Equine Parasitology

The Control of Gastro-Intestinal Nematode Parasites in Horses with Emphasis on Reducing Environmental Contamination.
"A New Control Strategy for an Old Problem."

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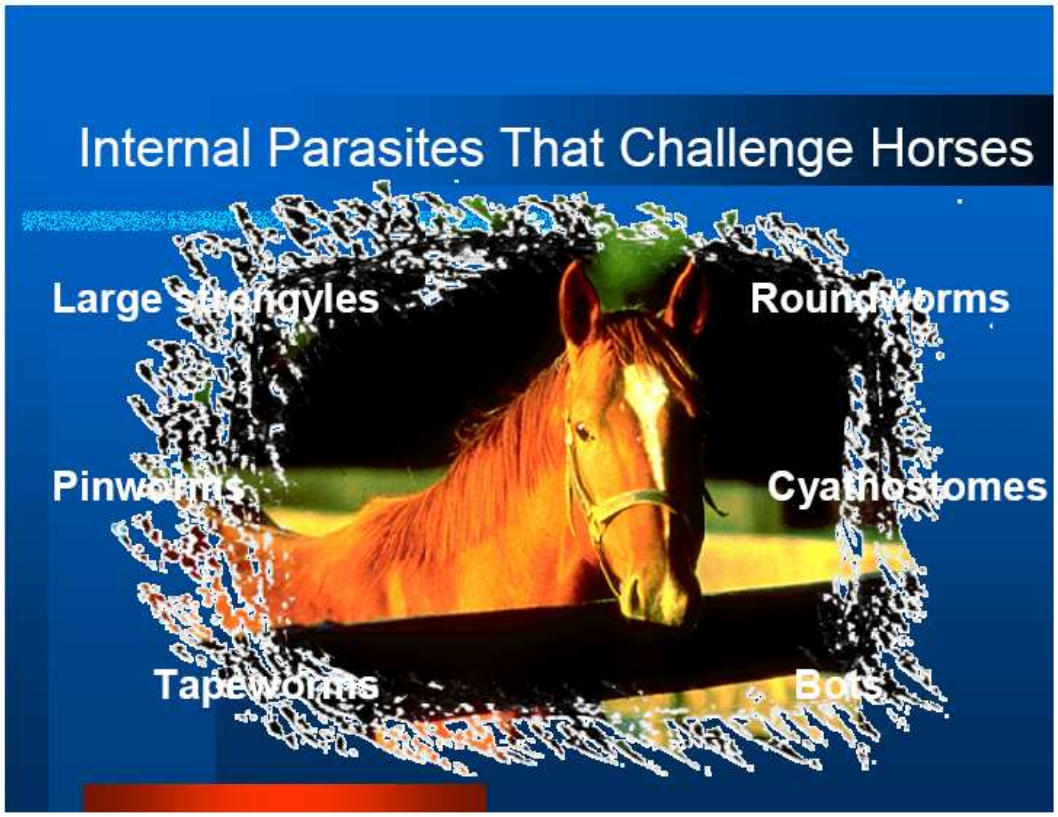
Swine, cattle and poultry raised in total confinement often exist free of gastro-intestinal nematode parasites, however, irregardless of how they're raised, horses rarely if ever, escape parasite exposure sometime in their life. Horses are said to have the largest collection of parasites of all domestic livestock. It is not unusual for a seemingly healthy horse to harbor over one-half million gastro-intestinal nematode parasites. These parasites cause damage to the animals both during the infection phase when the invading larvae are undergoing early development in various tissues of the body and then again after these larval stages have emerged and developed fully to adult parasites living in their final or predilection sites laying eggs back into the environment.

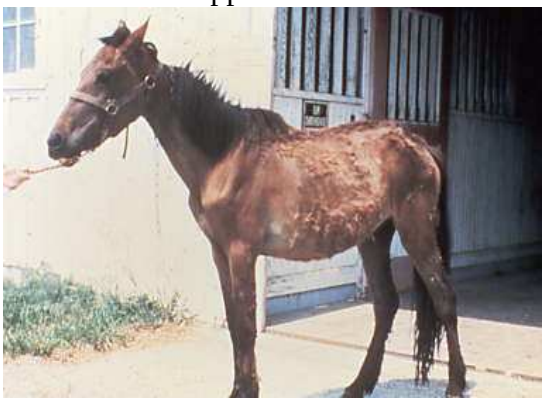
In the development phase, when tissue damage occurs, the immune system of the horse is negatively affected causing a cellular response directly proportional to the number of invading larvae. With daily exposure, the effect on the immune system can be very strong limiting the horse's ability to fight other disease problems at the same time trying to fight off a continuous stream of invading larvae. The problem comes from over-wintered larvae and worm eggs shed on the pasture early in the season that develop into infective larvae at an increasing rate as the temperature warms and summer approaches. Large numbers of these larvae can become infective over a short period, exposing the horse to high levels of parasitism. These free-living parasitic infective larvae present in the horse's environment are the foremost problem because they serve as the source for all new infections.

Overall, millions of dollars are spent every year for internal parasite control in horses; however, internal parasites remain one of the most important problems affecting the health and well-being of horses. The reason for this is that parasite control measures recommended and practiced over the past 25 years have provide limited protection to the horse because they remove infections after these infections have already developed and the damage to the horse is already done with little or no effect on reducing environmental contamination. If horse owners can develop a basic understanding of the infection process, changes in the way treatments are administered can be accomplished where the re-infection process that occurs in the environment can be reduced or prevented.



I. Gastro-intestinal Nematode Parasites Affect Horses in Many Ways.

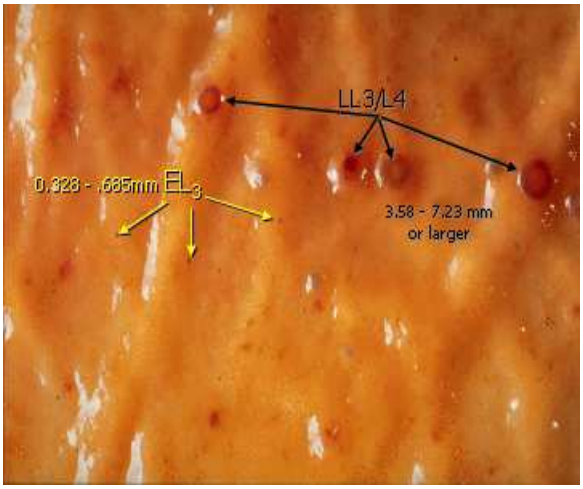
A. Clinical Parasitism is a condition where parasite numbers have reached a point that the negative effects of parasitism are visible. Animals with rough hair coat, potbelly, poor body condition and colic are examples of problems due to clinical parasitism. Clinical parasitism is complicated because it is interrelated to a number of variables including nutrition and immune status of the animals. Horses carrying heavy worm burdens can appear normal if nutrition levels are adequate to "feed the animal past the parasitism." If nutrition is inadequate, the animal may begin to develop signs of clinical parasitism (see Appendix II for a detail list of parasites in horses).



Animal that are allow to overgraze the pasture are at greatest risk for developing clinical parasitism because late in the season when the parasitic free-living larval populations on the pastures are at their greatest numbers, the nutrition of the pastures are usually at their lowest level. If left untreated, it is not uncommon for clinically affected horses to colic

and die from a heavy level of parasitism. Also, because of the coprophagic nature of horses, it is a very difficult task to maintain horses totally free of parasites without first reducing or eliminating environmental contamination by infective larval stages.

Most clinically infected horses harbor high numbers of adult parasites but also are carrying high numbers of encysted or inhibited larvae imbedded in the wall of the colon. As worm burdens build throughout the summer months, it appears that the physiology of the gastro-intestinal tract changes and conditions are no longer ideal for larval development. New incoming infective larvae then undergo a period of arrested development waiting in the tissues until the physiological condition of the gastro-intestinal tract returns to normal at which time these larvae resume development again. Since it is not in the best interest of the parasites to kill their host, the arrested development of larvae protects the host from being over-whelmed which also protects the parasites because if the host dies, the parasites also perish. The actions that triggers the release and re-development of encysted larvae into adult worms occurs when older worms die off naturally and are not rapidly replaced by new larvae especially during winter months or hot dry periods. The administration of a dewormer can also trigger the development of encysted larvae.



Once these larvae become encysted, their metabolism slows down and they become difficult to kill with conventional treatment because they are protected in the tissues. Since these larvae are in an inhibited state their uptake of chemicals dewormers intended to kill them is also reduced depending somewhat upon the chemical make-up of the deworming compound. Once encysted larvae begin development and emerge into the lumen of the colon, clinical disease can develop if high numbers of larvae emerge all at the same time. This is called "larval cyathostomiasis[1,2]." It's not unusually for some clinically infected horses to harbor over 1,000,000 encysted larvae at one time.

B. Subclinical parasitism is hard to see and measure. Subclinically infected animals appear normal but these parasitisms are responsible for reduced growth rates in foals; reduce reproductive rates in mares, reduced milk production for the young and a reduced ability of the infected animal's immune system to fight off other disease conditions. In performance horses, subclinical levels of parasitism can be very important because even slightly reduced performance may be very important. It only takes a few parasites to significantly reduce performance in a finely tuned animal.

Subclinical parasitism can be very costly because the owner is often unaware of the damage that is taking place since the parasites are not visible and lost performance can occur unknowingly. The most important aspect of subclinical parasitism, however, is the ability of subclinical infected animals to shed worm eggs into the environment producing

future infections. Subclinically infected animals, even with low worm egg counts, may be shedding thousands of eggs back in the horse's environment everyday.

Monitoring fecal worm egg counts is the best way to detect subclinical levels of parasitism (see Section IX). Positive results indicate a parasitic worm burden is present and contamination of the environment is taking place. High egg counts indicate a high level of contamination is already occurring. Also, fecal worm egg counts in horses often correlate better with numbers of adult parasites present than fecal worm egg counts in most other species. Horses with fecal worm egg counts conducted by the "Modified Wisconsin Sugar Flotation Technique" in excess of 300 eggs/3 gm sample are considered to be heavily infected while animals with egg counts over 1,000 eggs/3gm (150,000 eggs/pound of manure) sample are often showing signs of clinical parasitism

II. Parasites Develop Differently in Horses than in Cattle:

A. Parasitism in horses is most often an individual problem while parasitism in cattle is considered a herd disease because cattle often graze together in designated groups on the same pastures, are all exposed to the same infection level and subsequently develop similar parasite burdens. Domestic horses are different because they are seldom herded or handled in large groups. Millions of horses across the country are raised in isolation or semi-isolation where contact with other horses is limited to just a few animals. Even in equine operations with multiple numbers of horses, the animals are usually either maintained separately or in small groups with little cross-contamination between animals. In both cases, cumulative worm burdens are generated from exposure to the infective offspring found in the environment which developed from eggs shed by the horses themselves. Because of this auto-infection, parasitism in horses is a disease problem requiring special attention to individual animals and their immediate environment.

B. Horses routinely develop higher worm burdens than cattle especially under confined conditions. Grazing cattle can develop extremely high levels of parasitism depending upon their environment; however, unless cattle are overstocked on heavily contamination pastures, parasitism in cattle is usually subclinical in nature and can easily be controlled with strategic timed dewormings. The primary way cattle become infected is by eating forages contaminated with infective larvae. Feedlot cattle or mature dairy cattle on "full feed" seldom become re-infected while held in total confinement or dry lot conditions although calves can develop a "barnyard infection" with certain species of parasites while held in confinement especially when housed on a manure pack or in a crowded pen.

Horses tend to bit, chew or nibble at their surroundings often consuming parasite infected bedding and, therefore, can develop relatively high levels of parasitism even under arid conditions or while in total confinement[3]. Also, horses normally graze closer to the ground than cattle easily picking up large numbers of infective larvae while they graze. Because of these factors, horses can develop significant worm burdens depending upon environmental contamination whether they're housed in the stall, pen or on pasture.

C. Fecal worm egg output in parasitized horses is often much higher than parasitized cattle of similar age. Horses have higher average worm egg counts than cattle for a number of reasons. One reason is that horse manure is more concentrated and contains less moisture than cattle manure so the concentration of worm eggs shed per gram of manure is often much greater in horses. A second reason that horses tend to have much higher worm egg counts than cattle of similar age is that certain species of parasites such as the small strongyles are identified as more prolific egg layers than the common gastro-intestinal parasites (*Ostertagia*, *Haemonchus*, and *Trichostrongylus*) found in cattle. Overall, the most important aspect of high egg shedding is re-contamination of the environment leading to continued parasite exposure.

The number of worm eggs shed per gram of feces influences the contamination rate of the environment surrounding the animals. The more eggs that are shed into the environment, the greater the chance for re-infection to occur. Using the "Modified Wisconsin Sugar Flotation Fecal Technique" for floating worm eggs out of fecal material, it is not uncommon for a mature horse to have a fecal worm egg count greater than 1,000 eggs/3gm sample (150,000 eggs/pound of manure) whereas a count of greater than 100 eggs/3gm sample (15,000 eggs/pound of manure) is rare in mature cattle. As a general statement, the average fecal worm egg counts from horses routinely produce a 10 fold higher contamination rate when compared to cattle and is notably one of the main reasons for the ongoing failure to adequately control parasites in millions of horses across the country.

D. The economic value of deworming or cost of treatment is often less important with horse owners than with cattle producers. Cattle dewormers are often purchased and administered to the animals based on perceived economic benefit in terms of increased feed efficiency or growth whereas most horses are handled individually with personal care so treatments are given based on perceived need with less concern about cost versus benefit of the treatment given. Deworming costs for adult horses are also regularly more expensive that the cost for deworming cattle. Horse dewormers can cost from several dollars per dose to as high as $60.00/treatment with an average cost of treatment around $8.00 to $9.00/horse while treatment cost for adult cattle run from $1.80 per dose to a as high as $7.00/treatment with average costs of around $3.50/mature animal.

Many horse owners alternate dewormers to help prevent parasite resistance from developing, whereas, cattle producers often use products that are convenient, products that work well with their type of operation and products that match the season, i.e., cattle

producers may use an endectocide pour-on in late fall for lice and grub control while administering a medicated mineral or dewormer block in the spring. Because horse owners generally have lower concerns over product cost than cattlemen, the need to create a "least-cost most-effective" treatment program for horses has not been one of great concern for the horse industry. Because of this lack of economic concern, strategic deworming programs designed to reduce or eliminate environmental contamination by gastrointestinal internal parasites has not been widely researched or recommended.

E. The lack of scientific evidence that strategic deworming strategies are effective is a problem for horse owners when compared to cattle producers. Most cattle deworming programs are based on economic use data generated from carefully conducted trials measuring such parameters as growth rate, reproductive efficiency, and feed efficiency. These types of studies are seldom conducted with horses but rather horse owners are exposed to hundreds of treatment recommendations from a multitude of sources of which few provide scientific evidence that seasonal parasite control can be achieved by following the recommended program. Most of these deworming recommendations are confusing where one author recommends product rotation to prevent "resistance" while another author suggests that product rotation promotes "resistance" to all products use. Neither author provides any scientific evidence but rather provides their recommendations because it sounds like a "good recommendation." Horses all across the country are meanwhile suffering from unnecessary parasitism and parasite resistance has now become widespread such that horses can be exposed to increased levels of parasite exposure while their owners assume they're administrating an effective deworming strategy to their animals.

Equine dewormers are also easily available to horse owners where the owner purchases dewormers without knowing which products will work for their horses and which will not. The problem here is that if a particular product provides inadequate control due to the presence of parasite resistance, for example, millions of worm eggs can be shed into the environment before another deworming is given. A second deworming product may also not work. The only way owners can determine whether the products they're using are successful in their horses is to have fecal worm egg count exams conducted on a regular basis.

Another problem facing the horse industry is that thousands of horses are purchased and moved to new locations every year immediately contaminating the new location because care is seldom taken to confirm that animals are parasite-free before being moved. These horses usually have health records that outline deworming treatment history but animals can still be shedding worm eggs despite a recent deworming since parasite resistance could be present in the animals or sufficient time has elapsed allowing the animals to become re-infected since their last treatment. Two fecal checks should be conducted several weeks apart to determine a parasite-free status prior to moving the horse to the new environment. Using a larvicidal of fenbendazole (10 mg/kg daily for 5 days) is also recommended to remove all encysted small strongyle larvae.

III. Parasites Resistance to Dewormers has become a Major Problem:

A. Although horses throughout North America receive routine dewormings with excellent dewormers developed over the past 25 years, serious parasite problems have developed with "parasite resistance." The cause of parasite resistance to dewormers is not fully known. "Parasite resistance" is defined in the literature as determined by the extent to which parasites that survive drug treatment contribute their genes to future generation[4]. This contribution is influenced by the frequency and timing of treatment, drug efficacy, life expectancy and fecundity of the adult worms, rate of larval intake, egg deposition, grazing management and weather. With horses, the question exists, however, as to whether "parasite resistance" to dewormers is always true resistance or that some "resistance" may rather be "product tolerance" where many of the products used are unable to kill certain stages of the parasite especially while those parasites that are encysted in the wall of the intestine.

Control programs are numerous and recommendations vary from one expert to another[5,6,7,8,9]. Most experts agree that "parasite resistance" is a serious problem that is not going to go away on its own. However, little is being done to help the horse owner to prevent parasite resistance or to help solve the problem of resistance if it already occurs on an operation. Not only are programs not designed to eliminate environmental parasite contamination as the source of the problem, the widely recommended concept of repeated treatments given every 8 weeks to horses has caused and promoted the development of parasite resistance to many of the currently available deworming products.

B. Deworming horses often triggers the development of inhibited or arrested larvae in the wall of the colon making it appear as though "parasite resistance" is present. Most products have poor efficacy against encysted larvae which means that the larvae which survive treatment can emerge and develop to adult worms soon after the drug is gone from the horse's system. These newly developed adult parasite begin shedding eggs immediately and can give the appearance that "parasite resistance" had developed, instead, the eggs are coming from adults worms that developed from encysted larvae missed during the time of treatment. Depending upon the time of the year, the level of parasite infection present and the dewormer used, a population of inhibited larvae may be left in the animal after treatment. Within the population of arrested larvae that remain intact following treatment some of these larvae will begin development immediately following treatment such that in just a few days worm eggs can be detected in the feces of treated horses.

A recent investigation by the author for an occurrence of "parasite resistance" to fenbendazole in horses located in Ontario, Canada revealed a case where 100 yearling horses were housed in an outdoor snow-covered facility that experienced parasite problems despite treatment in early February with fenbendazole. Upon investigation, the horses had been on snow- covered ground since early November. On the 1st of December, all horses received ivermectin oral paste, on January 1, all horses received pyrantel paste at double dose and then on February 1, all horses received fenbendazole paste. Fecal samples taken two weeks following the fenbendazole treatment were positive for worm

eggs indicating “resistance.” In this case, however, parasite transmission had not occurred since early November when the ground was frozen and first covered with snow. The parasites found in the middle of February, therefore, must have survived treatment with ivermectin (December 1), pyrantel (January 1) and fenbendazole (February 1). The chance of the horses developing heavy infections between the time of the earlier treatments with ivermectin and pyrantel and the February treatment with fenbendazole is highly unlikely. This information indicates that the mature infections as indicated by positive worm egg counts probably developed from encysted larvae which survived the various treatments and emerged in February as adult parasites after the final treatment with fenbendazole. This more likely the case rather than the concept that these yearling horses suddenly became “resistant” to three different classes of dewormers all at same time.

C. Parasite control strategies recommended by manufactures for equine dewormers are not designed to prevent or to reduce environmental contamination. If the environment of an animal is heavily contaminated with infective larvae and treatment is given to an animal while exposed to daily ingestion of larvae, the administered drug treatment will kill the adults and those larval stages susceptible to treatment and leave those that are not susceptible. For example, most dewormers will not kill incoming infective larvae, early developing or encysted larvae so if these parasite stages are present at the time of treatment or are ingested during the next few days following treatment, they will survive treatment and continue development. Random treatment given to horses living in a heavily contaminated environment, therefore, accomplishes little in terms of controlling the existing parasitism, but rather only temporarily removes a percentage of parasites present and increases the risk of causing resistance to those present by being exposed but not killed by the deworming compound.

A parasite resistance study was conducted by the author with horses known to be harboring “parasite resistance” to fenbendazole. This study was conducted in four separate geographical regions of the country and demonstrated that when “parasite resistant” animals were removed from the source of parasite contamination and treated serially with the recommended dose of fenbendazole every 30-days, fecal worm egg counts were reduce to negative levels in 95% of the animals by the third treatment.

The question raised by this study was whether the parasites developed true resistance to fenbendazole or whether previous fenbendazole treatments removed only adult and late developing parasites allowing the encysted parasites to develop into adult egg-laying parasites soon after treatment making it appear like “parasite resistance” was present. Parasites that are truly resistant to a dewormer should be able withstand continued exposure to the compound and not be killed. In this case, the animals were move away from their original contaminated environment, thus no new parasites were ingested during the study. It is assumed, therefore, that the supply of encysted larvae in the colon was depleted by the third treatment thus worm egg counts dropped to negative levels in 95% of the animals following the third treatment.

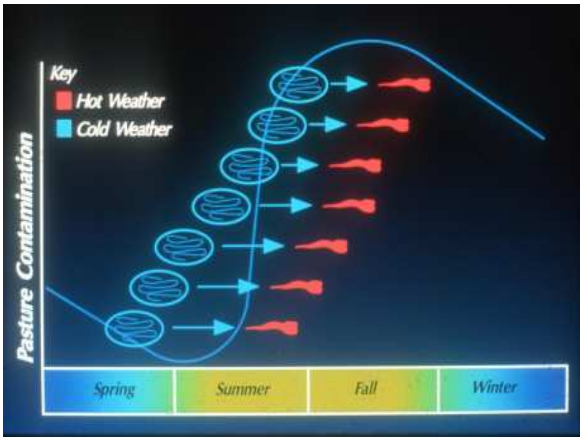
IV. The Seasonal Transmission of Parasites in the Horse:

A. Parasite infections come from the ingestion of infective larvae which develop in the environment of the horse. Most of the economically important gastro-intestinal parasites of horses have a direct life cycle as follows: adult parasites living within the horse lay eggs which are excreted in the manure; a larva develops within each egg, which then hatches, growing and molting twice until it reaches the infective third staged (L_3) larva. Egg development occurs outside the animal when weather conditions are warm and moisture is present. The development of eggs to infective larvae is slowed down or even stopped once temperatures fall below 45 degrees or when temperatures exceed 90 degrees. Development from the egg stage to an infective L_3 larva can take just a few days under optimal conditions or many months under suboptimal conditions.

Once infective, the larva needs to be consumed by a horse to re-establishes a new infection in the gastro-intestinal tract. In the horse, the ingested larvae undergo two additional molts during their growth process going from the infective L_3 stage to an L_4 larva and then to an L_5 or early adult stage. Male and female adult worms must both be present to produce viable eggs which then pass out with the feces starting the cycle over again. The time from when an infective larva is consumed until an egg-laying adult worm is present in the gastro-intestinal tract depends upon the worm species but can be anywhere from 4 weeks to 8 months.

The first (L_1) and second (L_2) staged larvae are not mobile but stay in the fecal material feeding off bacteria and fecal debris. These larval stages appear to be sensitive to adverse weather conditions; however, once the larva reaches the L_3 infective stage, it becomes more resistant to adverse weather condition and also becomes mobile, moving away from the fecal material by following moisture trails to reach nearby vegetation where it can be consumed by foraging animals. Infective larvae are encased with a sheath they acquire from the molt as L_2 larvae. This encasement protects the larvae from environmental conditions and immediately after ingestion (from stomach acids) before the outer sheath is shed and the new infection is initiated. The encasement covers the entire larva including the mouth parts and prevents the infective L_3 larvae from feeding. The infective L_3 larva must live off stored food material in the body of the larvae and, therefore, have a finite life span, once the stored material is gone, the larvae die. This especially important in the spring of the year when larvae which have over-wintered become active in the spring looking for a host animal. If a host is not found, these larvae will soon die because of limited energy from store food material.

Once infective, these L_3 larvae are mobile following moisture trails moving from the fecal matter onto the vegetation in order to re-infect the animals. The larvae that are consumed on a daily basis develop within the animals to mature adult worms releasing eggs back in the environment. These eggs develop into infective larvae which accumulate in the pastures often building up to very high levels by late summer or early fall. When the temperatures are cool, it takes longer for the infective larvae to develop than it does when the temperature is warm and moist. Often, the eggs that are deposited in the environment of the horse in early spring develop into infective at the same time as the



eggs deposited on the pasture later when the temperature is more favorable. Many times, larval build-up can reach as high as 10,000 larvae per square meters of grass collected. Once frost or freezing temperatures arrive, the larvae intake drops off as the larvae become immobile and are either killed or remain protected in the soil or under the vegetation. At this point, re-infection drops to low or negative levels.

Under heavy larval contamination of the environment, infective larvae can be found almost everywhere horses are present. Larvae can be found in the dirt, in bedding, around water troughs, on the animals themselves, and throughout the horses surroundings wherever the larvae can find protection from sunlight, desiccation, cold temperatures and other adverse environmental conditions. The infective L_3 stage survives in a protected micro-climate in the soil, in fecal pats, or under layers of herbage. These free-living infective larvae can live for an entire year in the environment if well protected from extreme heat, cold or drying. During periods of extreme cold or when conditions are very dry, the larvae become immobile waiting for warm moist conditions to return when it begins to become mobile again trying to find a host. Animals that are exposed to mild winter conditions or housed in heated barns can become exposed to parasitic larvae and develop active infections throughout the year.

B. Parasites can survive winter or hot summer conditions either as adult, inhibited larvae or infective larvae in the environment. The adult parasite within the horse have a finite life span, however, as the older parasites die off they are replenished by the larvae new incoming larvae or larvae that have emerged from the gut wall (in the case of the small strongyles), from the lungs (in the case of roundworms), and from the mesentery arteries (in the case of *Strongylus vulgaris)*. Infected horses then re-seed the pastures with parasite eggs which develop into infective larvae contaminating spring pastures. Animals that enter the spring months harboring parasites begin shedding worm eggs immediately while those which begin the spring season parasite free will not re-contaminate their environment until a new infection has developed from newly acquired infection off spring pastures. As temperature increase with spring developing, these eggs hatch and develop into infective larvae. The eggs that have been lying in the environment waiting for warm moist weather, many of these eggs will develop around the same time depending upon the weather causing high levels of contamination to occur once.

Pastures not grazed by horses from the beginning of the spring season for at least three months will become "parasite safe" pastures since the over-wintered larvae will have expired by this time and no new worm eggs have been released on the pastures. Any animals moving to "parasite safe" pastures should be dewormed prior to moving. Treating horses strategically to prevent shedding eggs during the first three months of the season will accomplish the same goal and the existing larvae will disappear by late June or early July and the pastures will be safe from parasites until fall.

C. Larval Transmission to the Horse: Transmission is the phase when infective larvae are consumed by animals and the infection process begins. The key transmission times are often in early morning or late evening when dew is on the vegetation and the animals are grazing. When the sun comes out it in the morning, the dew dries up and the larvae move back to the base of the grass. Rain also moves the larvae onto the vegetation where they can be consumed. When conditions are right for vegetation growth, conditions are also right for parasite development and infective larvae that survive the winters become active once temperatures warm up in the spring and move onto the vegetation where they are consumed by grazing animals. When the temperatures drop to low levels in the fall, the larvae become inactive and stay in the manure or in the soil out of harms way.

V. Seasonal Control of Parasites in Horses Can be Achieved by Reducing Parasite Contamination from the Environment:

A. Environmental contamination by infective larvae is the main deterrent to effective parasite control. If environmental contamination is not controlled, animals immediately become re-infected following treatment. Fecal worm egg counts taken weekly following treatment show that horses treated every eight weeks have little impact on reducing environmental contamination. Monitoring fecal worm egg counts following treatment also shows that many of these treatments are ineffective. This means with an eight week treatment cycle, the treated animals are free from shedding eggs for approximately four weeks and then shed eggs for four weeks every cycle. Even with a low egg count, the environment is quickly contaminated.

B. The concept of preventing parasite contamination of the environment can be accomplished by eliminating egg shedding back into the environment by strategically timed dewormings. Internal nematode parasites of horses can be controlled and, in some cases, eliminated with current deworming products by using these products at strategic times to prevent horses from re-infecting themselves or other horses that share the same environment. Auto-infection or re-infection comes from the environmental contamination of housing facilities, exercise areas, or pasture environment with infective larvae. Many times the environment of the horse, even in total confinement, can become highly contaminated with infective larvae such that dangerous levels of parasitism can develop within the horse unknowingly.

The most important part of keeping fecal worm egg counts at negative levels during the early part of the season is to monitor treatment to make sure the animals are not shedding

eggs into the environment. Whether a dewormer works in a particular horse depends on the species of parasites present, the number of parasite present, the number of inhibited small strongyle larvae present in the colon, and whether a particular infection is resistant to the dewormer used. Making sure the animals are not shedding worm eggs during the first three months of grazing reduces the build-up of infective larvae on the pasture later in the season.

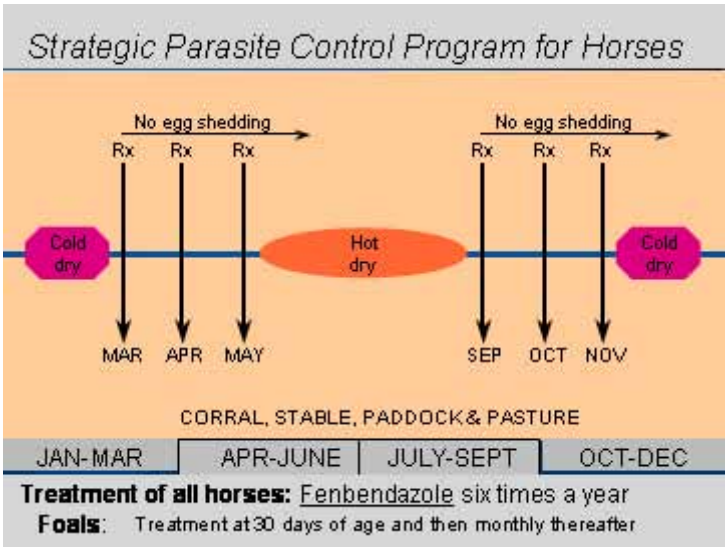
VI. General Recommendations for Strategic Timed Deworming for the Seasonal Control of Gastro-intestinal Parasites in Horses:

A. Successful deworming requires a seasonal program with multiple treatments given at strategic times to prevent the build-up of parasite contamination in the environment. A successful program is one that's designed to reduce both the number of parasites in the animal as well as subsequent parasite contamination of the environment. Treatments need to be strategically timed with the season when transmission is most likely to occur. Except for areas of the country where year around grazing is practiced, parasite transmission is limited during winter months. In most parts of the country, therefore, transmission occurs most successfully during the spring and again in the fall of the year. In parts of the country where year around grazing is practiced, all horses should receive repeated strategic dewormings during the rainy months so the animals will be free of parasites during the hot dry months of the year.

B. Phase I: **All animals raised in temperate areas of the country should be parasite-free during the winter months**. To begin the program, all horses should be parasite-free throughout the winter months and prior to the start of the transmission season in the spring. This includes making sure all animals are free from harboring encysted larvae acquired during the previous grazing season. The goal has multiple benefits, the first is to make sure the animals are free from harmful parasitism during the winter months, the second, is to make sure the animals are not shedding worm eggs at the beginning of the grazing season in the spring and, the third, is to make all mares are parasite-free at the time of foaling. The last treatment of the season should take place after the transmission season is over preferably in December. If post-treatment fecal exams indicate infections are still present after the December treatment, repeated treatment may be necessary including the use of the larvicidal dose of fenbendazole (10mg/kg daily for 5 days). All horses that are heavily parasitized (when fecal worm egg counts are over 300 eggs/3 gm sample) or horses that have not been dewormed on a regular basis should be dewormed with a larvicidal of fenbendazole to remove inhibited larval stages before starting the program.

C. Phase II: **Strategic Timed Spring Dewormings**: In the horse, treatment should be timed with the seasonal parasite life cycle on pasture where parasite development in the environment in most parts of the country is the greatest in the spring and the fall. To reduce the overall parasite contamination of the environment, three spring dewormings should be given one month apart in the spring and again in the fall. If the animals are parasite-free at the beginning of the spring season, the first treatment should be given approximately 30-days after the start of spring grazing. The repeated treatment works

because as animals pick up infective larvae which have over-wintered on the pasture in early spring, these larvae are killed with the first treatment before they can mature and begin laying eggs back in the environment of the horse. The horses continue to pick up more larvae, which are killed by the second and then the third treatment before they can shed eggs again. By preventing eggs from being shed for the first three to four months in the beginning of the grazing season significantly reduces parasite contamination for the next three months. With horses, three strategically timed dewormings given one month apart will provide approximately six months of safe grazing. The key to the success of this program is that the horses must be free of parasites at the start of the season so that the repeated treatments are simply removing the parasites picked up during each thirty day interval. If the treatments are successful no worm egg will be shed on the pasture for approximately 120 days, i.e., (1) clean to start, (2) three thirty-day treatments which provides 90 days without shedding and (3) another thirty days past the last treatment before mature worms can be present laying eggs into the environment.



Strategic timed deworming treatment should be given three times in the spring and fall one month apart as shown. The class of dewormer used can be interchanged as desired (see Section VIII). The last treatment should be given in late November or early December and may include both bot and tapeworm treatment if needed. Each "three-treatment strategically timed regime" provides approximately six months of control thus the spring treatment protects the horses until fall and the fall regime protects the horses until spring. These repeated treatments also help remove encysted larvae which may have survived in the horse through the winter months while preventing more from establishing throughout the entire grazing season by reducing the overall build-up of infective larvae in the environment of the treated animals.

VII. A Two Year Study Demonstrating the Value of Strategic Deworming to Reduce Environment Contamination:

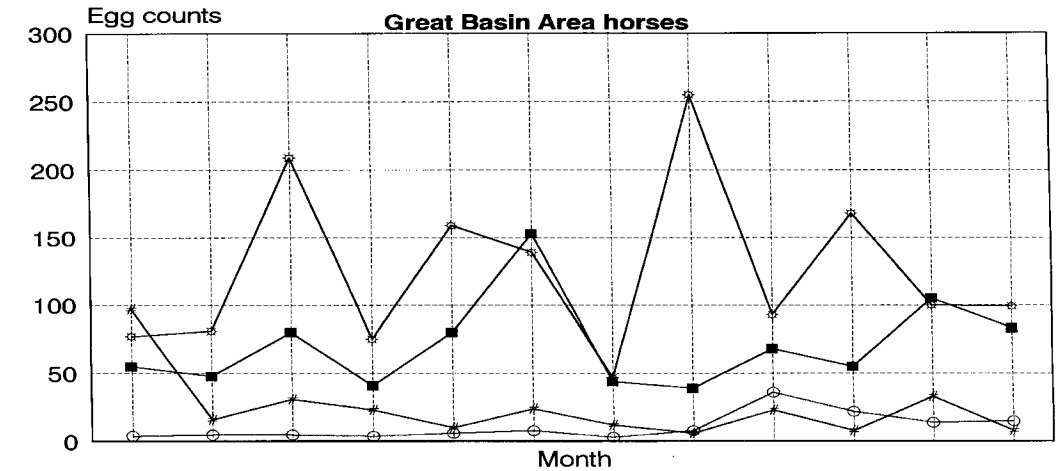
A two-year study was conducted with the University of Nevada to determine if strategic timed dewormings with fenbendazole (Safe-Guard®) given at three monthly intervals in the spring starting in March and three monthly intervals in the fall starting in October could be used to reduce environmental parasite contamination and to maintain horses safe from harmful levels of parasitism throughout an entire year. A total of 200 horses from local cooperating horse owners were used in the study. These horses were selected based on treatment history divided into five categories as follows: non-treated, infrequent

treatment (2 to 4 treatments a year), treatment every 8 weeks (6 treatments per year), and strategic treatment with Safe-Guard® (6 treatments per year) as describe above. The non-treated horses and the infrequent treated horses demonstrated a high parasite challenge throughout the year.

When fecal worm egg counts from strategically dewormed horses were compared with horses receiving no treatment, infrequent treatments and treatment every eight weeks, the difference was showing that worm egg counts stayed below 10 eggs/3 gram sample for eights months of the year increasing to a high of 36 eggs/3 gram sample in October but falling back below 20 eggs per gram by November (Figure 3). When comparing all treatments in the same figure, (Figure 3) the strategic deworming group maintained low levels throughout the year when compared to all of animals including those treated every eight weeks.

The non-treated horses average 125 eggs/3 gram sample throughout the two-year study period. The infrequently treated horses averaged 66 eggs/3 gram sample throughout the year while the horses treated every eight weeks averaged 24 egg/3 gram samples and the strategically dewormed horses averaged 11 eggs/3 gram sample. The fecal worm egg output in the strategically dewormed horses was reduced by 91.2 % compared to the non-treated horses, by 83.3% in the infrequent treated horses and 57.6 % in the horses treated every eight weeks (Figure 3).

FIGURE 3:



	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
None	77	81	209	75	159	139	47	255	93	168	100	99
Infrequent	55	48	80	41	80	153	44	39	68	55	105	83
6X/yr	97	16	31	23	10	24	12	6	23	8	33	8
Strategic	4	5	5	4	6	8	3	8	36	22	14	15

VIII. Equine Dewormers:

A. Equine dewormers currently on the market in the USA can be classified into three separate classes of compounds based on the mode of action (See Appendix I): These three major classes are: the benzimidazoles and pre-benzimidazoles (febantel, fenbendazole, oxibendazole, mebendazole and oxfendazole), the macrocyclic lactones (avermectin and moxidectin families), and the tetrahyo-pyrimidines (pyrantel). The mode of action is different for each class of compounds. The benzimidizoles are non-soluble compounds that destroy the metabolism of the parasites by interfering with the cell functions in the parasites and by preventing the uptake of food thus starving the worms to death. The macrocyclic lactones are very soluble compounds and affect the nervous system killing the parasites causing a non-spastic paralysis while the pyrimidines kill the parasites by acting on the nervous receptors causing a spastic paralysis.

All three classes of compound have excellent efficacy against the adult parasites, but since each dewormer class has a defined mode of action with a different level of activity against various developing and encysted larvae. The time it takes for larvae missed by treatment to develop into an adult parasite following treatment depends upon what larval stage the product is efficacious against. It takes longer for late L_3 larvae to develop into an adult parasite than it will for late L_4 larva. This difference can be measured in the time it takes for worm eggs to reappear in the feces following treatment. The longer it takes for eggs to reappear the more effective the product is against both the developing and encysted larvae.

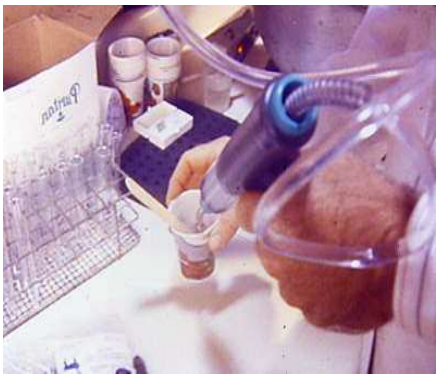
Using products correctly and understanding their characteristics can help keep all classes of products viable. Fenbendazole, for example, is an excellent product when used in a strategic deworming schedule. However, if parasite contamination is allowed to develop in the environment and parasite levels increases in the animals until a high population of encysted larvae are present in high numbers, the efficacy of fenbendazole at the recommended dose is drastically reduced.

Two key issues have been identified with fenbendazole that can affect its efficacy. The first issue is that this compound is not very soluble in liquids such as gastric juices or blood. The second issue is that it kills the parasite by destroying it's ability to metabolize food. Encysted larvae are in an arrested state with reduced metabolism and reduced absorption of nutrients. Because of fenbendazole's low solubility and reduced metabolism of the encysted larvae, the product needs direct physical contact to kill these encysted parasites. When fenbendazole is given at 10 times the recommended dose spread over a five-day period it is successful against both developing and encysted larvae (10 mg/kg given daily for five days). By flooding the gastrointestinal tract with molecules of fenbendazole, direct contact is made with the encysted larvae successfully killing them.

IX. Fecal Monitoring for Parasite Eggs is the Best Way to Determine Whether a Deworming Program is Effective:

The Modified Wisconsin Sugar Flotation Technique is a quantitative fecal exam that is conducted by floating worm eggs out of concentrated fecal material so they can be found and identified under a microscope**.** The fecal exam is a simple and effective way to check whether an animal is infected with gastro-intestinal parasites. Fecal worm egg counts, if conducted correctly, can also determine the general type of parasites present such as: pinworms, threadworms, roundworms (*Parascaris*), strongyles and tapeworms. The Modified Wisconsin Sugar Flotation Method is highly sensitive for use with horses (.3 egg/1 gram sample) such that negative counts indicate the absence of adult parasites. Repeated negative counts indicate a parasite-free status. Fecal worm egg count can also provide accurate information on total numbers of worm eggs shed per pound of feces which determines the rate by which an animal is re-contaminating its environment.

B. The best way to determine whether a dewormer is effective is to conduct a fecal worm egg reduction test (FECRT). This test is accomplished by conducting a fecal exam before treatment and again 10-14 days following treatment with a dewormer. Percent reduction in fecal worm egg counts due to treatment is calculated by subtracting the post-treatment egg count from the pre-treatment egg count divided by the pre-treatment egg count. If the percent reduction is not 90% or greater, "parasite-resistance" is a possibility and the animals in question should be monitored regularly until negative counts are achieved. Conducting a single fecal exam following treatment is also effective but knowing the level of egg shedding prior to treatment helps determine percentage reduction following treatment.



C. Taking Fecal worm egg counts should be an important part of the standard procedure for all equine operations. Monitoring fecals should be conducted at least semi-annually to monitor treatment progress. Fecal checks can be run any time of the year, however, one of the best time to sample horses is during the winter months to make sure the animals are parasite-free when environmental contamination is at it lowest point and to make sure the animals are not re-contaminating their environment immediately when grazing returns in the spring. Another good time to take fecals is in late summer or early fall in order to monitor whether the annual treatment program is reducing larval build-up on the pastures and subsequent worm build-up in the animals.

The Modified Wisconsin Sugar Fecal Technique: A 3-gram fecal sample is mixed with a saturated sugar solution designed to float the eggs out of the fecal matter. The samples are strained to remove large debris, then pour into a 15 ml taper test tube and centrifuged at a low RPM (<1000) for five minutes which increases the egg recover rate by a significant amount. After centrifuging, a few drops of sugar are added to form a meniscus on top of the tube. A cover slip is placed on the top of the tube for two minutes, removes and examined under the microscope at 40X magnification. Eggs are identified to type and counted.

The collection process is conducted using a sealable bag or baggie, invert the bag like a glove and pick-up freshly dropped fecal material the size of a golf ball from each horse to check for parasites. Keep each individual sample separate. Seal each bag and write on the bag to identify the horse and date of collection. Keep samples cool after collection since heat causes the eggs hatch and freezing distorts the eggs. For best results:

1. Refrigerate before shipping, then ship with a freezer pack or frozen water bottle.
2. Payment: Include a check for $5 for each sample sent.
3. Don't forget to include return address/fax number or e-mail address for returning sample results.

Ship to: **MidAmerica Ag Research**
3705 Sequoia Trail
Verona, WI 53593
Ph: 608-798-4901
midamericaagresearch.net

APPENDIX I:

VII. The Most Common Dewormers and Classes of Dewormers Available for Horses are as follows:

Class 1: **A. Benzimidizoles**

1. Fenbendazole: (Panacur®, Panacur ®Power-Pac, Safe-Guard®, Power-Dose™), Safe-Guard® Equibits™)
2. Oxfendazole: (Benzelmin®)
3. Oxibendazole: (Anthelcide® EQ)

B. Pre-benzimidazole

1. Febantel: (Rintal®, Cutter Paste Wormer®)

Class 2: **A. Macrocyclic Lactones**

1. Avermectins

i. ivermectin:

a. Zimecterin®, Rotectin®, Equimectrin®, Equalan®, Equell™, Equimax™, Ivercide™, Iversol™, Phoenectin™, Zimercterin® Gold, Combocare gel, IverEase™, Rotation™ 1, Agri-Mectin Equine Paste, Horse Health Ivermectin Paste.

2. Melbemycins

i. moxidectin:

a. Quest®, Quest® Plus, Combo Care™

Class 3: **A. Tetrahyopyrimidines**

1. Pyrantel pamoate:
   i. Strongid®P, Rotectin® 2,Pellet-Care P®, Strongid T®, TapeCare Plus™, Rotation™ 2, StrongyleCare™, Liqui-Care P™)
2. Pyrantel tartrate:
   i. Strongid®C, Strongid®C 2X, Nu-Image™ Guardian™, Continuex™, Equi-Aid CW™, Manna Pro™ Foal and Horse Pelleted Wormer)

APPENDIX II.

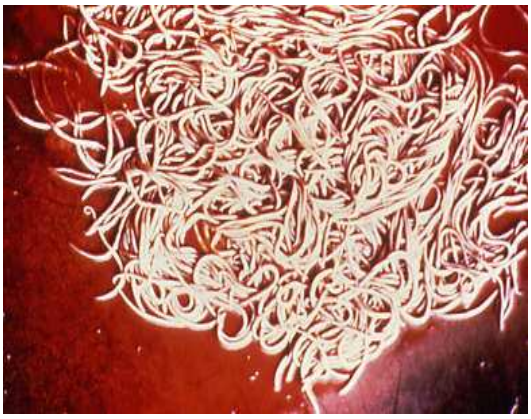
IIIa. The major gastro-intestinal nematode parasites of horses are:

A. Large Strongyles (*Strongylus vulgaris*, *S. edentatus*, *S. equinus* and *Triodontophorus*)
B. Small Strongyles (*Cyathostomes, Cylicocyclus, Cylicostephanus* and *Gyalacephalus*).
C. Large Roundworms (*Parascaris equorum*)
D. Pinworms (*Oxyuris equi*)
E. Threadworm (*Strongyloides westeri)*
F. Stomach worms (*Trichostrongylus axe, Draschia megastoma* and *Habronema musca, Habronema majus*).

III b. Other notable internal parasites of the horse:

A. Lungworms (*Dictyocaulus arnfieldi*)
B. Tapeworms (*Anopolocephalus magna, Anopolocephalus* perfoliata and *Paranoplocephala mammillana*)
C. Eyeworms (*Thelazia lacrimalis*)
D. Body Cavity Worms (*Setaria equina*)
E. Bots (*Gasterophilus intestinalus*).)

IIIc. Review of Internal Nematode Parasite of Horses:



A. Large Strongyles *(Strongylus vulgaris*, *Strongylus edentatus*, and *Strongylus equinus*) are reportedly the most damaging parasites in horses throughout the world, of which, *Strongylus vulgaris* is the most notorious of these parasites. Their life cycle is direct where the adults live in the large intestine (colon) and cecum and reproduce by laying eggs that pass back into the environment of the animals via the feces. As adults, these parasites are all plug feeders on the intestinal wall and are bloodsuckers. The prepatent period, which is the time it takes for an infective larva once ingested by a horse to reach an egg-laying adult parasite, can take as much as six and one-half months.

The reason it takes so long for these parasites to mature is because the immature stages undergo a period of migration through the body of the horse. These immature parasites cause considerable damage during the migration through the body on their way to the large intestine. Part of their migration is through the mesentery artery where excessive numbers can cause obstruction, fever, and shock-like symptoms. The larvae can remain

in the artery for three to four months causing severe problems including restricted blood flow resulting in diarrhea, aneurysm and colic, which may result in the death of the horse.

B. **Small Strongyles** (there are many species but most common are the Cyanthosomes) are non-migrating parasites that live in the large intestine. The immature

larval stages of this parasite develop in the walls of the intestine where they are protected until they emerge as early adult parasites. While they are in the walls of the intestine there are several stages of development when the larvae can encyst and undergo a period of inhibition or arrested development that is called hypobiosis; first, as newly arrived third staged larvae and second, as a late third staged larvae or early fourth staged larvae. When these larvae are encysted, they go through a period of arrested development or hypobiosis that can last from several months to as long as three years.

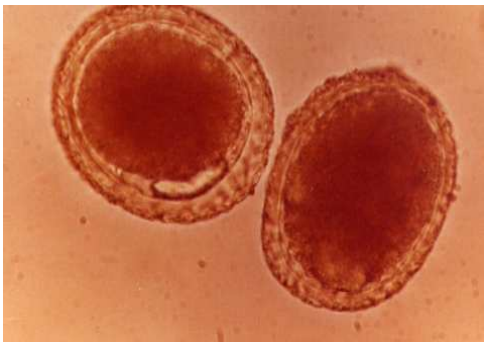
The physiological mechanism that causes hypobiosis to occur is unknown but hypobiosis is seasonal and occurs only when parasite populations or parasite density in the gastro-intestinal tract is the greatest. It's has become obvious, that the greater the parasite population, the more changes that occur in the physiology of the gastro-intestinal tract and the higher the percentage of incoming larvae that become encysted and undergo hypobiosis. The small strongyles as a group are the primary parasites of horses that are considered to the greatest "drug resistance" problems.

The problems for the horse come when these encysted cyanthostome larvae begin development again and emerge, rupturing through the intestinal wall. Usually this is a dynamic process with older worms dying off and new larvae replacing them. However, due a parasitic purge by a dewormer where a large number of worms are removed at the same time, mass emergence of encysted larvae often occurs causing severe gastrointestinal problems called cyathostomiasis. Clinical cyathostomiasis is often associated with the mass emergence of inhibited larvae from the mucosa of the cecum and colon which is a prominent cause of diarrhea in horses. Clinically, it is characterized by sudden onset of diarrhea, weight loss, subcutaneous edema and death. It is reported to be seasonal in occurrence and younger animals tend to be the most affected.

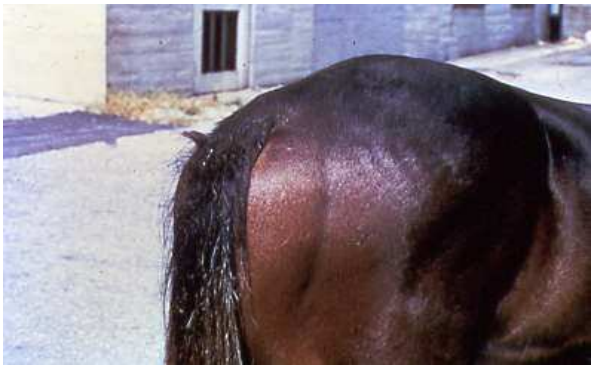
C. Large Roundworms (*Parascaris equorum*) is a very important parasite of horses especially for young foals. The infective larva remains within the egg and can survive for years in the environment. The prepatent period which is from the time on infection until an adult worm is present for Parascaris is reported to be between a1 and 12 weeks. Adult

roundworms are very prolific worms that can lay thousands of eggs, which pass back into the environment.

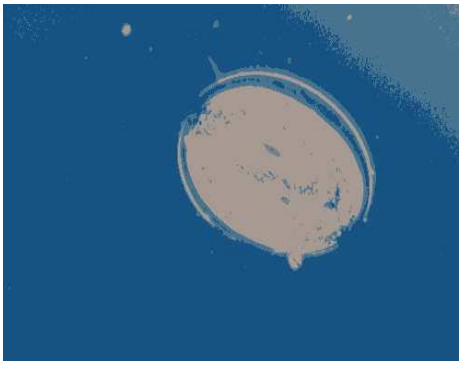
The transmission of this parasite is the results of foals ingesting the eggs from previously infected animals that have contaminated the environment where the foals are kept. The larvae hatch from the eggs soon after ingestion and migrate from the intestine through the liver to the lungs and then are coughed up and swallowed back into the small intestine where they grow to an adult parasite. Coughing and the development of pneumonia are common. Also, blockage of the small intestine by these very large adult parasites can be a problem in the young foal.



D. Pinworms (*Oxyuris equi*) are an annoying but not life threatening parasite of the horse. Visual evidence of this parasite is seen when infected horses continually rub their backsides against fence posts or other solid structures for relief. The female worm lives in the large intestine and rectum area and lays eggs in the skin of the perineal region, which can cause pruritus associated with this infection. The visual irritation of the horse observed by the owner makes this an important parasite. Dewormers that fail to control this parasite are obvious because owners consider continual rubbing by the horse a failed treatment.



E. Threadworm (*Strongyloides westeri*) is found in the small intestine of foals, usually less than six months of age. The parasite is transmistted either when suckling or by skin penetration from larvae living in the bedding. Foals can become infected during the first day of life and begin shedding eggs in the feces as early as six to ten days later. Young animals with unexplained diarrhea are often found to be infected with this parasite. Infections can be severe enough to require treatment, after which recovery will be rapid as the animals develop immunity against this parasite.

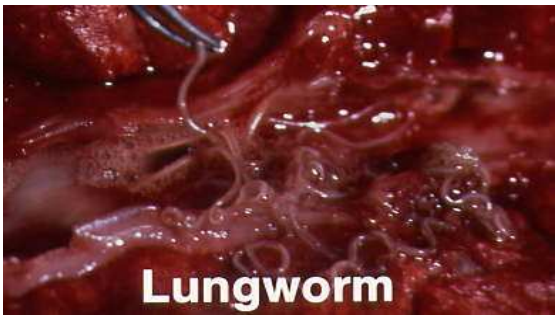


F. Stomach worms (*Habronema muscae*, *Habronema microstoma* and *Drachia megastome*) live in the horse stomach. Habronema species live under a thick mucus coat on the surface of the stomach while Drachia live in large nodules in the stomach wall. The housefly transmits both parasites.

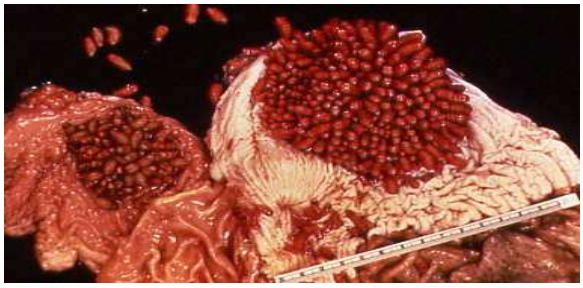
Other Parasites:

A. Tapeworms (*Anoplocephala magna, Anoplocephala perfoliate* and *Paranoplocephala mammillana*) are all acquire by the ingestion of orbit mites that live on pastures and in infested grain. Damage caused by tapeworms is limited to the attachment site causing thickening of the gut wall with some ulceration and enteritis. Tapeworms in horses are reportedly more common in northern parts of the country and in Canada than they are in southern parts of the country. Tapeworm presence is reported between 2% to 15% of the horses found infected[10].

B. Lungworms (*Dictyocaulus arnfieldi*) are more commonly found in donkeys and mules than in horses. Lungworms lay eggs that are coughed up and pass out of the gastro-intestinal tract as embryonated eggs in the feces. The eggs hatch soon after passing and the larvae develop into an infective free-living stage outside of the animals; move onto the vegetation with the aid of moisture (rainfall) to re-infect the horse. Young horses are somewhat more susceptible to this parasite than older animals and can show signs of respiratory stress which don't usually occur until two weeks after the onset of the infection.



C. Bots (*Gasterophilus intestinal* and *Gasterophilus nasalis*) are the larval stages of the non-feeding adult bot fly or nit fly. The adult fly deposits her eggs on the hairs of the legs, mane, or body and under the chin of the horses. The horses ingest the larvae that develops from the eggs when licking and then the fly bots eventually develop in the stomach of the horse. To complete the cycle, the bot larvae pass out in the feces and develop into the bot fly that starts the cycle over again. Bot larvae seldom are associated with any health problems in the horse.



D. Eye worms (*Thelazia lacrymalis*) are transmitted by the face fly. Some reports indicate that conjunctitis or keratitis is caused by this parasite. Necropsy reports prevalence as high as 30%.

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