Supplement to the Life History of *Strongyloides ransomi* Schwartz and Alicata, 1930 (Nematoda: Strongyloididae) of Pigs

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**ABSTRACT:** *S. ransomi* possesses three routes of infection: transmammary, percutaneous, and prenatal. The transmammary and percutaneous routes occur commonly in nature with prenatal occurring rarely. The larvae passed in colostrum and milk resemble filariform larvae with the exception of the genital primordium being longer, wider, and possessing more cells. Upon reaching the small intestines, the transmammary-passed larva reaches patency in 36 to 48 hr, consequently, the early egg passage by piglets. Barring further exposure, patency will persist for approximately 20 weeks. Percutaneous infection must occur for gilts and sows to store larvae in adipose tissue. Accumulation predominates in the ventroabdominal (mammary) area. Exposure prior to or as late as the final 4 to 6 weeks of pregnancy will insure transmammary passage of larvae. Without further exposure, shedding of larvae in colostrum and milk persists for at least three lactations. Hormonal changes occurring at parturition possibly influence migration and passage of larvae in colostrum and milk.

The life history of *Strongyloides ransomi* has been described in detail by Schwartz and Alicata (1930) and by Lucker (1934). Observations by Moncol and Batte (1966) revealed a transmammary route of infection.

The occurrence of intrauterine infection of pigs by *S. ransomi* has been proposed by Enigk (1952), Enigk et al. (1974), Stewart et al. (1963, 1969), Stone (1964), and Supperer and Pfeiffer (1967). The latter investigators found only one larva in the lungs of stillborn pigs or piglets killed immediately after birth, and interpreted this to mean that at birth the larvae are still in the vascular system and, therefore, scattered throughout the entire body. Yet eggs were regularly detected in the feces of baby pigs beginning the 4th day after birth. Enigk et al. (1974) observed larvae of *S. ransomi* in the tissues of only two of 47 piglets removed by Caesarean section. The sows had been infected at various stages of pregnancy, though both infected piglets occurred in sows infected in the first half of pregnancy. No patent infection followed.

Frickers (1953) observed *Strongyloides* eggs in the feces of 3- and 4-day-old piglets. Intrauterine infection was suspected yet no larvae were found in stillborn littermates. He ascertained that some other means of infection existed but did not elaborate.

Olsen and Lyons (1965) showed that larvae of *Ucinaria lucasi* were transmitted in the colostrum of the northern fur seal. The parasitic third-stage larvae were passed postpartum in the milk for only a short time. The intestinal phase of infection was shown to persist for approximately 3 months in the seal pups. The tissue phase, consisting of parasitic third-stage larvae, occurred in all age groups of seals particularly in the blubber along the belly region. In females, third-stage larvae were also present in the mammary glands and milk cisterns.

Being unable to demonstrate intrauterine infection with *S. ransomi* and having found no ova or larvae in the feces of near parturient gilts or sows, Moncol and Batte (1966) concluded that some other mechanism of infection had occurred. Larvae were observed in colostrum immediately prior to parturition and subsequently in milk. Piglets nursing these sows, or if given larvae collected from filtered milk, became infected and passed typical *Strongyloides* eggs on day 4 postpartum. Supperer and Pfeiffer (1967) and Stewart et al. (1969) subsequently confirmed the occurrence of transcolostral infection.

Other reported species of nematodes that are transmitted through milk are *Strongyloides westeri* in the horse (Lyons et al., 1969), *Strongyloides papillosus* in the sheep and cow (Lyons et al., 1970), *Toxocara cati* in the cat (Swerczek et al., 1971), and *Neoascaris cinctura* in cattle (Warren, 1969), *Ancylostoma caninum* and *Toxocara canis* in the bitch (Stone
and Girardeau, 1967), and Strongyloides papillosus in the goat (Moncol et al., 1973). Details on the biology and morphology of the transcolostral phase of S. ransomi as a supplement to the existing life history are presented in this paper.

Materials and Methods

Pigs with natural infections of S. ransomi were used as the source of ova. Larvae for experimental infections were obtained by culturing feces from infected pigs. Best results were obtained when feces were mixed with peat moss, covered with four layers of gauze, and incubated at 24 to 27 C. After incubation, only the top two layers of gauze were carefully removed to Baermann funnels for collection of filariform larvae, as this procedure proved to eliminate nearly all of the free-living forms.

Colostral larvae were obtained from naturally and experimentally infected sows. During experimental infection, pigs were always kept in concrete-floored isolation pens that were washed daily.

Following the intravenous injection of 10 IU of oxytocin, larvae were obtained from colostrum and milk that was collected by hand-stripping. The colostrum and milk were diluted with dechlorinated water and then passed through an AP-200 Millipore Filter (Millipore Filter Corporation, Bedford, Mass.). The filter was washed over a petri dish and the contents were examined under a dissecting microscope.

Larvae used for size determinations, whether from milk or tissue, were freshly collected and heat-fixed prior to being measured. Tissues to be examined for larvae were minced in a blender and then baermannized in 30 C heated funnels.

Experimental infections in most studies were accomplished either by subcutaneous injection or by direct application of larvae to the skin under moistened larvae-laden gauze pads. The exceptions to this involved experiments in which larvae were injected directly into adipose tissue.

Results

The life cycle of S. ransomi consists of two basic routes of infection: (1) transmammary (colostrum and milk), in which case the susceptible host (newborn piglet) receives advanced third-stage larvae that go directly to the small intestine and (2) percutaneous invasion by infective third-stage larvae (filariform) which undergo a period of tissue migration.

A. Morphogenesis

1. Morphological development of colostral (milk) larvae: Table 1 compares the measurements of filariform larvae, colostral larvae, and larvae collected from the mammary glands and adipose tissues. Larvae collected from colostrum of sows or gilts immediately prior to and during parturition measured 469 to 561 μ (519.9) long by 16 to 21 μ (19.0) wide in the region at the base of the esophagus. The length of the esophagus was 245 to 301 μ (279.0). Distance from anus to the tip of the tail was 53 to 75 μ (63.0). The genital primordium measured 12.7 to 23 μ by 4.6 to 6.9 μ (16.0 by 6.0). The tridid tail typical of the infective third-stage larva was present.

Except for being slightly larger and the genital primordium being longer, wider, and more conspicuous, colostral larvae are similar to filariform larvae. None of the larvae collected from colostrum, mammary, or adipose tissue were undergoing a molt or showed any evidence of an impending molt.

2. Development of colostral larvae within the small intestine of piglets: Table 2 compares the size of larvae collected from the small intestines of piglets at 28, 50, and 75 hr postnursing. The wide range in measurements reflects the fact that piglets continue to take in larvae with each nursing. The development of the reproductive system was quite advanced at 28 hr with ovaries extending anteriorly and posteriorly from the primordium of the vulva. Specimens were mature at 50 hr and developed ova were present in the uteri of female worms.

B. Natural infectivity

1. Duration of intestinal phase: Four pigs reared in the laboratory with only transmammary infection were shown to void eggs for at least 20 weeks. Egg production diminished to a point 12 to 15 weeks after infection where conventional flotation techniques would no longer give a positive test. After this time, eggs can only be demonstrated by centrifugation. Under normal conditions of rearing where percutaneous reinfection existed fol-
Table 1. Size comparison of *S. ransomi* filariform larvae, colostral larvae, and larvae collected from mammary glands and adipose tissues.

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Filariform larvae</th>
<th>Source of larvae*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(a)</td>
<td>(b)</td>
</tr>
<tr>
<td>Total length</td>
<td>504–635</td>
<td>500</td>
</tr>
<tr>
<td>Esophageal length</td>
<td>240–310</td>
<td>—</td>
</tr>
<tr>
<td>Esophageal width</td>
<td>15–19</td>
<td>—</td>
</tr>
<tr>
<td>Length of tail</td>
<td>60–90</td>
<td>—</td>
</tr>
<tr>
<td>Length and width of genital primordium</td>
<td>10–17</td>
<td>17</td>
</tr>
</tbody>
</table>

(a) Schwartz and Alicata, 1930.
(b) Lucker, 1934.
(c) Alicata, 1935.
* Average of 25 larvae each source.

Following initial transmammary infection, ova production diminished rapidly and was detectable for only 8 to 10 weeks.

2. Extent and duration of tissue phase:
The lactating sow is a vital link in the perpetuation of *S. ransomi*. For the sow to infect the next generation of pigs, it is understandable that she must be adequately exposed to infective larvae sometime prior to parturition and lactation. Ova produced by the young parasitic females during the first 3 to 4 weeks undergo homogenic development exclusively, thereby intensifying the infection in the piglet and replenishing the stores of larvae within the adipose tissues of the female host (Moncol, unpublished data). During this period, the sow develops a very transitory intestinal infection that lasts 3 to 4 weeks. Likewise, exposure to a large number ($10^5$) of filariform larvae immediately and within 1 week after parturition will extend the time of passage of larvae in milk. At this time only small numbers of larvae (<1 per cc of milk) were intermittently observed. A wide variation in the degree of infection exists among litters and even among littermates.

Naturally infected sows were held for three consecutive farrowings, without additional exposure to *S. ransomi*. Larvae passed in the milk at each succeeding parturition declined at the following rate: 5 larvae per cc colostrum, 1st lactation; 1 larva per cc, 2nd lactation; and 0.2 larvae per cc, 3rd lactation. The infection may have persisted longer, but this phase of the experiment was terminated after the third lactation.

Secretions were not taken from the mammary gland of the sow until immediately (12 to 24 hr) prior to parturition. It was not determined whether the larvae were present in the rapidly developing secretory portion of the mammary gland, or whether the larvae moved into the area during the final hours prior to parturition.

3. Distribution of larvae in tissue: The location and concentration of larvae in the tissues of the naturally infected sow are compared in Table 3. Larvae were found primarily in subcutaneous adipose tissue. The adipose tissue surrounding the mammary gland contained the greatest number of larvae. Internal adipose deposits were free of larvae. Relatively high proportions of larvae were found in the adipose tissue along the rib cage and dorsum of the back. The absence of any inflammatory reaction (Fig. 1) was seen upon histological examination of biopsied adipose tissue. In essence, the tissue appeared normal, with the exception of larvae being present. The larvae appeared to locate between the fat cells.
Figure 1. Photomicrograph of *S. ransomi* in adipose tissue—mammary area.

C. Experimental infections

1. Rate of development: Table 4 compares the rate of development of larvae taken by biopsy from adipose tissue at 3 and 7 days postexposure. The only outstanding morphologic feature was the increased size (length and width) of the genital primordium. Division progressed to the eight-cell stage. The total length, total width, and length of the tail were within the normal range for those of filariform larvae. Even though the length and width of the genital primordium had increased, it had not attained the size observed in colostral larvae or larvae recovered from adipose tissue and mammary gland.

2. Migration of tissue larvae: Biopsies of mammary glands were taken 4 to 7 days prior to parturition and entire glands were surgically removed 14 days prior to parturition. Material examined histologically and by baermannization gave negative results. In each case, however, the piglets became infected. Venous blood samples taken from sows during parturition and examined by filtration were negative for *S. ransomi* larvae.

3. Exposure of the pregnant female: Naturally infected gilts held in isolation for 3 to 4 months (no exposure during pregnancy) passed larvae in colostrum and milk and infected their offspring. Gilts reared with no exposure following colostral infection failed to have larvae in milk or to infect their offspring.

Five gilts reared free of colostral and percutaneous infection were exposed (4 divided
doses of approximately 1.3 million larvae 3 to 10 days apart) during late gestation (35 to 15 days before farrowing) had larvae in their milk and infected their offspring.

**Discussion**

The occurrence of transmammary infection by *S. ransomi* and a number of other nematode species has been well documented. The onset of occurrence, duration of persistence, and concentration of larvae per given volume of milk varies for each species. These variations are probably attributable to the location of stored larvae, factors responsible for inciting migration and migratory pathways.

It has been shown that larvae of *S. ransomi* are present in colostrum and later in milk of the sow. Piglets taken from the sow at birth and reared on an artificial diet were free of *Strongyloides* infection (Moncol and Batte, 1966).

Stewart et al. (1969) observed larvae in the tissues of fetuses of sows subjected to massive numbers (7 to 10 million) of filariform larvae during late gestation. However, it is not likely that the sow would naturally be exposed to such large numbers of larvae either at a given time or physiological state. Enigk et al. (1974) observed larvae in the liver, lungs, and small intestines of only two of 47 piglets taken by Caesarean section from sows heavily infected in the first half of pregnancy. The examination of fetal tissues of stillborn and live fetuses from sows reared under normal circumstances yielded no larvae.

Larvae often are present in great numbers in the colostrum prior to parturition and, subsequently, in milk. Passage of *Strongyloides* eggs in the feces of piglets in 2 to 4 days postpartum substantiates the theory that early infection exists. Also, the fact that sows and gilts were not voiding eggs in feces further confirms the theory that transmammary infection is the first and most common means of infection. Furthermore, since patency does not develop until 6 to 7 days following percutaneous infection, this negates the idea that infection occurs as a result of larvae coming from a contaminated environment. Actually, this initial infection serves to contaminate the environment, whereby the piglet increases its worm burden and permits the sow to rebuild her store of larvae for subsequent lactations. If this source of infection is great enough, the time that larvae are passed in milk of the sow may be extended.

Pigs receiving the only transmammary infections continued to void eggs for 6 to 8 weeks longer than pigs maintained under constant exposure conditions. This suggests that the intestinal infection resulting from transmammary infection failed to provide an antigenic stimulus that would enable the host to develop a detectable degree of immunity or sensitization. Limited observations of *S. ransomi* females indicated that they contained few eggs. It was not possible to determine whether pigs under constant exposure eliminated significant numbers of worms in addition to the suppression of egg production. Gilts and sows without recent exposure developed an intestinal infection of short duration (3 to 5 weeks). They voided only a limited number of eggs (100 to 300 epg) when compared to young pigs (20,000 to 100,000 epg). It would appear that a portion of the infecting larvae remained in the adipose tissues of sows and gilts. The time required for larvae to develop in tissue for transmammary passage was relatively short. This was demonstrated when pregnant gilts were exposed within the

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**Table 3. Location of *S. ransomi* somatic larvae in tissues of naturally infected sows.**

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Larvae/100 g</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subcutaneous dorsum of back</td>
<td>17</td>
</tr>
<tr>
<td>Subcutaneous lateral body wall (prefemoral)</td>
<td>15</td>
</tr>
<tr>
<td>Subcutaneous inguinal</td>
<td>1</td>
</tr>
<tr>
<td>Subcutaneous lateral body wall (costal)</td>
<td>92</td>
</tr>
<tr>
<td>Mammary gland and fat</td>
<td>200</td>
</tr>
<tr>
<td>Subcutaneous abdominal wall 10 cm lateral to mammary gland</td>
<td>225</td>
</tr>
<tr>
<td>Omentum</td>
<td>Neg</td>
</tr>
<tr>
<td>Pericardial fat</td>
<td>Neg</td>
</tr>
<tr>
<td>Submaxillary fat</td>
<td>&lt; 1</td>
</tr>
<tr>
<td>Subperitoneal fat</td>
<td>&lt; 1</td>
</tr>
<tr>
<td>Rear leg muscle</td>
<td>Neg</td>
</tr>
<tr>
<td>Lung</td>
<td>Neg</td>
</tr>
<tr>
<td>Heart</td>
<td>Neg</td>
</tr>
</tbody>
</table>

* Five samples of 50 g each.

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**Table 4. Development of *S. ransomi* in adipose tissue.**

<table>
<thead>
<tr>
<th>Days after injection</th>
<th>Measurements* in microns (mean)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Total length</td>
</tr>
<tr>
<td>3</td>
<td>560</td>
</tr>
<tr>
<td>7</td>
<td>539</td>
</tr>
</tbody>
</table>

* Based on 10 larvae each observation period.
final 4 to 6 weeks of gestation. Measurements of larvae from adipose tissue indicated that development of the genital primordium occurred quite rapidly (3 to 7 days).

Stone et al. (1967) applied S. ransomi larvae to the skin of 21-day-old pigs and found larvae in the lungs and other internal organs and leg muscles in 12 hr; juveniles were found in the small intestine within 36 hr. Egg voiding began on day 6 postinfection. This form of migration is commonplace in the young piglet and, to some extent, in the adult pig. On the other hand, young pigs (12 to 20 weeks old) and mature gilts (8 to 12 months old) that received frequent exposure stored larvae in adipose tissue. It was not determined whether males pigs stored larvae. Even if they did, the larvae would end up in a "dead-end" host. Olsen and Lyons (1965) observed that larvae of the seal hookworm taken from males and nonpregnant cows were not infective.

The ease of recovering filariform larvae from the adipose tissues of the subcutaneous ventral abdominal area (mammary area) from gilts and sows indicated a larval predilection for this tissue and anatomical location. Even though larvae were found in adipose tissues some distance from the ventral abdomen, the mammary area appeared to be the site of choice. Larvae are known to penetrate the dermis very rapidly; consequently, the posture of the animal (ventral or lateral recumbency during exposure) may explain this wide distribution of larvae. The reason for larval concentration in adipose tissue remains unknown. One theory that seems plausible is that adipose tissue receives limited circulation and, thereby, larvae may be free to develop and roam in an environment with few macrophages and antibodies.

Stewart et al. (1973) reported the recovery of viable Strongyloides larvae 20 to 30 cm from the site of injection in as little as 7 to 11 weeks. Even though S. ransomi as well as other members of the genus Strongyloides possesses this phenomenal ability to migrate, the mechanism of larval translocation from adipose tissue to milk ducts remains undetermined.

Webster et al. (1958) and Olsen and Lyons (1965) have postulated that migration is induced by hormonal changes occurring near or at the time of parturition. The fact that larvae were not found in biopsies of mammary tissues as late as 10 days prior to parturition would add support to this theory. The idea of vertical migration of larvae from adipose tissue adjacent to alveoli and ducts of the mammary glands seems most convincing.

Smyth (1962) discussed the evolution of the host–parasite relationship and cited several examples of parasites whose life cycle is apparently regulated by host hormones, particularly reproductive hormones. He further pointed out that this phenomenon leads to the synchronization of the life cycle of the parasite with that of its host. Dunsmore (1966) observed that during the breeding season the female rabbit Oryctolagus cuniculus developed a greater nematode infection than the male and ovariectomized females. He stated that "the host-parasite relationship has evolved in such a way that the parasite population is reproducing at a maximal rate at the most suitable time for ensuring the success of the next generation of the parasite." In the instance of S. ransomi, even though it is not reproducing, its presence in milk has been synchronized to infect the piglet, the most susceptible host. The role of hormones on parasitism has been the subject of numerous reports: Salisbury and Arundel (1970), Gibbs (1967), Dunsmore (1971), and Oshima (1961).

The reproductive state of the host, especially lactation, has been shown to inhibit the onset of the self-cure reaction (Connan, 1967, 1970, 1972). Dunsmore (1965) showed that a marked Ostertagia spp. egg count rise occurred in all lambing ewes at or closely associated with parturition. The ewes had not themselves picked up many Ostertagia spp. larvae during the latter part of their pregnancy; consequently, the parturient egg count increase resulted from larvae ingested prior to or early in pregnancy. Penmed ewes given repeated oral doses of Ostertagia spp. larvae during midpregnancy also delayed output of eggs till parturition.

A different model of host–parasite relationship probably exists for each species of parasite. The complexity of the relationship existing between the pig (sow) and S. ransomi is probably no greater than with others. In fact, it bears resemblance to the relationship existing between the ewe and Ostertagia.

Acknowledgment

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