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THE DIAGNOSIS OF BOVINE MASTITIS WITH PARTICULAR REFERENCE TO SUBCLINICAL MASTITIS: A CRITICAL REVIEW OF RELEVANT LITERATURE

W. H. GIESECKE* and L. W. VAN DEN HEEVER**

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ABSTRACT

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From a review of literature on bovine mastitis published between 1833 and 1973 it is concluded that although herd control has caused a shift of emphasis from the clinical to the subclinical forms, diagnostic criteria have remained basically unaltered. Concurrent with this shift, improved bacteriological and cytological techniques are more sensitive than those available at the turn of this century. The sensitivity of these methods apparently has led to great confidence in their use in the diagnosis of mastitis. This culminated recently in a definition of mastitis as well as standardization of diagnostic criteria by the International Dairy Federation (IDF) which stressed the significance of potentially pathogenic bacteria and elevated somatic cell counts (threshold 5×10^5 cells/ml) in aseptically sampled foremilk.

The validity of these widely accepted diagnostic criteria, particularly in the case of subclinical mastitis, is based on the assumption that an increase in the sensitivity of the methods should lead to an increase in diagnostic accuracy. Consequently, one would expect to find conclusive evidence of the diagnostic significance of the criteria used by the IDF.

In contrast, this review attempts to reveal a conspicuous lack of adequately controlled experimental evidence supporting the diagnostic significance of criteria laid down by the IDF. They are of particular importance when dealing with subclinical mastitis, since the demarcation between physiological and pathological changes is considerably less distinct than in acute clinical mastitis. Thus no reference to pathological alteration of the udder epithelium, the primary symptom of mastitis, could be found in the literature assessed in terms of examination of milk. Similarly, the misleading influence of teat canal infections or lesions on the diagnostic accuracy of bacteriological and/or cytological examination of milk samples, obtained by "aseptic" withdrawal via the teat canal, is disregarded. The physiological significance of Selye's General Adaptation Syndrome or Schalm's Leucocytic Udder Barrier as non-inflammatory causes of elevated epithelial or leucocytic counts in milk appear to have been overlooked by workers who regard the presence of somatic cells in general or leucocytes in particular as synonymous to pus in milk.

Due to absence of appropriately controlled experimental data, there is also no conclusive evidence to support the diagnostic significance of the IDF criteria referring to latent udder infections. Likewise it is not possible to distinguish between clinical and subclinical mastitis in udders with chronic indurative tissue changes in the absence of clinical alterations of the milk.

The above considerations do not preclude the control of certain types of mastitis by application of IDF standards. However, because such criteria may result in some 43,13 – 20,8% false positive diagnoses, it is conceivable that losses resulting from procedures based upon inaccurate diagnoses may far outweigh those caused directly by mastitis.

The criteria of the IDF could, however, be used to great advantage for the diagnosis and control of mastitis if augmented by a test capable of establishing the physiological and pathological state of the mammary epithelium. This is possible by means of a radial immunodiffusion test based on diffusion of bovine serum albumin (BSA), present in milk. The combination of BSA and the IDF criteria permits accurate assessment of udder health and teat canal infections.

*Veterinary Research Institute, Onderstepoort
** Faculty of Veterinary Science, University of Pretoria, P.O. Onderstepoort

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Introduction

Bovine mastitis caused by specific micro-organisms, such as *Streptococcus agalactiae, Corynebacterium pyogenes* or *Staphylococcus aureus* can be controlled with reasonable success by the application of currently available knowledge (Schalm, Carroll & Jain, 1971). However, bovine mastitis as a whole and especially subclinical mastitis, apparently remains an enigma and is considered to be anything but under control (Estes, 1964; Kaley, Brower, Safford & Hendershott, 1965; Morse, 1970).

The limited progress made in the overall control of bovine mastitis has resulted in extensive research being focussed on this complex disease. At present, bovine mastitis is regarded internationally as one of the most costly problems (Von Sivers, 1967) confronting the dairy industry (Janzen, 1970; Giesecke & Van den Heever, 1971; Giesecke, Van den Heever & Du Toit, 1971). This prompted the Expert Committee on Mastitis, which advises the International Dairy Federation (IDF), to standardize the definition and diagnosis of mastitis (Kästli, 1967; Tolle, 1971), thus promoting the application of more efficient control measures against this disease on a world-wide scale.

One of the most important reasons for the disappointing lack of success achieved in the control of bovine mastitis seems to be associated with the multitude of types of mastitis (Udall & Johnson, 1931; Ullner, 1959; Obiger, 1962a, b) resulting from the complex aetiology and symptomatology, as well as an apparently incomplete understanding of the pathogenesis of the condition. Major difficulties are still being encountered in the diagnosis, therapy and prevention of subclinical mastitis in particular (Müller, 1968; Reichmuth, 1968; Grambow, 1970; Dodd & Jackson, 1971).

The elaborate and expensive control measures against bovine mastitis would be justifiable if the currently accepted definition and diagnostic criteria for bovine mastitis, particularly with regard to the subclinical forms in lactating cows, were correct. However, recent investigations (Giesecke, Van den Heever, Du Toit & Beyer, 1973; Theodoridis, Giesecke & Du Toit, 1973) have cast serious doubts on these criteria.

To facilitate a better understanding of the difficulties encountered in the diagnosis of subclinical bovine mastitis, it has become necessary to review the relevant research which led to the formulation of currently accepted IDF standards.

The IDF Definition of Bovine Mastitis

The IDF definition of mastitis is based on recommendations made by Kästli (1967) and Tolle (1971). The definitions given by Kästli (1967) are cited as follows:

"i. *Normal udders* are those which show no outward signs of a pathological condition and the milk from which is free from pathogenic organisms and has a normal cell count.

ii. *Latent infections* are present when the milk shows the presence of pathogenic organisms, but nevertheless has a normal cell count.

iii. *Subclinical mastitis* shows no macroscopic evidence of inflammation but examination of the milk reveals udder infection, an increased cell count and also alterations in the chemical properties of the milk.

iv. *Clinical mastitis: acute mastitis* is present when there are obvious symptoms of inflammation of the udder such as heat, pain and swelling. The milk is macroscopically abnormal and the animals may have an elevated body temperature. *Subacute mastitis* is present when there are persistent clots especially in the foremilk.

v. *Non-specific or aseptic mastitis* is present when there is no recognisable infection and the symptoms may be subclinical or clinical.

All these definitions are connected with the cell counts of milk. Therefore....a treshold value (in milk from single udder quarters) of more than 500 000 cells/ml is suggested as indicating that the cell-count is abnormal and that a diagnosis of mastitis has been established. This threshold value is acceptable for diagnostic classification under the condition that the milk is sampled: from the first fractions of milking; from cows in normal lactation; aseptically at milking times".

Tolle (1971) elaborates further on the definition of mastitis:

"Mastitis is an inflammatory change of the mammary gland which, along with physical, chemical and microbiological changes, is characterized by an increase of somatic cells, especially leucocytes, in the milk, and by pathological changes in the mammary tissue.....

This definition applies to the examination of quarter milk samples taken at the usual milking time from the initial milk (after discarding the foremilk) of cows in lactation".

From the above it is apparent that the diagnosis of mastitis in lactating cows is currently based on clinical examination of the udder tissue and its secretion, the somatic cell content of milk, and bacteriological data. The reliance placed on these criteria is best understood if considered in the light of earlier research, which eventually resulted in the formulation of current concepts concerning bovine mastitis in general and the diagnosis of this disease in particular.

Evolution of the Diagnosis of Mastitis

1. *The diagnosis of mastitis prior to* 1889

Early literature suggests that clinical mastitis in cows and goats caused problems serious enough to attract the attention of a Swiss novelist (Gotthelf, 1849) as well as scientists. The scientific literature reveals a distinct tendency to describe, classify and define mastitis in terms of available diagnostic techniques and information on the pathogenesis. Early observations on the disease were therefore limited to the clinical signs observed by examination of udder tissue and secretion. Blaser (1833, cited by Heidrich & Renk, 1963) for instance, distinguished between benign and malignant forms of mastitis, the former characterized by absence and the latter by the presence of fever and malaise. Haubner (1848, cited by Heidrich & Renk, 1963) described superficial, parenchymatous and gangrenous forms of mastitis. The first was associated with slight redness, heat and pain of a somewhat swollen, firm, elastic udder tissue and an apparently unaffected secretion. The second was characterized by diseased glandular tissue exhibiting marked swelling, redness, pain and a viscous, greatly reduced secretion. In cases of the third type the udder surface was discoloured bluish red and the gland of doughy consistency and cold to the touch. Brennwald (1848), Gattiker (1848) and Rast (1848), all cited by Hess & Borgeaud (1888),

described an udder disease causing a progressive atrophy of the udder with concurrent decreasing secretion of a fluid which had an increasingly yellow appearance. A similar condition was observed in sheep and goats (Brusasco, 1871, cited by Hess & Borgeaud, 1888). This disease was referred to as "gelber Galt" or garget and was thought to result from low temperature, wet floors, incomplete milking, obstruction of lactiferous vessels due to inflammation of the abdominal wall, abnormal composition of digestive fluids or witchcraft.

Early suspicions concerning an infectious cause of udder disease (Zangger, 1854, cited by Hess & Borgeaud, 1888) apparently were verified by Nocard & Mollereau (1884) who isolated streptococci from the secretion of clinical cases of mastitis. With the advent of bacteriological research, efforts were intensified to further elucidate the types of udder diseases in general and the causes of udder inflammation in particular. Thus Crookshank (1888) describes summer mastitis and udder lesions associated with pox, foot-and-mouth disease and papillomatosis. Strebel (1888) reports on climatic factors in the aetiology of mastitis, discusses the theory on udder infections and proves the galactogenous nature of such infections. Bühler (1888), in assessing factors related to the aetiology of parenchymatous bovine mastitis, refers to infection as the new magic word and suggests, somewhat contemptuously, that witchcraft, frequently referred to by farmers as the cause of mastitis, is merely an excuse for abominable hygienic conditions. The rapidly advancing knowledge enabled Hess & Borgeaud (1888) to write a remarkably detailed paper dealing systematically with aspects such as symptoms (including altered chemical composition of milk), differential diagnosis (udder oedema, acute and chronic parenchymatous mastitis, aseptic mastitis), pathological anatomy, aetiology, bacteriology, pathogenesis, prognosis, therapy and prophylaxis.

Though limiting their efforts to observations on clinical mastitis, Hess & Borgeaud (1888) nevertheless set the stage for the more elaborate research on subclinical mastitis of subsequent decades by drawing attention to: the salty taste of mastitic milk; decreased lactose concentration; the separation by gravity of such milk into a serous top fraction and a bottom fraction consisting of floccules, fibrin and epithelial cells; the finding of degenerated cellular admixtures and streptococci by microscopic examination of mastitic milk; cultural characteristics of streptococci and the artificial reproduction of an identical type of mastitis in goats. Of particular interest is the early description of a condition comparable to what is presently termed "aseptic mastitis" and the finding of streptococci in the cow's environment accompanied by a suggestion that such streptococci, reaching the udder by contact with contaminated floors, bedding, excreta or milker's hands, initially proliferate in the drops of milk that frequently adhere to the teat orifice after milking. The streptococci were thought to progress from this drop of milk through the teat canal via a column of milk to cause mastitis on reaching the udder cavity. The streptococci were presumed to be the cause of the parenchymatous udder inflammation since they were always found in association with the latter, but never in other diseased or normal udders.

2. *The diagnosis of mastitis between* 1889 *and* 1899

A growing awareness of various aspects of mastitis is evident from papers published during this period (Casper, 1896; Jensen, 1897; Schlegel, 1898). Still depending on clinical examination as well as culture methods, Guillebeau (1890) and Guillebeau & Hess (1891) report on abnormal milk and mastitis in cows, and other domestic animals. Baum (1892) cites Franck (1876), Bang (1888), Kitt (1890) and Krüger (1890) as having isolated staphylococci, streptococci, bacilli and micro-organisms of unknown identity from cases of clinical mastitis.

Other researchers, presumably familiar with the bacteriological diagnosis of mastitis, thought it sufficient to rely merely on clinical examination. Kohl (1893) reports on a case of gangrenous mastitis and the recovery of the affected cow without proffering any bacteriological support for his diagnosis. For practical reasons Steuert, Albert, Notz & Schupp (1893) find clinical examination of the udder more suitable than the clinical examination of the secretion. Bacteriological diagnosis is not considered in their paper on udder inflammation. It appears that field workers and the more practical researchers thought the bacteriological diagnosis of mastitis too cumbersome, and it is not surprising to learn of early attempts to employ short-cuts to facilitate the rapid examination of more cows and milk samples; an additional incentive was provided by the concurrent increasing concern about milk-borne diseases such as tuberculosis and scarlet fever (Baum, 1892; Anon., 1893; Walley, 1893; Hintze & Lubarsch, 1894; Dürck, 1895; Squance, 1896; Woodhead, 1896; Anon., 1897; Délepine, 1897; Lubarsch, 1899). Baum (1892), for example advocated the macroscopic examination and determination of the pH of milk as suitable diagnostic methods. Zschokke (1897) found the macroscopic examination of milk, after separation of its constituents by gravity in particular, and a microscopic examination of the sediment for streptococci, very useful. He considered clinical examination of the udder and determination of milk production less suitable. By subjecting milk to macroscopic and microscopic examination for the determination of extraneous matter and cellular admixtures, Délepine (1897) found that the cell content of milk, which consisted of epithelial cells, leucocytes and many epidermal scales, varied considerably but was greatly elevated in tuberculous mastitis. He suggested the use of the guinea pig test for the detection of tuberculous milk. During the same year Stokes (1897, cited by Breed, 1914), and Stokes & Wegefarth (1897), both discussed the sanitary significance of cells, some of which they recognized as leucocytes and regarded as pus cells, in ordinary milk.

It appears in general that the advent to pollution problems causing sewage poisoning and mastitis in cattle (Malcolm, 1898), the strong promotion of joint medical and veterinary participation in milk hygiene (Woodhead, 1896) and the impact of young sciences such as medical bacteriology (Koch, 1890) and immunology (Behring, 1891) apparently resulted in considerable diversification of veterinary activities. In comparison to the progress during the period prior to 1889, the subsequent years 1889–1899 therefore seem to be less significant in mastitis research. However, although phagocytosis was still considered to be a puzzling new phenomenon at the beginning of the decade (Netschajeff, 1891; Hunter, 1892; Metschnikoff, 1894), a few years later leucocytic

infiltration and phagocytosis in acutely inflamed organs (Muir, 1898), the microscopic counting of somatic cells in milk sediment and their interpretation as pus cells (Stokes & Wegefarth, 1897) were acknowledged facts.

3. *The diagnosis of mastitis between* 1900 *and* 1909

Under the influence of rapidly advancing knowledge on bacterial diseases in man in general (Casper & Höchst, 1900/01; Piertik, 1902; Trautmann, 1903; Macé, 1904; Fromme, 1909; Koch, 1909) and milk-borne diseases (Délepine, 1908), particularly tuberculosis (Moser, 1902; Moussu, 1906) and scarlet fever (Hammer & Jones, 1909), together with the realization of the disconcertingly high bacterial content of milk (Houston, 1906; M'Fadyean, 1909; Savage, 1909) and the consequent fear of the occurrence of epidemics in the large cities, milk hygiene became considerably more important. The result was a distinct tendency to diagnose mastitis by laboratory examinations of milk rather than by clinical examinations of dairy cows in order to increase the efficiency of supervision over milk supplies. Klimmer (1900a, b, c) suggested the diagnosis of mastitis by considering the yield, macroscopic appearance and chemical composition of milk and emphasized that such examination should preferably be performed by veterinarians.

In contrast Zschokke (1904b) found chemical and physical examinations of milk unsuitable for the detection of mastitic milk and concluded that the only practical method to determine mastitic admixtures in market milk was veterinary examination of cows and their milk, the hygienic conditions on the farm and milk transport. He regarded centrifugal separation of milk sediment and examination for leucocytes and bacteria as the most suitable laboratory method. At the same time Bergey (1904, cited by Trommsdorf, 1909) and Bergey (1907) observed, on microscopic examination of milk sediment smears, that leucocyte counts of milk were elevated in the presence of mastitis streptococci. Contemporary workers such as Doane (1905), Stewart (1905; cited by Prescott & Breed, 1910), Savage (1906), Slack (1906, cited by Prescott & Breed, 1910) and Hewlett, Villar & Revis (1910) apparently diagnosed mastitic admixtures in market milk exclusively by so-called smeared sediment examinations and various volumetric methods capable of determining the number of cells per ml of milk. An account of these earlier methods is given by the Committee on Standard Methods of Bacterial Milk Analysis (Anon., 1910) appointed by the American Public Health Association. Russell & Hoffman (1907a, b; 1908) modified the earliest of these methods (Doane & Buckley, 1905, cited by Prescott & Breed, 1910) by heating the milk prior to centrifugation because they found more cells in the heated than unheated samples, a modification subsequently recommended as standard (Anon., 1910).

Examination of milk from 18 healthy cows (Russell & Hoffman, 1907a), for example, yielded the following results in terms of cell counts/ml of milk: $5\times10^4=31,1\%$; 5×10^4—$1\times10^5=18,5\%$; 1×10^5—$5\times10^5=36,6\%$; 5×10^5—$1\times10^6=10,3\%$ and $>1\times10^6=3,5\%$. Eleven cows showing various pathological conditions, but producing apparently normal milk, had corresponding values of 12,9; 15,2; 46,7; 15,0 and 9,3% respectively. The minimum and maximum values for the two groups of animals were $1,25\times10^3$ and $2,191\times10^6$ for the former and $7,5\times10^3$ and $4,952\times10^6$ cells for the latter. Other workers who elaborated on the cell count in normal milk were Doane (1905), Savage (1906), Bergey (1907), Kendall (1907), Stone & Sprague (1909) and Hewlett *et al.* (1910). Research also was performed on variations of cell counts in milk of individual quarters (Doane, 1905), individual animals (Russell & Hoffman, 1907a, b), day to day fluctuations (Doane, 1905; Stone & Sprague, 1909), foremilk, middle milk and strippings (Doane, 1905; Savage, 1906; Russell & Hoffman, 1907a, b), stage of lactation and age and breed of cow. Of particular interest is the report by Hoffman (1909) on experimental leucocytosis initiated by intramammary administration of distilled water, physiological solution of sodium chloride and boric acid.

It must be appreciated that most of these early workers had a medical background which is probably the reason why they regarded all leucocytes in milk as pus cells and evidence of inflammation. Hence, it is readily understood why the thought of ingesting these cells was repugnant.

At the termination of this decade, however, the attitude towards the presence of leucocytes in milk had changed considerably as exemplified by Miller (1909), a physician who had a very realistic approach to the significance of leucocytes and streptococci in milk. His data clearly show that leucocytes were observed in milk when histological examination of glandular tissue could not detect any evidence of inflammation. In fact, he found numerous leucocytes in the capillaries as well as in the connective tissue spaces between the alveoli of normally functioning mammary glands. Miller emphasized that although pus was present in milk from inflamed mammary glands, the leucocytes *per se* could not be regarded as deleterious and were an expected constituent of this animal product. He also referred to the contemporary belief that the demonstration of streptococci in milk invariably pointed to disease of the udder and that ingestion of such milk inevitably caused disease because the organisms were presumably capable of propagating in man. The author discussed this fallacy in the light of research which clearly demonstrated that the organisms responsible for spontaneous souring of the milk and mastitis were morphologically indistinguishable. He concluded that many leucocytes and streptococci are present in the normal milk of healthy cows, that leucocytes and streptococci are, as a rule, more numerous in the milk of diseased than healthy cows, and that unusually high leucocyte counts in milk should be followed up by veterinary examination of the herd concerned.

Concurrent to these American developments, European scientists were attempting to detect elevated leucocyte numbers in milk by indirect methods. This development apparently originated from observations on the gravity sedimentation of mastitic milk (Hess & Borgeaud, 1888; Radway, 1902), a technique which was refined by application of centrifugal sedimentation followed by the preparation of slides for microscopic demonstration of streptococci (Riddoch, 1902). Because histopathological findings showed that leucocytic infiltration was a feature of mastitis (Ibel, 1904) and increased concentrations of leucocytes were demonstrated invariably in udders with chronic mastitis or galactophoritis (Trommsdorf, 1906), the latter introduced the "milk pus test". He measured the concentration of leucocytes in 10 ml of milk by centrifugation in graduated tubes and suggested the test for milk hygiene control purposes. Milk from

healthy cows had values ranging from 0,5 to 1,0% (v/v) whereas those with a sediment of >1,0% (v/v) were considered to be mastitic. Trommsdorf (1906) also emphasized that, with few exceptions, increased numbers of leucocytes in milk usually coincided with the presence of many streptococci in milk. Because mastitis streptococci found in milk had to be considered a public health hazard and as pooled milk or milk from individual quarters were found to contain up to 2,5% (v/v) and 30% (v/v) of pus respectively, Trommsdorf (1906) warned against the consumption of unheated milk and considered large amounts of pus in mastitic milk aesthetically objectionable. Bergey (1907) confirmed the practical value of the Trommsdorf test for milk hygiene examinations and found the test more accurate for detecting mastitic milk than any other method then available.

Two years later Miessner (1909) and Trommsdorf (1909) elaborated on the advantages of the Trommsdorf test which had already become an accepted method in Europe for the diagnosis of mastitis in cows as well as for the detection of mastitic admixtures to milk supplies. However, Miessner (1909) also drew attention to data suggesting that milk may contain appreciable numbers of leucocytes in the absence of mastitis streptococci and this caused the Trommsdorf test to be seriously criticized. Trommsdorf (1909) agreed that such results may be obtained from incompletely milked cows, from those at the extremes of lactation, or those suffering from foot-and-mouth disease. He also considered the possible influence of feeding, method of milking and various diseases on the leucocyte test.

Due to these factors and instances where cases of chronic mastitis yielded milk with an increased leucocyte content on the one and a normal leucocyte content on the following day, Trommsdorf doubted the justification of an absolute leucocyte standard as proposed by American workers. He considered his test as a screening test and suggested that all positive results should be followed up by clinical and bacteriological examinations. Contrary to many of his contemporaries, Trommsdorf (1909) considered destruction of all milk containing streptococci unjustified, but supported the rejection of milk from cows with streptococcal mastitis, diagnosis of which Ernst (1909) claimed could be based with absolute certainty on capsule formation and other morphological characteristics visible on microscopic examination. On the diagnosis of human disease caused by streptococci, Savage (1909) suggested isolation of the micro-oganisms and intramammary injection of such cultures into goats. If mastitis resulted, the particular organisms were considered to be mastitis streptococci derived from bovine sources.

Concurrent with these developments, the appearance of case reports on clinical mastitis caused by mycobacteria, streptococci, staphylococci and *Escherichia coli* (Dubois, 1904; Joris, 1904; De Bruin, 1905; Carré, 1907), indicated an increasing interest in mastitis. Diphtheroid bacteria were found to be associated with teat lesions (Dean & Todd, 1902) and purulent udder secretions of mastitic cows (Glage, 1903; Künnemann, 1903). An extensively suppurating mastitis, closely resembling that caused by *C. pyogenes*, was found in heifers (Scott, 1902), and Dixson (1902) presumably dealt with clinical mastitis due to *S. aureus*.

Basic research was undertaken on the biology of *Streptococcus mastitidis contagiosa* (Stäheli, 1904) in clinical cases of mastitis, and Carré (1907) artificially infected bovine udders with streptococci. According to Zschokke (1904a) streptococci were undoubtedly the cause of garget, the mastitis resulting from galactogenous udder infections preceded by teat canal infections (Uhlmann, 1903, cited by Zschokke, 1904a). The pathogens were able to persist in the teat canal because phagocytic leucocytes were incapable of penetrating the transitional epithelium lining the teat canal. Zschokke (1904a) emphasized that udder infections by streptococci could be excluded from the diagnosis if there was no deposit in centrifuged milk and suggested that without pus there was no garget. However, on examination of udders 2–8 hours after slaughter, both normal and diseased udders were found to be inhabited by bacteria (Henderson, 1904). The bacteria seemed to exist as "epiphytes" in the teat canal whence they apparently spread into the udder cavity during periods of increased udder susceptibility. From the variety of bacteria found in the udder, Henderson (1904) concluded that they depend wholly on accidental advent from the external environment and upon their ability to exist either as "epiphytes" or as pathogens in the udder. Due to the occurrence of *Vibrio* spp. in mastitic udders the author suggested that an immense variety of pathogens are capable of inducing mastitis and that new types of mastitis will constantly emerge.

These findings were corroborated in principle by D'Heil (1906) with regard to the teat canal and teat cistern, whereas he noticed only few bacteria in the gland tissue proper, which showed a marked bactericidal effect. From his data D'Heil (1906) concluded that the teat canals of regularly milked cows contain a column of milk which forms a plug within a few days after cessation of milking and seals the teat canal. The bacterial population of this plug increases with time and the large number of bacteria found in the first jet of milk suggests the constant presence of bacteria in the teat canal. Most bacteria found in milk, however, appear to originate from inadequately cleaned milking machines.

4. *The diagnosis of mastitis between* 1910 *and* 1919

Research workers continued to be preoccupied with public health problems related to the consumption of milk (Beitzke, 1910; Helm, 1911; Kolle & Wassermann, 1913). Although mastitis streptococci were shown to be avirulent to man (Puppel, 1912), epidemics of appendicitis and parotitis (Rosenow & Dunlap, 1916) and of septic sore throat (North, White & Avery, 1914) caused controversy. *Brucella abortus*, known to cause contagious abortion in cattle (MacNeal & Kerr, 1910), was shown to be excreted in the milk of infected animals (Zwick & Krage, 1913). Investigations into the production of certified milk were conducted (Heinemann, Luckhardt & Hicks, 1910; Felix, 1916) and the bacterial content of market milk was monitored by microscopic examinations and/or colony counts (Goodrich, 1914).

The bacterial flora of normal udders was examined (Evans, 1916) and extensive studies were conducted on the aetiology of mastitis with particular reference to streptococci, staphylococci, *E. coli*, *Aerobacter aerogenes* and corynebacteria (Jones, 1918a, b, c, d).

European workers introduced the catalase test for the diagnosis of mastitis (Ernst, 1909) and continued to elaborate on this, the Trommsdorf test, pH-determinations and bacteriological culture methods (Frick, 1912; Gminder, 1912; Bahr, 1914a, b; Grinsted 1914; Höyberg, 1914). Since acute mastitis, characteristically associated with pronounced deviations of the pH, occurred less frequently than chronic mastitis,

Bahr (1914a, b) considered the catalase and Trommsdorf tests more suitable than pH determinations. Grinsted (1914) distinguished between distinct and slight mastitis and diagnosed the former by clinical examinations of tissue and secretion; for the diagnosis of the latter he found the Trommsdorf test more suitable than either pH or catalase determinations or the microscopic examination of sediment.

In America, Prescott & Breed (1910) introduced the Direct Microscopic Cell Count (DMC) method. Throughout their publication these authorsa re conspicuously noncommittal by referring to "body cells" in milk instead of the then more common reference to leucocytes or pus cells. Consequently it is presumed that the cell counts obtained by Prescott & Breed (1910) are equivalent to somatic cell counts according to present terminology. They regarded large numbers of somatic cells in milk as being hygienically undesirable because of their association with abnormal conditions of the udder. Due to the marked variability of somatic cell counts obtained from centrifuged milk, the authors concluded that cytological methods based on centrifugation are not satisfactory. In contrast, the DMC, tested on pooled milk in a series of 31 duplicate counts of 100 microscopic fields each, gave a total variation of 14,5% and a within-sample variation of ≤15%. Errors of 42,9% and 63,3% were recorded on 2 samples with $<2,5\times10^5$ cells/ml. Very few samples contained $<1\times10^5$ cells/ml; one sample of apparently normal milk showed $10,69\times10^6$ cells/ml. The average number of somatic cells in milk was $1,5\times10^6$/ml in contrast to 5×10^5/ml found previously (Russell & Hoffman, 1907a, b; 1908).

The introduction of the DMC by Prescott & Breed (1910) revealed such an inaccuracy in the older cytological methods that Breed & Stidger (1911) felt compelled to determine the number of body cells which may be expected in milk under normal and pathological conditions. The authors concluded that the number of cells in normal milk varies from almost negligible $<5\times10^4$ cells/ml, to 2×10^7 cells/ml and more. Nearly 30% of cows showed 5×10^5 cells/ml, just over 30% from $5\times10^5-1\times10^6$ cells/ml and almost 30% gave milk containing $>1\times10^6$ cells/ml. There was no evidence that pathological conditions were responsible for these high counts. Striking variations occurred from day to day. Individual cows maintained a certain constancy of cell numbers for several weeks at a time. The range of variation between individual quarters of a single udder was apparently as great as the range of variation between different cows. No constant relationship between fore-milk and middle-milk was apparent, but there was always an increase in the number of cells in the strippings when milking was complete. The relationship between these cells and pathological conditions was not at all clear (Breed & Stidger, 1911). These authors further state: "The statement that large numbers of these cells in the milk indicate a pathological condition is as unjustified by the evidence as is the converse that all pathological conditions produce an increased number of cells. Such evidence as there is indicates that very evident pathological conditions do not necessarily produce milk which has even an average number of cells for normal milk. Yet it is undoubtedly true that some pathological conditions do produce excessively large numbers of cells in milk. The evidence that there is a relation between the bacterial content of the udder, especially streptococci, and the number of these cells is very inconclusive when subjected to a critical analysis . . . there is at present no good evidence to show that the number of these cells has more than minor hygienic significance, if any at all, ..."

Ross (1912) thought it unwise to set any standard for the cell content of milk since he found considerable variations in the cell content of milk to be associated with individual cows, the stage of lactation or the production level. A viewpoint comparable with that of Ross (1912) is implied by the findings of Breed (1914), who concluded that the cellular elements in milk are a mixture of leucocytes of haematogenous origin and epithelial cells derived from the lining of the secretory portion of the udder, a situation which he regarded as perfectly normal. However, he conceded that pathological conditions could affect the discharge of these cells but states: "The cells certainly do not have the significance of pus cells under ordinary conditions nor does it seem probable that it will be possible to recognize the admixture of pathological with normal milk by means of these cells alone.....".

During this period, basic research was undertaken on the pathogenesis of streptococcal mastitis by means of artificial intramammary inoculations (Meyer, 1910). The pathology and bacteriology of various types of udder inflammation (Kitt, 1913; Wall, 1918) and the survival of mastitis streptococci and other bacteria (Frommeld, 1917) were also investigated. Data became available suggesting that mastitis not only results from galactogenous, but also from haematogenous or traumatogenous infections (Lockwood, 1913), and interest in the therapy of mastitis seemed to increase (Anon., 1913; Klein, 1914; Drennan, 1918). It is of interest to note that it was already suggested at this stage that prophylaxis, especially with regard to streptococcal mastitis, was more important than therapy (Anon., 1913).

5. *The diagnosis of mastitis between* 1920 *and* 1929

Public health considerations (Dieckerhoff, 1924; Hall, 1924; Brittelbank, 1925; Neukomm, 1927; Schultze, Schaaf & Seifried, 1927; Breed, 1928; Jordan, 1928; Rogers, 1928; Rosenau, 1928) continued to provide the incentive for an intensification of research on the control of bovine mastitis, in particular. However, more basic veterinary research on mastitis was coming into its own and can be regarded as having been inaugurated in this decade.

Steck (1920, 1921, 1922) examined the bacterial flora of normal udders. The investigations *per se* were not original, since comparable attempts had been made earlier, but the methods employed—careful aseptic sampling through the teat canal, bacteriological culture, DMC and udder palpation—seemed to justify the highly significant conclusions drawn. Steck (1922), for example, concluded that because sampling via the teat canal was performed aseptically, the bacteria isolated from normal udders are derived from the interior of the mammary gland. He found no sterile quarters, few had <10 organisms/ml, but most contained >100–1 000 organisms/ml. Such quarters differed in their cell content and were independent with regard to their respective bacterial flora, which remained sufficiently constant over months and years to permit identification of a cow by means of bacteriological results from the 4 individual quarters. He postulated that the excretion of bacteria from healthy quarters is parallelled by that of bactericidal cells and compounds, which are responsible for a balance between defence of the udder and its normal flora of bacteria such as micrococci, streptococci and corynebacteria. Because these bacteria exhibit low virulence,

they cause a very slight, chronic mastitis which can only be diagnosed by cell counts and is a normal phenomenon since it is found in otherwise normally functioning udders. However, due to incompetent milking such a normal (subclinical) mastitis may develop through various intermediate stages into the congestive (clinical) mastitis.

Harvey (1922) supported Steck by concluding from his work that because the first jet of milk almost always contains the greatest number of bacteria, teat canals and cisterns are similarly infected. Harvey further suggested that the bacterial invasion of the udder is pathological and that the condition termed mastitis is an expression of localized natural curative reactions.

From the discussion following on Harvey's paper it is also clear that the complexity of the mastitis problem was beginning to dawn on veterinarians. Most of all, their predicament appeared to be the lack of suitable remedies for the treatment of clinically acute types of mastitis. Considerable efforts were consequently made to improve mastitis therapy. In earlier decades this had consisted chiefly of oral administration of purgatives, diuretics, diaphoretics and sialagogues; external application of a variety of ointments or tinctures; intramammary administration of various antiseptics, and venepuncture (Hess & Borgeaud, 1888; Anon., 1913; Klein, 1914; Drennan, 1918). Attempts were therefore made to treat mastitis by intramammary administration of formalin (Udall, Gibbons & Bardwell, 1927) and salt solutions (Mattick & Wright, 1926). Dry cow therapy with intramammary administration of a bismuth paste and external application of tar, collodion or tincture of iodine was also advocated (Harvey, 1929). Fibrolysin therapy (Lebbin, 1924) and vaccination against mastitis (Moore, 1927; Seelemann, 1928) were tried and intramammary mastitis therapy with acriflavin derivatives was introduced (Lange, 1925; Schnorf, 1925; Schultz, 1925; Götze, 1926; Lange, 1926).

The general knowledge of conditions predisposing to mastitis (Pröscholdt, 1928), of udder diseases (Hudson, 1928), of the bacteriology of mastitis (Lamont, 1925; Minett, Stableforth & Edwards, 1929) and especially *C. pyogenes* mastitis (Jowlett, 1925; Brittelbank, 1926; Stewart, 1926) was consolidated and Seelemann (1929) introduced the modern concepts concerning coordinated mastitis control. Artificial intramammary infections with staphylococci, streptococci, *B. abortus, Pseudomonas* spp., *E. coli* and *Pasteurella* spp. furnished considerable knowledge on their pathogenicity (Carpenter, 1922).

The diagnosis of mastitis advanced considerably. Initially the merits of diagnostic procedures which relied more or less entirely either on lactose content determinations (Dieckerhoff, 1924), clinical examination (McCartney, 1924), or bacteriological data (Sheather, 1924), were emphasized. During later years particular attention was again paid to the determination of pH (Sheather, 1924; Fleischhauer, 1929; Gloy & Bischoff, 1929; Mundiger, 1929; Roeder, 1929), but this proved to be an unreliable diagnostic method (Sheather, 1924; Fleischhauer, 1929). The catalase, chlorine and chlorine & lactose tests were critically evaluated (Rühmedorf, 1927; Grubert, 1928; Seelemann, 1928) and the Trommsdorf test, performed with 30 ml instead of 10 ml of milk, was considered to be more efficient than the original since it gave a larger number of positive diagnoses (Walzberg, 1929/30).

Towards the end of the decade, some still relied solely either on clinical (Wilkinson, 1929) or bacteriological methods (Schlichting, 1927), but there was a distinct tendency to diagnose mastitis by a combination of methods. Thus Udall *et al.*, (1927) employed both udder palpation and bacteriological culture. Albiston (1927) relied on the microscopic examination of milk sediment for cells and bacteria. Pröscholdt (1928) employed sediment microscopy and bacteriological culture, a combination found to be most reliable by Seelemann (1928). Some research workers employed macroscopic examination of udder secretion in combination with pH, catalase, chloride and Trommsdorf tests and bacteriological culture (Gräub & Zschokke, 1929); others combined clinical examination of the udder with these tests, but placed particular emphasis on clinical and bacteriological data (Rüdiger & Mayer, 1929). The controversy about the most suitable methods for the diagnosis of mastitis suggests considerable confusion and uncertainty on matters related to bovine mastitis in general and the diagnosis of the disease in particular, which seemed to result mainly from variations in the terminology used to describe the various types of mastitis.

6. *The diagnosis of mastitis between* 1930 *and* 1939

Presumably the knowledge gained in the previous decade was not applied on a wide enough scale to satisfy the demands made by public health authorities, because one finds an increase in the number of publications expounding the dangers of milk-borne diseases (e.g. Brooks, 1930, 1932; 1935; Burrow, 1931; Gofton, 1931; Savage, 1931; Van Oijen, 1931; Minett, 1932; Rabagliati, 1932; Brown, 1933; Edwards, 1933b; Hofmann, 1933; Maynard, 1933; Dolan, 1935). The veterinary profession, whose competence for the hygienic control of milk had received further emphasis (Ernesti, 1930), was severely criticized for the persistently poor quality of milk supplies (Toman, 1933; Anon., 1936b). This was aptly expressed by the demand for sound milk from healthy cows (Von Ostertag, 1931). The situation was aggravated further by reports on the excretion of *Bacillus anthracis* (Weidlich, 1934), bacteria causing enteritis (Standfuss & Wilken, 1933) and *Streptococcus pyogenes humanis* (Hergestell, 1931; Dechigi, 1935) in milk. Although earlier, as well as contemporary, research had shown that *S. agalactiae* was not pathogenic to man, Haupt (1930) felt that all milk with streptococci should be considered as unfit for human consumption because some of them might well be harmful. Since Diernhofer (1930a, b), Seelemann (1930), Poppe (1931) and Seelemann & Hadenfeldt (1932b) clearly proved that *S. agalactiae* was avirulent to man, this demand was considered unjustified and flatly rejected. The vehemence of this controversy may best be gauged by referring to Skar (1928, cited by Diernhofer, 1930b) who suggested that "as a rule, rejection of all milk containing any streptococci is only demanded by those people who are incapable of completely grasping the practical implications of their demand".

Data which were accumulating on the high herd incidence of mastitis, tremendous economic losses and deleterious effects of mastitis on the dairy industry (Holford, 1930; Steck, 1932b; Rosell, 1933a; Seelemann, 1933c; Bryan, 1937; Gwatkin, Hawden & Legard, 1937; Van Rensburg, 1937; Ferguson, 1938) made the position of the veterinary profession even less enviable. Although some consolation (Ross, 1933) was derived from the limited effort made to

control mastitis (Pröscholdt, 1930; Seelemann, 1933b; Rosell, 1934; Udall, 1936b; Kästli, 1937), frustration arose from the inability to make significant progress in the control of the disease (Hodges, 1936). This is implied by the nature of mastitis research of this decade which resulted primarily in a re-assessment of problems dealt with by earlier research workers, using more advanced techniques. The object of these investigations was presumably to either confirm or refute earlier findings in terms of the contemporary knowledge so comprehensively reviewed by Munch-Petersen (1934), who concluded: "What constitutes normality so far as the bovine udder and mastitis is concerned? Nowhere in the literature perused in preparing this summary has the writer found adequate work on this fundamentally important point.... Even an adequate terminology of the complaint cannot as yet be agreed upon....".

In a detailed review Hudson (1934) dealt with the question whether to use the term "mastitis" or "mammitis" when referring to inflammation of the udder. Available general knowledge was also reviewed or summarized in several other papers (Jones, 1930; Minett, 1930, 1934; Udall & Johnson, 1930; Rosell, 1931a, b, 1933a, b; Sholl & Torrey, 1931a, b; Derrick, 1932; Udall, 1936a, 1937; Stableforth, 1939). Investigations were not arbitrarily aimed at mastitis in general. Some specificity of purpose is suggested by the work on clinical mastitis (Klimmer, 1930; Minett, Stableforth & Edwards, 1932; Stadler, 1935), subclinical mastitis (Tapernoux, 1931; Hucker, 1932b), latent udder infections (Steck, 1930a, b; Minett, 1931; Stableforth, 1931; Edwards, 1932, 1934; Hucker, 1932a; Minett *et al.*, 1932; Porcher, 1932; Seelemann & Hadenfeldt, 1932a; Stadler, 1935), non-specific mastitis (Peterson, Hastings & Hadley, 1938) and the classifications of the various types of mastitis (Minett, 1931; Stableforth, 1934; Stadler, 1935).

The pathogenicity of *S. agalactiae* and other bacteria for the udder was examined by direct intramammary inoculation (Diernhofer, 1930b) or the swab technique for inoculation of the teat canal (Hadley & Frost, 1933). From his investigations Diernhofer (1930b) concluded *inter alia* that a temporary mastitis could be caused experimentally by bacteria such as *Streptococcus lactis, E. coli* or *Aerobacter aerogenes*. Occasionally more permanent forms of mastitis were also established if the bacteria did not elicit too strong a defence reaction and survived in the udder. From their experimental work Hadley & Frost (1933) concluded that clinical examination of the udder and secretion should be employed as cow-side tests for the detection of permanent tissue alterations and the occurrence of floccules, and the pH test for alteration of the chemical composition of milk. Bacteriological examination, electrometrical pH testing, the catalase and the sediment (Trommsdorf) tests were recommended for laboratory use.

The bacterial flora of the udders of apparently healthy cows was re-examined by Ritter (1931) and Steck (1932c). The latter found that corynebacteria frequently inhabit normal udders. During mastitic episodes the numbers of these organisms decreased, probably due to some effect exercised by the pathogenic micro-organisms responsible for the mastitis. The corynebacteria re-appeared after the mastitis had subsided.

Cases of mastitis caused by streptococci were examined in detail (Roots & Karlson, 1931; Seelemann & Hadenfeldt, 1933; Klimmer & Haupt, 1935) and studies on staphylococcal mastitis were instituted (Parshall, 1934; Little & Foley, 1935; Minett, 1937). The latter organisms were recovered from normal and mastitic milk and from skin abscesses, and elicited mastitis when inoculated into the udder. Usually, the growth of staphylococci seemed to be confined to the teat canal (Little & Foley, 1935). Yeasts were found to be associated with mastitis (Derrick, 1932) and a filterable virus was isolated from a mastitic cow (Broadhurst, Cameron & MacLean, 1939).

Research showed clearly that garget did not occur in the absence of *S. agalactiae* (Seelemann, 1933a). However, elevated leucocyte counts and other secretional abnormalities also arose without concurrent streptococcal infections (Switzer & Gates, 1931; Hucker, Trudel & Jennings, 1932; Hucker, 1933; Hucker & Udall, 1933; Rosell & Miller, 1933; Klein & Learmouth, 1935; Miller, 1936; Starr, Prescott & Huffman, 1936; Hastings & Beach, 1937; Johns & Hastings, 138a, b) and milk of normal cellular composition frequently contained numerous pathogenic streptococci (Seelemann & Hadenfeldt, 1932a; Steck, 1939). The contradictory nature of these results presumably stimulated further investigations into the function of the teat canal and the histopathology of various types of mastitis. Thus Hopkirk (1934a) confirmed previous findings by Begg (1927) that a short teat canal is often associated with acute mastitis, probably secondary to a defect which permits passage of pathogenic organisms into the sinus. To his amazement he found that the apparently normal milk from less obvious cases of mastitis was heavily laden with leucocytes, streptococci, staphylococci and occasionally micrococci. On this phenomenon he remarked: "Many cases with these characteristics are not, in fact, instances of mastitis, but represent primary infection of the lactiferous duct. The infection may, and no doubt frequently does, extend to the lactiferous sinuses. If that occurs, and not otherwise, is the case truly one of mastitis". Hopkirk's lactiferous duct is undoubtedly synonymous to the *ductus papillaris mammae* or teat canal according to current terminology (Ziegler & Mosimann, 1960).

On examination of teat canals obtained from slaughtered cows and of swabs from teat canals of living cows, Hopkirk (1934a, b) found that, with the exception of corynebacteria, the organisms lodging in the teat canal were predominantly of a single type. If micrococci had taken possession of the duct, they were usually present in pure culture, and the same applied to streptococci or staphylococci. Corynebacteria, which apparently did not cause much tissue response were abundant in sloughed epithelial cells of the teat canal. On examination of teat and cisternal puncture samples, he concluded that there was an increase of leucocytes when the organisms became established in the teat canal. Leucocyte numbers decreased when the micro-organisms were destroyed. The author suggested that the micro-organisms infecting the teat canal elicit positive chemotaxis, which varies according to the organism and therefore results in a variable leucocyte response. To further quote from Hopkirk (1934a): "Much of the so-called chronic mastitis is an infection of the duct, and the ordinary cultural means employed do not demonstrate whether the gland itself is infected or not. Plating methods of examining milk and cream do show, however, that the cow is a carrier of various types of organisms found on culture and indicated by a high leucocyte count in the gravity cream....

The experiments described show that leucocytes are able to pour into the milk stream should invasion of the duct be attempted, and this is obviously the main line of defence. Leucocytes may invade the milk supply within the udder for several reasons, a slight toxic absorption from the infected area, bacterial breakdown products gaining entrance into the milk of the teat reservoir, or because of a commencing bacterial invasion of the milk in the sinuses causing alteration of pH or damage to milk producing cells". Never before had mastitis research been brought as close to an answer to the irksome question "what is mastitis" as with these observations.

From subsequent reports and the definition of mastitis suggested by the IDF (Kästli, 1967; Tolle, 1971) it is clear that the work of Uhlmann (1903, cited by Zschokke, 1904a), Hopkirk (1934a, b) and Munch-Petersen (1934) has unfortunately been disregarded in favour of previously accepted concepts pertaining to the close correlation between mastitis, elevated leucocyte counts and the almost constant presence of pathogens in the udder (Henderson, 1904; Evans, 1916; Jones, 1918d; Ayers & Mudge, 1922; Steck, 1922). The concept of bacterial infections of normal udder tissue, rather than teat canal infections, as being the precursors of septic mastitis was strengthened further by rather contradictory and/or inconclusive findings (Sholl & Torrey, 1931; Hucker, 1932a, b; Sweet, Miller & Graves, 1932; Hucker & Udall, 1933; Klimmer & Haupt, 1935; Holm, 1937; Gibbons, 1938; Morrill, 1938; Petersen *et al.*, 1938) which were accepted as evidence in favour of the contention that mastitis could only occur in the presence of bacterial infection (especially by *S. agalactiae*) of the udder parenchyma. This was apparently the reason why some workers regarded "udder infection" as synonymous to "udder inflammation" (Seelemann, 1932a, b, 1933 b; Seddon & Rose, 1934; Little, 1937a, b), a tendency supported by information obtained from studies on artificially induced streptococcal infections (Hadley, Frost, Gumm & Welsh, 1930; Schmidt-Hoensdorf & Schmidt, 1932; Seelemann & Siemonsen, 1932a, b; Edwards, 1933b; Hadley & Frost, 1933; Jones & Little, 1934; Miller, 1934; Little, 1937a, b) and from studies on the predispositions to internal udder infections (Ernst, Schmidt-Hoensdorf & Schmidt, 1931; Schmidt-Hoensdorf & Schmidt, 1932; Seelemann, 1932c; Seelemann & Siemonsen, 1932b; Rosell, 1933a, b; Miller, 1934; Anon., 1936a; Seelemann & Schaedla, 1937a, b; Peterson & Hastings, 1939). Teat and teat canal lesions were only discussed (Bendixen, 1934a, b, 1935) in terms of the formidable mechanical barrier provided by the teat canal.

The accumulation of information on internal udder infections stimulated research on intramammary mastitis therapy. Steck (1932a) administered liquid paraffin into the mammary gland and found that the resulting non-specific irritation caused clearance or depression of *S. agalactiae* infections and Toman (1933) used tuberculin for this purpose. Other research workers attempted specific cures by means of intramammary administration of ozone (Stadler, 1937), Kapff-gas (Andres, 1939) or sulphanilamide (Little, 1939). Particular attention was focussed on acriflavine derivatives (Seelemann, 1932d; Seelemann & Siemonsen, 1934; Steck, 1934a, b, 1935a; 1936a, b; 1937; Wolf & Leskien, 1937; Giebner, 1938; Klimmer & Haupt, 1938; Seelemann & Siemonsen, 1938a; Stableforth & Scorgie, 1938). Therapy of dry and lactating cows by means of acriflavine derivatives was attempted by Gerstner (1938).

Seelemann (1933b) emphasized the necessity of examining the entire herd prior to administration of mastitis remedies because success of therapy depends on the treatment of all infected cows, including those with latent infections. Due to relatively favourable results with the intramammary acriflavine therapy and advanced knowledge on the pathogenesis especially of streptococcal mastitis (Minett & Stableforth, 1931; Rautmann, 1931; Steck, Bachmann, Kästli & Gygax, 1933; Haupt, 1935; Klimmer & Haupt, 1935; Klimmer & Weitzke, 1936; Brown, 1937; Plastridge & Hartsell, 1937; Little & Minett, 1939), the salient points for the control of streptococcal mastitis, currently still applicable, were expounded (Steck, 1932a; Klimmer & Haupt, 1935) and large scale control of mastitis became feasible (Udall, 1936a, b).

Evidence on the contagious nature of mastitis due to *S. agalactiae* (Minett, Stableforth & Edwards, 1933), led to attempted prophylaxis by frequent testing and segregation of cows and by immunization against *S. agalactieae* (Krage & Gipmann, 1931; Seddon & Rose, 1934; Stableforth, Edwards & Minett, 1935; Seelemann & Siemonsen, 1938b; Seelemann, 1938; Von Sande, 1938). From experiments on the use of autogenous vaccines against streptococcal mastitis, Seddon & Rose (1934) concluded that the incidence of udder infection remained unaltered since vaccination apparently did not prevent the infection of udders by streptococci. The incidence of clinical mastitis was, however, decreased, the severity of such cases markedly reduced and the tendency to recurrent attacks of mastitis was significantly diminished. Stableforth (1939) argued emphatically against vaccination with autogenous streptococcal vaccines and expounded the distinct advantages of intramammary administration of mastitis remedies instead.

Most of this research was based on a terminology and diagnostic methods which were by no means standardized. General information on diagnostic methods employed during this decade is given by Ernesti (1930), Minett (1930), Minett, Stableforth & Edwards (1930), Roemmele (1930/31), Udall & Johnson (1931), Rosell (1931a), Seelemann (1931a, b), Switzer & Gates (1931), Götze (1932), Kiessig (1932), Porcher (1932), Schmidt (1932), Rosell (1933b), Rosell & Miller (1933), Buss & Möller (1934), Edwards (1934), Steck (1934c; 1935b), Bryan (1935), Plastridge, Anderson, Weirether & Johnson (1936) and Starr *et al.*, (1936). Until Udall & Johnson (1931) proposed a practical system of udder classification there was apparently no clinical standard method available whereby normal or diseased udders could be distinguished. The *Udall system* of udder classification, which is based on the degree of fibrosis in each udder quarter, was an important contribution. Data on age, stage of lactation and pregnancy, milk production and health of the individual cow as well as the entire herd, were also taken into consideration together with information from strip cup, thybromol and bacteriological tests.

The faith in clinical examinations was shared by Udall & Johnson (1931, 1935), Götze (1932) and Hucker & Udall (1933). Udall & Johnson (1935) found that cows, whose milk contained *S. agalactiae* or other bacteria capable of producing chronic mastitis, could with few exceptions be identified by means of udder and milk examinations made on the premises. This reliance, however, was based on rather contradictory evidence. Udall & Johnson (1931), for

example, already conceded to the presence of single, slight indurations in normal udders. Findings by Hucker & Udall (1933) were even more contradictory. These workers realized that scar tissue meant that the cow either was or had been infected, because they could not demonstrate a close relationship between streptococcal udder infections and slight or distinct fibrosis of the udder. Rosell & Miller (1933) diagnosed 65,6% of diseased quarters by means of clinical examinations alone and supported udder palpation as the most practical means for the detection of streptococcal mastitis. However, Rosell (1933b) conceded that, with the exception of typical cases of mastitis, chemical, biological and microscopic tests were superior to the clinical methods. This viewpoint was shared by many workers on account of the existence of latent udder infections or early stages of mastitis (Switzer & Gates, 1931; Porcher, 1932; Simpson, 1932; Seelemann, 1933b; Edwards, 1938; Rönnefahrt, 1938; Hadwen & Gwatkin, 1939). Consequently, the joint use of clinical examination and other methods, such as microscopic milk examinations (Steck, 1934c), bacteriological tests (Seelemann, 1932a, b; Seddon & Rose, 1934), catalase thybromol test (Münchberg, 1931), pH and rennin tests (Gwatkin *et al.*, 1937), bacteriological and pH tests (Udall & Johnson, 1930), or a wider range of laboratory tests (Römmele 1930/31; Rosell, 1931a, 1934; Götze, 1931; Edwards, 1934) were advocated.

In order to obtain the most reliable data on all types of udder infections, including latent infections, the bacteriological examination of milk samples was considered to be an essential part of mastitis diagnosis (Seelemann & Hadenfeldt, 1932a; Steck, 1939). Consequently, particular emphasis was placed on bacteriological diagnosis of mastitis in conjunction with clinical examinations (Seelemann, 1932a, b). Since udder infections were revealed more frequently by culture methods than by other laboratory procedures (Minett *et al.*, 1930; Edwards, 1932, 1934; Seelemann, 1932b; Schmidt-Hoensdorf & Schmidt, 1932; Pullinger, 1935; Bryan & Devereux, 1937; Murphy, 1939), this method of diagnosis was either regarded as most reliable (Edwards, 1934; Little, 1939) or as infallible (Bendixen 1934a, b; Engelbrecht, 1935). The importance attached to bacteriological findings was further emphasized by Hadwen & Gwatkin (1939), who concluded that normal milk must be free: "(1) from all visible forms of micro-organisms, whether they be considered harmless or not; (2) from leucocytes and fixed cells, except those that may be cast off in the normal process of milk excretion; (3) from polymorphonuclear leucocytes or other cells that occur during infection; (4) from pathogenic organisms on cultural examinations".

The processing of milk samples on selective and/or enrichment media (Clarenburg, 1930; Switzer & Gates, 1931; Edwards, 1933a, 1938; Steck, 1939) instead of solid media was promoted in order to improve the accuracy of the bacteriological diagnosis. Enrichment media in particular were found to be capable of determining the presence of very slight infections (Steck, 1939).

When enrichment media are employed the sampling technique becomes crucial. Since Steck (1920, 1921) apparently had shown convincingly that milk samples could be collected aseptically via the teat canal, subsequent workers also collected samples in this manner (Rosell, 1931a, b; Fay, Cave & Atkeson, 1938; Ferguson, 1938; Little, 1939; Steck, 1939). The precautions taken to ensure aseptic collection of such milk samples were frequently rather elaborate. For example Rosell (1931b) recommended that contamination of milk samples, collected via the teat canal, can be avoided by brushing and rubbing the cow's flanks, udder and tail with a moistened cloth; sampling should never be performed in a dusty place; the tails of neighbouring cows should be fixed; the udder should be washed with an antiseptic and after discarding the first jet of milk, samples should be collected in sterilized containers.

It is noteworthy that this absolute faith in aseptic sampling techniques via the teat canal persisted despite the reports on teat canal infections (Hopkirk, 1934a, b) and the inconclusive information on the sterility of normal udder tissue (Munch-Petersen, 1934).

The evaluation of various diagnostic methods with regard to non-specific mastitis (Switzer & Gates, 1931; Klein & Learmouth, 1935; Starr *et al.*, 1936; Johns & Hastings, 1938a, b), however, indicated that other methods (i.e. chemical, biological and microscopic cytological) were more suitable than bacteriological examinations (Rosell, 1933b). Consequently a distinction was made between screening tests, such as catalase and chlorine tests, and a confirmative diagnosis by bacteriological culture (Muchlinsky, 1930/31; Dietrich, 1932). Opinions varied appreciably on the diagnostic accuracy of chemical screening tests, such as pH, chlorine, chlorine & lactose, conductivity, rennin, iodine or milk-protein tests (Stableforth, 1930; Krenn, 1931; Schern, 1931; Hayden, 1932; Schmidt, 1932; Edwards, 1933b; Caulfield & Riddell, 1935; Tapernoux & Nicolas, 1936; Ritz, 1936; Sieber, 1937; Rowland, 1938; Rowland & Zein-el-Dine, 1938a, b). Other workers confirmed the advantages of direct or indirect leucocyte counts (Bachman, 1932; Johnson & Trudel, 1932; Hopkirk, 1933; Hadwen & Gwatkin, 1939).

At this stage it became standard practice to use individual fore-milk quarter samples, collected aseptically via the teat canal, for mastitis diagnoses (Seelemann, 1931a, b).

As indirect cytological methods, the Trommsdorf (Ehrlich, 1931; Rautmann, 1931), catalase and catalase-thybromol tests (Moser & Roeder, 1931; Rosell, 1931a, Seelemann, 1931a, b; Buss & Möller, 1934; Holm & Eveleth, 1937) were considered to posses particular diagnostic accuracy.

Whiteside (1939) reported on a new mastitis test consisting of the admixture of 2 ml N NaOH to 10 ml of milk which caused the development of viscous compounds in mastitic milk. Although Whiteside was probably unaware of the fact, this was the first step in the direction of rather intensive research on an appreciable number of new tests demonstrating elevated somatic cell counts in milk by means of increased viscosity.

With regard to the direct microscopic cytological examination of milk, earlier workers had already pointed out that certain factors are capable of causing elevated cell counts in the absence of mastitis. These observations were confirmed by Johnson & Trudel (1932), Helm (1937), Zinn (1937) and Johns & Hastings (1938b), who reported on elevated cell counts associated with advanced lactation, oestrus, milking methods, certain dietary changes, heating of milk and diurnal variations. Despite the above exceptions Hale (1939) was of the opinion that the presence of large numbers of leucocytes in milk reliably indicated the commencement of mastitis and Knoth (1930/31) emphasized that only the cytological examination of milk is capable of deciding whether

milk is suitable for human consumption. Consequently the direct microscopic leucocyte count of milk became a standard method of diagnosing mastitis (Jones & Little, 1934) and threshold values of 1×10^5 leucocytes/ml (Holm, 1934) or 5×10^5 leucocytes/ml (Rosell, 1933b; Fay *et al.*, 1938) were suggested to distinguish between normal and mastitic milk. Rosell (1933b) stated that leucocyte counts $\geq 5 \times 10^5$/ml always indicated pathological conditions of the udder, whereas Fay *et al.*, (1938) found this threshold too high since only 36,7% of samples with such counts showed streptococcal infections. Based on earlier work by Bourgeois (1927), Zinn (1937) evaluated smears from heated milk and found that the sediment of milk contained approximately 30 cells per microscopic field, whereas mastitis milk showed ≥ 100 cells per field. These values corresponded to 1–3 cells per field or 4–5 cells per field respectively in direct smears of unheated milk (Zinn, 1937). Other workers relied on microscopic examination of cream smears (Hopkirk, 1933). Although Prescott & Breed (1910) had found microscopic sediment examinations to be less accurate than the DMC, and Minett *et al.* (1930) also considered sediment or cream examinations to be inferior to other methods, the microscopic examination of sediment smears continued to be used as a simple and direct method of examining milk (Hadwen & Gwatkin, 1939). The latter also report that: "If the sediment samples are taken carefully and examined quickly, the only organisms present are those that have come directly from the udder. The arrangement of the leucocytes may be informative. For example, clumping indicates a defensive reaction; numerous lining cells indicate a destructive infection, and the causative organisms will probably be seen; phagocytic activity of the leucocytes may indicate whether a case of mastitis is in the acute or chronic phase. Other abnormalities in milk may be seen, such as blood, mucin, calcium calculi, and various micro-organisms. The concentration of the leucocytes in the sediment is sufficient in most instances to give a clear picture of the reactions that are taking place".

Due to the need for a reliable yet rapid means to diagnose all types of udder disturbances, both infectious as well as non-infectious, combined bacteriological and cytological evaluations were suggested by Little (1938). More samples could be processed accurately at lower costs by microscopic examination of the same milk or sediment smear for somatic as well as bacterial cells. Säuferlin (1930/31) examined a thick drop of milk. With regard to sediment smears, Steck (1934c) recommended examination of 100 microscopic fields and considered counts of $\leqq 5 \times 10^5$ somatic cells/ml as normal, of $\leqq 2\,000$ bacterial cells/100 microscopic fields as the threshold for slight and $\geq 2\,000$ bacterial cells as the threshold for severe udder infections. Other workers considered the concurrent appearance of increased leucocyte numbers and phagocytosed streptococci in fresh samples of milk as evidence of mastitis (Niemeyer, 1931; Rosell, 1931b; Helm, 1937; Seelemann, Wolf & Von Gartzen, 1937), whereas Clarenburg (1930) and Fay *et al.* (1938) found microscopic examination of sediment smears from preincubated milk for increased numbers of leucocytes and the presence of streptococci an efficient diagnostic approach. However, considerable difficulties were apparently encountered with the diagnosis of mastitis by sediment smears since Hadwen & Gwatkin (1939) report: "When large numbers of leucocytes are present in milk, it often means that few or no micro-organisms will be found microscopically or culturally. This has been observed in both staphylococcic and streptococcic infection... When resistance is high there will be many leucocytes and few organisms present. When many organisms and few leucocytes are found, it is probable that a new invasion of the udder will take place. Active phagocytosis indicates a return to normality".

7. *The diagnosis of mastitis from* 1940 *to date*

From the aforementioned it is apparent that the most important information which led to the current concepts on the diagnosis and definition of bovine mastitis had been established prior to 1939. The diagnostic methods employed prior and subsequent to 1939 are summarized in Appendix Table 1.

The knowledge on bovine mastitis which has accumulated since 1939 is considerable (Little & Plastridge, 1946; Merchant & Packer, 1952; Hughes, 1954; Schalm, 1962; Heidrich & Renk, 1963; Schalm *et al.*, 1971). However, since this was acquired principally by the use of methods already employed prior to 1939 and with the aid of advanced knowledge gained from related fields, it is not surprising to find that the salient research features for the period 1930 to 1939 still apply. In fact, by reconfirmation of earlier findings, current information on bovine mastitis in general seems to be regarded sufficiently dependable to justify the confidence of Weitz (1971) who states: "The activation of a nationwide scheme for the control and reduction of mastitis in our national dairy herd is a question of logistics which could be solved comparatively easily with the good will of the organizations concerned. It is rewarding to see the results of extensive research work finally realizing their practical objective. The application of the findings of such work in the field more than justifies the expense of research. The research worker has done his part effectively; it now remains for the field organisations to recognize the challenge and to put into effect the necessary measures". A comparable reliance is also implied by the proposal of an international definition of mastitis (Kästli, 1967; Tolle, 1971) which was summarized by Tolle (1971) as shown in Table 1.

TABLE 1 Assessment of cytological—bacteriological findings in mastitis diagnosis (Tolle, 1971)

Cell count/ml milk	Pathogenic micro-organisms	
	Not isolated	Isolated
$<500\,000$.....	Normal secretion...	Latent infection
$>500\,000$.....	Non-specific mastitis	Mastitis

This simplified basis for the diagnosis of mastitis facilitates the processing of large numbers of milk samples by udder health services. However, since the diagnosis of mastitis results in a chain-reaction involving considerable costs associated with treatment or culling of cows, disposal of milk contaminated with antibiotics, intensive preventive measures and mastitis research, it is apparent that a high turn-over of samples in a laboratory should be of secondary importance to the diagnostic accuracy achieved. Consequently it is essential that the IDF definition of bovine mastitis, which represents the sum of the most frequently employed clinical, bacteriological and cytological diagnostic criteria should furnish an accurate and reliable diagnosis.

The Reliability of the Current Diagnosis of Mastitis

1. *The clinical diagnosis of mastitis*

Munch-Petersen (1934), Little & Plastridge (1946) and Schalm *et al.* (1971) were undoubtedly correct in suggesting that the clinical diagnosis of mastitis is the oldest, most natural, logical and practical method to examine mastitic udders. This is borne out by the earlier data on the diagnosis of mastitis (Hess & Borgeaud, 1888; Ernst, 1909; Sanderson & Cleland, 1916; Ehrlich, 1929a, b; Gloy & Bischoff, 1929; Roeder, 1929; Laue, 1930; Udall & Johnson, 1931, 1935; Hucker, 1932a, b; Gätjen, 1933; Hucker & Udall, 1933; Kästli, 1933; Rosell & Miller, 1933). However, Murphy, Udall, Johnson & Tompkins (1944) already contradicted Udall & Johnson (1931, 1935) and Hucker & Udall (1933) by concluding that slight and even distinct indurations are not, in themselves, abnormal.

Presumably on account of the increasing frequency with which latent infections, non-specific and subclinical types of infectious mastitis were being diagnosed, a considerable change of attitude occurred towards the clinical diagnosis of mastitis. Thus Francis & Steward (1944), Breazeale, Kelly, Bartle, Hoerlein & Harschfield (1948) and Brown & Bryan (1950) still regarded clinical examination of the udder as very useful in the diagnosis of mastitis, since they could not only detect acute conditions, but also permanent udder changes due to chronic mastitis. Narayanan & Iya (1954) found that deep palpations were capable of detecting any tissue abnormalities. In contrast, Diernhofer (1950), Heidrich & Renk (1963), Kielwein & Johne (1968) and Klastrup (1970a) recommended clinical diagnosis mainly for acute mastitis and laboratory tests as supplementary methods, whereas the reverse was recommended for chronic mastitis. Schalm *et al.* (1971) suggested that there is a current tendency for clinical udder examinations to become a lost art. This tendency is made apparent by the IDF definition (Table 1) by Tolle (1971), which refers to bacteriological and cytological data as the main criteria in the diagnosis of mastitis.

Earlier recommendations to combine various diagnostic methods in order to achieve an objective diagnosis (Diernhofer, 1950; Götze, 1951; Ullner, 1959; Heidrich & Renk, 1963; Obiger, 1962b; Wendt & Leske, 1964) have therefore been waived in favour of a drastically reduced number of methods for the determination of udder inflammation as well as udder infection. There seemed to be some justification for this because the earlier reports (Seelemann, 1932 a, b; Udall & Johnson, 1931, 1935; Hucker & Udall, 1933; Murphy *et al.*, 1944) which cast doubt on the reliability of clinical examinations were also supported more recently by Heidrich & Renk (1963) who found that, with few exceptions, the physical examination of the udder is inadequate for the diagnosis of mastitis because quarters with no induration may be bacteriologically positive. These workers state: "This is not unexpected inasmuch as recent infections may not yet have elicited palpable tissue changes, and conversely, definite structural changes in the udder do not necessarily indicate an active mastitis. . ." More detailed studies on the relationship between chronic tissue alterations, somatic cell content of milk and bacterial infections support this point of view (Giesecke, Van den Heever, Hope & Van Staden, 1968). Thus the discrepancy between chronic tissue alterations and the presence or absence of inflammatory changes in the milk seems to result from the fact that in some indurated udders epithelial lesions persist whereas in others they have healed. Since inflammation in the former is still an active process, the secretion also shows inflammatory changes and the condition may be correctly diagnosed as chronic mastitis. If inflammation has subsided and scar tissue has been formed, such udders, judged by the normal somatic cell counts of milk (Giesecke, Van den Heever, Hope & Van Staden, 1968), are not mastitic, but indurated.

Two other clinical diagnostic methods are of doubtful value in cases of subclinical mastitis. Martyugin (1950), for example, reported that a drop in the yield of individual quarters was not always found to be accompanied by symptoms of "udder infection" and *vice versa*, while Reichmuth (1968) found a highly significant correlation between increased somatic cell counts and reduced production of individual quarters.

With regard to the strip-cup test, which is also a form of clinical examination, Neave, Higgs, Simpkin, Oliver & Dodd (1954) reported that small flakes do not seem to be a reliable indication of infection with streptococci or micrococci since 12–13% of quarters with such clots were not infected with the more common pathogenic organisms. Brown & Bryan (1950) and Merchant & Packer (1952) warned that the strip-cup only detects acute cases of mastitis since negative tests do not indicate absence of "infection". McFarlane, Blackburn, Malcolm & Wilson (1949) concluded that the strip-cup test permits many cases of sublinical mastitis to pass undetected, and McDonald (1955) pointed out that chronic clinical cases of mastitis show abnormal milk only at irregular intervals.

Clinical examination of the udder is a very subjective technique and depends, as pointed out by Little & Plastridge (1946) and Götze (1951), on the excellence and experience of the particular examiner. Hence there appears to be ample justification for its abandonment in extensive operations. However, the prognosis required for each individual animal and the feasibility of large scale therapy can be established only by considering laboratory as well as clinical data. Consequently it seems more reasonable to establish a diagnosis as suggested by Tolle (1971) and to examine the udders of mastitis positive cows subsequently by clinical means for prognostic purposes.

2. *The bacteriological diagnosis of mastitis*

Fresh raw milk usually contains a considerable number of micro-organisms which originate from the environment, the milker or milking utensils, the coat of the cow, the exterior and possibly the interior of the udder (Chalmers, 1955; Hammer & Babel, 1957; Judkins & Keener, 1960). The commensal bacteria found on the skin of the udder and teat are mainly micrococci (Diernhofer, 1950; Obiger, 1960, 1962a; Renk, 1966) and, to a lesser extent, certain *Staphylococcus* spp. (Davidson, 1961b, 1963; Wilson & Davidson, 1961; Orr & Taylor, 1968; McDonald, 1969, 1970b). These proliferate actively without causing any pathological changes (Davidson, 1961a). However, coagulase-positive staphylococci are not readily isolated from the normal udder skin (Slanetz & Bartley, 1953; Newbould, 1965; Cullen & Herbert, 1967; Nolting, 1968; Jasper, 1969) and according to *in vitro* tests it appears that their growth is inhibited by coagulase-negative staphylococci (Edwards & Jones, 1966; Heinemeyer, 1967).

S. agalactiae does not persist on the intact skin (Taylor, 1949; Beerwerth & Köser, 1965) but, like *S. dysgalactiae,* may grow in external lesions of the udder and teat (Taylor, 1949; Neave & Jackson, 1971). Generally *Streptococcus* spp. of groups C, E and G, especially *S. uberis,* are found more frequently on the udder than those of group B (Beerwerth & Köser, 1965; Cullen, 1966, 1969a, b).

Due to continuous contamination with soil and faeces, bacteria of the *E. coli—A. aerogenes* group and *Bacillus* spp. are often isolated from the skin of the udder and particularly from the tip of the teat (Schönberg, 1951; Dittmar, 1956; Schönberg & Kraus, 1956; Flörke, 1959).

Since contamination of milk by environmental micro-organisms is a long acknowledged fact it is not surprising that earlier and later workers alike took aseptic precautions during the collection of samples for mastitis examinations (Steck, 1920, 1939; Lamont, 1925; Rosell, 1931 a,b; Hucker & Udall, 1933; Fay *et al.,* 1938; Ferguson, 1938; Little, 1939; Little & Plastridge, 1946; Diernhofer, 1950; Merchant & Packer, 1952; Schalm, 1962; Heidrich & Renk, 1963; Schalm *et al.,* 1971).

The aseptic collection of milk samples was also recommended by Kästli (1967) and Tolle (1971) in the IDF definition of bovine mastitis. It appears that the above-mentioned workers unfortunately based their recommendations on the data established by Steck (1921, 1922), who wrongly concluded from his investigations that, since samples were drawn aseptically, the micro-organisms grown from such samples were representative of the bacterial contents of the udder parenchyma.

However, these conclusions were probably justified at the time in view of the findings by earlier workers that the lactiferous ducts harboured bacteria throughout their whole extent (Ward, 1898; cited by Hammer & Babel, 1957; Von Freudenreich, 1903, cited by Evans, 1916) and that the teat cistern and teat canal also usually contained bacteria (D'Heil, 1906). Consequently Evans (1916) considered it as conclusively proven that milk, when secreted by the glands of the healthy udder, is sterile, but becomes contaminated by bacteria normally present in all the lactiferous ducts. Further elaboration of this concept (Tanner, 1919; Fleischmann, 1922; Steck, 1930a, 1932c; Murphy, 1943) led to the following statement by Hammer & Babel (1957): "It is now generally agreed that milk contains bacteria at the time it is drawn as a result of contamination in the milk ducts and cistern. Growth of organisms in the passages during the periods between milkings provides the supply that makes possible continued contamination of milk".

Many publications have appeared pertaining to the:

i. poor relationship between mastitis and high bacterial counts (Hammer & Babel, 1957);

ii. relationship between total and mastitogenic bacterial counts (Moore, 1947, cited by Hammer & Babel, 1957);

iii. bacterial counts of milk drawn aseptically from healthy udders (Backhaus & Cronheim, 1897, cited by Birkner, 1964; Harding & Wilson, 1913; Copeland & Olson, 1926; Breed, 1928, all cited by Hammer & Babel, 1957; Wayne & Macy, 1933; Bacic, Jackson & Clegg, 1968);

iv. high bacterial counts of fore-milk (D'Heil, 1906; Harvey, 1922; Trout, 1929, cited by Faber, 1930; Munch-Petersen, Murnane & Bull, 1940; Murphy, 1943; Gadient, 1954a; Kästli, 1963b; Lerche, 1966; Bacic *et al.,* 1968; Honnens, 1968);

v. gradually diminishing bacterial counts in successive milk fractions (Russel, 1894; Backhaus & Appel, 1900; Lux, 1903; Stocking, 1906; Copeland & Olson, 1921, all cited by Birkner, 1964; Orla-Jensen, 1921);

vi. positive correlations between advancing lactational age and

(a) elevating bacterial counts (Bacic *et al.,* 1968);

(b) bacteriologically positive milk samples (Schürmann, 1964; Boge, 1965; Terplan & Honnens, 1967; Wollny, 1969; Grambow, 1970);

(c) secretional disturbances (Klein & Learmouth, 1935; Ward, Castle, How & Nielsen, 1945; Murnane, 1946; Murphy, 1946, 1947; Seelemann, 1947; Lancaster & Stuart, 1949; Ormsbee & Schalm, 1949; Spencer & Kraft, 1949; Neave, Dodd & Henriques, 1950; Oliver, Dodd, Neave & Bailey, 1956; Oliver, Dodd & Neave, 1956a, b; Plastridge, 1958; Schmahlstieg, 1960a, b; Rendel & Sundberg, 1962; Schürmann, 1964; Boge, 1965; Terplan & Honnens, 1967; Wollny, 1969; Grambow, 1970);

(d) dysfunction of the teat or teat canal (Ilgman, 1933; Little, 1937b; Glättli, 1961; McDonald, 1970a);

(e) isolations of S. *aureus* from milk samples (Boge, 1965; Schalm *et al.,* 1971); and

vii. isolation of micrococci, streptococci, corynebacteria or unidentified micro-organisms from normal udder tissue and/or their occurrence in aseptically drawn milk samples (Von Freudenreich, 1903; Harrison & Savage, 1912; Harding & Wilson, 1913, all cited by Evans, 1916; Ritter, 1931; Kowles, 1936; Jacquet, Richou, Steeg, Gerbeaux & Gerbeaux, 1952).

It would appear that at the time of Hammer & Babel (1957) the highly significant findings of Hopkirk (1934a, b) on the frequency of teat canal infections were completely forgotten.

The prevailing confidence in the reliability of the bacteriological diagnosis of the various types of bovine mastitis, including latent infections, on samples collected aseptically via the teat canal is also suggested by studies concerning:

i. the primary importance of galactogenous udder infections in the aetiology of bovine mastitis (Kitt, 1921; Steck, 1921; Frei, 1925; Götze, 1951; Pattison, 1958; Seelemann & Obiger, 1958; Renk, 1961a, b; 1962a, b; Heidrich & Renk, 1963; Kästli, 1963a; Newbould, 1964; Forbes & Herbert, 1968; Prasad & Newbould, 1968; Forbes, 1969; Hauke, Müller & Schönherr, 1969; Klastrup, 1969; Schalm *et al.,* 1971; Thomas & Thiel, 1971);

ii. the passage of bacteria through the teat canal (Davis, 1935; Johnston, 1938; Murphy & Stuart, 1953a, b; Plastridge, 1958; Bratlie, Salgsvold & Tollersrud, 1959; Teute, 1961; Stanley, Kesler & Bortree, 1962; Whittelstone & Olney, 1962; Brus, Cazemier, Jaartsveld, Politiek & De Rooy, 1964; Guss, 1964; Hauke, 1964a; Newbould, 1964; Newbould & Barnum,

1964; Guest, Stanley & Townsend, 1965; Walser, 1966a, b; Forbes, 1969; Thiel, Thomas, Westgate & Reiter, 1969; Tolle, Zeidler, Worstorff & Reichmuth, 1970); and

iii. the synonymous yet incorrect use of "udder infection" and "udder inflammation" particularly in the more recently published work on diagnosis, control and treatment of mastitis in dried-off dairy cows (Oliver, Neave & Sharpe, 1962; Neave & Oliver, 1962; Neave, Oliver, Dodd & Higgs, 1968; Dodd & Jackson, 1971; Ziv, 1971; Thomas, Neave, Dodd & Higgs, 1972) and others.

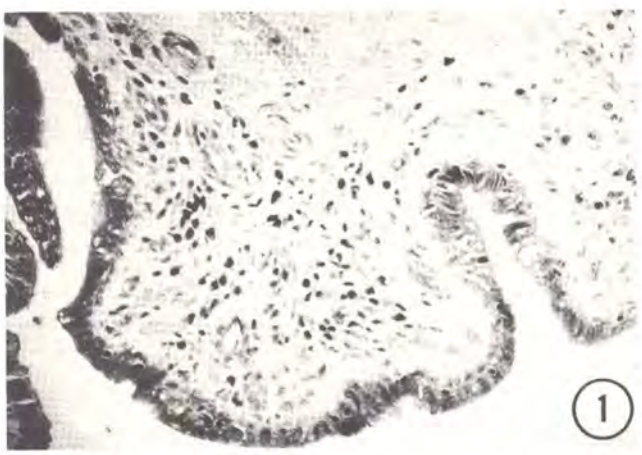
It is unfortunate that all these workers failed to prove conclusively that the bacteria isolated from samples collected aseptically via the teat canal in fact originated from udder rather than teat canal infections.

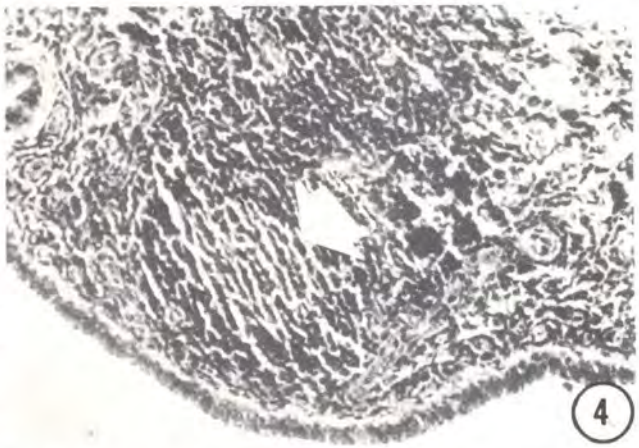
The importance of teat canal infections in the pathogenesis of mastitis (Hughes, 1953; Murphy & Stuart, 1953a, b; Sharpe, Neave & Reiter, 1962; Beech & Forbes, 1965; Forbes, 1968, 1970, a, b; Klastrup, 1969; Dodd & Jackson, 1971) suggests that this condition is very frequent. Lister (1874; 1878) and Roberts (1874) both cited by Breed (1928), Stohmann (1898), Löhnis (1910) and Kitt (1921) had already pointed out that milk in normal udders was free of bacteria and usually became contaminated on passage through the teat canal. Hopkirk (1934a, b) found that the majority of cases of mastitis diagnosed by laboratory methods were in fact teat canal infections. More recently, and prior to the first publication of the IDF standards (Kästli, 1967), Heidrich, Grossklaus & Mülling (1964) found that, depending on the efficacy of skin disinfection prior to gland cistern puncture, 20,8%; 86,2% and 100% of cistern samples from healthy udders were sterile. Birkner (1964) and Heidrich, Mülling & Birkner (1964) compared the bacterial content of samples collected aseptically via the teat canal with that of gland cistern samples and found that all (100%) of 108 teat samples, but only 7 (6,5%) of the cistern samples showed bacterial growth. Even more recently the significant differences between bacteriological findings in samples drawn aseptically via the teat canal and those drawn directly from the gland cistern were endorsed by Giesecke, Van den Heever, Hope & Van Staden, (1968). On examining 120 parallel teat and gland cistern samples they found no bacterial growth in 33,5% of the teat samples in contrast to 87,5% bacteriologically negative cistern samples. Similar results were also recorded by Wiesner (1969) and Black, Marshall & Bourland (1972). From the most recent work on teat and teat cistern samples from healthy cows and animals with subclinical mastitis (Giesecke & Viljoen, 1974), it is clear that the presence of bacteria in samples collected aseptically via the teat canal is more frequently associated with teat canal than udder infections. Since such teat canal infections often elicit a leucocytosis in the milk of the affected quarters prior to, or without actual damage to, the udder epithelium, this pre-inflammatory cellular reaction may simulate mastitis and cause some $43,13 \pm 20,8\%$ false positive diagnoses on application of the IDF standards proposed by Kästli (1967) and Tolle (1971).

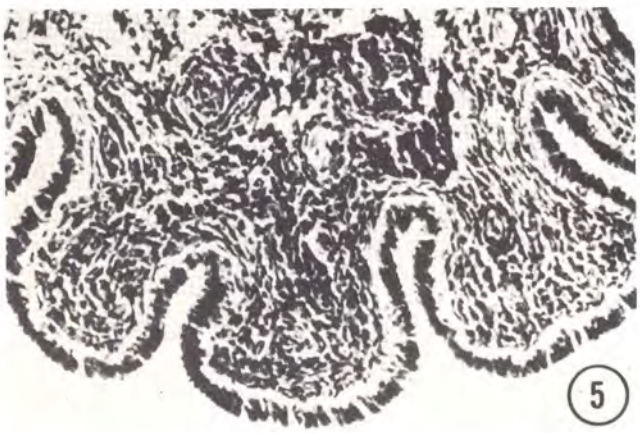
From the attempts at standardization of the definition and diagnosis of mastitis by the IDF (Kästli, 1967; Tolle, 1971) it is apparent that the findings of Stohmann (1898), Löhnis (1910), Kitt (1921), Hopkirk (1934a, b), Birkner (1964), Heidrich, Grossklaus & Mülling (1964), Heidrich, Mülling & Birkner (1964), Giesecke, Van den Heever, Hope & Van Staden, (1968) and Wiesner (1969) were disregarded. The reasons for this are probably twofold: (i) the lack of a practical method to determine teat canal infections which existed prior to the work by Giesecke & Viljoen (1974); (ii) data obtained from histological and bacteriological studies on tissue samples from slaughtered animals which indicated that there was a close correlation between bacterial isolates from teat samples and the infections of the udder cavity (Henderson, 1904; D'Heil, 1906; Runnells & Huddleson, 1925; Sholl & Torrey, 1931 a, b; Udall & Johnson, 1931; Hucker & Udall, 1933; Stradtmann, 1933; Nieberle & Cohrs, 1937; Gibbons, 1938; Morrill, 1938; Petersen *et al.*, 1938; and others reviewed by Munch-Petersen, 1934; Filion, 1948; Spencer & McNutt, 1950; Heidrich & Renk, 1963; Schalm *et al.*, 1971; Hübler, 1972). The presence of bacteria in the udder cavity, however, could also be explained by *post mortem* contamination with bacteria which spread from infected teat canals into the udder cavity by means of phagocytes, aspiration or capillary forces.

With regard to the phagocytes in the udder cavity it is unlikely that their death coincides with the death of the slaughtered animal *per se*. The physiological function of phagocytes in the secretion is in fact the maintenance of a phagocytic barrier which prevents dissemination of bacteria from the teat canal into the udder parenchyma proper. It is also conceivable that this phagocytic barrier transports limited quantities of bacterial antigen from the proximal regions of the teat canal to the diffuse lymphoid tissue surrounding the cisterns, thereby maintaining antigenic sensitization of this tissue and facilitating immune reactions. In either case phagocytes containing live bacteria might well be present in the milk of the udder cavity. This could result in the isolation of bacteria from such milk, particularly under conditions impairing the normal digestive activity of the phagocytes, e.g. pre-slaughter stress of the animal or premature death of the phagocytes. This hypothesis is substantiated by the following observations:

1. The sustained viability of leucocytes in milk removed from the udder (Nageswararao & Calbert, 1969; Lück, Giesecke & Van Tonder, 1970);
2. the leucocytic udder barrier (Schalm *et al.*, 1971) and positive chemotaxis of leucocytes elicited by teat canal infections (Hopkirk, 1934a; Giesecke & Viljoen, 1974);
3. the presence of subepithelial diffuse lymphoid tissue (Fig. 1–6) surrounding the normal gland and teat cistern;
4. local production of antibodies (Derbyshire, 1962; Campbell & Petersen, 1963; Lascelles, 1963; Campbell & Norcoss, 1964; Lascelles, Outteridge & Mackenzie, 1966; Outteridge & Lascelles, 1966, 1967; Lee & Lascelles, 1969; McDowell & Lascelles, 1969, 1971a, b; Lascelles, Mackenzie & Outteridge, 1971; Yurchak, Butler & Tomasi, 1971;
5. hypersensitivity reactions as the basis of the pathology of acute streptococcal mastitis (Zarkower & Norcoss, 1966a, b) and following intramammary administration of antigen (Lee & Lascelles, 1969);







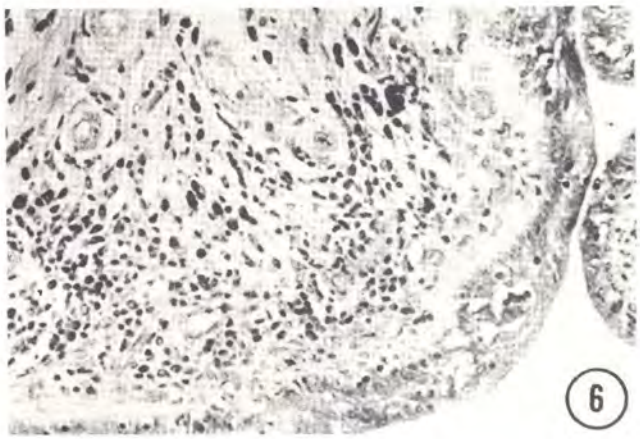


FIG. 1 Adult Friesland bull; longitudinal section through proximal portion of gland. ×200, Giemsa
FIG. 2 2 Month-old Friesian heifer calf; longitudinal section through proximal portion of gland. ×200, Giemsa
FIG. 3 6 Month-old Friesian heifer; horizontal section through proximal portion of gland. ×30, Giemsa
FIG. 4 The left solitary lymph nodule of Fig. 3 with germinal centre (see arrow). ×200, Giemsa
FiG. 5 The right solitary lymph nodule of Fig. 3 with slightly more diffuse lymphoid cells. ×200, Giemsa
FIG. 6 Lactating Friesland cow, longtudinal section through proximal portion of teat cistern. ×200, Giemsa

6. general information on the interaction between stress and infection (Selye, 1936, 1947; White & Dougherty, 1945; Mirick, 1951; Dougherty, 1952, 1953; Clawson & Neerenberg, 1953; Kass & Finland, 1953; Lurie, Zappasodi, Dannenberg & Cordona-Lynch, 1953; Kaliss, Hoecker & Bryant, 1956; White, 1963; Dougherty, Berliner, Schneebek & Berliner, 1964; Jenkins & Kruger, 1973).

The issue may also be confused by *post mortem* changes in the internal pressure of the udder and/or tone of teat canal musculature which would facilitate proximal movement of bacteria by aspiration or capillary forces. It is therefore doubtful whether research work based on dead animals can be used in this instance to elucidate subclinical mastitis in living ones.

The same applies in principle to cyto-bacteriological studies referring to "microscopically determinable pus" (Seelemann *et al.*, 1937; Schönherr, 1956; Obiger, 1962b; Lerche, 1966; Tolle, Zeidler, Heeschen, Kraack & Reichmuth, 1968). However, since such studies were usually conducted on living rather than slaughtered cattle it seems unfortunate that it was not attempted to determine whether: (i) microscopic pus was associated with damage of the udder epithelium; (ii) leucocytosis observed was pre-inflammatory or inflammatory in nature; (iii) phagocytosed bacteria originated from udder or teat canal infections.

3. *The cytological diagnosis of mastitis*

The history of the cytological examination of milk is complicated by many controversial findings. It is assumed that development of this diagnostic method resulted from the early recognition of pus cells (see pages 171–172) in milk, which frequently seemed to be associated with udder infections or with clinical types of mastitis. Consequently "pus cells" found in milk were considered to be indicative of mastitis and caused the milk to be declared as unfit for human consumption due to the repulsive "purulent" admixtures.

On comparing the early and current literature on mastitis a significant change in the actual meaning of pus cells is apparent. Whereas the pus cells in the original work of Stokes & Wegefarth (1897) were undoubtedly leucocytes, the IDF-standards (Kästli, 1967; Tolle, 1971), currently in use, are based on the somatic cell content of milk (including cells of haematogenous as well as of mammary origin) for the establishment of a cytological diagnosis of mastitis and hygienic grading of the milk (Tolle *et al.* 1968). Although the term pus cells is not used in current literature, the recommended somatic cell counts nevertheless imply that elevated numbers of such cells in milk, as determined by chemical, microscopic or electronic methods, are considered to be hygienically undesirable inflammatory admixtures. For example, Tolle *et al.* (1968) regarded increased somatic cell counts in milk as synonymous to "microscopically determinable pus" and stated: "Under consideration of physiological variations, an increased cell content in milk is to be considered as an obvious symptom of existing mastitis or secretional disturbances. Hence, the cell content represents a reliable indicator for irritation of the mammary gland. The cells concerned are leucocytic cells, glandular epithelial cells, epithelial cells from lactiferous ducts and connective tissue cells. The reliable determination of these criteria is of interest concerning mastitis diagnosis, quality grading of raw milk and various aspects of research".

The types of mastitis of major importance have also changed. During the early stages of mastitis research, particular attention was paid to clinical mastitis whereas at present sub-clinical mastitis is the main issue. Concurrently, selection of more and more sensitive cytological methods and the tendency to replace the microscopic by more convenient mechanized chemical methods and electronic cytological methods have occurred. These methods do not permit distinction between leucocytes and other somatic cells.

Cellular equivalence of DNA in milk

The determination of desoxyribonucleic acid (DNA) in milk is potentially the most accurate chemical method available at present for the indirect estimation of the number of somatic cells in milk (Hauke & Lüttigh, 1966). The bovine sperm cell, which is haploid, contains some $3{,}4\times10^{-6}$ μg DNA (Boivin, Vendrely & Vendrely, 1948). Consequently the DNA content of diploid bovine cells should be about $6{,}8\times10^{-6}$ μg DNA. According to Boivin *et al.* (1948) and Vendrely (1952) the mean value for the diploid bovine cells is $6{,}5\times10^{-6}$ μg DNA whereas it is estimated to be 7×10^{-6} and 9×10^{-6} μg DNA by De Langen (1967) and Hutjens, Schultz, Ward & Yamdagni (1970) respectively. From the above, the mean DNA value per diploid bovine cell is probably $\pm7{,}5\times10^{-6}$ μg DNA; 1×10^{6} diploid cells, as found in bovine milk, contain 7,5 μg DNA, which has been referred to as the *cellular equivalence of milk* (Giesecke *et al.*, 1973).

The use of DNA as a measure of cell numbers was first suggested by Davidson & Leslie (1950) and reports indicate a significant correlation between DNA levels of milk and cell content estimated by other methods such as the DMC, Michigan Mastitis Test, California Mastitis Test and catalase test (Paape, Synder & Hafs, 1962; Paape, Hafs & Tucker, 1963, 1964; Hauke, 1964b; Whittelstone & De Langen, 1965; Hauke & Lüttigh, 1966; De Langen, 1967; Siebels, 1969; Nageswararao & Calbert, 1969; Hutjens *et al.*, 1970). The DNA content of milk can be influenced by bacterial counts of $500–1\,000\times10^{6}$ micro-organisms/ml (Hauke & Lüttigh, 1966). Since bacterial counts of this magnitude are unlikely to be found in normal fresh milk, the DNA content of such milk must be derived primarily from intact cells. However, the macroscopically abnormal secretion of mastitic udders may also contain free DNA following cellular disintegration (Hauke, 1967). The DNA of normal milk is therefore cell-bound.

It has become customary to assess the accuracy of any new diagnostic method against the conventional DMC (Prescott & Breed, 1910). New cytological methods for mastitis diagnosis are therefore considered satisfactory if they show a significant correlation with the DMC (Bovim, 1949; Chu, 1949; Merilan, Herman, Edmonson, Taliman & Crisler, 1950; Narayanan & Iya, 1954; Seren, Ajmerito & Ferrini, 1954; Könz, 1955; Obiger, 1957; Mours & Obiger, 1960; Spencer & Simon, 1960; Dilbat, 1963; Scheidewig, 1964; Thompson & Postle, 1964; Kraack, 1965; Willits & Babel, 1965; Brazis, Reyes, Donelley & Peeler, 1965; Bieri, 1966; Whittelstone & Allen, 1966; Brazis, Reyes, Donelley, Read & Peeler, 1967; Giesecke & Van den Heever, 1967; Kroger & Jasper, 1967a, b; Luedecke, Forster & Ashworth, 1967; Miller & Kearns, 1967; Duitschaever & Ashton, 1968; Janzen, 1968; Astermark, Funke & Engan-Skei, 1969; Jensen & Beck, 1968; Jensen, Beck & Rusness, 1970; Whittelstone, Fell & De Langen, 1970; Bratlie,

1971). The same method was followed for the evaluation of modern optical and electronic counting methods (Dijkman, Schipper & Walstra, 1967; Phipps & Newbould, 1966; Tolle, Zeidler & Heeschen, 1966; Cullen, 1967a; Read, Reyes, Bradshaw & Peeler, 1967; Terplan & Schleyerbach, 1967; Dijkman, 1968; Kleinschroth, Richter & Schumann, 1968; Phipps, 1968; Dijkman, Schipper, Booy & Posthumus, 1969; Egger, 1969; Klein & Behnke, 1969; Pearson, Wright & Greer, 1970; Schultze, 1970; Seetzen, 1970; Tolle, Heeschen, Reichmuth & Zeidler, 1971).

This method of comparative evaluation has remained unaltered despite the acknowledged inaccuracy of the DMC (Prescott & Breed, 1910; Strynadka & Thornton, 1937; Leidl, Schlam & Lüps, 1961; Paape, Hafs & Snyder, 1963; Paape *et al.*, 1964; Baumgartner, 1965; Paape, Tucker & Hafs, 1965; Postle & Blobel, 1965; Schneider & Jasper, 1966a, b; Giesecke, Van den Heever, Hope & Van Staden, 1968; Hauke, 1969; Giesecke *et al.*, 1973).

However, in the absence of a reliable standard a good correlation between 2 methods does not necessarily result from true accuracy. According to Giesecke *et al.* (1973) highly significant correlations were repeatedly obtained between DMC, electronic counting (ECC) (Tolle *et al.*, 1966) and DNA content (Dische, 1930; cited by Chargaff & Davidson, 1955) when milk from cows in early to mid-lactation was tested. But with milk from cows in late lactation the correlation between the ECC and DNA and the DMC and DNA was poor compared to that of the DMC and ECC. This suggests that although there must be some relationship between ECC and DMC, it has, however, little to do with the actual somatic cell count if the latter is measured in terms of the DNA content/ml of milk. Since there is good reason to believe that DNA determinations give more accurate results than the DMC (Paape, *et al.*, 1962, 1964, 1965; Paape, Hafs & Tucker, 1963) it follows that the latter, which is the standard against which other methods are usually evaluated, probably has an error of sufficient magnitude to render the diagnosis by various direct and indirect cytological means rather empirical.

The cytological examination of milk gained a more sophisticated image by the use of the ECC, which gave the impression of greater accuracy by virtue of its excellent repeatability. The cellular DNA-equivalence of normal milk, however, suggests that electronic counting of somatic cells in milk is far from truly accurate (Giesecke *et al.*, 1973). This does not mean that the principle of electronic cell counting as such is inaccurate. The problem lies in the fact that the ECC has been standardized against the basically inaccurate DMC (Tolle *et al.*, 1966) possibly by inaccurate lower threshold settings of the Coulter Counter*.

From the above it appears that the DNA content of milk from healthy udders might be a more suitable standard than the DMC for the calibration of methods used for the counting of somatic cells in milk. Such a standard presupposes the availability of an adequately standardized method for the direct quantitative determination of DNA in milk. Hitherto DNA has been determined by the diphenylamine method of Dische (1930, cited by Chargaff & Davidson, 1955) or modifications thereof (Giles & Myers, 1965; etc.). The values obtained from bovine milk varied considerably. The DNA content of normal milk averaged $1\,540 \pm 440$ μg/ml (Siebels, 1969). Milk with an increased somatic cell content contained $4\,070 \pm 1\,470$ μg/ml, whereas milk from cases of mastitis caused by bacteria and yeasts averaged 6 000 μg/ml, and $2\,875 \pm 1\,206$ μg DNA/ml respectively (Siebels, 1969). Giesecke *et al.* (1973) obtained figures of 892 ± 316 μg DNA/ml from normal milk of cows in early to mid-lactation and $1\,725 \pm 1\,340$ μg DNA/ml from animals in late lactation. The DNA content of milk collected at these stages of lactation from cows with subclinical mastitis was $2\,803 \pm 1\,390$ μg/ml and $3\,804 \pm 2\,248$ μg/ml respectively. From these figures (Siebels, 1969; Giesecke *et al.*, 1973) it appears that milk from healthy udders in various stages of lactation may average some $2\,057 \pm 892$ μg DNA/ml.

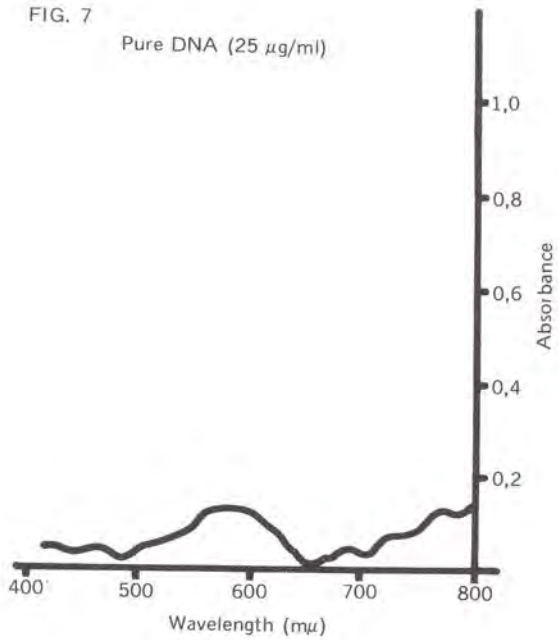
In contrast to this, a figure of 5μg DNA/ml is suggested by De Langen (1967), Hutjens *et al.* (1970) and Whittelstone, Kilgour, De Langen & Duirs (1970) for normal milk. The difference between the former and the latter values is considerable and all the more important if considered in terms of cellular DNA-equivalence of normal bovine milk. The figure of 5 μg DNA/ml implies a somatic cell count which lies within currently accepted standards for normal milk of $<1 \times 10^6$ cells/ml, whereas a minimum value of 700 μg DNA/ml suggests a cell count of close to 100×10^6 cells/ml of milk. Such high cell counts are unknown in normal milk and cast doubt on the validity of the corresponding DNA values. The discrepancies, however, may serve to accentuate some of the difficulties associated with use of the diphenylamine method, employed by all the above workers, to determine DNA in milk.

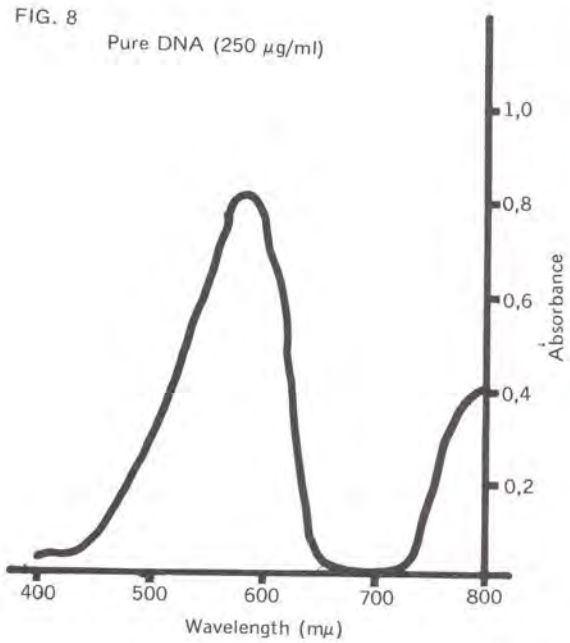
As indicated in Fig. 7–10, a possible explanation for these high values is that the diphenylamine reagent combines not only with DNA, but also with other undetermined compounds present in milk (Giesecke & Albrecht, unpublished data, 1973). With regard to the low DNA values it is not clear whether the temperature used for hydrolysis (37 °C for 20 h) by the workers concerned sufficed to break down all nuclei present in milk as would seem more likely to occur with a temperature of 100 °C for 10 min as used by Siebels (1969), Giesecke *et al.* (1973) and Giesecke & Albrecht (unpublished data, 1973). Moreover, the method used by Hutjens *et al.* (1970) results in considerable loss of DNA during filtration. The high DNA-ase activity in milk (Fig. 11–14) which is capable of diminishing the DNA content within a relatively short period of time, is also an important factor to contend with.

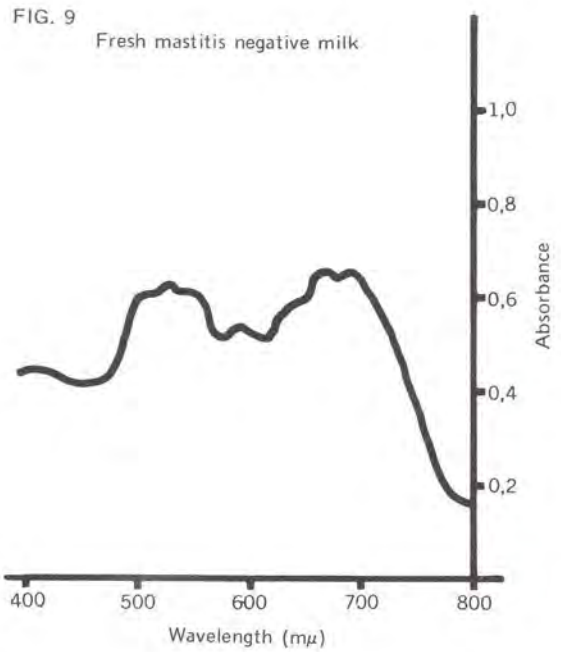
Furthermore, standardization of microscopic or electronic cytological counting methods against DNA could become an unpractical and tedious undertaking due to the wide variation in size of the cells present in milk. Darkfield fluorescence microscopy (Giesecke, unpublished data, 1972) of milk smears stained with acridin orange supports this contention. Small cellular particles are presumably equivalent to Nissen's bodies (Schönherr, 1956), whereas the larger cells represent the commonly encountered somatic cells. Because these cells all contain DNA, this would have to be considered when attempting to establish the correct correlation between the DNA content and somatic cell count of normal milk.

It is therefore clear that all data available on the DNA content of milk are unreliable and that present methods for DNA determinations in milk are in need of considerable improvement.

* Coulter Electronics







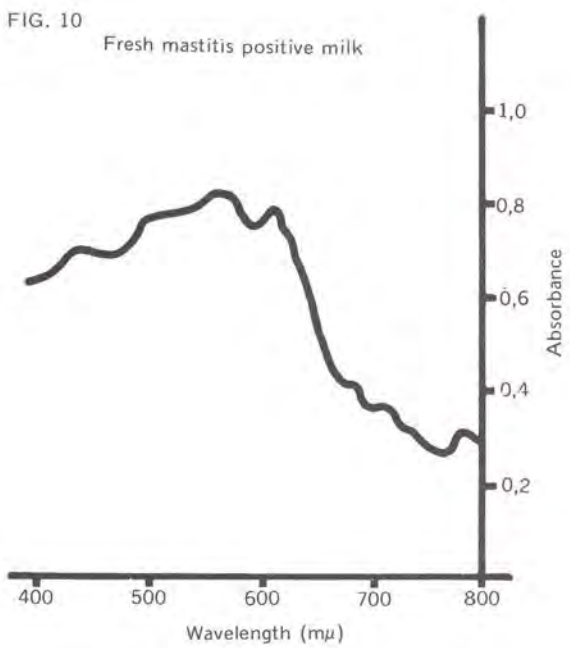


FIG. 7 and 8 Standard profiles of absorbance of pure DNA solutions containing 25 μg/ml and 250 μg/ml respectively
FIG. 9 Distinct double profile of absorbance of DNA purified from fresh mastitis negative milk suggests contamination by undetermined compounds which also react with diphenylamine
FIG. 10 Indistinct double profile of absorbance of DNA purified from fresh milk from a case of subclinical mastitis suggests contamination by undetermined compounds which also react with diphenylamine

FIG. 11

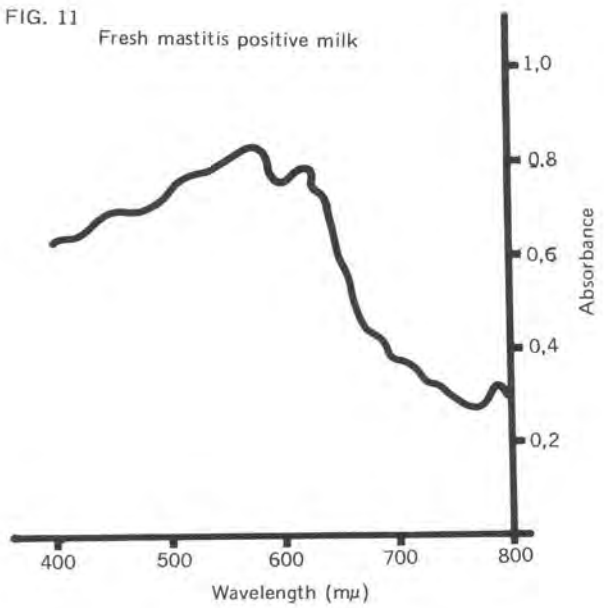


FIG. 12

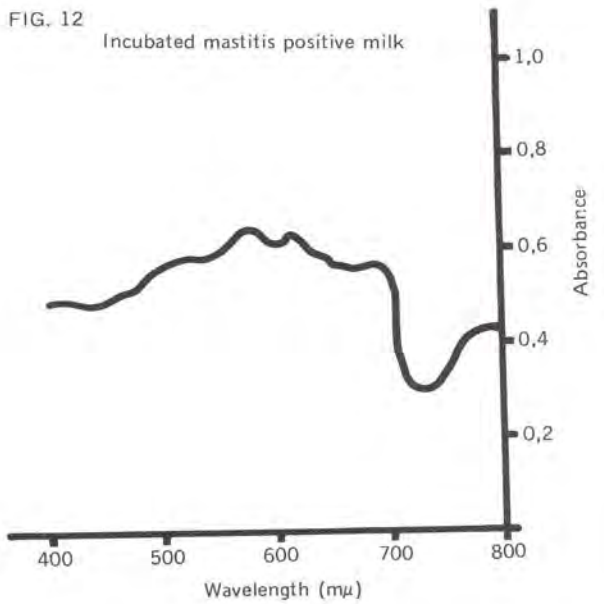


FIG. 13

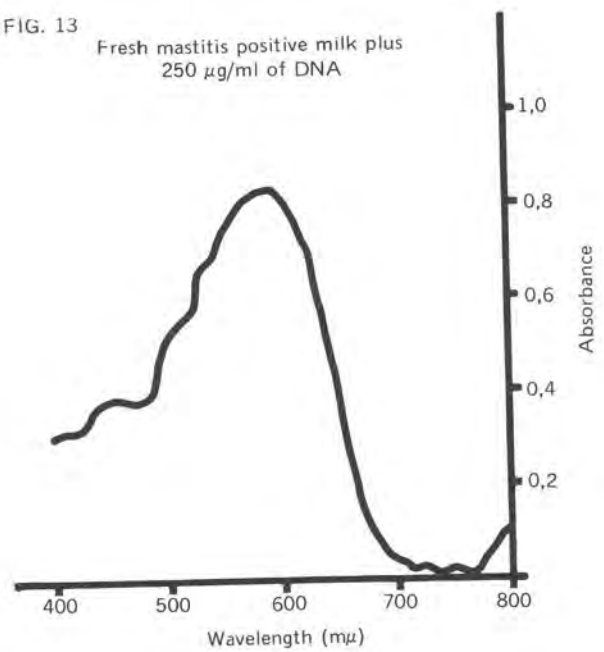


FIG. 14

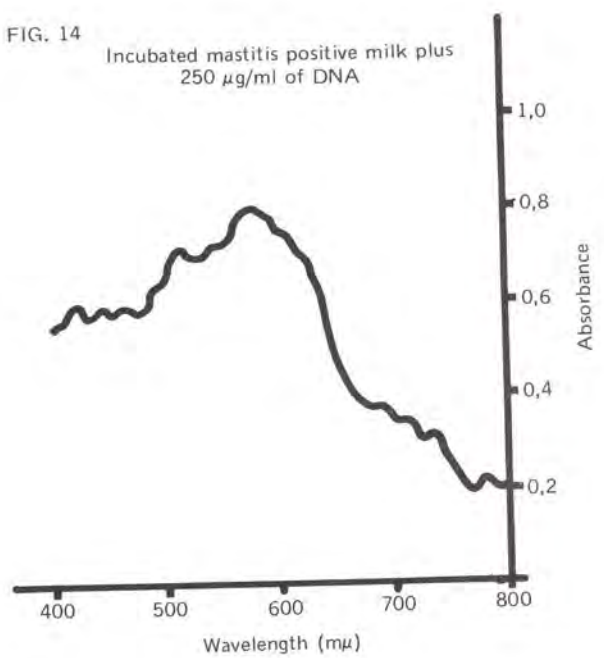


FIG. 11 and 12. The diminishing absorbance of DNA in fresh milk from a case of subclinical mastitis (Fig. 11) incubated for 120 minutes at 37 °C (Fig. 12) suggests DNA-ase activity in such milk

FIG. 13 and 14. The diminishing absorbance of 250 μg DNA/ml added to fresh milk from a case of subclinical mastitis (Fig. 13) incubated for 120 minutes at 37 °C (Fig. 14) suggests a high DNA-ase activity in such milk

Cellular fluctuations in milk

Even if the above difficulties are overcome and it is proved that DNA determination, because of greater accuracy, can replace routine cytological examinations of milk, as suggested by Hauke & Lüttigh (1966), it does not, necessarily, mean that determination of the somatic cell content is a reliable method for the diagnosis of subclinical mastitis. The reason for this is the wide range of variables to which this parameter is subject. The factors responsible for variations in somatic cell counts, even in milk from healthy udders, are too numerous and inaccurately defined to permit the application of compensating corrections during routine mastitis examination.

The following conditions are associated with variations in the somatic cell content:

(i) Lactational factors

a. fore milk samples (Steck, 1930a; Diernhofer, 1950; Hauke, 1961; Schalm, Lasmanis & Carroll, 1964; White & Rattray, 1965; Paape & Tucker, 1966; Schalm & Ziv-Silberman, 1967, 1968b; Schalm & Lasmanis, 1968);

b. strippings (Steck, 1930a; Kästli & Binz, 1948; Diernhofer, 1950; Kessler, Reid & Knodt, 1950; Hauke, 1961; Schalm, *et al.*, 1964; White & Rattray, 1965; Paape & Tucker, 1966; Mühlhäusler, 1967; Schalm & Lasmanis, 1968; Schalm & Ziv-Silberman, 1968b);

c. residual milk (Heidenreich, 1956; Smith & Schultze, 1964; White & Rattray, 1965; Paape & Tucker, 1966; Rüttiman, 1966; Natzke & Schultz, 1967; Schalm & Ziv-Silberman, 1968b);

d. different milk fractions (the cell content of cream, skim milk and sediment differs) (Prescott & Breed, 1910; Dilbat, 1963);

e. volume of milk produced (Daniel, Barnum & Rennie, 1966; Wilkens, 1968);

f. stage of lactation (Johnson & Trudel, 1932; Cone, 1944; Edelmüller, 1952; Merchant & Packer, 1952; McLeod & Anderson, 1952; MacLeod, Plastridge, Anderson, Gullet & Hale, 1953; Blackburn & MacAdam, 1954; Blackburn, Laing & Malcolm, 1955; Bratlie, 1955; Klastrup, 1960; Krieger, 1961; Braund & Schultz, 1963; Dilbat, 1963; Seelemann, 1964; Kaiser, 1965; Kraack, 1965; Blackburn, 1966; Daniel *et al.*, 1966; Daerr, 1968; Cullen, 1968; Schalm & Lasmanis, 1968; Schlüter, 1969; Tucker, 1969; Giesecke *et al.*, 1973);

g. pre-colostrum and colostrum (Rönnefahrt, 1938; Edelmüller, 1952; Schalm, 1960; Frank, 1963a, b);

h. number of lactations (Van Rensburg, 1947; Blackburn, 1956; Rendel & Sundberg, 1962; Braund & Schultz, 1963; Daniel *et al.*, 1966; Blackburn, 1968; Schalm & Lasmanis, 1968); and

i. hourly and daily fluctuations of somatic cell content (Wayne & Macy, 1933; Chu, 1949; Brown & Bryan, 1950; Gadient, 1954a, b; Narayanan & Iya, 1954; Hernholdt & Steinhardt, 1964; White & Rattray, 1965; Cullen, 1967b; Gronewald, 1967; Mühlhäusler, 1967; Schalm, 1967; Smith & Schultze, 1967).

(ii) Managemental factors

a. treatment with corticosteroid hormones (Carroll, Schalm & Lasmanis, 1965; Schalm, Lasmanis & Carroll, 1965; Wegener & Stott, 1968; Györvary, 1969; Tucker, 1969; Whittelstone *et al.*, 1970);

b. Intramammary administration of mastitis remedies or saline (Seelemann & Siemonsen, 1934; Garrison & Turner, 1936; Little & Plastridge, 1946; Götze, 1951; Keller & Boller, 1959; Cott, 1961; Obiger, 1961; Walser, 1963; Wetli, 1963; Fritsche, 1964; Katsube & Blobel, 1964; Giesecke, 1965; Natzke, Schultz & Kowalczyk, 1966; Jahnke, Wupper & Reuss, 1972; Majic, 1972; Thompson & Leaver, 1972; Neumeister, Barsch, Schmid & Kaltenegger, 1973);

c. general management (MacLeod, Plastridge, Anderson, Gullet & Hale, 1953; Elliott, 1968; Kilgour, 1968; Whittelstone *et al.*, 1970);

d. individual genetic differences (Young, Legates & Lecce, 1960; Schalm, 1962; Zeidler, Tolle & Heeschen, 1968);

e. use of milking machines (Cone, 1944; Dodd & Neave, 1951; Walser, 1966a, b; Behringer, 1967; Afifi, 1968; Miller & Finkner, 1969; Schneider, 1971);

f. mammary tissue injury caused by mechanical milking (Sørensen, 1962; Peterson, 1964; Schmidt-Madsen, 1967);

g. irregular milking intervals (Bachman, 1932; Anderson & McLeod, 1949; McFarlane *et al.*, 1949; Bogner, Burghkart & Laub, 1963; Kästli, 1963a, b; Natzke, Schultz, Barr & Holtmann, 1965; Gronewald, 1967; Daerr, 1968; Whittelstone *et al.*, 1970);

h. dietary changes (Moursy & Obiger, 1960; Obiger, 1961; Teute & Welz, 1961; Hernholdt & Steinhardt, 1964; Kraack, 1965; Daerr, 1968); and

i. movement and transport of dairy cows (MacAdam, 1954; MacLeod, Andersen & Plastridge, 1954; Daerr, 1968; Whittelstone *et al.*, 1970).

(iii) Environmental factors

a. climate (MacLeod *et al.*, 1954; Beckley & Johnson, 1966; Daniel *et al.*, 1966; Nelson, Shuh & Stott, 1967; Daerr, 1968);

b. ambient temperature (Wilkens, 1968; Whittelstone *et al.*, 1970; Zioleck, 1970);

c. premature regression due to diseases (Kurt, 1955; Nabholz, 1957; Kalich, 1958; Hoflands, 1961; Lovas, 1962; Renk, 1962a, b, c, 1966; Grunert & Weigt, 1964; Walser, 1966a, b; Heidrich & Renk, 1963; Schalm *et al.*, 1971; Theodoridis *et al.*, 1973).

(iv) Technical difficulties encountered with the cytological methods (Strynadka & Thornton, 1937; Peters & Trout, 1945; Leidl *et al.*, 1961; Paape *et al.*, 1963; Van der Schaaf, Jaartsveld & Kramer-Zeeuv, 1964; Schneider, 1965; Jasper & Dellinger, 1966; Scheider & Jasper, 1966a, b; Singh & Marshall, 1966; Tolle *et al.*, 1966; Duitschaever, Ashton & Singh, 1968; Kleinschroth *et al.*, 1968; Astermark, 1969; Tolle, Reichmuth, Zeidler & Heeschen, 1969; Zeidler, Tolle, Reichmuth & Heeschen, 1969;

Ward & Postle, 1970; Elliot & Jones, 1971; Gramatzki & Eichelmann (Udder Health Service, Lübeck, West Germany; personal communication, 1973); Merck, Raack, Kretschmer & Gramatzki (Free University, Berlin; personal communication, 1973); and

(v) a wide range of other factors discussed by Schürch (1950), Weihe (1969), Wollny (1969) and Grambow (1970).

Due to this variability of the somatic cell content, the contradictory data on the total and differential somatic cell counts of normal milk (Cooledge, 1918, cited by Thomas, 1941; Copland & Olson, 1926; Breed, 1929; Wayne & Macy, 1933; Eckl, 1937; Little, 1938; Thomas, 1941; Giesecke & Van den Heever, 1967; Guthy, Gedek, Kiermeier, Steinbrecher & Probst, 1970) and the inherent errors in the cell counting methods, resulting from the lack of a reliable standard, confidence in the cytological diagnosis of subclinical mastitis is not justified.

Cytological methods of increased sensitivity such as the Modified Whiteside Test (Murphy & Hanson, 1941), Field Whiteside Test (Schalm, Gray & Noorlander, 1955; Schalm, Pier, Gray & Noorlander, 1956), California Mastitis Test (Schalm & Noorlander, 1957), Brabant Mastitis Test (Jaartsveld, 1961), Wisconsin Mastitis Test (Postle, 1964), Aulendorfer Mastitis Test (Kielwein & Johne, 1968), and ECC (Tolle *et al.*, 1966) determine a wide variety of somatic cells, including connective tissue cells (Tolle *et al.*, 1968). The change of emphasis from clinical to subclinical mastitis (Munch-Petersen, 1934; Watts, 1940, 1944, 1949; Blackburn, 1952, 1956, 1958, 1960; Owen, 1954) together with this increased sensitivity of cytological methods has resulted in a considerable narrowing of the gap between cell counts due to true subclinical mastitis and those due to normal physiological fluctuations. This has rendered the cytological diagnosis of subclinical mastitis even more inaccurate.

An increase of the critical threshold value, which distinguishes normal from subclinically mastitic milk, would have been a reasonable attempt to retain a significant degree of diagnostic accuracy. Instead, the threshold value of only 5×10^5 somatic cells/ml, already in use at the beginning of this century (Prescott & Breed, 1910; Breed & Stidger, 1911; Ross, 1912; Breed, 1914), was adopted (Kästli, 1967; Tolle, 1971). It appears that as long as the physiological significance of somatic cells in milk is not clearly appreciated, the diagnosis of subclinical mastitis by cytological means will remain unaltered.

Physiological significance of epithelial cells in milk

From data on the physiology of lactation (Cowie & Tindal, 1971; Falkoner, 1971; Schmidt, 1971), the initiation of milk secretion at parturition (Reynolds & Folley, 1969), milk cytology (Mayer & Klein, 1961; Giesecke & Van den Heever, 1967; Schalm *et al.*, 1971) and the cell content of normal foremilk (Giesecke, Van den Heever, Hope & Van Staden, 1968), it is apparent that epithelial cells as well as leucocytes represent normal constituents of milk.

The appearance of epithelial cells in milk primarily depends on proliferative, maintenance and degenerative phases in epithelial activity, as suggested by studies on the fine structure of the cow's udder during gestation and lactation (Feldman, 1961). Studies on the effect of ephemeral fever on dairy cattle (Theodoridis *et al.*, 1973) strongly suggest that severe stress elicits premature degeneration of the lactating udder epithelium comparable to the types of the post-lactational involution described by Mayer & Klein (1961) or of the udder regression described by Hollmann & Verley (1966). This phenomenon, i.e. premature regression, may occur in appropriately stressed cows at any stage during lactation. It is slight but distinct during the ascending and marked during the descending phase of the lactation curve and is interpreted as an attempt by the cow to preserve energy. This contention agrees fully with the concept that abnormal function of the mammary gland, including mastitis, should not be regarded as a localized disease but, rather, a local expression of an environmentally and genetically induced occupational disease stressing the dairy cow *in toto* (Götze, 1951; Weigt & Aehnelt, 1965; Klastrup, 1970b). The above is also supported by general data on adaptation to stress (Selye, 1947; Jenkins & Kruger, 1973) and experiments conducted on cows by Harris (1955), Bianca (1964), Lee, Beatty & Roussel (1971), Gwazdaukas, Thatcher & Wilcox (1972), Willett & Erb (1972) and Rhynes & Ewing (1973). These workers suggested that animals react against stresses, such as temperature and humidity, by means of a non-specific defence syndrome Considerable energy leakage results from protein, especially casein, synthesis and can be limited by increased discharge of those epithelial cells which synthesize casein. Such epithelial cells, distinct from those synthesizing β-lactoglobulin, do exist in the udder (Giesecke & Osterhoff, unpublished data, 1973) and their discharge results in characteristic changes in the protein patterns of the secretions of involuting bovine udders (Giesecke & Osterhoff, unpublished data, 1973). Large numbers of degenerating epithelial cells have been found in udder secretions of involuting glands (Engel, 1953; Blackburn, 1966). The number of epithelial cells present in milk during premature regression associated with ephemeral fever was increased, particularly during the later stages of lactation (Theodoridis *et al.*, 1973). The above suffices to indicate that elevated epithelial cell counts of milk cannot be considered to be pathognomonic for subclinical mastitis or secretional udder disturbances as suggested by Tolle *et al.* (1968) or implied by the IDF-standards (Kästli, 1967; Tolle, 1971). It is presumed that epithelial cell counts in milk are also subject to the wide range of stress factors not associated with mastitis but affecting modern dairy cattle in general. In fact, premature regression, i.e. elimination of metabolically highly active cells of the udder epithelium, seems to be an essential part of the General Adaptation Syndrome (GAS) of Selye (1947). This contention is supported by elevated somatic cell counts in milk subsequent to treatments with corticosteroid hormones (Carroll *et al.*, 1965; Schalm *et al.*, 1965; Wegener & Stott, 1968; Gyövary, 1969; Tucker, 1969; Whittelstone *et al.*, 1970). Obviously the magnitude of premature regression during the GAS would not only depend on elevated blood levels of ACTH or corticosteroids as such but also on the actual dissipation of the energy made available during the alarm and resistance phases of the GAS. Hence it is understandable why somatic cell counts/ml of milk remained unaltered during investigations performed by Paape, Schultze & Miller (1973), who did not provide for appropriate dissipation of energy by the dairy cows concerned after the administration of ACTH or corticosteroids.

Physiological significance of leucocytes in milk

With regard to the leucocytes, it is known that PMN-leucocytes are part of the initial defence reaction (Anderson, 1961; Robbins, 1967; Smith & Jones, 1968). This reaction, which is a closely synchronized non-specific humoral and cellular response, is presumably an emergency reaction to prevent haemorrhage and localize, inactivate and eliminate pathogenic bacteria and their metabolites, that have penetrated interstitial tissues. This more or less instantaneous reaction also provides the time required for initiation of more specific defence reactions involving the reticulo-endothelial system.

However, conditions within the udder cavity differ from those in the interstitium. Although highly specialized to synthesize milk, the epithelial lining is a surface epithelium and is not left unprotected against bacterial destruction. The udder epithelium is in fact protected by various defence systems, the most important of all being the Leucocytic Udder Barrier (LUB) which operates beyond the epithelial barrier. The significance of the LUB has been elucidated in detail by Schalm (1970) and Schalm *et al.* (1971). The efficacy of this defence mechanism depends on the number of phagocytosing PMN-leucocytes present in milk when infection occurs (Schalm *et al.*, 1971). The number of PMN-leucocytes present in milk is subject to a wide range of factors affecting the cow *per se*, the udder in general and/or the epithelium lining the udder cavity in particular. Since infiltration of PMN-leucocytes seems largely dependent upon cellular damage or disintegration (Hesselberg & Loeb, 1937; Anderson, 1961; Robbins, 1967; Smith & Jones, 1968; Schalm *et al.*, 1971), leucocyte counts in milk are low where cellular damage in the udder cavity is slight and *vice versa*. The number of PMN-leucocytes is relatively low in fore-milk samples, apparently because the pressure within the filled udder prevents epithelial damage resulting from a mechanical stress such as intracellular biosynthesis of fat (Feldman, 1961). The percentage of butterfat is known to increase in successive milk samples and is especially high in residual milk (Smith, 1959), at a time when intramammary pressure is at its lowest (Zaks, 1962; Witzel & McDonald, 1965; Walser, 1966a). The secretion of fat globules from the udder epithelium is associated with damage to the cells concerned (Bargmann & Knoop, 1958/59; Reynolds & Folley, 1969; Cowie & Tindal, 1971). Hence apical epithelial damage increases considerably during removal of milk and the PMN-leucocyte count automatically increases at the end of a milking to reach peak levels some 4 hours later (Smith & Schultze, 1964; Schalm & Lasmanis, 1968). This post-milking leucocytosis may be regarded as an automatic and vitally important safety measure against bacterial infection of the udder. It is highly efficient if judged by the beneficial effect of frequent complete milking on mastitic udders (Heidrich & Renk, 1963). Conversely, incomplete milking of normal or mastitic udders is associated with induction of mastitis or acute flare-ups thereof respectively (Heidrich & Renk, 1963; Schalm *et al.*, 1971).

However, it should be emphasized that this defence mechanism has evolved to deal with events which occur during natural nursing of progeny, not the artificial milking act. Concurrent with the concentration of PMN-leucocytes during the milking interval (Smith & Schultze, 1964; Schalm & Lasmanis, 1968) the efficacy of the LUB is high for some hours after removal of milk and then gradually decreases. The post-milking leucocytosis therefore only safeguards the udder epithelium when milk is removed frequently. However, the number of PMN-leucocytes present in milk during milking intervals can be increased at any time by appropriate stimulation. Thus bacteria invading the udder cavity usually elicit an increase of PMN-leucocytes in excess of the physiological numbers present, i.e. the LUB is reinforced according to the defensive requirements of the udder cavity. The LUB may also be stimulated prior to bacterial infection by means of certain metabolites which, as suggested by Hopkirk (1934a, b), are released by bacteria inhabiting the teat canal or by those destroyed by the antibacterial compounds present in teat canal keratin (Adams, Rickard & Murphy, 1961; Adams & Rickard, 1963; Treece, Morse & Shah, 1964; Morse, Hubben, Treece, Willet & Platonow, 1965; Wagner, 1965; Hubben, Morse & Mealey, 1966; Treece, Morse & Levy, 1966; Treece & Morse, 1967; Hubben, Hanson, Ballard & Morse, 1968; Morse, Hubben & Mitchell, 1970; Hibbitt, Benians & Rowlands, 1972). Hibbitt & Cole (1968) and Hibbitt (1970) showed that cationic proteins of the normal teat canal keratin have a lytic effect on *S. aureus*. This micro-organism survives in abnormal teat canals for weeks (Beech & Forbes, 1965; Forbes, 1970a, b; Newbould, 1970), inhabits teat canal lesions of machine-milked cows (McDonald, 1970b), synthesizes various diffusible antigens (Elek & Levy, 1950), endotoxin (Schalm & Ziv-Silberman, 1968a), and mastitogenic exotoxins (Slanetz & Bartley, 1953; Brown & Scherer, 1958; Bernheimer & Schwartz, 1964; Stabenfeldt & Spencer, 1965; Ziv-Silberman & Risenberg-Tirer, 1970). These elicit positive chemotaxis of PMN-leucocytes (O'Gairbhidhe, Jain, Carroll & Schalm, 1970) without bacteria ever reaching the udder cavity and causing epithelial damage (Giesecke & Viljoen, 1974). A leucocytosis also occurs in response to irritation of the teat canal (Pearson *et al.*, 1971), caused either by mechanical milking (Udall, 1947; Happel, 1963a, b; Fell, 1964; McDonald, 1968a, b; 1970b; 1971; Appelman, 1969), or mastitis (Anderson, 1972).

The leucocytosis elicited by diffusible bacterial compounds or mechanical irritation of the teat canal may suffice to prevent bacterial infection emanating from the teat canal, provided the concentration of phagocytosing PMN-leucocytes in milk is high enough (Schalm *et al.*, 1971). Since this leucocytosis occurs prior to udder infection and pathological damage of the udder epithelium which truly characterizes mastitis, it has been termed pre-inflammatory leucocytosis by Giesecke & Viljoen (1974).

From these and other data on the general defensive activity of PMN-leucocytes (Anderson, 1961; Robbins, 1967; Smith & Jones, 1968) it is apparent that leucocyte counts are of limited significance in the diagnosis of subclinical mastitis in lactating cows. Though low leucocyte counts suggest the absence of mastitis, elevated counts are not pathognomonic for subclinical mastitis.

Towards the end of the lactation period the cytological diagnosis of subclinical mastitis becomes even less accurate than during lactation if judged by all the evidence on the physiological increase of somatic cells, particularly during bovine udder regression (Rönnefahrt, 1938; Edelmüller, 1952; Schalm, 1960; Frank, 1963a, b; Blackburn, 1966; Cullen, 1968; Tucker, 1969; Giesecke *et al.*, 1973). These data are further supported by comparable information (Cowie & Tindall, 1971) resulting from controlled experiments

performed on small laboratory animals (Kuramitsu & Loeb, 1921; Jeffers, 1935; Hesselberg & Loeb, 1937; Williams, 1941, 1942; Okada, 1956, 1957; Bargmann & Knoop, 1958/59; Mayberry, 1964; Verley & Hollmann, 1966; Hollmann & Verley, 1966; Lee & Lascelles, 1969; Lee, McDowell & Lascelles, 1969). It is therefore beyond comprehension how leucocyte counts, or direct and indirect estimations of the somatic cell content of udder secretions of cows at the end of lactation or during the dry period could be used as the major basis for the assessment of the currently widely acclaimed dry-cow therapy (Dodd & Jackson, 1970).

Somatic cells in milk and the diagnosis of mastitis

The available evidence indicates hat neither elevated leucocyte counts nor raised somatic cell counts in clinically normal milk are synonymous with mastitis. Neither are such counts necessarily indicative of purulent admixtures. In this regard it is particularly interesting to refer to Miller (1909), the physician, who found it scarcely reasonable to expect that a food so distinctly animal in origin as milk should contain no cellular elements. It is therefore difficult to understand why Tolle *et al.* (1968) and contemporary workers on mastitis should consider such cells (even if present in elevated numbers) in clinically normal milk as purulent admixtures, without determining conclusively that pathological damage of the epithelium of the udder is also present. Prior to Giesecke (1974), this latter most significant symptom of mastitis described concisely in mice by Chandler (1970), had been disregarded in the various definitions of bovine mastitis (Plastridge, Anderson, Williams & Hale, 1946; Zwerewa, 1970; Tolle, 1971). Nor was it attempted before Giesecke & Viljoen (1974) to diagnose such lesions by examination of milk and to assess their relationship to cellular elements of milk.

It is therefore incorrect to promote the rejection of clinically normal milk containing $>5\times10^5$ somatic cells/ml without providing conclusive evidence that the milk really does originate from udders with subclinical mastitis. The necessary proof is not provided by close correlations between elevated somatic cell counts in milk and:

i. increased incidences of udder infections (Zioleck, 1970; Booth, 1973) or subclinical mastitis (Tolle *et al.*, 1969) in the producer herds;
ii. reduced milk production in cows (Reichmuth, 1968) or altered chemical composition of milk (Van Rensburg, 1947; Kästli, 1963b; Tolle, 1970).

Such relationships can also result from environmental stress affecting the dairy cow *in toto* or individual quarters of an udder in particular. Due to the resulting premature regression a close correlation between histopathological, bacteriological and cytological subclinical changes (Hübler, 1972) must also be regarded as inconclusive evidence. This is supported by Chandler (1970), who found that "the (regressive) changes related to the secretory acinar cells (of healthy murine mammary glands) resembled in appearance a pathological degeneration ...", whilst Lee & Lascelles (1969) and other aforementioned workers on udder regression show clearly that leucocytes are abundant in the interstitium and secretion of regressive udders.

The diagnostic inaccuracy of somatic cell counts does, however, not preclude their usefulness as a convenient parameter for the detection of exposure of dairy cows to stress, thereby monitoring the efficiency of herd management and assessing relationships between management, milk yield and income over feed costs (Brown & White, 1973a,b); environment, milk production and udder conformation (Schmahlstieg, 1960a, b); ambient temperature, milk yield and composition (Lee *et al.*, 1971); mechanical milking and udder health (Schneider, 1971) and others. But without specific assessment of the health status of the epithelium of the udder, cytological data, even if supported by comprehensive statistical analyses (Zeidler *et al.*, 1968; Tolle, 1970) should not be regarded as "scientific evidence" (Tolle, 1971) for the establishment of diagnostic criteria and promoted internationally for the diagnosis of bovine mastitis (Kästli, 1967; Tolle, 1971).

Conclusions

From the review of the literature it is concluded that the diagnostic criteria used at the beginning of this century for the more advanced stages of bovine mastitis are of limited value in the diagnosis of the subclinical forms of mastitis in lactating cows, currently acknowledged to be the major problem.

Non-acute clinical changes in the udder parenchyma (i.e. fibrosis) and floccules in milk are not necessarily "macroscopic evidence of inflammation" and therefore do not provide a reliable distinction between clinical and subclinical mastitis as suggested by the IDF standards (Kästli, 1967; Tolle, 1971). Pathogenic bacteria isolated from and elevated numbers of somatic cells ($>5\times10^5$/ml) counted in clinically normal aseptic teat samples are also not necessarily pathognomonic of subclinical mastitis.

Alternative explanations for these phenomena are scar tissue in otherwise normal mammary glands, teat canal infections and lesions eliciting pre-inflammatory leucocytosis in the absence of pathological damage of the udder epithelium, and premature regression resulting from exposure of lactating dairy cattle to severe stress.

Pathological damage of the epithelium of the udder is regarded as the most significant criterium of mastitis. Current clinical, bacteriological or cytological methods applied to individual quarters can therefore only furnish valuable information if augmented by a method which assesses the physiological and pathological status of the udder epithelium as proposed by Giesecke & Viljoen (1974).

In comparison with the evaluation of individual aseptic quarter milk samples, the bacteriological and cytological monitoring of bulk milk consignments is of necessity less reliable in diagnosing mastitis. Such data could, however, be used to advantage to maintain continuous vigilance over the hygienic and managemental practices on dairy farms. High bacterial counts, including mastitogenic micro-organisms, in milk supplies could serve as an indication of inadequate methods of production and subsequent handling of milk, whereas raised somatic cell counts should be interpreted in terms of stress exposure of the cows *in toto* and the udder epithelium in particular.

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APPENDIX TABLE 1 Development or application of methods used for the diagnosis of mastitis

Authors	Year	Methods															
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Frank (cited by Baum, 1892)	1876	2+	×											2+			
Nocard & Mollereau	1884	2+	×											2+			
Bang (cited by Baum, 1892)	1888	2+	×											2+			
Hess & Borgeaud	1888	2+	2+		2+									2+			
Strebel	1888	2+	2+														
Guillebeau	1890	2+	×											2+			
Kitt (cited by Baum, 1892)	1890	2+	×											2+			
Kruger (cited by Baum, 1892)	1890	2+	×											2+			
Guillebeau & Hess	1891	2+	×											2+			
Baum	1892		2+			2+											
Kohl	1893	2+	×														
Steuert, *et al.*	1893	3+	×														
Zschokke	1897	+	2+	+									3+				
Malcolm	1898	2+	2+														
Klimmer	1900c		2+	2+	2+												
Radway	1902	2+	2+								2+	2+					
Riddoch	1902	2+	2+								2+	3+		2+			
Stäheli	1904	2+	×									2+		2+			
Zschokke	1904a, b											3+					
Doane	1905														3+		
Stewart (cited by Prescott & Breed, 1910)	1905														3+		
Savage	1906														3+		
Slack (cited by Prescott & Breed, 1910)	1906														3+		
Trommsdorf	1906										3+	3+		2+			
Bergey	1907										+	2+		2+			
Russel & Hoffman	1908											2+			3+		
Trommsdorf	1909										2+	2+					
Ernst	1909						2+							2+			
Meyer	1910		2+	2+	2+							2+					
Prescott & Breed	1910														+		3+
Frick	1912					2+					3+			2+			
Gminder	1912										3+			3+			
Kitt	1913	2+												2+			
Bahr	1914a, b					+	3+				3+						
Grinsted	1914	2+	2+			2+	2+				3+						
Steck	1920	2+	×											2+		3+	
Steck	1921	2+	×											2+		3+	
Dieckerhoff	1924								3+								
Sheather	1924		2+			+					2+	2+		3+			
Götze	1926											3+					
Albiston	1927											3+					
Rühmedorf	1927	3+						+	+			3+		3+			
Schlichting	1927	2+									+	3+					
Udall, *et al.*	1927	2+												2+			
Pröscholdt	1928											3+		2+			
Seelemann	1928						+	+				3+		2+			
Fleischhauer	1929						2+				3+			3+			
Gräub & Zschokke	1929		2+			3+	2+	2+			2+			3+			
Minett *et al.*	1929											3+		3+			
Rüdiger & Meyer	1929	2+				2+		2+	2+		2+			3+			
Wilkinson	1929	3+															
Clarenburg	1930											3+		2+			
Ernesti	1930	2+				2+								2+			
Knoth	1930/1931											3+					
Minett	1930	+	+			+					+	+		3+			
Minett, *et al.*	1930					+					+	+		3+			
Muchlinsky	1930/1931						2+	2+						2+			
Udall & Johnson	1930	3+	3+			3+								3+			
Krenn	1931							3+									
Minett	1931	2+	2+			2+					2+	2+		2+			
Moser & Roeder	1931					3+	3+										
Münchberg	1931	3+				3+	3+										
Niemeyer	1931											3+					
Rautmann	1931											3+		3+			
Rosell	1931a	2+	2+			2+	2+	2+	2+		2+	2+		2+			
Rosell	1931b											3+		3+			
Stableforth	1931		+			2+								2+		2+	
Switzer & Gates	1931	3+	+											2+			3+
Tapernoux	1931		+			2+	2+	2+	2+			2+		2+		2+	
Udall & Johnson	1931	3+	3+			2+								3+			
Dietrich	1932					2+	2+					3+					
Edwards	1932	2+	2+			2+					2+	+		3+			
Götze	1932	3+															
Hucker	1932 a, b	2+				2+		2+	2+			2+					
Kiessig	1932	2+				2+						2+		2+			
Porcher	1932					2+	2+	2+	2+								
Schmidt-Hoensdorf & Schmidt	1932	+	+											3+			
Seelemann	1932a	+										3+		2+			
Seelemann	1932b											3+		3+			
Hadley & Frost	1933	2+	2+			2+	2+				2+			2+			
Hucker & Udall	1933	2+											2+	2+			
Minett, *et al.*	1933					2+					2+			2+			
Rosell	1933a	2+	2+	+		2+	3+	3+			2+	2+		2+		3+	
Rosell	1933b		2+			2+	3+	3+			3+	2+		2+		2+	
Seelemann	1933b	+										3+		3+			
Bendixen	1934 a, b	2+												3+			
Buss & Moeller	1934					3+	3+										
Edwards	1934	2+	2+			2+					2+			3+			
Holm	1934							2+						3+		3+	
Miller	1934	2+	2+					2+	2+					2+			2+
Rosell	1934		2+			2+	2+	2+	2+			2+		2+		2+	
Seddon & Rose	1934	2+	2+											2+			
Bendixen	1935	2+				2+								2+			
Haupt	1935													3+			
Klimmer & Haupt	1935													3+			
Pullinger	1935												+	3+			
Stableforth, *et al.*	1935					2+					2+			3+			
Steck	1935b		2+		2+	2+		2+	2+		2+	2+		2+			2+
Udall & Johnson	1935	2+	2+											+			
Klimmer & Weitzke	1936													3+			
Ritz	1936	2+	2+			2+	2+					3+		3+			
Starr, *et al.*	1936	3+	2+			2+	2+	2+						2+			
Helm	1937											3+					
Little	1937 a, b													3+			
Seelemann, *et al.*	1937											3+					
Van Rensburg	1937	3+	+		+	+	+	+			+	2+	3+	3+		2+	
Zinn	1937															3+	
Edwards	1938													3+			
Fay, *et al.*	1938					+						2+	3+				2+

Authors	Year	Methods															
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Ferguson	1938													2+			
Little	1938					+		+						3+			3+
Gwatkin, *et al.*	1939	+				+								3+			
Hale	1939															3+	
Little	1939	2+	2+			2+								2+		2+	
Plastridge, Anderson, Williams & Weirether	1939		2+			2+		2+						2+		2+	
Stableforth	1939	+	+			+								3+			
Munch-Petersen, *et al.*	1940	2+	2+	2+										2+		2+	
Hadwen	1940/1941												2+				
Miller & Heishmann	1940	2+	2+	2+								3+		3+			
Ferguson	1941											+		3+			
Murphy & Hanson	1941						2+				2+			2+		2+	
Smith & Cameron	1941													3+			
Van Rensburg	1941					2+		2+					2+	2+			
Van Rensburg & Thorburn	1941	2+				2+							3+	2+			
Kleckner	1942												3+				
Murphy	1942					2+				3+				3+		2+	
Steck & Kästli	1942												3+	3+			
Murphy	1943a					2+			2+					2+			2+
Field & Smith	1944												3+	2+			
Livoni	1944													2+			
Malcolm, King & Campbell	1944	2+	2+						2+					2+		2+	
Schalm	1944											2+		2+			
Watts & Stirling	1944				+									3+		2+	
Roach	1945	2+	2+										2+				
Cunningham, Stirling, Raymont & Wilson	1946													2+			
Cunningham, Wilson, Ineson, Raymont & Mannan	1947													2+			
Monlux	1948		2+			2+	2+							3+			2+
Plastridge & Hale	1948	2+	2+										2+	2+		2+	
Spencer & Kraft	1948		2+			2+							2+	2+			2+
Chuh	1949					2+				3+				3+		2+	
Schalm, Bankowski, Ormsbee & Brownie	1949		2+			2+							2+	2+			
Dodd, Foot, Henriques & Neave	1950	2+	2+							2+				2+			
Diernhofer	1950	3+	2+	2+		2+	2+	2+			2+			3+			3+
Reid	1950												2+			2+	
Stableforth	1950					2+		2+				2+		3+			
Dodd & Neave	1951													2+			
Livoni	1953												2+	2+			
Murphy & Stuart	1953b	2+	2+											2+		2+	
Pittet	1953						2+				2+	2+		2+			
Blackburn & Mac Adam	1954													3+			
Edds & Trace	1954		2+			2+	2+							2+		2+	
Murphy & Stuart	1954a													2+			
Murphy & Stuart	1954b	2+	2+							2+				2+			
Neave, *et al.*	1954													2+		2+	2+
Klastrup & Pedersen	1955													2+			
Livoni	1955					2+								2+			
Olsen & Vindekilde	1956													2+			
Obiger	1957						2+	2+		2+		3+		3+			
Mac Adam	1958													2+		2+	2+
Sethi, Hale & Plastridge	1958												3+	2+			2+
Keller & Boller	1959									2+							2+
Lernau	1959													2+			
Slanetz	1959	2+	2+											2+		2+	
Krebs	1960					2+				2+	2+	2+		2+			
Obiger	1960													2+			
Schönherr	1960											2+		2+			
Jaartsveld	1961									2+				2+			
White & McDonald	1961									2+				2+			
Lernau & Sompolinski	1962													2+			
Klastrup	1963									2+				2+		2+	
Platnow & Blobel	1963													2+		2+	
Cazemier	1964									2+							
Cazemier, Jaartsveld, Politiek De Rooy & Brus	1964									2+							
Hickman	1964		2+							2+							
Howell, Wilson & Vessey	1964	2+	2+							2+				2+			
Jaartsveld, *et al.*	1964									2+				2+			
Schürmann	1964	2+	2+							2+				2+			
Aynsley & Buol	1965									2+				2+		2+	
Kielwein	1965													2+			
Postle & Blobel	1965						2+	2+		2+				2+		2+	
Salasci	1965									2+	2+	2+				2+	
Seelemann	1965									2+		2+					
Van den Heever	1965									2+				2+		2+	
Baumgartner & Lanz	1966									2+				2+			2+
Galli & Guallini	1966													2+		2+	2+
Post & Jaartsveld	1966	2+	×							2+				2+			
Schneider, *et al.*	1966									2+						2+	
Weigt & Möller	1966											2+		2+			
Wilson, *et al.*	1966	2+	2+	2+						2+			2+	2+		2+	
Schalm & Ziv-Silberman	1967									2+				2+			
Schleyerbach	1967									2+		2+					2+
Giesecke, *et al.*	1968	2+												2+		2+	
Kleinschroth, *et al.*	1968																2+
Roberts, *et al.*	1968													2+			×
Smith, *et al.*	1968													2+			
Bar-Moshe	1968									2+				2+			
Daniel & Seiffert	1969									2+				2+			
Dodd, *et al.*	1969		2+											2+			
Miljkovic, *et al.*	1969															+	3+
Münchberg	1969									2+				2+			2+
Neave, *et al.*	1969													2+			
Philpot	1969									2+				2+			
Probst, *et al.*	1969a													2+			×
Probst, *et al.*	1969b													2+			×
Roberts, *et al.*	1969	×	×							2+				2+			
Rosenzuaig	1969													2+			
Steele & Olsen	1969									2+							2+
Todorov	1969													2+			
Tolle	1969													2+			2+
Tolle, *et al.*	1969													2+			2+
Zeidler, *et al.*	1969													2+			2+
Kleinschroth, *et al.*	1969																2+
Ziv, *et al.*	1969													2+			
Botes	1970	2+	2+			2+	2+			2+				2+			
Brander	1970													2+			
Cargill & Bootes	1970	2+								2+				2+			
Daniel & Fielden	1970			2+						2+							

Authors	Year	Methods															
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Daniel, *et al.*	1970									2+						2+	2+
Dodd, *et al.*	1970	2+	2+							2+				2+			
Frey	1970	2+	2+							2+				2+			
Gedek & Aschenbrenner	1970													2+			
Grootenhuis	1970	2+	2+	2+										2+			
Guthy, *et al.*	1970													2+			2+
Ho	1970	2+	2+							2+				2+			
Kingwill *et al.*	1970	2+	2+											2+			
Lee & Frost	1970													2+			
McDonald	1970c									2+				2+			
Morse	1970													3+			
Natzke	1970		2+														
Pearson, *et al.*	1970									2+						2+	2+
Platonow & Kathein	1970									2+				2+			
Post	1970		2+											2+			
Reiter, *et al.*	1970													2+		2+	2+
Rosenzuaig & Mayer	1970a									2+				2+			
Rosenzuaig & Mayer	1970b									2+				2+			
Sachse	1970									2+							
Seetzen	1970											2+		2+			3+
Sullivan & Callan	1970	2+	2+											2+			
Tolle	1970													2+			3+
Tolle, *et al.*	1970													2+			2+
White	1970													2+			
Ziv	1970	2+	2+							2+				3+			
Dörrie	1971									2+		2+		2+			2+
Elliot & Jones	1971													2+		2+	
Giesecke, *et al.*	1971	2+	2+											3+			3+
Jahnke, *et al.*	1971	2+	2+											2+			
Morris & Hobbs	1971													2+		2+	
Neumann, *et al.*	1971													2+			2+
Pearson, *et al.*	1971			2+						2+							2+
Eberhart & Guss	1970									2+				2+			
Burgohain	1971	2+	2+											2+			
Wilson, *et al.*	1971	2+	2+							2+				2+			
Black, *et al.*	1972									2+				2+			
Blanc	1972	2+	2+		2+									2+			2+
Brander	1972													2+			2+
Feagan & Hehir	1972									2+				2+			
Fleet, *et al.*	1972				2+			2+	2+								
Giesecke, *et al.*	1972	2+	2+											2+			2+
Hoare & Barton	1972									2+				2+			
Hoare & Roberts	1972									2+				2+			
Jaartsveld	1972									2+							2+
Jacobs, *et al.*	1972									2+				2+			
Linzell & Peaker	1972			2+	2+				2+	2+	2+			2+			2+
Neumann	1972													2+			2+
Pearson, *et al.*	1972													2+			2+
Schulte	1972													2+			
Schultze & Smith	1972													2+			
Thornton	1972	2+	2+														2+
Wilson, *et al.*	1972	2+	2+							2+				2+			

LEGEND

Methods
1 = clinical examination of udder tissue
2 = clinical examination of udder secretion
3 = milk yield determination
4 = general chemical determination
5 = pH determination
6 = catalase determination
7 = chloride determination including conductivity
8 = lactose determination
9 = indirect cytological examination by detergent tests
10 = Trommsdorf test or equivalent thereof
11 = cyto-bacteriological examination according to or equivalent to Seeleman *et al.* (1937)
12 = cyto-bacteriological examination of pre-incubated milk samples
13 = bacteriological culture including enrichment methods
14 = PMN-leucocyte count or estimate
15 = DMC of PMN-leucocytes
16 = somatic cell count or estimate

Symbols
3+ = found to be particularly suitable or preferable
2+ = criterion is found to be suitable for the diagnosis of mastitis or it is used
\+ = found to be less suitable
× = text implies the use of this criterion