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The monthly magazine devoted to cashmere goats and their fiber

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C A S H M I R R O R

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Boring (Yet Important) Technical Information

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Cover photo:
"Linda Fox, Goat Knoll, Dallas, Oregon
"New kids on the block"

Six Years of Cashmere Business Conferences! A Comprehensive Reference for Cashmere Goats PCMA Proceedings Books For Sale—While They Last

CashMirror has inherited the last of the PCMA Proceedings books. They are for sale. Profits from sale of these books will be used to sponsor *CashMirror* contests and promote other cashmere goat events as we see fit. These books will be available to surfers on the internet as well and we expect them to disappear quickly, so if you want one, you should order one soon.

These books contain the proceedings of six years' Professional Marketing Cashmere Association Business of Cashmere conferences held 1995 - 2000. The books contain all papers presented at these six conferences, neatly printed, organized into sections and bound in an attractive (large) 3-ring binder.

Fifty-one papers are included on subjects including cashmere goat management, fiber issues, using goats for weed control and farm financial planning and management (see index below). This book is a valuable reference tool for new or prospective cashmere producers as well as a good perspective for experienced cashmere producers on the business aspects of raising cashmere.

These books are only $45 (plus $5 for shipping costs if we mail them to you, extra charge for postage outside of U.S.). Order from *CashMirror* by check or credit card.

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Reflections

by Linda Fox

New Arwen and attentive mother, Fancy.

It's 2002, a new goat year and this is the year we're going to do everything right. Just like we planned to last year—and the year before. However, this is the year we're really going to do it!

We're going to develop a better and easier recordkeeping system, we're going to ear tag our kids early, we're going to clean out the entire barn this fall, we're going to shear on time, we're going to write more legibly on the fleece bags, we're going to enter more fleeces in contests, we're going to work harder at marketing, and we're going to review the rules of kidding just before the kidding season starts. This last one just got added last weekend. We lost a kid that we shouldn't have.

In our years of raising cashmere goats (222 kids born), we've only lost two babies. Last Saturday we lost our third. The first one that we lost several years ago was four days old when it died. It was thin and weak when born and never gained strength, even with our care. The second, year before last, was too large for the doe to have. It died before the birth process even started and was removed by Cesarean by our Veterinarian.

This is the first kid that we have lost due to a poor kidding presentation, not corrected in time. The kid was presented for birth with its front feet back and tangled and we didn't correct the problem in time. We were in the barn and watched the normal birth of the first baby (Arwen—see photographs on pages 23 and 24). The doe was experienced, usually had twins and has always had uneventful births. We both watched the mother care for the first kid and intermittently lay down and briefly strain. We wondered if one kid was going to be all or if she was working on another. When we did finally assist, we untangled legs so that she could have the second kid, but only helped her deliver a perfectly normal, perfect little (dead) doe. She had gone too long without air.

We have a tendency to get complacent about cashmere goat births. In our seven kidding years, even though we've seen many births, we've only assisted in two—both involving resposition-ing tangled or caught front legs. The does seldom require our help and sometimes we wonder when we are there, if they would have an easier time of it if we were not there to worry them. However, we do need to remember that sometimes they do require our assistance.

I used to think that reading all the gruesome kidding articles that focused on kidding difficulties made us anxious and forgetful that most kiddings are uneventful. I have now decided that each year I need to re-read kidding articles before kidding starts to remind us of the things that can go wrong.

I debated even sharing this event with you as there are those of you who will no doubt label us fools for making the mistake and we are still somewhat sniffly over the whole affair. However, there are also those in the audience who may learn something from our error and perhaps a few of you who have done things equally unwise, but who have the "luxury" of not having to talk about it.

Paul with Aragon and Eowyn. They are a month old and demand his attention. Aragon likes his tummy scratched.

When Readers Talk...

Dear editor;
Have you ever written an article on meningeal worms in cashmere goats? If so, how do I get a copy of that issue? If you haven't written an article, please write one. I will probably lose one of my precious goats to this disease. I have a worming program, but apparently it was not often enough during the late summer when the deer were heavy in my pasture. Let people know that there are lots of natural predators for slugs, the intermediate host of the worm. I am going to start a flock of Khaki Campbell ducks because they are prolific slug eaters. We tried shots of steroids and ivermectin as well as panacur orally, it saved his life, but he doesn't have enough movement or strength in his hind legs to get up without assistance.

Sincerely,
Kim Reichart
Dayton, Maryland, December 26, 2001

Happy Ground Hogs Day from Shannon Atkinson. That would be February 2nd in case you're wondering.

You Know You Are a Cashmere Goat Farmer When:

You feel relieved when you discover the blood on your arm is just yours.

You don't think it's unusual to arrive at a fancy banquet in a pickup with hay in the back.

You think a jug is a kidding stall.

When you are no longer embarrassed to carry two goat kids into a major airport to catch a plane.

When you get livestock names confused with the names of your grandkids, nieces and nephews.

You know (and care) more about small ruminant health than your own.

You correct people who think goats eat tin cans and that the only good cashmere comes off the belly.

You are more concerned with your goats' hair than your own.

You know the Greek symbol for "micron", and can read a histogram.

You spell "dehairing" with one "r", know what the word means and are annoyed that your computer's spell checker doesn't.

Your definition of "style" has nothing to do with today's fashion scene.

You are reading this!

µ Computer Tip

If you want to insert the symbol for micron in any program, hold down your "Alt" key and type in 0181. When you finish typing and release the Alt key, the µ symbol will magically appear. This works in any program, including Word, Excel, Pagemaker, Notepad and Wordpad.

Overview of Missouri Goat Industry

From the Lincoln University Cooperative Extension
Marketing Program Newsletter, 11/01

In the past four years, interest in goat production in Missouri has grown by leaps and bounds. This has translated into the expansion of herds by existing producers and the entry of new producers into the industry, and led to an increase in the number of Missouri goats by approximately 1.9 million head. Throughout the United States, there has continued to be an upward trend in the number of goats. It is projected that this will continue into the future, holding everything else constant.

As previously reported in one of the last newsletters, different factors led to this conclusion. The demand for goat meat is steadily increasing annually by over two million. These individuals are coming from those countries where goat is a staple meat and a delicacy. There is a substantial increase in the number of United States citizens who are accepting goat as meat of choice. This is primarily because of its health-related attributes.

The following facts are true about goat meat:

· It is the most eaten meat in the world.
· It is exceptionally lean and low in cholesterol and therefore the focus of a growing demand from the health conscious communities in the country.
· It is the first choice meat for the majority of the world's population.
· No religious limits are imposed on its consumption.
· There is an emphasis on the increase of protein intake of populations in the United States as well as those in developing countries. Goat meat has become a recommended source of protein.

Review of Economic Factors Affecting the Goat Industry

The level of goat meat sales in Missouri and most other countries in a particular market are determined by the interaction of several factors. These include, but are not limited to, the following:

· Population and consumer income levels
· Dietary and religious tradition
· Competition from alternative meats
· Competition from domestic products and other imported products
· Trade policy arrangements that permit unlimited importation of these competing products

One major factor contributing to the demand for goat meat is economic growth. In most economies, it is generally assumed that during periods of high economic growth rates, there is strong growth in incomes, which translates into enhanced private consumption expenditure. This, in turn, leads to increasing demand for meat as an alternative source of protein.

Annual Federally Inspected Slaughter

Another evidence of the increases in activities in the goat industry is the number of goats slaughtered in the United States annually. As the data below indicate, there was a 59 percent increase in the number of goats slaughtered in the country between 1994 and the year 2000. This represents all the goats slaughtered only in federally inspected slaughterhouses. It does not include those slaughtered on farms and in backyards by ethnic groups and others who consume goats in large numbers. If these are added, the total number of goats slaughtered in the U.S. is projected to be more than 2 million head annually.

Annual Federally Inspected
Goat Slaughter

Year	Number of Goats
1994	344,7OO
1995	326,600
1996	401,700
1997	394,800
1998	445,723
1999	492,591
2000	549,371

Apart from 1995 and 1997, we notice a steady increase in the number of goats slaughtered in the country over the years.

Annabelle the Sheep

Have you found Annabelle the disco dancing sheep? She comes with Real Player, a video/audio player software program useful for listening to music, news, etc. It can be dowloaded from the internet free at http://www.real.com Find "Annebelle" under "Visualizations". Annebelle keeps time with the music being played and even has backup singers and other helpers who trot on from time to time.

Respiratory Diseases

By J. L. Ayers, Los Olivos, CA
From the USDA Extension Goat Handbook

Clinical pneumonia in goats is almost invariably preceded by some event or set of circumstances commonly referred to as stress. This very broad term, stress includes such factors as weaning, long distance hauling, weather factors including sudden temperature changes or low nighttime with high daytime temperatures, poorly ventilated barns especially those heated in extremely cold weather, overcrowding, malnutrition, feed changes, parasitism and worming. The microorganisms (germs) which produce the actual disease process are often normal inhabitants of the respiratory tract. These microorganisms are prevented from causing disease by the normal animal's body defense mechanisms. The relationship between the body and the microorganism is sometimes a very delicate balance especially in the very young animal. Stressing factors can tip the balance in favor of the microorganism and against the young animal.

Barn ventilation in extremely cold weather deserves special comment because it is often overlooked. Warming a barn increases the relative humidity, thus producing a stressful situation. Moisture should never be allowed to accumulate on the walls, ceilings and floors. The rule to follow, especially in barns where the bedding is allowed to accumulate and help maintain the warmth, is if the inside temperature is 5^0F above the outside temperature, an exhaust fan of adequate capacity to prevent moisture condensation must be used. Goats kept in well ventilated barns, which protect them from drafts and becoming wet, can readily tolerate temperatures below -25^0F.

Acute Pasteurella Pneumonia

In the United States of America the most common cause of pneumonia in goats is Pasteurella multocida and P. hemplytica. It is an acute disease causing extreme debilitation and often death. It occasionally has a systemic form in which the gastrointestinal tract is the other primarily involved system.

In sheep and presumably in goats, up to 40% of normal animals contain one of the above species of bacteria in their nasal passages.

Clinical Signs

While herd outbreaks do occur, individual cases also occur in goats. Morbidity and mortality figures are not available for goats. Depression, lack of appetite, mucopurulent ("pussy") discharge from the nose and occasionally the eyes, occasional coughing (but not as consistent as in cattle and sheep), fever (104^0 to 107^0F) are usually present. Difficult or increased breathing is often not noticed unless the animal is forced to exercise; in this case, panting and coughing occurs.

Tissue Changes

Small (petechial) hemorrhages may be present on the lining of the body cavities especially of the heart. The bronchiole lymph nodes are usually swollen and hemorrhagic.

The most consistent and striking change is seen immediately upon opening the chest and completely reflecting the ribs, especially of the right side. First there are marked adhesions of the visceral and parietal pleura (chest cavity lining) and pericardium. The heart and lung may be covered with yellow-gray gelatinous or clotted fluid, fibrin and fibrous connective tissue. This may completely or partially hide the underlying apical and cardiac lung lobe exposure of which reveals a very angry red or purple appearance. The lobes are necrotic, friable and often contain purulent exudate or even abscesses. The fluid may be dirty-yellow and have a fetid odor. When cut, the lungs may have a sharp line of demarcation between the less angry looking (merely consolidated or nearly normal) lung and the necrotic portion. A dark hemorrhagic band of 2 to 3 millimeters may separate the two zones. There are often necrotic cavitations containing purulent exudate or necrotic debris.

Diagnosis

Diagnosis is based upon the history, signs and necropsy lesions and is confirmed by isolation and identification of P. multocida or P. hemolytica. Because of the presence of the organism in normal animals, diagnosis cannot be made by culturing the organism without the signs and lesions. Differentiation from mycoplasma pneumonia (Mycoplasma mycoides subspecies mycoides) can be tentatively assumed at necropsy by the severe, angry appearance with marked necrosis which is characteristic of caprine pasteurellosis. Differentiation is important because treatment of pasteurellosis and mycoplasmosis is different.

Prevention and Treatment

Reducing stressful circumstances or giving antibiotics preventively when stress cannot be avoided, will help reduce the severity and the incidence of the disease.

Penicillin and sulfamethazine are approved for treatment by injection and sulfamethazine is approved for oral administration.

However, oxytetracycline and the long acting sulfonamide, sulfadimethoxine are also effective against the organism. The major limitation in using these antimicrobial drugs comes from the very short but severe course of the disease. It is difficult to detect, diagnose and treat before the severe necrotizing tissue changes occur. However, in an outbreak, an alert herdsman can detect additional new cases early.

Acute Mycoplasmal Pneumonia

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The mycoplasmal diseases of goats have taken some time for scientists to sort, classify and understand. The explanation is far beyond the scope of this article, but suffice to say, in the US there is a rather common disease characterized by acute pneumonia and arthritis. This disease is caused by Mycoplasma mycoides ss mycoides and is the most common cause of pneumonia in Arizona goats. The organism with the same name, but with very slight differences in growth characteristics (colony size), is the one which causes the dreaded Contagious Bovine Pleuropneumonia, eradicated from US cattle in the 19th century.

The disease occurs primarily in 2 to 10 week old kids. Two Arizona outbreaks were associated with the spring weather change of warm days but continued cool (30^0 to 40^0F) nights. The acute stage of severe disease and death loss lasted about one week. Sick animals which survived were ill about 3 weeks. The morbidity (percentage of the herd affected) was 70% and the mortality was 36%.

Clinical Signs
The most prominent signs were swollen joints, especially the carpi and stifles (front and rear knees) with or without lameness, fever (106^0 to 108^0F) and dyspnea. Coughing is not consistent unless elicited by forced exercise or laryngeal pressure. Swelling of the face or head is infrequently seen and results from mandibular or atlanto-occipital joint involvement.

Tissue Changes
The lung changes always occur on the right side and usually on the left. They consist of red-purple consolidation of the dependent or entire portions of the apical and cardiac and occasionally the diaphragmatic lobes. These portions are friable and moderate amounts of mucopurulent exudate can often be squeezed from them. Depending on the stage of the disease process, cut sections of affected lung vary from dark reddish-purple homogeneous tissue to a variegated color pattern of hepatized to necrotic lobules separated by interlobular edema or fibrosis. Occasionally thin walled abscesses are present from which the organism can be isolated in pure culture.

The copious yellow pleural exudate often contain large quantities of soft fibrin lightly adhering to the surfaces. Bronchial and mediastinal lymph nodes are generally enlarged and on cut section, very moist.

The major diarthrodial joints are most often affected. The inflammatory reaction varies from increased cloudy joint fluid with fibrin clots to marked erosions of articular cartilage with fibrosis of the joint capsule. Periarticular tissues in acute cases were often edematous and congested, with extensive fibrosis occurring in chronic cases.

Diagnosis
As with acute pasteurellosis, diagnosis is based on history, signs, necropsy lesions and isolation of the causative organism. However, the knowledge of which disease usually occurs in a particular area is of practical importance while awaiting laboratory confirmation. As previously stated, pasteurellosis usually produces a much more severe or angry appearance than mycoplasmosis; this is a subjective determination and subject to error.

The organism will grow on ordinary blood agar but many inexperienced technicians may not keep the plate long enough to notice the tiny areas of hemolysis in which a colony can only be seen under magnification. Ideally a special mycoplasma medium should be used, and if typical colonies are found, sent by the diagnostic laboratory to one of the few mycoplasma reference laboratories.

Prevention and Treatment
Tylosin is the drug of choice for mycoplasmosis. Prevention is as difficult as preventing the change in the weather. If outbreaks recur, the use of tylosin before an outbreak occurs may possibly prevent it. In the face of severe outbreaks, massive does of tylosin seem to be necessary to appreciably affect the disease process. Two grams of injectable tylosin given intravenously

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Respiratory Infections of Show Goats

Respiratory infections are very common after susceptible animals have been out to a show and often the infection will spread through the rest of the herd, even those that did not go to the show, especially if there was no isolation of the returning goats. Goats will cough and have a nasal discharge. They may run a fever, be off-feed, and stand around, in a depressed state with drooping ears.

There is no one specific infectious organism causing this. It is likely there are several agents involved, similar to the shipping fever situation in cattle. After several shows and bouts of respiratory infection, most goats develop some resistance. After that, it is usually only the newcomers that will be affected. However, some animals may remain as chronic coughers, and these often relapse into pneumonia following stresses such as a sudden change in weather.

Respiratory Diseases
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(slowly) followed by 1 gram given subcutaneously twice daily for 14 days appear to reduce the severity of an outbreak. It must be stated that tylosin in any dose is not approved for use in goats and inclusion in this section cannot be construed as a recommendation of its use. Indeed, at these high doses, there may be a risk of killing the animal. This must be weighed against the possibility of losing the animal without this treatment and needs to be discussed by the owner and the veterinarian. The author gratefully acknowledges Dr. Dale Brooks of the University of California at Davis as the initiator of the massive dose system. The above dosage schedule may not be the same as currently recommended by Dr. Brooks.

Mycoplasmal Pneumonia of Spanish and Angora Goats
A pneumonia disease of Spanish and Angora goats caused by Mycoplasma ovipneumoniae has been reported from Texas. The time changes are apparently quite similar to those produced by M. mycoides ss mycoides.

Further, the affected animals were subjected to extreme stress of inadequate handling, inclement weather and disease. In two of the four cases, only M. ovipneumoniae was isolated. The occurrence of the disease is associated with pasturing cows, sheep and goats together; transmission may take place from sheep to goats.

Chronic Progressive Pneumonia
This poorly documented pulmonary disease of goats has many of the characteristics of Progressive Pneumonia of sheep. It may be caused by a virus but it is complicated by the common pneumonia producing bacteria. It is found in breeding goats and invariably associated with the stress of bad ventilation or close confinement in dirty pens, especially kidding pens. Presumably the kids acquire the etiologic agent early, perhaps at birth from an infected mother. Each time the animal is stressed, another episode of acute pneumonia and more and more debilitation occurs; finally an acute episode causes death, often not until 6 or 7 years of age.

Clinical Signs
During each acute episode the animal is anorexic (won't eat), stands by itself with its ears down, acts completely lifeless. Auscultation of the lungs reveals some rales; however, in the advanced case, so little air moves through the lungs that the lung sounds are muffled. Difficult breathing (dyspnea) is present and worsens with repeated episodes. Hypoxia with blue tinting of the mucous membranes of the mouth, vulva or sheath continually worsen.

Tissue Changes
If an animal dies at a young age, lesions similar to but milder than acute pasteurella pneumonia may be seen. As the disease progresses, very small (miliary) foci containing mucous or mucopurulent exudate occupy more and more of the lung. Eventually the lung becomes essentially filled by these foci and by old fibrous connective tissue (scars) and abscesses. The lungs and bronchiolar and mediastinal lymph nodes become 3 to 5 times as heavy as normal. The animal has become extremely debilitated and has very little body fat.

Diagnosis
The history of chronic, recurring pulmonary illness and necropsy findings of chronic lung changes facilitate a diagnosis. Because the disease in goats is not well documented, and the real etiologic agent has not been identified, definitive diagnosis is not yet possible.

Prevention and Treatment
Treatment has been unsuccessful. This fact gives evidence for a viral etiology. Preventive measures should give good results. All does with a chronic cough and having acute pulmonary episodes should be culled from the herd. Kidding barns and all other types of winter housing should be kept clean and well ventilated.

Holy Goats

By Willa Cline
Overland Park, Kansas

An excerpt from Willa Cline's Journal, 12/98
http://www.Willa.com

Today we walked down to McDonald's for lunch so Misty and I could get A Bug's Life Happy Meals. While we were walking, Matt said something about a cashmere sweater he had seen and coveted, but didn't buy because it was ridiculously expensive; Misty mused aloud about why cashmere was so expensive. I told them this story:

The Story of Cashmere

Cashmere doesn't come from ordinary sheep. It comes from the incredibly rare Cashmere Goat. There are only five Cashmere Goats in existence, and they live on top of a very high mountain in Tibet, tended by Tibetan monks. The goats, since they are so very rare and precious, are considered to be sacred, and holy, and they are not allowed to be sheared.

Their hair, the sacred Cashmere Goat Hair, can only be collected after it sheds from the goat naturally. At least two monks attend each Holy Goat at one time—one monk to place a gold-embroidered white cloth in the path of the goat as it walks, so as to catch any sacred Cashmere Goat Hair that falls naturally from the goat, and one to walk behind and gather up the hair as it falls on the cloth.

Since the goats are cared for so religiously, they actually shed less than they would if they were left to their own devices, so very little hair is collected, only a few hairs per week, in fact. It takes almost a year to collect enough Sacred Goat Hair from the five Holy Goats to spin the wool to make one sweater. And only the very holiest of the monks are allowed to spin the wool. Only one monk is chosen every year, and it is considered a very great honor to be chosen to be the Sacred Spinning Monk.

Each year, if enough hair is collected for a sweater—and it doesn't happen every year—the Sacred Goat Hair is brought to the chosen Sacred Spinning Monk, who spins the hair into fine yarn which is then wrapped in one of the gold-embroidered white cloths that are used to collect the Sacred Goat Hair. The yarn is carried down the mountain in a golden casket and handed over to the best knitter in the village, known as the Sacred Cashmere Knitter.

The Sacred Cashmere Knitter knits the yarn into a sweater, using special golden needles that have been blessed by the Dalai Lama. When she has finished knitting, the Sacred Knitter's husband goes out into the valley and calls the monks back by blowing the Sacred Cashmere Horn. The monks then come back down the mountain, collect the sweater, and Federal Express it to a store selected by secret lottery, where it may be purchased at great price. And that is why cashmere sweaters are so expensive.

A sacred cashmere shawl? Well, uh, no...

Meningeal Worms (Brain Worms) & Liver Flukes (Deer Flukes) Two Uncommon Internal Parasites

J. S. Rook, D.V.M. & M. Kopcha, D.V.M., M.S.
MSU Extension & Ag Experiment Station Department of Large Animal Clinical Sciences
College of Veterinary Medicine, Michigan State University

In Michigan, clinical disease resulting from the deer fluke or the meningeal worm is rare, sporadic, and very localized (geographically). Clinical disease generally occurs during the months of September through March— not times when producers typically associate parasites with clinical disease and death losses. Producers residing in endemic deer fluke or meningeal worm regions are well aware of these parasites and the clinical signs they produce. However, producers purchasing feeder lambs and replacement ewes from endemic areas are often not as well versed in the clinical signs, treatment, prevention and available control options.

The deer fluke and the meningeal worm are found in very limited areas of the Upper Great Lakes Region. Each parasite requires close contact with: 1) white-tailed deer (definitive host) and 2) specific species of swamp residing snails (intermediate host) for completion of the parasite's life cycle. Furthermore, sheep are considered dead end hosts for both deer flukes and meningeal worms. The fact that sheep are a dead end host means that neither parasite can reproduce and complete its life cycle within the infected sheep. Once either parasite is ingested by a sheep it may migrate through various parts of the body wrecking havoc with that individual sheep, but the parasite will ultimately fail to reproduce within the affected animal. Thus, only infected deer (not infected sheep) can harbor the parasites over winter and spread either parasite to your flock. Additionally, this can only occur if certain types of snails (intermediate hosts) are also found in your pastures. Due to the relationship of the snail (intermediate host) and the white-tailed deer (definitive host) to the life cycle of the deer fluke and the meningeal worm, both parasites will be discussed as a group.

Meningeal worm ("brain worm")
The meningeal worm, scientifically known as Parelaphostrongylus tenuis and commonly referred to as the "brain worm", is a unique parasite that behaves quite differently from other "more normal" sheep parasites. Due to the location of the "brain worm" within the central nervous system of clinically infected sheep, treatment of animals exhibiting clinical signs is usually unrewarding. Therefore, to avoid significant losses producers need to recognize the clinical signs of meningeal worm infection and to understand how to prevent initial exposure to the parasite.

Deer fluke
The "deer fluke" (Fascioloides magna) is also known as the "giant liver fluke ". It originates in the upper Great Lakes Region and is not the same liver fluke as the "common liver fluke" (Fasciola hepatica) that affects cattle and sheep in most other parts of the country. While treatment of infected animals is usually more rewarding that in meningeal worm infection, deer flukes often cause both acute and chronic losses in endemic areas and impact the grazing management of fall pastures. They are also a common reason for condemnation of livers at slaughter.

Are your sheep at risk?
If either meningeal worm or deer fluke infection is going to be a problem on your farm, a peculiar set of events must all fall into place. In order for either parasite to survive and thrive on your farm, the following scenario must occur:

1. There must be an abundance of infected white-tailed deer.

2. You will usually have swampy pastures or small areas of swamp (pot holes etc.) that are grazed by sheep and goats during late summer and early fall.

3. The appropriate species of snails and slugs also have to reside in that pasture. Generally, deer fluke snails are found in grassy type swampy pastures and brain worm snails are more common in swampy areas that also harbor trees.

4. You also need sufficient rainfall during the grazing season to maintain snail habitat. However, this statement can often be misleading because in dry years sheep often penetrate further into infected swampy areas than they normally would if more water were present. Flock pressure on available forage also forces deeper penetration into swampy areas. In years when little rainfall occurs producers with grass-based production systems also tend to utilize low lying wooded areas to provide several weeks of grazing during dry summer periods.

How do deer contribute to the life cycle?

Common to both parasites
White-tailed deer are the definitive hosts (the animal in which meningeal worms and deer flukes complete their life cycles) and can be carriers of both parasites. However, outside of endemic areas, white-tailed deer are rarely infected with either parasite. The deer is the animal that harbors and protects the parasite through the long cold winter months, allowing winter survival of flukes and brain worms. Each spring infected deer start the infection by depositing fluke or brain worm infected manure onto swampy, snail infested areas. As spring and summer pro-

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Meningeal Worms/Liver Flukes
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gresses, snails eat the infective larva and become intermediate hosts for meningeal worms or deer flukes. While the larva are inside their intermediate hosts (snails) they undergo changes that allow them to become infective to deer (definitive hosts) and sheep or other small ruminants (dead end hosts). These changes inside the intermediate host (snails) take about 3 to 4 weeks. When complete, the snail contains infective larva.

Unlike most of the common sheep internal parasites with which producers are familiar, these unique parasites require snails as part of their life cycle. While deer are the definitive host, snails are important as an intermediate host for the parasites. These parasites can not infect sheep without a 3 to 4 week infective stage that takes place in the snail. Deer that inhabit swampy areas ingest infected snails and the cycle continues. Remove any portion of the equation—deer, parasite or snails—and clinical disease can not occur.

In Michigan, sheep pastures are usually considered "clean" or "free" of deer fluke or meningeal worm contamination shortly after a hard killing freeze each fall (usually November). Each fall a hard freeze kills infective snails—thus eliminating the problem until infected deer re-initiate the cycle the following spring. Therefore, clinical signs and the progression of deer fluke and meningeal worm cases into the winter months are intimately linked to local fall weather conditions. While under certain unusual weather conditions clinical disease could occur at any time, the majority of cases of clinical disease occur from November through March. In most parts of Michigan, September and October pastures are most likely to be contaminated with snails containing infective larva.

Brain worms only
Once infective snails are ingested by deer the meningeal worm larva migrates through the intestinal wall of the deer and eventually moves to an area just outside of the deer's spinal cord. In white-tailed deer the meningeal worm never actually enters the spinal cord—this is why deer do not seem to be clinically affected by the parasite. Much as a hen would lay her eggs in a nest and hatch her brood, the meningeal worm matures in this area (adjacent to the spinal cord), produces eggs, and these eggs incubate and hatch into larvae. As these larvae mature, they migrate to the lung and are coughed up into the mouth of the deer and swallowed. The larva that enter the intestinal tract (swallowed) are excreted in the manure and ingested by snails. The entire process—from ingestion by the deer to ingestion by the snails—encompasses about 3 to 4 months. Therefore, contaminated pastures will usually not occur until 3 to 4 months into the grazing season. For this reason, transmission to sheep is generally thought to be sometime after September 1st each fall.

Continued on next page

Different Types of Hosts

Definitive Host: the animal in which the parasite completes its life cycle—reproduce by laying eggs. For the meningeal worm, this is the deer.

Intermediate Host: Animal in which the parasite lives and grows, but does not complete its life cycle. The intermediate host is a necessary part of the parasite's life cycle. The parasite usually undergoes changes living here that allow it to become infective to the next host—either a definitive host or the hapless dead end host. For the meningeal worm, the intermediate hosts are certain land-dwelling snails and slugs.

Dead End Host: An animal in which the parasite lives where it cannot complete its life cycle. This is a dead end for the parasite—and sometimes the host. A dead end host is also referred to as an **aberrant** (unnatural) **host**. For the meningeal worm, the dead end hosts are goats, sheep, moose, elk and llamas.

Life Cycle of *P. Tenuis*

Adult worms living in White-tailed deer's brain and spinal cord lay eggs.

Eggs hatch into larvae.

Larvae migrate from deer's nervous system to deer's lungs.

Deer coughs up larvae and swallows them. Larvae pass through deer's digestive system (unharmed) and pass out with feces.

Snails eat feces containing larvae. Larvae grow inside snails.

Goats eat grass or browse containing snails.

Parasite migrates to the inside of the goat's brain and spinal cord.

Parasite matures and wanders through goat's nervous system causing damage until either the goat's system walls off

Meningeal Worms/Liver Flukes
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In contrast, when "dead end" or "aberrant hosts" (sheep, goats, moose, elk, llamas, and other exotic species) ingest infected snails the meningeal worm larva actually migrates to the inside of the spinal cord or brain. Once arrived, the larva mature and wander aimlessly through the nervous tissue until they are "walled-off" and killed by the body's defenses or the animal dies. The final destination of the parasite often involves the area of the spinal cord controlling function of the rear legs. This is why loss of function to the rear limbs is a common clinical sign. The resulting swelling and damage to the spinal cord produces the neurological deficits associated with meningeal worm infection. While reproduction does not occur in dead-end hosts, producers should remember that even individual worms migrating through the central nervous system can cause major damage to the spinal cord or brain!

Deer flukes only
The life cycle of the deer fluke is almost identical to that of the meningeal worm, except for the fact that the organ targeted by the parasite is the liver. When deer are infected, the larva migrate to the liver and reproduce, causing very little damage in that area. This is where the fluke winters. However, when sheep are infected, the flukes migrate to the liver and grow for 12 to 16 weeks, often becoming bigger than your thumb. All the while they are growing they continue to "swim" through the liver tissue causing tissue damage. Often they rupture blood vessels in the liver, causing sudden death (acute blood loss) or compromise liver function (chronic). Sheep often die from secondary clostridial disease initiated by the damaged tissue tracks from the migration. This disease, caused by Clostridium novi, is often referred to as Black Disease—due to the black color of the liver. Producers in endemic areas often vaccinate against Black Disease.

The life cycle of the deer fluke, similar to the meningeal worm, requires the same 3 to 4 months of development time from the onset of grass and snails each spring—before infected snails are capable of infecting sheep. Again, sheep usually come in contact with infected snails after September 1st each year. Clinical signs of deer fluke disease usually do not develop until flukes grow to what is known as the 8 to 12 week stage (related to the time after ingestion by the sheep). Thus, clinical signs generally develop during November through March.

When do clinical signs appear, and how do weather conditions and management practices affect onset?

Brain worms and deer flukes
In Michigan, it is unusual for clinical cases of deer fluke or meningeal worm infection to occur prior to September or after March. Normally, the majority of cases are concentrated in December, January and February. However, variations in weather conditions and grazing practices can greatly affect the onset, distribution and cessation of new infections.

Meningeal Worm

Taxonomic Classification
Metastrongylid nematode, Parelaphostrongylus tenuis (meningeal worm)

Definitive Host Spectrum
White-tailed deer (natural)
Elk, moose, caribou, black-tailed deer, red deer, llama, sheep, goats (accidental)

Intermediate Host
Snails and slugs— not as host specific as trematodes

Geographic Distribution
North America where white-tailed deer are found—primarily east of the Rocky Mountains

Morphology
Adults - 39 - 91 mm (1-1/2" - 3-1/2")
Larvae - 348 microns, with a dorsal spine near tail

http://www.missouri.edu/~vmicrorc/Nematoda/Strongylids/Metastrongylids/Ptenuis.htm

As discussed earlier, it normally takes about 3 months for either the meningeal worm or deer fluke to complete one life cycle in the deer and another month to produce numerous infective larvae in a population of snails. Grazing season in Michigan rarely begins prior to May 1st, therefore, it is unlikely that pastured sheep or goats will ingest any quantity of infected snails prior to September 1st each year. Once ingested by the sheep, larva of the meningeal worm or deer fluke must migrate from the intestine to the central nervous system (long ways for a parasite) or from the intestine to the liver (short time) of the infected animal. For the meningeal worm this migration can be as short as 10 days or as long as three months. Deer flukes generally reach the liver within several days after ingestion.

Considering these pertinent facts, the following assumptions can be made:

1. Large numbers of infective snail populations for either parasite species are unlikely to develop prior to September 1st.

2. Clinical signs of meningeal worm disease are unlikely to develop until at least 10 days after September 1st. Clinical signs

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Meningeal Worms/Liver Flukes
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of deer fluke disease are unlikely to develop until at least 6 to 8 weeks after ingestion of infected snails.

3. After a hard freeze infective snails disappear from pasture. In some years this freeze may occur as early as November 1st.

4. If clinical disease generally occurs 10 days to 3 to 4 months after ingestion of infective larva, new cases of clinical disease are unlikely (not impossible) after March

5. Yearly cycles of infection often occur. Clinical cases appear to be more common when an extremely wet summer is followed by a long, warm fall.

How can I protect my sheep and goats from meningeal worm or deer fluke infection?

We have briefly discussed the life cycle of the meningeal worm and the deer fluke in the deer, snails and aberrant hosts such as sheep. Utilizing this information, we should be able to determine what management practices will help to prevent infection in Michigan sheep.

Key Prevention Practices

Grazing management and strategic treatments
Grazing management and strategic treatments are the keys to preventing infection in Michigan. If swampy areas are to be pastured, if possible they should not be pastured by sheep after September 1st (August 15th might be a more conservative date). Many farms contain combination pastures of mixed high ground and swampy swales. If the pasture contains small areas where snails, slugs, swamp and deer coexist, then sheep should be fenced out of these areas. This practice is often the most sensible and economical preventative approach. Remember, a small finger-like projection of swamp may be the only source of infection in a 40 to 50 acre pasture. Fencing sheep out of this area is usually a more sensible alternative than repeatedly administering preventive medications.

Sheep and goats that are removed from infected pastures should be strategically dewormed with ivermectin or fenbendazole (for meningeal worms) or albendazole (for deer flukes). If treatment (discussed later in the article) is to target preventing meningeal worm infections then sheep can be medicated when they are removed from suspect pastures. If deer flukes are the issue, then treatment with albendazole should occur twice: 1) at removal and 2) again at six weeks after the first treatment. Albendazole is not effective against immature flukes (flukes less than 4 to 6 weeks old). Therefore, the second dose is needed 6 weeks later to treat immature flukes that were unaffected by the first dose. Farms that raise both cattle and sheep should also try to utilize cattle to graze swampy pastures after August 15th or during the entire grazing season. Cattle do not appear to be affected by either species.

Deer control?
Deer control is another not-so-practical method of reducing problems. The amount of deer needed to induce an infective population of snails is not known. Therefore, hunting pressure is not likely to influence infection. Fencing deer out of the area is usually cost prohibitive and swamp water and snails do not respect boundary fences.

Snail control
Snail control could, theoretically, reduce the likelihood of infection in sheep. While snail control products are available in Europe they are not currently approved for use in the United States. The environmental impact of these products makes their future approval unlikely.

How will I know if my sheep are infected with meningeal worms, and what can be done to treat them ? Animals infected with meningeal worms may exhibit a number of varying neurological signs. The severity of these signs will be dependent upon the number and location of parasites in the spinal cord. Larvae moving through the spinal cord can cause mild lameness in one or more legs, or total paralysis. If only the spinal cord is involved the affected animal will usually appear bright, alert, and have a normal appetite. Only the gait may be altered. A weakness or dragging of the rear legs is often noted. This often progresses to paralysis of the rear limbs with a classical "dog sitting" posture. If meningeal worms enter the brain, blindness and circling can develop, but this is unusual. Other diseases such as polioencephalomalacia, listeriosis and white-muscle disease need to be considered as alternative causes of similar clinical signs.

Diagnosis of meningeal worms
Diagnosis of meningeal worm infection is usually based upon clinical signs, seasonality and environmental and geographic conditions conducive to the life cycle of the parasite. Confirmation of the disease, however, is difficult. Currently, there is no safe, convenient and economical antemortem test for meningeal worm infection in sheep. Flock confirmation is based upon histopathological (fixed sections of spinal cord are viewed under a microscope) identification of: 1) the actual parasite in the tissue or 2) parasite migration tracts in the spinal cord of affected individuals. Confirmation involves a postmortem examination which includes removal and sectioning of the spinal cord.

What can I use to treat animals infected with meningeal worms? Meningeal worms are already present in the spinal cord when clinical signs are apparent. For this reason, treatment is usually unrewarding and "after the fact". Sheep producers should view diagnosis and confirmation as a warning to prevent further losses in the remainder of the flock. Flock treatment of affected and non-affected animals should involve the following:

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1. Removal of exposed animals from affected pastures
and
2. Immediate deworming of the entire flock with either fenbendazole (5 cc of a 10% solution/100 lbs of body weight, orally, non-approved use) or ivermectin (1 cc of cattle strength injectable/110 lbs of body weight, injected subcutaneously, non-approved usage), or ivermectin sheep drench as labeled on the approved product.

Sheep that are exhibiting clinical signs of meningeal worm infection may occasionally respond to treatment. This is the exception rather than the rule and generally there will follow prolonged nursing care. In most cases, nursing care is impractical. Practitioners also often use 2X to 5X doses of ivermectin for treatment.

What if I can't remove my sheep or goats from infected pastures? For some producers, removal of animals from infected pastures may be impractical. If removal can not occur, the following treatment regimen may help control losses.

1. Strategic de-worming with ivermectin in-injectable every 20 days until a hard freeze and sheep are removed from pasture.
Or
2. Daily strategic deworming with a medicated salt mix or salt block containing fenbendazole. Although not approved for sheep, commercial cattle salt blocks containing fenbendazole ("Safeguard") can be used in a free-choice feeding program.

It is very important to remember to continue these medicated routines until animals have been off the contaminated pasture for 10 days.

How will I know if my sheep are infected with deer flukes and what can be done to treat them ? Animals infected with deer flukes generally present the owner with sudden unexplained deaths during the late fall and early winter. These deaths generally result from blood loss following migration of a fluke through a liver blood vessel. A certain portion of the flock may also exhibit as "poor doers". The liver functions as a filter for many of the toxins and by-products produced in the body. When compromised, infected sheep often become more susceptible to other diseases. In feedlot lambs that arrive in the early fall, the sudden losses usually occur after the major losses from shipping fever have subsided and lambs are well on feed. Furthermore, lamb deaths are limited to groups of lambs from fluke infected areas. Postmortem exam generally reveals an abdomen full of thin, blood colored fluid and dark black tracks in the liver. Large, flat, flukes are also commonly observed and a generalized peritonitis is present.

What can I use to treat animals infected with deer flukes and how do I prevent losses?

Active cases, i.e. dead animals with confirmed deer fluke infection
Currently, there are no approved products for treatment of deer fluke infections in sheep. Only albendazole (Valbazen), fenbendazole (Panacur & Safegard) and clorsulon (Curatrem) are known to have some affect on mid-aged to mature flukes (>6 week old flukes). Clorsulon is not believed to be as effective as albendazole, so currently albendazole (at the bottle dose 4cc/1001bs) is considered the drag of choice. Neither drug is 100% effective, so some residual cases will remain even after treatment.

Breeding flock
When confronted with midwinter deaths due to deer fluke infection, affected animals are generally 8 to 12 weeks away from infected pastures and harboring 8 to12 week stage flukes. Single Valbazen treatments will be reasonably effective on these mature flukes. However, when breeding replacements are involved ewes should be treated twice—about 4 weeks apart. In severely infected flocks it also may prove useful to vaccinate ewes for Black Disease and treat prophylactically with long acting penicillin. Valbazen is also not labeled for use during the first 30 days of pregnancy.

Feedlot lambs
Valbazen (albendazole) is a good choice of medication for all lambs arriving at the feedlot. This alleviates the risk of missing some fluke infected lambs with an ineffective dewormer. However, some lambs from infected areas will be arriving fresh from infected pasture. Treatment upon arrival may treat resident 6—8 week old flukes, but will likely prove ineffective on less mature flukes. If lambs arrive from fluke areas, they should be treated upon arrival and again in about 6 weeks with albendazole. If it is known that lambs have been off of pasture for at least 6 weeks, one treatment may suffice.

Pastured animals
Treatment of pastured animals will be dependent upon the time of year and level of exposure. Most endemic fluke areas have early snows that run lambs off pasture by October or November. As mentioned earlier, deworming once with albendazole—upon removal from pasture—does not address the immature flukes missed by the treatment. Generally 2 treatments with albendazole is better—once at removal from pasture and again in 6 to 8 weeks is a good practice.

Prevention of deer fluke infection
Prevention of fluke infection generally centers around preventing contact on pasture. In endemic areas this is often impractical unless contaminated areas can be fenced or grazed by other species. Prevention generally has to involve a 2X serial fluke treatment with albendazole at removal from pasture and 6 to 8 weeks later.

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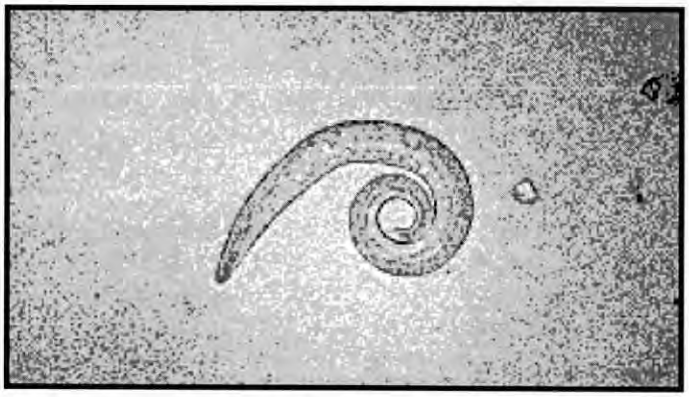

Meningeal worm

Table 1_Included in this table are various diseases that can look similar to "brain worm" infestation. Also listed are the target species that are susceptible to each of the diseases.

	Species		
Disease	Llamas and Alpacas	Sheep	Goats
Listeriosis	X	X	X
Caprine Arthritis-Encephalitis			X
Scrapie		X	Rare*
Rabies	X	X	X
Trauma	X	X	X
Copper Deficiency	X	X	X
Vitamin E/Selenium Deficiency	X	X	X
Spinal Cord or Brain Abscess	X	X	X
Polioencephalomalacia	X	X	X

A Goat Owner's Experience With Meningeal Worms

By Kimberly Reichart, Dayton, Maryland

I live in Dayton Maryland and currently own two three-year old cashmere wethers. In the beginning of December, I was in my barnyard on my way to feed my horses. I turned around at one point to greet my three-year old wether "Hopper" who I had just seen run down the hill five minutes before. When I turned around I found him lying on the ground on his back with a dazed look on his face. Startled, I helped him up and checked him for injuries. Finding none, I let him go. He got up, shook himself and ran away.

Over the next three days he periodically showed either slight weakness or a little tremor in his hind legs, but all of his vitals were normal. I didn't know what to make of it in the absence of other symptoms. On the fourth night I went down to the barn around 11 pm and found him unable to get up, but still normal otherwise, including appetite.

Unable to find anything resembling these symptoms in my two books on goat care, I turned to the Internet. An exhaustive word search of his symptoms led me to an article on meningeal worms in llamas, with a brief reference to goats. Everything seemed to fit, then I found the above article which gave even more details and really seemed to describe my goat.

I brought in my vet the next day. He prescribed five days of steroids and ivermectin (injections) and also fembendazole orally. It has been three weeks since those injections. He has continued to eat normally throughout but while he has movement in his hind legs, he is unable to stand unaided.

I have put him periodically in a sling and have massaged his leg muscles twice daily. He is also receiving a multi-vitamin and plenty of TLC. I have decided to give him another week and then I will probably have to put him down. He is living in a stall, but it has finally turned cold in Maryland after a warmer than normal fall and I will put him down before I let him freeze to death.

It seems that prevention is the best "treatment" for this disease. Worming with ivermectin as often as once every two weeks from late summer through early winter is recommended by some articles I have found on the Internet. Control of the intermediate host is also recommended. There are lots of wildlife that eat slugs including toads and ducks. I am going to start a flock of Khaki Campbells in the spring. They are prodigious slug and snail eaters and do not require a pond. They are available at many online hatcheries. Welch Harlequins are also good slug eaters. My farm is run with as few chemicals as possible, so pesticides are not an option.

I sincerely hope that my experience will save other owners of this magnificent breed the heartache that my farm has experienced this year.

Latest update on Hopper (January 14, 2002): Hopper continues to improve. His appetite has come raring back, and he seems to

Timeline of Events For Sheep and Goats

From the USDA APHIS
www.aphis.usda.gov/vs/ceah/cei/1sheep&g.pdf

1940's

- First reported case of scrapie in the US

1950's

- National Wool Act (1954)—Wool declared a strategic material. Act designed to increase production of wool resulting from the need during WWII and the Korean War to import half the wool required for military uniforms. Includes mohair, live lambs, and shorn wool.
- Scrapie Eradication Program

1960's

- Wool removed from Pentagon's list of strategic material
- Long term downward trend in sheep inventories begun

1970's

- Transgenics pioneered. Foreign section of DNA placed into an embryo to produce an animal with a trait determined by the inserted code.
- First of decades of surveys initiated to determine reason for declining sheep numbers.

1980's

- Goat cheese becoming popular—domestic production grows from nearly zero to 600 tons annually (80 tons more than is imported) within a decade [3]
- BSE first reported in England.
- "Pharm" animals—genetic modification of sheep and goats to produce proteins of therapeutic value in their milk.
- First cashmere goats imported.

1990's

- Hello Dolly—cloned sheep
- BSE becomes an issue for food safety
- NAFTA—Mexico will eliminate tariffs on US exports of live sheep, mutton, and lamb.
- National Wool Act phased out in part to appeal to public criticism of government spending; 1/3 payments go to Angora goat producers who export about 80% of their product, 1/2 payments go to top 1% of producers.
- Voluntary Scrapie Flock Certification Program established.
- Sheep numbers small enough to be considered "minor species" by FDA (for all issues except food-safety). Easier to get approval for using drugs already approved for use in major species such as cattle
- Underlying truism for all surveys regarding decline

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Sheep & Goats—Timeline of Events
Continued from previous page

in sheep numbers: profitability is too low. Reasons vary—inefficient processing and lack of progressiveness leading to higher retail prices and lower farm-level prices is one seemingly major one[4].

• Consumer surveys reflect that more people would buy lamb if it were in more convenient forms, and would try free lamb recipes if available—many consumers don't know how to prepare lamb[4].

• Dairy goat business produces about 24,000 tons of goat milk annually, yet about half of the states have no specific goat milk health regulation[7].

• First true meat goat imported (South African Boer). Dairy, Angora, and "Spanish" goats also used for meat—causes large diversity in production quality and carcass traits. Probably a serious impediment to orderly production and marketing. [6]

Trends

• Increasingly consumer driven market—uncertainty—lamb industry needs to focus on convenient, high quality, user friendly cuts.

• Slow, but persistent increase in goat production (goats in Texas are increasing at a rate of 9%/year)[1]. Since the majority of demand for goat meat comes mostly from the Hispanic and middle eastern ethnic groups, and since it is expected that these populations will grow in the U.S. in the coming years, the demand for goat meat will probably continue.[5]

• Decline in the sheep industry since WWII may continue, mostly as a result of decline in consumer demand.

• Continued withdrawal of government from price supports to farmers.

• Potential for export challenged by increased competition with foreign countries which are scrapie free.

• As agricultural lobbies become less powerful, politicians will respond more to environmentalists and recreationalists with regards to use of public lands (to graze or not?, to allow cheap leases or not?)

Uncertainties For The Future

Price

• Competitive disadvantage of goat/sheep milk vs cow milk. Gourmet restaurants, and deli can demand higher price—effective marketing can increase this.

• Competitive disadvantage of lamb verses beef. High retail price, inconsistent quality.

• Increasing popularity of goat cheese; are consumers willing to pay the higher price of goat cheese?

Health

• Increasing awareness of healthier foods—goat milk has more protein and less cholesterol, and less saturated fat overall—has more "benign" saturated fat[3].

• Increasing awareness of milk allergies may increase demand for goat milk.

• "Pharm Animals"[2]—"four legged drug factories for use down on the pharm" use as research animals—high-tech genetic research creates transgenic animals for use in drug production. The question is: are the proteins secreted in the milk of these animals safe and effective in humans?

• Potential for export of seedstock—US has a diverse gene pool, but may have to compete with countries which are disease free. This will increase interest in certifying flocks to be disease free, i.e. unknown prevalence of Johnes Disease

• Public awareness of scrapie and their concern for potential zoonotic infection could further impact demand.

• Since the majority of dairy goat milk production is not for commercial purposes , [6] how would Federal quality assurance programs (control of zoonotic infections) be implemented and controlled? How important might this market be in terms of disease control? Male kids from this industry would be sold to slaughter.

Other

• Use of sheep/goats for noxious weed control as restrictions on herbicide and pesticide use become stricter, and for range improvement—sheep reseed forest lands, and fire control since they eat chaparral and other undergrowth.

• Will need to add value to products (ground lamb, and lamb trimmings), in order to increase demand for parts of the lamb that are under utilized and under valued.

• As consumers spend more time eating outside of home, fast food becomes increasingly important—lamb has not been on the menu. Potential for expansion of this industry with use of value added products such as ground lamb burger seasoned to taste like gyro sandwich.

• Cloned animals—where will this go? Could this lead to a narrowing of genetic diversity with identical susceptibility to disease?

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Pre/post Kidding Preparations For Dairy Goat Does and Kids

Prepared by Dick & Anne Pigman
Wooden Bridge Ranch, Buellton, CA

Pre-Kidding Preparation

Vaccinations/shots (30 days prior to kidding)
1. 2cc—intramuscular—Combination: Perfringens CD & Tetanus toxoid
2. 1cc/40 lbs—subcutaneous—B0-SE (Vitamin E and selenium).

Grain/training (15 days prior to kidding).
1. Start does on goat ration 1/2 cup—gradually increase to 2 cups. When doe comes into milk, feed grain at the rate of 1 LB per day for maintenance plus 1/2 pound for every LB of milk produced. Divide total by 2 for amount to feed at each milking.
2. Feed doe on milk stanchion—start training by handling udder. Make it a happy time with lots of praise—especially important for first fresheners.

Kidding pen/supplies & equipment
(5 days prior to kidding).
1. Clip doe with #10 blade around tail and vent, down back legs and escutcheon, and under belly to navel.

2. Prepare kidding pen—bed with clean dry straw.

3. Collect kidding supplies:
    1. bucket for warm water
    2. betadine or provodone scrub
    3. surgilube or KY jelly
    4. 7% iodine & small cup
    5. OB loop
    6. newspaper—separated into single sheets
    7. scissors
    8. dental floss
    9. molasses or dark karo
    10. paper towels
    11. udder wash
    12. clean towels
    13. nail brush & nail clippers

4. Get milking equipment ready.
    1. stainless steel bucket & strainer
    2. gauze milk filters
    3. teat dip
    4. paper towels
    5. scale
    6. record sheets
    7. pasteurization equipment
    8. thermometer
    9. detergent

5. Collect kid feeding supplies.
    1. 16 Oz pop bottles
    2. Nipples—lambbar and pritchard
    3. bottle brushes
    4. funnel
    5. supply of heat treated colostrum

Kidding Preparation

Phase I—Early Labor
1. Isolate doe on her early due date (145th day)
    1. normal gestation is 145 - l55 days
    2. may be necessary to change bedding if doe is in kidding pen more than a day
    3. favorite time of day for delivery is late afternoon or early evening

2. Signs of early labor!
    1. appears restless
    2. eyes luminous
    3. smells the ground
    4. paws the bedding
    5. looks behind her
    6. stargazes
    7. hollows out
    8. tail lifts up
    9. rises and lies down frequently
    10. udder begins to fill
    11. vulva becomes flabby
    12. white discharge

Phase II—Actual Delivery
1. Prepare the doe.
    1. wash genital area with udder wash
    2. remove water bucket so babies won't drown
    3. stay with the doe in case she needs help
    4. watch color of discharge carefully

2. Signs of final labor!
    1. copious discharge
    2. strong labor pains—about 2 minutes apart
    3. ears stand out—lips curl
    4. doe begins to strain
    5. fluid filled bubble may appear
    6. water may break
    7. second bubble may appear
    8. feet and or nose may become visible

3. Normal presentations: The doe will usually deliver either of the following presentations without assistance:
    1. head lying on the forefeet with the chin about the knees and kid's back toward the doe's back.
    2. both rear legs in the birth canal with kid's back toward the doe's back.

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Pre/Post Kidding

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Phase III—Clean Up

1. Delivery of the afterbirth
    1. usually occurs within 3-4 hours
    2. may take as long as 12 hours
    3. if not hanging from the doe partially delivered look around in the bedding
    4. doe will sometimes eat the afterbirth
    5. if doe does not clean, may need infusion and/or veterinary assistance

2. Discharge of excess uterine fluid
    1. clear to slightly bloody discharge is normal
    2. may occur for 2 to 3 weeks after delivery
    3. if discharge is pus-like or reddish in color and/or increasing in volume, may require antibiotics and/or veterinary assistance

Problems With Delivery

1. Possible signs of trouble
    1. doe repeatedly starts pushing hard but gets up and stops labor, then lies down and starts again
    2. doe repeatedly gets up and down and arches her back and elevates her rear end as though trying to line up the babies
    3. discharge is rusty red and beginning to look septic
    4. parts of a baby are visible but doe is unable to deliver in spite of straining very hard
    5. doe is in hard grinding labor for more than 30 - 45 minutes with no results

2. Determine problem
    1. wash does vulva with mild soap and water
    2. wash your hands and arms and scrub finger nails well
    3. lather hands with betadine scrub and squeeze a generous ribbon of surgilube on the fingers
    4. have an assistant hold or restrain doe gently
    5. enter the vagina and dilate if necessary
    6. feel and identify the parts of the kid that are in the birth canal
    7. determine the problem and the corrective action necessary to rectify

3. Abnormal presentations
    1. <u>head first with one foreleg</u>; can be delivered this way but easier on the doe if you reach in and find the other leg and carefully pull it forward so the head is resting on both legs. The kid should deliver easily now. Just be sure the head and legs belong to the same kid.
    2. <u>head first with no legs</u>; cannot be delivered this way. Similar to the previous case, but you will probably have to reach in and push the kids head back to make room for the legs. Slide your hand along the head and neck until you find the shoulders, then locate the feet and gently bring them forward with the head resting on the legs. Kid should deliver easily now but you may need to help pull.
    3. <u>breach position with hocks first</u>; cannot be delivered this way but easy to correct. Just reach in and find the feet and carefully pull them forward so both rear feet are together and extended through the vulva. The kid should deliver easily now.
    4. <u>breach position with rump and tail</u>; similar to the previous case; but you will probably have to reach in and push the kids rump back to make room for the legs. Slide your hand along the rump until you find the legs, then locate the feet and carefully bring them forward so both rear feet are together and extended through the vulva. The kid should deliver easily now but you may need to help pull.
    5. <u>front feet first with head upside down</u>; can be delivered this way but may be easier on the kid if you rotate the entire kid so that the kid's back is upward toward the doe's back. Sometimes it is hard to turn the kid around if the feet and/or head are already visible. Just make sure that the kid curves around the doe's pelvic arch as much as possible even if it is slightly twisted. You will probably need to help pull the kid.
    6. <u>feet first with head thrown back;</u> cannot be delivered this way. This is probably the most difficult of the abnormal presentations to correct, especially if the doe has been in hard, unproductive labor for a considerable time and/or the kid is very weak. You will have to reach in and follow along the body and then along the neck until you locate the head. You may be surprised at how long the neck is and how deep you have to go (up to your elbow). The trick is to get the head forward and keep it there! If the kid is weak the head will keep flopping back every time you withdraw your hand to pull on the legs. In this case you will need your OB puller. A rubber one is best but you can use a thin noose made of nylon cord. Carry the noose in with your hand and slip it over the kids head. Position the head on the front legs and snug up the loop. Keep tension on the puller with your free hand and

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then withdraw your hand and grasp the feet. Pull on the feet and the loop at the same time and the kid should deliver just fine. Use plenty of surgilube as this is time consuming and things start to dry out. The kid may be weak and the doe tired. See also complications.

7. two heads with somebody's feet; cannot be delivered this way. Although this situation is somewhat intimidating at first, it is fairly easy to correct. The trick is to match up the head and feet of the same goat. Usually one kid's head will be more advanced than the other so push the other back and feel along the neck to the chest and down each leg until you can locate the feet of the kid whose head is more advanced. You may have to push the other kid back quite a ways to make room to work. Then carefully bring the feet forward until the head is resting on the legs. The kid should deliver easily now but you may have to help pull the kid.

8. mismatched head and feet; cannot be delivered this way. This usually occurs because one kid is presented head first with it legs back and a second kid's feet and legs have slid under the first kid's head. Since there is no room for the second kid's head it is usually turned back along its side. You will have to push the second kid back to make room to work. S1ide your hand along the first kid's head and neck to the chest and then down each leg until you locate the feet. Then carefully bring the feet forward until the head is resting on the legs. The kid should deliver easily now but you may have to help pull the kid. When you go back for the second kid the head will usually be presented normally or will come forward easily so that it is resting on its forelegs. The kid should deliver easily but you may have to help pull the kid. If you have trouble keeping the head forward see the previous discussion on using an OB loop.

9. no presentation; necessary to determine if the doe is sufficiently dilated and the cervix is open. The os, (opening to the cervix) should be dilated at least three fingers for normal birth. If not dilated then it may be too early. Wait a while and check the doe again. You can't hurt the doe by checking. If the cervix is open and all you feel is a side or ribs the kid is probably dead, but there may be live kids behind it: Push the dead kid back until you can turn it so that it is presented front feet/head first, or hind feet first. Use plenty of surgilube as the dead kids seem to be dry. You will have to pull the kid since the doe will probably not push very hard.

4. Possible complications
    1. infection; if invasion has been extensive or prolonged, may need antibiotics
    2. swelling, if excessive may need analgesic
    3. tears; either by the doe or the herdsman may require antibiotics and an analgesic
    4. depression; if severe, may need a lot of comforting
    5. an exceptionally traumatic delivery may require several days of treatment including intrauterine infusion. May need to call your veterinarian.

Post Kidding Preparations

1. Care of the doe..
    1. Give the doe a warm drink of water to which you might add a little molasses or dark karo.
    2. Keep the doe in a confined area until she cleans.
    3. After the doe has rested a bit take her into the milk room, get her up on the milk stanchion and milk out the colostrum.
    4. You may have to help her up on the stanchion. If the doe has had a really rough kidding, you may have to milk her flat.
    5. After she is milked out, you can medicate her if necessary.
    6. Keep the milk separate for three milkings as it still contains colostrum in decreasing amounts.
    7. Watch the doe's grain and water consumption carefully for the next couple of weeks. If she goes off feed or seems depressed you have to worry about ketosis. If you suspect ketosis, ask an experienced herdsman or your veterinarian for advise on treatment.

2. Care of the kid
    1. Clear the newborn kid's nose and mouth of any fluid. Then dry it off with single sheets of newspaper. Later you can use a towel but get as much of the slime off as you can with newspaper. Some herdsmen wash the kids in warm water in the sink and then blow dry them with a hair dryer. Good idea if you have the right setup in your barn. If the kid seems weak or chilled use a blow dryer or rub vigorously with a dry towel. Place the baby in a box of clean straw and put a heat lamp on it until it is dry. Kids should be trying to stand up within 10-15 minutes.

    2. Dip the naval with 7% strong iodine. Use a little cup

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and keep it off your hands. It is strong! Repeat two or three times within the first hour to make sure the entire umbilical area is covered. If the umbilical cord is bleeding, tie it off with dental floss about 1 and 1/2 inches from the body. If the umbilical cord is too long, tear it off about 1 and 1/2 inches long. Use your finger nails to tear it. Do Not Cut with scissors as it tends to bleed. Redip with strong iodine.

3. The newborn kid will need 4 oz to 8 oz of heat treated colostrum in the first 4 hours. Keep coaxing the newborn to nurse. They may do better if it is quiet and there are no distractions. Let the baby have all the colostrum it will drink usually about 4 ounces a feeding. If it refuses or drinks very little, wait 1/2 hour or so and try again. In subsequent feedings continue to feed colostrum until the baby has consumed at least one pint, and then switch to regular milk after 24 hours. Newborn kids should be fed 4 times a day for the first couple of days and then you can cut back to 3 times a day until they are consuming a least a pint at a feeding. Then you can cut them down to 2 times a day.

Giving new babies their first bottle(s) is sometimes a trial. Just be patient and keep trying until you get the baby to nurse. It is usually easier to hold the baby on your lap for the first few feedings. Most of them will start sucking right away but you may have to pry their mouth open to get the nipple in. Just remember, the baby is programmed to reach up to nurse its mother so you need to elevate the bottle and make the baby reach for it. Keep some tension on the nipple by pulling the bottle slightly away from the kid. Do not push the nipple into the baby's mouth. Apparently, this does not feel natural and the baby will try to back away.

After the babies are on regular milk, give them one to two pints of warm milk in the morning and one to two pints of warm milk in the evening. It is best to feed them at about the same time every day. DO NOT OVER FEED! The babies are greedy little pigs and will eagerly drink somebody else's bottle, so keep track of who has been fed. Too much milk will make them scour. Just be sure to keep everything clean. 16oz pop bottles work fine and are cheap. Lamb nipples work well but are hard to get on the bottles. A better choice are the lamb bar nipples. They fit most bottles and the babies seem to like them. However, if you run across a kid that is weak and/or just won't take the regular nipples, you might try a Pritchard flutter valve nipple which is very soft and pliable and much smaller. You can order any of these nipples from the major mail order suppliers.

4. Baby goats are usually disbudded when they are 4 or 5 days old. It seems to bother them less when they are younger. Many herdsmen use a disbudding box and plans are available for building your own. If you are new to this procedure, it will probably be best to have an experienced herdsman do it for you at first. Eventually you will want to get your own disbudding box and iron and learn to do it yourself. Have somebody that knows how to do it coach you until you gain confidence.

5. Tattooing the babies is usually done at the same time that they are disbudded, especially if you use a disbudding box. The tattoo tongs and letter/number sets are fairly expensive so you may want to have an experienced herdsman do it for you at first. After you start producing a lot of babies, you will probably want to get your own tongs. If you share tongs and/or letters with somebody just remember that the tattoo usually bleeds and you want to be careful exchanging blood with other goats. The CAE virus is present in all body fluids of a positive animal.

6. It is really important to establish a regular prevention program to control coccidiosis in the baby kids. There are several choices among the drugs that are recommended for the control of coccidia; all of, which are off label for dairy goats. One of the most popular is Corid (20% amprolium). The usual regimen is to treat the babies monthly beginning at one month of age and continuing until they are 7 months old. Treat for 5 consecutive days and then withdraw for 21 days. The simplest way to administer Corid is to add it to the milk at the rate of 1/2 level teaspoon to a gallon. This works well for an approximate 20 LB baby being fed 1 and 1/2 pints of milk per feeding. Larger babies will require proportionately more per gallon. If the babies are weaned then you will have to prepare a drench. Add 1 and 1/2 ounces (5 1/4 level tablespoons) of Corid to one quart (32 ounces) of water and then administer 3cc of the mix for every 10 pounds goat, once daily, for 5 days.

7. The babies acquire immunity to most diseases from their mother in utero and are protected for the first 30 days their life. After that they need their own vaccinations to remain protected. Assuming the doe received the requisite vaccinations for overeating disease, tetanus, and white muscle disease the babies will need the following vaccinations/shots at 30 days, followed by a booster at 60 days and a second booster at 180 days:

1. 2cc—intramuscular—Combination: Perfringens CD & Tetanus toxoid

2. 1cc/40 lbs—subcutaneou— B0-SE (Vitamin E and selenium).

This information was put together by the Computer Science project of the Irvine Mesa Charros 4-H Club by converting information provided by Dick & Anne Pigman. Project Leader: Michael Pazzani Department of Information and Computer Science, University of California, Irvine.

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A mother's tongue is never done.

Editor's Note: As usual, you will want to confirm that your pre- and post-kidding procedures are appropriate for your farm and locale by talking to other breeders in your area and your local veterinarian. Some of the procedures in the above article are specific to dairy goats; however, there is much good information here pertaining to all breeds of goats.

Birth—Things that come before.
Before Arwen was born (see next page), first appeared a bubble. Inside we could see tips of two tiny hooves.

Birth—Things that come after.
After birth, comes the afterbirth. Various fluid-filled sacs and other assorted goodies.

Birth of Arwen—1/12/02

Above: Fancy in labor. Arwen is born shortly after. At right, Arwen's hind feet are still inside mother and umbilical cord is still attached. Mother starts cleaning her.

Above: Fancy stands. Umbilical cord breaks and the job of cleaning begins in earnest. Arwen is lively, strong and hungry. She stands within ten minutes and begins the search for food. It doesn't take long for Arwen to connect with the food source.

Calendar of Events

March 22 - 24, 2002
Fibers Through Time 2002, Central Arizona College, Coolidge, Arizona. A conference sponsored by the Arizona Federation of Weavers and Spinners Guilds. Contact: Patricia Springer, 21609 N. 145th Dr., Sun City West, AZ 85375, 623-546-1691, rjsaz@worldnet.att.net

May 4 - 5, 2002
Maryland Sheep and Wool Festival, Howard County Fairgrounds, Baltimore, Maryland. For information: PO Box 99, Glenwood, MD 21738, 410-531-3647, email: info@sheepandwool.org

May 4 - 5, 2002
Tennessee Animal/Pet Exp & Sale, Nashville, Tennessee. Horses, miniature horses, miniature donkeys, miniature cattle, zebras, camels, llamas, goats, sheep, rabbits birds, poultry, reptiles, puppies, kittens, pocket pets, and exotics. Info: Doris or Ron Williams 615-449-6827, http://www.lucky11farm.com

June 1 - 2, 2002
Southwest Montana Flock and Fiber Festival, Dillon Montana. Farm management and fiber arts workshops, wool and commercial sheep show, mohair and cashmere goat show, fleece show and sale, vendor booths, demonstrations, children's events, farm photo contest. For more information: www.gjfarm.com/Festival.html or contact: Drin Becker, phone: 406-834-3444.

June 13 - 16, 2002
Estes Park Wool Market events and workshops, Estes Park, Colorado. Workshops, livestock exhibits, vendors, cashmere goat show (June 15th), other animal shows, handspun skeing competition. For a detailed schedule, see their website: http://www.estesnet.com/

June 21 - 23, 2002
Black Sheep Gathering, Lane County Fairgrounds, Eugene, Oregon.

September 17 - 20, 2001
Third National Small Farm Conference, Albuquerque, New Mexico. Training for specialists, technicians, farm advocates/entities. Focusing on helping the survival and economic viability of small farms as an "at risk" small business operation. Sponsered by New Mexico State University, US Dept. of Agriculture. For info: Denis Ebodaghe, USDA, email debodaghe@reeusda.gov, phone: 202-401-4385.

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Marti Wall (Washington) 360-424-7935

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North West Cashmere Association (NWCA)
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Internet listing of these breeders and a link to their email addresses and homepages, if they have one, can be found on the net at: http://www.cashmirror.com/breeders.htm

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November 2001
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December 2001
Refractions—Kids and Tags, Biologics Meetings, Multi-species Grazing, What is ATTRA?, Identifying Sheep and Goats, 2001 USA Grain Production, Tag Wars—Ear Tag Choices, Paul's Sad Tale, How to Ear Tag a Goat, Goat Knoll Tag Scheme, Tags in Wrong Ears!, Meat Goat Production in Georgia, Capricorn Cashmere Sale.

Someday, we will work on an alphabetical index by subject for the entire 13+ years of CashMirror, if there is enough interest—probably not to be printed here, but available if people ask. If you have an interest in an alphabetical index by subject, tell us, please. We will use these requests to gauge your interest level.

Texas "Fences Out" Colorado Deer and Elk

News release from the Texas Animal Health Commission.

Texas animal health officials have shut the door on the importation of live elk and several species of deer from Colorado after cases of Chronic Wasting Disease (CWD), a fatal, degenerative brain disease of elk and deer, were confirmed earlier this fall in farmed elk herds in that state. CWD belongs to the family of transmissible spongiform encephalopathies or TSEs, Other similar, but unique diseases, include BSE, or bovine spongiform encephalopathy, which affects cattle; and scrapie, a disease that can affect sheep and goats.

"The TAHC issued the quarantine on the entire state of Colorado, prohibiting the entry into Texas of live elk, mule deer, white-tailed and black-tailed deer. The quarantine is to prevent exposure to CWD and will remain in effect until it is modified or rescinded by the 12-member TAHC commission. The restrictions do not include hunter-killed animals," said Dr. Linda Logan, Texas state veterinarian and head of the TAHC. Texas has not had a case of CWD, and we want to provide as much protection against this disease as possible, while maintaining safe marketing and movement opportunities."

"The TAHC quarantine on Colorado was redundant until late November, when Colorado animal health officials lifted a movement ban that had been in place on domestic elk since October. Colorado officials will continue to restrict the movement of animals from quarantined facilities and any domestic elk that originate in the northeast corner of the state, where the disease is endemic.

Dr. Wayne Cunningham, Colorado state veterinarian, said his staff has nearly completed the disease investigation. As of end of November, they have detected 11 positive elk, resulting in the quarantine of nine herds, involving about 1,550 animals. The infected herds will be depopulated, beginning in the non-endemic area of Colorado.

Veterinarians from the Texas Animal Health Commission (TAHC), the state's livestock health regulatory authority, also have traced a dozen elk that were imported to Texas from two of the Colorado herds, prior to the detection of disease.

"Colorado officials acted swiftly to notify other states when they confirmed disease in the herds. Although this is extremely unfortunate, it's an indication that the detection and reporting system works among states, and we're handling this issue quickly to prevent potential exposure to Texas hoof stock," said Dr. Logan. "It should be noted that the ranchers who had imported the elk to Texas complied with all health regulations."

Dr. Logan said, before being imported into Texas, deer and elk must meet a number of health requirements. Besides entry permits, the animals must have had a certificate of veterinary inspection issued within the previous 30 days, meet stringent tuberculosis testing requirements and test negative for brucellosis, a bacterial disease that can affect cattle. The deer and elk also must come from a state with a CWD program that requires disease reporting and which imposes movement restrictions on suspicious or positive herds. If the animals originate in a state that has CWD in its wildlife, the animals must come from a herd enrolled in a CWD monitoring program for at least a year.

"We've located all of the imported elk, 11 of which were moved to a ranch in the Panhandle, and the 12th animal, which was sent to a facility in the Hill Country," commented Ken Waldrup, TAHC veterinarian and field epidemiologist. "When our veterinarians inspected these imported elk, they had no clinical signs of CWD, which can include extreme weight loss, unusual behavior, excessive salivation, weakness, and loss of body function."

Dr. Waldrup explained that the ranchers involved have excellent sale and movement records, making epidemiology work much easier for the TAHC veterinarians. The 11 elk on the Panhandle ranch were imported from Colorado prior to l998,

Two had been killed, and two each had been transported to Pennsylvania and Missouri. One had been returned to Colorado. Because there is no live-animal test for CWD, the four Colorado-imports remaining on the ranch were euthanized Friday, November 9, and their brain tissue was submitted to the National Veterinary Services Laboratory (NVSL) in Ames, Iowa, for examination. The carcasses were incinerated as an extra biosecurity measure, Dr Waldrup said.

"We've also notified Pennsylvania and Missouri animals health officials, so that they can locate the four Colorado animals that were transported to their states," said Dr. Waldrup. "While we await the report from NVSL regarding the health status of the Colorado-imported elk, the other animals in the Panhandle herd will be quarantined. If disease is detected, we'll take appropriate measures to cull and remove animals that may have been exposed."

Dr. Waldrup said the Colorado elk taken to the Hill Country ranch also is quarantined, along with its herd mates, while negotiations are finalized for the purchase of the imported animal for testing. "Federal CWD indemnity funds are limited to $3,000 per animal, and since many of these animals are worth much more, it is difficult to let go of an animal for testing," he said. "This animal has been in Texas less than three months, so there is little chance that this animal poses a threat to the rest of its herd."

Dr. Logan explained that Colorado officials have required mandatory CWD monitoring of farmed deer and elk herds in the state since May l998, due to the incidence of the disease in wildlife in the northeastern corner of the state. The monitoring

Continued on next page

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Colorado Deer and Elk—Continued from previous page

program involves testing animals that die, regardless of the cause of death.

The TAHC offers a voluntary CWD monitoring program in Texas, encompassing all cervids, including fallow and white-tailed deer. About 20 herds are enrolled, added Dr. Waldrup. He said TAHC veterinarians are working with staff from the Texas Parks and Wildlife Department to determine ways to increase surveillance for Texas white-tailed deer raised under permit by scientific breeders.

Dr. Logan said wildlife officials in Colorado, Wyoming and Nebraska also have collected brain samples for testing from hunter-killed animals in the targeted "endemic area," involving a small portion of northeastern Colorado, southeastern Wyoming and southwestern Nebraska. Hunters are notified when an infected carcass is detected. In Wyoming and Colorado, less than one percent of the elk and less than five percent of the deer have been found to be infected. Two hunter-killed infected mule deer have been detected in Nebraska.

"At this time, there is no evidence that CWD is transmissible to other hoof stock, such as axis or fallow deer. In the endemic area of Colorado, there has been no evidence of spread to cattle, sheep or pronghorn antelope," said Dr. Waldrup. "Experiments and monitoring are continuing in the area, so the veterinary and producer community can better understand this disease, which was unknown until 1967, when it was first seen in a captive wildlife research center in northeastern Colorado," he said.

Dr. Waldrup said that the first CWD-positive farmed elk herd was detected in 1997 in South Dakota. Since then, 16 other herds have been found: five more in South Dakota; three in Nebraska, five in Colorado, and one each in Oklahoma and Montana. By late October 2001, 10 of these herds had been depopulated, six remained quarantined, and one herd had been released from quarantine after rigorous testing and surveillance revealed no further evidence of disease. He said the disease also has been detected in several farmed elk herds and free-ranging mule deer in the Canadian province of Saskatchewan.

"All animal movement and trade entails a degree of risk," said Dr. Logan. "Besides disease eradication, our main duty is to assess and reduce risks to our state's herds and flocks. We cannot construct a fence around Texas, but we can set realistic standards, testing and monitoring requirements for imported animals. After Colorado officials complete the epidemiological work on these herds, the TAHC commissioners may want to revisit the issue of the prohibition on Colorado deer and elk imports in a year or more."

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"Isn't it interesting that the same people who laugh at science fiction listen to weather forecasts and economists?"

...Kevin Throop III (whoever he is)

"I'm always in favor of keeping dangerous weapons out of the hands of fools. Let's start with typewriters."

...Frank Lloyd Wright (1868-1959)

"Man is the only animal that blushes—or needs to. "

...Mark Twain

"Reality is merely an illusion, albeit a very persistent one."

...Albert Einstein

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