Should I be concerned about nutritional/metabolic diseases in sheep?

- Nutritional diseases in sheep are diseases often related to deficiencies or toxicities. The deficiencies can be in energy, protein or specific vitamins and minerals. Toxicities of some vitamins and minerals can also occur, such as copper or heavy metals.
- Typically, nutritional deficiencies are long-term conditions that can be corrected through dietary supplementation.
- Metabolic diseases of sheep are caused by productivity practices where the body reserves of certain nutrients such as calcium, magnesium or energy cannot meet the changing metabolic needs of a sheep.
- Metabolic diseases are generally rapid onset in nature and dramatically respond to the administration of the deficient nutrient, although affected animals may require subsequent dietary supplementation to avoid recurrence.
- An important aspect of dealing with production-induced metabolic diseases is accurate and rapid diagnosis.
- Correcting the diet for animals during particularly nutritionally stressful periods, such as from late pregnancy to peak lactation, is crucial in preventing these diseases.
- If these diseases occur frequently, it is essential to seek professional veterinary and nutritional advice.

What are some of the more common nutritional/metabolic diseases?

- **Ruminal Bloat:**
  - Ruminal bloat occurs when rumen gas production exceeds the rate of gas elimination. Gas then accumulates causing distension of the rumen.
  - Bloat can be a medical emergency and rapid treatment may be necessary to prevent death.
  - The rumen will be extended and tight and may be higher than the mid-line of the sheep.
  - Ruminants have four stomachs: the rumen which is a large fermentation vat and is located on the left side of the abdomen;

Metabolic diseases are generally rapid onset in nature and dramatically respond to the administration of the deficient nutrient.
the reticulum which is small and located in front of the rumen; the omasum which looks like a small soccer ball and helps to digest large fibres, is located to the right of the reticulum; and the abomasum – the true glandular stomach which is located on the lower right side of the abdomen. Once the lamb is weaned from milk, the largest stomach is the rumen.

- Some signs of bloat are:
  - Off feed and anxious.
  - Head down and extended if having trouble breathing.
  - Not belching.
  - Not chewing cud.
  - Normal rumen sounds absent (gurgling gastric sounds).
  - Grinding teeth.
  - A prominent swelling on the upper left abdomen behind the ribs, with tight skin. If percussed (tapped lightly), there is a hollow “drum-like” sound.
  - If on palpation high up on the left, the distended rumen is doughy or fluid-filled, this is not bloat but could be from another disorder, e.g. grain overload.

- Late stages of bloat signs:
  - Extreme distress and occasionally bleating.
  - Sometimes, green froth at the nose – a very dangerous sign.
  - Gasping for air.
  - Darkened (blue) tongue.
  - Sheep down.

- Bloat usually results from nutritional causes. There are two types of ruminal bloat – frothy and free gas:
  - **Frothy Bloat:** Commonly called pasture bloat, is usually associated with the consumption of lush legumes such as alfalfa.
    - May also occur in sheep and goats grazing lush cereal grain pastures or wet grass pastures or consuming grain that is too finely ground.
    - If a stomach tube is passed, usually no gas comes out and a green froth may be seen clogging the tube after it is removed.
  - **Free Gas Bloat:** Commonly called feed lot bloat, is associated with grain feeding and occurs when animals
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are not given enough of an adjustment period after feed is changed.

- Free-gas bloat may also be caused by choke or obstruction of the esophagus (e.g. apples, grain or a mass pushing on the esophagus). In these cases, a stomach tube cannot be passed.

- Occasionally, a feedlot animal may develop free-gas bloat secondary to damage to the nerve which controls the function of the rumen (the vagal nerve). These animals will bloat repeatedly and grow poorly. The nerve is often damaged from the inflammation associated with chronic pneumonia. The nerve is irritated where it runs through the chest from the brain to the rumen. A stomach tube can be passed, and free gas will be released, but the animal bloats again soon after.

  o Why does bloat kill and kill quickly?

    - The rumen expands to the point that it compresses the abdominal blood vessels, the heart and the lungs. The animal dies from lack of oxygen and blood to the brain.

- **Abomasal Bloat:**

  o Occurs in lambs pre-weaning and most often in lambs on milk replacer.

  o The most relevant risk factors for this condition are restricted feeding of milk replacer, usually fed warm, which encourages rapid drinking.

  o The large volume of milk replacer overwhelms the normal digestive juices in the abomasum. Bacteria grow rapidly and produce a stable foam.

  o The abomasum enlarges, rapidly causing extreme pain and compression on the lungs and heart.

  o In severe cases, the abomasum may rupture causing severe shock and death in the lamb.

  o All this can happen within an hour after feeding.

  o The signs have a fast onset.

  o The lamb is colicky and painful.

  o The abdomen is very large – larger on the right but also the left as it displaces the small rumen.

  o Often the lambs are found dead when the owner returns a few hours later.

  o Feeding milk replacer ad lib and cool reduces the risk of this disease.
• **Chronic Copper Toxicosis:**
  
  o Sheep are the most susceptible of all food-producing animals to copper toxicosis.
  
  o Sheep absorb copper from the diet in proportion to the amount of copper offered. It will store the excess copper in the liver until toxic levels are reached at which time the liver cells break-down releasing the copper into the blood stream where it destroys red blood cells.
  
  o Copper toxicosis may occur because of a recent exposure to high levels – but frequently the overdose may have occurred weeks to months or even years previously.
  
  o Some other stress can facilitate the release from the cells. This stress can be weather, poor nutrition, transportation, handling or lambing.
  
  o While all sheep are susceptible to copper toxicosis, the susceptibility of individual sheep is influenced by its breed (e.g Texel sheep are very susceptible and Finn sheep are very resistant); the levels of other minerals consumed (e.g molybdenum, zinc and sulphur all compete with the uptake of copper from the diet); and even the levels of some feed additives in the diet (e.g monensin in the diet will enhance absorption of copper).
  
  o Molybdenum is often added to sheep diets to try to help prevent copper toxicity if the copper levels have been found to be too high. However, molybdenum added at too high of levels can actually result in sheep having a copper deficiency, so it is important to know the content of the various minerals in the diet before making marked changes.
  
  o While the disease is called chronic copper toxicosis, the signs in the sheep are very acute and severe as a rule.
  
  o The haemolysis caused by the breakdown of the red blood cells releases haemoglobin into the blood serum to be converted to methaemoglobin, a form of haemoglobin that cannot carry oxygen to the tissues.
  
  o A sheep at this point is anaemic, with very pale mucous membranes, and lethargic.
  
  o It will be found down, staggering, trembling and appears to be in a great deal of pain.
  
  o The visible membranes rapidly yellow as jaundice sets in throughout the body.
  
  o The urine is a dark brown from the excreted methaemoglobin.
Almost all clinically affected sheep will die within two to three days. The carcass is yellow, the liver is enlarged and yellow and the kidneys are black.

Copper deficiency is a much rarer disease in Canada and usually results from excess molybdenum in the diet. The most common clinical sign is partial paralysis of the hind end of lambs – called enzootic swayback. A veterinary examination and often a necropsy are required to verify the diagnosis.

Copper should not be added to the diet unless the diagnosis is confirmed by an accredited veterinary diagnostic laboratory.

**Prevention and Treatment:**

- Prevention of copper toxicity is the most practical method of dealing with copper toxicity.

There are a number of strategies that can be used:

- Do not feed swine, cattle, horse or poultry feed to sheep; they contain high levels of copper by design.
- As a rule, there should be no more than 10 ppm (mg/kg) of copper in the complete diet – including forages. Molybdenum levels should be 1-3 ppm and even better if the ratio of copper to molybdenum is around 6:1.
- Communicate with feed company representatives that are supplying feed. It is important that mixers and augers are clean, and that feed delivery trucks are cleaned before being loaded with sheep feeds, especially if the company mixes and handles swine feeds.
- Routinely test forages for levels of copper.
- If copper toxicosis continues to be a problem despite low values on testing, have forages tested for molybdenum and sulphur, particularly if you are in an area known for deficiencies or high levels in any of these elements.
- Avoid grazing sheep on pastures where swine or poultry waste is applied as the feeds for these species are high in copper, which is passed in the manure and accumulates on pasture.
- Do not use copper piping for water.
- Do not use copper sulphate for treatment of lameness, or as a control for algae in ponds.
- Have post mortems done on dead animals; this is a good routine management practice.
• Treatment of sheep with copper toxicity should be done under the advice of a veterinarian; prevention is the best course of action.

• Enterotoxemia/Pulpy Kidney:
  o Enterotoxemia is one of the most common and costly disease problems in the sheep industry.
  o It is also known as “overeating disease” or “pulpy kidney” disease, it is most commonly caused by a bacteria, Clostridium perfringens type D and more rarely by C. perfringens type C. The latter usually causes bloody diarrhea in very young lambs and the former causes sudden death in lambs greater than five weeks of age up to adults.
  o C. perfringens type D is sometimes found in the intestinal tract of small ruminants but does not always cause a problem. The bacteria form spores that are passed in the feces and survive in the soil for years.
  o However, there are certain conditions that trigger excessive bacterial growth in which lethal amounts of toxin are produced, resulting in death of the animal.
  o Enterotoxemia is most commonly associated with nutritional stresses such as heavy concentrate feeding, pasturing on lush pasture, or an abrupt change in the diet, usually to a better feed or quality of pasture.
  o Feeding concentrate before forage can also cause enterotoxemia. Using this readily available carbohydrate source, the bacteria replicate rapidly and secrete toxins which are absorbed into the body. The most dangerous toxin, epsilon toxin, damages the brain and blood vessels. The lamb may briefly convulse before dying but is most often just found dead. It is not unusual for several lambs to die over a period of time unless action is taken.
  o On necropsy, little is seen except for some hemorrhages on the heart muscle and fluid around the heart – often with a “chicken-fat” clot inside the heart sac (pericardial sac). Gut contents, if taken fresh from the small intestine, can be submitted to a veterinary diagnostic laboratory for culture and typing to confirm the diagnosis.
  o To reduce the risk of continuing losses, remove the hot ration, e.g. stop feeding grain or bring in from the pasture and put on dry hay. A vaccination program should be started immediately.
  o Prevention of this disease is very simple: all sheep should be maintained on a proper vaccination program against clostridial diseases. All sheep need to be vaccinated twice six to eight weeks apart and then boostered annually. Lambs should be vaccinated twice as well, usually starting at 12 weeks of age. The directions
on the vaccine should be followed so that proper immunity can be maintained. Pregnant ewes should receive their booster one month prior to lambing.

- **Grain Overload/Ruminal Acidosis:**
  - This condition is most common in lambs post-weaning that are being pushed on a high-grain diet for market, or if there is an accidental over-feeding of grain.
  - The rumen micro-flora can only handle gradual changes in forage to grain ratio.
  - If the proportion, quantity or type of grain changes too quickly, then lactic acidosis will develop. This lowers the pH of the rumen, and when the pH drops below 5.5, protozoa and bacteria start to die. The lactic acid is then absorbed into the body, creating general acidosis.
  - If the pH is low enough, the rumen gets “burned” and if the animal survives, it often gets secondary rumen and liver infections from bacteria or fungi.
  - Fibre (for example, hay or silage) is important in all ruminant diets as well, as it stimulates the animal to chew, thus producing alkaline saliva which serves to buffer the rumen.
  - Animals eating diets with little fibre or chopped too finely are more at risk of lactic acidosis.
  - Chronic feeding problems will appear as:
    - Variable appetite.
    - Chronic laminitis is identified by fast-growing toes with “rings,” the quality of the horn is poor and flaky; animals will be lame and prone to foot abscesses.
    - Diarrhea smells acidic and is yellow in colour.
    - In very severe cases, there is no diarrhea because of total gut stasis.
    - It will go down and present symptoms that look very similar to milk fever, such as lowered body temperature and dilated pupils.
  - Management that will aid in prevention of the disease:
    - Rations should be formulated and balanced correctly for the correct production group.
    - Avoid sudden changes to the diet. There should be a gradual transition of two to three weeks when going from roughage to a highly concentrated ration.
    - Forage should be fed before grain.
    - Feed at regular intervals.

*Animals eating diets with little fibre or chopped too finely are more at risk of lactic acidosis.*
• Divide the daily feed into at least three separate feedings – make sure the bunk is not empty for very long if lambs are in a feedlot or on a self feeding system.
• Mix rations properly.
• Providing adequate feeder space.
• A total mixed ration (TMR) helps keep the rumen flora happy by not overwhelming them with carbohydrate at any one time.

• Grass Tetany:
  o Grass tetany is a metabolic disease caused by a lower than average blood magnesium (Mg) level. It is not common but can be mistaken for other diseases.
  o Low blood magnesium can be caused by low levels of magnesium in lush spring or fall pastures, grazing cereal grasses such as winter wheat, or by mineral imbalances such as high potassium and nitrogen or low calcium in the diet.
  o There is very little magnesium stored in the bones: absorption of Mg is dependent on the magnesium status of the animal, which is based on dietary intake.
  o In sheep it most commonly occurs, in acute form, within four to six weeks of lambing. It is often triggered by inclement weather.
  o Affected animals exhibit sensitivity to touch and trembling of the facial muscles; some are unable to move, others move stiffly; extreme cases collapse and show repeated tetanic spasms with all four limbs rigidly extended. Without prompt veterinary attention, most of these sheep will die.
  o Ewes with grass staggers are often low in calcium as well as magnesium.
  o Absorption of magnesium is also influenced by the amount of calcium, phosphorus and potassium in the diet.
  o Drought, heavily fertilized crops or pasture (fertilized with nitrogen and potassium or manure) and acidic soils all can reduce the absorption of magnesium in the animal’s system.
  o Prevention of the disease is possible through ration supplementation.
  o Livestock must receive adequate levels of calcium and magnesium through limestone and magnesium oxide.

• Congenital Goitre:
  o Goitre occurs when an animal does not get enough iodine in its diet and is most commonly seen as a cause of abortion, stillbirth and weak lambs that do not survive the first few days of life.
The goitre or swelling of the thyroid gland is caused by the thyroid gland enlarging as it tries to produce the thyroid hormones needed by the animal.

Soils in western and central Canada are deficient in iodine, so forage and grain grown there are deficient in iodine. The maritime provinces, because of their proximity to the ocean, usually have no problems.

Some diets can increase the need for iodine — mostly brassica plants such as turnips and forage rape. Pregnant ewes grazing these feeds must be supplemented with iodine throughout gestation to prevent this disease.

The salt supplements purchased at the feed store must have iodine added, along with cobalt. Read the label carefully. Salt-mineral premixes that also include selenium are a better purchase.

A minimum amount of l-salt should be “forced” into the rations of sheep by including it in a premix, concentrate or supplement.

Free choice salt should be available at all times for animals that want or need more.

It is important that the iodized salt not only be offered but that the sheep eat it. Blocks buried under bedding packs or by spoiled forage, or salt sprinkled on the forage are inadequate to prevent this problem. Again, pregnant ewes must consume iodine daily to ensure lambs will be born healthy.

- **Listeriosis:**

  - Listeriosis is also called “circling disease.”

  - *Listeria monocytogenes*, the bacteria that causes it is widely distributed in nature and is found in soil, feedstuffs, and feces from healthy animals.

  - It is most commonly associated with the feeding of poor-quality silage or spoiled hay, but because the organism lives naturally in the environment, listeriosis may occur sporadically.

  - Listeriosis usually presents as neurological disease, but may also cause abortion in ewes.

  - Sheep with the neurological form of the disease become depressed and disoriented; they may walk in circles with a head tilt and facial paralysis. Most often, affected sheep are down with the head pressed back against the flank. If you attempt to straighten out the neck, they become very distressed. They will also have a high fever (> 40 C). Most animals this sick will die.

  - Listeria thrives in aerobic conditions where the pH is 5.4 or higher. It does not do well in very acid conditions.
The top layers of silage or improperly preserved silage may harbour large numbers of organisms so it is important to make sure that silage is tightly packed to ensure proper fermentation. Silage stored on dirt, or contaminated with dirt or manure prior to feeding is also very high risk.

- Wet bales of hay or the spoiled hay around round bale feeders in a yard, may also harbour the bacteria.
- Sheep and goats are more susceptible to this disease than cattle; up to 30% of a flock or herd may be affected in an outbreak.
- It is important to seek veterinary input if a case of listeriosis is suspected. There are many other neurological diseases that may present similarly to listeriosis.

**Hypocalcemia (Milk Fever):**

- Milk fever, also known as parturient hypocalcemia, is a metabolic disease in ewes usually seen two to three weeks prior to lambing.
- Initially, the ewe may hop and stagger and appear nervous, but she quickly goes down into sternal recumbancy with her hind legs out behind her (frogged-leg). She is depressed, cold and poorly responsive. She often salivates as her swallow reflex is suppressed.
- Susceptible ewes are usually on a diet low in calcium (e.g. oat hay) or on a diet very high in phosphorus (e.g. poor quality grass hay with grain). In late pregnancy, the lambs’ skeletons are being mineralized, creating a large metabolic demand for calcium.
- Calcium is supplied from two sources:
  - Dietary calcium.
  - Mobilization of calcium from the bone.
- If the diet is low in calcium, it takes several days to adjust the body’s hormones to facilitate mobilization from the bones. In this gap, hypocalcaemia can occur.
- Hypocalcaemia presents similar symptoms to pregnancy toxemia but can be differentiated by the affected animal’s response to calcium therapy.
- It is unusual to see hypocalcaemia post-lambing but all milking ruminants require calcium rich diets after parturition.
- Alfalfa hay can provide this. Cereal crop forages such as wheat or oat hay are very low in calcium (0.15 percent and 0.24 percent dry matter basis, respectively) as opposed to alfalfa hay, and should be avoided unless the ration is balanced with other calcium sources.
The ration in late gestation and early lactation should also have a calcium to phosphorus ratio of greater than 1.75:1. Prevention of pregnancy toxemia will help to prevent hypocalcaemia as well.

- **Polioencephalomalacia:**
  - Polioencephalomalacia (PEM) is a disease characterized by a disturbance of the central nervous system.
  - The brain of infected animals becomes inflamed and swollen, and eventually the brain tissue dies.
  - PEM is caused by a thiamine (vitamin B1) deficiency.
  - Thiamine is normally manufactured by bacteria in a healthy rumen. However, small ruminants on high-carbohydrate diets may have an upset in normal rumen flora.
  - A change in bacterial types may cause either a deficiency of thiamine or production of an enzyme which inhibits thiamine activity.
  - Overdosing with amprolium (in the treatment of coccidiosis), exposure to high levels of sulphur in the diet, or grazing on mare’s tail (equisetum) can also result in PEM but these causes are unusual in comparison to high-carbohydrate diets. Using a molasses lick has been associated with PEM outbreaks, as has allowing sheep to graze “after-math” grain post-harvest.
  - PEM occurs suddenly. Affected animals stand or sit alone, are very stiff-gaited, may head-press indicating a severe headache, are blind and appear to “star gazers.”
  - Temperature and respiratory rate are usually normal but affected animals do not eat or drink.
  - Normally only a few individuals are affected. The animal may go down on its side with its head thrown back and will convulse with stiff legs.
  - If a light is shone in the eyes, the pupils will constrict but the animal will not react to a hand menace – indicating blindness.
  - If not treated on time, most animals with PEM will die within 48 hours.
  - Other disorders to rule out are tetanus (the animal is not blind), pulpy kidney, lead poisoning, listeriosis, and other toxins (e.g. organophosphates and organochlorines).
  - Sometimes the only way to make a diagnosis is through a response to treatment.
  - Early PEM cases often respond, at least partially if not completely, to thiamine administration. Often some response occurs within a few hours of initial treatment. However, PEM caused by high levels of sulphur in the diet, will not respond to thiamine.

*If a case of PEM is diagnosed in a flock, it is advisable to inject the remaining animals with thiamine as prevention.*
Because thiamine is water-soluble, it is quickly eliminated from the body through the kidneys and there is therefore little risk of overdosing.

Most other neurological diseases respond slowly or not at all to indicated treatments (except specific poisonings).

Because thiamine deficiency causes brain tissue to die, time is important. The longer treatment is delayed, the more likely irreversible brain damage may occur.

One case may not necessarily mean a herd problem, but the feeding management should be reviewed.

Some problem herds do require routine thiamine supplementation, but feeding management should be investigated first.

If a case of PEM is diagnosed in a flock, it is advisable to inject the remaining animals with thiamine as prevention.

Drinking water should be tested for sulphur contents, sources of thiamine, if any, should be removed and animals should be introduced to grain diets in steps to avoid a sudden increase in thiamine-producing bacteria in the rumen.

**Urinary Calculi:**

Urinary calculi or “water belly” is a common metabolic disease of male sheep.

The disease occurs when calculi (stones) lodge in the urinary tract and prevent urination.

The most common type of stones are struvite magnesium phosphate from high-grain diets and calcium carbonate from diets high in calcium.

Normally, phosphorus is recycled through saliva and excreted via feces in ruminants.

High grain, low roughage diets decrease the formation of saliva and therefore increase the amount of phosphorus excreted in the urine.

The primary cause of urinary calculi is feeding concentrate diets, which are excessive in phosphorus and magnesium and/or have a low ratio of calcium to phosphorus (<1.5:1).

Lack of water, and water sources that are high in minerals, are also contributing factors.

While urinary calculi can occur in intact males, wethers are at greatest risk because castration of young males removes the hormonal influence (testosterone) necessary for the penis and urethra to reach full size.
Lambs castrated within the first month of life are most vulnerable. For this reason, some veterinarians advocate delaying castration until after puberty.

If castration is performed after puberty, it should be done under anesthesia by a veterinarian.

It should be pointed out that this recommendation is for wethers to be kept after puberty as pets, for pack animals, etc. However, early castration of terminal (meat) animals does not usually pose a problem.

In females, calculi are formed, but excreted due to anatomic differences in the male and female urinary tracts.

The blocked animal will be uncomfortable and will strain, appearing as if constipated, will tread their hind legs and are depressed.

With careful observation, the producer may notice frequent dribbling of small amounts of urine which may be blood tinged.

If you are not sure if the ram is urinating, place him in an unbedded, cement pen by himself for several hours.

Preputial hairs may have dried crystals on the end.

If the blockage is not noticed and is total, the bladder ruptures in 24 to 36 hours.

After bladder rupture, the abdomen swells with urine and the animal appears more depressed.

He may live another few days before succumbing to the toxins in his system.

Occasionally the urethra ruptures and the urine pools under the skin. This condition is called “water belly.”

In rams, the penis can be exposed and the urethral process (vermiform appendage) examined.

Sand or stones, discoloration (red or purple) and swelling may be evident.

A normal appearing process may mean the blockage is higher.

In wethers, often the prepuce is adherent to the penis and it is difficult to expose the end.

As with most disease conditions, it is better to prevent urinary calculi than to treat it.

The disorder can be prevented by feeding rations which contain a calcium-to-phosphorus ratio of at least 2:1. The ratio of Ca:P should never be allowed to go below 1:1.

High calcium diets are effective at reducing the absorption of phosphorus from the GI tract.
Neither magnesium nor phosphorus should be added to concentrate diets.

In addition, diets high in potassium should be avoided.

All rations should contain adequate amounts of vitamin A.

Extra calcium is well tolerated by sheep, so where rations are unbalanced, they can be counterbalanced by adding ground limestone (not dicalcium phosphate).

Legume hays (e.g. alfalfa) are good sources of calcium.

In addition, roughage will increase salivation and rumination which will increase the amount of phosphate excreted in the urine.

Cereal grains (corn, barley, etc.), on the other hand, have an abnormally low calcium-to-phosphorus ratio: 1:4 to 1:6. Therefore, rations containing cereal grains need to be balanced with other feeds or mineral sources to form a complete ration that has the proper ratio of calcium and phosphorus.

Adequate water intake is also necessary to prevent urinary calculi – plenty of fresh, palatable water should always be available.

While vitamin A deficiency is unusual in sheep, lambs on stored forages and grain without A supplementation may have low stores. Low vitamin A is associated with abnormalities of the lining of the bladder, which may predispose to stone formation. Vitamin A should be supplemented in all diets.

Inadequate water intake causes the urine to be more concentrated, which makes the formation of stones more likely.

The use of ammonium chloride at a level of 0.5 percent of the total diet will help to acidify the urine and prevent the formation of calculi. This will have no effect on stones made of calcium carbonate or oxalate.

Most commercial lamb diets contain ammonium chloride, as well as the proper ratio of Ca:P.

**Uterine Prolapse:**

- A uterine prolapse occurs when the womb is turned inside out and pushed through the birth canal by the abdominal straining of the ewe.
- It occurs immediately after parturition and is a life-threatening situation. Veterinary medical attention should be sought immediately.
- A prolapsed uterus must be gently replaced back into the animal with the use of local anaesthetics to prevent straining. It is very easy to perforate the uterus causing severe internal infection.
The uterus should be cleaned with a warm, soapy, disinfectant solution prior to replacement and should be replaced before the tissues become dry or chilled. Deep sutures are necessary to keep the uterus in place. Difficult lambing is likely the most common reason and could be due to: large lamb size due to overfeeding of ewes carrying singles, over-conditioned ewes with pelvic fat; a hard assisted lambing, and less commonly due to metabolic disease such as hypocalcaemia.

- **White Muscle Disease:**
  - White muscle disease (WMD) is a degenerative muscle disease found in all large animals. It is caused by a deficiency of selenium and/or vitamin E.
  - Selenium (Se) deficiency is associated with selenium-deficient soils and the inadequate uptake of selenium by forages grown on these soils. Most of Canada is very deficient in selenium.
  - Vitamin E is plentiful in fresh forages and pasture, but depletes quickly in stored hay and is not present after one month of harvesting.
  - Both vitamin E and selenium are necessary to maintain the antioxidant system and to prevent build-up of free radicals. While a deficiency of either alone is sufficient to cause disease, a deficiency of both is more commonly found.
  - Deficiency causes acute and severe muscle necrosis known as white muscle disease.
  - Usually young, fast-growing animals are affected, at any time from birth to full grown. The young animals are in severe pain; they are reluctant to move, but may still look bright and may eat. Hunched appearance is common.
  - When the problem occurs in newborns, they are born weak and unable to rise.
  - Sudden exercise may trigger the condition in older lambs.
  - Sometimes the disorder manifests itself as sudden death because the heart muscle is affected and sometimes as respiratory distress as the diaphragm is affected.
  - In addition to WMD, selenium and vitamin E deficiencies can produce symptoms of ill thrift and reproductive losses: lower conception rates, fetal re-absorption, dystocia, retained placenta, reduced milk production, and reduced semen quality.
  - They can cause poor rate of growth or ill thrift in young lambs throughout the growing period.

**WMD can be prevented by supplementing the diet of susceptible animals with selenium and vitamin E.**
Sheep consuming selenium-deficient diets produce low wool yields and have increased incidence of periodontal disease.

Vitamin E also plays a key role in the animal’s normal immune response.

All breeds of sheep are susceptible to WMD, and the condition may develop under extensive or intensive management systems.

Treatment injection of lambs at birth with a quarter cc of a commercial Vitamin E-Selenium preparation is common practice (read the label to confirm dosage as there are several different formulations).

It is advisable to inject with a sterile 22 gauge (blue) needle under the skin (instead of into the muscle).

Ewes can be injected two to four weeks prior to lambing as well.

Lambs should be re-injected at one month of age if no feed supplementation is fed.

High concentrations of other minerals (e.g. calcium, sulphur, copper and zinc) and feed contaminants (e.g. nitrate, unsaturated fats, sulphates) may decrease absorption of selenium in the small intestine.

WMD can be prevented by supplementing the diet of susceptible animals with selenium and vitamin E.

Since it occurs mostly in lambs whose mothers were fed a selenium-deficient diet, supplementation of pregnant animals helps reduce disease in newborns. This is because selenium is transferred from dam to fetus across the placenta, and also is present in the colostrum.

While not much vitamin E is transmitted across the placenta, colostrum levels of vitamin E increase with ewe supplementation.

It is important to note that selenium in feed is governed by the Canadian Food Inspection Agency.

Injectable selenium compounds are available to prevent WMD in at risk-animals; however, injections are a poor alternative compared to routinely providing adequate selenium and vitamin E in the diet.

Ideally, the total diet for sheep and/or goats should contain 0.10 to 0.30 ppm of selenium.
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