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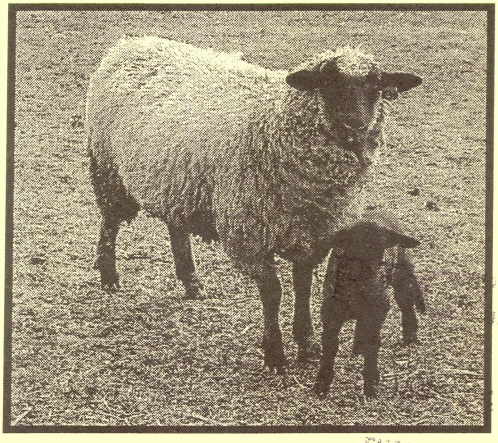

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Nancy J. Kerr completed the studies presented in this bulletin in partial fulfillment of the requirements for the master of science degree in animal and veterinary sciences at West Virginia University. A native of Carmichaels, Pa., she also holds a bachelor of science degree in animal and biological sciences from the Pennsylvania State University. **Robert A. Dailey** is station animal scientist in the West Virginia Agricultural and Forestry Experiment Station and a professor in the WVU College of Agriculture, Forestry and Consumer Sciences Division of Animal and Veterinary Sciences and advised Kerr during her graduate studies.

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By Nancy J. Kerr and Robert A. Dailey

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Abstract

Ringwomb is defined as failure of the cervix to dilate at parturition. Ringwomb causes sporadic dystocia, mainly in multiparous ewes bearing multiple fetuses. The most recognizable characteristic of ringwomb is the protrusion of placental membranes from the vulva with no sign of labor, but this is not without exception. Continued ineffective uterine contractions may cause separation of the placenta from the maternal cotyledons, which severs the blood supply from the dam to the fetal lamb. When this occurs, death of the lamb is inevitable unless it is promptly removed from the ewe. Because the cervix cannot be dilated by hand, a caesarean section usually is recommended. Various treatments with estradiol, progesterone, relaxin, prostaglandins, oxytocin, calcium borogluconate, and antibiotics have been used, but they have produced variable responses. The condition is not due to fetal malpresentation, premature birth, diseases that cause abortion, or mineral deficiencies. The condition may occur at the expected lambing date or be associated with a prolonged gestation of up to 14 days. Ringwomb typically does not affect ewes in two consecutive seasons, but consecutive cases have occurred. The condition may have a genetic component, because it frequently occurs in ewe families. It is proposed that ringwomb is determined by the genotype of the fetus. Families with the condition and ewes previously affected should not be used for breeding. In early dilation syndrome, incomplete dilation of the cervix occurs approximately seven to 14 days before term. Expulsion of the lamb(s) is not completed, and assistance is required. If assistance is not rendered the lamb dies with maceration of the lamb leading to septicemia, toxemia, and death of the ewe. Ewes delivering on their own have been found dead due to uterine prolapse or uterine tears with evisceration. The affected ewe might have little or no

udder development or a sudden, overnight blooming of the udder and have placental membranes protruding from the vulva when presented. The cervix can usually be dilated by hand, but the lambs, although born alive, are previable. The syndrome occurs most frequently in ewe lambs and first-lambing two-year-olds, but occasionally occurs in older ewes. Affected flocks can have over 30% morbidity. The syndrome occurs in all breeds and in all flock management situations.

INTRODUCTION

Parturition requires more than just contractions of the uterus. The increase in expulsive force must coincide with a decrease in resistance, as a result of softening of the collagen of the cervix, uterus, vagina and pelvic ligaments. Failure of these changes alone or in combination with fetal factors such as size, position, weight of fetal and placental tissues, and duration of second stage labor, might lead to a difficult birth (dystocia). Dystocia results in hypoxic or traumatic injury to the fetus that is unfavorable to its survival or traumatic injury to the ewe and fetus. Dystocia is a major cause of perinatal mortality, because it leads to neonatal as well as parturient deaths both during and soon after parturition. Dystocia can be a major cause of lamb losses in the flock and might result in great economic hardship to the producer. Caesarean section is in most cases an effective method for treatment of dystocia and is safe for the dam as well as the fetus, especially when performed as early as possible after onset of labor. Dystocia is usually higher in meat breeds than in wool breeds. One reason for the increased susceptibility of meat breeds may be that there has been a reduction in natural selection against dystocia, resulting from intensive shepherding and creating a demand for continuation of that practice.

Ringwomb is defined as failure of the ewe's cervix to dilate at parturition. Failure of the cervix to dilate may be caused by failure of secretion of the hormones that control labor or of the tissue response to hormonal secretions. Ringwomb causes sporadic dystocia, mainly in multiparous ewes bearing multiple fetuses. The condition is not due to fetal malpresentation, premature birth, diseases that cause abortion, or mineral deficiencies. Ringwomb has no predisposition associated with breed, age, or body condition score, but is associated with a significantly higher lambing percentage. The condition has been reported to be responsible for 15 to 32% of cases of ovine dystocia. [16]

The condition of incomplete dilation of the cervix has been the subject of many reports over the years. There are numerous theories as to its etiology, incidence and treatment. The objective of this bulletin is to present information toward a better understanding of the condition and its etiology, in order to treat affected ewes and to prevent or eliminate the condition from occurring in a flock.

Characteristics

The most recognizable characteristic of ringwomb is the appearance of placental membranes protruding from the vulva with no sign of labor, but this is not without exception. Ewes with ringwomb do not show typical signs related to first-stage labor. Ewes do not seek isolation from the remainder of the flock, nor do they lack an appetite. There is no noticeable swelling of the vulva or relaxation of the pelvic ligaments. Udder development appears slow but normal. Milk letdown sometimes is impaired and teat canals may be very hard. When examining the ewe, the producer or veterinarian will find an undilated cervix,

allowing only one or two fingers to be inserted into the uterus. Uterine contractions are weak and uncoordinated. The continued ineffective uterine contractions may cause separation of the placenta from the cotyledons, which severs the blood supply from the dam to the fetal lamb. The condition may occur at the expected lambing date or be associated with a prolonged gestation up to 14 days. Thirty percent of all ringwomb cases in one flock occurred in three-year-old ewes in second parity (Appendix, Table 2). Affected flocks can have up to 35% lamb mortality and 20% ewe mortality. Various manual, medical, hormonal, and surgical treatments either alone or in combination have been used to treat such cases, with varying degrees of success.

If a ewe with ringwomb suspected of being in the process of lambing is left alone for several hours, no change in dilation will occur. In non-productive labor, the cervical ring starts to close two to three hours after partial opening. [9] If the ewe is not provided assistance, spontaneous labor will occur after the fetus has died, usually 48 hours after initial onset of labor. The ewe, at this time, will require assistance to remove the remnants of the fetus or fetuses. Fetal autolysis can lead rapidly to septicemia and death of the ewe. If veterinary assistance is not available, an antibiotic should be administered to prevent systemic infection. In most cases, the ewe will breed the following season. Ringwomb does not typically affect ewes in two consecutive seasons, but consecutive cases have occurred. The condition may have a genetic component, because it frequently occurs in some ewe families.

BREED: Ringwomb has been observed in purebred and crossbred ewes. Hindson and Turner [11] reported cases in Clun Forest, Dorset Horn, Suffolk, Border Leicester, South Devon, Kerry Hill, and halfbred ewes.

BODY CONDITION: Body condition of affected ewes does not differ significantly from non-affected animals in the same flock. [11]

AGE OF EWE: Recorded incidences of ringwomb cases show that the majority of cases occur in ewes two years or older. Hindson and Turner [11] reported: four cases - two years old; nine cases - three years old; 17 cases, four years old; 14 cases - over four years old. The pelvic size of immature ewes has been suspected as one possible cause, but the majority of the cases recorded occur in ewes over two years of age. [20]

NUMBER OF LAMBS: Ringwomb has been associated with multiple bearing ewes. Hindson and Turner [11] reported the following observations: six ewes had one lamb; 32 ewes had two lambs; and three ewes had three lambs.

PARITY: Majeed [17] observed ringwomb in primiparous and two-year-old ewes having twin or triplet pregnancies. Hindson and Turner's study included ewes that had two known lambings. [11, 13] There is some confusion relating ringwomb to premature lambing detected by lack of milk. [11] This could result from an inability to differentiate between ringwomb and early dilation syndrome, or a hormonal interference (deficient relaxin) inhibiting lactogenesis. [23]

RELATED CONDITIONS

Early dilation syndrome (EDS) is similar to ringwomb but is considered to be separate from ringwomb. In early dilation syndrome, incomplete dilation of the cervix occurs approximately seven to 14 days before term. Expulsion of the lamb(s) is not completed and assistance is required. If assistance is not rendered, there is death of the lamb with

maceration leading to septicemia, toxemia and death of the ewe. Ewes delivering on their own have been found dead due to uterine prolapse or uterine tears with evisceration. The ewe has little or no udder development or a sudden overnight blooming of the udder and placental membranes protruding from the vulva when presented. The cervix usually can be dilated by hand, but the lambs, although born alive, are previable. The syndrome occurs most frequently in ewe lambs and first-lambing two-year-olds, but occasionally in older ewes. Affected flocks can have over 30% morbidity. The syndrome occurs in all breeds and in all flock management situations. No common link has been found from investigations of nutrition, physiology, toxicology, or infectious disease.

Ewes with diseases that cause abortion due to infectious agents may exhibit signs similar to ringwomb. Abortion with incomplete cervical dilation and abdominal straining may cause vaginal prolapse. Majeed [17] reported an association between ringwomb and vaginal prolapse in 18% (12 of 65) of the ewes observed. Pregnancy toxemia, consumption of poisonous plants, consumption of estrogenic plants, and lasalocid toxicity can cause ewes to display signs similar to ringwomb.

The cause of vaginal prolapse and the explanations for the development of the condition are still debatable. Edgar [6] was one of the first to suggest that sheep with vaginal prolapse could be suffering from hormonal imbalances. Since 1979, a hereditary predisposition has been suspected. [2] Stubbings [22] found lowered calcium concentrations in the plasma of animals with vaginal prolapse. Lowered calcium concentrations are known to be associated with elevated concentrations of estrogen. There are higher mean concentrations of plasma progesterone in the affected animals up to three days prior to parturition and a

subsequent fall until parturition. [21] The causes of hormonal irregularities are found partly in the dam, but probably mainly in the fetus and its membranes. The high progesterone in the animals with vaginal inversion and prolapse may be of placental origin and a manifestation of placental dysfunction. [10]

Hormonal Requirements During Pregnancy

The onset of parturition, although not completely understood, is brought about in part by a gradual increase in the secretion of estrogens from the placenta that occurs late in gestation. About 20 days before birth, the pituitary gland of the fetal lamb begins to increase the secretion of adrenocorticotropin (ACTH), stimulating the synthesis of the enzymes that convert progesterone to estrogen. Thus, placental progesterone production and consequently concentration of progesterone in maternal plasma fall and concentration of estrogen in maternal plasma rises. Estrogen stimulates secretion of prostaglandin $F_{2\alpha}$ by the placentomes and fetal membranes as well as increasing maternal secretion of oxytocin.

The changes in hormone secretion immediately before birth have been studied thoroughly in sheep. It is well established that the fetus influences its own birth. The sequence of events starts with activation of the hypothalamus and pituitary of the fetus. About five days before birth, a further increase in ACTH stimulates the fetal adrenal glands to secrete cortisol. Cortisol acts on steroid-secreting cells of the fetal cotyledon, which have been actively secreting the progesterone essential for maintenance of the pregnancy. Cortisol induces 17α-hydroxylase, an enzyme that switches steroid production from progesterone to estrogen. [1] Fetal estrogen crosses the placenta where it becomes unconjugated estradiol. At the

same time the clearance rate of progesterone increases.

The change to dominance of estrogen over progesterone is important in several respects, but particularly in the production of prostaglandins $F_{2\alpha}$ and E_2 ($PGF_{2\alpha}$ and PGE_2) in the cells of the myometrium and maternal placenta. Unconjugated estrogens promote, whereas progesterone inhibits, the synthesis of $PGF_{2\alpha}$ in sheep. Conversely, the enzymatic degradation of prostaglandins is enhanced by progesterone throughout pregnancy, but is depressed by the dominance of estrogen during the final days before birth. Thus, the endocrine changes promote the production of $PGF_{2\alpha}$ and of PGE_2, both of which are powerful stimulants of uterine muscle. Uterine contractions then facilitate the further release of the intercellular liposomal enzymes that synthesize prostaglandins, and the whole process becomes self-perpetuating.

The ultimate effect of uterine contraction, in combination with cervical relaxation and other changes, is to advance the fetus into the cervix and anterior vagina, which will elicit the release of oxytocin. Oxytocin augments the myometrial contractions, with the liberation of even more prostaglandins, so that the whole sequence takes on a cascading effect. The significance of this cascade and the reflex synchronized abdominal effort is to increase the efficiency and decrease the duration of second-stage labor, when the risks of anoxia and other hazards are maximal.

It is important that a number of workers have observed that exogenous progesterone cannot block parturition, either normal or induced, unless given in very high dosages (e.g. 200 mg/day).[3, 15] However, the experiments of Liggins, et al.,[15] showed that despite treatment of pregnant ewes with large amounts of progesterone, concentrations of progesterone in plasma failed to increase

greatly. It seemed that the progesterone was being cleared rapidly by the placenta and being converted partly to estrogen. Flint, et al., [7] showed that following the increase in the fetal cortisol before parturition, the placental enzymes are induced and that there is virtually a quantitative conversion of placental progesterone to estrogen.

Thus, when exogenous progesterone is administered in an attempt to block parturition, it probably increases circulating concentrations of estrogen. Consistent with this view, Liggins, et al., [15] reported a very high concentration of free estradiol-17β (700 pg/ml) in a ewe in which dexamethasone-induced parturition was blocked with progesterone. Even if a synthetic progestogen is used, the conversion of all the placental progesterone to estrogen as well as the conversion of C19 steroids from the fetal and maternal adrenals to estrogen may well lead to high concentrations of estrogen in the maternal circulation. In the sheep, large dosages of estrogen can induce uterine activity and parturition in ewes near term. [12, 5] However, this situation is associated frequently with prolonged labor and cervical dystocia. It would appear, therefore, that the failure of low dosages of progesterone to block parturition in sheep cannot be used to argue that progesterone is not a "myometrial blocker" in sheep.

PARTURITION

Parturition is divided into three stages: (a) preparation; (b) expulsion of the fetus; and (c) expulsion of the placenta. The preparatory stage is characterized by dilation of the cervix and rhythmic contractions of the longitudinal and circular muscles of the uterus. These contractions force the fetal fluids and membranes against the relaxed cervix, causing it to dilate. At the end of this stage, the cervix

expands, allowing the uterus and vagina to become a continuous canal. The fetus and chorioallantois are forced into the pelvic inlet where the chorioallantois ruptures, resulting in allantoic fluid flowing from the vulva.

The second stage begins when the distended amnion along with the head and part of the extremities are forced into the pelvic inlet. The presence of these parts of the fetus initiates reflex and voluntary contractions of the diaphragm and abdominal muscles. The passage of the fetus through the cervix into the vagina along with rupture of one or both of the fluid filled amnion and chorioallantois initiates reflex contractions, which force the fetus through the birth canal.

The fetus is expelled while still attached to the fetal membranes. The maternal cotyledons continue to supply oxygen from the ewe even if the expulsion is prolonged. The last fetal cotyledons are not detached from the maternal cotyledons until after the lambs are born, thus ensuring an oxygen supply until the young are able to breathe independently.

The expulsion of the fetal membranes is an active process associated with uterine contraction. Normally, the placenta of the ewe is delivered within two to eight hours following parturition.

RESULTS

Twenty-four cases of ringwomb out of 117 lambings occurred on one farm during the years of 1993 to 1997. Cases are listed below in five groups, classified according to common symptoms observed.

During the three weeks before parturition, the ewes were kept in a large indoor group pen at night and observed at 11 p.m. and 2 a.m. During nocturnal observations, characteristic labored breathing could be heard from ewes that were lying down. The ewes had a tendency to lie with their head and neck outstretched, appearing to have pain of the lower abdomen. All ewes appeared to have normal behavior while grazing and exercising during the day. Discharge of abnormally large amounts of thick, clear mucus from the vagina occurred during this period in affected ewes.

Ewes that were suspected to be lambing, but were making no effort, had either rupture of the chorioallantois with a significant amount of allantoic fluid expelled from the vulva, or a distended amnionic sac containing red/purple fluid. In two cases, the placenta and three to five cotyledons were protruding. In all cases, the cervix did not dilate sufficiently to allow more than two fingers to be inserted into the uterus. Manual dilation of the cervix was possible in the five cases in Group 5. In the 10 cases in Group 1, spontaneous dilation of the cervix occurred 48 hours after protruding membranes were first observed, yielding dead fetuses in the autolytic stage.

GROUP 1 - AUTOLYTIC TWIN OR TRIPLET FETUSES

YEAR	EWE NUMBER
1993	028, 189
1994	028, 102
1995	175, 306
1996	316, 215
1997	420, 102

An abnormally large amount of clear mucus discharge was observed every day up to three weeks prior to lambing. Incomplete cervical dilation was detected between seven and 15 days after the expected lambing dates. Udder development seemed slow but normal. Each ewe had placental membranes protruding from the vulva with no sign of labor. Between 24 and 48 hours later, the producer determined by vaginal examination that the cervical dilation was at least three fingers. Forceful abdominal straining began, and putrefied, autolytic lambs were delivered from the uterus. After parturition, the ewes received 20 cc of penicillin twice daily for three days. All ewes conceived the following season. In 1994, ewe 028 lost her wool three days after delivering putrefied lambs. The wool began growing back approximately one month after parturition. Ewe 028 remained in the flock and was exposed in the fall of 1994 and 1995, but did not lamb in either season.

GROUP 2 - ABDOMINAL STRAINING WITHOUT DILATION

YEAR	EWE NUMBER
1993	169, 178, 180
1995	04

Incomplete dilation was detected up to 12 days after the expected lambing date. Placental membranes protruded from the vulva. Forceful abdominal straining and extensive bleeding from the vulva occurred after moving the ewe(s) to lambing pens. The cervix was found to be hard and unyielding at vaginal examination. The ewes died within two hours of initial observation due to trauma and laceration of the uterus and cervix. A veterinarian was not available at the time of the emergencies for a caesarean section.

GROUP 3 - RECUMBENT WITH SIGNS OF SEPTICEMIA AND NO DILATION

YEAR	EWE NUMBER
1993	026
1994	05, 024

Incomplete cervical dilation occurred with protrusion of placental membranes between four and seven days after the expected lambing date. There were no signs of first-stage labor, no decrease in appetite, and no isolation from the rest of the flock. The ewes were checked every two hours for a change in behavior or cervical dilation. Forty-eight hours after initial observation, the ewes were recumbent. The pulse rate was subnormal, and the body

temperature was elevated to 103.5° F. Severe abdominal straining occurred with no cervical dilation and no vaginal discharge. The ewes were euthanized, and autolytic lambs were observed at the post-mortem examination.

GROUP 4 - CAESAREAN SECTION

YEAR	EWE NUMBER
1993	106
1994	04

Ewe number 106: the ewe was 14 days past the last possible lambing date. The ewe had full udder development. The ewe remained standing and shifted weight on all four legs for the last three days of gestation. The ewe was monitored for cervical dilation each of the last three days. Given the difficulty with the first two ewes of the season, the ewe was taken to the flock veterinarian for a caesarean section. The body temperature of the ewe was 104.5° F. Large twin ram lambs were delivered alive. One lamb died 30 minutes after birth after attempts to resuscitate failed. The second lamb was artificially reared. The ewe was treated with 20 cc of penicillin twice a day for three days. There was no evidence of uterine or cervical torsion. The ewe was bred in the following season but died three weeks before the expected lambing date.

Ewe number 04: a normal distended amnion was protruding from the vulva at the evening feeding. There was no sign of the fetal head or extremities. No other fluids were released to lubricate the vagina and vulva. The ewe showed no sign of labor and continued to consume feed. The ewe was placed in a lambing pen and examined for cervical dilation. No dilation or relaxation of the vagina or

cervix had occurred. The ewe was taken to the flock veterinarian for an emergency caesarean section one hour after initial observation. Large twin ram lambs were delivered. The first lamb died 10 minutes after birth after attempts to resuscitate failed. The second lamb died one hour after birth. The lambs were very weak and did not make any attempt to stand. The fetal lungs were pale in color and very firm. The umbilical cord was enlarged due to edema and was also pink in color in both lambs. The liver was also pale in color with slight necrosis in the center of the liver. Water green fluid was present in the abomasum, small intestine, and large intestine. The kidneys were submitted for diagnoses, along with cotyledons and amniotic fluid from the ewe. No *Campylobacter* or *Corynebacterium sp.* were isolated. Bacterial and fungal cultures, viral FA (fluorescent antibody) and microscopic examination of the fetal organs showed no evidence of infections organisms. The veterinarian preparing the necropsy report suggested ringwomb or early dilation syndrome as possible diagnoses. The ewe was treated with penicillin for three days and had a full recovery from surgery. The ewe was bred in the following season and died in labor (see group 2). Both ewes had a body condition score of 3.25 (scale: 1 = thin; 5 = fat).

GROUP 5 - EARLY DILATION SYNDROME

YEAR	EWE NUMBER
1993	148, 013
1994	011
1995	011
1996	011

Ewe number 148: incomplete dilation of the cervix occurred 13 days before the expected lambing date. Placental membranes protruded from the vulva with no sign of labor. Manual dilation was attempted for 30 minutes. Twin ewe lambs weighing four pounds each were delivered. The ewe had no udder development and no colostrum. The lambs were fed three ounces of goat colostrum. Both lambs were very weak and died six to 10 hours after birth. The ewe died 12 hours after delivery due to uterine and rectal tears with extensive internal bleeding.

Ewe number 011 (1994); incomplete dilation occurred 16 days before the expected lambing date. The cervix was dilated manually, and assistance was required to deliver large twin ram lambs weighing 14 pounds and 12 pounds, respectively. The ewe had slight swelling of the vulva and vagina, but no abnormal bleeding was present. The ewe had little udder development and the colostrum was extremely pale and water-like. The lambs were very weak and required assistance to stand and nurse. Colostrum supplement tablets were administered to both lambs, which remained with the ewe. Both lambs survived but were slow starters. The ewe increased milk production in the third week of lactation.

Ewe number 011 (1995): five days before the expected lambing date the ewe was suspected of lambing but making no effort. A small, distended amnionic sac containing red/purple fluid was protruding from the vulva. Cervical dilation was slow but progressed with manual dilation. Twin ram lambs were delivered alive but very weak and required assistance to stand and nurse. The ewe had little udder development and the colostrum was pale and water-like. Colostrum supplement tablets were administered to both lambs. Both lambs survived. The ewe was bred for the following season and repeated similar signs at lambing as in 1994 and 1995. The ewe developed mastitis and was culled from the flock after weaning.

Discussion

Over 65% of the 24 cases of ringwomb were in 1993 and 1994. There are several reasons why this may have occurred. First, the same service sire was used in 1993 and 1994. Not all of the same ewes were affected both years, but all lambs tended to have a higher than average birth weight. Second, a 14% protein ration with Bovatec (Hoffman-LaRoche, Basel, Switzerland) was fed during gestation and lactation in 1993. The same ration was fed in 1994 but without Bovatec. Bovatec (lasalocid) is used commonly as a feed additive at approximately 1 mg/kg of body for the prevention of coccidiosis and for improvement of feed efficiency. Studies in sheep and cattle have shown that lasalocid at increased levels can cause a toxic syndrome of congestive heart failure similar to that caused by the related drug monensin but at a higher dose. Monensin is reported to be toxic in sheep when fed at 8 mg/kg of body weight or at levels of 152 to 550 parts per million in feed. [8] A study on the effect of supplementation with concentrates and lasalocid during late pregnancy and lactation on productivity of Rambouillet ewes showed that

ram lambs were significantly heavier than ewe lambs only at birth. [19] Total weight gains between day 100 and day 146 of gestation were greatest in ewes supplemented with lasalocid. [19] Monensin (not approved for sheep) and Bovatec have been fed to ewes in late gestation to control toxoplasmosis (a protozoan parasite that causes abortion, encephalitis, and pneumonitis in sheep). Third, the ewes affected were all daughters, granddaughters, or great-granddaughters of one particular ram used on the farm as a service sire in 1990 and 1991. Finally, in 1993 and 1994, inclement weather changes occurred frequently in January, February, and March. The ewes were housed for long periods with little exercise due to continuous snowfall, below freezing environmental temperatures, and blizzard-like conditions.

Evidence for ringwomb as a hereditary condition that is sire-related is presented in Appendix Table 3. The data are from a closed ewe flock (1979-1998) where a new sire was purchased and introduced as the service sire every two years to prevent inbreeding. Sixty-eight percent (13 of 19) of the daughters of the sire 2 that was selected as a replacement were affected with either ringwomb or early dilation syndrome. Three of the six ewes not affected with ringwomb were only in the flock for one lambing season (one was hit by a car and killed, one was killed by dogs, and one was culled for a chronic foot abscess). The remaining three ewes not affected had single offspring and/or mostly male offspring. It is important to note that when sire 2 was the service sire to 46 ewes, no cases of ringwomb occurred (Appendix Table 3), but 66% of the ewes serviced required assistance at lambing (N.J. Kerr, unpublished data). However, when sire 4 was the service sire, 15 of 43 ewes were affected with ringwomb. It is interesting that 13 of 15 were daughters of sire 2, and two of 15 were granddaughters of sire 2 (Appendix Table 3 and

Figure 1). From these data, the author concludes that sire 2 introduced an autosomal recessive gene/gene mutation into the ewe flock and that the phenotype was not displayed until the carrier daughters were mated to another carrier. At least 13 of the 19 daughters kept as replacements were heterozygous for the recessive gene. Sire 3 was a non-carrier for this gene because zero of 33 ewes were affected when sire 3 was the service sire. However, sire 4 was a carrier of the recessive gene because 15 of 43 ewes were affected when sire 4 was the service sire. Sire 5 can also be classified as a carrier. All ewes affected when serviced by sire 5 were daughters (four), granddaughters (five), or great-granddaughters (one) of sire 2. Only one daughter of sire 5 was affected. She was a great-granddaughter of sire 2 and also was serviced by sire 5 to produce the condition (inbred). This particular ewe had a strong, maternal predisposition. Both the dam and the grandam of this ewe had the condition at least once (Appendix Figure 1). When sire 5 was the service sire in a neighboring flock, lambs exhibiting hereditary chondrodysplasia (spider lamb syndrome) were produced. Thus, sire 5 also was a carrier of spider lamb syndrome. Sire 2 had a gray pedigree, meaning that both the sire and grandsire of sire 2 had produced lambs with spider lamb syndrome, but sire 2 did not produce spider lamb syndrome in the closed ewe flock. Sire 1 was not a carrier because sire 1 was not in the pedigrees of 10 of the 19 affected ewes. It is concluded that sire 6 was not a carrier for ringwomb or early dilation syndrome.

One last important point to be established is that a large number of ringwomb cases occurred during the second and third weeks of February (Table 1). This was first established in the 1950s, but it was argued that mid-February was the peak time for lambing and therefore, the number of ewes affected for the number of ewes lambing

during this period was not statistically significant. But from communication with other producers who used multiple sires per breeding season, the author has noticed that the condition occurred mainly in mid or late February, independent of the peak of lambing season, in both fall and spring lambing operations. One theory to explain this effect is that carrier rams may be more likely to breed around October 1, independent of ram introduction, but dependent on environmental conditions. There is also an increased incidence in ewes that are either two years old at first parity or three years old at second parity.

Conclusion

Dystocia due to failure of the cervix to dilate is seen occasionally in the cow and ewe and very rarely in other domestic animals. Failure of the cervix to dilate properly at the time of parturition is associated with or observed in: uterine inertia, uterine torsion, metritis, placentitis, death of the fetus, diffuse peritonitis due to traumatic gastritis, hydrops of the fetal membranes, a terminal condition in severe septic or toxic diseases, mummified fetus, in certain abortions, preparturient paresis, and pregnancy toxemia in ewes.

Vaginal examination often permits identification of the cause of dystocia. It may be difficult to distinguish between the first stages of labor in a normal animal and failure of cervical dilation (ringwomb) or uterine torsion in an animal with non-productive labor.

Ringwomb is not caused by malpresentation, mineral imbalance or deficiency, or premature lambing. Ringwomb does not result from consumption of red clover or feedstuffs containing estrogens. Unconjugated estrogens promote, whereas progesterone inhibits, the synthesis of

$PGF_{2\alpha}$ in sheep.

Satisfactory results, either by surgical or medical treatment, depend on a correct early diagnosis and treatment of the condition. Ringwomb should be treated immediately upon detection. Treatment is important in reducing the mortality rate of newborns and dams. Caesarean section is still the best treatment for ringwomb cases, although it is not an economical practice.

The causes of ringwomb and early dilation syndrome are still not completely understood, although it is well established that the fetus influences its own birth. After careful review of lambing records from two flocks (one closed, one open) that spanned from 1986 to 1998, the author has found several associations that indicate that ringwomb and early dilation syndrome are genetically transmitted as an autosomal recessive gene.

1. Ringwomb and early dilation syndrome have appeared in certain maternal bloodlines, and when those bloodlines have been inbred, the frequency of the condition has increased.

2. In sets of twin ewes, one ewe may exhibit ringwomb and the other may never exhibit the condition at all. If the condition were to an intrauterine effect or an environmental factor such as nutrition, disease, or weather conditions, both ewes should be affected.

Because ringwomb does not typically affect ewes in two consecutive seasons, it is proposed that the occurrence of ringwomb is determined by the genotype of the fetus.

Fetuses that are homozygous recessive for the gene mutation responsible for ringwomb appear to cause the condition in the heterozygous ewe. Although ringwomb occurs more often in ewes bearing multiple fetuses, ewes bearing singletons have been affected. Further research should investigate the possibility of an autosomal recessive gene in the fetus of ewes affected with ringwomb, perhaps located on the same chromosome as Spider lamb syndrome.

It is important to remember that not all cases of incomplete cervical dilation are ringwomb. Every case of dystocia is individual and needs special treatment.

Recommendations

Recommendations for eliminating ringwomb from a flock include: culling affected ewes, culling female offspring from affected ewes, culling ewes with vaginal prolapse or a family history of vaginal prolapse, culling ewes that are carriers of the autosomal recessive disorder as indicated by its occurrence in their daughters, and culling ewes that do not lactate well. Families with the condition and those ewes previously affected should not be used for breeding. Therefore, each ewe that exhibits ringwomb should be culled immediately. All previous female offspring and the service sire when the condition occurred should be culled as well. Ewes should be fed a good quality ration and hay during the last six weeks of gestation. In addition, during the last six weeks of gestation, if a sudden change in weather occurs, precautions should be taken to minimize changes to the ewe (mainly stress, exercise, and feed related). Maintaining excellent flock records may allow the producer to prevent ringwomb from occurring in a flock by selecting replacements from superior performing ewes not displaying reproductive abnormalities. Ewes that

display any abnormality during gestation and/or parturition should be culled from the flock. Rams should be obtained from flocks that have adequate vaccination programs and have not experienced ringwomb or vaginal prolapse within the past five years. Mating large-breed yearling and two-year-old ewes to a medium-breed ram can reduce incidence of dystocia by decreasing fetal size and birth weight.

Treatment

Early signs indicating or identifying a ewe with ringwomb are characteristic labored breathing while lying and abnormally large amounts of thick, clear, vaginal mucus discharge during the last three weeks of gestation.

If a ewe is suspected of lambing with ringwomb with no abdominal straining, it is best to treat the ewe with an antibiotic and leave the ewe alone. The antibiotic will assist in prevention of septicemia in the ewe if death of the lambs occurs and the lambs cannot be removed within 24 hours. The antibiotic will also prevent the lambs from becoming putrefied. Monitor the ewe every two hours for abdominal straining and dilation. The ewe should deliver dead lambs within the next 12 to 48 hours with assistance. Prolonged manual dilation on an incompletely dilated cervix will result in uterine prolapse, vaginal tears, and/or uterine tears.

If ringwomb occurs with abdominal straining it is best to administer a spinal, epidural anaesthetic to subdue straining. If ringwomb is detected very early, a Caesarean section is the most effective treatment in yielding viable lambs. Caesarean section may not be an economical treatment in most flock situations.

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APPENDIX

TABLE 1. Parity and age of ewes with ringwomb and early dilation syndrome (EDS) in a Pennsylvania flock.

1993 EWES WITH RINGWOMB/EDS	DATE OF OCCURRENCE	PARITY	AGE	GROUP NO.
178	02-16-93	2	3	2
148	02-17-93	3	4	5
189	02-19-93	2	3	1
106	02-22-93	1	1	4
169	02-27-93	2	3	2
028	02-28-93	1	2	1
180	03-03-93	2	3	2
026	03-27-93	1	2	3
013	04-19-93	2	2	5

1994 EWES WITH RINGWOMB/EDS	DATE OF OCCURRENCE	PARITY	AGE	GROUP NO.
028	01-24-94	2	3	1
102	02-06-94	1	2	1
05	02-10-94	2	3	3
024	02-12-94	3	3	3
04	02-13-94	3	3	4
011	02-13-94	3	3	5

1995 Ewes with ringwomb/EDS	Date of occurrence	Parity	Age	Group No.
04	02-20-95	4	4	2
175	02-25-95	4	5	1
011	03-08-95	4	4	5
306	03-17-95	1	1	1

1996 Ewes with ringwomb/EDS	Date of occurrence	Parity	Age	Group No.
011	02-05-96	5	5	5
316	02-12-96	2	2	1
215	02-12-96	2	3	1

1997 Ewes with ringwomb/EDS	Date of occurrence	Parity	Age	Group No.
420	02-23-97	1	2	1
102	03-12-97	4	5	1

TABLE 2. Incidence of ringwomb and early dilation syndrome grouped by parity and age in a Pennsylvania flock during 1993-97.

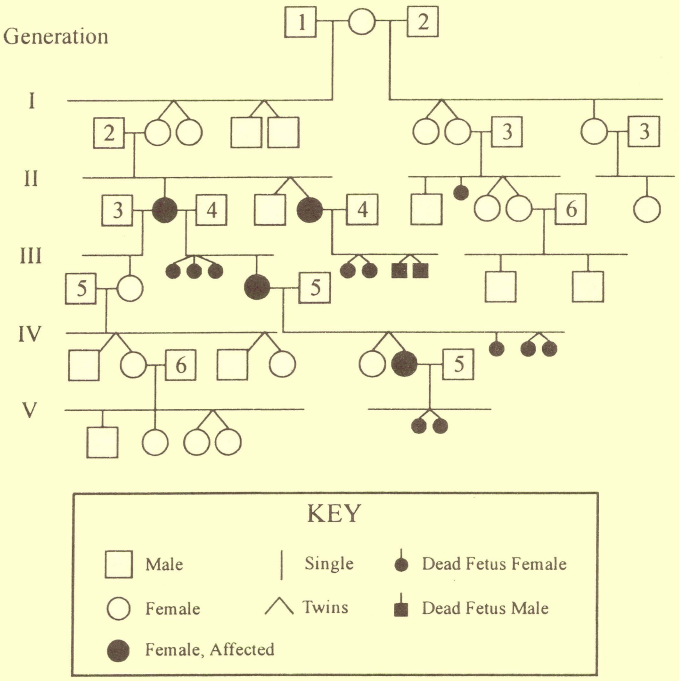
AGE	PARITY	NO. AFFECTED	% OF TOTAL CASES	NO. OF EWES LAMBING	% OF ALL EWES LAMBING
1	1	2	8.3	17	11.8
2	1	4	16.7	26	15.4
2	2	2	8.3	12	16.7
3	2	8	33.3	19	42.7
3	3	2	8.3	9	22.2
4	3	1	4.2	13	7.7
4	4	2	8.3	3	66.7
5	4	2	8.3	8	25.0
5	5	1	4.2	4	25.0
6	5	0	0	3	0
6	6	0	0	2	0
7	6	0	0	1	0
7	7	0	0	0	0
		24	99.9	117	

TABLE 3. Number of ewes serviced and number of female offspring with ringwomb in a Pennsylvania flock during 1993-97.

Service Sire						
Sire	1	2	3	4	5	6
Number of ewes						
With ringwomb	0	0	0	15	9	0
Without ringwomb	6	46	33	28	58	37
Total	6	46	33	54	67	37
Occurrence				33	13	

Female Offspring						
Sire						
Number of daughters						
With ringwomb	0	13	2	3	1	0
Without ringwomb	12	6	17	14	32	11
Total	12	19	19	17	33	11
Occurrence		68	11	18	3	

FIGURE 1. Occurrence of ringwomb in one selected ewe family showing the transmission of an autosomal recessive trait. Numbered males refer to sires and service sires in Table 3. Sire 2 introduced an autosomal recessive gene, which was not observed until the female offspring was serviced by sire 4. Sires 4 and 5 were both carriers of the gene. Notice that the affected daughter of sire 5 was also serviced by sire 5 to produce the condition. Female offspring of sire 2 were unaffected when serviced by sire 3. Producers suspecting ringwomb in their flock should first trace the maternal pedigree, followed by sires and service sires. Ewes were affected in the season that dead twin or triplet fetuses were produced.



GLOSSARY

Adrenocorticotropic Hormone (ACTH): A hormone released from the anterior lobe of the pituitary gland in response to the corticotropic releasing factor. ACTH stimulates the adrenal cortex to produce cortical hormones (corticosterone, cortisone, etc.) and to decrease the level of ascorbic acid in the adrenal cortex.

Anoxia: Lack of oxygen or reduction of oxygen levels in body tissues below normal physiological levels.

Autolysis: The destruction of cells or tissues by substances within them, as after death.

Autosomal: Any chromosome other than the sex chromosomes.

Blooming: Refers to early lactation, showing promise of good milk yield.

Dystocia: Difficult parturition.

Hypoxia: Abnormally low supply of oxygen in the blood preventing normal functioning of body organs.

Morbidity: The ratio or percentage of individuals exhibiting clinical signs of disease in relation to the number of individuals in the group.

Mortality: The percentage of deaths in relation to the number of individuals in the group.

Multiparous: Referring to females that have undergone more than one cycle of pregnancy and parturition.

Necrosis: Death of cells or a group of cells (tissue).

Neonatal: Pertaining to the newborn.

Parturient: Giving birth or about to give birth to young.

Previable: Inadequate lung development or maturation to survive.

Primiparous: Referring to females that have undergone only one cycle of pregnancy and parturition.

Prolapse: Protrusion of an organ or part of an organ from its normal position.

Ringwomb: Resistance or inability of the cervix to dilate, thus obstructing the natural delivery of the fetus.

Septicemia: A morbid condition of the blood due to the absorption of poisonous products of putrefaction; septic infection.

Toxemia: Any condition of blood poisoning caused by bacterial toxins transported through the blood stream from a focus of infection.

Notes

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