Myotonic Goat Review

A publication of the Myotonic Goat Registry

Summer 2021

Volume 13 Issue 2

Notes from the Pasture

Summer is almost done for another year, can you believe it!? Our oldest child recently headed off to kindergarten. I will admit I was not ready to see my baby girl board that yellow bus! Our second oldest will head off the Pre-K in a couple weeks. We are anxiously awaiting our fall kidding to start and hope that all of you who have kids in the fall have a great kid crop!

This issue is jam packed! I apologize for the length of the newsletter but hope it is well worth it! Several MGR shows have taken place since our last newsletter and it was a welcomed sight to see shows and fairs taking place after the year we had last year! We will catch up on show results, take an in depth look at Internal Parasites and also wrap up our Diseases and Testing series. Last but not least, we will travel to Utah and meet Jake and Wyatt Strahan for our Youth Spotlight!

As always, if you have ideas and suggestions for future newsletters, send them my way!

Until next time, happy goating!
Drew DeRiemacker, editor
info@fieldcrestfarms.com

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From The MGR Office...

The MGR office is happy to announce so many new things for this fall. MGR has newly surfaced annual fundraisers for the National Show and the Fall Finale. Anna Garrett will be hosting it on behalf of MGR. This fundraiser will take place September 1st- September 7th and we look forward to so many participating in this event. The fundraiser will take place on the Facebook group Myotonic Goat Registry. If you have any items you would like to donate to increase donations to MGR, please contact Anna Garrett (Facebook messenger or by phone:2529084380). Payment will be taken by Venmo and PayPal. We urge everyone to create a Venmo or PayPal account so they can join in on the fun!

As we approach 2021 MGR Nationals, breeders will be able to look forward to shirts being made. Ellen Pittman is hosting the sale and these shirts will have the list of the past winners of the National Shows. The sale will be posted to MGR's Facebook group soon and we hope everyone gets the opportunity to wear a National shirt this year! We are so proud of how advanced the myotonic breed has developed!

During this pandemic, we have all experienced hard ships. As a way for the breeders to have something positive, we included these fun photo contests! This photo contest was "Best Fall Photo." We try to have our members participate to promote the breed and since a lot of shows have been canceled in the past, this gave us all something to look forward to each month! Also, get your cameras out and look forward to "flashiest buck" photo contest soon and the first five places will receive a ribbon!

At this year's Fall Finale, we are hosting it in Kentucky. MGR is so excited to see everyone in person and show goats alongside other breeders! We also urge to reserve your hotel room and breeders can use the discount code "MGR", it is on the Fall Finale show forms. The discount is through Holiday Inn. Please pay special attention to the required medication report, this will be required of all goats participating in Nationals (not the Fall Finale Shows). To preserve our breed & ensure a fair playing field, MGR will be testing (at random) National Champions & Reserve National Champions beginning this year. We will be testing for performance enhancing substances.

Details will be available in the medication report & MGR newsletter set to publish today. Any additional questions regarding "can I use this" should be directed to your veterinarian. Enhancing substances have always been a violation of MGR Official Sanction Rules. Today, we take the first steps to be encouraged that we will be presenting supreme goats in the industry that need no artificial substance to be magnificent. For questions arising, MGR will be paying the bill to have animals tested. The animals will have blood pulled by staff from a local veterinarian office. They will be moved to a designated safe area prior to having pictures made and once the sample is collected, that animal will be released for photos. Any random National champion or reserve animal selected for testing will have to test. If you refuse testing, you will be disqualified. Ideal lab testing results can take two to three weeks to get back and the MGR office will receive results and confirm the wins. You will be contacted by email from MGR if we receive positive results for performance enhancing drugs. The medication report will contain specifics on acceptable or unacceptable medications and substances. These results will remain confidential between MGR and the owner of the animal.

~Tara

Myotonic Goat Registry

Medication Report

For National Show Exhibition

Effective 9/1/2021

Breeder # _____ Date _____

Owner Name _____ Phone Number ( ) _____

Animal Name_____

Animal Registration_____ Animal DOB _____ Animal Identification_____

CHECK ONE OF THE TWO CHOICES BELOW:

A. _____ **I CERTIFY THE ABOVE ANIMAL TO BE FREE OF MEDICATION, WHICH MEANS:**

I have not administered to and have no knowledge that this entry has received any substance not approved by the Food and Drug Administration (FDA) and/or the U.S. Department of Agriculture (USDA) for food animals.

IF YOU CHECKED THIS BOX, SIGN PART C BELOW AND **DO NOT COMPLETE THE TREATMENT RECORD.**

***PLEASE BE ADVISED THAT HEALTH PAPERS AND THIS DOCUMENT MUST BE PRESENTED IN PAPER FORM AT CHECK IN FOR ALL Myotonic Goat Registry National Shows effective 9/1/2021. ***

B. _____ **THE ABOVE ANIMAL HAS BEEN MEDICATED.** COMPLETE THE TREATMENT RECORD BELOW LISTING DRUGS FOR WHICH THE WITHDRAWAL PERIOD HAS NOT ELAPSED. PROVIDE COPY OF SCRIPT OR TREATMENT PLAN.

AFTER COMPLETING THE TREATMENT RECORD, SIGN PART C BELOW.

***PLEASE BE ADVISED THAT HEALTH PAPERS AND THIS DOCUMENT MUST BE PRESENTED IN PAPER FORM AT CHECK IN FOR ALL Myotonic Goat Registry National Shows effective 9/1/2021. ***

TREATMENT RECORD

Treatment Date	Condition Being Treated	Medication (Name)	Amount (Dose)	Instructed Withdrawal Milk/Meat	Date Withdrawal Complete

*IF THIS IS AN EXTRA LABEL OR l DRUG (refer to MGR sanctioned rules attached), LIST THE LICENSED VETERINATIAN'S NAME AND ADDRESS WHO PRESCRIBED OR DIRECTED THE TREATMENT:

_____

VETERINARIAN NAME STREET, P O BOX CITY, STATE, ZIP PHONE

C. Exhibitor compliance with all applicable rules and regulations is essential to the integrity of the Myotonic Goat Registry and its shows, programs, and exhibitions. Violation of any applicable rule or regulation may result in exhibitor sanctions. Sanctions may include but not limited to disqualification, suspension or the opportunity to exhibit at any future Myotonic Goat Registry sanctioned shows.

I HEREBY CERTIFY THAT THE INFORMATION ABOVE HAS BEEN READ AND IS CORRECT.

_____

PRINT CLEARLY – Name of owner SIGNATURE OF OWNER

_____

PRINT CLEARLY – Parent/Guardian of Owner SIGNATURE OF PARENT/GAURDIAN OF OWNER

Excerpt from MGR Sanction Rules

Myotonic Goat Registry Official Sanctioned Rules; B, 3, E; e**. Exhibitor shall use no artificial or unnatural substances to hide defects, to add bulk to an animal or to change natural tail carriage.**

Acceptable Practices and Substances

No person shall conspire with another person or persons to intentionally violate, or knowingly contribute or cooperate with another person or persons either by affirmative action or inaction to violate any part of these Rules & Regulations.

i. Permitted Practices and Substances

a. Drenches as needed, and as directed on the label, for animal health and well-being.

b. Drugs and other substances labeled for use in goats to treat health issues in an exhibited goat. Any extra-label usage requires a prescription, or statement for use from the attending veterinarian.

c .Grooming products that are not restricted from use in meat producing animals, including: brushes, combs, clippers, hoof trimmers, shampoos, conditioners, oils, mousse, alcohol/liniment, sprays, powder, and artificial coloring that enhances the goat's natural color.

i. Conditionally Permitted Practices and Substances

a. Therapeutic medications given for the legitimate treatment of illness or injury are permitted if ALL of the following conditions are met:

i. A completed medication report on file with show management before exhibiting the animal, which must include:

(a) Diagnosis of illness/injury, reason for administration, and name of administering and/or prescribing veterinarian.

(b) Signature of veterinarian or person administering the medication. If prescribed by written instructions, a copy must be attached to the medication report.

(c) Identification of the medicine; the name, amount, strength and mode of administration.

(d) Date and time of administration.

(e) Identification of the animal: MGR registration number, permanent identification, age, sex.

ii. The animal must be withdrawn and kept out of competition for not less than 24 hours after the medication is administered.

iii. The medication report must be filed with show management within one hour of administration of the medication or one hour after show management is available, if administration occurs at a time other than during competition hours.

iv. The medication report must be signed by show management and the time of receipt recorded on the report.

Prohibited Practices and Substances

a. Injection or external or internal administration via any orifice of any substance (including drugs, chemicals, and any other forms of products) prohibited from use in meat producing animals by the Food and Drug Administration (FDA), the U.S. Department of Agriculture (USDA), and/or any Federal, State or Local Law.

b. Extra-label use of any drug or substance approved for use on meat producing animals, but

not approved for use in goats (even though commonly used in goats) while on the show premises.

c. Injection or external or internal administration via any orifice of any allowed substance in any way that is inconsistent with the dosage and the route prescribed by the manufacturer or prescribing veterinarian.

d. Administration of any quantity of any diuretic, growth stimulant, or performance enhancing drug.

e. Filling and/or tubing of an animal.

f. Presenting any animal whose natural conformation and structure have been surgically altered in any way, exception for:

i. Removal of horns; and/or

ii. Removal of testicles in the case of wethers.

g. Using any inhumane or unethical treatments, including striking animals, using electrical contrivance, or other similar methods.

h. Artificial coloring that alters the natural color of a goat.

i. Attaching any objects, including hair or hair substitutes, cloth, or fiber to the animal for the purpose of deception.

j. Injection or external or internal administration via any orifice of any substance, whether gas, solid, or liquid, not conducive to continued animal health or marketability.

k. Tissue or fluid manipulation, removal, surgical attachment or otherwise to change, conceal, enhance, or transform the true conformation or configuration of the animal.

l. Administering any substance that artificially induces lactation.

m. Any substance, regardless of how harmless or innocuous it might be, which might interfere with the detection or quantization of any substance prohibited by MGR.

South Mountain Myotonic Goat Show - Thurmont, MD

South Mountain Myotonic Goat Show A: Judge Wade Buntin
Pam Mongan-Taylor Memorial Myotonic Goat Show B: Judge Josh Lichlyter
Gateway to the Mountains Myotonic Goat Show C: Judge Ashley Hadley
***Indicates verified MGR point**

Show A: May 28, 2021

Junior Champion Doe

Angelo's Fainters Alessandra	Anita Angelo

Reserve Junior Champion Doe

La Chevre d'Or Umbreon	Cheyenne Van Echo

Senior Champion Doe

Back 40 Sioux	Anita Angelo

Reserve Senior Champion Doe

Goat Flower Farm Tiarella	Carol Ellis

Grand Champion Doe

*Back 40 Sioux	Anita Angelo

Reserve Grand Champion Doe

Angelo's Fainters Alessandra	Anita Angelo

Junior Champion Buck

One Goat Farm Talking Bout' My White Pants	Tracy & Chloe Tumminello

Reserve Junior Champion Buck

Muddy River Ranger	Heather Beebe

Senior Champion Buck

Domino Goats Fearless for Real	Tracy & Chloe Tumminello

Reserve Senior Champion Buck

Twin Creek Harvey	William Smith

Grand Champion Buck

*Domino Goats Fearless for Real	Tracy & Chloe Tumminello

Reserve Grand Champion Buck

One Goat Farm Talking Bout' My White Pants	Tracy & Chloe Tumminello

Junior Champion Platinum Wether

La Chevre d'Or Liam	Gretchen Van Echo

Reserve Junior Champion Platinum Wether

Pahl's Farm Basswood	Donald Westen III

Senior Champion Platinum Wether

Fallen and Can't Get Up Sulley	Beth Ellen Smith

Reserve Senior Champion Platinum Wether

Mystic Kingdom Myotonics Chip	Beth Ellen Smith

Grand Champion Platinum Wether

*La Chevre d'Or Liam	Gretchen Van Echo

Reserve Grand Champion Platinum Wether

Fallen and Can't Get Up Sulley	Beth Ellen Smith

South Mountain Myotonic Goat Show - Thurmont, MD

Show B: May 29, 2021

Junior Champion Doe

La Chevre d'Or Umbreon	Cheyenne Van Echo

Reserve Junior Champion Doe

Bureau Creek Moonlit	Anita Angelo

Senior Champion Doe

Back 40 Sioux	Anita Angelo

Reserve Senior Champion Doe

Flipping Goat Farm Amanda	Cheyenne Van Echo

Grand Champion Doe

*La Chevre d'Or Umbreon	Cheyenne Van Echo

Reserve Grand Champion Doe

Back 40 Sioux	Anita Angelo

Junior Champion Buck

Bureau Creek Forged in Fire	Amy Taylor

Reserve Junior Champion Buck

One Goat Farm Talking Bout' My White Pants	Tracy & Chloe Tumminello

Senior Champion Buck

Domino Goats Fearless for Real	Tracy & Chloe Tumminello

Reserve Senior Champion Buck

Goat Flower Farm Denver	Carol Ellis

Grand Champion Buck

*Bureau Creek Forged in Fire	Amy Taylor

Reserve Grand Champion Buck

One Goat Farm Talking Bout' My White Pants	Tracy & Chloe Tumminello

Junior Champion Platinum Wether

Pahl's Farm Basswood	Donald Westen III

Reserve Junior Champion Platinum Wether

Quinn Beck Farm Mario	Quinn Ellis

Senior Champion Platinum Wether

Fern Hill Sponge Bob	Cheyenne Van Echo

Reserve Senior Champion Platinum Wether

La Chevre d'Or Liam	Gretchen Van Echo

Grand Champion Platinum Wether

*Fern Hill Sponge Bob	Cheyenne Van Echo

Reserve Grand Champion Platinum Wether

La Chevre d'Or Liam	Gretchene Van Echo

South Mountain Myotonic Goat Show - Thurmont, MD

Show C: May 30, 2021

Junior Champion Doe

Heavenly Hill Farm Allie Cat	William Smith

Reserve Junior Champion Doe

Delbay Farms Maybelline	William Smith

Senior Champion Doe

Goat Flower Farm Beretta	Heather Beebe

Reserve Senior Champion Doe

Over the Moon Fire	Sierra Weatherly

Grand Champion Doe

*Goat Flower Farm Beretta	Heather Beebe

Reserve Grand Champion Doe

Over the Moon Fire	Sierra Weatherly

Junior Champion Buck

Goat Flower Farm Je Suis Prest	Heather Beebe

Reserve Junior Champion Buck

Bailey's NonCents Farm Hornady	Elizabeth Bailey

Senior Champion Buck

La Chevre d'Or Savage	Elizabeth Bailey

Reserve Senior Champion Buck

Twin Creek Diesel	Tom Smith

Grand Champion Buck

*La Chevre d'Or Savage	Elizabeth Bailey

Reserve Grand Champion Buck

Goat Flower Farm Je Suis Prest	Heather Beebe

Junior Champion Platinum Wether

Pahl's Farm Basswood	Donald Westen III

Reserve Junior Champion Platinum Wether

Senior Champion Platinum Wether

Fern Hill Sponge Bob	Cheyenne Van Echo

Reserve Senior Champion Platinum Wether

Twin Creek Groudon	Tristan Van Echo

Grand Champion Platinum Wether

*Fern Hill Sponge Bob	Cheyenne Van Echo

Reserve Grand Champion Platinum Wether

Twin Creek Groudon	Tristan Van Echo

South Mountain Myotonic Goat Show - Thurmont, MD

Show A: May 28, 2021

Champion Light Weight Wether

Griffin Hill Farm Heathen	Jessica Wilfong

Reserve Champion Light Weight Wether

Griffin Hill Farm Deadpool	Morgan Leigh Shaffer

Champion Heavy Weight Wether

One Goat Farm Chevon	Tracy, Ricco, Chloe & Lydia Tumminello

Grand Champion Market Wether

*One Goat Farm Chevon	Tracy & Chloe Tumminello

Reserve Grand Champion Market Wether

Griffin Hill Farm Heathen	Jessica Wilfong

Show B: May 29, 2021

Champion Light Weight Wether

Griffin Hill Farm Deadpool	Morgan Leigh Shaffer

Reserve Champion Light Weight Wether

Griffin Hill Farm Heathen	Jessica Wilfong

Champion Heavy Weight Wether

One Goat Farm Chevon	Tracy, Ricco, Chloe & Lydia Tumminello

Grand Champion Market Wether

*One Goat Farm Chevon	Tracy & Chloe Tumminello

Reserve Grand Champion Market Wether

Griffin Hill Farm Deadpool	Morgan Leigh Shaffer

Show C: May 30, 2021

Champion Light Weight Wether

Griffin Hill Farm Deadpool	Morgan Leigh Shaffer

Reserve Champion Light Weight Wether

Griffin Hill Farm Heathen	Jessica Wilfong

Grand Champion Market Wether

*Griffin Hill Farm Deadpool	Morgan Leigh Shaffer

Reserve Grand Champion Market Wether

Griffin Hill Farm Heathen	Jessica Wilfong

Internal Parasites

Before learning about internal parasites several years ago, we used to deworm our herd on a quarterly basis, regardless if they "needed" deworming or not. We also used to alternate between dewormers from one quarterly deworming to the next. By doing so, we were giving the internal parasites an opportunity to become "immune" of sorts to the effectiveness of the dewormer used. There are scientific studies out there that back this up. We still deworm all kids born on our farm twice after birth. Beyond that, we only deworm on an "as needed" basis. If needed, we collect fecal samples and conduct a fecal egg count here at home. We only use 1 type of dewormer unless we have a parasite load that requires us to use a different type.

In the last issue of the newsletter, we looked at the different types of external parasites that we may encounter. Now we will take a look at the different internal parasites that can effect individual goats in our herds and can even effect the entire herd if not controlled. Hopefully the information in this article will give you a nice overview of internal parasites, and how to treat/manage the parasite load in your herd, the different types of dewormers available and also how to complete a fecal egg count. One of the nice things about our myotonics is that they are not as susceptible to internal parasites as other goat breeds, so we may not deal with internal parasites as often as other breeders.

*source American Consortium for Small Ruminant Parasite Control

Meet The Enemy

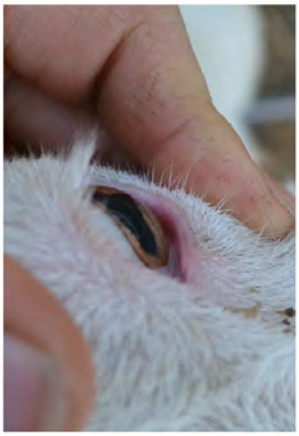
Worms are a normal part of the animal's ecosystem. A low level of worms is desirable to keep the animal's immune system active against worms. However, excessive worms causes disease. Excessive worms are caused by such things as a depressed immune system, consuming too many worm larva, filth and lack of sanitation, rainfall, close grazing etc. Worms function in the ecosystem to keep animals from overrunning the ecosystem when production conditions are good and they also prevent all animals from starving when there is a shortage of food. One cannot eradicate worms on your farm; you have to learn how to live with them and use management to control them to levels which do not harm animal production.

Barber Pole worm

The most common worm (especially in the Southeastern U.S.) that causes the majority of deaths as well as depressed animal performance is the Barber pole worm (scientific name Haemonchus contortus). The barber pole worm is a blood sucking-egg laying machine. It sucks about half to one drop of blood per day and produces 1,000-6,000 eggs per day. The barberpole worm can multiply rapidly under good conditions because of the large number of eggs that they lay. Since it sucks a half to one drops of blood per day, 1000 worms can suck two ounces of blood per day, two quarts in a month, which is why your goat will die from a heavy infection. The goat can make blood fast enough to replace that consumed by a low level of barberpole worm infection, but as the infection gets worse, the goat is unable to make components (red blood cells and blood protein) of blood fast enough to replace lost blood and the goat starts getting low on blood components. A low level of red blood cells (a component of blood) is called anemia (a thin layer of blood is pale because of the loss of red blood cells). The percent red blood cells in the blood can be measured in the laboratory to determine the extent of anemia.

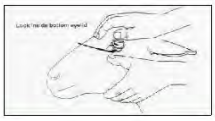
You can determine if an animal has anemia by looking at the color of the mucous membranes of the animal. Mucous membranes are areas of tissue where the capillaries are close to the surface of the skin and the color of the skin reflects the color of a thin layer of blood. When an animal becomes anemic these mucous membranes change from a healthy pink to a lighter pink and then if the anemia is severe, the mucous membranes will be white as a sheet of paper. When mucous membranes become white, the animal is critically low on blood and needs dewormed immediately.

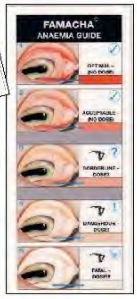
It must be remembered that other conditions that cause blood loss such as liver flukes or lice can also cause anemia. Mucous membranes that are easily observed are located on the inside of the lower eyelid (touching the eyeball), the gums (hard to see in animals with pigmentation in the mouth) and inside the vulva (often checked at goat dairies). A low level of blood protein also causes edema, due to a shortage of blood proteins to pull fluid back into the circulatory system. Edema is often seen as a pouch of fluid under the lower jaw. When an animal gets edema, he is severely low on blood components and needs dewormed promptly.



The FAMACHA© System

for assessing anemia and barber pole worm infection in small ruminants



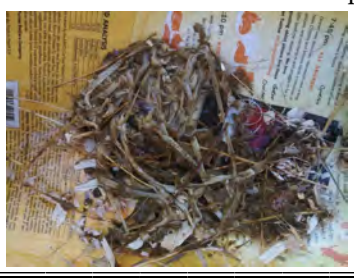


	Clinical Category	Color	PCV (hematocrit)	Deworming recommendation	
sheep	1	Red	≥ 28	No	goats
	2	Red-Pink	23-27	No	
	3	Pink	18-22	?	
	4	Pink-White	13-17	Yes	
	5	White	≤ 12	Yes	

The barber pole worm is a tropical worm; he likes a warm climate and therefore, predominates in the South, although he can cause major problems in northern areas, but for a shorter period of time. These worms require rain to infect small ruminants and therefore are much less of a problem in the West or any location where there is less than 25" of rain per year. The barber pole worm also has a short generation interval, being able to complete a generation as quick as 4 weeks which enables him to develop dewormer resistance quicker than for most other worms.

The barber pole worm is relatively large, and can easily be seen with the naked eye. He is about the diameter of paper clip wire and about an inch long. The barberpole worm pierces the stomach lining and establishes connections to small blood vessels to suck blood. If you open up the true stomach (abomasum) of a goat that has died from worms, you will see some floating free, but most are attached to the stomach and they may look like hair growing on the inside of the stomach. The barberpole worm will lose its coloration as it is exposed to air.

The average lifespan of a worm in the stomach is 6 months, but they can live longer than a year. The animal's immune system is constantly fighting against the worms and may suppress egg production of the worm or cause it to die prematurely. The immune system on some occasions may have a hyperimmune response and eliminate most of the worms in the animal. There are barber pole worms in sheep and cattle. But the strain in cattle will not infect sheep or goats (and vice versa) except under unusual circumstances.



(R)Tapeworms from a heavily infected Boer goat

Bankrupt worm & Brown stomach worm

There are two temperate species gastrointestinal nematodes which are important in goats and sheep. These are the Bankrupt worm (scientific name Trichostrongylus colubriformis) and Brown stomach worm (scientific name Telodorsagia circumcincta formerly known as Ostertagia).

Although these worms do not kill as many goats as the barberpole worm, under some circumstances they can cause important production losses as well as death of the goat. Many of the management practices which suppress the barberpole worm will also suppress these worms.

Since these worms are best adapted to temperate conditions, they are more of a problem in the fall and winter as compared to the barber pole worm which dominates in the summer. The main symptoms of a bankrupt worm or brown stomach worm infection is diarrhea , a slow growing animal, a rough haircoat and an unthrifty animal.

Tapeworm

The tapeworm is another worm that causes goat problems. It seldom kills goats, but causes poor performance, especially among young animals and may give them a pot-bellied appearance. Occasionally diarrhea is a symptom of tapeworms. Tapeworm segments can be readily observed in feces as they look like grains of rice making this parasite easy to diagnose. The general tapeworms that infect goats will infect sheep and occasionally cattle. Tapeworms are located in the small intestine and can grow to be several feet long. Tapeworms absorb nutrients from the digestive tract and therefore decrease nutrition available for the goat. The immune system of mature animals usually keeps the tapeworm suppressed.

The tapeworm eggs are consumed by a grass mite (like a chigger) and the egg develops to an infective stage in the body cavity of the mite over a 1 to 4 month period. The goat then eats grass on which the mite is crawling and becomes infected. It takes 40 days from the time a goat consumes infected grass mites until the tape worm segments appear in feces. Pasture areas that are infected usually remain infected for some time although the mites may be killed by winter weather.

Tapeworms are more of a problem under intensive production systems. If young animals get infected in a pasture, the general recommendation for cleaning the pasture up is to not graze a pasture for a year (make hay or graze with another species of animal). Tapeworms are not killed by all dewormers, but is controlled by the Benzamidole group of dewormers. Valbazen is one of the more popular dewormers used for tapeworm control.

Coccidiosis

Coccidiosis is a common goat parasite that appears when animals are stressed or sanitation is lacking. It mainly causes diarrhea, but unlike cattle, blood is seldom seen in the feces. Coccidia are normally present at low numbers in the digestive tract of the animal, but the infection level is low and the immune system is able to prevent them from causing disease. Coccidiosis is a disease of stress and filth. The main route of infection is the consumption of feces due to uncleanliness. The animal is usually stressed, depressing the immune system.

Coccidiosis is most commonly seen in just weaned kids due to stress, lack of a mature immune system and fecal contamination. Stressing animals by shipping is also a major cause coccidiosis. Animals often consume the infective stage coccidia from feces, such as fecal contamination in the feed trough or water trough.

Moisture whether by rain or humidity increases the time that infective coccidia live. Therefore, keeping the goat's environment clean and dry will help prevent coccidiosis.

During times of stress, a medicated feed containing Rumensin or Deccox can be fed which is quite effective at preventing coccidiosis. Occasionally coccidiosis will still occur despite feeding medicated feed, but fewer animals will be affected. Animals should be treated when diarrhea first starts if coccidiosis is suspected (history of stress) because delaying treatment can result in scarring of the intestine and an poor-doing animal for life. Coccidia are not observed when feces are examined under a microscope in early stages of disease, but they are very numerous later on.

Coccidiosis may be treated with Corid (Amprolium) or Albon (Sulfadimethoxine). If Corid is used at too high a dose, or for too long a time, animals may develop a thiamine deficiency called polioencephalomalacia (animals behave like they are drunk) which can be readily treated with thiamine and removal of the Corid treatment.

Meningeal worm

The meningeal worm or deer brain worm causes partial paralysis in goats, sheep and llamas that are exposed to the parasite by deer. The parasite occurs in deer and does not cause clinical symptoms as it does in goats. The larvae are passed in deer feces and are ingested by a variety of snails and slugs where they develop into infective larvae over a 3-4 week period. The snails or slugs are consumed by grazing goats. Inside the goat, the larvae penetrate the intestine and migrate to the spinal cord through the abdominal cavity over a 10 day period. The larva gets lost in migrating from the spinal cord to the brain because the goat anatomy is different from the deer. They end up destroying brain tissue causing differing degrees of paralysis.

Symptoms of the brain worm include paralysis of one or more limbs, excessive tail twitching, circling, abnormal head position, blindness, inability to get up, toe dragging, being in a dog-sitting position or difficulty or exaggerated movement of limbs when walking. The disease usually occurs in the fall and winter. There is no treatment for the brain worm that is very effective. Sometimes it is treated with high doses of various dewormers (fenbendazole and ivomec) and steriods, but treatment is often not effective.

Liver flukes

Liver flukes may be caused by the common liver fluke and less commonly by the large American liver fluke (also called the deer fluke). The flukes invade the liver and cause internal bleeding. A goat with high numbers of flukes will have an acute infection, where the animal stops eating, has pale mucous membranes, gradually does not get up and often dies within days. With fewer numbers of flukes, the symptoms may be milder and is called a chronic infection. The animal will have a poor appetite, lose weight for longer than a month, poor body condition, rough hair coat, rapid heartbeat, pale mucous membranes and sometimes edema, especially bottle jaw.

Liver flukes can infect wildlife, cattle sheep and goats and even man. The fluke lays eggs in the bile duct of the animal it infects and the eggs end up in the feces. The infective larva develop inside the egg over a 2-3 week period which then infect snails. These are the common pond snails which are in or around water and may range form 1/4" to nearly 3" in length. The larvae further develops in the snail over 5-7 days and then becomes a true infective larvae which leaves the snail and attaches to grass where the goats may consume it. Once consumed, the fluke further develops and penetrates the intestine on its way through the abdominal cavity to the liver. Once in the liver, it starts consuming the liver. Prevention includes fencing off ponds or marshy areas in the pasture.

Control of the liver fluke is dependent on the stage of the larvae, which depends on the time of the year. Chlorsulon and valbazen® is effective late in the year when flukes are mature. Chlorsulon is the only product that is effective against immature flukes, in early stages of infection as well as mature flukes. Consult local veterinary expertise for the time of the year to treat for flukes.

Lungworm

Lungworm infections results in respiratory distress such as painful breathing, chronic coughing, unthriftiness and death. There are several kinds of lungworms that live in the lungs of animals. Infection usually happens during the cooler months of the year. One kind of lung worm has a direct life cycle, the larvae are coughed up, go out in the feces, develop to infective worms in one to two weeks. The infective stage is killed by hard freezes or hot dry summer.

These larvae can live a long time in a cool damp environment. These larva develop into adults a month after being consumed. Several other kinds of lung worms have an indirect life cycle, that is they spend part of their lives developing in many species snails and slugs. Fortunately, these worms are easily controlled with the drug Fenbendazole (Panacur® or Safeguard®). Other dewormers such as levamisole and Ivomec are effective on some species of lung worms, but ineffective on other species.

Management

Most people think a parasite control program consists of (1) how frequently do we deworm, (2) which dewormer to use and (3) how much to use. This strategy has led us to high levels of dewormer resistance in the worm population, resulting in frustration, and animal deaths. We know that it is possible to have a parasite management plan that requires reduced or even no use of dewormer drugs, which is important to anyone who wants to stay in the goat or sheep business, but especially so to those raising an organic, chemical free or natural product. It is also important if a high level of dewormer resistance exists on a particular farm.

An integrated parasite management program consists of several components. The first is to identify the parasite that are causing the problem, which in the Southeastern US will be the Barberpole worm (Haemonchus contortus). In other drier and cooler areas, the Barberpole worm may be a minor or less severe problem. In cooler climates and times of the year, the Bankrupt worm (Trichostrongylus colubriformis) and the Brown stomach worm (Teledorsagia circumcincta) will be more important. Clinical symptoms of the Barberpole worm is anemia since he sucks blood which can be monitored by FAMACHA© . Clinical symptoms of the other worm species are usually diarrhea and fecal egg counts are the only available tool to monitor these other species.

The second step of integrated parasite control is to understand the biology of the parasite. Factors such as temperature and moisture are important since they are required for the eggs to develop to infective larvae and therefore determine how many infective larvae are available for your animal to pick up.

The third step is to develop a set of management practices to suppress the kind(s) of parasite that are applicable to your production system. This includes such practices as rotation grazing, making hay, tillage, not grazing close to the ground, managing for lower stocking rates, grazing browse and selecting for resistant animals. Making hay may be a viable management practice to reduce the level of infective larvae during the spring when grass growth is often excessive. If making hay is not possible, it may be possible to graze cattle or horses on the pastures following goats.

You may be able to incorporate sericea lespedeza into your pasture program since it has been shown to suppress worms.

The fourth part of integrated parasite management is monitoring the degree of infection and applying control (deworming) only when the level of infection of an animal depresses production. This may be done by monitoring fecal egg counts or by evaluating animals at regular intervals with the FAMACHA© chart. The latter is more convenient, but is only applicable when the Barberpole worm is the target parasite.

Fecal egg counts (FEC) can be used to monitor the level of worm infection in a herd. If the herd is small, all animals may be sampled whereas in a larger herd, a portion (10-20%) of animals are sampled. The same animals should be sampled at each time. When temperate species worms predominate (cooler climates and at cooler times of the year) fecal egg counts in conjunction with body condition and fecal consistency (temperate worms cause diarrhea in varying degrees) will need to be used.

One should deworm animals only when they need to be dewormed, not because it is that time of the year or because you are working the animals anyway or just to be sure there are no worm problems. The exception to this would be strategic deworming around kidding time to get arrested worms and slow down the rate of infection for lactating animals since they are more susceptible to worms. In sheep breeds that have significant resistance to worms, this may not be necessary. When temperate species of worms predominate (cooler climates and at cooler times of the year) fecal egg counts are very important since the FAMACHA procedure will not diagnose those worms.

Reducing the use of dewormers will reduce the rate of development of dewormer resistance in the worm population on your farm. Each time a goat or sheep is dewormed, it should be recorded. A small portion of your animals (20-30%) will carry a major portion of the worms (70-80%), presumably because their immune system is genetically weak for resisting worms. These animals are producing most of the eggs and larvae for infecting the rest of the herd. If we identify and cull these animals, we will substantially reduce our worm problems.

The next step is evaluation of how your parasite control management program is working. If very many animals need dewormed in any year, you should determine why. Once the reasons are identified, modify your parasite control program accordingly. As your parasite management program gets better and your genetic base of the flock or herd becomes more resistant to worms, less deworming will be necessary. However, continue to be cautious, because weather conditions change within a year and from year to year, and can increase parasite challenge. The introduction of new animals can create an additional challenge. Management changes related to pasture management, stocking rate, plane of nutrition may increase worm problems, even resulting in the death of animal(s) if we become complacent.

Five steps of Integrated Parasite Control

1)Identify worm(s) causing animal production problems (morbidity, mortality and reduced production). In the Southeast US, it will be the Barberpole worm.

2) Learn as much as you can about the biology of the worm causing problems so that you can utilize management practices which suppress parasite reproduction and development.. Evaluate the impact of your standard management practices on worms and revise as necessary.

3) Plan what management practices applicable to your operation. Stick to your plan unless it is obviously not working.

4) Evaluate the worm status of animals. Use either fecal egg counts or the FAMACHA system. Deworm only animals those that need to be dewormed.

5) Re-evaluate your worm problems and determine which management needs to be changed to control worms. Revise your parasite control plan for next year.

FAMACHA

The easiest way to monitor the need for deworming is to use the FAMACHA© chart if the Barberpole worm is the problem species.

The FAMACHA© chart was developed in South Africa in response to dewormer resistance that was causing major problems in sheep production systems. The name of the chart is an acronym from the name of a famous South African parasitologist Dr. Fafa Malan with chart added to
get FAMACHA. Although originally developed for use in sheep and it was successfully validated for use in goats in the USA through research support from Southern SARE. The validation exercise did include sheep as well as goats and involved several of the institutions that are currently a part of SCSRPC. This parasite management tool consists of a color chart for comparison of eye mucous membrane color and rules for proper use of the chart. Figure 1 shows a picture of the FAMACHA chart (actual chart is in color).

FAMACHA is an important tool in an integrated parasite management program. It identifies animals that have a high enough level of the Barberpole worm infection to reduce animal productivity. Only those individuals need to be dewormed. Since the Barberpole worm sucks blood, the of resultant degree of anemia. will cause lost production or even death of the animal. Anemia is reflected in the color of the mucous membranes i.e. a healthy reddish-pink color reflecting no anemia, whereas pale mucous membranes reflect a degree of anemia.

By monitoring the degree of anemia,(using eye mucous membrane color as an indicator) we can identify animals that need to be dewormed to prevent a loss in animal production and to prevent death. Usually only a portion of the animals in a herd need dewormed. The remainder of animals are not dewormed which reduces the development of dewormer resistance. This also reduces dewormer expense.

Mucous membranes that are readily observed are located on the inside of the eyelid, the gums (difficult to gauge anemia in animals with pigmented gums) and inside the vulva (often checked by dairy goat people when animals are being milked on a milk stand).

The FAMACHA© system was developed based on the eye mucous membrane, which is on the inside of the lower eyelid where it touches the eyeball. It is convenient to see the membrane by holding the animal=s head, slightly pressing down on the top of the eyeball (causes third eyelid to stay out of way), and pulling down on the skin immediately below the eyeball. The lower eyelid will roll out and can be readily compared to the FAMACHA© chart.

By using a series of color chips to match mucous membrane color, we can determine the degree of anemia and consequently, whether an animal needs to be dewormed. Since the Bankrupt worm and Brown stomach worm do not suck blood, FAMACHA© will not bean effective indicator for controlling these species of worms. These worms are more prevalent during cooler times of the year and need to be monitored with fecal egg counts or monitoring animals for diarrhea. Your local veterinarian or animal extension specialist should be able to help you identify times of the year when these worm species are more prevalent.

The FAMACHA© chart was originally brought into the US by the Southern Consortium for Small Ruminant Parasite Control. The agreement was that the chart could only be sold to Extension Educators, agricultural teachers and producers who had completed hands-on training following the specified curriculum (Some exceptions made for veterinarians and Extension Specialists who already had the prerequisite training). Training sessions are posted to the website (www.acsrpc.org) as well as further information about the FAMACHA© chart. Most states have qualified trainers which can be identified through your state sheep or goat extension specialist. Some trainers are listed on the ACSRPC web site.

The FAMACHA© chart is small enough to be fastened to the back of the hand for ready comparison when the lower eyelid is pulled down. The chart is calibrated for observing the eye of the animal in direct sunlight (the type of light may affect appearance of color). Animals should be observed in direct sunlight and matched to the chart. Color memory is not as good as a person thinks. Therefore, the chart should always be used for matching colors. If the animal's eye color is in between two chips, score as the lighter chip (higher number).

When scoring eyes, one should remember that some environmental factors can affect eye color and make the eye appear redder and make the animal not appear anemic. Factors include hot and/or dusty conditions which can irritate the eyes, infectious eye diseases (pink eye) and fever. In addition, remember that there can be other causes of anemia besides the Barberpole worm and deworming will not fix those anemias. Other causes includes liver flukes (most likely a problem in the Gulf Coast and Northwestern States), sucking lice, nutritional deficiency, bacterial and viral infections.

The frequency of checking eyes will vary with how suitable environmental conditions are for the Barberpole worm and age and class of the animal. Young animals need to be checked every two weeks because they are more susceptible to worms. In the spring, such as around kidding time, one may only check every 3-4 weeks, but when the weather warms up and you have rain, you may need to check as frequently as weekly.

Many producers will only need to check every two weeks except when it is warm and rainy when they need to go to weekly eye examination. With large herds of goats, a random sample may be checked (dont forget that animals that are anemic are often at the end of the line because they move slower due to anemia) and if 80% are 1 and 2's and there are no 4's or 5's then the herd is assumed OK. If there are 4's and 5's or more than 10% of the herd is a 3, then the whole herd should be examined.

Sheep or goats that score a 4 or 5 (pale) need to be dewormed and the rest turned back to the pasture.

However, when over 10% of the flock are dewormed, the 3's should also be dewormed because pasture contamination is building and the 3's will need dewormed shortly. The flock should be rotated to a new pasture since present pasture has become highly contaminated with infectious larvae. In addition, pregnant animals, lactating animals and animals under a year of age should be dewormed when they are 3's since their immune system is not fully functional. Animals with bottle jaw (swelling under the chin caused by edema) should be dewormed and animals that lag behind the rest of the herd or those that look wormy should be dewormed.

It is important to know that the dewormer that you are using works. The best way to determine this is to take a fecal sample before deworming on several animals then deworm those animals. Take another fecal sample 7-14 days later on the same animals for a fecal egg count. There should be less than 15% of the eggs in the second fecal count as compared to the first count. If there are more than 15% of the eggs in the second fecal egg count as compared to the first one, the dewormer is not effective in your herd or flock. The dewormer should be changed and be
sure to verify that the new dewormer is working for your sheep/goats.

Fecal Egg counts

* source:Improving Small Ruminant Parasite Control in New England

USDA Sustainable Agriculture Research and Education Program (LNE10-300)

The use of fecal egg counts is a necessary tool of a parasite control program. It is the best and quickest way to determine if your dewormer is/isn't working. It is also the only way to really tell the level of infection of the Brown stomach worm or the Bankrupt worm since these worms do not cause anemia (they do cause diarrhea though) which is monitored through the FAMACHA system. You can learn to do your own fecal egg counts rather easily.

The most common and efficient way to obtain fecal egg counts for sheep, goats, young cattle and horses is to use the Modified McMaster Test. This is a flotation test that separates parasite eggs from debris based on density; the eggs float to the surface of the counting chamber. This test uses a special microscope slide with a grid, which makes counting easier (Figure 1). Manure and flotation fluid is measured and mixed and only a small portion of the total mixture is counted. A calculation is performed to determine the number of eggs/ gram in the manure. This technique can be used to count strongylid (also called strongyle or trichostrongyle) eggs, including those of the barber pole worm (H. contortus).

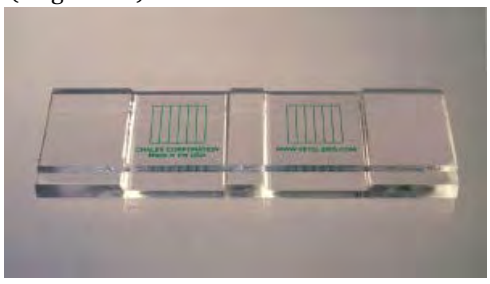
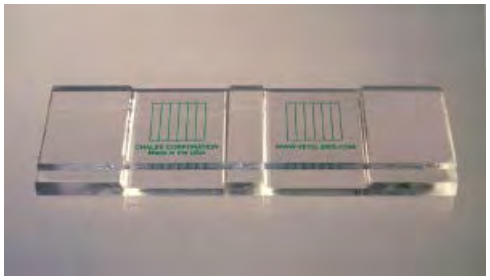


Figure 1. McMaster microscope slide.

Fecal Egg Counting Supply List:

- Scale to weigh fecal sample. Scale must weigh in 0.1 gram increments; a digital kitchen scale could be used.
- Two paper or plastic cups, at least 5 ounces
- Fecal flotation solution (can be Fecasol®, a commercially available solution, sugar solution, or a saturated solution of pickling salt (NaCl) or Epsom salts ($MgSO_4$) - see procedure notes on how to make this solution.)
- Dispenser bottle, measuring cup or large syringe for measuring flotation solution
- Tongue depressor / craft stick or spoon for mixing
- Straining technique – this can be a tea strainer; unfolded gauze 4" X 4" pads or squares; or cheese cloth cut into squares (6" squares preferred)
- 2-chamber McMaster slides. See procedure notes for more information including suppliers.
- 1 ml syringe, or eye dropper, or transfer pipette for filling slide
- Compound microscope with internal light source, moveable stage and 4X and 10X objective lenses. A binocular microscope is more comfortable than a monocular scope, but not essential.
- Timer



Fecal Collection Supply List:

- Exam gloves (powder free is best)
- Labels (1" x 3") to identify sample
- Permanent marker
- KY Jelly / lubricant
- Container with cooler/ice packs
- Refrigerator

Collecting a fecal sample:

1. Put on a clean glove. Apply a nickel size amount of water or water-based lubricant to index and middle fingers.
2. Insert index and middle fingers into the rectum of the animal, one finger at a time. No need to go very deep. Spread fingers to allow air into the rectum. The air duplicates fullness in the rectum and a wave of muscular movement will often move feces out into your hand.
3. Remove ~4 grams of fecal matter. A good sized adult pellet is about 1 gram.
4. Peel the glove off your hand keeping the fecal sample encased within it.
5. Squeeze as much air as possible out of the glove. Twist the wrist portion of the glove and fasten with a label (farm and animal ID) making sure the label sticks to itself, as it won't stick to the glove. You can also twist and tie off the glove and label the glove itself with an indelible marker.

Store the sample in the refrigerator until it can be analyzed (the sooner the better, but samples can be stored in the refrigerator for a week). If you are collecting many samples at one time, have a cooler with ice on hand to keep the samples cool until you can get them into a refrigerator.

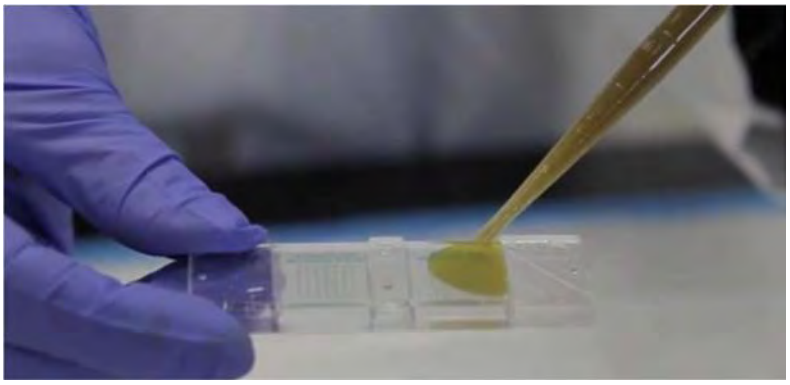
Don't use this collection method to sample very young animals. If you can't insert your fingers don't force them. Another option is to collect a sample immediately after it has been naturally deposited by t he animal. Rectal fecal sample collection is most successful when t he animals have been resting for a while, so if you need to pen them up to do the collection, let them rest there for a couple of hours before collecting the samples if possible.

Performing fecal egg count test:

1. Label two cups with animal ID as well as farm ID (if needed).
2. Tare one labeled cup on scale.
3. If manure is pelleted, crush the pellets in the glove and knead the manure in glove to mix. Cut off fingertip of glove containing feces to access fecal pellets, making sure to leave label intact.

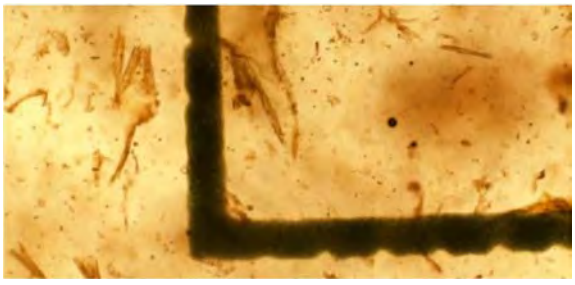
Step 3. Kneading manure in glove to mix fecal sample.

4. Measure two grams of fecal pellets into cup on scale.
5. Dispense 28 ml flotation solution into the cup, mix and let soak for approximately 5 minutes. *See following notes on flotation solution for how to make up your own saturated salt solution.
6. Once you are confident in the procedure you can weigh out multiple samples, add flotation solution and mix until 6-10 samples are set up.
7. Return to the first sample and mix again. Place tea or fabric strainer on top of the second cup (don't stretch fabric tight across the cup). Pour the mixture of feces and flotation solution through, pressing fluid through with the tongue depressor.
8. Immediately, fill both chambers of the McMaster slide using a transfer pipette, eye dropper, or syringe. If large bubbles are present, empty the slide and refill. Even if a large bubble is not actually under the grid, the slide should be refilled. Fill the entire chamber, not just the area under the grid.



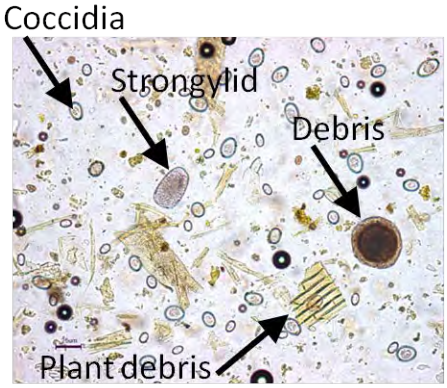
Step 8. Filling the first chamber of the McMaster slide using a transfer pipette.

9. Set slide aside for at least 5 minutes to allow parasite eggs to float to the surface. Read slides within about an hour of filling the slide. If slides are left too long, fluid evaporates and salt crystals form.
10. Place McMaster slide onto the microscope stage.
11. Bring the grid lines on the McMaster slide into focus using the low power (4X) objective and the coarse adjust knob. Turn to the 10X objective and refocus grid lines using the fine adjust knob. You can also focus on the air bubbles.



Step 11. Bringing the grid lines on the McMaster slide into focus.

12. Count all eggs inside of the grid areas using the 10X objective (include eggs on the grid line if greater than ½ of egg inside grid) in both chambers.



13. Always start at the same point on the McMaster slide (for example, top left or bottom right). That way, you won't lose track of whether you have counted only one or both chambers.
14. Count only strongylid eggs (oval shaped, ~80-90 microns long). Quantify Nematodirus eggs separately as they can be clearly distinguished. Other parasites present should be recorded and may be counted if desired, but numbers are often difficult to interpret.
15. Count both chambers.

Total egg count: (chamber 1 + chamber 2) * 50 = eggs per gram (EPG)

This multiplication factor of 50 is specific to the ratio of feces (2 grams) to flotation solution (28 ml) described in this procedure. Each egg observed represents 50 eggs/gram therefore, this procedure will not detect fewer than 50 eggs/gram, which is equivalent to seeing one strongylid egg on the McMaster slide.

Be consistent:
Many laboratories perform this test and you may see slight variations in the procedure described. The important thing is to always perform the test the same way each time—consistency is critical in order to monitor your animals over time or test the efficacy of drug treatment.

Flotation solution:
The following commercial solutions are commonly used by labs and can be obtained through your veterinarian:
Fecasol® - Vetoquinol, www.vetoquinolusa.com; Phone: 800-267-5707
Feca Med - Vedco Inc., Saint Joseph, MO, www.vedco.com; Phone: 816-238-8840
You can make up your own saturated salt solution using regular salt (sodium chloride) or Epsom salts (Magnesium Sulfate). A sugar solution is also available, but it is very viscous and sticky and results in difficult clean-up.
The approximate amounts of salt and water needed are provided on page 6. Add more salt as needed to fully saturate the solution. Add and mix the salt to lukewarm tap water until some of the salt no longer dissolves (the solution is saturated). Let it sit overnight. The amount of salt it takes to saturate the solution is affected by temperature, so the final test is to be sure you always see some un-dissolved salt at the bottom of your container. Pickling salt works better than table salt for making this solution because table salt contains anti-caking agent that doesn't dissolve and may mislead you into thinking that the mixture is saturated.

Sodium chloride (pickling salt):
Approximately 180 grams per 500 mls of water.
¾ of a cup of salt to 1 pint (16 ounces) of water – this would do about 16 fecal samples
Magnesium sulfate (Epsom salts):
Approximately 125 grams per 500 mls of water.
½ cup of salt to 1 pint (16 ounces) of water – this would do about 16 fecal samples

McMaster Slide:
The following are two U.S. Suppliers of this slide:

Chalex Corporation, 5004-228th Ave SE, Issaquah, WA, 98029.
Phone: 425-391-1169; www.vetslides.com

FEC Source, P.O. Box 601, Banks, OR 97106
Phone: 844-838-7543; www.fecsource.com

Parasite Egg Identification: The following two pages show the microscopic appearance of stronylid eggs, as well as other types of parasites, air bubbles and other material (such as plant material) commonly contained in sheep and goat fecal samples.

Parasite Egg Identification: Common Parasites in Small Ruminant Fecal Samples

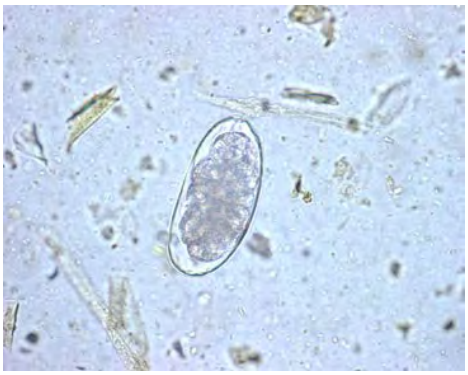


Figure 1. Strongylid egg

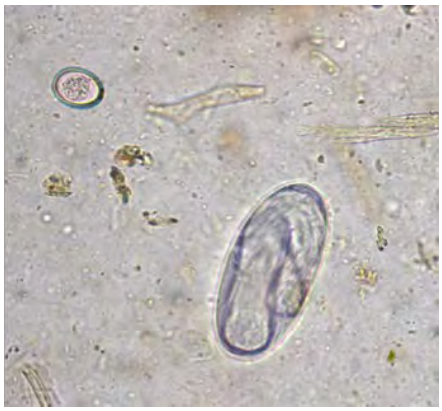


Figure 2. Larvated strongylid egg. These may be seen in old fecal samples

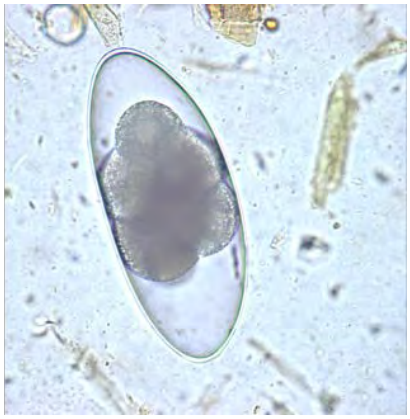


Figure 3. *Nematodirus* egg

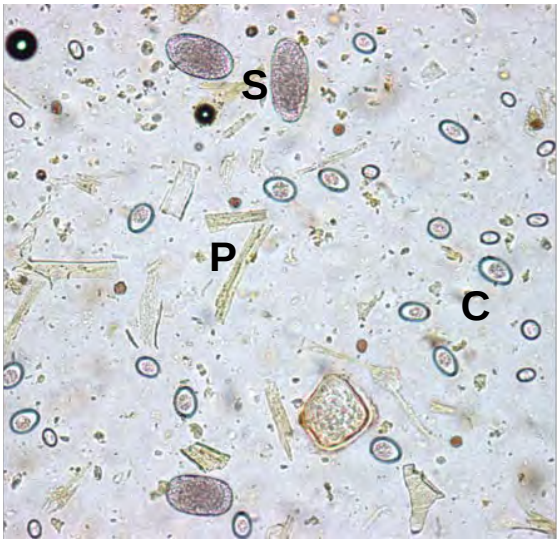


Figure 4. Strongylid egg (S), coccidia oocysts (C) and plant debris (P). Note size differences between eggs and oocysts.

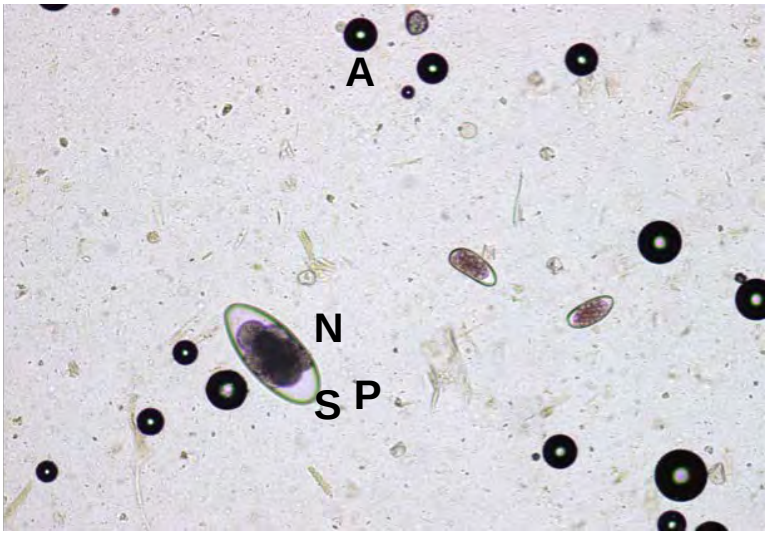


Figure 5. *Nematodirus* (N) and strongylid (S) eggs. Note size difference between eggs. Also note the presence of air bubbles (A).

Figure 6. Coccidia oocysts (C) and air bubble (A). Small ruminants are infected with several different species of coccidia that vary in size.

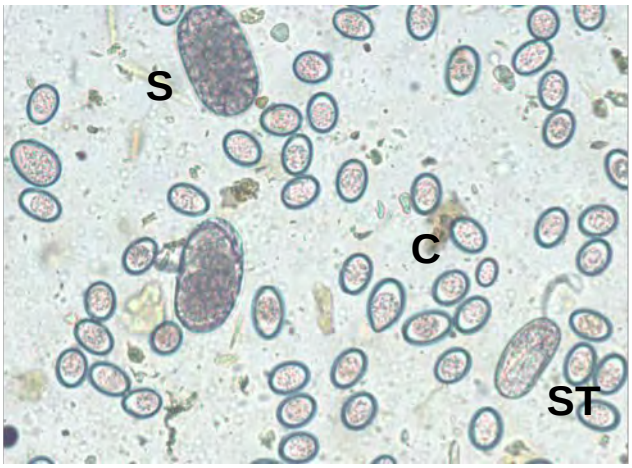


Figure 7. Strongylid (S) eggs and *Strongyloides* (ST) egg and coccidia (C) oocysts. Note size difference between eggs. *Strongyloides* eggs are larvated in fresh feces, strongylid eggs are not.

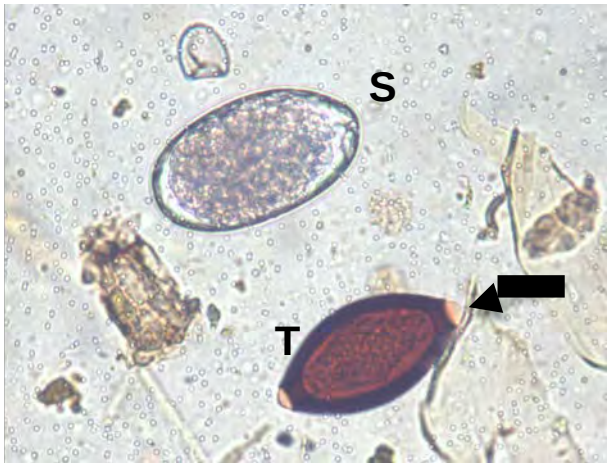


Figure 8. Strongylid (S) and *Trichuris* (T) eggs. Note similarity in size. *Trichuris* eggs have two distinctive polar plugs (arrow).

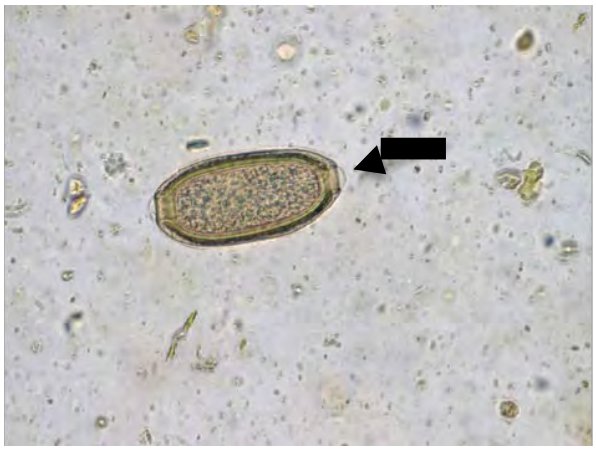


Figure 9. *Aoncotheca* egg. This egg looks like *Trichuris*, has polar plugs (arrow) like *Trichuris* but is about one- third smaller and is an uncommon finding.

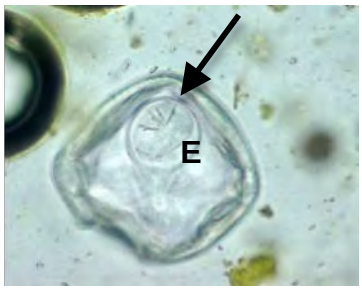


Figure 10. ***Moniezia*** **(tapeworm) egg. These eggs contain a small round embryo (E) with hooks (arrow). The embryo is difficult to see at 10X power. Eggs may appear square or triangular. The presence of the embryo distinguishes the egg from some confusing plant debris (see Figure 4).**

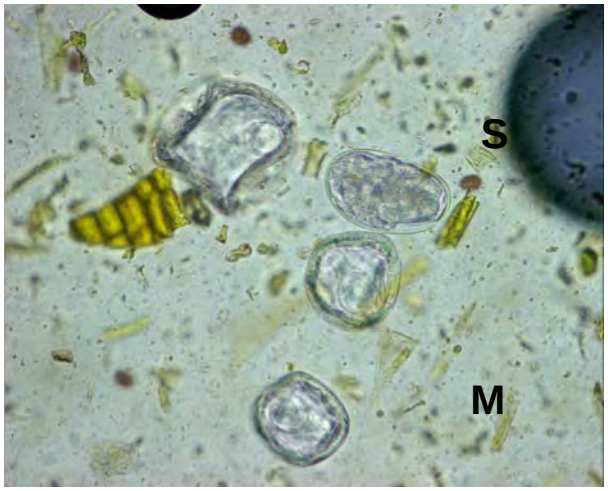


Figure 11. Strongylid (S) and ***Moniezia*** **(M) eggs. Note similarity in size.**

Dewormers & Resistance

Dewormers are a class of drugs used to kill gastrointestinal worm parasites in animals. There are two problems with dewormers for sheep and goats: the lack of being labeled for use in sheep/goats and dewormer resistance.

The greatest problem in use of dewormers is that worms have developed resistance to many dewormers due to the overuse and improper use of dewormers. Therefore, in some goat/ sheep herds there are only one or two dewormers that are still effective at killing worms, sometimes none. We need to use management techniques which suppress worms and then use dewormers only when management is not adequate to control worms. The second problem is that often an effective dewormer is not approved by the FDA for use in sheep/goats and therefore is not labeled for use in goat or sheep and must be used in an extra-label manner.

Dewormer Classes

Dewormers are divided into three classes based on their mode of action/chemical structure. Because of the chemical similarities and common mode of action, all drugs in a class are similar in many respects, but some members of a class may have unique characteristics as compared to other members of that class.

The first dewormers included phenothiazine which is no longer available. The next generation of dewormers were the benzimadoles. This class is also referred to as white drenches. They include Thiabendazole (TBZ) fenbendazole (Panacur®, Safeguard®), oxfenbendazole (Synanthic) and albendazole (Valbazen®). Since they are the oldest class of dewormers in use, the worms have had a longer period of time to develop resistance to them.

Thiabendazole is essentially useless and is no longer marketed. In more humid areas of the US where dewormers have been used quite a bit, the only one that may be effective is Valbazen® whereas in other parts of the US where there has been less dewormer used, all members of this class may be effective. All three drugs kill roundworms (such as barberpole worm, black scour worm and brown stomach worm), lungworms, worm eggs and tapeworms. In general, they have poor efficacy against arrested L4 larvae which may be improved by using a higher dose. In addition, Valbazen® also kills adult liver flukes, but has poor efficacy against the juvenile and immature stages.

The second class of dewormers are the imidazothiazoles (cell depolarizers). They have also been around quite a while, and may still be effective in many herds, a likely a consequence of not having been used as much in goats due to the narrow margin of safety. Levamisole (Tramisol, Levasole and Prohibit®) are water soluble powders that often require mixing, or may come in an injectable form.

Morantel Tartrate (Rumatel®, Positive Pellet Dewormer) is another member of this class which may be mixed with feed. Rumatel® must be mixed and used as per instructions on the bag because there are no provisions for “extra label” use of feed additives. This class is basically only effective against roundworms, arrested roundworms and lungworms.

Levamisole is typically given to goats at 1.5 times the sheep dose. However, at twice the sheep dose, toxicity can be observed; the animal walks like it is drunk, it may salivate and slobber, its eyes may tear like it is crying, and it may defecate and urinate. If it is only given a mild overdose, it will usually recover on its own in one to three hours.

Therefore, it is important to have a good idea of what your animals weigh before selecting a dose. Also, it would be good to take a break from deworming to watch animals to see how they are behaving 30 minutes after being dewormed. Since other classes of dewormers are quite safe (animals can usually tolerate at least several normal dose), it is easy to get into the habit that if in doubt whether an animals has swallowed the dewormer, give him another dose. However, don’t do that with this class of drugs because a second dose may be toxic and possibly fatal. When worms become resistant to Rumatel®, Levamisole will still work for a time. Since the gene for resistance to this drug in the worms is a recessive, drug resistance will likely be slower to develop as compared to other dewormers.

The third class of dewormers is the macrocyclic lactones (avermectins/milbemycins. Common members of this group include ivermectin (Ivomec®) dormectrin (Dectomax®), eprinomectin (Eprinex®) and moxidectin (Cydectin®). This is the most recently developed class of dewormers, and are effective against roundworms, arrested roundworms, lungworms and sucking lice. However, this class of dewormers are fat soluble and therefore drug withdrawal periods can be long because the drug is trapped in the fat of the animal.

Cydectin® is the most recent member of this class and appears to be the most potent, probably due to its long persistence in the body, but resistance is rapidly developing to this drug because it has been used so frequently.

Since only two dewormers are approved for use in goats, use of all the other dewormers is "off-label" which is described in boxed text. Goats require at least 1.5 times the sheep dose of dewormer due to having a proportionately larger liver to metabolize the dewormer and due to a faster rate of passage through the digestive tract.

Be careful on the dose with the Tramisol/Levasol/Prohibit® in that too much can be toxic as discussed above. The greater dose of dewormer in goats also requires a longer drug withdrawal period than for sheep. There is a list of withdrawal periods for giving the sheep dose to goats at http://www2.luresext.edu/goats/library/field/dawson02b.html. The withdrawal period will need to be lengthened when using a goat dose.

There is a dewormer dose chart for goats (goat dose) based on bodyweight at http://goatconnection.com/articles/publish/article_168.shtml. Administer dewormers over the back of the tongue so that they go to the rumen. There are extensions for syringes to facilitate correct placement of the dewormer. This will provide a prolonged residence time in the digestive trace and be more effective at killing worms than if the animal sucks the dewormer in the front of their mouth where most of it may go into the true stomach (abomasum).

Pour-on dewormers appear to work poorly in goats and should not be used. Injection of dewormers can promote dewormer resistance. If dewormer is given in the feed or in blocks, there is often a problem of non-uniform dosing of animals, feed hogs get a higher dose of dewormer and timid and wormy ones who aren't able to fend as well for themselves get little dewormer.

There are several strategies for increasing the efficacy of dewormers. These require more labor and may only be practical in special cases. Animals can be dewormed with a full dose of the Benzimidazole class of dewormers twice, 12 hours apart. This keeps a high level of the drug in the blood for a longer period of time, increasing the drug's effectiveness. Another trick is to fast overnight, deworm the next morning and keep off feed all day. This slows the passage of digesta and dewormer and increases its effectiveness. This should not be done with late pregnant does or ewes because of the risk of pregnancy toxemia.

There are several different strategies for using dewormers that have been used in the past and there are disadvantages to each one.

- One strategy is to deworm the herd when animals die or show symptoms of bottle jaw. The disadvantage is that animals are very wormy and the pasture has become heavily contaminated. There has already been a significant loss in animal production and health. It is much better to deworm animals before infection gets bad.

- We can deworm at stategic times when worms are most likely to be a problem such as prekidding and weaning. This can work very well. However, if refugia is low at this time, we can be promoting dewormer resistance. This also neglects year to year differences in weather which is a major factor in determining the level of worms.

- Deworming animals that don't need to be dewormed will increase resistance to the dewormer.

- We may deworm when weather conditions are favorable for worms. An old recommendation was to deworm 2 weeks after you have a month with more than 2 inches of rain and the mean temperature is over 60°F. While this is a good idea, the pasture infectivity may not be as high as we guess it to be and the animals may be withstanding the worm burden well.

We may deworm at regular intervals such as 30 or 45 days, to make sure that worms do not cause us a problem. This practice is effective at controlling worms in the short term, but is unsurpassed as the quickest way to develop dewormer resistant worms. The best strategy for deworming when the Barberpole worm is the dominant worm is to determine individually if an animal needs dewormed using the FAMACHA© program. This will reduce the development of dewormer resistance, control pasture contamination and prevent lost production due to worms. However, if the Bankrupt worm or Brown stomach worm are causing our worm problems, we must use fecal egg counts of the herd to determine when animals need dewormed and deworm the whole herd on this basis. Fortunately, in the US, these worms do not have as much dewormer resistance as the Barberpole worm.

Dewormer resistance has followed the use of dewormers. Where a given dewormer is used heavily over a period of time, resistance to that dewormer has developed. Resistance to the benzimidazole class of dewormers was documented in the 1960's. Resistance to levamisole was documented in the 1980's. Resistance to ivermectin was documented in the 1990's (was a good useful dewormer for a while). Moxidectin resistance was first documented in the 2000's.

In most of the southeastern US, there is documented resistance in sheep and goats to multiple dewormers. Several factors have contributed to the development of dewormer resistance. Veterinarians have pursued the eradication of worms instead of controlling them (deworming when the level of worms is high enough to impact the health or productivity of animals), producers found it easier to give a dewormer regularly (by the calendar rather than to evaluate whether there was a need for deworming (and dewormers were cheap!).

In addition, producers did not take the time to manage pastures to reduce parasitism. Pharmaceutical companies have encouraged heavy use of dewormers, driven by the profit motive. And, the worms were illiterate; they didn't read the pharmaceutical company ads to know which dewormers should kill them.

How do worms become resistant to dewormers?
Worms have a very genetically diverse genome. Therefore, one worm in millions of worms will have some genetic characteristic that enables him to be resistant to a specific dewormer. The potential for dewormer resistance exists in at least a few worms before a dewormer is ever used. Repeated use of that dewormer will gradually kill all the non-resistant worms in a specific population, leaving only resistant worms.

Over time, the level of resistant genes (and resistant worms) will increase in your worm population. Basically, resistance is permanent although there are a few rare exceptions. When worms in your sheep/goats become resistant to a dewormer, you can forget about ever using that dewormer again in your management program. Also, resistant worms are spread by animal movement. You may have not have worms that was resistant to a dewormer, but when you buy an animal, you get their worms for free, but worse, you get the dewormer resistance that may come with them.

That is the reason for recommending that all new animals be dewormed with two classes of dewormer (theory being that most worms will be resistant to only one class of dewormer) and a fecal sample of the sheep/goat needs to be checked a week or two later to verify the absence of eggs (Before putting them in the pasture with the rest of your animals!). Sheep and goats that originate in more humid areas, and higher value animals are more likely to have resistant worms due to greater exposure to dewormers.

One sobering fact of deworming resistance is that when our animals become resistant to all classes of dewormers, we are either going to have to do very intensive management to suppress worms or go out of the goat business. There is a recorded case of the worms being cleaned out of sheep with multiple doses of different dewormers and then dewormer susceptible worms were used to reinfect the sheep to replace the resistant worms. Also, there is a new class of dewormer which is in the FDA approval process and will likely take 5 more years to get approval. It is very potent against worms that are resistant to our present three classes of dewormers but worms can still develop resistance to it in time.

Dewormer resistance may be slowed down by;
- Not import resistant worms as described above
- Reduce deworming
- Increase refugia

Refugia
Refugia is defined as the proportion of the worm population that was not exposed to the dewormer. This includes eggs and larvae on pasture at time of treatment and the worms in animals that were not dewormed (reason for selective treatment with FAMACHA©). The importance of refugia can be illustrated by a case in Australia where it was hot and dry for 3 months and animals were dewormed in the dry period.

Deworming was very effective in that no eggs or larvae survived on pasture and only a few worms survived the deworming of the animal. However 100% of those few surviving worms were resistant. It took little time for the worm population to build (remember the barberpole worm can lay 5,000-6,000 eggs per day and every 4-5 weeks there is a new generation of worms producing eggs), but 100% of the worms were resistant because there were no susceptible worms surviving on pasture for them to mate with.

Worms in refugia are more susceptible to the dewormer and dilute out the dewormer resistant genes of the worms that survived deworming. So refugia is very important in reducing the rate of development of dewormer resistance. Note, that we do not prevent the development of dewormer resistance, but it may take two or three times longer for the worms to become resistant to the dewormer, enabling us to use the dewormer for a longer period of time.

There are three management practices that promote the development of dewormer resistance:
- Frequent deworming (more than 3 times per year)
- Underdosing the dewormer(increases proportion of worms with genes for dewormer resistance)
- Reducing refugia. Ways that we commonly reduce refugia are treating all animals at the same time, treating all animals and moving to another pasture that has few worm eggs or infective larvae, and treating when few larvae are on the pasture such as during a severe draught.

Selective treatment is very effective at increasing refugia and slowing the development of dewormer resistance. A low level of worms is normal and beneficial in that it keeps the immune system functioning against worms and provides refugia.

Therefore, the resistant worms that arise from eggs produced by dewormed animals will have many worms from eggs produced by animals that did not get dewormed. In this way, genes (for dewormer resistance) from the dewormed animals will be diluted and the resultant dewormer resistance will be increased only slightly. This is why FAMACHA© is so important to help slow the development of dewormer resistance. It is the only practical way that we can selectively deworm our animals and maintain a high level of refugia which slows the development of dewormer resistance.

Because dewormer resistance can put a producer out of business, especially in the more humid areas of the southeastern US, a producer needs to develop a plan to reduce the rate at which dewormer resistance develops. Management practices which suppress worms are going to have to be the foundation of the control program and dewormers are going to have to be used sparingly and intelligently and only when needed (selective deworming).

Unfortunately, dewormer resistance is inevitable, but how soon it develops it depends on your management. Using FAMACHA© will slow the development of dewormer resistance as compared to conventional deworming management. However, as mentioned already, the quickest way to get resistant worms is to buy them. Of course no one purposely buys resistant worms, but that expensive good looking animal that you just bought brings his worms along and they may be very resistant to dewormers.

Expensive animals tend to be dewormed excessively since the owners do not want to risk worms killing them. This is especially true if the animal comes from a humid area. Therefore, expensive animals are more likely to have resistant worms. Since buying resistant worms is the quickest way to get dewormer resistant worms, it is wise to make sure that those animals that you bring into your herd do not bring resistant worms in. This is done by deworming them two classes of dewormer (such as Valbazen© and Prohibit©; full goat dose of each at the same time) and then a week later, do a fecal egg count on the animal which hopefully will be zero or close to zero.

If not, you may have to resort to a 3-way combination of Valbazen©, Prohibit© and Cydectin©, all given at the goat dose given at the same time. If the fecal egg count is not zero in a week, you don't want these worms nor the animal on your farm. Highly resistant worms in one animal can become highly resistant worms in other animals in your herd in a short period of time. How are you going to control those worms? Sometimes, unfortunately, the solution is to go out of business although there are some solutions as discussed below.

When you have resistance to all dewormers i.e. none work, there are a few techniques which often require intensive management that can enable a producer to stay in the business for several more years. Hopefully, there will be another class of dewormers available within 5 years that will provide help. One can use a short duration (5 days on a pasture), long rest period (40 +days) grazing program so that sheep/goats pick up few worms and therefore do not need to be dewormed.

One may be able to use alternative dewormers (sericea lespedeza, copper oxide wire particles etc.) as discussed in the next article in this series. However, one is going to have to be good at parasite management to stay in the business. It is better to use good management to prevent your animals from getting into this difficult problem. Using a conventional dewormer at a higher dose (Prohibit© should not be given at more than 1.5 times sheep dose due to toxicity), giving combinations of dewormers or giving dewormers (or combinations) for 2 or 3 days in a row are techniques that can kill most resistant worms. You can select your animals for genetic resistance to worms.

All this makes good parasite management program to reduce dewormer resistance sound better all the time.

In conclusion, next time you deworm, do a fecal egg count to determine if your dewormer is working. If not, switch to another dewormer and check to see if it is working. Use a proper dose of the drug and know the drug withdrawal. Use FAMACHA© to slow down the development of dewormer resistance. Do not buy dewormer resistance and worm all incoming animals with two dewormers. Use management to prevent worms which reduces how often you deworm. Deworm only those animals needing it based on FAMACHA©. Cull animals requiring the most deworming.

Dewormer Chart for Goats

Important --Please read notes below before using this chart

1 ml = 1cc	Valbazen (albendazole) ORALLY	SafeGuard (fenbendazole) ORALLY	Ivomec Sheep Drench (ivermectin) ORALLY	Prohibit (levamisole) ORALLY	Cydectin Sheep Drench (moxidectin) ORALLY	Rumatel (morantel) Feed Pre-mix ORALLY
Weight Pounds (lbs)	20 mg/kg 2 ml/ 25 lb	10 mg/kg 1.1 ml/ 25 lb	0.4 mg/kg 6 ml/ 25 lb	12 mg/kg 2.7 ml/ 25 lb	0.4 mg/kg 4.5 ml/25 lb	10 mg/kg 45 gm/100 lb BW (Durvet)
20	1.6	0.9	4.8	2.2	3.6	
25	2.0	1.1	6.0	2.7	4.5	11 grams
30	2.4	1.4	7.2	3.3	5.4	
35	2.8	1.6	8.4	3.8	6.5	
40	3.2	1.8	9.6	4.4	7.3	
45	3.6	2.1	10.8	4.9	8.2	
50	4.0	2.3	12.0	5.5	9.0	23 grams
55	4.4	2.5	13.2	6.0	10	
60	4.8	2.7	14.4	6.6	11	
65	5.2	3.0	15.6	7.1	12	
70	5.6	3.2	16.8	7.7	12.7	
75	6.0	3.4	18.0	8.2	13.6	34 grams
80	6.4	3.6	19.2	8.8	14.6	
85	6.8	3.9	20.4	9.3	15.4	
90	7.2	4.1	21.6	9.9	16.4	
95	7.6	4.3	22.8	10.4	17.3	
100	8.0	4.6	24.0	11.0	18	45 grams
105	8.4	4.8	25.2	11.5	19	
110	8.8	5.0	26.4	12.1	20	
115	9.2	5.2	27.6	12.6	21	
120	9.6	5.5	28.8	13.2	22	
125	10.0	5.7	30.0	13.7	22.7	56 grams
130	10.4	5.9	31.2	14.3	23.6	
140	11.2	6.4	33.6	15.4	25.4	
150	12.0	6.8	36.0	16.5	27.3	68 grams

Valbazen Suspension (11.36 % or 113.6 mg/ml): 20 mg/kg orally; withdrawal time is 9 days for meat and 7 days for milk Do NOT use in pregnant does in the first trimester of pregnancy

Safe-Guard/ Panacur Suspension (10% or 100 mg/ml): the label dose in goats is 5 mg/kg, but a 10 mg/kg dosage is recommended. At 10 mg/kg, withdrawal time is 16 days meat and 4 days for milk. Add 1 day for each additional day the drug is used (e.g. if administered 2 days in a row then withhold milk for 5 days after 2nd dose).

Ivomec Sheep Drench (0.08% or 0.8 mg/ml): 0.4 mg/kg orally; meat withdrawal time is 14 days and milk withdrawal is 9 days.

Prohibit Soluble Drench Powder (Sheep): (Note that this drug is also sold as Levasol and Tramsiol) 12 mg/kg oral dose with meat withdrawal of 4 days and milk withdrawal of 3 days. Solution prepared by dissolving a 52 gram packet in 1 quart (943 ml) of water. This yields a solution with 49.6 mg/ml. If dosing kids, it is safer to dilute further (1 packet in 2 quarts of water), and then administer twice the amount listed on the chart. The larger volume administered will then provide a wider margin for safety if there are small errors in dosing.

Southern Belle Classic - Cuba, MO

Ring A: Judge Cindy Lynn Huggins
Ring B: Judge Jim Dowell
Ring C: Judge Shelly Strahan
***Indicates verified MGR Point**

Ring A- June 11, 2021

Junior Champion Doe

Naughty Goat Acres Scout	Colleen Reardon & Robert Lorenz

Reserve Junior Champion Doe

WP Willow	Renee Anderson

Senior Champion Doe

Oeltjenbruns' Farms Bessy	Debra Dockendorf

Reserve Senior Champion Doe

Twin Creek Rosebud	Colleen Reardon & Robert Lorenz

Grand Champion Doe

*Oeltjenbruns' Farms Bessy	Debra Dockendorf

Reserve Grand Champion Doe

Twin Creek Rosebud	Colleen Reardon & Robert Lorenz

Junior Champion Buck

WP You're My Boy Bleu	Leah Dockendorf

Reserve Junior Champion Buck

Naughty Goat Acres Hooligan	Janice Foster

Senior Champion Buck

Hillside Acres Carbon	Janice Foster

Reserve Senior Champion Buck

Wallace's Crazy Acres Oakley	Renee Anderson

Grand Champion Buck

*Hillside Acres Carbon	Janice Foster

Reserve Grand Champion Buck

Wallace's Crazy Acres Oakley	Renee Anderson

Junior Champion Platinum Wether

BDF Deluca	Leonie Dysart

Reserve Junior Champion Platinum Wether

Senior Champion Platinum Wether

Stray Eight Zane	Debra Dockendorf

Reserve Senior Champion Platinum Wether

Riverside Fainters Locked and Loaded	Alison Thielen

Grand Champion Platinum Wether

*BDF Deluca	Leonie Dysart

Reserve Grand Champion Platinum Wether

Stray Eight Zane	Debra Dockendorf

Southern Belle Classic - Cuba, MO

Ring B- June 12, 2021	
Junior Champion Doe	
BDF Jade	Drew & Amy DeRiemacker
Reserve Junior Champion Doe	
Bureau Creek Show Girl	Nikki Thummel
Senior Champion Doe	
Buck Creek Mystique	Drew & Amy DeRiemacker
Reserve Senior Champion Doe	
Twin Creek Rosebud	Colleen Reardon & Robert Lorenz
Grand Champion Doe	
*Buck Creek Mystique	Drew & Amy DeRiemacker
Reserve Grand Champion Doe	
BDF Jade	Drew & Amy DeRiemacker
Junior Champion Buck	
Naughty Goat Acres Hooligan	Janice Foster
Reserve Junior Champion Buck	
Stray Eight Vesuvius	Debra Dockendorf
Senior Champion Buck	
Wallace's Crazy Acres Oakley	Renee Anderson
Reserve Senior Champion Buck	
Buck Creek Hollywood Gold	Danielle Frost
Grand Champion Buck	
*Wallace's Crazy Acres Oakley	Renee Anderson
Reserve Grand Champion Buck	
Buck Creek Hollywood Gold	Danielle Frost
Junior Champion Platinum Wether	
BDF Deluca	Leonie Dysart
Reserve Junior Champion Platinum Wether	
Frosty Acres Tanner	Danielle Frost
Senior Champion Platinum Wether	
Bureau Creek Amazon Prime	Danielle Frost
Reserve Senior Champion Platinum Wether	
Oeltjenbruns' Farms Jager	Danielle Frost
Grand Champion Platinum Wether	
Bureau Creek Amazon Prime	Danielle Frost
Reserve Grand Champion Platinum Wether	
*Oeltjenbruns' Farms Jager	Danielle Frost

Southern Belle Classic - Cuba, MO

Ring C- June 12, 2021

Junior Champion Doe

Nine Acres Farm Halla	Nikki Thummel

Reserve Junior Champion Doe

Naughty Goat Acres Spitfire	Colleen Reardon & Robert Lorenz

Senior Champion Doe

Fern Hill Prudence	Drew & Amy DeRiemacker

Reserve Senior Champion Doe

WP Amaretto	Danielle Frost

Grand Champion Doe

*Fern Hill Prudence	Drew & Amy DeRiemacker

Reserve Grand Champion Doe

WP Amaretto	Danielle Frost

Junior Champion Buck

Nine Acres Farm Somethin Bout A Truck	Nikki Thummel

Reserve Junior Champion Buck

Naughty Goat Acres Hooligan	Janice Foster

Senior Champion Buck

Hillside Acres Carbon	Janice Foster

Reserve Senior Champion Buck

Naughty Goat Acres Boomer	Nikki Thummel

Grand Champion Buck

*Hillside Acres Carbon	Janice Foster

Reserve Grand Champion Buck

Naughty Goat Acres Boomer	Nikki Thummel

Junior Champion Platinum Wether

BDF Deluca	Leonie Dysart

Reserve Junior Champion Platinum Wether

Frosty Acres Tanner	Danielle Frost

Senior Champion Platinum Wether

Riverside Fainters Locked and Loaded	Alison Thielen

Reserve Senior Champion Platinum Wether

Bureau Creek Amazon Prime	Danielle Frost

Grand Champion Platinum Wether

*Riverside Fainters Locked and Loaded	Alison Thielen

Reserve Grand Champion Platinum Wether

BDF Deluca	Leonie Dysart

Diseases and Testing

Welcome to part three and the final part of the discussion series on Diseases and Testing. So far in this series, we have looked at two of the three important diseases that are common among goats- Caprine Arthritis Encephalities (CAE) virus, and Caseous Lymphadenities (CL). In this final part of the series, we are going to dive into the disease that I like to refer to as the "silent killer", Johnes disease. In my opinion, this disease is the most serious of the three and is also the most complex and difficult to understand. We will look at what Johnes disease is, and how it can be contracted. We will wrap up with a discussion about reasons to test, how to test, and ways to help prevent bringing CAE, CL and Johnes into your herds.

Johnes disease

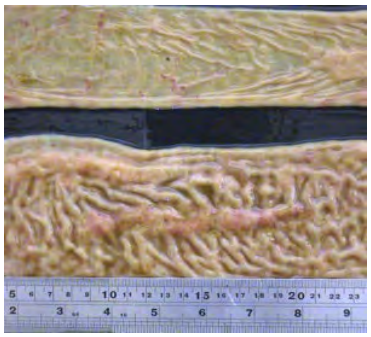
*source: Johne's Information Center; University of Wisconsin- Madison, School of Veterinary Medicine; www.johnes.org

A to Z

The History

"Hey Doc! While you're here, would you please look at Bella? She had a nice calf about three weeks ago and has been eating well, but she looks too thin and isn't milking as well as she should. She also seems to have rather loose manure."
This is the kind of question I imagine was presented to Dr. F. Harmes about a Guernsey cow in the Oldenburg region of Germany in 1895. Dr. Harmes preliminary diagnosis was intestinal tuberculosis (TB). TB in cattle was quite common in Germany then. But when he did the tuberculin skin test to confirm his diagnosis, the cow tested negative. So, the reason for the cow's condition remained a mystery.

A few months later, the cow died. Curious as to what killed the cow, Dr. Harmes sent intestines and other tissues to the Pathology Unit at the veterinary school in Dresden. There the tissues were examined by Dr. Heinrich A. Johne, Professor of Pathology (picture at right provided by Dr. J.B. Jørgensen, State Veterinary Serum Laboratory, Copenhagen, Denmark), and Dr. Langdon Frothingham, a visiting scientist from the Pathology Unit in Boston, Massachusetts.



They observed that the small intestine was quite a bit thicker than expected and that lymph nodes near this thick intestine were enlarged. The photo on the left shows a normal intestine at the top and the intestine thickened due to Johne's disease at the bottom. Lymphoid tissue, called Peyer's Patches, are also quite prominent (the raised and slightly red tissue running long-ways down the center of the thickened intestine). Interestingly, Dalziel in 1913 saw the same kind of pathology when he removed a section of intestine from a person with Crohn's disease remarking in his report that it resembled the cattle problem Dr. Johne had described.

Using what at the time were newly developed histopathology techniques, parts of the intestine were "fixed" (pickled in formaldehyde), sliced into very thin sections, placed on a microscope slide, and stained with special dyes – known as an acid-fast stain – designed to help visualize bacteria of the type causing TB. Under the microscope, Drs. Johne and Frothingham saw that the intestinal wall was filled with inflammatory cells of the kind to be expected in TB (macrophages and lymphocytes – the blue-colored stuff in the photo). In addition, they saw abundant red-staining bacteria (which microbiologists call acid-fast bacteria) throughout the inflamed tissues. Basically, it looked just like intestinal TB. But, when a sample of the fresh infected tissue containing the red-staining bacteria was injected into guinea pigs, it didn't cause TB. This took place shortly after Louis Pasteur had devised the "germ theory" of disease and before techniques for growing bacteria in the laboratory were widely available. Inoculating animals, therefore, was a routine way of detecting infectious microbes such as those that cause TB, and guinea pigs are quite susceptible to tuberculosis. So, the diagnosis on this cow remained a mystery.

Drs. Johne and Frothingham concluded that the disease seen in the very sick Guernsey cow was caused by a bacterium other than the one normally causing TB in cattle, namely Mycobacterium bovis. They speculated that perhaps the pathology was due to a related bacterial pathogen such as the one causing TB in birds, aptly named Mycobacterium avium. Considering their subject's gross pathology, microscopic pathology (histopathology) and animal inoculation findings, they proposed the name "pseudotuberculous enteritis" for the disease; a designation meaning inflammation of the intestine resembling intestinal TB but not actually the same as intestinal TB – somehow different. Soon after publication of their report, veterinarians began reporting outbreaks of this curious intestinal malady among dairy cows in Denmark, The Netherlands and elsewhere in continental Europe.

Hopping across the Atlantic

Dairy cattle came to the U.S. with the first European settlers. However, the dairy industry really started growing in the U.S. with the invention of the mechanical milking machine. As the dairy industry expanded, dairy cattle of diverse breeds and with high milk production potential were imported to the U.S. to build dairy herds using animals with the best genes. Most of these came from Europe, home to the major dairy breeds of cattle such as Holsteins, Guernsey's, Jerseys, Ayrshires, and Brown Swiss. Along with these imported cows, unknowingly and unfortunately, came a few that were incubating this mysterious malady that Dr. Johne had described.

In the early 1900s, Pennsylvania was home to a good proportion of all U.S. dairy cows, and it was there that this strange phenomenon of diarrhea and weigh loss was first recognized. In 1908, replicating what had been observed earlier in Germany, a farmer noticed a cow that was very thin and suffering from diarrhea. Seeking a diagnosis, the farmer sent this cow to the state veterinary school for examination by pathologists. Dr. Leonard Pearson (pictured to the right), who was serving as Dean at the University of Pennsylvania Veterinary School, examined this cow and became the first to publish a U.S.-based report of what we now call Johne's (Yo-nees) disease. Today the disease also goes by the name paratuberculosis.

The clinical signs of paratuberculosis – diarrhea and weight loss – are a bit vague, resembling those of many other cattle diseases. The pathology, however, is quite distinctive, which makes diagnosis by necropsy and histopathology quite straight-forward, especially when special stains are used to reveal the acid-fast (red-staining) bacteria. What eluded diagnosticians for several years was the ability to grow the microbe causing this new disease in the laboratory. This, after all, has long been the most common and reliable way of confirming the diagnosis of an infection.

Growing MAP in the lab

In the golden days of microbiology, once Pasteur had shown that germs cause disease and Koch had devised a method to grow bacteria in pure culture, the causes of many of the worst diseases plaguing humans – such as tuberculosis, typhoid fever, cholera, and diphtheria – were identified. The scientific standard for proving that the microbe scientists recovered from patients was in fact the cause of a given disease was to 1) show that the organism was abundant in sick patients or animals but not found in healthy ones, 2) grow the organism in pure culture from diseased tissues, 3) reproduce the disease by inoculating a culture of the microbe it into healthy animals, thereby causing the same disease, and finally 4) recover the same bacterium from the sick or dead animals. This standard of scientific proof became known as Koch's postulates after Robert Koch, the German microbiologist who formulated them. Koch soon abandoned these rigid criteria for proving causality after observing that some people were asymptomatic carriers of pathogens causing typhoid fever (remember Typhoid Mary?) and cholera.

For the next 17 years after Drs. Johne and Frothingham diagnosed their peculiar form of intestinal tuberculosis, the microbe causing this disease could be seen in tissue sections, but not cultured in the lab. Thus, it was not yet possible to figure out the identity of the microbe responsible for causing the thickened intestines, weight loss and diarrhea.

Finally, in 1912, a serendipitous observation by F. W. Twort, a British scientist, led to the successful isolation of the elusive microbe. Ironically, Twort's success derived in part from his failure to do a careful job cleaning laboratory glassware, along with his discriminating eye. Thus, Dr. Twort observed the presence of small bacterial colonies growing like satellites around larger colonies in old bacterial cultures that he was preparing to discard. These larger colonies were contaminants in his cultures, a species of Mycobacterium common in the environment, called Mycobacterium phlei. Suspecting that the M. phlei bacteria were providing some essential nutrient, Twort incorporated a heat-killed preparation of M. phlei into his culture medium. This new culture medium, he discovered, supported the growth of a new acid-fast bacterium from intestinal tissues of cows with Johne's disease. He named it "Mycobacterium enteriditis chronicae pseudotuberculosae bovis Johne" – a real mouthful. Fortunately for those biologists who pursued this microbe – as well as for anyone reading this article! – this name was soon shortened to Mycobacterium paratuberculosis, which, by microbiology convention, is simply written as M. paratuberculosis. Modern bacterial taxonomic methods have revised the name to Mycobacterium avium subspecies paratuberculosis (MAP).

Twort had witnessed two unique biological characteristics of M. paratuberculosis that even today are the benchmarks for defining the organism: 1) extremely slow growth (8-12 weeks of incubation before seeing colonies in culture tubes), and 2) the fact that they require a substance known as mycobactin (provided by other mycobacteria such as M. phlei) to grow in laboratory culture media, a characteristic referred to as mycobactin-dependence. Laboratories commonly culture tissue or manure samples on tubes of laboratory culture media with and without mycobactin. Slow growth of acid-fast bacteria on culture media with mycobactin (right-hand tube) and not on media without mycobactin (left-hand tube in the photo to the right) is strong evidence for isolation of M. paratuberculosis. Today, microbiologists can go one step further and test for the presence of genetic elements unique to M. paratuberculosis. These genetic markers are given seemingly arcane designations like IS900, HspX, Mav2, F57, and 251F.

Insidious and ignored

Johne's disease typically shows up as a curious combination of weight loss and decreasing milk production in cows with a healthy appetite and no fever. Other than being thinner than their herd-mates, these cows typically don't look or act sick. Of course, a dairy cow that's not giving her fair share of milk is not destined to stay around long. Most farmers simply send her to slaughter and replace her with another, better-producing cow.

Johne's disease has continued spreading among herds, between states and regions, and between countries for a multitude of reasons. First, the cattle industry failed to heed the warnings of veterinarians from the 1920s. Second, the disease spreads insidiously and the clinical signs are rather subtle, hence easily overlooked or ignored. Third, national and international veterinary regulations have either been too lax or too often ignored. Fourth, the economic impact of the disease is subtle and not readily apparent until a high proportion of the cows in a herd are infected.

As a consequence, over 50% of dairy herds in most major dairy producing countries are now MAP-infected. The official estimate in the U.S. from a survey conducted in 2007 and published in 2013 is that 91% of U.S. dairy herds are infected. This is up from USDA's 1996 estimate that 21.6% of U.S. dairy herds have paratuberculosis. The infection has spilled over into a variety of other animal species, including but not limited to; beef cattle, sheep, goats, bison, deer, elk, llamas, alpacas, camels, antelope and other exotic ruminants that can be found in zoos. There are also two reports of nonhuman primates having paratuberculosis, one in Rhesus macaques and the other in a Mandrill baboon. Multiple species of wildlife also have been found to be infected but in most instances these animals do not develop progressive disease or pass the MAP organisms in their feces and so are considered a "dead-end" host. This link takes you to a page on this website describing the many non-ruminant animals that have been found to be MAP-infected.

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The natural history of Johne's disease

The natural host for MAP is ruminants, meaning animals with a four-chambered stomach that chew their cud as they digest their diet of grass and hay. A natural host is one in which the relationship between host and microbe is relatively well-balanced. The host does not mount an immune response that ejects the microbe from the body and the microbe does not kill the host – at least not for a long time. The causes of TB in humans and cattle, and the cause of Johne's disease, M. paratuberculosis (MAP), are excellent examples of this balanced host-pathogen relationship. These situations allow MAP to silently multiply and leave one host via milk and manure to set up residence in another host, usually a newborn, after being consumed.

Newborn animals are most susceptible to MAP infection, which they get by ingesting the organism. In dairy calves, this can happen in a variety of ways. Often calves are born in a large maternity pen occupied by a number of pregnant cows on the verge of giving birth. Naturally, the pen is kept as clean as possible. However, cows produce about 60 pounds of manure a day and it can be challenging to keep things clean. Shortly after its birth, the newborn calf will try to stand and find its mother's teat for its first crucial drink of colostrum, rich in antibodies and nutrients. However, the calf will typically fall on its face as it tries to get those wobbly legs to work. And sometimes, not surprisingly, it does a face-plant right into a cow pie! Once on its feet, the calf tries to home in on mom's udder. Its efforts are not always on target and the first few efforts to suckle may be on mom's back leg or some other part of the cow. Eventually, the calf will successfully find the teat to which it latches and begins to suckle, just like a newborn baby. Of course, the teat surface – not to mention the mother cow's hind leg – has in contact with the bedding in the maternity pen and thus well may have also been in contact with manure from any of the cows in the pen that day. So, in these first few hours after being born, a calf has multiple opportunities to swallow a bit of MAP-laden manure.

Ingestion of MAP-contaminated manure is probably the most common means of exposure and infection for newborn calves. However, in cows that are in the more advanced stages of infection, the MAP bacteria escape from the infected intestinal wall and local lymph node, and are carried throughout the cow's blood stream. The cow's natural defenses will try to clear these bacteria from the blood, causing them to concentrate in the liver and spleen, organs that filter blood to remove microbes and aging red blood cells. But MAP will be found not only in the liver and spleen. It also will cross into the udder and then be found in the milk. And, if that cow is pregnant, the MAP bacteria in the blood will cross the placenta and infect the fetus resulting in a calf that is MAP-infected even before birth.

It is vital that calves drink three or four quarts of colostrum within six hours of birth. This is the only means by which they can get antibodies to protect them from viral and bacterial pathogens they will inevitably encounter during their first weeks of life. After their colostrum meal, calves are fed milk until they are weaned at 8-12 weeks of age. Many modern dairy farms will feed a product called "milk replacer." This is essentially pasteurized milk in powder form, just like human infant formula. However, on some farms raw milk, called "waste milk," is fed to calves. This milk is of a quality not suitable for human consumption. It comes from cows with mastitis, which causes the milk to contain too many white blood cells, or it is derived from cows that have been treated with antibiotics and thus is unsuitable for human consumption until after the drug has been cleared from the animal's system. Raw (not heat-treated) waste milk can harbor a variety of microbial pathogens. MAP is one of them.

MAP has cleverly devised a way to efficiently hop from infected adult cows to their calves. Other cattle pathogens like bovine brucellosis have adopted similar strategies. Indeed, there is an association between the MAP infectious status of a cow and her calves. This is truer in animal husbandry systems such as for beef cattle, where a calf stays at its mother's side nursing until it is six months old or more. This association is weaker in husbandry systems like that on most modern dairy farms, where the calf is removed from its mother soon after birth, reared well away from adult cows and fed milk artificially.

In cattle, the time lag between initial infection as a fetus or neonate until clinical signs of Johne's disease and death can be as short as 2 years or as long as 12, or even more. We don't really know how long cattle could survive with Johne's disease because farmers simply send them to slaughter when they start going downhill, before they die a "natural" death. The speed with which the infection progresses is governed mostly by the dose of MAP to which the calf was exposed. It may also be affected by the age of exposure or the genetics of the animal. Geneticists have found evidence that some cattle are more resistant to the infection than others. None are totally resistant, however. For most of this prolonged incubation period, before an animal shows visible signs of Johne's disease, MAP will be found in the wall of the intestine, in manure and in milk, although the abundance in milk is much lower than in manure.

This story of the pathogenesis of Johne's disease is similar for most animals that are considered its natural host, namely ruminants. In ruminants MAP has adopted a life style as an "obligate parasite." Although its ancestors were able to grow and replicate in soil and water, MAP has evolved along its own special track, adopting ruminant animals (specifically inside certain "white blood cells" called macrophages – blue cells in the adjacent photo. The MAP bacteria are stained red) as its host. In fact, it has lost the ability to replicate outside animals. It is obligated to a life inside the cells of its host; it is therefore called an obligate intracellular parasite.

Once MAP leaves its warm, nutritious intracellular home, it must wait patiently (it has no in dependent means of moving) until it is eaten by a susceptible host animal. Then, the infection and replication process begins again. Since this wait can be a very long one (imagine MAP sitting patiently, biding its time in a cow pie out in the middle of a pasture), MAP has evolved strategies for resistance to environmental conditions like heat, freezing, drying, and sunlight: factors that effectively kill most microbes. Under adverse conditions, MAP changes into a resting, or dormant form of bacterial cell called a spore. Bacterial spores are notoriously resistant to both physical and chemical factors that kill ordinary bacteria.

Why not treat animals with Johne's disease?

Treatment of Johne's disease in cattle has been attempted, but without much success. Although it may be theoretically possible to cure this infection, the antibiotics required are not legal for use in food-producing animals. Hence all the meat and milk from animals treated with such drugs would have to be discarded, not eaten. Moreover should anyone decide to attempt this, the drugs are expensive: dosages for a 1200 pound animal are large, and the duration of treatment must be 12 months or longer. Consequently, such a treatment regimen, if attempted, would cost 50 times the value of most dairy cattle, which simply does not make economic sense. So, animals that develop signs of Johne's disease or are found positive by diagnostic tests are simply sent to slaughter. There is no small irony here: Even if the cost of treating Johne's cattle were low enough to be economically feasible, regulations against human consumption of the relevant antibiotics would preclude doing so … and as a result, untreated cattle, ripe with untreated and virulent MAP bacteria, are made into meat – mostly hamburger – for direct human consumption! Recently, some investigators have claimed to be able to prevent or even cure Johne's disease with live cultures of a bacterium known as Deitzia – a "yogurt-like" probiotic approach. This work, while promising and potentially helpful to humans with Crohn's disease, has yet to be reproduced in a carefully controlled trial by scientists independent of the company selling the product.

How about vaccination?

There are vaccines for Johne's disease, but they are not very effective. In fact, there are no effective vaccines for any bacteria in the genus Mycobacterium, including those causing tuberculosis and leprosy. Mankind has been pursuing vaccines for mycobacterial infections without success since Robert Koch first described Mycobacterium tuberculosis in 1882. Scientists the world over continue trying.

There is only one USDA-licensed vaccine for Johne's disease in the U.S., known as Mycopar™. Its use is tightly regulated because vaccination with Mycopar™ causes animals to test positive for bovine tuberculosis, which seriously complicates U.S. efforts to eradicate bovine TB. The most recent clinical trial of Mycopar™ in dairy herds with Johne's disease was published in March 2013. In that case, every other calf born on three Wisconsin dairy farms was vaccinated with Mycopar™ before they reached 35 days old; they were then followed for several years. The authors reported that when vaccinated and non-vaccinated animals were compared, there was no significant difference in the rate of clinical Johne's disease, no improvement in the longevity of cows in the herd, and no difference in milk production or reproduction. They did, however, observe that the vaccinated cows had a lower frequency of detectable MAP in their feces.

Although the Mycopar™ vaccine may somewhat lower the frequency of clinical Johne's disease, many vaccinated cows still progress to this end stage of the infection. Pictured here is a cow that had been Johne's-vaccinated and yet still developed all of the classical signs of Johne's disease and the diagnosis was confirmed by detecting MAP in her feces. Notice the golf ball-size lumps in this cows brisket caused by the vaccine administered to this animal when it was less than 35 days old.

In addition to interfering with the bovine TB eradication program, vaccines for Johne's disease often cause significant damage at the site of inoculation, the brisket. These "vaccine-induced granulomas" range in size from golf balls to soccer balls. They tend to get banged around as the animal approaches a feed bunk and can get infected and ooze pus.

Animals are not the only ones that get vaccinated on the farm. Occasionally, when struggling with a rambunctious calf while trying to vaccinate it, animal holders or veterinarians will accidentally get stuck with a needle. Even the smallest prick from a needle that has Mycopar on it will lead to a painful swelling. This has happened often enough to warrant publications about these events, since the human pathology that results are typically quite graphic. For veterinarians, the painful lesions – that may take months or longer to resolve – can be incapacitating.

Then what CAN be done?

Control of Johne's disease in farm animals is fairly straight-forward, but requires consistency and lots of patience. To significantly reduce the infection rate in a dairy herd, herd managers must follow recommended changes in management, specifically designed to limit transfer of the MAP infection from cows to calves. The most critical changes for dairy herds are:

Only allow MAP test-negative cows into the main maternity pen,
Remove the newborn calf and cow from that pen within an hour of birth,
Feed the calf clean colostrum from a MAP test-negative cow, and then
Feed the calf pasteurized milk, meaning milk replacer or milk from a dairy herd that is pasteurized on the farm.
To find sources of MAP, herd owners must test every cow in the herd at least once during each lactation (basically once a year) and they must consistently act on the test results by culling (slaughtering) or segregating the test-positive animals. And, most importantly, they must do this consistently for a period of at least 6 or more years. Published evidence shows that such a program is very successful at reducing the infection rate in dairy herds. The strategies in other species are similar in concept but must be adapted to different animal husbandry methods.

Animals become or produce food

If you are a consumer, particularly if you are not a vegan, you may be thinking: "Hey! What happens to these animals with Johne's disease that are sent to slaughter? And, where does their milk go?" The troublesome truth is that every day, dairy cattle, beef cattle, goats and sheep with Johne's disease are sent to slaughter because they are no longer healthy productive animals. Veterinary inspectors who review the condition of live animals recognize that they may be a bit thin, but are otherwise healthy and are therefore deemed acceptable for use as food. On the slaughterhouse floor, veterinary inspectors who examine the insides of each carcass report seeing intestinal pathology indicative of Johne's disease on a regular basis, but they see nothing that by law requires carcass condemnation (as would be the case for pathology indicative, for example, of bovine TB). Thus, all these infected animals enter our food supply. Most become ground beef.

Likewise, dairy animals with Johne's disease – be they dairy cattle, goats or sheep – produce milk that enters the food supply. Most milk ends up as some form of cheese. The production of many but not all cheeses involves pasteurization of the milk as a first step in the production process. However, some cheeses can only be made from raw milk. By law, virtually all milk used for fluid consumption in developed countries is pasteurized. However, some "health food" advocates in the U.S. stridently argue that raw milk is better for you and have lobbied to change laws prohibiting sale of raw milk or have found other creative ways to circumvent pasteurization laws. Many of the raw milk producers and likely all of the raw milk consumers are unaware of the potential risks of drinking MAP. They seldom if ever ask if the animals producing their milk have Johne's disease. If the herd providing raw milk does have Johne's, then the milk is highly likely to be laced with MAP along with a long list of other zoonotic pathogens.

It is controversial whether MAP is killed by pasteurization or other food manufacturing practices. Suffice it to say that live MAP have been recovered form retail pasteurized milk in the U.S., U.K. Czech Republic, Argentina, Brazil and India.

Why aren't producers doing something about this?

MAP is a nagging but not very economically important problem in most herds and flocks of food-producing animals. As businesses with narrow profit margins, herd owners are not motivated to spend money to fix a problem that does not seem to be economically crucial. A common complaint of dairy herd owners is that a Johne's disease control program is more costly than the disease, a variant on "If it ain't broke, don't fix it" – "if it costs too much, don't bother."

Many countries have launched national programs to control Johne's disease. When government agencies subsidize the costs of Johne's disease control, more producers participate in control and herd certification programs. When the money disappears, they stop. As a result, MAP flows, quite literally, through the food chain to land on the consumer's plate as MAP-burgers, or in their glass of milk. Even scarier is the potential that MAP is in infant formula where, even if dead, it may trigger pathology. Since medical science does not currently label MAP a zoonotic pathogen – meaning an infectious disease transmitted from animal to humans – there is no legal way to stop MAP from contaminating foods. So, who decides if MAP is zoonotic? Good question!

The relationship between MAP, food-producing animals and humans is complex and still somewhat murky. Solutions will require more scientific investigation, and a rational dialogue based on mutual respect between food producers in animal agriculture, veterinarians, government agencies and medical doctors. Among these voices, consumers have been notably silent. The Johnes.org website is one step toward changing this situation.

Johnes in Goats

The previous pages have given us an over-view on Johnes disease. Much of what you have read has talked more about the disease in cattle than it has about goats. Johnes gets most of its attention from the cattle industry, but it is just as prevelant in goats.

EPIDEMIOLOGY

Prevalence

There are relatively few surveys for Johne's disease in goats. Three European studies reported a low MAP infection prevalence (see Nielsen & Toft (Prev.Vet.Med. 88:1-14, 2009). Norway recognized that paratuberculosis was a problem for a limited number of its goat herds and effectively eradicated the infection, according to their 2016 report. A systematic literature review published in 2014 found that in Latin America and the Caribbean for goats the animal-level and herd-level of paratuberculosis was 4.3 % and 3.7 %, respectively.

In Ontario , Canada, Dr. Cathy Bauman and colleagues surveyed 580 goats (lactating and 2 years of age or older) randomly selected from 29 randomly selected dairy goat herds. Animals were tested for MAP in fecal samples by both culture PCR, and for serum antibodies by enzyme-linked immunosorbent assay (ELISA). Using sophisticated statistical methods they concluded that the estimated farm-level true prevalence was 83.0% (proportion of dairy goat herds estimated to be MAP-infected).

In the U.S., Patrick Pithua and Nathaniel Kollias from the Dept. of Veterinary Medicine and Surgery, University of Missouri collected blood samples from goats ≥24 months of age in 25 Missouri Boer goat herds and tested them for MAP antibodies using a commercial ELISA kit. Herds were declared positive for MAP if one or more goats in the herd tested ELISA-positive. Of 629 goats tested only 12 were ELISA-positive giving an estimated true animal-level prevalence of 1.4%. However there was at least one ELISA-positive goat in 9 of the 25 herds tested giving an estimated true herd-level prevalence of 54.7%. They concluded that MAP infections are endemic in Missouri Boer goat herds. This study was published in Veterinary Medicine International.

In 2017, the Johne's Testing Center, University of Wisconsin received 709 goat fecal samples for testing by PCR. Of these, 75, or 10.5%, were PCR-positive for MAP and these originated from over 5 different states. That same year, 1,303 serum samples were tested for antibodies to MAP by ELISA, finding 69 (4.9%) were positive. States having the most goats testing positive for Johne's disease overall were Missouri, California, Indiana, Minnesota, Ohio, and Oregon. While these samples were submitted for the purpose of diagnosing Johne's disease, not a random survey, the results do indicate that Johne's disease is common among goats in the U.S. Many of the goats tested were pets such as Nigerian Dwarf goats and Pygmy Goats.

Sources of Infection

MAP is an obligate animal pathogen. This means that the only place MAP can multiply in nature is inside the goat. Most accurately, it is inside cells that are part of the animal's immune system called macrophages. When MAP leaves an animal, for example in the feces, it can survive at low numbers for a long time (up to a year) in environments such as soil and water, but it cannot multiply there. Consequently, the primary source of infection is a MAP-infected animal and its manure, and the resultant contaminated environment.

As MAP infection progresses in an animal, the number of bacteria being excreted steadily increases. Goat manure contaminates barns, pastures or fields into streams, ponds and groundwater. This means that the environmental burden of MAP steadily increases unless the source (MAP-infected goat) is removed from the herd.

Transmission of Infection

Most MAP transmission occurs from adult infected goats to young kids through the fecal-oral route. The organism is swallowed in manure-contaminated milk, water or feed; sometimes manure is swallowed directly. MAP is also shed directly into the milk and colostrum of infected dams in later stages of infection, providing another route of exposure for susceptible young animals. Pooling of milk from multiple nannies to feed multiple kids is a significant risk.

Goats are coprophagic (eat feces) and they also like to climb, getting into and defecating in feeders and waterers. These behaviors obviously facilitate spread of the MAP infection, making it extra important to detect and remove animals shedding MAP in their feces.

Another transmission route is in utero: a fetus may acquire the infection from its infected dam even before it hits the ground. There is no research in goats helping us to know the rate at which this occurs but if we borrow from cattle studies we can roughly guess that it happens 15-40% of the time, depending on how far advanced is the MAP infection in the dam.

There is no transmission risk of nose-to-nose fence line contact, through sneezed aerosols, or via artificial insemination or natural breeding. The most likely way MAP initially enters a herd is when a silently infected animal is purchased and introduced.

These transmission factors form the basis of MAP infection control: protect the future of your herd (the young kids) by making sure they are not exposed to potentially contaminated adult manure from potentially infected animals. The extent and duration of exposure to contaminated manure and milk from infected adult animals directly affects the likelihood of sufficient MAP exposure to cause a new case of infection. Clean, dry, birthing environments and housing of young kids away from the adult herd limits the possibility of infection transmission. Conversely, dirty maternity pens or lots and fecal contamination of feed and water supplies will promote spread of the infection.

PATHOLOGY

Knowledge Gaps

Much has been accomplished in understanding the interplay of MAP and its host in recent years using as new tools the genome of MAP and the many cellular, proteomic and reagents and assays recently developed. Most MAP research is done on cattle and studies specifically on goats are very few. Thus, sometime we have to make assumptions about how MAP infection progresses in goats. These are some of the core questions about MAP pathology and pathogenesis that remain unanswered:

What is the minimally infective dose by animal species and age?

Why are young ruminants much more susceptible than adult ruminants?

What proportion of infected animals recover (clear the infection), if any?

What factors affect MAP virulence?

Are all MAP strains equally virulent?

What are the innate immunologic responses to MAP infection in young animals?

Continued progress in comprehending host-MAP and MAP-environment dynamics is being made. This page summarizes our current knowledge about the impact of a MAP infection on goats.

Infection

The target for MAP is the goat's gastrointestinal (GI) tract. The end of the small intestine, called the ileum, is the primary site for infection. Goats swallow the organism (via MAP-contaminated milk, water or feed) which then invades through the intestinal wall localizing in specialized tissues called Peyer's patches where it is taken up by the goat's immune cells (macrophages). This microscopic infection of macrophages in the small intestinal persists for years without triggering any systemic response from the animal's immune system, meaning that the animal is MAP-infected but isn't sick and isn't responding to the infection in any detectable way. At some point MAP spreads beyond the intestinal tract to lymph nodes flanking the GI tract (the mesenteric nodes). Later, for reasons not yet understood, the infection spreads throughout the goat at which point it can infect the unborn fetus. Clinical symptoms of Johne's disease usually begin to appear at this point. When goats become thin due to the damage caused to their intestinal tract by MAP and the resulting inflammation, diagnostic tests are almost invariably positive, whether testing by fecal culture, fecal PCR, or by ELISA for serum antibodies.

Throughout this long sub-clinical phase (roughly 2-10 years) when the healthy although MAP-infected, it is capable of transmitting the infection by shedding MAP in milk and manure. Initially, the MAP bacteria may be only intermittently detectable but as the infection progresses, animals become steady shedders of MAP in feces.

Inflammation

MAP employs a variety of strategies to make sure it gets to where it can persist, and more importantly replicate, in an animal. One of these strategies is to use a component of the animal's defense system for its own home. After having been swallowed and travelled down the small intestine, MAP is picked up by host cells (with the intention of killing the foreign invader) and is carried inside these cells into the wall of the GI tract landing in the Peyer's patches (lymphoid tissue similar to tonsils) and sets up residence within macrophages (white blood cells) localized in that region and design to fight infection. Through complex mechanisms, MAP somehow turns off the bacteria-fighting mechanisms of the macrophages and instead creates a hospitable environment for itself. Far from alarming the immune system, this invasion seems to be ignored by the goat's immune system or actively suppressed by MAP. No detectable pathology appears at this phase. The photo on the right shows the red-stained MAP bacteria surrounded by a limited number of blue-colored goat inflammatory cells.

At some point, the goat mounts a stronger inflammatory response. Inflammation is defined as a protective tissue response to injury or destruction of tissues, which serves to destroy, dilute, or wall off both the injurious agent and the injured tissues. Through production of various cytokines (gamma interferon being an important one) the body begins this so called "cell-mediated" immune response. More macrophages are recruited to the site of infection and the result is called granulomatous inflammation, an aggregate of living, dying and dead MAP plus white blood cells from the goat, namely macrophages and lymphocytes. This lesion progresses, but remains a localized battle in the gastrointestinal tract – – – for a while.

As more MAP enter and replicate in the macrophages however, and as more cells are recruited to fight them, the lesion expands. This granulomatous inflammation spreads, and the infected macrophage may then leave the gastrointestinal tract inside macrophages and be filtered out by the neighboring lymph nodes. Eventually, the goat loses it's battle with MAP and the bacteria spread through the blood to other organ systems.

In many respects the lesion resembles that of leprosy (i.e., lesions produced by infection with Mycobacterium leprae) more than that of tuberculosis (caused by Mycobacterium tuberculosis). As in leprosy, some lesions may have numerous acid-fast (red) bacteria. Such lesions as shown on the left are referred to as lepromatous or multi-bacillary. When very few MAP bacteria are seen it is called paucibacillary.

This uncontrolled spreading inflammation is the primary reason Johne's disease is fatal. The GI tract is severely damaged and is no longer capable of absorbing nutrition. So, the goat keeps eating well but steadily loses weight. MAP-infected goats sometimes have pasty feces, but it is not like the diarrhea seen in cattle.

In this latter stage of infection the intestine is somewhat thickened. More noticeable, however, are the enlarged lymph nodes adjacent to the GI tract. When cut in half, these swollen lymph nodes often reveal pale whitish areas that are collections of white blood cells (macrophages and T-lymphocytes) that have come to the MAP infection site to try and deal with the invading microbe. Normally the lymph node with be a uniform, liver-like color. The white areas are all sites of granulomatous inflammation. Sometimes these lesions will be calcified.

DIAGNOSIS

Testing for Johne's disease can seem complicated. We try here to give some general guidelines but every herd and every owner is different. We urge goat owners to work with their local veterinarian to find the right testing program for their herd.

First, let's address: Why test?

There are a number of reasons to test your animals for Johne's disease? In fact, I think every single goat herd should be tested. The best test for your goats depends on how the testing information will help you accomplish your goals. Diagnostic testing will help you to:

-Determine whether or not MAP-infected goats are present in your herd.

-Estimate the extent of MAP infection (prevalence) in your herd.

-Control MAP (decreased the prevalence) in an infected herd.

-Eradicate MAP from an infected herd – yes this is possible.

-Monitor your herd to insure it remains MAP-free (surveillance).

-Make a diagnosis for a sick animal, does it have Johne's disease.

-Meet a pre-purchase or pre-shipping testing requirement.

Once your veterinarian knows the reason(s) you want to test for Johne's disease, s/he can tailor a diagnostic plan that best meets your needs. This plan should outline the type of test, when to test, which animals to focus on, the cost of testing, how to interpret the results and what actions to take based on test results. Lots to consider! That's why professional help is advised.

For all of these diagnostic assays, be sure you and your veterinarian use a laboratory that has voluntarily taken (and passed!) an annual "check test" to confirm that their test kits and methods are valid.

Which Test Should I Use?

Organism-based tests.

There are two types of these assays: (1) PCR, which looks for the MAP genetic material from living or dead MAP, and (2) Culture, which isolates the living MAP organism itself from manure or tissue. PCR has largely replaced culture as the most accurate and affordable way to detect MAP.

1. PCR, also called direct PCR: This test is used on manure or tissue samples. The assay looks for MAP's genetic material instead of the living organism. Most labs provide a result in 7-10 days. The accuracy of culture and PCR are comparable and PCR is faster and less expensive.

Pooling of samples (5 per pool with pooling done by the testing lab) is the most accurate and affordable way to test your herd if you have a low or zero prevalence of infection. Request "Direct PCR (pooled)" on the submission form and list your animals on the submission form in the order by decreasing age (oldest first). This insures that the pools are created with the maximum likelihood of having the MAP-infected animals in the same pool.

Paraffin block PCR is a special form of PCR that only some laboratories perform. When tissues are collected at a necropsy, they are embedded in paraffin to be thin-sectioned, stained and examined microscopically. The pathologist is looking for characteristic inflammation in tissues and for MAP itself within cells. Your veterinarian can request a PCR test on a portion of the paraffin block.

Most labs also use a PCR to confirm that the organism isolated during culture is actually MAP and not one of its closely-related mycobacterial cousins that live in soil and water.

2. Culture: A sample submitted for culture is monitored for eight weeks or longer because MAP is a very slow growing organism. If the sample is heavily contaminated with MAP, a positive result may be had sooner, but it can take two months of incubation or more until the lab feels confident that no MAP organisms are present in the sample tested and can report a "culture negative" result. Culture is effective for testing any animal species and can be done on manure or tissue samples.

Pooling of manure samples reduces the cost of a whole herd test. Individual samples are collected, then the laboratory mixes the samples (usually 5 samples per pool, 1 pool per culture). If a pool is test positive, the 5 animals contributing to the pool are then tested individually to find which one(s) are shedding MAP. Older culture methods used solid culture media that were examined visually on a weekly basis for at least 3 months. Newer methods use liquid culture media and automated instruments to "read" the culture for up to 8 weeks.

3. Antibody (blood or milk) tests: These assays look for antibody produced by a MAP-infected animal using a technology called ELISA (enzyme-linked immunosorbent assay). ELISAs detect antibodies in serum and the assay is performed in microtiter plates. To perform this test, 2-3 milliliters of blood is collected from an adult animal. The fluid part of blood samples (serum) is tested for anti-MAP antibody. The amount of antibody found (if any) is compared with positive and negative controls, and an interpretation is then assigned to the ELISA result. These numeric results (the actual amount of antibody) are useful: the higher the test result, the greater the certainty that the animal is infected and shedding MAP.

The ELISA is designed for testing large numbers of samples quickly (a few days) and this makes it a low-cost test. A number of ELISA kits have been approved for use in milk from individual cows as well as blood samples.

ELISAs are popular because they are fast and the least expensive of the available tests for Johne's disease. However, they are designed for rapid, low-cost screening of large numbers of animals. ELISAs are less sensitive than MAP-detection assays (PCR and culture), typically being positive in roughly 30%-50% of the animals that MAP-detection assays will identify as MAP-infected. This is generally because antibody production occurs later in the course of a MAP infection, months or even years after an infected animal has been passing MAP bacteria in its feces.

ELISAs are >99% specific. This means that there is a <1% chance that a positive ELISA is a false-positive. Typically, this means that animals found ELISA-positive should have a confirmatory test done by PCR or culture.

There are many nuances to testing recommendations that cannot be explained here. The best advice is to discuss your specific needs for Johne's disease testing with your veterinarian.

There are a variety of ways to test your herd that will give you the information that you need. The best testing program can be developed by you and your veterinarian since you know your operation best: its goals, resources, other animal health issues, etc.

Here are some approaches that have worked well for other goat herd owners:

Question: Is MAP present in my herd?

Recommendation: Use targeted testing (ELISA or fecal PCR) of oldest or thinnest goats (10% or more of the herd).

Question: How many of my goats are infected?

Recommendation: A good estimate can be made by blood testing (ELISA) all goats after their second kidding or older.

Question: What test should I use to control MAP in my infected herd?

Recommendation: For commercial herds, blood testing (ELISA) on all goats after their second kidding or older is economical. See the Control section for further information.

Question: What test should I use to eradicate Johne's disease in my herd?

Recommendation: Breeders must work to eradicate MAP. Pooled fecal culture is an economic way to eliminate the infection in the herd.

Question: Does this skinny goat have Johne's disease?

Recommendation: After ruling our parasites, fecal PCR is best. Even better is necropsy (autopsy) where a pathologist examines the tissues and a microbiologist tried to detect MAP in tissues by PCR.

Question: What test do I need to sell and transport this goat?

Recommendation: This is determined by the agency managing the shipment or the receiving owner. If I were advising the buyer, I would recommend a test on source herd (all adults or at least 30 head) by fecal PCR (pooling acceptable). Buying young animals from such a herd fairly safe. If the herd owner could show you 3 years of whole-herd negative test results that would be even better.

CONTROL

Johne's disease can be controlled in goat herds. Find the infection as soon as possible, keep good records, and make sure the goat kids have no chance to swallow MAP-contaminated milk, colostrum, hay or water.

Control is easy, it just takes time.

Johne's disease can be controlled and even completely eliminated from infected herds. However, it takes a thorough understanding of the disease by animal owners, consultation with a veterinarian, and requires use of one or more of the available goat diagnostic tests. Half-hearted attempts to control Johne's disease will generally fail. Control of Johne's disease also takes time and a strong commitment to management practices focused on keeping young animals away from contaminated manure, milk, water, etc. A typical herd clean-up program may take a number of years. Faster clean-up programs are possible, but they are usually more expensive.

The basics of control are simple: new infections must be prevented, and animals with the infection must be identified and removed from the herd.

#1 Prevent new infections in goat kids

Manure management

The most MAP bacteria excreted by infected goats are in the feces (manure). Farm sanitation and control over where manure goes on a farm are critical to control of Johne's disease. Because of the susceptibility of kids to MAP infection, it is important to keep them well away from goat pellets that may harbor the infection at least for the first 6 months of life, the "window" of maximum susceptibility.

Kids should be born in a clean dry environment with minimal fecal contamination. Individual kidding pens are optimal but if not feasible, you may establish test-negative and test-positive pens. To do this, you would test all your goats a few months prior to kidding season to have this test information available at the right time. Then try to establish an area outside the pen free of adult manure contamination ("the safe zone") for kids from test-negative does.

Manure contamination of water supplies, particularly ponds or streams that kids can drink from, must be avoided to limit spread of the infection. If you use water troughs, when cleaning them remove the sediment at the bottom and dump it away from where animals might graze – MAP apparently survives for a long time in this wet goo.

Pasture contamination with MAP is also important as a means of infection transmission, but it is less important than other modes of transmission and far more difficult to control. For the very concerned, you can till contaminated pastures and wait for time and environmental conditions (repeated changes in temperature, minimize shaded soil by cutting grass/crops/ shrubs) to kill off MAP on fields. While a majority of the organisms die within three months, a small population can remain for up to a year. Put off stocking contaminated pasture with young animals as long as feasible.

Milk and colostrum management

Many animals infected with MAP will excrete the bacterium in their milk. This happens most often in goats showing clinical signs of Johne's disease, but also occurs in infected animals that appear healthy.

A safe and effective alternative to milk replacers is to pasteurize milk on the farm. A recent study saw no difference in the number of new cases of Johne's disease arising in dairy herds (cattle) between those that pasteurized and those that used milk replacer (Recommended protocols: 145°F (63°C) for 30 minutes for batch pasteurization, or 162°F (72°C) for 15 seconds for flash pasteurization. The milk should be stirred or otherwise in motion to ensure even heat distribution.) Pasteurization kills virtually all MAP that may contaminate raw milk as well as other viral and bacterial agents that could affect the health of dairy doe replacements.

Colostrum, the antibody-rich milk produced by goats in the first few days after giving birth, also can contain MAP. Because colostrum is critical to the health and survival of newborns, feeding colostrum must be done. Many farms do not collect and store colostrum sufficiently carefully to minimize contamination if they choose to bottle-feed kids. However, the risk of transmitting MAP infections in colostrum can be minimized by following these three simple rules:

Use colostrum from Johne's test-negative animals only.
Do not pool colostrum from multiple animals.
Thoroughly wash and dry the udder and teats before collection of aseptic collection of colostrum into clean containers.
Pasteurization of colostrum is technically a fourth alternative. However, the thick viscous nature of colostrum makes it very difficult to pasteurize and so for practical reasons it is not advised.

#2 Identify and remove infected adult goats

Test-and-cull program

The majority of MAP infections in a herd are "invisible". Goats with clinical signs of Johne's disease (diarrhea and weight loss) are only a small fraction of the infected goats in a herd. The infection has the ability to silently spread from goats to kids long before signs of illness in infected animals are evident. For this reason, laboratory tests are important to determine which goats are infected. Test-positive goats are generally those most likely to be infectious (excreting MAP in milk and manure) and so they should be removed, or at least isolated from, the herd.

Referred to as a test-and-cull program, this practice is essential to successful control of Johne's disease in herds in a reasonable period of time. Clearly, there are situations where alternatives must be considered: testing and culling of all test-positive animals is not necessarily always required. For instance, some control programs retain goats with low or medium-level ELISA results to generate milk income but these goats are clearly tagged and strictly managed since they are almost certainly shedding MAP on the premises in their manure as well as in their milk. Farmers report that knowing the Johne's test status of a goat affects when the goat is culled if she develops any other health problems. Decisions on how best to implement testing in a Johne's control program should be made in consultation with your veterinarian. For details about available laboratory tests for Johne's disease, see the goat diagnosis section of this web site.

Disinfection

MAP is resistant to most disinfectants; washable tools, troughs, and feed dishes may be treated as directed on the bottle with a disinfectant labeled as "tuberculocidal". Since organic material deactivates the disinfectant, items should thoroughly cleaned with soap and water, rinsed and dried before the disinfectant is applied. Tuberculocidal disinfectants usually contain strong chemical compounds and should be used carefully. The instructions provided on the label for proper use and safe handling should be followed precisely.

Vaccination

No Johne's disease vaccines are available for goats in the United States. In Europe and Australia a vaccine (Gudair) is used in small ruminants and has been found to decrease the frequency of goats that develop clinical Johne's disease and reduces the amount of shedding of MAP. It does not appear to change the rate that goats get infected.

Considerable research is ongoing to devise better and safer vaccines for Johne's disease.

PREVENTION

Prevention is the most cost-effective way to manage Johne's disease.

It is far less expensive to block introducing Johne's disease into a herd than it is to control or eradicate the infection once it creeps in and invisibly starts to spread.

It is imperative that you know both the test status of any goat you plan to buy or lease AND the test status of the herd of origin. Herd history and test status are particularly vital if you are buying young animals as they are too young to be reliably tested for a MAP infection.

General principles

- Buy as few goats as possible from as few different herds as possible.
- Always insist on seeing the lab test results from the goat seller. Do not simply accept their word.
- Buy from test-negative herds: the more negative annual tests the lower your risk.
- Buy adult goats that have a long history of negative annual tests.
- Buy goat kids born to test-negative nannies.
- Rely more on fecal PCR test results than on ELISA results on blood samples.

Principles put into practice

Listed here are specific strategies to limit your risk for introducing MAP to your herd. This list starts with #1, the very best option, and goes down to the most risky practice.
1.Prove your herd is not MAP-infected (see the certification page) and then keep a totally closed herd: no live animals come onto your property.
2.Buy as few animals as possible and only buy them from herds that have at least 3 negative whole herd tests done using the fecal PCR (on individual or pooled samples). Ask to see the lab reports.
3.Buy as few animals as possible and buy them from herds that have at least 1 negative whole herd test done using the fecal PCR (on individual or pooled samples). Ask to see the lab reports.
4.Buy as few animals as possible and buy them from herds that have at least 1 negative whole herd test done using the ELISA on blood samples (on individual or pooled samples). Ask to see the lab reports.
5.Buy as few animals as possible and buy goats born to a test-negative nanny: ideally tested by both fecal PCR and ELISA.
6.Buy from reputable breeders buying from as few different herds as possible and from breeder who are well-informed about Johne's disease.

Risky business!

If you for some reason cannot follow these guidelines you must assume you will buy MAP-infected goats. So, you must quarantine them and test these goats by both fecal PCR and ELISA (blood test) before adding them to your herd. And, you should have strong MAP control measures in place to limit the spread of MAP once it gets into your herd, because it probably will.

CERTIFICATION

Definition

Herd certification is loosely defined on this web page as any official program designed to classify herds according to the probability that individual goats in those herds are infected with Mycobacterium avium subspecies paratuberculosis (MAP).

Goal

The primary aim of certification programs is two-fold:

- Provide a simple system to communicate to goat buyers the risk of buying a MAP-infected animal.
- Stop the spread of this infection to non-infected herds. Herd certification is the foundation of paratuberculosis prevention.

Basic Concepts

Johne's disease should be considered a herd health problem, not just an individual animal disease. Certification programs apply laboratory tests to classify herds by levels of infection ranging from not infected at all to very likely infected. Often such programs have regulations that participating herd owners must follow regarding the sources for replacement cattle. These rules are designed to help herd owners avoid bringing MAP-infected goats into their herds without compromising their ability to do business.

Diseases and Testing Wrap-Up

Now that we have learned about CAE, CL and Johnes, it is time to dive into the wrap up portion of the Diseases and Testing series we have been covering over the course of the last 3 MGR newsletters. The topic of diseases and testing can be the cause of some heated debates and opinions will vary on the need to test and the importance of it. I hope you will keep an open mind as we wrap up this series. I also hope that if you do not currently test your herd, this series will have opened your mind to consider the importance of doing so. A lot of what you will read in this wrap up comes from my own personal knowledge and beliefs about this topic. Anything said in what is to follow should in no way be taken personal. It comes from a desire to educate you. It is my sincere hope that you will leave this newsletter with a new found understanding of the diseases and also a desire to begin your own herd health management program, for the longevity of the breed, and the protection of your herd and everyone else's.

Why is testing important? I believe it is one of the most important herd management tools that you can implement. I also believe it is one of the most responsible and reputable things a breeder can do. Why? I believe the health and longevity of the myotonic breed, your own herd, as well as others herds' depends on us to test our herds! We all love our goats and this breed, otherwise we wouldn't own them, right? I believe that all herds are equally important when it comes to testing. This includes pet herds, breeding herds, show herds and commercial herds. As a breeder who is fully invested in testing my herd, I want the myotonic breed to continue to prosper and be around for generations to come! I want my herd to grow and prosper because the health of my herd is very important. I also want to protect the financial and emotional investment that my prospective buyers have placed into their herds' by providing them with myotonics that they can feel confident in buying.

In the last several years, I have seen friends and other breeders deal with CAE, CL or Johnes. I have seen the heartfelt devastation you have felt after receiving your test results, or buying an infected goat (unknown to you at the time). I have seen first class handling of the diseases by informing previous buyers of your goats and taking measures to attempt to eradicate the disease from your herd. On the flip side, I have also seen the deceitful handling of the diseases by breeders who continued to sell even though a disease problem in the herd was known. I have seen the devastation people have faced when goats they purchased came up positive from those infected herds.

Why should I test? If the health and longevity of the myotonic breed, your herd as well as others herds' isn't enough to convince you, here are some other things to consider.
1) I have a goat who has developed an external abscess in a location common for CL.
2) I have a goat who is excessively thin, cannot gain weight and is unthrifty.
3) I have had issues with goats dying on my property for unknown reasons.
4) My goat purchases have taken place at the local sale barn.
5) I raise other ruminant species and I have had disease problems with them in the past that can be common among goats.
6) I have purchased goats from herds that do not test and I am skeptical if I have just introduced a diseased animal into my herd.
7) I have goats from a non-tested herd. They have now started testing and have received positive test results.

The reasons listed above are just a few things to get you thinking. That list is not all inclusive, but covers the major of the reasons people decide to test.

Which diseases should I test for? I personally believe that CAE, CL and Johnes are all equally as important and therefore you should test for all 3. CL and Johnes seem to be the more common disease people deal with of the 3, but that shouldn't discount the importance of CAE. It is up to you to decide which diseases you test for. It is my hope that after learning about them, you would test your herd for all 3.

How do I test? If you are testing for the sake of establishing a benchmark condition of the disease status in your herd, the most common method is pulling a serum (blood) sample for testing by the Elisa test. The Elisa test will test for the presence of the CAE, CL and Johnes antibodies in the serum sample. If you are testing because you "think" or "know" you have a diseased goat(s), there are a couple routes to take with the tests. The most common ways to test are: For CL, you will either take a serum sample for the Elisa test or test the contents of the abscess (culture). For CAE, you will take a serum sample for the Elisa test. For Johnes, you will either take a serum sample for the Elisa test, or a fecal sample for the Direct PCR.

How do I collect the samples? There a videos on youtube that will walk you through pulling blood and collecting fecal samples. A lot of people collect the samples themselves or have fellow breeder friends that live close bye to help them do this. Some people have their vet collect the samples for them. Blood should be collected and submitted using the red top test tubes or the serum separator tubes. Collection of the contents of an abscess is a bit more tricky. In the unfortunate event the abscess is already draining, collection is easier. If the abscess is not, proceed with caution. Once the abscess is punctured, transmission will spread. Collection can be done using a syringe and then transferred to a red top test tube. I place the fecal samples in used pill containers. Be sure to check with your lab (if submitting yourself) to see how they prefer to receive the samples.

How much do I need for a sample? Whatever lab you choose to complete your tests should specify how much they require. In general, 2-3 ml of serum is sufficient. At least 15 grams (approx. 3 teaspoons) is sufficient for fecal samples.

Where do I send my samples? Whichever lab you choose, make sure they are an accredited lab for performing the tests you are wanting done. Many states have state run labs that will conduct the tests. I personally use the Washington Animal Disease Diagnostic Lab (WADDL) and the University of Wisconsin-Madison, both of which are accredited. Some labs will only accept samples collected and sent from a veterinarian. However, some labs will accept samples submitted by the owner.

When should I send my samples? Whichever lab you choose, check their testing schedule to see which days of the week they will perform the tests. Doing so will help you decide what day you need to send your samples so they arrive fresh and are not sitting in the post office over the weekend. After you get your samples pulled, keep them refrigerated until it is time to ship them.

How do I ship my samples? I like to send mine via UPS or FedEx. I used to send mine via USPS until a few years ago when I had my package lost in transit (even with tracking). I had to pull new samples and re-submit them, only to have the original samples arrive the day after my new samples arrived at the lab. I have a small foam cooler that I send the samples in and place cold packs in the foam cooler. The cooler is placed in a cardboard box. Make sure to get shipping with tracking!

What paperwork is needed? Whichever lab you choose will have forms that need to be completed and submitted with the sample. Make sure that your paperwork matches your samples (make sure your samples are labeled and the labels match the corresponding sample number on your paperwork).

How do I get the test results? If your veterinarian submitted the samples for you, most likely the lab will send the results to your vet who will then contact you when they receive them. If you use a lab that allows you to submit the samples, then the test results will go directly to you. WADDL emails me the test results directly. They also have an online portal for your account that you can access the results. The lab should have a schedule of when the results will be ready based upon when the sample(s) is received and when the tests are performed.

How much does testing cost? This will depend on what lab you choose, if you take the samples yourself or have your vet do it, how big your herd is and how many goats you are testing. I test our entire herd (anyone 1 year of age or older) all at once. In general, I am submitting about 25 samples at a time (1 serum sample can be used to complete all 3 Elisa tests) and am usually in the neighborhood of $1000 each year if I am just submitting serum samples. If you have to re-test any goats, of course you will be looking at additional costs.

At what age should I test my goats? All of the information that I have read over the years says you should wait to test until the animal is at least 1 year of age. Doing so will give you a more accurate test result as opposed to testing an animal younger than 1 year of age.

How often should I test? How many should I test? I test our entire herd on an annual basis. I do not break them down into groups. All goats are collected, submitted and tested at the same time. Depending on the size of your herd and cost, you may decide to break the herd into a spring group and a fall group for example. Anytime I purchase new goats, they are quarantined and tested (if they are a year or older). If you suspect you have a problem or a test result reveals you do, more frequent testing will be needed.

I have received my test results, now what? First, you need to understand the pathology of the diseases, especially Johnes, to get the full grasp of what the test results are telling you. Remember, your testing is just a snap shot in time. You also need to know that there is the possibility of false positives and also false negatives. Testing is a useful tool to determine the disease status of your herd at a given time. It does not always guarantee the future disease status of your herd. It is good to know the test threshold for the test your lab is using. This will let you know their cutoff from negative to suspect to positive. If you get all “negative” results, you should feel comfortable that at this given time, your herd is negative. If you were to get a “suspect” result, you should investigate further. This means the animal is not “negative” and not “positive”, but is approaching the positive side of the spectrum. What should you do? If it were me, I would immediately remove the goat from the herd and isolate it. I would consult with my veterinarian and the lab that conducted the test. I would conduct additional tests to determine if the first test generated a “false” positive or if this goat was trending towards the positive side of the spectrum. Depending on what the additional testing results showed, would determine my action plan from there forward. I would use this same process if I were to receive a “positive” result.

How do I interpret the test results? The answer to this will kind of overlap my answer to the previous question. If you receive negative results, you should feel comfortable that at this given time, your herd is negative for the disease(s) you tested for. I see the word “clean” used a lot when describing the disease status of a herd. I personally believe we should refrain from saying we have “clean tested herds.” I think of the word “clean” in terms of a freshly washed eating utensil that is free of germs and ready to be used again. In using the term “clean tested herd”, I believe we are saying we guarantee our herd is free from disease and that any goats purchased from us will never have a disease problem because our herd is “clean.” Especially with the pathology of Johnes, none of us are guaranteed our herd is Johnes “clean.” I believe the best and most honest term to use when describing the disease status of our herd is “health test negative.” Because after all, if you test your herd and have received negative results, your herd is “health test negative.”

How long should I test? This is entirely up to you. I personally will continue to test my herd as long as we have goats. With Johnes being capable of staying “dormant” within its host, a one and done test on your goat(s) isn’t enough. It is possible that the MAP bacteria that is Johnes will lie dormant for the entire lifespan of the infected goat and never become active. It can be triggered and become active at any given moment. Thus why testing is a snap shot in time.

Is testing a marketing tool? Don't begin testing for the purpose of having a way to market your goats and increase your sales. Go into it because of your desire to promote the longevity of the myotonic breed and to protect your herd as well as others herds'. It is a heard health management tool first and foremost. Does it become a marketing tool? It does because there are buyers out there who are looking to purchase goats only from tested herds. They are looking to herds who test for added security and peace of mind with their purchases. Just because someone doesn't test does not mean they have diseased goats or that their goats are of lesser quality than someone who does test!

How do I safeguard/protect my herd?

1)First of all, don't let your goats live in a bubble. If you want to show your myotonics for example, don't be afraid to take them to shows. It is true that anytime your goats leave your property, you are opening yourself up to bringing something un-wanted back to your herd. As added precautions, I can frequently be seen hanging tarps up on my goat pens at shows. I also disinfect my goats' hooves and wipe them off either before loading them on the trailer to head home or before unloading them upon arriving home.

2)Don't refrain from buying new additions to your herd because of the "what if's" out there. But me mindful and be cautious. There are quite a few myotonic breeders out there who test, but the grand majority of herds do not. I believe it is ok to ask a seller if they test and what the disease status is of their herd if they do test. I also believe it is ok to ask to see copies of their herd test results. I willingly provide all buyers with a copy of our most recent test results. If asked, I would gladly provide copies of entire herd test results from years past as well.

3)Implement biosecurity measures for visitors. Anyone who comes to our farm is provided with boot covers to wear over their shoes before entering our pasture. This includes our veterinarians and staff and buyers coming to look at or pick up a goat.

4)Only buy from tested herds. I will leave that up to you to decide if that is something you want to practice or not.

5)What about stud service to cover my does? This can be done safely in your driveway. However, I would think twice about allowing a buck from another herd spend any length of time in my pastures, dry lots or pens.

6)Someone wants to use my buck to breed their does? Again, it can be done safely in a driveway setting. However, I would think twice about letting my buck stay on someone else's property to breed their does.

7)Any equipment I use at shows (hay feeders, water buckets, etc) are just that. My show equipment. I have a completely different set of hay feeders, water buckets, etc that are used for the day to day operations of our herd. I disinfect all equipment before using it again at the next show. All show equipment stays in my trailer when not being used at shows.

8)We have separate pairs of shoes/boots that are worn when we go to shows. They are not the same pairs that are worn for doing chores and entering our pastures/pens/dry lots. Foot traffic is an easy way to spread disease!

9)Stay away from sale barns for your purchases. I am not discounting that there are reputable sale barns out there and that you can find a diamond in the rough at one. There is a huge world out there of myotonic breeders that you can purchase from regardless of what you are looking to purchase!

10)Be mindful when going to someone else's farm/property. As a precaution, you could wear boot covers without being asked. If you do so without being asked to, be tactful with your explanation as to why you are wearing them. The last thing you want to do is offend anyone! I take this approach: I hope you don't mind that I am wearing boot covers. I practice biosecurity and am looking out for the health of my animals as well as yours. I think if this approach is taken, you will be welcomed openly and respected for your effort to protect that persons animals!

11)Not only is foot traffic an easy way to spread disease but so is tire traffic. Keep that in mind when borrowing someone else's equipment who has been through their lots (if they also raise ruminants).

What else? This list of ways to safeguard/protect your herd is not meant to be all inclusive. You may have your own ways to accomplish this. I hope this list will give you a good overview of ways you can do so!

A few added notes about the diseases. They are out there, so be careful! When we started testing, I was just as engaged in social media as I am now. The amount of talk about diseases and testing has grown and grown! Has grown because more people are becoming interested in the topic or if the prevalence of diseases are becoming more widespread? I think it is a combination of both. Testing for the diseases by means of the Elisa test is the quickest and least expensive way to test. However, there is the potential for inaccurate results. If you choose to go this route to establish a benchmark condition for your herd and continue to test this way on an annual basis and continue to get consistently negative results, you will build a confidence level that your herd is negative and a low risk for the diseases. However, as I referred to Johnes earlier, I believe it is like carbon monoxide poisoning. It can be a "silent killer." With its capability to lie dormant, it can get you when you least expect it and most times when it is too late. The Elisa test as we learned, detects for the presence of the disease antibodies in the serum. The PCR test as we learned, detects amplified DNA of Map (the bacteria). When MAP becomes active in its host, it will be shed in its feces. Thus, you would get a positive fecal test result. However, MAP can be shed intermittently and can be shed at such low levels that the PCR test would not detect it. You will not get a true "positive" by the Elisa test unless the animal is shedding in its feces or has previously been shedding. If you get a true "positive" Elisa test result for Johnes, you could also get a true "positive" PCR test result and vice versa. Johnes is complicated to say the least! It is just as complicated for us to comprehend as it is for the researchers and veterinarians who study it! I would just strongly encourage you to learn all you can about the diseases and continue to learn about them as time goes on. I believe that doing so will help you on your testing journey.

Our Road to Testing

Anyone that has ever chatted with me about the diseases and testing topic knows how important I believe it is and knows how serious I am about it. I have invested a lot of time over the years learning all I can about the diseases and have gained a great grasp of them and a tremendous amount of knowledge. I am about to share with you my personal story about the road we took to testing. If what you have learned so far in the series we have covered in the newsletters isn't enough to convince you it is important to test and the diseases are serious and can be devastating, my road to testing should. I want anyone who reads this to know that what I will share with you did not impact any of our buyers at any point in the past because of the measures we took at the time. The diseases we have discussed are real. They are out there. Don't let anyone tell you they are not and that it wont ever happen to you.

Between my wife and I, we have been raising myotonics for over 20 years. The herd we had was small and consisted of about 10 goats, made up of goats that my wife started with in 2000 and also goats we purchased together. The goats my wife started with were her pets, her babies. She loved everything about them. She loved their color, their personalities. She loved sitting with them on a daily basis. She loved raising kids out of them. She simply loved them beyond measure. In 2011 I was at a point that I wanted to become a more reputable breeder of myotonics. Through a mentor, I was introduced to the testing and disease topic. From what I learned, I felt it was important to test and by establishing a herd health management program (testing), I could start to work towards the goal of becoming a reputable breeder.

So, in 2011, we started testing our herd. Our herd was a mixture of middle age goats and everything in between.That year, we tested everyone for CL (serum). I started with CL because one of our state universities performed the tests for CL and I felt like it was an easy one to start with. We did not start with CL because we had a CL problem or suspected that we did. All 2011 results were negative. At the start of the new year, we purchased a 5 year old doe from a tested herd. We paired her with our buck when we got her home and she kidded that spring. We were in a new year and decided to continue our testing. This time we tested for all 3 of the diseases by serum- CL, CAE, and Johnes. Our entire herd of 10 was tested that spring. Our results showed all negative for CL and CAE. 9 of the 10 goats were negative for Johnes. The lone positive was the 5 year old doe we had purchased a few months ago. Her Elisa test result was 2.408. I immediately removed her and her less than 1 month old twins and isolated them from the rest of the herd. Thinking I surely received a false positive result, I re-tested her a couple weeks later. I sent 2 different serum samples to 2 different accredited labs. Both additional serum tests came back with a positive result. 1 lab provided me with their negative to positive threshold cut-off. Anything over .2555 was considered positive. Her result from this lab was 2.222. At the same time, I also sent a fecal sample to be run by the Direct PCR method. This result came back as "not detected." This meant that she was either not shedding MAP in her feces at the time of sampling or she was shedding MAP at such low levels that it could not be detected. With this in mind, I felt somewhat comfortable that she was a "low" risk to the rest of our herd, but I there was no way to know for sure if she had ever shedded MAP in her feces (since shedding can happen intermittently). However, I made the decision to euthanize her and her twin kids. This prompted me to develop a timeline and brainstorm just what was going on. Did she contract Johnes from one of our other goats? I felt this was not likely since she was an adult when we purchased her and since the remainder of our herd just tested negative. Did she come infected from where we purchased her? Remember, we purchased her from a tested herd and because of her age and how long she had resided there, I felt this was very unlikely. My conclusion was that she contracted Johnes from the herd where she was born, either contracting it from her dam or from other infected goats in the herd.

Why at 5 years of age did she test positive?Remember what we have learned about Johnes. It can lie dormant for a long time before it starts to become active within its host. Remember, the herd we purchased her from purchased her as a baby. She had literally lived her entire life up until our purchase in the herd that purchased her as a baby. What causes MAP to mysteriously become "active" in its host when it had previously lied dormant? In our instance, what caused this 5 year old, previously tested doe, to become test positive for Johnes? I believe that 2 things could have caused the MAP to wake from its dormancy and become active in our doe. First, at 5 years of age, she became stressed moving after the 7 hour trip to our place from the place she had lived almost her entire life. Second, she kidded just a couple weeks before we ran that first test on her. Would an Elisa or PCR test given us any different results had we tested her right when we got her home? Quite possibly but quite possibly not. There is no concrete timeline to follow from MAP dormancy to activity. I was scared and unsure at the time I received those positive results. I was also saddened I was forced with a decision to make and at the loss of a beautiful doe and her twins. I would be lying if I said that no tears were shed during this process! We felt that based upon her PCR test result that she had not infected the rest of our herd and contaminated the place our herd resided.

As an added precaution, that fall our entire herd was tested again for Johnes by the Elisa test. All were negative. What a relief considering what we had already been through!

Spring of 2013 we purchased a couple young goats who were not old enough to be bred yet and kept them where we live (the rest of our goats were at the farm where my wife had grown up and had always had them when she started with goats in 2000). I implemented the biosecurity measure of wearing different boots at each place for choring. That fall, all of the original herd was tested again for CL, CAE and Johnes. All were negative for CL and CAE by the Elisa test. 4 goats (of the original herd) tested positive for Johnes by the Elisa test. 2 of them were 3-4+ years old, 1 was 6-7 years old and 1 was a yearling (her dam was one of the 3 positives). Again, remember that much of this original group were the goats that my wife started with and how important they were to her! At this point I was beside myself. I was left wondering why we were dealing with this "problem" again! I thought we had gotten past the bump in the road we previously experienced! I weighed out the options. I could isolate these 4, test them again by serum and also test their feces to determine if they were shedding and spend more money in the additional tests and quite possibly get the same test results. I could euthanize these 4 goats that tested positive and continue to run the original herd plus the new goats that lived at our place. We could also "start over" and shut down the original herd. I still cant believe my wife went along with the option that I chose. The option I chose broke her heart. There is not a day that goes by that she doesn't think about the goats in that original herd. I chose to euthanize the 4 that tested positive. The goats that remained of the original herd (test negative) were loaded up and were taken to the slaughter auction. I again would be lying if I said no tears were shed when the 4 were euthanized or when I delivered the rest to the sale barn and watched them go through the auction ring. Dealing with this was a literal hell to put it mildly! I can not even begin to describe the true emotional and mental stress this whole process caused! You may ask, was testing even worth it then? Looking back, from my standpoint alone, it was worth it. Why? I did the best thing I knew how and felt was right to do and that was to eradicate the disease and not pass it onto someone else. I felt doing this and striving to protect the longevity of the myotonic breed was worth it. I also believe that had we not started testing and took the drastic measures that we did to eradicate it, our herd wouldn't be where it is today.

We were fortunate to purchase additional land where we live and start over with the couple goats we had purchased in the spring of 2013 that already resided here. We had a fresh start and a clean slate. The land we purchased included a few acres of grass (where no livestock had been for at least 50 years) and also active cropland that we converted to pasture. Since then, we have built our herd to what it is today. I am blessed every day when I look outside and see the amazing herd we were fortunate enough to build!

We have continued to test annually and have consistently received negative test results for CL, CAE and Johnes. Our testing program consists of testing by serum for the Elisa test and also spot checking for Johnes by the PCR method. You have learned that the PCR method is more reliable than the Elisa. So why do I continue to use the Elisa test method? We have many years now of consistent negative results with the Elisa method. We have a solid concrete foundation built. I am extremely confident in the herd that we have and the results we are given annually. Can I guarantee we wont ever have a goat test positive for Johnes in the future? Knowing what I do about the disease, I cant guarantee that we wont ever encounter it again. I pray that we never do but I do feel extremely confident we are running a herd that promotes the health and longevity of the myotonic breed and are providing our buyers with myotonics they can feel comfortable and confident in buying to protect the health of their herds. If we ever get a positive result in the future, I will practice the same management practices I did all those years ago. I will isolate the goat, re-test by the methods available and then determine the mode of action. I will say that if I am ever faced with positive results in the future, I will have to take a deep look into if I can emotionally cull and re-test and continue raising myotonics.

I love our herd deeply and have developed a herd that we are very proud of. I know that all of us feel the same way about our own herds. If you currently test or have tested in the past but have let it slip away, please continue to test! If you do not currently test, I am urging you to begin testing! If what you read over the course of the newsletter series didn’t convince you to test, I hope that my personal story was enough to convince you. I don’t want to see anyone go through the headache and heartbreak we went through all those years ago! I want the myotonic breed to continue to thrive and be around for generations to come. I also want all of you to have health test negative herds, regardless of what you are raising your myotonics for!

MGR Youth Spotlight

Jake & Wyatt Strahan

13-year-old Wyatt Strahan and his 10-year-old brother Jake have lived on a farm their whole lives, in West Warren, Utah, about 50 minutes north of the state capitol of Salt Lake City.

They started showing Myotonic goats in 2013, when Wyatt was 5 years old, and Jake wasn't even old enough to lead his own goat around. At Wyatt's very first show, the judge asked him what kind of treats his goat liked during the Pee Wee showmanship, and Wyatt very matter-of-factly answered, "Goat treats," as though there weren't any other kind to give a goat. They have progressed with their showmanship skills since then, of course, and are now answering questions like "What is the average body temperature of a goat?"

In addition to raising and showing goats, Wyatt competes in Ice Hockey, Baseball and Floorball (floor hockey). Jake plays guitar, competes in Archery, and plays Basketball. Needless to say, the family doesn't have much free time!

Both boys currently have youth numbers with the MGR, and both have multiple goats registered in their own names. Wyatt hopes to participate in FFA once he reaches high school, and both Wyatt and Jake plan to continue raising Myotonic goats.

Top: Jake Strahan; Bottom: Wyatt Strahan

Iowa's Premier Myotonic Classic - Osceola, IA

Show A: Judge Shelly Strahan
Show B: Judge Maurice Erwin
Show C: Judge Debbie Mullins
***Indicates verified MGR Point**

Show A- June 4, 2021

Junior Champion Doe

Frosty Acres Valentine	Danielle Frost

Reserve Junior Champion Doe

Mar-Bob Red Flare	Brittany Frey

Senior Champion Doe

Twin Creek Red Hot Flame	Benjamin & Sheena Schmidt

Reserve Senior Champion Doe

Fern Hill Prudence	Drew & Amy DeRiemacker

Grand Champion Doe

*Twin Creek Red Hot Flame	Benjamin & Sheena Schmidt

Reserve Grand Champion Doe

Fern Hill Prudence	Drew & Amy DeRiemacker

Junior Champion Buck

Frosty Acres Legend	Danielle Frost

Reserve Junior Champion Buck

Nine Acres Farm Somethin Bout a Truck	Nikki Thummel

Senior Champion Buck

Buck Creek Moonshine	Brian & Mackenzie Treadwell

Reserve Senior Champion Buck

Fern Hill Notorious	Sarah Oeltjenbruns

Grand Champion Buck

*Frosty Acres Legend	Danielle Frost

Reserve Grand Champion Buck

Buck Creek Moonshine	Brian & Mackenzie Treadwell

Junior Champion Platinum Wether

Frosty Acres Tanner	Danielle Frost

Reserve Junior Champion Platinum Wether

Heavenly Hill Farm Frankenstein	Mackenzie & Philip Jurek

Senior Champion Platinum Wether

Oeltjenbruns' Farms Patron	Sarah Oeltjenbruns

Reserve Senior Champion Platinum Wether

Oeltjenbruns' Farms Frank	Danielle Frost

Grand Champion Platinum Wether

Oeltjenbruns' Farms Patron	Sarah Oeltjenbruns

Reserve Grand Champion Platinum Wether

Frosty Acres Tanner	Danielle Frost

Iowa's Premier Myotonic Classic - Osceola, IA

Show B- June 5, 2021

Junior Champion Doe

Oeltjenbruns' Farms Quinn	Sarah Oeltjenbruns

Reserve Junior Champion Doe

BDF Jade	Drew & Amy DeRiemacker

Senior Champion Doe

Oeltjenbruns' Farms Zendaya	Sarah Oeltjenbruns

Reserve Senior Champion Doe

Crow River Fainters Fabulous Flirt	Ashley Henklemann

Grand Champion Doe

*Oeltjenbruns' Farms Zendaya	Sarah Oeltjenbruns

Reserve Grand Champion Doe

Oeltjenbruns' Farms Quinn	Sarah Oeltjenbruns

Junior Champion Buck

Naughty Goat Acres Firetrucker	Colleen Reardon & Robert Lorenz

Reserve Junior Champion Buck

Muddy River Eris	Brittany Frey

Senior Champion Buck

Naughty Goat Acres Boomer	Nikki Thummel

Reserve Senior Champion Buck

Wallace's Crazy Acres Oakley	Renee Anderson

Grand Champion Buck

Naughty Goat Acres Boomer	Nikki Thummel

Reserve Grand Champion Buck

Wallace's Crazy Acres Oakley	Renee Anderson

Junior Champion Platinum Wether

Frosty Acres Tanner	Danielle Frost

Reserve Junior Champion Platinum Wether

Heavenly Hill Farm Frankenstein	Phillip & Mackenzie Jurek

Senior Champion Platinum Wether

Oeltjenbruns' Farms Jager	Danielle Frost

Reserve Senior Champion Platinum Wether

Bureau Creek Amazon Prime	Danielle Frost

Grand Champion Platinum Wether

*Oeltjenbruns' Farms Jager	Danielle Frost

Reserve Grand Champion Platinum Wether

Frosty Acres Tanner	Danielle Frost

Iowa's Premier Myotonic Classic - Osceola, IA

Show C- June 5, 2021	
Junior Champion Doe	
Oeltjenbruns' Farms Quinn	Sarah Oeltjenbruns
Reserve Junior Champion Doe	
Crow River Fainters Moxie	Ashley Henklemann
Senior Champion Doe	
Oeltjenbruns' Farms Zendaya	Sarah Oeltjenbruns
Reserve Senior Champion Doe	
Fern Hill Prudence	Drew & Amy DeRiemacker
Grand Champion Doe	
*Oeltjenbruns' Farms Zendaya	Sarah Oeltjenbruns
Reserve Grand Champion Doe	
Fern Hill Prudence	Drew & Amy DeRiemacker
Junior Champion Buck	
Naughty Goat Acres Jake	Brittany Frey
Reserve Junior Champion Buck	
Oeltjenbruns' Farms Zeus	Sarah Oeltjenbruns
Senior Champion Buck	
Oeltjenbruns' Farms Myotonics Kingsman	Benjamin & Sheena Schmidt
Reserve Senior Champion Buck	
Oeltjenbruns' Farms Case	Jessica Olmscheid
Grand Champion Buck	
*Oeltjenbruns' Farms Myotonics Kingsman	Benjamin & Sheena Schmidt
Reserve Grand Champion Buck	
Naughty Goat Acres Jake	Brittany Frey
Junior Champion Platinum Wether	
Heavenly Hill Farm Frankenstein	Phillip & Mackenzie Jurek
Reserve Junior Champion Platinum Wether	
Frosty Acres Tanner	Danielle Frost
Senior Champion Platinum Wether	
Oeltjenbruns' Farms Jager	Danielle Frost
Reserve Senior Champion Platinum Wether	
Bureau Creek Amazon Prime	Danielle Frost
Grand Champion Platinum Wether	
Heavenly Hill Farm Frankenstein	Phillip & Mackenzie Jurek
Reserve Grand Champion Platinum Wether	
Oeltjenbruns' Farms Jager	Danielle Frost

2021 MGR Show Schedule

**Sanctioned*

****September 3-4***
Antelope Island/Three Islands Crossing Show
Glenns Ferry, ID

****September 11***
International Goat Days
Millington, TN

****October 1-2***
MGR Fall Finale
Henry Co, KY

****October 2***
MGR Nationals
Henry Co, KY

MGR Myomania – Lebanon, TN

Show 1: Judge Lowell Walker
Show 2: Judge Jim Dowell
Show 3: Judge Debbie Mullins
***Indicates verified MGR Point**

Show 1- June 18, 2021

Junior Champion Doe

WP2 Jamie Diamonds	Samantha Wise or James Prather

Reserve Junior Champion Doe

Woody Creek Farm Friia	Richard & Emily Jorgenson

Senior Champion Doe

Outlaw Farms Ester	Tara and Joe Lawrence

Reserve Senior Champion Doe

Fieldcrest Farms Jetta	Collen Reardon & Robert Lorenz

Grand Champion Doe

*Outlaw Farms Ester	Tara and Joe Lawrence

Reserve Grand Champion Doe

Fieldcrest Farms Jetta	Colleen Reardon & Robert Lorenz

Junior Champion Buck

One Goat Farm Talking Bout My White Pants	Tracy, Ricco, Chloe & Lydia Tumminello

Reserve Junior Champion Buck

WP Fantastic Daydream	Tara and Joe Lawrence

Senior Champion Buck

Domino Goats Fearless For Real	Tracy, Ricco, Chloe & Lydia Tumminello

Reserve Senior Champion Buck

Buck Creek Aragon	Justin & Katie Bevels

Grand Champion Buck

*Domino Goats Fearless For Real	Tracy, Ricco, Chloe & Lydia Tumminello

Reserve Grand Champion Buck

One Goat Farm Talking Bout My White Pants	Tracy, Ricco, Chloe & Lydia Tumminello

Junior Champion Platinum Wether

LnS Laidback Ranch Remington	Suzette or Lowell Mason

Reserve Junior Champion Platinum Wether

Hourglass Acres Johnny Walker	Terin & Curtis McAllister

Senior Champion Platinum Wether

Rocky Ridge Tattoo	Ellen Pittman

Reserve Senior Champion Platinum Wether

Black Walnut Farm Oscar	Elisabeth Bevels

Grand Champion Platinum Wether

*Rocky Ridge Tattoo	Ellen Pittman

Reserve Grand Champion Platinum Wether

Black Walnut Farm Oscar	Elisabeth Bevels

MGR Myomania – Lebanon, TN

Show 2- June 19, 2021	
Junior Champion Doe	
Outlaw Farms Annalise	Tara and Joe Lawrence
Reserve Junior Champion Doe	
Outlaw Farms Cordelia	Tara and Joe Lawrence
Senior Champion Doe	
Outlaw Farms Ester	Tara and Joe Lawrence
Reserve Senior Champion Doe	
Twin Creek Rosebud	Collen Reardon & Robert Lorenz
Grand Champion Doe	
*Outlaw Farms Ester	Tara and Joe Lawrence
Reserve Grand Champion Doe	
Twin Creek Rosebud	Colleen Reardon & Robert Lorenz
Junior Champion Buck	
WP Fantastic Daydream	Tara and Joe Lawrence
Reserve Junior Champion Buck	
LnS Laidback Ranch Gunner	Suzette or Lowell Mason
Senior Champion Buck	
WP Don Julio	Kaely Prather
Reserve Senior Champion Buck	
Naughty Goat Acres Slapjack	Suzette or Lowell Mason
Grand Champion Buck	
*WP Don Julio	Kaely Prather
Reserve Grand Champion Buck	
WP Fantastic Daydream	Tara and Joe Lawrence
Junior Champion Platinum Wether	
Hourglass Acres Johnny Walker	Terin & Curtis McAllister
Reserve Junior Champion Platinum Wether	
Outlaw Farms Zinc	Tara and Joe Lawrence
Senior Champion Platinum Wether	
Rocky Ridge Tattoo	Ellen Pittman
Reserve Senior Champion Platinum Wether	
Amazing Grace Farms Lucky Charm	Grace Lawrence
Grand Champion Platinum Wether	
*Rocky Ridge Tattoo	Ellen Pittman
Reserve Grand Champion Platinum Wether	
Amazing Grace Farms Lucky Charm	Grace Lawrence

MGR Myomania – Lebanon, TN

Show 3- June 19, 2021	
Junior Champion Doe	
Outlaw Farms Annalise	Tara and Joe Lawrence
Reserve Junior Champion Doe	
WP Peanut Butta	Samantha Wise
Senior Champion Doe	
Twin Creek Rosebud	Colleen Reardon & Robert Lorenz
Reserve Senior Champion Doe	
Buck Creek Willow	Samantha Wise or James Prather
Grand Champion Doe	
*Twin Creek Rosebud	Colleen Reardon & Robert Lorenz
Reserve Grand Champion Doe	
Outlaw Farms Annalise	Tara and Joe Lawrence
Junior Champion Buck	
One Goat Farm Talking Bout My White Pants	Tracy, Ricco, Chloe & Lydia Tumminello
Reserve Junior Champion Buck	
WP Ghost Ryder	Krystal & Steve O'Bryan
Senior Champion Buck	
Buck Creek Aragon	Justin & Katie Bevels
Reserve Senior Champion Buck	
Muddy River Rebel	Justin & Katie Bevels
Grand Champion Buck	
*Buck Creek Aragon	Justin & Katie Bevels
Reserve Grand Champion Buck	
One Goat Farm Talking Bout My White Pants	Tracy, Ricco, Chloe & Lydia Tumminello
Junior Champion Platinum Wether	
Outlaw Farms Zinc	Tara and Joe Lawrence
Reserve Junior Champion Platinum Wether	
Hourglass Acres Johnny Walker	Terin & Curtis McAllister
Senior Champion Platinum Wether	
Rocky Ridge Tattoo	Ellen Pittman
Reserve Senior Champion Platinum Wether	
Black Walnut Farm Oscar	Elisabeth Bevels
Grand Champion Platinum Wether	
*Rocky Ridge Tattoo	Ellen Pittman
Reserve Grand Champion Platinum Wether	
Black Walnut Farm Oscar	Elisabeth Bevels

MGR Myomania – Lebanon, TN

Show 1: June 18, 2021

Champion Light Weight Wether

Black Walnut Farm I Heart Veggies	Elisabeth Bevels

Champion Heavy Weight Wether

One Goat Farm Chevon	Tracy, Ricco, Chloe & Lydia Tumminello

Reserve Champion Heavy Weight Wether

Rocky Ridge Under Pressure	Ellen Pittman

Grand Champion Market Wether

*One Goat Farm Chevon	Tracy & Chloe Tumminello

Reserve Grand Champion Market Wether

Black Walnut Farm I Heart Veggies	Elisabeth Bevels

Show 2 : June 19, 2021

Champion Light Weight Wether

Black Walnut Farm I Heart Veggies	Elisabeth Bevels

Champion Heavy Weight Wether

One Goat Farm Chevon	Tracy, Ricco, Chloe & Lydia Tumminello

Reserve Champion Heavy Weight Wether

Rocky Ridge Under Pressure	Ellen Pittman

Grand Champion Market Wether

*One Goat Farm Chevon	Tracy & Chloe Tumminello

Reserve Grand Champion Market Wether

Rocky Ridge Under Pressure	Ellen Pittman

Show 3: June 19, 2021

Champion Light Weight Wether

Black Walnut Farm I Heart Veggies	Elisabeth Bevels

Champion Heavy Weight Wether

One Goat Farm Chevon	Tracy, Ricco, Chloe & Lydia Tumminello

Reserve Champion Heavy Weight Wether

Rocky Ridge Under Pressure	Ellen Pittman

Grand Champion Market Wether

*One Goat Farm Chevon	Tracy & Chloe Tumminello

Reserve Grand Champion Market Wether

Rocky Ridge Under Pressure	Ellen Pittman

Hoosier Hysteria - Alexandria, IN

Heavenly Pines Family Farm Show 1: Judge Josh Lichlyter
Willow Tree Farm Show 2: Judge Ben Dauer
Fieldcrest Farms Show 3: Judge Tomas Redden
***Indicates verified MGR Point**

Show 1- June 25, 2021

Junior Champion Doe

Frosty Acres Barbie Girl	Danielle Frost

Reserve Junior Champion Doe

WP Spice Girl	Brian & Mackenzie Treadwell

Senior Champion Doe

WP Amaretto	Danielle Frost

Reserve Senior Champion Doe

Mar-Bob Fannie Mae	Suzette or Lowell Mason

Grand Champion Doe

*Frosty Acres Barbie Girl	Danielle Frost

Reserve Grand Champion Doe

WP Amaretto	Danielle Frost

Junior Champion Buck

Morelock Showstock Game On	Tyler Morelock

Reserve Junior Champion Buck

Oeltjenbruns' Farms Titan	Bryan & Debbie Monts

Senior Champion Buck

Buck Creek Moonshine	Brian & Mackenzie Treadwell

Reserve Senior Champion Buck

WP Huckleberry Finn	Brian & Mackenzie Treadwell

Grand Champion Buck

*Buck Creek Moonshine	Brian & Mackenzie Treadwell

Reserve Grand Champion Buck

WP Huckleberry Finn	Brian & Mackenzie Treadwell

Junior Champion Platinum Wether

Frosty Acres Tanner	Danielle Frost

Reserve Junior Champion Platinum Wether

Angelo's Fainters Antonio	Anita Angelo

Senior Champion Platinum Wether

Bureau Creek Amazon Prime	Danielle Frost

Reserve Senior Champion Platinum Wether

Bureau Creek Millionaire	Danielle Frost

Grand Champion Platinum Wether

Bureau Creek Amazon Prime	Danielle Frost

Reserve Grand Champion Platinum Wether

*Bureau Creek Millionaire	Danielle Frost

Hoosier Hysteria - Alexandria, IN

Show 2- June 26, 2021

Junior Champion Doe

Cornstalk Creek Rose	Bryan & Debbie Monts

Reserve Junior Champion Doe

Muddy River Prada	Kali Poore

Senior Champion Doe

Muddy River Gamora	Kali Poore

Reserve Senior Champion Doe

Fern Hill Gucci	J.R. Poore

Grand Champion Doe

*Muddy River Gamora	Kali Poore

Reserve Grand Champion Doe

Fern Hill Gucci	J.R. Poore

Junior Champion Buck

WP Ghost Ryder	Krystal O'Bryan

Reserve Junior Champion Buck

Taylor's Oh-Oh IMA Secret Weapon	Amy Taylor

Senior Champion Buck

Muddy River Recon	Kali Poore

Reserve Senior Champion Buck

WP Huckleberry Finn	Brian & Mackenzie Treadwell

Grand Champion Buck

*Muddy River Recon	Kali Poore

Reserve Grand Champion Buck

WP Huckleberry Finn	Brian & Mackenzie Treadwell

Junior Champion Platinum Wether

Frosty Acres Flash	Danielle Frost

Reserve Junior Champion Platinum Wether

Angelo's Fainters Antonio	Anita Angelo

Senior Champion Platinum Wether

Muddy River X-Force	Kinzie Poore

Reserve Senior Champion Platinum Wether

Bureau Creek Millionaire	Danielle Frost

Grand Champion Platinum Wether

*Muddy River X-Force	Kinzie Frost

Reserve Grand Champion Platinum Wether

Bureau Creek Millionaire	Danielle Frost

Hoosier Hysteria - Alexandria, IN

Show 3- June 26, 2021	
Junior Champion Doe	
Frosty Acres Valentine	Danielle Frost
Reserve Junior Champion Doe	
Muddy River Mi Amor	Jason Gray
Senior Champion Doe	
Mar-Bob Dewdrop	Brian & Mackenzie Treadwell
Reserve Senior Champion Doe	
Fern Hill Gucci	J.R. Poore
Grand Champion Doe	
*Mar-Bob Dewdrop	Brian & Mackenzie Treadwell
Reserve Grand Champion Doe	
Fern Hill Gucci	J.R. Poore
Junior Champion Buck	
Morelock Showstock Game On	Tyler Morelock
Reserve Junior Champion Buck	
WP Ghost Ryder	Krystal O'Bryan
Senior Champion Buck	
Buck Creek Hollywood Gold	Danielle Frost
Reserve Senior Champion Buck	
WP Huckleberry Finn	Brian & Mackenzie Treadwell
Grand Champion Buck	
*Buck Creek Hollywood Gold	Danielle Frost
Reserve Grand Champion Buck	
WP Huckleberry Finn	Brian & Mackenzie Treadwell
Junior Champion Platinum Wether	
Frosty Acres Tanner	Danielle Frost
Reserve Junior Champion Platinum Wether	
Angelo's Fainters Antonio	Anita Angelo
Senior Champion Platinum Wether	
Bureau Creek Amazon Prime	Danielle Frost
Reserve Senior Champion Platinum Wether	
Bureau Creek Millionaire	Danielle Frost
Grand Champion Platinum Wether	
*Frosty Acres Tanner	Danielle Frost
Reserve Grand Champion Platinum Wether	
Bureau Creek Amazon Prime	Danielle Frost

Hoosier Hysteria - Alexandria, IN

Show 1: June 25, 2021	
Champion Light Weight Wether	
Lloyd	Lora Watson
Champion Heavy Weight Wether	
Frosty Acres Chopper	Danielle Frost
Grand Champion Market Wether	
Lloyd	Lora Watson
Reserve Grand Champion Market Wether	
Frosty Acres Chopper	Danielle Frost

Show 3: June 26, 2021	
Champion Light Weight Wether	
Lloyd	Lora Watson
Champion Heavy Weight Wether	
Frosty Acres Chopper	Danielle Frost
Grand Champion Market Wether	
Frosty Acres Chopper	Danielle Frost
Reserve Grand Champion Market Wether	
Lloyd	Lora Watson

Illini Summer Spectacular - Princeton, IL

Show 1: Judge Josh Lichlyter
Show 2: Judge Chris Fleming
Show 3: Judge Ashley Hadley
***Indicates verified MGR Point**

Show 1- July 9, 2021

Junior Champion Doe

Oeltjenbruns' Farms Quinn	Sarah Oeltjenbruns

Reserve Junior Champion Doe

Oeltjenbruns' Farms Dixieland Delight	Sarah Oeltjenbruns

Senior Champion Doe

Oeltjenbruns' Farms Helen	Sarah Oeltjenbruns

Reserve Senior Champion Doe

Bureau Creek Layla	Benjamin & Sheena Schmidt

Grand Champion Doe

*Oeltjenbruns' Farms Helen	Sarah Oeltjenbruns

Reserve Grand Champion Doe

Oeltjenbruns' Farms Quinn	Sarah Oeltjenbruns

Junior Champion Buck

Oeltjenbruns' Farms Zane	Sarah Oeltjenbruns

Reserve Junior Champion Buck

Naughty Goat Acres Jake	Brittany Frey

Senior Champion Buck

WP Huckleberry Finn	Brian & Mackenzie Treadwell

Reserve Senior Champion Buck

Oeltjenbruns' Farms Myotonics Kingsman	Benjamin & Sheena Schmidt

Grand Champion Buck

*WP Huckleberry Finn	Brian & Mackenzie Treadwell

Reserve Grand Champion Buck

Oeltjenbruns' Farms Myotonics Kingsman	Benjamin & Sheena Schmidt

Junior Champion Platinum Wether

Echo Acres Theodore	Renee Anderson

Reserve Junior Champion Platinum Wether

Frosty Acres Tanner	Danielle Frost

Senior Champion Platinum Wether

Oeltjenbruns' Farms Patron	Sarah Oeltjenbruns

Reserve Senior Champion Platinum Wether

Bureau Creek Amazon Prime	Danielle Frost

Grand Champion Platinum Wether

*Oeltjenbruns' Farms Patron	Sarah Oeltjenbruns

Reserve Grand Champion Platinum Wether

Bureau Creek Amazon Prime	Danielle Frost

Illini Summer Spectacular
2021
Princeton, Illinois

Illini Summer Spectacular - Princeton, IL

Show 2- July 10, 2021

Junior Champion Doe

Naughty Goat Acres Gold Rush	Joe Prior

Reserve Junior Champion Doe

Crow River Fainters Moxie	Ashley Henklemann

Senior Champion Doe

Oeltjenbruns' Farms Helen	Sarah Oeltjenbruns

Reserve Senior Champion Doe

Buck Creek Ebony	Benjamin & Sheena Schmidt

Grand Champion Doe

*Oeltjenbruns' Farms Helen	Sarah Oeltjenbruns

Reserve Grand Champion Doe

Buck Creek Ebony	Benjamin & Sheena Schmidt

Junior Champion Buck

Naughty Goat Acres Grizzly	Benjamin & Sheena Schmidt

Reserve Junior Champion Buck

Oeltjenbruns' Farms Zac	Ashley Henklemann

Senior Champion Buck

Oeltjenbruns' Farms Lucky Charm	Phillip & Mackenzie Jurek

Reserve Senior Champion Buck

Buck Creek Hollywood Gold	Danielle Frost

Grand Champion Buck

*Oeltjenbruns' Farms Lucky Charm	Phillip & Mackenzie Jurek

Reserve Grand Champion Buck

Buck Creek Hollywood Gold	Danielle Frost

Junior Champion Platinum Wether

Frosty Acres Tanner	Danielle Frost

Reserve Junior Champion Platinum Wether

Echo Acres Theodore	Renee Anderson

Senior Champion Platinum Wether

Oeltjenbruns' Farms Patron	Sarah Oeltjenbruns

Reserve Senior Champion Platinum Wether

Bureau Creek Amazon Prime	Danielle Frost

Grand Champion Platinum Wether

*Frosty Acres Tanner	Danielle Frost

Reserve Grand Champion Platinum Wether

Echo Acres Theodore	Renee Anderson

Illini Summer Spectacular - Princeton, IL

Show 3- July 10, 2021

Junior Champion Doe

Naughty Goat Acres Poppy Seed	Betsy Muehleip

Reserve Junior Champion Doe

Heavenly Pines Reba	Brittany Frey

Senior Champion Doe

Buck Creek Tallulah	Benjamin & Sheena Schmidt

Reserve Senior Champion Doe

Oeltjenbruns' Farms Faith	Sarah Oeltjenbruns

Grand Champion Doe

*Buck Creek Tallulah	Benjamin & Sheena Schmidt

Reserve Grand Champion Doe

Naughty Goat Acres Poppy Seed	Betsy Muehleip

Junior Champion Buck

Oeltjenbruns' Farms Zane	Sarah Oeltjenbruns

Reserve Junior Champion Buck

Oeltjenbruns' Farms Zeus	Sarah Oeltjenbruns

Senior Champion Buck

Fern Hill Notorious	Sarah Oeltjenbruns

Reserve Senior Champion Buck

Heavenly Hill Farm Zorro	Phillip & Mackenzie Jurek

Grand Champion Buck

*Fern Hill Notorious	Sarah Oeltjenbruns

Reserve Grand Champion Buck

Heavenly Hill Farm Zorro	Phillip & Mackenzie Jurek

Junior Champion Platinum Wether

Echo Acres Theodore	Renee Anderson

Reserve Junior Champion Platinum Wether

Angelo's Fainters Antonio	Anita Angelo

Senior Champion Platinum Wether

Oeltjenbruns' Farms Patron	Sarah Oeltjenbruns

Reserve Senior Champion Platinum Wether

Heavenly Hill Farm No Boom Boom	Phillip & Mackenzie Jurek

Grand Champion Platinum Wether

*Oeltjenbruns' Farms Patron	Sarah Oeltjenbruns

Reserve Grand Champion Platinum Wether

Echo Acres Theodore	Renee Anderson

Myotonic Goat Registry
P.O. Box 141
Adger, AL 35006

Phone: 205-425-5954
E-mail: myotonicgoatregistry@yahoo.com
Website: www.myotonicgoatregistry.net

The Myotonic Goat Registry was formed in 2005 as a sole ownership registry by Gene McNutt with input from an initial Board of Advisors made up of Dr. Phil Sponenberg and Barbara Roberts. The current Board of Advisors includes Dr. Phil Sponenberg, Cindy Bene, Eve Williams and Anna Garrett. The owner and Board of Advisors will make decisions concerning the registry and its procedures. This method of governance is meant to provide Myotonic Goat breeders with a registry that will not have frequent changes, and will have the longevity and consistency needed to successfully promote the Myotonic Goat breed, while at the same time make it responsive to the needs and wishes of the breeders. In 2009, Gene retired and the registry was sold to Tara Lawrence. As the Myotonic Goat Registry grows, additional Board of Advisor members may be added in order to more broadly represent the breeders. The owner, along with the Board of Advisors, will be responsi-ble for providing for its own replacements and/or expansions.

The Myotonic Goat Registry takes into consideration all breeders, from pet owners to commercial meat growers. Regardless of which aspect of this breed appeals to you, the Myotonic Goat Registry is the place for all breeders to register their Myotonic Goats.

The Registry will help breeders promote their goats through sales, shows, and advertising, and will educate the public about the Myotonic Goat and its usefulness in a variety of settings.