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Mini - Dissertation submitted in partial compliance with the requirements for the Master's Degree in Technology: Homoeopathy in the Department of Homoeopathy at Technikon Natal.

I, Lindy Jane Clark, do hereby declare that this dissertation represents my own work in both conception and execution.

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Dr. C Hall B.Sc. (PU for CHE), M.Tech (T.Natal)
The Prophylactic Treatment of Bovine Mastitis using a Homoeopathic Complex

(Phytolacca decandra 200CH, Bryonia alba 30CH and Silicea terra 30CH).

Lindy Jane Clark

2001
This research is dedicated to my parents,
Nan and Graham Clark, for all their
encouragement and support, and to
Edward Spencer, for his encouragement during
the completion of my studies.
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Mr. K Le Roux                   Allerton Regional Veterinary Department
Mr K. N. Thomas                 Statistician
Natura Homoeopathic Laboratories Supplier of experimental medicine
ABSTRACT

The purpose of this research was to evaluate the efficacy of a homoeopathic complex of *Phytolacca decandra* 200CH, *Bryonia alba* 30CH and *Silicea terra* 30CH in the treatment of bovine mastitis. The efficacy of the complex was evaluated in terms of its effect on reducing the Somatic Cell Count (SCC) as well as its effect on reducing the total number of clinical cases occurring within the herd on treatment.

The aim of trying to reduce the SCC is the fact that it is related to the quality of the milk produced, the higher the SCC the lower the quality and yield of the milk produced and thus the lower the monetary return on the sale of such milk. It is also related to the volume of milk produced as the higher the SCC the lower the quantity of milk produced. The number of cases of clinical mastitis is also an important economical consideration, as there are a number of costs associated with each case including short and long-term losses in production as well as the cost of treatment and management of infected animals.
The treatment and prevention of mastitis is a challenging problem due to the complex nature of its aetiology. The numerous factors known to contribute to the high incidence of mastitis make it difficult to isolate only one factor as a variable for research purposes. Two separate, but identical trials were thus conducted for this research in order to try and verify any findings.

Each trial involved the selection of two herds, owned and managed by the same farmer in order to reduce the variables. Even so, there are still an enormous number of factors including environmental and stress factors that contribute to the high incidence of mastitis, some of which are virtually impossible to control or isolate.

They were also to belong to the Allerton Mastitis Control Scheme, which would involve bi-annual somatic cell count (SCC) tests. Treatment began straight after the first SCC test and continued for the six-month period until the second bi-annual SCC test. Records were to be kept during this period of any cases of clinical mastitis that occurred in either of the herds. Dairy staff are trained to check each animal for
any signs and symptoms of clinical mastitis when it is brought in for milking.

Treatment involved the mixing of 5ml of the Homoeopathic complex of *Phytolacca decandra* 200CH, *Bryonia alba* 30CH and *Silicea terra* 30CH with the animal’s food, once a week. This was then fed to the animal, when it was brought in for milking.

Once the results were obtained from the SCC tests, they were analyzed to determine if there was any statistically significant difference between the two herds before or after treatment, or between the before and after results for each herd. No significant difference was found in Trial 1 and in Trial 2 the only significant difference found was between herd A and herd B after treatment. Contrary to the aim of the research, the results from herd A were lower than those of herd B, but due to the complex nature of mastitis, this can be attributed to a number of other factors, the results are inconclusive.
A test for proportions between the number of clinical cases of mastitis that occurred in each herd was also conducted. A statistically significant difference was found in trial 1, showing an improvement in the proportion of clinical cases that occurred in the trial herd compared to the control herd. This was however not found in trial 2 and as there are so many possible variables that could have contributed to these results they become inconclusive.
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<table>
<thead>
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<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antibiotic</td>
<td>A chemical substance produced by a microorganism, which has the capacity to inhibit the growth of or to kill other microorganisms; antibiotics sufficiently non-toxic to the host are used in the treatment of infectious diseases. (Saunders 1989)</td>
</tr>
<tr>
<td>A.R.V.L.</td>
<td>Allerton Regional Veterinary Laboratory, the State Veterinary office.</td>
</tr>
<tr>
<td>Clinical</td>
<td>Pertaining to or to be found on actual observation and treatment of patients, as distinguished from theoretical or basic sciences. (Saunders 1989)</td>
</tr>
<tr>
<td>CMT</td>
<td>Californian Milk Cell Test, a cow side test that can be used for the detection of mastitis. (Mastitis Expert Committee 1/94)</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
</tr>
<tr>
<td>--------------</td>
<td>---------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Homoeopathy</td>
<td>a complete system of medicine which aims to promote general health, by reinforcing the body's own natural healing capacity (Hammond 1995)</td>
</tr>
<tr>
<td>Induration</td>
<td>process of hardening; an abnormally hard spot or place. (Saunders 1989)</td>
</tr>
<tr>
<td>Inflammation</td>
<td>a protective tissue response to injury or destruction of tissues, which serves to destroy, dilute of wall off both the injurious agent and the injured tissues. (Saunders 1989)</td>
</tr>
<tr>
<td>Lactation</td>
<td>the secretion of milk / the period of milk secretion. (Saunders 1989)</td>
</tr>
<tr>
<td>Mastitis</td>
<td>inflammation of the mammary gland (University of Kentucky)</td>
</tr>
</tbody>
</table>
Morbidity: the condition of being diseased or morbid. (Saunders 1989)

Side effect: a consequence other than that for which an agent is used, especially an adverse effect on another organ system. (Saunders 1989)

Sign: an indication of the existence of something; any objective evidence of a disease, i.e. such evidence as is perceptible to the examining physician. (Saunders 1989)

Somatic Cells: white blood cells and udder tissue cells. (Mastitis Expert Committee 1/94)

Subclinical: without clinical manifestations. (Saunders 1989)
Symptom any subjective evidence of disease or of a patient's condition (Saunders 1989)

Veterinarian a person trained and authorized to practice veterinary medicine and surgery

Veterinary pertaining to domestic animals and their diseases (Saunders 1989)
CHAPTER ONE

INTRODUCTION

Mastitis is a condition in which there is inflammation of the mammary gland or in the case of cattle, it is known as the udder. The epithelial cells of the udder, which are responsible for synthesising the protein, fat and lactose constituents of the milk can become damaged and are ultimately destroyed by the inflammatory process. This problem can become further exacerbated by the presence of opportunistic secondary infections, the most serious of which is a Staphylococcus Aureus infection.

Mastitis is the costliest disease of the dairy industry today. (Bray 2001).

The most recent South African approximation of the cost of mastitis by the Onderstepoort Veterinary Institute in 1996 was around R 529 million per year. In the United States just the milk losses due to mastitis exceed one billion dollars yearly (University of Nebraska –
Lincoln 2001). Other costs related to the incidence of mastitis include lost production due to decreased volumes that are produced by mastitic animals, veterinary and medical bills associated with its treatment as well as the cost of culling and subsequent replacement of animals lost through mastitis.

Even in well-managed farms, at least a third of the cows experience clinical mastitis each year (Morin et al. 2001). Mastitis results from a complex interrelationship between the environment, the cow, and a host of bacterial or other pathogenic organisms. The rate of infection is directly related to the bacterial invasion rate divided by the resistance of the cow to infection, thus any factors which increase the levels of bacteria within the cows surroundings, or which decrease the cows resistance to infection will contribute to the high incidence of mastitis in dairy herds. Good management techniques are thus essential in order to reduce the incidence of mastitis.

Currently the most favoured form of treatment involves the use of antibiotics. Antibiotics however are not only costly, but they are difficult to administer effectively. Antibiotics have proved effective in treating cases of clinical mastitis but they have not reduced the incidence of mastitis since their introduction (Duval 2000).
means that we are still no closer to solving the problem of mastitis, and to complicate matters further we are now finding strains of bacteria that are resistant to our current range of antibiotic treatments.

There is thus a great need for research especially into finding feasible alternatives to prevent and control mastitis (British Homoeopathic Journal 1995 volume 84).

Homoeopathy is a system of natural therapy, which offers a number of benefits to the treatment of mastitis. These include the fact that there are no side effects to the treatment, there are no drug residues left in the tissues or in the milk, it is cost effective and of course it has proved effective in a number of trials (Day 1995). If one-thousandth of the money and effort spent on research and promotion in conventional drug therapy were applied to Homoeopathic research and development, the results would be quite staggering (Day 1995).

With all this in mind, this research was designed with the purpose of evaluating the efficacy of the homoeopathic complex, firstly, in reducing the Somatic Cell Count and thus improving the quality of
the milk produced. The higher the quality of milk produced the higher the price per litre.

The second purpose of this research is to evaluate the efficacy of the homoeopathic complex in reducing the incidence of clinical mastitis while on treatment, as the milk produced by a cow with clinical mastitis is deemed unfit for sale.
CHAPTER TWO

LITERATURE REVIEW

2.1 INTRODUCTION

The word 'mastitis' is derived from the Greek words *mastos* (meaning breast) and *itis* (meaning inflammation). Thus the term 'bovine mastitis' would refer to the condition found in cattle, in which there is an inflammation of the breast, or in this case it is referred to as the udder.

Bovine mastitis is considered one of the most significant problems facing the dairy industry both from a health and a financial point of view. This disease has a number of serious economic implications, especially as it is generally the most under estimated single disease affecting dairy cattle (Giesecke et al. 1994).
2.2 CLASSIFICATION OF MASTITIS

Mastitis is divided initially into clinical and sub-clinical groups. This depends on whether or not there are any obvious signs or symptoms of mastitis visible when the cow is brought in for milking.

2.2.1 Sub-clinical Mastitis

In sub-clinical cases of mastitis there are no clinical signs or symptoms of mastitis that can be detected in the dairy, but the condition can be determined through an increase in the Somatic Cell Count (SCC) as well as a drop in the amount of milk that is being produced.

Sub-clinical mastitis can also be detected using the California Milk Cell Test (CMT). This test can be carried out in the dairy, and is used to detect any contaminated milk, which will produce a gel reaction and yield an acidic reading when combined with the reagent.
2.2.2 Clinical Mastitis

Clinical mastitis can be further divided into four categories according to the severity and duration of the condition.

- Peracute cases
- Acute cases
- Subacute cases
- Chronic cases

2.2.2.1 Peracute cases

A peracute case of mastitis is the most severe case that a cow can present with. It is characterised by: severely affected milk secretions which may cease altogether, with only a little exudate present at milking; the affected udder is distinctly swollen, hardened, painful and warm; the animal appears very ill, is possibly shivering and restless. Its eyes may be sunken due to dehydration and it shows irregular breathing, increased body temperature and no appetite; onset is rapid and severe and death can quickly ensue (Giesecke et al. 1994).
2.2.2.2 Acute cases

An acute case of mastitis is characterised by: the initial and further jets of milk showing purulent floccules in the strip cup; the affected udder quarter shows a reddened skin and is swollen, hardened and sensitive to the touch, hence the cow may be restless or bad tempered; the cow shows general symptoms of disease, including raised body temperature.

2.2.2.3 Subacute cases

A subacute case of mastitis is characterised by: the initial jets of milk showing purulent floccules; the milk glands appear normal on inspection, but on palpation of the empty udder one may pick up hardening of the gland tissue; the cow shows no general symptoms of disease, and behaviour is generally normal; this process is gradual and the development of the disease may take several days (Giesecke et al. 1994).
2.2.2.4 Chronic cases

The presentation of a chronic case of mastitis depends on the duration and severity of the particular case. There may or may not be alterations in the milk, even becoming serum-like. The present case of mastitis may or may not be due to a previous clinical case, but is usually an ongoing process that may last only a few weeks or may even affect subsequent lactations.

It causes progressive damage to the udder tissue, with acute flare-ups occurring from time to time. On the whole however the cow does appear healthy.
2.3 AETIOLOGY OF MASTITIS

There are a number of factors, which contribute to the high prevalence of mastitis among dairy cattle, which makes its prevention and control very difficult. There are however three main areas which if kept under tight control will ensure a drastic reduction in the prevalence of mastitis. These areas include, reducing the incidence of exposure of the cattle to infectious microorganisms, strengthening the immune system of the host cow, and reducing the incidence of physical injury especially to the udder of the cow (Farrow, 1997).

Thus, should the cattle suffer from any injuries or conditions, which may compromise their immune systems in any way, this will increase the likelihood that opportunistic microorganisms will be able to invade the animals and cause an increased incidence of mastitis. Therefore it is essential that the farmer implements effective management procedures in all areas of the dairy farming process, in order to ensure that the likelihood of this happening is reduced as far as possible.
2.4 COMMON CAUSATIVE MICRO-ORGANISMS IN MASTITIS

There are a number of micro-organisms that can be held responsible for the high incidence of mastitis, however in Kwazulu-Natal the most prevalent micro-organisms are:

- **Staphylococcus Aureus**
- **Streptococcus Agalactiae**
- **Streptococcus Dysgalactiae**
- **Streptococcus Uberis**
- **Corynebacterium Pyogenes** (also known as Actinomyces Pyogenes).

2.4.1 **Staphylococcus Aureus**

Mastitis caused by this particular micro-organism appears most often a few days after parturition, and can be highly fatal.

It is characterised by: the slow induration and ultimately atrophy of the udder tissue; a watery to purulent secretion with thick clots or flakes; the udder is swollen, warm and very painful to touch (the cow will avoid any contact between the legs and the udder); the
cow has an elevated temperature, a reduced appetite, and may appear depressed; there may also be changes in the colour of the udder, from pink to red to purple as the disease progresses. This may eventually result in the udder turning gangrenous in severe cases (Rebhun, 1995).

In chronic conditions there may be fibrosis of the glandular tissue, and permanent damage to the udder. This micro-organism is the most prevalent and results in the most damage caused by mastitis, but it is also the most difficult to eradicate (ARVL, 1997).

2.4.2 Streptococcus spp.

The morbidity rate in this form of mastitis is lower than with staphylococcal infections, reaching a maximum of around 28%, but the loss of production is still great. It is characterised by: a primary fever, swelling of the affected quarters and changes in the milk, which contains clots and flakes when viewed in the strip cup. The cows appetite is decreased, and its udder appears firm, swollen and oedematous (Rebhun, 1995).
2.4.3 *Corynebacterium Pyogenes*

Commonly referred to as 'summer mastitis', it is a condition that occurs more commonly in warmer conditions and it is primarily a disease of the teat canals.

This condition is characterised by: swelling and induration of the affected quarters, which produce an evil smelling, cheese-like secretion, which is difficult to expel. The cow has a raised temperature, loss of appetite, and an udder, which is hard, inflamed and painful.

There is often the development of an abscess(s) which can result in the permanent destruction of the glandular tissue, which results in the permanent loss of production from that quarter (Rebhun, 1995).
# TABLE 1 - THE MAIN CAUSATIVE ORGANISMS OF SECONDARY INFECTIONS IN MASITITIS CASES (Duval 2000)

<table>
<thead>
<tr>
<th>Species</th>
<th>Main Source</th>
<th>Living Conditions</th>
<th>Propagation Factors</th>
<th>Symptoms</th>
<th>Preventive Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Streptococcus agalactiae</em></td>
<td>Infected cows</td>
<td>Infected quarter and udder only</td>
<td>Using same rag for cleaning udders</td>
<td>Mild fever for about 24 hours</td>
<td>Wash udders after milking, reduces problem by 50%</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>Infected cows</td>
<td>On abnormal udder and teat, milkers, vagina, tonsils</td>
<td>Transmitted by hands or rags, enters during milking</td>
<td>Often quite acute for a few days after calving. May be fatal. Quarter swells and turns purple. Quickly affects entire system. In chronic state, udder hardens, aqueous secretion, eventual atrophy of the quarter. Intermediate form with granular secretion. Milk hotter than normal.</td>
<td>Cull infected cows</td>
</tr>
<tr>
<td><em>Streptococcus dysgalactiae</em></td>
<td>Infected cows</td>
<td>Infected quarter, injuries</td>
<td></td>
<td>Pronounced swelling of one or more quarters. Milk highly abnormal. High fever in serious cases. Pronounced swelling of one or more quarters. Milk highly abnormal. High fever in serious cases.</td>
<td></td>
</tr>
<tr>
<td><strong>Streptococcus uberis</strong></td>
<td>Contaminated environment</td>
<td>On cow's skin, mouth, ground</td>
<td>Neglected udder washing, insufficient drying, lack of bedding, muddy yards</td>
<td>Pronounced swelling of one or more quarters. Milk highly abnormal. High fever in serious cases. Affects mostly dry cows and heifers.</td>
<td>Wash udders only, dry well with disposable paper towels for each cow.</td>
</tr>
<tr>
<td>-------------------------</td>
<td>--------------------------</td>
<td>-------------------------------</td>
<td>-------------------------------------------------</td>
<td>---------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Escherichia coli</strong></td>
<td>Contaminated environment</td>
<td>Ground, bedding (sawdust and shavings), manure, water</td>
<td>Dirty calving stall, lack of bedding, inadequate udder washing</td>
<td>Often very serious. May lead to loss of quarter or even death. Thin yellow secretions, with granular texture resembling bran. Often high fever.</td>
<td></td>
</tr>
<tr>
<td><strong>Corynebacterium pyogenes</strong></td>
<td>Certain insects</td>
<td>Humid valleys, wooded areas</td>
<td>Pronounced systematic reaction due to toxins caused by bacteria. Often more than one quarter affected. They become hard, produce thick smelly secretion like cheese and difficult to eliminate. Followed by abscess that bursts, releasing creamy pus, and tissue loss.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


2.5 DETECTION OF MASTITIS

Cases of clinical mastitis are relatively easy to detect as the signs and symptoms are easily observable by the dairy staff when the cow is brought in for milking. These signs and symptoms can include a change in temperature, restlessness, swelling and inflammation of the udder as well as changes in the appearance and consistency of the milk. In order to make an accurate diagnosis of the cause of the mastitis as well as the recommended course of treatment requires a veterinarian to take samples for culture and sensitivity tests.

Subclinical mastitis is far more difficult to detect, as there are no easily observable signs and symptoms of the disease process. Diagnosis is made according to the outcome of specific tests. The two most commonly used tests to detect Subclinical mastitis are the Somatic Cell Count and the California Milk Cell Test.
2.5.1 Somatic Cell Count

The Somatic Cell Count test involves the laboratory testing of milk samples to determine the quantity of the so-called somatic cells. These cells are made up of the worn or damaged epithelial cells and the white blood cells that are excreted with the milk.

High herd Somatic Cell Counts have a negative correlation with milk production per cow and the quality of the milk (Mastitis Expert Committee 2/92). This means that the higher the Somatic Cell Count the less milk is produced and the poorer the quality of the milk. This is even more significant when one considers that the lost volumes of milk are lost earnings and that premiums are paid when low Somatic Cell Counts are maintained (Mastitis Expert Committee 2/92).

The results of the laboratory tests are graded according to a scale, from 0 (negative), through T (trace) 1-3 (ARVL, 1997 – Appendix C). Any sample of milk tested that has a SCC above 485 000 per milliliter of milk with no observable signs and symptoms, is considered a case of sub-clinical mastitis.
### TABLE 2 - HUMAN FACTORS IN MASTITIS (Duval 2000)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Associated factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low somatic cell count</td>
<td>Geographical position of the farm, treatment of dry cows, replacement cows produced on the farm, positive attitude towards milking, family enterprise.</td>
</tr>
<tr>
<td>High somatic cell count</td>
<td>Small herd, irregular maintenance of milking equipment, lack of bedding on cement floor, udder washing of dirty cows only, little ambition.</td>
</tr>
<tr>
<td>Low bacterial count</td>
<td>Treatment of dry cows.</td>
</tr>
<tr>
<td>High bacterial count</td>
<td>Tied housing, obsolete milking equipment, short withdrawal period after antibiotic treatment, slight tendency to seek out information.</td>
</tr>
<tr>
<td>High milk yield</td>
<td>Average herd, treatment of dry cows, average tendency to seek out information, elimination of cows that are too susceptible.</td>
</tr>
<tr>
<td>Low milk yield</td>
<td>Lack of hot water at milking, use of one cloth only for all cows, infrequent meetings with other farmers, strong will to continue traditional family farming, no vacation.</td>
</tr>
</tbody>
</table>

### TABLE 3 - MILK LOSS DUE TO MASTITIS (Mastitis expert committee 2/92)

<table>
<thead>
<tr>
<th>HSCC (Cells loss per ml herd milk)</th>
<th>Milk Daily Milk Production</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>600 L</td>
</tr>
<tr>
<td>250 000</td>
<td>3 %</td>
</tr>
<tr>
<td>500 000</td>
<td>6 %</td>
</tr>
<tr>
<td>1 000 000</td>
<td>10 %</td>
</tr>
</tbody>
</table>

### TABLE 4 - RELATIONSHIP BETWEEN THE CMT AND THE SCC RESULTS (Mastitis expert committee 1/94)

<table>
<thead>
<tr>
<th>CMT RESULTS</th>
<th>GEL FORMATION</th>
<th>SOMATIC CELL COUNT PER MILLILITRE MILK</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>None</td>
<td>100 000-200 000</td>
</tr>
<tr>
<td>T</td>
<td>Slight</td>
<td>150 000-500 000</td>
</tr>
<tr>
<td>1</td>
<td>Slight to moderate</td>
<td>400 000-1 500 000</td>
</tr>
<tr>
<td>2</td>
<td>Moderate</td>
<td>1 000 000-5 000 000</td>
</tr>
<tr>
<td>3</td>
<td>Heavy</td>
<td>Generally over 5 000 000</td>
</tr>
</tbody>
</table>
2.5.2 California Milk Cell Test

Sub-clinical mastitis can also be detected using the California Milk Cell Test (CMT). This test can be carried out in the dairy, and is used to detect any contaminated milk, which will produce a gel reaction and yield an acidic reading when combined with the reagent. The CMT measures not only the pH of the milk to detect any changes, but it also contains a reagent which reacts directly with the deoxyribose nucleic acid (DNA) present in the nuclei of the somatic cells to form a gel. It follows that the more cells that are present in the milk, the firmer the gel formed will be.

2.6 THE ALLERTON MASTITIS CONTROL SCHEME

The Allerton Mastitis Control Scheme was started in 1978 by Dr. Bryson at the Allerton Regional Veterinary Laboratory in Pietermaritzburg with the aim of trying to help Natal dairy farmers to improve the subclinical mastitis status of their dairy herds.
The aim of the scheme is to reduce the incidence of subclinical mastitis in the herd by:

- Identifying cows and quarters with existing subclinical mastitis infections in order to remove them from the herd, either by treatment or culling.

- Preventing new mastitis infections by proper maintenance and function of milking machines, as well as good milking procedure, dairy hygiene and herd management.

2.7 MANAGEMENT FACTORS IN THE PREVENTION OF MASTITIS.

Klastrup and his co-workers estimate that 25% of the susceptibility to infection is attributable to environmental factors, 20% to genetic factors and 50% to herd management. (Duval 2000)

The aim of management strategies is ultimately to reduce the rate of infection, which is directly proportional to the bacterial invasion
rate divided by the resistance level of the cow to infection (Giesecke et al, 1994). Thus, one must aim to keep the bacterial rate as low as possible, and the cow's level of resistance as high as possible. Good management is thus of cardinal importance (Mastitis Expert Committee 2/94). This can be achieved by taking precautions in two main areas:

2.7.1 Environmental Factors

It is essential to maintain high levels of hygiene, both among the workers and in the dairy itself, as well as to ensure that all equipment is regularly serviced and cleaned (ARVL recommends that the milking machines should be serviced at least once every six months).

During milking it is important to regularly hose down the dairy to prevent the build up of waste and to ensure that each cow's udder is thoroughly cleaned and dried using disposable tissue-paper before and after milking.
Chlorination of water for the dairy should take place at least 100m from the dairy to ensure that the chlorine has time to take effect. As far as possible it would help to eradicate flies from the dairy and to prevent them from feeding off any open wounds on the cattle.

Precautions should also be taken to ensure that the cattle are protected from extremes of temperature. Just as they foster our colds, rapid changes in temperature can encourage mastitis (Duval 2000). Heat causes tension, which is detrimental to udder health (Mastitis Expert Committee 2/94).

Stress is also an important factor in the development of mastitis. The more an animal is stressed in its environment the less efficient its immune system is, and the less it can resist microbial infections. Therefore the more stress there is the greater the chance of mastitis (Duval 2000).

Constant stress suppresses the animal's immune mechanism and resistance to disease is lowered (Mastitis Expert Committee 2/94).
2.7.2 Factors affecting cow-to cow spread.

It is important to try to keep cows affected with mastitis as far away from the healthy cattle as possible, and to milk them last ensuring that the milking machines are thoroughly cleaned between milkings.

It is also advisable to try to prevent any injuries occurring to the cattle, and their udders especially, and any injuries that do occur should be thoroughly cleaned and disinfected to prevent infections, which could be spread from one cow to another.

Any workers with open wounds on their hands should be advised to wear gloves as they can pass an infection on to the cow when handling the udder.

Cows should also not be permitted to lie down straight after milking until the udder has had a chance to dry off (Giesecke et al. 1994).
FIGURE 1 – FACTORS INFLUENCING THE INCIDENCE OF
MASTITIS (Duval 2000)

- Climate
- Milking machines
- Nonpathogenic bacteria
- Housing
- Bedding
- Pathogenic bacteria
- Feeding Stress
- Hierarchy
- Calving
- Hygiene
- Genetic
- Placenta retention
- Somatic cell count

24
2.8 DIET AND MASTITIS

Diet has an important effect on mastitis (Giesecke et al. 1994). Research into this area has discovered that selenium and vitamin E are important supplements to be included in the diet (Olson 1995), as they have shown to be useful in reducing the incidence of mastitis. It has also been noted that excessive nitrogen or protein in the feed, is often mentioned as one of the factors causing mastitis (Duval 2000). Naturally, a good diet with good nutrition will help to improve the cow's resistance to infection.

2.9 THE ANTIBIOTIC TREATMENT OF MASTITIS.

At present the main form of treatment for mastitis requires the use of antibiotics. Depending on the results of bacterial cultures, the following drugs are indicated in the treatment of:
2.9.1 Staph. infections

Commonly used antibiotics used in the treatment of these types of infection include:
Penicillin, Pirilimycin, Cloxacillin, Cephalosporins and Benzathine cloxacillin

2.9.2 Strep. spp.

Commonly used antibiotics used in the treatment of these types of infections include:
Penicillin, Cloxacillin, Erythromycin, Cephalosporin; *C. pyrogenes*
Penicillin (systemically).

There are however a number of problems and difficulties associated with the use of antibiotics. When treating with antibiotics it is very difficult to obtain effective levels of antibiotics in the milk when administering these drugs either systemically or intramammarily. Higher concentrations are needed systemically in order to ensure that the levels in the milk are high enough to be effective.
This level of dosage together with the frequency of dosage required to successfully eliminate the bacteria makes this process very costly, not only in the expense of the drugs but in the milk loss due to high levels of residues found in the milk (Rebhun, 1995).

Another problem with antibiotic treatment is that very often it may not reach the target area. This is normally due to swelling in the area or blockage of the ductules, both of which are usually present during a case of clinical mastitis. This can cause a reduction in the circulation to the affected area and thus a reduction in the amount of drug reaching its target.

The absorption of drugs into the milk from the blood is dependent on three factors:

- The lipid solubility of the drug - the higher the lipid solubility the easier the passage across the cell membranes
- The degree of ionisation - the lower the ionisation the greater the absorption
- The degree of protein binding - the less protein bound the drug the greater the degree of transfer to the milk.
With all these factors taken into consideration it is very difficult to be sure how much of the drug has actually made it into the milk. In order to combat this the dosage given may be more than apparently necessary. In this case residues of the drug may be forced into the tissues and should this animal then need culling the meat would be considered unfit for consumption. This would result in further financial loss to the farmer.

Drug absorption is also measured according to the pH of the milk, which is normally around 6.5 (Rebhun 1995) in comparison to the blood plasma, which has a pH of around 7.4 (Rebhun 1995). This creates a concentration gradient, which will favour the absorption of any drug that is of a basic nature (e.g. Erythromycin). The problem is accentuated during a bout of mastitis, as the pH of the milk is elevated, thereby decreasing the concentration gradient and thus also decreasing the amount of drug that is absorbed into the target area.

One must also consider that with such a concentration gradient any acidic drugs (e.g. Penicillin) are thus poorly absorbed and will
require high systemic administration (in the case of Penicillin a level of up to 37 times greater than that required in the milk has to be administered) (Rebhun 1995).

One also has to be careful when considering the site of administration as this affects the rate of absorption, as it is dependent on the blood circulation in the area as well as the condition of the cow.

The antibiotics, which finally reach the milk, are then often lost through the stripping out of mastitic quarters before they are able to take effect.

After the administration of antibiotics to treat the mastitis, one still has to wait for the drug to be eliminated from the milk before it can be placed into the bulk tank for sale, this can take up to 96 hours (Rebhun 1995) for some drugs.

With modern dairy farming an increasing problem with antibiotic treatment is that the micro-organisms involved are developing strains, which are resistant to all the normal antibiotic treatments. Antibiotic sensitivity tests can be carried out by the laboratory to
determine which remedies are most likely to be successful in the
treatment of mastitis (ARVL 1997a).

2.10 ECONOMIC FACTORS RELATED TO MASTITIS.

During 1987 it was estimated that the direct and indirect cost of
bovine mastitis in South Africa totaled almost R190 million per year
(Giesecke et al. 1994).

The most recent approximation of the cost of mastitis by the
Onderstepoort Veterinary Institute (1996) is around R529 million
per year. Making it the biggest single health problem confronting
the dairy industry.

Some of the costs involved include:

- The cost of treatments, this includes the antibiotics and
disinfectants. Veterinary practice economics are not at all easy if
mainly homoeopathy is used - a large proportion of
conventional veterinary income is derived from the sale of drugs
(Day 1995).
• It also includes the cost of the milk discarded as it is judged unfit for human consumption (due to antibiotic residues), as well as the cost of the veterinary consultation and extra labour required for the detection and treatment of the mastitis.

• The cost of lost milk yield, both temporarily and on a permanent basis is significant. The SCC is often a good indication of the percentage of milk lost through mastitis (a SCC of 250 000 - 500 000 is equivalent to a loss of 10% of milk production; a SCC of 750 000 - 1 000 000 is equivalent to a loss of 15% of milk production (ARVL 1997 a).) Over time this can amount to an enormous loss of income.

• Potentially one of the biggest costs comes in the form of lost lactations. Well-bred heifers should be highly productive for at least 5-6 lactations, of which the last two are often the most productive. In a cow with mastitis, the average number of lactations is only two. This would account for a staggering loss of income. (Giesecke et al 1994.)
The cost of the loss of dairy cows due to disposal of cows unproductive as a result of mastitis or cows with chronic recurrent mastitis or the disposal of cows due to acute cases, also amounts to an enormous loss of income.

The expense of feeding dairy cattle even though they may be unproductive due to mastitis may be considered a ridiculous waste.

It is also expensive, as well as an effort to breed and rear or buy new dairy cows to replace those lost through mastitis.

The price of milk is often determined by its quality and therefore the poor quality milk produced by cows with mastitis would account for a further loss of income.
2.11 HOMOEOPATHIC TREATMENT.

Homoeopathy is a very safe, gentle and effective means of therapy (Day 1995). Schuette (1992) states, "The main principle in cattle welfare is: as much homoeopathy as possible and as less allopathy as necessary".

Homoeopathic treatment has no side effects, which makes it a very humane, gentle and non-toxic form of therapy.

It is also impossible to overdose on the high potency remedies, as the content of toxic substance within the medicine is negligible.

Homoeopathic treatment does not rely on laboratory experimentation of drugs to prove their effectiveness. It is even safe enough that most of the provings of homoeopathic remedies have been done on humans, before clinical trials are carried out to prove their effectiveness in the treatment of animals.

Because of the minute doses given of the remedies there are no residues of the medicine left in the tissues or the milk and so any
meat or milk from the cow under treatment can be commercially sold.

The cost of the remedies is relatively inexpensive especially in comparison to the antibiotic alternative and, any cost that is incurred is usually compensated for by the value of the meat and milk salvaged from the animal that would normally be lost due to the presence of drug residues.

Often farmers will not treat sick or injured animals for fear of spoiling the meat with drug residues and so the animal is left to suffer. As the homoeopathic remedies do not leave any residues the animal can be treated to at least make it more comfortable or even to cure it without risking the salability of the meat / milk.

Homoeopathy is not dependent on diagnosis of the condition to the extent that allopatic medicine is and thus treatment can begin before the diagnosis is confirmed. This prompt treatment of the condition can often reduce the severity thereof.

Homoeopathy has proved an effective treatment in numerous fields, including that of veterinary (Schuette 1992). Wide-ranging
possibilities are presented for the cure or prevention of mastitis, so the relegation of Homoeopathy to the status of a minor system of medicine is unwise (Day 1995).

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). Dr. Christopher Day and his contemporary, the late Dr. George MacLeod, from the United Kingdom have used nosodes extensively in the treatment of mastitis and although the majority of the research has proved inconclusive they have commented favourably on their effectiveness (MacLeod 1979). Reliance on the nosode alone for treatment will almost certainly not produce a cure but can help a clinical situation (Day 1995). Following on from this research, Gregory Alan Farrow conducted further research into homoeopathic nosode therapy for the prophylactic treatment of bovine mastitis in South Africa, but his results also failed to conclusively prove the effectiveness of homoeopathic nosode therapy for the prophylactic treatment of bovine mastitis. He too however stressed the importance of further research into the use of homoeopathy in the treatment of bovine mastitis.
Searcy et al. (1995) conducted research into the preventative treatment of bovine mastitis using a homoeopathic complex of *Phytolacca decandra* 200CH (50%), *Phosphorus* 200CH (30%) and *Conium maculatum* 200CH. The results showed that the treatment group had a significantly lower incidence of clinical mastitis than the control group, but caution was advised in placing too much significance in the results, suggesting that experimentation should continue, with single and complementary homoeopathic medicines.

Dr. Christopher Day states that he considers the choice of potency to be much less important than the choice of remedy, but never the less the choice of potency remains a contentious issue. Searcy et al. (1995) used *Phytolacca decandra* in a 200CH and Jouanny (1984) recommends using *Bryonia alba* in a 30CH if prescribed clinically as in this research. “As a general guide, use higher potencies if you are very sure of your remedy; if not, tend to select lower potencies for these have a wider spread of action” (Day 1995). For this reason I chose *Phytolacca decandra* in a 200CH and due to *Bryonia alba* being more of an acute remedy in a 30CH. *Silicea terra* was also chosen in a 30CH as it is less specific for the treatment of bovine mastitis.
2.11.1 THE HOMOEOPATHIC REMEDIES USED IN THE 
MASITIS COMPLEX.

2.11.1.1 PHYTOLACCA DECANDRA.

This is a useful remedy for both acute and chronic cases of mastersis (Macleod 1991). In acute cases there is curdled milk retaining clots, the udder becomes hot, swollen, painful and inflamed. In chronic cases clots may appear in mid-lactation and the secretion generally becomes stringy, thickened and yellow. The cow may become restless and weak and is worse in cold, wet weather. Macleod (1991) states that it is "probably the most useful remedy for the average chronic case". Mettler (1990) in his discussion of the possibility of treatment of cow mastitis with homoeopathic remedies often mentions this remedy in conjunction with other remedies for the effective treatment of mastitis.

2.11.1.2 BRYONIA ALBA.

This remedy is especially important in acute cases, where the udder is swollen, hard, indurated and painful. The animal will often
lie down because the pain is better for pressure and worse for movement. Symptoms are worse in warm surroundings (Day 1995). The milk is diminished, watery and contains flakes of fibrin. The cow has a decreased appetite and an increased thirst generally. It is clinically indicated for acute mastitis with heavy painful breasts where pain is set off by the slightest movement (Jouanny 1984). *Phytolacca decandra* follows *Bryonia alba* when suppuration is inevitable and both are indicated in breast abscess and where conditions are worse for motion (Vermeulen 1994). *Bryonia alba* is also considered an acute of *Phytolacca decandra* (Ross 1997).

### 2.11.1.3 SILICEATERRA.

"A constitutional remedy of importance in cattle " (Day 1995). This remedy is specifically effective in *C. pyrogenes*, where purulent foci and sinuses have developed as a result of multiple abscesses. It has the ability to stimulate the bodies' responses, which makes it useful in chronic mastitis treatment. It is also a complementary remedy of *Phytolacca decandra* (Vermeulen 1994).
CHAPTER THREE

MATERIALS AND METHODS

3.1 SELECTION OF HERDS

The first step in this research project is to select suitable herds to participate in the study. Herds eligible to participate in this study must meet the following criteria:

1. They must belong to the Allerton Mastitis Scheme

2. They must have Allerton records dating back to a minimum of six months prior to the beginning of the trial period.

3. The farmers concerned must own at least two herds in the same area so that one can act as a control for the study on the other herd.
4. The study herd will consist of a minimum number of 75 cows at the start of the research. This is important for the statistical analysis as it insures that at least 30 of the animals will participate in the study over the entire trial period.

5. The study herd and control herd are to be under the same management and feeding schemes. This will help to reduce the number of variables that could account for any changes that may occur during the trial.

3.2 FARMER CONSENT

Once herds have been selected the farmers managing these herds must be approached regarding the research. Once the methodology and aims of the research have been explained to them as well as what their participation will involve, they will be given the choice of whether or not they would like to participate in the study. They will then be asked to sign a consent form giving permission for their herds to participate in the trial.
3.3 INITIAL TESTING OF SELECTED HERDS

Samples of milk are to be obtained from every animal in each of the selected herds, in accordance with the guidelines set out by the Allerton Regional Veterinary Laboratory (Appendix A) and sent to Allerton for testing of the Somatic Cell Count levels and subsequent culture of all samples with a Somatic Cell Count higher than 485 000 per millilitre.

3.4 TREATMENT OF SELECTED HERDS

Each cow in the study herd is then to begin treatment. Administration of the homoeopathic complex is to occur as follows:

1. Every cow in the herd is to receive 5ml (Medicine measure to be supplied) [5ml was considered an easy and readily acceptable unit of measure] of the complex once a week in the feed at milking. The farmer can decide which day of the week is most suitable, but this day must be maintained throughout the trial period (six months).
2. The complex, which will be supplied in a granule form, is to be mixed into the top cup of feed given to each cow. This will minimise the possibility of any of the medicine being lost and will ensure that even if the cow's appetite is poor that they will consume the medicine.

3. A specific member of staff is to be chosen by the researcher and the farmer and is to be trained in how to administer the remedy correctly. The researcher or farmer is to be on hand at all times when the remedy is being administered.

3.5 RECORDS TO BE MADE OF ALL CLINICAL CASES OF MASTITIS

During the trial a record is to be kept of all the clinical cases of mastitis that occur within the study herd as well as the control herd(s). Dairy staff are trained to perform a strip test on each quarter of every cow before milking. They have to examine the milk samples for any changes such as flocculation, clotting or changes in the colour of the milk, which would indicate the presence of mastitis. If
any changes in the milk are noted then that case of mastitis must be recorded, including the cow number, the date and the quarter(s) affected.

3.6 POST TREATMENT TESTING OF SELECTED HERDS

At the end of the six month trial period, milk samples will once again be taken from each cow and sent to Allerton for analysis of the Somatic Cell Count and subsequent culture of samples with a Somatic Cell Count higher than 485 000 per millilitre. All samples are to be taken and handled according to the Allerton guidelines as set out in Appendix A.

3.7 DATA ANALYSIS

Data analysis will be carried out using SPSS package. Parametric tests (since n ≥ 30) will be used to test whether there would be an improvement in the treatment group in comparison to the control group. In addition to the above a test for proportions will also be done to determine whether there is any significant difference in the
proportion of clinical cases of mastitis between the trial and control herds.

All the above tests will be carried out at $\alpha = 0.05$ level of significance and decisions made using appropriate P – values.

The tests listed below will be carried out for Trial 1 as well as for Trial 2:

3.7.1 Paired t-test to compare the Somatic Cell Count of each cow in Herd A (Control herd) before and after treatment.

3.7.2 Paired t-test to compare the Somatic Cell Count of each cow in Herd B before and after treatment.

3.7.3 Unpaired t-test to compare the Somatic Cell Count of Herd A and Herd B before treatment.

3.7.4 Unpaired t-test to compare the Somatic Cell Count of Herd A and Herd B after treatment.

3.7.5 Test for proportions to compare the incidence of clinical mastitis in Herd A to that of Herd B.
3.8 TRIPS TO THE FARM

The trips to the farm shall consist of visiting each farm individually, first of all to establish the viability of each farm for participation in the study as well as to ensure that all staff involved are aware of what the project is about and are competent to participate in the study. Regular trips are then to be made to the farms over the study period to ensure that everything is running smoothly, that there is sufficient medicine available and that records are being kept of any mastitis cases that occur during the treatment period.

3.9 ETHICS

This research involves the prophylactic treatment of bovine mastitis in dairy cattle and thus the ethical considerations involved in this research are aimed primarily at ensuring the well being of the animals involved.

Although the incidence of clinical mastitis is on the decline the incidence of clinical cases is still estimated at 20-40 cases/100
cows/year depending on the season and lactation (J. Eric Hullerton, 2000). It would thus be a benefit to any animal if the incidence of mastitis could be reduced. This research will also not compromise the control herds, as we would not be affecting their health in a negative way.

The well being of the treatment groups are also not being compromised in any way as the research does not involve any invasive or uncomfortable procedures and the homoeopathic remedies do not have any side effects. The remedies are administered in a form that is pleasant tasting and as it is administered with the feed there is no discomfort involved in the dosing.

The confidentiality of the farmer and the herds involved will be protected as far as possible. The herds to be used in the research will be allocated a number (e.g. Trial 1, Herd A (control)) and only these allocated numbers will appear in the final research data.
CHAPTER FOUR

RESULTS

4.1 INTRODUCTION

This chapter covers the results obtained from the statistical analysis of the data collected during the trial period. Two separate trials were run in order to reduce the influence of other uncontrollable variables on the outcome of the research. The two trials to be conducted will be referred to as Trial 1 and Trial 2 respectively. The control herd in each trial will be referred to as Herd A, and the test herd as Herd B.
4.2 CRITERIA GOVERNING ADMISSIBILITY OF DATA

4.2.1 EXCLUSION CRITERIA

1. Any animal whose results do not appear in the Allerton Somatic Cell Count test taken prior to the commencement of treatment.

2. Any animal whose results do not appear in the Allerton Somatic Cell Count test taken after the trial period.

3. Any animal removed from treatment during the trial period.

4.2.2 INCLUSION CRITERIA

1. Only Somatic Cell Counts obtained through Allerton Regional Veterinary Laboratory will be admissible for this research.

2. Results of the Somatic Cell Count tests are only admissible if collected according to guidelines set out by the Allerton Regional Veterinary Laboratory (Appendix A).


5. Only data obtained during the trial period is admissible for use in this research.
4.3 STATISTICAL ANALYSIS – TRIAL 1

4.3.1 PAIRED T-TEST HERD A (CONTROL)

The Paired T-Test for Herd A was an intra group comparison between the before treatment and the after treatment Somatic Cell Counts to determine if there was any significant difference between the two.

Two hypothesis were set up:

Ho: (The null hypothesis) There is no difference between the somatic cell counts for Herd A. \( \mu_a = 0 \)

H1: (The alternative hypothesis) There is a difference between the somatic cell counts for Herd A. \( \mu_a \neq 0 \)
The level of significance is set at $\alpha = 0.05$, therefore the null hypothesis is rejected if $P < \alpha$.

The outcome in this case was that $P > 0.05$, therefore statistically there was no difference between the Somatic Cell Count results for Herd A before and after treatments.
TABLE 4.1 - INTRAGROUP COMPARISON BETWEEN THE BEFORE AND AFTER TREATMENT SCC RESULTS WITH REGARDS TO HERD A (CONTROL) IN TRIAL 1.

T-Test

Paired Samples Statistics

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>N</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
</tr>
</thead>
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<td>86</td>
<td>4426.6394</td>
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<td></td>
<td>HERDAFT1</td>
<td>3379.9186</td>
<td>86</td>
<td>15135.6956</td>
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</table>

Paired Samples Correlations

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<td>.157</td>
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Paired Samples Test

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<tr>
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<th>Std. Error Mean</th>
<th>95% Confidence Interval of the Difference</th>
<th>t</th>
<th>df</th>
<th>Sig. (2-tailed)</th>
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<td>15087.7918</td>
<td>1626.9584</td>
<td>-2566.7556</td>
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<td>411</td>
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4.3.2 PAIRED T-TEST HERD B (TREATMENT)

The Paired T- Test for Herd B was an intra group comparison between the before treatment and the after treatment Somatic Cell Counts to determine if there was any significant difference between the two.

Two hypothesis were set up:

Ho: (The null hypothesis) There is no difference between the somatic cell counts for Herd B. ($\mu_a = 0$)

H$_1$: (The alternative hypothesis) There is a difference between the somatic cell counts for Herd B. ($\mu_a \neq 0$)

The level of significance is set at $\alpha = 0.05$, therefore the null hypothesis is rejected if $P < \alpha$.

The outcome in this case was that $P > 0.05$, therefore statistically there was no difference between the Somatic Cell Count results for Herd B before and after treatment.
TABLE 4.2 - INTRAGROUP COMPARISON BETWEEN THE BEFORE AND AFTER TREATMENT SCC RESULTS WITH REGARDS TO HERD B (TREATMENT) IN TRIAL 1.

T-Test

Paired Samples Statistics

<table>
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<td>HERDAFT1</td>
<td>1055.9683</td>
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Paired Samples Correlations

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Paired Samples Test

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<th>95% Confidence Interval of the Difference</th>
<th>t</th>
<th>df</th>
<th>Sig, (2-tailed)</th>
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<tr>
<td>Pair 1 HERDB4T1 - HERDAFT1</td>
<td>1551.9524</td>
<td>12867.6553</td>
<td>1621.1722</td>
<td>-1688.7245 - 4792.6294</td>
<td>957</td>
<td>62</td>
<td>.342</td>
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</table>
4.3.3 UNPAIRED T-TEST FOR HERD A (CONTROL) AND HERD B (TREATMENT) BEFORE THE TRIAL PERIOD.

The Unpaired T-Test for Herd A and Herd B involved an intergroup comparison of the Somatic Cell Counts of Herd A (control) and Herd B (treatment) before the treatment to determine if there was any significant difference between the two herds.

Two hypothesis were set up:

Ho: (The null hypothesis) There is no difference between the Somatic Cell Counts of Herd A and Herd B before the treatment ($\mu_a = \mu_b$).

$H_1$: (The alternative hypothesis) There is difference between the Somatic Cell Counts of Herd A and Herd B before the treatment. ($\mu_a \neq \mu_b$)
The level of significance is set at $\alpha = 0.05$, therefore the null hypothesis is rejected if $P < \alpha$.

The outcome in this case was that $P > 0.05$, therefore there was no statistical difference between the Somatic Cell Counts of Herd A and Herd B before the treatment period.

4.3.4 UNPAIRED T-TEST FOR HERD A (CONTROL) AND HERD B (TREATMENT) AFTER THE TRIAL PERIOD.

The Unpaired T-Test for Herd A and Herd B involved an intergroup comparison of the Somatic Cell Counts of Herd A (control) and Herd B (treatment) after the treatment to determine if there was any significant difference between the two herds.
Two hypotheses were set up:

\( H_0: \) (The null hypothesis) There is no difference between the Somatic Cell Counts of Herd A and Herd B after the treatment period \( (\mu_a = \mu_b) \).

\( H_1: \) (The alternative hypothesis) There is a difference between the Somatic Cell Counts of Herd A and Herd B after the treatment period. \( (\mu_a \neq \mu_b) \)

The level of significance is set at \( \alpha = 0.05 \), therefore the null hypothesis is rejected if \( P < \alpha \).

The outcome in this case was that \( P > 0.05 \), therefore there was no statistical difference between the Somatic Cell Counts of Herd A and Herd B after the treatment period.
TABLE 4.3 - INTERGROUP COMPARISON BETWEEN HERD A (CONTROL) AND HERD B (TREATMENT) GROUPS WITH REGARDS TO THE BEFORE AND AFTER SCC RESULTS FOR TRIAL 1.

T-Test

Group Statistics

<table>
<thead>
<tr>
<th>GROUP</th>
<th>N</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>HERDB4T1 1.00</td>
<td>86</td>
<td>4047.9884</td>
<td>4426.6394</td>
<td>477.3368</td>
</tr>
<tr>
<td>2.00</td>
<td>63</td>
<td>2607.9206</td>
<td>12621.4263</td>
<td>1590.1502</td>
</tr>
<tr>
<td>HERDAFT1 1.00</td>
<td>86</td>
<td>3379.9186</td>
<td>15135.6956</td>
<td>1632.1240</td>
</tr>
<tr>
<td>2.00</td>
<td>63</td>
<td>1055.9683</td>
<td>2121.2091</td>
<td>267.2472</td>
</tr>
</tbody>
</table>

Independent Samples Test

<table>
<thead>
<tr>
<th></th>
<th>Levene's Test for Equality of Variances</th>
<th>t-test for Equality of Means</th>
<th>95% Confidence Interval of the Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F</td>
<td>Sig.</td>
<td>t</td>
</tr>
<tr>
<td>HERDB4T1 Equal variances assumed</td>
<td>.017</td>
<td>.898</td>
<td>.980</td>
</tr>
<tr>
<td>Equal variances not assumed</td>
<td>.867</td>
<td>73.243</td>
<td>.389</td>
</tr>
<tr>
<td>HERDAFT1 Equal variances assumed</td>
<td>4.745</td>
<td>.031</td>
<td>1.209</td>
</tr>
<tr>
<td>Equal variances not assumed</td>
<td>1.405</td>
<td>89.531</td>
<td>.163</td>
</tr>
</tbody>
</table>
4.3.5 TEST FOR SAMPLE PROPORTIONS – TRIAL 1

This test is performed to compare the proportion of clinical cases that occurred during the trial in herd A, to the proportion of clinical cases that occurred during the trial in herd B.

Two hypothesis were set up:

$H_0:$ (The null hypothesis) There is no difference between the proportion of clinical case in herd A and the proportion of clinical cases in Herd B.

$H_1:$ (The alternative hypothesis) There is a difference between the proportion of clinical case in herd A and the proportion of clinical cases in Herd B.

The level of significance is set at $\alpha = 0.05$, therefore the null hypothesis is rejected if $P < \alpha$. The $Z$ level of significance rejects the null hypothesis if $-1.96 \geq Z \leq 1.96$.

In this case $Z = 2.743$ which falls outside the accepted region. This indicates that there is a significant difference between the two proportions.
4.3.5.1 Formulae used in test for sample proportions – Trial 1

\[ P = \frac{c}{n} \quad p = \frac{c_1 + c_2}{n_1 + n_2} \quad q = 1 - p \]

\[ Z = \frac{P_1 - P_2}{pq \left( \frac{1}{n_1} + \frac{1}{n_2} \right)} \]

- \( P \) – proportion of clinical cases to the number of cattle in herd
- \( c \) – number of clinical cases
- \( n \) – number of cattle in the herd
- \( p \) – a ratio between the sum of clinical cases and the sum of cattle in the herds
- \( c_1 \) – number of cases in Herd A
- \( c_2 \) – number of cases in Herd B
- \( n_1 \) – number of cattle in Herd A
- \( n_2 \) – number of cattle in Herd B
- \( Z \) – test statistic
- \( P_1 \) – proportion of clinical cases in herd A
- \( P_2 \) – proportion of clinical cases in herd B
TABLE 4.4 - TEST FOR SAMPLE PROPORTIONS OF CLINICAL CASES OF MASTITIS IN TRIAL 1.

TEST FOR SAMPLE PROPORTIONS

4.3.5 Table 1  P - Values

<table>
<thead>
<tr>
<th>Herd</th>
<th>n</th>
<th>c</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (control)</td>
<td>63</td>
<td>28</td>
<td>0.444'</td>
</tr>
<tr>
<td>B (treatment)</td>
<td>86</td>
<td>19</td>
<td>0.221</td>
</tr>
</tbody>
</table>

4.3.5 Table 2  Z - Values

<table>
<thead>
<tr>
<th></th>
<th>n1</th>
<th>n2</th>
<th>P1</th>
<th>P2</th>
<th>p</th>
<th>q</th>
<th>Z</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial 1</td>
<td>63</td>
<td>86</td>
<td>0.444'</td>
<td>0.221</td>
<td>0.3154</td>
<td>0.6846</td>
<td>2.743</td>
</tr>
</tbody>
</table>
4.4 STATISTICAL ANALYSIS – TRIAL 2

4.4.1 PAIRED T-TEST HERD A (CONTROL)

The Paired T-Test for Herd A was an intra group comparison between the before treatment and the after treatment Somatic Cell Counts to determine if there was any significant difference between the two.

Two hypothesis were set up:

$H_0$: (The null hypothesis) There is no difference between the somatic cell counts for Herd A. ($\mu_a = 0$)

$H_1$: (The alternative hypothesis) There is a difference between the somatic cell counts for Herd A. ($\mu_a \neq 0$)
The level of significance is set at $\alpha = 0.05$, therefore the null hypothesis is rejected if $P < \alpha$.

The outcome in this case was that $P > 0.05$, therefore there was no statistical difference between the before and after Somatic Cell Count results for Herd B.
TABLE 4.5 - INTRAGROUP COMPARISON BETWEEN THE BEFORE AND AFTER TREATMENT

SCC RESULTS WITH REGARDS TO HERD A (CONTROL) IN TRIAL 2.

Intragroup comparison between before and after treatment for Herd A (control) for trial 2

T-Test

Paired Samples Statistics

<table>
<thead>
<tr>
<th>Pair</th>
<th>HERDB4T2</th>
<th>HERDAFT2</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2453.6000</td>
<td>942.4870</td>
<td>115</td>
<td>9760.9129</td>
<td>910.2098</td>
</tr>
</tbody>
</table>

Paired Samples Correlations

<table>
<thead>
<tr>
<th>Pair</th>
<th>N</th>
<th>Correlation</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>115</td>
<td>.343</td>
<td>.000</td>
</tr>
</tbody>
</table>

Paired Samples Test

<table>
<thead>
<tr>
<th>Pair</th>
<th>HERDB4T2 - HERDAFT2</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th>95% Confidence Interval of the Difference</th>
<th>t</th>
<th>df</th>
<th>Sig. (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1511.1130</td>
<td>9290.7883</td>
<td>866.3705</td>
<td>-205.1602</td>
<td>3227.3863</td>
<td>1.744</td>
<td>114</td>
<td>.084</td>
</tr>
</tbody>
</table>
4.4.2 PAIRED T-TEST HERD B (TREATMENT)

The Paired T-Test for Herd B was an intra group comparison between the before treatment and the after treatment Somatic Cell Counts to determine if there was any significant difference between the two.

Two hypothesis were set up:

Ho : (The null hypothesis) There is no difference between the somatic cell counts for Herd B. 
(μa = 0)

H1 : (The alternative hypothesis) There is a difference between the somatic cell counts for Herd B. (μa ≠ 0)

The level of significance is set at α = 0.05, therefore the null hypothesis is rejected if P < α.

The outcome in this case was that P > 0.05, therefore statistically there was no difference between the Somatic Cell Count results for Herd B before and after treatment.
TABLE 4.6 - INTRAGROUP COMPARISON BETWEEN THE BEFORE AND AFTER TREATMENT SCC RESULTS WITH REGARDS TO HERD B (TREATMENT) IN TRIAL 2.

**T-Test**

**Paired Samples Statistics**

<table>
<thead>
<tr>
<th>Pair</th>
<th>HERDB4T2</th>
<th>HERDAFT2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1571.1489</td>
<td>4849.8511</td>
</tr>
<tr>
<td>N</td>
<td>94</td>
<td>94</td>
</tr>
<tr>
<td>Std. Deviation</td>
<td>10392.1912</td>
<td>20304.3618</td>
</tr>
<tr>
<td>Std. Error Mean</td>
<td>1071.8727</td>
<td>2094.2350</td>
</tr>
</tbody>
</table>

**Paired Samples Correlations**

<table>
<thead>
<tr>
<th>Pair</th>
<th>HERDB4T2 &amp; HERDAFT2</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>94</td>
</tr>
<tr>
<td>Correlation</td>
<td>-0.033</td>
</tr>
<tr>
<td>Sig.</td>
<td>.755</td>
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</tbody>
</table>

**Paired Samples Test**

<table>
<thead>
<tr>
<th>Paired Differences</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th>95% Confidence Interval of the Difference</th>
<th>t</th>
<th>df</th>
<th>Sig. (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pair 1 HERDB4T2 - HERDAFT2</td>
<td>-3278.7021</td>
<td>23109.3708</td>
<td>2383.5496</td>
<td>-8011.9593, 1454.5550</td>
<td>-1.376</td>
<td>93</td>
<td>.172</td>
</tr>
</tbody>
</table>
4.4.3 UNPAIRED T-TEST FOR HERD A (CONTROL) AND HERD B (TREATMENT) BEFORE THE TRIAL PERIOD.

The Unpaired T-Test for Herd A and Herd B involved an intergroup comparison of the Somatic Cell Counts of Herd A (control) and Herd B (treatment) before the treatment to determine if there was any significant difference between the two herds.

Two hypothesis were set up:

Ho: (The null hypothesis) There is no difference between the somatic cell counts for Herd B. ($\mu_a = 0$)

$H_1$: (The alternative hypothesis) There is a difference between the somatic cell counts for Herd B. ($\mu_a \neq 0$)
The level of significance is set at $\alpha = 0.05$, therefore the null hypothesis is rejected if $P < \alpha$.

The outcome in this case was that $P > 0.05$, therefore there was no statistical difference between the Somatic Cell Counts of Herd A and Herd B before the treatment period.

4.4.4 UNPAIRED T-TEST FOR HERD A (CONTROL) AND HERD B (TREATMENT) AFTER THE TRIAL PERIOD.

The Unpaired T-Test for Herd A and Herd B involved an intergroup comparison of the Somatic Cell Counts of Herd A (control) and Herd B (treatment) after the treatment to determine if there was any significant difference between the two herds.
Two hypothesis were set up:

$H_0$: (The null hypothesis) There is no difference between the Somatic Cell Counts of Herd A and Herd B after the treatment period.

$H_1$: (The alternative hypothesis) There is a difference between the Somatic Cell Counts of Herd A and Herd B after the treatment period.

The level of significance is set at $\alpha = 0.05$, therefore the null hypothesis is rejected if $P < \alpha$.

The outcome in this case was that $P < 0.05$, therefore there was a statistical difference between the Somatic Cell Counts of Herd A and Herd B after the treatment period.
TABLE 4.7 INTERGROUP COMPARISON BETWEEN HERD A (CONTROL) AND HERD B (TREATMENT) GROUPS WITH REGARDS TO THE BEFORE AND AFTER SCC RESULTS FOR TRIAL 2.

T-Test

Group Statistics

<table>
<thead>
<tr>
<th>GROUP</th>
<th>N</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>HERDB4T2 1.00</td>
<td>115</td>
<td>2453.6000</td>
<td>9760.9129</td>
<td>910.2098</td>
</tr>
<tr>
<td>HERDB4T2 2.00</td>
<td>94</td>
<td>1571.1489</td>
<td>10392.1912</td>
<td>1071.8727</td>
</tr>
<tr>
<td>HERDAFT2 1.00</td>
<td>115</td>
<td>942.4870</td>
<td>1844.4059</td>
<td>171.9917</td>
</tr>
<tr>
<td>HERDAFT2 2.00</td>
<td>94</td>
<td>4849.8511</td>
<td>20304.3618</td>
<td>2094.2350</td>
</tr>
</tbody>
</table>

Independent Samples Test

<table>
<thead>
<tr>
<th>GROUP</th>
<th>Levene's Test for Equality of Variances</th>
<th>t-test for Equality of Means</th>
<th>95% Confidence Interval of the Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F</td>
<td>Sig.</td>
<td>t</td>
</tr>
<tr>
<td>HERDB4T2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Equal variances assumed</td>
<td>.370</td>
<td>.543</td>
<td>.632</td>
</tr>
<tr>
<td>Equal variances not assumed</td>
<td>.628</td>
<td>193.430</td>
<td>.531</td>
</tr>
<tr>
<td>HERDAFT2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Equal variances assumed</td>
<td>19.326</td>
<td>.000</td>
<td>-2.054</td>
</tr>
<tr>
<td>Equal variances not assumed</td>
<td>-1.860</td>
<td>94.255</td>
<td>.066</td>
</tr>
</tbody>
</table>
4.4.5 TEST FOR SAMPLE PROPORTIONS – TRIAL 2

This test is performed to compare the proportion of clinical cases that occurred during the trial in herd A, to the proportion of clinical cases that occurred during the trial in herd B.

Two hypotheses were set up:

Ho: (The null hypothesis) There is no difference between the proportion of clinical cases in herd A and the proportion of clinical cases in Herd B.

H1: (The alternative hypothesis) There is a difference between the proportion of clinical cases in herd A and the proportion of clinical cases in Herd B.

The level of significance is set at $\alpha = 0.05$, therefore the null hypothesis is rejected if $P < \alpha$. The $Z$ level of significance rejects the null hypothesis if $-1.96 \leq Z \leq 1.96$.

In this case $Z = -0.205$ which falls within the accepted region. The null hypothesis is therefore accepted, as there is no significant difference between the two proportions.
4.4.5.1 Formulae used in test for sample proportions – Trial 2

\[ P = \frac{c}{n} \quad p = \frac{c_1 + c_2}{n_1 + n_2} \quad q = 1 - p \]

\[ Z = \frac{P_1 - P_2}{\sqrt{pq\left(\frac{1}{n_1} + \frac{1}{n_2}\right)}} \]

- P – proportion of clinical cases to the number of cattle in herd
- c – number of clinical cases
- n – number of cattle in the herd
- p – a ratio between the sum of clinical cases and the sum of cattle in the herds
- c₁ – number of cases in Herd A
- c₂ – number of cases in Herd B
- n₁ – number of cattle in Herd A
- n₂ – number of cattle in Herd B
- Z – test statistic
- P₁ – proportion of clinical cases in herd A
- P₂ – proportion of clinical cases in herd B
TABLE 4.8 - TEST FOR SAMPLE PROPORTIONS OF CLINICAL CASES OF MASTITIS IN TRIAL 2.

TEST FOR SAMPLE PROPORTIONS

4.4.5 Table 1  P - Values

<table>
<thead>
<tr>
<th>Herd</th>
<th>n</th>
<th>c</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (control)</td>
<td>94</td>
<td>13</td>
<td>0.138</td>
</tr>
<tr>
<td>B(treatment)</td>
<td>115</td>
<td>17</td>
<td>0.148</td>
</tr>
</tbody>
</table>

4.4.5 Table 2  Z - Values

<table>
<thead>
<tr>
<th></th>
<th>n1</th>
<th>n2</th>
<th>P1</th>
<th>P2</th>
<th>p</th>
<th>q</th>
<th>Z</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial 1</td>
<td>94</td>
<td>115</td>
<td>0.138</td>
<td>0.148</td>
<td>0.1435</td>
<td>0.8565</td>
<td>-0.205</td>
</tr>
</tbody>
</table>
CHAPTER FIVE

DISCUSSION

INTRODUCTION

This research was designed to investigate the efficacy of the Homoeopathic complex of *Phytolacca decandra* 200CH, *Bryonia alba* 30CH and *Silicea terra* 30CH in the prophylactic treatment of bovine mastitis.

Two sets of criteria were considered in the assessment of the efficacy of the homoeopathic complex, namely the results of the somatic cell count tests as well as the incidences of clinical mastitis that were recorded during the trial period.

Two separate trials were conducted in order to reduce the influence of other variables on the outcome of the results. This was considered necessary in light of the number of variables that are known to influence the incidence of mastitis.

Five statistical tests were carried out on each set of results obtained during the two trials. The first four tests involved the results of the
somatic cell counts tests obtained from the Allerton Regional Veterinary Laboratory, and the fifth statistical test involved the results obtained on the incidence of clinical mastitis.

The first test conducted was an intragroup comparison between the somatic cell count results of Herd A taken before the trial and those taken after the trial.

The second test conducted was an intragroup comparison between the somatic cell count results of Herd B taken before the trial and those taken after the trial.

The third test was an intergroup comparison of the somatic cell count result of herd A before the trial and those of herd B before the trial.

The fourth test was an intergroup comparison of the somatic cell count result of herd A after the trial and those of herd B after the trial.
The fifth test was a test for proportions. This test was used to statistically compare the proportion of clinical cases of mastitis that occurred in herd A during the trial to the proportion that occurred in herd B.

6.1 TRIAL 1

No statistically significant difference was found in either of the intragroup comparisons, of the results of the somatic cell count testing before and after the trial. For herd A or for herd B.

Similarly no statistically significant difference was found in either of the intergroup comparisons, of the results of the somatic cell count tests of herd A and herd B, before the trial or after the trial.

A statistically significant difference was however evident when the test for proportions was conducted on the incidence of mastitis during the trial. The proportion of the incidence of mastitis in herd A (the control herd) was significantly higher than that of herd B (the test herd). This means that there were proportionately fewer cases of clinical mastitis in herd B than in herd A.
6.2 TRIAL 2

No statistical difference was found in either of the intragroup comparisons, of the results of the somatic cell count testing before and after the trial. For herd A or for herd B.

Similarly no statistically significant difference was found in the intergroup comparisons, of the results of the somatic cell count tests of herd A and herd B, before the trial. A statistical difference was however found in the intergroup comparison, of the results of the somatic cell count testing of herd A and herd B, after the trial. It was found that the somatic cell count tests of herd B were in actual fact significantly higher than those of herd A, Which was in contradiction to the aim of the research.

No statistically significant difference was found when the test for proportions was conducted on the incidence of mastitis in each herd during the trial.
6.1 CONCLUSIONS

The results obtained during the study into the efficacy of the Homoeopathic complex of *Phytolacca decandra* 200CH, *Bryonia alba* 30CH and *Silicea terra* 30CH in the prophylactic treatment of bovine mastitis were in the end inconclusive.

There was an indication of an improvement in the incidence of cases of clinical mastitis that occurred in the treatment herd of trial 1, but this was not found to be the case in trial 2. This does give rise to the possibility that the homoeopathic treatment could be effective, but due to the high number of variables responsible for
the high incidence of mastitis, a positive effect in only one trial becomes nullified and is deemed inconclusive.

The only other significant difference found on statistical analysis of the data obtained was in trial 2. There was a significant difference between the somatic cell counts of herd A and herd B after the trial, which in fact demonstrated that the control herd had better somatic cell counts than that of the treatment herd. Again this can be attributed to a significant number of variables so the results are nullified especially as there were no intercurrent somatic cell count tests performed, which may have demonstrate a trend in the results.

It is clearly evident from this research that there is great scope for further research to be conducted into the homoeopathic treatment of bovine mastitis, that it should be conducted under far more controlled conditions.
6.2 RECOMMENDATIONS

When the concept of this research was first developed, it was designed with the idea of trying to conduct the research in such a manner as to make it viable for use in the commercial dairy industry. This may have been a good idea in concept, but unfortunately this type of research methodology may be a bit premature as relatively little research has been conducted into the use of homoeopathy in order to treat mastitis.

There are an incredible number of variables that can contribute to the high incidence of mastitis, any number of which could be held responsible for any anomalies that might be found in the results of research conducted using homoeopathic remedies to try and treat mastitis. For this reason it is recommended by the researcher that any future trials should be conducted in a far more controlled environment, ensuring that the trial and control herds are kept under identical conditions and are protected as far as possible from any environmental stresses.
The ideal of trying to isolate the homoeopathic treatment, as the only variable in the research is further compounded by the fact that homoeopathic treatment is dynamic, and not an exact science where only one solution to a problem exists. Homoeopathic remedies are not specific to a condition or pathogen, but rely on a homoeopath to accurately match up a remedy picture to what they perceive the signs and symptoms of the case to be. They must then also select a potency of the remedy that they feel will best effect the cure. When all this is considered one begins to appreciate why it can be so challenging to conduct research into the use of homoeopathy to treat mastitis.

The researcher thus recommends that anyone considering trying to conduct research into the homoeopathic treatment of mastitis should consider the possibility of following up on previous research and just altering one or two of the other variables to see if the outcome of the research remains the same. This could be far more beneficial to our knowledge and understanding of the treatment of mastitis than the results of research where the methodology has
been radically altered. It will also help to confirm or disprove any previous findings and in that way consolidate our knowledge.

Another possibility to consider would be to conduct more frequent somatic cell count tests throughout the duration of the trial. If somatic cell count tests are only conducted before and after the trial period, the validity of the results could be called into question as any deviance in the results could easily be attributed to other factors. More frequent somatic cell count tests would also allow the researcher to more closely monitor the progress of the treatment and the results obtained would be of more value. It would also be of value to conduct the tests more frequently if the research is to be conducted over a long period of time where the repeated dosing could lead to a homoeopathic proving which would give a false result to the test. The negative side of more frequent somatic cell count testing however is in the cost. It would probably only be a viable option if the test and control herds were kept relatively small.
After discussing the outcome of the trial with the farmers and other researchers, it is quite evident that future researchers should take more care when selecting the individual cows for both the control and the test herds. The validity of the results would be improved if the two herds were as homogenous as possible, so that the herds would be as identical as possible in relation to age, size, milk production, breeding and number of lactations.

The use of other parameters to validate any findings is another point to consider when designing future research. Taking somatic cell count readings from every cow can become expensive, but one possibility to consider would be to take somatic cell count readings of the milk in the bulk tank. This parameter used on its own as a way to assess the health of the whole herd instead of individual animals, or it could be used as a cheaper method of monitoring the progression of the research over the trial period. Another possible parameter would be to keep a record of the milk volumes produced. As mastitis results in a decrease in milk production it follows that the volumes of milk produced should
increase if the incidence of mastitis is reduced. This again could be done on an individual basis or for the whole herd.

For a more precise assessment of the incidence of clinical cases of mastitis one could also consider using the California Milk Cell Test as a more precise and scientific method of assessing the incidence of clinical mastitis. The cost however far outweighs any advantages it may have over the ability of the dairy staff to determine any cases of mastitis.

Classical homoeopathic treatment involves individualistic assessment and treatment of each case aimed at treating the patient as a whole, and does not focus on the treatment of the specific condition. When remedies are prescribed using this method the remedy is referred to as the similimum. This is the most effective method of prescribing homoeopathic remedies, but it requires skill and time and unfortunately does not lend itself to scientific research methodologies. Researchers may want to consider this when designing future research projects, possibly
comparing the effectiveness of an acute mastitis complex to the effectiveness of simillimum prescribing.

All things considered the researcher feels that there is an enormous amount of scope for research into the homoeopathic treatment of bovine mastitis, but recommends that future trials should be conducted in a far more controlled environment where the animals selected to participate in the trial are split into two homogenous groups as far as possible. The use of more frequent somatic cell count tests or of monitoring of the bulk tank somatic cell count throughout the duration of the trial is also recommended.
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APPENDICES
APPENDIX A

ALLERTON MILK HYGIENE LABORATORY: SUBMISSION OF MILK SAMPLES

A. BEFORE YOU SUBMIT YOUR MILK SAMPLES -
1. Please phone the Milk Hygiene Laboratory (033 3471931) in advance to book a test date.
2. Your milk samples MUST reach the laboratory on the previous day OR before 8:30am on the day on which they are booked.

B. IDENTIFICATION OF MILK SAMPLES
1. Number the sample tubes sequentially eg 1 to 100.
2. Number the cow identification sheets sequentially in the column headed "SAMPLE NUMBER" (eg 1 to 100).
3. Identify each cow in the column headed "COW NUMBER". This can be done before or during sample collection. Please if possible use the numbers rather than the names to identify your cows. Use the sample tube corresponding to the sample number allocated to each cow.
4. PLEASE DO NOT LEAVE ANY GAPS. If necessary substitute another or other samples in order to maintain continuity. Once your milk samples have been processed, the computer will sort them into alpha numerical order but it will not accept any gaps in the sequence of tubes.
5. Quarter samples can be identified in the column headed QUARTER.
6. Do not forget to write your name and the date on each sheet and to submit the completed sheets together with your samples.
C. SAMPLING PROCEDURE

AIM: TO COLLECT FOREMILK SAMPLES WHICH ARE FREE FROM CONTAMINANT BACTERIA FOUND ON THE TEAT SKIN AND HANDS OR IN DUST AND MANURE WITH CLEAN HANDS

1. Wash each teat (not the whole udder) with clean running water. Dry the teats with a piece of clean disposable paper towel.
2. Disinfect the teat ends with a piece of cotton wool soaked in methylated spirits.
3. Discard the first few squirts of milk into a strip cup in order to flush the teat canal.
4. Squirt the milk into the sterile sample tube provided. Hold the tube at an angle so that dirt does not fall in. Do not touch the lip of the tube or the part of the rubber stopper which comes into contact with the milk. Fill the tube to about two thirds full.

COMPOSITE SAMPLE: An equal amount of milk from each quarter in one sample tube.
QUARTER SAMPLE: Milk from one quarter in one sample tube.

5. NB KEEP THE SAMPLES COOL (BUT NOT FROZEN) UNTIL THEY REACH THE LABORATORY.

“LABORATORY DIAGNOSIS CAN ONLY BE AS GOOD AS THE SAMPLE RECEIVED.”
APPENDIX B

(ARVL Handout)

WHY IS SUB-CLINICAL MASTITIS IMPORTANT?

Sub-clinical mastitis is a costly erosion disease in terms of lost milk production and poor milk quality.

BULK MILK SOMATIC CELL COUNTS, LEVELS OF MASTITIS AND MANAGEMENT PROBLEMS, LOSSES OF MILK PRODUCTION AND MILK QUALITY

<table>
<thead>
<tr>
<th>Bulk milk somatic count per ml.</th>
<th>Level of mastitis and management problems</th>
<th>% Lost milk production</th>
<th>Milk cell quality</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 250 000</td>
<td>None to slight</td>
<td>5.0</td>
<td>Very good to good</td>
</tr>
<tr>
<td>250 000 - 500 000</td>
<td>Moderate to doubtful</td>
<td>10.0</td>
<td>Moderate to suspect</td>
</tr>
<tr>
<td>500 000 - 750 000</td>
<td>Unsatisfactory to distinctly unsatisfactory</td>
<td>12.0</td>
<td>Unsatisfactory</td>
</tr>
<tr>
<td>750 000 - 1 000 000</td>
<td>Serious</td>
<td>15.0</td>
<td>Unfit</td>
</tr>
<tr>
<td>&gt; 1 000 000</td>
<td>Very serious</td>
<td>&gt; 18.0</td>
<td>Unfit</td>
</tr>
</tbody>
</table>

In terms of hard cash, consider a herd of 100 cows producing an average of 5 000lt of milk each per lactation, with an average bulk milk somatic cell count of 500 000 somatic cells per ml. Production could be increased by 5% if the bulk milk somatic cell count were reduced to 250 000 somatic cells per ml. At a selling price of 50 cents per liter, a saving of R12 500 could be achieved.
APPENDIX C

FARMER CONSENT FORM

RESEARCH PROJECT TITLE: The Prophylactic Treatment of Bovine Mastitis using a Homoeopathic Complex of Phytolacca Decandra, Bryonia Alba and Silicea.

RESEARCH STUDENT: Lindy Clark

RESEARCH SUPERVISOR: Dr. Jane Fraser

PLEASE CIRCLE THE APPROPRIATE ANSWER:

1. Has the research project been explained to you? Yes/No
2. Have you had the opportunity to ask questions? Yes/No
3. Are you satisfied with how the research will be conducted? Yes/No
4. Do you have at least 2 herds of cattle that number over 75? Yes/No
5. Are all your herds presently on the Allerton Mastitis Scheme? Yes/No
6. Do you keep records of the number of cases of clinical mastitis? Yes/No
7. Do you understand the implications of your participation in the study? Yes/No
8. Do you feel you have enough information regarding this study? Yes/No
9. Do you agree to voluntarily participate in this study? Yes/No

Approximate no. of Cattle in herd:

NAME OF TRIAL FARM: 75-100/100-150/150-200/+200.

NAME OF CONTROL FARM 1: 75-100/100-150/150-200/+200

NAME OF CONTROL FARM 2: 75-100/100-150/150-200/+200

NAME OF FARMER: SIGNATURE:

NAME OF WITNESS: SIGNATURE:

RESEARCH STUDENT: SIGNATURE: