Goat Parasites: Management and Control

Drs. Sue Stehman and Mary C. Smith Taken from presentations during the ECA Symposium on Goat Health June 3, 1995 Revised September 2004 by Mary C. Smith DVM

A. Epidemiology

Goats are very sensitive to the effects of internal parasitism (such as anemia and low blood protein). Parasitism can cause decreased fertility, abortion, unthriftiness, increased susceptibility to disease, and death.

The effect of parasitism is determined by the interactions between the type of parasites present in your geographic area, parasite life cycles, the environment including weather patterns and type of farm management, and the host factors.

Environment/Parasite Interactions

The local climate determines which species of parasites are present and their life cycle. **Temperature** determines the speed of development and survival of the free-living stages of parasites. For example, larvae may be killed above 104°F, develop optimally at 86-95°F (in 8-9 days), have delayed development for 14 days at 68-77°F, and survive without hatching for more than 30 days at 32°F.

Moisture allows larvae to escape from feces onto vegetation and prevents the larval stages from drying out. **Vegetation** density and height also play a role in transmission of parasites. Confluent and dense grasses provide a cooler, moister microclimate for larvae by shielding them from sunlight.

Parasites adapt to different environments with their own strategies. During extremes of temperature, larvae halt development and reside in the host in an arrested state (hypobiosis) until favorable environmental conditions return. Some species of larvae adapt by burrowing into the ground or by staying within protective egg casings.

Environment/Host Interactions

1. Grazing behavior - browsing results in less infection than grazing.

2. Consistency of feces - hard pellets require breakdown by rain or insects to spread larvae; loose stools spread parasites more efficiently.

3. Stocking rate - increased consumption negatively affects the microenvironment but also increases contamination.

4. Management System - intensive confinement versus extensive grazing have an effect on the type of parasite exposure and transmission.

The northern United States has a definite pasture season which has an impact on parasite populations and transmission to animals on pasture: populations and transmission increase rapidly in the spring, decrease in the summer, then peak in the fall. Parasites survive the freezing temperatures of winter by persisting in an arrested larval state in the wall of the gastrointestinal tract during the winter. In the south the arrested state occurs during the heat of the summer; larvae can survive on pasture through the milder southern winters.

Parasite/Host Interactions

1. Parasites can evade control by developing drug resistance or by arrested development in the face of pre-existing host immunity. Resumed development occurs when host immunity wanes, as with concurrent disease, or during the hormonal changes of parturition and lactation (egg production is the highest 1-4 weeks post partum).

2. The host can be genetically less susceptible to parasites or acquire resistance with age in the face of continued, low exposure.

B. Diagnosis

Sampling of sentinel animals before and after grazing season plus information gathered at herd visit such as inspection of breeding, kidding and weight gain records, examination of pasture and housing, examination of different age groups in herd.

< Minimum of 5 samples or 5-10% of each group should be sampled, selecting animals that are representative of the problem, i.e. clinical as well as subclinical or asymptomatic animals should be sampled from each group.

Samples should be labeled and taken from well identified individuals rather than groups. Fecal samples should be taken from the rectum rather than the ground (unless very fresh) and submitted in plastic bags or tightly sealed containers. Samples may need to be sent refrigerated (but not frozen) during the summer. Samples should not be taken after anthelminthic treatment unless the efficacy of the treatment is being evaluated. When treatment efficacy is being evaluated, take samples after treatment takes effect, usually 7-10 days post treatment.

Interpretation - using fecal examination, the number of eggs in the feces may not always correlate with the number of parasites present *in the individual sampled*. Egg and larval counts are not always accurate for the following reasons: (1) eggs are sometimes intermittently secreted; (2) some worm species are more prolific egg producers than others; (3) host factors such as lactation and parturition result in increased output (periparturient rise) and immunity results in decreased egg output; (4) larval stages may be causing parasitism without production of eggs; and (5) hard fecal samples may contain more eggs than dilute, watery, unformed fecal samples. *However, if enough animals are sampled which are representative of the problem, egg counts usually reflect the type and severity of parasite infection <u>in the herd</u>.*

C. Nematodes - Stomach and Intestinal Parasites

General Life Cycle

1. Adults live in the stomach or intestine, produce eggs which are passed in the feces. 2. First stage larvae (Ll) develop within the egg within 1 day then break out of the egg and molt to L2. L2 molt to the infective stage L3 usually within 7-10 days if temperature and moisture are optimal.

3. L3 migrate to the tops of grasses, are ingested, burrow into the intestine, and develop into L4 in the host within 1-2 days. L4 remain in the tissue for up to 10 days, then return to the surface and molt into the adult stage. The whole cycle takes 3-4 weeks.

There are a large number of species of nematodes which live at different levels of the gastrointestinal tract. Most infections are mixed but three species stand out in their pathogenicity; *Haemonchus, Ostertagia (Teladorsagia)*, and *Trichostrongylus*.

Haemonchus contortus: economically important; one of the most serious parasite pathogens, 4th stage larvae and adults are aggressive blood feeders and live in the 4th stomach (abomasum). Clinical signs include: anemia, bottle jaw, weakness, reluctance to move, exercise intolerance, and weight loss.

Ostertagia (Teladorsagia) circumcincta: larvae live in the gastric glands of the stomach, cause inflammation and leakage of protein into the intestinal tract resulting in low protein, bottle jaw, and diarrhea.

Trichostongylus sp.: Larvae live in either abomasum or small intestine and cause problems similar to *Ostertagia*.

Clinical signs include loss of condition and appetite, poor growth, dull attitude, diarrhea, bottle jaw, pot-bellied appearance and death.

Treatment includes supportive care (fluids, good nutrition) and deworming with anthelminthics.

D. Protozoal Parasites:

Coccidia

Coccidia are host specific; some species are more pathogenic than others (*Eimeria ninakohlyakimovae* most pathogenic). Infection does not always equal disease. Resistance to infection increases with age but is specific to the infecting species of coccidia and depends on continuous exposure to the parasite. Adult goats normally shed low numbers of oocysts but are usually resistant to clinical disease unless they are exposed to a new species, an overwhelming dose, and/or stressors such as shipping, weather or feed changes, lactation, or concurrent diseases.

Clinical disease, which is usually seen in 3 - 5 month old kids, starts two weeks after ingestion and is caused by destruction of the intestinal cell wall by the organisms. Kids are most susceptible at weaning but coccidiosis can be seen as early as two weeks of age. Angora kids Goat parasites: management and control (revised September, 2004) Page 3

may present with coccidiosis in the fall when they are weaned, shorn and introduced to concentrates. Clinical signs in kids include: abdominal pain, diarrhea, +/- fresh blood (not as common in goats as in calves), anaemia, weakness, weight loss and dehydration. If kids survive clinical disease, compensatory weight gain occurs but survivors can be unthrifty and require additional time to match weights of uninfected animals of the same age. Subclinical infection can result in poor weight gain, poor hair coat, and an unthrifty appearance.

Life cycle: two to three weeks for the entire cycle.

1. Unsporulated oocysts passed in feces. Sporulate in 2 - 5 days if conditions are optimal (i.e. oxygen and moisture present, and temperatures between 24° C and 32° C or 75° -90°F). Oocysts do not survive below -30 or above 40°C. Oocysts and sporocysts can survive for more than a year in the optimal temperature range if not exposed to sunlight or extreme drying.

2. Sporulated oocysts ingested and immature stages go through a complex series of replications and changes in the wall of the intestine which amplifies the infection and causes damage to the intestine.

Diagnosis

Most goats shed oocysts in feces, therefore the presence of oocysts on fecal exam does not confirm a diagnosis of coccidiosis. Finding several thousand oocysts of a pathogenic species may be indicative of a herd problem. Appropriate clinical signs in conjunction with an appropriate history lead to a presumptive diagnosis of coccidiosis. Necropsy findings are diagnostic.

Treatment

Supportive care including oral or parenteral balanced electrolytes is indicated in clinical cases. In severe cases, antibiotics are indicated to prevent bacterial invasion of the disrupted intestinal wall with subsequent septicemia. Coccidiostats only control the early stages of development and may not be effective in animals showing clinical signs unless a mixed species infection is present with some organisms still in the early stages of infection. Sulfonamides, (avoid use in dehydrated animals) and amprolium (Corid®, MSD Agvet) given for 4-5 days can be used for treatment of clinical coccidiosis.

Control

Sanitation and reduction of stress are important to control. Dry bedding, prevention of fecal contamination of feed and water and reduction or removal of feces will decrease build up of coccidia in the environment. Most disinfectants do not work. Sunlight and drying will decrease numbers. Provide adequate nutrition; avoid overcrowding and handling stress to manage the disease. Do not mix younger kids with older kids or raise them in an area where older kids have been. Try to limit the age spread in a group to 2 weeks. Coccidiostats should be used for 30 days before anticipated outbreaks to control the severity of disease.

Coccidiostats:

Decoquinate (0.5 mg/kg - 1 mg/kg body weight; or 2 lbs of 6% Deccox® to 50 lbs of salt) and monensin (20 g/ton of feed) are approved for use in goats. Can be incorporated into Goat parasites: management and control (revised September, 2004) Page 4

creep feed or into salt but uptake must be monitored to ensure the appropriate dose. Treatment failures occur if the dose is inadequate. Treatment of late pregnant does will decrease oocyst shedding from dams and thus decrease contamination of the environment prior to kidding. Lasalocid (Bovatec®) is approved for use in sheep. Amprolium, though not approved, can be used by prescription in both species for prevention (25-50 mg/kg/day for 21 days) and treatment (5 days).

Cryptosporidiosis

Causes clinical disease in goats at less than four to six weeks of age. Disease presents as a self-limiting, usually nonfatal diarrhea in 5 - 10 day old kids. Infection is more severe in kids lacking colostrum. Death can occur if dehydration and electrolyte imbalance become severe enough. Treatment consists of supportive care against dehydration. The oocysts can infect other animals and humans and are very resistant to environmental conditions and disinfectants. Kids with diarrhea should be isolated and strict sanitation of feeding utensils, waterers and pens should be implemented to prevent buildup of the organism in the environment and spread of the infection. People handling sick kids should not handle healthy kids without using sanitary precautions between groups and to prevent their own exposure.

Toxoplasmosis

Causes late term abortion. There is no treatment though pregnant ewes treated with 15 - 30 mg of monensin daily had fewer abortions. Transmission is controlled by preventing contamination of feed and hay with feces from cats (especially young kittens).

E. Cestodes: Tapeworms

Moniezia spp. are the most common and usually do not cause clinical disease except with heavy infections. Eggs passed in proglottid segments in feces are taken up by mites living on pasture. An intermediate stage develops in the mite over 4 months which matures to the adult tapeworm in the host after the mites are consumed during grazing. (Prepatent period or time to egg production is 40 days.)

* Rarely causes ill thrift and weight loss in kids during first summer on pasture. Very severe infections can cause intestinal obstruction predisposing to enterotoxemia and, potentially, intestinal rupture.

*Treatment: fenbendazole at 15 mg/kg po; albendazole 10mg/kg; oxfenbendazole (10 mg/kg); and oral niclosamide 50 mglkg.

F. Trematodes: Flukes

General Life Cycle

Adults live in the bile duct; eggs passed in feces of host, develop in water to free living stage which infects certain species of snails (10-12 days). Usually in spring and summer.
 Immature stages develop in the snail, are released in water and attach to grass (5 weeks to 10 months)

3. Ingested by grazing host, penetrate intestinal wall, migrate to the liver, penetrate the capsule, migrate to the bile ducts (6-7 weeks). Mature to adults in 2-4 weeks. Clinical disease is seen in fall and winter.

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Fasciola hepatica - common liver fluke

* **Key Points**: need the right snail host; and the right combination of temperature and moisture. This species does not grow in the Northeast unless imported in infected animals from the south. Clinical signs include: poor appetite, lethargy, weight loss; acute cases die suddenly. Treatment: albendazole (not in first 3 months of pregnancy) at 15 mg/kg orally or clorsulon at 7 mg/kg (sheep dose); neither drug is labeled for use in goats and must be prescribed by a veterinarian.

Fascioloides magna - large American liver fluke, natural hosts are deer and elk and the parasite is found in the Adirondacks. Goats become infected in late summer when grazing swampy areas also inhabited by wild ruminants and develop clinical signs (usually sudden death) in January or February. One fluke can kill a goat. Control by fencing off marshy pastures or prophylactic treatment with albendazole one month after the first killing frost.

Dicrocoelium dendriticum - small liver fluke, life cycle includes snails and ants. Infection is widespread in North America; infection can occur in spring as well as late summer. Clinical signs such as chronic weight loss and depression are rare but livers may be condemned at slaughter. Treatment: albendazole (not in first 3 months of pregnancy) at 15 mg/kg orally.

G. Lungworms - 3 primary types:

Dictyocaulus filaria - direct life cycle, most pathogenic; adults live in the bronchi; Ll are coughed up, swallowed and passed in feces; L2 - L3 development takes 1-2 weeks; L3 (long lived in moist, cool environment) is eaten, and develops into L4 in the lungs where it can overwinter. Adults produce eggs within one month. Kids can develop signs in the spring but fall is a more typical time. Clinical signs include increased respiratory rate, difficult breathing and coughing. Ivermectin, levamisole and fenbendazole will kill larval stages and adults.

Muellerius and *Protostrongylus* - have an indirect life cycle with a snail intermediate host. *Protostrongylus* lives in the bronchi and usually does not cause clinical disease except in kids. *Muellerius* lives in the lung parenchyma and is usually subclinical but can cause weight loss and coughing if infestation is heavy. Levamisole is not effective. Fenbendazole: (2 doses) at 15 mg/kg or ivermectin will kill larval stages.

Parelaphostrongylus tenuis, the meningeal worm, naturally infects white-tailed deer with no apparent ill effects. In goats, *P. tenuis* causes severe neurologic disease due to aberrant migration of the parasite through the spinal cord. The life cycle is indirect with terrestrial snails as intermediate hosts. Keeping goats off fields frequented by deer will limit infections.

H. Practical Control Measures:

The type of control depends on the type of management system on the farm. Intensive, confinement housing controls most nematode infections but allows build up of coccidia. Semiconfinement with summer pasturing can lead to nematode problems in the summer and fall, especially in young kids. With extensive grazing or year round use of pasture, parasite problems will depend on interactions between climate, stocking rate and type of vegetation.

1. Strategic use of dewormers – these are times to consider using dewormers on susceptible, infected animals

- pre-breeding (fall) to decrease over wintering of arrested larvae and to improve body condition, reproduction, and kid birth weights.

- late gestation or during early lactation to treat peripanurient rise and to decrease parasite load being introduced on to pasture.

- treat and move to safe pastures which have not been grazed before or for at least 3 months. Treating before moving does select for parasite resistance.

- monitor fecal shedding during the summer and treat and move as needed.

2. **Good pasture management** - avoid over stocking, rotate before eaten to the ground, manage areas around feeders and watering areas, co-mingle grazing species. Strip grazing areas must be large enough to avoid severe buildup; small strips must be changed more frequently.

3. Increasing efficacy of the deworming program

* Goats metabolize anthelmintics differently than sheep; higher doses are required of levamisole (1.5x - more is toxic) and ivermectin (2x sheep dose needed to treat *H. contortus*).

* Weigh or tape representative animals to determine proper dose. A weight tape is available from Caprine Supply http://www.caprinesupply.com/ or 800- 646-7736.
* Hold off feed overnight to increase efficacy (not in late pregnancy, because of risk of pregnancy toxemia). Ideally, treat two days in a row.

* Avoid frequent rotation of dewormers - ideally, stay with one drug for an entire year.

* Do not use injectable or pour-on anthelmintics, as these select for resistant parasites.

* Do not use moxidectin (Cydectin®) unless no other drug works on your farm. This is the last resort drug and its efficacy must be preserved as long as possible.

* Do use an adaptor that allows delivery of the dewormer over the base of the tongue. This device is available from Pipestone at http://www.pipevet.com/ or 800-658-2523. * During the grazing season, monitor for clinical signs (anemia, diarrhea, bottle jaw) and treat the affected animals but leave goats that are in balance with their parasite load untreated. This slows the development of resistant parasites by leaving unselected worms in the population. Record the identity of animals requiring frequent treatment and cull them and their offspring to increase genetic resistance of the goats to parasites. The FAMACHA program is described at http://union.ifas.ufl.edu/03j-goatprogramj.doc>.

4. General Considerations

- Consult your veterinarian for fecal exams and control programs.

- Avoid small grassy exercise lots.

- Avoid deworming in the first month of pregnancy, when drugs might cause birth defects.

- Minimize treatments to slow development of resistance.

- Consider treating two days in a row to increase efficacy.

- Consider fasting overnight before treating to increase efficacy.

- Avoid milk residues in dairy goats; deworm while dry if possible or use morantel.

- Observe meat withdrawals in meat goats and culls.

- Inject liquids over the base of the tongue using a Sharpvet Dose Syringe (available from Pipestone at http://www.pipevet.com/> or 800-658-2523.

- If giving bolus (pill), use smooth metal balling gun very carefully to avoid injury to back of throat.

I. Chemical dewormers:

Thiabendazole, TBZ®

- Licensed for use in goats but no longer marketed.

- Withdrawal was milk 96 hours, meat 30 days.

Fenbendazole, Panacur®, Safeguard®

- labeled for goats at 5 mg/kg but 10 mg/kg correct dose for strongyles

- use by veterinary prescription.

- approved for dairy cows with zero milk withdrawal

Spectrum - abomasal and intestinal strongyles, lungworms, tapeworms

Dose - higher than sheep and cattle; at least 7.5 mg/kg; commonly use 10 mg/kg, 15

mg/kg for tapeworms

Route - oral

Withdrawal - milk recommendation 96 hours at 10 mg/kg based on goat studies; meat at least 8 days based on label for cattle.

Albendazole, Valbazen®

not approved for goats, is labeled for sheep at 7.5 mg/kg
Spectrum - abomasal and intestinal strongyles, flukes, tapeworms, lungworms
Dose -15 mg/kg
Route - oral
Withdrawal - meat 7 days (sheep label), not to be used in dairy animals

Morantel, Rumatel®

- approved and labeled for lactating dairy goats

Spectrum - abomasal and intestinal strongyles; not absorbed.

Dose - added to feed, same dose per pound as for cattle

Route - oral

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Withdrawal - milk zero; meat 14 days

Levamisole, Levasole®, Prohibit®

- not approved for goats, is labeled for sheep

- reputation for causing abortion

- causes frothing, muscle quivering in some goats even at normal dose.

Spectrum - abomasal and intestinal strongyles, large lungworms

Dose - 8 to12 mg/kg . DO NOT overdose - toxicity common.

Route - oral (some use cattle injectable but greater risk of toxicity)

Withdrawal - milk not known, not approved for dairy cows;

meat at least 3 days based on sheep oral label, at least 7 days based on cattle injectable label

Ivermectin, Ivomec®

- not approved for goats, is labeled for sheep and cattle

- use with veterinary prescription

- injectable product stings and should not be used, to avoid selecting for resistant worms. **Spectrum** - abomasal and intestinal strongyles, lungworms, biting lice (not tapeworms, flukes or biting lice).

Dose - routinely double that for cattle, sheep, horses; total resistance common in goats in Texas.

Route - oral (sheep drench, horse paste); pour-on (cattle product) not evaluated for efficacy in goats and would select for resistant worms.

Withdrawal - DO NOT USE IN LACTATING DAIRY GOATS.

Ivermectin contaminates the goat's milk. FARAD withdrawal recommendations for goats (JAVMA 217:668-671, 200) are

Oral 0.2-0.4 mg/kg - 14 days meat, 9 days milk SC 0.2 mg/kg - 35 days meat, 40 days milk

Moxidectin, Cydectin®

- not approved for goats.
- by veterinarian's prescription
- should not be used if any other dewormer is still effective on the farm
- FARAD withdrawal recommendations for goats are
 - Oral 0.2 mg/kg 14 days meat
 - Oral 0.5 mg/kg 23 days meat
- do not use SC
- do not use in dairy goats