PHASMID NEMATODE PARASITES OF MAN

SUBCLASS : *PHASMIDIA*
ORDER : *RHABDITIDA*
SUBORDER : *RHABDITINA*
SUPERFAMILY : *RHABDITOIDEA*

GENUS : STRONGYLOIDES (Grassi, 1871)

STRONGYLOIDES STERCORALIS

(Bavay, 1876) Stiles and Hassall, 1902

(*Producing strongyloidosis or strongyloidiasis in man*)

General consideration. *Strongyloides stercoralis* is a parasite of the small intestine of man, which may undergo sexual multiplication either in the intestine or outside the body. It is specially found to be prevalent and frequent in the tropics. It has two forms namely the **parasitic,** and the **free-living** form.

Incidence. It may exceed 25 per cent of population in the tropics and the highest incidence is often seen in children.

Geographical distribution. The worm is commonly found in Brazil, Cochin-China and other tropical countries.

Habitat. The adults (both male and female) are *primarily* found in the lung. *Secondarily,* the females lie buried in the mucosa of jejunum, and the males are found free, though rarely in the lumen of the small intestine.

Larvae and not eggs are passed in human stool. Infective larvae are found in the latrine, contaminated soil and water. Warm, moist soil is necessary to

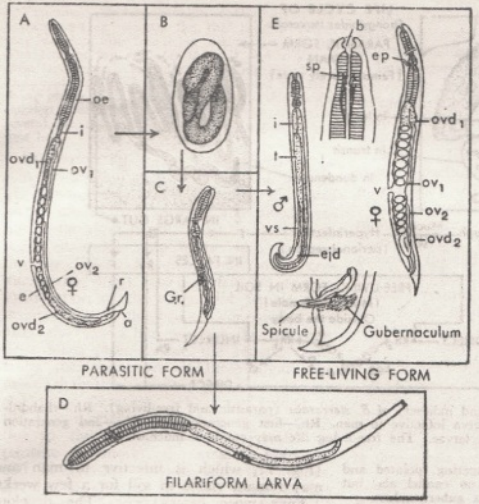

Fig. 6—*Strongyloides stercoralis*. A. Parasitic female ; oe-oesophagus ; i-midgut ; ovd_1-anterior oviduct ; ov_1-anterior ovary ; ov_2-posterior ovary ; v-vulva ; e-egg in utero ; ovd_2-posterior oviduct ; r-rectum ; a-anus.
B. Egg. C. Rhabditiform larva ; G.r.-genital rudiment. D. Filariform larva. E. Free-living male and female in soil ; b-buccal cavity ; sp-buccal spears ; ep-excretory pore ; t-testis ; vs-seminal vesicle ; ejd-ejaculatory duct.

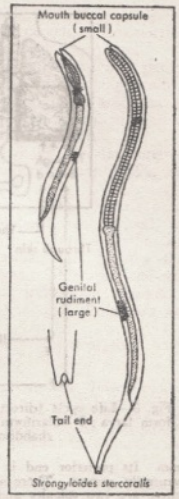

Fig. 7—Larvae of *S. stercoralis* ; left—*rhabditiform* ; right—*filariform*.

complete the life cycle of the free-living form outside the body.

Morphology. There are two kinds of the adult worm : (*a*) the parasitic form, and (*b*) the free-living form. Free living generation is the basic life cycle of the parasite, which is constantly present in warm-climates, where moisture and abundant faecal matter in the soil favour "*free-living existence*".

The Parasitic Form

Adults. The parasitic male is seldom seen and its morphology is not definitely known. The parasitic males are more often found in the lungs, than in the intestine. They are *not tissue parasites* and *do not penetrate* the epithelium. They are soon expelled with the faeces.

The parasitic females (Fig. 6A) are colourless, transparent, slender and filiform. They are 2.2 mm by 0.075 to 0.3 mm, with a finely striated cuticle. The posterior end is pointed. The anterior end is thin with a short buccal cavity and four indistinct lips.

Alimentary tract. In the female, the *oesophagus* is cylindrical and extends through the anterior one third of the worm, where it is continuous with the midgut. The ventral *anus* opens a short distance anterior to the posterior end.

Genitalia. The vulva opens ventrally at the junction of the middle and posterior third of the body ; from this a pair of uteri extend anteriorly and posteriorly to become the oviducts, which are joined by cylindrical ovaries. A number of 6-8 eggs lie in the uterus.

Eggs. The eggs are oval. thin-shelled transparent and measure 50μ by 30μ. They are segmented at the oviposition in the mucosa. They hatch out in a few hours to form rhabditiform larva, and this stage is passed in the human faeces.

Free-Living Form

Adult. The female (rhabditiform) is shorter than the parasitic female and measures 1 mm by 0.06 mm. The cuticle is smooth. The rhabditoid male is broad, short and measures 0.7 by 0.04

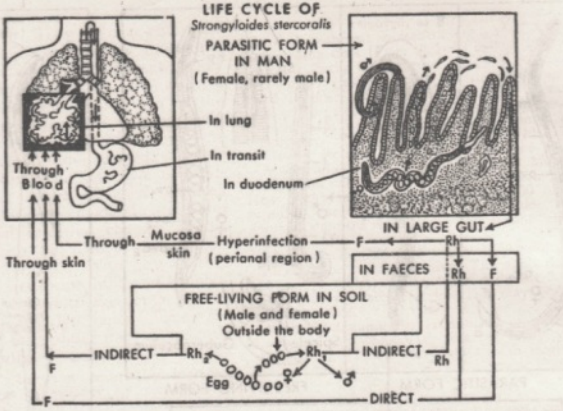

Fig. 8—Life cycle (direct and indirect) of *S. stercoralis* (parasitic and free-living). Rh—rhabditiform larva ; F—filariform larva infective to man. Rh_1—first generation and Rh_2—2nd generation rhabditoid larvae. The free-living life may continue indefinitely.

mm. Its posterior end is tapering, pointed and ventrally curved. There is no caudal ala, but there are two **spicules** and a **gubernaculum.**

ALIMENTARY TRACT. In both sexes the **oesophagus** is *rhabditiform.*

GENITALIA. The cylindrical testis in the *male* lies in the middle third of the body and opens by the seminal vesicle and ejaculatory duct into the cloaca. In the *female*, the genitalia resemble those of the parasitic female.

Eggs. The fertilised female deposits eggs, which are partially embryonated. The eggs are transparent, oval, thin-shelled and measure 70μ by 40μ.

LIFE SPAN in man is unknown as auto-infection may occur. But it is estimated to be about 5 years. The free-living form lives for a few days.

Life cycle (Fig. 8). The adult **parasitic female** of *S. stercoralis* lives within the serpiginous tunnels of the mucosa of the small intestine. **Parasitic males** are rarely seen. The partially embryonated (segmented) eggs are laid by the parasitic female in the submucous tissue of the small intestine. They become *mature* and *hatch out* within a few hours to **rhabditiform** larvae (Fig. 7), as found in the lumen of the small intestine, are passed in the faeces. As they are deposited on the soil, they feed on organic debris and **metamorphose** in a short time into **filariform** larvae (Fig. 7), which is infective to man and may remain viable in soil for a few weeks.

FREE-LIVING GENERATION. The developmental stages are seen outside the body in the case of free-living form : (1) *Indirect development* and (2) *Direct development.*

1. **Indirect development of larvae.** Under conditions of moisture and temperature in the tropics, the *rhabditiform larva* matures in 24-30 hours into free-living sexual generations of males and females. After fertilisation, the female lays second batch of approximately 60 eggs, which hatch out into *rhabditiform larvae.* These are hardly distinguished from those produced by parasitic females. After 3-4 days, they become *filariform* and may infect man, by penetrating the skin and migrate like the larvae of the parasitic form.

2. **Direct development of larvae.** Rhabditiform larvae undergo metamorphosis and directly develop in 3-4 days into *filariform larvae* on the surface of the moist soil ; the sexual phase being absent. A rhabditiform larva produces a single filariform larva after moulting.

The filariform larvae are **infective**, but may remain in the moist soil for several weeks under optimum conditions.

Variation. Under optimum conditions of the development of worms, the rhabditiform larvae may develop into free-living males and females. The females lay eggs on the soil and rhabditiform larvae hatch out and develop into free-living male and female adults and may repeat the free-living phase indefinitely. Alternatively, they might develop into filariform larvae. These die after several weeks unless infection takes place in a new host.

PARASITIC GENERATION. PENETRATING the skin or less commonly the buccal mucosa of human beings, the **infecting filariform larvae** are carried through the blood vessels to the right heart. They break through the pulmonary capillaries into alveoli, develop into *post-filariform* and *pre-adolescent stages* to *adolescent worms*. Fertilisation of the adolescent female may occur either in the bronchi, or trachea, or after the worm has reached the intestine. This takes place before the female penetrates the epithelium of these organs, be cause **rhabditoid adult parasitic males** are not tissue parasites. They are voided in the faeces after a brief stay in the body. But in the case of parastrongyloides (a parasite of shrew), the male is **filariform** and is a *tissue parasite* like the female. In the bronchi or trachea, some of the adolescent females after entering the columnar epithelium oviposit, but others pass to the epiglottis and are swallowed. After reaching the small intestine, they migrate into the submucosa, mature and begin to deposit eggs in the tissues. The incubation period is about 28 days.

AUTO-INFECTION OR HYPERINFECTION (Fig. 8). In heavily parasitised weak patients, the **rhabditiform larvae** from the parasitic female may develop into infective, dwarf **filariform larvae** in the bowel. By penetration of the perineal or the perianal skin, the *filariform larvae* might enter into the circulation and finally reach the intestine *via* lungs as in the parasitic form. The rhabditiform larvae in constipated stool may develop into filariform larvae within the colon and produce internal **auto-infection** in a similar way (**hyperinfection** of Faust).

Pathogenicity. Infection by *S. stercoralis* is known as strongyloidiasis or strongyloidosis.

The worm or its larvae may produce the following complications:

1. During *migration of the filariform larvae*, symptoms may be produced as follows:

SKIN. Dermatitis, creeping eruptions, urticarial rash or petechial haemorrhage due to sensitisation may take place.

INTESTINE. Occasionally in patients with low resistance, massive migration of larvae through the intestinal wall may be fatal. They may produce catarrhal, oedematous and ulcerative enteritis.

LUNGS. Haemorrhage in the lung alveoli, bronchopneumonia, eosinophilia, etc., may occur.

2. *Intestinal lesions.* The female adults may produce diarrhoea with blood and mucus. They form tunnels in the submucosa, through which they burrow their way to deposit eggs. They produce congestion, round cell infiltration, desquamation of the epithelium and sometimes haemorrhage.

Blood may show *leucocytosis* with marked *eosinophilia*.

Epidemiology. TRANSMISSION. Infection occurs by contact of the skin with soil contaminated with filariform larvae, but infection may occur through the mucous surface, or an auto-infection may occur from the perianal or perineal region or through the colon.

Laboratory diagnosis. The DIRECT METHOD of diagnosis is by the recognition of living or dead rhabditiform larvae in the faeces. In light infection and in chronic cases, *concentration method* with 33 per cent zinc sulphate may be needed (see appendix II).

Dwarf filariform larvae may be present in the faeces, if auto-infection is occurring. Larvae are occasionally found in the sputum, urine or duodenal fluid obtained by intubation. If infected stools are allowed to stand for at least 30 hours, free-living forms may be recognised.

SEROLOGICAL AND IMMUNOLOGICAL TESTS. (*a*) *Intradermal* and (*b*) *precipitation tests* with extracts of filariform larvae have been carried out successfully.

Prophylaxis. It is similar to those for hookworm infection.

Treatment. Dithiazanine has been used as a *specific anthelmintic* for strongyloides infection. **Gentian violet** (medicinal) has been found to be specific. Hetrazan may also be tried in doses of 2 mg per kg body weight, thrice daily for 3 weeks.

Thiabendazole has been found to be effective (Cuckler and Mezey, 1966) with a cure rate of 85%, when a single dose of 50 mg/kg body weight is administered (Coura, 1963; Chaia and Cunha, 1963).

Mebendazole ("**Mebex**": *Cipla*). The adult

dose is 200 mg (2 tablets) orally, twice daily for 3 successive days.

IMMUNOPATHOLOGICAL RESPONSE AS RELATED TO PROGNOSIS

In man no known specific response to Strongyloides has been demonstrated (Sheldon, 1937). Repeated experimental infections render dogs and cats partially immune. **Arthus type of skin reactions, haemagglutinin titres** in the sera, and **degranulation of mast cells** of immune animals by the specific Strongyloides antigen have been reported (Coldgrabber & Lewert, 1965). Fatal cases of strongyloidiasis due to chronic infection with altered immunity, or with treatment with immunosuppressive drugs have been referred in the literature (Cruz *et al.*, 1966 ; Rivera *et al.*, 1970 ; Rogers & Nelson, 1966).

The parasitic diseases and the **immunosuppressive drugs**, e.g., cortisone brings about alterations in the immune mechanism by disturbing the *immunologic balance* between the host and the parasite. Such alterations allow for increased **virulence** and **pathogenicity** on the part of the parasite. Heavily infected chronic cases with *hyperinfection, dissemination of the parasites* throughout the body and *failure of eosinophilic response* may be fatal. Patients from endemic areas with **altered immunologic response**, and also under **immunosuppressive therapy**, should be carefully handled, as they are prone to develop hyperinfective and disseminated stages of the disease, which may prove fatal, if not adequately treated early in its course.