

THE INTERACTION BETWEEN INTESTINAL HELMINTH INFECTION AND HOST NUTRITION. REVIEW

Saulius Petkevičius

Department of Infectious Diseases, Lithuanian Veterinary Academy, Tilžės g. 18, LT-47181 Kaunas, Lithuania.

Tel/fax +370 37 363559; e-mail: saulius.petkevicius@lva.lt

Summary. The regulation of helminth populations in the host's gastrointestinal tract is a complex process, influenced by host immunological and nutritional status, age and breed of the animal. The interaction between helminth infection and nutrition can be considered from two interrelated points of view: the influence of the helminth infection on the host's physiology and nutrition and the effect of host nutrition on the helminth populations, i.e. their establishment, persistence and reproductive capacity. The first point of view has been the subject of numerous investigations over the past decade. It was estimated that common features of infection with intestinal helminths are: a reduction in voluntary feed intake, reduction of the digestibility's of dry and organic matter, a decrease in efficiency of feed utilization, significantly higher nitrogen output, and a rise in plasma urea concentration. It was described that only a limited amount of studies have examined the effects of nutrition on the parasite response in the parasite infected host, and even fewer have considered the events occurring at the intestinal level, where absorption of nutrients occurs, intestinal parasites reside, and the gastrointestinal associated tissues play role in directing both the local and the more systemic responses. In this review the reference is made to the important literature related to the effect of different nutrients, e. g. carbohydrate, protein, fat, non-organic constituents and malnutrition on the helminth populations in host. It appeared that gastrointestinal helminths have very specific physico-chemical requirements of their host gut environment, and nutritionally mediated changes have a direct influence on the parasite population. Furthermore, the mechanisms by which different nutrients influence helminth infection are addressed.

HELMINTŲ INVAZIJOS IR GYVULIO ŠÉRIMO TARPUSAVIO SANTYKIAI (LITERATŪROS APŽVALGA)

Saulius Petkevičius

Užkrečiamų ligų katedra, Lietuvos veterinarijos akademija, Tilžės g. 18, LT-47181 Kaunas

Santrauka. Šioje literatūros apžvalgoje apibendrinta informacija apie įvairių maisto medžiagų – angliavandeniu, baltymu, riebalų, mikro ir makro elementų – badavimo poveikį virškinamojo trakto helmintų invazijai. Parazitų populiacijos reguliavimas šeimininko organizme yra sudėtingas procesas, priklausas nuo gyvulio imuniteto, šerimo, amžiaus ir veislės. Gyvulio šerimo ir parazitų santykiai dvejopii: parazitų invazija veikia gyvulio organizmo fiziologiją ir šerimą; gyvulio šerimas veikia parazitų populiaciją, t. y. jos pasireiškimą, persistenciją ir reprodukcines savybes. Paskutinį dešimtmetį atlakta daug tyrimų, paskelbta nemažai apžvalginiai straipsnių apie parazitų invazijos poveikį gyvulio organizmo fiziologijai ir šerimui. Nustatyta, kad virškinamojo trakto helmintų invazija slopina maisto medžiagų absorbciją, pašaro sausosios ir organinės medžiagos virškinamumą; gyvulys netenka daugiau maisto medžiagų, išskiria daugiau amoniako, šlapalo kraujo plazmoje.

Gyvulio šerimo arba dietos poveikis parazitų invazijai kol kas labai mažai tyrinėtas. Nedaug tyrimų atlikta nustatant gyvulio šerimo ir atskirų jo sudedamųjų dalių poveikį virškinamojo trakto parazitų invazijai, nors plonusiose ir storosiose žarnose vyksta aktyvus maisto medžiagų skaidymas ir absorbcija, o parazitai tiesiogiai kontaktuoja su virškinamojo trakto turiniu. Remiantis literatūra, gyvulio šerimo poveikį parazitinių helmintų invazijai galima suskirstyti taip: šerimo poveikis helmintų aplinkai, tiesioginis pašaro sudedamųjų dalių poveikis parazitams, dėl šerimo īvykę pasikeitimai šeimininko imuninėje sistemoje ir parazitams būtinų maisto medžiagų trūkumas. Virškinamojo trakto parazitų egzistavimui reikalinga specifinė biocheminė aplinka, priklausanti nuo gyvulio pašaro sudėties ir veikianti parazitų populiaciją.

Introduction. The gastrointestinal tract (GI) is not only an organ for digestion, absorption and excretion, but also it is a residence site to many parasitic organisms. The regulation of helminth populations in the host's GI is a complex process, influenced by host immunological and nutritional status, age and breed of the animal (von Brandt, 1979). Immunological status of the host is very important for helminth infections, because GI is one of the largest immunological organs of the body, and it serves as the first line of defense against orally administered antigens (e.g., feed proteins or carbohydrates) and

intestinal pathogens (e.g., parasites and bacteria). Gut associated lymphoid tissues make up about 25% by weight of the gut mucosa and submucosa and thus constitute the largest extrathymic site of lymphocytes (McBurney, 1993). Mucus secretion and formation of tight cell junctions prevent the entry of parasites and other pathogenic antigens, and rapid mucosal turnover enables the repair of epithelial or lymphoid cells damaged by parasitic infections. Furthermore, it is very important the interaction between helminth infection and nutrition, which are relevant to the population dynamics of parasites. This

interaction can be considered from two interrelated points of view, which are relevant to the population dynamics of parasites: (1) the adverse influence of the helminth infection on the host's physiology and nutrition and (2) the effect of host nutrition on the helminth populations, i.e. their establishment, persistence and reproductive capacity (Coop & Holmes, 1996).

The first point of view (the impact of helminth infection on the host's physiology and nutrition) has been the subject of numerous investigations over the past decade (cf. Stephenson, 1993; Solomons, 1993; Solomons & Scott, 1994; Edirisinghe & Tomkins, 1995; Coop & Holmes, 1996; Knox, 2000). Several reviews have concluded that animal offered a high plane of nutrition are better able to withstand the detrimental effects of nematode parasite infection than those less adequately nourished (Coop & Holmes, 1996; van Houtert & Sykes, 1996; Knox, 2000). The research on the complex interactions among host nutritional status, parasitic infection and immune responsiveness has mainly focused on the detrimental consequences of parasitic infections on host nutritional status and on mechanisms by which malnutrition impairs immunocompetence (Scott & Koski, 2000). Common features of infection with intestinal helminths are: a reduction in voluntary feed intake, reduction of the digestibility's of dry and organic matter, a decrease in efficiency of feed utilization, significantly higher nitrogen output, and a rise in plasma urea concentration (Blackburn *et al.*, 1991; Knox *et al.*, 1994). The most significant effect of gastrointestinal parasitism on the host is the depression in voluntary feed intake (Kyriazakis & Oldham, 1994; Knox, 2000). Large acute infections result in a very significant decrease in the rate of feed intake of parasitized animals (Sykes, 1987), but a degree of inappetence is present even in subclinical infections. In the latter case the degree of inappetence reported in the literature varies between 6 and 30% (Poppi *et al.*, 1990), and this variation has attributed to different nutrient contents of feed offered to parasitized animals. Host food intake is reduced depending on either the infective dose given to the host or the number of established parasites present (Crompton, 1991). Nutritional deficiencies as a result of intestinal helminth infections have been the subject of several investigations (cf. Hadju *et al.*, 1996; Lunn & Nothrop-Clewes, 1996). Intestinal helminths may affect the nutritional status by causing increased nutrient loss, in addition to decreased food intake and nutrient absorption (Edirisinghe & Tomkins, 1995). Detailed investigations of the mechanisms of gastrointestinal dysfunction of the parasitized host have shown that the increased endogenous loss of protein into the gastrointestinal tract is a key feature, partly as a result of leakage of plasma protein but also from increased exfoliation of gut epithelial cells and mucoprotein secretion (Bown *et al.*, 1991).

Curiously, the influence of host nutrition on helminth populations (the second type of host-parasite interaction) has received relatively little attention and limited information is available. Only a few studies have examined the effects of nutrition on the parasite response in the parasite infected host, and even fewer have considered the events

occurring at the intestinal level, where absorption of nutrients occurs, intestinal parasites reside, and the gastrointestinal associated tissues play role in directing both the local and the more systemic responses. Bundy & Golden (1987) described mechanisms by which host nutrition might influence helminth infection: nutritionally mediated changes in the helminth environment or nutritionally mediated changes in host defense and malnutrition of the parasite. Gastrointestinal helminths have very specific physico-chemical requirements of their host gut environment, and nutritionally mediated changes might have a direct influence on the parasite population (Crompton & Nesheim, 1976).

Carbohydrates. The effect of host diet and nutrition on parasites may be important in determining the overall transmission success, but this has received relatively little attention, at least in monogastric mammals like pig and human (Thamsborg *et al.*, 1999). Experimental infections in humans in order to elucidate effects of diet and nutritional status are for obvious reasons not possible. However, animal model systems employing closely related host and parasite species may help to determine the complex interaction between helminth infections and diet/nutritional status in the host (Johansen *et al.*, 1997). Due to anatomic, physiologic, immunologic, metabolic and nutritional similarities between humans and pigs, the pig has been paid extensive attention for use as a model for humans in many types of research, including parasitological research (Stephenson, 1993). The information provided on parasitic infections in pigs could lead us to believe that the model has a potential value for elucidating the infection and disease in humans (Wilingham & Hurst, 1996).

The carbohydrates, which include the low molecular-weight (LMW) sugars, starch and various cell wall and storage non-starch polysaccharides (NSP) are the most important energy sources for non-ruminant and ruminant animals (Bach Knudsen, 1997). The carbohydrates in the animal diet consist of mono-, di- and oligosaccharides and two broad classes of polysaccharides: starch and NSP (Cummings *et al.*, 1997). The NSP and lignin are the principal components of cell walls and are commonly referred to as dietary fibre (Theander *et al.*, 1993). It is now clear that dietary carbohydrates are a diverse group of substances with varied fates in the gastrointestinal tract and physiological properties of differing importance to animal health (Cummings & Englyst, 1995). The large differences in structural composition of the various NSP may explain part of the contradictory effects observed with different types of dietary fibre, as they can be expected to behave differently in the gastrointestinal tract depending on their chemical characteristics (Jacobs, 1986). Assimilation of dietary carbohydrates results principally in three groups of products: sugars (glucose, galactose, fructose), short-chain fatty acids (SCFA) and lactic acid (LA) (Bach Knudsen *et al.*, 2000). Glucose derives from the enzymatic breakdown of starch, of which the vast majority is broken down to glucose, maltose, maltotriose and α -linked dextrins, within the intestinal lumen by α -amylase, secreted via the pancreatic duct. On

the intestinal surface membrane, the oligosaccharides are cleaved to glucose, galactose and fructose and removed from the intestinal lumen either by a Na-dependent mucosal carrier and absorbed against concentration gradient (glucose, galactose) or by passive diffusion (fructose) (Gray, 1992). The main site for fermentation with SCFA production is large intestine (Fleming & Arce, 1986). The substrate for this fermentation is the dietary residues not digested in the small intestine, the main one being carbohydrate in form of NSP, resistant starch, various forms of oligosaccharides, dietary proteins and endogenous compounds such as mucus, enzymes and sloughed cells (Macfarlane & Cummings, 1991). The amount and type of carbohydrate potentially available for fermentation can be modulated through changes in the dietary composition, which will influence rate and amount of SCFA produced (Bach Knudsen *et al.*, 2000). The declining SCFA concentration from caecum and proximal colon to the distal colon in monogastric animals (Topping *et al.*, 1993) suggests a rapid absorption of SCFA from gut lumen (Fleming & Arce, 1986). The fermentable polysaccharides (PS) will act in the caecum and colon as an energy source for the microorganisms and be degraded mainly to SCFA, which are widely regarded as stimulators of intestinal tissue proliferation (Edwards, 1993). Highly fermentable PS will primarily be degraded in the proximal part of the large intestine, while the less fermentable PS, to a greater extent, will reach the distal part of the large intestine and may escape fermentation. The production of the SCFA changes as microbial adaptation takes place (Tulung *et al.*, 1987), suggesting that the time required by the gastrointestinal tract to fully adapt may depend on the type of PS and its fermentability. Other products of fermentation including lactate, which is an intermediate in starch breakdown, significant quantities may be found in the stomach and the large intestine but does not accumulate in the colon (Argenzio & Southworth, 1974). Carbohydrates are not only assimilated and provide energy to the host. Due to the cell wall structure, NSP may influence the rate and extent of starch digestion, blood glucose and insulin levels (Ellis *et al.*, 1995). The most notable effects have been related to soluble fibre sources (Jenkins *et al.*, 1978). The composition of the carbohydrate fraction influences the digestion and absorption processes of carbohydrates and other nutrients in the various parts of the gastrointestinal tract (Bach Knudsen & Jørgensen, 2001); it has profound influence on the secretory response of the gut to feed intake (Low, 1989), the volume flow (Bach Knudsen *et al.*, 1993), the mucosal architecture (Brunsgaard, 1998), composition of the gut flora (Jensen & Jørgensen, 1994) and the development of the gastrointestinal tract (Jørgensen *et al.*, 1996). However, it should be stressed that using the diet to manage gut health is not straightforward, since the expression of a pathogen in many cases requires the presence of other components of the commensal biota (Bach Knudsen, 2001).

Few investigations deal with the direct influence of host diet on parasitic helminths (host nutrition not severely affected). Studies on the influence of carbohydrates on parasite growth and establishment have been limited

mainly to cestodes and acanthocephalans (for reviews see Crompton & Nesheim, 1982; Nesheim, 1984). Increased fecundity, higher worm burdens and enhanced growth and sexual development of *Moniliformis dubius* (Acanthocephala) occurred in rats fed on host dietary fructose (Crompton *et al.*, 1982; Keymer *et al.*, 1983b). The survival, growth and reproduction of *Moniliformis moniliformis* are dependent on the carbohydrates liberated at different rates from the intestinal tract of the host during digestion and absorption (Nesheim *et al.*, 1977, 1978). In addition, Parshad *et al.* (1980) have found *M. moniliformis* worm dry mass at 5 weeks p.i. was greatest to smallest in order mannose, fructose, glucose and galactose 3% diets fed rats. Crompton *et al.* (1983) have found that *M. moniliformis* dry weights of male and female worm 5 weeks p.i. were greater in rats fed on fructose and fatty acids diet compared with the group fed by fructose and maize oil diet. Absence or restriction of availability of dietary carbohydrates resulted in decreased establishment, growth and reproduction of *Hymenolepis diminuta* in rats (Roberts, 1980; Keymer *et al.*, 1983 a). Dunkley & Mettrick (1969) have found that in rats fed by sucrose diet were found smaller *H. diminuta* worms than in rats on glucose or maltose diets and Roberts & Platzer (1967) pointed that absence of carbohydrates in the rats diet injured the worms reproductive systems. According Molan & James (1984) in mice fed on the milk diet 60 days p.i. were present more *Microphallus pygmæus* worms compared with the group on commercial pelleted diet. Additionally, it was found that *H. diminuta* worms from high starch diet rats were bigger than from low starch rats, which were bigger than from sucrose diet rats (Roberts, 1966).

However, the influence of carbohydrates in the host diet on nematodes has received less attention. Host dietary carbohydrates have been shown to have a positive influence on growth and reproduction of *Heterakis gallinarum* in chickens (Aboud, 1989). Thus, it has been found that the carbohydrate composition and the level of lignin play an important role in the physico-chemical environment in the gut lumen and for the microbial fermentation in the large intestine of pigs (Bach Knudsen *et al.*, 1993; Johansen *et al.*, 1997). These changes may have implications for maintaining optimal function of the epithelial cells lining the large intestine, as these cells obtain most of their energy from short-chain fatty acids (SCFA), in particular butyrate produced as a consequence of microbial fermentation in the large intestine (Sakata, 1995). A diet rich in carbohydrates is indispensable for development of the epithelial and ovarian cells and eggs of fully grown *Ascaris suum* (Movsesian, 1984). Moreover, a diet rich in carbohydrates, mainly undigested in the small intestine, stimulates peristalsis and increases the faecal bulk (Bach Knudsen & Hansen, 1991). Experiments in pigs, however, have repeatedly shown that a diet rich in non-starch polysaccharides (NSP) and lignin is beneficial to many intestinal parasites, particularly those which have predominantly anaerobic metabolism, such as *Oesophagostomum* spp. (Herbert *et al.*, 1969).

Protein and fat. Various dietary constituents other

than carbohydrates have also been found to be important to parasites. Although the diets used in the experiments were nutritionally sufficient for the host, apparent from high and comparable growth rates, they have shown different impact on parasites. The experimental sheep fed diet with low protein content were capable of eliminating a considerably lower proportion of *Oesophagostomum columbianum* larvae and were thus immunologically less competent than animals in adequate diets (Hunter, 1953). This conclusion is supported by the observations that in adequately fed sheep were found encapsulated *O. columbianum* larvae showing arrested development and adult female worms produced fewer eggs compared to sheep on low protein diets (Bawden, 1969). Above mentioned findings suggest that *O. columbianum* in poorly fed sheep frequently have shorter histotropic development and lower host immunity reaction to the parasite (Dobson & Bawden, 1974). Larvae of *Ascaris suum* established more readily in the intestines of pigs which were fed on oat diets than in pigs which received milk diets (Kelley *et al.*, 1959). Boddington & Mettrick (1981) have found that in rats fed by low protein diet were found reduced fecundity of *H. diminuta* female worms. Adding additional protein to the diet improves resistance and resilience to several nematode infections (Wallace *et al.*, 1998). Similarly, Crompton *et al.* (1985) have found that additional proteins in the diet increased *Taenia crassiceps* establishment and growth in mice. In young sheep offered a low quality roughage diet of oaten chaff and essential minerals, supplementation with urea reduced the effects of gastrointestinal parasitic infection by increasing weight gain and wool production; and reducing faecal egg output and parasite burden (Knox & Steel, 1996). It appeared that the establishment of *Teladorsagia circumcincta* larvae was depressed in lambs given urea supplemented diet (Stear *et al.*, 2000). Authors pointed that combination of relatively resistant sheep and nutritional supplementation is most efficient at controlling infection. Lambs fed lucerne diets were more resistant to *Oesophagostomum columbianum* than lambs fed straw and molasses (Dobson & Bawden, 1974), while lambs given supplementary fishmeal were more resistant to *Trichostrongylus colubriformis* (Houtert *et al.*, 1995) as were lambs given supplementary meat and bone meal as well as soyabean meal (Kambara *et al.*, 1993). Clarke (1968) showed that a low-protein diet given to *Nippostrongylus brasiliensis* infected rats resulted in expulsion of the worms. Studies on acute *N. brasiliensis*, *Nippostrongylus muris* and *Trichuris muris* infection in rodents have shown that dietary protein deficiency during primary infection can increase parasite establishment and survival (Bolin *et al.*, 1977; Michael & Bundy, 1991). In rats, fed by the low protein diet fewer of *Litomosoides carinii* worms developed, growth was reduced, embryogenesis retarded and onset of patency was delayed compared with hosts on normal protein diet (Storey, 1982). Low-protein diet increased the establishment rate of *Shistosoma japonicum*, favoured larger deposits of eggs in the liver and faecal egg excretion, reduced weight gains and caused anaemia and hypoalbuminaemia in young growing pigs as compared with a high-protein diet (Johansen M.

V. *et al.*, 1997). This is in agreement with study by Willingham *et al.* (1998), which showed that the serum albumin concentration in malnourished pigs was significantly affected by *Shistosoma japonicum* infection. Coutinho *et al.* (1992) concluded that low-protein and energy diet increased pathogenicity and reduced the fecundity of females of *Shistosoma mansoni* infection in mice. Significant reduction in growth, worm fecundity, worm recovery and a more proximal location in the gut of *Echinostoma caproni* was found in mice fed a high fat diet in the form of cottonseed oil, compared to mice fed a standard laboratory diet (Sudati, Reddy & Fried, 1996). Marked anterior migration in gut of *Hymenolepis diminuta* worms was found in rats 1h after feeding by dietary fat (olive oil) (Mettrick, 1971). Fewer *Litomosoides carinii* worms developed and growth of female worms retarded in rats fed on 10% glycerol diet (Kershaw *et al.*, 1975). The intensity and prevalence of *Ascaridia galli* infection were lower in chickens given a high protein diet (Zoltowska *et al.*, 1991). In mice fed a low protein diet *Trichinella spiralis* infection significantly increased, and there was a delayed and weakened inflammatory response to the invading parasites, compared to mice fed a normal protein diet (Gbakima, 1993). In sheep given additional dietary protein, egg counts of *Trichostrongylus colubriformis* in faeces were significantly lower (Brown *et al.*, 1991), worm expulsion significantly higher (Kambara *et al.*, 1993), and the animals developed a better resistance to parasites (Houtert *et al.*, 1995; Kambara & Mcfarlane, 1996). Chartier *et al.* (2000) have pointed that resistance and resilience of high productions goats to *T. colubriformis* infection may be improved by a protein supplementation in the diet. Increase of protein in the diet could decrease the periparturient rise of *T. colubriformis* egg count under natural conditions in goats (Etter *et al.*, 1999) and could improve resistance (EPG and eosinophil counts) to experimental *T. colubriformis* infection at the beginning of lactation (Etter *et al.*, 2000). Abbot *et al.* (1986) demonstrated that lambs fed a low protein diet were less able to withstand infections with *Haemonchus contortus*. Using different levels of a protein supplement (cotton seed meal) those sheep receiving the higher levels of supplement had the lowest *H. contortus* egg output (Datta *et al.*, 1998). Animals that had previously received the higher protein diets had higher antibody responses to both *H. contortus* and *Trichostrongylus colubriformis* infections and lower nematode egg counts than did the lambs previously offered the lower protein diets (Datta *et al.*, 1999). In controlled studies with sheep, Knox & Steel (1999) showed that supplementation of a low quality roughage diet of oaten chaff and essential minerals with urea reduced *H. contortus* and *T. colubriformis* faecal egg output and parasite burden. Studies in sheep showed that establishment of *Oesophagostomum columbianum* was not affected by the protein content of the diet, but worm establishment was higher at slaughter on day 56 p.i. in lambs on the low protein diet (Dobson & Bawden, 1974). In single infections with *O. dentatum* (Poelvoorde & Bergheen, 1978), or mixed infections with *Ascaris suum* and *Oesophagostomum dentatum* (Costa *et al.*, 1979), in

pigs that were fed a low protein diet, a reduction of faecal egg counts and numbers of worms was found compared to control animals. In contrast, Sinski *et al.* (1988) found that establishment of *Obeliscoides cuniculi* in rabbits and Kahn *et al.* (2000) pointed that faecal egg counts, worm burdens and per capita fecundity of adult female of *Trichostrongylus colubriformis* in lambs were not substantially influenced by protein deprivation.

Non-organic constituents. In addition to dietary carbohydrates, protein and fat, non-organic constituents in the diet have also been found to be of importance to the establishment of parasites (Coop & Holmes, 1996). In lambs infected with *Haemonchus contortus*, the addition of cobalt sulphate to the diet increased the total egg output (Lara *et al.*, 1974). Zinc deficiency (in the diet) impaired the expulsion of *Trichinella spiralis* (Fenwick *et al.*, 1990 a), enhanced the establishment of *Strongyloides ratti* in the intestine of rats (Fenwick *et al.*, 1990 b). Similarly, Boulay *et al.* (1998) reported prolonged survival of *Heligmosoides polygorus* in mice and El-Hag *et al.* (1989) of *T. spiralis*, *S. ratti* in rats and *H. polygyrus* in mice fed a zinc deficient diet (3mg zinc/kg diet). In contrast, a dietary zinc intake 5mg/kg did not increase the intensity of *H. polygorus* infection in mice (Minkus *et al.*, 1992) and had no effect on the number or size of *Nippostrongylus brasiliensis* in rats (El-Hag *et al.*, 1989). Scott & Koski (2000) concluded, that dietary zinc deficiency can improve the survival of intestinal nematode parasites in animal models under controlled experimental conditions. In chickens, addition of zinc and copper salts to the diets increased *Ascaridia galli* worm burdens (Gabrashanska, 1993; Gabrashanska & Timanova, 1993). Experimental results have indicated that adding selenium to the feed, either alone or in combination with zinc, significantly increased *Fasciola hepatica* egg output in ewes (Samak *et al.*, 1986). Laubach (1990) recovered more *Ascaris suum* larvae from the livers and lungs of mice given the same oral doses of infective eggs on low zinc rations during both primary and secondary infections. Mice infected with *A. suum* and fed low levels of dietary iron were found to harbor lower numbers of larvae in the lungs compared to mice receiving normal or light iron diets (Lauback, 1989). Studies on acute *Nippostrongylus brasiliensis* infection in rats have shown that dietary iron deficiency during primary infection can increase parasite survival and establishment (Bolin *et al.*, 1977). In contrast, the addition of molybdenum to the diet of lambs exposed to infection with *Trichostrongylus vitrinus* and *Haemonchus contortus* reduced worm numbers and length of adult worms (Suttle *et al.*, 1992 a; 1992 b).

Fasting. It appeared that diets with high content of insoluble dietary fibre and fast gastrointestinal transit time significantly increased efficacy of orally administered anthelmintics. Furthermore, Hennessy *et al.* (2000) have pointed that efficiency of anthelmintics is strongly depended on composition of diet administrated to the pigs. Some studies have been designed to analyze the influence of fasting on helminth parasites. Read & Rothman (1958) after fasting of rats for 48h have found marked polysaccharide depletion in *Moniliformis moniliformis* worms.

After fasting of mice up to 96h glycogen content of *Shistosoma mansoni* population, particularly of male worms was depleted (Cornford *et al.*, 1983). Glassburg *et al.* (1983) after food withheld in rats for 18, 24 and 48h have pointed anterior movement in the gut of *Nippostrongylus brasiliensis* worm populations and Clarke (1968) observed that in dietary deficiency rats more *N. brasiliensis* larvae reached lungs and gut compared with group on the normal diet. In the faeces of domestic horses starved for ten days, the number of strongylid eggs decreased, and expulsion of adults and larvae of the genera *Delafondia* and *Alfondia* was observed (Dvojnos & Timoshenko, 1994). Under natural winter conditions in Mongolia, wild horses had great difficulty finding food and showed a significant expulsion of adult strongylids (Dvojnos & Timoshenko, 1995). In rats fasted for two days, *Nippostrongylus brasiliensis* had a wider distribution in the small intestine and many individual worms (mainly females), moved from the small intestine to the caecum (Croll, 1976). Furthermore, the efficacy of anthelmintics could be increased by reducing amount of feed or even starving the animals for short periods of time before and after administration of the anthelmintic (Ali & Hennessy, 1995 b). The high diet fibre content and temporary starvation could increase the flow rate of digesta and it might be considered as a means to increase the availability of anthelmintic compounds in sheep (Ali & Hennessy, 1995 a; 1996), cattle (Sanchez *et al.*, 1997) and pig (Hennessy *et al.*, 2000). In contrast, Mueller *et al.* (1958) after food restriction in mice found no detectable effect on growth of *Spirometra mansonioides* and Van Cleave & Ross (1944) found *Neochinorhynchus emydis* worms live for many months when turtles were fasting. Apart from these studies, little is known about the influence of fasting or starvation on gastrointestinal helminths (Beisel, 1982; Shetty & Shetty, 1993).

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