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STRONGYLOSIS IN EQUINES: A REVIEW

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ABSTRACT

We review strongylosis covering all aspects as it is one of the most important internal parasitic diseases of equines caused by nematodes of strongylidae family affecting more than 80% equids in the world. Majority of the work published has been focused on incidence, epidemiology and control. Strongylus vulgaris, one of the large strongyle is the most prevalent and pathogenic. Small strongyles exhibit mild symptoms of diarrhea and weight loss in the host whereas large strongyles show major pathogenesis. The pathogenesis encompasses severe enteropathy, verminous arteritis, damage of visceral organs, embolisim / thrombosis leading to death and is mainly attributed to migrating larvae of parasites. In this report the scientific knowledge about development, pathogenesis, clinical findings, diagnosis, epidemiology, treatment and control of strongylosis in equines has been reviewed for a period of last nine decades. Inspite of substantial improvements in understanding the life cycle of strongyles, adopting latest diagnostic techniques at molecular level and implementing the most modern control measures / treatment the disease is still prevalent and could not be eradicated from any part of the world. It is currently impossible to measure or detect the encysted larval load in living horse, however there are exciting advancements in diagnosis of disease by means of molecular approaches such as cyathostomin gut-associated larval antigen-1 (Cy-GALA-1). The current strategy engaged in seasonal use of anthelmentics is the key to arrest the disease and overcome anthelmentic resistance. We conclude that there is still a need to thrash approaches / scientific knowledge towards understanding the problem to reduce economic / performance losses. The scientific studies for development of an effective vaccine are considered the need of the day.

Key words: strongylosis, equines, S. vulgaris, arteritis, faecal floatation, migrating larvae.

INTRODUCTION

Strongylosis has been reported from all parts of world and almost affects more than 90 % of horse population (Nielsen *et al* 2006). The small strongyles, also called cyathostomins, are among the most important intestinal nematodes of horses (Lyons *et al*., 1999) with almost 100% of horses infected with at least some species of small strongyles (Reinemeyer *et al*., 1984). Among the gastro-intestinal nematodes of horses, large strongyle infections were diagnosed with an infection rate of 58.5% (Saeed *et al*., 2010). S. vulgaris has long been considered as one of the most common and pathogenic parasites of the horse (Claire and Masterson., 1987; Krecek *et al*., 1987; Tolliver *et al*., 1987; Peter and Waller., 1997; Gasser *et al*., 2004; Hubert *et al*., 2004; Martin *et al*., 2007; Toscan *et al*., 2012). It is estimated that 45 to 90 per cent of horses harbor S. vulgaris (McCraw and Slocombe, 1976; Jubb *et al*., 1985). The prevalence of infection is high, and a few equines are likely to escape out of this disease by the end of their first year of life (0gbourne and Duncan, 1985). Infestation with strongyles is complex and produces an inflammatory enteropathy which results in impaired intestinal motility and microcirculation (Love *et al*., 1999; Bechera *et al*., 2010; Neils *et al*., 2011; Pilo *et al* 2012). Clinically, infection with small strongyles can cause mild disease symptoms such as weight loss, loss of appetite, poor hair coat, intermittent diarrhoea, lethargy, deterioration of condition, peripheral edema and disordered intestinal motility (McCraw and Slocombe, 1976; Love and Duncan, 1992; Love *et al*., 1992; Matthews and Morris, 1995). After ingestion, larvae travel through the digestive system to the large intestine, S. vulgaris migrate to the anterior mesenteric artery, S. endentatus to the liver / flank area and S. equinus migrate to the liver and pancreas (Owend and Slocombe., 1985; Monhan *et al*., 1995; Kuzmina *et al* 2012). Adult large strongyles live in the cecum and large intestine. Fourth (L4) and fifth (L5) stage larvae are responsible for arteritis, necrosis, and fibrosis of the cranial mesenteric artery and its branches (Patton and Drudge, 1977; Duncan and Pirie, 1985; Kuzmina *et al*., 2012). The occurrence of larvae in the cranial mesenteric artery was apparently known to the ancient Romans (McCraw and Slocombe 1974). Verminous arteritis in the cranial mesenteric artery and its branches has been reported as the cause of death in 10 to 33% of abdominal crises in the horse (Meads, 1969; Proctor, 1966) and in a few case studies of natural infections (Foster and Clark, 1937; Ottaway and Bingham, 1945; Deorani, 1966). Severe colic and death of horses is the consequence of thrombosis and embolism leading to infarction of the intestinal tract (Marinkovic *et al*., 2009). Many animals are not picked up early enough to interrupt the larval migration and are only diagnosed by faecal culture techniques indicating the presence of

fertile adults (Morgan *et al*., 1994). Determining the number of strongyle eggs per gram of faeces (EPG) has been the most widely used method for diagnosing infection with adult strongyles (Herd, 1995; Mathew and morris, 1995; Kaplan, 2010; Mahmood and Ashraf, 2010). Molecular approaches have been developed that enable species identification of pre-adult stages of strongyle nematodes (Gasser *et al*., 2004; Matthews *et al*., 2004). Routine treatment for the removal of adult worms is essential as the large egg output of strongyles can cause contamination of a restricted area very rapidly. Thiabendazole has been widely used and recently several other anthelmintics have been developed and approved for use in adult horses, including benzimidazole compounds (Drudge *et al*, 1975). The under dosing is the major cause of anthelmintic resistence (Toscan *et al*., 2012). Pasture management is considered a reliable control measure. The emergence of anthelmintic resistance demands review of old studies.

Development and Life Cycle: All species of equine strongyles have direct life cycles (Bucknell *et al*., 1995; Osterman, 2005; Kuzmina, 2006; Martin *et al*., 2007). There are significant differences in number of eggs in female uteri in various strongyle species where the egg number differs 50–100 times between species (Kuzmina *et al*., 2012). Eggs are laid by adult female strongyles and passed in the faeces into the external environment where they hatch to the first stage larvae (L1s), at 12– 39 ^{O}C with adequate moisture. The minimum temperature for eggs to hatch is 7–8 ^{O}C (Ogbourne, 1975). From the egg on the ground / pasture L1 emerges which grows and molts to L2 and then to the L3 stage. The L3 is the infective stage and under optimal summer conditions it requires about ten days to two weeks to develop from the time the egg is passed. L3s retain an outer protective sheath and are more resistant to chilling and dehydration than the L1s and L2s. Dehydration can prevent L3s from leaving the faeces and gaining contact to herbage. Most larvae climb no higher than 10 cm from the soil surface, can move 15 cm horizontally and during rain L3s migrate from the faeces to the surrounding herbage most efficiently. (Bucknell *et al*., 1995; Edward, 2007). After ingestion of L3 by the host, they pass to the small intestine, remove external covering and initiate the internal phase of development. Removal of protective covering depends upon the stimuli from physiological / biochemical conditions in the gut of the host. Larvae of large strongyles emerge from the sheath through an anterior cap, whereas larvae of small strongyle escape via a longitudinal slit in the region of the oesophagus (Kuzmina *et al*., 2012). Removel of outer covering at 38 ^{O}C within 3 hours using an artificial intestinal fluid comprising trypsin, pancreatin, sodium bicarbonate and sodium dithionite has been achieved experimentally (Kuzmina *et al*., 2006). Internal phase of large strongyles larval development encompasses a somatic migration, whereas those of small strongyles burrow into the glands in the caecum and colon, and become encysted with no further migration. (Eysker *et al*., 1986; Gasser *et al*, 2004). In the submucosa next molting occurs ie L4 on about day 4 or 5 PI. Working against the flow of blood, the L4s gradually move up the arterial system of the intestine. By eight days PI larvae have reached the cecal and ventral colic arteries. When these larger arteries are achieved, the route of migration is marked by a twisty thread of fibrin on the intima and by day 14 larvae may be found in mural thrombi. The ileo-ceco-colic and cranial mesenteric arteries are reached between 11 and 14 days PI. The traveling advance attains its climax by the 19th day at which time larvae may be found in almost any part of the arterial system but are always most abundant in arteries close to the origin of the cranial mesenteric artery (McCraw and Slocombe, 1976; Hopfer *et al*., 1984; Osreman, 2005). The molt to the fifth stage (L5) occurs as early as 9 days PI and by 120 days. At this stage most larvae are preadults measuring up to 18 mm long. S. vulgaris larvae tend to remain in the arterial site until they molt to the fifth stage, though many fourth stage larvae are apparently swept away before the last molt occurs. Larval size and the thrust from the flow of blood are important factors in the separation of larvae from arterial lesions. The preadult larvae reach the small arteries on the serosal surface of the large intestine and terminal small intestine. Unable to migrate further in arteries, the young one of large strongyle become encased in pea-sized nodules. These nodules are numerous four months after infection; after their escape from nodules into the lumen of the intestine, S. vulgaris require another six to eight weeks before reaching sexual maturity. The prepatent period (the time from ingestion of L3s to the excretion of eggs in the faeces) is 5–7 months (McCraw and Slocombe, 1976; Kuzmina *et al*., 2006; Martin *et al*., 2007; Niels *et al* 2011). The prepatent period among the species varies from 6 months to 12 months (Urquhart *et al*., 1996; Osterman, 2005). Prenatal infection lacks any evidence (Andersen, 2013). Deviant larvae can sometimes migrate to kidneys, thoracic cavity and testis. The pattern of larval migration among large and small strongyles differs; (1). Large strongyles larvae migrate widely within the host through extra-alimentary tissues with a minimum of 6 month prepatent period (Kaplan, 2004). (2) Small strongyles larvae invade the lining of the cecum and ventral colon where they grow within fibrous cysts in the mucosa or submucosa and can reside as long as 2.5 years (Reinemeyer, 2009).

Now it is understandable that the ecological requirements for larval development and insistence are matching for both groups as inferred from above mentioned studies and described by Hutchinson *et al*., 1989 too however, no dependable information is available on the environmental conditions required for

the development of L1s and L2s. For small strongyles, the life cycles of individual species have also not yet been determined. The question on influence of fecundity on proportion of species in strongyles community needs further studies.

Pathogenesis: Naturally infected horses usually carry a mixed load of large and small strongyles in the intestine (Owend and Slocombe 1985). The pathogenesis of strongylus has been studied based on elucidation of experimental mono specific infections (Drudge *et al*, 1966; McCraw and Slocombe, 1974; Malan *et al*., 1982; Alam *et al*, 1999). The damage caused by large and small strongyles is attributed to larval stages. Small strongyles have small buccal capsules and feed superficially on the mucosa (Ogbourne and Duncan 1985). Large strongyles have large buccal capsules which they attach to the intestinal mucosa, pull out a plug of tissue, absorb the host cells, crack the blood vessels and suck blood, feed on the mucosa and consume blood (Levine, 1980). Hemorrhage occurs subsequent to feeding at the injured site which eventually is marked by a scar. The larvae cause minimal inflammatory response as long as they remain encysted however their synchronous emergence of large number results in diffuse inflammation of the cecum and ventral colon (Love *et al*., 1999). In all the cases a normocytic, normochromic anaemia is observed in affected equines (Ogbourne, 1985). The L4s and L5s migrate through the arterial system and cause verminous arteritis with marked intima thickening infiltrated with inflammatory cells. They can cause mechanical damage and inflammation in the liver, pancreas and peritoneal cavity. A considerable reduction of lesions in the cranial mesenteric artery was found approximately nine months after infection with S. vulgaris larvae (Duncan and Pirie, 1985). Incidence of lesions is 86% in the cranial mesenteric artery followed by 62.5% in the cecal and colic arteries (Poynter, 1969). Depending on larval burden, infected horses show clinical signs of pyrexia, anorexia and colic. Colic has long been considered related to thrombosis or embolism in these vessels (Bueno *et al*, 1979). In older horses the cranial mesenteric and ileo-ceco-colic arteries are often encased in a large nodular mass. Occlusion of the right coronary artery was observed due to S. vulgaris larvae and in the kidney too (Cronin and Leader, 1982). S. vulgaris was also suspected as an important cause of cerebrospinal nematodiasis (Little *et al*., 1974). Several studies have reported alterations in blood parameters and blood chemistry as a consequence of S. vulgaris infection, including a decrease in RBC, PCV, total serum proteins and an increase in WBC. Intestinal haemorrhages lead to reduced RBC survival loss of albumin in intestine leads to increased albumin catabolism (Drudge *et al*., 1966; Duncan and Pirie, 1985; McCraw and Slocombe, 1976; Patton and Drudge, 1977; Peter and Waller 1997; Nielsen, 2012). In ponies with a natural infection, a reduced level of ileo-ceco-colic motility has been demonstrated with electromyographic techniques (Bueno *et al*., 1979). At necropsy, the crater-like ulcers caused by large strongyles are often more numerous than the worms, suggesting that they move periodically to new sites of attachment (Andersen *et al*., 2013).

Due to the difficulty in differentiating the effect of species in naturally acquired, mixed infections there is lack of detailed information on the pathological effects of individual species of small strongyles, (Ogbourne, 1976; Reinemeyer *et al.*, 1984; Lyons *et al*., 1997). Several studies have shown a marked decrease of S. vulgaris infection worldwide but on the other hand many studies showed that this parasite continues to exert its pathogenic role, even when its detection rate is quite low within the strongyle population infecting horses (Pilo *et al*., 2012).

Clinical Signs: The acute signs related with large strongyles are due to migrating larvae and are seen during the first few weeks after infection. The severity of signs is related to the number of larvae ingested, the age and previous occurrence of the host. (McCraw and Slocombe, 1985). Older horses are often observed to have arterial lesions without a history of specific signs, although signs detected in field cases can be correlated with findings at necropsy (Gasser *et al*, 2004). Anemia, emaciation, poor coat and poor performance are frequently attributed to large strongyles while in the intestine. Diaarrhoea is more common sign in small strongyle infection than with large strongyles (McCraw and Slocombe, 1976). The main clinical sign in small strongyle infection is weight loss (Love *et al.*, 1999). Other typical clinical signs are profuse / sudden onset of diarrhoea, loss of body condition, debility with normal appetite and subcutaneous oedema of the limbs / ventral abdomen. Death is relatively common with mortality rate of >50% (Love *et al.*, 1999). The encysted larvae of small strongyles can emerge synchronously from intestinal wall, leading to the clinical disease called ‘larval cyathostominosis’, which is associated with clinical signs of oedema, diarrhoea, pyrexia, weight loss, colic and can be fatal in up to 50% of cases (Gasser *et al*, 2004). Fever in S. vulgaris infection is attributed to tissue damage or a toxic substance elaborated by larvae. The most steady change in early S. vulgaris infection would result a rapid increment in total white cell (WBC) counts. These values rise sharply during the first three weeks to levels of 17,000 to 22,700/mm3. Eosinophils values will increase after the second week and demonstrate little change in acute infection. Increments in serum total protein and globulin fractions occur as early as the first week following infection. Thrombus formation can block arteries, causing infarction of intestinal walls and/or intermittent lameness, and is commonly associated with clinical signs of marked pyrexia, anorexia, severe colic

and death (Pilo *et al.,* 2012). Under natural conditions, severe symptoms are rarely seen because foals may tolerate large numbers of larvae ingested in small doses over a long period (Urquhart *et al,* 1996). Number of adult strongyles in the intestine required to provoke clinical signs are not yet known and need elaboration / further studies.

Diagnosis: Signs and symptoms are not valuable for diagnosis. It has usually relied on the use of the method of faecal flotation (Duncan and Pirie., 1985; Nautrup *et al*, 2003; Gasser *et al*., 2004; Kaplan and Nielsen., 2010; Andersen *et al*., 2013). Faecal egg counts are useful for comparing the efficacy of anthelmintic compounds, detecting drug resistance, and determining the correct gap between anthelmintic treatments (Herd, 1992; Warnick, 1992). Since it is not possible to distinguish strongyle eggs of different species morphologically, faecal samples are cultured to allow the development to L3s, which may be collected for study. The Baermann technique is also a successful method for the recovery of small strongyles immature larvae in the faeces of clinically diseased horses. In case of larval strongylosis fecal egg count technique is assumed to be of no value. It is impossible currently to measure or even detect the encysted larval load while the horse is still alive (Osterman 2005). A method for detecting mucosal larval stages would be valuable in the diagnosis of larval strongylosis. Recently cyathostomin (small strongyles) gut-associated larval antigen-1 (Cy-GALA-1) has been identified, which is a target of serum IgG (T) responses in experimentally and naturally infected horse populations (McWilliam *et al*., 2010). Within the past two decades, molecular approaches have been developed that enable species identification of pre-adult stages of strongyle nematodes like characterization of strongyle nematode ribosomal DNA sequences (Gasser *et al*., 2004). The first and second internal transcribed spacers (ITS-1 and 2) and the intergenic spacer (IGS) have been used as genetic markers for species identification. From the ITS sequences, species-specific oligonucleotide primers for some of the most common species (S. vulgaris, C. catinatum, C. nassatus, C. longibursatus and C. goldi) have been designed and used in a PCR system, thus allowing species-specific amplification of parasite DNA in eggs and larvae (Hung *et al*., 2000). IGS oligoporbes are used to study the effect of anthelmintic treatment at the species level (Matthews *et al*., 2004). IGS oligoprobes have been used in a PCR-ELISA (hybridisation assay) for the detection of PCR products from L4s collected from horses suffered from diarrhoea (Nielsen, 2012). A microchip-based capillary electrophoresis technology has been employed successfully for species differentiation of closely related cyathostomins (Posedi *et al*., 2004). A copro antigen ELISA has shown promise with moderate to good diagnostic sensitivity and specificity as well as a positive correlation with worm numbers (Kania and Reinemeyer, 2005; Skotarek *et al*., 2010). Change in the blood picture associated with S. vulgaris is not unlike that seen in bacterial infections (Drudge *et al*., 1984). Alterations in blood biochemical and haematological parameters can be detected in a proportion of infected horses. Hypoalbuminaemia is a common finding in naturally infected horses, which is probably due to the increased permeability of the intestines. A rise of b-globulin in serum has also been reported in natural infections. A marked reduction of serum fructose amines (glycated serum proteins) has been reported for horses with experimental cyathostomin infection (Dowdall *et al*., 2004)

Foregoing in view it is evident that recent advancements in molecular approaches lack specific diagnosis because of immunological cross-reactivity among species. The need of a reliable diagnostic assay to detect larval cyathostomins is still obligatory. There is a big need for specific criteria in the diagnosis of verminous arteritis in horses. Thus, at the present time, PCR cannot be recommended as an efficient and reliable means of surveillance. Cy-GALA-1 identification in small strongyles infestation is an exciting advancement, but a semi quantitative assay has to be developed for authentication.

Epidemiology and Prevalence: Strongyles infestation can involve millions of nematodes covering a large range of species (Ogbourne, 1975; Ogbourne, 1976; Reinemeyer *et al*., 1984; Bucknell *et al.,* 1995; Osterman 2005). In a single host 17 different species have been recorded (Bucknell *et al*., 1995). More than 50 species of equine strongyles have been reported (Lichtenfels *et al*., 2002). Small strongyles involve 80% of the total parasite population in a horse. The highest incidence of infection in yearlings (nearly 90%) and the lowest (46.6%) in foals has been recorded. Egg production varies seasonally and it has been demonstrated that least egg production occurs in winter, rising during the spring with maximal production during August / September (Poynter, 1954). In Ontario, it was found that egg counts were high in August but during May to July were lesser for thoroughbred, standardbred and show horses than for pleasure or commercial animals. (Slocombe and McCraw, 1973). The percentage of large strongyles viable eggs was lowest in winter but maximum in May and remained high throughout the summer (Ogbourne, 1975). Higher egg excretion has been recorded in spring and summer (Herd, 1990, Saeed *et al*., 2010). Season has no impact on the prevalence of strongyle infections but shedding intensity of strongyle eggs is affected by season and significantly higher egg excretion was recorded in spring and summer (Nielsen 2012). Higher EPG were recorded in young horse (≤ 3 year) as compared with

older horses, no difference in the prevalence of strongyle infections as influenced by sex and excretion of eggs was also not affected by the sex of the animals (Saeed *et al*., 2010). L3s survival on pasture was estimated at 2-4 weeks in the summer wet season and 8-12 weeks in the autumn-winter dry season (April- August). Hot dry spring weather (pre-wet season) is the most unfavourable for larval development, migration and survival (Hutchinson *et al*., 1989). L3s were recovered from herbage samples from plots 3-4 weeks after the faeces had been deposited during May-October (Ramsey *et al*., 2004). The proportion of larvae successfully surviving during winter appeared to be maximal in faecal deposited on pasture in September of the previous year (up to 42.0% of the initial number of larvae). Larvae were observed surviving winter in soil beneath the faecal pats (Kuzmina *et al*., 2006). L3s are capable of surviving severe cold, especially under a protective snow cover which somewhat stabilizes the climatic variations (Urquhart *et al*., 1996). Usually, 90% of the adult worms are distributed throughout the dorsal and ventral colon, and the remaining 10% are found in the caecum (Reinemeyer *et al* 1984; Gawor, 1995; Collobert-Laugier *et al*., 2002). Prevalence of strongylosis is not affected by age or sex (Saeed *et al*., 2010). Heavy worm loads may be found in individuals of all ages, and horses may contribute to infected pastures throughout their lives (Osterman, 2005). The occurrence of S. vulgaris has greatly decreased in recent decades (Herd, 1990). In herds where anthelmintics have not been used to control parasites, the prevalence has remained high (Gawor, 1995).

We believe that the available studies indicate major climatic and seasonal differences in rates of development and persistence of free-living stages, specifically the infective stage L3. This enables us to focus anthelmintic treatments at times when adequate refugia are present. In broad terms, it is therefore necessary to avoid treatments of strongylosis during the winter months in cold temperate climates and during summer months in warm/hot climates, in order to retard the development of anthelmintic resistance. Since understanding of larval ecology is prerequisite for development of rational anthelmintic control programs therefore, more research is needed for a thorough quantitative description of strongyle larval bionomics and a better understanding of their basic epidemiology.

Control and Treatment: Usually, equines are treated with anthelmintic drugs to eliminate adult strongyles from the large intestines to prevent excessive contamination of pastures with eggs and L3s. Thiabendazole has been widely used and several other drugs have been developed or approved for use in adult horses, including benzimidazole, tetrahydropyrimidines and organic phosphorus compounds (Drudge *et al*, 1975; Tolliver, 1987; Drudge *et al*., 1984; Saeed *et al*., 2008). Thiabendazole at the rate of 250 mg/kg body weight given through stomach tube on two consecutive days is useful (Coffman and Carlson, 1971). Currently, there are three main classes of commonly-used drugs, categorised by their mode of action: the benzimidazoles (e.g. thiabendazole, cambendazole, fenbendazole and oxibendazole), pyrantel and the macrocyclic lactones e.g. ivermectin and moxidectin (Gasser *et al*., 2004). In the 1990's, treatment intervals practiced for adult horses were 8 weeks for ivermectin and 4–6 weeks for other anthelmintics. Many combinations of macrocyclic lactones (abamectin, ivermectin, moxidectin), including ivermectin combined with pyrantel (tetrahydropyrimidine) and ivermectin combined with praziquantel (pyrazinoisoquinolin derivative), a pharmaceutically formed generic paste containing ivermectin 4% were tested for their effectiveness to control gastrointestinal nematodes of horses (Toscan *et al*., 2012). Alike formulations of ivermectins had different efficacies calculated by reduction of EPG (Mariana *et al*., 2010). Stages of efficacy of the tested drugs varied against S. edentatus, S. equinus and S. vulgaris. The generic paste (ivermectin 4%) was less effective than the conservative drugs. The efficacy of Oxafex, Ivomec and Farbenda has been established as 94.7, 98 and 81% respectively on day l4-post medication. On day 28th post medication it was 100%, 96% and 86%, respectively (Saeed *et al*., 2008). Now a days it is recommended to reduce the treatment intensity significantly to holdup further development of anthelmintic resistance (Kaplan and Nielsen, 2010). In severe enteropathy the administration of non steroidal anti inflammatory agent is also required. Single IV dose of 0.6 mg.kg-1body weight meloxicam once a day is recommended for horses (Mahmood and Ashraf, 2010). There is general agreement that the traditional treatment at frequent intervals should be abandoned, and that parasite control be maintained with far fewer anthelmintics (Nielsen, 2012). Prevention by routine deworming of horses is unnecessary in all regions during the 6 month period that comprises the unfavorable season for strongyle transmission. During this interval, environmental conditions largely prevent new parasites from developing. Even if horses have high egg counts during that period, relatively few of those eggs can develop into adults. Therefore, the goals of parasite control are being accomplished by the climate, and compound treatment is not required (Reinemeyer, 2009). Biological control, especially the predacious fungi have established good potential as an adjunct for strongyle control and such a product could easily have a market in equine establishments. There is a rising market for nematophagous fungal spores, and the biotech industry must be encouraged to finalize and market a product for equine usage. Commercial products are being promoted in Australia (Edward, 2007). Since small numbers of

infective larvae may have grave effects on foals thus mares in foal or newly foaled should be regularly examined for strongyle eggs and treated with suitable anthelmintics. Climatic influences cannot effectively clean pastures from one grazing season to the next (Martin *et al*., 2007). The use of herbal compounds as anthelmentics against strongylosis is yet to be explored.

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