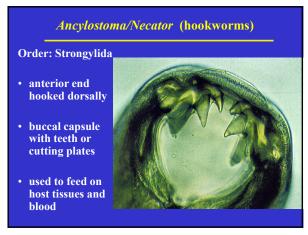


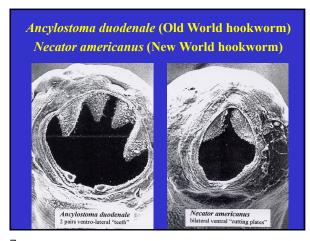
ENTERIC NEMATODES Trichuris trichiura Enterobius vermicularis Trichostrongylus orientalis enteritis Capillaria phillippinensis Ascaris lumbricoides Ancylostoma duodenale Necator americanus anaemia Strongyloides stercoralis

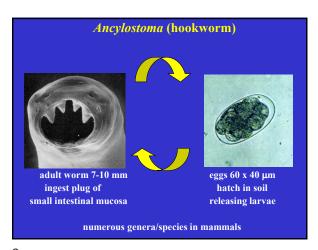
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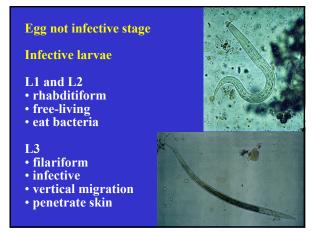
Hookworms in Australia Ancylostoma - with teeth A. caninum - dog, fox, cats (human) A. duodenale - human A. tubaeforme - cat A. braziliense - dog, cat, fox (tropics) Uncinaria – without teeth U. stenocephala - dog, cat, fox Bunostomum - without teeth B. phlebotomum - cattle B. trigonocephalum - sheep Necator - with plates N. *americanus* - human (probably extinct in Australia)

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Pulmonary cycle (tracheal migration)

• infective larvae penetrate skin

• larvae enter circulation and reach lungs

• perforate alveoli, migrate up trachea and swallowed

• larvae reach small intestines and mature to adults

pulmonary cycle

• minor haemorrhage

• pneumonitis (severity dependent on numbers)

• occasional sensitization (allergy)

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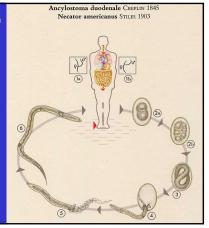
transmission through contaminated soil

cycle

2 months – 9 years

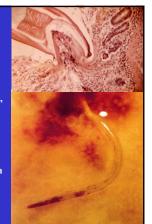
esp. prevalent where faecal contamination of moist/irrigated soil/lawns/gardens (also mines)

infections > 100 worms may cause severe disease



Pathogenesis

- biting worms cause tissue necrosis and abdominal pain (acute stage)
- feeding worms cause anaemia, hypoproteinaemia, iron deficiency (chronic stage) 0.03-0.2 ml blood lost per worm per day (wasteful feeders)
- impaired intestinal absorption (diarrhoea with blood/mucus)
- infections may be fatal in



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Pathogenesis

- · infections can also cause eosinophilic enteritis
- previously warranted exploratory surgery
- · excision and resection (considerable trauma)
- nowadays treated with anthelmintics



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Phenomenon of HYPOBIOSIS "arrested larval development"

e.g. A. duodenale

Order: Rhabditida

larvae may become "arrested" in gut or muscles and then recommence their development later

this means that hookworms may enter the intestine weeks or months after infection

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Infection routes

via skin

– larvae penetrate skin

enteric

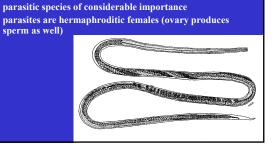
- larvae penetrate gut (A. duodenale)
- transplacental
- larvae cross placenta (A. caninum) and infect offspring

transmammary

- larvae may infect neonates via breast milk (A. duodenale/A. caninum)

via paratenic hosts

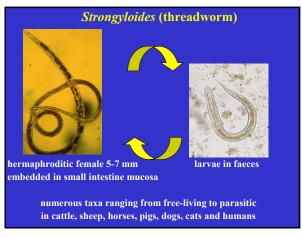
- larvae may survive in tissues of other mammals and even in insects



Strongyloides (threadworm)

typical species are free-living soil-dwelling nematodes, feed on bacteria, with "rhabditiform pharynx"

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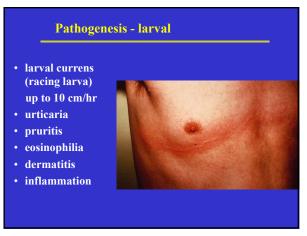
Thin-walled eggs hatch in mucosa

Larval development

L1 and L2 (350 µm)
• rhabditiform
• passed in faeces

L3 (600 µm)
• filariform
• infective
• penetrates skin

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Pulmonary cycle (tracheal migration)

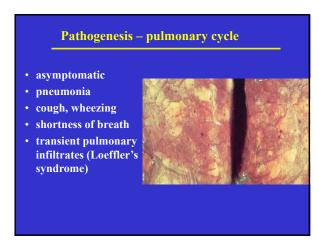
• infective larvae penetrate skin

• larvae enter circulation and reach lungs

• perforate alveoli, migrate up trachea and swallowed

• larvae reach small intestines and mature to adults

21 22



AUTO-INFECTION

- Eggs may hatch and emergent larvae moult twice in intestine to become infective

- Auto-infection may then occur by penetration of larvae in lower gut or peri-anal region

- Auto-infection allows infection to persist and to become very heavy if conditions are right

23 24

Free-living adults

- some rhabditiform larvae passed in faeces form free-living adults (male and female)
 [not in temperate climates]
- females produce eggs
- larvae hatch and moult twice to form infective filariform larvae
- infective L3 larvae penetrate skin (rarely ingested)
- · tracheal migration
- · form parasitic females



auto-infection

auto-infection

direct
(infective larvae)

indirect
(free-living adults form infective larvae)

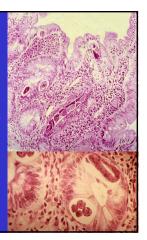
Adult in mucosa of small intestine

Penetrates skin Penetrates intestine or perhand skin Penetrates skin Penet

25 26

Pathogenesis

- many asymptomatic
- infections may persist for years (auto-infection or re-infection)
- · heavy infections damage mucosa
- sloughing, haemorrhage
- epigastric pain (may mimic peptic ulcer or Crohn's disease)
- peripheral eosinophilia
- reactive arthritis?



Pathogenesis - Hyperinfection

Heavy infections can develop when individuals are immunosuppressed/stressed

- · large numbers filariform larvae produced
- penetrate bowel and disseminate
- cause colitis
- polymicrobial sepsis
- pneumonitis
- meningitis



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TREATMENT - nematocides

Anthelmintic mebendazole albendazole pyrantel

Ancylostoma (hookworm) + - +

Strongyloides (threadworm) - +

SUMMARY

Simple life cycles - eggs ingested - larvae/adults in gut

e.g. Trichuris (whipworm)

Enterobius (pin worm)

Trichostrongylus (hair worm)

Capillaria (round worm)

More complex - egg ingested

- larvae migrate through lungs

- adults in gut

e.g. Ascaris (round worm)

Most complex - larvae penetrate skin

- larvae migrate through lungs

- adults in gut

e.g. Ancylostoma/Necator (hookworm) Strongyloides (threadworm)

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