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IMPACT, EFFECT AND CONSEQUENCES OF MASTITIS, TEAT CANAL INFECTIONS AND HEAT STRESS IN DAIRY CATTLE OF SOUTH AFRICA

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ABSTRACT

Mastitis, subclinical mastitis and teat canal infections (TCI), is the most important and expensive disease worldwide in dairy cattle because it is a multifactorial "occupational disease" in dairy cattle. No dairy herd is free of mastitis, subclinical mastitis and TCI. More than 137 pathogenic microorganisms can cause mastitis , subclinical mastitis and TCI. Mastitis affects about every fifth dairy cow in the world. The main access route for pathogens infecting the udder parenchyma tissue is through the teat canal (not less than 98%). The healthy undamaged and intact teat canal is the most important first-line defence mechanism against pathogenic microorganisms that cause TCI, subclinical mastitis and mastitis. Teat canal infection is inconspicuous and not visible. The diagnostic reliability of techniques for determining the prevalence of TCI is controversial and varies: bacteriological results obtained by examination of miniature teat canal swabs were 20% higher on average than those of foremilk samples examined and classified according to the criteria of the International Dairy Federation. Only with a miniature teat canal swab can teat canal infection be determined accurately. It is of utmost importance and a priority to understand the pathogenesis of bovine mastitis, subclinical mastitis and TCI to prevent and control the disease. Climate change which causes heat stress has the greatest negative impact as a stress factor in dairy cattle and impairs their immune system, making the cow more susceptible to infection of mastitis, subclinical mastitis and TCI. It is of utmost importance to understand the pathogenesis and consequences of bovine mastitis, subclinical mastitis and TCI in dairy cattle to prevent and control it. It is estimated that the milk loss due to mastitis, subclinical mastitis, TCI and heat stress in the dairy industry of South Africa is more than one billion Rand per year.

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Keywords: first-line defence mechanism, teat canal infection, lactogenic infection, heat stress, mastitis, immune system, udder parenchyma tissue, miniature teat canal swab

INTRODUCTION

Multifactorial "occupational disease" in dairy cattle: mastitis and teat canal infections

Mastitis, teat canal infection (TCI) and subclinical mastitis represent inflammation of the dairy cow's udder parenchyma tissue and teat canal. This is the most important disease worldwide in dairy cattle even though good prevention and control measures are available and implemented because the disease is a multifactorial "occupational disease" in dairy cattle with high milk production (Giesecke, *et al.,* 1994; Amritha, *et al.,* 2020). There is no dairy herd, regardless of the milk breed, location or size of the herd that is free of mastitis and TCI. Basically, mastitis is divided into clinical, subclinical, subacute, peracute, and chronic forms (Du Preez. 1987b). No less than 137 pathogenic microorganisms (of which more than 80 different bacteria) that can cause mastitis have been identified. Mastitis affects about every fifth dairy cow in the world. In South Africa, only eight out of ten dairy cattle have healthy udders. Milk production loss of udder quarters with clinical mastitis can be 10% to 80% or even more and those with subclinical mastitis from 5% to 25%. At least 10% to 15% of all dairy cattle are infected in one or more udder quarters with subclinical mastitis (Giesecke, 1979; Du Preez, 1988b; Giesecke W.H. *et al,.* 1994; Du Preez, 2020). Mastitis also plays an important role in milkborne diseases and milk-associated zoonoses (Du Preez & Du Preez, 2018). The udder quarter of a dairy cow that produces about 30 litres of milk per day has about 500 to 600 million alveoli (milk-producing epithelial cells [Giesecke W.H. *et al,.* 1994]). During the last decades, the dairy cattle have been improved by selection and breeding so that the dairy cattle's milk production has increased. Mastitis is the result of bacterial lactogenic infection of the dairy cow's udder that penetrates the teat canal and either weakens or overwhelms the immune system (Thompson-Crispi, *et al.,* 2014). The nutrients that possibly increase

the dairy cow's immunity and help prevent mastitis, are well-documented, namely vitamin E, selenium, zinc and vitamin A (Thompson - Crispi, K. *et al.,* 2016). Where the keratin is stripped in the teat canal, udder infection occurs much more easily, causing mastitis (Du Preez, 2020). The main access route for pathogens infecting the udder parenchyma tissue is through the teat canal (lactogenic route, not less than 98% infections). Haematogenous and traumatogenous mastitis infections in dairy cattle are very rare, namely less than 1%. The modern dairy cow is highly exposed to unnatural conditions, such as mastitis, heat stress, mud stress, handling stress, nutritional stress, negatively impacting grazing systems, poor housing systems, excessively large herds of dairy cattle, and many more that adversely affect their defence system and immunity. That necessitates an intact, healthy, undamaged and normal function of the teat canal (Giesecke, *et al.,* 1979; Du Preez, 1988a, 1994, 2014 & 2020; Sordillo, *et al.,* 1997). Specific and congenital immune factors related to the udder parenchyma tissue play an important role in protecting the udder from infections (Sordillo, *et al.,* 1997). Heat stress has the greatest negative impact as a stress factor in dairy cows because all systems and functions of the dairy cows suffer from it (Du Preez, 1994; Pragna, *et al.*; 2017; Dahl, *et al.,* 2020).

DISCUSSION

Connerstone for good udder health in dairy cattle

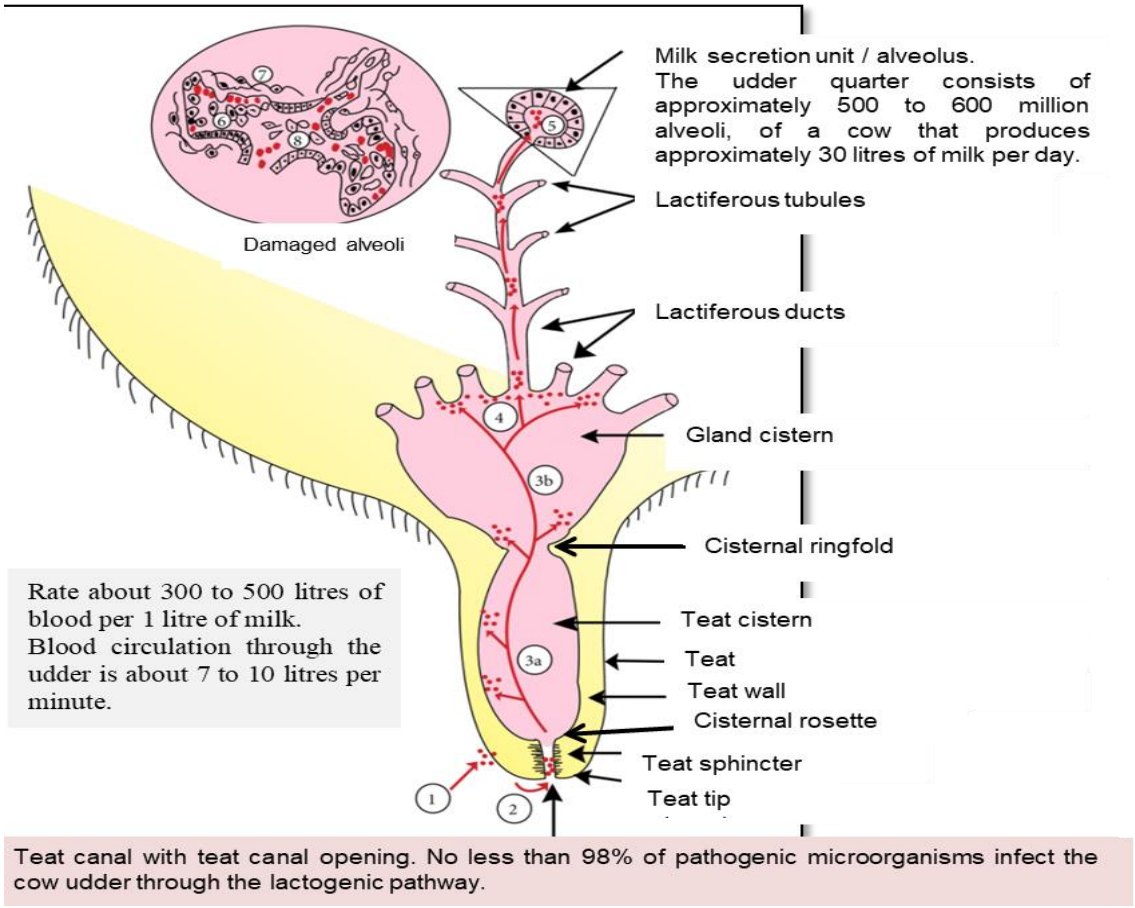
The healthy undamaged and intact teat canal is the most important first-line defence mechanism against pathogenic microorganisms that infect the cow udder and cause mastitis. Calcium deficiency leads to less complete closure of the teat muscle (Sordillo & Nickerson, 1998). The healthy cow udder with an optimal immune system, effective defence mechanism and healthy teat canal is the key and cornerstone for good udder health and minimal cases of mastitis (Sordillo, *et al.,* 1997; Du Preez, 2014 & 2020). Teat canal infection is not visible (subclinical), is inconspicuous, an entity in itself and is confined to the teat canal at the teat tip. It is independent of infection of the mammary ducts (galactophoritis) and mastitis involving the udder parenchyma tissue. Teat canal infection cannot be observed with the naked eye. Mastitis pathogenic and oppertunistic pathogenic bacteria can colonise the teat canal and cause teat canal infection as well as mastitis. The lower (distal) portion of the teat canal (± 1-2mm) that opens into the teat canal opening is in contact with the environment where the cow is located. Often saprophytes (e.g. *Arthrobacter* spp., *Flavobacterium* spp. and *Micrococus luteus*), soil bacteria (e.g. *Bacillus spp*.) and fungi (e.g. *Candida albicans*) colonise there and it is readily considered normal. The upper two-thirds (± 6-7mm) of the teat canal is usually free of bacteria. Sometimes pathogenic bacteria colonise there (*Streptococcus agalactia, Pseudomonas putretaciens* and many more). If bacteria enter the teat canal and infect the teat canal, it is called a teat canal infection which can result in a teat canal inflammation. A distinction must be made between teat inflammation (thelitis) and teat canal inflammation, which are two separate entities. (Du Preez, 1985, 2014 & 2020; Giesecke, *et al.,* 1979 & 1994). Teat canal infections cannot be determined accurately with the normal cytobacteriological milk sample examinations. Only with a miniature teat canal swab can teat canal infection be accurately determined. High incidence of TCI in a dairy herd indicates that there are many predisposing factors which make the herd susceptible to mastitis present on the dairy farm. The incidence of clinical mastitis is much higher in dairy herds where the incidence of teat canal infection is high (Du Preez, 1985 & 1988a). The overall management strategy on a dairy farm needs to be improved and optimised where the incidence of teat canal infection is high. The diagnostic reliability of techniques for determining the prevalence of TCI is controversial and varies: bacteriological results obtained by examination of the miniature teat canal swabs were 20% higher on average than those of foremilk samples examined and classified according to the criteria of the International Dairy Federation. Since they are based exclusively on the cytobacteriological results of the foremilk samples. The International Dairy Federation criteria for the classifications of the various forms of subclinical udder conditions do not permit an accurate classification of the health status of the udder (Du Preez, 1986).

Impact of teat canal infection, mastitis and heat stress on dairy cattle

Teat canal infections can serve as a source of infection for the udder parenchyma tissue and can cause subclinical and clinical mastitis. A high incidence of TCI in dairy herds indicates that there are many predisposing factors for mastitis present on the dairy farms (Du Preez, 1985, 1986 & 2020). The most important negative factors to dairy cattle are: a cow-unfriendly environment; milking machines not functioning properly; poor and impermissible milking techniques; inefficient management practices; heat stress; mud stress; handling stress and many more (Du Preez, 1994). The TCI can persist indefinitely or the cow udder can eliminate it by itself or it can lead to subclinical (invisible) or clinical (visible) mastitis with all the adverse effects. Teat canal infection is not necessarily a precursor to mastitis, but can be caused directly by bacteria moving through the teat canal (especially if the teat canals' keratin are stripped) into the udder parenchyma tissue and then causing infection (Du Preez, 1986). Dry cow teat canal therapy effectively eliminates existing TCI and to a large extent prevents the development of new udder infections during the dry period of the dairy cow before she goes into calving. Although teat canal therapy with antibiotics is enormously effective in eliminating TCI, its commercial use has not

yet been finalised (Du Preez, 1986). Regular teat dipping (with a disinfectant) of the cows after milking can help prevent mastitis and TCI. The incidence of TCI in dairy cattle that are regularly teat dipped after milking will usually not decrease (Du Preez, 1987a). The incidence of TCI decreases in dairy herds where good management, animal care and hygiene are applied (Du Preez, 1985). The healthy and undamaged teat canal plays the most important role in udder health and prevention of mastitis (Du Preez, 2020). Heat stress in dairy cattle compromises and impair their immune system, making the cow more susceptible to infection by microorganisms in the environment that can cause mastitis and teat canal infection (Dahl, *et al.,* 2020; Du Preez, 2020). Heat stress and mastitis are one of the biggest problems affecting the production potential of dairy cattle in almost all parts of the world. The effects of mastitis and heat stress on dairy cattle are reduced milk production, decreased milk quality, increased somatic cell count, endocrine imbalance, involution of udder tissue, apoptosis, necrocystosis and autophagy (cleaning out damaged cells), decreased feed intake, pathological changes in the udder and mastitis (Du Preez, 2000; Pragna *et al*., 2017; Dahl, *et al.,* 2020). Milk producers must strive to produce milk with a somatic cell count (SCC) between 300 000 to 350 000 per ml of milk. South Africa's threshold value for SCC of the bulk milk tanks is 500 000 per ml of milk. Scientists have already shown that if the SCC is 500 000, the dairy farmers cows have already produced 6 to 9% less milk (Alhussien & Dang, 2017). The impact of heat stress, mud stress, handling stress, nutritional stress etc. is enormously negative on the immune system and milk production of dairy cattle (Dahl, *et al.,* 2020).

Figure 1: It is of utmost importance to understand the pathogenesis and consequences of teat canal infections and bovine mastitis in dairy cattle to prevent and control TCI and mastitis (Giesecke, 1979; Frost, *et al.,* 1980; Du Preez, 1985; Giesecke, *et al.,* 1994; Sordillo, *et al.,* 1997; Kerro Dego, *et al.,* 2002; Zecconi, 2005; Amritha, *et al.,* 2013; Zhang, *et al.,* 2016; Pal, *et al.,* 2019; Du Preez, 2020).



Caption of Figure 1: Potential mastitis pathogenic bacteria (1) infect the teat tip; bacteria spread and multiply (2) in the teat canal; bacteria gain access to the teat and glandular cisterns (3a, 3b) and infect the glandular epithelium; bacteria multiply and spread further through the lactiferous ducts and tubules (4) until they finally reach the alveoli (milk-producing units) that produce and secrete milk; the alveoli become infected (5); the milk-secreting epithelium of the alveoli (6) is damaged and some epithelial cells are destroyed and rejected. Epithelial damage worsens and the mammary gland

epithelium (udder parenchyma tissue) of the alveoli is destroyed (7). Pus forms in the alveolar cavities and tubular cavities is flushed out by milk that is still formed by the undamaged alveoli, thus making the mastitis clinically observable. Connective tissue is deposited around and in the damaged alveoli and forms scar tissue (8) which replaces the alveoli and thus eliminates and renders it dysfunctional (Du Preez, 1988b; 2000 & 2020). As large parts of the milk-secreting tissue are destroyed, milk production decreases and the udder parenchyma tissue become hardened and abnormal (becoming fibrotic). The destruction of the alveoli is permanent and irreversible and the udder quarter produces less milk for the rest of the cow's life.

Figure 2: Electron micrograph (10,000 x magnified). Normal healthy and functional alveoli of the cow udder. Fat (lipid) droplets in the alveoli are clearly visible (Du Preez, 1985 & 2020).

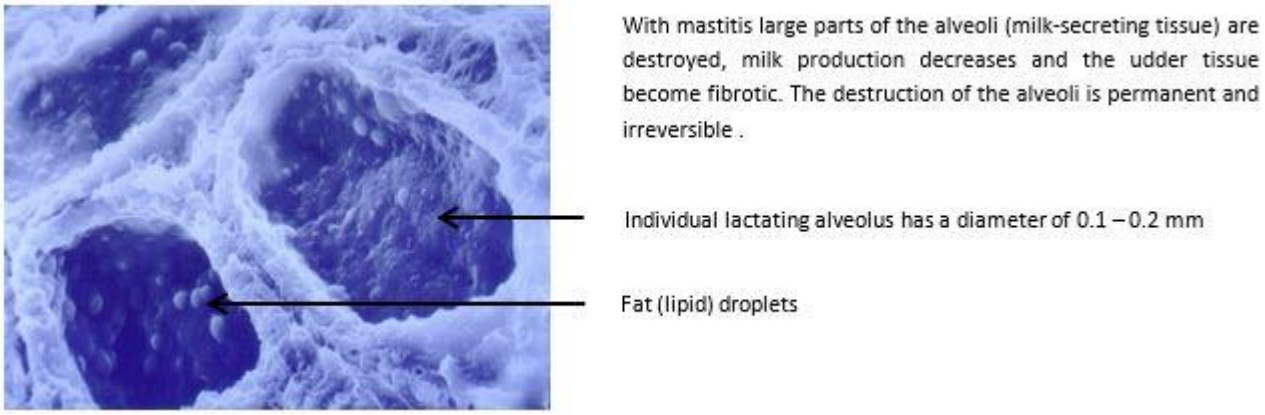
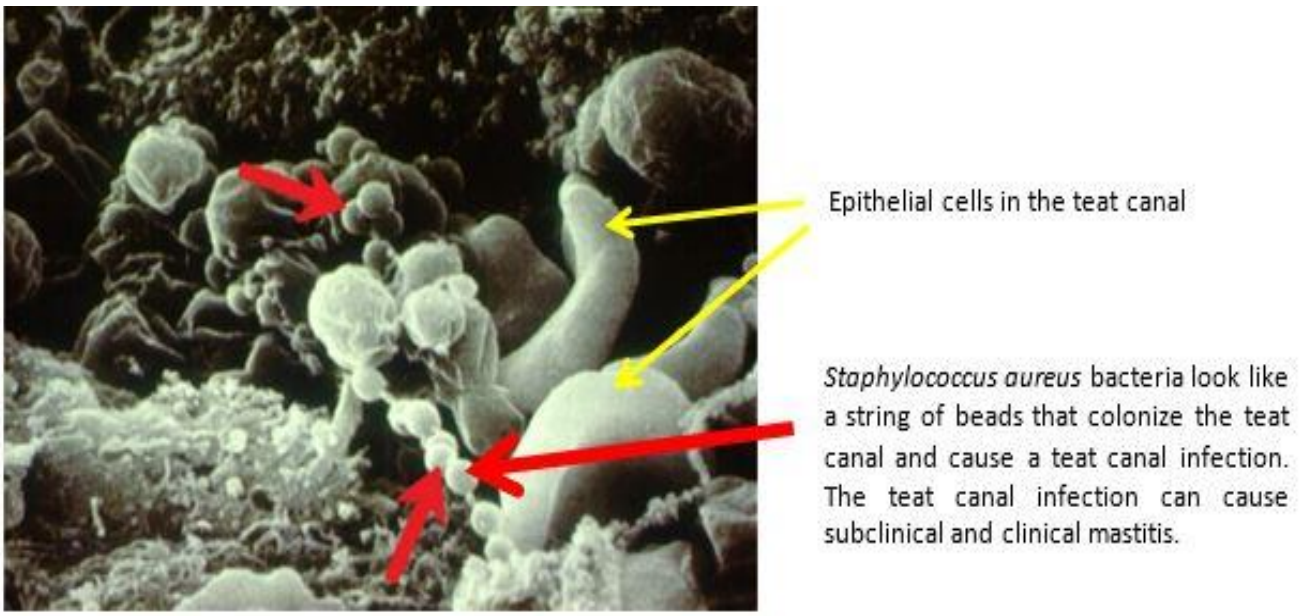


Figure 3: Teat canal electron microscopy

Electron microscope photograph (10 000 x enlarged) of the bacteria *Staphylococcus aureus* that colonise the teat canal and cause teat canal infection. Bacteria look like a string of beads. *Staphylococcus aureus* bacteria can produce enterotoxin (vasoconstrictor) which can cause Toxic Shock Syndrome as well as narrowing of the udder's blood vessels. Numerous other pathogenic bacteria colonise the teat canal (Du Preez, 1985 & 2020).

Figure 3: Teat canal electron microscopy



Important pathogenic and oppertunistic pathogenic bacteria colonise the teat canal are *Enterobacter cloacae, Streptococcus uberis, Corynebacterium bovis, Serratia marcescens, Streptococcus faecalis, Streptococcus agalactiae, Staphylococcus aureus, Escherichia coli, Trueperella pyogenes, Klebsiella oxytoca, Pseudomonas aëruginosa*, *Pseudomonas putrefaciens, Streptococcus epidermidis, Streptococcus bovis* (Du Preez, 1985 & 2020).

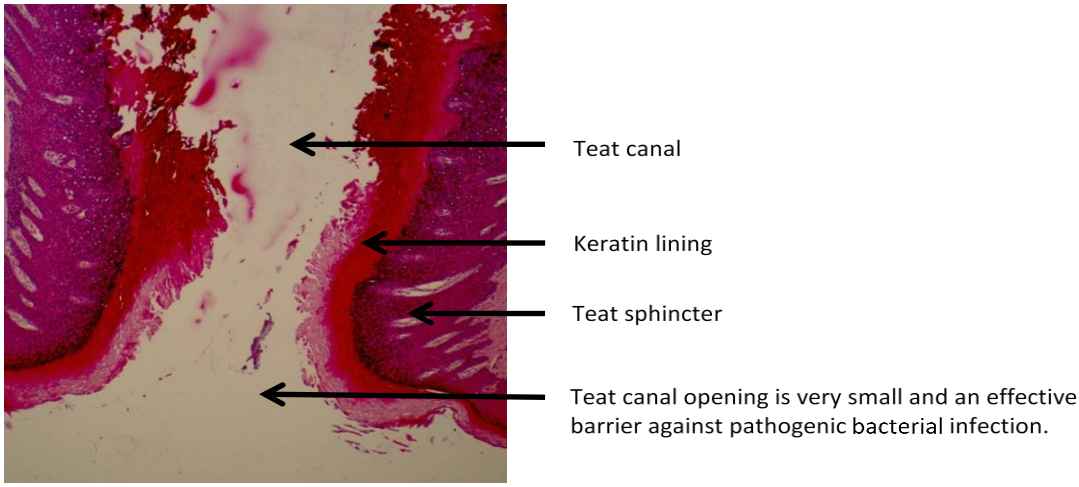


Figure 4: The healthy undamaged and intact teat canal (under the microscope) is the most important first-line defence mechanism against pathogenic microorganisms that infect the cow udder and cause mastitis (Sordillo, *et al.,* 1998; Du Preez, 1985 & 2020).

Bacteriostatic acids that protect the teat canal against pathogens are myristic, palmitoleic and linoleic acids. Bacteriocidal factors also protect the teat canal against pathogens are immunoglobulin, phagocytes, complement factors, lizozyme, lactoferrin, lacoperoxidase and also humoral and cellular immunity. The optimal teat canal defence machanism and functions are: keratin lining of the teat canal; bacteria are trapped by epithelial cells and peel off every day and are removed by milking (rinsing effect); keratin (waxy material) that traps bacteria; natural antimicrobials; after milking, the teat canal is dry (hydrophobic teat canal); cationic proteins and fatty acids; rapid epithelial regeneration and teat canal is funnel-shaped. Teat sphincter ensures efficient closing of the teat canal. Takes 15 to 20 minutes, after milking to close, preferably cows should then stand (Sordillo, *et al.,* 1997; Giesecke, *et al.,* 1994; Du Preez, 2020). The number of lactating dairy cattle in South Africa is about 600,000 (Swart, 2020). It is estimated that the milk loss due to mastitis, TCI and heat stress in the dairy industry of South Africa is more than one billion Rand per year (Du Preez & Swart, 2020).

Figure 5 and 6: The diagnostic reliability of techniques for determining the prevalence of teat canal infections is contraversial and varies: bacteriological results obtained by examination of teat canal swabs were 20% higher on average than those of foremilk samples (FMS) examined and classified according to the criteria of the International Dairy Federation (IDF). Since they are based exclusively on the cytobacteriological results of the FMS, the IDF criteria for the classifications of the various forms of subclinical udder conditions do not permit an accurate classification of the health status of the udder (Du Preez, 1986).

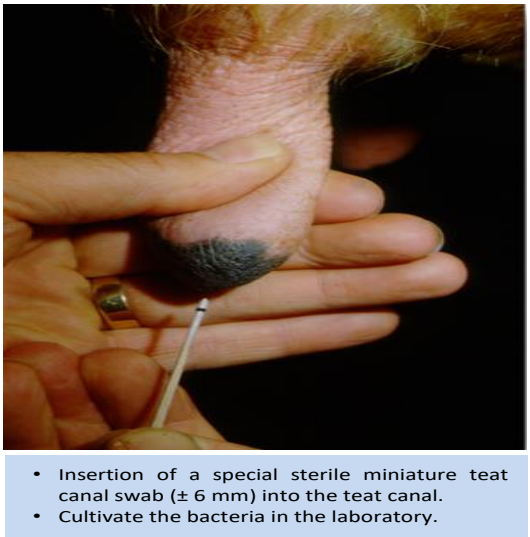
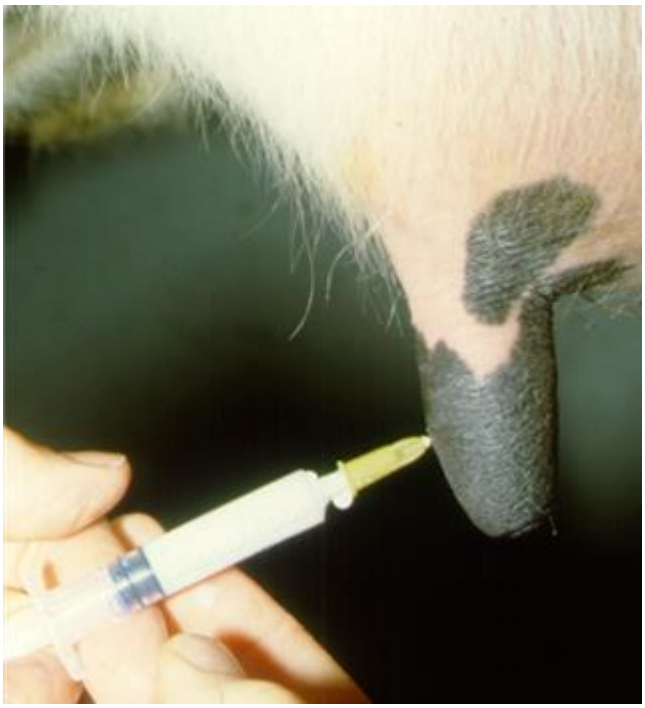


Figure 5: Taking a teat canal swab sample for bacterial culture (Du Preez, 1988a)



- Disinfection of the teat.
- Insert the sterile needle through the teat wall into the teat cavity (cistern).
- Aspiration of 1ml milk with a sterile syringe.
- Cultivation of the milk sample in the laboratory to confirm that the milk is free of bacteria.
- If the milk sample contains no bacteria and the teat canal swab sample is positive, it confirms the presence of bacterial colonization in the teat canal.

Figure 6: Confirming bacterial colonization in the teat canal with teat wall puncture milk sample (Du Preez, 1988a)

CONCLUSION

The impact, effect and consequence of mastitis, subclinical mastitis, TCI and heat stress in dairy cattle in South Africa is an enormous problem. Prevention and control as far as possible of mastitis, subclinical mastitis, TCI and heat stress is dairy cattle in South Africa is therefore extremely relevant and a priority. Practical understanding of the pathogenesis to prevent and control bovine mastitis, subclinical mastitis, TCI and heat stress are indispensable and also a priority.

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COMPETING INTERESTS

The authors declare that they have no competing interests.

AUTHORS CONTRIBUTIONS

JH prepared the initial version of the manuscripts. JH and TD assisted in literature collection. JH and TD drafted and

revised the manuscript for critical scientific corrections. All authors read and approved the manuscript.

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