

THE PROPHYLACTIC TREATMENT
OF BOVINE MASTITIS USING
HOMOEOPATHIC NOSODE THERAPY

by

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requirements for the Master's Degree in Technology in the
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ABSTRACT

The purpose of this study was to determine the efficacy of homoeopathic nosode therapy as a prophylaxis for bovine mastitis. It was hypothesised that the nosode would decrease the somatic cell counts in milk samples taken from the experimental group and hence the incidence of clinical and subclinical mastitis.

A placebo-controlled double-blind experimental design was chosen. The sample comprised 69 Friesland cows selected from a dairy farm in the Karkloof area of KwaZulu-Natal and was divided randomly into two groups of approximately equal size. Each group was tagged, thus easily identified for treatment purposes. During the trial the two groups intermingled freely, and were therefore exposed to the same external influences.

The medication and placebo were provided in identical 50 ml bottles, each labelled with a code corresponding to one of the groups. For each treatment, five millilitres from each bottle were

diluted into two separate containers, each holding one litre of drinking water. The dilutions were administered orally to the animals with 100 ml syringes filled to give a 50 ml dose per animal.

A baseline somatic cell count was taken from the two groups, after which treatment and sampling took place on a monthly basis over a five month period. The milk samples were taken by a trained animal health technologist and sent to Allerton Regional Veterinary Laboratory for analysis.

The somatic cell counts were obtained using a coulter counter, which records the number of cells counted per millilitre. Any count over 485 000 was automatically considered subclinical mastitis, and was cultured to determine the infective microorganisms. In addition, all cases of clinical mastitis identified were noted on a monthly basis.

At the conclusion of the five month period, only 57 cows remained in the study, i.e. 28 in the experimental group and 29 in the placebo group. Each cow had six somatic cell count results, as well as a

record of clinical or subclinical mastitis.

These data were then analysed using two methods, viz. the two sample t-test to compare differences between the two groups throughout the treatment, and Wilcoxon's two-sample tests to test whether animals within the same group improved as the treatment progressed. The level of significance used was $\alpha=0,05$.

No significant differences were noted between the two groups, based on the mean somatic cell counts and the incidence of clinical and subclinical mastitis. Although significant improvement was noted on seven occasions within each group as the trial progressed, this was difficult to ascribe to the intervention as it occurred randomly and equally within both groups.

The study thus failed to provide conclusive evidence that homoeopathic nosode therapy is an effective prophylaxis for bovine mastitis. The results did, however, highlight several weaknesses in the study design. If resolved, these aspects would increase considerably the data base from which valid observations could be made.

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CHAPTER 1 - INTRODUCTION

1.1 THE PROBLEM OF MASTITIS

Mastitis probably effects a greater loss to the dairy industry than does any other infectious disease. It is an inflammation of the mammary glands and generally is caused by one of several different organisms. It may cause milk to be unsaleable, or result in temporary or permanent loss of one or more quarters of an udder. (Judkins and Keener 1960: 182.)

In terms of the problem in South Africa, figures from the Allerton Regional Veterinary Laboratory (ARVL) mastitis scheme for KwaZulu-Natal (1993-1994) show that 45,64 % of the samples had somatic cell counts (namely, of the white blood

and udder epithelial cells) high enough to be considered an indication of subclinical mastitis (ARVL 1994d).

The most common causative organism at present is *staphylococcus aureus* (30,6%), which is also the most difficult to treat (ARVL 1994d). This gives an indication of how prevalent mastitis is, despite widespread measures to control it. The cost to each dairy farmer and to the dairy industry as a whole is enormous due to reduced yields, discarded milk during antibiotic treatment and the culling of severely affected cows (Stopes and Woodward 1988).

The situation is unlikely to improve, since the demand for milk is increasing constantly, placing the onus on dairy farmers to produce increasingly higher yields, and presenting a vicious circle: mastitis causes unsaleable milk, farmers are thus forced to increase yields to meet demands, so exacerbating the incidence of mastitis (Day 1992).

1.2 TREATMENT

1.2.1 Allopathic treatment

Current allopathic treatment with antibiotics, rather than providing the solution, presents a number of problems.

Firstly, these drugs pollute the milk with antimicrobial residues (Steele and Beran 1984), rendering it unacceptable with regard to human nutrition and health (Giesecke *et al.* 1994: 59).

Secondly, antibiotic treatments consistently encounter bacterial resistance problems, necessitating the development of new drugs (Day 1992).

A third drawback is that antibiotic treatments are often unsatisfactory in effect under field conditions, since they treat only the infectious agents and not the udder inflammation. This causes long term damage to the udder

tissue. (Schalm *et al.* 1971.) In addition, the allopathic treatment of bovine mastitis has generally followed a reactive approach, with antibiotic treatment being administered only after signs of infection have been diagnosed (Giesecke 1979).

Such a symptomatic approach is expensive, unsuccessful and inefficient for two reasons. Firstly, by the time the infection is diagnosed, it will have reached such an advanced stage that udder tissue is damaged irreparably. Secondly, preoccupation with individual cases of clinical mastitis ignores completely the problem of subclinical mastitis, which is far more prevalent and therefore demands urgent attention. (Giesecke 1979.)

1.2.2 Homoeopathic treatment

Comparing homoeopathic with allopathic remedies, it is clear that homoeopathy offers an attractive alternative to the use of

antibiotics. Individual remedies can be prescribed for specific cases of mastitis, depending on the presenting symptoms of each cow, as opposed to administering just one or two drugs to the entire herd and hoping, despite differing symptoms and conditions, that they will work.

In addition, homoeopathic remedies are far cheaper than antibiotics, making them a prudent choice, particularly in the large amounts required by a herd of dairy cows. In the long term, they effect no harmful residues in the milk, and bacterial resistance does not occur. Most significant is that the remedies can be used prophylactically, in the form of nosodes, thus preventing the onset of disease; this form of treatment, therefore, stands in stark contrast to the reactive methods employed by allopathic medicine. (MacLeod 1979.)

1.3 THE NOSODE SOLUTION

The aim of this study was to attempt to solve the problem of bovine mastitis prophylactically; if successful, prophylactic treatment would of necessity reduce the incidence of both clinical and subclinical mastitis.

Previous research in this field includes a clinical trial on bovine mastitis conducted by Dr Christopher Day in England. This trial, a pilot study, employed homoeopathic nosodes made from the bacteria commonly causing mastitis. Favourable results prompted Day to conclude that the study was worthy of repetition and further research. (Day 1986.)

It was hoped, therefore, that by repeating the study - this time under South African conditions - firmer evidence would be obtained to prove the effectiveness of homoeopathic nosodes in the treatment of bovine mastitis.

CHAPTER 2 - REVIEW OF THE RELATED LITERATURE

2.1 INTRODUCTION

The word 'mastitis' is derived from the Greek words *mastos* (breast) and *itis* (inflammation). Hence, the term 'bovine mastitis' defines the inflammation of the mammary glands of a cow. (Campbell and Marshall 1975: 330.)

Man has been faced with the problem of mastitis ever since he chose to breed cattle specifically for their milk-producing qualities, thereby creating an intensive industry with its main objective being high yields. This industry has produced an

animal with an abnormally large udder and therefore an enormous capacity to produce milk, but the cost is an increased susceptibility of the bovine mammary glands to infection. (Foley *et al.* 1972: 401.)

2.2 HISTORICAL PERSPECTIVE

The first cases of clinical mastitis in South African dairy cattle were reported at the turn of this century; by this time 70 years of similar research had already been conducted in Switzerland, France and Germany. (Giesecke *et al.* 1994: 163.)

Between 1910 and 1937 no records of any noteworthy progress on mastitis control in South Africa were kept, suggesting that the control of clinical mastitis remained limited to the 'single

cow' approach, a tradition still overemphasised by many milk producers. (Giesecke *et al.* 1994: 163.)

In 1938, however, the attention of South African research switched to aspects of subclinical mastitis. Advancement graduated, between 1945 and 1965, to a non-standardised type of specific single herd approach, adapted if necessary to supplement the single cow approach in herds with serious problems of clinical mastitis. (Giesecke *et al.* 1994: 163.)

Complications, however, occurred between 1968 and 1970, when dairy farmers began indiscriminately to administer antibiotics instead of using them as recommended, to supplement good parlour management. (Giesecke *et al.* 1994: 163.)

From 1976 onwards, mastitis control progressed to a standardised extensive regional approach supported in a limited, though growing, number of herds. There was a

repeated monitoring of somatic cell count values in herd milk as well as veterinary advice on various aspects of the prevention and control of subclinical and clinical mastitis. (Giesecke *et al.* 1994: 163.)

2.3 ECONOMIC PERSPECTIVE

During 1978 it was estimated that the direct and indirect costs of bovine mastitis in South Africa totalled almost R 190 million per year (Giesecke *et al.* 1994: vii). The most recent tally approximates R 529 million per year, making it the biggest single health problem confronting the dairy industry (Onderstepoort Veterinary Institute 1996).

Such losses are almost incredible unless one appreciates the wide range of deleterious effects which mastitis has on the productivity of dairy farming, as well as on the hygiene quality, safety and general usefulness of milk as a valuable food and industrial raw product (Giesecke et al. 1994: vii).

2.4 AETIOLOGY

The intramammary development of mastitis depends on three interactive key elements: intramammary bacterial challenge, intramammary defence, and intramammary destruction of tissue (Giesecke et al. 1988). The most common mastitis pathogen at present in KwaZulu-Natal is *staphylococcus aureus* (ARVL 1994d).

Also prevalent in the province are the pathogens *staphylococcus epidermidis*, *streptococcus uberis*, *streptococcus dysgalactiae* and *streptococcus agalactiae*. *Corynebacterium* (generally assumed to be *clostridium bovis*) is a minor mastitis pathogen which has nevertheless been prevalent over the past 10 years among dairy herds in KwaZulu-Natal. It is regarded predominantly as a teat canal 'contaminant' due to ineffective post-milking teat disinfection, or to physical damage of the teat canal as a result of overmilking. (ARVL 1994d.)

A cow's metabolic adjustments and immunological responses are its most important natural mechanisms for protecting the integrity of its tissues and its life (Giesecke et al. 1994: 70). The lactating cow and its udder have high metabolic requirements. They are thus particularly susceptible to the stressful conditions associated with metabolic environmental factors (including a hot climate, exertion, transportation, starvation

and lactation) which may readily affect the metabolism and integrity of mammary tissue. (Giesecke *et al.* 1988.)

In addition, the udder is almost constantly exposed to various microbial environmental factors, for example a number of bacteria, soiled milking equipment or bedding and flies. Deficient immunological responses to bacterial challenges may thus promote udder infections and elevate the risk of mastitis. (Giesecke *et al.* 1994: 72.)

Each case of mastitis, regardless of its specific type of conditions, amounts to a fight at the cellular level between the cow and a harmful agent causing tissue damage in the udder. Normally, the development of the intramammary lesion precedes mastitis. (Giesecke *et al.* 1994: 6.)

The time difference between the two is increased because detection of mastitis by means of a diagnostic technique -

visual detection of mastitis floccules in the milk and slight swelling of the udder or erythema - usually occurs only once the infection has progressed to an advanced stage. Since increased time delays result in increased tissue damage, minimising time delays is a factor of vital importance for the successful therapy of mastitis. (Giesecke *et al.* 1994: 6.)

2.5 DIAGNOSIS

Mastitis can be classified into three basic types: acute clinical mastitis, chronic mastitis and subclinical mastitis.

2.5.1 Acute clinical mastitis

This form of mastitis is recognised easily, by the abnormal appearance of the milk from the affected quarter as seen when milk is expressed from the udder. The milk may contain clots of pus, be watery, or contain blood. In addition, the affected quarter may be swollen, reddened, hot and painful. (ARVL 1994a.)

The cow will also exhibit general symptoms of disease, with distressed behaviour and an increased body temperature (Giesecke *et al.* 1994: 5). In severe cases of acute clinical mastitis the somatic cell count values frequently exceed 15-20 million cells per millilitre of milk, and tend to clump together to form floccules in the milk (Giesecke *et al.* 1994: 112).

2.5.2 Chronic mastitis

Frequently developing from other types of mastitis, chronic mastitis progresses over a period of weeks, possibly from lactation to lactation. It causes progressive damage to the udder tissue, with acute flare-ups occurring regularly. In long standing cases the affected quarter(s) may be smaller and more solid than those not affected, with no marked swelling or pain necessarily evident on palpation, and the cow may be generally healthy. (Giesecke *et al.* 1994: 5.)

2.5.3 Subclinical mastitis

Subclinical mastitis cannot be diagnosed with certainty in the milking parlour. The udder quarters generally appear normal and the milk unaffected on the strip cup. However, the milk will

produce a gel reaction and yield an acidic reading on the California Milk Test (CMT). (ARVL 1994α.)

The CMT comprises a reagent containing a purple pH indicator dye, which gives an indication of the number of somatic cells present in the milk of each udder quarter tested. It also indicates whether the milk is alkaline or acidic, i.e. its pH value. The reagent reacts directly with the deoxyribonucleic acid (DNA) present in the nuclei of the somatic cells, forming a gel; the more cells present in the milk sample, the stronger the gel formation. This reaction can be graded on a scale:

0 (negative), T (trace), 1, 2 or 3. (ARVL 1994α.)

While a CMT produces inconclusive results when interpreted independently, it is a fairly accurate and speedy 'cow side' test which is useful in three ways: to differentiate between normal udder quarters, to distinguish between clinical and

subclinical mastitis, and to identify affected quarters in the case of subclinical mastitis (ARVL 1994a).

2.6 ANALYSIS

If clinical or subclinical mastitis is suspected, samples of infected milk are submitted by the farmer or veterinarian to a laboratory, for bacteriological examination. The number of somatic cells per millilitre of milk is ascertained primarily using a coulter counter. This, essentially, is a particle counter capable of rapidly and accurately determining the number of particles in a particular suspension. It does not specifically count somatic cells in milk, but must be calibrated to count particles of a certain diameter or larger. (ARVL 1994a.)

All composite milk samples with a somatic cell count of higher than 485 000 per millilitre of milk are cultured on blood agar plates, which are then incubated for 48 hours in a normal atmosphere at 37°C. If bacteria are present, they are identified according to cultural characteristics, gram stain, and catalase or oxidase tests. Samples with somatic cell counts higher than 485 000 per millilitre of milk, as well as positive bacterial growth, are considered cases of subclinical mastitis. (ARVL 1994b.)

The somatic cell count in milk is a very sensitive indicator of udder health. Increased somatic cell count values signal udder disease, decreased milk production, changed composition and dairy technological usefulness of milk, reduced hygienic quality (therefore safety for human consumption) of milk, deficient management of udder health, and most importantly increased production costs and decreased profits for the farmer. (Giesecke *et al.* 1994: 111.)

For optimum efficiency in modern herd management of udder health, therefore, obtaining somatic cell counts of herd milk is the most practical way presently available to farmers to monitor and control mastitis (Giesecke *et al.* 1994: 111).

This viewpoint is confirmed by that of van den Heever *et al.* (1983), who state that the level of somatic cell count escalation in milk is directly correlated to the tissue damaging effects of the udder irritation and the degree of irritation elicited. The authors conclude that the somatic cell count in milk is a sensitive parameter for monitoring udder health.

2.7 MASTITIS CONTROL

2.7.1 Preventive control

Practices which prevent mastitis follow one of three methods: reducing the incidence of exposure to infectious micro-organisms, strengthening the immune system of the host, and reducing physical injury (Campbell and Marshall 1975: 343).

The milking machine plays a crucial role in the control of mastitis in a dairy herd. There is no doubt that a poorly designed, badly constructed machine used negligently can wreak havoc in an otherwise well run herd of dairy cows. (Redostits and Blood 1985: 102.)

This is shown in the following ways. The milking machine:

- carries infection among cows via the contaminated cups;

- aids in the transfer of infection from quarter to quarter in the same cow during milking;
- encourages the penetration of the teat canal, through particles of milk carrying bacteria derived from the colonised skin of the teat, the environment (for example dirty washing water) or another (affected) teat;
- causes damage to the skin, especially at the teat end, in the form of erosions which are prime sites for colonisation with major pathogens. (Radostits and Blood 1985: 102.)

The milking machine and pipelines need to be serviced and checked every six months, or immediately the condition of even one cow's teats deteriorates or increased incidence of clinical or subclinical mastitis is diagnosed (ARVL 1994c).

To reduce further the incidence of exposure of healthy cows to mastitic pathogens, cows must be milked in the order of healthy first, beginning with healthy calf heifers, then healthy older

cows and finally mastitic cows (Giesecke 1979).

Dipping of the teats of all cows immediately after each milking and dry period treatment is the backbone of the modern mastitis control programme. Previously the standard recommendation was to disinfect the teat before milking. However, after tests were conducted between 1980 and 1985, it was concluded that teat dipping at this time completely revolutionised mastitis control. (Radostits and Blood 1985: 105.)

This conclusion was based on the observation that bacterial colonisation of the teat skin occurred during the period between milkings, and that if the skin could be disinfected and kept sterile between milkings the rate of new infection would be reduced significantly. (Radostits and Blood 1985: 105.)

It is generally recognised that the combined functioning of integrated mechanisms of genetic resistance and immunity

determines how successfully a cow defends her udder against microbial challenges and other adverse environmental factors. Such mechanisms become more critical as dairy farming methods advance and intensify. (Giesecke *et al.* 1994: 76-77.)

One of the most significant and prevalent predispositions to mastitis is stress. South African dairy cattle, particularly Friesian and Holstein breeds, frequently are subjected to poor nutrition and heat stress. These animals require carefully monitored feeding, sufficient drinking water and shade. (Giesecke *et al.* 1994: 76-77.)

The most effective day-to-day preventative method against mastitis is the efficient management of the herd. Disinfectants, antibiotics and other remedies have been developed to control the growth of bacteria and other microorganisms; these compounds are totally ineffective, however, against the mechanical faults of the milking machine as well as other

management deficiencies. Against these there is only one remedy, i.e. the stringent and knowledgeable supervision of each cow in each herd. (Giesecke 1979.)

2.7.2 Therapeutic control

2.7.2.1 Clinical mastitis

Comparatively satisfactory results can be expected only if correct emergency treatment is administered within the first six to eight hours after the onset of the udder infection directly responsible for the development of clinical mastitis (Giesecke *et al.* 1994: 62). The longer the delay the greater the irreparable damage to the mammary epithelium, which in turn affects its secretional performance (Giesecke 1979).

Before treatment is instituted a milk sample is to be collected

from the affected quarter(s) for bacteriological examination at a laboratory (ARVL 1994c). The standard treatment, then, consists of infusing antibiotics into the udder. However, this is relatively ineffective in eliminating the microorganisms if the cow is lactating. (Foley *et al.* 1972: 406.)

The single most important factor for the successful treatment of clinical mastitis is the complete and almost continuous removal of secretion from the affected quarter(s). The more frequently such a removal is repeated, the better the chances of rapid and successful therapy. (Giesecke 1979.)

Parenteral therapy is generally unnecessary unless there is systemic involvement with oedema, causing the blockage of ducts within the udder. Much larger doses of drugs are required for intramuscular or intravenous injections (because they are distributed throughout the animal's body) than for

infused drugs (which remain within the mammary glands).
(Campbell and Marshall 1975: 343.)

Prompt consultation with a veterinarian and attention to the results of the antibiogram ensure that the correct antibiotic is selected, thus increasing the chances of completing the therapy successfully (Giesecke 1979).

For generalised clinical mastitis, further systemic treatments can include analgesics to combat pain, multivitamins to support liver function, cortisone for relief of swelling and inflammation, antihistamines to combat shock and inflammation, liquids administered orally to further combat shock, and heart stimulants and calcium replacements if necessary (Giesecke *et al* 1994: 297).

Allerton Regional Veterinary Laboratory recommends that a cow which has had clinical mastitis on more than three occasions

during a lactation, which does not respond to dry cow treatment, or which has individual quarters persistently infected with *staphylococcus aureus*, should be culled (ARVL 1994d).

2.7.2.2 Subclinical mastitis

Cows with subclinical mastitis should be treated at drying off and not during lactation. Possible exceptions are cows infected with *streptococcus agalactiae* or young cows which are normally good producers and which have calved recently. (ARVL 1994c.)

2.7.2.3 Dry period treatment

Opinions differ as to the form of treatment to be given when cows are dried off at the end of their lactation period. Allerton

Regional Veterinary Laboratory (1994c) recommends universal treatment of all quarters of all cows, using antibiotic dry cow treatments recommended by the veterinarian.

Radostits and Blood argue that the cost of treating uninfected quarters is high, and that there might be some disadvantages in removing all infections from the quarters. This argument has gained strength from recently acquired knowledge about the role of minor pathogens in encouraging greater resistance to other infections (Radostits and Blood 1985: 99).

The authors suggest blanket dry period treatment only when the quarter infection rate is more than 15 %, when the bulk milk cell count is greater than 500 000 per millilitre of milk, or when four or more clinical cases per 100 cows are diagnosed over a period of 30 days (Radostits and Blood 1985: 98).

2.7.2.4 Vaccination

The only vaccination prospect in bovine mastitis is that aimed at *staphylococcus aureus*. Commercial vaccines administered parenterally are available for *staphylococcal* mastitis, and they may reduce the severity of the disease sufficiently to warrant using them to treat a problem herd not experiencing success with its current treatment. However, much of the experimental work regarding these vaccines has been done on sheep and may not be relevant or applicable to cattle. (Radostits and Blood 1985: 108.)

It is obvious that not much progress has been made in the search for a viable vaccine. As far back as 1971, Schalm *et al.* (1971) cited the following limitations in their book:

- Large and repeated doses are required to produce high antibody titres, and these are maintained for only a few months.

- No continuous resistance can be expected unless booster doses are administered every three to six months.
- Vaccines are strain-specific, thus do not give adequate protection against heterologous strains.
- Circulating antibodies do not pass into the milk of the mammary glands, thus protection against initial infection does not result from vaccination.

Intramammary infusions of vaccines have also been tried commercially, but the results claimed are at most a reduction in the severity of subsequent mastitis attacks and in loss of milk yield (Radostits and Blood 1985: 108).

2.7.2.5 Shortcomings

The major shortcoming of conventional antibiotics is bacterial resistance. Most mastitogenic bacteria inherit and acquire

characteristics which make them resistant to a single antibiotic or several antimicrobial drugs. For example, certain strains of the most common mastitogenic microorganism, *staphylococcus aureus*, produce a substance called *penicillinase*, which destroys *penicillin* and renders the bacteria resistant to this antibiotic. (Giesecke et al. 1994: 295.)

Further limitations to this form of treatment include poor blood-udder penetration in certain antibiotics. Others, for example *streptomycin*, dissolve poorly in fats and are thus unable to move efficiently or in sufficient concentration through the tissues to reach the bacteria and destroy them. (Giesecke et al. 1994: 295.)

Certain antibiotics, furthermore, chelate with the magnesium and calcium in milk, and are thus rendered inactive. Others still have a very short half life; if not administered repeatedly as prescribed, the duration of therapeutic concentration in these

antibiotics required to kill the bacteria in the udder is too short and the treatment fails. (Giesecke *et al.* 1994: 295.)

In addition, it has become increasingly important with regard to public health to keep antibiotics out of the human food chain. Penalties for not keeping contaminated milk out of the bulk tank are stringent. (Radostits and Blood 1985: 108.) In South Africa it is an offence to sell milk polluted with residues of remedies, disinfectants, cleaning agents and other chemicals (Giesecke *et al.* 1994: 56-59).

Unfortunately, data suggest that in South Africa antibiotic drug residues in milk supplies are still a considerable problem requiring increased attention as well as appropriate counter measures from health authorities, milk processors, and in particular from dairy farmers (Giesecke *et al.* 1994: 56-59). There is also valid concern over the effects of antibiotic residues disturbing the agro-ecosystem and

potentially reducing the efficacy of antibiotics in human medicine (Stopes and Woodward 1988).

To ensure antibiotic drug residues do not contaminate milk supplies, the milk from a cow treated with a quick release udder infusion has to be withdrawn from the bulk supply for 72 hours. This means that a large quantity of saleable milk is lost, further increasing the financial losses to the farmer. (Radostits and Blood 1985: 108.)

The widespread use of antibiotics in the conventional treatment of bovine mastitis clearly has resultant problems, chiefly the development of bacterial resistance and contamination of milk destined for processing and, ultimately, human consumption. When these factors are taken into account, it is evident that an efficient veterinary approach, one which avoids the use of antibiotics, would be of major interest to farmers and researchers. (Stopes and Woodward 1988.)

2.8 THE HOMOEOPATHIC APPROACH

Homoeopathy is a system of medical treatment founded by the German physician Dr Samuel Hahnemann (1755-1843), based on the principle that 'like cures like'. In practice, this means that a medicine capable of producing certain effects when taken by a healthy human being is capable of curing any illness that displays similar effects. (Sankaran 1992: 1.)

Using this approach, Hahnemann developed an extensive array of substances to help him combat disease, in a powerful and humane manner yet with no side effects. He later discovered that the substances' curative powers grew more potent, yet had less chance of inflicting harm, when they were diluted.

He furthermore subjected his remedies to succussion (violent mixing) at each stage of dilution, discovering that the

process of succussion harnesses the vital energy of the substances, thus enabling them to be curative. (Day 1992.)

2.9 HOMOEOPATHY IN MASTITIS CONTROL

Veterinary homoeopathy has gained significant recognition over the past decade, since it has proven successful in the treatment of many diseases and ailments. Mastitis has received notable attention, its prevalence and frequent occurrences causing serious financial constraints on the dairy industry. (Day 1992.)

All cases of acute mastitis call for the employment of various remedies according to the difference in apparent symptoms. For example, when the udder becomes hot, swollen and

painful the homoeopathic remedies *aconite* and *belladonna* would be used frequently in alternation. In contrast, *bryonia alba* may be needed in those cases where induration without pain or heat is the main symptom. The toxic form of summer mastitis in the non-lactating animal may require remedies such as *pyrogenium*, *echinacea*, *phosphorus* or *hepar sulphuris*. (MacLeod 1979.)

For prophylactic treatment, a different system of therapeutics has been created through the use of nosodes (Day 1992). The Thorsons Encyclopaedic Dictionary of Homoeopathy defines a nosode as a homoeopathically prepared remedy from disease products with its own full, distinct drug picture (Gaier 1991).

Nosodes undergo the same dilution and potentisation process as do other homoeopathic remedies, so that none of the original substance remains in the solution. There is, therefore, no infective potential in these medicines, nor are they strictly

speaking vaccines (in which one would expect to find demonstrable 'antigenic' material. (Day 1992.)

Nosodes are useful in preventative programmes, and although prepared homoeopathically work in much the same way as do vaccines, both prior to and in the face of outbreaks of specific infectious disease. More attention is paid to the causative agent of the symptoms than to the symptoms themselves, enabling treatment to be instituted on a wide scale. This is advantageous since less time and energy are required by the veterinary practitioner. (Day 1992.)

Most of the research to date into the use of nosodes to treat bovine mastitis has been conducted by Dr Christopher Day (1986), a British veterinary homoeopath. His contemporary, the late Dr George MacLeod, also used nosodes extensively in the treatment of mastitis, and commented favourably on their effectiveness (MacLeod 1979).

The pathogens used by Day to make up his nosode were obtained from the disease material discharged from cows with infected udders. The organisms present included *streptococcus uberis*, *streptococcus dysgalactiae*, *streptococcus agalactiae*, *escherichia coli* and *staphylococcus aureus*. (Day 1986.)

Day's choice of administration was oral. Five millilitres of the nosode in the 30cH potency was added once a month to the dairy's main drinking water tank. (Day 1986.) Further to this method, Day suggests the addition of five millilitres of the nosode to the water trough twice weekly for three weeks and then once a month thereafter (Day 1994).

The research on this subject conducted by Day (1986) showed promising results, with underlying trends Day considered too powerful to ignore. Veterinary homoeopathy remains a largely unexplored field of medicine in South Africa.

For this reason, research into a problem such as bovine mastitis is a prudent choice, since successful results would indicate the benefits of this form of treatment and encourage further exploration and study into the problem specifically and its related field generally.

CHAPTER 3 - MATERIALS AND METHODS

3.1 OBJECTIVE

The objective of this research was to determine whether homoeopathic nosode therapy is an effective prophylaxis for bovine mastitis. It was hypothesised that treatment would cause a decrease in somatic cell counts, as well as reduce the incidence of clinical and subclinical mastitis.

3.2 STUDY DESIGN AND PROTOCOL

A placebo controlled double-blind study was chosen to eliminate bias from the trial. Two coded bottles of medication were supplied by an independent homoeopathic pharmacist. The two groups of cows were tagged so that each group corresponded to one of the coded bottles; in this way it was ensured that each group received the same treatment throughout the duration of the study.

The two groups remained together as a herd throughout the trial so that they were exposed to the same daily routine, diet, milking procedures and environmental stressors. This strict uniformity ensured that favourable results could be attributed to the medication and not to an unknown factor.

3.2.1 Sample selection

A suitable herd of Friesland dairy cows was located in the Karkloof area of KwaZulu-Natal, with the assistance of an animal health technologist at the Allerton Regional Veterinary Laboratory in Pietermaritzburg. The farm owner's written permission was obtained (Appendix 3) after the entire study procedure had been explained to him.

The initial sample size consisted of 69 animals, namely 34 in the treatment group and 35 in the placebo group. The cows were divided randomly into the two groups using a random number generator (de Klerk 1994).

Due to the subsequent sale of 12 of the original sample, only 57 cows completed the full trial. At the end of the trial the researcher was informed which the treatment and placebo groups had been. It was then determined

that a total of 28 animals comprised the treatment group and 29 the placebo group.

3.2.2 Exclusion and inclusion criteria

The study excluded the following cows:

- those with active clinical mastitis
- those with recognisable structural changes to the udder due to chronic mastitis
- those which were not lactating
- those which did not form part of the dairy herd selected by the researcher

3.3 INTERVENTION

The combined mastitis nosode was produced from infected milk samples supplied by the Allerton Regional Veterinary Laboratory. The pathogens isolated from these samples included *staphylococcus aureus*, *staphylococcus epidermidis*, *streptococcus uberis*, *streptococcus dysgalactiae*, *streptococcus agalactiae* and *corynebacterium* species. These samples, which also contained blood, pus and other cell debris, were mixed together in equal parts and then added to 70% alcohol in a ratio of one part combined sample to nine parts alcohol, to create the mother tincture.

The mother tincture was then subjected to a serial dilution process: one drop was added to 99 drops of 70% alcohol and the mixture succussed 100 times to make the 1cH potency. One drop of the 1cH potency was then added to 99 drops of 70%

alcohol and the mixture succussed 100 times to make the 2cH potency. This process was repeated until the 30cH potency was reached. (The suffix cH is used to indicate that it is the centissimal scale introduced by Hahnemann that is being used to potentise the remedies [Gaier 1991].)

The placebo underwent the same procedure, the mother tincture comprising a drop of 70% alcohol and the dilution and succession processes repeated to reach a dilution of the 30cH potency. Both of the final potencies were diluted in 30% alcohol, then dispensed to the researcher in identical 50ml bottles complete with encoded labels.

3.3.1 Dose and route of administration

For administration purposes a five millilitre dose of the contents of each bottle was transferred into correspondingly

encoded plastic water containers, each holding one litre of drinking water. Thus, a 1:200 dilution was created. After the water was stirred thoroughly, the containers were placed in the dairy parlour to be accessible for dosing purposes. The animals were dosed individually by mouth, using sterile 100ml syringes filled to the 50ml mark. Doses were administered immediately after the animals had been sampled and milked, ensuring a high degree of dosing accuracy.

3.3.2 Treatment regimen

Treatment of the animals was planned to coincide with milk sampling in order to minimise the disruptions to normal milking routine. Milk sampling was conducted by an animal health technologist on a monthly basis, following the routine prescribed in Appendix 1.

The first milk sample, taken at the time of the first treatment, was the baseline somatic cell count for each animal, against which future trends could be compared. Sampling and treatment took place over a five month period, which allowed five treatments and six somatic cell counts for each cow.

3.4 MEASUREMENTS

Somatic cell counts were obtained from the Allerton Regional Veterinary Laboratory using a coulter counter (as described in the chapter two, review of the related literature). This ensured a high degree of accuracy in measuring as well as the standardisation of results.

All samples with counts higher than 485 000 per millilitre of milk were considered cases of subclinical mastitis (ARVL 1994b) and were cultured for 48 hours on blood agar plates at 37°C to determine accurately which microorganisms were present. Samples displaying obvious signs of clinical mastitis, namely clumping and flaking in the milk with blood and pus, were diagnosed with clinical mastitis; in these cases the confirmation by somatic cell count was unnecessary.

3.5 STATISTICAL ANALYSIS

Once the study was completed the results were tabulated, so that each cow had six sequential somatic cell counts as well as a record of occurrences of clinical and subclinical mastitis.

These results were analysed by a statistician using the Statgraphics Version 6+ program. Two sample t-tests and the Mann-Whitney test were used to compare the differences between the two groups throughout the treatment, while Wilcoxon's two-sample tests were used to determine whether animals within the same group improved as the treatment progressed. The level of significance was $\alpha=0,05$.

CHAPTER 4 - RESULTS

4.1 CRITERIA GOVERNING ADMISSIBILITY OF DATA

- Only data obtained from milk samples taken in the approved manner (Appendix 1) were admissible.
- Only official results of the somatic cell counts and cultures received from Allerton Regional Veterinary Laboratory were used.
- Only samples not contaminated in any way were used.

4.2 STATISTICAL ANALYSIS

Statistical analysis was simplified in this study, as both the treatment and placebo groups numbered more than 25 animals. The following assumptions were satisfied due to the large sample size.

4.2.1 Assumptions

Let X = sample 1 and Y = sample 2.

(a) $X \sim N(\mu^1, \sigma_1^2)$

(b) $Y \sim N(\mu^2, \sigma_2^2)$

(c) $\sigma_1^2 = \sigma_2^2 = \sigma^2$ (unknown)

(d) the two samples are independent

4.2.2 Two-sample t-test statistics

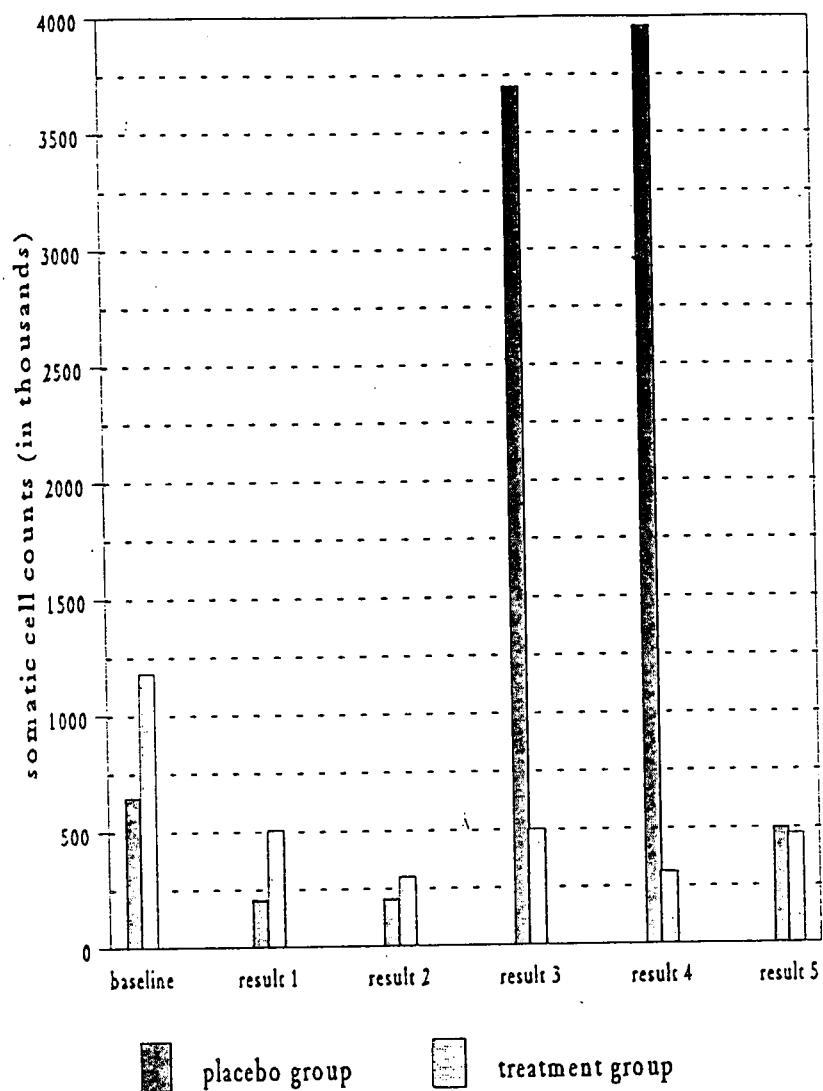
Two-sample t-tests were used to test for significant differences in the means of the somatic cell counts between the two groups over the treatment period. The null hypothesis (H_0) was accepted if there was no significant change in the two means, i.e. if $\mu^1 = \mu^2$. The alternative hypothesis (H_1) was accepted if there was a significant change in the two means, i.e. if $\mu^1 \neq \mu^2$. The level of significance used was $\alpha = 0,05$.

All six t-test results were insignificant, indicating that the treatment was no more effective than the placebo in reducing the somatic cell counts over the period of the trial. The detailed results are tabulated in Table 4-1 and in Graph 4-1.

TABLE 4-1

Results of two-sample t-test analysis from baseline to result 5

| | | MEAN | MEDIAN | STANDARD DEVIATION | T- STATISTIC | SIGNIFICANCE LEVEL | RESULT |
|----------|---|---------|--------|-----------------------|-----------------|-----------------------|---------------|
| baseline | P | 691621 | 201000 | 1572110 | -0,863157 | 0,3918 | insignificant |
| | E | 1181680 | 219500 | 2605930 | | | |
| result 1 | P | 208034 | 81000 | 469852 | -1,01405 | 0,315001 | insignificant |
| | E | 537536 | 83000 | 1683740 | | | |
| result 2 | P | 200241 | 153000 | 146770 | -0,545641 | 0,587519 | insignificant |
| | E | 237143 | 147000 | 332244 | | | |
| result 3 | P | 3698860 | 215000 | 1852180 | 0,918635 | 0,362298 | insignificant |
| | E | 478464 | 222000 | 929835 | | | |
| result 4 | P | 3942520 | 265000 | 18502500 | 1,03528 | 0,305067 | insignificant |
| | E | 320286 | 172000 | 457476 | | | |
| result 5 | P | 541345 | 352000 | 733913 | 0,178752 | 0,858789 | insignificant |
| | E | 499929 | 289500 | 999628 | | | |



GRAPH 4-1
Comparison of somatic cell counts
from baseline to result 5

4.2.3 Mann-Whitney test statistics

A comparison of the means of the somatic cell counts of the two groups was made using the Mann-Whitney test, where the null hypothesis would be rejected if half the two-tailed Z-value were less than the α value. None of the six tests rendered a significant result at the $\alpha=0,05$ level, thus the same conclusion as the two-sample t-test results was reached. The detailed results are tabulated in Table 4-2.

TABLE 4-2

Results of Mann-Whitney tests from baseline to result 5

| | Z-STATISTIC | $z/2$ | CONCLUSION |
|----------|-------------|-----------|---------------|
| baseline | 0,725412 | 0,362706 | insignificant |
| result 1 | 0,854329 | 0,4271645 | insignificant |
| result 2 | 0,87945 | 0,439725 | insignificant |
| result 3 | 0,904695 | 0,4523475 | insignificant |
| result 4 | 0,267254 | 0,133627 | insignificant |
| result 5 | 0,179957 | 0,0899785 | insignificant |

4.2.4 Wilcoxon's two-sample test statistics

Wilcoxon's two-sample test was implemented to ascertain whether the means of the somatic cell counts of the cows in the same sample decreased significantly as they progressed from baseline to treatment 1, 1 to 2, 1 to 3, 1 to 4, 1 to 5, 2 to 3, 2 to 4, 2 to 5, 3 to 4, 3 to 5, and 4 to 5.

Improvement was noted if the absolute value of Z was greater than 1,96 (the tabulated value of Z at the $\alpha=0,05$ level of significance). Significant improvement was noted randomly on seven occasions in each of the two groups; the findings were therefore insignificant. These results are tabulated in Tables 4-3 and 4-4.

TABLE 4-3

Results of Wilcoxon's two-sample tests for the placebo group

| | LARGE SAMPLE TEST STATISTIC | TWO-TAILED PROBABILITY OF EQUALLING OR EXCEEDING Z | SIGNIFICANT IMPROVEMENT |
|----------------------|--------------------------------|---|----------------------------|
| baseline vs result 1 | 4,0853 | 0,0000440437 | yes |
| baseline vs result 2 | 1,48556 | 0,137394 | no |
| baseline vs result 3 | 0 | 1 | no |
| baseline vs result 4 | 0 | 1 | no |
| baseline vs result 5 | 1,85695 | 0,0633175 | no |
| result 1 vs result 2 | 2,22834 | 0,0258574 | yes |
| result 1 vs result 3 | 4,0853 | 0,0000440437 | yes |
| result 1 vs result 4 | 2,97113 | 0,00296724 | yes |
| result 1 vs result 5 | 3,34252 | 0,000830335 | yes |
| result 2 vs result 3 | 1,70084 | 0,0889726 | no |
| result 2 vs result 4 | 1,11417 | 0,265204 | no |
| result 2 vs result 5 | 2,97113 | 0,00296724 | yes |
| result 3 vs result 4 | 0,188982 | 0,850102 | no |
| result 3 vs result 5 | 2,22834 | 0,0258574 | yes |
| result 4 vs result 5 | 1,11417 | 0,265204 | no |

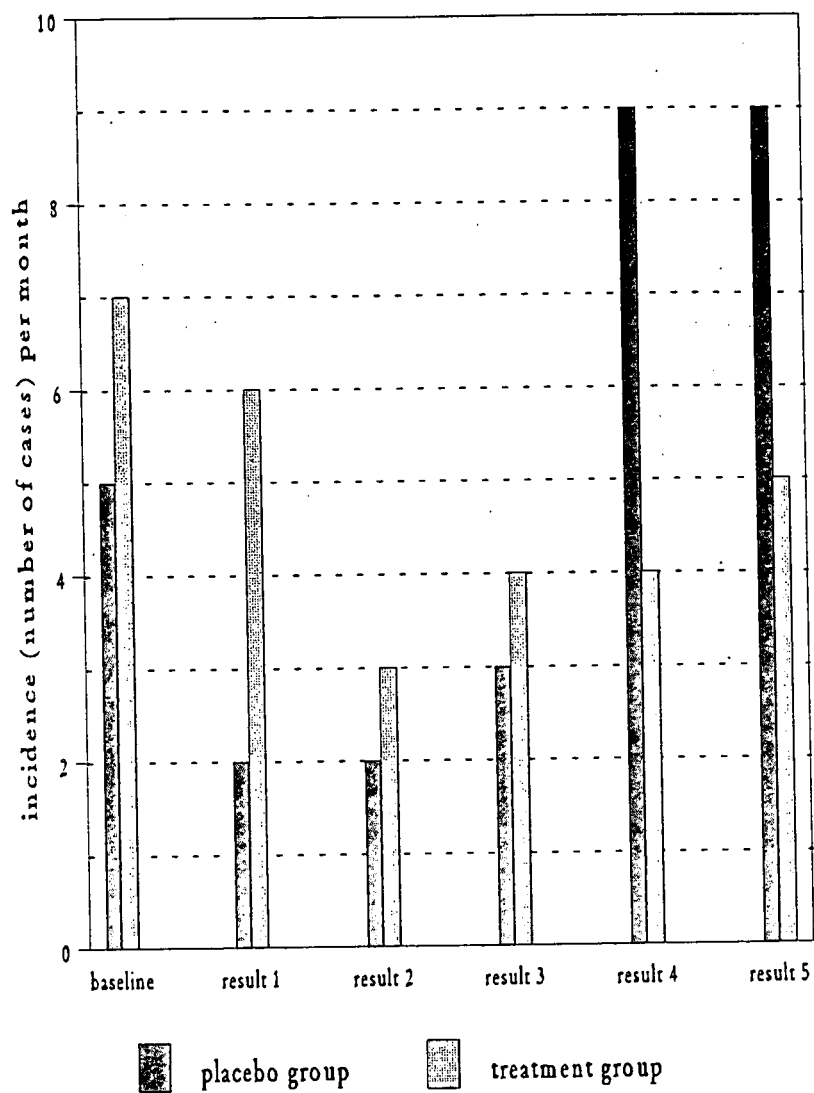
TABLE 4-4

Results of Wilcoxon's two-sample tests for the treatment group

| | LARGE SAMPLE TEST STATISTIC | TWO-TAILED PROBABILITY OF EQUALLING OR EXCEEDING Z | SIGNIFICANT IMPROVEMENT |
|----------------------|--------------------------------|---|----------------------------|
| baseline vs result 1 | 3,2127 | 0,00131507 | yes |
| baseline vs result 2 | 2,83473 | 0,00458651 | yes |
| baseline vs result 3 | 1,32288 | 0,185876 | no |
| baseline vs result 4 | 1,70084 | 0,0889726 | no |
| baseline vs result 5 | 0 | 1 | no |
| result 1 vs result 2 | 1,70084 | 0,0889726 | no |
| result 1 vs result 3 | 3,2127 | 0,00131507 | yes |
| result 1 vs result 4 | 2,0788 | 0,0376351 | yes |
| result 1 vs result 5 | 2,45677 | 0,0140192 | yes |
| result 2 vs result 3 | 2,45677 | 0,0140192 | yes |
| result 2 vs result 4 | 0,566947 | 0,570747 | no |
| result 2 vs result 5 | 2,45677 | 0,0140192 | yes |
| result 3 vs result 4 | 1,70084 | 0,0889726 | no |
| result 3 vs result 5 | 0,188982 | 0,850102 | no |
| result 4 vs result 5 | 1,32288 | 0,185876 | no |

4.4 SUBCLINICAL MASTITIS INCIDENCE

The treatment group started off with a higher incidence of subclinical mastitis (seven cases versus five cases), which persisted until result 3. In results 4 and 5 the incidence was higher in the placebo group. This is illustrated in Graph 4-2. These results were found to be insignificant when analysed using the two-sample t-tests and Wilcoxon's two-sample test statistics ($\alpha=0,05$).



GRAPH 4-2
Comparison of diagnoses of subclinical mastitis
from baseline to result 5

4.5 CLINICAL MASTITIS INCIDENCE

Only one case of clinical mastitis was recorded, in results 4 and 5 of the placebo group. No statistical inferences can be made from this isolated result.

CHAPTER 5 - DISCUSSION

This study attempted an investigation into the effectiveness of homoeopathic nosode therapy as a prophylaxis for bovine mastitis.

5.1 ANALYSIS OF THE TRIAL

Two parameters were used to test the hypothesis, namely the individual somatic cell counts of the cows taken on a monthly basis, and the monthly incidence of clinical and subclinical mastitis. Such tests yielded accurate results concerning the status of mastitis within the herd, and allowed for objective

comparisons to be made between the treatment and placebo groups throughout the duration of the trial.

The two-sample t-tests compared the means of the somatic cell counts as the trial progressed. This comparison proved indicative of the response of each group on a collective basis over the five month period. Graph 4-1 illustrates that the treatment group started off with a higher mean somatic cell count which persisted until result 2, while results 3 and 4 indicate an increase in the mean of the placebo group. It is important to note that Day (1986) recorded that the cell counts in the bulk milk he tested remained lower in the treatment group throughout the trial.

Day furthermore suggested in a letter to the researcher (1994) that the somatic cell counts would be reduced over a period of time after, possibly, an initial increase. After this, the clinical incidence, severity and duration of cases of mastitis should

decrease. In this trial, however, the researcher found that the somatic cell counts decreased at first and then rose slightly towards the conclusion of the trial.

Further analysis of the two-sample t-tests led the researcher to the conclusion that results 3 and 4 were in reality insignificant, since the change can be attributed to only one animal in the placebo group, which had a somatic cell count of 99 999 000 recorded over two consecutive months. This analysis is verified in result 5, which shows the means of the two groups as almost identical once the isolated high cell count had normalised.

The results of the Wilcoxon's two-sample tests (tabulated in Tables 4-3 and 4-4) also proved to be inconclusive. Both groups showed significant improvement in seven of the 15 tests, thus improvement cannot be attributed to the intervention. Furthermore, the improvements took place at similar times over the test period, which further complicates any attempt at differentiation.

All cases of subclinical mastitis are compared in Graph 4-3. From this graph it can be seen that the treatment group included more cases of subclinical mastitis from baseline to result 3. Results 4 and 5 subsequently show an increase in incidence in the placebo group while the incidence in the treatment group remained stable.

The difference in incidence proved insignificant. However, the results indicate a favourable trend in the treatment group. Further research might have substantiated this claim; unfortunately, though, the trial ended at this stage.

5.2 SUGGESTIONS FOR FUTURE TRIALS

5.2.1 Alternative method of analysis

The results of the trial were inconclusive with regard to statistical analysis. This might be due partially to the narrow parameters that were used to determine whether improvement occurred over the trial period. The choice of somatic cell counts as a means of detecting mastitis is a good one as it is both accurate and objective.

However, it is not the only possible indicator of whether or not the treatment is working. In order to obtain a more holistic viewpoint the researcher suggests that future trials use other indicators as well.

In Day's (1986) trial cell counts on bulk milk were recorded as a measure of the mastitic status of the herd. By combining milk drawn from all cows in the herd, this practice minimises the impact of wide fluctuations from the norm; however, the progress of the mastitic status of individual animals is far more difficult to monitor using this method of analysis.

A possible alternative would be to substitute the mean for the median, since the median is not influenced by isolated extreme variations to the norm (Reich 1993). Using this method, therefore, one could continue to monitor the individual somatic cell counts while simultaneously minimising extreme variations.

5.2.2 Sample selection

There is a certain amount of controversy regarding sample selection. Day (1986) did not split the herd according to yield, age or calving date, because he believed that this approach could affect the results materially. This method and system was adopted by the researcher.

Searcy *et al.* (1995), on the other hand, contend that the two groups should be made as homogenous as possible with regard to age, monthly milk production and status of mastitis. This approach is advantageous in short term trials in that significant differences between the two groups would be identified quickly. In long term studies, however, initial differences become negligible as the study progresses.

5.2.3 Treatment regimen

No set protocol has been laid down as to the frequency of dosage and route of administration required. Day (1994) suggested in a letter to the researcher that five millilitres of the remedy should be added to the water trough twice weekly for three weeks, then once a month.

Searcy *et al.* (1995) administered individual doses orally using an aspersion pressurised tank, starting with a dose every 48 hours for the first two weeks. In the third week only two doses were given, with a single dose being administered in the last week. This trial of just four weeks' duration is incomparable to Day's trial, which lasted nine months, and frequency of dosage must therefore be seen in the light of the trials' lengths.

5.2.4 Extending the data base

Stopes and Woodward (1988) conducted a survey of farms using the mastitis nosode as part of their mastitis control programme. A significant outcome of their survey was that the attitude and approach of the farmer was very important in determining the perceived success of the treatment. In this regard, a questionnaire to discover the attitudes of host farmers included in the trial would be valuable, and the results taken into consideration.

The monthly records should be expanded to include antibiotic usage, quarter(s) of udders affected, and various fluctuations in milk yield. In addition, duration and severity of infections should be noted in all cases of clinical mastitis, as this could provide evidence to support the use of the nosode.

An interesting finding was noted concerning the subclinical mastitis incidence and the fluctuations in rainfall during the trial. When illustrated on a graph (Appendix 4) it is evident that the incidence of subclinical mastitis in the placebo group increased in relation to the increased rainfall, while the treatment group remained relatively stable.

Wet conditions are known to encourage the development of mastitis (Searcy and Guajardo 1994), thus these results suggest that the treatment group was more resistant to this particular environmental stressor. Future trials could possibly include the testing of rainfall on a more advanced level, as well as other environmental stressors, including temperature, with regard to their effect on the results of the trial.

CHAPTER SIX - CONCLUSION AND RECOMMENDATIONS

6.1 CONCLUSION

It is evident that this trial was ineffective in proving the hypothesis, namely the effectiveness of homoeopathic nosode therapy as a prophylaxis for bovine mastitis. The reasons include insufficient data to make a detailed comparison, ambiguity of the analysed results, and too short a trial period.

There was no significant difference between the two groups based on the mean somatic cell counts and the incidence of clinical and subclinical mastitis.

Significant improvement within each group was noted on certain occasions during the trial, but was difficult to ascribe to the intervention as the results occurred randomly and equally in both groups.

6.2 RECOMMENDATIONS

Future trials should focus on collecting a wider variety of data which can be used to support the hypothesis. Age, state of lactation, severity of infection and treatment of individual animals should be taken into account. In addition, external influences such as the weather or stress should be monitored.

A questionnaire to be completed by the dairy farmer would identify his perceptions of the treatment, having a possible

effect on the outcome of the trial.

Easy and frequent access to the herd would be essential for regular dosing and monitoring. Furthermore, continuation of the trial for a full 12 months would make statistical analysis simpler and more accurate, and this minimum period is therefore recommended.

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APPENDICES

APPENDIX 1

The composite milk sampling procedure and methods
undertaken by Allerton Regional Veterinary Laboratory

- (1) The milk hygiene laboratory to be telephoned (0331 - 471931) to book a test date.
- (2) Milk samples to reach the laboratory the day before the test, or before 08:00 on the day of the test.
- (3) Aim: to take foremilk samples (free from contaminating bacteria normally found on the teat, skin of technician's hands or in dust and manure).
- (4) Method:
 - (a) bottles numbered sequentially (eg. 1 to 100)
 - (b) cow's number or name written against corresponding number on result sheet
 - (c) no gaps left in the sequence of tubes
 - (d) with clean hands, each teat washed with clean, running water and dried with paper towel

- (e) teat end disinfected with cotton wool soaked in methylated spirits, using one piece of cotton wool per teat
- (f) first few squirts of milk discarded into a strip cup, in order to flush teat canal
- (g) milk squirted into sterile sample tube provided, holding the tube at an angle to prevent dirt entering it
- (h) lip of tube or part of rubber stopper which comes into contact with milk not to be touched

Addendum

A composite sample is formed from an equal amount of milk from each quarter in one sample tube, while a quarter sample is formed from milk taken from one quarter of any given sample tube.

APPENDIX 2

Statistics taken by Allerton Regional Laboratory mastitis scheme for
Kwazulu-Natal (1992 - 1994)

| <i>analysis conducted on:</i> | <i>1993/1994</i> | <i>1992/1993</i> |
|-------------------------------|------------------|------------------|
| herds on the scheme | 195 | 200 |
| cows on the scheme | 24 699 | 24 952 |
| new members | 30 | 32 |
| herds off the scheme | 35 | 23 |
| somatic cell counts | 60 917 | 56 803 |
| bacterial cultures | 27 803 | 20 697 |
| pathogens isolated | 14 861 | 23 971 |
| contaminated samples | 3 549 | 3 551 |
| antibiograms | 218 | 260 |

Of the pathogens isolated, the following were identified most often :

staphylococcus aureus (30.6 %)

streptococcus uberis (14.7 %)

corynebacterium species (29.1 %)

streptococcus dysgalactiae (3.9 %)

staphylococcus epidermidis (16.3 %)

streptococcus agalactiae (1.3 %)

APPENDIX 3

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CONSENT FORM FOR PARTICIPATION IN A HOMOEOPATHIC RESEARCH DISSERTATION

I,, of the farm agree to allow eighty (80) cows of my dairy herd to be used in a research programme to test the prophylactic treatment of bovine mastitis, using homoeopathic nosode therapy.

The following terms and conditions have been agreed to:

1. The nature of the research and the methodology involved have been explained fully to me, and I understand the role that my herd is to play.
2. I understand that the eighty cows will be divided into a treatment and a placebo group, which implies that only half the cows will be receiving medication.
3. I understand that I am under no obligation to continue with the research until its completion, and that I may withdraw at any time if I am dissatisfied in any way.

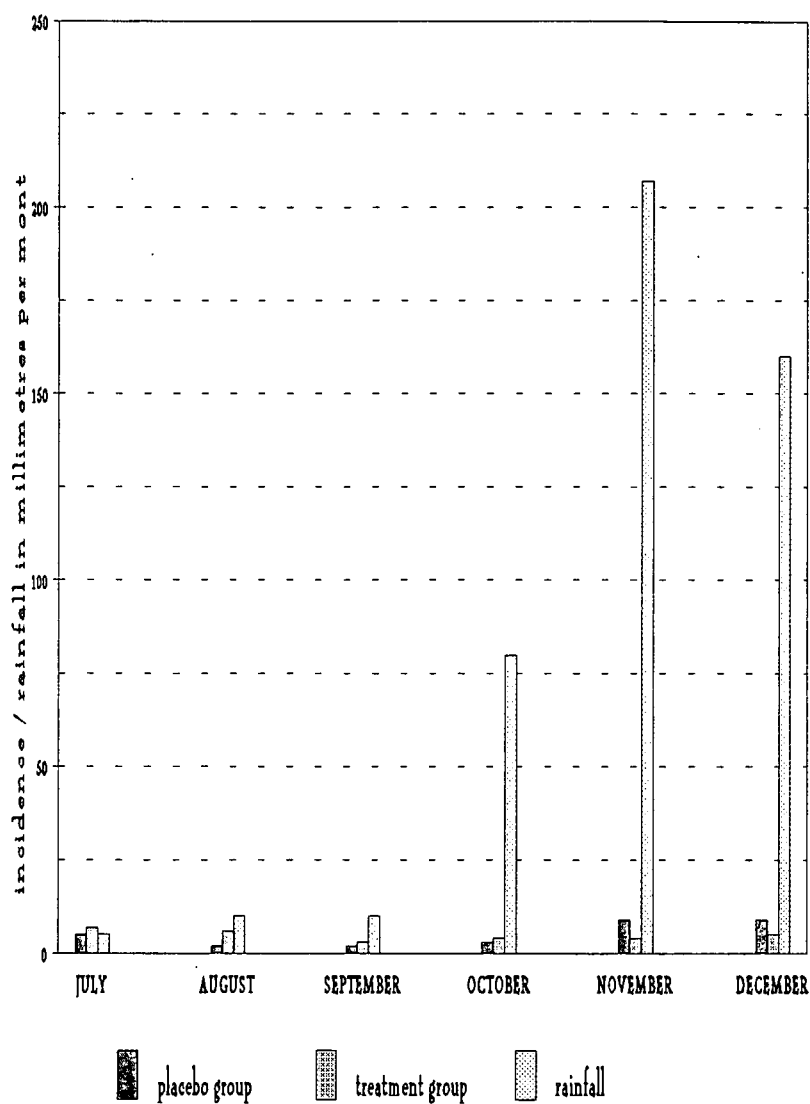
Signed at on

Farmer

Allerton Animal Health Technologist

Researcher

APPENDIX 4



GRAPH 4-3

Comparison of subclinical mastitis to rainfall figures over the trial period

reference:

Allerton Regional Veterinary Laboratory 1995