

**HELMINTHIASIS OF UNGULATES**

ANIMAL GROUP AFFECTED	TRANSMISSION	CLINICAL SIGNS	FATAL DISEASE ?	TREATMENT	PREVENTION & CONTROL
Artiodactyls, perissodactyls	Faeco-oral, by ingestion of infective eggs, larvae or intermediate hosts	Loss of condition, diarrhoea	Occasionally	Anthelmintics	<i>In houses</i> Hygiene <i>in zoos</i> monitoring and prophylaxis, paddock management

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Susceptible animal groups Artiodactyls and perissodactyls, especially species from arid and semi-arid regions when kept on grass.	
Causative organism A range of cestodes, trematodes and especially nematodes, including <i>Haemonchus</i> , <i>Ostertagia</i> and related genera, particularly <i>Camelotrongylus mentulatus</i> , <i>Trichostrongylus</i> , <i>Nematodirus</i> , <i>Cooperia</i> , <i>Trichuris</i> , <i>Capillaria</i> , <i>Strongylus</i> , <i>Ascaris</i> , <i>Dictyocaulus</i> sp.	
Zoonotic potential Majority of ungulate parasites pose no threat. Main exception is <i>Trichinella spiralis</i> , which can infect humans if they eat meat containing encysted larvae.	
Distribution World-wide, but species of parasites will vary according to the climate.	
Transmission a) Direct life cycle with passage of resistant ova in faeces, survival/development in, and contamination of, the environment and ingestion by new host (eg. <i>Ascaris</i> , <i>Trichuris</i>). b) The same, but with hatching of larvae from ova and development in the environment. Entry to new hosts by percutaneous infection (eg. <i>Bunostomum</i> hookworms) or ingestion of 3 rd stage larvae (most G-I nematodes). c) Passage of ova in faeces and direct ingestion by intermediate host or hatching and infection of an intermediate host. Development within one or more intermediate hosts before ingestion by the final host of the infected intermediate host, or a final larval stage (e.g. flukes such as <i>Fasciola</i> sp. and tapeworms, e.g. <i>Moniezia</i>).	
Incubation period Variable, depending on the rate and degree of infection, non-pathogenic developmental stages within the host, and the host's innate resistance, and nutritional and immune status.	
Clinical symptoms a) GI helminths cause poor growth or loss of condition, often with diarrhoea, which may be liquid and cover the perineal skin and hindlegs; hookworms may additionally cause anaemia. b) Type II ostertagiasis presents as severe depression, collapse and death with, or without, diarrhoea. c) Lungworms may cause coughing, respiratory distress and loss of condition. d) With liver fluke infections there may be signs of liver failure (jaundice, loss of condition, anaemia, etc.). e) Large numbers of migrating helminth larvae may also cause liver damage, and larvae of <i>Strongylus vulgaris</i> in equids may be associated with colic, hindlimb lameness or sudden death.	

**Post mortem findings**

- a) Fatal GI helminthiasis: thin/emaciated carcass, +/- fat atrophy, diarrhoea. Abomasal nodules and/or enteritis. Nematodes visible upon gross examination if *Haemonchus*, or *Bunostomum*.
- b) Severe abomasitis with haemorrhagic parasitic nodules.
- c) Excess mucus in the respiratory tract, presence of lungworms, secondary lung changes.
- d) Liver necrosis and/or fibrosis, presence of flukes.
- e) liver necrosis associated with larval tracks from the liver capsule. *Strongylus vulgaris* larvae in thromboemboli of the cranial mesenteric or other arteries.

Diagnosis

- a) History, clinical signs, high parasite egg counts (McMaster count, or similar) in the individual and herd, raised plasma pepsinogen concentration (abomasal nematodes), gross PM findings and presence of large numbers of adult helminths in abomasal and/or small intestinal samples examined grossly and under a dissecting microscope.
- b) History of stress, occurrence in winter, raised plasma pepsinogen, gross PM findings and presence of large numbers of nematode larvae in samples of abomasal contents, c) history, clinical signs, presence of lungworm larvae in faeces (Baerman technique).
- d) History, clinical signs, elevated bilirubin and liver enzymes, hypoalbuminaemia, presence of ova in faeces (flotation solutions denser than saturated salt, eg. zinc sulphate, sugar).
- e) History of grazing heavily contaminated pasture, clinical and PM findings.

Material required for laboratory analysis

Faeces from clinically affected individuals. Faeces from the herd; several samples, or a well-mixed group sample, collected and tested regularly. Faecal samples collected in the wild can be preserved in sodium acetate formalin and examined later for helminth ova and protozoal cysts. Blood samples from affected individuals for haematology, routine biochemistry and plasma pepsinogen assay. Samples (eg. 2%) of abomasal, small intestinal and large intestinal contents stored in 4-5% formalin for counting and identification of adult worms. Collections of discrete adult helminths in 70% ethanol (or 4% formalin). Histopathology of mucosal, liver, arterial and any other relevant lesions.

Relevant diagnostic laboratories

Parasitology Department, Veterinary Laboratories Agency, Weybridge, Surrey KT15 3NB, UK
Natural History Museum, London, SW7 5BD, UK
Royal Veterinary College, Camden Town, London, NW1 0TU, UK
Governmental and private veterinary laboratories

Treatment

Anthelmintics:

Nematodes: benzimidazoles (fenbendazole, albendazole etc.), levamisole, avermectins (ivermectin, doramectin, moxidectin etc.), morantel and pyrantel

Trematodes: oxclozanide, rafoxanide, nitroxylnil, triclabendazole

Cestodes: praziquantel, niclosamide, bunamidine.

Resistance has been found to many anthelmintics, so it is important to treat at full therapeutic dose rates and for sufficient time to eliminate worms, and to alternate anthelmintic drug types for prophylactic treatment (see below).

Prevention and control in zoos

- Quarantine, testing +/- treatment prior to introduction of new stock.
- Routine anthelmintic treatment for susceptible species, frequency dependent on susceptibility and strategic timing to reduce egg output.
- Regular monitoring to detect increases in group, faecal egg counts before the onset of clinical disease.
- Reduce faecal contamination by removal of faeces, or rotation of paddocks.
- Reduce stocking density, improve nutrition and avoid behavioural, and other, stresses.

Suggested disinfectant for housing facilities

Removal of faecal contamination, cleaning and allowing facilities to dry will eliminate the majority of ova. All major disinfectants may be used for further reduction in contamination.

Notification

None are notifiable.

Guarantees required under EU Legislation**Guarantees required by EAZA Zoos**

Ungulates should be free of endoparasites when transferred between collections, and this is achieved by



testing faeces and treatment if any ova present.

Measures required under the Animal Disease Surveillance Plan**Measures required for introducing animals from non-approved sources****Measures to be taken in case of disease outbreak or positive laboratory findings****Conditions for restoring disease-free status after an outbreak****Contacts for further information****References**

1. Courtney, C. H., and G. V. Kollias. 1983. Management concepts for endoparasitism in exotic ungulates. AAZV Annual Proc., Tampa, Florida. Pp.62-63.
2. Flach, E. J. 1997. Investigation and control of gastrointestinal parasitism in zoo ungulates. Verh. Ber. Erkr. Zootiere 38: 359-365.
3. Isaza R., and G. V. Kollias. 1999. Designing a trichostrongyloid parasite control program for captive exotic ruminants. Chapter 87. *In*: Fowler, M. E., and R. E. Miller (eds). Zoo and Wild Animal Medicine: Current Therapy, 4th Ed. WB Saunders Co., Philadelphia, Pennsylvania. Pp. 593-597.
4. Mikolon A. B, W. M. Boyce, J. L. Allen, I. A. Gardner, and L. F. Elliott. 1994. Epidemiology and control of nematode parasites in a collection of captive exotic ungulates. J. Zoo Wildl. Med. 25: 500-510.