
THE PROCEEDINGS OF THE
NEW ZEALAND SOCIETY
OF
ANIMAL PRODUCTION
VOLUME 62 2002

NUTRITION AND EMBRYO SURVIVAL IN THE EWE

T. N. EDEY
University of New England, Armidale, Australia

SUMMARY

Results published in the past decade permit reasonably accurate definition of the effects on embryo survival of various nutritional regimes. Severe undernutrition lasting 1 to 3 weeks during the first month of pregnancy often causes measurable loss of embryos. The induced loss rarely exceeds 15%, and is usually higher in twin than in single ovulators. In some experiments increased duration of undernutrition, and in others low ewe body weights have been associated with higher embryonic loss. Age of ewe appears relatively unimportant. The few studies extending into the second 21 days of pregnancy indicate that by this time embryos are more resistant to undernutrition. High planes of nutrition (*e.g.*, 200% maintenance) in the pre-implantation period can also cause increased embryonic loss. Little progress has been made in defining the physiological basis of nutritional effects on embryo survival. Progesterone insufficiency has been excluded as a likely cause, and the necessary biochemical studies of the endometrium have been slow to develop in any species, but are now gaining momentum.

INTRODUCTION

IN AN ERA when profitability of sheep enterprises depends so greatly on high reproductive rates, techniques have been evolved to allow the partitioning of reproductive wastage into components so that management strategies for each phase of reproduction can be evaluated critically. One such component of the whole complex is embryo mortality, which is strictly the loss of cleaving ova and of embryos which occurs between fertilization and the end of organogenesis — about day 40 in the sheep. However, the term embryo mortality is often used loosely to cover losses through the whole of pregnancy, and while this does not usually involve great inaccuracy, this paper will be confined to the embryonic period during which most prenatal losses occur.

Recognition of the importance of reproductive losses during pregnancy goes back over half a century (review by Edey, 1969) but it is only in the last two decades that reasonably accurate estimates for the sheep, of the extent, timing and cause of loss have been made. Obviously the level of loss will vary with genotype and environmental conditions, but a high proportion of the

TABLE 1: SELECTED ESTIMATES OF BASAL PRENATAL MORTALITY IN THE EWE.

Author(s)	*No. of Ewes*		
Dutt (1954)	180	Mixed	32.7
Quinlivan *et al.* (1966)	676	Romney	22.1
Averill (1955)	131	Suffolk	22.5
Mattner and Braden (1967)	100	Merino	20.8
Mattner and Braden (1967)	200	Merino	6.1

reliable estimates place the normal or basal prenatal loss, occurring in the absence of identifiable stress, at 20 to 30%. However, several lower estimates illustrate the variation which can occur (see Table 1).

Some embryonic mortality, and in the view of some authors (*e.g.*, Bishop, 1964) most of it, is inevitable and even desirable in disposing of unfit genetic material. Generally over half the loss occurs before day 13, allowing the ewe to return to service within a normal cycle length (Edey, 1967) and thus without external evidence that a death has occurred. Most of the remainder occurs by day 18 and the ewes will usually return to service after some extension of the cycle length, but generally in time to be remated before the rams are removed. In addition to the basal embryonic loss, further loss can be caused by any condition imposed upon, or existing within the dam, which can give rise to an abnormal utero-tubal environment. The operation of nutritional intake in this context will now be examined.

ENERGY

Any nutritional treatment may have not only a short-term effect reflected in blood nutrient levels, but also long-term effects represented by a change in body weight. Early studies of nutrition and reproduction showed a lack of awareness of this distinction. The classic example was in the work on nutrition and ovulation, but it has extended also into the work on nutrition and embryo mortality.

The first series of experiments which examined the response to different energy intakes in early pregnancy was conducted by a Wisconsin group, and has often been misinterpreted by reviewers. In that series, the emphasis was on the effects of long-term pre- and post-mating supplementation programmes using hay and grain-based rations. Undernutrition was not involved, the planes of nutrition being best described as "moderate" and "high", and much of the interest in successive experiments centred on a possible detrimental effect of the high grain-based ration. Differential feeding commenced several months before mating, large differences in body weight were generated (*e.g.*, 17 kg in El Sheikh *et al.*, 1955) resulting in big differences in ovulation rate (20 to 62%; Casida, 1964), but small and equivocal differences in embryo mortality. In reviewing the work of El Sheikh *et al.* (1955), Foote *et al.* (1959) and Bellows *et al.* (1963), Casida (1964) concluded that in none of the six trials was the percentage survival of embryos greater in the high-plane grain group. However, when the data are recalculated for number of embryos surviving, the effect of increased ovulation rate was such that in four out of six experiments a greater number of embryos survived in the high-plane groups. Further reference will be made later to this apparent conflict between high ovulation rates and embryo survival.

A suggestion which sprang from the above work, and from results in the pig (review by Scofield, 1972), that high-plane feeding in early pregnancy can reduce embryo survival, has not been studied exhaustively. However, some support comes from a well-designed experiment by Cumming *et al.* (1975) which indicated that Merino but not Border Leicester × Merino crossbred ewes showed higher embryo mortality on a 200% maintenance compared with a 100% maintenance ration from day 2 to day 16 post-mating.

The report of a significant drop in lambing percentage following severe under-feeding during the first 90 days of pregnancy in 2-year-old Merino ewes was the first linking undernutrition and early prenatal mortality (Bennett *et al.*, 1964). The initial report by Edey (1966) of a 16% loss of embryos in mature Merino ewes subjected to severe 7-day nutritional stresses during the first 20 days of pregnancy, was followed by a series of papers on the embryonic period. In these studies, pre-ovulatory differences in nutrition were excluded and post-mating treatments ranging from 15% maintenance for 7 days to 50% maintenance for 30 days were imposed. It was concluded (Edey, 1970d) that in only one experiment out of four was embryo mortality clearly induced, though following a 50% maintenance treatment from days 7 to 37 in another experiment, the occurrence of late returns to oestrus suggested that some embryos had succumbed.

About 1970 a group at Werribee, Victoria, commenced a detailed study of nutrition and embryo mortality. They have made significant progress in clarifying the effects of time of application and duration of nutritional stress. Cumming (1972b), using mature Perendale ewes in a factorial giving combinations of 1, 2 or 3 weeks of undernutrition (0.2 kg hay/day) during the first 21 days of pregnancy concluded that week of application was not critical, but that increasing duration of underfeeding was detrimental to embryo survival. The main loss was in ewes which started with twins and progressively lost one and then two embryos with time. The suggestion of Van Niekerk *et al.* (1968) that a few days of fasting could kill embryos was investigated by Blockey *et al.* (1974) in 1189 mature Merino and Border Leicester × Merino cross ewes. It appeared that 3 days of fasting killed up to 10% of single embryos in the first 10 days after mating, but there was no effect in twin ovulators. The authors concluded that, because deaths occurred early, re-mating could occur and there would normally be little or no effect on lambing percentage.

The same group (Cumming *et al.*, 1975) studied the effects of 25%, 100% and 200% maintenance level rations over days 2 to 16 in 1000 Saxon Merino and Border Leicester × Merino cross ewes, and concluded that embryo survival was greatest on the 100% diet. There were some breed differences, however; twin survival on all diets was higher in the crossbreds than in the Merinos, and on the 200% diet the Merinos were much worse ($P < 0.01$) than the crossbreds.

Because of the known vulnerability of the ovum in its early cleavage stages (Edey, 1969) attempts have been made to magnify the effect of early post-mating stress by adding a period of undernutrition immediately before mating. Thus, McKenzie and Edey (1975a) examined the effect of combinations of severe underfeeding for 7 days pre-mating and 14 days post-mating. In mature Merino ewes, ovulation rate was unaffected by the premating treatment but ovum wastage was apparently increased (9 to 15%) in all underfed groups. A group receiving both treatments showed an increased number of long cycles, indicating the death of embryos after day 13. In the same paper, 1½-year-old maiden Merino ewes which received 30% of their maintenance requirement for 14 days post-mating showed a 14% higher embryo mortality than their controls, and an increase in the number of long cycles.

More prolonged but less severe undernutrition, lasting 8 weeks pre-mating, was found to have no effect on embryo survival in Merino ewes with low ovulation rates (McKenzie and Edey, 1975b). These ewes were gaining weight in early pregnancy, and Cumming (1972a) found that ewes which lost weight for a month pre-mating and then gained weight produced significantly more lambs than a group on the reverse treatments. Embryonic survival was poorer in the group which lost weight after mating mainly owing to additional losses from twin ovulators.

BODY WEIGHT EFFECTS

There is evidence that ewes at very low body weights have relatively poor embryo survival (Edey, 1970a, 1970d; Guerra *et al.*, 1971; Gunn *et al.*, 1972) and a relatively high rate of barrenness (Coop, 1962; Guerra *et al.*, 1972). At higher weights there is general agreement (Killeen, 1967; Edey, 1970d; Cumming *et al.*, 1975) that body weight *per se* has no important influence on the survival of embryos.

OVULATION RATE

The effect of ovulation rate on basal prenatal mortality was discussed by Edey (1969) who pointed out that even on a random-loss basis a higher percentage of eggs shed is likely to be lost from twin than single ovulators. This is because, when one embryo of twins dies, the pregnancy continues but the second embryo is still at risk. The question of whether or not basal embryonic death is random is unresolved. However, from the data of Edey (1966) and Cumming (1972a, b) it is clear that there are some circumstances in which induced mortality falls more heavily on twin embryos, to the point at which more twin ovulating ewes than single ovulators are barren. In some cases twin ovulators will lose only one embryo but, but when uterine conditions which are inimical to embryo survival are induced, all embryos will be lost. Thus unless data are interpreted carefully it is possible to attribute increased embryo mortality to higher body weight where the operative relationship is between body weight and ovulation rate.

DISCUSSION OF ENERGY EFFECTS

So far in this review there has been deliberate emphasis on the positive results. It should now be pointed out, however, that many workers (*e.g.*, Hodge, 1966; Coop and Clark, 1969; Bennett *et al.*, 1970; Edey, 1970b, c; Braden, 1971; Gunn *et al.*, 1972; Dobbie *et al.*, 1975) have reported little or no effect of undernutrition on embryo mortality. In explanation of the differing results between experiments and between workers the following points are important:

(1) When detected, levels of induced mortality have usually been less than 15%, so fairly sensitive experimental designs are required to decide if loss is occurring.
(2) With rare exceptions, positive results have been associated only with severe undernutrition for periods of 7 to 21 days during the first month of pregnancy.
(3) The same sheep can give different results between seasons in response to apparently similar treatments.
(4) Breed and strain of sheep can influence the response.
(5) There is a need to distinguish between basic experiments which aim to determine if mortality can be induced and more applied ones which investigate the possibility that it may occur under normal management systems.

PROTEIN

Hoxsey *et al.* (1960) and Van Der Westhuysen (1971) used high protein supplements in early pregnancy, but, as might be expected in a ruminant animal, there is no evidence from their papers of a beneficial effect on embryo survival.

There appear to be no studies of embryo survival under the low protein conditions which may exist in drought.

MINERALS

Selenium is the only mineral clearly implicated in embryo mortality. Hartley (1963) reported that in certain areas of New Zealand a reduced fertility condition characterized by a high incidence of barren ewes was eliminated by a single dose of selenium. Embryonic death apparently occurs between 3 and 4 weeks post-conception. Beneficial effects of selenium were also reported by Dobbie *et al.* (1975). Better lambing results, presumably due to decreased embryo mortality, were obtained following administration of vitamin E and selenium in selenium-deficient areas of Scotland (Mudd and Mackie, 1973).

Reports of improved fertility in ewes treated with selenium while grazing oestrogenic pastures in South Australia (Godwin *et al.*, 1970) are not necessarily related to the same syndrome.

SPECIFIC FEED HAZARDS

There are few confirmed cases where ingestion of a particular feedstuff has caused embryonic death. One such case was the feeding of kale reported by Williams *et al.* (1965); another was the implication of the weed *Veratrum californicum* as a cause of embryonic death and subsequent foetal abortions (Van Kampen *et al.,* 1969).

Long-term grazing of ewes on oestrogenic pastures can cause a severe drop in fertility, but it appears that impaired sperm transport and neonatal loss are the main factors involved (Moule *et al.,* 1963). Embryo mortality, if involved at all, is a minor factor.

MECHANISMS OF EMBRYONIC DEATH

To date little progress has been made in explaining the reasons for failure of survival of apparently normal embryos in the uterus. The basic biochemical studies required to define the nature of the uterine environment are proceeding steadily, but comparatively little is known as yet about endocrine control of the endometrium and its secretions. Some studies are starting to appear which relate hormonal changes to biochemical changes in the uterus and a few which indicate the hormonal changes which can occur in response to environmental stresses.

An obvious area of activity has been on the role of progesterone in embryo survival. Early studies tended to rule out progesterone deficiency as a likely cause of embryonic death (*e.g.,* Moore *et al.,* 1960) and, in confirmation, Cumming *et al.* (1971) have recently shown that undernutrition causes an increase in plasma progesterone levels.

AGE-NUTRITION INTERACTION

Edey (1969) reported that basal prenatal mortality rates appeared to be similar in 1½-year-old and mature ewes. From limited evidence (Mackenzie and Edey, 1975a) young ewes appear just as resistant as mature ewes to embryo mortality induced by short-term severe undernutrition. However, further experiments are required using young ewes with higher ovulation rates. Also, in view of the increase in the practice of mating ewe lambs, embryo mortality studies are required for this age-group, particularly in view of their lower progesterone status (Chu and Edey, unpublished data).

In the study of Bennett *et al.* (1964) involving undernutrition for the first 90 days of pregnancy, 2-year-old maiden ewes suffered higher prenatal loss than mature ewes.

EMBYRO MORTALITY AND NUTRITIONAL MANAGEMENT SYSTEMS

The desire to achieve high body weights before mating and adequate nutrition in late pregnancy-early lactation have left the early- to mid-pregnancy period as a time when restriction of intake is commonly practised. Coop and Clark (1969) evaluated this system and concluded that, where ewes were restricted for 5 to 8 weeks after the third week of pregnancy so as to lose about 5 kg, there was no decline in reproductive performance. There can be little quarrel with this conclusion provided the dangers are recognized. The main hazard is that producers may equate joining with the start of pregnancy, and, by commencing severe restriction too early, both limit ovulation rate and risk some embryonic loss.

It is clear, however, that the known benefits of additional premating feed in giving high body weight and increased ovulation rates of up to 50% in most breeds outweigh the risk that postmating restriction may cause embryo mortality which, at worst, should not exceed 20%. Moreover, re-mating will offset some of this loss, though it should not be overlooked that, when long cycles follow embryonic death, fertility is depressed at the next oestrus (Edey, 1972) and a few barren ewes may result.

Because of the inter-relationship between the various reproductive phases, unduly severe restriction for any lengthy period must reduce reproductive rate. Thus, systems which aim to maximize productivity per unit area face the problem that, as they increase the reproductive rate, the greater numbers of lambs increase the competition with the ewes, and if adjustments are not made some phase of reproduction will suffer. However, sufficient knowledge is now available on the effects of undernutrition on all phases of reproduction, including early pregnancy, to allow the making of sound decisions.

REFERENCES

Averill, R. L. W., 1955: *Proc. Soc. Study Fert.*, *7*: 139.

Bellows, R. A.; Pope, A. L.; Chapman, A. B.; Casida, L. E., 1963: *J. Anim. Sci.*, *22*: 101.

Bennett, D.; Axelsen, A.; Chapman, H. W., 1964: *Proc. Aust. Soc. Anim. Prod.*, *5*: 70.

Bennett, D.; Nadin, J. B.; Axelsen, A., 1970: *Proc. Aust. Soc. Anim. Prod.*, *8*: 362.

Bishop, M. W. H., 1964: *J. Reprod. Fert.*, *7*: 383.

Blockey, M. A. de B.; Cumming, I. A.; Baxter, R. W., 1974: *Proc. Aust. Soc. Anim. Prod.*, *10*: 265.

Braden, A. W. H., 1971: *Aust. J. exp. Agric. Anim. Husb.*, *11*: 375.

Casida, L. E., 1964: *Proc. 6th int. Congr. Nutr., Edinb., 1963*: 366.

Coop, I. E., 1962: *N.Z. Jl agric. Res.*, *5*: 249.

Coop, I. E.; Clark, V. R., 1969: *J. agric. Sci., Camb.*, *73*: 387.

Cumming, I. A., 1972a: *Proc. Aust. Soc. Anim. Prod., 9*: 192.
——— 1972b: *Proc. Aust. Soc. Anim. Prod., 9*: 199.
Cumming, I. A.; Blockey, M. A. de B.; Winfield, C. G.; Parr, R. A.; Williams, A. H., 1975: *J. agric. Sci., Camb., 84*: 559.
Cumming, I. A.; Mole, B. J.; Obst, J.; Blockey, M. A. de B.; Winfield, G. C.; Goding, J. R., 1971: *J. Reprod. Fert., 24*: 146.
Dobbie, J. L.; Welch, R. A. S.; Ackerley, J. G.; Lake, D. E., 1975: *Proc. N.Z. Soc. Anim. Prod., 35*: 91.
Dutt, R. H., 1954: *J. Anim. Sci., 13*: 464.
Edey, T. N., 1966: *J. agric. Sci., Camb., 67*: 287.
——— 1967: *J. Reprod. Fert., 13*: 437.
——— 1969: *Anim. Breed. Abstr., 37*: 173.
——— 1970a: *J. agric. Sci., Camb., 74*: 181.
——— 1970b: *J. agric. Sci., Camb., 74*: 187.
——— 1970c: *J. agric. Sci., Camb., 74*: 193.
——— 1970d: *J. agric. Sci., Camb., 74*: 199.
——— 1972: *J. Reprod. Fert., 28*: 147.
El Sheikh, A. S.; Hulet, C. V.; Pope, A. L.; Casida, L. E., 1955: *J. Anim. Sci., 14*: 919.
Foote, W. C.; Pope, A. L.; Chapman, A. B.; Casida, L. E., 1959: *J. Anim. Sci., 18*: 453.
Godwin, K. O.; Kuchel, R. E.; Buckley, R. A., 1970: *Aust. J. exp. Agric. Anim. Husb., 10*: 672.
Guerra, J. C.; Thwaites, C. J.; Edey, T. N., 1971: *J. agric. Sci., Camb., 76*: 177.
———; ———; ——— 1972: *J. agric. Sci., Camb., 78*: 245.
Gunn, R. G.; Doney, J. M.; Russel, A. J. F., 1972: *J. agric. Sci., Camb., 79*: 19.
Hartley, W. J., 1963: *Proc. N.Z. Soc. Anim. Prod., 23*: 20.
Hodge, R. W., 1966: *Aust. J. exp. Agric. Anim. Husb., 6*: 314.
Hoxsey, V.; Hoversland, A. S.; Blackmore, D. W.; Van Horn, J. L., 1960: *J. Anim. Sci., 19*: 959 (Abstr.).
Killeen, I. D., 1967: *Aust. J. exp. Agric. Anim. Husb., 7*: 126.
Mackenzie, A. J.; Edey, T. N., 1975a: *J. agric. Sci., Camb., 84*: 113.
———; ——— 1975b: *J. agric. Sci., Camb., 84*: 119.
Mattner, P. E.; Braden, A. W. H., 1967: *Aust. J. exp. Agric. Anim. Husb., 7*: 110.
Moore, N. W.; Rowson, L. E. A.; Short, R. V., 1960: *J. Reprod. Fert., 1*: 332.
Moule, G. R.; Braden, A. W. H.; Lamond, D. R., 1963: *Anim. Breed. Abstr., 31*: 139.
Mudd, A. J.; Mackie, I. L., 1973: *Vet. Rec., 93*: 197.
Quinlivan, T. D.; Martin, C. A.; Taylor, W. B.; Cairney, I. M., 1966: *J. Reprod. Fert., 11*: 379.
Scofield, A. M., 1972: In *Pig Production* (ed. D. J. A. Cole). Butterworths, London.
Van der Westhuysen, J. M., 1971: *Agroanimalia, 3*: 91.
Van Kampen, K. R.; Binns, W.; James, L. F.; Balls, L. D., 1969: *Am. J. vet. Res., 30*: 517.
Van Niekerk, C. H.; Belonjé, P. C.; Hunter, G. L., 1968: *Proc. 6th int. Congr. Anim. Reprod. artif. Insem., Paris. 1*: 455.
Williams, H. Ll.; Hill, R.; Alderman, G., 1965: *Brit. vet. J., 121*: 2.