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Review article

# A review of dystocia in sheep

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ABSTRACT

This review aims to describe the nutritional and non-nutritional factors that may affect parturition and dystocia in sheep. Dystocia is associated with fetopelvic disproportion, uterine inertia, failure of the cervix to fully dilate, malpresentation and disease or congenital defects in lambs. Dystocia can result in lambs that are born dead, or lambs that survive parturition but sustain birth injury including central nervous system damage. Dystocia risk is increased with high or low birthweight lambs, high (fat) or low liveweight ewes, and small first parity ewes. Other factors implicated include low muscle glycogen, pregnancy toxaemia, mineral imbalance causing hypocalcaemia, and a lack of antioxidant nutrients. Addressing these risks requires differential nutritional management for single and multiple bearing ewes. There is also evidence for stress and environmentally related dystocia. The stress related hormones cortisol, adrenaline and ACTH play a major role in the initiation and control of parturition in the sheep indicating a need for adequate supervision during lambing, provision of adequate feed and shelter at the lambing site, and small flock size to reduce physical and environmental stress. Hormonal control of parturition can be further disrupted by xenoestrogens or phytoestrogens in clovers and medics. Oestrogenic plants are still widely grown in mixed pastures but should be not be grazed by pregnant ewes. There is clearly a genetic component to dystocia. This is partly explained by incompatibility in physical size and dimensions of the ram, ewe and lamb. A rapid reduction in dystocia through direct genetic selection is problematic with low heritability of dystocia and some of its indicator traits such as lambing ease. This review provides broad interpretation of the literature, but conclusions are not definitive with widespread inconsistency in reported results. Further research is required to investigate dystocia under commercial production conditions, and this should be complemented by focussed studies under controlled conditions. Priorities include defining the fitness of the ewe to lamb, the role of stress and environment on parturition and the use of indicator traits to select for ease of birth.

## 1. Introduction

The incidence of lamb mortality prior to weaning is high. Hinch and Brien (2014) summarised results from 15 Australian publications to conclude that, on average, 20–30% of lambs die before weaning and 74 % of these deaths are within three days of birth. Others have reported lamb mortality rates from 10 to 25% in Australia, UK and New Zealand (Mellor and Stafford, 2004; Celi and Bush, 2010; Ferguson et al., 2014). It is likely there are similar losses in all other extensive sheep production systems including those in China and South America. The major causes of perinatal mortality are dystocia (19–67%) and starvation-mismothering-exposure (SME; 30–48%) (Dennis, 1974; Holst et al., 2002; Refshauge et al., 2016). Dystocia is defined as a difficult birth due to a long, unassisted parturition or prolonged delivery requiring assistance (Arthur, 1975; Zaborski et al., 2009).

Neonatal mortalities have been estimated to cost the Australian sheep industry approximately AU$540.4 million each year with approximately AU$219 million estimated to be from dystocia (Lane et al., 2015). A 50 % reduction in dystocia has been predicted to gain the industry approximately AU$77 million (Lane et al., 2015). These estimates do not account for possible production loss from surviving ewes and lambs in subsequent years.

Ewe mortality is also a potential source of economic loss associated with dystocia, but there is little information on the incidence and cause

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of ewe deaths associated with parturition. Annual ewe mortality has been estimated at 2–10 % in New Zealand and Australia, with higher susceptibility in multiple-bearing ewes (Trompf et al., 2011; McGrath et al., 2013; Ferguson et al., 2014). The highest risk of ewe mortality is during the periparturient period (Mavrogianni and Brozos, 2008), but few data are available to support this expectation or to indicate the risks associated with dystocia.

Peri-natal lamb and periparturient ewe mortality not only have financial costs associated with production losses but also present an animal welfare concern. There is an increasing demand for improved ethical treatment of farm animals. Ferguson et al. (2014) summarised some of the consequences of these demands as:

- Increasing pressure to include welfare standards in trade agreements;
- Differentiation of products and brands based on animal welfare standards;
- Increased consumer pressure on governments to introduce a social license to operate;
- Products required to include welfare standards in provenance certification.

Failure to address and improve sheep welfare including ewe and fetal or lamb mortality during and after pregnancy will compromise the brand, consumer trust and confidence, and may even threaten the licence to operate in some markets.

This review aims to identify the nutritional and non-nutritional stressors that affect parturition and dystocia in ewes.

## 2. Impact of dystocia on sheep survival, health and production

### 2.1. Maternal and fetal dystocia

Dystocia may originate from either the ewe (maternal dystocia) or lamb (fetal dystocia); (Table 1). Fetopelvic disproportion may be considered as both maternal (e.g. inadequate size of pelvis/birth canal) or fetal (e.g. oversized foetus) dystocia (Majeed and Taha, 1995; Noakes et al., 2001), however in this review has been considered with other causes of maternal dystocia consistent with other studies (Cloete et al., 1998). The causes of maternal dystocia are inconsistent and vary between breed with reports of fetopelvic disproportion in South African Mutton Merinos, uterine inertia in Dormer ewes and incomplete cervical dilation in other breeds (Thomas, 1990; Cloete et al., 1998; Sirinivas and Sreenu, 2009; Mostefai et al., 2019). Fetal dystocia explains between 36 % and 75 % of cases and is mostly caused by malpresentation (Majeed and Taha, 1995; Cloete et al., 1998; Sirinivas and Sreenu, 2009; Ennen et al., 2013; Mostefai et al., 2019).

#### 2.1.1. Maternal dystocia

Maternal dystocia due to fetopelvic disproportion is the result of incompatibility between fetal size and the dimensions of the pelvis. This can occur over successive lambings (McSporran and Fielden, 1979) and is commonly associated with high lamb birthweight. In ewes, the related dimensions of the pelvis including conjugate diameter (distance between pubic symphysis and sacral promontory) and pelvic area are negatively correlated with the length of parturition and incidence of dystocia in many breeds (Fogarty and Thompson, 1974; McSporran and Fielden, 1979; Kilgour and Haughey, 1993; Cloete et al., 1998).

Ringwomb, vaginal prolapse, uterine torsion or inguinal hernia may all result in maternal dystocia due to obstruction of fetal delivery. Ringwomb refers to inadequate softening and dilation of the cervix during parturition and is often reported as an important contributor to maternal dystocia (Thomas, 1990; Sirinivas and Sreenu, 2009; Ennen et al., 2013; Mostefai et al., 2019). In Australia, ringwomb has been reported to vary between seasons (Jackson, 2004). Obstruction due to failure of cervical dilation may also be observed in association with vaginal prolapse, cervical damage, uterine inertia, or breech and other malpresentations of lambs (Jackson, 2004; Menzies, 2006). Uterine torsion is uncommon in ewes, though may be confused with incomplete cervical dilation during clinical examination (Scott, 2011). Inguinal herniation of the uterus may also be associated with fetal obstruction but is uncommon in ewes (Sirinivas and Sreenu, 2009; Mostefai et al., 2019).

Uterine inertia is failure of the uterus to expel the fetus. Primary inertia results when uterine activity is reduced or disrupted in ewes with metabolic diseases such as hypocalcaemia and pregnancy toxaemia, endocrine disruption such as with consumption of oestrogenic clover, or physical injury including abdominal wall rupture, abdominal hernia, umbilical hernia or perineal hernia (Moule, 1961; Robalo Silva and Noakes, 1984; Jackson, 2004; Barbagianni et al., 2015). Uterine activity and progression of labour can also be inhibited by stress during lambing (Naaktgeboren, 1979). Secondary uterine inertia due to myometrial exhaustion may be the consequence of prolonged labour, fetal malpresentation, loss of uterine tone, or stress (Dwyer et al., 1996; Cloete et al., 1998). Overstretching of the myometrium or poor uterine tone following delivery of other fetuses may cause uterine inertia in multiple-bearing ewes (Jackson, 2004).

Uncommon causes of maternal dystocia include hydropic conditions (hydrops allantois and hydrops amnion) and sciatic nerve paralysis impacting function of the hindquarters (Patil et al., 2014; Prasad et al., 2014).

#### 2.1.2. Fetal dystocia

Fetal dystocia due to malpresentation of lambs is associated with at least 50 % of dystocia cases in studies based on requirement for intervention at parturition (Dwyer et al., 1996; Cloete et al., 1998). Common malpresentations include flexions of the neck, shoulder or carpus but dystocia may also result from breech presentation (posterior presentation with retention of the hind limbs) or simultaneous presentation of lambs (Jackson, 2004). Congenital defects that result in arthrogryposis or fetal malformation (monsters) may result in malpresentation of the fetus. Congenital defects such as hydrocephalus, monsters, hydrops fetalis are uncommon in sheep, but may present as fetopelvic disproportion (Jackson, 2004; Basher, 2006).

#### 2.1.3. Other causes of dystocia

Failure of the initiation of birth and subsequent dystocia may result from fetal death *in utero* (Jackson, 2004). *In utero* death may be caused by compromised placental function, infection, exposure to toxic agents, metabolic disease, stress, or congenital defects. Autolysis and emphysema of dead fetuses can compromise the uterus and increase the risk of vaginal or uterine damage during delivery (Menzies, 2006).

**Table 1**
Causes of maternal and fetal dystocia.

| Maternal dystocia | Fetal dystocia |
|---|---|
| Fetopelvic disproportion | Malpresentation |
| Failure of the cervix to fully dilate (ringwomb) | Fetal disease and death |
| Vaginal prolapse | Congenital defects |
| Uterine torsion | |
| Inguinal hernia | |
| Uterine inertia | |

### 2.2. Dystocia and lamb survival

Irrespective of the aetiology of dystocia, the consequences of prolonged parturition and dystocia are an increased risk of asphyxia and circulatory compromise leading to central nervous system (CNS) lesions and oedema. In one study, up to 33 % of newborns experienced severe asphyxia with risk 15.6 times higher for twin-born lambs compared to singles (Dutra and Banchero, 2011). Hypoxia and acidaemia evident as

haemorrhaging and congestion of the meninges of the CNS have been reported in dystocic deaths (Haughey, 1973a). More recently, Dutra et al. (2007) reported that all autopsied lambs presented with hypoxic ischemic lesions of the CNS indicating a lack of oxygen. Lesions are usually produced ante- or intra-partum, although forming post-partum lesions is possible if the lungs fail to properly inflate. Increased neuronal death in the dentate gyrus of the hippocampus has been observed in lambs that died from of dystocia compared to lambs that died from SME or barbiturate overdose (Lashley et al., 2014). Haemorrhage of the CNS and blood vessel dilation may be artefactual and gross lesions can occur in absence of neuronal degeneration or CNS necrosis (Robertson et al., 2020). Severity of lesions is correlated with mortality risk (Haughey, 1980, 1982) and Barlow et al. (1987) proposed that maternal contribution to neonatal lamb mortality through intrapartum asphyxia and reduced placental efficiency warrants further investigation.

#### 2.2.1. Classification of dystocia and SME

Dystocia is classified into three broad categories based on evidence injury including tissue autolysis, inflammatory lesions, haemorrhage, and oedema. These lesions are assessed in combination with observations that indicate viability following birth such as aeration of lung tissue, evidence of walking, the metabolism of brown adipose tissue, and the presence of milk in the abomasum (Table 2). Meningeal lesions were first characterised by Haughey (1973a, 1973b) and, using these, Holst (2004) subsequently defined the three categories as Dystocia A, B and C (Table 2). The categories were later modified by Refshauge et al. (2016) to classify lambs with subcutaneous oedema that may have breathed or walked into Dystocia A (Table 2). The critical categorisation of lambs into one of the three classes of dystocia relies on the presence of oedema and CNS lesion scores.

Starvation-mismothering-exposure cases may have signs consistent with birth injury (Dystocia C) except for CNS lesions (Table 2). While this categorization is now well defined, the assessment of CNS lesion scores is subjective, and categorization of lambs into the death categories relies heavily on assessor experience and bias. Dutra et al. (2007) concluded that lesions explain most deaths within 6 days of birth despite categorising 49 % of lambs to starvation-mismothering-predation and 40 % to dystocia-stillbirth-birth injury. Other studies have reported that 20–60 % of lambs categorised as SME also experienced birth stress apparent as CNS lesions (Alexander et al., 1980; Duff et al., 1982; Haughey, 1982, 1983; Knight et al., 1988).

These studies demonstrate the complexity in differentiating both within dystocia categories and between dystocia and SME and indicate that the proportion of deaths due to dystocia is likely to be underestimated.

#### 2.2.2. Prospects for survival of lambs born alive

Long and difficult labours are associated with reduced ewe behaviours important for establishing maternal bond with the lamb, and lower lamb survival (Arnold and Morgan, 1975; Cloete et al., 1998; Dutra and Banchero, 2011). For example, ewes display a reduced frequency of low-pitched bleats, less grooming of the lamb by the ewe, and an increase in rejection behaviour following a prolonged or difficult birth (Dwyer et al., 2003). Lambs with poor vigour in their first hour of life are less likely to survive (Murphy and Lindsay, 1996). Longer parturition has been associated with poorer lamb viability at birth, including latency to stand or suckle (Dwyer et al., 1996; Dutra and Banchero, 2011; Fonsêca et al., 2014), although this is not consistent in all studies (Duff et al., 1982). Delayed appearance of righting movements, impaired suckling ability and depressed neonatal behaviours have been reported for lambs assisted at birth (Dwyer, 2003; Dwyer and Bünger, 2012). Lower suckling ability reduces access and absorption of colostral immunoglobulins (Hinch and Brien, 2014).

Lambs impacted by dystocia are vulnerable to wind chill when exposed to cold temperatures (Haughey, 1980) and to dehydration during hot conditions. Lambs with impaired suckling behaviours have reduced energy and fluid intake. Heat production by the lamb may also be impaired by hypoxia or placental insufficiency (Haughey, 1980; Eales et al., 1982). Severe hypoxia and acidaemia may be associated with depressed heat production for up to 72 h post-partum (Eales and Small, 1985; Darwish and Ashmawy, 2011). Dystocia-induced hypothermia may be a direct result of placental insufficiency and chronic hypoxaemia (Mellor and Stafford, 2004) or acute hypoxaemia from short term occlusion of the umbilical cord (Eales and Small, 1985).

Sub-acute CNS injuries associated with dystocia are not always fatal. However, the impacts on suckling, heat production and the ewe-lamb bond are often so challenging that the neonate is unable to survival without human intervention, particularly under cold or hot conditions. When kept warm, well fed and with careful attention to managing risks for infectious disease, very few hand reared lambs die and even very low birthweight lambs can be kept alive with appropriate care (Greenwood et al., 1998).

### 2.3. Dystocia and ewe health and survival

Dystocia-associated impacts on the ewe may be attributable to trauma, haemorrhage and septicaemia (Mavrogianni and Brozos, 2008). There are few studies that describe the incidence of complications in extensive sheep production systems or the consequences on ewe survival and production.

Trauma associated with dystocia and/or obstetrical intervention may result in haemorrhage, subsequent sepsis or organ prolapse (Hindson and Winter, 2007; Roger, 2009). Examples of injuries that may occur during dystocic parturition include uterine rupture, and

**Table 2**
Methodology developed by Holst (2004) and refined by Refshauge et al. (2016) to classify lamb deaths associated with dystocia or starvation-mismothering.

| | Dystocia A (Holst, 2004) | Dystocia A (Refshauge et al., 2016) | Dystocia B (stillbirth) | Dystocia C (birth injury) | Starvation-mismothering |
|---|---|---|---|---|---|
| Oedema* | + | + | – | – | – |
| Central nervous system lesion score ** | ≥2 | ≥2 | ≥3 | ≥3 | < 2 (no lesion) |
| Walked | – | +/- | +/- | +/- | + |
| Breathed | – | +/- | +/- | + | + |
| Fat deposits metabolised*** | – | – | – | + | + |

* subcutaneous oedema of head or shoulders.
** Central nervous system (cranial, spinal) lesion score scale 1–5 (1 = no lesion; 5 = severe haemorrhage, obvious blood clots & congestion).
*** Pericardial and perirenal fat deposits metabolised.
+ presence of observation.
- absence of observation.
+/- observation sometimes present.

tearing of the cervix, vagina, vulva or rectum.

For ewes, dystocia increases the risk of acquiring an infection related to parturition. Dystocia, and related conditions (uterine or vaginal prolapse, retained placenta or fetal tissue or post-parturient ketosis) predispose ewes to metritis that can result in septicaemia and death if untreated (Leontides et al., 2000; Tzora et al., 2002). Ewes with metritis often respond to treatment with an antimicrobial agent, oxytocin and non-steroid anti-inflammatories with no adverse consequences to future fertility (Mavrogianni and Brozos, 2008; Roger, 2009). Clostridial infections including *Clostridium chauvoei* (post-parturient gangrene) subsequent to trauma during parturition or obstetrical interference may progress to severe necrotizing myositis, toxaemia and death (Lewis, 2007; Roger, 2009). Good hygiene during lambing reduces the risk of metritis, especially where obstetrical assistance is provided. Vaccination of pregnant ewes and good hygiene during lambing also reduces the risk of complications associated with clostridial diseases.

### 2.4. Consequences for future production of surviving ewes and lambs

The 'carry-over' impacts of dystocia on the subsequent reproductive and productive performance of surviving ewes and lambs are not well studied. Haughey (1982) reported no permanent effect on growth or fleece production for up to two years following a prolonged or stressful birth. Ewes with dystocia at their first lambing may have a higher risk of dystocia at subsequent lambings (Horton et al., 2018).

## 3. Physiological risk factors for dystocia

### 3.1. Birth weight, conformation and litter size

Dystocia is a significant contributor to lamb deaths across all litter sizes (Holst et al., 2002; McHugh et al., 2016; Refshauge et al., 2016; Holmoy et al., 2017; Kenyon et al., 2019). When adjusted for birthweight, the difference in dystocia between litter size groups is reduced but not eliminated (Woolliams et al., 1983; Brown et al., 2014). This indicates that dystocia is caused by factors additional to high birthweight. Lamb conformation is a factor, Brown et al. (2014) reported some categories of death from dystocia were positively correlated with lamb thorax circumference adjusted for birthweight and negatively correlated with crown-rump length at a given birthweight. In contrast, the risk of birth injury was negatively correlated with thorax circumference and positively correlated with crown-rump length at a given birthweight. Therefore, selection for longer and thinner lambs may not reduce the incidence and consequences of dystocia (Brown et al., 2014). Fetal entanglement, malpresentation and prolonged birthing process are more significant contributors to dystocia in multiple-born lambs (Woolliams et al., 1983; Hinch et al., 1986; Speijers et al., 2010; Dwyer and Bünger, 2012; Kenyon et al., 2019).

A quadratic relationship exists between birthweight and the risk of peri-parturient lamb death (Knight et al., 1988; Geenty et al., 2014). Although optimal birthweight varies between breeds and birth types, studies on the Australian Sheep Cooperative Research Centre Information Nucleus Flock indicated that the risk of dystocia is lowest for lambs with birthweight between 4.5 and 5 kg with higher risks at the birthweight extremes in singles, twins and triplets (Brown et al., 2014). Similarly, Everett-Hincks and Dodds (2008) observed in singles, twins and triplets that the risk of death due to dystocia increased as the birthweight of the individual lamb deviated from the mean for its birth type. Horton et al. (2018) divided dystocia into low and high birthweight dystocia and reported that the rate of low birthweight dystocia increased with litter size while high birthweight dystocia was not affected by litter size. Refshauge et al. (2016) also reported that lighter multiple-born lambs were more likely to display dystocia than lighter singles. Interestingly, Horton et al. (2018) reported that the optimum birthweight to minimise the risk of dystocia in singles may be lower than that for multiple-born lambs.

Combined, these findings indicate that there is an optimum range in birthweight for each birth type that will reduce the risk of dystocia. Management of lamb birthweight to avoid dystocia is however challenging, with large variation in birthweight (1.5–10 kg) reported for lambs born to ewes similarly managed to body condition targets (Geenty et al., 2014).

### 3.2. Ewe live weight and body condition score

The relationship between either ewe liveweight or body condition score and dystocia is inconsistent across studies. Body condition score offers advantage as an objective measure because it is independent of frame (skeletal) size whereas liveweight is a combination of both frame size and condition. Hall et al. (1994) reported that ewe liveweight at joining had no influence on dystocia rates. In contrast, Horton et al. (2018) reported positive relationships for both ewe liveweight and body condition score at joining with the risk of both low and high birthweight dystocia. Further, they reported a positive relationship for ewe liveweight at days 60 and 90 of pregnancy and the risk of both low and high birthweight dystocia.

Liveweight gain in the last six weeks of pregnancy was reported by Scales et al. (1986) to be positively associated with increased rates dystocia in singles but decreased rates in twins. However, ewe liveweight just prior to lambing had little effect on the proportion of ewes requiring assistance. Horton et al. (2018) found a positive relationship between ewe liveweight at day 120 of pregnancy and the risk of dystocia, though high condition score at day 120 of pregnancy was associated with a reduced risk of low birthweight dystocia and a tendency for lower rates of high birthweight dystocia. Holst et al. (2002) observed greater CNS lesion scores for offspring of fatter ewes and concluded a 'dam fitness quotient' was present whereby ewes with high fat level were predisposed to dystocia, fetal birth injury and increased lamb mortality. Morris (1973) and Holst et al. (1986) also reported that overfeeding during certain 'windows' of gestation increased gestation length. This may result in slightly larger lambs and increased dystocia risk. In beef cattle, maternal obesity has been linked to dystocia (Arnett et al., 1971). Loss of ewe body condition during pregnancy and low condition score at lambing is also a risk factor for dystocia. Behrendt et al. (2019) observed higher incidence of dystocia C (birth injury) and lower rectal temperature for lambs born to ewes managed to reach body condition score 2.4 or 2.8 at lambing compared to lambs born to ewes managed to reach body condition score 3.2 or 3.6.

It is likely that ewes with low or high body condition score at lambing will be at a higher risk of dystocia and that an appropriate supply of energy, protein, minerals and vitamins are required to reach the desired condition. The optimum condition score for reduced dystocia may vary between single- and multiple-bearing ewes. Further studies that link the risk of dystocia with ewe weight and condition profile during pregnancy and/or meta-analysis of existing data are required before firm conclusions and recommendations can be made (Horton et al., 2018).

### 3.3. Time of lambing

Time of lambing has implications for ewe nutrition and therefore ewe weight, condition score and lamb birthweight. Pregnant ewes predominantly graze in seasonal environments so feed supply and pattern of availability vary for different mating and lambing times. There are few studies comparing the risk of dystocia across different times of lambing, however, there is some evidence that variation in the availability of energy, protein, minerals and vitamins influence the incidence and category of dystocia (Osuagwuh et al., 1980).

### 3.4. Ewe parity and age

The rate of dystocia and lamb birth injury have historically been

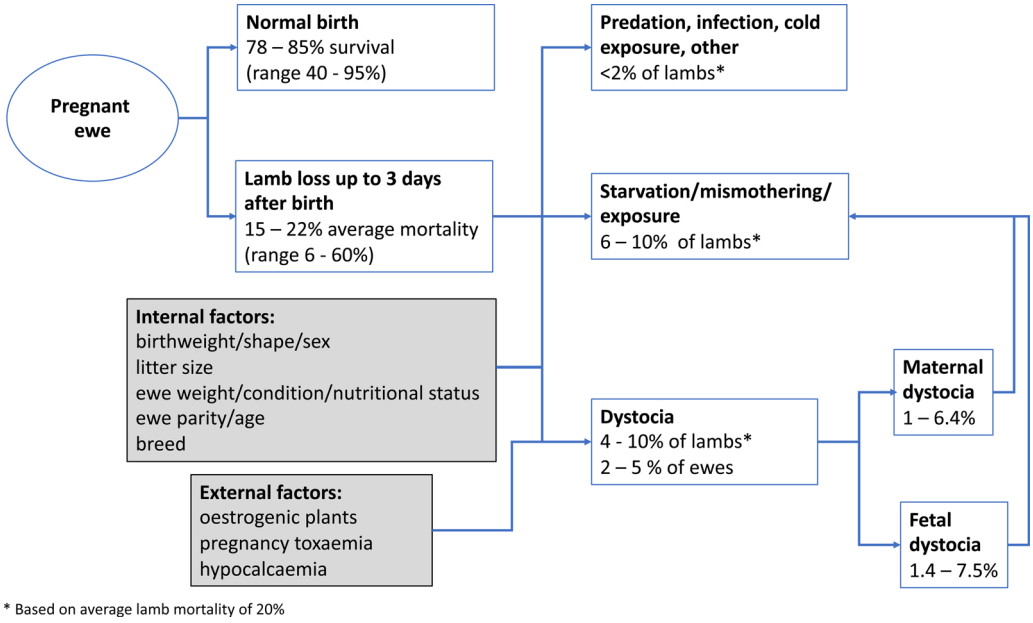


**Fig. 1.** Schematic summary of lamb survival and dystocia in Australian sheep flocks.

reported to be highest in first parity ewes, with little or no difference between ewes of greater parity or age (Woolliams et al., 1983; Speijers et al., 2010; McHugh et al., 2016; Refshauge et al., 2016). Li and Brown (2016) reported that lambing difficulty decreased as dam (ewe) age increased, but only up to 4.5 years. After this age, lambing difficulty increased. Horton et al. (2018) reported that the incidence of dystocia increased with ewe age, however, older ewes with dystocia were predominantly triplet-bearing with low birth-weight lambs indicating that this increased risk was specific to low birthweight dystocia. The distribution of high birthweight dystocia was more even across ewe age groups (Horton et al., 2018).

Younger ewes have been reported to have a longer parturition and require more birthing assistance than mature ewes (Everett-Hincks et al., 2007; Matheson et al., 2012). Parity and age are often confounded (e.g. ewe age increase as parity increases), and the association between younger age and dystocia is often attributed to the smaller, younger ewes having a disproportion between the size of the ewe and lamb (McMillan, 1983). This is supported by the positive relationship between ewe liveweight and pelvic dimensions, with increasing pelvic conjugate diameter associated with a lower incidence of dystocia and greater lamb survival (Knight et al., 1988; Kilgour and Haughey, 1993; Cloete, 1994). Size is not the full explanation, others have reported that birthing difficulty rates did not differ between first parity ewes lambing at approximately one or two years of age (McHugh et al., 2016). Redmer et al. (2004) observed that over-nourishing pregnant adolescent ewes results in rapid maternal growth at the expense of the nutrient requirements of the gravid uterus. Subsequent nutrient deprivation to the feto-placental unit and uterus could impact on the normal mechanical and endocrine parturition processes (Wallace et al., 2005).

### 3.5. Breed

The influence of breed on the risk of dystocia has been inconsistent between studies. Differences in the number and types of breeds compared is likely to explain a significant proportion of this variation. Dam breed has been reported to influence the rate of dystocia (Knight et al., 1988; Kerslake et al., 2005; Dalton et al., 2012; Dwyer and Bünger, 2012; McHugh et al., 2016), possibly due to differences in pelvic size (Knight et al., 1988; Cloete et al., 1998; Dalton et al., 2012). This is more likely to occur in breeds selected for terminal sire traits including muscling (Dwyer and Bünger, 2012; McHugh et al., 2016). However, ewe breed has not affected the rate of dystocia in all studies (Woolliams et al., 1983; Geenty et al., 2014; Refshauge et al., 2016).

Speijers et al. (2010) reported that sire breed influenced the risk of dystocia risk. This was driven by variation in lamb birthweight and malpresentation of lambs between sire breeds. Others, (Knight et al., 1988; Geenty et al., 2014; Refshauge et al., 2016) have reported inconsistent or no effects of sire breed.

Combined, these results indicate that there is scope for producers to manipulate the risk of dystocia through selection of dam and/or sire breed. However, further work is required to compare the risks across different genotypes and environments before firm recommendations can be made.

### 3.6. Sex of lamb

Dystocia rates and need for birthing assistance have been reported to be higher in male lambs (Everett-Hincks and Dodds, 2008; Matheson et al., 2012; McHugh et al., 2016). This difference may be at least partly due to heavier birthweight of males although, McHugh et al. (2016) reported that the difference still existed after adjustment for birthweight.

### 3.7. Summary

The physiological and environmental factors contributing to dystocia and the relative contributions to lamb mortality are summarised in Fig. 1. This figure is based on an average lamb mortality of 20 % and is at the lower end of the range reported by Hinch and Brien (2014). Estimates of lamb deaths and causes are therefore likely to be conservative.

## 4. Genetic options for dystocia management

The pursuit for highly productive breeding stock has its compromises. Domestication and intensive selection has led to longer parturition and less favourable maternal behaviour in ewes (Dwyer and Lawrence, 2005). Selection for accelerated growth rate, increased muscling, optimal fat coverage, the use of larger sires, and the retention of ewe and ram lambs from dystocic births (e.g. births requiring assistance) in the breeding flock may be contributing to more difficult parturitions and reduced lamb vigour (Speijers et al., 2010; Dwyer and

Bünger, 2012). The positive genetic correlation between yearling greasy fleece weight with Dystocia A, B and C and yearling weight with Dystocia A and C (Brown et al., 2014) provides further evidence of unfavourable genetic correlations with some production traits.

There is limited potential for a rapid reduction in dystocia through direct genetic selection. Heritabilities for Dystocia B and C and for lamb survival range from 0.02 to 0.04, with a moderate level of genetic correlation between dystocia types (Brien et al., 2010; Brown et al., 2014). This is consistent with observations of Everett-Hincks et al. (2014) who also reported low heritabilities for dystocia (0.01 to 0.07) in a large study in New Zealand. Matheson et al. (2012) determined the heritability of birthing assistance to be 0.26 and reported genetic correlations with lamb vigour and the need of suckling assistance of 0.68 and 0.54 respectively.

Dystocia indicator traits include lambing ease (Lamb Ease score), birthweight, lamb thorax circumference, lamb crown rump length and yearling fleece weight. Lamb Ease score is a semi-quantitative score derived from the level of lambing difficulty. Of these, lambing ease has the highest genetic correlation with dystocia of up to 0.45 followed by lamb thorax circumference of up to 0.44 (Brown et al., 2014). These correlations indicate that it may be possible to select against dystocia using indicator traits including lambing ease and lamb size, weight and conformation at birth, combined with other lamb survival and ewe rearing ability traits. Everett-Hincks et al. (2014) reported a moderate level of heritability for the indicator trait of lamb birth-weight (0.32 to 0.43) while Brien et al. (2010) reported a low heritability of 0.09 for lamb ease. Li and Brown (2016) reported a low to moderate genetic correlation between lamb ease and birth weight (0.31) and between lamb ease and gestation length (0.24).

Horton et al. (2018) found that prediction of future dystocia from previous dystocia was not reliable observing that the repeatability of both low and high birthweight dystocia was not consistent. However, the authors suggested that a small proportion of ewes could be identified as high risk and managed differently from the rest of the flock.

In cattle, positive correlations are found between heifer body height, hip height and shoulder height with calf birth weight (Basarab et al., 1993) indicating that larger heifers with a larger pelvic inlet give birth to correspondingly larger calves (Zaborski et al., 2009). In studying 14 cattle breeds, Laster (1974) found heavier cows have calves with greater weight deviation from the mean and were tending to have larger pelvic area. Selection to increase pelvic size relative to calf birth weight or pelvic size relative to cow body weight should lead to favourable reductions in dystocia (Taylor et al., 1975). Morrison et al. (1986) estimated a genetic correlation between pelvic area and cows weight at 0.57, similar to those by Upton and Bunter (1995) with liveweight (0.5 − 0.6) and with hip height (0.53 − 0.71), while Meyer et al. (2010) reported correlations to hip height at 0.21 − 0.44. Calf birth weight is however, strongly correlated with pelvic area at 0.73 − 0.75 (Benyshek and Little, 1982; Cook et al., 1993), suggesting that selection for reduced birth weight will see concomitant reductions in progeny pelvic area.

## 5. Endocrine, nutritional and metabolic relationships with birth and dystocia

The length of gestation is tightly regulated and is determined by fetal genotype. Prolonged gestation could increase the risk of dystocia due to fetal overgrowth, however, changes related to lamb birth type, size and sex and the age and nutrition of the ewe are small (Forbes, 1967) with no evidence of increased dystocia. The dominant hormone during gestation in sheep is progesterone secreted by the placenta. It is often called the "hormone of pregnancy" as it has many roles relating to the development of the fetus (Liggins, 1982). One of these roles is to block uterine myometrial activity to prevent early expulsion of the fetus (Heap and Flint, 1984). Hence, the needed increase in contractility of the myometrium at parturition to expel the fetus necessitates the removal of this hormonal inhibition. The way that progesterone dominance is removed, at least in the sheep, is an example of how placental endocrine function is influenced by the near-term fetus.

### 5.1. Parturition – the neurohormonal cascade

The chain of events leading to parturition begin with connective tissue changes in the cervix which precede uterine contractions that, in turn, lead to cervical dilatation to allow expulsion of the fetus. The fetus is crucial in this process as it initiates a neurohormonal cascade, via activation of the fetal hypothalamic-pituitary-adrenal (HPA) axis, which sets in motion the physiological, endocrine and biochemical changes needed (Fowden et al., 1998). Appropriate and timely cervical remodelling and uterine contractions are key for successful birth. It has been suggested that hormonal imbalances related to activation of the fetal HPA axis may result in dystocia due to incomplete dilation of the cervix or uterine inertia (Braun, 2007) however, for sheep, there is no published evidence to support this.

### 5.2. Role of the fetus

A general characteristic of fetal endocrine maturation across different species is the enhanced activity of the fetal HPA axis during late gestation (Challis et al., 2001). Activation of this axis has been linked to a changing intra-uterine environment, such as hypoxemia (Braems et al., 1996) acting as a stressor to the fetus. HPA development is associated with increased levels of adrenocorticotropic hormone (ACTH) and adrenal corticosteroids (cortisol in sheep) in the fetal circulation (Challis and Olsen, 1988), and increased corticotrophin releasing hormone (CRH) synthesis in the fetal hypothalamus (Challis, 1995). At term, increased levels of cortisol act on the placenta to increase expression of prostaglandin (PG) synthase (Challis, 2013). Increased synthesis of PG in turn increases the activity of enzymes in the fetal membranes that result in increased local generation of cortisol from cortisone, with further paracrine/autocrine stimulation of PG output (Challis, 2013). Increased fetal cortisol contributes to the maturation of organ systems required for post-natal survival of the lamb in the extra-uterine environment (Challis and Olsen, 1988), and starts the cascade of events leading to parturition. Thus, the level of fetal HPA activity is crucial not only for determining gestation length, but also in preparing the fetus for extra-uterine life.

### 5.3. Role of the placenta

Cortisol from the fetal adrenal gland provides the signal for the subsequent maternal endocrine changes. In sheep this is primarily driven by changes in the hormonal output of the placenta (Flint et al., 1975). During gestation, the myometrium is rendered quiescent under placental progesterone dominance via refractoriness to stimulation by PG-F2α and oxytocin. Removal of progesterone dominance is essential to start the cascade of events leading to birth.

The increase in fetal cortisol prior to parturition causes an increase in the activity of the enzyme CYP17A1 (formally known as P450 C17) in the placenta. As a result, C21 steroids reaching the placenta can be metabolised to C19 steroids and then another placenta enzyme, P450 aromatase, converts the C19 steroids to oestrogen. The prepartum increase in oestrogen results in increased output of PG-F2α from the placenta which in turn acts on uterine tissues (Challis et al., 2001). More recently it has been proposed that fetal cortisol may also act directly on placental tissue to stimulate PG secretion (Challis, 2013), particularly PG-E2. It has been shown that PG-E2 has a primary role in the regulation of cervical softening and dilation at term (Ledgert et al., 1983; Owiny and Fitzpatrick, 1990). Braun (2007) suggested that hormonal imbalances could result in dystocia due to the cervix not being completely dilated. This has not been investigated but could occur via alteration of placental PG-E2 actions.

### 5.4. Role of the ewe

Two changes must take place in a ewe's reproductive tract for parturition to occur. First, the uterus must be converted from a quiescent structure to a contracting organ. This requires the formation of gap junctions between myometrial cells to allow for transmission of the contractile signal. The second change is that the cervical connective tissue and smooth muscle must be capable of dilatation to allow the passage of the fetus from the uterus. Myometrial activity becomes synchronised and uterine contractions increase in both frequency and amplitude just before the time of parturition (Liggins, 1982). With the fetus being pushed onto and through the cervix there is a neurohormonal reflex release of oxytocin from the maternal pituitary gland, which further increases the contractions and leads to expulsion (Heap and Flint, 1984). These changes are accompanied by a shift from progesterone to oestrogen dominance (Heap et al., 1977), increased responsiveness to oxytocin by means of up regulation of myometrial oxytocin receptors, increased PG synthesis by the uterus (Jenkin, 1992), increased myometrial gap junction formation (Garfield et al., 1979), decreased nitric oxide activity and increased influx of calcium into myocytes leading to increased myometrial activity (Crankshaw et al., 1979; Massmann et al., 1999).

### 5.5. Endocrine changes associated with dystocia

Most commonly dystocia associated with hormonal imbalances is linked to endocrine disrupting compounds (EDC). The main EDCs are xenoestrogens and phytoestrogens. Xenoestrogens are man-made synthetic products whereas phytoestrogens are derived from plants.

#### 5.5.1. Oestrogenic pastures

Many forage legumes contain oestrogenic compounds that cause infertility in grazing animals. The richest sources of phytoestrogens in sheep diets are green clover although other legumes such as medics (including Lucerne), particularly when dry and rank and under fungal attack are also a risk (K Foster pers comm). Phytoestrogens are structurally like mammalian oestrogen, 17β-oestradiol, and thus are potent binders with mammalian oestrogen receptors. The effects of phytoestrogen metabolites are variable as they can act as oestrogen agonists or antagonists (Usui et al., 2002). The oestrogenic activity of phytoestrogens depends on the chemical structure of the compound, bioavailability, responsive tissue and its oestrogen receptor sub-type (ERα or ERβ), and metabolites resulting from plant fermentation and digestion (Shutt and Cox, 1972; Lundh, 1995). Phytoestrogens can alter reproduction of females on different levels because endogenous oestrogen receptors are widely distributed in the entire reproductive axis (hypothalamus, pituitary, ovary and reproductive tract). Phytoestrogens generally inhibit endogenous oestrogen production, leading to disturbances in the follicular development and lack of the occurrence of oestrus (Rosselli et al., 2000). Phytoestrogens may also disturb oestrus and ovulation through their effects on the CNS (Wocławek-Potocka et al., 2013).

It has long been known that high levels of oestrogens found in older varieties of subterranean clover (*Trifolium subterraneum* L) can cause 'clover disease' with associated ewe infertility, prolapsed uteri, dystocia and increased lamb mortality (Bennetts et al., 1946). Ewes grazing oestrogenic varieties of subterranean clover have reportedly had rates of dystocia of 40 %, with 20 % ewe mortality and 60 % mortality of lambs (Moule, 1961). Davies et al. (1970) also reported higher mortality of lambs born to ewes grazing highly oestrogenic varieties (30–47 %) compared with other varieties with a lower oestrogen concentration (16–30%).

Clover disease remains one of the most significant environmental factors in poor ewe reproductive performance in southern Australia. Adams (1995) estimated that approximately four million ewes were affected in south Western Australia, and in 2002 it was reported that between 10–15 million sheep in the Australian flock were affected to varying degrees by oestrogenic clovers (Walker et al., 2002). This number is still applicable to the current flock, with a recent survey in Western Australia, South Australia, New South Wales and Victoria indicating 25 % of pastures tested contained potentially potent concentrations of phytoestrogens (Foster et al., 2019) with the figure being over 65 % in some districts. This high percentage is not surprising, as a recent survey indicated 70 % of producers have not re-sown a paddock to one of the newer subterranean clover varieties in the last 25 years (Foster et al., 2019).

The pathogenesis of clover-induced dystocia is not well understood. Bennetts et al. (1946) suggested that dystocia resulted from primary uterine inertia. However, Maxwell (1970) and Adams and Nairn (1983) suspected that some outbreaks of dystocia resulted from failure of the cervix or vulva to dilate. Experimental treatment of ewes with diethylstilbestrol, a synthetic oestrogen, can produce a syndrome in which the expulsive effort of uterine musculature is normal but the cervix or vulva fails to dilate (Clark, 1965; Hindson et al., 1968), in a fashion similar to the dystocia ascribed to phytoestrogens. However, the effect of phytoestrogens may not be direct because treatment of ewes with oestrogen in late gestation does not result in dystocia (Clark, 1965). It is possible that the condition arises from a refractoriness of the genital tract to oestrogenic stimulation. If, for example, the ability of oestrogen to induce the synthesis of PG were impaired, both uterine motility and dilation of the cervix could be impeded (Anderson et al., 1981). Woclawek-Potocka et al. (2005) found that soy-bean (*Glycine max* L) derived phytoestrogens altered the production of luteolytic PGF2a in cattle during the oestrous cycle and early pregnancy. The effect of phytoestrogens on uterine and cervical function during parturition have not been investigated.

#### 5.5.2. Endocrine disrupting compounds

Endocrine disrupting compounds (EDCs) "interfere with the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body that are responsible for the maintenance of homeostasis, reproduction, development, and/or behaviour" (Crisp et al., 1998). Animal exposure to EDCs can occur via ingestion of food, dust and water, inhalation of gases and particles in the air and through the skin. EDCs can also be transferred from pregnant sheep to the developing fetus or to the lamb through the milk. The most abundant EDCs in the environment are the xenoestrogens. Xenoestrogens are exogenous oestrogen-mimicking compounds that are related, either structurally or functionally, to 17β-oestradiol (E2). This can allow them to bind to oestrogen receptors (ERs), though the degrees of affinity and selectivity of this binding do vary between xenoestrogens (Paterni et al., 2017).

Although there is no direct evidence for a link between EDCs and dystocia, several studies have indicated that EDCs can disrupt uterine structure and/or function in animal models (Gore et al., 2015; Feng et al., 2016). Moreover, EDCs like dioxins, polychlorinated biphenyls (PCB) and organochlorine pesticides, have been or are used extensively in agriculture. Defects in fetal development were noted when pregnant ewes were exposed to a mix of EDCs contained in human sewage sludge, following its application to pasture as a fertiliser (Paul et al., 2005; Fowler et al., 2008).

#### 5.5.3. Environmental and management stress and interruption during parturition

Stress causes an arousal of the sympathetic nervous system, leading to a release of adrenaline, other catecholamines and glucocorticoids such as cortisol (Selye, 1956; Naaktgeboren and Slijper, 1970; Naaktgeboren and Bontekoe, 1976). in vitro studies indicate that adrenaline is able to either excite or to inhibit uterine activity (Rüsse, 1963; Naaktgeboren and Slijper, 1970; Naaktgeboren and Bontekoe, 1976), however, little is known about the impact of environmental stress on the incidence of dystocia in sheep. One study investigating the

underlying endocrine mechanism reported that both stress (psychological) and exogenous adrenaline inhibited uterine motility in sheep, but only when plasma levels of oestrogen were high (Bontekoe et al., 1977). It is known in many species that adrenaline inhibits oxytocin release during the milk-ejection reflex during lactation (Whittlestone, 1954; Barowicz, 1979; Bruckmaier et al., 1997). However, the effect of stress (adrenaline) on oxytocin release during parturition and the role this plays in dystocia in sheep is less clear. The responsiveness of the HPA axis to stressors has been shown to be progressively attenuated during late pregnancy (Neumann et al., 1998; De Weerth and Buitelaar, 2005), and remains suppressed during parturition in other species (Gilbert et al., 1997; Douglas et al., 2003).

There is an established link between cold stress and lamb survival (McCutcheon et al., 1981). Challenges that threaten energy homeostasis, such as cold exposure, will normally lead to activation of the HPA axis resulting in the release of adrenal hormones which facilitate the mobilisation of energy substrates and supports energy homeostasis. These endocrine changes could interfere with parturition. Verbeek et al. (2012) reported that thin ewes with restricted feed intake had reduced stress responses to an acute cold challenge, suggesting that the ewes had an impaired ability to temporarily adjust their physiology to the cold challenge. The possibility of heat stress impacting on fetal development and parturition should also be considered. Sustained heat stress in sheep during late gestation appears to be associated with intra-uterine growth restriction (Alexander and Williams, 1971; Brown et al., 1977), which may lead to fetal dystocia due to developmental abnormalities. Heat stress does not appear to alter the duration of parturition for ewes (Stephenson et al., 1984), though research in this area is limited.

Much more is known about the impacts of environmental and management stress on dystocia in dairy cattle as reviewed by Mee (2008). Incomplete dilatation of the cervix and vulva is more common when there is environmental stress or premature assistance during the periparturient period. Also, the increased risk of vulval stenosis and dystocia in heifers calving in stalls compared to pens or paddocks has been attributed to parturient stress and adrenalin and cortisol release. Moving heifers or cows during the early stages of calving is associated with increased risk of dystocia. Assistance at calving before the cervix and vulva are fully dilated can result in iatrogenic dystocia due to cervico-vulval stenosis. Providing assistance less than one hour after the fetal hooves appear increases the risk of dystocia and reduced perinatal vigour, while delaying assistance for more than two hours prolongs calving and induces hyperlactataemia. Environmental disturbances at calving caused by the continuous presence of an observer, confinement, or overcrowded calving accommodation can lead to reduced uterine motility, cervical dilatation and abdominal contractions with resultant prolonged calving and dystocia. The effects of many of these environmental and management stressors on dystocia in sheep are either unknown or anecdotal.

### 5.6. Energy and protein

Energy and protein intake may influence the size of the lamb, length of gestation, condition of the pregnant ewe and plays a direct role in the process of parturition. The effects of nutrient manipulation during pregnancy on fetal development and gestation length in sheep are subtle and inconsistent (Morris, 1973; Holst et al., 1986; Redmer et al., 2004), and may depend on the level and length of manipulation as well as the stage of pregnancy. Under-feeding sheep during late gestation is associated with intra-uterine growth restriction (Oliver et al., 2001). This may impact on dystocia as both oversized and undersized lambs are more susceptible. The hypoglycaemia associated with underfeeding the pregnant ewe shortens gestation length (West, 1996), though reversing this by experimentally inducing a state of hyperglycaemia does not always extend gestation length (Stevens et al., 1990). There is some evidence that over-feeding slightly increases gestation length. Longer gestation may be associated with dystocia through increased birthweight (Morris, 1973; Holst et al., 1986; Redmer et al., 2004).

Energy is also associated with the parturition process as it is required for muscle contraction. The specific energy expended during labour and parturition is not high relative to daily energy requirements (0.8–1.2 MJ) (Brockway et al., 1963) but, in other species, glycogen in the myometrium increases dramatically just prior to parturition and decreases significantly during birth. This indicates that glycogen may serve as an important energy source for uterine contraction (Chew and Rinard, 1979), with exhaustion of glycogen contributing to secondary uterine inertia. Exogenous oestrogen also increases glycogen in the uterus (Bitman et al., 1967), so the increase in glycogen close to parturition is aligned with the change in the ewe from progesterone to oestrogen dominance. In peri-parturient dairy cows a relationship between serum calcium (Ca) and energy metabolites indicates that Ca may also influence energy mobilisation and utilisation around parturition (Lean et al., 2014).

There is some evidence of interactions between nutrition and endocrinology that may influence the process of parturition. McMillen et al. (1995) suggested hypoglycaemia in the fetus may result in increased production of ACTH and cortisol and thereby play a role in the initiation of the HPA cascade. Others have postulated that progesterone stored in adipose tissue of overfat ewes may cause a hormonal imbalance at parturition leading to a dysfunctional birth process. This has not been tested experimentally (Holst et al., 2002).

### 5.7. Minerals and vitamins

#### 5.7.1. Macrominerals, vitamins and uterine activity

Acute hypocalcaemia has been observed in ruminants for 200 years and, if untreated, may result in mortality of the pregnant ewe and fetus. Transient acute or subclinical hypocalcaemia is less well understood but has implications for the birth process and maternal dystocia. In the uterus, myometrial contractions are triggered by a rise in intracellular Ca caused by an influx of Ca from the extracellular space and mobilisation of intracellular Ca (Tribe, 2001). Low Ca allows sodium (Na) to enter the nerve cells to cause spontaneous contractions and fasciculations (Friend et al., 2020). Secondary or subclinical hypocalcaemia increases dystocia, retained placenta and uterine prolapse in dairy cattle (Curtis et al., 1983; Risco et al., 1984). Less is known of secondary effects in the ewe. Older ewes are more susceptible to hypocalcaemia and the incidence of hypocalcaemia increases from about 6 weeks before lambing. Hypocalcaemia in cattle is usually observed later in pregnancy and early lactation and may therefore coincide with parturition. Although hypocalcaemia is less likely to coincide with parturition in the ewe, induction of hypocalcaemia by infusion of the disodium salt of ethylene-diamine tetra acetic acid causes a reduction in uterine activity (Silva and Noakes, 1984), indicating a likely relationship between Ca and dystocia in sheep. In field studies, Caple et al. (1988) reported subclinical hypocalcaemia in ewes was associated with lower lamb survival.

Hypocalcaemia is primarily due to a failure of the endocrine system to respond to increased demand for Ca. There is uncertainty around the role of mineral and vitamin intake in mitigating the susceptibility in ewes (Friend et al., 2020). Modern pastures grazed around parturition often provide an imbalanced supply of Ca, magnesium (Mg), potassium (K), phosphorus (P), Na, vitamin D and dietary cation-anion difference (DCAD) (Masters, 2018; Masters et al., 2019). High K and low Na in pastures depress absorption of Mg and may induce hypomagnesaemia, while low Mg and vitamin D and high P and DCAD will decrease Ca absorption or mobilisation. Hypomagnesaemia predisposes the grazing ruminant to hypocalcaemia (Herd, 1965). Further studies are required to determine if mineral imbalance induces subclinical hypocalcaemia during late pregnancy and whether this subclinical hypocalcaemia causes uterine inertia and maternal dystocia in grazing ewes.

#### 5.7.2. Oxidative stress and maternal dystocia

Dystocia is associated with acute inflammation of the uterus and cervix that leads to oxidative stress. Cytokine production and reactive oxygen species (ROS) in serum increase significantly at this time (Rizzo et al., 2008). Change from anaerobic to aerobic respiration also increases the production of ROS in the neonate. If untreated this may cause pathological conditions, disease or death (Mutinati et al., 2014). A direct relationship between oxidative stress and impaired uterine contractility has also been suggested in the ewe (Celi, 2010). Selenium (Se), manganese (Mn), zinc (Zn), copper (Cu), sulfur (S), vitamin E and vitamin A function synergistically in a series of antioxidant reactions that provide a defence against ROS (Masters, 2018). An imbalanced supply of these minerals and vitamins may slow recovery or compromise survival and long-term production. Moreover, magnesium sulfate has been shown to act as a neuroprotectant through reduction in the production of cytokines and free radicals following hypoxia-ischemia (Marret et al., 2007; Plush et al., 2016). Similarly caffeine may reduce the effects of hypoxia and has been associated with improved lamb survival (Robertson et al., 2017).

While there is insufficient evidence to indicate a lack of antioxidants is a primary contributor to the high rates of dystocia in ewes and subsequent production loss, there is evidence to support a strategy ensuring reproducing ewes have an adequate and balanced supply of antioxidants during the periparturient period, particularly when lambing on rapidly growing pastures in regions with a history of Se deficiency.

### 5.8. Pregnancy toxaemia

Pregnancy toxaemia is a metabolic disease that may occur during late pregnancy. It is most likely to develop in fat or lean ewes when high energy requirements are not being met or when feed intake is depressed (Pethick et al., 2005; Mavrogianni and Brozos, 2008). The resulting high blood ketone levels and low blood glucose causes ewe mortality and, even if the ewe survives, increases lamb mortality (Henze et al., 1998). Barbagianni et al. (2015) observed a higher incidence of dystocia in ewes with pregnancy toxaemia and suggested this may be caused by impaired hormonal mechanisms due to poor ewe nutrition or reduced gestation length. Ewes that recover may develop dystocia or have a slow birth process resulting in metritis and stillborn lambs (Andrews, 1997). Hypocalcaemia may also reduce abomasal and rumen motility and may precipitate pregnancy toxaemia in ewes (Friend et al., 2020). A combination of pregnancy toxaemia and hypocalcaemia would likely cause uterine inertia. Pregnancy toxaemia has been reported to have relatively low incidence of 0.5–2% of the flock (Andrews, 1997; Lane et al., 2015) and is therefore unlikely to be a major contributor to dystocia in ewes.

### 5.9. Toxins

A wide variety of toxins have been demonstrated to have teratogenic effects that impact embryo survival and can be associated with congenital defects and malformation of the fetus. Ingestion of several teratogenic shrubs, such as locoweeds (*Astragalas* spp.), lupine (*Lupinus* spp.), and pine (*Pinus* spp.), predispose sheep to prolonged gestation (Coppock and Dziwenka, 2017). Ergopeptine alkaloids from ergot or grazing of fescue pasture infected with endophytes during late gestation may be associated with prolonged gestation, dystocia, agalactia and foal dysmaturity in horses (Porter and Thompson, 1992; Evans et al., 2004; Coppock and Jacobsen, 2009). Sheep are less susceptible to endophyte toxicosis than cattle and dystocia has not been reported.

The pesticide carbaryl has been shown to have teratogenic effects via stillbirths and malformations in pigs and dogs, and dystocia associated with uterine atony has been observed in bitches exposed during pregnancy (Smalley et al., 1968). Carbaryl exposure is unlikely to cause dystocia in sheep except in circumstances where ewes are grazing treated pasture during late gestation.

Scorpion venom exposure in late pregnancy can cause abortion or dynamic dystocia (Ben Nasr et al., 2007a, b).

## 6. Conclusions

Causes of dystocia are multifactorial with a range of nutritional and non-nutritional stressors contributing to occurence and outcome. Nutrition related factors include lamb birthweight, ewe liveweight, condition and fatness, glycogen in uterine muscle and mineral status. The risk of dystocia is increased in both high or low birthweight lambs, high (fat) or low liveweight ewes, and small first parity ewes. Other factors implicated include low muscle glycogen, pregnancy toxaemia, mineral imbalance causing hypocalcaemia and a lack of antioxidant nutrients. Addressing these risks requires differential nutritional management for single and multiple bearing ewes although there are no research tested optimum nutritional profiles for either class of animal.

There is evidence for stress and environmentally related dystocia. The stress-related hormones cortisol, adrenaline and ACTH all play a major role in the initiation and control of parturition in the sheep, they are under both endogenous control and exogenous influence. Extrapolating from our knowledge on the role of these hormones in normal birth suggests adequate (but not excessive) supervision during lambing, provision of adequate feed and shelter at the lambing site, and small flock size may reduce exposure to physical and environmental stress. The requirement that the ewe shift from progesterone to oestrogen dominance during the onset of parturition is also subject to exogenous interference through the consumption of plants containing phytoestrogens or xenoestrogens. Phytoestrogens in clovers and medics, in particular, cause ewe infertility, prolapsed uteri, dystocia and increased lamb mortality. These plants are still widely grown in mixed pastures but should be not be grazed by pregnant ewes.

There is clearly a genetic component to dystocia. This is at least partly explained by incompatibility in physical size and dimensions of the ram, ewe and lamb. Use of a large ram breed over a smaller ewe breed increases the likelihood of dystocia. Domestication and intensive selection has led to longer parturition, increased muscling and less favourable maternal behaviours. While there is a genetic component to dystocia, rapid reduction through direct genetic selection is problematic with low heritability of dystocia and some of its indicator traits such as lambing ease. The expected impact of culling or modifying management for individual ewes following dystocic birth is uncertain, with studies indicating low repeatability of dystocia.

This review provides broad interpretation of the available literature, but conclusions are not definitive. Widespread inconsistency in the literature indicates further research to investigate dystocia under commercial production conditions is required, and this should be complemented by focussed studies under controlled conditions. Priorities include defining the fitness of the ewe to lamb, the role of stress and environment on parturition and the use of indicator traits to select for ease of birth.

## Declaration of Competing Interest

The authors have no conflict of interest.

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