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The high producing dairy cow and its reproductive performance

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Contents:

Intensive genetic selection has resulted in modern dairy cow with very high milk yields but reduced fertility, due mainly to an increase in postpartum clinical problems, poor expression of oestrus, defective oocytes/embryos and uterine infections. It is a challenge to get enough food into these cows to meet the high demands of peak milk yields in early lactation and the animals require considerable veterinary attention in the early period after calving. Both genetic and management changes to increase the persistency of lactations would reduce the number and intensity of clinical risk periods throughout a cow's life without compromising milk output.

Introduction

The high-yielding dairy cow produces a lot of milk; and the main emphasis for dairy farmers is to sell as much milk as possible with maximum efficiency, both financially and in terms of animal welfare. The number of calves produced is of secondary importance, and this should be remembered by managerial/agricultural advisors and veterinarians. Of course, a cow must have a calf to begin lactating and the need to create the next generation must not be forgotten. However, the prime commodity for the dairy industry is milk of a quality appropriate for further processing to drink (the liquid market), or manufacture into cheese, butter, cream, yoghurt, etc.

Why we are where we are in 2007

What is ‘high’ milk production? This depends on the breed of cow; for example Channel Island herds (Jerseys, Guernseys) have average 305-day lactations of ~4000 litres with substantial constituents of ~4.5% fat and ~4% protein; in comparison, Friesians have typical 305-day lactations of ~7000 litres at ~4% fat and 3.5% protein; whereas, Holstein cows can achieve ~10,000 litres (~3.5% fat and 3% protein). Hence, the total lactation yields of fat are 180kg, 280kg and 350kg for Channel Island, Friesian and Holstein cows, respectively. However nowadays, most milk is divided into its constituents during the initial manufacturing process (even drinking milk), and then reconstituted with varying percentages of fat (and protein to a lesser extent) depending on the final product, milk, cream, butter or cheese.

Cows must be fed appropriately to produce high yields. However, there is a maximum output that can be mainly supported by forage, the natural diet of ruminants (~6000 litres per 305 day lactation). Herds that depend on grazing ± conserved forage for the majority of the diet will be constrained by environmental opportunities to grow herbage. Thus, their calving patterns are dictated by the need to maximise the efficiency of ‘milk-from-forage’. Nevertheless, these animals may suffer the same problems as those herds housed all-year-round producing 10,000 litres. Thus, the implications of ‘high-producing’ can not be defined

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in finite litres of milk or kg fat; but must also take into account the breed and management systems involved.

Financial efficiency and welfare depend in large part on the correct feeding of the cow. When working out feed-rations, there is a component for maintenance of the cow's own body and a further requirement depending on the amount of milk produced. For example, each day a mature 666kg Holstein requires 60 MegaJoules each day for maintenance plus 5 MegaJoules per litre of milk produced. Whatever the breed or size of cow, financial efficiency depends on the amount of milk that can be produced per kg cow requiring maintenance. Table 1 compares current financial aspects of producing milk from a typical cow from three different breeds in the UK; there may be variances between different countries but the nuances will not have major effects on the comparison.

Defining the problems and providing solutions

The financial comparisons in Table 1 emphasise why many dairy farmers enthusiastically engaged in the genetic transformation to breed high-producing cows – the comparison between breeds is stark but the same principles apply even within a breed. But at what cost has this change occurred? There is now considerable evidence available suggesting that cattle fertility (expressed in terms of pregnancy rates to first insemination) has declined over the last 50 years during the rapid increase in yields (Royal et al. 2000). Thus, genetic correlations between fertility and production are generally unfavourable, although there are also some breed exceptions, such as the Norwegian Red (Chang et al. 2006). Nevertheless, are the majority of our modern cows trying to tell us something? Cows of high genetic merit for milk yield mobilise more body tissue in early lactation than cows of average genetic merit (Pryce et al. 2003). Thus, there is a negative correlation between milk yield and BCS in early lactation raising the question whether we are able to adequately feed these high genetic merit cows. The critical limiting factor is total Dry Matter Intake (DMI) - bigger cows are capable of higher milk yields but there is a limit to the size of the rumen and appetite! Higher energy density feeds have been employed but eventually these interfere with efficient functioning of the rumen leading to digestive problems (Krajcarski-Hunt et al. 2002). In spite of this, some high milk producers continue to maintain high reproductive performance avoiding the impact of yield on fertility – however, close examination of such herds reveals that very high veterinary attention is required (Lopez-Gatius et al. 2006).

Indeed, from a welfare point-of-view, examining the incidence of (sub)clinical problems around calving is disturbing: the vast majority of veterinary attention is paid to cows from one week before to ten weeks after calving (Zwald et al. 2004). Clearly, calving presents a considerable welfare risk to a dairy cow – so why make her calve so frequently? Taking an extreme example for emphasis, think of a three-year period (this is the UK average cow life-span after first calving) during which a cow calves at intervals of 1.5 years. She would only experience two periods of high risk compared to three if she calved once per year. Obviously, to be financially viable, the cow would need to continue to produce adequate amounts of milk until the necessary 6-8 weeks dry period prior to the next calving. Persistent lactations are characterised by achieving lower and later peak yields but reasonable production is maintained for longer (Fig. 1).

Persistency is flexible in that it can be enhanced by more frequent milking, feeding more concentrate during declining lactation, and by calving in the winter rather than the summer in the UK (Knight 2001). In part, persistence is also due to avoiding pregnancy soon after calving: pregnancy leads to depressed milk production in the second and third trimester. In other words, farmers need to voluntarily delay first insemination – and there is evidence to show that pregnancy rates per insemination in later lactation are equivalent or better than those soon after calving. Retnayake et al (1998) found no significant differences in

pregnancy rates for cows managed to calve at 12, 15 or 18 month intervals. Furthermore, high-yielding cows deliberately rebred at 11.5-13 months greatly improved profitability without any other deliberate action to improve lactation persistency (Arbel et al. 2001).

Some people may be concerned that there will be a reduction in the number of calves born per year – but remember, dairy farmers are in business to sell milk, not surplus calves. Other people may be worried about an interruption in the availability of replacement heifers. In a reasonably well-managed stable herd, with an annual culling rate of ~20% (which could decrease with improved cow welfare due to lowered peri-parturient clinical risk), only ~20% replacements are required. The increasing use of sexed semen will also overcome this problem, especially when genetic changes are required (Klinic et al. 2007).

To be financially acceptable, the milk yield from two calvings over three years will have to be equal or greater than that delivered by three annual calvings in the extreme example used above; but currently this extreme is not achievable. However, the heritability for persistent lactation has been estimated at 0.09-0.18; compared to 0.03-0.19 for fertility (Dekkers et al. 1998; Haile-Mariam et al. 2003; Muir et al. 2004). This is in comparison to the heritability for 305-day milk yield of 0.45 that has increased UK production from 3000 litres in 1960 to 7000 litres in 2000. So, it should be possible with the widespread use of AI in dairy cattle to make quite considerable advances in persistency so that farmers can effectively manage voluntarily extended calving-to-calving intervals. Predictions are encouraging – the economic value of persistency almost triples when the calving interval increases from 12 to 13 months (Dekkers et al. 1998). However, caution has been urged as cows with mean high somatic cell counts have reduced persistency of yield but this can be overcome by attention to udder health (Haile-Miriam et al. 2003).

Understanding the causes behind the constraints to reproductive performance

Until the full genetic delivery of persistent lactation, it is instructive to reflect on exactly how reproductive performance is being limited in high-producing cows. Knowing the precise nature and extent of the constraints will enable melioration of the effects.

a) Clinical conditions—Some infectious diseases do have indirect effects through compromise of immune status in high-producing cows or even direct effects on pregnancy rates (for example, *Leptospirosis*: Dhaliwal et al. 1996; *BVDv*: Fray et al. 2002). Other infections, such as *Brucella* or *Neospora* cause abortions and hence reduce the milk output of herds and sometimes fertility. However, even though infectious diseases are damaging in individual herds, on a larger scale the impact of infectious diseases can be overcome by eradication schemes (eg *Brucella*) or by vaccination programmes (eg *Leptospirosis, BVDv, IBR)*.

More insidious are the ‘management/production’ diseases such as undernutrition (poor BCS or loss of BCS), hypocalcaemia, mastitis, and lameness that all lead to reduced reproductive performance compared to unaffected contemporaneous herd-mates. Cows with high milk yields and low BCS in the early postpartum period, take >10 days longer to conceive (Lopez-Gatius et al. 2003; Garnsworthy 2006), and those succumbing to hypocalcaemia take 13 extra days to get pregnant (Parker 1992). Indeed, the calving-to-pregnancy interval is extended for at least 7, 8, 26 and 31 days in cows treated for mastitis, retained fetal membranes, hypocalcaemia or endometritis, respectively, compared to healthy herd-mates (Borsberry and Dobson, 1989; Schrick et al. 2001). Lameness is associated with even worse reproduction performance, as up to 40 days are lost to get lame cows in-calf again even though the lameness has been treated (Collick et al. 1989 Melendez et al. 2003; Hernandez et al. 2005; Fig. 2). In part, these poor fertility data may be related to delayed resumption of ovarian cyclicity after calving. For example, if cows have mastitis soon after calving, luteal

activity starts 7 days later than healthy animals (Huszenicza et al., 2005). Similarly, lame cows begin post-partum luteal activity later than unaffected herd-mates (50 *versus* 33 days; Petersson et al. 2006) However, delayed cyclicity is not the whole cause of lowered fertility as this only accounts for approximately half the delay in getting lame cows pregnant again; the ability to express oestrus is also important (see later).

Obviously, prevention of all the above clinical conditions by the implementation of good management practices will be rewarding, both financially but perhaps more importantly with respect to animal welfare. Prevention may be partially achieved via selective breeding as genetic correlations for production diseases are typically between +0.10 and +0.40 (Weigel 2006).

With an ironic downwards twist in the tail, while high milk yields make dairy cows more susceptible to production diseases (including lowered fertility), the converse is also true - several production diseases reduce milk yield. Systemic or local mastitis, lameness, ketosis or hypocalcaemia are associated with respective losses of 160, 75, 75-100, 88 or 45 kg milk over the first 140 days of lactation (Bareille et al. 2003).

b) Expression of oestrous behaviour and return to ovarian cyclicity— Establishing a pregnancy is risky for animals because, in the later stages, the fetus makes huge demands on the dam (mainly nutritional). So following the survival instinct, animals will choose to not show signs of oestrus in order to avoid conception. Indeed, there is a higher incidence of ‘silent’ ovulations (no oestrus) in Holsteins with increasing levels of milk production (1.6 versus 0.7 silent oestruses; for 36 and 28 kg/day, respectively; Harrison et al., 1990). Furthermore, cows loosing most weight (BCS) in the early postpartum period, take 30 days longer to display the first post-partum oestrus (Butler 2003), and once started, periods of oestrus are shorter in higher producing Holstein cows (Table 2). Other aspects of oestrus are also affected by high milk production (Table 2).

If cows have mastitis around the time of the first ‘silent’ oestrus (15-28 days postpartum), luteal activity resumes 39 *versus* 32 days postpartum; but the onset of oestrous behaviour does not occur until 91 *versus* 84 days, in mastitic and healthy cows, respectively (Huszenicza et al., 2005).

Concerning the mechanisms involved behind these observations, we have provided considerable evidence to show that both acute and chronic stressors (including some of the production diseases cited above) are associated with interactions between the hypothalamus-pituitary-adrenal gland and hypothalamus-pituitary-ovarian axis (Dobson and Smith 2000; Dobson et al. 2001, 2003). For example, acute stressors reduce GnRH and hence LH pulse frequency, leading to short–term decreases in follicular oestradiol production, as well as delaying and reducing the magnitude of the LH surge.

However, there do appear to be different consequences of chronic stressors as revealed by our more detailed behavioural studies in high-producing lame cows. While lame cows display the same incidence of oestrus (56% of low milk progesterone periods associated with any oestrus behaviour), the duration and intensities of behavioural signs are decreased mainly in terms of reduced sniffing of the vulva and less mounting activity (Table 2). Furthermore, although milk oestradiol profiles are unexpectedly the same in chronically lame and non-lame cows, progesterone concentrations (especially in a period 6 days before oestrus) are lower in lame cows and this may have consequences for pheromone production and/or detection mechanisms (Walker et al. 2006).

c) Oocyte and embryo quality—In the real world, reproductive performance is not dependant on one factor: there are many facets. Even if sufficient oestrus signs are exhibited and insemination does take place, the fertility of high-producing cows is still compromised.

Reproductive performance can be affected by changes during the breeding period; for example, heat stress severely reduces fertility in cows with pregnancy rates decreasing to as low as 10% in environmental temperatures of 33°C (Hansen and Arechiga, 1999). On the other hand, some events have more long-lasting effects; for example, the signs of dystocia, or immediate post-partum hypocalcaemia, endometritis or mastitis can be 'cured' within days by clinical treatment but the cows are subfertile many weeks later during the breeding period (Borsberry and Dobson, 1989; Huszenicza et al. 2005). Detailed data reveal that heat stress also has long lasting consequences, and fertility is still lower when environmental temperatures have decreased in the autumn (September, October and November). Furthermore, these effects are exacerbated by increasing milk yields (Fig. 3). Clearly, high-producing post-partum cows are surviving on a knife-edge and in the long-term are more susceptible to challenge.

Follicular growth takes place over 3-4 months (Webb et al. 2004) involving several critical stages that can be disrupted during environmental or physiological insult leading to production of defective oocytes, and subsequent poor embryo quality. Acute heat stress during early stages of antral follicular development in high-yielding cows reduces steroid production in pre-ovulatory follicles several weeks later, and oocyte quality improves slowly and gradually only towards the end of the cooler period in hot countries (Roth et al. 2001a, b). In addition, there is an interaction between high milk production and body condition score (BCS) as oocytes collected from high-producing cows with poor BCS have lower *in vitro* rates of cleavage than those derived from high BCS animals (Snijders et al. 2000). One stage further during *in vivo* development, Leroy et al. (2005) categorised only 13% embryos as excellent during collection on Day 6 after oestrus from high-yielding first lactation cows compared to 62.5% from non-lactating heifers.

d) Uterine infection—With increasing milk yields from 1980 to 1998, there has been an increase from 7% to 17% in the incidence of persistent corpora lutea as determined by milk progesterone profiles (Royal et al. 2000; Lucy 2001). This increase in atypical progesterone profiles is also associated with a greater incidence of uterine infection (21.4 *versus* 11.5%) and longer calving intervals (402 *versus* 382 days) compared to animals with normal progesterone profiles. Indeed, one of the major risk factors for prolonged luteal phases in high-yielding dairy cows is uterine infection (endometritis; Opsomer et al. 2000).

If cows are seen in oestrus and inseminated, the uterus must be fit to receive sperm and then the fertilised zygote. Unfortunately, there is a considerable decrease in fertility caused by contamination of the uterus after calving, with 40% animals developing uterine infection that persists for more than three weeks in 15% high-yielding cows (Sheldon et al. 2002; Sheldon and Dobson 2004). *Eschericia coli* is the most common isolate from the uterus; and lipopolysaccharide (LPS), the main pathogenic product from *E. coli*, occurs in plasma of cows with postpartum uterine infection (Sheldon et al. 2002; Mateus et al. 2003). The cells of the endometrium express the specific receptor complex for detection of LPS, and LPS switches prostaglandin secretion from $F_{2\alpha}$ to E_2, which likely disrupts luteolysis (Herath et al. 2006). The subfertility is not only associated with uterine damage but also with disruption of ovarian cycle control mechanisms. Uterine disease associated with *E. coli* or infusions of LPS both suppress follicular growth, decrease oestradiol production and delay the LH surge and ovulation (Suzuki et al. 2001; Sheldon et al. 2002). Although plasma FSH concentrations *in vivo* are not affected, LPS treatment *in vitro* down regulates the expression of aromatase mRNA in granulosa cells, however, LHR expression remains unaffected

(Williams et al. 2007a). Thus, folliculogenesis appears to be directly perturbed by the inability of ovarian granulosa cells to aromatise androstenedione to oestradiol in response to FSH. Furthermore, plasma progesterone concentrations are lower after ovulation of dominant follicles in cows with uterine infection, or those infused with LPS (Williams et al. 2007b). Taken together, it appears that uterine disease not only causes endometrial pathology that would perturb fertility but also modulates the endocrine function of the uterus and ovary.

Conclusions

Most mammals lactate to nurture their off-spring but domestication of the dairy cow has involved removal of the neonate(s), thus eliminating biological feedback systems regulating the volume and pattern of milk production. Exploitation of this reduced feedback has led to very high milk yields; however, we are starting to pay the cost in terms of reduced reproductive performance. It is possible that there is a solution to this problem available through genetic enhancement of persistent lactations that will lead to a reduction in the number of calvings and hence avoidance of postpartum energy deficits, stress-induced disruption of follicular development and oestrus behaviour, production of defective oocytes and embryos, and a reduction in the impact of uterine infections.

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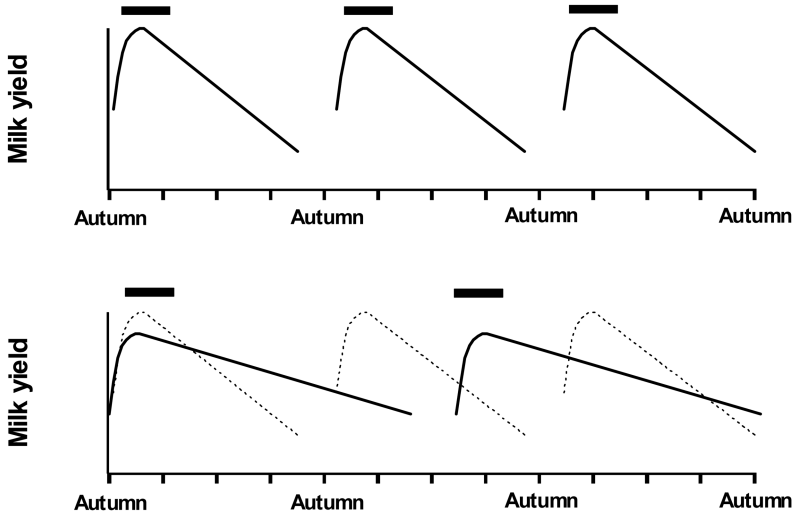

Fig. 1.
Diagram of milk yields in cows that a) calve every year b) calve every 1.5 years. The horizontal bars represent periods of risk around calving. The dotted line in b) represents yields in cows calving every year.

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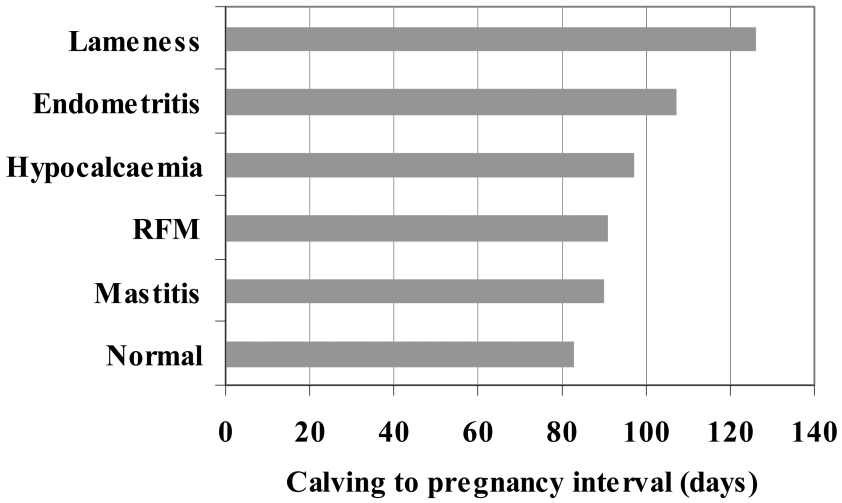

Fig. 2.
Calving to pregnancy intervals for cows treated for various clinical conditions (data from: Borsberry and Dobson 1989; Collick et al. 1989)

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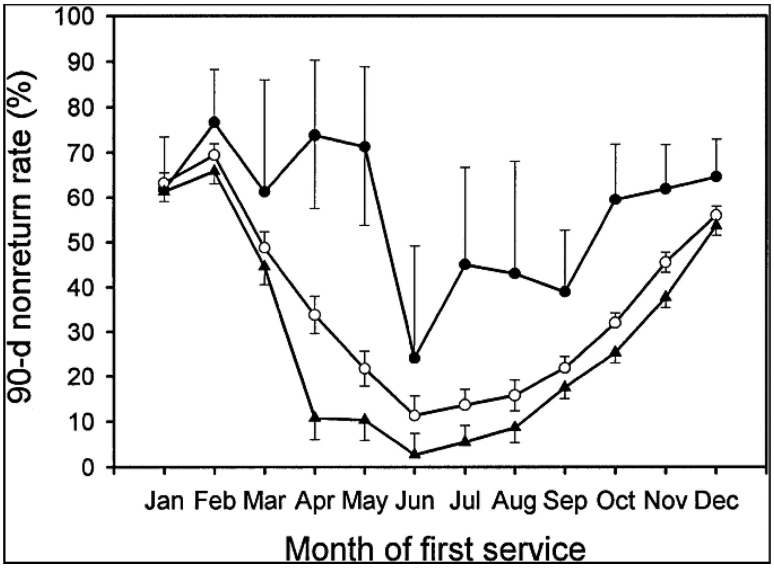

Figure 3.
Seasonal variation in 90-day nonreturn rate to first service as affected by mature equivalent milk yield. Results represent least squares means ± SEM adjusted for interval to first service when cows were grouped according to milk yield ((●) < 4536 kg; (○) 4536 to 9072 kg; (▲) >9072 kg). Reprinted from Al-Katanani et al. 1999 with permission.

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Table 1

Financial aspects of feeding a cow of different breeds compared to milk yield

	Channel Island	Friesian	Holstein
Average 305-day yield (litres)	4,000	6,000	10,000
Average daily yield (litres)	13	20	33
Mature weight (kg)	444	555	666
Feed costs £ (euro)	£536 (788 €)	£666 (979 €)	£808 (1187)
Milk income £ (euro)	£764(1123 €)	£1116(1640 €)	£1640(2410 €)
'Profit' (milk minus feed)	£228 (335 €)	£450 (661 €)	£832 (1223 €)

These calculations assume average costs of £125 (184 €) per tonne concentrates, and £25 (38 €)) per tonne conserved forage, and income of 18p (0.26 €)) per standard litre (4% fat, 3.3% protein) with 2p (0.03 €)) per litre adjustment per 1% above/below standard for fat and protein. Fixed costs and labour per cow are the same irrespective of breed but have not been included in the 'profit' figure.

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Table 2

Effects of milk yield or lameness on aspects of oestrus

	Milk yield[1]		Lameness status[2]	
	Average	*High*	*Not lame*	*Lame*
Duration of being mounted (h)	11	6*	11.0	7.5*
Incidence of vulval sniffing	-	-	13	6*
Incidence of standing in oestrus	8.8	6.3*	9.5	5.6*
Duration of standing (sec)	28.2	21.7*	-	-

* $p<0.05$ within a study;

[1] Lopez et al. 2004 (33 versus 46 kg/day);

[2] Walker et al. 2005 (yield 43kg/day)