Production diseases of the transition cow

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Abstract

Production diseases of the dairy cow are caused by a level of production inconsistent with nutrient intake, provision of an inadequate diet, an unsuitable environment, an inappropriate breeding policy or various combinations of these factors. Although the transition period of 3 weeks pre-calving until 3 weeks post-calving is associated with a peak incidence of production disease, the effects of these diseases on dairy cow health and productivity extend far into the following lactation.

Recent advances in understanding of production diseases include the emergence of propylene glycol and rumen protected choline as the supplements of choice for preventing fatty liver and the absence of any preventative effect of increased energy density in the close-up dry period diet on this condition; the linear negative influence of dietary cation anion difference (DCAD) on the incidence of milk fever regardless of urinary pH or the target level of dietary DCAD achieved; the inflammatory response associated with subacute rumen acidosis and its effect on feed intake; an increased awareness of the potential for antioxidant status to improve immunity and health in the transition period; the development of more standardised diagnostic criteria and treatment protocols for uterine infection.

A significant body of knowledge already exists which should allow for the optimal management and prevention of bovine production diseases. One of the important challenges facing the dairy industry is the development, implementation and economic assessment of practical, integrated, blueprints of best practice for prevention of the production diseases and other diseases of the dairy cow.

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Introduction

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 an animal welfare concern. While traditionally regarded as encompassing the significant metabolic disorders of dairy cows (hypocalcaemia, hypomagnesaemia and ketosis), the term ‘production disease’ has been broadened to include conditions such as retained placenta, displacement of the abomasum and laminitis. Over the past several decades dairy cows have undergone intensive genetic selection, which has increased milk yield to a level where the demand for nutrients from the diet and body tissue reserves often results in ill-health and infertility. During the same period systems of dairy production have been significantly developed, with the objective of improving producer profitability as the main driving force.

In both extensive and intensive systems, it can be argued that dairy cow health and welfare have only been considered relevant where economic benefit results. Thus, the objective of increased output per unit cost underpins intensive systems while ‘least-cost’ or ‘easy-care’ dairy farming systems may also be detrimental to animal health in extensive grassland-based dairy farming systems. Production diseases such as hypocalcaemia, hypomagnesaemia and ketosis are associated with ‘imbalances in the ‘input’ and ‘output’ of metabolites required for production or deviations in the normal ‘throughput’ pathways of the body’ (Payne et al., 1973).
Production diseases may be considered ‘a man-made problem’ resulting in ‘a breakdown of the various metabolic systems of the body under the combined strain of high production and modern intensive husbandry’ (Payne, 1972). More recent definitions of production disease have been informed by the influence of high production and management on ‘factors such as animal behaviour, immunity and gene expression’, thus the definition has been expanded to include not only ‘metabolic and nutritional diseases’ but diseases of an ‘infectious and genetic nature’ (Herdt, 2006). It is important to state that both production and environmental factors are equally implicit in the onset of production diseases. Drackley (2006) stated that ‘triggers for production disease likely lie at the interface between environmental stressors and productivity’. The common theme of all these diseases is their association with management and selection of animals for ‘efficient’ agricultural production (Herdt, 2006).

Recent trends in the occurrence of production diseases

Despite the significant advances in our understanding of production diseases at the clinical, subclinical, biochemical and molecular levels, the incidence rates of these diseases in many well-managed herds remain similar to those published decades ago. From the experience of University College Dublin’s Dairy Herd Health Group, incidence rates remain at an unacceptably high level in many dairy herds. This observation is consistent with the findings of Kelton et al. (1998) who reported increasing incidence rates for clinical hypocalcaemia (milk fever), retained placenta, metritis, ketosis, abomasal displacement, cystic ovarian disease, lameness and mastitis from 1979 to 1995.

While many would conclude that this increase in the incidence rate of production diseases could be largely attributed to the increase in milk yields of dairy cattle over the same period, the relationship between milk yield per se and production diseases is complex. Ingvarsten et al. (2003) found that with the exception of cystic ovarian disease, mastitis and lameness, higher yielding cows do not have an increased risk of production diseases (including milk fever, ketosis, abomasal displacement, metritis and retained placenta). The authors make the point that for some production diseases, such as displacement of the abomasum, the pathogenesis is not directly related to milk yield per se, but to other variables, such as feed intake and feeding errors.

For other production diseases at the herd level, such as retained placenta, there is no direct association with milk yield. However, in considering the complex relationship between milk yield and production diseases, it is important to remember that production diseases are more likely to be caused by ‘imbalances’ in ‘inputs’ and ‘outputs’ or ‘throughput’ rather than just output, i.e. milk yield alone. Ingvarsten et al. (2003) developed this point by stating that no relationship exists between energy balance and milk yield in early lactation, but rather energy balance in early lactation is a function of both feed intake and milk yield. Thus the hypothesis that high yielding cows automatically have higher levels or production diseases is likely to be as false as the hypothesis that lower yielding cows suffer from lower levels of production diseases. In other words, a cow of relatively low milk yield may be more likely to suffer from a specific production disease if exposed to relevant nutritional or environmental stressors (inadequate feed intake) than a cow of high milk yield potential not exposed to the same stressor (adequate feed intake).

The transition cow

Grummer (1995) described the transition period as the period from 3 weeks pre-calving until 3 weeks post-calving. Although some authors define the transition period slightly differently, the definition of this period has been widely accepted (Drackley, 1999). The importance of the transition period has been highlighted in several review articles (Grummer, 1995; Drackley, 1999; Ingvarsten et al., 2003). The period is characterised by marked changes in the endocrine status of the animal that are much more dramatic than at any other time in the lactation–gestation cycle, and a reduction in feed intake when nutrient demand for the developing conceptus and the impending lactogenesis are increasing (Grummer, 1995). The initiation of milk production has the direct effect of increasing calcium output from the cow at a time when input of calcium cannot be increased in the short-term in many dairy cattle, while the reduced feed intake at this time creates an imbalance of energy yielding inputs relative to energy outputs.

Transition dairy cows also experience immunosuppression in the periparturient period and often have to cope with sudden dietary changes that cause digestive disturbances. In addition to the metabolic, endocrine, and immune system perturbances experienced by transition dairy cows, they are also likely to experience environmental stressors arising from the normal group changes that are associated with dairy farm 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 the period immediately after parturition.

Ingvarsten et al. (2003) summarised data from 93,000 first parity and 58,000 third parity Danish dairy cows which demonstrated that the highest incidence of total disease (mastitis, ketosis, digestive disorders, and laminitis) occurred in a period from the day of calving until 10 days post-calving. Many nutritional and management strategies of the pre-calving cow have been reported to alter the degree of negative energy balance, hypocalcaemia, immunosuppression and digestive disorders experienced by the transition cow. Thus, alterations in the nutrition and management of dairy cows during the transition period have an enormous capacity to alter the health status, fertility and productivity of dairy cattle and are ultimately key determinants of dairy cow welfare and producer profitability.
The importance of production diseases of the transition cow

Individual production diseases of the transition cow are best not considered in isolation. Ketosis, fatty liver, clinical hypocalcaemia, retained placenta, metritis, and displacement of the abomasum are all aetologically inter-related. For example, over-conditioned dry cows are more likely to suffer from ketosis and fatty liver, both of which may suppress immunity directly or through an excessive negative energy balance route (Ingvartsen et al., 2003).

Immunosuppression is thought to be the main cause of retained placenta (Le Blanc, 2008). Over-conditioned dry cows are also more likely to suffer from hypocalcaemia, which exacerbates immunosuppression and may cause dystocia and retained placenta (Houe et al., 2001). Thus, ketosis and milk fever are both related to each other and to retained placenta via more than one aetiological pathway. Because of these inter-relationships, production diseases of the transition cow regularly result in cascade effects that increase the incidence of infectious disease or other production diseases, reduce fertility, reduce milk production and increase lameness. Therefore, prevention of production diseases has consequences for dairy cow welfare and producer profitability long after the transition period ends.

Some examples of these relationships include the observations that over-conditioned dry cows are four times more likely to experience milk fever (Houe et al., 2001); dairy cows with milk fever are eight times more likely to suffer from mastitis in the following lactation (Curtis et al., 1983); dairy cows in negative energy balance (NEB) in the pre-calving period are more likely to develop displacement of the abomasum in the following lactation (Le Blanc et al., 2005a,b); those that have excessive NEB after calving or milk fever have reduced fertility performance (Borsberry and Dobson, 1989; Buckley et al., 2003) and those with ruminal acidosis are likely to suffer from immunosuppression, excessive NEB and laminitis (Enemark, 2008).

Recent advances in production diseases of the transition cow

Fatty liver and energy balance

The mobilisation of fatty acids from adipose tissue to support the nutrient demands of lactation is a natural biological phenomenon that occurs in most lactating mammals orchestrated by the endocrine system. However, increasing concentrations of non-esterified fatty acids (NEFA) in the blood of the dairy cow result in the accumulation of triglycerides in hepatocytes and the impairment of liver function. This fatty infiltration of the liver is particularly significant for dairy cows as approximately 85% of glucose for metabolism is derived from the liver, which also plays a central role in feed intake regulation, fertility and immunity.

It has recently been shown that increasing the energy density of transition period diets by increasing the non-fibrous carbohydrate or fat content had no significant effect on liver triglyceride accumulation (Grummer, 2008). It may in fact be advantageous to maintain a highly fibrous or low energy diet beginning at drying-off throughout the dry period (Dann et al., 2005). Of the feed supplements that are available, it appears that propylene glycol and rumen-protected choline are useful in preventing fatty liver. Propylene glycol prevents lipolysis, while choline facilitates the export of fatty acids from the liver as very-low-density-lipoproteins. Because of their different modes of action, both may act synergistically to form a very effective supplementation strategy (Grummer, 2008).

It is interesting that cow management (group changes, diet changes, etc.) has been implicated as a factor that may potentially be more important than nutrition in the development of fatty liver. One such management strategy that has proven useful in reducing the severity of NEB and triglyceride accumulation in the liver is shortening or eliminating the dry period. While nutrition and management during the transition period are obviously relevant to the development of fatty liver, late lactation feeding and historical energy status are also important, as cows that are over-conditioned at drying-off mobilise more adipose tissue pre-calving than thinner herd mates (Kim and Suh, 2003).

Clinical and subclinical hypocalcaemia

Both clinical and subclinical hypocalcaemia act as classical 'gateway diseases', as the presence of either subclinical or clinical hypocalcaemia is linked with many other transition cow disorders. The list of conditions that have been linked with hypocalcaemia includes dystocia, uterine prolapse, retained placenta, endometritis, infertility, mastitis, displaced abomasum, ketosis and immunosuppression (Houe et al., 2001; Ducusin et al., 2003). The typical incidence rates for clinical hypocalcaemia vary between 3.5% and 7% (De Garis and Lean, 2008). However, it has recently been reported that incidence rates of subclinical hypocalcaemia as high as 33% are associated with incidence rates of milk fever of the order of 5% (Roche, 2003). Similarly, approximately 50% of older cows may fall into the subclinical hypocalcaemia category (Goff, 2008), which is consistent with an increased risk of milk fever of the order of 9% as lactation number increases (De Garis and Lean, 2008). In field circumstances, the Dairy Herd Health Group at UCD have encountered several recent cases where the incidence of milk fever ranged from 30% to 75% in Irish dairy herds.

In the context of milk fever prevention, current reviews place strong emphasis on the role of dietary cation anion difference (DCAD). De Garis and Lean (2008) present data from a recent meta-analysis, which indicate that the effect of DCAD on milk fever incidence is a linear one. This implies that reducing DCAD will decrease the risk of milk fever even if the accepted specific thresholds for milk fever prevention after dietary acidification (such as targeted transition cow urine pH or dietary DCAD) have not been met. Goff (2008) suggests that in field situations diets may be ini-
tially formulated to contain 0.5% less chloride (Cl) than potassium (K) in order to achieve a suitable DCAD, with further modification possibly necessary to bring dietary Cl to within 0.4% or 0.3% of dietary K.

It is very clear that hypomagnesaemia is a significant risk factor for the development of hypocalcaemia and magnesium (Mg) concentration of the diet has been cited as the most important dietary factor affecting milk fever occurrence (Lean et al., 2006). Apart from dietary Mg concentration, the levels of both dietary calcium (Ca) and phosphorus (P) have been reported to have an effect on milk fever incidence. The peak of milk fever risk has been associated with a dietary Ca concentration between 1.1% and 1.3% of dry matter (DM), while higher than required concentrations of dietary P have also been reported to increase milk fever risk.

De Garis and Lean (2008) reported that the protective effect of high Ca diets on milk fever occurrence may be due to the counteracting of a hypercalcuric state after prolonged exposure to DCAD diets pre-calving. In their review, De Garis and Lean (2008) revealed that increasing the period of exposure to pre-calving diets increased milk fever risk. While a protective effect of low Ca pre-calving diets is well established for milk fever prevention, it may be necessary to use Ca binders to induce a pre-calving hypocalcaemia necessary to prevent milk fever (Goff, 2008).

**Subacute ruminal acidosis (SARA)**

SARA has been reported to be prevalent in 19% of early lactation and 26% of mid-lactation dairy cows in the USA. Furthermore, recent Australian and Irish data indicate that between 10% and 15% of dairy cows grazing perennial ryegrass-based pastures have the condition (Bramley et al., 2005; O’Grady et al., 2008). SARA has been implicated in the aetiology of laminitis (Enemark et al., 2002; Oetzel, 2000), reduced and erratic feed intake, low body condition score in lactating cows (Oetzel, 2000), low milk fat syndrome, caudal vena caval syndrome, abomasal displacement/ulceration (Olson, 1991), ruminitis (Enemark, 2008), immunosuppression (Kleen et al., 2003) and inflammation (Plaizier et al., 2008). Early lactation cows and cows at peak DM intake are most at risk from SARA: the early lactation cows are at higher risk due to reduced absorptive capacity of the rumen, poorly adapted rumen microflora, and the rapid introduction to high-energy dense diets (Dirksen et al., 1985), while cows at peak DM intake are at increased risk due to the greater amount of acids produced in the rumen (Oetzel, 2005).

Recent research has contributed significantly to our understanding of the condition. For instance, the frequently accepted view that a reduction in feed intake or cyclical feeding patterns are indicative of SARA has been questioned by Plaizier et al. (2008), who conclusively demonstrated that reduced feed intake does not accompany all reductions in rumen pH, consistent with SARA. The authors present the hypothesis that the reduction in feed intake that is associated with SARA may be due to inflammatory changes occurring in various organs of the cow, rather than any effect on fibre digestion in the rumen. This hypothesis is well developed by Plaizier et al. (2008) and makes a significant contribution to our understanding of SARA. They observed that SARA results in increased concentrations of the acute phase proteins (APPs) serum amyloid-A and haptoglobin in blood. However, while grain-induced SARA has reportedly increased the concentrations of these APPs in blood, SARA induced by reducing fibre particle size did not. Whereas the acute inflammation may be caused by Gram-negative bacterial lipopolysaccharide endotoxin produced in the rumen (and or large intestine), and translocated to the liver via portal blood, other pathogenic factors produced by bacteria such as *Escherichia coli* and other *Enterobacteriaceae* may be involved in this inflammatory process.

Enemark (2008) describes a strong physiological association between SARA and immunosuppression. The metabolic acidosis that results from SARA may result in reduced glucose-dependent insulin secretion, increased cortisol secretion, reduced phagocytic activity and migratory speed of neutrophils (Enemark, 2008).

In the field, the diagnosis of SARA remains problematical due to a combination of factors, including questions over the validity of rumen pH measurements, the occurrence of clinical signs after a SARA episode, various clinical manifestations of SARA, and the overlap of the clinical signs with many other phenomena. However, a rumenocentesis technique has been well developed in the USA and significant work has been done on the appropriate interpretation of herd-level data. Other advances in the diagnosis of SARA include developments in technology leading to the use of in-dwelling rumen pH probes, the use of rumen valerate and urinary net acid base excretion (Enemark, 2008). However, it is likely that diagnosis of SARA at herd-level will continue to be based on a thorough herd investigation that takes cognisance of the inherent diagnostic difficulties, together with the fact that many of the associated clinical signs occur much later than the SARA episode itself. The negative and irreversible consequences of SARA on cow welfare and producer profitability mean that prevention will remain a priority in addressing this production disease.

**Oxidative stress, antioxidants and immunity**

One of the reasons that infectious diseases such as mastitis may be associated with a poorly managed transition period is that the dairy cow experiences a substantial periparturient immunosuppression. The severity of this immunosuppression is exacerbated by factors such as NEE (Ohtsuka et al., 2006), hypocalcaemia (Ducusin et al., 2003) and increased circulating levels of cortisol for prolonged periods around calving (Burton et al., 2005). In addition, it has been established that dairy cattle subjected to the demands of late pregnancy, parturition or peak lac-
ivation may be subjected to oxidative stress or the production of reactive oxygen metabolites (Spears and Weiss, 2008). Immune cells are sensitive to oxidative stress as their membranes contain high concentrations of polyunsaturated fatty acids that are vulnerable to lipid peroxidation and they produce large quantities of reactive oxygen metabolites when stimulated (Spears and Weiss, 2008).

Several trace elements and vitamins are potentially useful in maintaining an appropriate balance of antioxidants in the dairy cow to cope with the increased production of reactive oxygen metabolites around parturition. The list of potentially useful trace elements that function as or are key components of antioxidants includes copper (Cu), selenium (Se), zinc (Zn) and chromium (Cr). In addition, vitamin-E and β-carotene have useful antioxidant properties.

There is a substantial amount of research data, which indicate beneficial effects of these trace elements and vitamins on mastitis and retained placenta incidence in periparturient dairy cows. For example Cr supplementation during the last 9 weeks of pregnancy significantly reduced the incidence of retained placenta (Villalobos et al., 1997); heifers fed supplemental Cu from 60 days pre-partum to 42 days post-partum had lower E. coli and somatic cell counts in milk, lower clinical scores and lower rectal temperatures than controls after an intramammary challenge of E. coli at 34 days in milk (Scaletti et al., 2003). The effects of vitamin-E on the incidence of mastitis and retained placenta have been demonstrated by Weiss et al. (1997) and Miller et al. (1993). However, as is also the case with β-carotene, responses are probably dependent on pre-supplementation vitamin-E or β-carotene/vitamin-A status. In particular, plasma concentrations of vitamin-E and α-tocopherol decrease during the dry-period in cows fed stored forages including hays, alfalfa hay and corn silage (Spears and Weiss, 2008).

For the trace elements, the form of supplementation may have a large effect on the amount of the supplements absorbed from the gastro-intestinal tract (GIT) (Andrieu, 2008). It has been suggested that by using chelation technology to produce an electrically neutral form of trace element supplement, absorption may be enhanced. The negative effects of inter-mineral interactions in the (GIT) are avoided, as are the negative interactions encountered by negatively charged mineral ions on the reaching the unstirred water layer and the negatively charged mucus layer on small intestinal villi (Andrieu, 2008). There are numerous reports of enhanced health status via reductions in somatic cell counts in dairy cattle following supplementation with organic ‘chelated’ trace element supplements in comparison to inorganic ‘non-chelated’ trace element supplements.

**Parturition and uterine health**

Dystocia is associated with acute pain and is one of the most significant welfare issues in dairy cow production. Production systems that have resulted in breeding larger frame dairy cattle, inbreeding, inappropriate calving assistance, stresses at parturition and an increased tendency to breeding heifers at young ages, have resulted in this condition being more common in domesticated cattle rather than their feral counterparts (Mee, 2008). Furthermore, as more and more attention is being placed on managing dairy cows at herd level, with the emphasis on subclinical conditions and suboptimal performance, individual cows may receive less attention, as in cases referred to as the ‘lost in the herd syndrome’ or ‘loser cow’. In addition, the benchmarking of farmers against their peers in terms of husbandry hours per cow per year has the potential to reduce the necessary husbandry involved in the care of the individual at parturition. For primiparous cows, feto-pelvic disproportion is the predominating risk factor, while for multiparous cows fetal malposition is the most important risk factor (Mee, 2008). It is likely that the use of selection indices to include calving ease and continued farmer education offer the best approaches to the problem of dystocia.

Retained placenta and uterine infection after parturition are both manifestations of a reduced immunity in the periparturient period. It is interesting that retained placenta has been linked to elevated NEFA levels and reduced feed intake in the pre-partum period and that the most appropriate preventative strategies are those associated with good periparturient health and immunity (Le Blanc, 2008). In terms of dealing with uterine infection, the differentiation between cows with normal expulsion of lochia in the early postpartum period and a metritis cow with elevated rectal temperature, reduced feed intake, history of retained placenta and dullness is paramount. For dairy herds with high levels of clinical or subclinical endometritis, it is unlikely that any substantial benefit can be obtained from identification of affected cows and treatment with antibiotics over and above the use of two injections of prostaglandin (PG)F₂ at approximately 35 and 49 days post-partum (Le Blanc, 2008).

It is apparent that the quality of research data, as well as field diagnosis and treatment, has been hampered by inconsistent definitions of uterine disease. Sheldon et al. (2008) have proposed a system of definition that should bring uniformity and consistency to this issue. Examination of the vaginal mucus and a consistent scoring system will allow veterinarians in the field to differentiate between the various forms of uterine infection and facilitate optimal strategies for treatment and prevention. The main consequence of uterine infections and retained placenta is poor reproductive performance, which is a key contributor to reduced profitability for most dairy producers internationally.

**Conclusions**

Production diseases of the dairy cow continue to cause economic loss to the dairy industry and animal welfare concerns. The challenges facing us in the rapidly changing
international agricultural industry of the 21st century are to prevent disease, enhance animal welfare and farmer profitability while taking cognisance of food safety issues, the consumer and the environment. The former agricultural policy of the European Union that was primarily concerned with increasing production has been replaced by a European rural development policy that focuses on agri-competitiveness, animal welfare, food quality and safety and environmental sustainability.

Although there will always be more to learn about individual diseases of production, a significant body of knowledge already exists which should allow us to prevent these diseases. Furthermore, while a reductionist approach concentrating on the pathophysiology of individual diseases of production has been essential to the creation of the sophisticated knowledge of these diseases. In the complex milieu of the modern dairy farm, veterinarians are rarely presented with uncomplicated herd-level problems or conditions such as hypocalcaemia. Thus, cows in NEB are more likely to develop clinical ketosis, mastitis, retained fetal membranes and left displaced abomasum with obvious implications for herd health and welfare, milk production and fertility. It is the complex interaction of these diseases, their relationship with nutritional strategy and housing environment, their ability to impact on the expression of infectious disease such as mastitis and the fundamental influence of social and attitudinal factors that make prevention and control of these diseases such a challenge.

One of the important challenges facing the industry is the development, refinement, implementation and economic assessment of practical, integrated, blueprints of best practice for prevention of the production diseases and other diseases of the dairy cow. Ideally based upon principles of risk management, these blue-prints will assist farmers in providing reassurance regarding the health status of the farming enterprise, thus increasing trust, transparency and acceptability on issues of animal health and welfare to the dairy industry, retailers and consumers. This is particularly important in the context of consumer concerns about food safety and animal welfare and the rapidly changing international agricultural environment, with a requirement for cross-compliance and an increasing emphasis on profitability with sustainability as opposed to increased production per se. The Dairy Herd Health Group at UCD has recently proposed a multidisciplinary plan for the management of production diseases of the transition cow (Mulligan et al., 2006) involving the farmer, the veterinarian, and other farm advisors.

The challenges facing us have clear implications for research in bovine health management, as there will be an increasing need to engage social science methodologies such as action research, as well as behavioural economics, which will facilitate evaluation of the economic benefits of consumer perceptions of novel preventive approaches as well as conventional cost-benefit analyses. Action research is being employed in agricultural developmental research in many parts of the world and involves the use of the knowledge and experience of farmers, their agricultural advisors and veterinarians in the various stages of the research process from problem identification, design and application of projects to implementation of results. Disease prevention, in its broadest sense is no longer the sole preserve of veterinarians and rising to this challenge will require the adoption of a multidisciplinary team approach involving not only the farmer, the veterinarian and the farmer’s advisors, but also nutritional and animal breeding consultants (Mulligan et al., 2006; Le Blanc et al., 2005a,b). Allied to this will be the need to place significant emphasis on dissemination of knowledge, training, motivation and the encouragement of fundamental attitudinal changes to disease prevention within the industry.

Animal health policy in the European Union now emphasises the need for a preventative approach to animal disease and the European Commission White Paper on Food Safety emphasises the need for ‘an integrated farm-to-table approach’ with a requirement for risk management as its foundation. It underlines the importance of integrating animal health and welfare with food policy as well as the need for sustainable agriculture where the emphasis will be on achieving profitability in the context of optimal animal health, welfare and food safety along with increased environmental awareness. The development and implementation of practical on-farm risk management programmes addressing production diseases and other disease of dairy cows will allow us to face these challenges.

Conflict of interest statement

Neither of the authors (F.J. Mulligan and M.L. Doherty) has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the paper entitled Production diseases of the transition cow.

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