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Review

Nutrition and production diseases of transition dairy cows and their effect on subsequent reproduction

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Introduction

Production diseases of dairy cows are caused by a level of production inconsistent with nutrient intake, provision of an inadequate diet, an unsuitable environment, an inappropriate breeding policy or combinations of these factors. The transition from gestation to lactation is a perilous period for the dairy cow. The events occurring during this period are probably the most significant factors that predispose dairy cows to ill-health. The production diseases of the dairy cow are a manifestation of the cow's inability to cope with the metabolic demands of high production, and they continue to be a cause of economic loss to the dairy industry and a welfare concern. Traditionally, the term production disease encompassed hypocalcaemia, hypomagnesaemia and ketosis. However, the term has recently been broadened to include conditions such as retained placenta, metritis, fatty liver, displacement of the abomasum and laminitis. Such metabolic problems affecting early postpartum cows extend the period of negative energy balance and delay resumption of ovarian cycles, reducing subsequent fertility (Butler, 2000). Prevalence of endometritis remains high at 50 d postpartum and reduces fertility (Gilbert *et al.,* 2005). Improvements in fertility in lactating dairy cows can be achieved by feeding management during the transition period. Many nutritional and management strategies of the pre-partum cow alter the degree of negative energy balance, hypocalcaemia, immunosuppression and digestive disorders experienced by the transition cow (Mulligan and Doherty, 2008; Mezzetti *et al*., 2021).

***The transition cow*:** About 30 to 50 per cent of dairy cows are affected by metabolic or infectious disease around calving (LeBlanc, 2010). A smooth transition from the late dry period through early lactation affects lactation and conception. Grummer (1995) described the transition period as the period from 3 weeks pre-partum until 3 weeks

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post-partum. The period is characterised by marked changes in the endocrine status of the animal, and a reduction in feed intake when nutrient demand for the developing conceptus is increasing. The initiation of milk production increases calcium output when input cannot be increased in the short-term in many dairy cattle, while the reduced feed intake creates an imbalance of energy input relative to output. High-producing dairy cows drastically adjust their metabolism during this period so that nutrients can be partitioned to support milk synthesis.

Transition dairy cows also experience immunosuppression in the periparturient period and often have sudden dietary changes that cause digestive disturbances (Mulligan and Doherty, 2008). They are also likely to experience environmental stress arising from the group changes associated with management of dry and lactating cows. When these effects are combined with the exertions of parturition, it is not surprising that the period of highest risk for production disease is immediately after parturition.

***Parturition and utero-ovarian changes*:** After calving, the uterus must return to a condition to support a new pregnancy. This begins with the clearance and involution of the uterus, regeneration of the endometrium and elimination of bacterial contamination followed by resumption of ovarian activity by a set of interactions between the brain, liver, ovaries and uterus (Wathes *et al.,* 2007). However, stress or negative energy balance (NEB) influences normal return to reproductive function. Parturition is a source of stress, and interaction between stress and fertility has been underlined (Dobson *et al.,* 2001; Alam and Dobson, 1986; Dobson and Alam, 1986). Dystocia is associated with acute pain and is a significant welfare issue in dairy cows. Breeding larger cattle, inbreeding, inappropriate calving assistance, and breeding heifers at young ages, have resulted in this condition being more common (Mee, 2008). Acute or chronic stress results in reduced pulsatile patterns of GnRH release and LH pulse frequency. Such altered hormone levels exert a negative effect on follicle growth, development and probably ovulation. Moreover, energy balance, to an extent, determines when normal reproduction resumes after parturition (Schneider, 2004). In cattle, under-nutrition has been linked with reduced frequency of LH pulses (Schillo, 1992), which support follicle growth, maturation, and ovulation. A favourable nutritional status favours the release of GnRH and gonadotrophins. Mechanisms that regulate energy and nutrient distribution in the body may affect the hypothalamo-pituitary-ovarian axis (Chagas *et al.,* 2007). FSH is essential for the recruitment of antral follicles in the ovary and LH pulse frequency is related to follicle development and ovulation. Energy deficiency delays the first ovulation after calving as NEB limits GnRH and gonadotrophin secretion (Schneider, 2004). Abnormal oestrous cycles postpartum with reduced oestrus expression, anoestrus, cyst formation and delayed first ovulation led to prolonged calving to first insemination intervals (Lopez *et al.,* 2004; Vanholder *et al.,* 2006). Low postpartum insulin and glucose concentrations may suppress GnRH secretion and subsequent LH release. As

insulin locally stimulates follicular growth, maturation and steroidogenesis, reduced postpartum concentrations are linked to ovarian dysfunction. At the ovarian level, non-esterified fatty acids (NEFA) may affect follicular growth and development by acting directly on follicular cells. There may be decreased conception rates and increased incidence of early embryonic mortality (Bilodeau-Goeseels and Kastelic 2003). Up to 70 - 80% of the total embryonic and fetal losses typically occur during the early embryonic, pre-attachment period which seems to be a sensitive time. In early lactation, milk production is favoured over fertility, because if nutrition becomes scarce, the lactating dam will preferentially invest the limited resources in the survival of living offspring rather than gambling on a future pregnancy. This leads to delayed exhibition of first oestrus postpartum, delayed conception and increased inter-calving intervals.

***Metabolic changes during the transition period*:** During the last 3 weeks of pregnancy the endocrine status of the cow changes very quickly (Tufarelli *et al*., 2014). As calving approaches, concentrations of progesterone in blood decrease and those of oestrogen remain high or increase (Grummer, 1995). There is a marked increase of nutrient demand to satisfy fetal development and mammary activity; in contrast, dry matter intake is often lower than requirements and body reserves need to be mobilized. High circulating oestrogen is believed to contribute to decreased dry matter intake around calving (Grummer, 1993), resulting in NEB. Drastically reduced insulin concentration because of the NEB brings about energy mobilisation and partitioning of energy to the udder, promotes the production of glucose in the liver and the mobilization of fat reserves. The mobilized NEFA serves as an alternative energy source for other tissues to preserve glucose, which is preferentially used by the mammary gland to form lactose. NEFA are predominantly transported to the liver where they are oxidized to provide energy or transformed into ketone bodies, again an alternative energy source elsewhere in the body. An excessive loss of body condition during the transition period has been identified as a major risk factor for health and fertility (Roche *et al.*, 2007), which stresses the importance of monitoring body condition early postpartum (Chagas *et al*., 2007; Caixeta and Omontese, 2021; Mezzetti *et al*., 2021; Ghaffari *et al*., 2024).

***Diseases during the transition period*:** During the postpartum period, dairy cows are at risk of developing hypocalcaemia, metritis, ketosis and displacement of the abomasum (Curtis *et al.,* 1983). Cows with dystocia with or without retained placenta should be monitored carefully because they are at greater risk of developing metritis, fever and hypocalcaemia. All dairy cattle experience: a period of insulin resistance, reduced feed intake, NEB, lipolysis, and weight loss in early lactation; hypocalcaemia in the days after calving; and bacterial contamination of the uterus for 2 to 3 weeks after calving. There are also changes in circulating progesterone, oestrogen, and cortisol concentrations, which contribute to reduced immune function for 1 to 2 weeks

before, and 2 to 3 weeks after calving (Goff and Horst, 1997). Moreover, social stress, negative energy balance, heat stress, endotoxin exposure, and oxidative stress promote inflammation, suppress feed intake, and impair metabolic and immune function during the transition period.

***Milk fever and subclinical hypocalcaemia*:** Many cows experience subclinical hypocalcaemia after parturition, but some experience severe hypocalcaemia (milk fever). During the dry period, calcium needs are minimal, but after parturition large amounts of calcium are exported into milk (DeGaris and Lean, 2008). This sudden calcium drain must be countered by increased absorption from the gut or mobilization from bone. Hypocalcaemia results when these mechanisms are insufficient. Milk fever is an economically important disease and significantly increases a cow's susceptibility to mastitis, retained placenta, displaced abomasum, dystocia and ketosis, which can reduce a cow's productive life (Curtis *et al.,* 1983). Milk fever and subclinical hypocalcaemia exacerbate the immunosuppression experienced by periparturient dairy cattle (Kimura *et al.*, 2006). Milk fever results in reduced fertility due to its effect on uterine muscle function, slower uterine involution (Borsberry and Dobson, 1989) and reduced blood flow to the ovaries (Jonsson and Daniel, 1997). There are also indirect effects of milk fever on fertility, mediated through dystocia, retained placenta and endometritis. Kamgarpour *et al.* (1999) reported that cows with subclinical hypocalcaemia have fewer large follicles at days 15, 30 and 45 post-partum and smaller follicles at first ovulation than normal cows. Hypocalcaemia reduces smooth muscle contraction, which results in reduced rumen and abomasal motility leading to displaced abomasum and reduced feed intake (Goff, 2008; Espiritu *et al*., 2025). Calcium treatment early postpartum, particularly of those cows affected with dystocia or retained placenta can help promote normal function of the uterus, rumen and abomasum.

***Displaced abomasum*:** Displaced abomasum (movement of the abomasum from the right to the left of the abdomen) is occurred mainly one to six weeks post-partum. It is preceded by significant changes in adipose mobilization and energy metabolism up to 3 weeks before (LeBlanc *et al.*, 2005). Displaced abomasum may indicate energy-related metabolic disorders in the dairy herd. Early lactation (Constable *et al.*, 1992), higher parities (Rasmussen *et al.*, 1999), a high body condition score at calving (Rasmussen *et al.*, 1999; Rukkwamsuk *et al.*, 1999) and other diseases (Correa *et al.*, 1990) have been associated with displaced abomasum. Overfeeding and high body condition during the dry period increases the risk of displaced abomasum (Rukkwamsuk *et al.*, 1999). Ketosis, hypocalcaemia, retained placenta and metritis may increase the risk of displaced abomasum (Massey *et al.,* 1993). Analyzed serum markers like L-FABP, TFF-3, and leptin in 48 cows affected by LDA or RDA compared to healthy controls. Findings suggest leptin may help differentiate between left- and

right-sided cases—though overall diagnostic performance remains limited (Ider *et al*., 2023).

Ketosis: Ketosis (acetonaemia) of dairy cattle may appear as a primary disease or in association with other conditions. It is a serious problem, occurring during the first 10 to 60 days after calving (Ameni *et al*., 2025). It is associated with poor management of the transition period resulting in decreased dry matter intake and decreased body condition score (BCS), increasing blood concentration of ketone bodies. The three-week period after calving seems to be the most critical time. The disease results from a lowered blood glucose and resultant fat mobilization, causing the formation of ketone bodies. Signs of ketosis in dairy cattle include dullness, depression, a staring expression and rapid loss of weight, a drop in milk production, constipation, mucus-covered faeces, incoordination and partial paralysis. Ketosis can be complicated by retained placenta, metritis and displaced abomasum etc. causing cows to go off feed. It has been associated with compromised fertility. Circulating concentrations of ketone bodies have been used as indicators of excessive lipid mobilization. The concentration of beta-hydroxybutyrate (BHB) and the duration of elevated BHB concentration were negatively associated with the probability of pregnancy following the first service (Walsh *et al*., 2007). Furthermore, the rate of pregnancy after parturition was lower among cows with higher blood BHB (Ospina *et al*., 2010).

***Retained placenta and Uterine infections*:** The uterine lumen of most post-partum cows is contaminated with environmental bacteria. Elimination of contamination is dependent on uterine involution, and regeneration of the endometrium and uterine defence mechanisms. The first and most important phagocytic cells to enter the lumen are neutrophils. In many post-partum cows, the capacity of neutrophils is reduced and retained placenta, metritis, and endometritis affect up to half of all dairy cows in the first 60 days post-partum (Sheldon *et al.,* 2006). Uterine infections lead to decreased fertility and in some cases may be fatal. Retained placenta, metritis and endometritis are strongly associated with immune function in the transition period (Kimura *et al.,* 2002). NEFA and BHB have been implicated in immunosuppression in postpartum dairy cows. Disease early postpartum can further accentuate the adverse effects of NEB, as sick cows have reduced appetite and often lose more body weight than healthy cows. Cows that fail to clear bacterial infection and have clinical endometritis and chronic subclinical inflammation have substantially decreased pregnancy rates (LeBlanc *et al.,* 2002; Gilbert *et al*., 2005).

The placenta is normally expelled within 6 h of expulsion of the calf but if not expelled by 24 h, it is defined as a retained placenta. The incidence is higher in cows with twins and after dystocia and abortion. Delayed placental separation predisposes cows to acute metritis and endometritis through bacterial multiplication and impairment of immune function of endometrial macrophages and neutrophils by bacterial endotoxins. Cows that had retained placenta had substantially higher serum

cortisol for several days before parturition (Peter and Bosu, 1987) which may be one contributor to impairment of neutrophil function. Normal expulsion of the placenta involves three components: (1) placental maturation associated with the endocrine changes in late pregnancy (2) exsanguination of the fetal side of the placenta allowing shrinkage and collapse of the villi with separation from the crypts, and (3) uterine contractions with distortion of the placentomes. There may be genetic or immunological components to the problem associated with expression of major histocompatibility complex (MHC) molecules (Joosten *et al.,* 1991). Retained placenta is an important predisposing factor for uterine infection. Typically, 25–40% of animals have clinical metritis in the first 2 weeks after calving and disease persists in up to 20% of animals as clinical endometritis (Rohmah *et al*., 2023). Metritis involves infection of the deeper layers of the uterus, whereas endometritis is infection of the lining of the uterus. Endometritis causes changes in the endometrium which can lead to delayed service, repeat breeding, decreased milk production and impairment of ovarian function, delay in first ovulation and impaired growth of dominant follicles. Santos *et al*. (2011) observed that cows with dystocia, metritis or clinical endometritis were 50 to 63% less likely to resume ovarian cyclicity and 25 to 38 % less likely to become pregnant following the first artificial insemination (AI) postpartum than healthy cows. Moreover, cows with dystocia or clinical endometritis were 67 and 55 % more likely to lose their pregnancies during the first 60 d of gestation than healthy cows.

Conclusions

The transition period, now often defined as ~90 days, is a critical phase for herd health marked by major metabolic, immune, and nutritional challenges. Dairy cows undergo physiological, metabolic and nutritional changes during transition to lactation. The multitude of disorders that dairy animals face during this period, such as retained placenta, metritis, milk fever, displaced abomasum and ketosis, directly affect fertility and hence pose a major concern for dairy producers and veterinarians. The majority of these problems are metabolic in origin because the demand for energy is high immediately post-partum and dry matter intake is lower than that required to sustain milk production. Dairy cows mobilise body reserves to meet this demand and thus enter a state of negative energy balance, which leads to diseases such as ketosis, milk fever and displaced abomasum. This state of energy deficiency decreases the release of hormones from the hypothalamo-pituitary-ovarian axis causing delay in first oestrus, fertilization and conception, and the survival of the embryo may be compromised. Dystocia, retained placenta and metritis delay reproduction and can decrease milk production, impair ovarian function, delay first ovulation and impair growth of dominant follicle, thereby delaying conception and decreasing pregnancy rates.

The transition period constitutes a turning point in the productive cycle of the cow. The manner in which the changes occur in the transition period and how they are managed are of great importance, as they are closely linked to lactation performance, clinical and subclinical postpartum diseases, and reproductive performance, which significantly affect profitability. Management strategies should focus on the early detection of metabolic imbalances using innovative biomarkers and precision tools. Supplementation with rumen-protected amino acids and polyunsaturated fatty acids is increasingly recognised for its potential to enhance health outcomes and productivity. To further improve cow welfare and dairy farm profitability, more integrated, predictive, and technology-driven approaches are needed.

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