

HOMOEOPATHY IN HYPERCHOLESTEROLAEMIA

by

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To whom it may concern, this dissertation represents my own work, both in conception and execution. _____

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ABSTRACT

The purpose of this study was to evaluate the effect of Chelidonium 3X in the treatment of hypercholesterolaemia in terms of changes in the blood cholesterol levels in order to determine the extent to which homoeopathic medicine could be used in the treatment of this condition. It was hoped that there would be a drop in the total cholesterol levels and an increase in the ratio of high density / low density lipoprotein.

Participants in the trial were drawn from the staff at Technikon Natal, convenience sampling being used. For acceptance into the trial, participants had to have an elevated total cholesterol level together with a raised low density lipoprotein value according to age. Drawing of blood was performed following an overnight fast. Of those participants meeting the above criteria thirty were chosen to participate in the trial. Half constituted the control and were given a placebo and the other half made up the experimental group and were treated with Chelidonium 3X. This was a double blind study with the medicine being dispensed on a random basis by a qualified pharmacist. After commencement of the trial venous blood was collected once a month for three consecutive months from both the control and experimental groups. The participants were asked to make no changes to their normal lifestyle.

Statistical analysis of the results using paired T-tests revealed no statistical difference between the initial and final total cholesterol or ratio reading of the control group. However, with the experimental group a statistical difference was noted between both the initial and final total cholesterol reading ($p=$

0.023), and the initial and final ratio reading ($p= 0.019$). The unpaired T-tests revealed a difference between the final total cholesterol readings of the control and experimental groups ($p= 0.046$) which indicated a 2.7 % drop in the levels of the total cholesterol of the experimental group as compared to the 0 % drop in the control group.

In concluding, it would seem that experimentally, Chelidonium 3X caused a decrease in the total cholesterol level and an increase in the ratio of high density / low density lipoprotein of the experimental group. However, the margin of improvement does not seem to warrant the use of this substance in the treatment of high to severe hypercholesterolaemia. It could perhaps be used in patients with a moderately raised cholesterol level, but then probably only when used in conjunction with behavior modification such as diet and exercise.

UITTREKSEL

Die doel van die studie was om die uitwerking van Chelidonium 3X te bepaal in die behandeling van hipercholesterolaemia in terme van die verandering in die bloed cholesterolvlak om vas te stel tot watter mate homeopatiese medisyne gebruik kan word in die behandeling van die toestand. Hopelik sou daar 'n verlaging in die totale cholesterol vlak en 'n verhoging in die verhouding van hoë / lae intensiteit lipoproteïene wees.

Die deelnemers aan die studie is uit die personeel aan Technikon Natal gewerf. Die kandidate wat tot die studie toegelaat was, moes 'n verhoogte totale bloedcholesterol vlak, sowel as 'n verhoogte lae intensiteit lipoproteïene waarde volgens ouderdom gehad het. Bloedmonsters is na 'n oornag vastydperk geneem. Dertig pasiënte wat aan die vereistes voldoen het, is tot die studie toegelaat, en in twee groepe ingedeel. Die vyftien pasiënte in die kontrolegroep is placebo toegedien. Die vyftien in die eksperimentele groep is Chelidonium 3X toegedien. Die medisyne is deur 'n gekwalifiseerde apteker op lukraakte grondslag toegedien. Gedurende die studie is binne-aarse bloed maandeliks vir drie agtereenvolgende maande van al dertig pasiënte getrek. Deelnemers aan die studie is versoek om nie hulle lewensyl te verander nie.

Statistiese ontleding van die kontrole groep se resultate het volgens die een-monstertoets geen verskil getoon tussen die aanvanklike en finale totale bloedcholesterol vlak. In die eksperimentele groep, is daar 'n statistiese verskil tussen die aanvanklike en finale totale cholesterol vlak ($p=0.023$), en die aanvanklike en finale ratio lesing ($p=0.019$) gevind. Die twee-monstertoets

toon dat daar 'n verskil is tussen die finale totale cholesterol vlak van die kontrole en eksperimentele groepe ($p=0.046$). Daarvolgens is daar 'n 2.7% verlaging in die totale cholesterol vlak van die eksperimentele groep, maar geen verskil in die kontrole groep se lesings nie.

Ter opsomming: Chelidonium 3X het eksperimenteel 'n verlaging teweeg gebring in die totale bloedcholesterol vlak en 'n verhoging in die ratio hoë / lae inensiteit lipoproteien. Die graad van verbetering blyk nie die gebruik hiervan te regverdig nie in die behandeling van hoë tot ernstige hoë cholesterol nie. Die geneesmiddel kan wel gebruik word vir matige hoë cholesterol, maar dan net wanneer dit gepaard gaan met 'n veranderde lewenstyl.

TABLE OF CONTENTS

CHAPTER ONE

1.1) INTRODUCTION _____	1
1.2) PROBLEM STATEMENT _____	3
1.3) HYPOTHESIS _____	3
1.4) DELIMITATIONS _____	3
1.5) ASSUMPTIONS _____	4

CHAPTER TWO

2.1) INTRODUCTION _____	5
2.2) THE ROLE OF THE LOW DENSITY LIPOPROTEINS _____	5
2.3) THE ROLE OF HIGH DENSITY LIPOPROTEINS _____	6
2.4) DIET _____	9
2.5) EXERCISE _____	12
2.6) BODY WEIGHT _____	13
2.7) THE ROLE OF SMOKING AND OF TRIGLYCERIDES _____	14
2.8) ALLOPATHIC DRUGS AND THEIR SIDE-EFFECTS _____	14
2.9) THE USE OF CHELIDONIUM 3X _____	15

CHAPTER THREE

3.1) THE CRITERIA GOVERNING THE ADMISSIBILITY OF THE DATA ____	17
3.2) THE RESEARCH METHODOLOGY _____	19

CHAPTER FOUR

4.1) RESULTS OF THE PAIRED T-TESTS _____	21
4.2) RESULTS OF THE UNPAIRED T-TESTS _____	23
4.3) FREQUENCY TABLES _____	27
4.4) GRAPHICAL REPRESENTATION OF THE RESULTS _____	29

CHAPTER FIVE

5.1) EVALUATION OF THE RESULTS _____	32
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CHAPTER SIX

6.1) CONCLUSION _____	35
6.2) RECOMMENDATIONS _____	35

REFERENCES _____	36
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LIST OF TABLES.

TABLE ONE

(2 1)

The mean difference between the initial and final total cholesterol reading together with the mean difference between the initial and final HDL / LDL ratio reading over the three month treatment period for both the control and experimental groups.

TABLE TWO

(2 3)

The mean (x) and standard deviation (SD) of the initial total cholesterol and HDL / LDL ratio reading for both the experimental and control groups.

TABLE THREE

(2 4)

The mean (x) and standard deviation (SD) of the total cholesterol and HDL / LDL ratio readings after one month of treatment for both the control and experimental groups.

TABLE FOUR

(2 5)

The mean (x) and standard deviation (SD) of the total cholesterol and HDL / LDL ratio readings after two months of treatment for both the control and experimental groups.

TABLE FIVE

(26)

The mean (\bar{x}) and standard deviation (SD) of the total cholesterol and HDL / LDL ratio readings on completion of the three month trial for both the control and experimental groups.

TABLE SIX

(27)

Frequency table depicting any changes in the total cholesterol values for both the control and experimental groups over the three month trial period.

TABLE SEVEN

(28)

Frequency table depicting any changes in the HDL / LDL ratio reading for both the control and experimental groups over the three month trial period.

LIST OF FIGURES

FIGURE ONE (29)

The mean total cholesterol readings of the control and experimental groups taken before the start of medication and then once a month for three consecutive months.

FIGURE TWO (30)

The mean HDL / LDL ratio readings of the control and experimental groups taken once before the start of medication and then once a month for three consecutive months.

FIGURE THREE (31)

The difference between the means of the total cholesterol for the control and experimental groups taken before the onset of medication and then once a month for three consecutive months.

TABLE OF ABBREVIATIONS

HDL - high density lipoproteins

LDL - low density lipoproteins

TC - total cholesterol

CHAPTER ONE

1.1) INTRODUCTION

The clear relationship between coronary heart disease and raised blood cholesterol levels has led to many attempts to lower cholesterol levels (Leitch 1989). Smith (1992) reports on the Whitehall study, which showed a continuous positive relationship between cholesterol concentration and coronary heart disease. The Adult Treatment Panel of the National Education Programme in the United Kingdom has, however, identified low density lipoprotein as the most important atherogenic lipoprotein, while low levels of high density lipoprotein are seen as the major risk factor for coronary heart disease (Grundy et al. 1987). Many researchers have shown that the lowering of serum cholesterol levels has led to a decrease in mortality due to coronary heart disease, (Cooper et al. 1992), and that total mortality has also been decreased by a reduction in the total cholesterol levels in the serum (Canner et al. 1986 ; The Multiple Risk Factor Intervention Trial Research Group 1990).

The degree of atherosclerosis in coronary heart vessels has been found to be a linear function of serum cholesterol levels, with a proportionate increase in atherosclerosis occurring as the concentration of serum cholesterol levels in the blood increases. This condition is exacerbated by the presence of other risk factors such as hypertension, obesity, cigarette smoking and diabetes mellitus (Reed et al. 1989).

Cooper et al. (1992) has established that there is a regression of atherosclerotic lesions in coronary blood vessels resulting from the lowering

of serum total cholesterol. In confirmation of this claim, several experiments have shown that endothelial healing of the arteries appears to be one of the first results of a decrease in the plasma cholesterol level, thereby decreasing the prevalence of atherosclerotic heart disease (Brown et al. 1990; Ornish et al. 1990).

1.2) PROBLEM STATEMENT

The purpose of this investigation is to evaluate the effect of Chelidonium 3X in the treatment of hypercholesterolaemia in terms of changes in blood cholesterol levels in order to determine the extent to which Homoeopathic medicine can be used in the treatment of this condition.

1.3) HYPOTHESIS

It is hypothesized that the medicine will cause a decrease in the total cholesterol levels and an increase in the ratio of high density / low density lipoprotein.

1.4) DELIMITATIONS

1) This study will not attempt to explain the following:

- a) The mechanism of action of the medicine used.
- b) The effect of diet or exercise on hypercholesterolaemia.
- c) The role played by triglycerides in hypercholesterolaemia.

2) This study will also not attempt to identify the type of hypercholesterolaemia e.g. familial hypercholesterolaemia.

3) People on alternate medication for hypercholesterolaemia may not participate in this trial.

4) Pregnant women will not be used in this trial.

1.5) ASSUMPTIONS

1. It is assumed that:

- a) the patients will take the medicine as prescribed.
- b) the pathogenesis of the remedies to be used is correct.
- c) the medicine will be prepared as set out in the Homoeopathic Pharmacopoeia.
- d) the participants in the trial will make no lifestyle changes.

CHAPTER TWO

2.1) INTRODUCTION

Cholesterol is a subject that has received much attention from the medical fraternity over the years. Although considerable research has been undertaken in this field there is still much that needs to be understood.

In the following literature review, the roles of high density and of low density lipoprotein will be looked into and briefly discussed. Other aspects that have pertinence to cholesterol as a whole, such as exercise and weight management, together with the effects of smoking and the role of triglycerides will be dealt with. There will also be a brief review of some of the most commonly used allopathic drugs, their effects and side-effects. To conclude, the reason for the researcher using the homoeopathic drug, Chelidonium 3X, will be explained.

2.2) THE ROLE OF THE LOW DENSITY LIPOPROTEINS

It has been well established in many trials that high levels of low density lipoprotein correlate directly with the risk of developing coronary heart disease (Brown et al. 1990 ; Council on Scientific Affairs 1983). At present, it is accepted that coronary artery disease is aggravated independently by factors such as an elevated low density lipoprotein level , hypertension , smoking, diabetes mellitis and low levels of high density lipoprotein (Grundy et al. 1987). However, Brown et al. (1990) feels that the above may only be risk factors for coronary atherosclerosis when the low density lipoprotein level is high enough to set the stage for atherosclerosis.

A further description of the role of low density lipoproteins in coronary heart disease is unwarranted, and would be repetitious as many researchers have adequately discussed it. This then brings us to the role of the high density lipoproteins.

2.3) THE ROLE OF HIGH DENSITY LIPOPROTEINS

There is considerable epidemiological data available that indicates an inverse relationship between levels of high density lipoprotein and the incidence of coronary artery disease (Hillery 1983). As the high density lipoprotein level decreases, so the risk of coronary artery disease increases in a fairly linear manner. For each 1mg/dL decrease in high density lipoprotein levels, the risk increases by approximately 2% -3% whereas for each 1mg/dL increase in the low density lipoprotein level the risk of coronary artery disease increases by 1% (Kreisenberg 1993). From this it would seem that high density lipoproteins are more important than low density lipoproteins in predisposing one to coronary heart disease. Gotto (1993), suggests that in patients with dyslipidaemia, the evaluation and treatment decisions, should be based not only on total cholesterol and low density lipoprotein levels, but also on high density lipoprotein and triglyceride levels.

Given the epidemiological data indicating that a low high density lipoprotein level is a risk factor for coronary heart disease, there has been interest in increasing the high density lipoprotein level, although how this would protect against atherosclerosis remains unclear (Kreisenberg 1993). In a review of one hundred and thirteen consecutive patients in a cardiac rehabilitation

programme undertaken by Mailander et al. (1993), low levels of high density lipoprotein were prevalent in cardiac rehabilitation patients, and were far more widespread than elevated low density lipoprotein levels both before and after rehabilitation. He therefore recommended that the National Cholesterol Education Programme should re-evaluate the pivotal role of high density lipoproteins in its assessment and treatment guidelines, particularly in patients with known coronary heart disease, since he felt that emphasis on both low density and high density lipoproteins is needed for optimal primary and secondary prevention of coronary artery disease. It has not yet been shown though whether increasing low high density lipoprotein levels to a more optimal level gives the same protection that would occur had the high density lipoproteins always been at that level.

There is relatively little information on the mechanism behind low high density lipoprotein levels. However, according to Brinton (1991) there is an increase in the catabolism of the high density lipoprotein rather than a decrease in its production. This was found in patients both with and without hypertriglyceridaemia. Other research may seem to show that a low high density lipoprotein level reflects defects in the metabolism of very low density lipoproteins and that it is these lipoproteins that are responsible for atherogenesis and not the low high density lipoprotein levels (Kreisenberg 1993).

Not all people with low high density lipoprotein levels are at risk of developing coronary artery disease. Vegetarians have low high density lipoprotein levels and are not at increased risk of the above, probably because their low density lipoprotein levels are also very low (Kreisenberg 1993). There are, however,

no firm guidelines on the management of patients with isolated low high density lipoprotein levels. There is some agreement though that positive lifestyle changes should be promptly implemented. Such measures would include a weight reduction regime, a cessation of smoking, regular exercise, prudent amounts of alcohol and the use of lipid lowering drugs for other medical conditions (Kreisenberg 1993).

It must be noted that the use of a low-saturated-fat, low cholesterol diet in patients with an isolated low high density lipoprotein level usually further decreases the high density lipoprotein level (Frick et al. 1987). Regular exercise has also shown to be important in preventing the decrease in high density lipoprotein levels that accompanies the Step I diet (Brown et al. 1990), as well as maintaining the fat intake at 35%-40% of caloric need by the substitution of monounsaturated fats for saturated fats in a low-fat diet (Cashin-Hemphill et al. 1990).

It therefore now seems apparent that the indicator of the likelihood of developing atherosclerotic heart disease is not total plasma cholesterol, but rather the ratio of plasma low density cholesterol to plasma high density cholesterol i.e. at any given plasma cholesterol concentration the higher the high density lipoprotein level, the less the risk (Canter 1990). Rossouw et al. (1988) states that since the low density lipoproteins carry cholesterol to the tissues while the high density lipoproteins are involved in the reverse transport pathway. The high density / low density lipoprotein ratio should, theoretically, have a greater predictive power for coronary heart disease than the total cholesterol value alone as it takes into account both the factors which play a role in determining cholesterol levels.

The Council on Scientific Affairs verifies this view by stating that a ratio of total cholesterol / high density lipoprotein or of high density / low density lipoprotein would be an even more precise measure of risk for coronary heart disease than any single component alone (Council on Scientific Affairs 1983).

Consequently, in this study, it was decided to use the ratio of high density / low density lipoprotein as well as the total cholesterol levels when recording the participants' cholesterol levels before and after medication.

2.4) DIET

A diet that has been demonstrated to decrease cholesterol levels is one that is moderately low in total fat, particularly in saturated fats, and low in cholesterol content, but adequate in protein (Council on Scientific Affairs 1983). The diet proposed by the American Health Association includes a daily intake of no more than 30 mg of cholesterol; no more than 30% - 35% of calories as fat with only 10% derived from saturated animal fats and 10% or more being from polyunsaturated fatty acids. (American Heart Association Steering committee for Medical and Community Programs 1980).

According to the American Heart Association a phased approach to dietary management is recommended. The above mentioned diet is phase I. In phase II which is used for unresponsive and severe cases, the fat and cholesterol intake should not exceed 30% of calories and 250mg / day respectively. Phase III calls for a restricted diet containing only 20% -25% of calories as fat, with less than 10% from saturated fat and less than 100mg / day of cholesterol (Council on Scientific Affairs 1983). A programme emphasizing lean meats,

low fat dairy products, fruits, grains, and vegetables is the basis of these recommendations (Russel et al. 1993).

In practical terms this means that the patient should be instructed to limit the intake of all animal fats derived from meat and dairy products; and to use vegetable oil whenever possible. Findings indicate that dietary cholesterol is incompletely absorbed and that those individuals who have the highest levels of low density lipoprotein appear to have the highest percentage of absorption of cholesterol (Cooper et al. 1992).

The Council on Scientific Affairs (1983) states the following as regards diet:

- i) Decreasing saturated fats in the diet and increasing polyunsaturated vegetable oils will generally reduce plasma cholesterol levels.
- ii) Some vegetable oils are more effective than others, and this depends on whether they replace saturated fats or carbohydrates.
- iii) Oils obtained from corn and soyabeans will lower plasma cholesterol when substituted for carbohydrates.
- iv) Substituting monounsaturated oil such as olive oil for saturated fat has the same lowering effect.

Ripsen et al. (1992) found that incorporating oat products into the diet causes a modest reduction in blood cholesterol levels. However, Stewart et al. (1992), in a similar study, found that 50 g of oatbran ingested daily by patients on a low fat diet showed no influence on serum lipid levels.

A high dietary fibre content has been shown to cause a greater average reduction (13%) in serum cholesterol than a low-fat diet (9%)(Anderson et

al. 1992). According to Harris et al. (1990) fish oils in the diet lower plasma triglyceride and very low density lipoprotein levels mainly by inhibiting the synthesis of very low density lipoproteins and triglycerides.

It should be noted though, that the reduction of plasma cholesterol levels attained by dietary means only in the subjects of the Diet Heart Feasibility Study averaged only 10% to 12% (National Diet Heart Study Research Group 1968), while the reduction of plasma cholesterol levels obtained by dietary means only, in the Multi Risk factor Intervention trial, was only 6.5%, despite special attention to patient motivation and dietary instruction (Canter 1990). Thus, while a few patients may achieve a substantial cholesterol reduction in response to diet, many will achieve only a slight improvement (Council on Scientific Affairs 1983).

Haines (1989) and Vander et al. (1985) state that the homeostatic control of cholesterol synthesis by the body is the major reason why it is difficult to alter plasma cholesterol very much in either direction by altering only dietary cholesterol.

Taking the above into account, diet will not be altered in this study and the participants will be advised to continue eating as they had previously. It is accepted that people may change their diet in some way once they realize that they have a raised cholesterol level, but, as the study design includes a control group, any changes in the experimental group, should be reflected in the control group, hopefully making the results of the medication still visible.

2.5) EXERCISE

Relatively consistent strenuous exercise should lower the concentration of triglyceride and low density lipoprotein levels and raise the high density lipoprotein level (Wood and Haskell 1979). Other findings indicate that adults who walk for 2.5 to 4.0 hours a week tend to have a 3% decrease in total cholesterol level and a 3.4% increase in the high density lipoprotein level as compared to subjects that do not walk regularly (Tucker and Friedman 1990 ; Sorg 1993).

According to Taylor and Ward (1993), in a study conducted only on women, exercise training in the absence of other interventions does not cause high density lipoprotein levels in older women to be raised appreciably. He noted though that high volumes of exercise by younger women may cause high density lipoprotein levels to rise.

In an independent study on premenopausal women (Boyden et al. 1993), exercise was shown to cause a decrease in the total cholesterol and low density lipoprotein levels, but no significant changes were noted in the high density lipoprotein or triglyceride levels.

Although the data suggests that exercise plays an important role in cholesterol reduction, participants in the immediate trial were not advised on the role of exercise or asked to embark on some form of physical activity, as the researcher wished only to note the effect of Chelidonium 3X.

2.6) BODY WEIGHT

An excess of body weight is associated with deleterious changes in the lipoprotein profile. A higher Body Mass Index is also associated, at all ages, with higher plasma triglyceride levels, lower high density lipoprotein and higher total cholesterol and low density lipoprotein levels. These results were verified by Denke et al. (1993).

In young men the higher total cholesterol level was reflected mainly in the low density lipoprotein fraction, and in middle aged and older men in the lower high density lipoprotein fraction according to Kasim et al. (1993). He therefore suggested that programmes to decrease coronary artery disease by improving lipid levels should therefore include more emphasis on achieving and maintaining an ideal body weight.

MacDonald et al. (1992) confirms this view by stating that an elevated blood cholesterol level increased with an increased Body Mass Index and Waist / Hip ratio. However, (Majeroni et al. 1992) in his study found that neither weight nor Body Mass Index was associated with elevated serum cholesterol levels, suggesting that screening must be offered, without regard to level of obesity, in order to find those patients who will benefit from intervention.

Wolf and Grundy (1983) state that when an obese patient loses weight, a large decline in triglyceride level usually occurs (about 40%) together with a smaller decline in the total cholesterol or low density lipoprotein levels (about 10%) and an increase in the high density lipoprotein level (about 10%).

Noting that excess body weight plays a role in raised cholesterol levels, and that a loss of weight in an obese or overweight person would seem to lower cholesterol levels, the benefits of weight reduction was not discussed with any of the overweight participants in this particular trial.

2.7) THE ROLE OF SMOKING AND OF TRIGLYCERIDES

Smoking increases low density lipoprotein and triglyceride levels, but decreases high density lipoprotein levels (Brischetto et al. 1983).

With respect to the triglycerides, data is mixed and the evidence on a causal relationship is incomplete. According to a study done by Criqui et al. (1993), the plasma triglyceride level showed no independent association with coronary mortality.

Mindful of the above, these two aspects pertaining to cholesterol were not taken into account at all in this trial.

2.8) ALLOPATHIC DRUGS AND THEIR SIDE-EFFECTS

The three main groups of drugs used in the treatment of hypercholesterolaemia are as follows:

1) Nicotinic acid: This inhibits the secretion of very low density lipoproteins and also decrease the catabolic rate of high density lipoproteins. Side-effects are gastro-intestinal symptoms, flushing and hyper-uricaemia (Rossouw et al.

1988 ; Council on Scientific Affairs 1983).

2) Bile acid binding resins: These are mainly effective in lowering total and low density lipoprotein levels. Side-effects such as gastro- intestinal symptoms and the increased absorption of folic acid , warfarin, digoxin, thyroxine and tricyclines are common (Rossouw et al. 1988 ; Brown et al. 1990).

3)Hydroxymethyl Co A reductase inhibitors: These are the most potent agents available for lowering the low density lipoprotein levels. They also modestly increase the high density lipoprotein levels (Rossouw et al. 1988 ; Brown et al. 1990).

In view of the fact that any drug regimen may have undesirable side effects in some patients and because treatment must be pursued for a lifetime if risk factor reduction is to be maintained, drugs are not recommended except for patients whose lipid profile level remains abnormal in spite of an adequate trial of diet therapy and weight reduction. (Council on Scientific Affairs 1983 ; Oliver 1991).

2.9) THE USE OF CHELIDONIUM 3X

In a study by Bol et al. (1987) oral administration of Chelidonium 3X twice a day was found to partly counteract the increase in serum cholesterol seen in rabbits after cholesterol feeding. After 34 days the rabbits treated with Chelidonium 3X displayed about 25% lower serum cholesterol concentrations than those in the controls group.

The researcher is unaware of any trials having been done using Chelidonium 3X to lower serum cholesterol in humans. Therefore, it was felt that an endeavour to assess the efficacy of this medicine in the treatment of hypercholesterolaemia would be of great value.

CHAPTER THREE

3.1) THE CRITERIA GOVERNING THE ADMISSIBILITY OF THE DATA

- 1) Only data collected from the trial was accepted.
- 2) Only those individuals with increased total cholesterol levels, defined according to specific age cut-off points in combination with an elevated low density lipoprotein level were used in the trial.

The guide to low density lipoprotein action limits in mmol/l by the Lipid Research Council Programme Prevalence Study was used to determine whether the patients had raised low density lipoprotein levels (Heiss et al. 1980), and is as follows:

- < 30 yrs - moderate risk = 2.88 - 4.15
- high risk = > 4.15
- > 30 yrs - moderate risk = 3.40 - 5.20
- high risk = > 5.20

The cut-off points for a raised total cholesterol in mmol/l were those as defined in the Coronary Risk Factor Study (Rossouw et al. 1985) and are as follows:

- < 30 yrs - moderate risk = 4.00 - 5.6
- high risk = > 5.6
- > 30 yrs - moderate risk = 5.2 - 7.3
- high risk = > 7.3

3) All drawing of blood samples had to be carried out either by a registered nurse who drew the blood samples in the researcher's presence, or at the collection rooms of the pathology laboratory being used for the analysis of the samples.

4) Analysis of blood samples for high density lipoprotein, low density lipoprotein and total cholesterol had to be done at a reputable pathology laboratory.

5) No pregnant women were to be used in the trial as total cholesterol and low density lipoprotein levels are increased during pregnancy (Cooper et al. 1992).

6) Blood drawn for the testing of high density lipoprotein, low density lipoprotein and total cholesterol levels had to be taken after an overnight fast.

[Fasting for twelve to fourteen hours is desirable before a test specimen is collected for a battery of lipid measurements. This is essential for triglyceride assays but not generally for total cholesterol measurements. Since the high density lipoprotein level decreases postprandially due to involvement in metabolism of triglyceride rich lipoproteins, it is preferable to analyze a fasting sample for measuring the high density lipoprotein level (Cooper et al. 1992).]

7) No one was accepted within one month of a myocardial infarction as this is associated with a decrease in the total cholesterol and low density lipoprotein concentrations (Cooper et al. 1992).

8) Participants were not to have recently undergone surgery.

9) Those individuals used in the trial were not to be on cholesterol reducing medication.

3.2) THE RESEARCH METHODOLOGY

On commencement of the trial, convenience sampling was used in order to locate participants. A letter was sent to all the staff at the Technikon Natal stating that a cholesterol study was being undertaken, and inviting those interested to undergo a free cholesterol test. Those individuals responding to the letter were then screened for elevated total cholesterol levels according to specific age cut off points, using a Boehringer Reflotron. This was done in a non-fasting situation.

Those individuals with an elevated total cholesterol level were then tested for raised levels of low density lipoprotein as well as a raised total cholesterol level, in order to determine a baseline for the study. The blood samples were collected after an overnight fast with the patient seated, and then analyzed by a pathology laboratory using the Technicon Method (Allain et al. 1974 ; Lie et al. 1976).

Of those individuals who were found to have an elevated low density lipoprotein cholesterol level according to age, thirty were chosen to participate in the three month trial. Their levels of low density lipoprotein, high density lipoprotein and total cholesterol, together with their age and sex were noted

and tabulated. Fifteen were then given a placebo and fifteen were treated with Chelidonium 3X. The medication was to be taken twice a day on waking and at bedtime. This was a "double blind" study with the medicine being dispensed by a qualified pharmacist on a random basis.

After commencement of the medication, venous blood was then collected once a month for three consecutive months from both the control and the experimental group. This procedure was performed following a fast on the part of the trial participants. The blood samples were then analyzed at the same pathology laboratory for the levels of total cholesterol, high density lipoprotein and low density lipoprotein. The figure for total cholesterol was utilized alone, whilst the latter were recorded in the ratio high density / low density lipoprotein.

One control sample was sent in each month to ensure that the results were valid and reliable i.e. two samples from the same participant, but under a different name, were sent in for analysis.

Upon completion of the trial period, changes in weight, diet, use of medication, smoking habits and exercise status as related by the participants, were recorded.

CHAPTER FOUR

4.1) RESULTS OF THE PAIRED T-TESTS

TABLE ONE

The mean difference between the initial and final total cholesterol reading together with the mean difference between the initial and final HDL / LDL ratio reading over the three month treatment period for both the control and experimental groups.

GROUP	TOTAL CHOLESTEROL			HDL / LDL RATIO		
	d1	SD	95 % CI	d2	SD	95 % CI
TREATED	0.321	0.491	0.049;0.593	-0.049	0.068	(0.084)(0.008)
CONTROL	0.106	0.493	(0.167);0.379	-0.027	0.1	(0.082);0.28

d1= mean difference between the initial and final total cholesterol readings.

d2= mean difference between the initial and final ratio readings.

CI = confidence interval.

()= a negative number.

SD = standard deviation.

The results of the paired T-test were as follows:

i) No statistical difference was found between the initial and final total cholesterol or ratio readings of the control group.

ii) A statistical difference was, however, found between the following:

a) the initial and final total cholesterol reading in the experimental group

($p=0.023$).

b) the initial and final ratio reading of the experimental group ($p=0.019$).

4.2) RESULTS OF THE UNPAIRED T-TESTS

TABLE TWO

The mean (x) and standard deviation (SD) of the initial total cholesterol and HDL /LDL ratio readings for both the experimental and control groups.

GROUP	TOTAL CHOLESTEROL			HDL / LDL RATIO		
	X1	SD	95 % CI	X2	SD	95 % CI
TREATED	6.684	0.872	0.338 ; 0.1607	0.29	0.101	(0.120);0.027
CONTROL	5.711	0.822	0.338 ; 0.1607	0.337	0.961	(0.120);0.027

X1 = mean total cholesterol reading.

X2 = mean HDL / LDL ratio reading.

CI = confidence interval.

() = a negative number.

Analysis of the results in Table. 2 indicated a statistical difference between the initial total cholesterol readings of the two groups (p= 0.0039), with no statistical difference being found between the initial ratio readings of the same groups.

TABLE THREE

The mean (x) and standard deviation (SD) of the total cholesterol and HDL /LDL ratio readings after one month of treatment for both the control and experimental groups.

GROUP	TOTAL CHOLESTEROL			HDL / LDL RATIO		
	X1	SD	95 % CI	X2	SD	95 % CI
TREATED	6.583	1.133	0.122;1.751	0.308	0.119	(0.162);0.024
CONTROL	5.651	1.056	0.122;1.751	0.378	0.13	(0.162);0.024

X1= mean total cholesterol reading.

X2= mean HDL /LDL ratio reading.

CI = confidence interval.

()= a negative number.

A significant difference was found between the total cholesterol readings of the experimental and control groups after one month of treatment (p=0.027).

There was, however, no significant difference found between the ratio readings.

TABLE FOUR

The mean (x) and standard deviation (SD) of the total cholesterol and HDL /LDL ratio readings after two months of treatment for both the control and experimental groups.

GROUP	TOTAL CHOLESTEROL			HDL / LDL RATIO		
	X1	SD	95 % CI	X2	SD	95 % CI
TREATED	6.634	1.068	(0.060);1.545	0.324	0.141	(0.151);0.051
CONTROL	5.892	1.078	(0.060);1.545	0.374	0.128	(0.151);0.051

X1= mean total cholesterol readings.

X2= mean HDL / LDL ratio reading.

()= a negative number.

CI= confidence interval.

No significant difference was found between either the total cholesterol or the ratio values of the control and experimental group after two months of treatment.

TABLE FIVE

The mean (x) and standard deviation (SD) of the total cholesterol and HDL / LDL ratio readings on completion of the three month trial for both the control and experimental groups.

GROUP	TOTAL CHOLESTEROL			HDL / LDL RATIO		
	X1	SD	95 % CI	X2	SD	95 % CI
TREATED	6.362	1.141	0.014;1.500	0.377	0.141	(0.140);0.085
CONTROL	5.605	0.818	0.014;1.500	0.364	0.159	(0.140);0.085

X1= mean total cholesterol reading.

X2= mean ratio reading.

()= a negative number.

CI= confidence interval.

A statistical difference was noted on completion of the trial between the total cholesterol readings of the control and experimental groups (p=0.046). However, no statistical difference was noted between the ratio readings of the same two groups.

4.3) FREQUENCY TABLES

TABLE SIX

Frequency table depicting any changes in the total cholesterol values for both the control and experimental groups over the three month trial period.

TOTAL CHOLESTEROL

GROUP	IMPROVED	NO CHANGE	INCREASED	TOTAL
TREATED	12	0	3	15
CONTROL	12	0	3	15

TABLE SEVEN

Frequency table depicting any changes in the HDL / LDL ratio reading for both the control and experimental groups over the three month trial period.

HDL / LDL RATIO

GROUP	IMPROVED	NO CHANGE	DECREASED	TOTAL
TREATED	12	1	2	15
CONTROL	7	1	7	15

4.4) GRAPHICAL REPRESENTATION OF THE RESULTS

FIGURE ONE

The mean total cholesterol readings of the control and experimental groups taken before the start of medication and then once a month for three consecutive months.

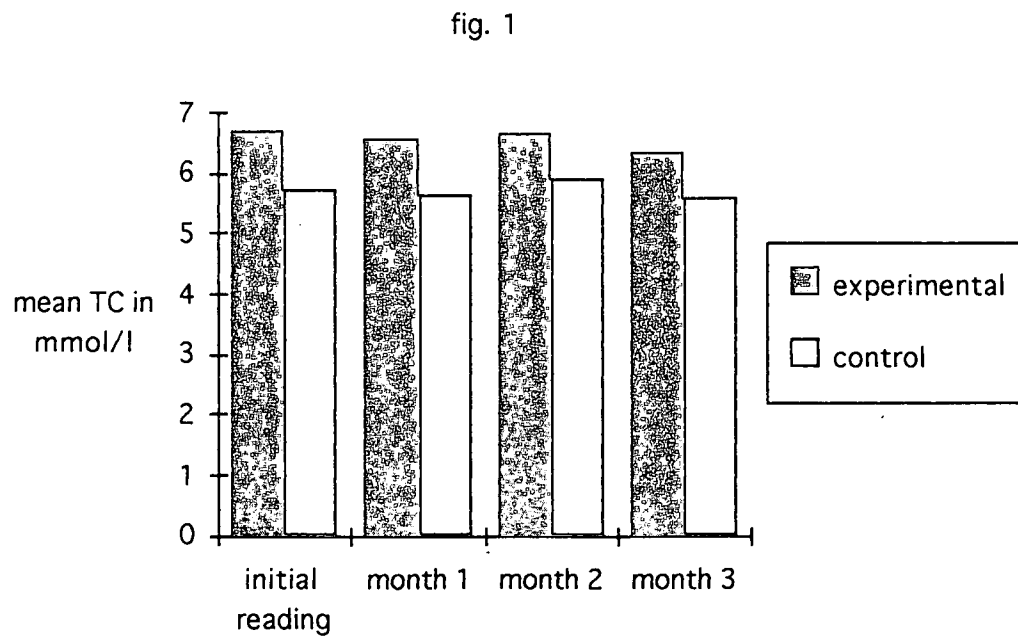


FIGURE TWO

The mean HDL / LDL ratio readings of the control and experimental groups taken once before the start of medication and then once a month for three consecutive months.

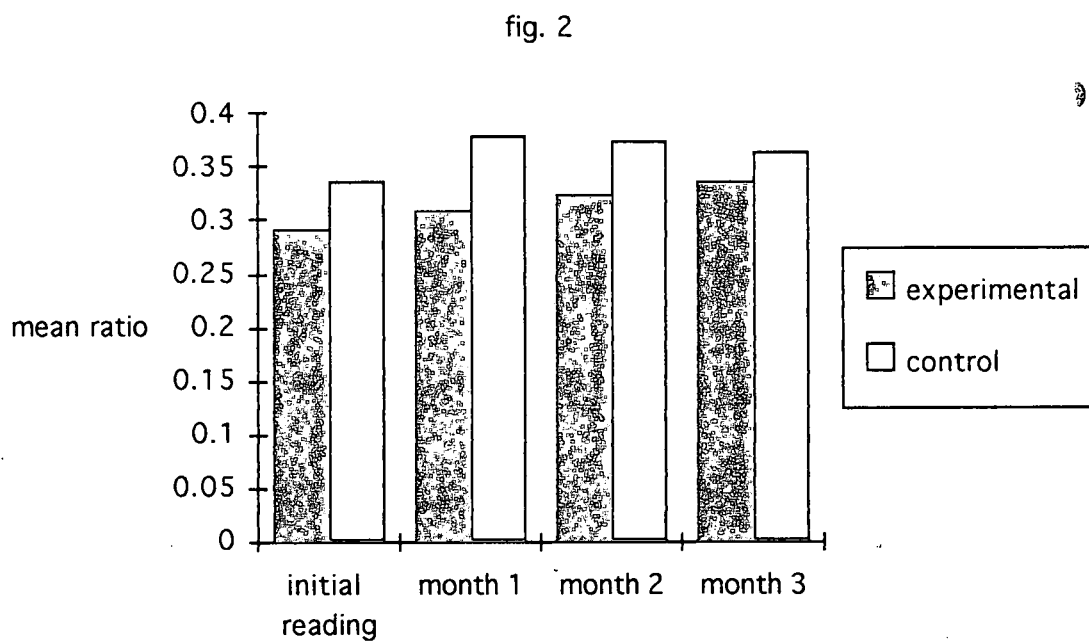
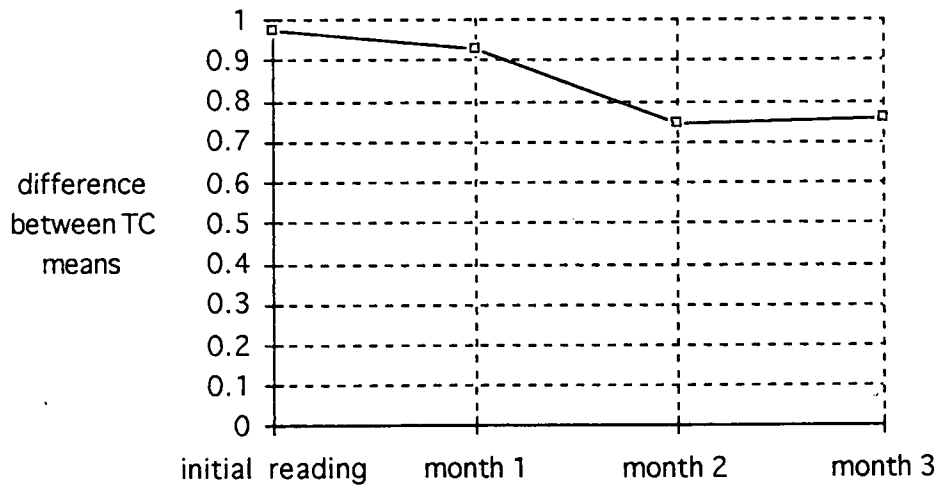


FIGURE THREE

The differences between the means of the total cholesterol for the control and experimental groups taken once before the onset of medication and then once a month for three consecutive months.

fig. 3



CHAPTER FIVE

5.1) EVALUATION OF THE RESULTS

In the attempt to evaluate the effect of Chelidonium 3X in the treatment of hypercholesterolaemia in terms of changes in blood cholesterol levels, the results showed a decrease in the levels of total cholesterol found in the experimental group, with the same group depicting an increase in the ratio value.

The results of the paired T-tests performed on the control group showed no statistical difference between the initial and final total cholesterol readings, or between the initial and final ratio readings (table. 1) after the three month trial.

A small statistical difference was noted between the initial and final total cholesterol readings of the experimental group ($p= 0.023$) with mean total cholesterol readings dropping from 6.684 mmol/l to 6.362 mmol/l which indicates a mean decrease of 4.8 % over the three month trial period (fig. 1). A statistical difference too was found between the initial and final ratio readings of the same group ($p = 0.019$) with a mean increase in the ratio readings from 0.29 to 0.337 which is a 13 % increase (fig. 2).

The unpaired T-tests depicted a statistical difference between the initial total cholesterol readings of the experimental and control groups ($p= 0.0039$), with a mean total cholesterol of 6.684 mmol/l in the experimental group and control mean total cholesterol of 5.711 mmol/l. Thus a 14.6 % difference. Upon

completion of the three month trial period a statistical difference was still noted between the final mean total cholesterol readings of the two groups ($p=0.046$), but it was a smaller difference, with the treated mean total cholesterol being 6.362 mmol/l and the placebo mean total cholesterol being 5.605 mmol /l which was an 11.9 % difference (table. 5). As no statistical difference was found between the initial and final total cholesterol readings of the placebo group we can assume that the mean drop of 2.7% found between the final total cholesterol levels of the two groups indicates a 2.7% improvement in the cholesterol levels of the treated group. This does not seem as favourable as the 25% drop in total cholesterol levels found by Bol et al. (1987) in his trial using rabbits fed a high cholesterol diet for one month and compared to a placebo group on a cholesterol free diet. However, this method is questionable, as normal subjects cholesterol levels are controlled via the body's own homeostatic mechanisms. People with hypercholesterolaemia, among other factors, may have a dysfunction of their normal cholesterol pathways for absorption, metabolism and excretion. Therefore, a drop in cholesterol levels in a normal subject who has artificially induced hypercholesterolaemia should be greater than that of a one that has a physiological abnormality causing increased cholesterol levels. By extrapolation therefore, we should see similar results to Bol et al. (1987) if diet was the only factor causing the raised cholesterol levels in healthy subjects.

It should also be noted that the rabbits in the experimental group were given medication daily. With the human participants in this trial it was not possible to ensure that the medication was taken twice daily as prescribed. However, when questioned on the regular use of the medication as prescribed, only one participant admitted to not taking the medication twice daily as indicated. This

fact may have played a role in the outcome of the trials results.

During the trial period four participants dropped out due to various reasons. None of the participants noted any side-effects of the medication; and one reported that the medication increased bowel regularity.

It is of interest to note that of the treated group three embarked upon an exercise programme (Appendix A, no's 3, 9 & 13). Of the three, two were found to have a small drop in the total cholesterol levels and all had an increase in the ratio readings. Participant no.1 made an attempt to follow the dietary guidelines as set out by the Heart Foundation of Southern Africa and experienced a decrease in the total cholesterol level and an increase in the ratio reading.

In the placebo group two participants lost weight (Appendix B, no's 8 & 13). Both were found to have a decreased total cholesterol level upon completion of the trial with one having an increase in the ratio reading. Participant no. 4 started aerobics and was found to have a decrease in the total cholesterol level and an increase in the ratio reading.

CHAPTER SIX

6.1) CONCLUSION

Experimentally, Chelidonium 3X has been shown to decrease total cholesterol levels and increase the ratio of high density / low density lipoprotein, both of which are of concern in the management of coronary heart disease. However, the margin of decrease in the total cholesterol levels and increase in the ratio levels, does not seem to warrant its use as a mainstay regimen in the treatment of severe hypercholesterolaemia. Its use could lie in patients though who have only a moderately raised cholesterol level, and should probably be used in conjunction with behavior modification. More research needs to be done however, to substantiate the validity of this last statement.

6.2) RECOMMENDATIONS

It is recommended that further trials be done using Chelidonium 3X, but over a longer time period. It would also be of value to note the effect of the above mentioned medicine in conjunction with behaviour modifications such as a healthy eating programme, regular exercise, and in very overweight individuals, the loss of weight.

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APPENDIX A

The results as recorded for both the total cholesterol(TC) and HDL /LDL ratio reading for the experimental group over the three month treatment period.

EXPERIMENTAL GROUP

	TC 1	TC 2	TC 3	TC 4	R 1	R 2	R 3	R 4
1	7.39	5.83	8.28	7.2	0.37	0.43	0.36	0.41
2	5.89	5.8	6.05	4.88	0.26	0.35	0.31	0.36
3	6.99	7.81	7.33	6.8	0.24	0.35	0.36	0.4
4	5.61	5.62	6.38	6.1	0.44	0.42	0.41	0.42
5	8.15	8.54	8.06	8.66	0.19	0.18	0.19	0.17
6	7.85	7.48	6.92	7.53	0.2	0.19	0.22	0.22
7	6.85	6.46	6.79	6.08	0.51	0.59	0.75	0.73
8	7.02	7.28	6.78	6.41	0.27	0.24	0.25	0.27
9	6.62	7.33	6.48	6.96	0.21	0.2	0.25	0.22
10	7	6.45	7.02	6.94	0.3	0.29	0.32	0.28
11	6.63	6.51	6.59	6.5	0.19	0.21	0.21	0.22
12	6.71	7.42	6.4	5.88	0.25	0.21	0.23	0.3
13	7.02	6.98	7.48	6.51	0.18	0.21	0.2	0.21
14	4.28	4.4	4.48	4.03	0.39	0.35	0.4	0.41
15	5.71	4.84	4.48	4.96	0.36	0.41	0.4	0.44

TC 1 = initial total cholesterol reading

TC 2 = reading after 1 month.

TC 3 = reading after 2 months.

TC 4 =reading after 3 months.

R 1= initial ratio reading

R 2 = reading after 1 month.

R 3 =reading after 2 months.

R 4 =reading after 3 months.

APPENDIX B

The results as recorded for both the total cholesterol and HDL / LDL ratio reading for the control group over the three month treatment period.

CONTROL GROUP

	TC 1	TC 2	TC 3	TC 4	R 1	R 2	R 3	R 4
1	5.66	6.88	6	6.59	0.24	0.2	0.29	0.2
2	5.37	5.7	5.94	5.2	0.26	0.28	0.27	0.29
3	6.4	7.13	7.21	6.35	0.27	0.28	0.3	0.34
4	7.6	6.46	7.2	6.51	0.19	0.25	0.22	0.25
5	5.81	5.9	7.13	5.58	0.47	0.64	0.58	0.73
6	5.38	5.89	5.11	5.89	0.29	0.29	0.26	0.23
7	4.53	3.86	3.87	4.6	0.29	0.35	0.37	0.26
8	5.51	5.59	6.18	4.89	0.42	0.36	0.42	.044
9	6.29	6.68	7.18	6.27	0.31	0.25	0.24	0.3
10	5.91	6.2	6.38	6.32	0.48	0.52	0.42	0.39
11	4.68	3.99	4.51	4.6	0.38	0.47	0.41	0.35
12	6.72	5.54	5.73	6.71	0.28	0.37	0.34	0.28
13	4.66	3.84	4.27	4.5	0.27	0.35	0.32	0.24
14	5.29	5.05	5.73	4.71	0.44	0.55	0.54	0.68
15	5.86	6.06	5.95	5.36	0.47	0.51	0.64	0.49

TC 1 = initial total cholesterol reading.

TC 2 = reading after 1 month.

TC 3 = reading after 2 months.

TC 4 = reading after 3 months.

R 1 = initial ratio reading.

R 2 = reading after 1 month.

R 3 = reading after 2 months.

R 4 = reading after 3 months.