

ENVIRONMENTAL STRESS AND REPRODUCTION IN COWS AND SHEEP

Dobson, H.

Department of Veterinary Clinical Science, University of Liverpool, Leahurst, Neston, Wirral, CH64 7TE, United Kingdom

SUMMARY

The husbandry of ruminants has not kept pace with genetic advances in potential performance. Adequate feeding and housing have great influence on animal comfort and reproductive performance. Increasing severity of mastitis or lameness both reduce fertility, in spite of treatment. In cows and sheep, reductions in LH pulse and/or surge patterns contribute to decreased fertility. For a short-term solution, animals need to be fed better, provided with improved housing, and subject to fewer production-related diseases. In the long-term, genetic strategies such as by increasing persistency of lactation in dairy animals and by increased use of gene markers should provide a solution.

Keywords: genetics, feeding, housing, LH, production diseases, persistent lactation

INTRODUCTION

The aim of this paper is to high-light the pressure points (vulnerable links) in the interaction between the environment and reproductive performance in cows and sheep. This will be achieved by reflecting on

- the problems encountered;
- the principal mechanism by which these are mediated;
- suggesting appropriate solutions.

‘Progress’ from extensive to intensive husbandry within the cattle and sheep industry has led to animal under-performance (and heightened public concern in some countries). For example within the dairy cow population, there is substantial evidence to show that production-related diseases reduce both fertility and potential milk yields (Borsberry & Dobson 1989; Bareille et al 2003).

THE PROBLEMS

Genetics and breeding

Approximately 60–65% of the increased yield per dairy cow over the past 20 years has been achieved by selective breeding i.e., via genotype. In other words, since 1983 an extra 120–135 kg fat per year is now being produced per cow, 70–89 kg of which are attributable to genetic gain (JP Chenais and F Miglior, *personal communication*). Regrettably this means that the way we look after animals, i.e., ‘husbandry’, is not keeping pace with genetics. Many countries now have a

national dairy herd that produces at least 7000 litres milk per cow in 305-day lactations but concerns are being expressed about a concurrent reduction in fertility (Royal et al 2000; Butler 2003). Our inability to appropriately feed and house these very high-yielding animals is leading to a failure to meet genetic potential, and we must provide realistic solutions soon if we want a sustainable agricultural industry.

There is little information widely available comparing genotype to phenotype success in the sheep and beef industries. Increased prolificacy is one approach to increase sheep productivity; however, considerable research has revealed that incorporation of 'prolificacy' genes in sheep leads to greater embryo losses. For example, within the highly prolific Cambridge breed, pure bred ewes with no copies of the prolificacy gene(s) have 2–3 ovulations and subsequently 2–3 lambs; ewes with one copy have 4–5 ovulations and 4–5 lambs; whereas, those with 9–13 ovulations produce only 1–2 lambs (DAR Davies, *personal communication*). Furthermore, there are some breeds of sheep, for example the Inverdale, that possess a gene motif that can result in very poor development of the uterine tract, obviously resulting in infertility of part of the population (Davis et al, 1992).

Feeding

A major characteristic that genetics has introduced into many national dairy cattle herds is the sudden large increase in milk yield in early lactation. As suggested above, one of the most common difficulties encountered by modern dairy cows is achieving sufficient food dry matter intake during the early-mid postpartum period. This is revealed by poor body condition scores in early lactation and a marked improvement in pregnancy rates once dry matter intake increases resulting in positive energy balance (Lucy 2001).

The phenomenon of feed 'flushing' has been incorporated into both sheep and beef husbandry for many years to hasten seasonal breeding periods or overcome oestrus suppression due to suckling (Clark 1934). There are several recipes for increasing fertility by increasing energy availability as day-length begins to decrease in sheep (with or without pre-exposure to teaser rams; Walkden-Brown et al 1999); and weaning, even if temporary, has positive effects on return to optimal fertility in beef cows (Stagg et al, 1998). However, the converse of all these situations is also true, leading to considerable restriction on efficiency in some commercial herds and flocks.

Housing

Adequate cubicle size and number are important for cow comfort, but while the provision of straw-yards increases dairy cow lying time, a compromise has to be made with an increased incidence of udder infections (Whitaker et al, 2000). Dairy cows housed on (slatted) slippery floors express oestrus less intensively with major impact on pregnancy rates to artificial insemination (AI; Britt et al, 1986). High environmental temperatures (>30°C, either within housing or outside without shade) reduce fertility, and this is even more dramatic when dairy cows are under the pressure of producing high milk yields (Al-Katanani et al, 1999). Appropriate levels and locations of lighting are also important as increasing exposure to light in the cubicle area during winter increases lying time, and would thus hinder signs of oestrus (Phillips et al, 1998).

It is unusual to house sheep or suckler cows in the traditional extensive husbandry practice, although it is accepted that this does occur in some parts of the world. However, there are few documented data in the scientific literature concerning the influence of housing on fertility in sheep flocks and beef herds.

Production-related diseases

Ask any dairy farmer what are the main three concerns regarding the herd's performance and a reply will given: mastitis, lameness and fertility. While there is variation between farms, this is only with regard to the priority of these three aspects. Increasing severity of mastitis (or somatic cell counts, SCC) or lameness both reduce fertility and expression of oestrus in spite of treatment (Dobson et al 2001; Walker et al 2005). Several of these post-partum 'production' diseases have their origins in inappropriate feeding either in late pregnancy (dystocia, milk fever) or early post-partum (ketosis) which may also lead to compromised immunity (mastitis, endometritis). Lameness often arises due to changes of diet before and after calving (resulting in changes in hoof growth), but stone-free walkways, comfortable cubicles (to increase lying times) and absence of slurry-pools (predisposing to digital dermatitis) are all important aspects related to the environment in which cows are kept.

There have been few studies in the UK attempting to evaluate clinical reasons for lowered fertility in commercial sheep flocks. However, one report did high-light an incidence of >15% anatomical abnormalities capable of interfering with establishment of a pregnancy (e.g., uterine tract adhesions, blocked fallopian tubes, mucometra; Winter & Dobson 1992). These abnormalities are probably the result of damage and/or infection incurred during lambing.

MECHANISM BEHIND THE PROBLEMS

Females must produce a fertile egg and attract the male (or an AI technician) at the right time to achieve successful fertilisation. In order to do this effectively, follicles must grow in the ovaries, egg(s) need to be released into the female reproductive tract, and hormones produced not only to control pheromone release but also to prepare the uterus to receive the conceptus and maintain the corpus luteum during the 'maternal recognition of pregnancy'.

In more detail for both cows and sheep, neurotransmitters in the higher brain control gonadotrophin releasing hormone (GnRH) and hence luteinising hormone (LH) pulse secretion. Initially, LH is secreted in small discrete pulses, the frequency and amplitude of which drive growth of ovarian follicle(s) and oestradiol production with consequent pheromone release. Towards the end of the follicular phase, there is a precisely-timed surge of LH secretion. This causes resumption of meiosis in the egg(s), further egg cytoplasmic maturation, ovulation and formation of a corpus luteum in the residual follicular tissue. Sequential exposure to correct concentrations of oestradiol (from the growing follicle) and progesterone (from the subsequent corpus luteum) prepare the uterine environment, and in concert with signals from the conceptus, a successful pregnancy will be established.

In evolutionary terms, establishing a pregnancy is a very high-risk strategy for female mammals to pass genes on to the next generation. It is hypothesised that if conditions are not appropriate, LH pulse frequencies and amplitudes will be disrupted, as well as the timing and amplitude of the LH surge, resulting in failure to initiate a pregnancy. Of necessity, this disruption is usually temporary so that when prevailing conditions improve, normal fertility will resume.

Many examples exist in the literature to support this hypothesis and link in with aspects of the environment referred to above as 'the problem'. In all these situations, there is evidence available in cows and sheep to show that either LH pulse or surge patterns (or both) are disrupted and no doubt contributory to changes in fertility (feeding: Butler 2003; male effect: Walkden-Brown et al 1999; day-length: Karsch et al, 1993; weaning: Staggs et al 1998; heat: Badinga et al, 1994;

mastitis: Schrick et al, 2005, Kaneko and Dobson *in preparation*; lameness: Morris and Dobson *in preparation*). In summary, most aspects of environment-induced changes in fertility are orchestrated by LH secretion profiles which in turn are dependant on GnRH patterns and ultimately hypothalamic neurotransmitter system(s) in the brain.

THE SOLUTIONS

Pay greater attention to the environment in which we keep animals!

In the short-term, get more food into post-partum animals, provide better housing/environment, and reduce the incidence of production-related diseases. But this is easier said than done; the vast majority of farmers are not wilfully starving and mistreating their animals – with the increasing reduction in fertility this would be an ultimately self-destruction strategy. However, there is evidence to show that some farmers can keep digestive diseases, mastitis and lameness to a minimum: 8% clinical mastitis rate, and 2.5% clinical lameness (Whitaker et al, 2004).

In the longer-term, upon reflection, it is apparent that the genetic make-up of the breeds being farmed has out-stripped any advances in husbandry of cows. Perversely, perhaps we should be using genetic strategies to provide a solution? For dairy cows, this would mean exploiting the heritability for persistent lactation estimated at 0.09–0.18 (Haile-Mariam et al 2003). Increases in persistency will decrease peak milk yield 60–80 days after calving (thus relieving the importance of high dry matter intake in early lactation). It will also allow extension of the calving-calving interval thus reducing the frequency of calving that is the major risk factor for the production diseases that occur within the first 60–80 days postpartum. More thoughtful selective breeding within the sheep industry might reduce the emphasis on major leaps in prolificacy and concentrate more on the survivability of the embryo, fetus and neonate. In both species, increased use of SNPs (genetic markers) have the potential to help.

The problems we currently have are important for mankind in terms of reduced (re)productive performance; not for the animals. They are only responding to the abuse imposed by the environment in which they have been ‘domesticated’. The solution to maintain a sustainable fertile population is now an urgent remit for agriculturalists, veterinarians, geneticists and scientists.

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