COURSE CODE: **VPR 302**

COURSE TITLE: **INTRODUCTORY VETERINARY PARASITOLOGY**

NUMBER OF UNITS: **2UNITS**

COURSE DURATION: **1 HOUR (L), 3 HOURS (P)**

COURSE DETAILS:

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COURSE CONTENT:

**general characteristictics and taxonomy of helminthes, protozoa, and artropods of veterinary imporatnce. Economic importance of parasite of domestic animals and poultry. Host immune mechanism of parasite destruction. Principles of the diagnosis, treatment and prevention of parasitic diseases.**

COURSE REQUIREMENTS:

**Compulsory**

READING LIST:

1. **Ejl Soulsby, Helminths, Arthropods And Protozoa Of Domesticated Animals. 7th Edition. London,Uk Bailliere, Tindall 1986.**
2. **Urquhart, G.M., Armour J., Duncan J.L. ,Dun A.M., Jennnings F.W. Veterinary Parasitology 2nd Edition 2003.**
3. **Dwight D. Bowman, Georgis’ Parasitology For Veterinarians. 8th Edition 2003.**

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LECTURE NOTES

**PROTOZOANS**

**GENERAL CHARACTERISTICS OF PROTOZOANS**

The protozoa are unicellular animal in which the various activities of metabolism, locomotion, etc, are carried out by organelles of the cell, comparable forms occur in the plant kingdom (unicellular plants) and, in general, protozoa are differentiated from these by the absence of chlorophyll-containing chromatophores and their mode of nutrition (holozoic). The unicellular plants are frequently bounded by a fairly rigid cell wall made of cellulose and the nuclear material is often dispersed in the cell. The protozoa, on the other hand, have a well defined nucleus and do not have a rigid cell wall, allowing, at a times, a marked variation in size and shape. Never the less, this distinctions cannot be rigidly applied to all form and there is an assemblage of organisms which share the characters of both plants and animals. The term protista was introduced for such forms, but this has not been generally adopted.

Since the discovery of protozoa by Antoni van Leeuwenhock, some 45,000 species have been described. The majorities of these is free living and are found in almost every habitat on land and in water. Although the parasitic protozoa are smaller in numbers, they never the less assume an important role as producers of global disease which, apart from producing death or deformity, saps the energy and initiative and decays the world.

Of no less important is the untold loss of livestock and livestock products which is frequently a burden in those communities and areas of the world that can least support it.

***STRUCTURE OF PROTOZOA***

***Nucleus***

Protozoa are eukaryotic (nucleus enclose in a membrane) where as the bacteria are prokaryotic (nucleus disperse in cytoplasm).

Usually only one nucleus is present, although in some forms more than one nucleus may be present in some or all stages of development. The vesicular type of nucleus consist of a nuclear membrane which bounds the nucleoplasm in which, lying more or less central, is an intranuclear body, the endosome (or kayosome) or the nucleus. An endosome is devoid of deoxyribonucleic acid, where as a nucleolus possess DNA. Chromatin material frequently occurs in the inner surface of the nuclear membrane and many also be seen as strands radiating from the kayosome to the nuclear membrane. The vesicular type of nucleus is seen most commonly in the Mastigophora and the Sarcodina.

**Cytoplasm:** This is the extra nuclear part of the protozoan cell. It may be differentiated into an outer ectoplasm and an inner endoplasm, the former often being homogeneous and hyaline in appearance and the latter frequently containing granules, vacuoles and sometimes pigment. In some forms (e.g sarcodina) there is no definite limiting membrane, but usually a pellicle serves as such in the majority of species.

**Locomotion** Protozoa may move by gliding or by means of pseudopodia, flagella or cilia.

Pseudopodia are used by amoeba-like organisms, the structures being temporary locomotory organelles which are formed when required and retracted when not needed.

Flagella are whip-like filamentous structures which arise from a basal granules or blepharoplast in the cytoplasm of the organism. They are composed of a central axial filament, the axoneme, which is surrounded by a contractile cytoplasmic sheath. Ultra-structural studies indicate the axoneme to be composed of two central filaments surrounded by nine peripheral filaments. In some forms the flagellum may be attached to the body of the protozoan by an undulating membrane. Flagella are typically seen in the mastigophora.

Gliding is seen in Toxoplasma, Sarcocystis and other forms, this being achieved without the aid of cilia or flagella.

Cilia are fine, short, flagella-like structures originating from a basal granule embedded in the pellicle or ectoplasm. They are the organs of locomotion in the ciliates, but they may also aid the ingestion of food or serve as tactile structures. The ultra-structure is similar to that of the flagella and may usually occur in large numbers, arranged in rows over the body of the protozoan.

**ORGANELLES OF NUTRITION**

In the amoeba-like form, particulate food materials are acquired by means of pseudopodia. An advance on this is a specialized opening called the cytostome through which food particles are engulfed and passed to food vacuoles. In the ciliates the cytostome may be lined with cilia which further assist in the ingestion of food. Food vacuoles occur in the cytoplasm and contain particulate materials at various stages of digestion. Non-digestiable material may be extruded from the cell either via a temporary opening or through a permanent cytopyge.

Excretion of waste products may occur directly through the body wall or by means of contractile vacuoles which periodically discharge waste material through the body wall or, in a few instances, through an anal pore.

**Phylum Sarcomastigophora (the protozoa)**

* **Subphylum Mastigophora (the flagellates)**
  + **Class Zoomastigophorea**
    - **Order Retortamonadida**
      * ***Chilomastix mesnili* (a commensal)**
    - **Order Trichomonadida**
      * ***Dientamoeba fragilis***
      * ***Trichomonas vaginalis* (trichomoniasis)**
    - **Order Diplomonadida**
      * ***Giardia lamblia* (giardiasis)**
    - **Order Kinetoplastida**
    - **Family Trypanosomatidae**
      * ***Leishmania* spp. (leishmaniasis)**
      * ***Trypanosoma* spp. (African trypanosomiasis, "sleeping sickness")**
      * ***Trypanosoma cruzi* (American trypanosomiasis, Chagas' disease)**

**FamilyCRPTOBIIDAE**

***Cryptobia* sp.**

* **Subphylum Sarcodina (the amoebae)**
  + **Superclass Rhizopoda**
    - **Class Lobosea**
      * **Order Amoebida**
        + ***Endolimax nana* (a commensal)**
        + ***Entamoeba coli* (a commensal)**
        + ***Entamoeba histolytica* (amoebiasis, amoebic dysentery)**
        + ***Iodamoeba bütschlii* ( a commensal)**

**Phylum Apicomplexa**

* **Class Sporozoea**
  + **Subclass Gregarina**
    - **Order Eugregarinidia**
      * **Suborder Septatina**

***Gregarina* sp.**

* + **Subclass Coccidia**
    - **Order Eucoccidiorida**
      * **Suborder Eucoccidea**

***Hemogregarina* sp.**

* + - * **Suborder Eimeriorina**
        + **Family Eimeriina**

***Eimeria* spp. (coccidiosis)**

***Isospora* spp. (coccidiosis)**

**Isospora belli**

**Tyzzeria**

**Wenyonella**

* + - * + **Family Sarcocystidae**

***Sarcocystis* spp.**

***Toxoplasma gondii* (toxoplasmosis)**

* + - * + **Fa*mily Cryptosporidiidae***

***Cryptosporidium parvum* (cryptosporidosis)**

***Cyclospora cayetanesis***

***Pneumocystis carinii***

* + - * **Suborder Haemosporoina**

***Plasmodium* spp. (malaria)**

* + **Subclass Piroplasmasina**
    - * + **Family Babesiidae**

***Babesia bigemina* (babesiosis)**

**Phylum Myxozoa**

* **Class Myxosporea**
  + **Order Bivalvulida**
    - **Suborder Platysporina**
      * ***Myxobolus* (= *Myxosoma*) sp. ("whirling disease")**

**Phylum Ciliophora (the ciliates)**

* **Class Oligohymenophorea**
  + **Subclass Hymenostomatia**
    - **Order Hymenostomadia**
      * ***Ichthyophthirius multifiliis* ("ick")**
  + **Subclass Peritrichia**
    - **Order Sessilida**
      * ***Epistylis* sp.**
    - **Order Mobilida**
      * ***Trichodina* sp.**
* **Class Litostomatea**
  + - **Order Vestibuliferida**
      * ***Balantidium coli***

**ARTHROPODA**

**GENERAL CHARACTERISTICS OF ARTHROPODS**

The name of this phylum, derived from Greek word *arthros,* a joint, and*podos,*a foot, refers to the fact that the members of the phylum have jointed limbs. The primitive limb of arthropods was biramous, consisting of an unbranched basal piece, the *protopodite*, which branched into an inner *endopodite* and an outer exopodite. Some of the limbs of some species of arthropod are still of this type.

Arthropoda have probably descended from ancestors which also gave origin to the soft-skinned annelid worms, an example of which is the earth worm, but the arthropod have developed an outer covering of chitin, which forms an exo skeleton in which the whole body is enclosed. This chitinous covering is secreted by chitogenous cells beneath it and it not only covers the external surface of the body, but also passes through the mouth into the anterior part of the alimentary canal called protodaeum both of which arise as invaginations from the exterior into the body. The excoskeleton is usually present in the form of chitinous plates called sclerite, called a tergum, a ventral sclerite, called a sternum and a lateral plate between the tergum and sternum, which is called a pleuron. The tergum, sternum and pleuron of each segment are united by more flexible portions of the chitinous exoskeleton.

As the arthropod grows it becomes too big for its chitinous covering and periodically this is cast off and a new exoskeleton is formed. Each casting off of the exoskeleton is called **ecdysis.**

Arthropods are metamerically segmented animals. The segment of arthropods are associated in groups, the anterior segments forming the heads, the middle ones the thorax and the posterior ones the abdomen.

The appendages found on the body of an arthropod are typically paired, one pair usually being found on each segment. The appendages on the head are typically one or two pairs of sensory antennae and behind these paired appendages modified for feeding, commonly there is one pair of mandibles and behind these two pairs of maxillae .

Behind these again there may be **maxillipedes**, which are walking legs adapted for feeding. The next group of appendages belongs to the thorax and they are walking legs. Behind them, in aquatic species such as the crustacean, there are a variable number of abdominal appendages, some or all of which are use for swimming, terrestrial species usually loss these or some of them may become modified to perform other functions.

A dominant feature of the internal anatomy of the Arthropoda is the fact that the general body cavity is not a coelom. It is a space full of blood, which is called the haemocele. The blood in it bathes all the organs of the body. The heart pulsates; it sucks in blood from pericardium through openings in its walls called ostia. It then pumps the blood into the haemocele through short arteries which are usually the only blood vessels in the body.

The respiratory organs of arthropods are also characteristic of the phylum. They are:

1. Gills (branchiae) of various kinds found in larvae, nymphs and adults of species that are aquatic.
2. Tracheae, which are fine, elastic tubes, with a thin, chitinous lining which are held open by rings or spiral thickenings of the chitinous lining tracheae branch and ramify among the internal organs, to which they take air that enters them through their external openings or stigmata; trachae are especially characteristic of insects.

Other respiratory structures are lung-books and gill-books of spiders and crabs respectively. In some forms, e.g the parasitic mites, respiration is through the cuticle.

The alimentary canal varies in the different classes of arthropods. In all, however, it consist of (a) the stomodaeum mentioned above, which is lined by chitin and may be divided into a sucking phayrnx, a provetriculus (crop) and a gizzard, (b) the proctodaeum mentioned above, which is also line by chitin, (c) a mid-gut, or mesenteron, which connects the protodacum with stomodacum.

The excretory organs of arthropods vary in the different classes of the phylum. Those of the class Crustacea are a pair of nephridia which open on the bases of the second antennae. The excretory organs of the insecta are tubules, called malpighan tubules, which are arranged in a ring round the alimentary canal. Usually they open into the anterior end of the proctodaeum. Arachnida also have malpighian tubulethat open into the anterior end of the proctodaeum, but they have, in addition, coxal glands, which open on the coxae of the legs. These latter are true nepridia homologous with the nephridia of the crustacean.

The nervous system of arthropods consists of cerebral ganglia in the head, united circumoesophageal commissures to a ventral double nerve cord that runs along the ventral side of the body and has nerve ganglia on it. Typically, there is one ganglion in each segment, but fusions of segment carry with them fusions of the ganglia associated with them. Associated with this central nervous system are eyes, sensory setae and other special sense organs.  
The sexes of arthropods are usually separated.

**ARTHROPODA**

* **Subphylum Crustacea**
  + **Class Maxillopoda**
    - **Subclass Ostracoda**
    - **Subclass Copepoda**
      * **Order Cyclopoida**
        + [***Lernea* sp. ("anchor worms")**](http://www.biosci.ohio-state.edu/~parasite/lernea.html)
      * **Order Poecilostomatoida**
        + [***Ergasilus* sp.**](http://www.biosci.ohio-state.edu/~parasite/ergasilus.html)
    - **Subclass Branchiura**
      * **Order Argulidea**
        + [***Argulus* sp.**](http://www.biosci.ohio-state.edu/~parasite/argulus.html)
  + **Class Insecta**
    - * **Order Dictyoptera**
        + [***Periplaneta americana* (American cockroach)**](http://www.biosci.ohio-state.edu/~parasite/periplaneta.html)
      * **Order Anoplura**
        + [***Pediculus humanus* (body louse)**](http://www.biosci.ohio-state.edu/~parasite/lice.html)
        + [***Phthirus pubis* (pubic or crab louse)**](http://www.biosci.ohio-state.edu/~parasite/lice.html)
      * **Order Hemiptera**
        + [***Cimex* spp. (bedbugs)**](http://www.biosci.ohio-state.edu/~parasite/cimex.html)
        + [***Panstrongylus megistus***](http://www.biosci.ohio-state.edu/~parasite/panstrongylus.html)
        + [***Rhodnius prolixus***](http://www.biosci.ohio-state.edu/~parasite/rhodnius.html)
        + [***Triatoma* sp. (assassin bug)**](http://www.biosci.ohio-state.edu/~parasite/triatoma.html)
      * **Order Coleoptera**
        + [***Tenebrio molitor* (grain beetle)**](http://www.biosci.ohio-state.edu/~parasite/tenebrio.html)
        + [***Tribolium confusum* (flour beetle)**](http://www.biosci.ohio-state.edu/~parasite/tribolium.html)
      * **Order Siphonaptera**
        + [***Ctenocephalides* sp. (fleas)**](http://www.biosci.ohio-state.edu/~parasite/catflea.html)
        + [***Tunga penetrans* (sand flea)**](http://www.biosci.ohio-state.edu/~parasite/tunga.html)
      * **Order Diptera**
        + [***Anopheles* spp. (mosquito)**](http://www.biosci.ohio-state.edu/~parasite/anopheles.html)
        + [***Cuterebra* spp. (bot fly)**](http://botfly.ifas.ufl.edu)
        + [***Glossina* spp. (tsetse fly)**](http://www.biosci.ohio-state.edu/~parasite/glossina.html)
        + [***Tabanus* spp. (horse and deer flies)**](http://www.biosci.ohio-state.edu/~parasite/tabanus.html)
* **Subphylum Chelicerata**
  + **Class Arachnida**
    - **Order Acari**
      * **Suborder Metastigmata**
        + **Family Ixodidae (hard ticks)**

[***Amblyomma americanum* (lone star tick)**](http://www.biosci.ohio-state.edu/~parasite/amblyomma.html)

[***Boophilus microplus*(southern cattle tick)**](http://www.biosci.ohio-state.edu/~parasite/boophilus.html)

[***Dermacentor* spp. (dog tick)**](http://www.biosci.ohio-state.edu/~parasite/dermacentor.html)

[***Ixodes scapularis* (deer tick)**](http://www.biosci.ohio-state.edu/~parasite/ixodes.html)

* + - * + **Family Argasidae (soft ticks)**

[***Ornithodorus turicata* (relapsing fever tick)**](http://www.biosci.ohio-state.edu/~parasite/ornithodorus.html)

* + - * **Suborder Astigmata**
        + [***Notoedres cati* (face mange)**](http://www.biosci.ohio-state.edu/~parasite/notoedres.html)
        + [***Sarcoptes scabiei***](http://www.biosci.ohio-state.edu/~parasite/sarcoptes.html)

**Suborder Prostigmata**

* + - * + [***Demodex* spp. (follicle mites, demodetic mange)**](http://www.biosci.ohio-state.edu/~parasite/demodex.html)

**. Astigmata**   
**.** **Sarcoptidae**   
**-** [*Sarcoptes scabiei*](http://icb.usp.br/~marcelcp/Sarcoptes.htm) **(itch mite)**   
**-** [*Notoedres cati*](http://icb.usp.br/~marcelcp/Notoedres.htm) **(mange mite)**   
**-** [*Knemidocoptes*](http://icb.usp.br/~marcelcp/Knemidocoptes.htm)spp **(scaly mite,depluming mite)**   
**. Psoroptidae**   
**-** [*Psoroptes*](http://icb.usp.br/~marcelcp/Psoroptes.htm)spp **(scab mite)**   
**-** [*Otodectes cynotis*](http://icb.usp.br/~marcelcp/Otodectes.htm) **(ear mite)**   
**-** [*Chorioptes bovis*](http://icb.usp.br/~marcelcp/Chorioptes.htm) **(chorioptic mange mite)**   
**. Listrophoridae**   
**-** [*Lynxacarus radovskyi*](http://icb.usp.br/~marcelcp/Lynxacarus.htm) **(cat fur mite)**   
**. Analgidae** **(feather mites)**   
**-** [*Megninia ginglymura*](http://icb.usp.br/~marcelcp/megninia.htm)

*Economic importance of Parasites of domestic animals and poultry*

Overt manifestations of the effects of parasitism may take a number of forms and almost invariably result in loss associated with product or with productivity. The most drastic form that this can take is, of course, where parasitism results in death of the affected animals. Most severe forms of parasitic disease can produce this effect. For example, Theileriosis or East Coast fever in newly infected susceptible cattle frequently results in heavy mortality in the affected animals. These effects are often most pronounced in younger animals because of lack of resistance. This lack of resistance may be immunological since in many instances the age at which the young are affected is too early in their development for the immune system to mount an adequate response. In other cases the physical attributes of the younger animals, because of requirements for growth and development, do not allow for the additional stresses impose by heavy parasitism and this results in death. Parasitic organisms other than protozoans can also cause death. Many of the nematode parasites, like *Haemochus contortus*, particularly if there is overcrowding leading to extensive build-up of the infective stages, will produce fulminating outbreaks of disease with heavy mortality.

In some instances, there may be interaction between mortality due to parasitism and other facets of productivity. Thus, the effects of parasitism actually represent a complex interplay of a variety of factors. Such as age of animal movement of animals, treatment as well as weather, all influence the size of worm burdens that directly affect the productivity of animals.

Of the ectoparasites of livestock, ticks cause the greatest economic losses. These effects include the production of toxins and loss of blood, their role as vectors of protozoan, nutritional, bacterial and viral diseases and a variety of nuisance, irritative and anorectic effects as well as their predisposing of affected animals to other diseases like screw-worm infestation (myasis). On a global basis, their economic toll is staggering. They transmit such major diseases as babesiosis, anaplasmosis, East Coast fever (therleriosis) and heart water (Cowdriosis).

It has been estimated that every tick completing it life cycle on an animal consumes 1 – 3ml of blood.

Animal fibre production can be adversely affected by parasitism by some of the trichostrongyle worms like *Trichostrongylus colubriformis* and trematodes. Sheep infected with ectoparasites like Psoroptes ovis show broken and light weight fleeces because of the effects of intense pruritis. This is reflected adversely in the quality of the product from animals but additionally can result in increased stress on the animal under conditions of environmental adversity such as cold.

Because of the debilitating nature of many forms of parasitic diseases in animals, there may be a number of indirect deleterious effect on reproduction. Lowered rates of fecundity as a result of poor condition leads to reduction in the number of offspring being produced by these animals. Lesions caused by the chorioptes mites that extended over one third of the scrotum of rams lowered the quality of semen similar to that noticed when testicular temperature was raised and could thus interfere with fertility. Some parasites like *Trichomonas fetus* by causing abortion and chronic sterility, can severely compromise breeding programs. They not only cause loss of product by abortion but result in lengthening of the breeding intervals, through fertility, with the attendant economic loss.

Because of the effects on breeding programs, parasitism may make it impossible for the livestock producer to maintain either the desired age structure or rate genetic gain through selection. Parasitism may distort the value of some parameters used for genetic selection and thereby influence selection process.

Additionally, parasitic infestation can cause economic losses by adversely affecting the quality of whole carcasses, or parts thereof, may be condemned as unfit for human consumption. Thus there is condemnation of livers of sheep and cattle, because of the presence of flukes, and potions of meat, because of the occurrence of the cysts of *Taenia saginata* or *Taenia solium*. Parasitism may also influence the product characteristics, including composition, so that the consumer obtains less value from its use. Hides may be severely damaged by the activities of larval flies like Hypoderma bovis, or by mites like Demodex folliculorum necessitating down grading of the product for leather manufacture.

Immeasurable losses may result by consumer knowledge concerning the potential presence of a parasite in product, thus discouraging its consumption and lowering the market price. Such an example would be the suspected presence of Trichinella in pork.

This is also the matter of public health considerations. Diseases like trichinosis, cysticercosis and toxoplasmosis are readily transmissible to man by consumption of inadequately cooked meat. Because of the actual or potential presence of some of these agents in various animal products, important international economic sanctions may be imposed on the transfer of these products from one country to another. The cost of monitoring by various regional and national agencies concerned with quality of meat, milk and other animal products can impose a substantial economic burden. Coupled with these are, of course, all those additional costs imposed on the individual producer as well as the whole livestock industry of a country in the farm of medication cost, veterinary fees, lost labour, lost feed and other management practices aimed at reducing level of infection, minimizing the effect of parasitism and controlling the causative organisms, their vectors and intermediate host.

*HOST IMMUNE MECHANISMS OF PARASITE DESTRUCTION.*

***Immune Response***

The immune response to parasitic invaders is in principle based on the same components as the response to other infectious agents. The relative importance of the single components, the interaction between them, and the resulting effector mechanisms are highly variable, depending both on the species of parasite and host.

***Mechanism of Immunity***

The immune system exhibits a range of responses involving humoral factors, immunoglobulin, and sensitized calls of the lymphoid system. The immunoglobulin which posses antibody activity comprise distinct classes of which IgG, IgM, IgA and IgE are the most important, IgG is the predominant class circulating in the blood stream. IgA may be involved in local immune response through its capacity to cross-epithelial surfaces of e.g. the gut, the bronchi or the mammary gland. IgE may be associated with cells in immediate hypersensitivity reactions. Also additional humoral factors like complement are involved in many immune responses. The cells may be lymphocytes derived from stem cells in the bone marrow. There are two major categories of these cells, namely; the B-cell lymphocytes which give rise to plasma cells whose main function is the production of immunoglobulin and the T-cell which develop under the influence of thymus, and transform into cell populations such as the so-called helper and suppressor cells. There is a close and complex cooperation between the B-and T-cell systems and other cells like macrophages, mast cells, and polymorphonuclear leucocytes. One cell type within great number in helminth infection especially where the association of parasite with host tissue is close. Several arms of the immune response must interact before the animal can effectively control its parasites. Thus, simple neutralization by antibody, which may operate against bacteria and viruses, is usually incapable of affecting parasite. Composite effector mechanism like antibody mediated cell lysis by lymphocytes may be effective although much more complex reactions are usually required. Immediate hypersensitivity reactions are often encountered in the local environment of the parasite in that mast cells become sensitized with antibodies of the IgE class.

On contact with a parasite antigen, the mast cell releases so called vasoactive amines, which cause smooth muscle concentrations and increased permeability of blood vessels. Evidence suggests that this may demolish parasites either because of physiological changes of their environment or because they come in closer contact with antibody and other defense factors of the host. Other parasites induced tissue lesions, with or without an immune background may also create unfavourbale conditions. This it is believed that parasites may be trapped and immobilized by granulomas and connective tissue formations.

***Manifestation of Immunity on the Parasite Population***

Whereas immunity to bacteria or viruses is often able to protect the animal from initial attack or to destroy the invaders – this is not characteristic for immunity to parasites. There immunity develops slowly, and seldom cause total elimination of the parasite population.

In helminth infections immunity may affect parasites in at least three characteristic situations:

i. The parasite population or sub-population may be markedly reduced

either because it is trapped and killed in the tissues or because it is expelled from the host, e.g. via the gastro-intestinal tract. In domesticated animals, probably the best known of all expulsion phenomena is the so-called self-sure reaction described in haemonchosis in sheep. The major responsible factor for this phenomenon may be local hypersensitivity reaction of the immediate type triggered off by the antigenic stimulus provided by the introduction of infective larvae, although it has been indicated that under certain conditions it may be induced by dietary factors.

This pattern of expulsion may also operate in other helminth infections like dictyocaulosis and ostertagiasis of ruminant. In these infections, immunity is associated with a terminal exponential expulsion of adult worms.

ii. A number of parasite species may become arrested in their larval

development as a result of acquire immunity. Larvae may also become temporarily arrested due to prior experience of certain climatic and environmental factors. This process referred to as hypobiosis is comparable with the diapause phenomenon encountered in insect physiology.

iii. Reduction of size and egg-laying capacity of adult worms observed

in chronic infections may be a result of acquire immunity.

In protozoan infections, acquire immunity may exert the influence in a number of ways including agglutination, immobilization, lysis or phagocytosis, and reproduction/multiplication may be inhibited.

In both helminth and protozoan infections, a complete elimination of the parasite population may not always be entirely beneficial. Long-term protection by the continuing stimulation of the immune apparatus may acquire persistence of at least a few parasites, a state that is usually designate premunity or concomitant immunity.

It is evident that the capability of the host to acquire immunity to a given parasite plays an important direct role in limiting the incidence and severity of clinical disease.

***Evasion of the Immune Response***

Many parasites appear not to elicit or not to be affected by their hosts' immune response. Any parasite which can survive in its mammalian host for appreciably more than nine days must be assumed to have some mechanism for avoiding or mitigating their hosts' immune response. The evasion strategies can include: surface absorption of host antigen, molecular mimicry, loss or masking of surface antigens, antigenic variation, the occupation of immunologically incompetent sites and immunosuppression. There is no evidence for specific immunological tolerance.

Invertebrates have innate, but not aquired immune responses, invertebrates have no 'immunological memory'. However, the evasion strategies used by parasites of invertebrates show many parallels with those used by the parasites of mammals.

There is a complex interaction between parasites and their hosts' immune system and parasites may provide unique systems in which to study immune responses.

Many parasites may survive for long periods in immuno-competent hosts. A number of mechanisms have been recognized which may explain how the parasite avoid the potential lethal consequences of the host response. Three relative well-described mechanisms are;

i. Parasites may be shield from interaction with immune factors in

special anatomical sites or by intracapsular or intracellular locations, e.g. it is believed that the blood protozoa *Trypanosoma brucei* may be secluded from the host reaction, when it is localized in the central nervous system, and *Toxoplasma gondi*, another protozoa protected with host cells.

ii. The parasite may avoid the host’s immune response by shifting

antigenic structure. This is a prominent feature of trypanosomiasis and it helps to explain why the immune response is more or less in capable of controlling the disease. Trypanosomes are apparently able to synthesize an unlimited number of variant antigens – at intervals of a few days. The immune response (although effective) occurs invariably too late to affect the parasite because it has altered its antigenicity.

iii. The parasite may present surface which the host cannot detect or

attack. In chronic schistomiasis, concomitant immunity is a characteristic feature.

Adult worms persist unaffected in hosts which may be highly resistant to re-infection. The parasite apparently acquires a coating of host antigens on its surface preventing the host from identifying it as non-self. Alternatively, the parasite itself may produce antigens indistinguishable from those of the host, it is also possible that the surface layer (tegument) turns over so rapidly that adhering antibodies or cells are discarded. Immuno suppressive effect exerted by certain protozoan e.g. trypanosomes may be another important factor for the parasite to avoid host reactions.

# *NEONATAL UNRESPONSIVENESS*

The capacity of neonates to respond immunologically to infection varies with the species of animal and with the kind of antigen to which it is exposed. Among domestic animals, sheep fail to develop significant resistance to helminth infection until they are several months old. This has important implications in the field because the animals may be exposed to high levels of infection during the vulnerable period. The phenomenone has been described for *Haemonchus contortus* and *Trichostrongylus colubriformis* and it has been demonstrated in neonatal cattle affect with eggs of the Cestode *Taenia saginata*.

### D  Pathology of Helminth Infections:

In terms of pathology both adult and larval helminths may cause pathology and disease. An important difference between infection with parasitic helminths, and infection with bacterial, viral or protozoan parasites is that, in most cases, the parasites do not increase in numbers within their hosts, (exceptions to this general rule may however be found with larval helminths, or some nematodes such as *Strongyloides* sp.). That is, each larval helminth that infects the definitive host will give rise to only one adult parasite. Therefore, as pathology due to helminth infection is usually density dependent, (i.e. only with high worm burdens is severe pathology present) this parasite density, and therefore degree of pathology, is governed by the rate at which larval parasites enter the definitive host. This aspect of these diseases has important implications for the control of helminth parasites in that the diseases that they cause may be reduced or even eliminated by control measures that do not completely eliminate the parasite. This is the basis for control of many trichostrongylid nematodes of veterinary importance, where it is impractical to completely eradicate the parasite, but the disease caused by the parasite may be eliminated by controlled use of drugs at strategic times of the year. This is not the case with parasites that can divide asexually in their hosts such as bacterial, viral or protozoan parasites, where, for example, a single malaria parasite is capable, (at least in theory), of causing a fatal illness.  
In terms of veterinary importance the strongylid nematodes are of greatest economic importance. As has been said above, larval helminth infections in their intermediate hosts may also be important disease organisms, for example hydatid disease in man and domesticated animals (caused by *Echinococcus granulosus* infection), or hyper infections of *Trichinella spiralis* L3 larvae in their host's muscle tissues. With viviparous helminths larvae may also cause problems in the definitive host. The most important example of this is with river blindness, due to *Onchocerca volvulus* infection, with microfilaria (a pre larval L1 stage) migrating through cutaneous tissues (causing skin pathology) and the eye (eventually causing blindness). Pathology with infection of adult helminths may be due to a number of reasons, including:

P  Immune responses to adult, larval or eggs stages of the lifecycle (for example with the schistosomes).   
  
P  High densities of adult parasites feeding on host tissues, causing tissue damage (for example many of the digenean flukes), or obstruction of the gut, (as may be the case with *Ascaris* infections) or lymphatic drainage (as seen with lymphatic filariasis, although in this case this is a gross simplification of what happens).   
  
P  Depletion of nutrients or other metabolites required by the host (for example vitamin B12 depletion, leading to pernicious anaemia with infection by *Diphylobobrium latum*)   
  
P  Parasites feeding on blood, causing anaemia (for example infection with hookworms)   
  
P  Other reasons

Other parasitic infections however, where the association is much closer, are generally less pathogenic, extreme pathology generally only being associated with high parasite loads. That both parasite and host undergo this evolution may be seen with the association between the protozoan parasite *Trypanosoma brucei brucei*, which in its natural hosts (wild animals such as Zebra and Antelope) do not cause disease. Domesticated animals such as cattle or horses (which have not undergone this co-evolution) when introduced into trypanosome endemic areas are rapidly killed by the same parasites however. The reason for this co-evolution is probably because it is not in the interest of the parasite to kill its host, and the parasite may have evolved ways of reducing any pathogenic effects it may have. This growing association may even go to the extent of the parasites limiting their own densities of infection, (for example the concomitant immunity reported with schistosome infections).   
There are however important exceptions to this, particularly for example with infection of intermediate hosts with larval helminth parasites. Here it is often the case that the intermediate host must be eaten by its definitive host to complete its lifecycle. If this is the case, the larval parasites have often evolved to facilitate this, either actively or passively. Examples of parasites actively aiding the predation of their intermediate hosts have been reported from a number of species of helminths that modify the behaviour of these intermediate hosts. For example metacercarial infections of ants by *Dicrocoelium dendriticum* causes the ants to change their behaviour, by running up, and attaching themselves, by their jaws, to the tops of blades of grass, where they can be accidentally ingested as their herbivorous definitive hosts graze. Other behavioural modifications include the intermediate host not hiding itself from predators, as has been reported in fish infected with larval pseudophyllidean cestodes (e.g. *Schistocephalus solidus*), or arthropod intermediate hosts infected with larval acanthocephalans. Other more passive means include infection with many larval cyclophyllidean cestodes, where the larval cestode may, as it develops to maturity, cause extreme pathology (for example infection with species of *Echinococcus*), which will eventually kill the intermediate host. The dead intermediate host is then available for scavenging by the carnivorous definitive hosts, who then become infected, completing the parasites lifecycle.   
Larval helminths in accidental or paratenic hosts may cause pathology, two types of condition being important. Firstly **Visceral Larval Migrans**, as seen with *Anisakis* and *Angiostrongylus* infections, and importantly with the larvae of the nematode *Toxocara canis*, where migrating larvae may causes blindness in infected humans (usually children). Here larvae migrate deep within the paratenic hosts tissues. Secondly **Cutaneous Larval Migrans**, where the larvae migrate through the skin and subcutaneous tissues. Examples here include dog hookworms of the genus *Ancylostoma*. Some larval helminths may cause both conditions in paratenic hosts, such as is the case with plerocercoids of Pseudophyllidean Cestodes such as *Spirometra*, where the condition is known as sparganosis.

**THE PRINCIPLE OF DIAGNOSIS, TREATMENT AND PREVENTION**

## Immunodiagnosis of Parasitic Infection

The available immunodiagnostic procedures in parasitism comprises serodiagnostic test base on detection of specific antibody in the serum.

## Serodiagnosis

The humoral immune response to parasitic infection involves the occurrence of varying levels of specific antibodies in the blood stream. Description of fluctuation in antibody titre (and immunoglobin levels) has for many years been included in studies on immunology of parasite/host relationship. The importance of serology lies in its diagnostic capacity. Routine diagnosis usually depends on direct identification of the parasite, but there is a number of infections where parasite stages are not excreted with faeces or urine or released to the blood stream, and therefore not directly identifiable by isolation and microscopy. This applies to infections like trichinelosis, hydatid disease, toxoplasmosis etc., where indirect method such as serology are needed. Serology may be relevant in other infections because direct demonstration of the parasite may be tedious and time-consuming. In addition, direct and indirect approached may be complementary. Serology may, for instance, be applied during prepatent, hypobiotic - or other non-reproductive phase of helminth infections. Serodiagnosis may also be helpful in many haemoprotozoan diseases where parasitaemia is restricted to certain phases of the infection.

The qualification of a serological method in animal parasitology is determined by its degree of sensitivity, specificity and reproducibility, weighed against cost and capacity for routine applications. Methods in use include the Complement Fixation Test (CFT), the Indirect Haemagglutination Test (IHT), Indirect Flourescent antibody Test (IFT) and the Enzyme Linked Immunosorbent Assay (ELISA). Whatever the test may be, it’s major limitation seems to be specificity and to a lesser extent sensitivity. Parasite antigens are very complex mixtures and no serological test can be more specific than the antigen used.

***Examination of the Body***

The body is searched for external parasites or their eggs (bots, oxyurids), not only the surface but also in the ears and in the conjunctival sac (eye worm), and the skin should be palpated to determine the presence of subcutaneous larvae. If mange like lesions are present, the hair round the affected area should be clipped and scrapings made with a scalpel, the blade being held at such angle that the material scraped away falls into a piece of card or paper or a microscope slide held underneath. A little oil on the blade used will cause the material to adhere tot he blade, so that it is not lost. Scraping should continue until suspected. The lesion should then be dressed and the material examine for the presence of mites or of fragments of them. Some material may be examine directly, either in water or saline or in light oil, e.g. clove oil. It is too dense for direct examination, it should be brought just to the boil in 10% caustic soda or caustic potash to break it up. It may then be examined in the hydroxide used, but it is better to centrifuge it lightly and examine the sediment. It may be possible to find mites in the external auditory canal by rotating a cotton-wood swab in this canal. A little oil on the swab will help to capture the mites. Examination with an illuminated auriscope may be useful.

***Examination of Excretions***

Excretions of the body may contain parasite eggs or larvae. The nasal discharge and the sputum may, therefore, aid in the diagnosis of parasites in the air-passages, the vomit may bear evidence of parasites in the stomach, and the urine may contain eggs which can be concentrated by centrifuging. The faeces are by far the most important, as eggs or larvae of gastro-intestinal parasites and may others leave the body in the faeces. It should be remembered, however, that no eggs may be found if the worms are still immature of it only male are present.

***Faeces***

Faeces are examined in the first place for adult parasites, larval stages insect (e.g. bots) or segments of tapeworm. If a tapeworm infection is suspected, a purgative may be given to cause the expulsion of segments in case they are not readily found.

Birds that have caeca, as the domestic fowl for example pass two kinds of faeces, those from the small intestine being relatively coarse and loose with particles of varying colour, while those from the caeca are of a fine, pasty nature with a homogenous brown or brownish-green colour. The eggs of small intestine worms will be found in both types of faeces, while those of caecal worms are found only in the caecal faeces.

***Blood Examination of Larvae***

The microfilariae of most filarial worms are found in the blood and the diagnosis of filariasis is made by finding them in blood examination.

1. A drop of fresh blood is placed on a slide, covered with a cover-slip and examined immediately. The microfilariae will be seen moving about. This method can be carried out as a preliminary, but is not suitable for a specific identification.
2. If the larvae are abundant, a thin smear can be made; if they

are rare; a thick film is made and this gives better results in the majority of cases. The films are completely air-dried as quickly as possible and the thick film is them placed into vessels of distilled water in a slanting position and facing downwards, until it has been completely dehaemoglobinished. It is then air-dried again, fixed in methyl alcohol for ten minutes and then stained.

1. When the microfilariae are rare concentration techniques such

as the knot technique are applicable.

***DIAGNOSTIC METHODS IN PROTOZOLOGY***

***Faeces or Intestinal Contents***

Where motile organisms are to be searched for (e.g. Hexamita, trichomonas etc), material should be examine as soon as possible since the organisms lose their motility in cold material. The preparation should be kept warm until examination and a simple smear is made, mixing the specimen with warm saline. Direct examination is useful for the detection of motile organism, and the use of a phase contrast or dark field microscope greatly facilitate this.

Intestinal contents or faeces may need to be stained for more accurate identification of intestinal protozoa. Cover glass preparations are the most suitable and these should be coated with albumen fixative to facilitate adhere of faeces etc to the glass. A thin layer of faeces or intestinal contents is spread on the cover glass, and this is fixed before it is allowed to drip. The most satisfactory fixative is Schadinn's fluid and cover glasses should be gently dropped into this or floated on it, smear down, and left for about 10 - 15 minutes.

***Blood or Tissue Fluids***

Wet blood smear can be examined for living trypanosomes, the search for organisms been greatly facilitated by phase contrast or dark field illumination. Blood smears should be stained by one of the Romanowsly stains (methylene blue-eosin combination). Where organisms are plentiful, a thin blood smear is satisfactory examination.

Slide should be absolutely clean and the blood smear should be absolutely clean and the blood smear should be spread evenly and thinly. Thick blood smears cannot be used with avian or camel blood because of the nucleated erythrocytes. Thick blood smears need to be dehaemoglobinized before staining and this may be done by placing them in distilled water until the colour has disappeared. This smear or dehaemoglobinized smears are fixed in absolute methanol and may then be stained by Leishman, Giemsa, Wright's or Field's stain.

***Tissue***

Usually, the most satisfactory method of examination of tissue is for sections to be cut and stained with haematoxylin and eosin, Giemsa or other appropriate stain. Frozen sections provide a rapid means of examination. A diagnosis may often be made by mixing a scraping, or a small sample, of the tissue with a little saline and examining the preparation in the fresh state (e.g. schizonts of coccidia, toxoplasma, pseudocysts, sarcosporidia etc). Such preparation may also be stained to achieve a more critical examination.

**TREATMENT AND CONTROL OF PARASITES**

***Arthropods***

Although ticks/insects are in themselves important parasites, and should be combated for this reason, control measures are, as a rule, directed against the disease of which the ticks are the vectors and therefore based on the epizootiology of these diseases as well as on the habits of the ticks/insects.

Because ticks attach to various parts of the bodies of animals, treatment has to be applied to the whole body and may be carried out by dipping the animals in a suitable tank containing the dip in an aqueous solution, suspension or emulsion, however, spray races, showers etc are replacing conventional plunge dips since they are labor-saving and economical. In some cases ticks attached to the legs and under-sides of the bodies of animals and consequently shallow dips, through which hosts are made to walk, may be sufficient to give control.

These modern forms of apparatus drain the dipping fluid and filter it after it has been sprayed on the animals and return it for use again.The various stages of ticks may stay on their host for only a few days during each year and are often on the hosts only at certain times of the year.

Dipping for control of ticks/insects is therefore planned with knowledge of the biology of each species of tick, the duration of each of its stages and its feeding times and the duration of the whole life-history. An important consideration is whether the tick is one-host tick, a two-host species which uses one individual host for the larvae tick, each stages of which require a separate individual host. The one-host tick is obviously much easier to control than the other. Acaricides/Insecticides may act differently on the different stages of the life-cycle.

Other measure which have more limited value, are useful more specially against two-and three-host ticks, which spend relatively long period of their lives off the host and on the pastures. These methods include:

1. Burning of pasture, this may kill large number of the larvae and other stages, especially if it is done during the times of the year when these stages may be expected to be off the hosts.
2. Cultivation of land: this undoubtedly tend to reduce tick life by controlling the movement of domestic and wild animals, as well as by creating conditions unsuitable for ticks, for instance exposing of eggs to sunlight, or burying them decreasing the humidity on which the tick depend.
3. Repellant may be useful in certain circumstances

Tick control on your pet

Keeping pets out of grasses and woods helps to reduce their exposure to ticks. But any animal outside can quite easily have a tick crawl on board. Products that kill and repel ticks are needed.

Once-a-month Topicals: Once-a-month topical insecticides which are applied to the back of the pet, are probably the easiest product to use, and generally, last the longest. Some kill fleas and ticks, and others just fleas, so check the label carefully. Ingredients generally include permethrin, pyrethrin, imidacloprid, or fipronil.

Sprays: Flea and tick control sprays can come as aerosols or pump bottles. When using a spray, you do not have to soak the pet with the spray, but be sure to spray all parts of the animal. Spray a small amount on a cotton ball to apply the product around the eyes and ears. Do not get any of these products in the eyes.

Powders: Powders are generally easy to apply but can create a mess. If you or your pet has asthma, powders may not be the best choice of product since the powder could be inhaled. Be sure to use powders in well-ventilated areas. Powders often contain pyrethrin.

Dips: Dips and rinses are applied to the entire animal. They generally have some residual activity. It is helpful to put cotton balls in the pet's ears and ophthalmic ointment in the pet's eyes. Even with these precautions, be very careful not to get any of the product in the pet's ears or eyes. Dips and rinses may contain permethrin, pyrethrin, or organophosphates.

Shampoos: Shampoos help to primarily rid the pet of the ticks it already has on it, although some have residual activity. To properly use a flea & tick shampoo you must be sure to work the shampoo in over the entire body and then leave it on at least 10 minutes before you rinse it off. This is true of almost any medicated shampoo. Again, remember to protect the eyes and ears of the pet.

Collars: Collars can be effective, but must be applied properly. To get the right degree of snugness, you should just be able to get two fingers between the collar and the neck of your pet. Be sure to cut off any excess portion of the collar after you have properly applied it. Otherwise, that animal or other pets may try to chew on the end.

***Helminth***

Differences in epidemiology under different climatic conditions require different approaches for control. Control based on management incorporates the knowledge of the life-cycle, larvae ecology and epidemiology of the parasites. It is influenced by grazing management provision of clean pastures alternate by immunologically resistant hosts of the same species, stocking rate, and timing of reproductive events. Control based on management factor will be aided by the strategic use of anthelmintic. Alternatively, particularly where permanent pastures are utilized, formers may rely solely on anthelmintics for control, treatments being given as often as every three to four weeks, but this is likely to be uneconomical.Intermediate host of the trematode could be removed to break the life cycle of the parasites.

The principle of a parasite control is to keep the challenge to young livestock by the pathogenic parasites at a minimum rate. This is achieved in the following ways.

(a) Controlling the density of livestock (stocking rate). Overstocking forces the animals to graze closer to faecal material and closer to the ground, and may result in the consumption of a higher number of infective larvae.

(b) Periodic deworming.

(c) Strategic deworming when conditions are most favorable for larval development on the pasture.

(d) Separating age groups in the more intensive production systems.

(e) Reducing the effects of gastro-intestinal parasites by ensuring an adequate plane of nutrition.

(f) Using grazing management to minimize the uptake of infective larvae and to create safe pastures.

The development of such programme requires a thorough knowledge of the types of parasites present (including their biology and epidemiology), herd structure and grazing management, parasite seasonal availability and survival and the weather conditions in particular areas.

**Control of gastro-intestinal nematodes**

The ideal approach is an integration of:

· adjusting stocking rate  
· optimum use of safe pastures  
· strategic use of anthelmintics  
· use of resistant breeds or genotypes

Overstocking is a major problem in large parts of the world particularly in Africa outside the tsetse-infested areas. In addition to contributing to pasture degradation and soil erosion in certain marginal areas, it also forces the animal to graze closer to faecal material which inevitably results in the uptake of higher number of infective larvae. Reducing the stocking rate can significantly reduce the parasite burden of grazing livestock.

Improving grazing management and introducing the safe pasture concept can reduce the use of anthelmintics, minimizing the risk of developing anthelmintic resistance. Ungrazed pastures are parasitologically safe at the end of a prolonged period of dry weather (10 weeks or more). Other types of safe pasture are those used for hay/silage production and those previously grazed by other species. In some countries safe pastures are created by letting cattle graze pasture first, and following with sheep/goats. Grazing different species of livestock together may reduce the overall parasite burden of the species in question but this will not usually be sufficient for efficient parasite control. Fields of harvested cereal crops are also safe. If safe pastures are available, treat young stock with an anthelmintic at the onset of the rains and place them on the safe pastures entirely separated from the older animals.

Based on the seasonality of development and survival of (L3) on the pasture, the timing of strategic anthelmintic use can be determined and integrated into control programmes

## Anthelmintics

An anthelmintic is a compound which destroys or removes helminths from the gastro-intestinal tract and other tissues and organs they may occupy in their hosts.

Currently a good selection of safe anthelmintics is available, some with broad spectrum activity and others with activity against specific helminth infections. Many modern anthelmintics are effective against both adults and larval stages and an increasing number are efficacious against arrested or dormant larvae.

Due to their cost and their tendency to delay or interfere with natural host immunity mechanisms, anthelmintics may not be the most desirable method of managing helminth problems. However, in many circumstances the sensible use of anthelmintic drugs is likely to be the only available method of controlling helminth parasites. They should not be used indiscriminately.

### Characteristics and selection of anthelmintics

The ideal anthelmintic has the following properties:

(a) A broad spectrum activity against adult and larval helminth parasites.

A number of factors influence the efficacy of an anthelmintic drug. Animals often harbour several different species of helminths, which may not have the same sensitivity to a given anthelmintic. In addition, there is usually a difference in sensitivity between adults and larval stages, with immature stages being less sensitive than the adult parasites.

Very few if any of the anthelmintics are completely effective at the recommended doses under field conditions. Some anthelmintics may be very effective in sheep but not in cattle, or *vice versa.*

(b) A rapid metabolism in the body and short-lived presence at low levels in the milk and/or tissues.

Animals should not be slaughtered for human consumption and milk not distributed to consumers until the drug residues have reached acceptably low levels. The withdrawal period of the drug should be considered before its use.

(c) A low toxicity in the target species. The ratio of the therapeutic dose to the maximum tolerated dose should be as large as possible.

It is desirable that an anthelmintic has a safety margin of at least six-fold.

(d) No unpleasant side-effects to the animal or to the operator.

Drugs may cause vomiting, or pain at the injection site. Some drugs irritate the skin of humans.

(e) Suitable for practical and economical integration into various management systems.

The selected drug(s) should be competitively priced and ready to use in a simple way. They should be stable and not decompose on exposure to normal ranges of temperature, light and humidity, and have a long shelf life.

### Administration of anthelmintics

It is important to first identify the nature of the parasitic problem in order to select the appropriate drug to treat the infection. The optimal time and mode of administration of the drug should then be considered.

A wide variety of formulations and preparations have been developed to provide methods of dosing animals, which are convenient for a wide range of species and circumstances.

#### Dosing by mouth

The majority of anthelmintics are given by mouth as liquid preparations, pastes, boluses and tablets.

Liquid preparations are usually sold ready to use. Several devices such as syringes, bottles and drenching guns can be used for delivering the dose. Drenching guns are generally preferable and a wide variety, including single dose, multi-dose and automatic types, are available. It is important to keep the drenching equipment clean after use. The dose to be delivered should be checked before-and several times during-dosing to ensure that the correct dose is given to all animals. A graduated cylinder should be included in the field equipment for calibration purposes. It may be necessary to fit a short piece of rubber tubing on the end of the dosing nozzle to protect the mouth and pharynx of dosed animals.

Pastes are relatively easy to administer if a proper dispenser is available. If that is not the case, care should be taken to ensure the animal receives a full dose.

Boluses and tablets can be placed deep in the mouth of the animal by using a dosing gun or a pair of long-handled forceps, both of which can be manufactured locally. Bolus and tablet formulations have the advantage that if the dose is rejected, it is usually the total dose and a replacement can then be administered.

Prolonged protection of grazing livestock can be achieved by incorporating anthelmintics into medicated salt-molasses blocks and prepared mineral mixes, but animals do not always consume the amount required for an efficient treatment. Controlled-release preparations, such as slow release boluses allow the effective delivery of anthelmintics over several months.

#### Dosing by injection

A number of anthelmintics are available for injection. The size of needles should be appropriate for the formulation and the site of injection. In order to avoid local reactions (such as abscess formation at the injection site) the highest possible hygienic standards should be maintained.

#### Dosing by external application

Several dewormers are now available in a formulation for external application, termed "pour-on" preparations. The active ingredient of the drug is absorbed through the skin reaching its target via the circulatory system. This application form, which is particularly convenient for animals kept under range conditions, has the advantage that only minimum restraint of animals is needed, as the dose is applied to their back while passing through a crush or standing at a feeding trough.