CANADIAN MEAT GOAT Association
CANADIENNE de la CHÈVRE de BOUCHERIE

Pregnancy Toxaemia/Ketosis

Does carrying multiple fetuses require a much higher level of energy than does carrying singles.

This is a condition of late pregnancy and early lactation most commonly occurring in the last six weeks of gestation in does with multiple fetuses and in the first 4 weeks in heavily lactating does. Pregnancy toxaemia is a more common condition than ketosis in goats. Through recognition of early signs and symptoms and avoidance of the predisposing factors, it can be reduced to a sporadic condition.

Predisposing Factors

Factors that predispose does to develop pregnancy toxaemia can be divided into two types: inadequate nutrition (they are not offered the correct quantity or quality of the required ration) and adequate nutritional offerings but external or animal factors (e.g. disease) affecting intake.

Inadequate Nutrition

Does carrying multiple fetuses require a much higher level of energy than does carrying singles. These increased needs are compounded by a decreased capacity to consume. When offered free choice balanced feeds, does in late pregnancy will voluntarily increase energy consumption but expanded uterine contents limit dry matter intake, putting fecund does at even greater risk of developing pregnancy toxaemia. To counteract this, the producer must offer a ration that is more energy and protein dense, e.g. 35% grain to 65% forage. Prolific does on pasture may have difficulty meeting their energy needs through grazing.

Factors Affecting Intake

EXTERNAL FACTORS: Healthy, well fed goats may tolerate many external factors but does chronically underfed are not able to compensate. Inclement weather (e.g. rain or snow storms or extreme heat) may interrupt intakes. Feeder space may have been calculated for non-gravid does and may be inadequate for heavily pregnant does. When hand feeding, the producer should be warned to watch for excessive competition. Restricted water intake or poor quality water will reduce dry matter intakes. Rapid feed changes, transport may all temporarily reduce intakes. Poor quality forage that is too woody may also reduce dry matter intake. Preventive treatments such as vaccinating, etc. performed in late gestation may reduce intakes, particularly if they are held off feed for the procedure. Exercise has been shown to increase voluntary intake.

ANIMAL FACTORS: Factors that will reduce intakes include dental disease, old age, smaller body size than group (see feeder space), and other concomitant disease such as hypocalcaemia, lameness (e.g. CAE arthritis), gastrointestinal parasitism, Johne's disease, etc. The body condition score of the doe entering into late gestation is important. Does that are very thin (< 2.5) have little fat or muscle reserves to draw upon and are then at increased risk despite a good ration. Very fat does (> 4.0) will readily use body fat reserves in late gestation but also experience decreased voluntary intakes, thus

predisposing them to formation of ketone bodies that further suppress appetite.

Clinical Picture

The course of the disease varies but generally develops over three to ten days. A more sudden onset is usually associated with a sudden stress or poor producer observation. Does will start to decrease grain intake, followed by silage and then forage. They separate from the herd, lag behind, and become depressed and gaunt. Other signs of predisposing disease may also be present. Producers that are vigilant when hand feeding does will easily recognize these animals. If missed, the doe may go on to exhibit neurological signs which include an abnormal gait and stance, apparent blindness, stargazing and severe depression followed by recumbency and coma. Some of these signs are attributable to ischemic necrosis of the brain cortex secondary to prolonged hypoglycaemia (low blood sugar). Polioencephalomalacia, hypocalcaemia, toxic mastitis (if near or after kidding), grain overload, listeriosis and lead poisoning are important differential diagnoses. Some producers can smell acetone on the goat's breath (not everybody has this capability). This is the ketone bodies being produced because of the disease.

Clinical Pathology

Most changes are attributable to primary hypoglycaemia resulting from the failure of nutrient intake to meet the combined needs of the doe and fetuses or of the doe's milk production. Circulating glucose demands in late gestation and early lactation are extremely high. The doe must either manufacture this from the production of propionic acid precursor in the rumen or from gluconeogenesis of amino acids derived from the diet or body muscle reserves. This prolonged hypoglycaemia results in suppressed insulin production, which in turn increases fat mobilization. This is limited by the availability of the glucose precursor oxaloacetate for the tricarboxylic acid cycle. This results in a build-up of acetyl-CoA which is broken down in the liver to the ketone bodies acetoacetate and ß-hydroxybuterate.

Low blood glucose is a consistent finding in clinical and subclinical pregnancy toxaemia (often much less than 2.0 mmol/L) but may disappear in cases of severe disease, probably due to the death of the fetuses.

Ketonaemia (blood ketones) and ketonuria (urine ketones) are also consistent findings. The serum level of ß-hydroxybuterate (ß-HB) is inversely correlated with serum glucose. Serum ß-HB levels may be used to provide a screening test for flock nutritional status in late gestation. Interpretation of values are presented in table 1 (sheep values). ß-OH levels have been reported at pregnancy toxaemia levels up to 10 days before the ewe develops clinical signs.

Table 1. Interpretation of serum ß-hydroxybutyrate levels in the evaluation of late gestation ewe nutritional status

Status	Serum ß-hydroxybutyrate (mmol/L)
Normal	< 0.70
Moderate under feeding	0.80 - 1.6
Severe under feeding (subclinical pregnancy toxaemia)	1.6 - 3.0
Pregnancy toxaemia	> 3.0

Treatment

It is important that the producer recognizes the predisposing factors to pregnancy toxaemia and takes action to prevent the disease. Does with reduced appetite and mild depression with no neurological signs, may respond to the following conservative treatment regime: supplementation with propylene glycol (600 mg/ml) at a rate of 60 ml/ BID per OS for a minimum of 3 days;

improved nutrition and feeding management; and treatment of any predisposing condition. More severely affected does require aggressive therapy which includes:

- A single injection of glucose IV (more frequent injections have been associated with insulin suppression and rebound hypoglycaemia)
- Oral propylene glycol at the above dosage regime if not comatose
- Oral and/or intravenous fluid therapy using balanced electrolyte solutions
- Correction of ketoacidosis using bicarbonate or bicarbonate precursors.
- Since hypocalcaemia is often a secondary disease associated with pregnancy toxaemia, clinical signs of hypocalcaemia should be evaluated.
- Corticosteroid therapy using a single dose of dexamethasone.
- Removal of the fetuses. Abortion is the preferred method as it is more affordable and less stressful to the doe. If the kids are more than 2 to 3 days premature, they will be unlikely to survive but are already at great risk of death in a severely ill doe. Consult your vet about this procedure. Before a caesarian section is performed, the doe should be stabilized using appropriate fluid therapy.
- Systemic antibiotics.
- Nursing care. Does need to be encouraged to eat and will need extra attention.
- Correction of other diseases (e.g. if CAE arthritis, a non-steroidal anti-inflammatory could be considered so the doe is willing to get up to eat)

This is a disease that needs to be prevented rather than treated. It is generally a management disease and should be initially investigated as a herd level problem rather than an individual sick goat. If one doe is clinically ill, many more in the herd are likely at risk.

Dr. Paula Menzies, Department of Population Medicine,
Ontario Veterinary College, University of Guelph