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7431 P

The Fecundity of Parasitic Female Strongyloides.*

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In human and animal infections with *Strongyloides stercoralis* several undetermined factors operate to produce a variability and instability of the strains of the organism.[1] Possibly the presence of parasitic male worms during the period between adolescence and maturity of the parasitic females may play an important part in determining the type of their progeny.[2] In order to obtain information on the fecundity of the parasitic females, we have inoculated 17 dogs with 5 known human strains of *Strongyloides stercoralis* and a rhesus monkey with a chimpanzee strain. The abdominal skin, the buccal cavity and the intracecal mucosa were used as sites of entry for the infective stage (filariform) larvae. Detailed information was obtained concerning the source, type and number of larvae in each inoculum, the prepatent period, the daily number of *Strongyloides* eggs or larvae recovered from the feces, and, on sacrificing the host, the position, number and condition of the parasitic females. The prepatent periods usually ranged from 11 to 18 days, although one dog was positive on the fifth day, one was not detected until the 143rd day and one apparently never passed larvae in the stools.

The number of eggs produced has been shown to rise rapidly to a maximum, after which it declines to a lower level which is maintained for a time and then is gradually reduced to a base level of zero.

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[1] Faust, E. C., and Kagy, E. S., *Am. J. Trop. Med.*, 1932, **13**, 47.

[2] Faust, E. C., *Am. J. Hyg.*, 1933, **18**, 114.

Culture of larvae in the feces, as recommended by Sandground,[3] is a poor index of production of progeny, because so many larvae are passed in a non-viable condition. Likewise, samplings of less than 5 gm. of feces daily are frequently inadequate to detect the larvae present. Even then great care is needed to discover and identify disintegrating larvae, which frequently number 5 to 90% of the total yield.

At the beginning of egg production or soon afterwards the parasitic female worms invade the intestinal mucosa, usually in the region of the duodenum or jejunum, where they deposit their embryonating eggs in the tissues. In the canine hosts the eggs usually hatch in the tissues and the rhabditiform larvae gradually work their way out into the lumen of the bowel, pass down and are evacuated in the feces. In the monkey infection eggs are usually discharged in the unhatched condition. Daily and average weekly counts of progeny in the feces were usually far in excess of uterine eggs in the parasitic females, indicating that several full clutches of eggs were oviposited daily. Furthermore, the yield of parasitic females at autopsy was frequently in excess of expectations, based on the number of infective-stage larvae in the inocula. Twice the number of parasitic females recovered was actually more than the number of larvae in the inocula. This excess can only be explained by the mechanism of internal (hyper-) infection, by which daughter rhabditiform larvae transform into dwarf filariform larvae as they pass down the bowel, thence penetrating the mucosa of the large bowel to effect a migration *via* the blood stream to the lungs and return to the intestine, to develop into and settle down as adult worms usually in the mucosa of the duodenum and jejunum. Likewise, adolescent and recently matured parasitic female worms, found by us in the duodenal and jejunal wall, along with post-productive females, several months after the host has been inoculated, quite likely belong to this type. Many female worms recovered at autopsy were non-fecund. In several instances the feces had yielded no larvae for weeks or months. Nevertheless, these females were still living and became active in physiologic saline solution. Other females were encapsulated; still others were surrounded by phagocytic white cells, which had occasionally broken down the capsules and were attacking the worms. Hence reduction in egg-production and the eventual loss of adult female worms by the host is probably due, not to migration of these worms out of the tissues and their loss in the passed feces, but to host tissue

[3] Sandground, J. H., *Am. J. Hyg.*, 1926, **6**, 337.

reaction, involving 2 mechanisms, namely, encapsulation and phagocytosis.

Although infection of the dog with human strains of *S. stercoralis* is maintained only for a period of weeks or months, in contrast to years in man, the natural host, it is unlikely that man remains infected indefinitely without outside exposure or hyperinfection. Suspected patients should never be pronounced "negative" for *Strongyloides* until prolonged intensive fecal examinations have been made. Even then the patient may be harboring tens or hundreds of post-productive females, which may account for characteristic symptoms of chronic strongyloidosis. Likewise the number of larvae recovered from the feces is not necessarily an index of the degree of infection in the intestinal wall.

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Filarial Periodicity in the Dog Heartworm, Dirofilaria Immitis, After Blood Transfusion.

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Lane[1, 2] believes that the mechanism of filarial periodicity in *filariasis bancrofti* in man is due to a simultaneous daily parturition of the females, involving the destruction daily of as many microfilariae as are produced. O'Connor[3, 4] has found histological evidence to support Lane's hypothesis, and concludes that this occurs about midday in Puerto Rico. On the other hand, Low and Manson-Bahr[5] criticize the theory, because of the effects of absorption of these millions of dead microfilariae in the body, and because microfilariae have been kept alive outside the host for a week. Murgatroyd,[6] however, injected citrated blood containing 720,000

[1] Lane, C., *Lancet*, 1929, 1291.

[2] Lane, C., *Lancet*, 1933, 399.

[3] O'Connor, F. W., *Porto Rico J. Pub. Health and Trop. Med.*, 1931, **6**, 263.

[4] O'Connor, F. W., and Hulse, C. R., *Trans. Roy. Soc. Trop. Med. and Hyg.*, 1932, **25**, 445.

[5] Low, G. C., and Manson-Bahr, P. H., *Lancet*, 1933, 455.

[6] Murgatroyd, F., *Lancet*, 1933, 610.