Sustainable Non-synthetic Gastrointestinal Parasites Control in Small Ruminants

Tuskegee University
Cooperative Extension Program

Editor: Byeng Ryel Min, Ph.D.
July 2017
Sustainable Non-synthetic Gastrointestinal Parasites

Control in Small Ruminants
Preface and Acknowledgements

This handbook has been developed to train field level Extension and Technical assistance personnel, who are involved in educating and helping goat producers. A wide range of topics relevant to alternative parasites control management, especially focusing on small ruminants, are included in this handbook.

Development of this handbook became possible from the funding support of Southern Sustainable Agriculture Research and Education (SSARE) and George Washington Carver Agricultural Experiment Station (GWCAES), Tuskegee University, AL. Experts from various institutions (Fort-Valley State University, GA, Louisiana State University, LA, University of Georgia, GA, and Langston University, OK. I greatly appreciate the contributions of all the authors and reviewers.

Byeng Ryel Min, Ph.D.
Editor
TABLE OF CONTENTS

Chapter 1. Understanding of gastrointestinal parasites in small ruminants

Chapter 2. How and why resistance to worm remedies develops

Chapter 3. Conventional method to control internal parasites. Combination of Dewormers: The Time is Now!

Chapter 4. Potential solutions to highly resistant worms in sheep and goats

Chapter 5. Alternative Methods for Managing Gastrointestinal Parasites and *Eimeria* spp. in Small Ruminants

Chapter 6. Non-chemical control of parasites in small ruminants through grazing management and nutrition

Chapter 7. Overview on Enhancing Immune Responses to Internal Parasites in Small Ruminants

Chapter 8. Decreasing Barber Pole Populations on Grass Pastures: Is Liquid Nitrogen Fertilizer a Viable Alternative?

**Seminar presentation**
Chapter 9. An update on anthelmintic resistance on goat farms in the United States

Chapter 10. *SERICEA LESPEDEZA* for parasite control

Chapter 11. Sustainable Non-synthetic Parasites Control in Our Farm The way to go?

Chapter 12. Nutrition, Feeding, and Immunity against parasites
Chapter 1. Understanding of gastrointestinal parasites in small ruminants

James E. Miller DVM MPVM PhD DipACVM

Department of Pathobiological Sciences
School of Veterinary Medicine, Skip Bertman Dr., Louisiana State University
Baton Rouge, LA 70803
225-578-9652 (Office), 225-578-9701 (Fax), jmille1@lsu.edu

Gastrointestinal nematodes (worms)

There are a number of worms found in small ruminants. The predominant and most pathogenic ones are found in the abomasum (stomach) and small intestine. Haemonchus is the most pathogenic and predominates in warm climates. Teladorsagia is secondary and predominates in cool climates. Trichostrongylus is can be important in both climates.

General life cycle

It is important to understand some aspects of the life cycle of these worms. Part of their life cycle is spent inside the animal and part on the pasture (Figure 1).

Worms mate in the host and eggs produces by females pass out in the feces. Eggs hatch and develop through 2 larval molts to infective larvae while remaining in the feces. The infective larvae then migrate out of the feces, when a moisture medium (rain, heavy dew, irrigation, etc.) is present, onto the surrounding forage where they can be consumed during grazing thus completing the cycle. The prepatent period is the time from ingestion of infective larvae to egg laying adults, and is about three weeks. Development from egg to infective larvae can be short (7-10 days during warm summer months), so pasture contamination and reinfection can be quite rapid. Larval development on pasture is delayed during cooler months and can take a month or two to reach the infective larvae stage, thus pasture contamination and reinfection is minimized. The infective larvae have a sheath that protects them from adverse environmental conditions so they can survive for months,
which extends transmission potential. As long as temperature and moisture conditions are warm and wet, pasture contamination accumulates, but if the temperature gets too hot/cold and/or the moisture conditions become dry, larvae are threatened and pasture contamination is less. Worm transmission can be reduced by implementing control measures to remove worms from the animal (deworming) and/or using management to reduce reinfection.

![Life Cycle of Haemonchus contortus, the barber pole worm](image)

**Figure 1.** Typical worm life cycle

Another way to look at the life cycle is in four phases, the epizootiologic cycle (Figure 2). Phase 1 is the Parasitic Phase (host and the parasite interaction). Phase 2 is the Contamination Phase (eggs that are passed in the feces). Phase 3 is the Free-Living Phase (larval development and survival in feces and on pasture). Phase 4 is the Infection Phase (consumption of infective larvae during grazing). A number of factors affect what happens and influences control strategies during these phases.

During Phase 1, infective larvae are ingested, lose their protective sheath and invade the mucosa (lining) of the gastrointestinal tract (abomasum, small intestine or large intestine) depending on the worm. Larvae develop to the next larval stage and then emerge back surface where they develop to adult worms. The immune system is the major defense mechanism against worms. A series of activities act to mobilize various components (antibodies, killer cells, etc.) that then affect the ability of the worms to survive. The immune system has to mature with age. Young animals are more susceptible to infection and become more resistant with age. Young animals, therefore,
harbor the heaviest worm burdens and suffer the most severe consequences. Adult animals have stronger immunity and harbor lower worm burdens. Infection level is measured by counting the number of eggs that are passed in the feces. High and low egg counts are usually seen in young and adult animals, respectively. Signs of infection and clinical disease infection in young animals can include diarrhea, rough hair coat, anemia, weight loss, bottle jaw, etc. Infection in older animals usually is subclinical with weight loss and poor condition score. Nutrition and/or stress can alter a host’s immune competence. Poor nutrition and/or stressful conditions compromises the immune system which then can’t respond adequately. Therefore, the effects of infection can become worse no matter what the age of the animal. The prepatent period can be extended for worms that enter a period of delayed or arrested development in the host called hypobiosis. This occurs when the environmental conditions are unfavorable for development and survival of larvae on pasture. This happens during summer in warm/dry climates and during the winter in colder climates.

During Phase 2, the magnitude of pasture contamination is affected by stocking rate (number of animals per grazing area), age of the animals, season of the year and hypobiosis. The lower/higher the stocking rate, the less/more feces are deposited, thus more/fewer eggs on pasture. Young animals pass more eggs than older animals. Worms have a definite seasonality, so more eggs are produced and passed during their ‘season’. The phenomena called the peri-parturient rise in fecal egg output is common in small ruminants. This occurs around parturition and extends through most of lactation. These two times are stressful and the dam’s immune system is compromised. Nutrients are used primarily to support fetal development and then lactation and ability to maintain an effective immune response to worm infection is compromised. This results in female worms increasing egg production and the number of eggs deposited in the feces. During hypobiosis, development time to the adult stage is extended which results in fewer worms and fewer eggs deposited in feces. However, when larvae resume development, massive numbers become adults over a short period of time. The resultant increase in number of worms results in increased egg production and deposition in the feces, thus more contamination.

During Phase 3, development and survival of the free-living larvae depends on environmental (temperature and moisture) and nutritional (oxygen and energy) conditions. The first stage larvae
develops in the egg. After hatching, development to second-stage and finally third-stage (infective) larvae occurs in the feces. The unprotected first- and second-stage larvae need oxygen and energy (feed on nutrients and microorganisms present in the feces) to grow. Because infective larvae are enclosed in protective sheaths, they do not feed. Normal development and survival occurs between 65-85 degrees F. As the temperature increases or decreases from this range, development and survival is reduced. Moisture is very crucial for development and survival. Within feces, moisture is usually adequate to complete development to the infective larvae. However, if temperatures are high and/or feces are physically disrupted, the first- and second-stage larvae are susceptible to desiccation and will die. If feces remain intact and conditions remain favorable, infective larvae can survive for months. A moisture medium (rain, dew, irrigation) is necessary for infective larvae to migrate out of feces. Once outside the feces and on the forage, they are relatively resistant to environmental conditions due to their protective sheath. Temperature is usually the determining factor that adversely affects the survival of infective larvae on pasture. They can survive very low temperatures, but may die off during hard freezes. Temperatures above 95 degrees F are usually lethal if exposed for long periods of time. The higher moisture and lower temperature conditions at ground level under forage cover is usually adequate for infective larvae to survive for extended periods of time. Their length of survival depends on how fast they use up their energy reserves because they can’t feed. As temperature increases, the faster they move; therefore, energy stores are used up quicker and survival is shorter. Infective larvae remain close to the fecal pat (within 12-24 inches) and when there is a moisture medium present (i.e. advancing and receding dew, rain), they move up and down the blades of grass (2-3 inches). So, as the animals graze closer to the ground or closer to feces, consumption of infective larvae increases.

During Phase 4, stocking rate again is important. As the stocking rate goes up, there is more contamination (Phase 2) of the pasture and, consequently, more infective larvae will be available for consumption, and vice versa. It is well known that animals tend to avoid grazing close to feces so the further the distance between fecal deposits, exposure is reduced. When feces eventually disintegrate, grass grows well (fertilization) and animals grazing over that area will be exposed to more infective larvae. Forage grows well along the edge of natural sources of water (streams, ponds or lakes). When animals congregate to drink, they consume the attractive forage. Defecation in these areas also results in increased contamination and eventually infective larvae build up. The
same can be said for areas where supplements, especially hay, are fed on the ground. Similarly, trees provide an area for animal congregation and shade. Under all these situations, a high stocking rate has been artificially created in smaller areas, i.e., hot spots for reinfection.

**Figure 2.** Epizootiologic cycle of gastrointestinal worms

**The Worms**

*Haemonchus contortus* (Barberpole Worm)

*Haemonchus contortus* is found in the abomasum. They are blood feeding worms that gets the name from the barberpole appearance of white ovaries twisted around the red blood filled gut (Figure 3). They feed by disrupting the surface of the abomasum with a lancet which results in blood flow (Figure 4). They then ingest the blood through the lancet, like a straw. They are rather large compared to other gastrointestinal worms, measuring up to 3/4 of an inch. If one opens up
the abomasum, they can be readily seen as thin red hair-like worms on the abomasal surface. Female worms are prolific egg layers and with a heavy burden, the environment is contaminated with a very large number of eggs. These worms are found predominantly in tropical and subtropical regions of the world where they thrive under hot and moist environmental conditions. These conditions prevail in the southeastern US, but where similar environmental conditions are encountered during the summer, *H. contortus* can also be a problem.

**Figure 3.** Adult female *Haemonchus contortus*

**Figure 4.** Head of *Haemonchus contortus* with lancet

*H. contortus* transmission and infection is at the lowest level during the winter. As temperatures get warmer and moisture increases during the spring, transmission and infection increases and peaks during the summer. As temperature and moisture dissipates during the fall, so does transmission and infection. Hypobiosis has not been observed to occur to any great extent in the southeastern US because the life cycle can be maintained year around, but it does occur in more northern/western temperate (cold/dry) regions of the US.
The primary sign associated with *H. contortus* infection is anemia due to blood loss. Mucous membranes will be pale (most visible by looking at the inside of the lower eyelid) and bottle jaw (an accumulation of fluid under the chin) may appear. As the worm burden increases, the more blood is lost and eventually animals may die.

*Telodorsagia (Ostertagia) circumcincta* (Brown Stomach Worm)

The second abomasal worm of importance, also found in the abomasum, is *Telodorsagia circumcincta* which is smaller than *H. contortus*. It is not as readily visible as *H. contortus* since it is about a quarter to half inch long and very thin. These worms feed mostly on nutrients along the surface and are not a blood feeder. Female worms produce fewer eggs than *H. contortus*. Infection causes direct damage to the abomasal surface which interferes with digestion. As a result, infection is usually considered a production disease because animals do not grow well. That said, when infection reaches a level that causes clinical disease, the primary symptom is diarrhea and death can result under very high infection conditions. This worm thrives in cooler wet environmental conditions such as the more temperate regions of the US outside most of the SE. This worm takes advantage of hypobiosis when environmental conditions are too cold (winter) or too dry (summer).

*Trichostrongylus colubriformis* (Bankrupt worm)

*Trichostrongylus colubriformis* is the most predominant small intestinal worm and is very small and threadlike. It is present throughout the US, but similar to *T. circumcincta*, it thrives better when conditions are more cool and wet. In the southeastern US, *T. circumcincta* is not very prevalent, so *T. colubriformis* is the next most common and important worm after *H. contortus*. As with *T. circumcincta*, this worm feeds on nutrients on the surface of the mucosa and interferes with digestive function. The most common clinical sign is diarrhea accompanied by weight loss or reduced weight gain. It is called the bankrupt worm because animals become poor doers leading to loss of production and income.
**Figure 5.** Relative difference between *Hamonchus*, *Teladorsagia* and *Trichostrongylus*

*Nematodirus* spp. (Long-necked bankrupt worm)

*Nematodirus* spp. are found in the small intestine and are relatively large worms (easily seen, like *H. contortus*). They are found throughout the US, but usually in rather small numbers and thrive better in cooler climates. Problems are rare in the southeast US, but in cooler areas greater numbers of worms can accumulate and if this occurs, production and income losses will result (similar to that of *T. colubriformis*).

*Oesophagostomum* spp. (Nodular worm)

*Oesophagostomum* spp. are found in the large intestine and are relatively large (easily seen, again like *H. contortus*). They are found throughout the US, but usually in rather small numbers. The larvae are found in the mucosa of both the small and large intestine where they form nodules, thus Nodular worm. After leaving these nodules, the larvae develop to adults in the large intestine where they feed on blood and can contribute to anemia caused by *H. contortus*. They also disrupt function of the large intestine (water resorption) which contributes to diarrhea.
**Trichuris spp. (Whipworm)**

*Trichuris* spp. are found in the large intestine usually in small numbers. The posterior end of the worm is rather large and can be readily seen while the anterior end is thread-like, thus Whipworm. These worms are also blood feeders and, like *Oesophagostomum* spp., contribute to anemia due to other worms. In addition, it contributes to disruption of the function, thus diarrhea.

**Diagnostic methods (measure how wormy animals are)**

The general clinical signs of worm infection include rough hair coat, diarrhea, depression, weight loss (or reduced weight gain), bottle jaw and anorexia (off feed). Supporting diagnostic findings from laboratory testing include the fecal egg count (FEC, increases with higher worm burden) and blood pack cell volume (decreases with higher worm burden). These are tests usually done by a veterinarian, but can be by others with proper training.

During the FEC exam, the eggs of all the worms mentioned above look similar (called trichostrongyle-type, Figure 6) except for *Nematodirus* spp. (large football shape, no end plugs, Figure 7) and *Trichuris* spp (small football shape with protruding end plugs, Figure 8).

![Figure 6. Typical trichostrongyle-type eggs](image)
**Figure 7.** Typical *Nematodirus* spp. Egg

**Figure 8.** Typical *Trichuris* spp. eggs
Chapter 2. How and why resistance to worm remedies develops

Ray M. Kaplan, DVM, PhD, DipACVM, DipEVPC

University of Georgia College of Veterinary Medicine, Department of Infectious Diseases, Athens, Georgia, USA


Introduction

The age of modern chemistry and pharmaceutical solutions to infectious disease ushered in a renaissance of improved health and productivity of livestock. Starting in the 1960’s, the availability of highly effective, safe and broad-spectrum worm remedies (anthelmintics) made parasite control simple and effective. Frequent regularly scheduled treatments with worm remedies kept animals healthy and productive, and had a great costbenefit return. However, this strategy has turned out to be shortsighted and unsustainable. This is because gastrointestinal nematodes (GiN; commonly referred to as roundworms) of sheep and goats have become resistant to multiple drugs. This problem is extremely common and widespread around the world, and is particularly serious in *Haemonchus contortus* (barberpoleworm or wireworm), but also in other species of GiN (Kaplan and Vidyashankar, 2012). In fact, in many areas of the world, including South Africa, there is a great risk of having no effective worm remedies to use in the near future. Some farms are already in such a predicament. This leads to several important questions. First, why does drug resistance in worms seem to happen so readily and how does this happen? And second, what can be done to slow down the development of drug resistance?
What is drug resistance and how does it start?

First we should define resistance to worm remedies. Resistance is defined as a heritable genetic change (meaning it is passed genetically directly from a worm to its offspring) in a population of worms that enables some individual worms to survive drug treatments that are generally effective against the same species or worm and stage of infection at the same drug dose rate. In practical terms resistance to worm remedies is present in a population of worms when the effectiveness of the drug falls below that which is historically expected, when other causes of reduced efficacy have been ruled out. Really, we should not be surprised that worms have become resistant to worm remedies. Everyone is aware that that bacterial resistance to antibiotic drugs is becoming a major problem worldwide. Very similar evolutionary processes also occur with worms. In fact, we now understand quite well that drug resistance is an inevitable consequence of using drugs to kill bacteria, viruses and parasites. With regard to parasitic worms, these organisms have many biologic and genetic features that favor the development of drug resistance, such as short life cycles, high reproductive rates, high genetic mutation rates, and extremely large population sizes. These biologic characteristics lead to rapid rates of evolution and exceptionally high levels of genetic diversity. Therefore, even though mutations that cause a worm to become resistant are very rare, they are constantly occurring due to the enormous sizes of worm populations. For instance, each female *H. contortus* worm produces approximately 5,000 eggs per day. If a flock of sheep averages 1,000 worms per animal (a very modest level of infection), then there are 500 females so that a single sheep will pass 2.5 million eggs per day. In other words, a flock of 100 sheep will shed almost 2 billion eggs per week. These huge numbers provide the opportunities for rare mutations that lead to drug resistance to occur. So – what happens to make these very rare mutations increase in numbers to such a great extent that many or even most worms on a farm have the resistance trait? As discussed above, initially worms bearing mutations that make them resistant to the drug are very rare within the midst of a very large genetically diverse population, which may number in the hundreds of millions or billions. These resistant individuals and their offspring will remain rare within a worm population, and may even disappear from the population unless they gain a survival advantage over their parasitic competitors. The way they gain such a competitive advantage is by treating animals with worm remedies. Treatment per se does nothing positive for resistant worms, but by killing the drug-sensitive worms, which comprise the vast majority of a parasite population, resistant individuals are able to reproduce for a given interval in the relative
absence of competition. As a result, following each and every treatment with a worm remedy, the numbers of resistant worms increase incrementally. The development of drug resistance to levels that are clinically important is usually a slow and gradual process, requiring numerous generations under drug selection (usually taking many years). This can be best understood with a simple illustration. If say one in one million worms in the population are resistant, and then numbers of resistant worms double with each treatment we will see the following change in frequency of resistant worms over 13 treatments: 1/1,000,000; 1/500,000; 1/250,000; 1/125,000; 1/62,500; 1/31,250; 1/16,000; 1/8,000; 1/4,000; 1/2,000; 1/1,000; 1/500; 1/250; 1/125. So – with just 13 treatments we went from 1 in 1,000,000 to 1 in 125. However, even though this signifies an almost 10,000-fold increase, the drugs are still 99% effective and the resistant worms are not noticed. But then it takes just 6 more treatments to reach complete treatment failure (1/125; 1/62; 1/31, 1/15; 1/8; 1/4, 1/2). In this illustration it took just 19 treatments to change the frequency of resistant worms from one in one million (0.000001%) one in two (50%). But only in the last 3 treatments would there be enough resistant worms present to cause a problem. Of course this is just an illustration, and this process can occur faster or slower depending on numerous factors. Thus from a practical perspective, drug resistance develops slowly over time, during which time it is impossible to detect. But then levels of resistance increase very rapidly in the last phase, where it is then perceived as a clinical event (treatment failure). Alternatively, resistant worms can be purchased, thus bypassing the many years of worm evolution and drug selection necessary to reach high levels. Depending upon how many animals are purchased harboring resistant worms, treatment failures can occur practically instantly or over a relatively short period. This has great clinical relevance because in either case, resistance can transition from undetectable, to clinically important levels over a very short period of time. Consequently, unless a surveillance program is in place that closely monitors the effectiveness of drug treatments over time (see paper on diagnosis of drug resistance), resistance will not be noticed clinically until levels of resistance are extremely high. This is a major problem because once resistance reaches detectable levels, irreversible changes in the genetic structure of the worm population have occurred, ensuring that “resistance” is fixed in that population forever(Roos et al., 1995). Thus, once resistance is diagnosed as a clinical problem “reversion” to susceptibility likely will never occur.
Factors that affect the development of drug resistance

It is easy to understand how resistance may evolve when worm remedies are administered frequently. But what other factors regulate the rate with which resistance develops? Why does resistance develop so much quicker in some parasites and in some hosts than in others? We don’t fully know all the answers to these questions but there is much we do know. Firstly, it is important to administer quality drugs at the proper dose level, and to deliver the dose using optimal drenching technique. Under dosing either because of underestimating the animal’s weight or sub-optimal delivery of the dose can greatly accelerate the rate with which resistance develops. Similarly, the use of poor quality or degraded drugs (old or stored poorly) can also accelerate resistance because all of these practices allow partially resistant worms to survive that would otherwise be killed by the full dose. However, the most important factor affecting the rate of development of resistance to worm remedies is the proportion of the worm population under drug selection. In other words, of all the worms on the farm, both in the animals and on the pasture, what percent of these worms is experiencing the effect of the worm remedy treatment? The more worms that “experience” or “see” the drug, the more opportunity there is for the resistant worms to gain an advantage. The fewer the number of worms that “see” the drug, the less advantage the resistant worms gain because many of the drug-susceptible worms are still around to dilute the resistant ones. We use the term “Refugia” to describe the portion of a parasite population that is not exposed to a worm remedy during a treatment event (Van Wyk, 2001). Parasites in refugia escape selection pressure from the drug, thus parasites in refugia constitute a reservoir of drug-susceptible parasites that keep the resistant worms diluted to low levels. Examples of refugia include eggs, infective third-stage larvae (L3) and pre-infective larvae (L1, L2) in the environment (on the pasture) and parasitic stages in those individual animals that are not dewormed whenever other herd members are treated. It is noteworthy that at times of the year when worm transmission is high (rainy season) thenumbers of parasites on pasture often comprise >99% of the total parasite population on a farm. In contrast, during hot and dry times of the year few parasitic stages can survive on the pasture, and therefore most of the worms are inside the animals. To summarize, if we are using worm remedies to treat and control worm infections in our livestock, we cannot stop drug resistance from developing. However, there are things we can do to greatly slow down the rate of development of resistance. Managing refugia is the most important and direct way that we can achieve this. The more parasites that are in refugia, the slower the development of resistance will be to worm
remedies. This is because the resistant worms that gain advantage every time an animal is treated are greatly diluted by the untreated refugia. Though managing refugia cannot prevent resistance from eventually occurring, managing refugia on a farm is critical to delaying the inevitable development of resistance and improving the sustainability of worm control programs.

Conclusion

Despite the occasional development of new types of worm remedies (anthelmintic classes), history clearly demonstrates that the development of resistance consistently outpaces the introduction of new drugs. Clearly then, major changes need to be made in the way that worm control is practiced. It is no longer acceptable for veterinarians or farmers to view GIN parasite control in terms of a “deworming program”. Over the past decade a paradigm shift has occurred in how GIN parasite control must be viewed and practiced. Worm remedies can no longer be viewed as a relatively inexpensive management tool to be used with little thought to maximize animal productivity. Rather, they must be viewed as extremely valuable and limited resources. We must balance our desire for simplicity and ease with the reality that effective long-term control of parasitic worms will only be possible if worm remedies are used intelligently with prevention of resistance as a goal, and as part of a sustainable integrated parasite management (sIPM) system.

References


Resistance to dewormers is a fact of life, and the situation has worsened greatly in recent years. Surveys indicate that most farms have worms resistant to at least two of the three major groups of dewormers. Many have resistance to all three groups, and some farms now have resistance to all available dewormers.

There is now very strong evidence that using a combination treatment is the best method for using dewormers and should be implemented immediately on all farms that have gastrointestinal parasite resistance problems.

- In New Zealand and Australia, products are sold that contain a combination of dewormers, so only one product needs to be administered. In contrast, in the USA, no dewormers are yet sold in this formulation, so the dewormers need to be bought and administered separately. This increases the cost as compared to the products available in these other countries. In the USA, the different groups of dewormers available on the market are not chemically compatible, thus they CANNOT be mixed together in the same syringe. Rather, they need to be administered separately, but can be given one
immediately after the other. Products that contain a combination of dewormers as those available in New Zealand and Australia, however, are being considered by the Food and Drug Administration,

- *When using dewormers in combination, meat and milk withdrawal times will be equal to the dewormer used with the longest withdrawal time period.*

- All dewormers should be administered at the full recommended dose whether administered singly or in combination. Check the *Parasite Control* section of the NCSU Meat Goat Portal for recommended dewormer dosages and meat and milk withdrawal times.

- If using dewormers in combination, it is critical to maintain refugia; thus, one should be using a selective treatment approach based on FAMACHA© (see FAMACHA© section of the ACSRPC website for more information on this method and for further explanations of refugia). The presence of refugia is essential to realize the full benefits from combinations. In fact, if refugia are not maintained then you will not get the necessary dilution of the resistant survivors, and this will then lead to having multiple-resistant worms that can no longer be controlled with the combination treatment.

- If the efficacy of your dewormers are >80%, it is possible you may not notice any difference in the clinical response of treatments when applied singly vs. in combination.

- Any safety precautions that exist for a single dewormer will also exist when used in a combination. Nevertheless, there are no known additional risks with using more than one dewormer at the same time.
Chapter 4. Potential solutions to highly resistant worms in sheep and goats

- **By Steve Hart**  
  Goat Extension Specialist  
  Langston University

- A parasite management program uses the principles of biology of the worm and manages pastures and animals in such a way to suppress worms. This implies a plan of certain management actions. This would include such things as rotational grazing with a 40-day (or more) rest period, not grazing within 4 inches of the ground, and using browse and/or sericea lespedeza. Higher stocking rates encourage worms.

- Finding an effective way to deworm animals (discussed later) will only provide short-term relief for the problem and not solve the problem in the long-term. If you have significant dewormer resistance, you are going to have to develop a parasite management plan if you want to stay in the sheep/goat business for the long-term.

- Other management practices that can be useful to control worms by reducing the number of infective larvae on a pasture include baling hay which removes most of the infective larvae from the pasture (the larvae in the hay bale die within a month). Tillage, such as used to plant wheat or sorghum-sudan pasture for grazing, is an effective method of cleaning pasture, since larvae that are tilled an inch under the soil do not survive.

- Grazing with another animal species (horses or cattle; sheep and goats share the same parasites and will not help one another) is another way to clean larvae off the pasture. Pasture rest (at least 6 weeks during a hot summer, longer in cooler climates) will also clean a pasture, especially when the weather is hot. With improved pasture species, the pasture forage quality will decline with this amount of rest. Solutions include making hay on the pasture while it is rested, grazing with another animal species, or even mowing to keep the grass in a vegetative stage.

- There have been several cases where resistant worms have been replaced with susceptible worms, but this requires superb management and the services of a parasitologist. But,
without an effective parasite management program that minimizes the use of dewormers, this is a solution for only a few years.

- Another way to overcome parasites is to select animals that are more resistant to parasites. This begins by culling animals. Fifteen percent of the animals in the herd carry 50 percent of the worms, whereas 20 to 30 percent of the animals in the herd carry 70 to 80 percent of the worms. If you get rid of the animals that carry the most worms, then there are many fewer infective larvae on the pasture to infect the rest of the animals, and you have prevented these animals from contributing their weak parasite resistance genes to the herd.

- Everyone knows that some of their animals are always wormy, and those are easy to identify and cull. There are two ways that wormy animals can be identified. One is by doing FAMACHA© across the season and the animals that require the most deworming can be culled. One should categorize animals by litter size (singles, twins and more) and cull those within each group that require the most deworming. The reason for categorizing by litter size is that does/ewes that raise more kids will often show more symptoms of worms.

- Another way to identify the animals with the most worms is to do a fecal egg count on all animals and cull the ones within each litter size with the highest fecal egg counts. The buck or ram accounts for 50 percent of the herd genetics. You should take several fecal egg counts during the summer from your bucks/rams and get rid of those with the highest fecal egg counts. FAMACHA© could also be used, but fecal egg counts are better for the bucks since fecal egg counts are more accurate and can be justified for the bucks/rams due to their importance.

- Yearlings will tend to have higher fecal egg counts since their immune systems are not fully developed. This should be factored into selection. If you identify the most resistant rams/bucks using this method, they should be bred to the best females (determined by FAMACHA©) and the resulting offspring saved as replacements.
How to kill resistant worms

- First, you need to incorporate the support of the animal’s immune system which will require good nutrition to support an immune response. There are three classes of dewormers: benzimidazoles (Panacur®, Safeguard®, and Valbazen®), cell-depolarizers (Prohibit® and Rumatel®) and avermectins and milbemycins (Ivermectin and generics, Eprinex®, Dectomax® and Cydectin®).

- In general, Valbazen® is the most potent benzimidazole and Cydectin® is the most potent member of the avermectins and milbemycin group. The use of the more potent members may prevail over resistant worms. Doubling the dosage will help only little, increasing the percentage of worms killed often by only 25 percent. Goats should be given twice the sheep dose of Benzimidazoles and the Avermectin/milbemycin group. They should be given only one and a half times the dose of Prohibit® due to the narrower margin of safety.

- Animals can be given combinations of dewormers at the same time, one from each of the different classes of dewormer (usually the most potent members). These can be quite effective against resistant worms.

- Benzimidazoles can be given every 12 hours for several doses to increase the effectiveness. The last dose can be given with a dewormer of another class to increase the effectiveness. Copper oxide wire capsules have been shown to be effective against the Barberpole worm (Haemonchus contortus).

- The only long term solution to highly-resistant worms is an effective parasite management program. Combinations of dewormers may work for a while, but will fail without an effective parasite management program. It is possible to raise sheep/goats without dewormers (they did exist before dewormers were available and some people manage to raise them without dewormers), but the weak ones died, they rotated themselves, did not graze close to the ground, and goats browsed and ate a variety of plants. It is our management that has caused parasite problems (Adapted by American Consortium for Small Ruminant Parasite).
Chapter 5. Alternative Methods for Managing Gastrointestinal Parasites and *Eimeria* spp. in Small Ruminants

B. R. Min*, N. Gurung, A. Elliott, and S. Solaiman

Department of Agricultural and Environmental Sciences
Tuskegee University, Tuskegee, AL 36088
*minb@mytu.tuskegee.edu,
334-524-7670

Who cares and why?

Infection with gastrointestinal nematodes (GIN) and coccidia (*Eimeria* spp.) remains one of the primary constraints to sustainable production of small ruminants world-wide, and with resistance to synthetic anthelmintics now reaching epidemic proportions in sheep and goat GIN, alternative (non-synthetic) control strategies are being explored. Infected animals have lower growth rates, reduced reproductive performance, and have higher rates of illness and death. In the past, sheep and goat producers relied heavily on anti-parasitic drugs. Unfortunately, GI parasites have become increasingly resistant to many of the ruminant anthelmintics. Alternative methods of GI parasite control for animals raised primarily on forages are vital for the sustainability and profitability of sheep and goat farms in the southern United States. Consequently, alternative, sustainable, and affordable methods of parasitic control are required. There is also a need to examine plant-based alternatives to control coccidiosis in livestock because there are no FDA approved drugs to treat coccidiosis in small ruminants.

Alternative approaches to control of parasites in goats

If internal parasites are resistant to drenches, grazing management is essential to maintain animal production as shown follow:

1. Grazing management
2. Safe pasture
3. Selective breeding for host resistance
4. Feed additive
   - CT-containing diets (Table 1).
   - Copper Oxide Wire Particles - fungi

**Pasture management**

1. Smart grazing management:
   Avoid graze below about 5-6 cm (2-3 inches) pasture height.
   Over 80% of larvae are within 3 cm of the soil surface.

2. The Gumboot rule;
   Avoid pasture height shorter than the toe of your gumboot (Figure 1).

![Figure 1](image.png)

**Figure 1.** The effect of forage height on fecal egg counts (FEC) in sheep.

**Manage pasture quality:**

To ensure high quality regrowth for next time it is grazed

1. Goat to cattle system; 5-6 cm deep pasture left behind by the goats, it is acceptable to production from cattle.

2. Goats + cattle Integrated system: cattle and goats prefer different species of forage.
   - Goats + cattle
     (not share the same parasite species)
   - Goats + sheep
     (share the same parasite species)
   - Cattle + Sheep
What is safe pasture?

Safe pasture is pasture with few or no parasite larvae

1. New pasture: the time to cultivate new pasture allows larvae to die before being grazed.
2. Forage crops: tannins containing forages.
   - Sericea lespedeza, *Lotus corniculatus*
   - Sainfoin, mimosa
3. Hay and silage regrowth: These paddocks will probably have been closed for 4 to 8 weeks. So most of larvae on the herbage will have been removed or died

Tannin-containing diets

Research has shown that legumes such as Sericea lespedeza (*Lespedeza cuneata*) contain condensed tannin (CT) with anti-parasitic properties. The anti-parasitic properties of CT have been demonstrated to reduce GI parasitic infection in goats in Oklahoma and Georgia and in sheep in Louisiana and Arkansas. In a series of studies with goats and sheep of differing breeds, life stages (young, mature), and geographic regions of the southern and eastern U.S., including sericea lespedeza in the diet as fresh (grazed) or dried (hay, leaf meal, pellets) material has resulted in a reduction of gastrointestinal parasites eggs and coccidial oocysts in feces (up to 97 percent), lower numbers of adult parasites in the animals’ stomach and intestines (up to 70 percent), and reduced development of gastrointestinal parasites eggs to infective larvae in simulated pasture conditions (Burke et al., 2012 a,b; Min et al., 2004; 2005; Terrill et al., 2009). Researchers at Tuskegee University found potential benefits of pine bark (PB) supplementation on anti-parasitic effects and improved feed efficiency. Pine bark is one of the abundant forest byproducts in the southern United States and contains 11-to-13 percent CT on a dry matter basis. By raising sheep and goats primarily on forage, farmers can reduce feed costs. Research demonstrates that goats fed on a PB diet have up to 30 percent fewer worms in a total ration, as well as lower instances of fecal egg count and fecal coccidian oocyst count. Feeding a pine bark diet reduced both male (64 percent) and female (59 percent) worm counts compared with the control (without PB) diet. In addition, mean dry matter intake, feed efficiency, average daily gain, and cold carcass weight were also greater for goats fed the pine bark diets. On-farm research with local farmers in Alabama also has shown that goats on a pine bark diet had 74 percent lower fecal egg counts and 5 percent better animal weight gain compared to control diets during three-month trials. Ground pine bark as a feed
ingredient has the potential to improve animal performance while decreasing internal parasites and coccidian infection. On-going our study indicated that using pine bark in combination with sericea seems to be even more effective with artificially infected drug-resistant *H. contortus* (Wright et al., 2016). The extract from the bark of the pine tree (*Pinus radiata*), which is rich in condensed tannins, was reported to inhibit the life cycle of Coccidia as evidenced by decreased sporulation of the oocysts of *E. tenella*, *E. maxima*, and *E. acervulina* (Molan et al. 2009). Thus, developing plant-based alternatives such as pine bark and other natural resources for GI parasites control would be expected to have a greater impact on the goat and sheep industries. This will allow development of Best Management Practices to prevent or treat coccidiosis and GI parasites in ruminant livestock. Ultimately, by raising sheep and goats primarily on forage, farmers can reduce feed costs.

**What is the tannins?**

Tannins (plant secondary compounds) are plant compounds that bind to proteins and other molecules. Tannin is related to “tanning”, as in preserving hides, and tannins are found in many plants. There are two main types of tannins; hydrolyzable (HT), some of which may have toxic effects on animals, and condensed tannins (CT), which are found in forage legumes (including sericea lespedeza) and other plants. Effects of tannins vary depending on type of tannin, concentration, and on the animal consuming the tannins. Ruminants (sheep, goats, cattle, deer etc.) consume a wide range of plant materials (grasses, legumes, browse shrubs, tree leaves and bark), and some of the plants contain tannins that have potential effects on animal nutrition, parasites, and gut microbiome diversity. The CT-containing forage diets typically reduce ruminal protein digestion, increase rumen bypass protein, and can increase average daily gain and improve health of sheep, cattle and goats. The potential benefits of tannins-containing diets are not the same for all the tannins, but rather depend upon the concentration and structure of the tannins.

Negative effects may include reduced intake and reduced digestibility, leading to a decline in animal productivity (Figure 2). Negative effects are seen more often when CT concentration is high (above 55 g CT/kg DM in the forage) (Min et al., 2003). Positive effects may include an increase in by-pass protein (causing the animal to use protein more efficiently), a reduction in bloating, increased milk production, and a reduction in internal parasite numbers, egg output, and hatchability.
According to Min et al. (2003), low concentrations of CT (20-45 g CT/kg DM) are helpful to animals, while high forage CT concentrations (>55 g CT/kg DM) may have negative effects. Results vary according to CT concentration and structure and the animal that is grazing the forage, however. Researchers have shown that big trefoil, sulla, sanfoin, and sericea lespedeza are useful in controlling internal parasite infection in sheep and goats. Providing condensed-tannin-containing forages is one way to boost the health of sheep or goats.

Figure 2. The effect of forage CT concentration on wool production (Min et al., 2003).

Impact Statement (tannin-containing diets)
Goats fed on a PB diet have up to 30 percent fewer worms in a total ration, as well as lower instances of fecal egg count and fecal coccidian oocyst count.

Goats on a pine bark diet had 74 percent lower fecal egg counts and 5 percent better animal weight gain compared to control diets during three-month trials. Ground pine bark as a feed ingredient has the potential to improve animal performance while decreasing internal parasites and coccidian infection. Thus, developing plant-based alternatives such as pine bark and other natural resources for GI parasites control would be expected to have a greater impact on the goat and sheep industries. Development of Best Management Practices to prevent or treat coccidiosis and GI parasites in ruminant livestock. Raising sheep and goats primarily on forage can reduce farmers feed costs.
Table 1. Condensed tannin (CT; % DM) content in different forage species

<table>
<thead>
<tr>
<th>Forage</th>
<th>% DM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perennial ryegrass</td>
<td>0.18</td>
</tr>
<tr>
<td>Chicory</td>
<td>0.31</td>
</tr>
<tr>
<td>Crabgrass/tall fescue mix</td>
<td>0.32</td>
</tr>
<tr>
<td>Alfalfa</td>
<td>0.05</td>
</tr>
<tr>
<td><em>Lotus corniculatus</em> (Birdsfoot trefoil)</td>
<td>4.8</td>
</tr>
<tr>
<td><em>Lotus pedunculatus</em> (Big trefoil)</td>
<td>7.7</td>
</tr>
<tr>
<td>Sainfoin</td>
<td>2.9</td>
</tr>
<tr>
<td>Sulla</td>
<td>5.1-8.4</td>
</tr>
<tr>
<td>Sericea lespedeza</td>
<td>4.6-15.2</td>
</tr>
</tbody>
</table>

Adapted from Min and Hart, (2003); Min et al., (2005).

Conclusion
This research has enabled goat production to be a more sustainable and low-cost enterprise by mitigating internal parasites and coccidian infections of grazing animals, thus enhancing overall meat production, protecting animal and human health, increasing producer profits, and providing a stable and safe food supply. Producers should not rely on one method, e.g., sericea as the sole method for controlling internal parasites. However, sericea can be useful as one part of a complete parasite management strategy.

What research is needed?
More research is needed to: study the direct mechanisms of tannins and parasite interactions; look for ways of validating the anti-parasitic plants of sheep and goats production systems; and to continue testing the effectiveness of alternative natural dewormers for use in ruminants.
References
Chapter 6. Non-chemical control of parasites in small ruminants through grazing management and nutrition

Steve Hart
Langston University, OK

Introduction

It is well established that worms are the leading cause of morbidity, mortality and loss of profit in small ruminants in regions where there is sufficient rainfall. Worms or roundworms refers to gastrointestinal nematodes of the *Trichostrongylus* superfamily. Although there are more than 30 species of worms that parasitize small ruminants, three species cause most of the infections and death here in the Southeastern US. They are the barberpole worm (*Haemonchus contortus*; a tropical roundworm), the brown stomach worm (*Teledorsagia circumcincta* or *Ostertagia ostertagia*, a temperate species roundworm) and the black scour worm (*Trichostrongylus colubriformis*, a temperate species roundworm). The same grazing management practices and nutritional strategies used against these three major roundworms will usually discourage infection by the other thirty species. The barberpole worm will tend to dominate in warmer regions such at the Southeastern US. Even in cold Canada, the barberpole worm is a problem, but only for 6 to 8 weeks. The brown stomach worm and black scour worm, being temperate species worms are greater problems in the spring and fall. Before the advent of dewormers, sheep and goat producers used management to prevent worms. Animals with poor resistance to worms usually died. A producer who did a poor job of managing against worms was not a producer very long.

Worms are normal, we cannot, nor should we try to eradicate worms. They have a useful role in animals in the wild, preventing a population from overrunning an area when conditions for forage growth are good (high rainfall and warm weather) and killing the more susceptible animals when growing conditions are bad (drought forcing animals to graze close to the ground), ensuring that the strong survive. We want animals to have a few worms to keep the immune system functioning
against worms, but not so many as to impair animal production. An excessive number of worms causes pathology, such as anemia, poor appetite, diarrhea, edema and death. Our goal should be to manage animals to keep worm numbers from becoming excessive. It is possible to raise small ruminants using good grazing management, appropriate nutrition and good animal genetics without having to deworm them. This is particularly important for organic producers where dewormers are prohibited, but it is becoming very critically important for all small ruminant producers as worms become more resistant to our dewormers.

**Rotational grazing**

Rotational grazing can be used to prevent animals from picking up infective larvae in the pasture if the grazing period (time animals are grazing a specific area) is short enough so that infective larvae do not develop and the rest period (time between successive grazings of the same area) is sufficiently long so that few larvae survive on the pasture. So the first question we must answer is how long does it take for worm eggs to develop into infective larvae available for small ruminants to consume them. This depends on temperature and moisture. Eggs are in the fecal pellets of small ruminants, being laid by adult worms in the digestive tract and then end up in the fecal pellet or pat. Generally about 50°F is the minimum temperature for hatching eggs. Eggs hatch slowly at this temperature. The optimal temperature for hatching eggs is about 86°F and eggs will hatch in 4-24 hours at this temperature. This is why when it is colder than 50°F, worms are much less of a problem. Higher temperatures can reduce hatch rate slightly. Once the egg is hatched, it has to be developed to an infective third stage larva and then migrate from the fecal pellet or pat onto the grass. The hatched egg is referred to as a first stage larva which wanders through the fecal pellet eating bacteria such as E. coli and growing. It then molts (sheds skin like
a snake) to a second stage larva which does the same thing, wondering through the fecal pellet eating bacteria and growing until becoming a third stage larva. Larva are very small, being about as long as the period at the end of this sentence and finer than a spiderweb. The first and second stage larvae are subject to being killed by drying out. Hot, dry weather at this time can kill developing larvae. When the second stage larva molts to a third stage larva (infective stage larva), it is an incomplete molt. That is, the skin of the second stage larva slips up, but does not come off. Because the larva has an extra layer of skin, it is more resistant being dried out by the heat. However, when the skin slipped up, it now covers his mouth preventing him from eating. Now as a third stage infective larva, it must be released from the fecal pellet or pat and float on a layer of moisture from dew or rain onto the grass to be picked up by sheep or goats. The main trigger for release is rainfall, requiring two inches of rain in a month’s time based on studies in Australia. Several days of heavy dew can also release some larva, but rain is the major factor for release. This is why worms are much less of a problem in areas with less than 25” of rainfall.

It takes a minimum of 4 days for an egg to develop into an infective larvae at optimal temperatures if there is sufficient humidity (O’Conner et al., 2006). It may take as long as 14 days at 50°F. Actual field studies determined 4 days as being the minimum and 5-6 days under practical conditions. So, then the amount of time for the grazing period should be limited to 3 or 4 days, assuming there has been sufficient rain to release infective larvae from the feces, and then animals are moved to prevent consumption of infective larvae. Animals can remain on pasture until there is sufficient rain to release larvae from the fecal pellet. If the fecal pellet is exposed to the summer sun for a significant period of time, the temperature in the pellet may rise to 150°F, killing the eggs and larvae in the pellet.
The next question that needs to be answered is how long dolarvae survive on the pasture so that the time to rest a pasture can be determined. Basically, infective larvae die when they run out of body reserves since they cannot eat, although heat and low humidity can enhance mortality. Since infective larvae are cold-blooded, their metabolism reflects ambient temperature. Hot temperatures speed up metabolism and loss of body stores and therefore cause death of the larvae. In the review by O’Conner et al. (2006) survival times ranged from 30 days in the tropics to 10 weeks in more temperate areas. One study has noted that as little as 2 weeks of hot, dry weather could reduce infection rate (H. Jordan, personal communication).

Barger et al. (1994) grazed sheep in a tropical environment (Fiji islands) with a 3.5 day graze period and 31.5 day rest period and were successful at avoiding worms. Pomroy et al. (2002) grazed goats on a pasture for 5 days and rested pastures for 65 days. This reduced fecal egg counts from 309 to 121 epg and eliminated the need for deworming. Pasture contamination was greatly reduced 630 vs 40 worms/animal for tracer animals demonstrating that rotational grazing can successfully control worms in the Southeast US. In the French West Indies, a compilation of data from several grazing studies showed that infective larvae numbers started to increase after a week on pasture and were reduced to 10% of peak infective larval numbers after 35 days of rest (Mahieu et al., 2008). Colven et al. (2008) was successful at controlling worms in grazing sheep with a 5 day grazing period and 100 days of rest period. But a problem with such long rest periods, is that forage quality declines to the point that animal production is markedly reduced.

**Stocking rate**

Stocking rate is an important consideration in worm control because increased animals density results in an increased number of infective larvae on the pasture and there are more animals picking up infective larvae. With a stocking rates below two ewes or does and their lambs or kids per acre, stocking rate is not a significant factor, but as it increases, so do parasitism problems. Unfortunately, stocking rates must be higher than two animals per acre in many cases if the small ruminant enterprise is to be profitable. The producer should be aware that as stocking rates increases, so will parasite problems. Furthermore, the benefit of some technologies used to
increase stocking rate such as high levels of fertilization and irrigation may be somewhat offset by increased parasite problems.

Trees, barns and water sources can act to increase stocking rates in localized areas, exacerbating parasite problems. Water sources generally increase humidity and often increase soil moisture, improving conditions for egg hatch and development to infective larvae. If animals are being rotationally grazed, but use a common lane and water source, the area may accumulate high levels of infective larvae contamination, causing severe parasite problems despite having a rotation grazing system that should suppress infective larvae. Trees can be big offenders in this regard as they shade infective larvae from the sun, increase humidity, encourage animals to congregate which increases concentration of infective larvae, and encourages close grazing (picking up infective larvae) around the trees they rest under. The great effect of trees promoting worms was previously identified by Pomroy et al. (2002), and it was noted that rotation grazing reduced the effect of trees, but did not eliminate it.

The residual height or height of forage when animals are removed is an important factor in that the infective larvae are concentrated in the lower 2-3 inches of forage. Therefore, removing animals before they graze pastures down to 4 inches will reduce the consumption of infective larvae. Irrigation and periods of high rainfall may move infective larvae to higher levels necessitating higher pasture residual levels. Irrigation also creates a humid environment that increases the success of egg hatch and larval development. Sod forming grasses such as bermudagrass create a humid microenvironment protected from the sun that promotes the development of infective larvae as compared to a bunchgrass which allows some sun to penetrate and allows the ambient air to blow humidity from plant transpiration away. When small ruminants are consuming browse, they consume very few infective larvae and seldom need to be dewormed.

Pasture contamination may be reduced by resting the pasture or by grazing with another species such as cattle or horses which consume the infective larvae and then the larvae die in the digestive tract. Even grazing small ruminants with cattle or horses has been demonstrated to effectively reduce worm problems (Jordan et al., 1988). Making hay on pastures will clean many of the larvae off in the hay where they die in about a month. Those left on the field are killed by direct sunlight and low humidity. Cornell University recommends that in areas where hay cannot be made, the pasture can be mowed short at the end of the graze period to help infective larvae to
die. If the ground can be clean tilled and the larvae buried by an inch, they die. Kentucky State University recommends planting sorghum X sudan or millet for goats to graze. This takes advantage of tillage to bury the infective larvae at the beginning of the season and with these grasses the animals graze high away from the ground and avoid picking up infective larvae. Animals that are born early in the spring have fewer parasite problems than animals born later that must contend with a greater level of pasture infection due to warmer weather.

**Nutrition**

Nutrition helps to control parasites by providing nutrients for a strong immune system. The immune system is the body’s first line of defense against worms. The immune system requires nutrition (protein, energy, minerals and vitamins) for effective functioning, but genetics and stress are also factors in the strength of immune response against worms. The immune response of very young animals is reduced, whereas immunity wanes in elderly animals. Without appropriate nutrition, the immune response is compromised. The effect of nutrition on parasite infection has been extensively studied in Australia and New Zealand.

It appears from a number of studies that growth, pregnancy and lactation have a priority in terms of nutrient partitioning and with functioning of the immune system impaired if nutrient intake is insufficient for these functions (Coop and Kyriazakis, 1999). Therefore, nutrition of the animal is very important for the immune system to suppress worms. High producing dairy goats have been shown to be more susceptible to worms than low producers. Generally, an increased energy supply improves immune response little except for the effect that it may have on microbial protein synthesis (Houdijk et al., 2012), even though a fever response greatly increases energy requirement. Rumen undegradable protein supplementation from animal origin supplements appears to be superior to microbial protein for promoting immune response due to amino acid balance. There are many studies that demonstrate a reduction in fecal egg count with protein supplementation, especially with rumen undegradable protein.

Supplementation with protein has also been shown to reduce fecal egg counts of high producing dairy does. It has been demonstrated that animals infected with worms have greater protein secretion into the gut. This loss of protein is exacerbated by the reduction in appetite
associated with a worm infection. Protein supplementation not only stimulates the immune system, but often increases the level of animal production.

It is well known that during lactation, the immune response to worms is depressed (periparturient relaxation of immunity). Houdijk et al. (2000) demonstrated that increased intake of protein could overcome much of this phenomena, significantly reducing fecal egg counts. Fecal egg counts were substantially reduced in suckling lambs as compared to weaned lambs (Iposu et al., 2010) and worms had reduced fecundity (likely due to enhanced immune response of the animal). It appears that suckling (provision of milk protein) enhanced the immune response of lambs early in life. Although protein supplementation has been very well demonstrated to reduce worm problems in small ruminants, it is seldom utilized due to the cost of the supplement and feeding on a daily basis.

Several minerals are known to be important for a healthy immune system and deficiencies of these minerals will reduce immune system functioning. However, there has been little research addressing direct effects of minerals on worms or fecal egg counts. Hence, it is assumed that mineral supplementation improves immune response, it should improve suppression of worms. In general, when various minerals are deficient, supplementation will improve immune system functioning. There is little confirmation of supplementation of a mineral at supranutritional levels (levels which are substantially above requirements) is useful for immune system functioning. However, the requirements of various minerals for goats have not been adequately determined. In addition, the biological responses used to determine the adequacy of minerals are parameters such as growth and not immune functioning.
Copper, zinc, iron, selenium, cobalt, magnesium, manganese and possibly chromium (ultra-trace mineral) are all important for immune system functioning (Carroll and Forsberg, 2007). Excess iron, above 400 ppm, can be antagonistic to other minerals, so more is not necessarily better. Excess zinc may also be antagonistic to immune responses. Selenium works in conjunction with vitamin E as an antioxidant and is well-recognized as an immunostimulant in animals. Mineral contents of forages and feeds from nutrition tables cannot be relied upon to be accurate for a specific location. Forages and other feedstuffs need to be analyzed for mineral content. Liver is a very good tissue to analyze for determining mineral status of an animal, although blood may be necessary for a few minerals (Kincaid, 1999). Mineral supplements should be formulated to meet the deficits in a given area. They need to be monitored to insure adequate consumption.

Vitamins E, A, B6, B12, folic acid, carotenoids and C are well known for their support of the immune system (Carroll and Forsberg, 2007). Vitamin E of course works in conjunction with selenium and its functions in the immune system have been well documented. Vitamin A has been shown to improve immune functioning in deficient animals. Carotenoids have been shown to benefit immunity aside from being precursors of vitamin A. Vitamin C, apart from its function as an antioxidant, has been shown to stimulate immunity in several species. Although only vitamins E and A are required by the ruminant, we should make sure they are provided in adequate quantities, whether through green grass or as mineral/vitamin supplements.

References


Chapter 7. Overview on Enhancing Immune Responses to Internal Parasites in Small Ruminants

Zaisen Wang
The American Institute for Goat Research, Langston University, Langston, OK

Internal parasitism poses a major problem in the small ruminant industry. The USDA National Animal Health Monitoring System reported that 74% of sheep in the U.S. encountered gastrointestinal nematode parasitism (USDA, 2003). The level in goats may be more severe since goats may be more susceptible to nematode parasites than sheep (Lightbody et al., 2001). In clinical cases during 1993 to 2000 at the Auburn University Veterinary Medical Teaching Hospital, 91% of goats examined and treated related to internal parasite diseases (Pugh and Navarre, 2000). Parasitism significantly reduces performance of animals, including decreased body weight, fertility (older age at first kidding and longer intervals of kidding cycles), birth weights and growth rates, and markedly increased mortality of kids (Gatongi et al., 1997). Clinical signs may not develop until just prior to death. Sudden onset of weakness or death is a common finding in gastrointestinal parasitism cases (Valentine et al., 2007). The current management of internal parasitism mainly relies on uses of anthelmintics. This practice is challenged by widespread of anthelmintic resistance in the US and other parts of the world (Zajac and Gipson, 2000; Terrill et al., 2001; Kaplan, 2004). Mortenson et al. (2003) described resistance to two classes of drugs on 14 of 15 goat farms and resistance to 3 classes of anthelmintics on 5 of 15 goat farms. Kaplan et al. (2005) reported a total anthelmintic failure on a meat goat farm in Arkansas. Furthermore, the anthelmintic resistance in goats seems to be more prevalent than in other species of animals. Cabaret (2000) searched the literature and found that 30% of the anthelmintic articles on goats concerned anthelmintic resistance as compared to 22% for sheep, 18% for horses, and 6% for cattle. From these facts, it is not surprised that alternative approaches have attracted much attention. These approaches include enhancing resistance by nutritional manipulation, genetically selection, and vaccination; feeding or browsing forages with helmintic-suppressing properties; and avoiding contaminated pastures by grazing management.
Overview of immune responses to nematode parasites

Immunity to internal parasites in small ruminants has been evidenced by the existence of genetically resistant animals (Miller et al., 1998; Pena et al., 2006) though the mechanism of such resistance is not clearly demonstrated. Infections of nematode parasites result in mastocytosis, eosinophilia, and raised levels of serum immunoglobulins (Huntley et al., 1992, 1995; Lacroux et al., 2006). Immune responses to pathogens are mediated by cytokines. The most studied cytokines include interferon (IFN)-γ, interleukine (IL)-2, IL-12, and tumor necrosis factor (TNF)-α for Th1 type, and IL-4, IL-5, IL-10, IL-11, and IL-13 for Th2 type immunity. Nematode parasites (such as *Haemonchus contortus*) infection induces a decline in IFN-γ, increases in IL-4, IL-5 and IL-13 expression (Gill et al., 2000; Lacroux et al., 2006). These changes in cytokines may lead to recruitment of mast cells, eosinophils, macrophages, and lymphocytes at the infected site (Huntley et al., 1995; Balic et al., 2002; Perez et al., 2003). Mast cells may mediate expulsion of some worms from the host gut. A study found that mast cells induced intestinal paracellular permeability during helminth infection and the increased permeability was necessary for expulsion of worms from the intestine in mouse (McDermott et al., 2003). Eosinophils kill certain types of parasites by adhering to the worms or larvae and releasing their granule proteins and (or) superoxide radicals that are toxic to the tegumental membrane of parasites (Caulfield, 1980). Despite the fact that *H. contortus* infection induces eosinophilia and mastocytosis in goats and sheep, the eosinophil and mast cell counts in animals infected with *H. contortus* are not consistently related to worm resistance (Huntley et al., 1995; Chiejina et al., 2002; Muturi et al., 2005). In addition to these cells, immunoglobulin A (IgA), IgE, and IgG4 increase when animals are infected with *H. contortus* (Gill et al., 1994; Strain and Stear, 2001). These manifestations indicate a strong Th2 biased immune response. However, the long time survival and active fecundity of parasites in host animals, even in genetically parasite-resistant animals (Gill et al., 1994; Strain and Stear, 2001; Pena et al., 2006) suggest that the outcome of immunity is not sufficiently protective to the host. The situation is further complicated by possible regulation by parasites. Emerging evidence indicates that some nematode parasites are able to regulate their host’s immunity to create a favorable environment for themselves (Maizels et al., 2004). The alteration of immune responses by nematode infection has been evidenced in patients infected with other pathogens. The infection of human T cell lymphotropic virus type 1 activates T cells, triggers an uncontrolled proliferation.
of lymphocytes and a Th1 response characterized by a high level of IFN-γ and low levels of IL-5 and IL-10 expression (Porto et al., 2005). The expressions of these cytokines are reversed when patients are coinfectected with helminthic parasites (Porto et al., 2005). If the ineffective immune response in ruminants is resultant of parasitic regulation, it becomes possible that a protective immunity may be achieved by enhancing expression of certain cytokines of the hosts to overcome the parasitic regulation.

**Nutritional manipulation**

Protein and amino acids. Protein intake affects resistance to internal parasites in both ruminants and non-ruminants (Ing et al., 2000; Ford et al., 2001; Sykes and Coop, 2001). Compared with those fed a straw-based diet (low in protein), sheep fed a lucerne-based diet (high in protein) harbored less nematodes at 56 days after infection (Bawden, 1969). Supplementation of a hay-based diet with fish meal (van Houtert et al., 1995) or soybean meal (Strain and Stear, 2001) substantially reduced worm burden in sheep infected with nematodes *Trichostrongylus colubriformis* and *H. contortus*. In spite of the fact that goats are more susceptible to gastrointestinal nematode infections than sheep (Pomroy et al., 1986; Lightbody et al., 2001), goats respond to protein supplementation similarly. One investigation (Singh et al., 1995) showed that worm burden was lower in goats fed supplemental protein (cottonseed meal) than in control animals. The increased resistance to parasite infection by protein supplementation has been attributed to improved immunity. This is because protein supplementation increases availability of amino acids (Ipharraguerre et al., 2005) for immune cell proliferation and synthesis of immunoglobulins. On the other hand, many of the amino acids, including arginine, cysteine and glutamine, have been demonstrated to be involved in regulation of immune responses.

The regulating effect of glutamine on immunity in ruminants is evidenced by selectively promoting certain subset of lymphocytes. Postruminal glutamine supplementation in dairy cows consuming a corn silage and hay based diet increased the relative abundance of CD4+ cells (Doepel et al., 2006). CD4+ cells are shown to be responsible for resistance to parasites in genetically resistant sheep (Gill et al., 1993; Pena et al., 2006). Glutamine is used at a high rate by lymphocytes and even higher if the lymphocytes are activated by mitogens (Brand et al., 1989; Calder, 1995). The concentration of plasma glutamine decreased after extensive exercise and athletic training in humans (Hack et al., 1997), which are associated with an increased
susceptibility to infections suggestively due to decline in glutamine supply to lymphocytes (Newsholme and Calder, 1997). Indeed, increasing availability of glutamine enhances the production of cytokines, such as IL-2, IL-10, and interferon-γ, by cultured lymphocytes (Rohde et al., 1996; Yaqoob and Calder, 1998).

Cysteine may affect immunity through glutathione (GSH). The availability of cysteine is a limiting factor for synthesis of GSH in animals. Supplementation with cysteine or its precursor, methionine, to low protein diets increases tissue GSH concentrations (Alhamdan and Grimble, 2003). Glutathione plays a vital role not only in detoxification of free radicals and other oxidants (Deneke, 2000; Lu, 2000) but also in modulation of immunological functions in humans and animals (Droge et al., 1994). Clinical studies in humans indicate that HIV patients are more susceptible to other infections and GSH deficiency is a key determinant of survival in HIV disease (Herzenberg et al., 1997). Intracellular availability of GSH is critical to activity of many types of cells including immune cells. Rats fed a lipid peroxide diet showed a marked depression of intestinal mucosal proliferation and the depression was reversed by GSH supplementation (Tsunada et al., 2003). Lymphocytes cannot proliferate when GSH is depleted (Hamilos et al., 1989; Kavanagh et al., 1990). Intracellular concentration of GSH even determines the immune responsive types. Antigen-presenting cells direct T-cell responses toward a Th1 or Th2 type response, depending on the level of intracellular GSH. Th2 responses occur when GSH levels are low (Peterson et al., 1998). This may be because the antigen-presenting cells cannot process the antigen when their cellular concentration of GSH is low (Short et al., 1996). In addition, parasites may alter the concentration of GSH in the locale where they are dwelling. An in vitro study with nematode *Ascaris suum* indicated that the parasites could utilize exogenous GSH to replenish their GSH level (Hussein and Walter, 1996). If this finding is plausible to other nematodes, the internal parasites in animals will induce a GSH deficiency in their micro-environment. The low concentration of GSH may lead to a lack of immune response in the animal.

Arginine as an immune regulator plays a critical role in the development of immunity. Arginine supplementation in humans is associated with increased lymphocyte and monocyte proliferation and Th cell formation (Barbul, 1990; Cerra, 1991), activation of macrophage cytotoxicity, and increased cytokine production (Kirk and Barbul, 1990). Arginine deficiency depresses early B cell maturation (de Jonge et al., 2002). This may explain why arginine supplementation increases antibody production and enhances humoral immune response (Shang
et al., 2003; Moriguti et al., 2005). Furthermore, arginine may affect immunity by regulating lymphocyte migration between blood stream and other tissues. Sufficient supply of arginine is essential for mononuclear cell migrating through the layer of endothelial cells prestimulated with cytokines (Isenberg, 2003).

Doubtlessly the amino acids play a crucial role in immune regulation and it is very likely that they act orchestrally upon immune responses. More work focusing on the exact mechanisms by which the amino acids finely tune the immunity will be necessary for efficiently using amino acids in the strategy of parasite control in goats. In addition to elevating availability of amino acids, protein supplementation increases production of ruminal volatile fatty acids (VFA) (Ipharraguerre et al., 2005). The VFA are not only a major energy source for ruminants but also involved in immune regulation.

Volatile fatty acids. The three major ruminal VFA, acetic, propionic and butyric acids, can influence different arms of the immunity. Butyrate depresses production of IL-12 and IFN-γ, and increase expression of IL-4 and IL-10 in monocytes (Saemann et al., 2000). Since the IL-4 and IL-10 are important cytokines for Th2 immunity which is responsible for expulsion of some nematode parasites (Finkelman et al., 1997; Miller and Horohov, 2006), butyrate may be a potent player in the immune response to internal parasites in ruminants. Acetate and propionate are able to activate G protein-coupled receptors GPR41 and GPR43 in adipocytes and monocytes (Brown et al., 2003) and mast cells (Karaki et al., 2006). The activated GPR41 and GPR43 stimulate leptin production (Xiong et al., 2004; Covington et al., 2006). It has been reported that leptin tends to skew the immune response to a Th1 immunity (Lord, 2002), which may result from the negative regulation of regulatory T cells by leptin (De Rosa et al., 2007). From these findings it becomes possible that an enhanced immunity to internal parasites may be achieved by manipulating ruminal VFA ratio. Indeed the ratios of ruminal VFA can be altered by dietary protein sources (Litherland et al., 2000), and the proportion of butyric acid increases when animals are fed a diet high in protein (Ipharraguerre et al., 2005). Given the immune regulating nature, more work is needed to define the importance of VFA in immunity to internal parasites.

Minerals. The effects of minerals on immunity to internal parasitism have been reviewed recently in detail (McClure, 2008). Here we briefly review the application of copper oxide wire particles (COWP) in treatment of parasitism. The COWP, a supplementary form for copper deficiency in small ruminants, has been found to be effective in treatment of internal parasites in
sheep (Bang et al., 1990; Knox, 2002; Burke et al., 2004, 2006, 2007). The research in goats has produced mixed results. Glennon et al. (2004) observed a 32% to 41% reduction in fecal egg counts (FEC) in yearling goats receiving 5 g of COWP and a 57% reduction with a 10 g dose. In their subsequent study (Lugginbuhl et al., 2006) COWP did not seem to have any effect on FEC in weanling goats receiving 2.5 g COWP. However, Burke et al. (2007) found that COWP capsules were effective as an anthelmintic for up to 21 days after dosing and a dose as little as 0.5 g was effective for weanlings and 5 g for mature goats. Furthermore, COWP is more effective against H. contortus (Burke et al., 2007) and less effective to Teladorsagia, Trichostrongylus or Trichuris genus (Pomroy and Adlington, 2006). Nevertheless, more studies are needed in the area to use COWP capsules as an alternative anthelmintic.

Selection of resistant animals

The capability of resistance to internal parasites may be achieved through genetic selection. The genetic trait is measured generally by FEC. It has been noticed that some breeds of goats are more resistant to internal parasites than others. For example, the Small East African goats have lower FEC and higher PCV as compared to the Galla goats throughout the year and the difference is even greater during lactation (Baker et al., 1998). Similarly, Kiko or Spanish breeds seem to be more resistant than Boer goats (Browning et al., 2007). Pralomkarn et al. (1997) also observed that Thai native goats had much lower FEC than crosses of 50 and 75% Anglo Nubian. Only 8% of a trickle infection became established in the Thai native goats as compared to 17% for the Anglo Nubian crosses. Even in the same breed, FEC of individual animals are not normally distributed, but are rather over dispersed with a positive skewness (Hoste et al., 2001). A few individual animals of the population have most of the worms (Costa et al., 2000). Typically 20-30% of the goats in a herd have 70-80% of the worms and the FEC are relatively repeatable (Hoste et al., 2001). Vlassoff et al. (1999) observed a considerable individual variability in FEC of Angora goats following either a natural or experimental challenge infection. Repeatabilities of 0.22 to 0.41 are similar to those observed in sheep. This indicates that they are heritable since the repeatability is the upper limit for heritability. Though the heritability estimates for FEC are moderately low in goats (average 0.29; range 0.19 to 0.37) (Mandonnet et al., 2001; Olayemi et al., 2002), establishing a resistant sub-breed is possible. Ruvuna et al. (1984) was able to select divergent groups of animals. The selected group of animals which comprised 11% of the initial
population had a FEC of only 15% of the remainder of the population. Rohrer et al. (1991) reported a heritability of FEC in this population to be 0.4. The does of cashmere goats that were selected for seven generations for parasite resistance in Scotland had 35% lower fecal egg counts than their unselected counterparts (Jackson, 2002).

The mechanism for heritability is not well established. The genes for the major histocompatibility complex of the immune system in sheep have been identified as candidates (Benavides et al., 2002). The genetic trait measured (FEC) may be affected directly by genetic effects on immune system (Fakae et al., 1999), or indirectly through improved diet selection for improved nutrition, or grazing habits which reduce exposure to infective helminth larvae (Hutchings et al., 2007). There may also be other factors involved as Hutchings et al. (2007) identified that animals with lower FEC picked up fewer larvae due to their grazing habit. Nevertheless, there is a great need for the establishment of a breeding program utilizing the major goat breeds available in the US. Genetically selected lines of goats that are resistant to *H. contortus* could be distributed to aid in developing genetic resistance in goat herds. In addition, this research would increase our understanding of mechanisms of inheritance as well as biology of the host-parasite interaction. If such program is initiated by a national breed registry, the production losses, cost of treatment, morbidity and mortality in the goat industry could be greatly reduced. Producers could also use FAMACHA scores and/or the number of times that a goat required deworming as the basis for culling and gradually eliminate those animals (and their genes) from the flock. This would be very easy to implement. The meat goat industry could do well to follow the program initiated by the Katahdin sheep breeders to measure resistance under field conditions by taking fecal samples according to a standardized protocol and then calculating expected progeny differences for parasite resistance. Such information can be used for genetic selection of animals for resistance to internal parasites.

**Vaccination**

Early research showed that sheep could be vaccinated with X-irradiated *H. contortus* larvae and develop immunity to the worm (Jarrett et al., 1959). In subsequent research immunoprotection against *H. contortus* was conferred after immunization with cysteine protease enriched protein fraction (Ruiz et al., 2004). A degree of immunity in goats can be developed by natural parasite infection (Pomroy et al., 1989). There has been a tremendous effort in Australia and New Zealand
to develop a vaccine for control of helminth. Although most vaccines would be specific for only one species of helminth, this would still be effective since usually only one species predominates in a given season.

**Summary and future research**

Internal parasitism significantly reduces performance of goats and increases costs of production. In the face of anthelmintic resistance, alternative approaches have been explored extensively. Nutritional, especially protein, supplementation plays an important role in immune response. Amino acids do not only serve as substrates for immunoglobulin synthesis but also act as regulators of cytokine expression. The same importance is for VFA that exert regulatory roles in immunity. This area warrants more work to clarify how these molecules mediate the immune responses. The results of using COWP to treat internal parasites in goats are not consistent and more studies are needed to substantiate the effectiveness and define the dosage. Genetically selection for resistant populations is a promising approach and there is a great need for the establishment of a breeding program utilizing the major goat breeds available in the US. The tannin-containing plants and nematophagus fungi possess a potential anthelmintic activity. More research is needed to finalize the dosages and a strategy to apply them in the practical goat production. Most importantly, the mechanisms of immune responses to internal parasites are not understood fully. Much work is needed to demonstrate how the host to react to the infection of parasites and whether the parasites manipulate the immune outcome of the host. Only if the specific pathways of immune responses are clearly elucidated, a precise manipulation on immunity becomes possible.

**Reference**


As it is well known, barber pole larvae show increased resistance to commercial anthelmintics drenched to small ruminant livestock, and producers are looking for other alternatives. What about bypassing the livestock entirely and applying a larvicidal product directly on the pasture itself?

Studies we conducted at North Carolina State University showed that 96.6% L3 barber pole larvae were not moving or dead when immersed in solutions of liquid nitrogen fertilizer (containing 32.7% urea and 42.2% ammonium nitrate [21.1% ammonium and 21.1% nitrate), corresponding to field applications of 30 lb of nitrogen per acre. Another laboratory study showed that a 10% solution of household bleach (5.25% sodium hypochlorite) resulted in 99.1% of L3 larvae not moving or dead. Higher solutions of household bleach caused lysis (disintegration) of the larvae.

The larvicidal action of liquid nitrogen fertilizer has been attributed to the toxic qualities of ammonia and nitrate, as well as to the increased osmotic pressure created with an
accompanying water loss when in direct contact with L3 larvae. Do you remember as a child how water leaked out of slugs when dribbling table salt on them?

One agricultural practice recommended by the N.C. Cooperative Extension Service is to apply 50 lb of nitrogen fertilizer per acre to cool-season grasses such as fescue and orchardgrass both in early spring and late summer, and one or several applications during summer for warm-season grasses such as bermudagrass, millet and sorghum. So, could we pop two balloons with one dart by fertilizing pastures with liquid nitrogen fertilizer to promote forage growth and at the same time reducing pasture nematode larvae population, their subsequent ingestion by grazing animals and ultimately reducing gastrointestinal parasite loads?

**Experimentation**

We conducted 3 experiments to test this hypothesis on predominantly tall fescue pastures, one in spring and two in fall. In each of these experiments, a field was grazed by does heavily infected with gastrointestinal parasites for 21 to 26 days until the ground was well covered by fecal pellets. The goats
were then removed from the field, which was immediately flail-chopped (forage was cut and moved off-site) to a height of 5 inches. Then, according to experimental principles that took into account differences in slope, soil, etc., the fields were divided into several sub-pastures so that each experimental treatment was replicated 3 or 4 times. Sub-pastures within each replication were then randomly assigned to be either treated with liquid nitrogen fertilizer or untreated (control). Granular urea was applied to the untreated (control) sub-pastures to ensure that forage would grow at an equal rate in all sub-pastures and because granular urea has a very limited negative effect of L3 larvae survival. As infective L3 larvae migrate up the blades of forage crops during mornings and evenings with the dew, we sprayed pastures with liquid nitrogen fertilizer starting at 6:30 AM or after 4PM depending on weather conditions. Large amounts of liquid nitrogen can be lost from evaporation to the atmosphere if applied under full sun, and may also burn the tip of leaves.

A 4th experiment was conducted on bermudagrass during the summer. The differences in preparing the field compared to the previous 3 experiments were the following: the field was only contaminated for 11 days, forage was flail-chopped 3 days later, and liquid nitrogen fertilizer was only applied 11 days later.

Five days after applying liquid nitrogen or urea, ‘clean’ goats were randomly assigned to each plot and control-grazed. Why the 5-day delay? Because we were concerned with nitrate poisoning if goats had been turned immediately into the sub-pastures that had been sprayed with liquid nitrogen.

Fecal egg counts were performed on all goats as they were entering the sub-pastures to get a baseline count. We also took blood samples to determine packed cell volume, and
FAMACHA-scored the lower eyelids of each goat. All sampling and scoring took place on a weekly basis.

The Bottom Line

Nothing is simple when dealing with biology and dynamic environmental conditions one cannot control.

1. Results from experiments conducted in the laboratory do not necessarily translate into the same outcome in field situations. The field experiments described above necessitated a large amount of coordination, resources and labor, the results were disappointing with the exception of Expt. 1 conducted under ideal environmental conditions (rainfall and temperature), and many questions still remain unanswered.

2. The main limitation of liquid nitrogen is that it kills or incapacitates larvae on contact and that its effectiveness is very short-lived, such that on a heavily contaminated field another crop of larvae may hatch and molt as soon as the effect of liquid nitrogen has disappeared (Expt. 4). In addition, as larvae can bury in the forage mat or the soil, or stay at the base of forage plants for protection from desiccation, it is important to spray liquid nitrogen very early in the morning when the dew is still present, or late in the day under cloudy skies, on forage that is not more than 5 inches in height. Therefore, another limitation is owning or having access to a flail-chopper, a tractor and a boom-sprayer. Finally, not everybody has easy access to a source of liquid nitrogen fertilizer.

3. Adequate temperature and moisture are necessary for larvae to hatch, molt and migrate onto the leaves of forage crops. In the absence of rainfall during the contamination period (Expt. 2), only a minimum number of larvae will hatch and molt, whereas the majority will stay protected in the fecal pellets and wait for outside conditions that will favor their survival.

4. Spraying liquid nitrogen on pastures during dry conditions (Expt. 2) will be less effective or not effective at all due to the combination of the short effectiveness of liquid nitrogen and the low number of larvae that hatched, molted and migrated to grass blades. In addition, liquid nitrogen will have no effect on eggs still in the fecal pellets. If adequate amounts of rain falls following a drought, fecal egg counts will shoot up within the next 2 to 3 weeks (Expt. 2), even on the plots previously sprayed with liquid nitrogen.

5. Too much rain on a field having a certain slope may mean that most of the feces and larvae could be washed away from the experimental plots, resulting in low fecal egg counts for more than a month (Expt. 3) due to a hurricane passing through the day after nitrogen was applied. Nonetheless, larvae and feces may accumulate on a field.
located downslope that may belong to you or your neighbor, and it could become heavily contaminated without goats having grazed it.

6. Should we have used another approach, such as waiting for a longer period of time between contamination and spaying to give the majority of fecal eggs the opportunity to hatch and molt to achieve a better ‘kill’? Or, should we have used split applications to also achieve a better kill? Possibly! In the case of split applications, fast forage growth may decrease the direct contact of liquid nitrogen fertilizer with L3 larvae. Or should we have used applications of liquid nitrogen fertilizer and in addition several separate applications of household bleach solutions?

7. The objectives of the experiments described herein were never to pretend that liquid nitrogen be used as the sole source of a gastrointestinal parasite control program, but that hopefully it could be included as part of an integrated program. Environmental conditions and their interaction with forage growth and the dynamic life cycle of gastrointestinal nematodes represent a complex challenge that may greatly affect outcomes.
Chapter 9. An update on anthelmintic resistance on goat farms in the United States

Sue B. Howell, Bob E. Store, and Ray M. Kaplan

Why is resistance important?

- Anthelmintic (dewormer) resistance is highly prevalent in parasites of livestock worldwide.
- Multiple-drug resistance and "total anthelmintic (dewormer) failure" are common.
- Resistance is present in worm species of all livestock hosts:
  - Problem worst in small ruminants (goats especially)
  - Becoming increasingly severe in horses, cattle, farmed deer, camelids, exotic ungulates (2003)

IMPORTANT GASTROINTESTINAL (GIN) PARASITES OF GOATS AND SHEEP

1. Haemonchus contortus  
   barberpole worm

2. Teladorsagia (Ostertagia) circumcincta  
   brown stomach worm

3. Trichostrongylus colubriformis  
   bankrupt worm
**Introduction**

**BUT MOST IMPORTANT PARASITE IN THE SOUTHERN U.S. IS...**

*Haemonchus contortus*

(barberpole worm)

**WHY??**

- Short life cycle — less than 3 weeks
- High fecundity (high egg production)
- Each female worm produces ~5,000 eggs per day
- Little acquired immunity for young animals
- Kids and lambs are highly susceptible to parasites

**Introduction**

**ALSO, *HAEMONCHUS CONTORUTUS* IS...**

- Highly pathogenic — potential for rapid and acute blood loss
- Which causes severe anemia
- Hypoproteinemia (low protein) because of blood loss (Bottleneck)

**Introduction**

*Teladorsagia and Trichostrongylus*

Important parasites of sheep/goats in cool climates and/or those with summer rain
- e.g., U.S., Scotland, NZ, parts of Australia

- Affect appetite, digestion and nutrient utilization
- Clinical symptoms: diarrhea, reduced appetite, reduced growth — weight loss

**Introduction**

**GIN Life Cycle**

**Introduction**

**So how do we detect resistance in goats??**

- **in vivo** (fecal egg count reduction test)

- **in vitro** testing

**Introduction**

**In vitro bioassay**

- DrenchRite<sup>®</sup> larval development assay (LDA) has been used in our lab since 2000
- History of D'Rite
- We have been collecting data on drug resistance in GIN; mainly *Haemonchus contortus* (barberpole) in small ruminants since that time
Introduction

Drug conc doubles from low to high every well

EC50: "Effective conc" of drug = where 50% of the L3 are killed by that drug conc. = determining the level of resistance for that drug

Materials and Methods

- Eggs were isolated from feces then stored and filtered over 30 μm mesh
- Eggs were purified using a sucrose gradient
- Plated into wells of the plate, with nutritive media, incubated for 7 days at 25°C
- Assay was terminated with 50% Lugo's solution after the incubation period
- L3 larvae were counted and identified to species

Evaluation

- D'Rite data were evaluated to determine the effective conc (EC50) for BZ, LEV, IVM, and EC95 for ivermectin
  - based on these data resistance status were determined
  - EC50 = where 50% larvae are killed
  - EC95 = where 95% larvae are killed

- Data were analyzed
  - to examine trends over time intervals

Objectives

- Evaluate the increase in prevalence of resistance in the most recent goat samples tested (and compare with other samples)
- Look at the drug conc. which can measure the increase in the degree of resistance to Barberpole worm in goat farms

Materials and Methods

- Fecal samples from 21 goat farms (15 states) from 2013 - 2016 were evaluated for resistance
  (and compared to previous samples)
- Samples were packaged anaerobically, shipped to UGA and processed within 72 hours
- Eggs were isolated for the D'Rite and coprocultures were performed for each farm

<table>
<thead>
<tr>
<th>Years</th>
<th>Goats</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>2013</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>2014</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>2015</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>2016</td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>
Analysis

- Statistical analyses were performed using Graph Pad Prism 6.0 (p < 0.05 was considered significant).
- Differences in EC_{50} and EC_{95} values were analyzed using one-way ANOVA followed by Keuskal-Wallis test for the individual species and yearly grouping for goats.
- Differences in EC_{50} and EC_{95} were analyzed using t-test with Welch's correction for the species by yearly groupings.

Results

<table>
<thead>
<tr>
<th>YEARS</th>
<th># Farms</th>
<th>% IVM Resistant</th>
<th>% MOX Resistant</th>
<th>% TOTAL Drug Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>2013-16</td>
<td>21</td>
<td>95%</td>
<td>62%</td>
<td>8/21 = 38%</td>
</tr>
</tbody>
</table>

Summary

21 goat farms (2013-16) were evaluated for resistance to BZ, LEV, IVM, MOX and compared to previously tested goat samples to look at recent trends in resistance to goats.

Summary

- Prevalence of resistance for BZ was 100% for 2013-16.
- Prevalence of resistance for LEV was 57% for 2013-16.
- A sizeable increase in the mean drug concentration trended steadily upward for BZ, IVM (EC_{50}) and MOX (EC_{95}) since 2000.
- Although the mean drug concentration (EC_{50}) for LEV remained relatively stable, the number of resistant goat samples increased steadily since 2000.
Summary

Total dewormer failure was detected in
- 2000 – 04 - 1 of 37 farms (3%)
- 2013 – 16 - 6 of 21 farms (30%)

Conclusions

These data show that drug resistance is at a critical level for goats in the U.S.
Clearly, there is need for sustainable integrated parasite management (IPM) programs to be broadly implemented.

Summary

- Prevalence of resistance for IVM was 95% for 2013-16
- Prevalence of resistance for MOX was 82% for 2013-16 (0% in 2001)

Conclusions

“Novel Approaches” to parasite management
1. Selective Treatment (FAMACHA)
2. Copper oxide wire particles
3. Sericea lespedeza (condensed tannins)
4. Using drug combinations
5. Improved pasture management (can include co-grazing)
6. Nematode trapping fungus
www.wormx.info

Acknowledgments

Dr. Min and Tuskegee University
Dr. Ray Kaplan
Dr. Joan Burk
American Consortium for Small Ruminant Parasite Control Group (ACSRPC)

Questions?

For more information about the DrenchRite assay:
E-mail: tcdb@uga.edu
Chapter 10. SERICEA LESPEDEZA for parasite control

Thomas H. Terrill, Ph.D.
Fort Valley State University, Georgia
Sericea Lespedeza Cultivars

<table>
<thead>
<tr>
<th>Name</th>
<th>Year Available</th>
<th>Institution</th>
</tr>
</thead>
<tbody>
<tr>
<td>72</td>
<td>1920</td>
<td>USGA</td>
</tr>
<tr>
<td>72</td>
<td>1921</td>
<td>SCS</td>
</tr>
<tr>
<td>72</td>
<td>1922</td>
<td>SCS</td>
</tr>
<tr>
<td>72</td>
<td>1960</td>
<td>Alabama Ag. Ext. Sta.</td>
</tr>
<tr>
<td>72</td>
<td>1961</td>
<td>Georgia Ag. Ext. Sta.</td>
</tr>
<tr>
<td>72</td>
<td>1962</td>
<td>Mississippi Ag. Ext. Sta.</td>
</tr>
<tr>
<td>72</td>
<td>1977</td>
<td>Alabama Ag. Ext. Sta.</td>
</tr>
<tr>
<td>AU Grazer™</td>
<td>1957</td>
<td>Alabama Ag. Ext. Sta.</td>
</tr>
</tbody>
</table>

Sericea Lespedeza in USA today
- Older cultivars used for stabilizing soils from surface-mine coal sites, roadbanks, and other disturbed or eroding sites, improving wildlife habitats
- ‘Common’ sericea lespedeza is growing on thousands of acres in the mid-western states
- AU Grazer™ is the primary cultivar planted as grazing and hay crop

Agronomic Advantages of Sericea Lespedeza
- Grows on a wide range of soil types, including acidic, infertile sites
- Legume, needs no N fertilization
- Deep rooting, reduces need for P fertilization
- Drought tolerant once established
- Insect damage, disease problems minimal
- Tendency to shed lower leaves, leading to:
  ✓ Improved soil fertility, soil structure
  ✓ Reduced soil erosion

Sericea lespedeza - Forms
- Fresh forage
- Hay
- Leaf meal
- Pellets
- Silage

Dry Matter Production of Sericea Lespedeza
- 3-4 tons of hay per acre in Georgia (Hoveland et al., 1990)
- Up to 2 tons per acre of leaf meal from SL in Alabama

Nutritional Value of Sericea Lespedeza
- Older cultivars had thick, woody stems that reduced nutritional value
- High-CT cultivars developed with finer stems
- High concentration of condensed tannins (CT) reduced intake, digestibility
- Sun-drying of high-CT SL improved intake and digestibility
- Low-CT cultivars developed with higher digestibility
Nutritional Value of Sericea Lespedeza

- High-CT sericea adequate nutrition as pasture and hay crop for beef cows and calves, animal performance similar to bermudagrass
- Not recommended to graze growing calves on high-CT SL
- Cattle and sheep grazed on “AU Lotan” in South Africa
- Hay, pelleted SL readily consumed by all classes of livestock (cattle, sheep, goats, horses, llamas, exotic hoofstock)

Nutritional Value of Sericea Lespedeza for Small Ruminants

- Goats readily graze high-CT SL
- Sheep graze SL after an adjustment period
- Adequate nutrition for older animals (bucks, does, rams, ewes)
- Good nutrition as short-term feed (no more than 8 weeks) for weaned lambs and kids
- Reduced gains in growing kids and lambs compared to perennial grasses after 8-10 weeks

Health Benefits for Livestock

- Anti-bloat
- Reduces somatic cell count in goat milk
- Anti-parasitic

Ethno-medical Uses of Sericea Lespedeza in China

- Ye men guan (pinyin)
- Whole plant boiled extract for:
  - Anemia
  - Dysentery
  - Dental caries
  - Dog bite
  - Hemorrhage
  - Hemin
  - Infnantile meningitis
  - Skin ulcers
  - Snake bite
  - Tuberculosis

Other benefits

- Lower ruminal methane production
- Reduced urinary losses of N as urea
- Kills houseflies in animal feces

CONDENSED TANNINS IN FORAGES

CT concentration, composition varies with:

- Cultivar
- Plant part (Leaves vs stems)
- Plant maturity (leafy vs reproductive growth)
- Processing method
  - Sun-curing for hay
  - Grinding and pelleting
  - Ensilage

(Puchalski et al., 2005; Min et al., 2008; Naumann et al., 2013)
ANTHELMINTIC (AH) EFFECTS OF CONDENSED TANNINS

- Associated with their ability to form complexes with both plant and parasite proteins
- Mechanism of action explained by two hypotheses:
  - Indirect effects
  - Direct effects

Anti-parasitic effects of grazed SL - Summary

Grazing Sericea Lespedeza Works for GIN Management

- Different locations
  - USA (North Carolina, Georgia, Louisiana, Arkansas)
  - South Africa (KwaZulu Natal)
- Different animal species, breeds, ages
  - Pre-weaned, weaning lambs, weaned kids (6 to 12 months)
  - Spanish, Dorset cross
- Different SL cultivars
  - AU Grazer
  - AU Lotan
  - Other cultivars not tested

Grazing trials with SL

- 57% reduction in GIN egg counts in does grazing SL compared with tall fescue pasture
- Lower numbers of adult worms in "tracer" kids grazing SL
  - Haemonchus contortus (94%)
  - Teladorsagia circumcincta (20%)
  - Trichostrongylus colubriformis (46%)
  - Min et al., 2002; 2003
- 95.4% and 71.4% reduction in GIN egg counts in kids grazing SL or SL + bermudagrass (BG) pasture compared with BG pasture only
  - Mecham et al., 2014

Grazing of Sericea Lespedeza

- Limited to late spring, summer, early autumn months, areas where SL is adapted
- Ensiling SL, or sun-drying, processing into leaf meal, pellets
  - Gives farmers flexibility in use of SL on-farm
  - Facilitates storage, transport to areas where SL is not adapted
  - Generates heat, reduces extractable CT, increases CT bound to protein

Does sun-drying, grinding, pelleting or ensiling of sericea lespedeza affect its anti-parasitic properties?
Effect of Feeding Sericea Lespedeza Hay on Fecal Egg Count (FEC) of Goats

% Larval survival in fecal cultures

Effects of Feeding SL Hay on Adult Worm Species in Goats

Effects of Feeding SL pellets on goat FEC

Effects of Feeding Ensiled SL on goat FEC

Hay trials with sericea lespedeza
- Goats
  - FVSU, USDA/ARS Booneville, AR
- Sheep
  - LSU, USDA/ARS
- Cattle
  - LSU
- Llamas
  - FVSU
**Sericea lespedeza pellet experiments**

- Gastrointestinal nematodes (*H. contortus*)
  - Sheep (LSU, USDA/ARS)
  - Goats (FVSU, NCA&T, USDA/ARS)
  - Beef cattle (FVSU)

- Coccidia (*Eimeria* spp.)
  - Sheep (USDA/ARS, LSU)
  - Goats (FVSU)

**Bottom Line**

**DRYING AND PROCESSING SERICEA LESPEDEZA DOES NOT REDUCE IT’S ANTI-PARASITIC EFFECTIVENESS**

---

**Other Questions about Anti-parasitic Properties of SL**

- How much is needed to achieve anti-parasitic effect?
- What parasites is it most effective against?
- Can parasites develop resistance to the effects of SL?

**Effects of Feeding SL Hay on goat FEC: Dose Titration Study**

---

**Effect of feeding whole plant and leaf only SL meal at 25% of the diet on goat FEC**

**Gastrointestinal Nematodes**

<table>
<thead>
<tr>
<th>Species</th>
<th>% reduction of adult females relative to control</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Haemonchus contortus</em></td>
<td>77%</td>
</tr>
<tr>
<td><em>Trichostrongylus colubriformis</em></td>
<td>50%</td>
</tr>
<tr>
<td><em>Teladorsagia circumcincta</em></td>
<td>36%</td>
</tr>
</tbody>
</table>
**Eimeria spp. (Coccidia) OPG**

Can parasites develop resistance to the effects of Sericea Lespedeza?

**Adaptation to High-Tannin Diet**
- Increased production of “proline-rich” saliva
- Increased production of tannin-degrading ruminal bacteria
- Recent results with pelleted SL showed lack of effectiveness against sheep and goat GIN in Arkansas
- The same pellets were effective against GIN and coccidia in Georgia in both lambs and kids

**Anti-parasitic Effectiveness of Sericea Lespedeza**
- Consistently effective in all forms tested with goats in Georgia and North Carolina
- Consistently effective with sheep in trials in Louisiana
- Variable results with sheep and goats in Arkansas
- Infection level with *Haemonchus contortus*?
- Over-heating of pellets?
- Possible regional differences in parasites?
Sericea Lespedeza Feeding Recommendations for Parasite Management

- Can be fed fresh (grazed, cut-and-carry), dried (hay, leaf meal, pellets), or preserved (ensiled)
- 25% or more of the diet
- Supplement energy or protein to meet nutritional needs of specific classes of animals

Sericea Lespedeza Feeding Recommendations for Parasite Management

- For control of Coccidia or Barberpole worm, begin feeding 2 weeks prior to periods of stress, at least 6 weeks afterwards
- Susceptible animals
  - Kids and lambs at weaning
  - Does and ewes during kidding/lambing, in early lactation (particularly with twins or triplets)

Sericea Lespedeza Feeding Recommendations for Parasite Management

- Currently, recommend feeding SL for no longer than 8 weeks at a time with young kids and lambs because of possible binding of trace minerals in some locations/farms
- Not a problem with older animals

Future Research with Sericea Lespedeza for Parasite Control

- Grazing trials with sheep and goats
  - Pure stands
  - In combination with other forages
  - SL as deworming paddock
- Research with SL as dried or ensiled feed
  - Ingredient in complete feeds
  - Pasture supplement
  - Component of TMR for feedlot, confinement feeding
  - Beef and dairy cattle, dairy goats, llamas and alpacas, pigs, poultry, zoo animals

Future Research with Sericea Lespedeza for Parasite Control

- Establishment for organic or grass-fed small ruminant production
- On-farm trials
  - Use of SL grazing, hay, pellet, or silage feeding for as part of a parasite management program in combination with FAMACHA, copper oxide wire particles, other novel control technologies
Commercial sources for sericea lespedeza seeds and pellets?

- Seeds and leaf meal pellets of 'AU Grazer' can be purchased from a company in Alabama:

  Sims Brothers Seed Company  
  Union Springs, AL  
  Phone: 334-738-2619  
  Email: simsbrothers.com
Chapter 11. Sustainable Non-synthetic Parasites Control in Our Farm
The way to go?

Byeng R. Min, Ph.D.
Tuskegee University, AL
Minh@mytu.tuskegee.edu
Condensed tannins on DM intake of sheep and goats

Sheep

Condensed tannins & dry matter digestibility of sheep and goats

Sheep

Condensed tannins & nitrogen digestibility of sheep and goats

Sheep

Condensed tannins on rumen ammonia-N concentration in sheep and goats

Sheep

Condensed tannins and ratio of nitrogen retention/N-intake of sheep and goats

Sheep

Condensed tannins & ADG

Sheep
The effect of forage CT concentration on wool production.

(Min et al. 2003)

Alternative Sustainable Approaches to Parasite Control

Sheep and goats - Parasites of concern

- Three main species
  - *Haemonchus contortus*
    - "Barber pole worm"
    - Kills sheep
  - *Trichostrongylus colubriformis*
    - "Black Sour worm, Bankrupt worm"
    - Lies in the mucous layer of the small intestine and feeds on mucus
  - *Ostertagia circumcincta*
    - "Small Brown stomach worm"
    - Lives in mucus layer and feeds on mucus

Break the life cycle of parasites infection

- It is increasingly evident that parasite control programs based on anthelmintics are failing.
- Alternative parasite control strategies are necessary.
- Alternative non-chemical control strategies have been suggested.

Gastrointestinal parasite nematodes population in goats (n = 90) over 18 farms (Feb. to June) in 9 counties of AL, 2010.

- More than 60% of the goats were considered wormy having more than 500 FEC per gram of feces.
- *Trichostrongylus and Ostertaga* followed by *Haemonchus* prevalent in numbers.
Opportunities to Control Internal Parasites:

- Selicea lespedeza
- Pine bark
- Peanut skin

Tannins containing Selicea lespedeza and Parasites control

- 50 Anthelmintic-drenched Angora does were randomly allocated to three treatments.
- Group 1: Goats grazing on predominant by Selicea lespedeza (SL; 10 does; set stocked; 31 days)
- Group 2: Goats grazing on non-CT-containing mixed forage only (20 does; set stocked; 31 days)
- Group 3: Rotational grazing with 2 vaci and without CT-containing forages (20 does)

The effect of lespedeza on the liveweight gain and dry matter intake by Angora does and kids

<table>
<thead>
<tr>
<th>Forage</th>
<th>SL</th>
<th>CF</th>
<th>Rotation</th>
<th>SEM</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total tannins (% DM)</td>
<td>15.0</td>
<td>0.3</td>
<td>11.0</td>
<td>0.8</td>
<td>-</td>
</tr>
<tr>
<td>Does (kg/d)</td>
<td>1.1* 1.0* 2.4*</td>
<td>1.7</td>
<td>0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kids (kg/d)</td>
<td>97</td>
<td>104</td>
<td>105</td>
<td>11.6</td>
<td>NS</td>
</tr>
<tr>
<td>Does DMI (kg/d)</td>
<td>3.2</td>
<td>4.1</td>
<td>3.4</td>
<td>0.75</td>
<td>0.9</td>
</tr>
</tbody>
</table>

SL: Selicea lespedeza; CF: crested wheat; rotation, SL=CF


The effect of lespedeza on fecal egg count (FEC), parasite egg hatch and packed cell volumes (PCV) in Angora does and kids

<table>
<thead>
<tr>
<th>Forage</th>
<th>SL</th>
<th>CF</th>
<th>Rotation</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEC (egg/g)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Does</td>
<td>186*</td>
<td>1,148*</td>
<td>428*</td>
<td>0.01</td>
</tr>
<tr>
<td>Kids</td>
<td>550*</td>
<td>3,600*</td>
<td>2,757*</td>
<td>0.01</td>
</tr>
<tr>
<td>Total egg hatch (%)</td>
<td>7.7*</td>
<td>14.5*</td>
<td>16.3*</td>
<td>0.01</td>
</tr>
<tr>
<td>PCV (Does)</td>
<td>27*</td>
<td>24*</td>
<td>26*</td>
<td>0.01</td>
</tr>
</tbody>
</table>

SL: Selicea lespedeza; CF: crested wheat; MEX: SL=CF


Condensed tannins containing forage S. lespedeza and fecal egg counts (egg/g) in Angora does


78
Worm burden and species recovered from the abomasum and small intestine of Angora kids.

<table>
<thead>
<tr>
<th></th>
<th>Abomasum</th>
<th>Small intestine</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HM</td>
<td>TD</td>
<td>TS</td>
</tr>
<tr>
<td>SL</td>
<td>17a</td>
<td>0</td>
<td>17</td>
</tr>
<tr>
<td>CF</td>
<td>267a</td>
<td>133a</td>
<td>33</td>
</tr>
<tr>
<td>Rotation</td>
<td>83a</td>
<td>25a</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>SEM</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>P-value</td>
<td>0.01</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Haemonchus (HM), Teladorsagia (TD), and Trichostrongylus (TS) recovered from Ab., Trichostrongylus (TS) and Nematodirus (NT) recovered from Sl.

SL, Tetanus turgidus, CF, subgrass favor, rotation, SL+CF

Mun et al. (2005). Vet. Parasitol. 130: 105-113

---

**Pine park**

- Pine bark contains 11% CT
- Readily available forest by-product
- Natural product and generally regarded as safe (GRAS)

---

**Pine bark**

- Alabama has 23 Million ac of commercial timberland
- Alabama is the 2nd largest timberland in the US, producing $15 billion worth product in 2005
- Among those, 40% of timberland is pine tree, especially long-leaf pine.

---

**Materials and Methods**

- 22 antherminic-drenched Kiko-cross goats were randomly allocated to 3 treatments.

- Group 1: 0.17% CT DM
- Group 2: 1.8% CT DM
- Group 3: 3.2% CT DM

Control: 0% 15% 30%
**Ingredient of different experimental diets containing ground pine bark**

<table>
<thead>
<tr>
<th>Item, % as-fed</th>
<th>Diet</th>
<th>Control</th>
<th>15% PB</th>
<th>30% PB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pine bark, ground</td>
<td>0</td>
<td>15</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>Wheat straw, ground</td>
<td>10</td>
<td>15</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Corn</td>
<td>19</td>
<td>19</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>SBM, 48%</td>
<td>18.5</td>
<td>20</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>Soyhulls</td>
<td>4.5</td>
<td>5</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Alfalfa meal</td>
<td>5</td>
<td>3</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Molasses</td>
<td>6</td>
<td>6</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Vitamins &amp; minerals</td>
<td>0.5</td>
<td>0.5</td>
<td>0.6</td>
<td></td>
</tr>
<tr>
<td>Bermudagrass hay</td>
<td>15</td>
<td>15</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Condensed tannins</td>
<td>0.19</td>
<td>1.63</td>
<td>3.20</td>
<td></td>
</tr>
</tbody>
</table>

**Carass measurement**

**Fecal Egg & Coccidia counts**

Min et al., 2012

**Haemonchus contortus Worm counts**

Min et al., 2012

**Apparent nutrient digestibility**

<table>
<thead>
<tr>
<th>Item</th>
<th>Diet</th>
<th>Control</th>
<th>15% PB</th>
<th>30% PB</th>
<th>SEM</th>
<th>Contrast (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DMI, g/d</td>
<td>1020</td>
<td>1018</td>
<td>1099</td>
<td>80.1</td>
<td>0.49</td>
<td>0.68</td>
</tr>
<tr>
<td>Digestibility, %</td>
<td>66.3</td>
<td>63.3</td>
<td>65.0</td>
<td>1.67</td>
<td>0.59</td>
<td>0.26</td>
</tr>
<tr>
<td>CP</td>
<td>73.5</td>
<td>71.1</td>
<td>69.6</td>
<td>1.64</td>
<td>0.09</td>
<td>0.84</td>
</tr>
<tr>
<td>NDF</td>
<td>48.8</td>
<td>39.4</td>
<td>36.5</td>
<td>2.84</td>
<td>0.01</td>
<td>0.36</td>
</tr>
<tr>
<td>Lignin</td>
<td>41.1</td>
<td>27.7</td>
<td>18.2</td>
<td>4.50</td>
<td>&lt;0.01</td>
<td>0.73</td>
</tr>
</tbody>
</table>

**Animal performance and feed efficiency**

Min et al., 2012

**Packed cell volume**

Min et al., 2012

**ADG (g/d):**
- Control: 91.1
- 15% PB: 114.3
- 30% PB: 136.2
- SEM: 6.91
- P-value: 0.001
- Q-value: 0.94

**DMI (Kg/d):**
- Control: 1.3
- 15% PB: 1.3
- 30% PB: 1.5
- SEM: 0.05
- P-value: 0.001
- Q-value: 0.39

**G:F ratio:**
- Control: 0.07
- 15% PB: 0.09
- 30% PB: 0.09
- SEM: 0.004
- P-value: 0.04
- Q-value: 0.51
Rumen fermentation parameters

<table>
<thead>
<tr>
<th>Item</th>
<th>Control</th>
<th>15% PB</th>
<th>30% PB</th>
<th>SEM</th>
<th>L</th>
<th>Q</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ammonia-N, mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D 0</td>
<td>10.3</td>
<td>14.1</td>
<td>12.4</td>
<td>1.70</td>
<td>0.45</td>
<td>0.25</td>
</tr>
<tr>
<td>D 40</td>
<td>12.5</td>
<td>11.5</td>
<td>11.4</td>
<td>1.36</td>
<td>0.61</td>
<td>0.03</td>
</tr>
<tr>
<td>D 83</td>
<td>11.6</td>
<td>9.8</td>
<td>8.2</td>
<td>1.70</td>
<td>&lt;0.01</td>
<td>0.99</td>
</tr>
<tr>
<td>Acetate:propionate</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D 0</td>
<td>2.45</td>
<td>3.29</td>
<td>3.69</td>
<td>0.36</td>
<td>0.05</td>
<td>0.66</td>
</tr>
<tr>
<td>D 40</td>
<td>4.45</td>
<td>3.88</td>
<td>2.63</td>
<td>0.29</td>
<td>&lt;0.01</td>
<td>0.40</td>
</tr>
<tr>
<td>D 83</td>
<td>3.45</td>
<td>2.73</td>
<td>2.85</td>
<td>0.10</td>
<td>0.01</td>
<td>0.21</td>
</tr>
</tbody>
</table>

Major mineral utilization

• Apparent retention of P, Mg, Mn, Zn, and Fe was greater linearly, but K, S, and Ca was lower linearly as PB increased in the diets.

Fatty acids composition (mg/g of tissue) of subcutaneous fat from Kiko crossbred male goat kids

<table>
<thead>
<tr>
<th>Item</th>
<th>Control</th>
<th>15% PB</th>
<th>30% PB</th>
<th>SEM</th>
<th>L</th>
<th>Q</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of animals</td>
<td>8</td>
<td>7</td>
<td>7</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SFA</td>
<td>694.8</td>
<td>835.1</td>
<td>1001.7</td>
<td>85.05</td>
<td>0.01</td>
<td>0.91</td>
</tr>
<tr>
<td>MFA</td>
<td>89.9</td>
<td>99.3</td>
<td>122.5</td>
<td>14.3</td>
<td>0.10</td>
<td>0.71</td>
</tr>
<tr>
<td>PUFA</td>
<td>45.6</td>
<td>53.3</td>
<td>76.3</td>
<td>7.47</td>
<td>0.008</td>
<td>0.43</td>
</tr>
<tr>
<td>PUFA:SFA</td>
<td>0.06</td>
<td>0.06</td>
<td>0.08</td>
<td>0.003</td>
<td>0.03</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Necropsy test in goat’s liver and kidney

Peanut skin

Objective

➢ To determine whether the phytochemical CT-containing peanut skin would have effects on animal performance and drug-resistant H. contortus control in meat goats.
Materials and Methods

**Diet & Fecal Egg Count**

- Animal performance, FAMACHA, fecal egg count (FEC) were recorded every two wk.
- Blood samples were collected twice, at the beginning and the end of the study.

**Statistical Analysis**

- Data were analyzed by the Proc GLM procedure of the SAS for completely random design. Animals were the experimental unit and were treated as random effects.

---

**Results**

**Table 1. Chemical composition of control, 25%, and 50% peanut skin (PS) diet offered to goats**

<table>
<thead>
<tr>
<th>Item</th>
<th>Treatment group (g/kg)</th>
<th>SEM</th>
<th>.05</th>
<th>.10</th>
</tr>
</thead>
<tbody>
<tr>
<td>DM</td>
<td>82.0</td>
<td>0.1</td>
<td>81.9</td>
<td>81.1</td>
</tr>
<tr>
<td>CP</td>
<td>26.0</td>
<td>0.1</td>
<td>25.6</td>
<td>25.4</td>
</tr>
<tr>
<td>ADL</td>
<td>82.9</td>
<td>0.1</td>
<td>82.7</td>
<td>82.4</td>
</tr>
<tr>
<td>Mh</td>
<td>0.00</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Th</td>
<td>0.00</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>TBH</td>
<td>7.00</td>
<td>0.1</td>
<td>8.0</td>
<td>8.0</td>
</tr>
</tbody>
</table>

**Table 2. The effect of peanut skin (PS) supplementation on daily dry matter intake in meat goats**

<table>
<thead>
<tr>
<th>treatment</th>
<th>0%</th>
<th>25%</th>
<th>50%</th>
</tr>
</thead>
<tbody>
<tr>
<td>DM</td>
<td>10.0</td>
<td>10.2</td>
<td>10.4</td>
</tr>
<tr>
<td>CP</td>
<td>26.0</td>
<td>25.6</td>
<td>25.4</td>
</tr>
<tr>
<td>ADL</td>
<td>82.9</td>
<td>82.7</td>
<td>82.4</td>
</tr>
<tr>
<td>Mh</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Th</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>TBH</td>
<td>7.0</td>
<td>8.0</td>
<td>8.0</td>
</tr>
</tbody>
</table>

**Table 3. The effect of peanut skin (PS) supplementation on gastrointestinal focal egg count (FEC) and FAMACHA score in meat goats**

<table>
<thead>
<tr>
<th>Item</th>
<th>Treatment group (g/kgDM)</th>
<th>SEM</th>
<th>Linear</th>
<th>Quadratic</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEC</td>
<td>10.0</td>
<td>1.1</td>
<td>0.15</td>
<td>0.45</td>
</tr>
<tr>
<td>250</td>
<td>15.0</td>
<td>2.2</td>
<td>0.21</td>
<td>0.68</td>
</tr>
<tr>
<td>500</td>
<td>20.0</td>
<td>3.3</td>
<td>0.23</td>
<td>0.19</td>
</tr>
<tr>
<td>FAMACHA</td>
<td>10.0</td>
<td>1.1</td>
<td>0.15</td>
<td>0.45</td>
</tr>
<tr>
<td>250</td>
<td>15.0</td>
<td>2.2</td>
<td>0.21</td>
<td>0.68</td>
</tr>
<tr>
<td>500</td>
<td>20.0</td>
<td>3.3</td>
<td>0.23</td>
<td>0.19</td>
</tr>
</tbody>
</table>

---

**Figure 3. The effect of peanut skin (PS) supplementation on daily dry matter intake in meat goats**

---

**Table 4. The effect of peanut skin (PS) supplementation on plasma biochemical parameters in meat goats**

<table>
<thead>
<tr>
<th>Item</th>
<th>Treatment group (g/kgDM)</th>
<th>SEM</th>
<th>Linear</th>
<th>Quadratic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein</td>
<td>10.0</td>
<td>1.1</td>
<td>0.15</td>
<td>0.45</td>
</tr>
<tr>
<td>250</td>
<td>15.0</td>
<td>2.2</td>
<td>0.21</td>
<td>0.68</td>
</tr>
<tr>
<td>500</td>
<td>20.0</td>
<td>3.3</td>
<td>0.23</td>
<td>0.19</td>
</tr>
<tr>
<td>Lipid</td>
<td>10.0</td>
<td>1.1</td>
<td>0.15</td>
<td>0.45</td>
</tr>
<tr>
<td>250</td>
<td>15.0</td>
<td>2.2</td>
<td>0.21</td>
<td>0.68</td>
</tr>
<tr>
<td>500</td>
<td>20.0</td>
<td>3.3</td>
<td>0.23</td>
<td>0.19</td>
</tr>
<tr>
<td>Glucose</td>
<td>10.0</td>
<td>1.1</td>
<td>0.15</td>
<td>0.45</td>
</tr>
<tr>
<td>250</td>
<td>15.0</td>
<td>2.2</td>
<td>0.21</td>
<td>0.68</td>
</tr>
<tr>
<td>500</td>
<td>20.0</td>
<td>3.3</td>
<td>0.23</td>
<td>0.19</td>
</tr>
</tbody>
</table>

---

**Figure 4. The effect of peanut skin (PS) supplementation on plasma biochemical parameters in meat goats**
Animals received condensed tannin-containing diets

- Reduced FEC and possible reduce worm burden.
- May increased/decreased blood metabolites
- break the life-cycle of parasite infection

The CT may react directly with parasite larvae during passage through the gut.

Therefore, it seems that CT may counteract parasites by one or more of the above mechanisms, and that the mechanism involved may differ between forage species and IP.

Thank You Very Much
Chapter 12. Nutrition, Feeding, and Immunity against parasites

N. Gurung, Ph.D.
Tuskegee University

Goat Industry Challenges

- Internal parasites (production disease)
- Fencing costs
- Predators
- Lack of knowledge

Nutrition and Feeding

- Goats are natural browsers and given the opportunity, they select over 60% of their daily diets from brush and woody and broadleaf plants (Luginbuhl, 2005).

- Brush and woody plants include multiliora rose, brambles, sumac, privets, honeysuckle, etc., and broadleaf plants such as pigweed, dock, horseweed, lambsquarter, etc.
### Diet Preference Differences (% of diet)

<table>
<thead>
<tr>
<th>Animals</th>
<th>Grass</th>
<th>Weeds</th>
<th>Browse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horse</td>
<td>90</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>Cattle</td>
<td>70</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>Sheep</td>
<td>60</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>Goats</td>
<td>20</td>
<td>20</td>
<td>60</td>
</tr>
</tbody>
</table>

Source: An Peischel, 1999

### Bipedal Stance

- Browsing plants

### Browsing Plants

- Ideal mix should be both browse and pasture unlike sheep
- A good mix is about two acres of browse for every one acre of open grassland
- Problem is growing browse back after grazing goats for more than two seasons

### Browsing/Parasites

- Browsing goats have less parasites problem due to higher grazing heights and due to tannin-containing browse species
- Goats grazing grass on high stocking rates are become susceptible to parasites

### Barber Pole Worm

- The Barber-pole worm has become resistant to most of the available dewormers:
  - Frequent and indiscriminate uses
  - Use of many under doses
- Goats graze to the ground level:
  - Ideally should not be grazed below 6 inches from the ground

### Nutrition and Feeding...

- Ideal mix should be both browse and pasture unlike sheep
- A good mix is about two acres of browse for every one acre of open grassland
- Problem is growing browse back after grazing goats for more than two seasons
### Nutritional Management: Integrated Approach

- Many approaches
- Nutritional management is one of the integrated approaches for parasite control
- Well-fed animals are able to defend themselves better

### What is immunity?

- Means that an animal is protected from catching a certain disease or infection
- **Immune System:**
  - Protects body from infectious agents and toxins
  - Enables the body to repair damaged cells

### Immunity...

- Active or passive
  - Active – animal is more or less permanently immune
  - Passive – animal is only temporarily immune

### Immunity...

- Animals are born with some immunity
- Colostrums is rich in antibodies:
  - Serve the new animal until its own immune system can take over

### Nutrition and Immune System...

- Animals prioritize immune system for nutrients over growth
- Nutrients not available for growth, thus, immune system depresses growth and feed efficiency
- Sick or infected animals reduce feed intake which further depresses immune system
Animals may be deficient at levels that affect immunity without displaying clinical signs of deficiency.

**Vicious Cycle**
- Poor nutrition
- Worms
- Anorexia

Nutrition and Immune System…
- When antigens (infectious agents) act on the lining of the stomach.
- Damage to host tissues caused by excessive activity of the immune system (immunopathology).

Host immunity
- Goats tend to be more susceptible to parasites than sheep, especially hair sheep.
- Periparturient females
  - Ewes and does suffer a temporary loss of immunity around the time of parturition.
  - The length and intensity of the periparturient egg rise varies by species, breed, individual, and season of kidding.
  - Management of periparturient egg rise
    - Deworm females during late gestation
    - Increase protein content of late gestation ration.
    - Alter dates of kidding and lambing
    - Keep herd in dry lot during PPP.
- Weanlings
  - Have no natural immunity to parasites.
  - Will develop immunity to parasites over time; by 4-9 months of age, but varies by species, breed, and individual.
  - Development of immunity requires continued exposure to parasites.

Source: Susan Schoenian, 2015

Young animals are relatively susceptible to infection and become more resistant with age.
- Immunity also wanes around the time of kidding/lambing.

Nutrition for Optimum Immune System?
Energy Sources

- Gold standard:
  - EFA rich (linoleic)
  - Antioxidants
  - 95% digestible
- By-product feeds such as soybean hulls, corn gluten feed, beet pulp, and brewers’ grains, etc.

Energy Sources

- Most important but it’s not labelled in a feed bag
- Flushing: 10% increase in drop rate

Protein

- Low protein; low concentrations of antibodies
- Body proteins are broken down to provide energy and amino acids for the immune system
- Low levels of antibodies weaken animals’ ability to fight off diseases and parasites

Effect of Protein Levels on Worm Burdens in Lambs

Minerals

- Magnesium and phosphorus
- Trace minerals such as copper, zinc, iron, manganese
Copper and Immunity
• Forage surveys conducted across the United States are marginally to severely deficient in copper.
• Also higher levels of antagonistic minerals (sulfur, molybdenum and iron) bind with copper making it unavailable for use by the goats.

Vitamins
• Vitamin A and D play important roles in regulating immunity:
  o Reduced resistance to all types of diseases including parasites
• Vitamin D is required to activate the killer cells of the immune system (e.g., T-cells)
• Vitamin E is the most important antioxidant

Dietary requirements set by the National Research Council (NRC)
• Optimal immunocompetence may occur at nutrient levels that are: higher than the NRC requirement (panel A); equal to the NRC requirement (panel B); or less than the NRC requirement (panel C).

Wholesale Costs for Supplying 100% of Mineral Needs of a 150 lb Goat for Various Minerals in 1 Year

<table>
<thead>
<tr>
<th>Minerals</th>
<th>Costs ($)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium</td>
<td>1.15</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>4.50</td>
</tr>
<tr>
<td>Salt</td>
<td>0.40</td>
</tr>
<tr>
<td>Magnesium</td>
<td>1.11</td>
</tr>
<tr>
<td>Potassium</td>
<td>1.50</td>
</tr>
<tr>
<td>Trace minerals</td>
<td>0.45</td>
</tr>
<tr>
<td>Other minerals</td>
<td>0.65</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>9.70</strong></td>
</tr>
</tbody>
</table>

Source: Steve Hart, 2005

Effect of copper oxide wire particles (COWP; n = 8/dose) on fecal egg counts (FEC; A) and blood packed cell volume (PCV; B) from d 0 through 21 after administration of COWP in weaned kids on pasture (Burke et al., 2007)
Vitamin E Levels in Milk and Plasma in Healthy and Mastic Cows

Milk

Plasma

Supplementation of Vit E (1500 IU) and Se (0.3ppm) increases plasma concentration of vitamin E and reduce incidence of mastitis (Smith, 1986)

Plasma concentration alpha tocopherol

Source: Aroshi et al., 1986

Effects of Supplemental Dietary Vitamin E on Morbidity and Performance of Transport-Stressed Calves

<table>
<thead>
<tr>
<th>Item</th>
<th>Control</th>
<th>Vitamin E*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily gain, kg</td>
<td>0.8</td>
<td>0.9</td>
</tr>
<tr>
<td>Dry matter intake, kg</td>
<td>7.5</td>
<td>7.5</td>
</tr>
<tr>
<td>Feed to gain ratio</td>
<td>12.4</td>
<td>9.0</td>
</tr>
<tr>
<td>Morbidity</td>
<td>55</td>
<td>48</td>
</tr>
</tbody>
</table>

*Vitamin E levels were 400 to 1,000 IU/head/day

Source: Secrist et al. (1997); Means of 5 trials

Role of Trace Minerals on Immune Systems

- Their roles goes beyond stimulating the activity of immune cells, it includes protecting the host from toxic substances formed by macrophages
- Macrophages use superoxide to destroy pathogens and other foreign substances

Role of Trace Minerals on Immune Systems...

- Superoxide, however, is also toxic to host cells and therefore, needs to be converted to compounds that are less toxic
- The crucial step in this scavenging of superoxide requires superoxide dismutase, an enzyme in which zinc, manganese, and copper are essential co-factors.
Practical Dietary Recommendations

<table>
<thead>
<tr>
<th>Items</th>
<th>% Protein</th>
<th>% TDN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Growing kids (0.3 lb/day)</td>
<td>12 - 13</td>
<td>58 - 63</td>
</tr>
<tr>
<td>Dry does and bucks</td>
<td>9 - 10</td>
<td>54 - 58</td>
</tr>
<tr>
<td>Lactating goats</td>
<td>12 - 13</td>
<td>62 - 68</td>
</tr>
</tbody>
</table>

Source: Pinkerton and Pinkerton, 2000

Two Sample Creep Feeds for Goat Kids

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Sample 1</th>
<th>Sample 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cracked corn</td>
<td>50%</td>
<td>50%</td>
</tr>
<tr>
<td>Soybean hulls</td>
<td>30%</td>
<td>----</td>
</tr>
<tr>
<td>Oats</td>
<td>----</td>
<td>30%</td>
</tr>
<tr>
<td>Soybean meal</td>
<td>15%</td>
<td>15%</td>
</tr>
<tr>
<td>Molasses</td>
<td>5%</td>
<td>5%</td>
</tr>
</tbody>
</table>

Source: Rankins, 2004

Two Sample Diets for Growing Young Kids

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Sample 1</th>
<th>Sample 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ground hay</td>
<td>25%</td>
<td>----</td>
</tr>
<tr>
<td>Cottonseed hulls</td>
<td>----</td>
<td>25%</td>
</tr>
<tr>
<td>Cracked corn</td>
<td>44%</td>
<td>41%</td>
</tr>
<tr>
<td>Soybean hulls</td>
<td>15%</td>
<td>15%</td>
</tr>
<tr>
<td>Soybean meal</td>
<td>10%</td>
<td>13%</td>
</tr>
<tr>
<td>Molasses</td>
<td>5%</td>
<td>5%</td>
</tr>
<tr>
<td>Trace mineral salt</td>
<td>0.5%</td>
<td>0.5%</td>
</tr>
<tr>
<td>Dicalcium phosphate</td>
<td>0.5%</td>
<td>0.5%</td>
</tr>
</tbody>
</table>

Source: Rankins, 2004

Summary and Conclusion

- Inadequately fed goats cause reduced goat performance:
  - Reduced feed intake
  - Reduced feed efficiency
  - Protein leakage, gut damage
  - Nutrients are diverted away from production

Summary and Conclusion...

- Adequately fed animals can tolerate increasing levels of parasites
- Interrelationship which exists between nutrition and immunity must be taken into consideration

Thank You!