A STUDY OF BOVINE MASTITIS CONTROL IN NYERI, KENYA. //

BY

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A THESIS

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UNIVERSITY OF NAIROB

DECLARATION:

I hereby declare that this thesis is my original work and has not been presented for a degree in any other University.

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This thesis was submitted with our approval as University supervisors.

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Prof. G.M. Mugera

DEDICATION.

This work is dedicated to my wife, Dr. Jane Nyawira Maina, who in and out of sickness patiently and enthusiastically encouraged me through the period of study, and to my child Grace.

ACKNO ILEDGELIEN".

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ABSTRACT

This investigation was carried out to determine the incidence of mastitis in a specified area, King'ong'o in Nyeri, to evaluate the effectiveness of the various methods in reducing the level of mastitis under the /Kenyan environment.

One hundred milking cows were used in the investigation. This included 25 milking cows picked from each of 4 selected dairy herds. Each herd had to be in a handmilking practice. The investigation was done in two phases:-

(a) Dairy herd management was evaluated and recorded. Then, using Mastitis Indicator Test solution (MIT) on quarter milk samples, the numerical values of milk reactions were recorded for a period of sever months to establish the incidence of clinical and subclinical mastitis. Bacterial survey through culturing proceedures and sensitivity tests was done on random samples.

(b) The second phase included all the procedures in the first phase of investigation but with control methods being applied in all herds. The control methods included:-

> (i) Teat sanitation: An Iodophor solution was used in teat dipping immediately after milking.

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- (ii) Udder sanitation immediately before
 milking. The udder washing was done with
 a dilute solution of a quartenary ammonium
 detergent.
- (iii) Dry cow therapy (DCT).
 - (iv) Clinical case treatment and
 - (v) Equipment sanitation using the quartenary ammonium solution.

All these mastitis control methods were used in combination for a period of seven months. The result was a drop in subclinical mastitis incidence from 57.5 percent (from the first phase) to 37.8 percent (in the control phase). Simultaneously, there was a reduction in the incidence of clinical mastitis from 4.8 percent to 2.4 percent.

117 quarters were dried and infused with a dry cow antibiotic preparation. Of these quarters, 91 were subclinically mastitic. In the subsequent lactation only 11 were found to be subclinical. This meant that 80 quarters had been cleared of this category of mastitis. This is a 87.9 percent clearing of subclinical mastitis from the dry cows.

The most predominant bacterial isolates were: <u>Staphylococcus aureus, Streptococcus agalactiae</u> and <u>Klebsiella pneumoniae</u>. All the isolates from the clinical cases were found to be sensitive to the most commonly used antibiotics. There was clear evidence that under the conditions in which the dairy industry is operated in King'ong'o, Nyeri, with proper application of the recommeded mastitis control methods, the incidence of both subclinical and clinical mastitis should be lower than is actually observed.

However, for the control methods to be appreciated, the milkman and the farm owners should understand the basic principles of each control method and be convinced of the advantage there in. The udder and the milkman's hygiene should be further emphasised.

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INTRODUCTION.

1.

Mastitis is the inflammation of the udder (Mugera, 1979; Carrol, 1977). It is characterized by physical, chemical and usually microbiological changes in the milk and pathological changes in the glandular tissue (Blood, 1974). The characteristic changes in the milk include discolouration, the presence of clots and large numbers of leukocytes and at times erythrocytes (Schalm, 1977). The mammary gland is swollen, painful and hot in infectious mastitis. Milk production is greatly reduced (Dobbins, 1977).

Mastitis can be classified into several categories. When all of the cardinal signs of inflammation are readily apparent, this form is called peracute mastitis. In addition, there are accompanying systemic signs of fever, depression, shivering, loss of appetite and rapid loss of weight. Acute mastitis is also characterized by the five listed signs of inflammation with, generally, an accompanying fever and drop in milk production. When the cardinal signs of mastitis are subdued and not accompanied by systemic effects, the mastitis is called subacute. The existence of infection in the abscence of gross signs is referred to as a latent infection (Schalm, 1977).

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Milk is a major product in the livestock industry and is widely consumed in Kenya and the world at large. Milk is thus of economic and nutritive importance in the country. However, if the udder is infected, alot of pathogenic microorganisms could be consumed with the milk. When there is mastitis, milk producion is drastically reduced (Shaw and Beam, 1935; Dobbins, 1977).

No work has been done on the effects of mastitis in the Kenyan environment. Work carried out in Kenya has mainly concentrated on identification of causative organisms and classification of types of mastitis that exist (Hamir, 1978: Hamir, 1979). Secondly, the countrywide incidence of mastitis in Kenya has not been documented in detail. No reports are available for the incidence of mastitis in the Central province of Kenya, where the investigation was done. The microorganisms causing mastitis differ from country to country and from one climatic region to another in regard to strains present or even susceptibility to antimicrobial agents. It is important to have this data in relation to drawing control measures or a control program (Dodd, 1977).

In this investigation, some recommended measures of mastitis control by way of hygiene (Teat, Udder and personell sanitation) and treatment (Clinical case and dry cow therapy) were applied to some selected animals.

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The clinical and subclinical quarter cases were recorded numerically as evaluated using Mastitis Indicator Test, before, during, and after the control period. Using standard laboratory culture methods, quarter samples were analysed from each of clinical, subclinical and clean quarters to show the causative agents involved. The bacteria isolated were then analysed for antimicrobial agent sensitivity.

During the investigation, however, some constraints were foreseen:-

(a) To be able to realize lowered subclinical infection, the stipulated time of one year was not enough or adequate. Further investigation would need to be undertaken.

Mastitis control is a long-term program, but improvement should be evident in 6 months; although three years or more are needed for maximum effect (Jasper, 1983.

(b) The sample size would not be as large as may be necessary due to:- time (as in (a) above), the laboratory's processing capacity and personnel limitations.

(c) The investigation would very expensive if all the subclinical mastitis quarters were to be infused with an antibiotic (Blitz therapy). Only the clinical cases would be infused with the appropriate antibiotic. However during the start of the dry period all quarters would be infused with a dry cow antibiotic preparation.

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2 LITERATURE REVIEW.

Mastitis has been defined as the inflammation of the mammary gland regardless of the cause. With inflammation, there is found physical, chemical, and usually microbiological changes in the milk and also pathological changes in the glandular tissue. The inflammation can be caused by many reasons. The commonest is bacterial infection (Ogaa, 1981).

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The typical pathological change in milk is increased number of somatic cells, particularly white blood cells. This is brought about by the emigration of these leukocytes into the udder and milk as the body's natural defence force fight the infection. Usually the farmer is drawn to a mastitic problem by the changes in milk that are physical including: changes in the colour and the consistency of milk, presence of clots in milk, or reduction in the amount of milk produced. This is referred to as "Clinical Mastitis" (Schalm, et al., 1971).

A large proportion of mastitic glands are not evident by visual examination or by manual palpation of the gland. This is inapparent to the farmers. It is referred to as "Subclinical Mastitis"[.] (Schalm, <u>et al.</u>, 1971). However, it is detectable most easily by the estimation of the number of leukocytes in milk. The number of leukocytes can be quantified chemically. The use of a strip cup in the first strips of quarter milk, can detect presence of clots or flakes. The subclinical mastitis affects the performance of the mammary gland quarter in varying degrees, and the state is a dynamic one progressing to a clinical state in one extreme and clearing naturally on the other extreme. The effects of subclinical mastitis are therefore difficult to evaluate either on the udder health or on the dairy industry's economy.

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Some inflammatory processes persist over many months or from one lactation to the next and are referred to as "Chronic Mastitis". These types exist for most part in a subclinical form with periodic flare-ups producing subacute or acute clinical signs which commonly subside shortly there after reverting to the subclinical forms.

2.1. INCIDENCE OF SUBCLINICAL MASTITIS.

Mastitis is frequently encountered in exotic breeds of cattle in Kenya, where the climatic conditions make it possible to rear these animals (Hamir <u>et al.</u>, 1979). In most countries, surveys of the incidence of mastitis, irrespective of cause, show comparable figures of about 40 percent morbidity amongst cows and a quarter incidence of about 25 percent. The incidence is similar in goats and buffaloes kept in dairies (Kalra et al., 1964). A survey of the incidence of bovine mastitis made during the year 1971 to 1973 in the Kabete area of Kiambu district, Kenya (Laverman, et al., 1973), showed the incidence of clinical mastitis to be 2.5 to fax. 3.0 percent per annum, whereas the incidence of subclinical mastitis was about 48 percent (Hamir et al., 1979) and 49 percent (Lauerman, et al., 1973).

In the ambulatory practice area of the Faculty of Veterinary Medicine, clinical mastitis forms about 12 percent of the total patient load; the incidence of the disease clinically in other high potential dairy practice areas of Kenya is estimated to range from 3.5 to 25 percent (Anon, 1980).

From the annual report of the Veterinary practice in the Central Province districts of Kenya, the incidence of clinical mastitis is high (Appendix 1). This agrees with the report of Hamir <u>et al</u>. (1979) that bovine mastitis is the most encountered clinical disease in Kenya.

The incidence of mastitis, at clinical and subclinical level in Kenya has not really been worked out and although there has been some studies on mastitis in countries with farm practice similar to those found in Kenya (Frost, 1962., Kalra and Dhanda, 1964), research in this country has been very limited (Hamir et al., 1979).

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2.2. DETECTION OF SUBCLINICAL MASTITIS.

In the diagnosis of subclinical mastitis the methods of diagnosis aim at revealing one or more of the features of inflammation i.e. presence of leukocytes, fibrin clots and change of milk to a more watery form. Also used is the detection of changes in chemical composition of milk due to supression of secretion. This is followed by the transfer of sodium chloride and bicarbonate from blood to milk bringing about a shift of pH to an alkaline level.

The first of these detection methods, was developed as early as 1916 (Moak, 1916). Since then the list has enlarged to include the following:-

- (a) The strip cup(Moak, 1916).
- (b) The pH determination using:-
 - (i) Bromocresol purple developed in
 1919 (Baker and Van Slyke, 1919)
 and (Baker and Breed, 1920).
 - (ii) Bromothymol blue....(Fay et al.,
 1938., Hayden and Johnson, 1934).
- (c) Milk chloride detection.... (Hammer and Bailey, 1917., Rosell, 1932).
- (d) Catalase test in milk.....(Merchant and Packer, 1944., Monlux, 1948., Spencer and Simon, 1960).

(e) Whiteside test......(Whiteside, 1939).

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- (f) The California Mastitis tests... (Schalm and Noorlander, 1957).
- (g) The Brabant Mastitis test(Jaartsved, 1961).
- (h) The Wisconsin Mastitis test....(Postle, 1964).
- (i) The cell count (Blackburn, 1968).
- (j) Prescott and Breed count (Prescott and Breed, 1910).
- (k) Electronic cell counter (Phipps and Newbould, 1966).

While all these methods are used to some extent today, tests that directly detect the presence of increased numbers of somatic cells in milk, primarily the leukocytes, due to inflammation, have assumed a position of greater importance (Schalm <u>et al.</u>, 1971). These tests are also capable of detecting mastitis in its advanced stages.

In Kenya, only the strip cup and California Mastitis test are used. However, their use is also limited (Hamir <u>et al.</u>, 1979). The ambulatory service at University of Nairobi, Kenya, uses the California Mastitis test (CMT) for teaching purposes and detection of the subclinical mastitis. However, the Veterinary Practitioner in Kenya does not use any of these methods (Kenya annual report, 1982). Few samples from clinical mastitis are submitted to the diagnostic laboratories for microbial cultures and drug sensitivity tests.

2.2.1. The strip cup.

This procedure, developed by Moak, (1916) is used for examination of fore milk for evidence of mastitis. The first streams of milk are drawn into a pail covered by a 100 mesh brass wire. Small flakes of milk and larger clots are held back by the mesh and are readily visible. Modification of the method to use hand held cup became known as the strip cup (Schalm <u>et al.</u>, 1971). The strip cup is illustrated in appendix 2.

Examination of milk as it flows over a black surface has advantages over the wire mesh screen in that watery milk is readily detectable. In chronic mastitis (Schalm <u>et al.</u>, 1971) a watery appearance of the first stream of milk is just as important a diagnostic sign as flakes or clots in milk.

To be effective as a means for the detection of mastitis the strip cup should be used at every milking as an intergral part of the milking routine. The strip cup serves a number of useful purposes; namely:-

- (a) The detection of abnormal milk,
- (b) Removal of the first milk aiding in stimulation of milk let down, and
- (c) Discarding of the fore milk leading to a lower bacteria count, since the first milk commonly has the largest number of bacteria per millilitre.

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Although the routine use of a strip cup oy the milker should be encouraged, it is not sufficiently sensitive for mastitis detection to be solely depended upon as the method of diagnosis.

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2.2.2. California martitis test (CMT).

A French physician, Alfred Donne (Schalm, 1971), is credited with the observation that pus in urine can be detected indirectly by adding a crystal of either sodium or potassium hydroxide to centrifuged sediments (Donne, 1957). In 1939, Whiteside, reported that on the addition of 2 cc NaOH to 10 cc of the milk from cows suffering from mastitis, and subsequently beating of the mixture with a glass rod, a viscid mass was formed. Murphy and Hanson, (1941) modified the test to what is in use to-day, and suggested the designation, "Modified Whiteside Test". On fresh warm milk, 2 drops of NaOH are added to 5 drops of the test milk. In the state of New York, the modified whiteside test is employed as a screening test for milk quality control on the blended milk of entire herds. On bulk farm milk, trace reactions begin at the level of about 500,000 cells per millilitre (Schalm, 1971). Studies on the nature of the whiteside test reaction clearly show that fresh somatic cells are necessary for the reaction to take place (Dunn et al., 1943., Petersen et al., 1950, and Schalm and Noorlander, 1957).

One explanation that has been given as a basis for the reaction is that the nucleic acid forms a sodium salt in the presence of NaOH producing a gelatinous mass to which serum solids and fat globules become absorbed to produce the characteristic precipitate of the reaction (Whiteside, 1939). Increasing intensity in the modified test compared well with increasing total somatic cell count and polymorphonuclear (PMB) leukocyte count per millilitre of milk.

Beginning in March 1955, a large scale whiteside testing program was initiated in Sacrament County California (Schalm <u>et al.</u>, 1956). Although correlation was good between results with the original and modified whiteside tests, the former was not quite as sensitive for the detection of milk having somatic cell counts of 1 million per millilitre or less. It was suggested that a surface active agent added to sodium hydroxide would enhance the rupture of somatic cells and cause flakes and shreds to become more prominent. An anionic surface active agent, alkyl arylsulfonate was found to improve materially the field whiteside test.

A solution consisting of 0.5 percent alkyl arylsulfonate and 1.5 percent NaOH proved to be much more sensitive in detecting abnormal cell counts.

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This combination was designated formula 2 (Schalm et al., 1971). Later on results using formula 2 were found to vary with the fat content in milk. Further study revealed that 3 percent alkyl arylsulfonate reacted with mastitic milk without involving the milk fat in the visible reaction. The indicator dye bromocresol purple was added in final concentration of 1:10,000. The dye colour changes towards deep purple as the pH of the milk increases in mastitis. The colour reaction is especially useful to indicate mammary quarters that have depressed secretory activity. To draw attention to the fact that the method was entirely new, the method was designated California mastitis test or CMT (Schalm and Noorlander, 1957).

Investigations into the nature of the positive CMT reaction led to the conclusion that the active principle in mastitis milk was deoxyribonucleic acid (DNA) from the nuclei of somatic cells (Carroll and Schalm, 1962., Jaartsveld, 1961, and Paape <u>et al.</u>, 1963). The proof rests on the fact that nucleated but not non-nucleated red blood cells added to normal milk produced the typical CMT reaction (Schalm <u>et al.</u>, 1971). Further proof that the CMT reaction is a test for DNA is found in the close parallel between CMT positive results on milk and positive DNA specific Feulgen reaction (Paape <u>et al.</u>, 1963). The discovery of this anionic detergent that reacts with mastitis milk to produce a visible effect that can be scored

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numerically with reference to content of somatic cells made possible the introduction of a new and practical test for mastitis that could be:-

- (a) concluded at the side of the cow at the milking parlour.
- (b) performed in the laboratory on mixed milk from the four mammary quarter's bucket samples and on blended milk taken from the farm bulk tank.

A number of modifications of CMT have been putforward under different names. Milk quality test (MQT) is the same as CMT with methylene blue dye replacing the indicator bromocresol purple. Mastitis Indicator Test (MIT) is also similar to CMT but without any dye incoorporated. Both MQT and MIT do not evaluate pH of the milk by colour changes.

When CMT is applied to individual mammary quarters, it is common practice to conduct it on the first streams of milk at milking time to assess the health status of separate quarters of the lactating cow. Such milk gives positive reactions with more intensity and the reactions tend to weaken or become negative in succeeding streams. This is especially true of glands in which inflammation is slight (Schalm et al., 1971).

2.3. AFTIOLOGY OF SUBCLINICAL MASTITIS.

Many kinds of bacteria, fungi and yeast can produce mastitis, and more than one organism can be present in one herd and even in different quarters of the same cow (Mugera <u>et al.</u>, 1979). Survey of the various infections in cattle show a great deal of similarity in different countries (Blood and Henderson, 1974). A list of organisms that have been associated with bovine mastitis is given in appendix 3. However, no viruses have been shown as causative agents of bovine mastitis. Work done by Fetlow and Ferer (1982), did not show significant association between bovine leukemia virus and mastitis.

There are predisposing factors that render cows more prone to infection by certain microorganisms. These are:- unsatisfactory milking machines and poor milking techniques; exposure to cold and wet weathers; blows, horn injuries, bruises, abrasion of the udder and wounds on teats. Also of consequence are irregular and incomplete milking, the introduction of unsterilized instruments up the teat canal and any systemic distubances of animal's health. Large pendulous udders are more prone to injury and consequent infection (Schalm <u>et al.</u>, 1971., Blood and Henderson 1974., Mugera <u>et al.</u>, 1979., Norman, 1973., and Weisner, 1974).

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No work has been done on the incidence of subclinical or clinical mastitis or actiological agents from the geographical area of the present investigation. The practising veterinarians do not go into the causes for mastitic cases attended. However, at the Veterinary Investigation Laboratory Karatina, the annual reports (Kenya annual report 1982(b) give figures on isolates from the milk samples submitted for diagnostic purposes (appendix 4).

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2.4. CLINICAL AND TATHOLOGICAL FINDING IN SUBGLINICAL MATITIS.

Inflammation in a lactating gland is unique in that products of inflammation enter the milk and are readily available for study (Schalm, 1977).

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There may be all degrees of variation in signs from the gradual onset of fibrosis, through acute inflammation without systemic signs, to severe toxaemia with systemic signs in clinical mastitis. Subclinical mastitis however, is characterized by finding of products of inflammation in milk and a reduction in milk production that is not obvious to detect (Stang, 1977 and Schalm <u>et al.</u>, 1971). The condition of the udder at a given time depends upon the resistance of the mammary tissue, the virulence of the invading bacteria, (Blood and Henderson, 1974) and sometimes the number of microorganisms that have access to the mammary tissue (Schalm <u>et al.</u>, 1964., Neave <u>et al.</u>, 1950).

Subclinical mastitis, a disease that is inapparent, is to-day diagnosed most populary and with a high degree of accuracy by methods that detect presence of inflammatory cells (Jain et al., 1969., Jain <u>et al.</u>, 1971., Schalm <u>et al.</u>, 1971). Variation in somatic cell numbers in different fractions of milk (Carrol <u>et al.</u>, 1963., Paape <u>et al.</u>, 1963., Smith and Schultze, 1967) is of use in early detection of subacute mastitis (Schalm, 1977) and can be so used to position the site of pathology in the affected gland (Blood and Henderson, 1974).

The first pathologic change in cows during mastitis is passage of serum albumin from blood to milk. This is a direct result of increased capillary permeability (Carrol and Jain, 1969). Sodium chloride and Sodium bicarbonate follow, causing a shift in pH towards alkalinity. Synthesis of lactose is suppressed in propotion to the concentration of sodium entering the milk. This is a mechanism designed to maintain isotonicity of the altered udder fluid with blood. Synthesis of casein is reduced, resulting in a fall in total nonfat solids.

Emigration of neutrophil leukocytes into milk can be demonstrated within three hours from beginning of an inflammatory process (Carrol et al., 1963). Evidence suggests that the neutrophil leukocyte is involved in initiation of this inflammation process in bovine udder (Jain et al., 1968). Experimentally, it had been concluded that in the early phase of acute inflammation the neutrophil leukocyte is an important agent influencing the magnitude and duration of the

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inflammation process (Jain <u>et al.</u>, 1969; and Jain <u>et al.</u>, 1971). Fibrin escaping into milk is converted to fibrin strands which enmesh leukocytes, epithilial cells, bacteria, and other debris to form flakes and clots. Flakes and clots are easily detected at subclinical level by a strip cup (Moak, 1916).

Injury is an important predisposing factor to infectious mastitis (Norman, 1973). It is suggested that the response of the neutrophil leukocytes to injured tissue is the initial step upon which the vascular permeability change is dependent for its development (Ramsey and Grant. 1974). The suggestion is based on evidence that cows experimentally made neutropenic are unable to respond to an udder irritant in the normal manner. Cardinal signs of mastitis do not develop at all or are delayed in neutropenic cows. Leukocyte emigration appears to lag behind the change in vascular permeability. This is due to the time required for neutrophil leukocyte to migrate throu n tissue into the milk, whereas serum albumin readily filters through altered capillary cells (Jain et al., 1968).

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2.5. IMMUNITY OF THE MAMARY GLAND TO MASTITIS.

At present, work is going on by way of research to establish the importance of intrinsic and extrinsic factors of immunity in the mammary gland. For this reason some information is included in this work to give some background on the control of mastitis.

The mammary gland is an external body surface at the skin. The protective system at body surface is equipped with physical defence mechanism and further re-enforced by a surface immunoglobulin system, designated the secretory immunoglobulin (S.I.) system. The skin on the udder, acts as a mechanical barrier to microorganisms that would otherwise gain access to the udder. It also produces chemicals that prevent the proliferation of pathogenic bacteria on this body surface. The udder, has a flushing activity provided when the milk flows out during milking or suckling (Jean-Francois 1982).

2.5.1. Mucosal immunoglobulin system (MIS).

Four of the antibody classes are involved in the mucosal immunity, i.e. IgN, IgE, IgC and IgA. (Bourne and Newby, 1981).

In most domestic species, IgG is the major immunoglobulin, present in higher concentration in colostrum than in serum (Newby <u>et al.</u>, 1982). In contrast, in man and in rodents, IgA predominantes in colostrum with much smaller amount of IgG. IgG and IgM do appear in intestinal secretions, IgG mediating a number of immune reactions (Bourne and Newby, 1981). The principal subclass of IgG in colostrum is IgG₁, and it is known to be derived from serum during colostral formation (Larson and Kendall, 1957).

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IgG₁ is the principal immunorlobulin cleo in mil-(Mach and Pahud, 1971), while IgG_2 , IgM and IgA are present in small amounts (Merby <u>stal.</u>, 1982). The IgG_1 is derived from serum and is concentrated many times above serum levels (Pierce and Feinstein, 1965). Only small proportions of the IgA and IgM present in colostrum are drawn from local synthesis within the mammary gland (Newby and Bourne, 1977). Since over 80 percent of the milk immunoglobulin is serum derived, in contrast to other species, there is no significant local immune synthesis in the udder of the cow (Newby <u>et al.</u>, 1982).

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IgA is also a major immunoglobulin of secretions (Bourne and Newby, 1981), secreted by the B lymphocytes underlying the mucosal epithelium. The IgA is selectively transported across the epithelial cell-wall into the mucus by the mediation cra secretory component. This immunoglobulin, IgA, was demonstrated by Butler et al. (1972), using invitro methods along with IgG and IgM to be synthesized by mammary tissue in small amounts and was also smaller in amount than is found in other secretory tissues.

It was postulated by Jean-Francois (1982), that there is a link between the gut and the mammary gland in the cow, such that antigenic stimulation within the intestine lead to an IgA response in the mammary gland, and since this IgA is locally synthesized, it must be mediated by the traffic of primed lymphocytes from the gut to the mammary gland. This work has been supported and extended. In several species, including humans, rats, rabbits and swine, oral immunization during pregnancy has been shown to result in the presence of antibodies, mainly IgA in Colostrum and milk (Saif.<u>et al.</u>, 1972; Montgomery <u>et al.</u>, 1974., Michaleck <u>et al.</u>, 1976). Antibody forming precursor cells from Peyer's patches give rise in rabbits to plasma cells synthesizing IgA in the lamina propria of the intestine (Rudzick <u>et al.</u>, 1975., Rudzick <u>et al.</u>, 1975(b); Craig and Cebra, 1975) as well as in the bronchi (Rudzick <u>et al.</u>, 1975(b).

In reference to the gut and the mammary gland relationship, Bienenstock (1974) suggested a universal linkage between all mucosal immune systems and the term "common mucosal system" has been coined to suggest this relationship. However, this hypothesis awaits critical experimental testing before it can be generally accepted (Bourne and Newby, 1981). The extent to which this gut-mammary lymphocyte migration in ruminants operates is far from clear (Newby <u>et al.</u>, 1982). In the absence of intramammary infusion there is only little local antibody synthesis in bovine udder (Newby and Bourne,

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1977). Plasma cells do accumulate in late gestation and some colostrum IgA and IgH is locally produced. It is probable therefore that some movement of lymphocyte from the gut to the mammary gland does occur in cattle but that this decreases soon after parturition (Newby et al., 1982). This is consistent with other findings which showed that the other secretory bovine tissue (intestine and respiratory tract) are more active than the bovine mammary gland (Butler et al., 1972).

The presence of the reagenic antibody (IgE) in the mammary secretions has received relatively little attention (Newby <u>et al.</u>, 1982). IgE is present in human breast milk in low consentrations (Turner <u>et al.</u>, 1977). Petzoldt and VonBenten (1978), demonstrated some activity in the serum and colostrum of cows. The antibody was shown on the basis of heat stability and presence in the skin, to be IgE-like.

Nany attempts have been made to increase the level of antibody at mucosal surface by various immunization regimes:

- (a) Intramuscular (Bohl et al., 1975);
 (b) Parenteral (Chidlow and Porter, 1973);
- (c) Intramammary direct injection (Bohl
 <u>et al.</u>, 1972; Bourne <u>et al.</u>, 1975;
 Chidlow and Porter, 1978), and

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(d) Intramammary infusion (Kerr et al.,

1959).

The simplest method has been to use parenteral immunization (Newby <u>et al.</u>, 1982), although for the purpose of immunity in the bovine udder, because of the anatomical structure of the teat canal, it is easy to introduce antigen into the lumen of the udder, thereby producing a true mucosal immunization (Newby <u>et al.</u>, 1982). However the use of "bovine udder immunization" in the field is yet to be seen. Newby <u>et al.</u> (1982) findings showed that there was an increase in total concentration of immunoglobulin secreted by the bovine udder after antigenic stimulation by direct intramammary infusion. The speed of the local response to intramammary infusion is remarkable. Sarwar <u>et al.</u> (1964) found agglutinins present in milk only 24 hours after primary infusion.

2.5.2. Vaccination.

Vaccination has proved to be of limited value in the control of mastitis, as has been indicated in the discussion above on "Mucosal Immunoglobulin System". Its effeciency will depend largely upon the antigenicity of the causative agent. While the vaccination is by no means complete and further research is indicated, it seems safe to say that vaccination against <u>Streptococcus analactiae mastitis is unlikely</u> to be

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effective since most recovered animals have little, if any, immunity. The use of an autogenous bacterin against <u>Staphylococcus</u> <u>aureus</u> may be of some use in herds where the infecting organism is highly antigenic. In these herds it is unlikely to be sufficiently effective to completely prevent infection but it may reduce the incidence and severity of clinical mastitis (Norcross, 1963., Norcross, 1964.).

2.6. CONTROL OF MASTITIS.

Bovine mastitis is not an eradicable disease in terms of practicality of control. It is therefore not possible to legislate mastitis control, but rather the control is purely a voluntary dairymen's programme. The programme should aim at reducing the incidence and maintaining the infection rate at a low level. The justification for control of mastitis is therefore based on each individual farms ability to apply the control procedures. Any national programme can only be in the form of providing incentives and assistance to individual dairymen who wish to participate (Blood and Henderson, 1974).

An effective mastitis control programme must (a) provide an economic advantage, convincing the dairymen of its necessity; (b) be within the scope of the dairymen's technical skill and understanding; and be able to handle all the procedures required in the programme: and (c) be capable of merging into the management system already employed. To-day, in Kenyan farms, mastitis control depends on the treatment of clinical cases and the use of hygienic techniques poorly effected in the milking parlour (Hamir, <u>et al</u>.. 1979 and Lauerman <u>et al.</u>, 1973). As a result, subclinical cases go undetected and the continuous spread to further quarters goes on relatively unchecked. In most dairy populations the percentage of quarters

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infected is at the rate of 25 - 50 percent (Schalm, 1977). The resulting financial losses are likely to be very heavy. The two criteria necessary to the biological objective of limiting quarter infection rate are:-

- (a) reduction of the new infection rate,
- (b) reduction in the duration of infected status of infected quarters.

Based on those two criteria, National Mastitis control programmes have developed (Dodd and Neave,(1970); Dodd <u>et al.</u> (1977); and Kingwill <u>et al</u>. (1970). (England, U.S.A., Sweden, Norway).

In reduction of new infection rate, the programme includes:-

- (a) dipping all teats after each milking;
- (b) adequately servicing and maintaining milking machines;
- (c) backflushing cups after each milking and rinsing udders before milking, preferably with running water.

Reducing the duration of infection includes -

- (a) treating all quarters of all cows at drying off;
- (b) treating clinical cases as they occur;
- (c) detecting subclinical cases in line filter: and
- (d) culling chronic clinical cases.

2.6.1. Detection of intested quarters.

This is the most important option in any control programme. However, milk sampling of all cows is expensive and would put a great strain on laboratory resources if routinely used. For control purposes, general herd sampling should be done when a given farmer has a problem and calls for assistance. Then a herd survey or significant sample is taken for laboratory survey. For use alongside a control programme, the general survey of the herd should be omited until those farms on which the programme does not work well become noticeable. This will be at about the end of the first year, when it will be apparent that the clinical mastitis rate is not significantly reduced. The detection of infected quarters can then proceed (Blood, 1978).

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The recommended procedures for the detection and management of infected quarters which give satisfactory results at a reasonable cost (Blood and Henderson, 1974) are:-

- (a) subjection of quarter samples from all cows to the california mastitis test (CMT);
- (b) bacteriological examination of CMT positive quarters;
- (c) identification of coagulase-positive
 staphylococci and CAMP test positive
 streptccocci.

(d) determination of the sensitivity of the pathogens.

2.6.2. Treatment of infected quarters.

Control systems based solely on diagnosis and treatment of the infection are expensive and not foolproof because antibiotic therapy is not always effective (Gustafson, et al., 1977). However, therapy remains an important part of the solution for mastitis control (Ogaa, 1980) in combination with other control measures.

Effective Veterinary practice would dictate that the causative microorganisms be identified and the antibiotic sensitivities be determined before initiation of treatment. While this approach may be useful in treating mild or chronic mastitis, it is not practical in the treatment of paracute or acute syndromes because these often are very severe and dc not allow time for completion of laboratory diagnostic procedures if the animal's life is to be saved. Farmers can only detect about 30 percent (Ogaa, 1980) or 40 percent (Dodd. <u>et al.</u>, 1977) of the clinical cases.

Although it is difficult to make an aetiological diagnosis on the clinical picture alone, this is about the best criterion for the practitioner in the field. The history of the case, the general condition of the animal, findings on palpation of the udder and the appearance of the secretion should five a guide to what type of mastitis one is dealing with. A wide range of medicaments is available to treat cows with mastitis. Bacteriological and sensitivity determinations made with previous cases of mastitis in the herd or a geographical area can be of value in selecting the antibiotic to use for treatment of acute and peracute cases. However, it is preferable that samples of mastitic milk be taken to the laboratory for diagnosis and drug sensitivity testing (Norman, 1973; and Gustafson, et al., 1977).

It is imperative that an adequate dose be administered and that medication be continued for at least one day after a favourable response is observed; or for a minimum of three days. In Kenya, clinical runs give the veterinarian only a roadside crush contact with the animal and therefore the continued medication is the responsibility of the farmer. These being laymen, they may not apply the drug as instructed at times leading to failure in therapy.

To-day numerous mastitis preparations are presented in either tubes or injections for practical and easy intramammary administration. These medicaments are also fairly easily accessible to farmers over the counter (Mutuiri, 1980). It is therefore common to

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encounter cases in clinical runs that have already been treated with various preparations. Thus once highly reputed efficiency of broadspectrum drugs is getting lost due to such use which has lead to increased drug resistance for drugs like Chloramphenicol and the Tetracycline (Weist, 1973., Huber, 1977 and Kariuki, 1977). Ziv (1978), has summarized adequately the criteria to be used in choosing the therapeutic drug for mastitis.

Generally, it is expected that every practitioner should therefore endeavour to stock in his drug store, at least one drug from each group.

Many supportive medicaments have been tried but opinions differ among veterinarians as to their usefulness.

Among such are:

(a) <u>Oxytocin</u>: This may prove useful in facilitating milking and maintaining the milk duct patency making it easy to remove any toxins in the udder (Ogaa, 1980).

(b) <u>Parenteral corticosteroids</u>:

When given at a high dose they are reported to aid in reducing effects of toxins and swelling (Stang, 1977).

(c) Intramarrary cortionstoroids:

These have given good recovery rates at the University of Nairobi Veterinary Clinic (Ogae, 1980). However, it is argued that the drugs interfere with the body's attempt to fight the infection (Weigt, 1973; Black, 1977).

(d) Hydrotherapy:

Application of hot or cold water on the udder surface has been reported to be useful in reducing the swelling of inflammation (Gustafson, et al., 1977).

(e) Enzymes:

Trypsin, veridose, fibrotan, leucocillase, among others, have proved useful in the treatment of mastitis because they help digest and remove necrotic tissue (Heidrich and Renk, 1965; Weigt, 1973). However, the value of these substance as therapeutic agents is limited.

(f) Fluid therapy:

The acutely sick cow is often very markedly dehydrated but reluctant to drink water. Fluid therapy is therefore absolutely important in the treatment of such cases. Water is given by stomach tubes or drenching bottles in large amounts at regular intervals. With dehydration toxaemia is often found. Treatment of toxic cows with 500 ml. of 50 percent dextrose solution and 500 ml. of 25 - 40 percent Calcium borogluconate often leads to rapid improvement. However, Calcium borogluconate and Sulphonamides must not be administered within 36 hours of each other because death can occur as a result of a dramatic precipitation reaction (Gustafson <u>et al.</u>, 1977).

(g) Mild laxatives:

These are recommended where bowel stasis is likely to occur.

Success of a control program depends to a marked degree on reducing the duration of udder infection. The duration of infection can be reduced only by increasing the rate of elimination of infections. Infections that are not eliminated spontaneously are eventually eliminated by therapy (Dodd <u>et al.</u>, 1977., Wilson and Kingwill, 1975). Whatever regime of treatment is followed, and irrespective of the constraints that will be encountered, therapy of the clinical cases has its place in the control of subclinical and clinical mastitis.

2.6.3. Dry com therapy.

The lactating pregnant cow be ins to decline in milk production at about the fifth month of pregnancy (Schalm, 1971). A non-lactating period of 55 to 60 days is optimum for maximum production in the following lactation (Klein, 1934). Drying-off is a physiological process, but it can occur due to pathologic cases (Schalm, 1971). Natural drying-off of normal glands occurs gradually and the increase in cell numbers in the milk is mainly due to epithelial cells (Johnson and Trudel, 1932). Drying-off occuring as the result of mastitis is characterized by the presence of a variable number of neutrophils in the secretion. Therefore, cell counts are higher in infected quarters than in normal quarters (Kaiser, 1966).

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Cows are commonly dried off at the end of a lactation by abrupt cessation of milking or by milking them at gradually increasing intervals (Blood and Henderson, 1974). These methods of drying-off the cows were found to affect the bacterial and cell content of milk (Wayne and Macy, 1933). In comparison, intermittent and incomplete milking usually resulted in somewhat higher bacterial and cell counts during the drying-off period than complete cessation of milking. Oliver at al. (1956) in a comparative study of abrupt cessation of milk and intermittent milking at increased intervals, found that the quarters of cows that were not infected on drying off showed a higher incidence of new infection following abrupt stopping of milking than following the intermittent method of drying off. However, it is argued that internal pressure resulting from abrupt stopping of milking would be highest among those cows secreting the largest volumes of milk, and that once milk production falls to 10 pounds daily, it is safe to use the abrupt method for inducing non-lactation (Schalm, 1971).

Some evidence has been obtained to indicate that a fall of intrinsic bactericidal activity may occur in the early part of the dry period (McEwen and White, 1950). In another observation including infection due to Streptococcus, Staphylococcus and other bacteria, new infections were found to relate to the presence and concentration of these organisms on the skin of the teats at the time of drying-off the udder. Most of the new infections that persisted were caused by Staphylococcus, and more than half the quarters developed clinical signs of mastitis at calving or shortly thereafter due to Staphylococcus (Neave et al., 1950). In an investigation by Parisi and Baldwin (1963), Streptococcus agalactiae was reported to persist through dry periods of 140 and 371 days. The persistence of Streptococcus analactiae

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through the non-lactating period was investigated in 69 cows. These cows had dwy periods ranging from 16 - 350 days (average 75 days) and 85 percent of the cows shed <u>Streptococcus avalactiae</u> again at the beginning of the following lactation. 22 percent of the non-infected quarters were shedding <u>Streptococcus</u> <u>agalactiae</u> at the beginning of the following lactation.

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It is not uncommon to find that cows can acquire an infection during the dry period or can enter the dry period with udder infections and freshen with the same infections, or even can go through several lactations with the same infections (Schalm, 1971). Work in England (Neave et al., 1950., Oliver et al., 1956; Oliver et al., 1962; Smith et al., 1966, and Smith et al., 1968) indicated that when new infections appear in the dry period, they appear early rather than late. They tend to occur more frequently in those cows with previous history of infection and in cows with already existing infection. Since the success of a control programe depends to a marked degree on reducing the duration of under infection (Gustafson et al.. 1977) it seems inevitable to draw attention to the treatment of non-lactating cows. Whether treatment would include all the quarters of all cows at drying-off or only those quarters tested and found infected, depends on economics (Dodd et al.,

1977). This refers not to the average cost of eliminating each infection by antibiotic but to the total economics of a control program incoorporating a specific antibiotic routine. Teat disinfection after the last milking has been reported to be effective in reducing the number of new infections after the dry period (Oliver <u>et al.</u>, 1956; Oliver <u>et al.</u>, 1962). This is mainly for Staphylococci which dominate the skin (Neave <u>et al.</u>, 1950).

It has been reported that half of the new infections occuring during the dry period occur during the first 21 days after the last milking (Neave <u>et al.</u>, 1950). Therefore, the effective treatment in a non-lactating udder should be done early and preferably with a long acting antibiotic preparation. To increase protection, the teat skin should be bathed in a disinfectant before drying the cow.

2.6.4. Teat dipping.

Excellent results have been obtained by the use of suitable teat dip disinfectants in preventing new infections (Blood and Henderson, 1974). Disinfectants have been used as teat dips during lactation (Newbould and Barnum, 1960) and at the time the cows enter a dry period (Roberts <u>et al.</u>, 1969). Teat disinfection alone, was shown to have achieved

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a reduction in incidence of new udder is ection of 53 percent (Newbould and Barnin, 1960). Although hygiene alone is not enough to control bovine mastitis, Roberts, et al. (1947) noted that disinfection of tests after milking is regarded as an effective means for reducing the spread of mastitis. On the basis of available data, test disinfection combined with intramammary therapy has been recommended as a practical means for mastitis control (Dodd, et al., 1964).

Observation on new infection by <u>Streptccoccus</u>, <u>Staphylococcus</u>, and other bacteria, showed that new infections by <u>Staphylococcus</u> were related to the presence and concentration of these organisms on the skin of the teats (Neave, <u>et al.</u>, 1950). Since then, various teat disinfectants have been put forward for use.

Although the factors determining the ability of microorganisms to colonize teat skin and to invade the mammary gland are well recognized, great variations do occur and these have not been determined (Cullen and Herbert, 1967). Any practical procedure that keeps teat skin colonization under control is likely to reduce the prevalence of mastitis. Commonly used chemicals for teat skin disinfection are:-

(a) <u>Iodine:</u> This is used as a 5 percent tincture of icdine, after the last milking, the

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disinfectant was reported to be effective in reducing the number of new infections caused by <u>Staphylococcus</u> in the subsequent dry period (Oliver <u>et al.</u>, 1956).

A separate solution of iodine in the form of Iodophor has been used with success (Wesen and Shultz, 1970). Iodophor dips are expensive but effective.

(b) <u>Chlorhexidine</u>: Dipping the teats for 20
seconds in a 2 percent chlorhexidine solution has
given good protection against new infections caused
by Staphylococci (Oliver <u>et al.</u>, 1962). A one
percent chlorhexidine (Hibitane) in polyvinylpyrrolidine
solution has also been used with success (Gehring
<u>et al.</u>, 1968).

(c) Sodium hypochlorite: Control of mastitis, including Streptococcus mastitis by teat dipping method, using 40,000 ppm of sodium hypochloride (Clorox^(R)) has been done in New York State (Roberts et al., 1969) and in England (Neave et al., 1966). This solution has been shown to kill bacteria on the teat skin and remain effective until the next milking. even if the cows are walking through long wet grass. It is also not irritating to the skin of the teat and hands of milkers (Newbould and Barnum, 1960).

Although teat skin disinfection has been used alone to achieve a degree of reduction in mastitis level, it will be seen from the above that all the

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various control methods have to work synergistically.

This investigation aims at answering the following questions:

- (i) What is the incidence of subclinical and clinical mastitis in King'ong'o area of Nyeri, Kenya.
- (ii) To what extent can the level of subclinical mastitis and clinical mastitis be lowered by the application of selected, recommended mastitis control methods, namely:
 - (a) Sanitation of milking equipments,
 - (b) Washing of the udder with sanitized water,
 - (c) ~ Teat dipping into an effective sanitizer,
 - (d) . Treatment of all clinical quarters and(e) ... Dry cow therapy.

3. MATERIALS AND METUODS.

The research was carried out around ing'ong'o area in Nyeri District (Appendices 1 and 2), in the Central Province of Kenya. This area had numercus small farms with one to five milking cows each. There were however some few large herds with about twenty milking cows in each. Most of the dairy animals were crosses of Fresians, Guenseys, Ayrshires and Jerseys, and Zebu cattle; pure breeds could also be found.

Milk yield was generally low. Handmilking was the main method of milking. Although some farms had adequate facilities, the general standard of hygiene was very low, especially in the smaller herds. Mastitis preventive measures were hardly in practice.

In choosing the farms for this trial, the following factors were considered:

- (a) The herd size had to be one of 25 milkers or more,
- (b) The farms had to be within a close range from the Investigation Laboratory, due to financial constraints,
- (c) There had to be some kind of record keeping,
- (d) There had to be some willingness by the management to participate in the exercise and
- (e) The farm had to be practicing handmilking.

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The following farms wer selecter for the investigation:

- (a) Kyanyange farm (Ky).
- (a) Keremara farm (K).
- (c) Wambugu farm (W).
- (d) Seremwai farm (S).

3.1. FARM L'ANAGEMENT EVALUATION.

The rating of management for the dairy enterprise in the four chosen farms was done in consideration of the following factors:

- (a) Grazing fields in relation to bush clearing.
- (b) Fencing with reference to type and its maintenance.
- (c) The holding ground during milking time, the size of the paddock, the possibility of mud or dust during the respective wet or dry seasons.
 - (d) The milking procedure, considering the udder scrubing cloth, udderwash water, the milkman's attention to hygiene at the parlour, and the milking method.
 - (e) The milking equipment and its sanitation.
 - (f) Record keeping for the individual animal

and the herd in general.

In each of the factors considered, a numerical value was given subjectively as follows:

Poor rating	1	point.
Fair rating	2	points.
Average rating	3	points.
Above average	4	points.
Excellent rating	5	points.
 I walno was then sizes for ask for		

A final value was then given for each farm management.

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3.2. SUBCLINICAL MASTITIS EVIL. TICN.

A cowside chemical test, the Mastilis Indicator Test (MIT), was used on all the quarters, both before control method application (BCHA), and during control method application (DCM.).

A microbial survey was done on random samples from each of the following:

- (a) Normal quarters (those quarters that did not give a positive reaction on the MIT).
- (b) Subclinically infected quarters (those quarters that had a positive reaction on MIT, but without observable changes in the milk or the udder).
- (c) The clinically infected quarters. This was once a month for a period of 14 months for each quarter. The first period of seven months was the control period (BCMA) while the next seven months were the experimental months (DCMA).

3.2.1. Mastitis indicator test (MIT).

Various tests for the diagnosis of clinical and subclinical mastitis, have been in use since 1916. In this investigation, MIT, a method modified from California Mastitis Test (CMT), was used. As in CMT, the milk was drawn from each of the four quarters into respective cups with no attempts to obtain equal amounts (fig. i). The cups held on one paddle

Fig. 1. PROCEEDURES IN MIT TEST.



Fig. (a) A = MIT Paddle B = MIT Solution C = MIT Squart bottle



(b) Drawing of test milk



(d) Addition of a MIT



(c) Pouring excess milk



(e) Reading the reaction solution to the test numerical value. The values (Fig. 1,c,), were tipped to a near vertical position to drain off excess milk leaving approximately 2 mls. in each cup. The reagent was then added by squarting from a plastic siphon bottle (Fig. 1,d), until an amount equal to the volume of the test milk was added by estimation to each cup. The mixing was accomplished by a circular motion of the paddle in a horizontal plane (Fig. 1,e). The reaction developed almost immediately with the milk containing a high concentration of somatic cells. The peak of the reaction was obtained in about 10 seconds and was read, since the weak reactions tended to fade on continued motion of the paddle.

After completing one test, the mixture was discarded into a waste container and the paddle rinsed in cold water. This left the paddle ready for use with the next cow without having to dry the paddle. A trace of moisture did not interfere with the reaction.

3.2.2. Milk samples collection.

Milk samples were taken from random cases that were found either clinical, subclinical or negative on MIT test. Three cases were sampled from each category except for clinical cases that would be fewerwhen less than 3 cows were clinically infected.

In total 9 cows would be sampled at each of the visits made to the farms.

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To obtain a milk sample for microbial diagnosis, the udder was washed with a dilute solution of mastrite^(R). The solution draining on the skin was wiped using one paper towel per cow and a different surface of the paper per test. The milk was expressed into a universal bottle. The universal bottles were then labelled for the cow and the quarter position on the udder for identification.

The samples were transported to the laboratory shortly after collection for culturing and storage. The samples were stored refrigerated prior to culturing the next day.

Mastrite^(R) Wellcome, Kenya Ltd, Nairobi; A dark brown liquid containing 1.1 percent w/w available iodine in the form of iodophor. with lanolin based emollients and sulfactant.

3.3. MICROBIAL ISOLATION AND DRUG SEBSITIVITYS.

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Milk was inoculated onto sheep blood agar and McConkey agar plates and incubated at 37°C. for 24 hours. Isolated colonies were subcultured onto the same media and identified to species level (Ministry of Agriculture Fisheries and Food, 1978). For bacterial drug sensitivity each organism was streaked densely and uniformly on Mueller-Minton agar plates. Various sensitivity disks were then applied onto the plates (Appendix 7). The plates were incubated for 24 hours at 37°C and diameter of the inhibition zone was measured. Manufacturers guides for the various types of disks used were followed to rate the organisms as either sensitive or resistant to the respective drugs. 3.4. APPLICATION OF MASTITIS CONTROL METHODS:

Having done the initial mastitis evaluation for a period of seven months, mastitis control methods were introduced and applied for the subsequent seven months for four farms. These control methods included: (a) Milking equipment hygiene (b) Milkers-hands and udder hygiene (c) Teat skin sanitation after milking (d) Dry-cow therapy (DCT) (e) Clinical case treatment.

3.4.1. Milking equipment hygiene:

The milking technique was handmilking using the following equipments: Milking buckets, udderwash buckets, weighing buckets, water jars, milk can and towels. For their sanitation immediately after and before use, a sterilizing <u>detergent solution, Bactergent^(R)</u>, was used in cold water. Bactergent^(R) - Wellcome, Kenya Ltd., Nairobi; A clear slightly soapy liquid, containing powerful quarternary ammonium genicide and detergent. The metal equipment was brushed with Hot Bactergent^(R) solution prepared at the rate of 1:400 in water. After washing, the equipment was left to drain and dry. For the next milking they were rinsed with clean cold water before using them again.

3.4.2. Udder hygiene.

The udder hygiene before and after milking was considered. For disinfection, Mastrite^(R), a liquid containing 1.1. percent (W/W) available iodine in the form of iodophor with lanolin based emolient and a sulfactant, was used as described below:-3.4.2.1. <u>Udder hygiene before milking:</u> To prevent introduction of bacteria into the udder from the milkers' hands and to avoid contamination from the soiled udder to the next cow, the cow's udders were washed prior to milking using a dilute solution of Mastrite^(R), at a concentration of 1:300 (1 tablespoonful or 15 ml. of Mastrite^(R), in 4.5 litres or one gallon of water). The udder cloth was wrung out in this solution and carefully used to wipe the udder and the teats.

3.4.2.2. <u>Udder hygiene after milking</u>: To prevent microorganisms from ascending into the teat canal from the skin of the teat, a strong solution of Mastrite^(R) (1 part Mastrite^(R) to 1 part water) was used in a teat cup as a teat dip. The dipping of teats in this solution aims at destroying most mastitis causing organisms that would otherwise cause scending infection; and because of the Lanolin base, the sat surface would be safe from sores.

3.4.3. Dry cow therapy:

All those cows that were in their last 2 months of gestation and had reduced milk production per day to one kilcgram were dried up in consultation with the farm management. Drying of cows is not routine in Nyeri area with or without the use of dry cow therapy. The mammary quarters were stripped of all milk and Novomast D.C. (R), a dry cow antibiotic preparation was infused into each quarter. The dry cows were then left with the antibiotic until the next lactation.

Novomast D.C. ^(R) - Wellcome, Kenya Ltd., Nairobi; A dry cow intramammary antibiotic preparation containing procaine Penicillin P.B. (3,000,000 Iu) and Novobiocin sodium (250 mg) per tube.

3.5. GLINICAL CASE TREATMENT.

During the initial evaluation period (ECMA), only the visible clinical mastitis was attended to. This means that only those cases that the farmer himself noticed, were treated. This helped to obtain the incidence of subclinical mastitis without interfering with the normal dairy management that existed. However, during the mastitis control method application period (DCMA), advice and assistance was given in spoting the clinical cases. Using the black surface of the MIT paddle, it was easier to spot the physical changes in the milk that were associated with clinical mastitis and these were confirmed using the MIT evaluation. However, not all the cases with an interpretation of over 500,000 polymorphonuclear leucocytes/ml. were treated since clinical mastitis has as well as an increase in leukocytes, physical chemical and other pathological changes. The MIT results were supplemented with physical palpation of the udder, the appearance of the milk, its consistency and stage of lactation.

Before treatment of a case, a sample for bacteriological analysis was taken. One antibiotic preparation multimast ^(R) was always used for treatment awaiting a sensitivity test. The sensitivity tests were done with every positive bacterial cultures. Only then could another preparation be used as indicated through <u>the sensitivity patterns ob</u>tained.

Multimast^(R) = Wellcome, Kenya Ltd., Nairobi; 250 mg. Neomycin as Neomycin Sulphate B.P. 250 mg. Streptomycin as Streptomycin Sulphate B. 100,000 units Procaine Penicillin B.P., 50 mg Oxytetracycline hydrochloride B.P., 10 mg Predinisolone E.P.

4. RESULTS.

This work was carried out for a period of fourteen months between June 1982 and July 1983.

4.1. HERD MANAGEMENT EVALUATION RESULTS.

The evaluation for the general management of the general method applications are given below and results in table 1.

4.1.1. Grazing fields.

The grazing fields in relation to thorn bush clearing in this area was given little attention by the farmers. As in fig. 2 and fig. 3, it appeared that the main clearing was done in areas where the thorny bush would help to maintain the integrity of the fence. The grazing area per cow was good. 4.1.2. Holding ground/fencing.

The holding ground generally was small. The periphery fence in these paddocks was poorly maintained so that the animals would find their way through barbed wire easily (fig. 4) and they stood deep in mud during the wet seasons (fig. 5).

4.1.3. The milking parlour.

The milking parlours had been well constructed although the maintainance of the structures was poor. Fig. 6 shows one of the parlours whose floor was broken and the ground in the holding paddock had building stones scattered around. The roof was old and during the rainy season it was seen to leak. The



Fig. 2. Short thorn bushes seen collected in small heaps after clearing at Kyanyange farm.



Fig. 3. Loose wire fence seen supported with thorny bushes after clearing.

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Fig. 4. A holding ground during milking showing broken posts and loose wire.



Fig. 5. A cow after milking seen leaving the milking parlour within a holding paddock during a milking session.

ELELE 1.	NUMERICAL EVALUATION OF DAIRY HERD MANAGEMENT FOR THE VARIOUS FACTORS CONSIDERED AND THE
	OVERALL MANAGEMENT.

HERD	NC. OF COWS	BREED	GRAZING FIELD	HOLDINC GROUND	MILKING PARLOUR	MILKING PROCEDURE	DRY COW/MILK PRODUCTION	CLINICAL CASE	OVERALL MANAGEMENT
X	25	M	3	2	2	2	1	2	2(below average)
KY	25	M	2	2	3	2	1	2	2("")
S	25	G	2	1	2	2	1	2	1.7(" ")
W.	25	F/G	3	4	4	2	3	3	3.2 (Average)

Li = Mixed

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G = Guernseys

F = Friesians

K = Keremara Farm

- Ky= Kyanyage Farm
- S = Seremwai Farm
- W = Wambugu Farm.

rcof run-off water was poorly drained and therefore this water fell directly onto the holding ground whose drainage was also poor. The holding grounds in fig. 6 was therefore wet most of the time. Fig. 7 shows another parlour where two walls were missing making the shed too open and dusty during the dry season. 4.1.4. The milking procedure.

Handmilking was used in all the four farms surveyed. The technique used was poor as it included the use of thumb and the index finger with some considerable amount of pulling on teats. Milking jelly was provided by the management but the milkmen kept the jelly in an open place so that most of the time, the jelly was dusty.

The washing of the udder was done with unsanitized water, either cold or slightly warm. The udders which were often soiled were washed using old torn and dirty cloths. This was rarely changed during one milking session. Usually one bucket of water and one udder cloth was used for over 10 cows and at times shared between 3 milkmen. In some occassions the milkers only greesed their hands with jelly and milked without washing the udders especially during the dry seasons.

No feed was provided during milking generally, and the animals were sometimes forced into the milking parlour, causing an unnecessary stress.

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Fig. 6. Broken ground seen infront of a milking parlour. The roof was poor allowing rain water to pour in. The ground infront of the milking parlour is deep with mud.



Fig. 7. The roof above, is good bu the walls are open on the two sides.

4.1.5. Dry cow and milk production.

Generally the animals were not allowed to dry between one lactation and the next. Milk production was between 2 kg. - 4 kg., and at times it was down to 0.5 kg. per milking session per cow. At Wambugu Farm, cows would be dried. It was the little milk that dictated drying the cow and not the gestation stages. Other farms simply went on milking till the next calving.

4.1.6. Clinical cases diamosis.

The clinical cases were not attended, since treatment was at the discretion of the milkmen. Even when a clinical case was diagnosed, the line of action was: the milkman, then the foreman, the manager and sometimes the farm owner who decided what to do. Usually antibiotic preparations were in the farm store and any of the available intramammary tubes were used; the dosage of which was decided and judged by the milkman. At times the milkmen ignored the diseased cases and just milked the clinical quarter milk directly on to the floor. However, Government or private veterinary personnel were also consulted for some cases.

The results are shown on table 1 for each factor and the final evaluation of each dairy herd.

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4.2. MASTITIS EVALUATION MASTITIS INDICATOR TEST (MIT).

4.2.1. Clinical mastitis.

The first visit to the four selected dairy herds in the period DCMA showed a pocled farm total of 26 quarters to be clinically mastitic out of 370 examined quarters. This is a 7 percent clinical mastitis incidence. However, only a few were under treatment, and the actual number under treatment could not be ascertained due to the fact that records of treatment were not available.

The distribution of these clinical cases was 8 quarters at Ky farm, 8 quarters at W farm, 6 quarters and 4 quarters at K and S farms, respectively.

The whole period of 7 months BCMA had a total of 126 clinically mastitic quarters, out of the 2634 tests that were done with 4.8 percent clinical mastitis. However, the subsequent period of 7 months DCMA had 58 clinical quarters out of 2370 tests done. This period had 2.4 percent clinical mastitis incidence (Tables 2 and 3). The clinically mastitic quarters were those that had an estimated 500,000 Polymorphonuclear cells/ml, accompanied also by observable changes in the secretion or udder pathology.

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4.2.2. Subclinical mastitis.

The pooled farm subclinical mastitis tests on the first visit gave 204 quarters to be subclinically mastitic out of the 370 tests that were performed, with a result of 55.1 percent subclinical mastitis incidence.

The whole period of 7 months BCMA showed 1514 pooled farm subclinical quarters out of the 2634 examined quarters. The percent of subclinical mastitis in this period was 57.5 percent. However, in the subsequent period of 7 months DCMA, the encountered subclinical mastitis was 897 quarters out of the 2370 tested quarters with 37.8 percent incidence (Tables 2 and 3).

4.2.3. FARM DISTRIBUTION OF MASTITIS.

The distribution of clinical mastitis and subclinical mastitis in the investigation period for the individual farms is shown on tables 2 and 3. The lowest and the highest recorded subclinical mastitis in the period RCMA were 52.5 percent and 65.7 percent respectively while those for the period DCMA were 27.1 and 54.6 percent. For the clinical mastitis the lowest and the highest recordings were 2.8 and 7 percent in the BCMA while those from the period DCMA are 0.5 and 5.4 percent respectively.

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4.4. MICROBIAL SURVEY.

In total 347 organisms were isolated from all the quarter milk samples cultured (Table 4). 187 isolates were from clinically infected quarters, 129 isolates from subclinical quarters, and 31 isolates from latent infections (normal quarters), as shown on table 4.

Isolates from each of the four farms are also presented on table 4 for each of normal; subclinical; and clinical categories of quarter conditions. The most predominant organism from pooled farm and quarter state was <u>Staphylococcus aureus</u>. Other organisms isolated in order of decreasing frequency in isolation were: <u>Streptococcus spp</u>. <u>Klebsiella pneumoniae</u>. <u>Escherichia coli</u>, <u>Bacillus cereus</u>, <u>Pseudomonas aeruginosa</u> <u>Yeast</u>, and <u>Pasteurella multocida</u>. Samples from where mixed growth was obtained were 21 (Table 4).

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4.4. SENSITIVITY TEST.

Antibiotic sensitivity tests against the most commonly used intramammary preparations were done. The sensitivity tests were done for those organisms isolated from clinically infected quarters. The sensitivity results for both periods BCMA and DCMA pooled for clinical quarters, multiple drug restance results pooled for the farms, and the multiple drug resistance per farm are given in table 5. The occurence of resistant isolates for each of the discs used were very low. All the Streptococcus organisms isolated were found to be sensitive to Penicillin and Neomycin while the highest sensitivity by Staphylococcus organisms were observed with Neomycin and Erythromycin.

The sensitivity discs used are listed in appendix 7 against their sources.

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4.5 ORGANISMS ISOLAT D IN RELATION TO QUARTER UDDER POSITION.

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In total 347 organisms were isolated. The distribution of the isolates to quarter position on the udder was: 86 from the right front quarters, 102 from the left front quarters, 102 from the right hind quarters and 57 from the left hind quarters. All the right side quarters yielded 188 organisms, while the left side quarters yielded 159 organisms. The front quarters had 188 while the hind quarters had 159 organisms. The frequency of organisms isolated in quarters position is shown in table 6 for both periods BCMA and DCMA.

4.6. DRY COW THERAPY (DCT).

In total 30 cows were dried and infused with a DCT antibiotic preparation in the period DCMA. A total of 117 quarters were dried. Three cows had lost a quarter each. The numerical MIT values were recorded before drying and at the start of the subsequent lactation (table 7). Quarters found infected at subclinical level were 91 while at the begining of the next lactation only 11 were found infected. There were therefore 79 quarters cleared of infection. This reduction is from 77.8 percent to 10.3 percent, a drop of 67.5 percent units.

Table	6.	ISOLATE	D OF	RCANIS	S POOLI	ED	FOR	QUARTER	POSITION
		DURTNG	मम	WHOLE	PERTON	OF	TNU	TPSTT GA T	[OII .

(a)

(b)

(c)

Tot	al	RF	lf	RH	LH	R	L	F	H
Normal quarters	31	11	5	8	7	19	12	16	15
Subclinical quarters	129	19	41	47	22	66	63	60	69
Clinical quarters	187	56	56	47	28	103	84	112	75
TOTAL	347	86	102	102	57	188	159	188	159
FOR THE PE	RIOD	BCM	1:						
Te	otal	RF	LF	RH	LH	R	L	F	H
Normal quarters	14	4	1	5	4	9	5	5	9
Subclinical quarters	69	8	20	26	15	34	35	28	41
Clinical quarters	131	41	37	34	19	75	56	78	53
TOTAL	214	53	58	65	38	118	96	111	103
FOR THE PE	RIOD	DCM	A :						
T	ota].	RF	LF	RH	LH	R	L	F	H
Normal quarters	17	7	4	3	3	10	7	11	6
Subclinical quarters	60	11	21	21	7	32	28	32	28
Clinical quarters	56	15	19	13	9	28	28	34	22
Total -	133	33	44	37	19	70	63	77	56

RF	=	Right front quarter.
RH	=	Right hind quarter.
LF	=	Left front quarter.
LH	=	Left hind quarter.
R	=	Right quarter.
L	=	Left quarter.
H	=	Hind quarter.
F	=	Front quarter.

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Table 7.	PCOLED	FARM	MIT	ULTS	FOR	DRY	COW	TERAPY

	-					
	MIT N	lemerical	Values	; Berd	ere DCT	
	Negative MIT value		1	2	3	Total
No. of Quarters	26	12	8	23	48	117
Total			91			

MIT numerical values after DCT at the start of the subsequent lactation

	Negative MIT value	I	1	2	3	Total			
No. of Quarters	106	3	8	0	0	117			
Total			11						
	uarters of infect:	ion	80						

KEY:

DCT = Dry cow therapy.

5. DISCUSSION.

Various fectors of mastitis control were used in combination in this investigation: Teat dipping, dry cow treatment, udder hygiene, equipment hygiene, and a guided clinical case treatment. A reduction in the prevalence rate of subclinical mastitis was observed. In the period before control method application (BCMA), the prevalence rate was 57.5 percent while the prevalence rate for the period during control method application was 37.8 percent. This is an improvement in the elimination of infection of 19.7 percent units within the seven months duration, during which the control methods were in application.

Teat dipping is beneficial in lowering the incidence of subclinical mastitis, whereas dry cow treatment is beneficial in shortening the duration of infection. Crist <u>et al.</u> (1982) and Funk <u>et al.</u> (1982), showed that a programme of dry cow therapy plus teat dipping was superior to dry cow treatment alone, teat dipping alone or none at all. In Kenya, where handmilking is the more practised procedure, various combinations and single control methods need to be tried so as to arrive at the most effective method of mastitis control. Such records are not available for comparison with other field trials done elsewhere or for future research to improve on. In a field trial, Pankey et al. (1982) was able to effectively eliminate 80 percent of <u>Stanhvlococcus aureus</u> infections using dry cow therapy alone. Higher cure rates were obtained against <u>Streptococcus SDD</u>. (Philpot, 1979). The proven advantages of dry cow therapy are (a) Higher efficacy compared to treatment during lactation (Philpot, 1979 and Funk <u>at al.</u>. 1982). (b) Improved milk production at calving. The economic effect was demonstrated by Smith <u>et al</u>. (1979) cited by Pankey <u>et al</u>. (1982). Clinical cases reduced at calving with the use of dry cow treatment (Philpot, 1979). (c) No milk is discarded as would occur with treatment during lactation (Philpot, 1979).

Bramley (1982), suggested that penetration of the teat duct by <u>Escherichia coli</u> occured in the period between contamination and milking. In teat dipping the aim is to obtain a seal on the teat skin and the teat duct opening with the teat dip chemical. Fig. 7 below shows a drop of the teat dip solution in a position to seal the teat duct opening. However, in this investigation, all the farms used a form of milking jelly for lubrication of hands and teats during milking. The teat dipping was latter done. This means that the teat dip chemical was applied on the milking jelly coat left on the teat skin after

71 -Fig. 7.

A drop of teat-dip-solution at a position to seal the teat opening.

milking. This brough about the question of efficiely of this procedure and thus investigation into this problem should be done. It was further suggested that antiseptic properties be incoorperated into the jelly. In the process of lubricating the teat skin on antimicrobial cover for both the hands and the teats would be provided.

Since new infection is related to the presence of, and concentration of organisms (<u>Staphylococcus</u>. <u>Streptococcus</u> and <u>Escherichia spp</u>.) on the teat skin (Neave, <u>et al.</u>, 1950), any form of teat skin protection would be beneficial. This hypothesis remains to be tested.

5.1. GRAZING FIELD AND LASTITIS.

Milking animals are frequently seen with teat or udder wounds. The general management results showed that fences were not well maintained and there were spaces between the lose fence wire. These spaces are very tempting to animals and in the event of a cow going through the fence, where barbed wires are used, the udders and teats can be injured. Other bruises and wounds can be caused by the low lying thorn bushes in the pastures. Animal grazed in poorly kept pastures frequently have thorny bushes dangling around their udders causing some form of lesion. Although this investigation did not collect data on teat lesions, a study by Seiber and Farmsworth (1981), showed that glands with acute teat lesions, and those cows in which the teats had been traumatized or leaked with milk had higher rates of infection. Other reports showing a direct relationship between teat lesion and subclinical mastitis are those of Udall, (1947)., Weisner, (1974)., Michel <u>et al</u>. (1974), and Turner, (1952).

Surfice it to say that all farms should have well maintained fences and grazing pastures so as to have better results in the mastitis control programme. It is possible that these frequently observed teat and udder lesions affected the results on the present mastitis control investigation.

5.2. SUBCLINICAL MASTITIS AND MILK PRODUCTION.

One of the cardinal signs of inflammation, loss of function in the udder, is reduction in amount of milk normally produced. In subclinical mastitis, this is never obvious to the farmer. In this investigation there was no direct evidence to show these loses. However work done at Washington State University (Dobbins, 1977) using CMT in the comparison of the infected and the non-infected quarters of the same cows, showed that there was milk loss corresponding to the state of infection (Appendix 2). This information does show that the improvement in state of infection by 19.7 percent units in the prevalence of sub-clinical mastitis in the present investigation, indirectly reflects on improvement in milk production for the farmer.

Smith et al. (1968) showed that milk production is reduced upto 3.5 percent in quarters infected at calving. In the present investigation 80 quarters out of 91 quarters that were infected before calving, and infused with a suitable antibiotic at drying, were found to have cleared of the sub-clinical infection at the subsequent lactation. This is an 87.9% reduction in infection. The information from the work by Smith et al. (1968) does indicate that dry cow therapy improved milk production for tested farms. Actual work on the effect of sub-clinical mastitis to milk production in Kenya is however necessary.

5.3. DRY COW THERAPY.

Dry cow therapy reduced the sub-clinical infection at the start of the next lactation by 67.5 percent. In the 91 infected quarters that were dried and infused with a dry cow antibiotic preparation, 80 quarters were found to have been cleared of the infection.

Although dry cow therapy was effective in the present investigation in reducing the incidence of sub-clinical infection at calving, drying of cows with or without intramammary infusion with an antibiotic was not done at Kyanyange, Keremara, and Seremwai

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farms before the present investigation. All the cows were dried and infused in the interest of the investigation for all the farms, except for Wambugu Farm where drying of cows was practiced without the use of the dry cow antibiotic.

The results in this investigation are similar to those obtained by workers elsewhere under different conditions (Brookbanks, 1971., Dodd <u>et al.</u>, 1977., Kingwill <u>et al.</u>, 1970., Phipps, 1966., and Philpot, 1979. A trial by Pankey <u>et al.</u> (1982), was effective in eliminating more than 80 percent of <u>Staphylococcus</u> <u>aureus.</u> Although these high effects with dry cow therapy have been reported by many workers, spontaneous recovery from subclinical infection in the dry period of 30 percent have been observed (Pankey <u>et al.</u>, 1982).

It would appear that the first step towards the control of mastitis, using infusion of antibiotic into the quarters of a dry cow, is getting the farmers to appreciate the need to dry a cow for the two months before calving. Only then would the second step of introducing the advantages of dry cow therapy be effectively done.

5.4. UDDER WASHING.

Udder washing was observed to be done using cold or warm water. The udder washing water, usually in a bucket was not changed for the whole of

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milking time. For this reason the last cows in a milking row were washed with very dirty and greasy water. This would indicate that those last cows in the milking line were exposed to a higher contamination risk than the first cows on the line. Milking in these farms did not follow a given order or sequence, and therefore no data was available to verify this hypothesis.

It is expected that the clinically mastitic cows would be milked last or in isolation. This fact was not followed. The mastitic cows came for milking unmarked and when noticed, the mastitic milk was drawn directly on to the ground. Without the use of any disinfectant in the udder wash water, the cow that followed would be exposed to contamination by the same organisms from the sick cow. This contamination would easily be done through the uddercloth and the milkers hands.

The chances of spread of infection in this experiment was lowered, not only by the sanitation of the udder wash water but also by renewing the water after every five cows or less. However, the effect solely brought about by the udder hygiene introduction was not evaluated. The MIT results were as a result of an added effect of all the mastitis control methods used in combination. Work should be done to evaluate the use of udder washing before handmilking as a factor in the control of mastitis.

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5.5. QUARTER POSITION ON THE UDDER AND MICROBIAL ISOLATION.

It would appear that some quarter positions on the udder are less prone to infection than others. In this investigation the right side quarters and the fore side quarters yielded more organisms than the left side quarters or the hind side quarters. The left hind quarters yielded 38 organisms out of 214 organisms isolated in the period before control method application (BCMA), and 19 organisms out of 133 organisms isolated in the period during control method application (DCMA) as in table 6.

It could be urgued that, since hand milking is mainly done from the right side of the animal, the left side quarters and especially so the left hind quarters have the less contact with the milker. In those milking procedures where sanitation is not taken care of, then there are chances that the quarters with the more contact with the milker would come down with infection much more frequently than others. However, the picture of the frequency of organisms isolation and quarter position was the same for both periods ECMA and DCMA separately and when combined.

5.6. MASTITIS CONTROL AWARENESS.

Many factors on the control of mastitis at all levels have been put forward and investigations continue to be done. In the present investigation, it was clearly observed that some methods of mastitis control were already known by the farmers. However, even those measures that were known were not being applied in a desirable manner. Lack of proper application of a control measure that was already known by the farmer. was atributed to unawareness or wrong interpretation of the benefits attached to the use of the particular control measure.

The implication is that effective mastitis control may have to delay until the seriousness of sub-clinical mastitis is fully understood by the farmers. The need to control sub-clinical mastitis has to be appreciated before any effective and economical control can be realized. Above all, the benefits have to overshadow the costs of the recommended control methods. When the actual figures of the economics of mastitis control would be worked out, they would serve as probably the guide lines for urging the farmers to participate in a control programme.

It was also observed that the use of the control methods will require alot of ground work in the way of education of the dairy management force before they can reach an effective level of application.

6. CONCLUSION.

The present investigation set out to establish the level of mastitis in the King'ong'o area of Nyeri, Kenya and to find out the extent to which sub-clinical mastitis could be controlled by the application of the recommended methods of mastitis control.

Results showed that mastitis was high with a prevalence of 57.5 percent for sub-clinical mastitis and 4.8 percent for clinical mastitis. The intervention using hygiene and therapy during lactation and in the dry period improved the level of mastitis from 57.5 to 37.8 percent for sub-clinical mastitis, a drop of 19.7 percent and clinical mastitis improving from 4.8 percent down to 2.4 percent at the end of the intervention period, a drop of 2.4 percent.

While the present investigation shows some improvement after the use of the recommended mastitis control methods used in combination, further work should be done to evaluate the benefit of using each of these methods separately, in so doing, the contribution of each method can be evaluated versus its sole economic effect to the whole mastitis control programe. This investigation did not consider the economics of the inputs versus the improvements observed. When and if the actual figures of the economics of mastitis control would be worked out, they could probably serve as the guidelines for urging the farmers to participate in a possible mastitis control programme.

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It was also observed that the use of the control methods will require alot of ground work to be done by way of education of the dairy management force before they can reach an effective level of application.

REFERENCES.

- 82 -

Anon, (1980).

Selected monthly reports of Veterinary Clinics for 1977 - 1980; Department of Veterinary Services, Ministry of Livestock Development, Kenya.

Anon, (1965).

Screening test for the determination of abnormal milk. U.S. U.D.E.W. Washington D.C. Baker, J.C. and Breed, R.C. (1920).

> The reaction of milk in relation to the presence of blood cells and of specific bacterial infection of the udder. N.Y. State Ag. Exp. Sta. Tech. Bull; 80.

Baker, J.C. and Van Slyke, L.L. (1919). Amethod for the preliminary detection of milk abnormality based on the hydrogen ion concentration. J. Biol. Chem. <u>40</u>: 357 - 360.

Barto, P.B., Bush, L.J. and Adams, G.D. (1982). Feeding milk containing <u>Staphylococcus</u> <u>aureus</u> to calves.

J. Dairy Sci. <u>65:</u> 271 - 274. Bienenstok, J. (1974).

> The physiology of the local immune response of the gastrointestinal trac. In: progress in immunology <u>Vol. 4</u> Ed. L. by Brent and J. Holbrow, Nor n Holland publishing, Holland, Pg. 197 - 205.

Blackburn, P.S. (1968).

The cell count of cow's milk and the

microorganisms cultured from milk.

J. Dairy Res. 35: 59 - 62.

Black, W.D. (1977).

Usefulness of Ancillary drugs in Mastitis

Therapy. J. Am. Vet. Med. Ass. <u>170</u>: 1187 - 1191. Blood, D.C. (1978).

The control of subclinical mastitis.

The Kenya Veterinarian, 2: 1, 3 - 6.

Blood, D.C. and Henderson, J.A. (1974).

Veterinary Medicine, Fourth edition. The English Language Book Society and Bailliere Tindall, Pullman, Washington, Pg. 257 - 292.

Bohl, E.H., Gupta, R.K.P., Olquin, M.V.F. and Saif. L.J. (1972).

> Antibody response in serum, colostrum and milk of swine after infection or vaccination with T.G.E. virus.

Infect. Immun. 6 (3) 389 - 300.

Bohl, E.H. and Saif, L.J. (1975).

Passive immunity in T.G.E. of swine; Immunoglobulin characteristics of antibody in milk after inoculating virus by different routes.

Infect. Immun. 11: 23 - 32.

Bramley, J.A. (1982).

Sources of <u>Streptococcus</u> <u>uberis</u> in the dairy herd. I. Isolation from boving faeces and from straw bedding of cattle.

J. Dairy Res. 49: 369 - 373.

Bramley, A, John, Kelvin S. Godinho, and Robert J. Grindal (1981). Evidence of penetration of the bovine teat duct by <u>E</u>. <u>coli</u> in the interval between milking.

J. Dairy Res. <u>48</u>: 379 - 386.

Bramley, A. John (1978).

The effect of <u>Staphylococcus epidermidis</u> infection of the lactating bovine udder on its susceptibility to infection with <u>Streptococcus agalactiae</u>, or <u>Escherichia coli</u>. Br. Vet. J. <u>134</u>: 146 - 151.

Bourne, F.J. and Curtis, J. (1973).

The transfer of immunoglobulin IgG, IgA and IgM from serum to colostrum and milk in the sow. Immuno. <u>24</u>: 157 - 162.

Bourne, J. and Newby, T. (1981).

Mucosal immunity.

In Practice. Sept. Pg. 5 - 11.

Bourne, F.J.; Newby, T.J.; and Chidlow, J.W. (1975). The influence of route of vaccination on the systemic and local immune responce in the pig. Res. Vet. Sci. <u>18</u> (3): 244 - 248. Brown, P.J., Bourne, F.J. and Denny, H.R. (1975). Immunoglobulin containing cells in Pig mammary gland.

J. Anatomy, <u>120</u>: 329 - 335.

Brookbanks, E.O. (1971).

Bovine mastitis in Relation to Intensive Farming. Aust. Vet. J. <u>47</u>: 226 - 232. Butler, J.E., Maxwell, C.F., Pierce, C.S., Hyton, M. B., Asofsky, R. and Kiddy, C.A. (1972). Studies on the relative synthesis and distribution of IgA and IgG, in various

tissues and body fluids of the cow.

J. Immunol. 109: 38 - 46.

Bushnell, R.B., Brazil, L. and Douglas, (1982). Evaluation of hygiene in controlling mastitis on large dairies. Proceedings of the II International Congress

of Hygiene, 1982. Pg. 19 - 20.

Carrol, E.J. (1977).

Environmental Factors in Bovine mastitis.

J. Am. Vet. Med. Ass. <u>170</u> (10) 1143 - 1149. Carrol, E.J. and Schalm, O.W. (1962).

Effect of Deoxyribonuclease on the California test for mastitis.

J. Immunol. 109: 38 - 46.

Carrol, E.J., Schalm, O.W. and Lasmanis, J. (1963) Experimental colliform (<u>Aerobacter aerogenes</u>) Mastitis: Characteristics of the endotoxin and its role in pathogenesis.

Am. J. Vet. Res. 25: 720 - 726.

Carrol, E.J. and Jain, N.C. (1969).

Bactericidal activity of normal milk, mastitic milk, and colostrum against <u>Aerobacter</u> <u>aerogenes</u> bacteria.

Am. J. Vet. Res. 30: 1123 - 1132.

Chang, C.C., Winter, A.J. and Nocross, N.L. (1981). Immune response in the bovine mammary gland after intestinal local and systemic immunization. Infect. and immun. <u>31</u>: (2) 650 - 659.

Chidlow, J.W. and Porter, P. (1978).

The role of oral immunization in stimulating <u>E. coli</u> antibody of the IgM class in percine colostrum. Res. Vet. Sci. <u>24</u>: 254 - 257.

- Craig, S.W. and Cabra, (1975).
 Rabbits peyer's Patches. Appendix and
 popliteal lymphnode B Lymphocytes. A
 comparative analysis of their membrane.
 J. Immunol.114: 492 502
- Crist, W.L., Heinder, L.E., Sears, P.M., Barr, H.L., Konalski, J.J. and Schimdt, G.M. (1982). Effectiveness of education efforts in implementing mastitis control procedures in commercial dairy herds. J. Dairy Sci., <u>65</u>: 823 - 834.

Cullen, G.A. and Hebert, C.N. (1967).

Some ecological observations on micro-organisms inhabiting Bovine skin, Teat canals and milk. Br. Vet. J. <u>123</u>: 14 - 25.

Donne, J. (1957).

Donne's Corpuscle Test. Dorland's illustrated Medical Dictionary, 23rd Ed. W.H. Sauders, Philadelphia, Pg. 1382.

David, L.H. (1953).

Experimental methods to control the spread of <u>Streptococcus agalactiae</u> in a dairy herd.

Vet. Rec. 65: 1 - 12.

. Dobbins, C.N. (1977).

Mastitis losses.

J. Am. Vet. Med. Ass. <u>170</u> (16) 1129 - 1132. Dodd, F.H. and Jackson, E.R. (1971).

Control of Mastitis.

Reading, Berks: British Cattle Veterinary Association NIRD. Pg. 390.

Dodd, F.H. and Neave, F.K. (1970).

Mastitis control. In biann.Review NIRD. Reading, England. Pg. 21.

Dodd, F.H., Westgarth, D.R., Griffin, T.K. (1977). Strategy of Mastitis control.

J. Am. Vet. Med. Ass. 170 (10) 1124 - 1132.

- Dodd, F.H., Neave, F.K. and Kingwill, R.G. (1964). Control of udder infection by management. J. Dairy Sci. 47: 1109 - 1112.
- Dunn, H.O., Murphy, J.M. and Garrett, O.F. (1943). Nature of the material in milk responsible for the modified whiteside test of mastitis. J. Dairy Sci. 26: 295 - 302.
- Fay, A.C., Cave, H.W., Atkenson, F.W. (1938). Detection of mastitis by the Bromothymol blue test, leukocyte count and the microscopic examination of incubated samples of milk. Cornell Vet. <u>28</u>: 40 - 50.

Fetlow, L. and Ferer, J.F. (1982).

Bovine leukemia virus infection and mastitis J. Dairy Sci. 65: 881 - 882.

Frost, A.J. (1962).

Incidence of Udder Infection and Mastitis :-South East Queensland.

Aust. Vet. J. 38: 110 - 113.

Funk, D.A., Freeman, A.E., and Berger, P.J. (1982). Environmental and physiological factors affecting mastitis at drying off and post calving. J. Dairy Sci. <u>65</u>: 1258 - 1268.

Gehring, E.L., Hall, R. and Sandoe, A.J. (1968).

The evaluation of Teat-dipping Formulation

Chlorhexidine.

Vet. Rec. 83: 112 - 114.

- 88 -

Gray, D.M. and Schalm, O.W. (1962). The mastitis variable in milk yield as estimated by California Mastitis Test.

Am. J. Vet. Res. 23: 541 - 547.

Gray, D.M. and Schalm, O.W. (1960).

Interpretation of the California Mastitis Test results on milk from individual mammary quarters, bucket milk, and bulk herd milk.

J. Am. Vet. Med. Ass. <u>136</u>: 195 - 198. Griffin, T.K. (1971).

> The control of bovine mastitis. (Editors: F.N. Dodd and E.R. Jackson of the national institute of research in dairying) Reading, England.

Gustafson, D.P., Amstutz, H.E., Beiber, L., Dodd, F.H., Fox, F.H., Arthur, F., Gale, C., Jarrett, J.A., McCallon, W., Miller, R.E., Natzke, R.P., Parker, R.A., Postle, D.S., Schalm, O.W., Stowe, C.M., Van, C.D. and West, R.L. (1977). Colloquium on Bovine Mastitis.

J. Am. Vet. Med. Ass. <u>170</u> (10) 1119 - 1123.

Hamir, A.N., Gehring, W., and Muhammed, S.I. (1978). Incidence of bovine mastitis in Kenya. Bull. Animal Health Prod. Afric. 26: 55 - 61.

Hamir, A.N., Gehring, W., and Muhammed, S.I. (1979). Control of subclinical mastitis by intramammary antibiotic infusion during lactation (Blitz therapy) in Kenya.

Bull. Animal Health Prod. Afric. 27: 119 - 202.

Hammer, B.W. and Bailey, D.E. (1917).

A rapid volumetric method for the approximate estimation of chlorine in milk.

Iowa Ag. Exp. Sta. Res. Bull. <u>41</u>: 365 - 370. Hayden, C.E. and Johnson, S.D. (1934).

> A non-alcoholic bromothymol blue solution. Cornell Vet. 24: 270 - 282.

Hicks, O.G., Kennedy, T.J., Keister, D.M. and Miller, M.L. (1981).

> Evaluation of a teat dip of Chlcrohexidine Digluconate (5%) with Glycerin (6%).

J. Dairy Sci. 64 (11) 2266 - 2269.

Heidrich, H.J. and Renk, W. (1967).

In: Diseases of the Mammary Glands of Domestic Animals. Translated by L.W. Van den Heaver. W.B. Saunders Company. Philadelphia and London. Huter, W.G. (1977).

Antibacterial drug effectiveness against mastitis pathogens.

J. Am. Med. Ass. 170: 1182.

Jaartsveld, F.H.J. (1961).

Contribution to diagnostics of mastitis in cattle in connection with the mastitis control. Thesis, University of Utrecht. The Netherlands. Jain, N.C., Schalm, C.W., Carrol, E.J. and Lasmanis, J. (1968).

> Experimental mastitis in leukopenic cows: Immunologically induced neutropenia and response to intramannary inoculation of <u>Aerobacter aerogenes.</u>

Am. J. Vet. Res. <u>29</u> (nov.) 2089 - 2097.
Jain, N.C., Schalm, O.W. and Lasmanis, J. (1969).
Comparison in normal and leukopenic cows
of experimental mastitis due to <u>Aerobacter</u>
<u>aerogenes</u> or <u>E. coli</u> Endotoxin.

Am. J. Vet. Res. 30 (May) 715 - 724.

Jain, N.C. Schalm, O.W. and Lasmanis, J. (1971). Experimentally induced coliform (<u>Aerobacter</u> <u>aerogenes</u>) Mastitis in normal cows and in cows made neutropenic by an Equine Anti-Bovine Leukocyte Serum.

Am. J. Vet. Res. <u>32</u> (Dec). 1929 - 1935. Jain, N.C., Vegad, J.L., Jain, N.K. and Shrivastava, A.B. (1982).

> Haematological studies on normal lactating Indian water buffaloes.

Res. Vet. Sci. <u>32</u>: 22 - 56.

Jasper, D.E. (1982).

Personnal communication and comments. University of California, Davis, U.S.A.

- 31 -

Jean-Francois Back., Immunology, Edition (1982).

A Willey Medical Production Pg. 195, 196, 344 and 526.

Johnson, S.D. and Trudel, F.G. (1932).

Observations of the significance of leukocytes in milk.

Cornell Vet. 22: 354.

Kaiser, E. (1966).

Cell content of milk at the end of the lactation cycle.

Dairy Sci. Abst. 1576.

Kalra, D.S. and Dhanda, M.R. (1964).

Incidence of mastitis in cows and buffaloes in North West India.

Vet. Rec. 76 (8) 219 - 222.

Kariuki, D.P. (1977).

The use and abuse of antibiotic in livestock. The Kenya Vet. <u>1</u>: 8 - 9.

Kenya annual reports, 1982(a).

Nyeri district annual reports, Ministry of Livestock Development.

Kenya Annual Reports, 1982(b).

Veterinary Investigation Laboratory, Karatina, Kenya.

Ministry of Livestock Development.

Kenya annual report, (1982)(c).

Provincial annual reports, Central Province, Kenya.

Ministry of Livestock Development.

Kerr, W.R., Pearson, J.K.Z. and Rankin, J.E.F. (1959). The bovine udder and it's agglutinins. Br. Vet. J. 115: 105 - 119.

Kingwill, R.G., Neave, F.K., Dodd, F.H., Griffin

T.K. and Westgarth, D.R. (1970).

The effect of mastitis control systems on levels of sub-clinical mastitis in two years. Vet. Rec. <u>87</u>: 94 - 112.

Klein, J.W. (1943).

Influence of length of dry period upon the quantity of milk produced in the subsequent lactation.

J. Dairy Sci., 26: 705.

Kowalski, J.J. (1977).

Microbial agents and bovine mastitis.

J. Am. Vet. Med. Ass. <u>170</u>: 1175.

Landrey, J.S.A. (1965).

The effect of mastitis on herd milk production and composition.

J. S. Afr. Vet. Med. Ass. <u>36</u>: 515 - 519. Lamm, M.E. (1976).

Cellular aspects of Immunoglobulin A.

Advances in Immunology, 22: 223 - 290.

Larson, B.L. and Kendall, K.A. (1957).

Changes in specific blood serum protein level associated with parturition in the bovine,

J. Dairy Sci. <u>40:</u> 559 - 666.

Lauerman, L.H., Greig, W.A., Buck, H.A. and Lutu, W.Z. (1973).

Bovine mastitis in Kenya.

Bull Epizoot. Dis. Afr. 21: 167 - 170.

McEwen, A.D. and White, M.B. (1950).

Variation in bactericidal and bactericstatic properties of milk.

Vet. Rec. 62: 27 - 30.

Mach, J.P. and Pahud, J.J. (1971).

Secretory IgA, a major immunoglobulin

in most bovine external secretions.

J. Immunol. 106: 552.

McDermortt, M.R. and Bienenstock, J. (1979).

Evidence for common mucosal immunologic system. I. Migration of B immunoblasts into intestitial respiratory and genital tissues.

J. Immunol. 122: 1892 - 1898. -

Merchant, I.A. and Packer, R.A. (1944).

Etiology, Diagnosis and Control of infectious Mastitis.

Burgess Pub. Co., Minneapolis, Minn., Pg. 35.

Michaleck, S.M., McGhee, J.R., Mestecky, J., Arnold, R.R. and Bozzo, L. (1976). Ingestion of <u>Streptococcus mutans</u> induced secretory IgA and Carries imunity.

Science, <u>192</u>: 1238 - 1240.

- Michel, G. Seffner, W., Schultz, J. (1974). Zur Frage der Hyperkeratose des Strichkanalepithels der Zitze des Rindes Monatsschr Vet. Med. <u>29</u>: 570 - 574.
- Ministry of Agriculture Fisheries and Food, (1978). Manual of Veterinary Investigation Laboratory Technique.

Waybridge, Britain.

Moak, H. (1916).

Control and eradication of infectious mastitis in dairy herds.

Cornell Vet. 6: 36.

Monlux, A.W. (1948).

The Catalase test in the diagnosis of infectious bovine mastitis.

Cornell Vet. 38: 389. - 390..

Montgomery, P.C., Rosner, B.R. and Cohen, J. (1974). The secretory antibody response. Anti DNP antibodies induced by dinitrophenylated type 11 pneumococcus.

Immunol. Commun. 3: 143 - 156.

Mugera, G.M., Bwanganci, O., Wandera, J.G. (1979).

Diseases of cattle in Tropical Africa. Kenya Literature Bureau, Nairobi.

Pg. 172 - 189.

Murnane, D. (1946).

Clinical bovine mastitis, treatment and control. Aust. Vet. J., 22: 156 - 168.

Murphy, J.M. and Hanson, J.J. (1941).

A modified whiteside test for the detection of chronic bovine mastitis.

Cornell Vet. 31: 47 - 55.

Murphy, J.M. (1942).

Further observation on the modified whiteside test for the detection of bovine mastitis. Cornell Vet. <u>32</u>: 439 - 442.

Mutuiri, S.N. (1980).

Animal diseases in Kenya: A priority appraisal. A report of the National Council for Science and Technology. No. 5. 439 - 444.

Neave, F.K., Dodd, F.H. and Henriques, E. (1950). Udder infection in the dry period. I. J. Dairy Res. <u>17</u>: 37 - 38.

Neave, F.K., Dodd, F.H. and Kingwill, R.G. (1966).

A method of controlling udder diseases.

Vet. Rec. 78: 521 - 522.

Neave, F.K. (1968).

Mastitis in dairy cattle.

J. Dairy Res. <u>35</u>: 127 - 129.

Newbould, F.H.S. and Barnum, D.A. (1960).

The reduction of the microflora of milking machine, inflaction by teat dipping and teat cup pasteurising.

J. Milk Food Tech. 23: 37 - 39.

Newbould, F.H.S. (1974).

Microbial disease of the mammary gland in lactation: "A comparative Treatise". Editors: Larson, B.L. and Smith, V.R. Acadermic Press, Inc. New York. II: 269 - 316.

Newby, F.J., Stokes, C.R. and Bourne, F.J. (1982). Immunological activities of milk.

Vet. Immunol. and Immunopathol 3: 67 - 94.

Newby, T.J. and Bourne, J. (1977).

The nature of the local immune system of the bovine mammary gland.

J. Immunol. 118 (2) 461 - 465.

Norman, B. (1973).

The dairy farmers vet. book. Farmers press

Ltd. Wharedale road, Ipswich.

Norcross, N.L. (1963).

Antigenic substances purified from <u>Streptococcus</u> <u>agalactiae</u> I. Antibody response in infected cattle.

Cornell Vet. <u>53</u>: 301 - 308.

Norcross, N.L. (1964).

Antigenic substances purified from <u>Streptococcus</u> <u>agalactiae</u> extracellular products.

Am. J. Vet. Res., <u>25</u>: 1457 - 1461. Norcross. N.L. and Stark, D.M. (1970).

Immunity to mastitis. A review.

J. Dairy Sci. 53: 387 - 393.

Ogaa, J.S. (1980).

Strategy of effective treatment of acute mastitis in the field.

The Kenya Veterinarian, 4: 2, 16 - 19.

Ogaa, J.S. (1981).

Personal communication: The control of Bovine mastitis by management.

Cliver, J., Dodd, E.H. and Neave, F.H. (1956). Udder infection in the "dry period" V. The effect of teat disinfection at drying off on the incidence of infection in the early dry period.

J. Dairy Res. 32: 212 - 214.

Oliver, J., Neave, F.K. and Sharpe, M.E. (1962). The prevention of infection of the dry udder. J. Dairy Res. <u>29</u>: 95 - 98. Paape, M.J., Hafs, H.D. and Tucker, H.A. (1963). Relationship of Feulgen - DNA in milk to the number of somatic cells.

J. Dairy Sci., 46: 625 - 634.

Pankey, J.W., Barker, R.M., Towmet, A. and Durrs, G (1982). A note on effectiveness of dry cow therapy in New-Zealand Gairy herds. N. Z. Vet. J. 30: 50 - 52.

Parisi, J.T. and Baldwin, J.N. (1963).

The incidence and persistence of certain strains of <u>Staphylococcus</u> <u>aureus</u> in dairy herds. Am. J. Vet. Res. <u>24</u>: 551 - 556.

Petersen, W.E., Grimmel, J.F. and Schipper, I.A. (1950). Factors involved in the whiteside reaction.

J. Dairy Sci. 33: 384 - 386.

Petzoldt, K. and Benten, C. Von. (1978).

Passive allergisation of calves and lambs due to colostral antibodies.

Ann. Res. vet. 2: 235 - 238.

Philpot, W.N. (1979).

Control of mastitis by hygiene and therapy.

J. Dairy Sci. 62: 168 - 176.

Phipps, L.W. and Newbould, F.H.S. (1966).

Determination of leucocytes concentration

in cow's milk with a coulter counter.

J. Dairy Res. 33: 57 - 62,

Pierce, A.E. and Feinsten. A. (1965).

Biophysical and immunological studies on bovine immune globulin with evidence for selective transport within the mammary gland from maternal plasma to colostrum. Immunology 8: 106 - 123.

Prescott, S.C. and Breed, R.S. (1910).

The determination of the number of body cells in milk by a direct method. J. inf. Dis. <u>7</u>: 632 - 635.

Porter, P., Noakes, D.E. and Allen, D.W. (1970). Secretory IgA and antibodies to <u>Escherichia</u> <u>coli</u> in porcine colostrum and milk and their significance in the alimentary canal of the

young pig.

Immunology <u>18</u>: 245 - 257.

Postle, D.S. (1964).

The Winsconsin mastitis test.

Proc. U.S. Livestock Sanitary Ass. <u>68</u>: 488 - 499. Ramsey, W.S. and Grant, L. (1974).

> Chemotaxis in the inflammatory process, Vol. I. Edited by B.W. Sweifact et al. Academic press. NY: 316 - 328.

Roberts, S.J., Neave, F.K. and Oliver, J. (1969).

Concepts and recent developments in mastitis control.

J. Am. vet. med. Ass. 155: 157 - 158.

Rusell, J.M. (1932).

Determination of chlorides in milk, N.Y. State Ag. Exp. Sta. Tech. Bull. <u>199</u>: 1 - 102. Rudzick,O., Perey, D.Y.E. and Bienenstock, J. (1975). Differential IgA repopulation after transfer of autologous and alogenic rabbit Peyer's patches cells.

J. Immunol. <u>144</u>: 40 - 41.

- Saif, L.J., Bohl, E.H. and Gupta, R.K.P. (1972). Isolation of porcine immunoglobulins and Determination of T.G.E. viral antibodies. Infect. Immun. <u>6</u>: 600.
- Sarwar, M., Campbell, B. and Petersen, W.E. (1964). Production of antibody in mammary gland of pregnant non-lactating and lactating cows evoked by polyvalent antigen with reference to damage and frequency of immunization. Can. J. Comp. vet. Med. <u>28</u>: 184 - 192.

Schalm, O.W. and Lasmanis, J. (1968).

The leukocytes: Origin and function in Mastitis. J. Am. vet Med. Ass. <u>153</u>: (Dec. 15): 1688 - 1694. Schalm, O.W. (1977).

Pathologic changes in the milk and udder of cows with mastitis.

J. Am vet. Med. Ass. <u>170:</u> 10. 1137 - 1140. Schalm, O.W., Carroll, E.J., Jain, N.C. (1971). Bovine mastitis: Lea and Fabiger, Philadelphia. Schalm, O.W. and Lasmanis, J. (1957).

Distribution of micrococci and other bacteria in milk samples from a single herd after twelve years of mastitis control. Am. J. vet. Res. 18: 778.

Schalm, O.W., Lasmanis, J. and Carroll, E.J. (1964). Pathogenesis of experimental Coliform (<u>Aerobacter aerogenes</u>) Mastitis in cattle. Am. J. vet. Res. 25: 75 - 82.

- Schalm, O.W., Gray, D.M. and Noorlander, D.C. (1955). Procedures for the use of whiteside test on milk in the laboratory or barn. North Am. Vet. 36: 1011.
- Schalm, O.W., Pier, A.C., Gray, D.M. and Noorlander, D.O. (1956).

Application of the whiteside test to bucket milk and foremilk in herd programmes for the prevention and control of mastitis.

Calif. vet. 2: 24 - 26.

Schalm, O. W. and Noorlander, D.O. (1957).

Experiments and observation leading to development of the California Mastitis Test.

J. Am. vet. Med. Ass. 130: 199.

Seiber, R.L. and Farnsworth, R.J. (1981.

Prevalence of chronic teat-end lesions and their relationships to intramammary infections in 22 Herds of Dairy Cattle.

J. Am. vet. Med. Ass. 178 (2) 1.53 - 1257.

Shaw, O.A. and Beam, A.L. (1935).

Effects of mastitis on milk production. J. Dairy Sci. <u>18</u>: 353.

Smith, A., Dodd, F.H. and Neave, F.K. (1968). The effect of intramammary infection during the dry period on the milk production of the affected quarter at the start of the succeeding lactation.

J. Dairy Res. 35: 287 - 290.

Smith, K.L., Muir, L.A., Ferguson, L.C. and Conrand, H.R. (1971).

> Selective transport of IgG₁ in to the mammary gland: Role of oestrogen and progestrome.

J. Dairy Sci. 54: 1886 - 1894.

Smith, J.W. and Schultze, W.D. (1967).

Variation in cell content of milk associated with time of sample collection. 1. Diurnal variation. J. Dairy Sci. <u>50</u> (July): 1083 - 1091.

Smith, A., Neave, F.K. and Dodd, F.H. (1966). Methods of reducing the incidence of udder infection during the dry period.

Vet. Rec. 79: 233.

Spencer, G.R. and Simon, J. (1960).

The catalase, California, and cell count tests for detecting abnormalities in milk. Am. J. vet. Res. 21: 578 - 584.

Stang, A.M. (1977).

Pharmacological principles of systemic and intramammary mastitis therapy.

J. Am. vet. Med. Ass. <u>170</u>: 10, (2) 1180 - 1192. Turner, C.W. (1952).

The mammary gland. I. The anatomy of the udder of cattle and domestic animals.

Columbia, Mo. Iucas Brothers, Publishers, Columbia.

Turner, M.W., McClelland, D.B.L., Medlen, A.R. and Stokes, C.R. (1977).

IgE in human urine and milk.

Scand. J. Immunol. <u>6</u> 343 - 348.

- Udall, D.H. and Johnson, S.D. (1931). The diagnosis and control of mastitis. Cornell vet. 21: 190 - 192.
- Udall, D.H. (1947).

Teat erosions.

Cornell Vet, 37: 73 - 77.

Udall, D.H., Johnson, S.D. and Ferguson, J. (1938). The control of mastitis in New York State. Vet. Rec. 50: 1417 - 1419.

Wayne, R. and Macy, H. (1933).

The effect of various methods for drying up cows on the bacterial and cell content of milk.

J. Dairy Sci. 16: 79 - 91.

Weigt, U. (1973). Gezielte therapie der akuten mastitiden in der praxis. Der praktische Tieraszt. Colloquim vetenarium, 53: 26 - 30. Wesen, D.B. and Schultz, L.H. (1970). Effectiveness of a Fost-milking Teat-Dip in preventing new udder infections. Whiteside, W.H. (1939). Observation on a new test for the presence of mastitis in milk. Can. Pub. Health J. 30: 44 - 47. Weisner, H.W. (1974). Ubersicht screferat: Gewebeschaden des Rinderesulers beim. Maschinellen milchentuzug Ditsh Tieraerztl Wolchenschr. 81: 413 - 417. Wilson, C.D. and Kingwill, R.G. (1975). A practical mastitis control routine. In: Proc. Int. Dairy Fed. Ann. Bull. 85: 422 - 438. Wilson, C.D., Richards, M.S. (1980). A survey of mastitis in British dairy herds. Vet. Rec. 106:(21) 431 - 435.

- 105 -

Yokomizo, Y. and Norcross, N.L. (1978).

Bovine antibody against <u>Streptococcus agalactiae</u> Type 1a produced by parturient intramammary and systemic vaccination.

Am. J. vet. Re.s. 39: 511 - 516.

Ziv, G. (1978).

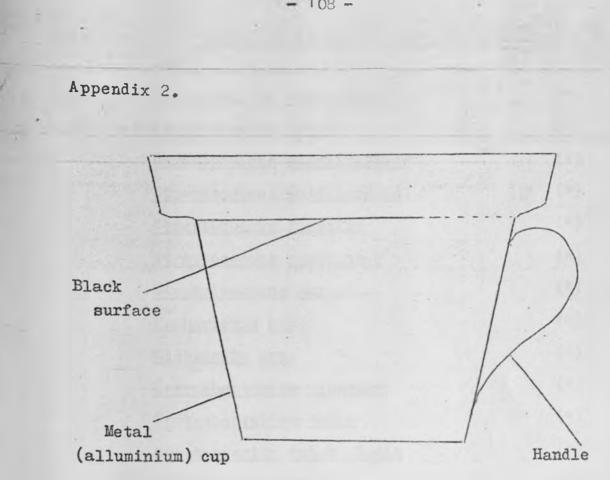
Practical pharmacokinetic aspects of mastitis therapy. Pro-Bovine Mastitis Regional Seminar sponsored by University of Minnesota, Minnesota Assn. of Bovine Practitioners and Beecham Labs. P.L.

Appendix 1. <u>DISTRICT ANNUAL REPORT ON CLINICAL</u>

MASTITIS.

District				
-	Year	1980	1981	1982
NYERI		444	2598	4814
KIRINYAGA				1999
MURANG'A		570	1610	4034
NYANDARUA		1200	1081	4473 .
KIAMBU		595	1166	2657

Source: Kenya annual report (1980, 1981 and 1982) Ministry of Livestock Development, Provincial annual report, Central Province.



An illustration of a strip cup.

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Appendix 3. CAUSATIVE AGENTS OF MASTITIS IN CATTLE.

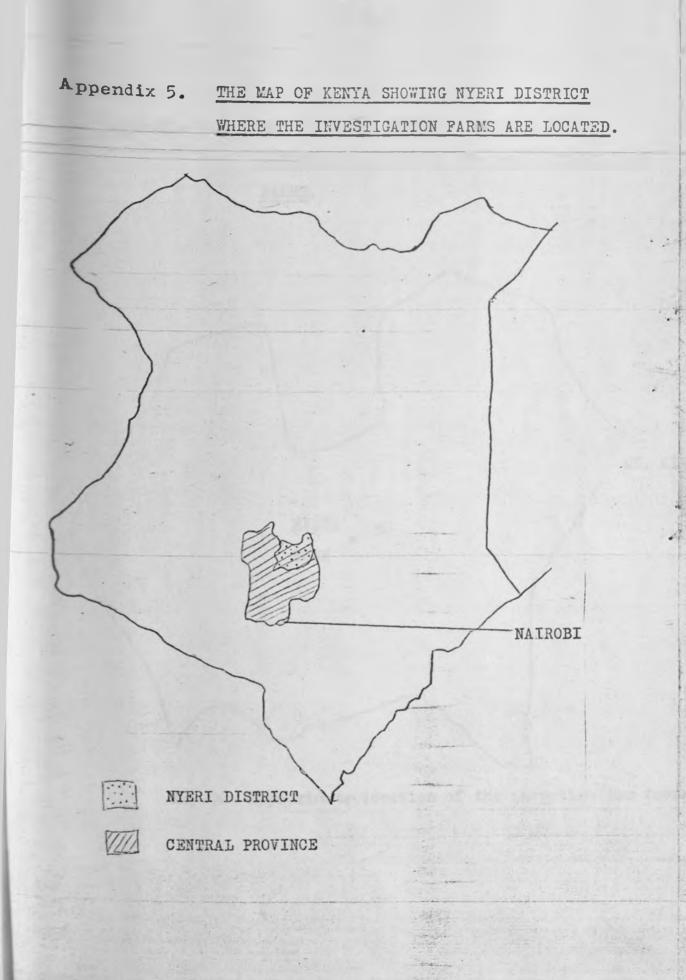
acteria	Streptococcus agalactiae	(*)
	Streptococcus uberis	(*)
	Streptococcus zooepidemicus	(*)
	Streptococcus dysgalactiae	(*)
	Streptococcus faecalis	(*)
	Streptococcus pneumoniae	(*)
	Staphylococcus aureus	(*)
	Escherichia coli	(*)
	Klebsiella spp.	(*)
	Corvnebacterium pyogenes	(*)
	Corynebacterium bovis	(*)
	Mycobacterium tuberculosis	(*)
	Mycobacterium spp.	(**)
	Bacillus cereus	(*)
	Pasteurella multocida	(*)
	Pseudomonas pyccyaneous	(*)
	Sphaerophorus necrophorus	(*)
	Serratia marcescens	(*)
	Mycoplasma spp.	(*)
	Norcadia sop.	(*)
Fungus	Trichosporon spp.	(*)
Yeast	Candida spp.	(*)
	Cryptococcus neoformans	(*)
	Saccharomyces spp.	(*)
	Torulopsis spp.	(*)

1974.

ACTERIA.			Y	EARS		
	1978	1979	1980	1981	1982	Total
Streptococcus spp.	15	19	18	40	18	1 10
Staphylococcus spp.	2	9	18	25	52	106
Escherichia coli	11	10	23	16	5	65
Klebsiella spp.	4	19	42	70	27	162
Corynebacterium spp.	. –	-	9	6	12	27
Pseudomonas spp.	_	4	6	9	-	19
OTHER BACTERIA*	9	19	2	8	31	69
FUNGUS						
Norcardia spp.	-		-	-	-	-
YEAST: **	-		_		-	-
Total	41	80) 58	3 104	149	562

Development, Karatina Veterinary Investigation Laboratory, (1978 - 1982).

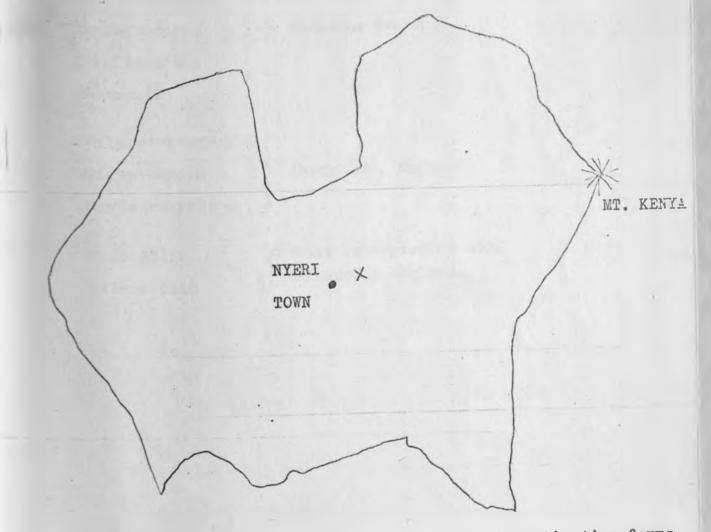
Note- * Other bacteria: <u>Bacillus cereus etc.</u> ** Yeast : <u>Candida spp.</u>



4

Appendix 6. MAP OF NYERI DISTRICT SHOWING THE

APPROXIMATE LOCATION OF THE INVESTIGATION FARMS.





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Appendix 7. THE DRUGS FOR WHICH MICROBIAL ACTIVITY WAS TESTED AND THEIR SOURCES.

Chloramphenicol)	Sources	
Penicillin)		
Erythromycin	A.B. Biodisk pyramidvagen 7	171
Trimethoprim	36 Solna Sweden.	
Sulfisomidin }		
Neomycin)		
Chlorotetracycline Streptomycin	Oxoiā Ltd. England.	
Oxytetracycline)		
Ampicillin Cloxacillin	Mast laboratories Ltd, Liverpool England.	

Appendix 2. ESTIMATED LOSSES DUE TO VARIOUS DEGREES OF INFECTION.

1

CMT infection levels	Production loss 10/quarter/day
Negative	0
Trace	0.96
1	2.18
2	3.88
3	5.74
Source: Dobbins, C.N.	(1977).

Mastitis losses.

J. Am. vet. Med. Ass. 170 (16) 1129 - 1132.