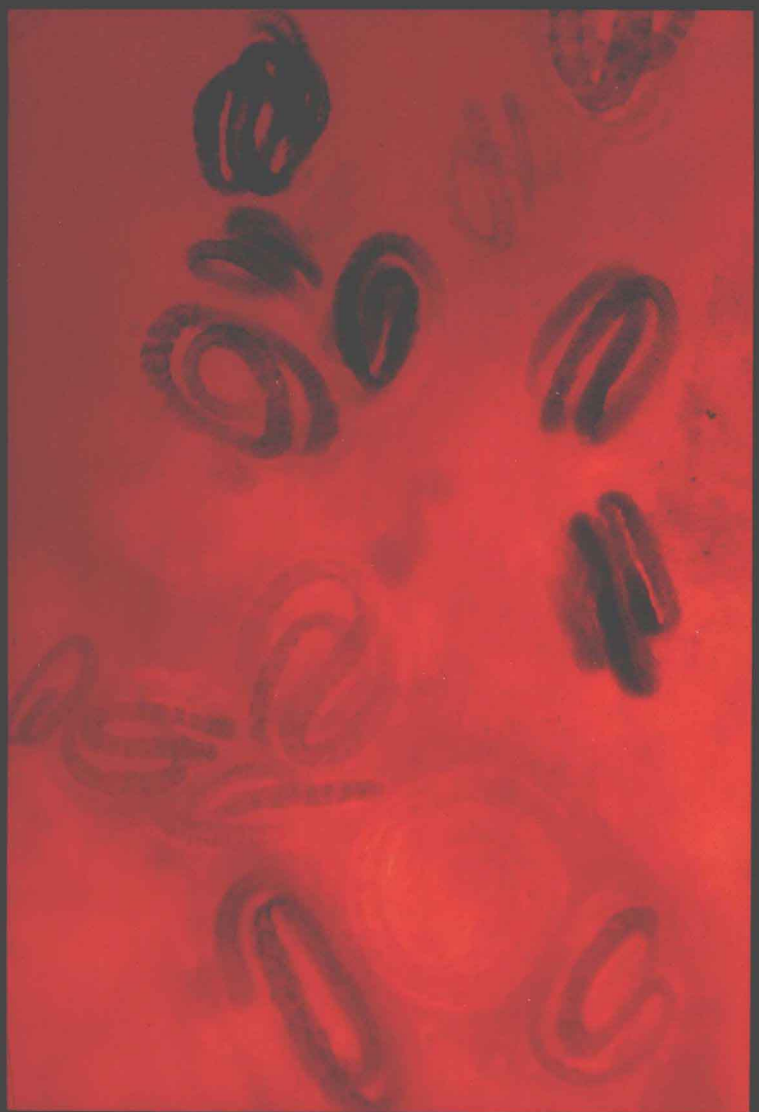
VETERINARY PARASITOLOGY



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Veterinary Parasitology

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Veterinary Parasitology

FOREWORD

This book is intended for students of veterinary parasitology, for practising veterinarians and for others requiring information on some aspect of parasitic disease.

Originally intended as a modestly expanded version of the printed notes issued to our students in the third and fourth years of the course, the text, perhaps inevitably, has expanded. This was due to three factors. First, a gradual realisation of the deficiencies in our notes; secondly, the necessity of including some of the comments normally imparted during the lecture course and thirdly, at the suggestion of the publishers, to the inclusion of certain aspects of parasitic infections not treated in any detail in our course.

We should perhaps repeat that the book is primarily intended for those who are directly involved in the diagnosis, treatment and control of parasitic diseases of domestic animals. The most important of these diseases have therefore been discussed in some detail, the less important dealt with more briefly and the uncommon either omitted or given a brief mention. Also, since details of classification are of limited value to the veterinarian we have deliberately kept these to the minimum sufficient to indicate the relationships between the various species. For a similar reason, taxonomic detail is only presented at the generic level and, occasionally, for certain parasites, at species level. We have also trod lightly on some other areas such as, for example, the identification of species of tropical ticks and the special significance and epidemiology of some parasites of regional importance. In these cases, we feel that instruction is best given by an expert aware of the significance of particular species in that region.

Throughout the text we have generally referred to drugs by their chemical, rather than proprietary, names because of the plethora of the latter throughout the world. Also, because formulations are often different, we have avoided stating doses; for these, reference should be made to the data sheets produced by the manufacturer. However, on occasions when a drug is recommended at an unusual dose, we have noted this in the text.

In the chapters at the end of the book we have attempted to review five aspects of veterinary parasitology, epidemiology, immunity, anthelmintics, ectoparasiticides and laboratory diagnosis. We hope that this broader perspective will be of value to students, and particularly to those dismayed by the many complexities of the subject.

There are no references in the text apart from those at the end of the chapter on diagnosis. This was decided with some regret and much relief on the grounds that it would have meant the inclusion, in a book primarily intended for undergraduates, of hundreds of references. We hope that those of our colleagues throughout the world who recognise the results of their work in the text will accept this by way of explanation and apology.

We would, however, like to acknowledge our indebtedness to the authors of several source books on veterinary parasitology whose work we have frequently consulted. These include *Medical and Veterinary Protozoology* by Adam, Paul and Zaman, *Veterinaermedizinische Parasitologie* by Boch and Supperer, Dunn's *Veterinary Helminthology*, Euzéby's *Les Maladies Vermineuses des Animaux Domestiques*, Georgi's *Parasitology for Veterinarians*, Reinecke's *Veterinary Helminthology*, Service's *A Guide to Medical Entomology* and Soulsby's *Helminths, Arthropods and Protozoa of Domesticated Animals*.

Any student seeking further information on specific topics should consult these or, alternatively, ask his tutor for a suitable review.

The ennui associated with repeated proof-reading may occasionally (we hope, rarely) have led to some errors in the text. Notification of these would be welcomed by the authors. Finally we hope that the stresses endured by each of us in this collaborative venture will be more than offset by its value to readers.

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Secondly, to the following individuals and companies who kindly allowed us to use their photographs or material as illustrations or plates:

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Thirdly, to the pharmaceutical companies of Crown Chemical, Kent, England; Hoechst UK, Bucks; Merck Sharp & Dohme, Herts; Pfizer, Kent; Schering, New Jersey; Syntex Agribusiness, California. Their generosity enabled us to present many of the photographs in colour, thus enhancing their value.

Finally, to those members of the Faculty of Veterinary Medicine, Glasgow, whose cooperation was essential in the production of this book. We would especially like to thank Kenneth Bairden, our chief technician, who prepared much of the material for photography, often at inordinately short notice; Archie Finnie and Allan May, of the Photographic Unit, who, almost uncomplainingly, undertook the extra work of photographing many specimens; our two departmental secretaries, Elizabeth Millar and Julie Nybo without whose skill and attention to detail this book would certainly not have been written.

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September 1985

CONTENTS

VETERINARY HELMINTHOLOGY

PRINCIPLES OF CLASSIFICATION

All animal organisms are related to one another, closely or remotely, and the study of the complex systems of inter-relationship is called **systematics**. It is essentially a study of the evolutionary process.

When organisms are examined it is seen that they form natural groups with features, usually morphological, in common. A group of this sort is called a **taxon**, and the study of this aspect of biology is called **taxonomy**.

The taxa in which organisms may be placed are recognised by international agreement, and the chief ones are: **Kingdom**, **Phylum**, **Class**, **Order**, **Family**, **Genus** and **Species**. The intervals between these are large, and some organisms cannot be allocated to them precisely, so that intermediate taxa, prefixed appropriately, have been formed; examples of these are the **Suborder** and the **Superfamily**. As an instance, the taxonomic status of one of the common abomasal parasites of ruminants may be expressed as shown in the next column.

Kingdom	Animalia
Phylum	Nemathelminthes
Class	Nematoda
Order	Strongylida
Suborder	Strongylina
Superfamily	Trichostrongyloidea
Family	Trichostrongylidae
Subfamily	Haemonchinae
Genus	*Haemonchus*
Species	*contortus*

The names of taxa must be adhered to according to the international rules, but it is permissible to anglicise the endings, so that members of the superfamily Trichostrongyloidea in the example above may also be termed trichostrongyloids.

The names of the genus and species are expressed in Latin form, the generic name having a capital letter, and they must be in grammatical agreement. It is customary to print foreign words in italics, so that the name of an organism is usually underlined or italicised. Accents are not permitted, so that, if an organism is named after a person, amendment may be necessary; the name of Müller, for example, has been altered in the genus *Muellerius*.

The higher taxa containing helminths of veterinary importance are:

Major
Nemathelminthes (Roundworms)
Platyhelminthes (Flatworms)

Minor
Acanthocephala (Thornyheaded Worms)

Phylum NEMATHELMINTHES

Though the phylum Nemathelminthes has six classes only one of these, the **nematoda**, contains worms of parasitic significance. The nematodes are commonly called roundworms, from their appearance in cross-section.

Class NEMATODA

A system of classification of nematodes of veterinary importance is given in Table 1.

It must be emphasised that this is not an exact expression of the general system for parasitic nematodes, but is a simplified presentation intended for use in the study of veterinary parasitology. It is based on the ten superfamilies in which nematodes of veterinary importance occur, and which are conveniently divided into **bursate** and **non-bursate** groups as shown in Table 1.

Table 1 Parasitic Nematoda of veterinary importance
Simplified Classification

Superfamily	Typical features
Bursate nematodes	
Trichostrongyloidea *Trichostrongylus*, *Ostertagia*, *Dictyocaulus*, *Haemonchus*, etc.	Buccal capsule small Life cycle **direct;** infection by L_3.
Strongyloidea *Strongylus*, *Ancylostoma*, *Syngamus*, etc.	Buccal capsule well developed; leaf crowns and teeth usually present. Life cycle **direct;** infection by L_3.
Metastrongyloidea *Metastrongylus*, *Muellerius*, *Protostrongylus*, etc.	Buccal capsule small. Life cycle **indirect**; infection by L_3 in intermediate host.
Non-bursate nematodes	
Rhabditoidea *Strongyloides*, *Rhabditis*, etc.	Very small worms; buccal capsule small. Free-living and parasitic generations. Life cycle **direct**; infection by L_3.
Ascaridoidea *Ascaris*, *Toxocara*, *Parascaris*, etc.	Large white worms. Life cycle **direct**; infection by L_2 in egg.
Oxyuroidea *Oxyuris*, *Skrjabinema*, etc.	Female has long, pointed tail. Life cycle **direct**; infection by L_3 in egg.
Spiruroidea *Spirocerca*, *Habronema*, *Thelazia*, etc.	Spiral tail in male. Life cycle **indirect**; infection by L_3 from insect.
Filaroidea *Dirofilaria*, *Onchocerca*, *Parafilaria*, etc.	Long thin worms. Life cycle **indirect**; infection by L_3 from insect.
Trichuroidea *Trichuris*, *Capillaria*, *Trichinella*, etc.	Whip-like or hair-like worms. Life cycle **direct** or **indirect**; infection by L_1.
Dictophymatoidea *Dioctophyma*, etc.	Very large worms. Life cycle **indirect**; infection by L_3 in aquatic annelids.

STRUCTURE AND FUNCTION

Most nematodes have a cylindrical form, tapering at either end, and the body is covered by a colourless, somewhat translucent, layer, the cuticle.

The cuticle is secreted by the underlying hypodermis, which projects into the body cavity forming two lateral cords, which carry the excretory canals, and a dorsal and ventral cord carrying the nerves (Fig. 1). The muscle cells, arranged longitudinally, lie between the hypodermis and the body cavity. The latter contains fluid at a high pressure which maintains the turgidity and shape of the body. Locomotion is effected by undulating waves of muscle contraction and relaxation which alternate on the dorsal and ventral aspects of the worm.

Most of the internal organs are filamentous and suspended in the fluid-filled body cavity (Fig. 2).

The **digestive system** is tubular. The mouth of many nematodes is a simple opening, which may be surrounded by two or three lips, and leads directly into the oesophagus. In others, such as the strongyloids, it is large,

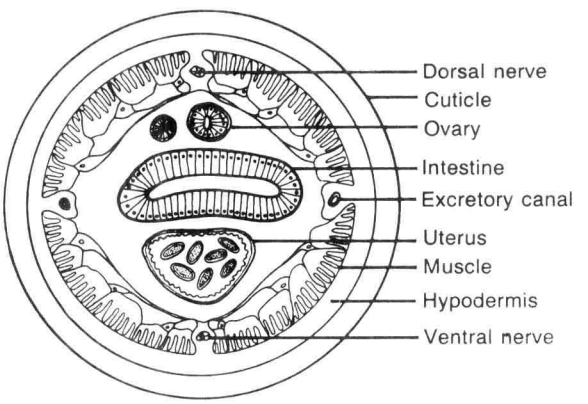

Fig 1 Transverse section of a typical nematode.

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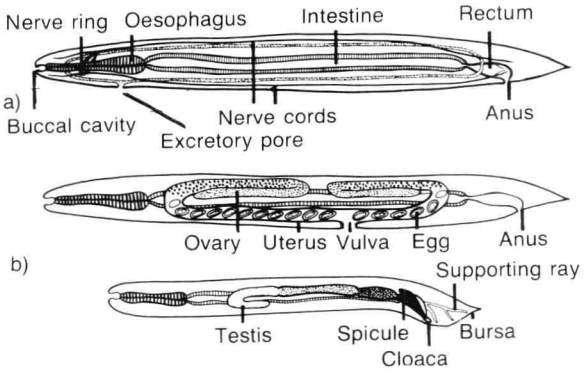


Fig 2 Longitudinal sections of a nematode illustrating:
(a) Digestive, excretory and nervous system.
(b) Reproductive system of female and male nematodes.

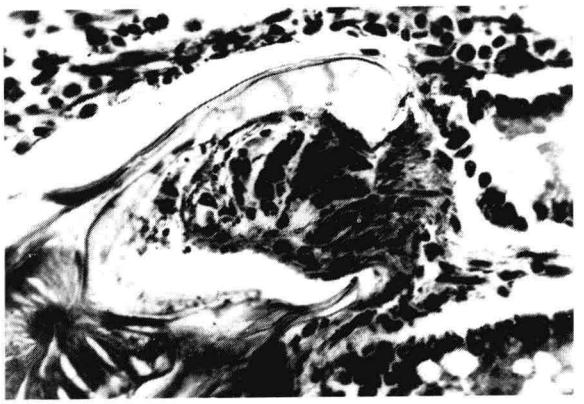


Fig 3 Large buccal capsule of strongyloid nematode ingesting plug of mucosa.

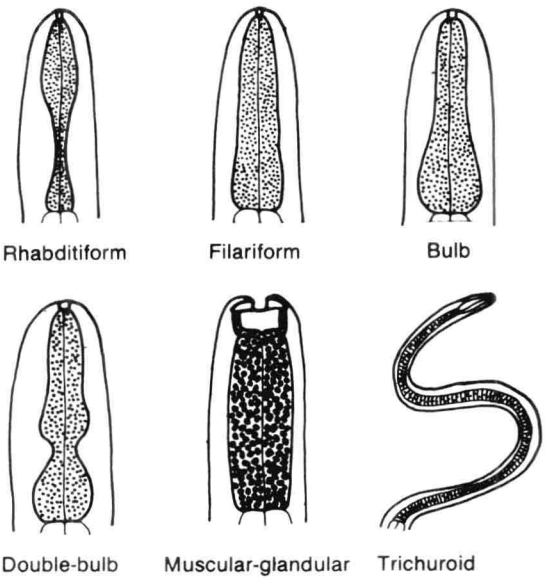


Fig 4 The basic forms of oesophagus found in nematodes.

and opens into a **buccal capsule**, which may contain teeth; such parasites, when feeding, draw a plug of mucosa into the buccal capsule (Fig. 3), where it is broken down by the action of enzymes which are secreted into the capsule from adjacent glands. Some of these worms may also secrete anticoagulant, and small vessels, ruptured in the digestion of the mucosal plug, may continue to bleed for some minutes after the worm has moved to a fresh site.

Those with very small buccal capsules, like the trichostrongyloids, or simple oral openings, like the ascaridoids, generally feed on mucosal fluid and cell debris, while others, such as the oxyuroids, appear to scavenge on the contents of the lower gut. Worms living in the bloodstream or tissue spaces, such as the filarioids, feed exclusively on fluids.

The **oesophagus** is usually muscular and pumps food into the intestine. It is of variable form (Fig. 4), and is a useful preliminary identification character for groups of worms. It may be **filariform**, simple and slightly thickened posteriorly, as in the bursate nematodes; **bulb-shaped**, with a large posterior swelling, as in the ascaridoids; or **double bulb-shaped**, as in the oxyuroids. In some groups this wholly muscular form does not occur: the filarioids and spiruroids have a **muscular-glandular** oesophagus which is muscular anteriorly, the posterior part being glandular; the **trichuroid** oesophagus has a capillary form, passing through a single column of cells, the whole being known as a stichosome. A **rhabditiform** oesophagus, with slight anterior and posterior swellings, is present in the preparasitic larvae of many nematodes, and in adult free-living nematodes.

The **intestine** is a tube whose lumen is enclosed by a single layer of cells or by a syncytium. Their luminal surfaces possess microvilli which increase the absorptive capacity of the cells. In female worms the intestine terminates in an anus while in males there is a cloaca which functions as an anus, and into which opens the vas deferens and through which the copulatory spicules may be extruded.

The so-called **'excretory system'** is very primitive, consisting of a canal within each lateral cord joining at the excretory pore in the oesophageal region.

The **reproductive systems** consist of filamentous tubes. The **female organs** comprise ovary, oviduct and uterus, which may be paired, ending in a common short vagina which opens at the vulva. At the junction of uterus and vagina in some species there is a short muscular organ, the ovejector, which assists in egg-laying. A vulval flap may also be present (Fig. 5).

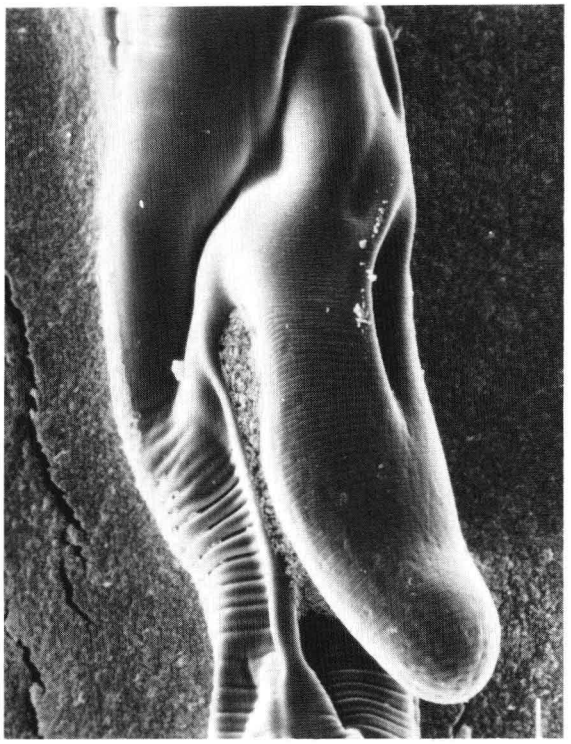


Fig 5 Scanning electron micrograph of a vulval flap of a trichostrongyloid nematode.

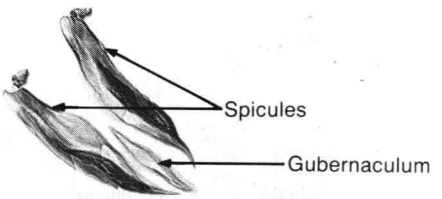


Fig 6 Spicules and gubernaculum of a trichostrongyloid nematode.

The **male organs** consist of a single continuous testis and a vas deferens terminating in an ejaculatory duct into the cloaca. Accessory male organs are sometimes important in identification, especially of the trichostrongyloids, the two most important being the spicules and gubernaculum (Fig. 6). The **spicules** are chitinous organs, usually paired, which are inserted in the female genital opening during copulation. The **gubernaculum**, also chitinous, is a small structure which acts as a guide for the spicules. With the two sexes in close apposition the amoeboid sperm are transferred from the cloaca of the male into the uterus of the female.

The **cuticle** may be modified to form various structures, the more important (Fig. 7) of which are:

Leaf crowns consisting of rows of papillae occurring as fringes round the rim of the buccal capsule (external leaf crowns) or just inside the rim (internal leaf crowns).

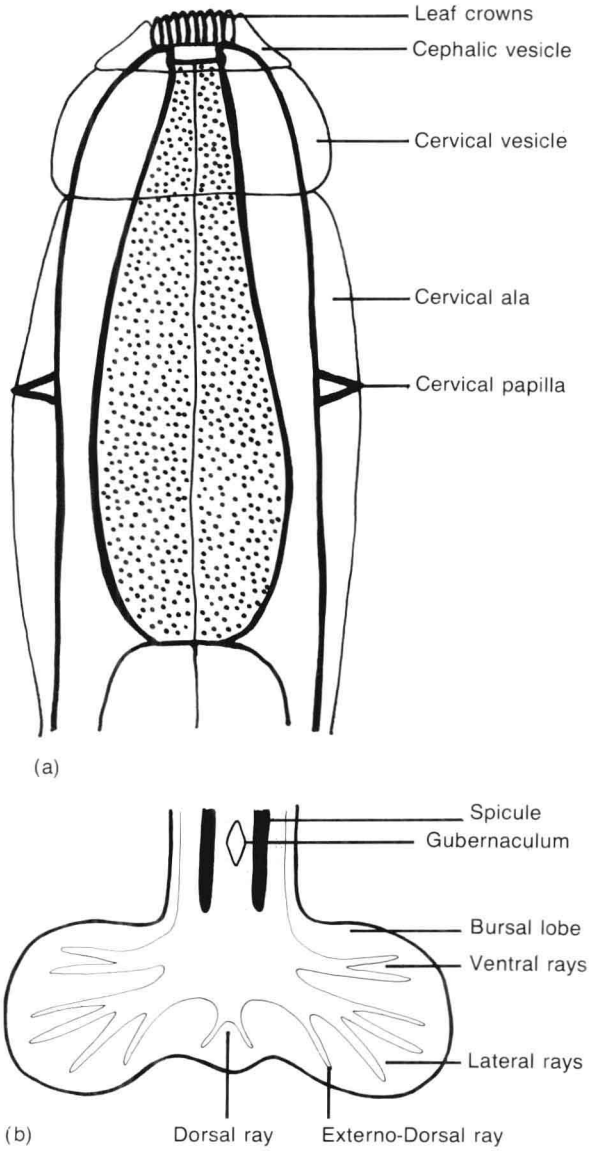


Fig 7 Nematode cuticular modifications.
(a) Anterior.
(b) Posterior of male.

They are especially prominent in certain nematodes of horses. Their function is not known, but it is suggested that they may be used to pin a patch of mucosa in position during feeding, or that they may prevent the entry of foreign matter into the buccal capsule when the worm has detached from the mucosa.

Cervical papillae occur anteriorly in the oesophageal region, and **caudal papillae** posteriorly at the tail. They are spine-like or finger-like processes, and are usually diametrically placed. Their function may be sensory or supportive.

Cervical and **caudal alae** are flattened wing-like expansions of the cuticle in the oesophageal and tail regions.

Cephalic and **cervical vesicles** are inflations of the cuticle around the mouth opening and in the oesophageal region.

The **copulatory bursa**, which embraces the female during copulation, is important in the identification of certain male nematodes and is derived from much expanded caudal alae which are supported by elongated caudal papillae called **bursal rays**. It consists of two lateral lobes and a single small dorsal lobe.

Plaques and **cordons** are plate-like and cord-like ornamentations present on the cuticle of many nematodes of the superfamily Spiruroidea.

BASIC LIFE CYCLE

In the Nematoda, the sexes are separate and the males are generally smaller than the females which lay eggs or larvae. During development, a nematode moults at intervals shedding its cuticle. In the complete life cycle there are four moults, the successive larval stages being designated L_1, L_2, L_3, L_4 and finally L_5, which is the immature adult.

One feature of the basic nematode life cycle is that immediate transfer of infection from one **final host** to another rarely occurs. Some development usually takes place either in the faecal pat or in a different species of animal, the **intermediate host**, before infection can take place.

In the common form of **direct** life cycle, the free-living larvae undergo two moults after hatching and infection is by ingestion of the free L_3. There are some important exceptions however, infection sometimes being by larval penetration of the skin or by ingestion of the egg containing a larva.

In **indirect** life cycles, the first two moults usually take place in an intermediate host and infection of the final host is either by ingestion of the intermediate host or by inoculation of the L_3 when the intermediate host, such as a blood sucking insect, feeds.

After infection, two further moults take place to produce the L_5 or immature adult parasite. Following copulation a further life cycle is initiated.

In the case of gastrointestinal parasites, development may take place entirely in the gut lumen or with only limited movement into the mucosa.

However, in many species, the larvae travel considerable distances through the body before settling in their final (predilection) site and this is the migratory form of life cycle. One of the most common routes is the **hepatic-tracheal**. This takes developing stages from the **gut** via the portal system to the **liver** then via the hepatic vein and posterior vena cava to the **heart** and from there via the pulmonary artery to the **lungs**. Larvae then travel via the bronchi, trachea and oesophagus to the **gut**. It should be emphasised that the above is a basic description of nematode life cycles and that there are many variations.

DEVELOPMENT OF THE PARASITE

EGG

Nematode eggs differ greatly in size and shape, and the shell is of variable thickness usually consisting of three layers.

The inner membrane, which is thin, has lipid characteristics and is impermeable. A middle layer which is tough and chitinous gives rigidity and, when thick, imparts a yellowish colour to the egg. In many species this layer is interrupted at one or both ends with an operculum (lid) or plug. The third outer layer consists of protein which is very thick and sticky in the ascaridoids and is important in the epidemiology of this superfamily.

In contrast, in some species the egg shell is very thin and may be merely present as a sheath around the larva.

The survival potential of the egg outside the body varies, but appears to be connected with the thickness of the shell, which protects the larva from desiccation. Thus parasites whose infective form is the larvated egg usually have very thick-shelled eggs which can survive for years on the ground.

HATCHING

Depending on the species, eggs may hatch outside the body or after ingestion.

Outside the body, hatching is controlled partly by factors such as temperature and moisture and partly by the larva itself. In the process of hatching, the inner impermeable shell membrane is broken down by enzymes secreted by the larva and by its own movement. The larva is then able to take up water from the environment and enlarges to rupture the remaining layers and escape.

When the larvated egg is the infective form, the host initiates hatching after ingestion by providing stimuli for the larva which then completes the process. It is important for each nematode species that hatching should occur in appropriate regions of the gut and hence the

stimuli will differ, although it appears that dissolved carbon dioxide is a constant essential.

LARVAL DEVELOPMENT AND SURVIVAL

Three of the important superfamilies, the trichostrongyloids, the strongyloids and the rhabditoids, have a completely free-living preparasitic phase. The first two larval stages usually feed on bacteria, but the L_3, sealed off from the environment by the retained cuticle of the L_2, cannot feed and must survive on the stored nutrients acquired in the early stages. Growth of the larva is interrupted during moulting by periods of lethargus in which it neither feeds nor moves.

The cuticle of the L_2 is retained as a sheath around the L_3; this is important in larval survival with a protective role analogous to that of the egg shell in egg-infective groups.

The two most important components of the external environment are temperature and humidity.

The optimal temperature for the development of the maximum number of larvae in the shortest feasible time is generally in the range 18–26 °C. At higher temperatures, development is faster and the larvae are hyperactive, thus depleting their lipid reserves. The mortality rate then rises, so that few will survive to L_3. As the temperature falls the process slows, and below 10 °C the development from egg to L_3 usually cannot take place. Below 5 °C movement and metabolism of L_3 is minimal, which in many species favours survival.

The optimal humidity is 100%, although some development can occur down to 80% relative humidity. It should be noted that even in dry weather where the ambient humidity is low, the microclimate in faeces or at the soil surface may be sufficiently humid to permit continuing larval development.

In the trichostrongyloids and strongyloids, the embryonated egg and the ensheathed L_3 are best equipped to survive in adverse conditions such as freezing or desiccation; in contrast, the L_1 and L_2 are particularly vulnerable. Although desiccation is generally considered to be the most lethal influence in larval survival, there is increasing evidence that by entering a state of anhydrobiosis, certain larvae can survive severe desiccation.

On the ground most larvae are active; although they require a film of water for movement and are stimulated by light and temperature, it is now thought that larval movement is mostly random and encounter with grass blades accidental.

INFECTION

As noted previously, infection may be by ingestion of the free-living L_3, and this occurs in the majority of trichostrongyloid and strongyloid nematodes. In these, the L_3 sheds the retained sheath of the L_2 within the alimentary tract of the host, the stimulus for exsheathment being provided by the host in a manner similar to the hatching stimulus required by egg-infective nematodes. In response to this stimulus the larva releases its own exsheathing fluid, containing an enzyme leucine aminopeptidase, which dissolves the sheath from within, either at a narrow collar anteriorly so that a cap detaches, or by splitting the sheath longitudinally. The larva can then wriggle free of the sheath.

As in the preparasitic stage, growth of the larva during parasitic development is interrupted by two moults, each of these occurring during a short period of lethargus.

The time taken for development from infection until mature adult parasites are producing eggs or larvae is known as the **prepatent period** and this is of known duration for each nematode species.

METABOLISM

The main food reserve of preparasitic nematode larvae, whether inside the egg shell or free-living, is lipid which may be seen as droplets in the lumen of the intestine; the infectivity of these stages is often related to the amount present, in that larvae which have depleted their reserves are not as infective as those which still retain quantities of lipid.

Apart from these reserves the free-living first and second stage larvae of most nematodes feed on bacteria. However, once they reach the infective third stage, they are sealed in the retained cuticle of the second stage, cannot feed and are completely dependent on their stored reserves.

In contrast, the adult parasite stores its energy as glycogen, mainly in the lateral cords and muscles, and this may constitute 20% of the dry weight of the worm.

Free-living and developing stages of nematodes usually have an aerobic metabolism whereas adult nematodes can metabolise carbohydrate by both glycolysis (anaerobic) and oxidative decarboxylation (aerobic). However, in the latter, pathways may operate which are not present in the host and it is at this level that some antiparasitic drugs operate.

The oxidation of carbohydrates requires the presence of an electron transport system which in most nematodes can operate aerobically down to oxygen tensions of 5.0 mm Hg or less. Since the oxygen tension at the mucosal surface of the intestine is around 20 mm Hg, nematodes in close proximity to the mucosa normally have sufficient oxygen for aerobic metabolism. Otherwise, if the nematode is temporarily or permanently some distance from the mucosal surface, energy metabolism is probably largely anaerobic.

As well as the conventional cytochrome and flavoprotein electron transport system, many nematodes have 'haemoglobin' in their body fluids which gives them a red pigmentation. This nematode haemoglobin is chemically similar to myoglobin and has the highest

affinity for oxygen of any known animal haemoglobin. The main function of nematode haemoglobin is thought to be to transport oxygen, acquired by diffusion through the cuticle or gut, into the tissues; blood-sucking worms presumably ingest a considerable amount of oxygenated nutrients in their diet.

The end products of the metabolism of carbohydrates, fats or proteins are excreted through the anus or cloaca, or by diffusion through the body wall. Ammonia, the terminal product of protein metabolism, must be excreted rapidly and diluted to non-toxic levels in the surrounding fluids. During periods of anaerobic carbohydrate metabolism, the worms may also excrete pyruvic acid rather than retaining it for future oxidation when aerobic metabolism is possible.

The 'excretory system' terminating in the excretory pore is almost certainly not concerned with excretion, but rather with osmoregulation and salt balance.

Two phenomena which affect the normal parasitic life cycle of nematodes and which are of considerable biological and epidemiological importance are **arrested larval development** and the **periparturient rise** in faecal egg counts.

ARRESTED LARVAL DEVELOPMENT

(Synonyms: inhibited larval development, **hypobiosis**).

This phenomenon may be defined as the temporary cessation in development of a nematode at a precise point in its parasitic development. It is usually a facultative characteristic and affects only a proportion of the worm population. Some strains of nematodes have a high propensity for arrested development while in others this is low.

Conclusive evidence for the occurrence of arrested larval development can only be obtained by examination of the worm population in the host. It is usually recognised by the presence of large numbers of larvae at the same stage of development in animals withheld from infection for a period longer than that required to reach that particular larval stage.

The nature of the stimulus for arrested development and for the subsequent maturation of the larvae is still a matter of debate. Although there are apparently different circumstances which initiate arrested larval development, most commonly the stimulus is an environmental one received by the free-living infective stages prior to ingestion by the host. It may be seen as a ruse by the parasite to avoid adverse climatic conditions for its progeny by remaining sexually immature in the host until more favourable conditions return. The name commonly applied to this seasonal arrestment is **hypobiosis**. Thus the accumulation of arrested larvae often coincides with the onset of cold autumn/winter conditions in the northern hemisphere, or very dry conditions in the subtropics or tropics. In contrast, the maturation of these larvae coincides with the return of environmental conditions suitable to their free-living development, although it is not clear what triggers the signal to mature and how it is transmitted.

The degree of adaptation to these seasonal stimuli and therefore the proportion of larvae which do become arrested seems to be a heritable trait and is affected by various factors including grazing systems and the degree of adversity in the environment. For example, in Canada where the winters are severe, most Trichostrongyloid larvae ingested in late autumn or winter become arrested, whereas in southern Britain with moderate winters, about 50–60% are arrested. In the humid tropics where free-living larval development is possible all the year round, relatively few become arrested.

However, arrested development may also occur as a result of both acquired and age immunity in the host and although the proportions of larvae arrested are not usually so high as in hypobiosis they can play an important part in the epidemiology of nematode infections. Maturation of these arrested larvae seems to be linked with the breeding cycle of the host and occurs at or around parturition.

The epidemiological importance of arrested larval development from whatever cause is that, first, it ensures the survival of the nematode during periods of adversity; secondly, the subsequent maturation of arrested larvae increases the contamination of the environment and can sometimes result in clinical disease.

PERIPARTURIENT RISE (PPR) IN FAECAL EGG COUNTS

(Synonyms: Post-parturient rise, Spring rise).

This refers to an increase in the numbers of nematode eggs in the faeces of animals around parturition. The phenomenon is most marked in ewes, sows and goats.

The etiology of this phenomenon has been principally studied in sheep and seems to result from a temporary relaxation in immunity associated with changes in the circulating levels of the lactogenic hormone, prolactin. It appears that a decrease in parasite-specific immune responses occurs following elevation of serum prolactin levels. These are rapidly restored when prolactin levels drop at the end of lactation or more abruptly if lambs are weaned early and the suckling stimulus removed.

The source of the periparturient rise (PPR) is threefold:

(i) Maturation of larvae arrested due to host immunity.
(ii) An increased establishment of infections acquired from the pastures and a reduced turnover of existing adult infections.
(iii) An increased fecundity of existing adult worm populations.

Contemporaneously, but not associated with the relaxation of host immunity, the PPR may be augmented by the maturation of hypobiotic larvae.

The importance of the PPR is that it occurs at a time when the numbers of new susceptible hosts are increasing and so ensures the survival and propagation of the worm species. Depending on the magnitude of infection, it may also cause a loss of production in lactating animals and by contamination of the environment lead to clinical disease in susceptible young stock.

Superfamily TRICHOSTRONGYLOIDEA

The trichostrongyloids are small, often hair-like, worms in the bursate group which, with the exception of the lungworm *Dictyocaulus*, parasitise the alimentary tract of animals and birds.

Structurally they have few cuticular appendages and the buccal capsule is vestigial. The males have a well developed bursa and two spicules, the configuration of which is used for species differentiation. The life cycle is direct and usually non-migratory and the ensheathed L_3 is the infective stage.

The trichostrongyloids, including *Dictyocaulus*, are responsible for considerable mortality and widespread morbidity, especially in ruminants. The most important alimentary genera are *Ostertagia*, *Haemonchus*, *Trichostrongylus*, *Cooperia*, *Nematodirus*, *Hyostrongylus*, *Marshallagia* and *Mecistocirrus*.

Ostertagia

This genus is the major cause of parasitic gastritis in ruminants in temperate areas of the world.

Hosts: Ruminants

Site: Abomasum

Species:

Ostertagia ostertagi	cattle
O. circumcincta	sheep and goats
O. trifurcata	sheep and goats

Minor species are *O.* (syn. *Skrjabinagia*) *lyrata* and *kolchida*, in cattle and *O. leptospicularis* in cattle, sheep and goats

Distribution:
Worldwide; *Ostertagia* is especially important in temperate climates and in subtropical regions with winter rainfall

IDENTIFICATION

The adults are slender reddish-brown worms up to 1.0 cm long, occurring on the surface of the abomasal mucosa and are only visible on close inspection. The larval stages occur in the gastric glands and can only be seen microscopically following processing of the gastric mucosa.

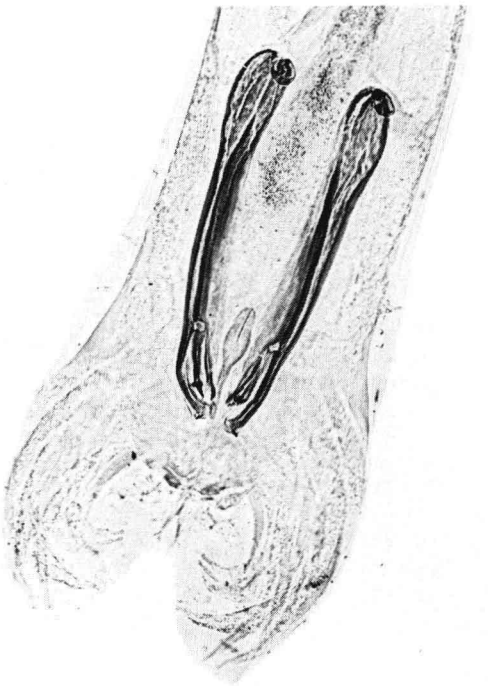
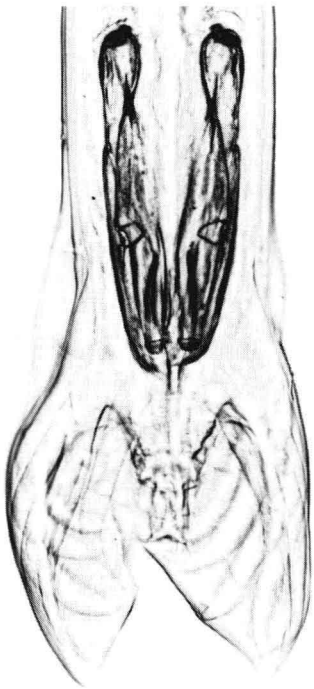


Fig 8 Structure of spicules from five *Ostertagia* species.
(a) *O. ostertagi*.



(b) *O. lyrata*.

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