE REVIEW

*Ascaris suum* Goeze, 1782, the large roundworm of pigs (*Sus scrofa domestica* Linnaeus, 1758), is a common parasite with a cosmopolitan distribution. It is the largest, most well known and probably most important helminth in farmed swine throughout the world (Roberts, 1934; Levine, 1980; Anderson, 1992). The prevalence and intensity of this parasitic infection remains surprisingly high in many countries in spite of regular control measures, including intensive barn management with sanitation practices directed against the free-living stage of *A. suum* plus the regular use of the highly effective anthelmintics available to pig producers worldwide (Mercy et al., 1989b; Roepstorff and Nansen, 1994; Roepstorff et al., 1998). The success of this parasite in the face of these control measures is impressive and is due to a number of factors, including the high fecundity of the female worms, each producing as many as 2,000,000 eggs per day (Olsen et al., 1958), the resilience of the eggs, which are somewhat resistant to desiccation and highly resistant to most common disinfectants (Roberts, 1934; Seamster, 1950) and their ability to remain viable for at least five years in the external environment (Anderson, 1992). These biological features of *A. suum* may lead to high levels of facility contamination even if only a few animals are infected and may also prevent complete elimination of the contamination.
A. suum is known to be present in Saskatchewan pigs. A survey reported in 1980 indicated a high prevalence and moderate intensity of infection (Polley and Mostert, 1980). The presence of this parasite in the provincial pig herd is a potential cause of economic loss as a result of reduced production efficiencies in growing pigs and liver condemnations resulting from lesions associated with larval migrations. Research into the biology and epidemiology of A. suum in Saskatchewan pigs is important to assist in "fine-tuning" control program recommendations.

Ascaris suum is also capable of infecting people. Increased knowledge of the local transmission features of this parasite is crucial. Governments in western Canada, including Saskatchewan, are currently promoting increased hog production and therefore the potential for contact between people and ascarid eggs in fecal material from pig farms may increase. Understanding the risk posed to people by this parasite is an important aspect of the debate regarding the establishment of new, large scale pig production units in Saskatchewan as well as in determining the most efficient and safest manure disposal methods.

1.1 Biology of Ascaris suum

For many years, researchers considered the ascarid of people, A. lumbricoides, and the ascarid of pigs, A. suum, to be the same species. Roberts (1934), working exclusively with pigs, indicated they were the same species with minor physiological differences allowing for some host specificity. The adult
worms are essentially indistinguishable on gross examination and cross-infections are possible, though this may be rare (Roberts, 1934; Anderson, 1992). Taffs (1961), reviewing the literature, concluded on the basis of epidemiological and morphological evidence that the two species are distinct. More recent work has described genetic differences between the two species (Anderson, 1995).

The taxonomic description is as follows (Anderson, 1992):

Phylum: Nemathelminthes
Class: Nematoda
Order: Ascaridida
Superfamily: Ascaridoidea
Family: Ascarididae
Subfamily: Ascaridinae
Genus: Ascaris
Species: suum

*Ascaris suum*, in common with most other ascarids, is a large yellowish white nematode inhabiting the small intestine of its host. Occasionally worms are found in the stomach, the bile duct or the large intestine. The adult females range from 20 to 40 cm in length with the males slightly shorter at 15 to 25 cm. Adult males are also more slender (Soulsby, 1982). The head of adult worms is quite distinct with three prominent triangular lips surrounding a small buccal capsule. Male worms have two equal spicules measuring 1.8 to 2.4 mm in length (Roberts, 1934). The uterus containing the characteristic eggs occupies a large part of the adult female *A. suum* (Roberts, 1934). The eggs are infective to many mammal species. Ingested eggs will
hatch and larvae will migrate but will not usually mature in “abnormal” hosts
(Schwartz, 1959)

1.1.1 Life-cycle

Roberts (1934) completed the first successful experimental infection of pigs with *A. suum* and the first detailed description of the life cycle in the normal host. Prior to that, there was debate regarding the nature of this parasite’s life cycle: was it indirect requiring some type of intermediate host (Stewart, 1917)? was it direct with the embryonated eggs hatching in the small intestine and larvae remaining there maturing to adults in that part of the gut? or was there migration of larvae through the body of the host before maturation was completed in the intestine (Ransom and Foster, 1919)? Many early workers attempted experimental infections in a number of mammal species but, according to Schwartz (1959), were only able to find ascarid larvae in the process of hatching in the small intestine of these animals shortly after infection. Stewart (1916) (repeated by Ransom and Foster, 1919) was the first to discover the migration route of *A. suum* larvae from the small intestine to the liver and the lungs and then to the small intestine. These observations were made not in pigs but in rats and mice. Stewart (1916) believed that these small mammals might function as intermediate hosts and that infective larvae would be spread in the environment by contamination with mouse or rat feces (Ransom and Foster, 1919). Stewart’s and other researchers’ lack of success in establishing experimental infections in pigs with embryonated *A. suum* eggs contributed to this hypothesis.
(Stewart, 1917; Ransom and Foster, 1919). Ransom and Foster (1919) were able to complete the life cycle in both a kid and a lamb and also had success finding migrating larvae in guinea pigs and rabbits but were unsuccessful with experimental infections of pigs. The initial observation of Roberts (1934) on the development of *A. suum* in experimentally infected pigs have been supplemented by results from several subsequent studies: Schwartz (1959) determined the fate of *A. suum* larvae which failed to complete development in the pig; Douvres et al. (1969) modified the descriptions of larval stages during migration; Murrell et al (1997) described a revised migratory pathway; and Roepstorff et al. (1997) related differences in ascarid migration patterns in pigs to the administration of different numbers of infective eggs.

The current understanding of the life cycle is as follows. The adult nematodes live in the small intestine of the pig where eggs are shed. These eggs are passed in feces into the external environment where they develop to an infective stage within the egg. Once ingested, these infective eggs hatch very quickly, some in the stomach but most in the small intestine (Murrell et al. 1997). The freed larvae, which are 224-263 μm in length (Douvres et al., 1969), then penetrate the gut wall. Recent research (Murrell et al., 1997) indicates this occurs in the colon and caecum and not the small intestine as previously believed (Murrell, 1986) and occurs rapidly with the majority of larvae out of the intestinal lumen by 18 hours post infection (PI). The larvae travel from the intestinal wall, via the hepatic portal system, to the liver (Anderson, 1992) where they arrive as quickly as six hours PI (Douvres et al., 1969;
Murrell et al., 1997). Maximum numbers are present there three (Roepstorff et al.,
1998) or four (Kelley et al., 1957; Douvres et al., 1969) days PI. The parasites moult
during this phase of the migration from a second stage (L2) 172-252 μm in length to
a third stage larvae (L3) 533-619 μm in length (Douvres et al., 1969). Migration of
these larvae through the liver results in macroscopic lesions known as “milk-spots”
(Corwin and Stewart, 1992). Subsequently the L3 move via the vasculature
(Soulsby, 1982) to the lungs beginning to arrive there at approximately day four PI
(Douvres et al., 1969). By day seven PI (Roepstorff et al., 1997) or day nine PI
(Kelley et al., 1957) the L3 are present in maximum numbers and are 0.98-1.68 mm
in length (Douvres et al., 1969). In the lungs the third stage larvae leave the
branches of the pulmonary artery and enter the alveoli, then the small bronchioles
and eventually reach the trachea (Soulsby, 1982). The larvae then pass through the
pharynx and are swallowed, entering the small intestine at between eight to 10 days
PI (Roepstorff et al. 1997). The third moult is completed at approximately day 10 PI
(Douvres et al., 1969). At this stage of the cycle, the larvae are 1.57 – 1.87 mm in
length (Douvres et al., 1969). Roberts (1934) reported that the last moult occurred in
the small intestine between day 21 and 29 PI and Schwartz (1959) found that many
worms were spontaneously eliminated in association with this moult. This
elimination is likely immune mediated with larger egg inoculations resulting in more
effective expulsions in experimentally infected pigs (Roepstorff et al., 1997).
Worms mature in the small intestine and egg production begins at approximately 42
days PI (Roepstorff et al., 1997), 49-52 day PI (Schwartz, 1959), or 60-62 day PI
(Olsen et al, 1958). Adult worms may live for up to 55 weeks but most are eliminated at about six months (Olsen et al, 1958; Corwin and Stewart, 1992). There is no evidence that *A. suum* can be transmitted prenatally (Soulsby, 1982).

1.1.2 Egg development

Nematode egg shells are considered to be “one of the most resistant biological structures” (Wharton, 1980). *Ascaris suum* eggs measure, on average, 54 by 70 μm (Roberts, 1934) although egg sizes reported by that author varied from as large as 59 by 84 μm to as small as 40 by 45 μm. The eggs are oval in shape and brown in color (Soulsby, 1982). The thick shell has a rough, mamillated appearance. Ascarid egg shells consist of four definable layers which are, from inside out: a selectively permeable lipid layer primarily involved in gas and water exchange; a chitin/protein layer providing strength and structure (the thickest layer); a vitelline layer; and an outer albuminous uterine layer (Wharton, 1980) which is very sticky and allows eggs to adhere to barn fixtures and to the pigs themselves (Anderson, 1992). This remarkable shell allows the eggs to survive harsh environmental conditions and assists them in remaining viable for long periods of time at any stage of development from single cell to completely larvated (Brown, 1928a). The egg’s shell also provides resistance to many types of disinfectants (Seamster, 1950). Eggs are somewhat susceptible to desiccation. Eggs cease development and will die within days in dry soils or when exposed to direct sunlight (Gaasenbeek and
Borgsteede, 1998). Factors influencing the environment near the eggs that modulate drying are very important in egg survival (Caldwell and Caldwell, 1928). Eggs are also susceptible to pig urine; eggs will not develop after a few days exposure (Roberts, 1934; Nilsson, 1982).

Eggs of *A. suum* are not immediately infective; containing only a single cell when passed in feces. They must undergo a period of development in the external environment. Traditionally, it was believed that only one moult occurred during egg development and a second stage larvae was present inside the infective egg (Soulsby, 1982). Maung (1978) reported the occurrence of a second moult that was initiated inside the egg but often not completed until the larvae reached the liver of the definitive host.

Egg development to infectivity is affected by a number of factors including temperature, humidity (Seamster, 1950), oxygen tension (Brown, 1928a), and sunlight (Caldwell and Caldwell, 1928). Most work studying ascarid egg development has focused on the effects of temperature and humidity. Eggs begin development when the temperature reaches 14.5°C, the development threshold below which no egg maturation is observable (Seamster, 1950). This author did not observe eggs past a multi-celled stage at 14.5°C but did report observing larvated eggs in 888 hours at 16.7°C. Earlier, Roberts (1934) observed slight egg development when eggs were exposed to 13.5-15.5°C. More recently the development threshold has been reported to be 16°C with eggs requiring more than 700 hours to become larvated at this temperature (Arene, 1986). Roberts (1934)
found that eggs develop most quickly at between 31 and 33°C with larvae appearing within 8-9 days. Seamster (1950) reported the optimum temperature to be 31.1°C with larvated eggs seen within 210 hours and Arene (1986) observed larvated eggs in about 220 hours at a temperature of 31°C +/- 1°C. Egg development actually slows above these relatively high temperatures and temperatures above 37°C will kill eggs within a few days (Seamster, 1950). Any eggs exposed to 55°C are killed within 6.5 minutes (Barnard et al., 1987). The rate of egg maturation increases linearly as temperature rises within the development range (Seamster, 1950).

The viability and survivability of the L2 within the eggs varies depending on incubation temperature. Larvae from eggs developing too quickly (incubated at between 28 and 34°C) were less motile and had reduced ability to penetrate a paper membrane under laboratory conditions when compared to eggs incubated at a lower temperature (22°C +/- 1°C) (Arene, 1986). It is not certain how significant this information is when applied to barn conditions but the high summer temperatures (similar to external environmental temperatures) in Saskatchewan pig barns may reduce the viability of eggs developing over the summer months.

1.2 Epidemiology

Many of the helminths that infect wild or domestic pigs kept outdoors have a reduced prevalence in, or are eliminated from, animals raised in intensive indoor systems (Roepstorff and Nansen, 1994). These include nematodes that require
intermediate hosts which do not find an appropriate environment inside a modern pig barn (e.g. *Metastrongylus* spp.) and those with free-living intermediate stages that do not survive well in indoor conditions (e.g. *Stephanurus dentatus*) (Roepstorff and Nansen, 1994). *Ascaris suum* is one of the few endoparasites that has managed to survive and thrive in the transition to indoor production systems (Roepstorff and Murrell, 1997). Based on abattoir surveys of liver lesions, *A. suum* prevalence increased steadily in Northern Ireland between 1961 and 1991 (Menzies et al., 1994). This parasite’s success is most likely associated with the very large numbers of eggs produced by each female worm and the ability of these eggs to survive for long periods in varied environmental conditions (Roepstorff and Murrell, 1997).

Frequently, *A. suum* is the only helminth parasite reported from large, intensively managed herds (Roepstorff and Jorsal, 1989; Roepstorff, 1991).

The epidemiology of *A. suum* in domestic pigs varies depending on management systems, housing practices (pigs raised outdoors have a higher risk of infection (Biehl, 1984)), and regional climatic conditions. These factors have a tremendous influence on the age related prevalence and intensity profiles of this parasite (Pattison et al., 1980; Roepstorff and Nansen, 1994).

Indoor pig herds are usually separated into different age groups within the barn with piglets remaining with the sow in the farrowing crates for varying lengths of time, after which they are weaned and moved to a nursery where they spend about two months. Subsequently, the pigs are moved into the grow/finish area of the barn where they remain until market. Surveys reported over the last 65 years in Europe
(Jacobs and Dunn, 1969; Pattison et al., 1980; Roepstorff and Jorsal, 1989; Roepstorff, 1991; Dangolla et al., 1996b; Roepstorff et al., 1998), Australia (Roberts, 1940; Mercy et al., 1989a), the United States (Biehl, 1984; Morris et al., 1984; Marti and Hale, 1986) and Canada (Martin et al., 1974; Polley and Mostert, 1980; Wagner and Polley, 1997b) indicate that *A. suum* is still common worldwide and is a parasite primarily seen in grower pigs. These surveys also demonstrated that adult pigs consistently had lower infection prevalence and intensity compared to younger animals. This is probably related to the strong age related immunity induced by *A. suum* (Roepstorff and Nansen, 1994). Ascarid eggs require time in the external environment to become infective, approximately 30 – 35 days in the farrowing house (Moncol, 1993). In traditionally managed indoor herds with late (>5 weeks) weaning, infected sows in the farrowing crates pass ascarid eggs that have time to become infective and therefore are an important source of infection for the piglets. In herds where management intensity is increased and early weaning is practiced (<4 weeks) piglets are removed before eggs passed by sows become infective (Moncol, 1993). Some researchers believe that development conditions for ascarid eggs in the farrowing crates of modern pig barn are poor (Roepstorff and Nansen, 1994). Marti and Hale (1986) studied one herd in Georgia practicing early weaning and observed a nearly complete absence of patent parasitic infections in pigs in the nursery over five years. Pigs from intensively managed barns with early weaning and early movement from the nursery do not become infected until late in the growing phase (Roepstorff, 1991) when fecal contamination increases and the pigs are in pens for periods long
enough for transmission to occur. It is possible that a small number of piglets become patent early in the production cycle and eggs passed by these animals begin to contaminate the grower barn environment. One consequence of late infection with *A. suum*, recognized only recently, is that in some intensively managed operations the replacement gilts are the most heavily infected animals on farm. This may be due to the lack of exposure to this parasite early in the life of the pig and consequently a lack of immune protection (Roepstorff and Nansen, 1994; Dangolla et al. 1996b). Sows occasionally have increased infection levels when management practices include early weaning and lack of regular cleaning of pens (Dangolla et al., 1996b).

The *A. suum* population in the barn is not limited to adults in the intestines of the pigs. A large proportion of the population is present as free-living eggs at various stages of development and heavily contaminated areas may contain billions of eggs (Miskimins et al., 1994). The presence of these eggs and the development rates influence the epidemiology of this nematode. Eggs of *A. suum* develop at varying rates throughout the seasons of the year (Connan, 1977; Stevenson, 1979; Nilsson, 1982; Wagner and Polley, 1999). In the European studies no egg development was observed during the late fall and winter months. As average air temperature rises in the spring the eggs accumulating in the barn over the winter months all begin to develop at the same time and reach the infective stage in early summer. Seasonal differences in *A. suum* prevalence have been reported in Europe (Roneus, 1966; Connan, 1977; Stevenson, 1979; Menzies et al., 1994) and in Canada.
(Humik and Dohoo, 1995) based on abattoir surveys of liver lesions. This increased incidence of condemned livers in the summer is thought to be associated with the large numbers of infective eggs present in the barn at this time of year.

A feature of many helminth populations within groups of animals is an aggregated distribution (overdispersion) within the hosts. That is, most of the parasites in a host population are contained in only a few animals. This feature is recognized in pigs both naturally and experimentally infected with *A. suum* (Polley and Mostert, 1980; Roepstorff et al., 1997; Boes et al., 1998). There are a number of factors influencing this overdispersion including host density and distribution, parasite infective-stage density and distribution, individual host feeding behavior, and individual host immune response (Anderson and Gordon, 1982). In animals experimentally infected with *A. suum* where most of the aggregation factors were controlled, other than host immune response, Roepstorff et al. (1997) observed overdispersion. Boes et al. (1998) found that treated pigs reacquired *A. suum* infections at a high or low intensity that corresponded to the pretreatment intensity suggesting that the animals with high infection intensity may have a predisposition to this parasite.

Infection dose level appears to have an inverse relationship with the number of worms able to establish in the gut. In an experiment infecting pigs with high numbers (10,000) or low numbers (50) of eggs, Jorgensen et al. (1975) found that *A. suum* larvae migrated in both groups of pigs but those infected with the higher dosage lost all worms from the gut through the prepatent period. All pigs infected
with 50 eggs became patent. Roepstorff et al. (1997) observed that the level of larval expulsion from the gut during late migration was linearly related to the infective dose in experimental infections of 100, 1,000 or 10,000 eggs per pig.

1.3 Pathogenesis

It is believed that *A. suum* is a rare cause of clinical disease in pigs (Roberts, 1934; Primm et al., 1990; Miskimins et al., 1994). Even so, damage caused by migrating larvae is observed in tissues and organs of infected hosts and these larvae are thought to be the most important cause of pathological changes associated with the parasite (Soulsby, 1982). Speculation regarding respiratory disease in young pigs related to *A. suum* larval migration ("thumps") began shortly after the life cycle was postulated by Stewart (1916) (Ransom and Foster, 1919). Respiratory symptoms are obvious in infected young animals (Ransom and Foster, 1919; Roberts, 1934) and in older, ascarid-naïve pigs exposed suddenly to large numbers of infective eggs (Miskimins et al. 1994; Hurnik and Dohoo, 1995). Clinical signs include dyspnea and coughing caused by larvae migrating through the lungs, usually at between day six to ten following initial exposure. Symptoms vary in severity depending on level of exposure and some animals may die (Soulsby, 1982; Miskimins, et al., 1994). An elevated respiratory rate has also been reported in a pig experimentally infected with "an enormous dosage of infective eggs" (Roberts, 1934). Also, opportunistic secondary bacterial and viral infections are observed in lungs of heavily infected
pigs, their entry made easier by damage done by migrating larvae (Humik and Dohoo, 1995). Heavily infected young pigs may become stunted and remain unthrifty for the remainder of their lives and may develop diarrhea (Roberts, 1934; Soulsby, 1982). This decreased growth rate may be due to malabsorption of nutrients from the small intestine caused by the presence of adult worms (Stephenson et al., 1980).

Post-mortem examinations reveal other pathological changes caused by migrating larvae. Liver lesions in market weight pigs have been observed for many years but not positively attributed to ascarid migration until Schwartz and Alicata (1933) described larvae in some of these lesion as consistent in size and shape with *A. suum*. These lesions are now one of the most recognized aspects of ascarid infections in pigs. Schwartz and Alicata (1933) described affected livers as “striking as the liver takes on a mottled appearance, due to the striking contrast between the normal color of this organ and the pale color of the cysts; the contrast is particularly marked when the cysts are numerous.” Attempting to find these lesions in pig livers at slaughter is a now common surveillance technique to determine the *A. suum* infection status of individual herds (Bernardo et al. 1990b). These lesions are referred to as “white-spots” or “milk-spots” and have been described in detail by Roneus (1966). He observed three types of lesions in experimentally infected pigs. Two are referred to as granulation-type (GT), both small (up to 5 mm in diameter) and large (up to 30 mm in diameter); the third is described as lymphonodular-type (LT). Both types of GT lesion appear diffuse, or web-like (Roepstorff et al., 1997),
with visible, gray-white interlobular septa and intense eosinophilic infiltration. The small lesions may be a result of a successful larval migration (Roneus, 1966). The large lesions have, in addition, a central gray-white tissue mass up to 10 mm in diameter, which often contains a degenerating larva (Schwartz and Alicata, 1933; Roneus, 1966). The GT lesions reach maximum number at day 7 PI (Roepstorff et al., 1997) and maximum size at between days 10-14 PI (Roneus, 1966). The damaged tissue will heal within about 25 days (Corwin and Stewart, 1992), 35 days (Miskimins et al., 1994; Copeman and Gaafar, 1972), 40 days PI (Roepstorff et al., 1997) or 2 months (Roneus, 1966). Lymphonodular-type lesions appear to develop from large GT lesions and are pearl-like, semi-transparent, well defined nodules of lymphocytes (Roneus, 1966). These nodules appear beginning approximately day 10 PI and may persist until between days 90-170 PI (Roneus, 1966). Some authors have observed that, during first exposure to *A. suum* larvae, no macroscopic lesions are produced in the liver (Bindseil, 1972). Others have observed only small GT lesions on first infection (Copeman and Gaafar, 1972) with all three types observed on secondary infection. Roneus (1966) reported all three types of lesions in an initial infection. Copeman and Gaafar (1972) also reported greater persistence of lesions after a second experimental inoculation. Roepstorff et al. (1997) found that the number of “white-spots” is directly correlated with the size of the experimental dose of eggs, which conflicts with Bindseil’s (1972) finding that there is no such relationship. Other ascarids, including *Toxocara canis*, *T. cati* and *Parascaris equorum* will cause similar liver lesions in pigs (Roneus, 1966). It is interesting to
note that even with an extremely heavy infection, with very large numbers of “milk-spots” on a single liver, that liver function apparently is not impaired (Roneus, 1966).

Migrating larvae move on to the lungs where they cause damage by breaking out of capillaries into the alveoli producing many minute petechiae (Miskimins et al., 1994). In heavy infections this damage induces varying degrees of pneumonia, with the lungs becoming edematous and congested. Histologically, lung sections display “severe, acute, eosinophilic and granulomatous interstitial pneumonia” (Perry and Strokappe, 1993) and cross-sections of migrating larvae may be observed (Miskimins et al., 1994).

Adult ascarids in the small intestine are generally not thought to do much harm (Soulsby, 1982) but sometimes they cause problems. Large numbers of parasites may occlude the intestinal tract, they may perforate the intestinal wall producing a peritonitis, or they may travel up and occlude the bile duct (Soulsby, 1982). Martin et al. (1984) observed villous atrophy and fusion in experimentally infected animals caused by adult worms and reported reduced absorptive surface area. Stephenson et al. (1980) found in animals infected with post-migratory *A. suum* larvae collected from the intestinal tract of rabbits that the small intestine weight increased due to hypertrophy of the longitudinal and circular muscles. The authors were uncertain of the actual cause of hypertrophy but suggested a combination of physical, biochemical, and immunological stimuli. The authors also
implied, from data gathered during digestibility work in the same trial, that *A. suum* infection interferes with protein, fat and carbohydrate absorption from the gut.

Recent case reports indicate that clinical disease and mortalities due to *A. suum* infection do still occur in modern facilities and in an age of highly effective anthelmintics. Miskimins et al. (1994) investigated an outbreak in Illinois where market weight pigs were moved to a dirt pen for two hours while their concrete pen was cleaned. Ten days later, 35 of 150 pigs from the pen displayed respiratory signs and eventually 13 animals died. Clinical symptoms and *post-mortem* findings, in these apparently ascarid-naïve pigs, were consistent with those described above. Diagnosis was confirmed by finding larvae in the lungs and ascarid eggs in the soil of the dirt pen. A similar outbreak, in Alberta, occurred one week following the movement of 1200 pigs from indoor pens to a large outdoor pen. In this instance 300 pigs displayed clinical respiratory symptoms and 120 animals died over a period of three months (Perry and Strokappe, 1993). *Post-mortem* findings on four pigs included severe, diffuse, pulmonary edema, acute multifocal pulmonary hemorrhage and severe parasitic hepatitis. One pig, dead after three weeks, also had severe secondary bacterial pneumonia caused by *Actinomyces pyogenes* and *Pasteurella multocida* (Perry and Strokappe, 1993). A third report, from Ontario, investigating causes of large numbers of liver lesions in pigs from five farms, attributed the damage to the sudden exposure of naïve animals to large numbers of ascarid eggs (Sanford et al., 1991).
Ascaris suum is able to cause clinical disease or death in other animal species. There have been a number of examples reported. From Australia, there is a report of serious respiratory problems of in cattle fed pig manure as part of their normal ration (McLennan et al., 1974). Larvae in the lungs were identified morphologically as *A. suum*. From Ontario, there is a case of calves displaying severe respiratory problems about 10 days after being housed in a pen previously occupied by pigs (McCraw and Lautenslager, 1971). *Ascaris suum* eggs were isolated from bedding material and larvae were isolated from lungs at post-mortem. There is a report from Saskatchewan of a group of lambs exhibiting respiratory symptoms following access to dirt pens formerly inhabited by pigs (Clark et al., 1989); ascarid larvae were found in the lungs and eggs were found in the feces of flock mates. Also, there is a report of liver condemnations over a number of years in a flock of British lambs. Upon investigation, it was discovered the lambs were rotationally grazed with infected pigs and the lamb’s liver lesions were attributed to ascarid larval migration (Mitchell and Linklater, 1980). It is probable that ascarid larval migration may cause disease problems in animals other than pigs, cattle and sheep.

1.4 Economic importance

*Ascaris suum* is the most important swine parasite throughout the world (Corwin and Stewart, 1992). Unfortunately, there is minimal information on the
economic effects of this parasite in the pig industry in North America, although it is considered to have a major impact (Murrell, 1986). Hale et al. (1985) claimed parasites cost United States pig producers “millions of dollars each year”. There are no published data on the dollar value of losses caused by A. suum or other swine parasites in Canada, only two estimates of losses due to liver condemnations (Polley and Mostert, 1980; Hurnik and Dohoo, 1995).

Researchers have divided economic losses due to ascarid infection in pigs into three main categories. These are: 1) losses due to liver condemnations at packing plants; 2) losses associated with subclinical levels of infection which may reduce growth rates and feed conversion and may result in costs associated with regular anthelmintic treatment; 3) losses due to morbidity and mortality together with subsequent diagnostic and treatment costs (Stewart and Hale, 1988).

Murrell (1986) reports losses of $50,000,000 (43,000,000 lbs.) from liver condemnations in the United States in 1980. In Saskatchewan, Polley and Mostert (1980) reported that 2.5% of livers were condemned because of “milk-spots” and a further 7.9% relegated to animal feed. This damage is a direct loss suffered by the meat packing industry in Canada where producers are paid on dressed weight of animals and any by-products are the property of the packer (Hurnik and Dohoo, 1995). A large abattoir in western Canada has implemented premium pricing for high quality pigs which must have unscarred livers (Julia Keensliside, personal communication, 1999). In Europe, liver condemnations due to A. suum migration appear to be an ongoing problem and, in Northern Ireland, the prevalence of these
liver lesions in market weight pigs increased between 1961 and 1991 (Menzies et al. 1994).

Subclinical reductions in growth rates and feed efficiency are insidious because they are easily overlooked although they constitute a major expense for the pig producer (Stewart and Hale, 1988). There is excellent evidence that infection reduces production efficiencies in experimentally infected pigs but less information on how naturally infected pigs are affected. Spindler (1947) undertook the first published study to determine the effects of A. suum infection on the growth of pigs. He observed dramatic reduction in growth rates in experimentally infected animals. A young pig with 109 worms at post-mortem actually lost weight during the 129 day course of the trial, a pig with 39 worms gained only 48% as much as its paired control, one with 20 adult nematodes gained 55% as much as its paired control and a pig with only 12 worms did not differ in growth rate compared to its control. It is possible, however, that an inadequate diet may have influenced these findings (Bernardo et al. 1990c). Hale et al. (1985), in a more detailed experimental infection study, found that young pigs fed with 600, 6,000, or 60,000 infective A. suum eggs had reduced final weight, average daily gain, and feed to gain ratio varying from between 5 and 13% depending on infection level over the 91 day study period. The average number of worms found at post-mortem was 11.5, 16.3, and 18.5 respectively for the three infected groups. These authors also reported that infection decreased digestion coefficients for dry matter, crude protein, and gross energy as well as reducing nitrogen retention.
There is less information on the effect of ascarids on naturally infected pigs under farm conditions. On Prince Edward Island, Bernardo et al. (1990c) indicated a modest growth rate reduction that was associated with worm load and that control on heavily infected farms could improve daily gain by as much as 1%. Hale and Stewart (1987) calculated that in the United States, increased feed and maintenance costs of light infection to be $1.92 per pig, of medium infection $3.21 and of heavy infection to be $5.56. It is extremely difficult to determine the actual dollar value of losses to the pig industry due to ascarid infection because of fluctuating prices of pigs, feed, anthelmintics and pig by-products, and the lack of information relating to the cost of subclinical infections in barn settings (Moncol, 1993; Roepstorff and Nansen, 1994). It seems likely, however, that the parasite is of economic importance on certain farms.

The case reports mentioned in the previous section (1.3) indicate that there are still costly losses incurred due to morbidity, primarily respiratory symptoms, and mortality caused by *A. suum* infection in pigs (Sanford et al., 1991; Perry and Strokappe, 1993; Miskimins et al., 1994) although these cases may be rare (Primm et al., 1990). Respiratory symptoms and death in other animal species is another source of financial loss (McCraw and Lautenslager, 1971; McLennan et al., 1974; Mitchell and Linklater, 1980; Clark et al., 1989)
1.5 Control measures

The aim of control programs against *A. suum* is the maintenance of infection at levels that do not adversely affect pig health or production. The complete elimination of this parasite, once present in a facility, is virtually impossible (Myers, 1986). Effective control programs will use a combination of sanitation, removing a proportion of the free-living ascarid population, and anthelmintic treatments that will remove parasites from host animals (Roepstorff and Nansen, 1994).

Traditionally, control programs have been designed to accommodate most facilities with a particular management system. The United States Department of Agriculture developed the McLean County System in 1921 (Levine, 1980) for extensively managed pigs. Steps taken to constrain parasite numbers in this system included the cleansing of farrowing pens before introducing the sow, washing and treating the sow prior to farrowing, moving litters by trailer to clean pastures, not herding them through contaminated alleyways, and rotating animals frequently through clean pastures (Murrell, 1986). A system developed in 1979, the North Carolina Parasite Control Program, focused on confined animals (Murrell, 1986). Recommendations from this program included treating sows with an anthelmintic 5–10 days prebreeding and again pre-farrowing, treating piglets at 5–6 weeks of age and again a month later and treating boars once per year.

Currently, the use of anthelmintics alone is the most common control procedure used by producers (Roepstorff and Nansen, 1994). Modern anthelmintics
available to pig producers are highly effective against *A. suum*. They are available in a variety of formulations and are easily administered in either feed or water.

Injectable anthelmintics are also available (Canadian Animal Health Institute, 1995). Treatments may be strategically targeted to specific animal groups in the barn at various production stages (e.g. sows pre-farrowing) or, more usually target the entire facility at certain times of the year (Roepstorff and Nansen, 1994). Unfortunately, the use of these medications alone may be ineffective. In barns contaminated with large numbers of ascarid eggs animals are continuously infected (Roepstorff and Nansen, 1994). Mercy et al. (1989b) found that 80% of farms using anthelmintics as the primary method of parasite control still had evidence of nematode infection.

Menzies et al. (1994) observed, on the basis of increased numbers of condemned livers over a number of years, that current control procedures are not effective.

The most recent parasite control recommendations are not based on general management systems, intensive or extensive, within the pig industry. Instead they are based on evaluating individual herds and their management, housing, and parasitological status then incorporating a combination of management, primarily sanitation, practices followed by anthelmintic treatment only when necessary (Moncol, 1993; Roepstorff and Nansen, 1994). The impact of *A. suum* will vary between herds depending on a variety of management factors (Primm et al., 1990). Non-treatment factors that reduce *A. suum* prevalence in indoor housing systems include a high levels of sanitation, early weaning (3-4 weeks) of piglets, use of slatted floors at all stages of production, and all-in all-out production systems.
(Primm et al., 1990; Moncol, 1993; Menzies et al., 1994; Roepstorff and Nansen, 1994). Incorporation of these features into management systems may adequately control parasite numbers making anthelmintic treatment unnecessary (Roepstorff and Nansen, 1994; Roepstorff, 1997). Anthelmintic treatment would be recommended only if intensive management practices did not maintain parasites at acceptable levels as determined by regular fecal monitoring of the various production groups within a barn. Roepstorff (1997) observed this to be an acceptable procedure in a three-year study, but more research must be carried out. Programs such as this that rely primarily or exclusively on management may also be beneficial in preventing the development of anthelmintic resistance among pig helminths (Roepstorff and Nansen, 1994; Dangolla et al, 1996a; Roepstorff, 1997).

1.6 Zoonotic aspects

*Ascaris suum* is zoonotic (Anderson, 1995; Hurnik and Dohoo, 1995) but there is still debate regarding frequency and importance of human infections; some authors indicate it is rare (Anderson, 1992) while others believe that it is greatly under-reported (van Knapen et al., 1992; Anderson, 1995).

Governments in western Canada are currently encouraging expansion of pig production. In Saskatchewan there are plans to triple the number of market hogs from the current one million animals to three million by 2000-2002 (Saskatchewan Pork Profile, 1997). This increase in animal numbers raises several concerns
including issues of sustainability, of environmental contamination, of odor problems, and of manure utilization.

As well, there are specific issues raised because of the known presence of *A. suum* in the provincial pig herd (Polley and Mostert, 1980; Wagner and Polley, 1997b) which are summarized in two questions “Are current methods of manure utilization a public health hazard due to the presence of pig ascarid eggs?” and “Will increasing production levels increase risk of human, or other animal, infection?”

Pig manure is a valuable fertilizer adding organic matter and other nutrients to soil. The current pattern of manure management on Saskatchewan pig farms is first temporary storage of liquid manure in pits beneath the barn. These pits are regularly emptied into earthen sewage lagoons. Current regulations for new barns require at least 400 days of storage capacity in these lagoons. There is somewhat conflicting evidence related to ascarid egg survival in the sewage lagoons. A recent report from Holland by Gaasenbeek and Borgsteede (1998) describes complete loss of egg viability, based on microscopic examination, by 16 weeks in the lagoon, while Johnson et al. (1998), in Australia, determined that 42-49% of non-embryonated eggs remained viable and were able to develop and infect mice after 29 weeks in a sludge lagoon. In the same study 90% of eggs already larvated were able to survive and were infective to mice after 29 weeks in the lagoon environment.

It is usual practice in Saskatchewan that approximately once per year the long-term storage lagoons are emptied with the contents spread on, or injected into, cropland. Ascarid eggs may be more susceptible here than in the wet environment of
the lagoon. It has long been known that a combination of desiccation and sunlight kills eggs quickly (Caldwell and Caldwell, 1928) and recent research by Gaasenbeek and Borgsteede (1998) has confirmed this. In their study eggs survived for only two to four weeks under dry/sunny conditions. Optimal conditions for egg survival were in wet/shaded areas where 90% of eggs survived for longer than eight weeks. Roepstorff and Murrell (1997) observed that in Denmark, during a dry summer, almost all the eggs deposited on a pig pasture by infected pigs died very quickly. Saskatchewan croplands vary in moisture level but they all suffer from a lack of shade and this may influence egg survival rates. There are, however, no published data on the microclimate as it influences ascarid egg survival and development on Saskatchewan fields.

Occasionally, *Ascaris* infection in people is reported from areas where *A. lumbricoides* is non-endemic. These are thought to be cross-infections with *A. suum* (Anderson, 1995). There have been approximately 66 of these instances reported over the last 38 years: seven from Canada (Phills et al., 1972; Eaton, 1985; Anderson, 1995); 37 from the United States (Bullock, 1961; Jaskoski, 1961; Lord and Bullock, 1982; Shoemaker-Nawas et al., 1982; Anderson, 1995) five from England (Phillipson and Race, 1967; Crewe and Smith, 1971; Denham, 1984; Eddy, 1985) and 17 from Japan (Maruyama et al., 1996). Most of these cases were diagnosed as infection with *A. suum* based on circumstantial evidence but Anderson (1995) was able to confirm *A. suum* infection in North America in nine instances based on molecular differences between the two species. In Sweden, another 20
local cases with unknown histories, except lack of travel, were diagnosed as *A. lumbricoides* (Willcox, 1985) but may well have been *A. suum* (Anderson, 1995). Anderson (1995) questioned why cross infections such as these are rare while *A. suum* infections are common in domestic pigs worldwide. The author suggested that this paradox is associated with differences in host and parasite genetics and physiology. Only individuals immunocompromised in some way may be susceptible or perhaps only a fraction of the *A. suum* population contains alleles allowing them to establish in people. It is interesting that in one study in Guatemala, where both *A. suum* and *A. lumbricoides* are endemic, it was observed that the two species maintain separate host-specific transmission cycles (Anderson et al., 1993). Anderson (1995) did not indicate that *A. suum* infection in people might depend on the degree of exposure to pigs and pig manure. Taffs (1985) observed that when the source of human ascarid infection is obscure, investigators should determine if the patient has had contact with pigs or pig manure. Eaton (1985), a Canadian physician, reported observing many such cases and he “eventually adopted a practice of inquiring on each occasion as to whether there was any association with pig farms or manure” and was never disappointed.

Forty-nine of the 66 human cases referred to above had regular access to pigs, pig manure, or old pig sties. In two cases, researchers were infected accidentally in the laboratory (Jaskoski, 1961; Anderson, 1995). Only nine of these 51 cases were reported as symptomatic (Maruyama et al., 1996), suggesting that *A. suum* infection in people is not commonly associated with significant clinical
problems. Three cases with no history were diagnosed as *A. suum* based on molecular evidence (Anderson, 1995). Four reported cases were the result of an intentional infection with apparent criminal intent, and all these individuals were symptomatic (Phills et al., 1972). The remaining cases had no history (7) or a recent travel history (1) to areas with endemic *A. lumbricoides* and could not, therefore, be attributed to *A. suum*.

Saskatchewan has a huge land base with a total of 161.1 million acres (570,264 square kilometers) of which approximately 50 million are cultivated farmland (Saskatchewan Agriculture and Food, 1997a). Current levels of pig production use only about 300,000 acres (0.6% of the total) for manure disposal. Production increases to projected levels will require approximately one million acres (2.0% of the total) (Saskatchewan Agriculture and Food Manure Management Spreadsheet calculations, 1998). This province also has a low human population density with only 1.73 people per square kilometer. It also currently has a pig population of only three pigs per square kilometer of the 47% of provincial land suitable for agricultural use (Saskatchewan Agriculture and Food, 1997b). In contrast, the Netherlands has a population density of about 364 people and about 340 pigs on each of 41,160 total square kilometers (Gaasenbeek and Borgsteede, 1998; Saskatchewan Pork Profile, 1997). There is little evidence of *A. suum* related public health problems associated with manure utilization in the Netherlands although one study suggested an exposure level of 7-10% based on seroprevalence (van Knapen et al., 1992). These authors also report that pig manure is spread on public parks in that
country. It seems reasonable to suggest, therefore, that direct contact between people and infective eggs of *A. suum* on Saskatchewan cropland would be extremely unlikely.

Unless grossly under-reported, human infection with *A. suum* appears to be associated with repeated and intensive exposure to pigs or soil contaminated with pig manure. Saskatchewan’s huge land base and low human population density assist in preventing this type of contact. Infective eggs may be present on cropland but contact is limited to those working in pig production and related fields (pig farmers and families, packing plant workers, and employees of companies specialized in manure disposal). It is important, however, that care is employed if fertilizing gardens with pig manure (Shoemaker-Nawas et al., 1982) and risks should be assessed before spreading pig manure on cattle, horse, or sheep pastures (Murrell, 1986).
2.0 OBJECTIVES

The objectives of this study were: 1) to assess the prevalence and intensity of *A. suum* in Saskatchewan market weight pigs through an abattoir survey; 2) to investigate the current anthelmintic use on Saskatchewan pig farms and assess producers' perceptions of the problems associated with *A. suum* through the use of a postal survey; and 3) to investigate the epidemiology of the parasite in intensively managed pigs in Saskatchewan by studying egg development and infection patterns within barns.
3.0 *Ascaris suum* Prevalence and Intensity: An Abattoir Survey of Market Hogs in Saskatchewan

3.1 Introduction

*Ascaris suum*, the large roundworm of pigs, is found commonly in grower pigs worldwide (Pattison et al., 1980; Marti and Hale, 1986; Mercy et al., 1989a; Roepstorff and Jorsal, 1989) and is widespread in Canadian animals (Martin et al. 1974; Polley and Mostert, 1980; Bernardo et al., 1990a).

It is possible that improvement and increased intensity of pig management over the past 15 years, as well as the introduction of broad spectrum anthelmintics into the Canadian market, have affected the prevalence and intensity of *A. suum*. This paper reports the results of a 1995 abattoir survey of the prevalence and intensity of *A. suum* in market weight (100-110 kg) hogs from Saskatchewan and compares them to results from a similar survey conducted here in the late 1970's (Polley and Mostert, 1980).
3.2 Materials and Methods

A local abattoir, handling the majority of the province’s market weight pigs, was visited one to three times per week for four months in early 1995 (January to April) and two prevalence and intensity surveys were undertaken: the first of liver lesions associated with larval ascarid migration (Survey 1: 2500 pigs); the second of both hepatic lesions and adult ascarids in the small intestines (Survey 2: 500 pigs). The survey methods used were as described previously (Polley and Mostert, 1980). Briefly, in the first survey, liver surfaces were examined for milk-spot lesions and scored according to the following scale; 0 = no lesions, 1-4 = number of major liver lobes affected, and 5 = all four major lobes affected with some liver scarring. In the second survey, each liver was examined and scored in the same manner. Additionally, the corresponding small intestine was removed from the mesentery, the contents squeezed into a collection tray and then the entire intestine opened longitudinally to recover and count the immature and mature adult ascarids. Almost all pigs included in the present study were identified by farm of origin based on tattoo numbers. Descriptive statistics were calculated using Statistix® (Analytical Software, 1994). Prevalence and intensity distributions in the 1995 survey were compared with those from the earlier study, carried out from November to February (Polley and Mostert, 1980) using a chi-squared test of heterogeneity (Statistix®, Analytical Software, 1994).
3.3 Results

Survey 1:

The distribution of liver lesion scores is shown in Figure 3.3-1. One thousand one hundred (44%) of 2500 animals examined were positive for “milk-spots” with most affected livers (897 (35.9%)) displaying relatively minor damage (scores of 1 to 3). Only 203 (8.1%) of pigs were more severely affected (scores of 4 or 5). Animals from approximately 400 farms were examined, and infected pigs were found to originate from 325 (81%) of these. The numbers of farms included may be an underestimate because pigs from several small farms are often assembled at one location where they are all given the same identification number prior to transportation to the abattoir.

Survey 2:

Adult ascarids were found in 88 (17.6%) of 500 pigs examined. The mean intensity, among infected animals, was 2.5 (range 1-52). Only one pig contained more than 50 parasites. Figure 3.3-2 presents the frequency distribution of adult parasites. Seventy-two pigs (14.4%) had both liver lesions and adult worms while 16 (3.2%) harboured adults without displaying liver lesions. Of the animals examined, 179 (35.8%) were positive for “milk spots” without adult parasites and 233 (47%) had neither “milk-spots” nor adult worms. In total, therefore, 267 (53%) of the 500 pigs examined displayed evidence of A. suum infection. Market weight animals
from approximately 211 farms were examined, and infected pigs were found to originate from 152 (72%) of these. The distribution of liver scores was very similar to that for Survey 1. In this survey, the presence of hepatic lesions predicted the presence of intestinal stages with a sensitivity of 82% and a specificity of 57%. The positive and negative predictive values were 29% and 94% respectively.
Figure 3.3-1. The distribution of the severity of milk-spot lesions in 2500 market weight pigs in 1980 (Polley and Mostert, 1980) and 1995 (Survey 1).
Figure 3.3-2. The intensity of infection by adult ascarids in 2500 (1980) (Polley and Mostert, 1980) and 500 (1995) market weight pigs (Survey 2).
3.4 Discussion

On the basis of milk-spot liver lesions the prevalence of *A. suum* changed little between the late 1970's and 1995 (Polley and Mostert, 1980). The liver score distributions in the two surveys are heterogeneous ($\chi^2=112.52$, $p<0.0001$) but the practical significance of this is uncertain.

The majority of the hepatic lesions observed in both surveys were probably caused by *A. suum* although other migrating helminths, especially other ascarid species, may cause similar lesions in pigs. It would be unusual, however, for intensively managed, confined pigs, such as those raised in Saskatchewan, to be exposed to these other parasites (Polley and Mostert, 1980).

The prevalence and intensity of adult *A. suum* in 1995 were both reduced compared to 1980 (Polley and Mostert, 1980). These reductions are significantly different ($\chi^2=71.39$, $p<0.0001$). It is possible that this is a result of improved management as well as increased anthelmintic use, including the use of ivermectin for the control of helminths and sarcoptic mange. In the two surveys, the unchanged prevalence and intensity of liver lesions, together with the reduced prevalence and intensity of adult *A. suum* infections, suggest that although animals are continuing to ingest infective eggs and that the larvae released are migrating to the liver, either they do not complete the migration to the intestine or, once in the intestine, they are removed by more effective anthelmintics or improved treatment schedules. This hypothesis is supported by the results of studies in Denmark where liver ascarid
lesion prevalence has been consistent over the past 20 years but the prevalence of *A. suum* adults (as measured by fecal examination) appears to be decreasing (Roepstorff and Jorsal, 1989). It is possible that anthelmintic treatment occurs most commonly once the infections have become patent and the adult worms have contaminated the barn environment with large numbers of eggs.

In Quebec Canada, Martin et al. (1974) found that 39% of 90 market weight animals examined were infected with adult ascarids. On Prince Edward Island, Bernardo et al. (1990a) found liver milk-spot prevalence to be 82% (313/380) in market weight pigs but the animals included originated from farms selected because of a history of ascarid infection and no anthelmintic treatment. In addition, thirty-four percent of the Prince Edward Island animals harboured adult *A. suum*. The study demonstrated that abattoir surveillance of hepatic lesions as an indicator of adult ascarid infection had a sensitivity of 90.8%, specificity of 22%, positive predictive value of 37.7% and negative predictive value of 82.1%. Thus both the Prince Edward Island and Saskatchewan surveys indicate that the presence of milk-spot lesions does not always predict the presence of intestinal worms, but the absence of such lesions usually indicates the absence of ascarids in the intestine (Bernardo et al., 1990b). Larval ascarid lesions in the liver will heal in time and if there is no further ingestion of infective eggs there may be adult worms in the intestine with no visible hepatic damage (Bernardo et al., 1990a). On the basis of these two studies, therefore, it seems that the presence of liver lesions is the most useful indication of ascarid infection in a pig herd.
Worldwide, there are few published studies of abattoir surveys of parasites of pigs. Pattison et al. (1980) in England, found 11.8% of market weight animals infected with adult ascarids with a mean worm count of three among infected pigs. Jacobs and Dunn (1969) in Scotland found adult parasites in 28.6% of market weight pigs with a mean count of 4.5 adults per infected pig. The differences in prevalence and intensity between the United Kingdom and Saskatchewan may be due to differences in climate and in management or treatment practices.

The prevalence and intensity data from this survey are applicable only to this age group of pigs. Detailed cross-sectional and longitudinal studies of in-barn prevalence and intensity need to be carried out to develop a complete picture of the epidemiology of *A. suum* in confined pigs in Saskatchewan.
4.0 ANTHELMINTIC USE ON SASKATCHEWAN PIG FARMS: RESULTS FROM A POSTAL SURVEY.

4.1 Introduction

There are several highly effective anthelmintics available to pig producers in Canada (Canadian Animal Health Institute, 1995). Manufacturers of most products claim greater than 95% efficacy against intestinal stages of *A. suum*. In addition, some anthelmintics are also effective against larvae migrating in the host tissues. In Saskatchewan, abattoir surveys of market-age animals have demonstrated *A. suum* prevalences of 60% and 53% based on the presence of adult parasites and typical liver lesions (Polley and Mostert, 1980; Wagner and Polley, 1997b). That *A. suum* remains common in pigs in the province may be in part a consequence of the extent and timing of anthelmintic treatments. There are limited published data on the use of these products in parasite control programs (Mercy et al., 1989b; Dangolla et al., 1996a) and none for North America. This paper reports the results of a postal survey of anthelmintic use by Saskatchewan pig producers during 1995.
4.2 Materials and Methods

Survey Sample Selection

Saskatchewan Pork International (SPI) is a provincial agency that, at the time, marketed all of the pigs sent to abattoirs in the province. Data from SPI were used to select 580 pig farmers from the approximately 3000 in Saskatchewan (SPI Marketing Group, 1994). Less than 10% of the province's market weight pigs are produced by approximately 2100 small farms (1-200 pigs annually) and 42% are produced by approximately 60 large farms (>4000 annually) (SPI Marketing Group, 1994). Producers were selected in a random stratified manner to ensure appropriate representation of each farm size category; 1-200; 201-1000; 1001-2000; 2001-4000; and >4000. The stratification was based both on the number of farms in each size category as well as that group's contribution to the total pig production in the province. Farm size may be associated with management intensity, the larger farms being more intensively managed.

Survey Form

The following information for 1995 was sought using a multiple choice survey form (Appendix A) distributed in March, 1996:

1) farm size confirmation (see above);

2) production system (farrow to finish; farrow to weaning or weaning to finish)
3) presence or absence of perceived parasite problem and how detected by the farmer;

4) "High Health" herd (Specific Pathogen Free (SPF) or similar) or not;

5) anthelmintics used in 1995 or not;

6) perceived efficacy and benefit of anthelmintic treatment;

7) age categories treated, treatment patterns and anthelmintics used in each age category (weanlings: 3 to 8 weeks; growers: 8 weeks to 6 months; and breeding stock);

8) reasons for anthelmintic selection and sources of product information.

Descriptive statistics were calculated using Statistix® (Analytical Software, 1994)

4.3 Results

Farm Characteristics

The overall survey response rate was 33% (190/580) and included responses from 49 of the approximately 200 (24.5%) farms in the province that market more than 1000 pigs per year. These farms represent approximately 67% of all animals marketed. Of all farms responding, 61% were farrow to finish, 31% were weaning to finish and 8% were farrow to weaning or "other". More than half the producers selling more than 1000 pigs per year considered themselves to have "High Health" herds. Overall, 48% of respondents believed they had a parasite problem in their
animals, most often determined by the farmer's observation of adult parasites in the
feces (52%) or a perceived production problem (50%) (Table 4.3-1).

Seventy-six percent of all respondents treated some portion of their pig herd
with an anthelmintic in 1995. Although less than half of the largest farms (>4000
pigs marketed annually) had used anthelmintics during the year, almost all of these
farms had followed a "planned treatment program" (Table 4.3-1). Among all
producers, 96% believed anthelmintics to be effective and beneficial when used on
their farms. These medications were most often chosen on the basis of this perceived
effectiveness (on 81% of farms) and ease of use (66% of farms). Cost was a factor
for 16% of respondents. Veterinarians were the main source of product information
in all farm size categories (57%), followed by feed companies (36%) and advertising
(24%). Other producers were consulted only by respondents in the two smallest
farm size categories.

Treatment Characteristics

These were similar among farms in all size categories (Tables 4.3-2 to 4.3-5).
A macrolide was the only injectable used by respondents. Figure 4.3-1 shows the
distribution, by age group of pigs, of the anthelmintics administered in-feed.
Hygromycin B was used on only one farm in the smallest size category. In-water
treatments were used on only one of the larger farms. Both of the anthelmintics
currently registered in Canada for in-water administration to pigs (levamisole - an
imidathiazole, and piperazine) were used by respondents to treat animals in all age groups.

**Weanlings (approximately 3 weeks (age at weaning) to 8 weeks of age)**

Among all respondents, 86% had treated weanling pigs during 1995 (Table 4.3-2). Most commonly, in all farm size categories, all weanling pigs were treated. Two of five producers from the largest farms that treated weanlings did not treat all animals in this age group (Table 4.3-2).

**Growers (approximately 8 weeks to approximately 6 months of age)**

Sixty nine percent of all respondents had treated grower pigs in 1995. On approximately half of these farms, pigs in the grower barns were treated two or three times during the year (Table 4.3-3).

**Breeding Animals (sows and boars)**

During 1995, sows were treated by 90% of producers. On approximately half the farms they were treated pre-farrowing and, on approximately 20%, pre-breeding (Table 4.3-4). Boars were treated on 75% of farms and approximately half of respondents treated two or three times during the year (Table 4.3-5).
Table 4.3-1. Postal survey of anthelmintic use on Saskatchewan pig farms: Survey response rate and overall anthelmintic use.

<table>
<thead>
<tr>
<th>Farm Size Sold/Year</th>
<th>Survey Response Rate (%)</th>
<th>Farmers Perceiving a Parasite Problem (%)</th>
<th>Parasite Problem Determined By (%):</th>
<th>Farms Treating Anthelmintic In Treatment 1995 (%)</th>
<th>Farms Using a Planned Treatment Program (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-200</td>
<td>80/370 (22)</td>
<td>58</td>
<td>56** 56 7 7</td>
<td>76</td>
<td>57</td>
</tr>
<tr>
<td>201-1000</td>
<td>61/140 (44)</td>
<td>53</td>
<td>56 41 9 6</td>
<td>90</td>
<td>61</td>
</tr>
<tr>
<td>1001-2000</td>
<td>16/20 (80)</td>
<td>29</td>
<td>50 25 25 0</td>
<td>69</td>
<td>73</td>
</tr>
<tr>
<td>2001-4000</td>
<td>12/20 (60)</td>
<td>42</td>
<td>80 0 20 0</td>
<td>67</td>
<td>50</td>
</tr>
<tr>
<td>&gt;4000</td>
<td>21/30 (70)</td>
<td>19</td>
<td>50 50 25 25</td>
<td>48</td>
<td>90</td>
</tr>
<tr>
<td>Overall</td>
<td>190/580 (33)</td>
<td>48</td>
<td>52 50 10 7</td>
<td>76</td>
<td>62</td>
</tr>
</tbody>
</table>

*1, adult worms in feces; 2, poor performance; 3, fecal flotation/egg count; 4, slaughter check of livers for ascarid migration lesions; ** a total of >100% in any farm size category indicates more than one choice by individual farms.
Table 4.3-2. Postal survey of anthelmintic use on Saskatchewan pig farms: Weanling treatment.

<table>
<thead>
<tr>
<th>Farm Size (Pigs Sold/Year)</th>
<th>Number of Farms</th>
<th>Number of Farms Treating Any Weanlings (%)</th>
<th>Percentage of Farms Treating any Weanlings</th>
<th>Percentage of Farms Using Anthelmintic Administration Route:</th>
<th>Injectable</th>
<th>In-Feed</th>
<th>In-Water</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-200</td>
<td>61</td>
<td>60</td>
<td>93 3 0 2 2</td>
<td>70** 45 42</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>201-1000</td>
<td>55</td>
<td>49</td>
<td>84 4 4 8 0</td>
<td>80 57 22</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1001-2000</td>
<td>11</td>
<td>8</td>
<td>100 0 0 0 0</td>
<td>50 63 0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2001-4000</td>
<td>8</td>
<td>3</td>
<td>100 0 0 0 0</td>
<td>33 67 0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;4000</td>
<td>10</td>
<td>5</td>
<td>60 20 0 0 20</td>
<td>40 60 20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall</td>
<td>145</td>
<td>125</td>
<td>89 4 1.5 4 1.5</td>
<td>70 52 30</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*1, all weanlings; 2, 75% of weanlings; 3, 50% of weanlings; 4, 25% of weanlings; 5, other;
** a total of >100% in any farm size category indicates more than one choice by individual farms.
Table 4.3-3. Postal survey of anthelmintic use on Saskatchewan pig farms: Grower treatment.

<table>
<thead>
<tr>
<th>Farm Size (Sold/Year)</th>
<th>Number of Farms With Growers</th>
<th>Number of Farms Treating any Growers (%)</th>
<th>Percentage of Farms Treating any Growers</th>
<th>Percentage of Farms Using Anthelmintic Administration Route:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>1* 2 3 4</td>
<td>Injectable In-Feed In-Water</td>
</tr>
<tr>
<td>1-200</td>
<td>58</td>
<td>49</td>
<td>16 57 20 7</td>
<td>57** 49 51</td>
</tr>
<tr>
<td>201-1000</td>
<td>51</td>
<td>36</td>
<td>17 47 25 11</td>
<td>50 50 22</td>
</tr>
<tr>
<td>1001-2000</td>
<td>10</td>
<td>4</td>
<td>25 25 0 50</td>
<td>50 75 20</td>
</tr>
<tr>
<td>2001-4000</td>
<td>8</td>
<td>1</td>
<td>0 0 100 0</td>
<td>0 100 0</td>
</tr>
<tr>
<td>&gt;4000</td>
<td>10</td>
<td>4</td>
<td>25 25 25 25</td>
<td>50 75 0</td>
</tr>
<tr>
<td>Overall</td>
<td>137</td>
<td>94</td>
<td>17 50 22 11</td>
<td>53 52 35</td>
</tr>
</tbody>
</table>

*1, >3X/year; 2, 2-3X/year; 3, 1X/year; 4, other; ** a total of >100% in any farm size category indicates more than one choice by individual farms.
Table 4.3-4. Postal survey of anthelmintic use on Saskatchewan pig farms: Sow treatment.

<table>
<thead>
<tr>
<th>Farm Size (# Pigs Sold/Year)</th>
<th>Number of Farms With Sows</th>
<th>Number of Farms Treating Any Sows (%)</th>
<th>Percentage of Farms Treating any Sows Using Anthelmintic Administration Route: Injectable In-Feed In-Water</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-200</td>
<td>40</td>
<td>33</td>
<td>24 33 16 9 18</td>
</tr>
<tr>
<td>201-1000</td>
<td>38</td>
<td>37</td>
<td>38 16 14 16 16</td>
</tr>
<tr>
<td>1001-2000</td>
<td>9</td>
<td>7</td>
<td>43 14 0 14 29</td>
</tr>
<tr>
<td>2001-4000</td>
<td>8</td>
<td>8</td>
<td>37 13 13 0 37</td>
</tr>
<tr>
<td>&gt;4000</td>
<td>9</td>
<td>9</td>
<td>56 0 0 0 44</td>
</tr>
<tr>
<td>Overall</td>
<td>104</td>
<td>94</td>
<td>35 20 12 11 22</td>
</tr>
</tbody>
</table>

*1, regularly prefarrowing; 2, occasionally prefarrowing; 3, regularly prebreeding; 4, occasionally prebreeding; 5, other; ** a total of >100% in any farm size category indicates more than one choice by individual farms.
Table 4.3-5. Postal survey of anthelmintic use on Saskatchewan pig farms: Boar treatment.

<table>
<thead>
<tr>
<th>Farm Size (# Pigs Sold/Year)</th>
<th>Number of Farms With Boars</th>
<th>Number of Farms Treating Any Boars (%)</th>
<th>Percentage of Farms Treating any Boars Using Treatment Pattern:</th>
<th>Percentage of Farms Using Anthelmintic Administration Route:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>1* 2 3 4</td>
<td>Injectable In-Feed In-Water</td>
</tr>
<tr>
<td>1-200</td>
<td>40</td>
<td>27</td>
<td>4 48 44 4</td>
<td>48** 37 41</td>
</tr>
<tr>
<td>201-1000</td>
<td>38</td>
<td>30</td>
<td>7 53 40 0</td>
<td>67 33 27</td>
</tr>
<tr>
<td>1001-2000</td>
<td>9</td>
<td>7</td>
<td>0 43 57 0</td>
<td>57 57 0</td>
</tr>
<tr>
<td>2001-4000</td>
<td>8</td>
<td>7</td>
<td>0 14 72 14</td>
<td>43 57 0</td>
</tr>
<tr>
<td>&gt;4000</td>
<td>9</td>
<td>7</td>
<td>14 72 14 0</td>
<td>33 44 0</td>
</tr>
<tr>
<td>Overall</td>
<td>104</td>
<td>78</td>
<td>5 48 44 3</td>
<td>55 41 24</td>
</tr>
</tbody>
</table>

*1, >3X/year; 2, 2-3X/year; 3, 1X/year; 4, other; ** a total of >100% in any farm size category indicates more than one choice by individual farms.
Figure 4.3-1. Anthelmintics used in feed for various age categories of pigs.

(A, benzimidazole; B, imidathiazole; C, macrolide; D, organophosphate;
E, piperazine; F, tetrahydropyrimidine).
4.4 Discussion

The results of this survey provide data on perceived parasite problems and on anthelmintic treatment use for approximately 20% of the pigs marketed each year in Saskatchewan. It is possible that producers responding may have been those most interested in these aspects of management and the results may emphasize what is done on these farms rather than in the industry as a whole.

Based on this survey, almost half of respondents believed that endoparasites were a problem in their herds in 1995. This belief was most often based on somewhat subjective evidence: either the detection of adult parasites in feces, which does not provide accurate data on prevalence or intensity of infection, even for *A. suum*, and which may not detect smaller helminths such as *Oesophagostomum* spp. or *Trichuris suis*; or “poor performance”, which may be difficult to accurately assess and which may be due to factors other than endoparasitism. More objective measures, such as information from fecal flotations and/or egg counts, or slaughter checks for liver lesions due to ascarids, were less commonly used, the last primarily by producers marketing more than 4000 pigs per year.

Other published surveys of anthelmintic use on pig farms do not include data on assessment of parasite problems by farmers (Mercy et al., 1989b; Dangolla et al., 1996a). In a recent study in Denmark, detection of parasites in feces after treatment along with “improved general appearance of pigs” and slaughter checks of livers
were used by farmers to assess anthelmintic efficacy. The authors concluded, however, that in their study these methods were not reliable (Dangolla et al., 1996a).

In 1995, approximately three-quarters of survey respondents used anthelmintics for endoparasite control with most following a “planned treatment program”. The number of producers treating for endoparasites may actually be higher than indicated by the survey because of the use of ivermectin for the control of sarcoptic mange. A larger proportion of farmers used anthelmintics than believed they had a parasite problem. Although the data do not provide a clear explanation, it is possible that during the year these medications were administered by some producers to treat a perceived problem that, as a result, had resolved, and by others to prevent the development of a parasite problem. In Australia, fecal flotation demonstrated parasitic infection on 79% of farms that treated regularly with anthelmintics and routine or occasional treatments did not affect the prevalence of parasites except in porkers (equivalent to young growers) (Mercy et al., 1989b).

In the current study, weanling pigs were the second most commonly treated age group, after sows. Similar results have been reported from a survey in Australia, where weanlings were treated on 83% of farms (Mercy et al., 1989b). In Denmark however, weanlings were treated by only 33% of producers (Dangolla et al., 1996a). Five years’ data from a farm in Georgia on which untreated young pigs were weaned at 5-7 weeks revealed prevalences, based on fecal flotations, of 0 to 2.2% for *A. suum*, *Oesophagostomum* spp. and *Trichuris suis* (Marti and Hale, 1986). In Denmark, in a similar study of 66 farms weaning at 3 to 10 weeks and with unknown
treatment histories, *A. suum* was found in 10% of weanlings, *Oesophagostomum* spp. in 7% and *Trichuris suis* in 1% (Roepstorff and Jorsal, 1989). In a subsequent Danish survey of untreated pigs aged less than 12 weeks on 8 farms with different management systems, patent *A. suum* infections were found in 0 to 78% of the animals, *Oesophagostomum* spp. in 0 to 82% and *Trichuris suis* in 10% of the animals on only one farm. In general, prevalences on the more intensively managed farms, where pigs were weaned at 4 to 5 weeks, were significantly lower than in more traditionally managed indoor herds where weaning was later (up to 14 weeks of age) (Roepstorff, 1991). In Australia, *A. suum* was found in weanlings on 17.7% of farms, *Oesophagostomum* spp. on 14.7%, *Trichuris suis* on 7.3% and *Hyostrongylus rubidus* on 4.2%. Of the farms included in this survey, 83% used anthelmintics routinely or occasionally (Mercy et al., 1989b). There are no prevalence data from Canada specifically for weanlings. In a survey in Quebec, based on fecal flotations, between 50% and 100% of “young pigs” from farms with unthriftiness or excessive sow culling were “positive for parasites” (Martin et al., 1974).

The least commonly treated age group in Saskatchewan were pigs in the grower barns. In Denmark, growers were treated on only 14% of farms (Dangolla et al., 1996a) but, in Australia, on 81% (Mercy et al., 1989b). On-farm surveys in the United States (Morris et al., 1984; Marti and Hale, 1986), the United Kingdom (Jacobs and Dunn, 1969; Pattison et al., 1980), Australia (Mercy et al., 1989a) and Denmark (Roepstorff, 1991) have demonstrated that it is grower pigs that have the highest *A. suum* prevalence and egg counts, ranging up to more than 90% and more
than 2000 eggs per gram in traditionally managed, untreated, indoor herds. In Canada, in a longitudinal study on Prince Edward Island, approximately 30% of pigs were found by fecal examination to be infected with adult ascarids during their time in the grower barn and, when slaughtered at market weight, liver lesions associated with larval ascarid migration were found in 82% of these animals (Bernardo et al., 1990a). In this study, however, farms were selected in part because they had a history of ascarid infections. In a Saskatchewan abattoir survey, 60% of market weight pigs, of unknown disease history, showed evidence of ascarids (Polley and Mostert, 1980). In on-farm surveys of grower pigs in North America and Europe, prevalences of \textit{Oesophagostomum} spp. ranged from 8.5% to 80% and of \textit{Trichuris suis} from 0% to 29% (Jacobs and Dunn, 1969; Martin et al., 1974; Pattison et al., 1980; Morris et al., 1984; Marti and Hale, 1986; Roepstorff and Jorsal, 1989; Roepstorff, 1991). In Australia, \textit{A. suum} was found in growers on 25% of farms, \textit{Oesophagostomum} spp. on 24%, \textit{Trichuris suis} on 15.3% and \textit{Hyostrongylus rubidus} on 4.8% (Mercy et al., 1989a).

In Saskatchewan, sows were the most commonly treated age group, as they were in Australia (83% of farms; Mercy et al., 1989b) and Denmark (91% of farms; Dangolla et al., 1996a). There are no published reports on the prevalence of helminth parasites in sows in Canada. Elsewhere, on-farm surveys conducted over the past 30 years have shown prevalences of patent \textit{A. suum} in sows housed indoors ranging from 3.5% in northern England 1980 (Pattison et al., 1980) to 16.7% in Georgia (Marti and Hale, 1986) and, of patent \textit{Oesophagostomum} spp., from 40% in
Denmark (Roepstorff and Jorsal, 1989) to 89% in Scotland (Jacobs and Dunn, 1969). In addition, *Hyostrongylus rubidus* prevalences ranged from 28.5% of sows in England (Pattison et al., 1980) to in 50.7% in Scotland (Jacobs and Dunn, 1969). *Strongyloides ransomi* was not found in sows in any of these surveys and *Trichuris suis* found only sporadically. In Australia, *A. suum* was found in sows on 22.7% of farms surveyed, *Oesophagostomum* spp. on 60.0%, *Trichuris suis* on 12.1% and *Hyostrongylus rubidus* on 20.0% (Mercy et al., 1989a). Surveys conducted in England (Pattison et al., 1980) and Denmark (Roepstorff, 1991) demonstrated that sows in more traditionally managed indoor herds (late weaning age and reduced levels of barn hygiene) provided a steady supply of parasite eggs, while sows in intensively managed indoor herds (early weaning and better hygiene) were not an important source of parasite infection for the young pigs. Boars were treated regularly on three-quarters of the farms surveyed in Saskatchewan. There are no other published data on treatment of boars.

From on-farm surveys of parasite occurrence in Canada (Bernardo et al., 1990a), Georgia (Marti and Hale, 1986) and Denmark (Roepstorff, 1991) it seems possible that anthelmintic treatments may not always be administered at points in the pig production cycle to ensure maximum effects on the parasites. For anthelmintics to be used efficiently, treatment programs should be based on the epidemiology of the targeted parasites. Except for one study of market weight hogs (Polley and Mostert, 1980) there are no such data for Saskatchewan and little for North America. In-barn studies of the prevalence and intensity of pig endoparasites for the various
age categories of pigs are needed before efficient anthelmintic treatment programs can be developed. There is also a need for continuing studies on treatment and local management effects and how these factors affect endoparasitic populations in Saskatchewan pigs.
5.0 *ASCARIS SUUM*: SEASONAL EGG DEVELOPMENT RATES IN A
SASKATCHEWAN PIG BARN.

5.1 Introduction

*Ascaris suum* is common in Saskatchewan pigs and in pigs raised in intensive
management systems throughout the world (Wagner and Polley, 1997). This parasite
is associated with liver damage ("milk-spot" lesions caused by migrating larvae)
resulting in organ condemnations at slaughter. Also, infection with this helminth has
been found to adversely affect various production parameters such as rate of weight
gain and feed efficiency (Humik and Dohoo, 1995). The main control measure for
*A. suum* is the regular use of anthelmintics (Roepstorff and Nansen, 1994). These
treatments remove the parasites present in the infected animals but these constitute
only a proportion of the total ascarid population in an infected hog barn. Many of the
parasites are present as eggs on the barn floor and other fixtures. Knowledge of the
biology of these free-living stages may assist in developing appropriate control
programs.

*Ascaris suum* eggs are not immediately infective when passed in pig feces but
must undergo a period of development in the external environment. This
development, to the infective second larval stage (L2), is affected by a number of
factors including temperature and humidity (Seamster, 1950). Egg development has been studied under laboratory conditions (Seamster, 1950) and in pig barns in Great Britain (Connan, 1977; Stevenson, 1979) and Sweden (Nilsson, 1982). In these barn studies, there were marked seasonal differences in egg development rates. There are no published data describing *A. suum* egg development under North American production and climatic conditions. This study describes seasonal development patterns of ascarid eggs under barn temperature conditions in Saskatchewan, Canada and on the subsequent longevity of the eggs once found infective.

5.2 Materials and Methods

Once each month, from July 1997 to July 1998, fertilized eggs were collected from mature female *A. suum*. At a local abattoir at least 60 adult worms were removed from a minimum of five recently killed market hogs and transferred to the laboratory in an “egg-laying solution” (phosphate buffered saline pH 7.3, with 0.0015N sodium hydroxide and 11mM glucose) then washed several times in sterile distilled water. The female worms were maintained alive for five days at 37°C in 1L pharmaceutical graduates (Nalge), each containing 30 worms and sufficient egg-laying solution with added gentamycin sulphate (125 mg/L) to fill the container to a volume of 1L. Eggs released settled to the bottom of the vessel and were collected at 12-hour intervals using a pipette. After each egg collection, enough egg-laying solution was added to again fill the container to 1L. The outer protein coat of the
egg was removed in 1% sodium hypochlorite to ease handling, washed several times in distilled water, and suspended in 0.1N aqueous sulfuric acid (Jeska et al., 1986). The resultant egg suspension contained approximately 10,000 egg/ml as higher concentrations of eggs appear to have an inhibitory effect on their development (Eriksen, 1990). Fifty ml of this egg suspension were added to a plastic 750-ml tissue culture flask (Falcon) and stored at 4°C until required, to a maximum of seven days. When placed on its side the depth of fluid in the container was 3 mm and the surface area was 175cm²; this permitted adequate aeration of the egg suspension.

The study was conducted in a grow/finish barn on pig farm approximately 50-km northeast of Saskatoon, Saskatchewan, Canada. The barn was insulated but had no heat source besides body heat from the pigs. Temperature was controlled through negative pressure ventilation. Once a month, beginning in July 1997 and continuing until July of 1998, two of the Falcon culture flasks containing recently collected *A. suum* eggs were placed in a plastic box (45 cm x 30 cm x 45 cm) on a shelf 1.5 m above the barn floor and out of reach of the pigs. The shelf was located on an interior, dividing wall in the center of the barn. Once per week, a 0.5 ml sample from each flask in the barn was collected and transported to the laboratory. The eggs in these samples were examined microscopically for development. As well, all culture flasks were opened and agitated for 2 minutes each week to assure gaseous exchange. Once motile larvae were present in a particular month’s flask, the viability of the eggs was determined using a mouse bioassay. Four mice were infected, each with 0.25 ml of the egg suspension from the culture flasks using a
gastric gavage technique and euthanized two days post-infection. Migrating stages from the liver were isolated using the “spin method” of Johnstone et al. (1978). Once infective eggs were present in a particular flask the ova were examined monthly by passage in mice to monitor the duration of viability. This was done until the study ended in early August, 1998. To determine the viability of the eggs released from the female parasites, control culture flasks from each month’s abattoir collection were incubated at 28°C until infective as assessed by the mouse bioassay. The mouse bioassay experimentation was approved by the University of Saskatchewan Animal Care Committee and complied with Canadian Council on Animal Care (CCAC) principles and regulations.

In-barn temperature was recorded hourly beside the flasks using an electronic monitor (HOBO Temp, Onset Computer Corporation). Floor temperatures were also recorded hourly from mid-April to July 1998 again using a HOBO Temp monitor. Both temperature monitors used in the barn were consistent with one another as well as with a mercury maximum – minimum thermometer when placed side by side for two days in the laboratory. External environmental temperatures were recorded at an Environment Canada weather station located 35 km southwest of the study farm.

5.3 Results

Eggs in all months’ culture flasks reached the infective L2 stage. Although development times did vary depending on season, there did not appear to be a period
of the year where egg development ceased completely. Eggs within individual culture flasks, as observed by weekly collections, progressed at very similar rates with approximately 50% of eggs containing active larvae when first inoculated into mice. In all months' experiments, two weeks later almost 100% of the eggs examined from each flask were larvated. A summary of the development periods is presented Table 5.3-1.

Egg development in the summer (July, August and September, 1997 and June and July, 1998) progressed at a consistent rate in both 1997 and 1998, taking between 21 and 28 days to reach the infective L2 stage (eggs may have reached maturity at any point between the weekly collection periods). Fall and winter egg development rates decreased as mean barn temperature fell. The longest development time was between 77 and 84 days for those eggs placed in the barn in January. The in-barn temperature and ova development rate decreases corresponded to the decrease of the external environmental temperatures for 1997 (Figure 5.3-1). In the spring of 1998 the egg development rates increased with mean environmental temperature (Figure 5.3-1). The greatest acceleration in development rates occurred from mid April to May with eggs from March and April experiments reaching maturity at about the same time (Table 5.3-1). The development rate appeared to be slower for those eggs placed in the barn in November. The reason for the reduced rate is unclear. Least squares linear regression analysis showed a strong correlation between temperature and development period throughout the year (r² = 0.8954; p < 0.0001). Mean floor temperatures were within 1°C of the mean temperatures.
measured adjacent to the culture flasks. Eggs in all months’ experiments, once infective, remained viable through to the end of the study.

Seamster (1950), working on development rates of *A. suum* eggs in relation to temperature, calculated from laboratory cultures that eggs of this helminth require 3070 development units (DU) to contain motile larvae. The DU total is defined as the number of hours required to reach this stage multiplied by the difference between the culture temperature and a threshold of 14.5°C (Seamster, 1950). In the current study, motile larvae were consistently observed in eggs at the point in time when monthly egg cultures had been exposed to 3070 DU based on daily mean temperatures (Table 5.3-2). Although there were motile larvae present in eggs at this DU total, this stage of development was not infective to mice. Total DU required to reach the infective stage were calculated for each months’ experiment and generally required exposure to approximately twice the DU as the 3070 reported by Seamster (1950) (Table 5.3-2).
Table 5.3-1. *Ascaris suum* seasonal egg development rates in a Saskatchewan pig barn.

<table>
<thead>
<tr>
<th>Date egg suspension placed in barn</th>
<th>Date eggs first found infective¹</th>
<th>Development period (days)²</th>
<th>Mean in-barn temperature during development (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>July 10, 1997</td>
<td>August 6, 1997</td>
<td>21-28</td>
<td>25.3</td>
</tr>
<tr>
<td>August 6, 1997</td>
<td>September 3, 1997</td>
<td>21-28</td>
<td>24.2</td>
</tr>
<tr>
<td>September 3, 1997</td>
<td>October 1, 1997</td>
<td>21-28</td>
<td>23.6</td>
</tr>
<tr>
<td>October 1, 1997</td>
<td>November 19, 1997</td>
<td>42-49</td>
<td>20.0</td>
</tr>
<tr>
<td>November 5, 1997</td>
<td>January 21, 1998</td>
<td>70-77</td>
<td>17.4</td>
</tr>
<tr>
<td>December 10, 1997</td>
<td>February 11, 1998</td>
<td>56-63</td>
<td>17.2</td>
</tr>
<tr>
<td>January 7, 1998</td>
<td>March 4, 1998</td>
<td>77-84</td>
<td>16.8</td>
</tr>
<tr>
<td>February 11, 1998</td>
<td>April 1, 1998</td>
<td>63-70</td>
<td>17.8</td>
</tr>
<tr>
<td>March 4, 1998</td>
<td>April 29, 1998</td>
<td>49-56</td>
<td>18.9</td>
</tr>
<tr>
<td>May 6, 1998</td>
<td>June 10, 1998</td>
<td>28-35</td>
<td>22.1</td>
</tr>
<tr>
<td>June 3, 1998</td>
<td>July 1, 1998</td>
<td>21-28</td>
<td>23.8</td>
</tr>
<tr>
<td>July 1, 1998</td>
<td>July 29, 1998</td>
<td>21-28</td>
<td>25.4</td>
</tr>
</tbody>
</table>

¹ Larvae were recovered from at least one of the four mice inoculated.
² The first value indicates the last without infective eggs.

The second value indicates the first day with infective eggs.
Table 5.3-2. *Ascaris suum* seasonal egg development rates in a Saskatchewan pig barn, a basic model.

<table>
<thead>
<tr>
<th>Date egg suspension placed in barn</th>
<th>Date eggs exposed to 3070 DU(^1)</th>
<th>Date motile larvae first observed</th>
<th>Total DU exposure when infective larvae first observed(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>August 6, 1997</td>
<td>August 19, 1997</td>
<td>August 19, 1997</td>
<td>5208-6634</td>
</tr>
<tr>
<td>September 3, 1997</td>
<td>September 16, 1997</td>
<td>September 18, 1997</td>
<td>4651-6204</td>
</tr>
<tr>
<td>October 1, 1997</td>
<td>October 19, 1997</td>
<td>October 22, 1997</td>
<td>6213-6919</td>
</tr>
<tr>
<td>November 5, 1997</td>
<td>December 4, 1997</td>
<td>December 17, 1997</td>
<td>6946-7684</td>
</tr>
<tr>
<td>March 4, 1998</td>
<td>April 9, 1998</td>
<td>April 8, 1998</td>
<td>5068-6439</td>
</tr>
<tr>
<td>April 1, 1998</td>
<td>April 21, 1998</td>
<td>April 22, 1998</td>
<td>4651-5880</td>
</tr>
</tbody>
</table>

\(^1\) DU = development units (see text)

\(^2\) The first value indicates the last without infective eggs.

The second value indicates the first day with infective eggs.
Figure 5.3-1 Ascaris suum: Seasonal egg development rates in a Saskatchewan pig barn, environmental temperatures during the study period and 30 year mean temperatures (1966-1996)
The seasonal differences in development rates of *A. suum* eggs in Saskatchewan pig barns are consistent with other published studies. The time to the infective stage varied from between 3-4 weeks in the spring and summer to as long as 11-12 weeks in mid-winter. This variation in seasonal development times is not as marked as in reports from the three studies from northern Europe, where no development of eggs was observed through any of the winter months (Connan, 1977; Stevenson, 1979; Nilsson, 1982). In-barn temperatures in these studies were much cooler than in Saskatchewan and, from approximately October to May, were below the development threshold for *A. suum* eggs. In the current study, the mean in-barn temperature was below the development threshold only very rarely. In late spring and into summer in Europe, as temperatures rose, the eggs which had accumulated through the winter began to develop and reached the infective stage at approximately the same time. This seasonal peak was associated with a rise in liver condemnations due to milk-spots in late summer and fall in England (Connan, 1977; Stevenson, 1979). This pattern was also observed between 1969 and 1991 in Northern Ireland (Menzies et al., 1994). An autumn increase in liver condemnation rates has also been reported in Atlantic Canada (Hurnik and Dohoo, 1995).

During the summer in Saskatchewan in-barn temperatures were very similar to ambient external temperature, well above 14.5°C. During winter the in-barn temperatures were within the range recommended for Saskatchewan pig production.
and slightly above the *A. suum* development threshold. The pattern of *A. suum* egg development in Saskatchewan hog barns suggests limited seasonal variation in liver damage relative to Europe. Control measures for this part of the world may differ when compared to the European situation. The barn cleaning procedures recommended for the British and Swedish conditions, including a thorough cleaning in the spring to remove accumulated fecal matter and *A. suum* eggs, might not be as effective in production conditions in western Canada. Because of the slow but continuous winter egg development here, a more regular cleaning protocol may be required to assist in control.

This study was designed to determine the effect of temperature variation alone on *A. suum* egg development rates. As in Connan’s (1977) barn experiments, decorticated eggs maintained in a mildly acidic medium were used. There are no published data on the effects of decortication or of an acidic medium on *A. suum* egg development or mortality rates. Other conditions influencing development rates, such as humidity, were optimized. Similar experimental systems were used in the three European studies (Connan, 1977; Stevenson, 1979; Nilsson, 1982). In addition, Nilsson (1982) conducted experiments exposing feces containing ascarid eggs to the humidity found on the floor of a pig barn adjacent to the pig pens. Embryonated eggs were not recovered from any of these cultures, where humidity varied from 63.5% - 78.5%. Embryonated eggs were consistently recovered, however, from the cultures which were watered daily (Nilsson, 1982). Seamster (1950) found that *A. suum* eggs spread on glass slides required 100% humidity to
complete development. Areas of excess water accumulation in barns may enhance the success of developing eggs (Nilsson, 1982). Also, in the three previous reports of *A. suum* egg development, egg cultures were maintained on the barn floor in an alleyway with temperatures recorded beside the cultures (Connan, 1977), in an empty loose-box housing pen with temperatures recorded at an outdoor site about 1 mile from the study facility (Stevenson, 1979), or on the concrete floor adjacent to a pig pen with temperatures recorded beside the cultures (Nilsson, 1982). In our study egg suspensions were maintained above the floor. Defining all the actual conditions to which *A. suum* eggs are exposed in a pig barn is very difficult. Eggs in fecal material in pens may be exposed to fluctuations in all the environmental conditions that affect egg development. The effect of the pigs on the microclimate surrounding the eggs may be substantial. None of the studies replicate conditions in a populated pen where the animals themselves are a source of both heat and moisture and may strongly influence egg development. It should be noted that in the current study the eggs used were released by adult female parasites in culture rather than being dissected from the adults and mouse inoculation started as soon as larvated eggs were observed in the culture flasks in the barn.

Because of the great diversity of climate and management systems in pig producing areas of the world, data from studies such as these are primarily applicable only to local conditions (Nilsson, 1982). Based on the high prevalence of *A. suum* in Saskatchewan pigs (Wagner and Polley, 1997) conditions conducive for successful egg development must exist in western Canadian barns. Egg development
model systems such as that described in this paper are useful, therefore, in understanding the biology of the free-living stages of this parasite. Results from these experiments may assist in developing more efficient control measures for *A. suum*, combining both properly timed anthelmintic treatments and properly timed barn cleaning procedures.
6.0 *ASCARIS SUUM*: LONGITUDINAL MONITORING OF INFECTION IN GROWING PIGS

6.1 Introduction

Liver lesions due to *A. suum* migration are a common problem in market hogs in Saskatchewan (Polley and Mostert, 1980; Wagner and Polley, 1997b), in other parts of Canada (Bernardo et al., 1990a) and in pig producing areas in many areas of the world (Menzies et al., 1994). Condemnation of these organs is considered a significant cause of economic loss (Hurnik and Dohoo, 1995). It is well established that in modern production facilities with intensive management, including early weaning of piglets, pigs do not become infected with *A. suum* until the growing/finishing period (Moncol, 1993; Roepstorff and Nansen, 1994; Hurnik and Dohoo, 1995). This conclusion is based primarily on cross-sectional surveys of the various age groups found within pig barns in several parts of the world (Martin et al., 1974; Mercy et al., 1989a; Morris et al., 1984; Roepstorff and Jorsal, 1989; Roepstorff et al., 1998). There are few detailed longitudinal studies published. Marti and Hale (1986) in Georgia, U.S.A., Bernardo et al. (1990a) in Prince Edward Island, Canada and Roepstorff (1991) in Denmark have studied pigs throughout the production cycle.
In Saskatchewan, nothing is known about the changes that occur in the prevalence and intensity profiles in pigs infected with *A. suum* between birth and market weight: when do the animals first become infected? what proportion of a population of pigs becomes infected with *A. suum* at various stages of the production cycle? how heavily infected do they become? and do prevalence and intensity levels decline as pigs approach market weight? This chapter describes a longitudinal study carried out in Saskatchewan to determine the transmission dynamics of *A. suum* in growing pigs within a single herd in Saskatchewan.

6.2 Materials and Methods

This study was conducted on a pig farm approximately 50 kilometers north east of Saskatoon. Management of this 60-sow farrow-to-finish operation included weaning of piglets at approximately four weeks of age and transfer of the weaned animals to a nursery in which each pen has an expanded metal (perforated) floor allowing minimal contact between pigs and feces. Piglets stayed in the nursery for approximately four weeks. Groups of approximately 14 weanlings assembled in the nursery remained as penmates through the remainder of their stay on the farm although there was sometimes limited mixing of the groups in the grow/finish barn. From the nursery, the pigs were moved to a separate barn housing all the pigs from early grower to market weight. Early growers were kept in pens occupying about one third of the barn and this area was separated by a floor to ceiling wall from the pens.
for the finishing animals. The floors of the pens in the grow/finish barn were two-thirds solid concrete (next to the central alleyway) and one-third slatted concrete (next to the outside wall) draining into manure pits below the barn. Excess fecal material was scraped daily from the solid concrete to the slatted portion of the pen. No other cleaning was carried out in this portion of the barn. No anthelmintics had been used in the grow/finish barn for at least 10 years although sows were treated occasionally with dichlorvos. This barn was chosen because of known ascarid infection based on the presence of liver lesions at slaughter. Also, the farmer was extremely cooperative, allowing unlimited access to the barn.

Two groups of piglets, the first farrowed January 9, 1998 (Group A) (day 0) and the second (Group B) on March 3, 1998 (day 0) were monitored from 40 and 36 days of age respectively, when they were in the nursery, until market weight. Fecal samples were collected every two weeks until day 145 (Group A) and day 155 (Group B) and then weekly until the animals were sold. All samples were collected rectally unless the animal was observed defecating, in which case the feces was collected from the pen floor taking care to obtain only fresh material. Samples were processed using a quantitative Wisconsin fecal flotation (Cox and Todd, 1962).

These two groups of weanlings were moved from the nursery to the grow/finish barn at day 64 (Group A) and day 68 (Group B) and then from the grower to the finisher area on days 101 and 124 respectively. The pigs were penned as individual groups (A and B) throughout the study with no other animals added to either group.
At slaughter, livers of the study animals were examined for “milk-spots” and scored according to the following scale; 0 = no lesions, 1-4 = number of major liver lobes affected, and 5 = all four major lobes affected with some liver scarring. Also, the small intestine of each study animal was removed from the mesentery, the contents squeezed into a collection tray and then the entire intestine opened longitudinally to recover and count the immature and mature adult ascarids (Wagner and Polley, 1997b).

6.3 Results

At the first sampling, nursery pigs in both groups were positive for *Isospora* sp. (2/14, Group A; 5/14 Group B). No eggs or oocysts were found in the fecal samples of either group until day 166 (Group A) or day 155 (Group B) when *A. suum* eggs were detected. Subsequently, *A. suum* eggs were found in the feces of from one to three of the Group A pigs at each of the 5 weekly samplings prior to the pigs going to market. Egg numbers were extremely low with counts ranging from less than one to three eggs per gram (epg) of feces. No other positive fecal samples were observed in Group B pigs and the positive sample from day 155 had less than one egg per gram of feces. None of the *A. suum* eggs observed were larvated and no other helminth eggs were observed during the study. One animal in Group B died on approximately day 106.
Pigs in both groups were marketed at approximately 200 days of age.

Twenty-three of 25 livers from the two groups examined at slaughter had "milk-spot" lesions. The liver scores are displayed in Figure 6.3-1. Only one adult *A. suum* was found in the intestinal contents of a pig in Group B. The liver from this animal had been given a score of five. None of the fecal samples collected at the abattoir were positive for ascarid eggs. Two animals were not observed at the abattoir.
Figure 6.3-1. Liver score distribution of 23 pigs from groups A and B.
6.4 Discussion

It is not surprising that patent helminth infections were not found in the weanling pigs at the beginning of this study. This finding is consistent with previous reports in which young pigs under early weaning management systems were negative for *A. suum* (Marti and Hale, 1986; Roepstorff, 1991; Moncol, 1993). Under optimal conditions, eggs require approximately 30-35 days in the farrowing crates to become infective (Moncol, 1993). Early weaning at 3-5 weeks removes piglets from the environment before eggs from the feces of the sows become infective and, as a result, pigs moving to the nursery area are usually ascarid free (Moncol, 1993; Roepstorff and Nansen, 1994; Hurnik and Dohoo, 1995). Thus, even if sows are passing eggs in the feces they are not believed to be a major source of infection for the piglets in this type of system (Roepstorff, 1991). In addition, Roepstorff and Nansen (1994) as well as Hurnik and Dohoo (1995) reported that in intensively managed facilities with early weaning, transmission of *A. suum* does not begin until pigs are in the grow/finish area of the barn. Animals are kept here long enough for the transmission cycle (including egg development and the long prepatent period) to be completed (Hurnik and Dohoo, 1995). These authors also indicated that pigs may not be infected until late in the finish phase (70-150 days of age) and market weight may be reached before the pigs become patent. Roepstorff (1991) also found that on farms in Denmark with late weaning (>6-14 weeks) there was a high level of *A. suum* transmission in the farrowing crates.
In the present study, eggs were first detected on either day 166 (Group A) or 155 (Group B) indicating that the infection producing the eggs had probably been acquired in the grow/finish barn approximately 7-8 weeks earlier (Schwartz, 1959). It is possible, however, that not all the *A. suum* positive fecal samples collected in this study reflected patent infections in the pigs. Boes et al. (1997) stated that low fecal egg counts must be interpreted carefully. These authors observed that false-positive egg counts in pigs housed indoors do occur because these animals are coprophagic. It is, therefore, difficult to determine the validity of the low egg counts detected in the present study especially when only one worm was found at post-mortem. Average egg counts in similar studies have been as high as 800 epg (sodium chloride McMaster technique) in Prince Edward Island with infected pigs containing, on average, 12 worms (Bernardo et al., 1990a) and 984 epg (zinc sulphate McMaster technique) in Georgia (Marti and Hale, 1986). Bernardo et al. (1990a), following naturally infected pigs in a similar manner to the current study also reported positive egg counts from pigs that had no intestinal worms at post-mortem and believed that these eggs resulted from coprophagia. Roepstorff and Nansen (1994) indicate that egg counts below 200 epg are should be considered false positive.

It is also not surprising that of the 25 pigs observed at post-mortem, 23 had some degree of liver damage but only one adult worm was recovered. Other published surveys have reported liver lesions at post-mortem without observing macroscopic *A. suum* worms in the gut (Polley and Mostert, 1980; Bernardo et al.,
Bernardo et al. (1990a) found that 51.3% of 386 pigs from 15 Prince Edward Island pig farms had liver lesions but no worms in the intestine. This was especially apparent on three of the farms where lesions were present in 94.5% of 18, 89.3% of 28 and 70.5% of 18 market animals but only two of these pigs contained one or two adult worms. The authors proposed two possible explanations: either the parasites resulting from a recent infection were successful migrating in the pigs but the animals were marketed before stages of *A. suum* visible to the naked eye were present in the intestine and were therefore not recovered (the presence of ‘milk-spots’ generally indicates infection within the previous 3-6 weeks (Hurnik and Dohoo, 1995) although lesions may remain for periods of 60 to greater than 90 days (Roneus 1966)) or, secondly, the larvae of *A. suum* were cleared from the host some time after the hepatic migration due to host immune responses.

Based on the number of damaged livers observed, it appears that the study barn was contaminated with *A. suum* eggs. There may be infective *A. suum* eggs present in this barn that have accumulated over many years without treatment and limited sanitation. Pigs may be ingesting these eggs shortly after entering the barns and throughout their time there. Due to immune response or late infection, most animals do not develop patent infections. It is interesting that Jorgensen et al. (1975) observed that migration of larvae occurred in the pigs receiving a dose of 10,000 eggs but the immature worms were expelled from the animal on their return to the intestine and none of these experimental animals developed a patent infection. Conversely, it may be that the levels of environmental contamination with *A. suum*
eggs in the barn used in the current study were insufficient to produce patent infections in the pigs.

Control recommendations in this barn would include anthelmintic treatment although this measure alone may not be extremely effective. In barns contaminated with large numbers of ascarid eggs animals are continuously reinfected (Roepstorff and Nansen, 1994). A high level of sanitation does, however, reduce *A. suum* prevalence in pigs in indoor housing systems by removing infective eggs from the environment (Primm et al, 1990; Moncol, 1993; Menzies et al., 1994; Roepstorff and Nansen, 1994). Incorporation of this measure into management systems may adequately control parasite numbers making anthelmintic treatment unnecessary (Roepstorff and Nansen, 1994; Roepstorff et al., 1997). On the farm used in this study, however, a combination of sanitation and medication would likely be most appropriate.

Continued longitudinal studies of in-barn prevalence and intensity need to be carried out. The formation of a more complete picture of the epidemiology of *A. suum* in confined pigs in Saskatchewan will assist in the development of maximally effective and efficient control programs.
7.0 GENERAL DISCUSSION AND CONCLUSIONS

The worldwide pig production industry is both diverse and dynamic. Changes in production systems, most notably the movement of pigs indoors, as well as the increasing sophistication and scale of intensive management systems, have affected the patterns of infectious diseases of pigs, including *A. suum*. This parasite remains common in pigs in many parts of the world (Wagner and Polley, 1997b; Roepstorff et al., 1998) despite considerable published information on its biology and the wide availability and use of very effective anthelmintics. Part of this success of *A. suum* may be a result of the very high fecundity of the adult parasites (Olsen et al., 1958) and the ability of the eggs to survive very well in the environment (Brown, 1928b). Another factor may be the “implementation gap” between what is known about the parasite and what is done on the farm, particularly in terms of parasite control.

Moncol (1993) stated that many current parasite control practices are based not on recent research findings but on information from at least 25 years ago for pigs raised on dirt lots.

Probably the two key factors affecting the occurrence of *A. suum* in domestic pigs are management, which varies from farm to farm and does change as knowledge related to pig production improves, and climate, which varies from region to region. Consequently, for *A. suum*, epidemiological patterns and optimal control measures
are to a certain extent locally defined. Other than an abattoir survey carried out in Saskatchewan 20 years ago, there is no published information on the epidemiology and control of this parasite in pigs in the Canadian prairies and very little for other areas of Canada (Martin et al., 1974; Bernardo et al., 1990a). The four inter-related projects described in this thesis were designed to increase knowledge and understanding of the epidemiology and control of *A. suum* in pigs in this part of the world.

In the abattoir survey (Wagner and Polley, 1997b) it was observed that approximately 50% of market pigs in this province have evidence of *A. suum* infection and that 81% of the farms included marketed pigs infected with *A. suum* during the survey period. The survey also demonstrated that the prevalence and intensity of intestinal adult *A. suum* in Saskatchewan market pigs has declined somewhat during the past 20 years while, over the same period, the prevalence and intensity of liver lesions associated with ascarid larval migrations have remained unchanged. The reasons for these patterns are unclear but it is interesting that similar trends have also been observed in Denmark (Roepstorff and Jorsal, 1989).

The survey of anthelmintic use in Saskatchewan (Wagner and Polley, 1997a) showed that these drugs are used by 75% of the 190 producers who responded to the questionnaire. This widespread use occurs despite apparently limited attempts to assess the prevalence and intensity of ascarids in the pigs and our very limited ability to relate these parameters to production efficiency. Further, it appears that anthelmintics are used in Saskatchewan, and elsewhere in the world, in the absence
of the clear understanding of local epidemiological patterns that is needed to optimize their application in parasite control programs.

In the in-barn egg development project (Wagner and Polley, 1999) there were seasonal differences in the rates of this development in Saskatchewan that were comparable to those reported from northern Europe (Connan, 1977; Stevenson, 1979; Nilsson, 1982)

Further, the longitudinal study of *A. suum* prevalence and intensity in two small cohorts of pigs followed from the nursery to market demonstrated that a group of pigs can develop a high prevalence of liver lesions in the absence of evidence of widespread infection with intestinal adult ascarids.

There are few published reports on the epidemiology of *A. suum* in pigs in Canada and, not including the work from this thesis, only one from western Canada (Polley and Mostert, 1980). There remain, therefore, many opportunities for future work.

For example, extending an abattoir survey for a full year may provide an indication of seasonal changes in *A. suum* prevalence and intensity. Examination of the intestinal contents for parasites not visible to the naked eye, perhaps using a recently published extraction technique (Roepstorff et al., 1997) together with a detailed examination of the intestinal parasites recovered to include data on size, sex and stage of sexual maturity, may provide useful information. The collection and examination for eggs of fecal samples from the animals included in the survey, something difficult to do in an abattoir setting, may also be helpful.
Regarding the postal survey of anthelmintic use, to avoid non-response bias (Roush, 1998), follow-up by letter or telephone may increase the response rate among those selected for the survey. There are, however, concerns that this approach might have reduced the accuracy of the information collected.

Only three in-barn *A. suum* egg development studies have been reported in the literature, two in England (Connan, 1977; Stevenson, 1979) and one in Sweden (Nilsson, 1982). A problem with investigations of this type, including the one described in this thesis, is that the culture conditions used for the eggs do not accurately mimic the environment to which they would be exposed on the floor of a pen full of pigs. Given the required duration of such experiments and the behavior of pigs, to design such a study is a major challenge.

If longitudinal monitoring of growing pigs similar to that described in this thesis is to be attempted in the future, the use of parasite-naïve “tracer pigs” in various stages of the production cycle with *post-mortem* examination of these may assist in assessing infection pressures in a group of pigs or in a particular environment. Unfortunately, this may be very difficult to accomplish in modern production facilities because of the high levels of biosecurity practiced on many pig farms. Alternatively, it may be beneficial to sacrifice some of the experimental animals at predetermined times during the experiment but this too would be difficult in small groups of monitored animals. Also, it might be possible in the future to assess *A. suum* exposure serologically throughout the study period.
As indicated earlier, there are many aspects of the biology, epidemiology and production significance of *A. suum* yet to be understood. Future research possibilities are numerous. Recent studies have noted that the distributions of adult ascarid populations are overdispersed in groups of experimentally infected pigs and in small groups of animals on pasture (Boes et al., 1998; Roepstorff et al., 1997). Determining the distribution of adult parasite populations in infected pig barns would be very helpful. This can be accomplished to some extent on the basis of fecal examination for eggs but recently protocols used widely to investigate gastrointestinal helminths in people have been applied to *A. suum* in pigs. These include treating experimental animals with an anthelmintic, collecting adult worms from the feces ("treat and recover") and then allowing reinfection in the previously occupied contaminated environment ("treat and reinfect") (Boes et al., 1998).

Even as the understanding of the epidemiology of *A. suum* in Canadian prairie production systems improves there is still a need for recommendations concerning optimal parasite control. Traditional control programs based on general management features of intensive or extensive pig production systems appear not to be effective in Saskatchewan barns. Future control should be based on the parasite status of individual barns as assessed by routine fecal and liver lesion monitoring (Moncol, 1993; Roepstorff and Nansen, 1994; Hurnik and Dohoo, 1995). Effective control may be accomplished through management factors alone, such as regular barn sanitation, but anthelmintic treatments must be incorporated when fecal monitoring indicates need (Moncol, 1993; Roepstorff, 1997).
Even when these goals are met we still need to better define the economic effects of subclinical parasitism within commercial pig barns. This has been done in experimental infections but very little work has been conducted in active pig operations. It may be, however, that given the tenacity of the parasite, reducing the prevalence and intensity of the infection in modern production conditions may be very difficult and not cost-effective.

Regarding public health, although it appears that, in Saskatchewan risk, of human infection with *A. suum* is minimal, the zoonotic aspect of this parasite should not be minimized. More detailed risk assessments may be conducted to determine egg density and survival on cropland fertilized with pig manure. An attempt to define risk differences between those involved in the pig industry versus the rest of the population may also be beneficial. A seroprevalence survey of exposure within these populations may also produce interesting and useful information.

Finally, it is unfortunately true that the development of the most up-to-date control recommendations, focusing on parasite status in individual herds and based on the most recent information available, may not be of any benefit to the industry if not properly communicated to practitioners and producers.
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development of ova of pig and human *Ascaris* under natural conditions, and


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SURVEY OF ANTHelmintIC USAGE ON SASKATCHEWAN HOG FARMS

We are attempting to gather information on the use of deworming (anthelmintic) medications on Saskatchewan hog farms. Filling out the following survey form will be of great help in the completion of this project. Your response will be used only for this research project and the source of all information will remain confidential.

1. Name and Address (optional)

2. Farm Type
   - ☐ farrow to finish
   - ☐ farrow to weanling
   - ☐ weanling to finish
   - ☐ other (please specify)

3. Farm Size (number of market hogs or weanlings sold per year).
   - ☐ 1-200
   - ☐ 201-1000
   - ☐ 1001-2000
   - ☐ 2001-3000
   - ☐ >4000

4. I have a High Health Herd.  ☐ Yes  ☐ No

5. a) I have noticed a problem with internal parasites of pigs on my farm.  ☐ Yes  ☐ No
   b) This was determined by:
      - ☐ parasite egg count
      - ☐ poor performance
      - ☐ adult parasites seen in feces
      - ☐ other (please explain)

6. I have treated my pigs with a dewormer in the past year (1995).  ☐ Yes  ☐ No
   If YES, please continue with the questionnaire. If NO, please stop and return this form in the self-addressed, stamped envelope.

7. I find deworming medications to be effective and beneficial when used on my farm.  ☐ Yes  ☐ No

8. I treat with dewormers following a planned program.  ☐ Yes  ☐ No

9. When treating weanling pigs in 1995 I treated:
   - ☐ all weanling pigs
   - ☐ about 3/4 of all weanling pigs
   - ☐ about 1/2 of all weanling pigs
   - ☐ about 1/4 of all weanling pigs
   - ☐ no weanling pigs
   - ☐ other (please explain)

10. In the past year (1995) I have used these dewormers to treat weanling pigs: (check all that apply)
    - ☐ injectable
    - ☐ in feed
    - ☐ in water

11. I treat pigs in the grower barn with a dewormer:
    - ☐ more than 3 times per year
    - ☐ 2-3 times per year
    - ☐ once per year
    - ☐ never
    - ☐ other (please explain)

Please continue on the back of this page.
12. In the past year (1995) I have used these dewormers to treat grower pigs: (check all that apply)

<table>
<thead>
<tr>
<th>Injectable</th>
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</tr>
</thead>
<tbody>
<tr>
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<td>Atgard</td>
<td>Piperazine</td>
</tr>
<tr>
<td>Levamisole</td>
<td>Banminth II</td>
<td>Mecadox</td>
</tr>
<tr>
<td>other</td>
<td>Exhelm E</td>
<td>Pro-banminth</td>
</tr>
<tr>
<td></td>
<td>Tramisol</td>
<td>Safe-Guard</td>
</tr>
<tr>
<td></td>
<td>Co-op wormer</td>
<td>other</td>
</tr>
</tbody>
</table>

13. I treat sows/gilts with a dewormer:
- □ regularly, prefarrowing
- □ regularly, at breeding
- □ occasionally, prefarrowing
- □ occasionally, at breeding
- □ rarely
- □ never
- □ other (please explain)

14. In the past year (1995) I have used these dewormers to treat sows/gilts: (check all that apply)

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</tbody>
</table>

15. I treat boars with a dewormer:
- □ greater than 3 times per year
- □ 2-3 times per year
- □ once per year
- □ never
- □ other (please explain)

16. In the past year (1995) I have used these dewormers to treat boars: (check all that apply)

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17. I have used the dewormers marked off for these reasons: (check all that apply)
- □ effectiveness
- □ advertising
- □ price
- □ ease of use
- □ other (please specify)

18. I use these sources of information to decide on products and treatments used: (check all that apply)
- □ advertising in the farming press
- □ other pig producers
- □ drug company representatives
- □ feed companies
- □ veterinarians
- □ other (specify)

END

THANK-YOU FOR YOUR TIME AND EFFORT