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Investigating premature lactation in pregnant dairy females

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BACKGROUND

A veterinarian in charge of animal health for 766 heifers transported from Western Australia to Mexico, reported a prevalence of 11% of heifers with a distension of the mammary gland. He reported an incidence of distension of the gland of 17% of heifers during the voyage, with a higher prevalence of distension in more heavily pregnant animals (there was 14 times the prevalence in heifers 5 months pregnant than 2 months pregnant). Of the 766 heifers, 8 had clinical signs consistent with clinical mastitis and 6 expressed milk. A small number of heifers appeared to reduce distension following dietary change from pellets to chaff and redeveloped distension following the reintroduction of the pellet diet (McCarthy pers comm. – Appendix 1).

EXECUTIVE SUMMARY

- Mastitis does occur in heifers that are unbred and in pregnant heifers – the prevalence estimates vary from 8 to 90%, with a moderate rate of infection (20 to 30% being reported relatively consistently). These infections are usually subclinical and are associated with coagulase negative staphylococci (CNS), however, major mastitis pathogens such as *Staphylococcus aureus* and *Streptococcus agalactiae* have been identified.

- Risk factors for mastitis especially relevant to the shipping situation include, crowding, environmental contamination of the udder, exposure to dietary change and fly worry.

- Clinical mastitis in heifers is most frequently described as the 'summer mastitis' complex. This clinical syndrome involving flies, environmental contamination and multiple organism infection with *Archanobacterium pyogenes* and anaerobic gram negative organisms is extremely difficult to treat and results in a high prevalence of blind quarters as detailed in Mexico (McCarthy pers comm.).

- Heifers should be treated for mastitis with systemic therapies rather than intramammary therapies. Specific recommendations are given in Figure 1.

- Udder oedema, the accumulation of fluid in the mammary region is more specifically related to the immediate pre-calving period than the first 2-6 months of gestation and is unlikely to be a cause of the observed problems. However, concentrations of salt and potassium in the diet should be tightly controlled to reduce the risk of oedema contributing to the clinical syndrome observed.

- Oestrogen is the dominant hormone in mammary development, however, many other orchestrated hormonal changes occur and progesterone also has a very substantial role in development. These contentions are supported by the potential to induce lactation in non-pregnant heifers using these hormones.

- Corticosteroids play little role, identified to date, in premature mammary development. Consequently, stress associated with transport, shipping and group assembly is unlikely to play a major role in causing the mammary distension seen.

- Exogenous sources of oestrogen in feed are well-reported as a cause of premature udder development. Many of these reports have been from Western Australia, but the syndrome of 'clover udder' is well recognised in other regions. This syndrome can also be associated with ingestion of damaged lucerne (personal observation).

- Figure 1 shows a flow diagram to differentiate between the different causes of udder distension in heifers and appropriate diagnostic approaches and responses for veterinarians in charge.

- It appears likely that feed factors are leading to premature development of the mammary gland. This is increasing the risk of bacterial penetration of the teat end under circumstances where serious environmental contamination of the udder is inevitable.

- A series of recommendations are given (in Recommendations Page 15) that should minimise the risk of this problem.

1. PREPARTUM MASTITIS IN HEIFERS

1.1. Introduction

Heifer mastitis can be detected either pre- or postpartum (Trinidad et al. 1990a) and is most often subclinical (Daniel et al. 1986). Intramammary infection (IMI) in breeding age and pregnant heifers are much higher than previously thought. Many of these infections can persist for long periods of time, are associated with elevated somatic cell counts (Hallberg et al. 1995; Trinidad et al. 1990b), and may impair mammary development (Trinidad et al. 1990b) and affect milk production after calving. Immune function during the periparturient period is impaired (Kehrli et al. 1989), and the mammary gland is susceptible to mastitis during transition. Until recently, little attention has focused on methods of controlling mastitis in heifers. Consequently, there are few management practices for controlling mastitis in heifers extensively supported by research data. Intramammary infections at parturition, either clinical or subclinical, will lead to considerable losses for the farmer, as observed in Mexico. The prevalence of mastitis in heifers and strategies for control are discussed.

1.2. Prevalence of intramammary infections in heifers

A high percentage of pregnant heifer mammary glands are infected during late gestation, at calving and during early lactation, but these infections are associated with elevated somatic cell counts, that is subclinical infection, in most cases (Oliver, 2000). Munch-Peterson (1970) found that 22% of all quarters in heifers were already infected by the first day of lactation. Trinidad et al. (1990c,d) found that up to 90% of heifers had quarters infected before parturition, while other researchers in the USA and Europe (Munch-Petrson, 1970; Meaney, 1981; Oliver and Mitchell, 1983; Pankey et al. 1991; and Mathews et al. 1992) claimed that the IMI rate in heifers was moderate (13 to 39%).

The prevalence of IMI in unbred heifers and heifers during different stages of pregnancy may also be high (Trinidad et al. 1990a). Unbred heifers can have a higher percentage (86.7%) of infected quarters compared with the overall mean for pregnant heifers (70%).

Staphylococcus (*S.*) species were observed frequently and 8 different species were isolated; the three most common species isolated from unbred and pregnant heifer mammary glands *S. Chromogenes*, *S. hyicus* and *S. aureus*. *Coagulase-negative staphylococci* (CNS) accounted for 67.4% of bacteria isolated (Oliver and Mitchell, 1983; Boddie et al. 1987; Trinidad et al. 1990a,b; Pankey et al. 1991; Shearer and Harmon, 1993; Fox et al. 1995; Nickerson et al. 1995). The prevalence of teat canal and teat skin colonisation in unbred and primigravid heifers, with either CNS or *S. aureus*, has been reported to be equally high (Boddie et al. 1987; White et al. 1989; Trinidad et al. 1990c,d; Roberson et al. 1994). Location, herd, season, and trimester of pregnancy all significantly influenced prevalence of IMI in heifers. Heifers in the third trimester of pregnancy had the highest prevalence of IMI. Thus, based on all studies reported so far, CNS will likely cause the majority of IMI in unbred and pregnant heifers and variation in the prevalence of CNS IMI in heifers should be expected among herds.

Studies of Oliver et al. (1987, 1992, 1997) found that 8 to 10% of heifer mammary glands were infected with major mastitis pathogens near calving. Infections with major mastitis pathogens were caused by environmental contaminants, primarily *Streptococcus* species, a pattern which was consistent with IMI in lactating cows in these herds (Oliver et al. 1990). Conversely, other studies (Fox et al. 1995; Trinidad et al. 1990a) indicated that *S. aureus* was the most prevalent major mastitis pathogen isolated from unbred and pregnant heifer mammary glands. Differences in the incidence of IMI and types of bacteria causing IMI in pregnant heifers are likely due to the prevalence of mastitis pathogens in the herds evaluated. Thus, it is reasonable to assume that heifers from herds with a high prevalence of contagious mastitis will likely be infected predominantly by contagious mastitis pathogens such as *S. agalactiae* and *S. aureus*. Similarly, environmental mastitis pathogens such as *S. uberis* and gram-negative bacteria such as *Escherichia coli* will likely

be the predominant major pathogens isolated from heifer mammary glands from herds with an environmental mastitis problem. The percentage of infected quarters with environmental *Streptococci* at any one point in time is generally low and seldom exceeds 10% of quarters.

Mammary secretion from infected mammary glands had significantly higher somatic cell counts than secretions from uninfected quarters (Oliver, 2000). Also tissues from mammary glands of unbred heifers infected with CNS had more leukocyte infiltration and increased connective tissue compared with tissue from uninfected mammary glands (Trinidad et al. 1990b). Thus, infection of heifer mammary glands by mastitis pathogens may occur at a very early age and some of these infections may impair mammary growth and development and influence future milk production.

1.3. Summer Mastitis

Summer mastitis is a severe form of clinical mastitis in heifers. It is associated with the presence of *Archanobacterium pyogenes* and anaerobic gram negative bacteria. Mammary glands are asymmetrically distended and often hot and painful, and the mastitis is characterised by thick, yellow-green, putrid-smelling pus that can be extracted from the glands of some heifers (Lean et al 1987; Radostits et al. 1996). The incidence of summer mastitis has been reported to be as high as 10% of some groups, but lower rates of 2% are more typical (Berry, 1998).

1.4. Risk Factors

Most cases of clinical mastitis occur postpartum, and the most important predisposing factors such as improper milking and hygiene are well known. Only a few studies have focused on risk factors for clinical mastitis in heifers, and those studies included prepartum as well as postpartum cases (Myllys and Rautala, 1995; Waage et al. 1998). Following are the risk factors that may contribute to occurrence of prepartum mastitis in heifers:

1. An increase in the mean milk yield per cow in the herd, an increase in the overall herd incidence of clinical mastitis, and a decrease in bulk milk somatic cell count (SCC) have been associated with increased risk of clinical mastitis in prepartum heifers (Myllys and Rautala, 1995).

2. Season: heifers calving late in spring or during summer are at higher risk for clinical mastitis than those calving at other times of the year. Schultze (1985) also reported that incidence of IMI before parturition has been considerably higher during warm than during cool weather. During summer, mastitis caused by *Archanobacterium pyogenes*, often together with some other bacteria (Hillerton et al. 1987), is prevalent in heifers and appears to be closely related to the activity of the fly *Hydrotaea irritants* (Madsen et al. 1992). Summer mastitis has been observed in closely housed heifers in the presence of other biting flies (Lean et al. 1987).

3. There is a significant association between the risk of mastitis in heifers and the total incidence rate for clinical mastitis in the corresponding herd (Waage et al. 1998).

4. Heifers in herds that are kept on pasture are at lower risk for clinical mastitis than heifers in non-grazing herds (Bendixen et al. 1986; Waage et al. 1998).

5. Type of ration and proportion of hay to silage in the diet is also considered a risk for clinical mastitis in heifers (Waage et al. 1998).

6. Milk leakage before calving has been associated with an increased risk for mastitis.

7. An increase in age at first calving is associated with an increased risk for clinical mastitis in heifers (Erb et al. 1985; Waage et al. 1998). However, this relationship is not linear as heifers calving at an extraordinarily high age are less at risk for clinical mastitis. (Waage et al. 1998)

8. Heifers in the third trimester of pregnancy had the highest prevalence of IMI. (Fox et al. 1995)

9. Udder oedema can be a significant risk factor for clinical mastitis in prepartum heifers. (Waage et al. 2001).

10. Confinement: heifers during the prepartum period may pick up IMI from dry cows under confinement conditions (Waage et al. 2001; Schukken et al. 1990; Jones and Bailey, 1998).

11. Genetic effects on prevalence of mastitis in heifers have been estimated to be small relative to the environmental effect (Shook, 1993).

1.5. Control and treatment

There has been little research focused on management strategies for controlling mastitis in heifers. Evidence suggests that IMI in pregnant heifers occurs frequently and that some infections are detrimental to mammary gland development and subsequent lactational performance (Oliver, 2000). In general, bacteria are highly susceptible to all antibiotics evaluated, however, some variability to antimicrobial susceptibility of bacteria may be observed among herds (Oliver, 2000). The following strategies can be used for controlling mastitis in unbred or pregnant heifers;

1. Visually examine udders whenever working with heifers. Look at the condition of teats, especially for scabs from fly bites, check for swollen or hard quarters and examine mammary fluid. It should appear honey-like. Watery flakes or clots are signs of mastitis infections.

2. Prepartum intramammary antibiotic infusion of heifer mammary glands a few weeks before calving is an effective procedure for eliminating many infections in heifers during late gestation and for reducing the prevalence of mastitis in heifers, both during early lactation and throughout lactation. It can be economically beneficial to treat heifers with antibiotic infusions before calving, because treated heifers will produce more milk and have less mastitis throughout lactation (Oliver, 2000). Nickerson et al. (1995) also reported that a one-time infusion of antibiotic into infected quarters 45 days precalving reduced the incidence of IMI by 59% at calving compared with the pretreatment level. Prophylactic treatment of uninfected quarters 45 days prepartum reduced the rate of IMI by 93%. The mean SCC was 50% lower at calving for treated heifers, and milk yield over the first 2 months of lactation was 10% greater than that of untreated heifers. Collectively, the treatment of heifers prepartum with a dry cow product (60 to 40 days before calving) or lactating cow therapy (14 to 7 day before calving) are effective and the cure rate will be higher than during lactation (Trinidad et al. 1990c,d).

3. One disadvantage of prepartum antibiotic administration for controlling mastitis in heifers is the potential for antibiotic residues in milk. This is especially important if heifers calve

sooner than expected. Residue testing should be carried out before the milk from treated heifers postpartum is mixed with herd milk.

4. Flies can carry a number of mastitis-causing organisms that can colonise teat lesions. Eliminating fly breeding sites is one aspect of fly control. Heifers from herds using fly control have a lower prevalence of IMI than herds without fly control (Yeomen and Warren, 1984; Jones and Bailey, 1998).

5. Environment: control programs for pregnant heifers pre-calving should be designed to reduce the number of mastitis causing bacteria at the teat end. Well drained paddocks are preferred for late pregnant heifers, because muddy areas increase the risk of mastitis.

6. Selenium and vitamin E: The risk of mastitis in properly supplemented heifers is lower (Weiss et al. 1997). Supplementing heifers 60 days pre-calving with 0.3 pmm of selenium and 800 to 1000 IU/d vitamin E, and another injection of 50 mg selenium 3 weeks before calving will reduce the risk of IMI significantly.

The future of the dairy herd depends upon mimimising mastitis infections, including prevention of infections in heifers. Once udders are infected in heifers before calving, these will not respond easily to antibiotic treatment during lactation. Since most mastitis infections pre-calving are caused by bacteria, treatment of heifers with intramammary infusion products formulated for treating cows at drying off can be successful. **Systemic administration of antibiotics would be more advantageous as this avoids the difficulties associated with restraining the heifers and administering product via the teat canal.**

2. PREPARTUM UDDER OEDEMA

2.1. Introduction

Udder oedema is one of the most common causes of enlarged mammary gland and results from excessive accumulation of intercellular fluid in the mammary gland. Clinically, cattle have a symmetric, hardening and swelling of the mammary gland. The distension can extend anterior to the udder and rarely down the escutcheon and hind legs. Development of at least some mammary oedema is associated with pregnancy and parturition especially in late primigravid heifers (Schmidt, 1971; Malven et al. 1983). The primary importance of this condition is the need to differentiate it from the pathologic oedema associated with IMI. An udder swollen with lymphatic fluid loses its flexibility, hence, it is more susceptible to physical damage or mastitis and an extra management burden is required to care for the oedematous animals.

2.2. Risk factors

1. Older heifers at calving and gestation length are more likely to have prepartum oedema (Malven et al. 1983; Hayes and Albright, 1966, 1976).

2. The calf birth weight in heifers is negatively related to prepartum oedema (Malven et al. 1983).

3. An inherited trait may contribute to the occurrence of udder oedema, especially in Jerseys (Lema et al. 1992; Al-ani et al. 1985).

4. Overfeeding grain (only one study; Emery et al. 1969).

5. Excessive salt in dry cow rations (Nestor et al. 1988).

6. High potassium forages: grass hays and pastures can contain high level of potassium, especially if they have had manure applied to them (Randall et al. 1974; Nestor et al. 1988).

7. Endogenous concentrations of reproductive hormone prepartum; such as oestradiol-17-α and oestrone that are positively associated with the prevalence of udder oedema (Malven et al. 1983).

2.3. Control and Treatment

1. Limiting salt in the diet to reduce the risk of overfeeding salt mixed with other feed.

2. Limiting potassium in the diet, especially if udder oedema is a significant problem in a herd.

3. Moderate exercise.

4. Diuretics, primarily thiazide derivatives and furosamide have been used, but their potential benefit may be offset by serious side effects, including dehydration, if used for more than 24h.

5. Corticosteroids have been used in the past, but appear not to alter the clinical course of the disease process.

3. MAMMARY GLAND DEVELOPMENT

3.1. Normal mammary gland development

3.1.2. Introduction

The mammary gland is unusual in that most of its development occurs in the adult, when it undergoes repeated cycles of development, function, and differentiation, involving extensive proliferation and apoptosis of secretory tissue (Knight, 2000). The physiological performance of the adult gland (amount of milk produced) is highly correlated with secretory tissue mass (Linzell, 1966). The mammary gland duct system expands markedly during the prepubertal period and, in cattle, there is evidence that inappropriate nutrition will restrict this process and subsequently cause milk yield to be reduced (Sejrsen, 1994). There are two sets of potential interactions during gestation. The dam (and her environment) can affect foetal mammary gland development, and there is the possibility that the foetus influences maternal mammary gland development. Given that the phenotypic expression of the mammary gland (milk) is for the benefit of the young rather than the mother, it is not surprising that this influence does occur (Collier et al. 1995). Although gestational development is essential to lactational output, it is not a critical window in the classical sense of having an effect that is only manifest some considerable time later.

3.1.2. Isometric growth phase

The basic structures and outer shape of the mammary glands are formed in foetal life, but the epithelial tissue (ducts) is still rudimentary at birth. In the first period after birth, the mammary fad pad grows isometrically (at the same rate as the rest of the body). Very little mammary development occurs during this phase. True lobulo-alveolar development is limited in the period before first pregnancy. This relatively small contribution to the total mammary growth, however, belies the importance of the prepubertal period to mammary development and function, because it is during this phase that the framework for the lobulo-alveolar development of the gland occurs that determines the efficient function of the gland.

3.1.3. Allometric growth phase

Well in advance of the onset of puberty, mammary growth becomes allometric. During this period, the mammary gland undergoes a period of growth and development which is significantly greater than that for body surface area. In this period, there is rapid growth of both the fat pad and the ducts that branch into it. The allometric growth phase is initiated as early as 2 to 3 months of age. At this stage of development the mammary glands of heifers weigh about 2 to 3 kg, of which only 0.5 to 1.0 kg is parenchymal tissue containing the mammary ducts (Sejrsen et al. 1982). The parenchymal tissue usually contains 10 to 20% epithelial cells, 40 to 50% connective tissue and 30 to 40% fat cells. This period is well in advance of puberty, which is variable in onset, generally from 6 to 7 months of age (Sinha and Tucker 1969). In cattle, allometric growth (prepubertal mammary development) is closely linked to the gradual maturation of the ovaries, and early ovariectomy abolishes mammary growth completely (Wallace 1953; Purup et al. 1993). In cattle, this allometric phase of growth is influenced by nutrition and by hormonal manipulation (Cowie et al. 1949).

Following the allometric growth phase, glandular growth decreases and resumes an isometric pattern until conception. During pregnancy, mammary growth is both qualitatively and quantitatively much more extensive than during puberty. In early pregnancy, growth of the mammary ducts continues and from mid pregnancy there is extensive lobulo-alveolar development. This is fastest during the later stages of pregnancy, which coincides with the most rapid period of foetal growth. Extensive lobulo-alveolar development occurs only during pregnancy. The lactating mammary glands can weigh as much as 25kg (Foldager and Sejrsen, 1991), and lactating parenchyma consists of 40-50% epithelial cells (ducts and alveoli), 15-20% lumen, about 40% connective tissue and almost no fat cells (Harrison et al. 1983). In cattle, mammary development is essentially complete at calving. It is therefore likely that factors affecting mammary development in the rearing period also influence the milk yield capacity of the heifers once calved. During the allometric growth phase there is rapid growth of the mammary fat pad and extension of the duct system into the fat pads. This duct development is primarily a result of cellular growth rather than cellular proliferation.

3.1.4. Hormonal control of mammary gland development

Mammary development is a function of hormonal and nutritional inputs and the gland is quite sensitive to hormonal stimuli even shortly after birth, as evidenced by the occasional observation of marked mammary development in neonates in association with high maternal levels of mammogenic hormones at the time of parturition. The key hormones which stimulate mammary growth are oestradiol-17ß, progesterone, prolactin, growth hormone and placental lactogen. Insulin appears to be an important facilitator of growth, while not playing a key stimulatory role in mammary growth. Local hormones and growth factors are also involved including the insulin-like growth factor

I (IGF-I), epidermal growth factor, transforming growth factors, chalones (growth inhibitors) and prostaglandins. These local hormones have been demonstrated to be capable of stimulating or controlling mammary growth *in vivo* and *in vitro*, although the importance of their role in the normal physiology of mammary is still to be defined. A further group of hormones influencing mammary development include the mineralocorticoids and glucocorticoids including aldosterone and cortisol, however the role of these is largely limited to the period immediately before calving. During the allometric growth phase there is rapid growth of the mammary fat pad and extension into of the duct system into the fat pads. This duct development is primarily a result of cellular growth rather than cellular proliferation.

Optimal mammary growth requires both oestrogen and progesterone. Both hormones are elevated during pregnancy, which is why there is no lobulo-alveolar growth during the oestrous cycle, when only one of these hormones is elevated at a time. During pregnancy, the mammary tissue has oestrogen and progesterone receptors. In the cows, progesterone concentrations are elevated throughout gestation, while oestrogen is particularly elevated during the second half of gestation when gland development is accelerated. Together, these result in lobulo-alveolar growth.

Oestrogen and progesterone are released in association with the ovarian cycle - oestrogen being dominant in the follicular and ovulatory phase and progesterone dominating in the luteal phase. Oestrogen acts to increase the number of progesterone receptors and stimulates duct growth, while duct branching and DNA synthesis in the duct ends is promoted by progesterone. Progesterone appears to inhibit the DNA synthetic and cellular mitotic activities observed during oestrogen dominant phases of the oestrus cycle. Lobulo-alveolar development is a function of the combined effects of progesterone and oestrogen. In general, during the post-pubertal period (puberty to conception) oestrogen causes cell multiplication at the tip of the terminal end buds and enlargement of ducts (lengthening and branching of ducts), while progesterone causes duct and ductule cells to multiply to ductule development and duct enlargement or widening (Hurley, 2000). The asynchronous release of these hormones through the post-pubertal period of cyclicity before pregnancy contrasts with the high levels of both hormones present during pregnancy. The protein hormones prolactin, somatotropin, and insulin also play an important role in alveolo-lobular development as laboratory animal and *in vitro* studies show that steroid hormones will not stimulate lobulo-alveolar growth in the absence of the protein hormones (Cowie et al. 1980).

Treatment of heifers aged 8-12 months with exogenous bovine somatotropin will result in an increase in the amount of mammary parenchyma present and in a decrease in the amount of mammary adipose tissue (Sejrsen et al. 1986; Sandles et al. 1987). The action of somatotropin on the growing mammary gland is most probably mediated through the actions of IGF-I. However, increased milk yields associated with this increase in parenchyma have not been consistently demonstrated. Heifers of the same age fed on rations which stimulate insulin release develop glands with excessive levels of mammary adipose and which are associated with reduced future milk yields (Little and Kay 1979; Sejrsen et al. 1983). As a result of these and other observations of impaired lactational performance in heavily fed heifers, it has been suggested that growth rates of prepubertal heifers should not exceed 0.75 kg/day. While low planes of nutrition and low protein diets have been associated with impaired mammary development in some species, extremely low growth rates (0.1 kg/day) were not associated with impaired milk yields or mammary DNA content in cattle. Mammary tissue is not related to concentration of insulin or glucocorticoids. Mammary secretory tissue is negatively correlated with blood serum concentration of prolactin; however, this relationship disappears after adjustment for concentration of growth hormones, insulin and glucocorticoids (Sejrsen et al. 1983). Transforming Growth Factor (TGF) also play a role in mammary gland development. When animals are implanted with TGF-β, mammary gland growth and morphogenesis is inhibited reversibly, suggesting a role as a negative growth regulator (Silberstein and Daniel, 1987). By contrast, TGF-α in combination with insulin, prolactin,

aldosterone, and hydrocortisone promoted lobulo-alveolar development in the mammary gland primed with oestrogen and progesterone.

3.2 Premature mammary gland development

The term premature mammary gland has been assigned to describe the development of the gland in postpubertal heifers that are 4 to 6 months pregnant. In these heifers, mammary gland development is similar to that of cattle pending parturition and there is a fullness of the gland, but no evidence of inflammation in the quarters.

Risk factors: Steroid hormones, for example oestrogen and progesterone, are the main hormones that are involved in controlling the normal development of mammary gland during pregnancy. The action of the steroid hormones is facilitated by the stimulatory effect of some protein hormones. In heifers as puberty approaches, the increased frequency of luteinizing hormones pulses stimulates the development of large dominant ovarian follicles that secrete increased amounts of oestrogen (Bergfeld et al. 1994). The prepubertal allometric growth phase of the mammary gland is caused in a large part by the elevated concentrations of oestrogen (Kinder et al. 1995). Once puberty occurs, secretion of progesterone by the ovaries causes an end to the prepubertal allometric growth phase (Knight and Peaker, 1982).

Steroid hormones have been used in the dairy industry to induce lactation in nulligravid heifers (Smith and Schanbacher 1974) and cows (Smith and Schanbacher 1973). Subcutaneous injections of 17β-oestradiol and progesterone can initiate lactation, in non-pregnant heifers, with 17β-oestradiol-progesterone ratios of 1:2.5, 1:2 and 1:1 when constant 17β-oestradiol was maintained (Smith and Schanbacher 1974). Results of a study by Lammers et al. (1999) showed that oestrogen implants accelerated mammary gland development in prepubertal heifers (1000 g/day).

Melengestrol acetate has progesterone-like activity; however, it promotes persistent ovarian follicles and increases secretion of oestrogen from the ovaries (Kojima et al. 1995). When heifers, from 2.5 months of age until breeding size, were fed a diet with and without melengestrol acetate, total mammary gland DNA increased by 60% in heifers at breeding size with melengestrol acetate (Pritchard et al. 1972). It was also shown (Pritchard et al. 1972) that a high feeding level reduced mammary development at first oestrus in untreated heifers, whereas feeding level did not affect mammary development in heifers fed melengestrol acetate.

There is some evidence that an elevated blood concentration of oestrogen is the main risk factor for premature udder development in non-pregnant heifers (Vestweber et al. 1986, Shore et al. 1988; Thain 1965, Lammers et al. 1999, Maple, 1998) and ewes (Clark, 1964). Shore et al. (1988) was the first to describe premature udder development in cattle as the result of eating forage containing oestrogens. There are other reports of lactation in non-pregnant cattle that were the result of non-steroidal phyto-oestrogens present in alfalfa (Adler and Tranin, 1960) or subterranean or berseem clover (Clark, 1964; Thain, 1965). Oestrogen implants can stimulate a large increase in teat length growth during the treatment period, but this enlargement is lost post-treatment and teat length growth reduced by 30% (Lammers et al. 1999).

Thain (1965) investigated the cause of infertility in a dairy herd that were on predominantly subterranean clover pastures, and found that the ingestion of phyto-oestrogen in the plant caused udder development in maiden heifers. Udder swelling was also reported where cows were on clover pasture (Schoop and Klette, 1952). A similar syndrome has been reported when ewes were on clover pasture (Clark, 1964; Bennets et al. 1946). There is a report (Vestweber et al. 1986) that premature mammary development in a heifer with abdominal mesothelioma was due to the

neoplastic tissue that had affected the physiologic function of the ovary, resulting in excess production of oestrogen. Silberstein and Daniel (1987) also highlight the role of oestrogen on mammary growth, as TGF-α can influence the development of mammary gland if the udder is primed with oestrogen and progesterone. This indicates that the role of other hormones in mammary gland development depends on the presence of steroid hormones.

These data clearly indicate that oestrogen can play a major role in premature udder development in non-pregnant heifers and during early pregnancy in heifers. Treating or feeding heifers with food which contain oestrogen for a long period of time may stimulate premature udder development. This process can be stopped or reduced simply by removing the oestrogen source in animal food or treatments.

4. RECOMMENDATIONS TO REDUCE THE RISK OF MAMMARY DISTENSION AND MASTITIS

While definition of the problem is secondary to immediate prevention, prevention strategies will be improved by the better diagnosis and definition of risk factors:

- Provide detailed epidemiological descriptions of the condition including: prevalence of distension (and milk leakage) in heifers at initial assembly, prevalence of distension (and milk leakage) after loading and every second week during passage.
- Obtain samples aseptically of mammary secretion from heifers leaking milk for visual and microbial assessment.
- Obtain samples of feed from introductory diets and on the boat for visual appraisal and detailed nutritional analysis through Dairy One (DHIA), Laboratory in New York. Samples can be retrospectively analyzed for oestrogenic activity.

Specific recommendations to reduce the risk of distension of the mammary gland:

- Do not use legume-based forages as a base for diets used on the ship or limit these in the diet to a small proportion of the diet.
- Control the energy density of the diet and target a modest 0.4-0.5 kg per day growth rate for heifers.
- Limit potassium to <1% of diet and sodium to 0.5% of diet to control udder oedema risk.
- Ensure that selenium and vitamin E inclusions are consistent with concentrations used to reduce the risk of mastitis.
- Use fly treatments, eg Coopafly, Arrest, to control insect annoyance and irritation of teat ends.

- Improve environmental conditions to reduce exposure of the teats to faecal contamination and abrasive surfaces.

- Heifers showing mammary distension should be teat dipped with an iodine-based teat dip, placed in cleaner pens and fed on a diet higher in chaff.

- Veterinarians in charge of cattle should have a copy of Figure 1 (appendix II) to assist with decision making and problem definition. Specific therapeutic recommendations and management options are detailed on the sheet.

Further recommendations that may follow should these steps not sufficiently reduce the problem

- Regular teat dipping or use of a long acting external teat dip to reduce environmental exposure.

- Further evaluation of data gathered on subsequent shippings.

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APPENDIX I:

Udder Development and Lactation

As previously mentioned, 766 heifers loaded at Fremantle were monitored closely for udder development and the expression of milk. The passage between Fremantle and Portland enabled these heifers to be monitored in cool conditions in contrast to the previous voyage where much of the udder development appeared to be associated with heat an/or heat stress. Udder development in these heifers was evident early in the voyage suggesting that the development is linked to the stressful experiences associated with the export process and not related specifically to heat stress.

These heifers were monitored by undertaking a "walk through" on a regular basis. The eartag number of any heifers demonstrating udder development was noted to determine the incidence, the pattern of development any linkages to any known stage of pregnancy and/or property of origin.

This procedure was hampered later in the voyage as eartags became hard to read, however, there was plenty of data to make the linkages described.

- Of the 766 head monitored, 129 demonstrated udder development at some stage, (17%).
- The incidence of heifers at any one time ranged from 7-11% with most heifers demonstrating udder distention remaining distended throughout the voyage.

Inspection	At Loading	1st Inspection	2nd Inspection	3rd Inspection	4th Inspection	5th Inspection
Incidence	1.2%	7.8%	6.7%	6.9%	7%	10.6%

- A significant number of heifers, however, showed some easing of the distention (or recovery) and the distention seemed to be closely correlated to stressful incidents.
- Several "waves" of udder distention were observed.
- The incidence increased as the voyage progressed.
- Of those that showed udder development, only 6 head were observed to be expressing milk. This is likely to be a conservative figure since a larger number were observed to express milk at time other than the formal inspections. Nevertheless, the incidence may not be as high as first thought.
- Only 8 heifers were identified as having red, swollen or distorted udders that suggested mastitis. The actual incidence is reported be much higher than this, based on feed back from the receivers.
- 10 heifers showing udder development were placed in a central crush and more closely inspected. Of these, 3 head were found to have mastitis and treated accordingly. One of these

did not show any outward visual signs of mastitis. It is important not to confuse colostrum with mastitis affected milk (per com. Ian Lean). Milk was not cultured (nor dry slides for laboratory confirmation) to confirm that mastitis was present.

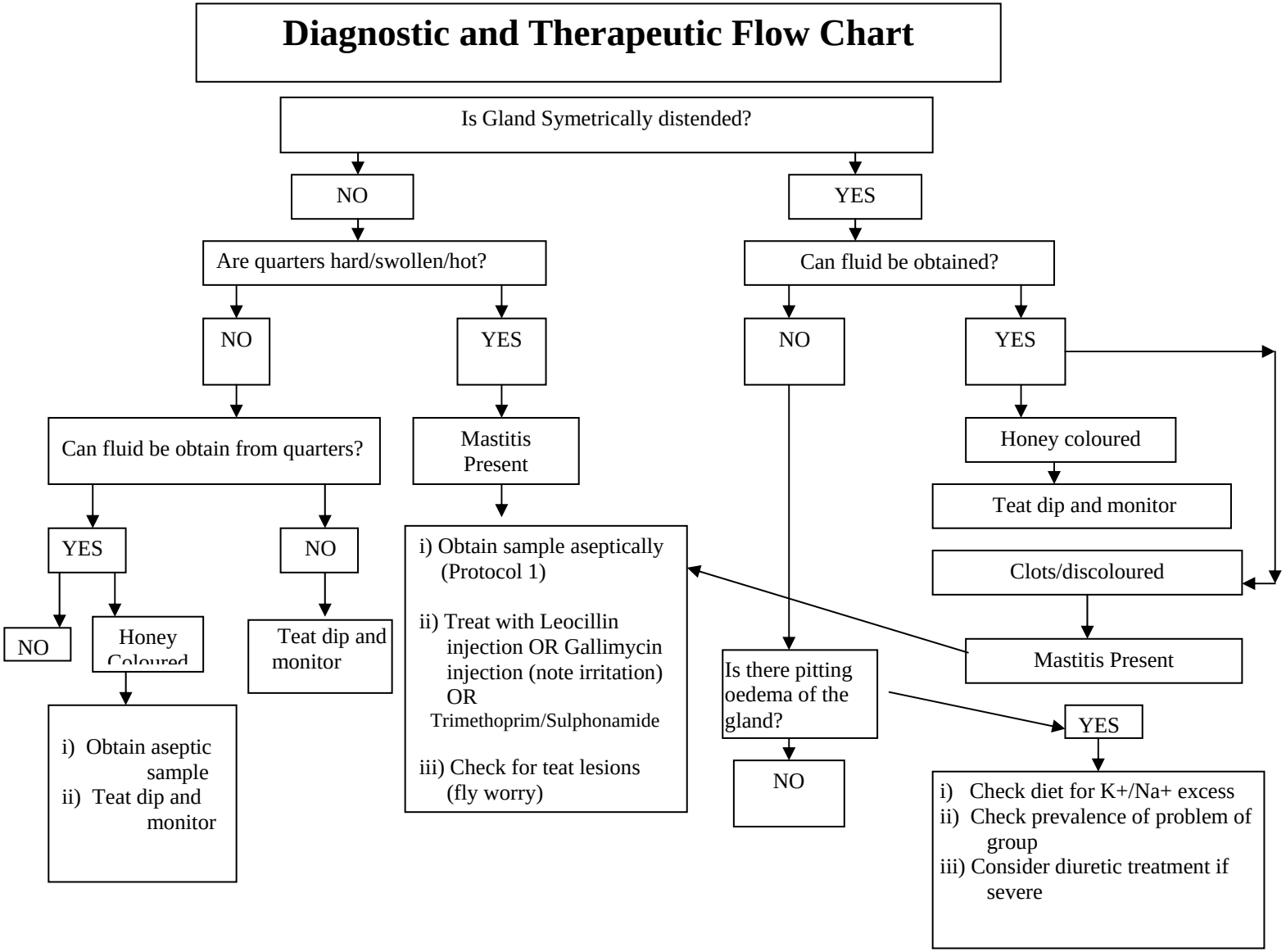
- The incidence of udder development was greater in those animals that were more advanced in pregnancy status, although it also occurred in heifers considered to be 2-2 ½ months pregnant.

Pregnancy	2 Mths	2 ½ Mths	3 Mths	3 ½ Mths	4 Mths	4 ½ Mths	5 Mths
Incidence	2%	12%	13%	14%	20%	27%	29%

- The incidence also ranged greatly between heifers sourced from different properties. Although some of the sample sizes of the groups was small, significant differences occurred, with only some of the difference being explained by differences in the stage of pregnancy. In those heifers that were 5 mths pregnant the incidence ranged beween 19-38%. In those heifers diagnosed as 4 ½ months pregnant the incidence ranged from 9-48 % and in those heifers diagnosed as 4 mths pregnant the incidence ranged from 8-60%. From this it can be assumed that property of origin is a pre-disposing factor.

- Nutrition has been identified as a contributing factor (Per com. Ian Lean). Even though the pellets are designed for maintenance, the high daily intake results in a relatively plane of nutrition. 5 heifers demonstrating udder development were removed to a sick pen where they were fed chaff only. The extent of udder development in these cattle subsided over a 4-6 day period but returned after these cattle were re-introduced to the pellet diet.

APPENDIX II:


Diagnostic and Therapeutic Flow Chart
Is Gland Symetrically distended?
NO
YES
Are quarters hard/swollen/hot?
Can fluid be obtained?
NO
YES
NO
YES
Can fluid be obtain from quarters?
Mastitis Present
Honey coloured
Teat dip and monitor
Clots/discoloured
YES
NO
NO
Honey Coloured
Teat dip and monitor
i) Obtain sample aseptically (Protocol 1)
ii) Treat with Leocillin injection OR Gallimycin injection (note irritation) OR Trimethoprim/Sulphonamide
iii) Check for teat lesions (fly worry)
Mastitis Present
Is there pitting oedema of the gland?
YES
NO
i) Obtain aseptic sample
ii) Teat dip and monitor
i) Check diet for K+/Na+ excess
ii) Check prevalence of problem of group
iii) Consider diuretic treatment if severe