Oxyurina
(Syphacia obvelata, Syphacia muris, Aspiculuris tetraptera, Dentostomella translucida)

Host species
• Syphacia (S.) obvelata: mainly mouse (also rat, hamster, gerbil, wild rodents)
• S. muris: mainly rat (also mouse, hamster, gerbil, wild rodents)
• Aspiculuris (A.) tetraptera: mouse, rat (rarely), wild rodents

Organotropism
• intestinal tract: Syphacia spp. primarily caecum / rectum, A. tetraptera primarily colon

Life cycle
Syphacia spp.
Direct cycle which requires only 11-15 days. Gravid females deposit their eggs in the perianal region. The eggs become infectious within 5-20 hours after release (Pritchett 2002).
Three possible infectious routes:
• direct: by ingestion of embryonated eggs from the perianal region
• indirect: by ingestion of food or water contaminated with embryonated eggs
• retroinfection: when eggs hatch in the perianal region and the larvae migrate back into the colon by way of the anus (Flynn 1973; Baker 2007)

A. tetraptera
Direct cycle requires 23-25 days. Females lay their eggs in the colon and the eggs leave the host on faecal pellets. The eggs become infectious after 6-7 days at room temperature.
Infection by ingestion of infectious eggs (Flynn 1973; Baker 2007)

Susceptibility
• the prevalence of pinworms in an infected rodent population depends on age, sex and host immune status
• in enzootically infected colonies, weanlings develop the greatest parasite loads, males are more heavily parasitized than females
• Syphacia numbers diminish with increasing age of the host (Wescott 1982)
• athymic (Foxn1nu) mice have increased susceptibility (Jacobson and Reed 1974)
• Mastomys coucha is more susceptible than the BALB/c mouse (Higgins-Opitz et al.1990)
• in rats, the infestation rates of S. muris are higher in the WKY strain than in the SHR strain (Lübcke et al. 1992)
• increase in resistance to pinworm infection with advancing age of rats (Wagner 1988)

Clinical disease and pathology
• subclinical (Flynn 1973; Wescott 1982)
• infections with pinworms generally are considered to be non- or mildly pathogenic in animals with normal immune system (Taffs 1976; Levine 1968; Harkness et al. 1995)
• symptoms are poor condition, rough hair coats, reduced growth rate, rectal prolaps (Hoag 1961; Harwell and Boyd 1968; Jacobson and Reed 1974)
• experimentally with S. muris infected animals grow slower than uninfected animals (Wagner 1988)
• infection with S. muris retards the growth of young mice and accelerates the development of their hepatic monooxygenase system (Mohn and Philipp 1981)
• pinworms of laboratory rodents are generally not considered pathogens (Flynn 1973; Wescott 1982)
• somatic extract of S. muris adults was examined for proteins with mass spectrometry. 359 proteins were identified and the largest protein families consisted of metabolic enzymes and those involved in the nucleic metabolism and cell cycle (Sotillo et al. 2012)

**Morbidity and mortality**
• none

**Zoonotic potential**
• S. obvelata seems to occur in humans, but it has no known health significance (Flynn 1973; Kellogg and Wagner 1982; Ross et al. 1980; Wescott 1982)

**Interference with research**
• infection with pinworms reduces the occurrence of adjuvant-induced arthritis (Pearson and Taylor 1975)
• infection alters the humoral response to nonparasitic antigenic stimuli indicating that infection might modulate the immune system (Sato et al. 1995)
• infection with S. obvelata induces a proliferation of T- and B-lymphocytes in spleen and lymph nodes and occasional germinal centre formation (Beattie et al. 1981)
• athymic mice infected with pinworms develop a lymphoproliferative disorder which eventually leads to lymphoma (Beattie et al. 1980; Baird et al. 1982)
• animals infected with pinworms are not suitable for growth studies (Wagner, 1988)
• in rats, infection with Syphacia muris caused impaired transport of water, sodium, and chloride in the intestine (Taylor et al. 1995)
• infection with S. obvelata in mice causes a significant reduction of activity in behavioural studies (McNair and Timmons, 1977), however infection with S. muris in rats does not affect activity levels (Webster 1994)
• in rats, intestinal transport of water and electrolytes is significantly decreased due to pinworm infection (Lübcke et al. 1992)
• pinworm infection in neonatal mice causes a strong Th2 response including high levels of IL-4 and IL-5 production. This Th2 response ended immediately after pinworm eradication (Agersborg et al. 2001)
• infection with S. obvelata induces a transient TH2-type immune response with elevated interleukin 4 (IL-4), IL-5, and IL-13 cytokine production and parasite-specific immunoglobulin G1 (IgG1) in BALB/c mice. BALB/c mice deficient in IL-13, IL-4/13, or the IL-4 receptor alpha chain showed chronic disease with a >100-fold higher parasite burden, increased gamma interferon production, parasite-specific IgG2b, and a default Th2 response. In addition helmith-infected mice immunized against ovalbumin (Ova) elicited more severe anaphylactic shock with reduced Ova-specific IL-4 and IL-5 than did noninfected controls, demonstrating that S. obvelata infection is able to influence non-related laboratory experiments (Michels et al. 2006)
• infection with S. obvelata in mice causes hematopoietic alterations, characterized by increased myelopoiesis and erythropoiesis (Bugarski et al. 2006)
• S. obvelata-infected mice show altered sensitivity to IL-17 (Bugarski et al. 2006)
• natural S. obvelata infection induced significant alterations in murine bone marrow cells manifested at the molecular level. Infection induced sustained phosphorylation of the members of the three major groups of distinctly regulated mitogen-activated protein kinases (MAPKs), the p38, the c-Jun amino-terminal kinase (JNK) and the extracellular signal-regulated kinase (ERK), as well as enhanced expression of mRNA for the inducible nitric oxide synthase (iNOS) in the bone marrow cells. Obviously, S. obvelata is able to manipulate signal transduction pathways in the hosts’ bone marrow cells (Ilić et al. 2010)
• infection with *S. muris* in Wistar rats induces an immunity against subsequent infection with *Echinostoma caproni*. The worm recovery was significantly decreased in rats primarily infected with *S. muris* (Trelis et al. 2013)
• in mice infected with *A. tetraptera* adult worms can penetrate the colonic wall and cause granulomas (Mullink 1970)
• rats occasionally may develop focal submucosal granulomas associated with pinworm infection (Percy and Barthold 2001)
• infection with *Dentostomella translucida* in gerbils was found to be associated with a mild diffuse eosinophilia of the lamina propria of the anterior small intestine (Smith and Snider 1988). These results are contradicted by finding a similar eosinophilia in both infected and control animals (Berger 1991)
• research data support the concept that infection with helminth parasites can reduce the severity of concomitant disease. Infection with helminth parasites has been tried as therapy in inflammatory bowel disease of humans (Matisz et al. 2011)
• rodent helminth infection can be used as models to uncover the workings of mammalian immune response to metazoan parasites

**Notice**
• the eggs of pinworms survive for months in the animal room environment (Flynn 1973; Klement et al. 1996; Meade and Watson 2014)

**References**


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