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PREGNANCY TOXEMIA OF EWES, DOES, AND BEEF COWS

Joseph S. Rook, DVM

Pregnancy toxemia is a metabolic disease that commonly affects pregnant ewes and does during late gestation. The terms *twin lamb* or *kid disease, lambing* or *kidding sickness, lambing* or *kidding paralysis,* and *lambing* or *kidding ketosis* are commonly used to describe the condition in sheep and goats. Pregnancy toxemia is common to both range and farm flock sheep operations in the United States and also in meat, hair, and dairy goat production systems. The disease is rare in cow/calf operations; however, under certain management conditions, brood cows carrying multiple fetuses may be affected. Independent of species, the clinical signs associated with pregnancy toxemia can occur in pregnant individuals with normal body condition, in pregnant animals that appear overconditioned, or in thin pregnant animals. Affected individuals are generally pregnant with multiple fetuses and in their last month of gestation. Clinically, cases are typically limited to older ewes and does during their second or subsequent pregnancies. The disease is uncommon in dams pregnant with a single fetus, in pregnant replacement ewe lambs or does, or in yearlings bred for their first pregnancy. In sheep and goats, unlike dairy cattle, pregnancy ketosis is much more common than lactational ketosis and occurs more commonly in highly prolific "improved" breeds.[19]

From the Department of Large Animal Clinical Sciences, College of Veterinary Medicine, Michigan State University, East Lansing, Michigan

PREGNANCY TOXEMIA—OCCURRENCE, CLASSIFICATIONS, AND RELATIONSHIP TO PRODUCTION PRACTICES

Pregnancy toxemia in ewes, does, and beef cows typically occurs during the last month of gestation. Clinical cases generally follow a period of negative energy balance resulting in hypoglycemia, increased fat catabolism, ketonemia, and ketonuria in susceptible animals. Although pregnancy toxemia of food animal species is usually included in a long list of other metabolic diseases, classification and discussion as a management/nutrition/economic–related metabolic disease is more appropriate and descriptive. Additionally, understanding the importance of nutrition, economic, and management factors in the progression of the disease is extremely important if practitioners and producers desire to develop practical flock or herd-based prevention programs. Producer-initiated feeding, management, and economic decisions involving a hypothetic ewe flock may be helpful in briefly illustrating management's relationship to pregnancy toxemia issues.

Ewe Flock Example

Traditionally, annual feed costs for the ewe flock account for about 50% of yearly out-of-pocket expenses for producers (Fig. 1). Profitability is dependent on optimizing ewe productivity within the confines of a cost-effective, practical, adequate feeding program. Commercial operations typically target cost-reduction strategies in the direction of greatest economic impact—ewe feed expense. Superficially, nutritional manage-

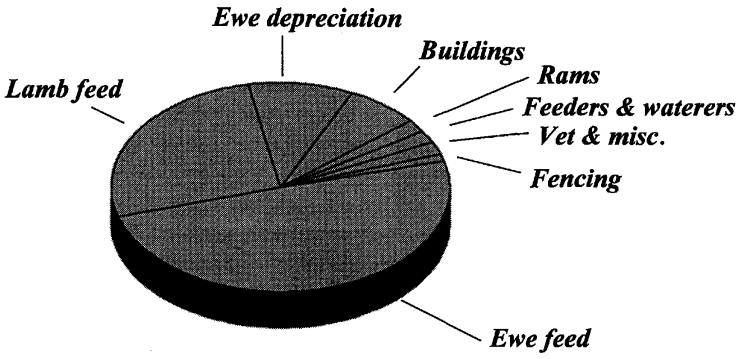

Figure 1. Proportional annual expenses for a traditional Midwestern farm flock production system. Notice the importance of ewe feed costs in the production budget. The economic success of the production unit is dependent on reducing feed costs while maintaining adequate production levels. Nutritional management decisions related to cost containment greatly affect pregnancy toxemia. (*Data from* Jordan RM, Egertson K, et al: Increasing profitability with sheep. Minnesota Extension Service, AG-FO-2925:1–4, 1986.)

ment decisions may appear solely motivated by economics; however, nutritional management connects producers and veterinarians to a mechanism that contributes to, or detracts from, flock health and productivity—all within the confines of normal production practices.

Furthermore, the potential for increased prolificacy within a flock is unquestionably linked to genetic selection decisions made by flock managers; however, realization of that potential must be coupled with adequate nutrition. Recognizing and providing for the increased nutritional requirements of late gestation should be a major concern to sheep producers and their veterinarians. Failure to provide adequate nutrition during late gestation can result in individual animal cases and flock or herd outbreaks of pregnancy toxemia.

When discussing management issues, it is also important to recognize that commercial sheep, goat, and cow/calf production systems are composed of a variety of nutritional, metabolic, genetic, physiologic, environmental, health, and management inputs that singularly or as a group influence the clinical expression of pregnancy toxemia. Independent of species involvement, the cause, prevention, and treatment of pregnancy toxemia focuses on these six production issues: (1) increased late-gestation nutritional demands associated with the developing fetal-placental unit, (2) reduced rumen capacity resulting from competition with fetal growth, (3) improper, declining, or interrupted nutrition, (4) stresses related to management, weather, transport, shearing, or predators, (5) concomitant diseases, and (6) individual animal susceptibility to pregnancy toxemia (genetic). When investigating pregnancy toxemia outbreaks, it is often helpful to focus treatment and prevention programs on these six basic production issues.

Additionally, some practitioners and authors find it useful to divide the types of pregnancy toxemia into the following four broad metabolically descriptive categories that can also be used to focus prevention and treatment programs: (1) primary pregnancy toxemia, (2) fat-ewe pregnancy toxemia, (3) starvation pregnancy toxemia, and (4) secondary pregnancy toxemia. *Primary pregnancy toxemia* is common and customarily results from a decline in the plane of nutrition during late pregnancy or management changes that create a brief period of fasting. *Fat-ewe* or *doe pregnancy toxemia (estate ketosis)* results from an overconditioning of the flock, herd, or individual during early pregnancy—sometimes followed by a late gestational decline in nutrition. Similar to the space-occupying pregnant uterus in late gestation, excessive abdominal fat can reduce rumen capacity in the abdomen. Obese pregnant goats are also more at risk for developing lactational ketosis once kidding occurs.[19] *Starvation pregnancy toxemia* involves excessively thin ewes whose condition usually results from mismanagement or unavailability of feed resources following periods of drought, heavy snows, or flooding. *Secondary pregnancy toxemia* has a more sporadic occurrence and is the result of concurrent disease in affected ewes.[16]

To understand the factors initiating pregnancy toxemia, practitioners should also recognize that clinical cases of pregnancy toxemia in sheep

and goats occur sporadically and unpredictably, even when adequate management and feeding practices are in place. When this occurs, morbidity is typically quite low, involving less than 1% to 2% of the mature ewe flock or goat herd; however, in contrast, mortality rates often exceed 80% of affected animals—especially if treatment is delayed until affected individuals are unable to stand. As a rule, infrequent cases of pregnancy toxemia in a flock or herd generally involve animals with concurrent health problems (postshearing pneumonia, age-related poor dentition, lameness, chronic pneumonia, and so forth) that spawn a cycle of anorexia, fat catabolism, and ketosis. Occurrence of sporadic, unexplained cases of pregnancy toxemia, even in well-managed flocks, also suggests that genetic lines or families of sheep and goats may be predisposed to metabolic disorders within a given management system, and progeny from affected animals should probably be excluded from flock replacements. In contrast to the relatively frequent occurrence of pregnancy toxemia in sheep and goats, twinning and related pregnancy toxemia in cow/calf operations are rare.

In sheep and goat production systems, pregnancy toxemia may also occur at a flock or herd outbreak level, characterized by the onset of numerous clinical cases of pregnancy toxemia during the last month of gestation. Clinical history typically indicates that the majority of affected animals are in their last 2 to 3 weeks of pregnancy. When flock or herd outbreaks occur, morbidity is often high, approaching 5% to 20% of the ewes or does on the premise. Mortality rates often exceed 80% of untreated individuals, and economic losses can be substantial. When confronted with a flock or herd outbreak, management history is extremely important. Increased incidence of pregnancy toxemia in sheep and goats usually follows unsuitable feeding or management decisions, sudden weather changes, or environmental or psychologic stresses that impact the production group. Somewhat dissimilar to other food animal production systems, it is important to note that the entire flock or herd may lamb or kid during a brief 20- to 30-day period. This creates a scenario whereby 80% to 90% of the animals in a given production system are at an identical stage of gestation and at risk for developing pregnancy toxemia. This concentration of high-risk individuals is a critical issue for practitioners to understand and address on a flock or herd basis. When flock or herd problems occur, the first clinical cases are merely the tip of the iceberg.

The relative contribution of management-related issues towards pregnancy toxemia is clear if one understands that most sheep, goat, and cow/calf production is designed around forage-based feeding systems. Traditionally, forage-based systems rely on the economic advantages of pasture, home-stored local forages, and unprocessed grains. Likewise, housing and machinery influences on these operations are often minimal. Whereas the nutritional requirements for individual animals in various parts of the country may be reasonably similar, nutritional delivery systems are not. Nutritional stresses, rare during periods of maintenance and early gestation (50% to 75% of the year), often result

from failure of feeding programs to address elevated requirements of late gestation. Increased prolificacy (common to sheep and goats) and harsh environmental conditions coinciding with late gestation, shearing, and lack of shelter further increase nutritional demands on the ewe, doe, or beef cow. Additionally, these increased nutritional demands of late gestation often coincide with total reliance on producer-stored and supplied forages and grain. Nutritional management decisions by the producer are more at risk of inducing pregnancy toxemia than would be the same scenario in a lush pasture setting.

Additionally, a 365-day-per-year management philosophy (common to high-producing dairy and hog units) is often conceptually foreign to many sheep, goat, and cow/calf producers attuned to pasturing animals and conducting minimal feeding management for much of the production year. Philosophically, this may explain the difficulty that many sheep, goat, and cow/calf producers have adjusting feeding programs to meet the increased nutritional demand of late gestation. Feeding programs often "crash and burn" during critical high-requirement production periods, such as late gestation and lactation.

PREGNANCY TOXEMIA—A DISRUPTION OF GLUCOSE HOMEOSTASIS

The exact pathophysiology of pregnancy toxemia is poorly understood. Nutritional research and clinical experience suggest that most clinical cases of pregnancy toxemia can be prevented by matching nutritional intake with the increased late gestation requirements of the maternal-fetal unit. For example, late gestation fetal growth and pregnancy requirements are roughly 150% (ewe with a single lamb) to 200% (ewe with twins) above maintenance levels. These increased late gestation requirements are caused by the fact that nearly 80% of fetal growth occurs during the last 6 weeks of gestation.[8, 13] During late gestation, the fetal-placental unit is supplied almost entirely by glucose and lactate, consuming nearly 30% to 40% of maternal glucose production.[2, 14] Fetal glucose requirements drastically expand just as rumen capacity is being compromised by the developing fetus, placing additional demand on the maternal glucose pool. Furthermore, fetal uptake of glucose appears to function independently of blood glucose regulation in the dam, and as glucose production in the dam declines, fetal glucose demands remain satisfied. This unidirectional flow of glucose toward the fetus occurs at the expense of glucose homeostasis in the dam. Although declining blood glucose levels are unarguably detrimental to the dam (and ultimately the fetus), this fetal glucose safety mechanism ensures short-term fetal viability.[4]

Ultimately, disruption of the glucose homeostatic mechanism in the late pregnant dam is theorized as the metabolic event that initiates pregnancy disease[20]; however, this theory alone fails to explain why ewes and does frequently develop low blood glucose levels (20 to

40 mg/dL) during late pregnancy, yet seldom develop the ketonemia, ketonuria, and clinical signs associated with pregnancy toxemia. Simple hypoglycemia also fails to explaiin experimental production of pregnancy toxemia–like symptoms in wethers and nonpregnant ewes.[15]

Whereas disruption in blood glucose homeostasis in susceptible dams probably triggers the symptoms associated with pregnancy toxemia, variability among animals and susceptibility to pregnancy toxemia probably results from individual differences in the dams' ability to maintain glucose homeostasis.[21] It is important for practitioners to recognize that production units contain individual animals that are susceptible or resistant to pregnancy toxemia. Research suggests that susceptible animals probably exhibit impaired insulin function in the form of insulin resistance. Insulin-resistant animals, unable to regulate glucose homeostasis during late pregnancy, may be more susceptible to pregnancy toxemia than are insulin-susceptible individuals. In theory, constant fetal drain of glucose from the dam may result in reduced insulin production by pancreatic beta-cells. Reduced insulin production would diminish the ability of the dam to respond to fluctuations in her own blood glucose levels. Impaired intravenous glucose tolerance tests, associated with pregnancy toxemia–susceptible individuals, suggest that pregnancy toxemia may be very similar to insulin-dependent diabetes mellitus in humans or type II ketosis in dairy cows (see the article by Herdt [pp 215–230] elsewhere in this issue).[17, 20] Furthermore, genetic diversity, regarding insulin resistance or susceptibility, may explain the wide variations observed in flock management that appear to precipitate clinical cases of pregnancy toxemia. Genetics and individual variation could best explain why long-established flocks with inadequate feeding practices experience very few cases of pregnancy toxemia. Perhaps, over a period of time, insulin-resistant individuals have been naturally culled from the flock's genetic pool.

CLINICAL SIGNS OF PREGNANCY TOXEMIA

A diagnosis of pregnancy toxemia should be considered whenever late pregnant ewes, does, or cows exhibit neurologic signs or motor weakness leading to death within 3 to 10 days. Differential disease considerations commonly include hypocalcemia, listeriosis, polioencephalomalacia, hypomagnesemia, trauma, parasitism, and meningeal worm infection (in small ruminants). Clinical signs associated with pregnancy toxemia include anorexia, hypoglycemia, ketonemia, ketonuria, weakness, depression, incoordination, mental dullness, and impaired vision, followed by recumbency and death. Recumbency is generally indicative of a poor prognosis. Unless compounded by severe weather stress, acute deaths are uncommon. Characteristically, affected animals linger for several days to a week before dying. As mentioned previously, animals affected with pregnancy toxemia are usually pregnant with multiple fetuses and in the last several weeks of gestation.

During the initial stages of the disease, mild clinical signs often go unnoticed. Affected animals appear sluggish, often lagging behind the flock or herd while in transit to feeding or watering areas. If observed closely, affected animals commonly approach feeders with the flock or herd, yet fail to eat. As the disease progresses, affected animals separate themselves from the group, appear blind or disoriented, wander into objects or stand in the same area, or fail to flee from approaching people, dogs, or equipment. Affected individuals are typically constipated, and teeth grinding is common. In the later stages of the disease, weakness and mental dullness increase, leading to recumbency and death. When helped, affected ewes and does will often stand, yet only walk several steps before collapsing to a recumbent position. As metabolic acidosis develops, an increased respiratory rate may mimic signs of pneumonia.[19] In the terminal phases of the disease, affected animals are unable to stand. Head pressing, muscle tremors, subtle convulsions, lip twitching, and star-gazing postures are common. Helpful diagnostic aids include a strong ketone-positive reaction on urine ketone test strips and a "fruity" ketone smell to the breath. If left untreated, recumbency usually develops 3 to 4 days after initial observation of early clinical signs, followed by death in another 3 to 4 days. Fetal death and associated toxemia are common sequelae. Recovery may occur if parturition occurs, or if the fetuses are removed by cesarean section or corticosteroid-induced parturition.[16] Ewes that do recover commonly develop dystocia at lambing and often retain fetal membranes following parturition.[1]

TREATMENT OF PREGNANCY TOXEMIA

Practitioners faced with individual animal treatment for pregnancy toxemia should first review various treatment options, costs, and prognoses with owners. Treatments generally range from inexpensive on-the-farm medical management to expensive surgical intervention. Sheep and goat owners, unfamiliar with the seriousness of pregnancy toxemia, often place unrealistic expectations on treatment outcome. Unless affected animals are of valuable breeding stock, producer concern is generally focused on the potential economic value of the unborn fetuses, not the salvage value of the dam. Production economics and practicalities often restrict individual animal treatment to inexpensive medical support until parturition, recovery, or death occurs. This is a reasonable approach. Also, producers should be reminded that prevention in the remainder of the production unit is usually more important and cost-effective than is individual animal treatment. Prevention programs generally focus on addressing weaknesses in management and feeding programs.

In hospital situations, animals with pregnancy disease are often presented in the recumbent, terminal stage of the disease, when treatment is difficult and unrewarding. If available, ultrasound can be useful in determining fetal viability. Usually, multiple electrolyte, acid-base,

and fluid imbalances are present, requiring intensive therapy that is often economically unfeasible for most producers. If practical, valuable individuals should receive intravenous replacement electrolytes, fluids, and glucose based on serum chemistry profiles. An intensive treatment protocol for a ewe or doe might involve the placement of an indwelling intravenous catheter for administration of appropriate electrolyte fluids with 5 to 7 g of glucose administered intravenously every 3 to 4 hours. Additionally, affected ewes or does might receive an intramuscular injection of 20 to 40 units of protamine zinc insulin every other day for 3 days.[10] In most ambulatory practice situations, this type of intensive, individual, glucose and fluid therapy is usually impractical, unless an owner is extremely conscientious and knowledgeable.

In practice situations, traditional therapy focuses on reversing the negative energy balance, raising blood glucose levels, and stimulating the appetite. Producers with ewes or does exhibiting early signs of pregnancy toxemia often initially drench affected animals with 100 to 200 mL of propylene glycol twice daily for several days. Some authors believe a smaller dose (60 mL twice daily) of propylene glycol is more appropriate and less likely to cause side effects.[19] Mildly affected animals often respond to simple treatment and continue their pregnancy to lamb or kid. More intensive on-the-farm treatment of ewes and does generally includes two to four times daily oral drenching with 100 to 200 mL of propylene glycol solution, corn syrup, or glycerol, along with intravenous administration of 250 mL of 20% dextrose solution or 500 mL of 10% dextrose solution. Because of solution availability and convenience of syringe administration, food animal practitioners commonly choose intravenous administration of 120 mL of a 50% dextrose solution; however, lower concentrations of intravenous dextrose (20% or 10% dextrose solution) may enable the dam to better use the product. Additionally, B vitamins and 50 to 125 mL of 20% calcium borogluconate solution are often administered subcutaneously (or added to the intravenous fluid solutions) to stimulate appetite and rumen motility in the anorectic animal.[8] Corticosteroids are also commonly used to assist in gluconeogenesis, increase appetite, and induce abortion. Similarities in the clinical appearance of pregnancy toxemia and hypocalcemia in sheep and goats suggests that calcium preparations be included in field treatment protocols. It is estimated that 20% of ewes exhibiting clinical signs of pregnancy toxemia are also hypocalcemic. It is theorized that hypocalcemia may be caused by high levels of circulating cortisol and fatty liver interference with hydroxylation of vitamin D.[1] Oral calcium gel preparations may be safer than intravenous administration of calcium. Additionally, separating affected individuals from the flock or herd facilitates observation and treatment and allows decreased competition for available feed resources.

Additionally, newer information suggests that treatment for pregnancy toxemia in ewes and does should include oral drenching (three to four times daily) with 160 mL of a commercial concentrated calf scour rehydration solution containing 45 g of glucose, 8.5 g of sodium chloride,

6.17 g of glycine, and various other electrolytes. Research suggests that this type of therapy elevates blood glucose levels higher than the levels achieved following propylene glycol drench.[16] This may be explained by the fact that hepatic generation of glucose (required for the use of propylene glycol) is compromised (poor liver function) in animals affected with pregnancy toxemia. As a variation, other authors suggest oral administration of 3 to 4 L of a commercially available calf diarrhea rehydration solution (mixed as directed) via oral gastric intubation.[8] Commercial calf scour rehydration solutions typically contain varying concentrations of glucose, sodium chloride, glycine, and other electrolytes often needed by affected ewes or does. Additionally, fluid rehydration and electrolytes would be useful in addressing the dehydration and constipation commonly observed in affected animals.

Further treatments may also include removal of the source of glucose drain on the dam. Typically, this necessitates removal of multiple fetuses either by cesarean section or chemical induction of parturition. Generally, this approach focuses treatment on salvaging the dam at the expense of her fetuses. Customarily, exact breeding dates are unknown; under normal production conditions, neonatal survival rarely occurs if parturition is induced prior to the last week of gestation. Generally, fetal removal by induction of parturition or cesarean section should be reserved for the early stages of pregnancy disease, before the affected dam's condition is irreversible or fetal death has occurred. Cesarean section may also be appropriate for animals that fail to show some response to medication within 24 hours after initiation of treatment.[19] In the ewe, a parenteral dose of 20 mg of dexamethasone sodium phosphate is commonly used to induce parturition. Lambing usually occurs about 48 to 72 hours post injection.[16] Intramuscular doses of 10 mg of dexamethasone for induction of parturition in the ewe and 20 to 25 mg for induction of parturition in the doe are also cited[5]; however, during the later stages of pregnancy toxemia, response to corticosteroid-induced parturition is variable and unreliable. It is theorized that this unreliable response to parenteral corticosteroid medications may be related to elevated endogenous corticosteroid levels.[1] In the doe, if due dates are known and it is the last week of pregnancy, a 10-mg dose of prostaglandin $F_{2\alpha}$ can also be used to abort the fetus; however, if survival of the kids is important, or breeding dates are unknown, 20 to 25 mg of dexamethasone is more commonly used to induce parturition.[19]

Although not currently approved for use in sheep, preliminary reports suggest that subcutaneous injection of recombinant bovine somatotropin, in combination with concentrated oral electrolyte and glucose drench solutions, may be more effective than drenches alone. A recent report from the United Kingdom suggests that a singular subcutaneous injection of 160 mg of a slow-release formulation of recombinant bovine somatotropin combined with concentrated electrolyte and glucose drenches may show promise for increasing both ewe and lamb survival. It is theorized that bovine somatotropin may improve the efficiency of glucose and ketone body usage at a cellular level.[1, 18]

PREDICTING PREGNANCY TOXEMIA IN INDIVIDUAL ANIMALS AND THE FLOCK

When pregnant ewes or does are placed under starvation stress, changes in blood chemistry parameters occur, yet none appear to accurately predict which individuals will exhibit clinical signs of pregnancy toxemia. Blood glucose and calcium concentrations typically decline, nonesterified fatty acids and acetoacetate concentrations increase, and urea concentrations remain normal. Although hypoglycemia is associated with late pregnancy starvation stress, the degree of hypoglycemia does not appear to predict which individuals express clinical disease. Only an impaired intravenous glucose tolerance test appears to serve as a predictive index for clinical signs associated with pregnancy disease in individual animals.[20] In practice situations, this type of testing is impractical.

Whereas the predictive value of blood chemistry tests is of little practical value for individual animals, flock or herd profiling may be useful in preventing pregnancy disease at the flock level. Furthermore, profiling may predict improper feeding practices before clinical cases of the disease occur. In the ewe flock, increased serum levels of betahydroxybutyrate (BHB) and decreased levels of serum fructosamine may be useful in evaluating the nutritional status of the flock. Serum BHB values in well-fed ewes are normally below 0.5 mM/L, with ketonuria developing when BHB values exceed 0.7 mM/L. Ewes exhibiting clinical signs of pregnancy toxemia reportedly exhibit elevated serum BHB values (normal ewes 0.47 mM/L mean concentration versus affected ewes 8.50 mM/L mean concentration) and elevated ketone body (acetoacetate plus BHB) concentrations in excess of 3.0 mM/L. A small, composite, late gestation sample of BHB values might be helpful in assessing adequacy of the feeding program.[9, 22] Generally, profiled flock BHB concentrations below 0.8 mM/L are considered normal; however, BHB concentrations between 0.8 and 1.6 mM/L suggest moderate malnutrition, and BHB concentrations above 1.6 mM/L indicate severe energy undernutrition. Ewes exhibiting clinical signs of pregnancy toxemia usually have BHB levels in excess of 3.0 mM/L. As a rule, when flock BHB levels exceed 0.8 mM/L, the potential for clinical cases of pregnancy toxemia to develop increases. Additionally, postmortem aqueous BHB concentrations above 2.0 mM/L are considered diagnostic for pregnancy toxemia.[1]

Decreased serum fructosamine might also be supportive as an early indicator of potential pregnancy toxemia, even in the case of normal BHB values. Fructosamine is a ketoamine compound formed when glucose reacts with amino groups on proteins. When protein concentrations are stable, serum fructosamine concentration is related to the average glucose concentration over the prior 3- to 4-week period. Serum fructosamine concentrations should be lower in animals with persistent hypoglycemia.[3] Although both BHB and fructosamine serum concentrations may be useful indicators of potential problems with pregnancy toxemia,

a combination of forage analysis and late gestation ration balancing is probably a more practical and realistic means of averting flock or herd problems.

NECROPSY AND HISTOPATHOLOGY

Gross necropsy findings vary according to the body condition of the ewe at the onset of disease and the initiating cause. The carcass can appear emaciated or in good condition. Sporadic cases of pregnancy toxemia are often defined by concurrent disease conditions having little to do with feeding or management practices; however, as a rule, the uterus of most affected individuals contains multiple fetuses in various stages of decomposition. Overconditioned individuals typically exhibit severe fatty degeneration of the liver (enlarged, friable, and pale yellow in color); however, practitioners should use caution when interpreting hepatic fat levels. Elevated hepatic fat levels (normal 3% fat, elevated up to 30% fat) are a normal occurrence during late pregnancy in the ewe. Additionally, during postmortem examination, the adrenal glands may appear enlarged, with hemorrhagic cortices. Signs of constipation are also common.[1, 8] In contrast, thin ewes or does may exhibit few of the above signs. Thin animals typically exhibit necropsy signs consistent with starvation. These include serous atrophy of perirenal and cardiac fat and enlarged adrenal glands. A large single fetus is not an uncommon necropsy finding in very thin ewes or does. Histopathologic investigation of the brain is rarely performed; however, neuronal necrosis along with astrocytic nuclear swelling, hypertrophy, and proliferation are reported. These findings support the theory that clinical signs of pregnancy toxemia result from a hypoglycemic encephalopathy.[6]

ECONOMIC IMPLICATIONS FOR THE PRACTITIONER AND PRODUCER

In the United Kingdom, pregnancy toxemia is estimated to affect about 2% of the national ewe flock each year. Furthermore, in severe outbreaks in the United Kingdom, pregnancy toxemia is recognized to affect up to 40% of the pregnant ewes in the affected flocks.[1] In this country, however, economic reports concerning pregnancy toxemia in the sheep, goat, and cattle population are rare. Nonetheless, the following personal observations concerning the ewe flock appear appropriate and, perhaps, could be extrapolated to other species.

Ewe Flock Example

In sheep flocks, economic losses from pregnancy toxemia result from loss of the ewe and her unborn lambs and expenses related to

treatment and prevention of the disease. Commonly, producers fail to associate fetal loss caused by pregnancy toxemia with loss of potential income; however, loss of the ewe at the extreme end of the winter feeding period is an expense that is apparent to most producers. Depending on the production system, ewe feed costs (mostly wintering costs) account for about 50% of the annual ewe budget (see Fig. 1).[7, 11] Estimated costs for each ewe lost to pregnancy toxemia (estimates for 1999 for commercial flocks in the upper Midwest) would amount to about $105 to $180/ewe, plus any incurred treatment costs. This figure would include the replacement value of the ewe ($80 to $120 for ewes delivered in the fall) and winter feed costs ($25 for grazing-oriented flocks to $40 to $60 for semiconfinement operations).

It is important that veterinarians understand producer expenses and their impact on pregnancy toxemia treatment decisions. Death from pregnancy toxemia occurs very late in pregnancy when, to a great extent, the annual cost of maintaining (wintering) that animal (except for lactation diet) has already been incurred. Affected individuals fail to generate an annual income from the sale of lambs, kids, milk, fleece, hair, or as a cull animal; however, the economic loss of $105 to $180/ewe creates an expense barrier that, except in the case of valuable breeding stock, limits individual animal treatment options. In practice, economic necessity suggests that recommendations focus on prevention of pregnancy toxemia in the remainder of the flock or herd, not treatment of individual animals.

PREVENTION OF PREGNANCY TOXEMIA: A FLOCK OR HERD APPROACH

Flock- or herd-level pregnancy toxemia issues can usually be resolved through the use of practical nutritional management protocols designed around the production requirements of the flock (herd); however, nutritional advice is useful only if producers understand the issues and are willing to comply with management suggestions. Currently, nutritional programs for the flock or herd are best described by (1) the level of production and (2) the associated nutritional requirements.[12, 13] Production segments of the calendar year are commonly divided into periods of (1) maintenance, (2) breeding (flushing), (3) early gestation, (4) late gestation, and (5) lactation. Because pregnancy toxemia occurs during late gestation, nutritional management during breeding, early gestation, and late gestation primarily impacts the disease. Also important for proper nutrition of the flock or herd are production requirements further defined by (1) the number of fetuses, (2) the mature weight of the dam, and (3) the concurrent production requirements for milk, hair, and wool, weather, shearing status, housing, and other factors.[12, 13] Generally, the lactation and maintenance portion of the production cycle are relatively unimportant to a discussion of pregnancy toxemia. To simplify the discussion of nutritional and management

issues relating to pregnancy toxemia, maintenance and lactation feeding periods are not considered.

Breeding Issues Associated with Pregnancy Toxemia

Typically, species prone to pregnancy toxemia are forage-based (pasture, crop residues, and hay) animals that, over their adult lifetime, receive limited amounts of concentrate. Grains, if fed at all, are generally limited to breeding (flushing) and late gestation, when quality pasture in most production systems is often unavailable. Maintenance and early gestation requirements for mature ewes, does, or beef cows are relatively low and usually well served by free-choice access to medium-quality forages; however, energy requirements for ewes and does increase slightly during the breeding season. This brief (about 30 days) increased plane of nutrition (primarily energy) is associated with the breeding season and is commonly referred to as the *flushing period.* Proper flushing management should create a slight weight gain in the ewe or doe resulting in a 10% to 20% increase in lambing or kidding rates. Increasing the frequency of multiple fetuses in the flock or herd affects the potential number of individuals at risk to pregnancy toxemia. During this time, animals should be handled regularly and maintained at a body condition score level of 3.

Early Gestation Issues Associated with Pregnancy Toxemia

The early gestation feeding period begins immediately after flushing and extends to about 4 to 6 weeks prior to parturition. Nutritional mismanagement during this period can create excessively fat or thin ewes or does at risk for pregnancy toxemia as they enter late gestation. To control production costs, commercial producers rarely supplement grain in the early gestation ration (unless dairy use). When grain is supplemented in the ration, it is usually because (1) hay quality is extremely poor, (2) pasture/forages are scarce and expensive, or (3) grain prices and labor supply warrant supplementation.

To reduce early gestation production costs, crop residue, stockpiled pasture, and cool season annuals are often pastured during the fall and early winter months; however, as pregnancy advances, fall and winter forages often decline in quantity, quality and availability (weather) at a time when susceptible animals are approaching late gestation. Customarily, as the fall grazing season progresses, crop residues and mature forages that are low in energy and protein may warrant some level of hay or grain supplementation. Supplementation can be useful in preventing excessive weight loss in the pregnant ewe and associated starvation-induced pregnancy toxemia. As availability of fall and winter feed sources diminishes, early gestation body condition scores (2.5 to 3) can

be maintained by daily supplementation of 1 to 2 lb of legume hay for each pregnant ewe or doe. Weather stress and lack of housing—common to larger herds or flocks—may further increase early gestation energy requirements; however, in direct contrast, overly abundant energy-dense early gestation feeds, such as turnips, rape, kale, grain-saturated corn stalk residue, corn silage, or small-grain hays or haylages, can generate excess condition in the pregnant animal. Overconditioning during early gestation frequently contributes to fat-ewe pregnancy toxemia, especially if the late gestation diet becomes marginal or is energy deficient.

A body condition scoring system is a useful tool for documenting the suitability of the feeding program. Body condition scoring should be monitored on a regular basis to ensure that (1) ewes and does are not overconditioned during early gestation, and (2) the plane of nutrition rises during the second half of gestation (Table 1). Ewes and does are generally scored on a 1-through-5 system, with a condition score of 1 being extremely thin and a score of 5 being obese. It is common for body condition scores to decline by about one-half score during the second and third months of pregnancy; however, this condition should be corrected by the time the dam enters the fourth month of pregnancy. Variations in excess of one half of a body condition score during early pregnancy can lead to overly thin or overly fat ewes during late gestation.[1] Typically, recommended body condition scores fluctuate with the major production periods of the animal (Table 1). It is important that producers and veterinarians regularly score a small percentage of the flock or herd to determine average values; however, producers who view their flock on a daily basis are often poor evaluators of body condition (gradual change goes unnoticed). It is often easier for an infrequent and impartial observer, such as a local veterinarian, to detect gradual condition changes that might go unnoticed by producers.

Early detection of body condition excesses or deficiencies is important. In general, small ruminants are capable of rapid response to dietary changes. Also, the health, production, and economic implications of overfeeding and underfeeding are especially severe if either occurs at

Table 1. SUGGESTED BODY CONDITION SCORES FOR EWES AND DOES DURING VARIOUS STAGES OF PRODUCTION (1 TO 5 SCALE)*

Maintenance	2.0–2.5
Breeding	3.0
Early pregnancy (first trimester)†	3.0
Midpregnancy (second trimester)	2.5–3.0
Late pregnancy (third trimester)	3.0–3.5
Lambing	3.5
Weaning	2.0–2.5

*Where 1 indicates extremely thin and 5 indicates obese. Variations are associated with different production periods.

†Due to fetal wastage, excessive weight loss during early pregnancy should be avoided.

Data from Nutrient Requirements of Sheep, ed 6. Washington, DC, National Academy Press, 1985; and Smith MC, Sherman DM: Goat Medicine, ed 1, Philadelphia, Lea & Febiger, 1994, pp 546–549.

a critical phase of production. Moreover, increased fecundity related to flushing may be diminished if ewes are obese (body condition scores of 4 or 5) or extremely thin (body condition scores of 1 to 1.5) prior to breeding. Producers should be encouraged to adjust pasture stocking rates or feed quality to maintain an average condition score of 2 to 2.5 during maintenance. Ewes should gain weight during flushing, but condition scores should not increase past the 3 to 3.5 level.

Late Gestation Nutritional Issues Associated with Pregnancy Toxemia

Although breeding and early gestation nutrition and management set the stage for pregnancy toxemia problems, ultimately, it is late gestation nutrition and management that initiates clinical disease. To prevent pregnancy toxemia, ewes and does entering the late gestation feeding period should not be overly thin or fat. To detect problems and allow time for feed changes, scoring during late pregnancy should occur on a weekly basis. Pregnant ewes and does maintained at a 2.5 to 3 body condition score—until about 6 weeks prior to parturition—are in ideal condition to respond to appropriate energy-dense late gestation rations. As a rule, during the last 6 weeks of pregnancy, mature ewes and does pregnant with single fetuses should increase body weight by about 10%. Correspondingly, ewes or does carrying twins should exhibit an 18% increase in body weight. Average body condition flock/herd scores should be maintained at or increased to a 3 to 3.5 score at parturition.[16]

Substantial increases in late gestation requirements and associated weight gains are all necessary for the growth of the fetus and increased fat stores and udder development related to approaching lactation. Until the final weeks of gestation, fetal growth is rather minimal; however, roughly 70% to 80% of the fetal birth weight is accumulated during the final 4 to 6 weeks of pregnancy. Additionally, late gestation nutrition influences neonatal survival rates. As an illustration, optimal individual birth weights for twin lambs born to a 150- to 160-lb commercial ewe should be about 9 to 11 lb. Newborn lambs weighing less than 7 lb or more than 11 lb are subject to increased neonatal mortality rates. Mortality observed in lighter lambs appears associated with hypothermia and starvation loss, whereas dystocia contributes to mortality in larger lambs. Late pregnancy nutritional management is useful in addressing both pregnancy toxemia and neonatal lamb loss issues—two extremely important production diseases. Interestingly, combined birth weights for lambs born to an individual ewe plateau at about 25 to 30 lb—independent of fetal numbers. This suggests that prolific ewes have an upper limit of birth-weight response to increased late gestation nutrition. In addition to late gestation body condition scoring, the average birth weight of the first few lambs born each season can be a useful

assessment tool for evaluating the appropriateness of the late gestation ration.

The magnitude of late gestation nutritional demands confronting ewes and does pregnant with multiple fetuses might best be illustrated using the following dairy cow/ewe analogy. A combined twin or triplet birth weight of 20 to 30 lb represents about 20% of the maintenance weight of a mature 150-lb ewe. To a dairy practitioner, these birth weights correspond to a 1500-lb cow delivering 100- to 150-lb twin calves.

Whereas the previous example illustrates the extreme nutritional demands placed on prolific ewes and does, practitioners working with late gestation rations need to appreciate (1) the absolute amounts of nutrients needed for fetal growth and (2) reduced rumen capacity (volume) available for nutrient intake and use. During late pregnancy, the increasing size of the gravid uterus competes with the rumen for a finite quantity of abdominal space. Although somewhat unique to small ruminants, reduced rumen capacity ultimately spawns the physiologic factors associated with pregnancy toxemia.

During late gestation, energy and dry-matter intake are prime factors limiting production and contributing to pregnancy toxemia. Poor forage quality further compounds the problem of reduced rumen capacity by (1) limiting intake and (2) creating multiple nutritional deficiencies (energy, protein, calcium, and phosphorus), all at a time when fetal growth requires adequate nutritional support. To prevent pregnancy toxemia, late gestation rations need to address a 65% increase in energy requirements above those reported for early pregnancy (Fig. 2*A* and *B*). Additionally, late gestation feeding recommendations for ewes and does should recognize a reduction in forage intake associated with declining rumen capacity. Reduced rumen volume and increased nutritional demand require more energy-dense rations than were necessary during early gestation.

Late Gestation Management Issues Associated with Pregnancy Toxemia

In traditional sheep and hair goat production systems, the last 4 to 6 weeks of gestation commonly involve shearing, confinement to drop-lot areas, and feeding management systems using stored forages and grains. Unless parturition is scheduled to occur on lush spring pasture, nutritional input is often 100% producer dependent. To prevent pregnancy toxemia, late gestation feeding should begin about 4 weeks prior to the earliest expected parturition date; however, in commercial production systems, initial feeding of the late gestation ration is influenced by breeding and management schemes involving the group, not individual animals. Because of the seasonal breeding pattern of small ruminants, ewes and does bred (in August and September) to deliver in January and February conceive over a prolonged period of time. Parturition

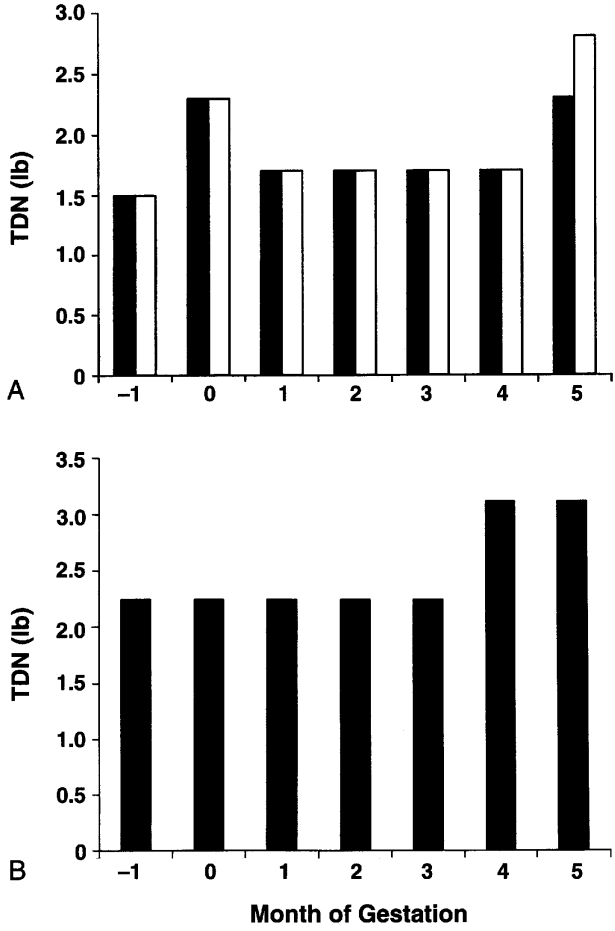

Figure 2. *A*, Energy requirements for a commercial ewe (154 lb) at a 130% to 150% *(solid bars)* or 180% to 220% *(open bars)* expected lambing rate. (*Data from* Nutrient Requirements of Sheep, ed 6. Washington, DC, National Academy Press, 1985.) *B*, Energy requirements for a 154-lb, pregnant, nonlactating doe at medium activity levels *(solid bars)*—assumes no concurrent lactation demands. Current National Research Council requirements assume pregnant doe with twin fetuses. TDN = total digestible nutrients. (*Data from* Nutritional Requirements of Goats, ed 15. Washington, DC, National Academy Press, 1981.)

within this management scheme often occurs over a long, drawn-out 8- to 12-week period. In contrast, production units managing for parturition in April and May usually concentrate breeding and birthing seasons into a more narrow 3- to 4-week window. Sheep, hair goats, and meat goats, unlike dairy goats, are commonly housed and fed in large groups. In most commercial situations, individual animal breeding dates are unknown, and the initial feeding date for a late gestation ration becomes

an important health and economic consideration for the flock or herd, especially if a lengthy birthing season is expected.

To illustrate this management issue, obesity in ewe flocks that lamb in January and February often results from a management decision to initiate feeding of an energy-dense late gestation ration 6 weeks prior to the earliest projected parturition (based on ram turnout dates). Odds related to seasonal breeding suggest that much of the ewe flock will receive a high-energy late gestation ration (in excess of requirements) for about 8 to 14 weeks instead of the customary 4 to 6 weeks prior to parturition. As a consequence, obesity may develop in a portion of the flock, yet producers often fail to recognize this management-induced obesity problem until midway through lambing season. Once alerted, the producer response to mounting late gestation ration costs (because of the lengthy feeding period) and a drop-lot full of obese pregnant ewes is the related management decision to reduce the energy content of the ration; however, reducing the energy intake of obese ewes during late pregnancy commonly results in fat mobilization and ketosis, leading to fat-ewe pregnancy toxemia. As a rule, feeding alterations to improve the condition of obese ewes should not occur during late gestation. Instead, reduction in body condition scores should be reserved for normal physiologic weight loss associated with lactation or a planned weight loss during periods of maintenance.

Producers whose flocks or herds traditionally lamb or kid over an extended period of time should also consider the health, economic, and labor implications associated with a prolonged lambing or kidding season, not just pregnancy toxemia. Obesity can develop if, because of seasonal breeding factors, a large portion of the production group remains on an energy-dense late gestation ration for a prolonged interval. Prolonged birthing season–associated obesity problems can be minimized if dams are sorted into early and late parturition groups and fed accordingly. Group feeding decisions can be managed by weekly *bagging* (udder palpation) and sorting of pregnant ewes. Additionally, sorting (1) reduces feed costs, (2) focuses observation, (3) maximizes use of housing facilities, (4) reduces crowding, and (5) improves ventilation and sanitation.

Addressing the increased nutritional requirements associated with multiple fetuses is also crucial to pregnancy toxemia prevention programs, especially in production units where multiple fetuses are common. Practitioners working with late gestation feeding management need to recognize and accommodate the expected or known lambing or kidding rate of the targeted animal unit. In a ewe flock, for example, late gestation rations designed for less prolific flocks (130% to 150% lambing rate) typically require about 20% less energy than do rations for prolific production units (180% to 225% lambing rate). Prolific flocks that historically approach or exceed a 200% lambing rate should use rations designed around National Research Council (NRC) requirements for ewes producing a 180% to 225% lambing rate.[12, 13] As a rule, if

producers with prolific flocks or herds cannot identify and separate animals carrying multiple fetuses, then the entire flock should be fed for the higher level of production.

If equipment and expertise are available, ultrasound scanning can be used to identify and divide the flock or herd into two economical and efficient feeding groups based on fetal counts; however, scanning is not widely practiced in this country. Although scanning is considered state of the art, justification should be based on economics and the ability of the production system to separate feeding groups during late gestation; however, in many production units, separation and feeding of distinct production groups is not always possible or economically feasible.

The following ewe flock examples should help illustrate these issues. Scanning, sorting, and feeding late gestation ewes for lower production levels (130% to 150% lambing rate) conserves about 0.6 pounds of total digestible nutrients (TDN) per ewe per day. Flocks selected for prolificacy and seasonal fertility usually contain a very small percentage of open adult ewes or adult ewes that are carrying singles. Midwest farm-flock economics suggest that the difference in late gestation feed costs between ewes with a 130% or a 200% predicted lambing rate amounts to 2 or 3 cents per day. In the Midwest, this is equal to savings of $0.60 to $0.90 per ewe, distributed over the relatively short late gestational feeding period. Scanning does allow open ewes to be culled from the flock prior to late gestation; however, conception rates (over 90%) for healthy adult ewe flocks exposed during the normal breeding season suggest that scanning the entire flock may not be cost-effective. In the author's opinion, scanning for fetal count may be economical for (1) production units with historically low lambing or kidding rates (many singles), (2) flocks or herds that breed out of season (more open animals), (3) flocks on accelerated lambing programs, (4) flocks or herds with poor reproductive history (many open ewes), and (5) pregnancy screening of replacement ewe lambs or does.

When ultrasound scanning is performed, it is important to remember that scanning data represent the result—not the cause—of the reproductive performance of the flock. For example, if scanning data predict a low lambing rate (120% to 130%), management's short-term response might be to assign separate single or multiple fetus feeding groups to prevent pregnancy toxemia while reducing late gestation feed costs; however, more essential to the long-term economic survival of the flock may be addressing the broader issue of management's contribution to reduced fetal numbers. Diverting scanning dollars toward (1) increased selection pressure for fecundity and maternal traits in replacement ewe lambs, (2) optimal nutrition during breeding season, (3) purchase of proven prolific maternal-type sires, (4) prevention of abortion diseases, and (5) fertility testing of rams might be more economically responsible and appropriate management responses.

Shearing Stresses

An additional pregnancy toxemia late gestation management-related issue involves late gestation shearing. In many production systems, pregnant ewes and hair goats are sheared or clipped about 4 weeks prior to the start of the lambing season. Shearing during late pregnancy aids in visual assessment of average body condition scores for the ewe flock. This management practice allows producers a window of opportunity for ration adjustment prior to parturition. Although regular handling of ewes to assess body condition is encouraged throughout the year, realistically, it does not always occur. Late gestation shearing (if occurring at least 4 to 6 weeks prior to parturition) coupled with visual assessment of body condition offers a crutch to producers or veterinarians who do not body condition score the flock on a regular basis. On the positive side, shearing reduces bunk space requirements, improves ventilation and sanitation, affords better visualization of parturition, and contributes to reduction of neonatal mortality. On the negative side, however, weather stresses may increase the housing and energy requirements of the flock. Compounding this issue is the fact that extensive-type production units generally have minimal housing available for weather protection. For this reason, pasture-based production units depend on favorable weather conditions during the lambing or kidding season. Especially vulnerable to losses associated with outbreaks of pregnancy toxemia are large production units with thin, recently shorn animals. In extensive production systems, late gestation exposure to unseasonably cold temperatures, rain, wind, ice, or snow often contributes to enormous losses that are difficult to predict, prevent, or treat.

Late Gestation Feeding Recommendations to Prevent Pregnancy Toxemia

Hay

In farm-flock production units, standard late gestation daily feeding recommendations for the commercial ewe or doe (150 to 160 lb with twin fetuses) would normally include 3.5 to 4 lb of medium to good-quality hay and 1.25 to 1.5 lb of concentrate. Supplemental concentrate should be initiated 2 to 4 weeks prior to the first predicted due dates dependent on ewe condition, management system, and flock genetics. Increased fiber levels in poor-quality, overly mature hays (especially grass hays), combined with reduced rumen capacity (because of the developing fetuses), can limit daily hay consumption to 3 to 3.5 lb per animal, contributing to negative energy balance and pregnancy toxemia. Poor-quality hays should be avoided during the late gestation period. If only poor-quality hay is available, practitioners formulating feeding programs should use caution when estimating late gestation consump-

tion figures for poor-quality forages, even in a free-choice feeding system. Pregnancy toxemia issues are less likely to develop if daily forage consumption figures err on the conservative side. Late gestation rations using poor-quality forages should be designed around realistic, reduced forage intake and increased amounts of concentrate.

High-Moisture Feeds (Assumes 150- to 160-lb Ewe or Doe)

A similar reduced intake situation occurs when stored high-moisture forages are fed during late gestation. Because of reduced rumen capacity, late gestation daily individual consumption of high-moisture feeds may be limited to 6 to 8 lb of silage or haylage, compared with daily free-choice consumption levels approaching 10 to 12 lb of the identical high-moisture feed during early gestation. This example illustrates several pregnancy toxemia concerns for practitioners dealing with high-moisture forages for pregnant sheep and goats. As a rule, free-choice early gestation feeding of haylages and silages generally leads to overly fat ewes or does during late gestation. In contrast, limited intake in late gestation often contributes to a negative energy balance if grain is not included in the diet. Forage testing, ration analysis, and reasonable knowledge concerning intake of various feeds are all necessary to formulate rations designed to prevent and treat pregnancy disease.

As a rule, most 100% forage diets typically fail to meet individual animal late gestation requirements for ewes and does pregnant with multiple fetuses, unless forage quality, digestibility, and availability are exceptional. For this reason, cost-conscious large production units often rely on a 4- to 6-week grazing period on highly nutritious lush spring pasture or free-choice availability of excellent-quality legume hay or alfalfa pellets to supply late gestation requirements; however, if pasture is to be used as the late gestation ration, the breeding season is generally arranged so that parturition occurs shortly after spring pasture growth begins. An additional reason for all forage, late gestation rations involves the feeding frenzy associated with grain feeding systems. Larger commercial production units often choose to reduce the feeding frenzy associated with feeding grain to sheep and goats by discontinuing grain feeding when parturition begins or avoiding concentrate feeding altogether. Under this scenario, clinical cases of pregnancy toxemia associated with limited or no concentrate feeding appear to decline over time as pregnancy toxemia–susceptible individuals and families are culled from flocks or herds.

Condition Scoring as a Tool

Body condition scoring of ewes or does 4 to 6 weeks prior to the expected date of parturition allows detection and adequate time for correction of problems. Late gestation body condition scores should

increase to a 3 to 3.5 level at parturition. Additionally, as late gestation approaches, energy levels exceeding the NRC requirements may be necessary if (1) pregnant animals are thin, (2) weather conditions are extreme, (3) housing is inadequate, or (4) prelambing shearing is practiced. The goal of the late gestation diet is to have ewes and does in optimal condition at the time of parturition.

Practitioners faced with the task of preventing thin or fat ewe or doe pregnancy toxemia at a flock or herd level need to develop two separate feeding management strategies designed for each group of animals. Thin late gestation ewes and does should be sorted (if possible) into separate feeding groups and provided a more energy-dense diet than the NRC-recommended levels for ewes with normal body condition scores. The energy level and density of the diet should also be increased to adjust for housing, weather condition, and fleece/hair status. If thin ewes or does are numerous and logistics prevent sorting, it is usually wise to feed the entire production unit at the higher energy level. In contrast, obese ewes should not be fed to reduce body condition during late pregnancy. Pregnancy toxemia is commonly a consequence of weight reduction during late pregnancy. Fat ewe pregnancy toxemia that results from overfeeding during early pregnancy is a common problem for (1) small farm flocks or herds, (2) beginning producers, and (3) systems where high-energy forages are fed ad libitum during early pregnancy. In contrast, pregnancy toxemia resulting from underfeeding and starvation is a common problem with larger, grazing-oriented commercial flocks or herds. Because of ewe or doe numbers, larger commercial flocks and herds are severely impacted by drought-induced feed shortages, adverse weather conditions, and lack of housing facilities.

Basic Feeding Guidelines for Prevention of Pregnancy Toxemia in Ewes and Does

Although feeding recommendations vary from operation to operation, the following guidelines might be useful to practitioners unfamiliar with feeding practices for pregnant ewes or does. The guidelines presented assume a 150- to 160-lb commercial ewe, pregnant with twins. Guidelines for meat and hair goats are similar; however, dairy goats often have additional concurrent lactation requirements based on the level of production. Early gestation rations usually consist of 100% forage diets that incorporate about 4 lb of medium-quality first-cutting grass/mixed hay (8% to 10% crude protein, 50% TDN). Late gestation diets usually include 3.5 to 4 lb of a better-quality hay (over 10% crude protein and over 55% TDN) and a once-daily feeding of 1.5 lb of concentrate (whole shelled corn is most economical in the Midwest). In commercial operations, concentrate feeding generally begins about 4 weeks prior to the first projected parturition date. Initially, concentrate is fed at the rate of about 0.5 lb/head/d and gradually increased to about 1.5 lb/head/d at about 2 weeks before parturition. If ewes or does

are thin, shorn, or clipped and weather stresses are severe, concentrate is often increased to as much as 2 to 3 lb/head/d, split into two feedings to prevent grain overload. Pregnancy toxemia is avoided by increasing the energy density of the diet at a time when requirements increase and rumen capacity declines.

As mentioned earlier, hay and concentrate late gestation rations are the norm for most flocks in the Midwest; however, in practice, flock outbreaks of pregnancy toxemia commonly occur when producers switch to or are forced to use unfamiliar feeds. These feeds commonly include energy-dense small-grain hays and high-moisture, bulky feeds such as corn silage and haylage. Small-grain hays are extremely energy dense (65% to 75% TDN) and palatable. When available for free-choice feeding during early gestation, consumption of small-grain hays often approaches 6 to 7 lb/head/d (on an as-fed basis). Early gestation energy intake from small-grain hay diets can exceed early gestation energy requirements by as much as 200% to 300%. Free-choice feeding of small-grain hays during early gestation can lead to excessive body condition scores and fat-ewe pregnancy toxemia.

Silage and haylage can lead to similar problems if fed free-choice during early gestation. Haylage is extremely palatable, and corn silage is both a palatable and a high-energy feed. Overconsumption of either haylage or corn silage can lead to excessive energy intake during early gestation. Ad libitum consumption of corn silage or haylage during early gestation often approaches 12 lb/head/d (on an as-fed basis). Most early gestation energy requirements are met with just 6 to 7 lb of corn silage or haylage. The moisture content of these feeds may also contribute to pregnancy toxemia when these feeds are continued into late gestation. Reduced rumen capacity combined with the bulk and moisture content of the feed often limits intake. Forage analysis and ration formulations are especially important when feeding high-moisture feeds, small-grain hays, and other atypical feeds. Most flock or herd outbreaks of pregnancy disease can be prevented with proper feeding practices.

Numerous other less-tangible feed management factors can also contribute to or prevent flock or herd outbreaks of pregnancy toxemia. When possible, separate ewes and does into feeding single-, twin-, or triplet-status feeding groups and feed accordingly. Ultrasound scanning can be helpful. When scanning is not possible, producers should be encouraged to feed the flock or herd for a lambing rate consistent with recent flock history. Sufficient bunk space (1 foot per dam) should also be available to allow equal access to concentrate and roughage.

Producers should also be aware of flock behavioral differences when adding purchased ewes or does to native flocks or herds. This is especially important when western range animals are imported into existing farm flocks. For example, ewes who have spent their entire life in extensive range management systems are often unfamiliar with machinery noise and intensive bunk-feeding practices typical of farm flocks. When imported to the Midwest, range ewes are often timid and fail to

compete with native ewes for supplemental concentrate feeds. Many have never been fed grain. As is the case with any production-related disease, pregnancy toxemia may be limited to newly introduced range ewes, while native ewes appear to be free from clinical signs. Housing and feeding new additions as a separate group (the first season) may help to decrease competition and reduce the likelihood that timid ewes develop pregnancy toxemia. Finally, adjustments in feeding programs also need to address weather conditions, fleece status, and availability of housing. Producer attention to a variety of factors that influence body condition scores is necessary if pregnancy toxemia is to be prevented.

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Address reprint requests to
Joseph S. Rook, DVM
Department of Large Animal Clinical Sciences
Michigan State University
College of Veterinary Medicine
A113 Veterinary Clinical Center
East Lansing, MI 48824

e-mail: rook@cvm.msu.edu