The effectiveness of a homoeopathic complex (Caladium seguinum 30CH, Nux vomica 30CH and Staphysagria delphinium 30CH) compared to a tautopathic preparation of the cigarette smoked in the management of nicotine withdrawal syndrome.

By

Catherine Joy Riggien

Dissertation submitted in partial compliance with the requirements for the Master's Degree in Technology: Homoeopathy, in the Faculty of Health at Durban University of Technology

I, Catherine Joy Riggien do declare that this dissertation is a representative of my own work

_____ _____

Catherine Riggien Date

APPROVED FOR FINAL SUBMISSION

_____ _____

Dr D. F. Naude M.Tech: Hom Date

(Supervisor)

Dedication

This dissertation is dedicated to my wonderful children who may not have insured swift work, but who inspired me on a daily basis and to my loving husband, parents, sister and friends for their endless love, patience and support throughout my studies.

Thank you.

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I would like to extend my sincere appreciation to all those who have assisted me, and made this dissertation possible.

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Abstract

Introduction

‘Cigarette smoking is a modern day epidemic that poses a substantial health burden’, it has been proven that smokers die on average fourteen years earlier than non smokers as a direct result of their smoking. An abundance of evidence indicates that the health risks associated with cigarette smoking can however be reversed with a sufficient period of abstinence. Thus achieving life-long abstinence must be a health priority for both developing and developed countries (Caponnetto &, Polosa, 2008).

Over 80% of smokers express a desire to stop smoking and 35% of them try to stop each year. However, less than 5% are successful in un-aided attempts to quit (American Psychiatric Association, 1995).

The greatest challenge facing smokers who wish to quit are nicotine withdrawal symptoms; these include dysphoric or depressed mood, insomnia, irritability, frustration, anger, anxiety, difficulty concentrating, restlessness, decreased heart rate and increased appetite or weight gain (American Psychiatric Association, 1995).

The aim of this double blind placebo controlled quantitative study was to determine the effectiveness of a homoeopathic complex (*Caladium seguinum 30 CH, Nux vomica 30 CH and Staphysagria delphinium 30 CH*); a tautopathic preparation and the combined effect thereof, in the treatment of nicotine withdrawal syndrome as determined by the Tolerance Dependence Questionnaire, Smoking History and Perceptions of Treatment Questionnaire.

Methodology

Forty participants recruited by means of convenience sampling were randomly and equally divided into one of four treatment groups, namely tautopathic group, homoeopathic group, combined tautopathic and homoeopathic group and placebo group. The duration of the study was 2 weeks and two consultations with each participant were conducted.

The respective interventions were administered in oral spray format; participants were asked to spray their respective preparations directly into their mouth three times daily and to repeat the dose whenever they had a craving for a cigarette.

Measurements in the form of the Tolerance Dependence Questionnaire (Appendix D), Perceptions of Treatment Questionnaire (Appendix H), and Smoking History (Appendix G) were used to quantify response to treatment. Non-parametric statistical analysis was conducted to analyse the data.

Results

All four research groups experienced a statistically significant reduction in the amount of cigarettes smoked, favourable perceptions of their response to treatment and improved tolerance. Statistically however when the groups were compared with each other they were similar with respect to their tolerance to nicotine, perception of response to treatment and reduction in amount smoked.

Although interventions were statistically similar in terms of effectiveness, the data does suggest that Tautopathy as an intervention warrants exploration. The Tautopathic group achieved the highest reduction in the number of cigarettes smoked when comparing medians (11 less smoked per day), achieved the highest percentage of participants who experienced reduced cravings, and the highest percentage of participants who would continue using the intervention (90% respectively) as well as improvements in 6/9 variables of the Tolerance Dependence Questionnaire.

Conclusions

The study concludes that each of the four subject groups (including placebo) proved to be successful in aiding the participant to cease smoking. The results showed a significantly positive perception of the participants to the interventions used. The influence of the placebo effect however was very evident in this study; in addition other factors such as the unique method of administration of the medication (oral spray format on demand) the Hawthorn effect and the participants' desire/commitment to quit smoking may have contributed to the positive results obtained. Notwithstanding the above and although not statistically significant; positive trends within the data do suggest that the Tautopathic approach used in this study should be further investigated in future.

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Definition of Terms

Addiction: the fact or condition of being addicted to a particular substance or activity (Oxford Dictionaries, 2010).

Dependence: the state of relying on or being controlled by someone or something, addiction to drink or drugs (Oxford Dictionaries, 2010).

Hawthorne effect: the alteration of behaviour by the subjects of a study due to their awareness of being observed (Oxford Dictionaries, 2010).

Homoeopathy: a system of complementary medicine in which ailments are treated by minute doses of natural substances that in larger amounts would produce symptoms of the ailment. Often contrasted with Allopathy(Oxford Dictionaries, 2010).

Placebo: a substance that has no therapeutic effect, used as a control in testing new drugs (Oxford Dictionaries, 2010).

Placebo effect: a beneficial effect produced by a placebo drug or treatment, which cannot be attributed to the properties of the placebo itself, and must therefore be due to the patients belief in that treatment (Oxford Dictionaries, 2010).

Tautopathy: a practice of alternative medicine that is similar to homoeopathy in that it uses very diluted substances to treat illness, However, Tautopathy does not rely on the law of similars as homoeopathy does. According to tautopathic practitioners, dilute solutions of lead and arsenic can cause the body to secrete excess amounts of these toxic metals. Also according to advocates, a patient exhibiting negative side effects from conventional medication could be treated by a dilute solution of the same medication, to give the same healing effect while lessening the side effects (Bhatia, 2006).

Potency: the number of times a remedy has been diluted and sucessed, taken as a measure of the strength of the effect it will produce: "*she was given a low potency twice daily".*

Homoeopathic complex: a system of homoeopathic medicine that has more than one remedy combined together in one dosage form (The Free Dictionary.com).

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Chapter one

1.1 Introduction

According to the World Health Organization (2002); smoking is the single largest preventable cause of death, being a prime factor in chronic lung disease, a range of cancers, stroke and heart disease. Half of long term smokers are predicted to die from tobacco, if the existing trends continue it is proposed that smoking will kill one in six people, with each cigarette reducing predicted lifespan by about five minutes.

‘Cigarette smoking is a modern day epidemic that poses substantial health burden’, it has been proven that smokers die on average fourteen years earlier than non smokers as a direct result of their smoking. An abundance of evidence indicates that the health risks associated with cigarette smoking can in fact be reversed with a sufficient period of abstinence. Thus achieving life-long abstinence must be a health priority for both developing and developed countries (Caponnetto, Polosa, 2008).

Over 80% of smokers express a desire to stop smoking and 35% of them try to stop each year. However, less than 5% are successful in un-aided attempts to quit (American Psychiatric Association, 1995).

The need to manage tobacco addiction and the need for help in doing so is thus apparent and thus the rationale for the conducting of this particular study.

The greatest challenge facing smokers who wish to quit are nicotine withdrawal symptoms; these include dysphoric or depressed mood, insomnia, irritability, frustration, anger, anxiety, difficulty concentrating, restlessness, decreased heart rate and increased appetite or weight gain (American Psychiatric Association, 1995).

The individual remedies comprising the experimental homoeopathic complex used in this study were chosen specifically to match and thus treat the effects of the nicotine withdrawal syndrome.

1.2 Aim of the study

The aim of this double blind placebo controlled study is to determine the effectiveness of a homoeopathic complex (Caladium seguinum 30 CH, Nux vomica 30 CH and Staphysagria delphinium 30 CH); a tautopathic preparation and the combined effect thereof, in the treatment of nicotine withdrawal syndrome as determined by the Tolerance Dependence Questionnaire, Perceptions of Treatment Questionnaire and Smoking History.

1.3 Objectives

1.3.1 Objective one

To determine the effectiveness of a tautopathic preparation of the combusted cigarette smoked (6CH) in managing nicotine withdrawal syndrome as determined using a Tolerance Dependence Questionnaire, Perceptions of Treatment Questionnaire and Smoking History.

1.3.2 Objective two

To determine the effectiveness of the homoeopathic complex (Caladium seguinum 30CH, Nux vomica 30CH, Staphysagria delphinium 30CH) in managing nicotine withdrawal syndrome as determined using a Tolerance Dependence Questionnaire, Perceptions of Treatment Questionnaire and Smoking History.

1.3.3 Objective three

To determine the combined effectiveness of a tautopathic and the homoeopathic complex in managing nicotine withdrawal syndrome as determined using a Tolerance Dependence Questionnaire, Perceptions of Treatment Questionnaire and Smoking History.

1.3.4 Objective four

The fourth objective is to compare the effectiveness of the tautopathic preparation, the homoeopathic complex and the combined approach (tautopathy and homoeopathic complex) with that of the placebo, in the management of nicotine withdrawal syndrome as determined using a Tolerance Dependence Questionnaire, Perceptions of Treatment Questionnaire and Smoking History.

1.4 Statement of hypotheses

1.4.1 The first hypothesis

It was hypothesised that the tautopathic preparation would be effective in reducing the number of cigarettes smoked daily and the effects of nicotine withdrawal symptoms as measured by the Tolerance Dependence Questionnaire, Perceptions of Treatment Questionnaire and Smoking History.

1.4.2 The second hypothesis

It was hypothesised that the homoeopathic complex (Caladium seguinum 30CH, Nux vomica 30CH, Staphysagria delphinium 30CH) would be effective in reducing the number of cigarettes smoked daily and the effects of nicotine withdrawal syndrome as measured by the Tolerance Dependence Questionnaire, Perceptions of Treatment Questionnaire and Smoking History.

1.4.3 The third hypothesis

It was hypothesised that a combination of the tautopathic preparation and the homoeopathic complex (Caladium seguinum 30CH, Nux vomica 30CH, Staphysagria delphinium 30CH) would be effective in reducing the number of cigarettes smoked daily and the effects of nicotine withdrawal syndrome as measured using the

Tolerance Dependence Questionnaire, Perceptions of Treatment Questionnaire and Smoking History.

1.4.4 The fourth hypothesis

It was hypothesised that the tautopathic preparation, the homoeopathic complex (Caladium seguinum 30CH, Nux vomica 30CH, Staphysagria delphinium 30CH) and the combined approach would be superior to placebo in reducing the number of cigarettes smoked daily and the effects of nicotine withdrawal syndrome as measured by Tolerance Dependence Questionnaire, Perceptions of Treatment Questionnaire and Smoking History.

1.4.5 The fifth hypothesis

It was hypothesised that the combined approach (the tautopathic preparation and the homoeopathic complex in combination) would be superior to the tautopathic and homoeopathic complex (alone) in reducing the number of cigarettes smoked daily and the effects of nicotine withdrawal syndrome as measured by the Tolerance Dependence Questionnaire, Perceptions of Treatment Questionnaire and Smoking History.

Chapter two

Review of Related literature

2.1 Historical perspective on smoking

The tobacco plant used to make cigarettes began growing in the Americas as early as 6000 BC; however inhabitants only started utilising these plants in the form of enemas and smoking in 100 BC. In the 12th century pots were found depicting a man smoking a roll of tobacco leaves fastened with string. It was in 1492 that the developed world was first exposed to the act of smoking by Columbus and tobacco was introduced into England in 1564 by Sir John Hawkins (Lewis, 2007).

It was in 1603 that physicians complained about tobacco for the first time, at that stage they were upset due to the fact that tobacco was being used without their prescription and their concerns were brought it to the attention of King James the 1st. King James was the first person formally recorded who disapproved of tobacco and in 1604 wrote “A Counterblaste to Tobacco” saying it is “loathsome to the eye, hateful to the nose, harmful to the brain, and dangerous to the lungs”. Formal punishment for the selling or smoking of tobacco began in 1628 with Shah Sefi pouring hot lead down the throats of merchants who sold tobacco. Sultan Murad 4th of Turkey ordered tobacco users to be executed as infidels in 1633. A year later in 1634 Czar Alexis of Russia formulated strict punishment regimes for smokers, with a first offence being punishable by whipping, a slit nose, and transportation to Siberia, and the second offence being execution. Use or distribution of tobacco was made punishable by decapitation in China in 1638. In 1650 a new law was passed by the Colony of Connecticut General court that prohibited smoking unless it was conducted with a physicians order. In 1701 Nicholas Boisregard warned young people of the symptoms of tobacco use, such as trembling and unsteady hands, staggering feet and a withering of their noble parts. The first mention of the causal relationship between tobacco and cancer occurred in 1761, when Dr John Hall performed the first clinical study of the effects of tobacco in snuff users and their vulnerability to cancer of the nose. At the same time Dr Percival Pott theorised with regard to the connection between soot exposure and cancer of the scrotum as noted in many chimneysweeps at that time. Thirty five years later in 1795 Samuel von

Soemmering shared his findings of cancer of the lip in pipe smokers. The first formal sale of cigarettes took place in 1847 when Phillip Morris opened a tobacconist shop; smoking became even more convenient with the introduction of matches in 1852. Between 1853 and 1856 British soldiers involved in the Crimean War learned of the cheap and convenient cigarettes used by their Turkish allies and subsequently brought the practice back to England. In the United States the mass production of cigarettes began in 1884 when Duke and Sons bought two cigarette rolling machines and began producing 744 million cigarettes per year and subsequently consolidated their rivals into the American Tobacco Company. To combat this American take-over British tobacco companies united to form the Imperial Tobacco Group in 1901. One year later these two companies agreed to remain in their own respective countries but to unite to form the British American Tobacco Company (Lewis, 2007).

After many years of silence, the effects of smoking resurfaced when Dr I Adler suggested that smoking was related to lung cancer in 1912. In 1928 a Journal article in the New England Journal of Medicine, by Lombard and Doering reported on 217 deaths due to cancer, and that 34 out of 35 site-specific cancer sufferers such as those suffering from cancers of the lung, lips, cheek and jaw were heavy smokers. The sales of cigarettes were at an all time high between 1939 and 1945 at end of the Second World War. In 1941 Dr Michael DeBakey noticed and reported on the increase of the sale of tobacco and the subsequent increase in lung cancer in the US. In 1950 two epidemiological studies highlight some very important facts, linking smoking to lung cancer; Journal of American Medical Association (JAMA) found that 96.5% of patients suffering from lung cancer were moderate to heavy-to-chain smokers and Doll and Hill reported in the September 30 edition of The British Medical Journal that heavy smokers were 50 times more likely to suffer from lung cancer. In 1953 evidence of the first definitive biological link between smoking and cancer was produced when Wynder induced tumour growth on mice by painting cigarette tar on their backs. In 1954 mass action was taken in an attempt to warn people about the health hazards of cigarette smoking. This action occurred in response to a two page advertisement which was placed in 448 newspapers and a booklet that was published by the Tobacco Industry Research Committee. This advertisement destined to reach 43 million people, quoted cancer statistics, while the

booklet which was published was distributed to 176 800 doctors quoting 36 scientists who questioned the assumed link of smoking to health problems (Lewis, 2007).

Despite all the evidence highlighting the consequences of smoking, the Marlboro Cowboy was created in 1954. Three years later in 1957 an internal report by the British tobacco industry referred to cancer by a code name, Zephyr and stated the following; “as a result of several statistical surveys, the idea has arisen that there is a causal relation between Zephyr and tobacco smoking, particularly cigarette smoking”. In 1962 in the UK the first report of the British Royal College of Physicians (RCP) of London called, “Smoking and Health” was published. Three years later cigarette advertisements on TV were taken off the air in the UK, and a year later in 1966 health warnings began to appear on cigarette packs. In 1967 the Surgeon General of the United States reported a link between smoking and heart disease and concluded that smoking was the principal cause of lung cancer. In 1971 a second RCP report which likened the cigarette death toll as “this present holocaust” and a cigarette smoking and health report by an interdepartmental parliament group concluded “all things considered, tobacco use brings in more money than it costs in health and disability”, this report was undisclosed to the public until May 6, 1980 when the Guardian newspaper published it accordingly (Lewis, 2007).

In 1984 the first Nicotine replacement therapy was approved by the FDA (Food and drug administration) in the form of nicotine gum and was called the “new drug” and quit smoking aid. In 1988 the World Health Organisation sponsored the first No-Tobacco Day. In 1992 the nicotine patch was introduced as another form of nicotine replacement therapy in an attempt to aid people to quit smoking. In this same year the famous Marlboro Man, Wayne Mclaren died of lung cancer at the age of 51. In 1994 seven tobacco company executives began testimonies in congressional hearings. In the 1995 July issue of JAMA, we find a report devoted to tobacco papers that found unequivocal evidence that the US public had been deliberately misled by the tobacco industry and the author believed that all citizens should be outraged and force the removal of “this scourge from our nation...” In 1995 the second Marlboro Man, David Mclean died of lung cancer. In 1996 Science Magazine published an article that showed that benzo(a)pyrene a derivative in cigarette tar damages the p53 suppressor gene. In 2004 the city of New York published 1 year results of its smoking ban quoting unheralded success, global cigarette production

declined by 2.3%, the lowest since 1972, and at that stage China produced 32% of the worlds cigarettes, while in the UK Wanless Report concluded that cutting the smoking of cigarettes was key to success, in meeting the Government's public health targets and that the National Health Services needed to concentrate its efforts on smoking prevention (Lewis, 2007).

In 2006 in Scotland ban on smoking in enclosed public spaces was implemented, and one year later in 2007 the National Health Service is officially smoke free, with bans in Wales, Northern Ireland and England, and the legal minimum age to purchase cigarettes was raised to 18 (Lewis, 2007).

2.2 Development of South African legislation on smoking

The legislation of tobacco/smoking in South Africa has undergone significant changes; the 1993 Tobacco Products Control Act was amended and replaced by the Tobacco Control Act of 1999, and in 2006 amended and replaced by the current Tobacco Control Amendment Act of 2006 (Tobacco Products Control Amendment Bill, 2006).

These recent changes in the smoking legislation limit outdoor smoking, protect children from secondary smoke and decrease the risks of fires caused by burning cigarettes (Langa, 2009).

The changes include an increase in fines for smoking in prohibited areas such as partially enclosed areas e.g. patios or walkways, smoking in vehicles where a child under the age of 12 is a passenger and smoking in any area including private homes used for the purpose of child care facilities, schooling or tutoring. In addition it is now prohibited for anyone under the age of 18 to be allowed into a smoking area, this includes parents taking babies into designated smoking areas (Langa, 2009).

There have also been changes in the anti-smoking legislation that affect the marketing of tobacco products; the tobacco industry may not hold parties marketed towards young people and prohibition of using the internet or cell phone marketing to overcome the ban on tobacco advertising in the media which came into effect in 2000 was implemented. The sale of tobacco products is prohibited to anyone under

the age of 18 as is the sale of sweets or toys that resemble tobacco products. Cigarette vending machines may only be placed in areas where minors may not have access and may also not be used to sell any other products such as sweets, chips or soft drinks (Langa, 2009).

There are more positive changes in the Tobacco Control Act to be expected in the near future, these include; the use of vivid picture based health warnings on tobacco based product packaging, further restrictions on smoking areas, the introduction of new self- extinguishing cigarettes, the misleading terms “mild” and “low tar” will no longer be permitted, and manufacturers will need to disclose all the harmful additives used in the manufacture of their tobacco products (Langa, 2009).

2.3 Smoking constituents

Mainstream cigarette smoke has 4700 identified constituents and an estimated 100 000 unidentified components. As many of these constituents appear in miniscule concentrations, it has become useful to categorize the complex cigarette smoke into four parts; the first part consists of carbon monoxide, the second any other vapour phase components such as acetaldehyde, acrolein, nitrogen oxides, formaldehyde and carbon dioxide, the third part consists of tar which is the residue one finds in the cigarette filter apart from the nicotine and water, and the fourth part consists of the particulate phase or nicotine. These four components of cigarette smoke are simultaneously delivered to the active smoker as a complex aerosol composed of combustion gasses and semi-liquid particles (Smith, Fischer, 2001).

Tobacco contains an estimated 2500 different chemical compounds; cigarette smoke on the other hand contains over 4000 compounds, thus several compounds are generated by the mechanisms such as combustion, distillation and pyrolysis which make up the action of active cigarette smoking i.e.: the burning of the tobacco (Baker,1987; Newhouse, Singh, Potter, 2004a; Frishman, Mitta, Kupersmith, Ky, 2006). The exothermic combustion zone which is easily visible at the end of an ignited cigarette ranges in temperatures from 700-950 degrees centigrade, below that is the distillation and pyrolysis zone which has a lower temperature ranging from

200-600 degrees centigrade which is where most ingredients or compounds are formed during endothermic reactions (Baker, 1987).

The individual composition of cigarette smoke is also influenced by many factors that differ between the different brands of cigarettes, such as the type and composition of the paper and filter, composition of the tobacco, length of the cigarette (Frishman et al., 2006). The added flavourings such as vanillin and bergamot oil are used to mask the odour, irritation effects and visibility of the environmental smoke. Theobromine and caffeine enhance bronchodilation and increase the bio availability of the nicotine by regulating the ph, while levulinic acid, ammonia and sugars temper the harsh feeling of the smoke. These additives and flavourings can add up to 10% of the total weight of the cigarette (Talhout, Opperhuizen, van Amsterdam, 2006; Rabinoff, Caskey, Rissling,Park, 2007; Connolly, Wayne, Lymperis, Doherty, 2000; Keithly,Ferris Wayne, Cullen, Connolly, 2005; Hammond, Collishaw, Callard, 2006; Henningfield, Pankow, Garrett, 2004) and serve to make each brand slightly different from the next although the main ingredients are the same.

Although the cigarette smoke has four different parts it can also be defined and further simplified into the gaseous and particulate phases; the gaseous phase contains ammonia, nitrates, alcohols, ketones (acetone, butanone), volatile sulphur containing compounds (hydrogen sulphide), hydrocarbons, aldehydes (formaldehyde and acetalhyde), inter alia nitrogen oxide, carbon monoxide, carbon dioxide, free radicals and other oxidants such as hydrogen peroxide; which has been theorised to have a role in airway tumorgenesis, superoxide anion and peroxynitrite (Frishman et al.,2006; Zevin, Benowitz, 1999; Khan, Lanir, Danielson, Goldkorn, 2008; Swan, Lessov-Schlaggar, 2007; Hatzinikolaon, Lagesson, Stavridou, Pouli, Lagesson-Andrasko, Stavrides, 2006; Pryor, Stone 1993).

The particulate phase consists of tar which consists of several radioactive compounds, free radicals, metallic ions and polycyclical aromatic hydrocarbons, water and alkaloids (Frishman et al., 2006; Zevin, Benowitz, 1999) of which nicotine is the most abundant at approximately 95% (Cai, Liu, Lin, Su, 2003) and can easily and rapidly cross the blood brain barrier and has a high affinity for organs such as the liver, spleen, kidneys and lung, with minor alkaloids being anatabine, anabasine and nornicotin (Dome, Lazary, Kalapos, Rimer, 2009).

The whole tabaccum plant is poisonous and the leaves which are used to make cigarettes are the most dangerous. Ingestion of this plant can cause anxiety, irritability, confusion, dizziness, drowsiness, nausea, vomiting, loss of appetite, cough, palpitations and an irregular pulse (Vermeulen, 2002).

2.4 Smoking Addiction and Nicotinic effects on the body

According to the Diagnostic and Statistical Manual of Mental Disorders (1995), over 80% of individuals who smoke, express a desire to stop smoking and 35% of them try to stop each year. However, less than 5% of those who attempt to stop smoking un-aided are successful.

Nicotine is proven to be the main substance responsible for habitual smoking; the molecular mechanisms responsible for the reinforcing properties of nicotine are mirrored by those underlying the reinforcing properties of other psychostimulants (Picciotto, 1998).

There is much evidence to demonstrate the reinforcing properties of nicotine, the simplest of which is an experiment whereby rodents will choose to drink a solution of nicotine and water over water alone (Smith, Roberts, 1995; Glick, Visker, Maisonneuve, 1996; Robinson, Marks, Collins, 1996). The behavioural sensitization as seen in nicotine challenges in locomotor activity tests can be observed in other known psychostimulants, such as amphetamine and cocaine and are believed to be responsible for the development of addictive behaviour (Kalivas, Sorg, Hooks, 1993; Robinson, Berridge, 1993).

The biochemical response to both nicotine and cocaine, namely the release of various neurotransmitters such as acetylcholine, dopamine, glutamate, GABA, norepinephrine and serotonin indicates similar mechanisms are involved in the development of dependence to these drugs of abuse. These addictive properties include those of reinforcement, anxiolytic effects, reinforced motor patterns and stimulation of reward centres within the brain (Picciotto, 1998).

Active cigarette smoking is the most effective form of nicotine intake, being more bio available at 80-90 % than other forms of nicotine replacement therapy such as

nicotine containing gum, lozenges, sprays and patches. Cigarette smoking is also a much faster method of nicotine delivery with high levels of nicotine reaching the brain 10-20 seconds after taking a puff, this is even faster than venous delivery (Hukkanen, Jacob, Benowitz, 2005), the reason for this effective delivery system can be attributed to the rapid perfusion of the nicotine into the large blood perfused alveoli which have a large surface area in the lungs (Brewer, Roberts, Rowell, 2004). Routes of drug administration like this rapid delivery of nicotine to the brain that have an added effect of inducing euphoria and self reports of great pleasure increase the propensity of that drug to induce addiction (Samaha, Yan, Yang, Robinson, 2005a; Samaha, Robinson, 2005b).

An active smoker is capable of regulating their own nicotine intake by adjusting the way they smoke, namely the puff volume, inter-puff interval, puff velocity and the number of puffs per cigarette (Hammond et al., 2006) thus inducing the desired effects of smoking.

Many recent studies have suggested that cigarette smoking is reinforced by non nicotine chemical, environmental and behavioural factors, not only the addictive nature of nicotine (Rose, 2006; Samaha et al., 2005a; Samaha, Robinson, 2005b; Talhout, Opperhuizen, von Amsterdam, 2007). These findings have led to the hypothesis that there are other addictive ingredients within cigarettes besides nicotine.

What adds to the addictive nature of the cigarettes is the pleasure that the smoker gains whilst smoking, as well as the psychosocial factors that also play a role in the continued use of cigarettes, despite the known harmful effects of smoking (Waxler, 2006).

2.5 Smoking and Alcohol

There is an undeniable association between heavy alcohol use and cigarette smoking, with approximately 80% of those that are alcohol dependent being smokers (Romberger, Grant, 2004).

Despite a decrease in the number of smokers in general, more than 60% of people seeking treatment for substance abuse are active smokers (Kohn, Tsoh, Weisner, 2003), with heavy smoking being a predictor of unrecognised alcohol dependence and abuse (Kozlowski, Jelinek, Pope, 1986).

It has been extensively researched and proven that cigarette smokers drink more alcohol, more often compared to non smokers (Anthony, Echeagaray-Wagner, 2000; Chiolero, Wietlisbach, Ruffieux, Paccaud, Cornuz, 2006; Dawson, 2000; Falk, Yi, Hiller-Sturmhofel, 2006; Kahler, Strong, Papandonatos, Colby, Clark, Boergers, Niaura, Abrams, Buka, 2008b).

The significance of this concomitant use of alcohol and cigarettes is of concern for public health due to the multiplicative effect these combined behaviours have on the greater risks of such individuals in developing oral, pharyngeal, laryngeal and oesophageal cancers (Pelucchi, Gallus, Gravello, Bosetti, La Vecchia, 2006; Romberger, Grant, 2004). Men who drink alcohol and smoke cigarettes are 38 times more likely to develop head and neck malignancies than men who neither drink alcohol or smoke cigarettes. Non smoking men who drink alcohol are 5.8 times more likely to develop these cancers while a non-drinking smoker is 7.4 times more likely to develop head and neck cancers (Romberger, Grant, 2004).

Combined use of cigarettes and alcohol has also been reported to have a synergistic influence on the biological processes leading to hepatocellular, pancreatic, oesophageal and laryngeal cancers, and increasing the risk of secondary tumours in patients with existing upper aeorodigestive tract malignancies (Romberger, Grant, 2004).

The risk of developing non-malignant diseases such as cirrhosis of the liver, periodontal disease, pancreatitis and cardiovascular disease may also be influenced by the combined use of alcohol and cigarettes (Romberger, Grant, 2004; Schroder, Marrugat, Elosua, Covas, 2002).

Tobacco is indicated as the leading cause of death in those previously treated for alcohol dependence, not alcohol as one would expect (Hurt, Offord, Croghan, Gomez-Dahl, Kottke, Morse, 1996), thus demonstrating the further health risks of

smoking even in those suffering the effects of heavy alcohol use (Romberger, Grant, 2004).

Moderate alcohol consumption has been linked to a few health benefits, such as a positive effect on cardiovascular disease, dementia and hearing loss. These effects are lost if the moderate alcohol consumption is combined with cigarette smoking, or if the intake of alcohol becomes more than moderate (Romberger, Grant, 2004; Schroeder et al, 2002).

With all the recent evidence highlighting the prevalence of cigarette smoking among patients in alcohol treatment programmes and the detrimental effects of combined drinking and smoking on these patients health, it becomes clear that establishing optimal approaches to smoking cessation in alcohol dependence treatment is essential (Romberger, Grant, 2004).

2.6 Smoking, Health and Psychology

2.6.1 Chronic Obstructive Pulmonary Disease

The incidence of chronic obstructive pulmonary disease (COPD) is on the rise and it is predicted that it will be the third leading cause of death worldwide by 2020 (Tashkin, Murray, 2009). COPD often severely limits the day to day activities of the patient impairing their health related quality of life (Rennard, Decramer, Calverley et al, 2002), and the resultant morbidity and mortality exerts a substantial burden on our economy and healthcare system (National Heart Blood and Lung Institute, 2007).

COPD may present itself with, exertional dyspnoea which is a limitation of physical activity which may not become evident until the progression of the airflow obstruction is significantly advanced. COPD is categorised into the following stages by the Global Initiative for Chronic Obstructive Lung Disease (GOLD); mild, moderate, severe and very severe based on spirometry values measuring the ratio of forced expired volume in 1 second to forced vital capacity. A value of less than 70% that does not increase to greater than 70% after the use of a bronchodilator is indicative of COPD, while a value of less than 80% is considered Mild COPD, 50-79% is considered to be moderate COPD, 30-49% is considered to be severe COPD and a

value of less than 30% is considered to be indicative of severe COPD (Tashkin, Murray, 2009).

COPD is often under-diagnosed and is subject to under recognition and subsequent under treatment, with COPD being mainly defined on the basis of its physiology and effect on air flow limitation within the lungs that is not fully reversible, it is primarily diagnosed using spirometry. This progressive limitation of airflow within the lung is associated with an abnormal inflammatory response within the lung to noxious gases and particles, such as those resulting from cigarette smoking (Tashkin, Murray, 2009).

The main risk factor associated with COPD is cigarette smoking, with up to approximately 90% of fatalities due to COPD being directly attributed to cigarette smoking (Anthonisen, Connet, Kiley,et al, 1994), the most effective means of slowing down or halting COPD is smoking cessation (Anthonisen et al, 1994; Scanlon, Connett, Waller, Altose, Bailey, Buist, 2000). Ironically, 30%-43% of those with moderate to severe COPD continue to smoke (Calverley, Anderson, Celli et al, 2007; Tashkin, Celli, Decramer et al, 2005; Wedzicha, Calverley, Seemungal, Hagan, Ansari, Stockley, 2008).

Smoking cessation is the most effective means of slowing down the progression of COPD and given that a third or more of these sufferers continue to smoke it is evident that a more effective intervention for smoking cessation is needed especially in patients suffering from serious conditions like COPD (Tashkin, Murray, 2009).

2.6.2 Pneumococcal disease

Recent studies show an alarming increase in hospitalization and mortality due to pneumococcal disease over the last decade. Cigarette smoking is the strongest independent risk factor for developing pneumococcal disease (Nuorti, Buttler, Farley, Harrison, McGeer, Kolczak, Breiman, 2000).

Smoking can irritate the respiratory tract and result in immune-suppression, predisposing the smoker to such infections as pneumococcal infections among others (File, 2000; Murin, Biello, Matthay, 2000).

Prevention of these pneumococcal infections especially in high risk groups of individuals such as smokers is becoming of the utmost importance as the rate of morbidity and mortality increases. Physicians and primary health care practitioners should therefore warn smokers of the risk of development of pneumococcal disease and highlight the importance of smoking cessation (Looijmans-van den Akker, van den Heuvel, Verheij, van Delden, van Essen, Hak, 2007).

2.6.3 Age-related maculopathy and age-related macular degeneration

Age-related maculopathy (ARM) and the associated age-related macular degeneration (AMD) are degenerative disorders of the central portion of the eye; they are the leading causes of impaired vision and subsequent blindness (Chopdar, Chakravarthy, Verma, 2003).

ARM is characterised by yellow deposits under the pigmentation epithelium of the retina, while AMD is characterised by areas of atrophy, choroidal neovascularisation, detachment of pigment epithelia called dry AMD or disciform scarring known as exudative or wet AMD (Gottlicb, 2002).

Smoking is the most modifiable risk factor associated with ARM and AMD. Active smoking increases this risk by reducing the serum antioxidant levels which this in turn may reduce the antioxidant enzyme levels resulting in greater oxidative damage, a reduction in choroideal blood flow to the eyes, promoting hypoxia and increasing the susceptibility of the macula to degenerative changes (Evans, 2001).

2.6.4 Arteriosclerosis and Coronary Heart Disease

Smoking has a substantial negative impact on world health and has been proven to have a causal effect to many forms of cardiovascular disease which result in death and morbidity; such as myocardial infarction, stroke, coronary artery disease, and peripheral artery disease among others (Jockel, Lehmann, Jaeger, Moebus, Mohlenkamp, Schmermund, A., Dragano, Stang, Gronemeyer, Seibel, Mann, Volbracht, Siegrist, Erbel, 2009).

Cardiovascular diseases have many risk factors such as genetic predisposition, blood pressure, cholesterol, diabetes, age, and weight with smoking being one of the most modifiable; this makes smoking cessation a key issue for the prevention of these cardiovascular diseases. It takes approximately 2-30 years for a smoker who quits smoking to regain the status of a non-smoker or never smoker in terms of the risk of coronary heart disease. Even if a long term smoker ceases to smoke they may add about 6 years onto the ageing progression of their coronary arteries, leading to major ischemic heart disease events even 20 years after of giving up smoking (Jockel et al., 2009).

Research has described a definite link between smoking and coronary calcium burden, which indicates subclinical coronary atherosclerosis and the disposition to coronary heart disease (Jockel et al., 2009).

Nicotine in the bloodstream due to cigarette smoking, triggers platelet activation, fibrogen production and provokes endothelial cell damage, this causes vascular inflammation and leads to an increase in plasma fibrogen (Mitsiakos et al., 2008).

Arteriosclerosis is associated with both coronary artery calcium accumulation and an increase in fibrogen production. The excess fibrogen production decreases after smoking cessation occurs with only the acquired coronary artery calcium burden persisting, causing the premature ageing of these arteries (Jockel et al., 2009).

2.6.5 Smoking and Pregnancy

Maternal smoking during pregnancy is associated with many adverse outcomes of pregnancy, such as low birth weight, attention deficit hyperactivity disorder, conduct disorder, offspring smoking, cognitive dysfunction (Knopik, 2009), placental abruption and placenta previa (Ananth et al., 1996; Higgins, 2002; Mortensen et al., 2001).

Tobacco, the main ingredient in cigarettes and many of its 3000 compounds are able to cross the placental barrier causing multi organ foetal damage, with an increase risk of perinatal morbidity and mortality as well as an elevated risk of intracranial haemorrhage in preterm infants (Mitsiakos et al., 2008).

Surprisingly evidence exists that smoking reduces the risk of pre-eclamsia although the exact cause for this protective mechanism is unclear (Cnattingius et al., 1997), suggestions include effects of nicotine and other substances in tobacco smoke on the placental vasculature (Conde-Agudelo et al., 1999), an increase in the amount of trophoblasts (Larsen et al. 2002), a less expanded plasma volume or the hypotensive effect of the thiocynate found within tobacco (Castles et al., 1999). However should pre-eclampsia develop; heavy smoking is associated with placental abruption, small gestational age and a greater risk of perinatal death (Cnattingius et al., 1997).

Smoking whilst pregnant is associated with many known socio-demographic and health behavioural risks, namely; unmarried status (Luo et al., 2004), young maternal age (Jolly et al., 2000), lower social status with alcohol consumption (Hannigan, Armant, 2000), low level of education (Rutter, Quine, 1990), and previous pregnancy terminations (Ancel et al. 2004).

It has been proven that reducing the number of cigarettes smoked daily during pregnancy is likely a protective mechanism against the known risks of maternal smoking whilst pregnant, such as growth restriction, low birth weight and small gestational age infants and infants born with low Apgar scores (Raatikainen, Huurinainen, Heinonen. 2006).

2.6.6 Smoking and Hip Fracture

The micro vascular effects of nicotine cause a reduction in tissue perfusion, which if prolonged is implicated as a contributing factor leading to osteoporosis and fracture occurrence. Smokers at the age of 60 years have a 17% increase in the risk of suffering a hip fracture, while 70 year old smokers have a 41% increase in risk of hip fracture and at 80 years old a 71% increase in risk of suffering a hip fracture. One hip fracture in eight is directly related to smoking. Smoking is also a significant risk factor for complications after arthroplasty of the hip or knee leading to increase in risk of admission into intensive care unit and a longer hospital stay. Smoking has also been proven to reduce the age at which one would sustain a hip fracture, with smokers being on average nine years younger than non smokers when they sustain

a hip fracture, this has been explained to be due to the physiological ageing effects of smoking on the tissues (Johnston, Gurusamy, Parker, 2005).

2.6.7 Smoking and Cancer

Cancer as a result of smoking has been a well documented, with tobacco smoke being proven to be carcinogenic to the lung as early as 1950 (Doll, Hill, 1950; Wynder, Graham, 1950).

Tobacco smoking is the predominant cause of cancer death worldwide, with it being responsible for an estimated 16% of all cancers (Stewart, Kliehues, 2003), 91% of all lung cancers in men, 69% of lung cancers in women, 43-60% of cancers of larynx, oesophagus and oral cavity, 11% of cancer of stomach in men, 4% of cancer of stomach of women in developing countries and, 17% of stomach cancers of men and 11% among women in developed countries all attributed to tobacco smoking (Boyle et al., 2003).

The first sites of cancer due to smoking were listed as the lungs, oral cavity, pharynx, oesophagus, pancreas, urinary bladder and renal pelvis. More recent studies have added the nasal cavities, paranasal sinuses, nasopharynx, stomach, liver, kidneys (renal cell carcinoma), cervix, oesophagus (adenocarcinoma) and myeloid leukaemia to the list of cancers directly related to smoking (Sasco, Secretan, Straif, 2004).

Many of the compounds within tobacco and cigarettes are carcinogenic; Benzene is a well established carcinogen capable of inducing myeloid leukaemia, this risk is increased as the duration of smoking and number of daily cigarettes increases (Sasco et al., 2004).

The risk of death due to smoking related complications is highest in those smoking high-tar cigarettes; this may be due to the fact that some of the high-tar brands of cigarettes do not make use of a filter. There has been no evidence to prove that low-tar cigarettes in any way reduce the smoking related risks, as smokers who smoke these lower-tar yielding cigarettes may compensate for the smaller amount of tar by taking larger and more frequent puffs, making the risk factors for low and medium tar yielding cigarettes the same. Smoking cessation considerably reduced the risk of lung cancer, in those that quit before the age of 35 having similar risk as non smokers (Gallus, 2004).

Pancreatic cancer is a therapy resistant form of cancer which is aggressive and difficult to diagnose, tobacco smoking is a main risk factor for developing pancreatic cancer, this could be due to many of the carcinogenic compounds or other ingredients such as aromatic amines polycyclic aromatic hydrocarbons or metal components such as cadmium, within tobacco smoke reaching the pancreas via the blood or refluxed bile (Talamini et al., 2009).

2.6.8 Smoking and Hearing loss

Disorders affecting ones hearing are not only a great cause of psychological stress but also a form of invisible handicap affecting ones means of communication, a burden said to be greater than that of blindness (Keller, Morten, Thomas et al., 1999).

Smoking has been implicated as a risk factor for hearing loss in those not exposed to occupational noise, as there is a multiplicative effect of smoking and age on hearing loss (Nomura, 2003).

The exact mechanism of ototoxicity and the resulting hearing loss in un clear, however the suggestion of nicotine causing vasoconstriction of the cochlea and resulting in anaemia of the cochlea seems biologically plausible (Giacomo, Pietro, 1961).

2.6.9 Smoking and Menopause

Menopause occurs naturally in women when the number of ovarian follicles decreases below a critical level. Evidence shows that women who smoked more than 14 cigarettes a day at their last menstrual period experienced menopause earlier than non-smoking women of the same age, however those smoking less than 14 cigarettes a day showed no difference in age of onset of menopause compared to non-smokers. Women who quit smoking before their last menstrual period did not seem to experience early menopause (Kinney, Kline, Levin, 2005).

There are many mechanisms hypothesised to cause the early onset of menopause (2.5 months – 2.5 years earlier) due to smoking; current smokers are found to have lower levels of estradiol in the middle and luteal phase of their cycle than non smokers, nicotine causes the loss of follicles within the ovary and blocks the enzyme aromatase, which converts androgens into estrogens, smokers also demonstrate nonexistent or delayed peaking of luteinizing hormones in the middle of their cycle (Parente et al., 2008).

Smoking has an antiestrogen effect due to the increase in androgen production by the suprarenal glands as well as polyaromatic hydrocarbons which induce microsomal cytochrome P-450, they serve to metabolise steroildal hormones and increase the production of catechol metabolites of estradiol, a weaker alternative to estrogens thus reducing the beneficial effects of estrogen. A further association has been proven between the pre-apoptotic protein Bax and the hydrocarbons found in cigarettes which induce the expression of Bax in the oocytes causing apoptosis and resulting in premature ovarian failure (Parente et al., 2008).

Infertility, another common problem associated with smoking may also be attributed to this reduction in ovarian reserves (Parente et al., 2008).

2.6.10 Smoking and Depression

Affective disorders particularly major depressive disorders are proven to have an association with nicotine dependence with 22% to 60% of current smokers experiencing depression (McClave et al., 2009), although the exact mechanism is unclear (Breslau et al. 1993; Anda et al. 1990). Theories serving to explain this association include; smoking may be a coping mechanism for stress (Revel et al., 1985), or act as a form of self medication to reduce depressed mood (Covey et al., 1997).

The elevated risk of smoking in those with mood disorders such as depression has also been explained by these three theories; firstly the self medication theory, which suggests that the nicotine and other components of cigarettes have anti depressive effects and are smoked for this reason to manage pre-existing depression, the second theory highlights the common genetic and environmental risk factors of both

smoking and depression, and the third theory suggests that depression may be a consequence of smoking (Duncan, Rees, 2005).

Some pharmacological smoking cessation aids are in fact antidepressants, this further strengthens the belief that nicotine dependence and depression share many neuronal substrates (Picciotto et al., 2008).

There is evidence to suggest that smokers who have a history of depression who stop smoking are at an increased risk of suffering another episode of major depression; this risk may remain for about six months (Kinnunen et al., 1999; Glassman, 2001).

There is an increased risk of mortality due to cardiovascular disease or suicide in those with major depression; although depression can also be viewed as an independent risk factor for diseases such as cardiovascular disease and cancer, the combined increased rate of smoking as found in those suffering from depression would further strengthen the risk of mortality through the association of depression (Surtees et al., 2008; Cuijpers, Schoevers, 2004; Prozuelo et al.,2009; Oerlemans et al., 2007).

2.6.11 Smoking and Diabetes

Smoking increases ones risk of developing type 2 diabetes; the known smoking-associated health risks such as cardiovascular and kidney disease can also exacerbate conditions that accompany or precede diabetes. Diabetic smokers are often deterred from smoking cessation as they may experience short term worsening of some of their diabetic symptoms and weight gain (Tonstad, 2009).

Smokers with type 2 diabetes have been shown to have an increase in blood glucose concentration, insulin and C-peptide responses with increased responses to oral glucose load when compared to non smoking type 2 diabetes patients. This increase in insulin resistance is a major risk factor for the development of metabolic syndrome, which mediates cardiovascular diseases such as coronary heart disease and stroke, responsible for a large proportion of type 2 diabetes associated mortalities (Tonstad, 2009; Kong et al., 2001).

Smoking in type 1 and 2 diabetics exacerbates markers for kidney failure such as microalbuminuria and promotes the onset and progression of nephropathy; smoking is therefore a major risk factor for kidney dysfunction and diabetic kidney disease (Tonstad, 2009).

Cigarette smoking and type 1 diabetes are both known to be associated with accelerated progression and increase risk of retinopathy (Eliasson, 2003), although some studies have indicated that smoking has no relation to retinopathy in type 2 diabetics (Guillausseau et al., 1998; Owens et al, 1988).

An additional complication of smoking in diabetic patients includes an increased risk of hypoglycaemia (Tonstad, 2009).

Smoking cessation is beneficial to diabetic patients; however the positive effects are not immediately obvious. Smoking cessation may affect the patient's pharmacokinetics or treatment regime with insulin (particularly inhaled insulin) absorption rates being altered as quickly as twelve hours following smoking cessation, this would require careful monitoring to decrease the risk of hypoglyceamia (Tonstad, 2009).

Despite a short term increase in body mass index which can be expected in smoking cessation, there is an increase in insulin sensitivity which results in an improvement of the lipoprotein profile of the patient (Tonstad, 2009).

2.7 Passive Smoking

Passive smoking also known as 'involuntary smoking' occurs when a non smoker is exposed to the environmental or 'second hand' tobacco smoke from a smoker in near proximity. This second hand smoke is composed of the mainstream tobacco smoke being exhaled by the smoker and the side stream smoke from the smouldering cigarette both of which are diluted in the surrounding air. Although the chemical breakdown of second hand smoke is different to the smoke inhaled by a smoker, it still contains many known carcinogens and nicotine and some toxins of which are found in higher concentrations in side stream smoke (Sasco, Secretan, Straif, 2004).

Passive smokers include spouses who live with a spouse who smokes in the house, employees who work with smokers and children of smoking parents. Evidence shows that long time non-smoking spouses of smokers had a 20-30% increase in risk of lung cancer, while non-smokers exposed to second hand smoke at the workplace had a 16-19% increase in risk of developing lung cancer. Observations have been made that the risk factors of passive smoking increase with exposure. Passive smokers have also been found to present with other diseases associated with smoking such as, increased morbidity and mortality linked to cardiovascular diseases, eye and nose irritation, lower respiratory tract infections in children, exacerbations of conditions such as asthma and other chronic respiratory conditions as well as middle ear infections. There is also a strong link between passive smoking and a higher incidence of sudden infant death syndrome (Sasco, Secretan, Straif, 2004).

Passive smoking is a well known risk factor for upper respiratory tract infections in children; some other conditions include chronic cough, wheezy bronchitis, ear discharge, mouth breathing, pneumonia, snoring and sudden infant death syndrome (Gryczyska, Kobos, Zakrzewska, 1999).

2.8 Smoking Interventions

Health care practitioners are in the unique position to intervene and offer advice to smokers in an effort to stop them smoking. Brief advice to quit smoking offered by a physician can have a significant health effect of 5-10% abstinence rates if provided routinely, however surveys of smokers indicate that they receive such advice at less than half of their visits to the physician (Cornuz, 2007).

Physicians become despondent and hesitate to recommend smoking cessation as so few patients follow their advice, however they need to realise that even when such advice does not produce the immediate desired response, it may edge the patient closer towards the difficult decision to stop smoking (Cornuz, 2007).

Every smoker needs to be encouraged not only to reduce the number of cigarettes smoked daily which is often proposed as an alternative but stop smoking completely. Smoking cessation programmes that encourage the patient to gradually reduce the

number of cigarettes smoked daily without medication compared to the complete and immediate cessation of cigarettes often prolong withdrawal symptoms and craving. Those trying to quit smoking have been found to compensate for a reduction in the number of daily cigarettes by taking more and deeper puffs per cigarette. A gradual reduction in the daily amount of cigarettes smoked has only been found to be effective when used in conjunction with (NRT) nicotine replacement therapy (Cornuz, 2007).

When initiating and assessing an intervention for tobacco users a 5 A guideline has been proposed, the 5 A's comprise:

1) Ask the patient about their tobacco use: identify and record tobacco use in each patient at each visit.

2) Advise the patient to quit: using a firm and personalised manor urge patient to quit smoking, use personalised advice highlighting the individuals situation, for example "your smoking can be detrimental to the health of your children" or "your smoking will prolong your cough and put you at risk for long term respiratory problems like emphysema and chronic bronchitis".

3) Assess willingness of patient to attempt to quit: Is the patient willing to try quit smoking?

4) Assist in attempt to quit smoking: use counselling and pharmacotherapy to assist the patient.

5) Arrange follow up: maintain contact with patient preferably within first week after proposed quit date, which should prevent relapse (Cornuz, 2007).

It is a common fact however that intervention efforts will be unsuccessful if the patient is unwilling to quit smoking or without successful motivation. For these unwilling patients the United States practice guidelines recommend using the "5R's".

1) Relevance: bring to the individuals attention why quitting is personally relevant to them, such as being pregnant with risks of pre term delivery, a family history of strokes, cancers, bypass surgery, or health of children exposed to secondary smoke.

2) Risks: identify negative consequences and risks of tobacco use such as cardiovascular problems, cancer, risks to children exposed to smoke and a increased probability that children will also take up smoking.

3) Rewards: Emphasise the positive implications associated with cessation of smoking such as improved health, setting a better example for children, financial gains, improved sense of smell and taste and car, clothes and breath will not smell like smoke.

4) Roadblocks: identify potential barriers and problems the patient may anticipate such as depression or weight gain; the clinician should offer treatment to address such barriers such as problem solving and pharmacotherapy.

5) Repetition: this motivational intervention must be repeated with each visit to the doctor until the desired outcome of smoking cessation is achieved (Cornuz, 2007).

In preparation for the quitting process a clinician should assist the patient by helping them establish a quit plan, providing counselling and intra-treatment social support and recommending approved pharmacological assistance using a “STAR” plan according to Cornuz (2007).

1) Set a quit date.

2) Tell family and friends about the quit plan and request support and understanding.

3) Anticipate setbacks and challenges especially due to nicotine withdrawal syndrome in the first few weeks.

4) Remove tobacco products from individual’s environment where possible.

The individual who is trying to quit smoking will require some essential information:

1) Smoking is an addiction and cessation needs to be taken as seriously as any other drug addiction, willpower alone may be insufficient and the individual needs to make quitting a top priority.

2) Total abstinence after the proposed quit date is the goal.

3) Nicotine withdrawal symptoms such as mood disturbance, irritability, difficulty concentrating, insomnia, and weight gain with increased appetite can be expected. These symptoms usually peak after a few days of quitting but dissipate within 3-4 weeks.

4) The Physician must help identify high risk situations that would increase the risk of failure or relapse due to past experience with smoking such as, drinking alcohol, being with other smokers or stressful situations. Such situations need to be avoided if at all possible.

5) Strategies of cognitive and behavioural coping skills need to be selected for when the patient experiences cravings for tobacco. These include reminding one's self why they want to quit, telling one's self the urge will subside, repeating phrases such as "smoking is not an option", to leave the situation and engage in a distracting activity, take deep breaths and/ or seek social support (Cornuz, 2007).

2.9 Smoking and diet

Smokers are more vulnerable to damage of the cardiovascular system due to free-radicals from their cigarette smoke and generally have a lower antioxidant vitamin intake and plasma level. This may render a smokers' low density lipoprotein cholesterol more susceptible to lipid perioxidation and consequent atherosclerosis (Waart, Smilde, Wollersheim, Stalenhoef, Kok, 2000).

The beneficial effect of a smoker consuming a diet high in fruits and vegetables is small in comparison to the effects of quitting smoking, thus the greatest benefit and reduction in the risks for cancer of the lung would be achieved with the combination of smoking cessation and an increase in the amount of fruit and vegetables consumed (Skuladottir, Tjoenneland, Overvad, Stripp, Christensen, Raaschou-Nielsen, Olsen, 2004).

According to Scala (1993), a smoker is likely to have diminished levels of vitamin A, B, C, E, folic acid, calcium, selenium and zinc. Smokers require more anti oxidants

and vitamins such as vitamin C and E than non smokers which reduce the risk of cancer, cataracts and heart disease. Therefore smokers can reduce the risks of smoking by consuming a diet high in vitamins and anti oxidants (Scala, 1993).

2.10 Orthodox Medical Management of Cigarette Addiction

Orthodox medicine commonly uses two forms of smoking cessation medication; nicotine replacement therapy (NRT) and bupropion (Zyban). NRT doubles the success rate of smoking cessation and long term abstinence by replacing some of the nicotine a smoker would have obtained through cigarettes, thereby reducing the severity of nicotine withdrawal symptoms (McRobbie, Lee, Juniper, 2005). There have been concerns raised regarding the limited medical supervision of those on NRT, some patients may stay on the NRT for longer than recommended and could even become dependent on the NRT (Etter, 2009).

NRT provides a safer and lower dose of nicotine, and allows the smoker to develop coping strategies for associated behavioural aspects of smoking while the physiological component of their addiction is being satisfied. The NRT provides limited nicotine which should prevent intense withdrawal symptoms as well as not providing the associated reinforcing peaks of smoking (Mitrouska, Bouloukaki, Siafakas, 2007).

2.10.1 Nicotine Gum (Polacrilex)

The long term use of NRT gum is common, and in the US 5-6% of NRT gum users exceeded the recommended three month period, while 9% of the UK users used the gum for a year or longer (Etter, 2009).

NRT gum was approved by the US-FDA in 1984 and can be purchased over the counter since 1996. It contains nicotine in different strengths in a resin base. The absorption of the nicotine takes place through the buccal mucosa, which is the preferred route of administration due to the gastro-intestinal side effects experienced by swallowing nicotine. Once the nicotine has been absorbed it undergoes metabolism by the liver, resulting in minimal levels of nicotine in the blood. NRT gum

is most effective when it is chewed slowly for 2-3 times until a tingling sensation in felt then left between the cheek and gums, this routine of chewing intermittently and then re positioning the gum, should take place for 15-30 minutes. The gum is best utilised without the presence of any acidic environments, as one would find in fruit juices and so these need to be avoided (Mitrouska, Bouloukaki, Siafakas, 2007).

A few common side effects of NRT gum found in 25% of users includes headaches, dyspepsia and jaw ache. There are no significant differences in success rate between the two different doses of nicotine gum, the 4 mg dose is used for smokers with a higher nicotine dependence (20-25 cigarettes per day) with which the 2 mg dose has been un successful. The recommended duration of use of the gum is 1-3 months with a daily intake of 10-15 pieces of gum. The use of NRT gum increases the quit rate by 8% (Mitrouska, Bouloukaki, Siafakas, 2007).

2.10.2 Nicotine Nasal Spray

This form of NRT introduced in 1996 is the most rapidly absorbed NRT as absorption occurs via nasal mucosa. The effects peak within 5-10 minutes and a dose which is classified as a spray in each nostril provides 0.5mg of nicotine with a maximum recommended dose being 40mg. This form of NRT is contraindicated in individuals suffering from asthma or similar hyper reactive air way diseases. Common side effects include throat and nasal irritation, cough, watering eyes, sinusitis, nausea and palpitations. The cessation success rate with use of the nicotine nasal spray increases by 12.5% (Mitrouska, Bouloukaki, Siafakas, 2007).

2.10.3 Nicotine Patch

The transdermal nicotine patch introduced in 1992, provides a slow release of nicotine through the skin. Once applying the patch initial absorption and transfer of nicotine is rapid from the adhesive layer but there after it takes between 2-7 hours for blood nicotine levels to mimic levels achieved through smoking. The transdermal patch produces a reservoir type nicotine state which will be maintained for a short time after the patch is removed (Mitrouska, Bouloukaki, Siafakas, 2007).

Transdermal nicotine patches are available in a variety of strengths from a 5mg dose to a 21mg dose and are designed to be used for 16-24 hours. The 24 hour dose is available in a 7, 14 and 21mg dose and is applied each morning. The 16 hour dose available in a 5, 10 or 15mg dose is designed for day time use and is removed before bedtime. The 16 hour and 24 hour preparations offer similar benefits with the dose being progressively weaned over 8-12 weeks. Skin irritation, a common adverse effect can be reduced by rotating the site of patch application. Nightmares and sleep disturbances can be associated with the 24h dose although removal of the patch before bed can reduce these side effects. The quit rate using a transdermal nicotine patch increases by 19% in both men and women (Mitrouska, Bouloukaki, Siafakas, 2007).

2.10.4 Nicotine lozenge

The Nicotine lozenge is a sublingual tablet, it offers easy to use NRT with slightly more nicotine than the nicotine gum, available as a 2 or 4mg dose the lozenge shares the same pharmacokinetic properties as the nicotine gum, offering a rapid delivery of nicotine via buccal mucosa which declines over time (Mitrouska, Bouloukaki, Siafakas, 2007).

This form of NRT has produced an increase in smoking cessation rates of 8.7% and has been proven to be particularly effective in those unable to quit using other forms of pharmacotherapy (Mitrouska, Bouloukaki, Siafakas, 2007).

2.10.5 Nicotine inhaler

Introduced in 1998 the nicotine inhaler contains 4mg of nicotine within a plastic cartridge that is inserted into a mouthpiece, and is available by prescription. This form of NRT mimics smoking and it takes approximately 80 inhalations to obtain the equivalent amount of nicotine as one would get from smoking a cigarette. This offers a rapid delivery of nicotine and is favoured by heavier smokers increasing smoking cessation rates by 12.3% (Mitrouska, Bouloukaki, Siafakas, 2007).

The adverse effects of a nicotine inhaler including a cough, mouth and throat irritation are minimal, with the recommended dose being 6-16 cartridges a day for 6-12 weeks with a further three months of tapering the dose (Mitrouska, Bouloukaki, Siafakas, 2007).

2.10.6 Electronic cigarette

The electronic cigarette is similar to the nicotine inhaler in its application, but uses a tiny battery to produce the nicotinic vapour. This is not a registered form of NRT, has not been approved by the Food and Drug Administration (FDA) and the packaging inserts state that the product is not designed to help one stop smoking. The electronic cigarette does not contain any health warnings and is not governed by the same smoking laws thus they can be purchased by anyone even those under the age of 18. The FDA is also concerned that the application comes in a variety of flavours including, mint, cherry, apple, vanilla, strawberry and chocolate which may entice users, increase nicotine addiction and may lead younger users to try other tobacco products. The FDA is concerned not only that the products contain known harmful substances such as carcinogenic nitrosamines and diethylene glycerol a toxic chemical found in anti freeze, but also that clinical studies about the safety and efficacy of these products have not been submitted to the FDA. Those consumers who use electronic cigarettes do not know what concentration of hazardous chemicals or nicotine they are inhaling or whether they are safe for their intended use (FDA consumer health information, 2009).

2.10.7 Bupropion Hydrochloride

Bupropion is an atypical antidepressant phenylaminoketone first introduced in the USA under the brand name of Zyban is the first non nicotinic agent to be approved by the FDA for smoking cessation and is considered a first line pharmacological treatment for nicotine dependent smokers (Mitrouska, Bouloukaki, Siafakas, 2007).

Nicotine is known to activate the mesolimbic system by releasing dopamine from the nucleus accumbens, the pathway thought to be responsible for reward and cravings

while withdrawal is thought to be related to an altered noradrenergic activity in the locus coeruleus (Mitrouska, Bouloukaki, Siafakas, 2007).

Bupropion has a direct inhibiting effect on the neuronal uptake of the catecholamines noradrenalin and dopamine with a much lesser effect on the re uptake of indolamines like serotonin as well as no inhibitory effect on monoamine oxidase, although not proven the mechanism of action of Bupropion in the treatment of nicotine dependence may be due to this reduced re uptake of dopamine in the mesolimbic system and noradrenaline in the locus coerulus (Mitrouska, Bouloukaki, Siafakas, 2007).

Bupropion is available in a 150mg and 300mg dose, the target dose is 300mg per day in a dependent individual. The treatment is started for at least 7 days before expected quit date at a dose of 150mg per day then increased to 150mg twice a day for 3-6 days so that adequate levels are obtained before the quit attempt. The reduction in urge to smoke and craving for cigarettes facilitates cessation (Mitrouska, Bouloukaki, Siafakas, 2007).

The first formulation of Bupropion became available in 1988; however it was an immediate release form which showed adverse events at maximal plasma concentration, thus the FDA approved the sustained release Bupropion in 1996 which presented with an improved safety profile associated with much fewer adverse effects (Mitrouska, Bouloukaki, Siafakas, 2007).

Bupropion concentrations have been found to be higher in breast milk than in plasma concentrations and thus its use should be avoided in breastfeeding mothers, Bupropion should also be used with caution in those taking antipsychotics, antiarrythmics, b-blockers and certain anti depressants as there is a possibility for drug interactions. Adverse effects of Bupropion include insomnia, agitation, dry mouth, headache, skin rash, pruritis, hypersensitivity, dizziness and the most serious adverse effect being seizure in approximately 0.1% of users. Bupropion is thus contraindicated in patients suffering from epilepsy or any history of seizures, those with a history of eating disorders such as bulimia and anorexia, uncontrolled hypertension or severe hepatic necrosis (Mitrouska, Bouloukaki, Siafakas, 2007).

Combination therapy of Bupropion and NCT can be used in cases where there have been prior unsuccessful smoking quit attempts or those who have a high level of nicotine dependence. Evidence suggests the Bupropion may also be used to prevent smoking relapse and has been found to be particularly effective for smoking cessation among difficult to treat patients with cardiovascular disease, COPD, depression and schizophrenia. Quit rates are improved to between 39-44% when using Bupropion (Mitrouska, Bouloukaki, Siafakas, 2007).

2.10.8 Second line pharmacotherapuetics

Certain antidepressants such as imipramine, doxepin and moclobemide, antihypertensives such as clonidine , anxiolytics like buspirone, diazepam, meprobamate , b-blockers and sensory replacement therapy such as silver acetate have been used to aid in smoking cessation. Clonidine and Nortriptilyne are seen as second line smoking cessation drugs (Mitrouska, Bouloukaki, Siafakas, 2007).

Although Clonidine has been found to increase smoking cessation rates by 11%, this orally or transdermally provided antihypertensive that acts to suppress sympathetic activity and reduce withdrawal symptoms associated with abstinence of alcohol or drugs such as opiates, is still considered a second line therapy due to its adverse side effects of dizziness, sedation and hypotension (Mitrouska, Bouloukaki, Siafakas, 2007).

The tricyclic antidepressant Nortriptyline has been shown to improve smoking cessation rates by 12% with its mostly noradrenergic properties; however it has not been licensed for this indication. Despite its similar success rates to Bupropion its side effects and limited clinical trials make it a second line intervention (Mitrouska, Bouloukaki, Siafakas, 2007).

2.11 Behavioural therapy and Management of Nicotine withdrawal Syndrome

Drug trials often use the medication together with various behavioural therapies (Piasecki, 2006).

Even with the development of extensive smoking cessation programmes, smoking relapse is the modal outcome, even in those using intensive psychosocial treatment and pharmacotherapy (Piasecki, 2006).

It has been suggested that treatments with extensive social support or extended treatment contact could increase cessation rates by not only providing more opportunities to provide behavioural treatments like educational information, empathy or coping skill training, it also promotes abstinence by providing accountability with the individual knowing they are being monitored and that they would have to admit any lapses or failure to quit (Piasecki, 2006).

General problem solving, a form of cognitive-behavioural therapy constitutes relapse prevention therapy, aiming to help patients identify high risk situations and avoid such situations (Piasecki, 2006).

Aversive smoking such as rapid smoking, are a form of behaviour therapy techniques which use the discomfort of oversmoking as a punisher or substrate for conditioned taste aversion. These methods popular in the late 1970s lost popularity with the introduction of nicotine gum in the 1980s along with the concern about the health risks and safety of oversmoking techniques. Problem solving approaches and other cognitive therapies within clinical psychology replaced the aversive smoking techniques (Piasecki, 2006).

2.12 Nicotine Withdrawal Syndrome

The diagnostic criteria for Nicotine Withdrawal, as stated in the fourth edition of the Diagnostic And Statistical Manual of Mental Disorders, includes the following symptoms as directly related to the abrupt cessation of nicotine or decrease in the amount of nicotine used by those who had previously used nicotine for at least a few weeks.

(1) Dysphoric or depressed mood

(2) Insomnia

(3) Irritability, frustration, or anger

(4) Anxiety

(5) Difficulty concentrating

(6) Restlessness

(7) Decreased heart rate

(8) Increased appetite or weight gain

These symptoms impair social, occupational or other important areas of functioning (American Psychiatric Association, 1995).

The effects of nicotine withdrawal are both physiological, which normally only lasts about a week, and psychological which can take weeks or years to overcome (Arden, Christen, Kenneth, Cooper, 1979).

Withdrawal is normally accompanied by certain physiological changes like decreased heart rate. Evidence suggests withdrawal syndrome begins within the first 2-24hours of abstinence, peaking within one week and gradually decreasing to pre-cessation levels within 4 weeks (Morrell, Cohen, Absi, 2008).

2.13 Negative effects of smoking cessation

2.13.1 Weight Gain

Smoking cessation has numerous well documented health benefits; however weight gain is common after smoking cessation, with most successful quitters gaining 4-5kg and 13% of quitters gaining 11kg or more, which has been found to be related to relapse (Pisinger, Jorgenson, 2007).

2.13.2 Depression

Depression has been studied as moderators of smoking relapse; however major depression is inconsistently related to relapse. Any depressive symptoms at the start of smoking cessation attempts dramatically lower expected quit rates (Piasecki, 2006).

Bupropion an anti depressant is a first line pharmacotherapy used in smoking cessation attempts and would seem to be an obvious choice drug for an individual who wishes to reduce post cessation negative effect, however evidence suggests this is not the case with Bupropion and reduces its efficacy. Bupropion is known to ameliorate depression but not particularly post cessation depression associated with negative effects of smoking cessation. In cases of smokers with a depressive history, cognitive behavioural therapies are recommended (Piasecki, 2006).

Depression may make it more difficult to cease smoking and smokers who attempt to stop smoking may become depressed as a result of unsuccessful quit attempts. Smokers who are depressed have been shown to have a higher nicotine dependence, which could explain their higher incidence of multiple chronic diseases like diabetes and cardiovascular disease (McClave et al. 2009).

2.13.3 Ulcerative colitis

Smoking has been shown to offer some benefit to those suffering from ulcerative colitis, new research shows that NRT such as nicotine gum may offer the same benefit (Watson, Lewis, 1995). The implications of this study warn smokers who have ulcerative colitis of possible exacerbations of their symptoms if they cease smoking and that these undesirable effects may be avoided with the use of NRT such as nicotine gum.

2.13.4 Parkinson's Disease

There has been a link found to exist between nicotine use such as smoking and Parkinson's disease with an increase in the interest of the role of nicotine as a form of treatment for Parkinson's disease. Nicotine has been found to reduce tremors, rigidity, bradykinesia and improve the characteristic gait and sleep disturbances common in Parkinson's patients (Balfour, Fagerstrom, 1996). This would explain the need for NRT in Parkinson's patients who want to stop smoking but who still want to receive the ameliorative effects from the nicotine.

2.14 Alternative Management of Nicotine Withdrawal Syndrome

2.14.1 Acupuncture

Acupuncture has been used to treat chemical withdrawal syndrome associated with chemical dependence. Auricular acupuncture showed a 6.3-60% success rate for abstinence from smoking with a recorded decrease in withdrawal symptoms through release of endogenous opioid peptides, thus acupuncture is a safe intervention for smoking cessation with only minor side effects of tenderness, slight bleeding and nausea. However the results are still unsatisfactory and should be combined with behavioural counselling and NRT (Wu et al. 2007).

2.14.2 Hypnosis

Hypnosis has been used with success rates varied from 21% to 96% quit rates with an average of 36%. Participants were hypnotised for two sessions according to recommendations from the Handbook of the American Society of Clinical Hypnosis for 45 minutes each in an effort to remove the unwanted bad habit of smoking, however the statistical and clinical evidence is inconclusive and it cannot be said that hypnosis is any more successful than any other cessation techniques or spontaneous cessation (Valbo, Eide, 1996).

2.14.3 Self Help Books

Self help books (SHB) are widely prescribed by therapists despite the limited positive evidence of efficacy. SHB appeal to the general public and may be effective in changing certain problematic behaviours such as smoking, and can be used more effectively in conjunction with cognitive behavioural therapy. Many SHB describe exaggerated claims of effectiveness by the author who despite having credentials may offer bias hunches, over generalised or superficial theories that have no substantiated evidence. The appeal of SHB may be that they offer the reader motivation to change with a decrease in self recrimination or self reproach and, a non threatening form of advice free from judgement or embarrassment as one may experience in therapy (Pantalon, lubetkin, Fishman, 1995).

2.15 Homoeopathy in Management of Nicotine Withdrawal Syndrome

Homoeopathy is capable of working within an individual on not only a physical level but also the emotional and mental level (De Schepper, 2001). This makes Homoeopathy an ideal approach to treating nicotine withdrawal syndrome which manifests symptoms on all three levels. The Diagnostic And Statistical Manual of Mental Disorders highlights the physical symptoms such as decreased heart rate; the mental manifestations including restlessness and difficulty concentrating, with emotional disturbances such as irritability depression, anger and frustration (American Psychiatric Association, 1995).

2.15.1 Homoeopathic Complex Materia Medica and Potencies

The homoeopathic complex used in this study was made up of Caladium seguinum, Nux vomica and Staphysagria delphinum, all in a 30CH potency. All three of these remedies are indicated in the Repertorium Homeopathicum Syntheticum to increase ones disgust for tobacco, to be used for those who desire to smoke tobacco and in which tobacco causes a form of aggravation (Schroyens, 2001). These remedies are also indicated due to the mental states they are indicated for which coincide with anticipated symptoms of nicotine withdrawal syndrome as outlined before.

2.15.1.1 Caladium seguinum 30 CH

Caladium seguinum exhibits mental symptoms such as “irritable and depressed mental states, headache of smokers, restless and cannot control himself after smoking, confused and cannot concentrate” (Vermeulen, 2000).

2.15.1.2 Nux vomica 30 CH

Nux vomica exhibits mental symptoms of “causeless anxiety, taciturn, angry and impatient, quarrelsome if disturbed indisposition to mental exertion, very irritable and sensitive to all impressions” (Vermeulen, 2000).

2.15.1.3 Staphysagria delphium 30CH

Staphysagria delphium exhibits the following relevant symptoms, “moody, irritable, sad and irritable, depressed and prefers solitude, sleep greatly disturbed, weakness of memory”, “effects of smoking” including “excoriated tongue, gastralgia” (Vermeulen, 2000).

2.16 Tautopathy in Management of Nicotine Withdrawal Syndrome

According to Dr Bhatia (2006), Tautopathy is the method of curing or removing the bad effects of conventional drugs by means of identical homoeopathically potentised drugs. For example, if you are suffering from bad effects or side effects of the antibiotic Penicillin, you can use potentised Penicillin to remove its side effects. This idea has been confirmed not only clinically by a large number of homoeopaths but has also been proven scientifically. There have been studies in which potentised lead and potentised arsenic have been used to promote excretion of the same substances in relevant cases of poisoning. The results have shown that such use of potentised substances can help remove symptoms caused by toxicity, by enhancing the elimination of the toxin from the tissues (Bhatia, 2006).

2.17 Placebo Effect

The placebo effect is still not properly understood; while surgical placebo interventions are viewed as unethical the use of placebos in a randomized controlled trial (RCT) remains a valuable tool. There is also the possibility that what was seen as a placebo effect could have been due to spontaneous remission, response to nursing care or consultation, or natural variability. Placebo responders are labelled as such if they respond to a single administration of placebo substance, while they could in fact be partial responders or reliable responders. Previous studies highlighting consistency or reliability of a placebo response are flawed and contradictory (Kaptchuk et al. 2008).

2.18 Related homoeopathic research

De la Rouviere completed a study in 1996 to test the effectiveness of both acupuncture and homoeopathic treatment to help people stop smoking. Thirty participants were randomly divided into two treatment groups. Each participant completed questionnaires such as the Fagerstrom Tolerance test which assessed nicotine dependence at the start and end of the study, and a daily smoking log was kept by each individual prior to and for the duration of the study. The data produced was statistically analysed using the Mann-Whitney U test and the Wilcoxon signed rank test to compare results within each group before and after treatment. Both forms of treatment were found to help participants stop smoking, with the homoeopathic treatment group showing a 40% quit rate compared to the acupuncture group which showed a 33% quit rate. The particular homoeopathic intervention used in this study was a form of hetero-isotherapy whereby the participants were given a remedy produced from a tincture of the specific brand and strength of cigarette they smoked.

In 1998 Pautz completed a double-blind randomised trial to test the effectiveness of isotherapy together with homoeopathic similimum compared to isotherapy and placebo to help people stop smoking. Of the thirty participants each received the isotherapy which consisted of a remedy made as one would make Nicotinum tobaccum but out of the individuals own brand and strength of cigarette, they then either received a homeopathic similimum as indicated by a homoeopathic consultation or a placebo substance. Questionnaires and daily log sheets were completed for statistical analysis which consisted of the Mann-Whitney U tests and Wilcoxon signed rank tests. Results indicated that both groups showed a reduction in cigarette consumption with the homoeopathic similimum and isotherapy group being more effective than the isotherapy and placebo group.

In 2001 Hellberg performed a double blind placebo-controlled study to test the effectiveness of a homoeopathic complex (Avena sativa D3, Ignatia amara D4, Daphne indica D6, Nux vomica D6, Caladium seguinum D60, Nicotinum D60 and Nicotiana tabacum D60) to help people stop smoking. Thirty participants took part in the study; each participant received five treatments over the duration of the study which was four weeks.

Daily smoking log sheets were completed as well as questionnaires at the start and end of the study, enabling the researcher to analyse the results statistically. The Mann-Whitney and Wilcoxon signed rank test were performed, with results indicating that both the homoeopathic complex and placebo showed a significant reduction in cigarette consumption, with the researcher deducting that the remedies used in the homoeopathic complex were not necessarily effective in reducing cigarette consumption.

Lutchman-Maharaj completed a study in 2002 that compared the effectiveness of the homoeopathic complex (Caladium seguinum 30CH, Nux vomica 30CH and Staphysagria delphinium 30CH) and the indicated homoeopathic similimum in the management of tobacco addiction. Three groups were recruited one received the homoeopathic complex, one the similimum and one the placebo treatment. Each participant completed the tolerance dependence questionnaire at the start and end of the study and completed daily smoking log sheets. These results were analysed using the Wilcoxon Signed rank test and Kruskall Wallis tests. Each of the groups including the placebo group showed a reduction in the number of cigarettes smoked, thus the researcher concluded the homoeopathic remedies were not effective in the management of tobacco addiction as they offered no more benefit than placebo.

Chapter Three

Materials and Methods

3.1 Advertisements

Advertising was done in the form of posters on the notice boards at the DUT campus and an advert was placed in the Berea Mail, which is a local paper, in order to obtain participants for the study (See Appendix J).

3.2 Sampling method and sample size

Non-probability sampling in the form of convenience sampling was applied as a sample frame for the population was not available. The first 40 patients to respond to the advertisements, who met the inclusion criteria and provided consent, were recruited accordingly.

3.3 Subjects

3.3.1 Inclusion Criteria

The following criteria had to be met by potential patients who responded to the advertisements in order to be considered suitable for inclusion:

- The participant had to be between the ages of 18 and 60.
- The participant had to be a smoker of cigarettes, not one who uses a pipe or chews tobacco.
- The participant had to smoke at least 10 cigarettes a day.
- The participant had to have been smoking for at least one year.
- The participant had to smoke one of 15 predetermined popular cigarette brands and of a strength from which a tautopathic preparation was made in advance (See Appendix O).
- Participants of both genders were included.
- Participants had to be in good health and could not be pregnant.

- The participants were not permitted to change their basic routine or lifestyle for the duration of the study.
- The participants were to be able to attend a first and final consultation.
- The participants were to be of sound mind and may not have been suffering from any mental disorder that may have been exacerbated by nicotine withdrawal symptoms.

3.3.2 Exclusion Criteria

The following criteria were used to exclude those who were not suitable participants:

- Pregnant or lactating mothers.
- Those suffering from a mental illness.
- Those who smoked a brand of cigarette which did not correspond to the predetermined 15 brands of cigarette determined by the researcher.
- Those in which the consumption of ethanol was contraindicated.
- Those who could not agree to the terms and conditions of the study as outlined in the participant information sheet (See Appendix I).

3.4 Randomisation process

Each participant was allocated a number, one through to forty. The research supervisor drew up a random placement roster, dividing the participants into four equal groups consisting of ten participants. The laboratory technician allocated the names of each patient against a position in the placement roster and was the only one who knew which participant was in each group, thus making the study double blind.

3.5 Preparation of experimental medication

3.5.1 Tautopathic preparation

The tautopathic preparation was manufactured according to the German Homoeopathic Pharmacopoeia (Method 6A) up to 6Ch potency, along with an

additional process, whereby the lactose base was infused by the smoke of the burning cigarette (See Appendix K).

3.5.2 Homoeopathic complex

The complex was manufactured by the laboratory technician at the DUT Homoeopathic day clinic according to the German Homoeopathic Pharmacopoeia (method 6A) (See Appendix L).

3.5.3 Combined tautopathic and homoeopathic complex

This preparation was produced by combining the tautopathic preparation and homoeopathic complex in equal proportions, each of which were manufactured individually according to the German Homoeopathic Pharmacopeia (Method 6A) (See Appendix M).

3.5.4 Placebo preparation

This preparation was manufactured using the same batch of distilled water and ethanol used to manufacture the tautopathic and homoeopathic complex and the same water/ethanol concentration maintained thus excluding the influence of any extraneous variables and thus prepared in a manner such that it was indistinguishable from the other two preparations with respect to taste and appearance (See Appendix N).

3.6 Dosage and posology

Fifty millilitres of each participant's respective intervention (tautopathic, homoeopathic complex, combined tautopathic and homoeopathic complex or placebo in 30% water/ ethanol solution) was dispensed in a spray bottle container and participants were asked to spray the medication directly into the mouth three times a day, and again should they crave a cigarette. Each 50ml spray bottle

contained approximately seven hundred metered doses which was sufficient to last for the duration of the study. Should the participant have required more of their specific medication, they were able to collect a refill from the technician at the DUT Homoeopathic Day Clinic.

Participants will be asked not to use the spray directly before or after meals.

3.7 Consultation process

Advertisements requested potential participants phone the researcher directly; at which point they were asked certain screening questions to ascertain if they met the inclusion criteria (see 3.3.1 & 3.3.2). If they were found to be suitable their name, contact number and cigarette preference were recorded and an appointment was scheduled with the researcher for an initial consultation at the DUT Homoeopathic Day Clinic.

3.7.1 Procedure followed at first consultation:

Upon arrival the DUT Homoeopathic Day Clinic potential participants were asked to complete the following:

- Participant details sheet (Appendix A)
- Questionnaire on types of smokers (Appendix E)
- Questionnaire on hazards of smoking (Appendix C)
- Questionnaire on tolerance and dependence (Appendix D)

During the first consultation with the researcher the participant was issued with and completed the following documentation:

- Information sheet regarding the study (Appendix I)
- Informed consent document (Appendix B)
- Instructions on how to take the medication.

The researcher reconfirmed that the potential participant met all the required inclusion criteria and explained the study accordingly; the potential participant was issued the information sheet (Appendix I) and given an opportunity to ask any questions that he/she had regarding the study. Once the participant had agreed to participate, they signed the informed consent document (Appendix B) and were formally recruited for the study.

During the first consultation the researcher ascertained the participants smoking history (Appendix G) and conducted a brief consultation, checking the patients' vital signs to make sure that the participant was able to take part in the study. Thereafter the participants were given the opportunity to ask any questions regarding the study. Once the participants were satisfied with what was expected of them, they were taken back to the waiting room, at which point their respective intervention was dispensed according to the randomisation schedule. A follow up consultation was then scheduled for two weeks later.

3.7.2 Procedure followed at the second (final) consultation:

At the final consultation participants completed the tolerance dependence questionnaire once again as well as a perceptions questionnaire. Upon completion of the trial all participants were notified as to which intervention they had received; those who were within the placebo group were offered free treatment (tautopathic and homoeopathic complex).

3.8 Measurement tools

3.8.1 Smoking History

This questionnaire (See Appendix G) was given to the participant to complete at the start of the study, determined at what age they started smoking, which brand of cigarette they smoked, how many cigarettes they smoked daily and if they had ever tried to stop smoking before. This information was used by the statistician to evaluate the efficacy of the study.

3.8.2 Questionnaire on Health Hazards of Smoking

This questionnaire (See Appendix C) was completed at the start of the study and consisted of two parts; part A assessed the participants' own estimate of the risk of smoking and the subsequent change in their life expectancy. According to Goldstein (1988), an estimated decrease in life expectance due to health risks of smoking of 5-10 years is most accurate.

Part B consists of 17 commonly used statements that smokers use to explain their continued tobacco use. An endorsement to any of these statements indicated an underestimation of the health hazards of smoking and may be linked to a lack of motivation to quit smoking (Glodstein, 1988).

3.8.3 Tolerance Dependence Questionnaire

The Tolerance Dependence Questionnaire (See Appendix D) adapted from Goldstein (1988) was completed by each participant at the start and upon completion of the study. It consisted of nine questions with a multiple choice style answer format which provided information for statistical purposes. This questionnaire assessed issues such as number of cigarettes smoked daily, time between cigarettes, relative assessment of addiction to smoking and the participants desire to quit smoking.

3.8.4 Questionnaire on Types of Smokers

This questionnaire (See Appendix E) adapted from Goldstein 1988, was completed by participants at the start of the study divided smokers into three groups according to their main reason for smoking.

The Three Types of Smokers:

1) Positive-affect smoker, one who obtains pleasurable relaxation or stimulation from cigarettes.

2) Negative-affect smoker, one who uses smoking to relieve tension, depression, hostility or anxiety.

3) Habitual-addictive smoker, one who demonstrates an addictive type of smoking with automatic smoking behaviour.

This questionnaire consists of twenty three questions with graded answers, never, seldom, occasionally, frequently and always, these questions are divided into the different types of smoker responses, thus the highest score reflects which kind of smoker the participant is, i.e. what the main reason for smoking is for that individual.

3.8.5 Perceptions of Treatment

This questionnaire (See Appendix H) was used for statistical data; it recorded the number of cigarettes used by the participant at the start and end of the study. It determined if the participant believed the treatment helped them reduce their smoking or quit smoking all together, if the treatment helped them manage their nicotine withdrawal symptoms, and if they would continue to use the medication to help them stop smoking or control their nicotine cravings. The data generated indicated whether the treatment was perceived as helpful by each individual.

3.9 Data analysis

SPSS version 15.0 (SPSS Inc., Chicago, Illinois, USA) was used to analyse the data. A p value <0.05 was considered as statistically significant. Baseline data and demographics were compared between the four treatment groups to ensure that randomization was complete and that the groups are comparable at baseline using ANOVA tests in the case of quantitative data and Pearson's chi square tests where the data were categorical.

3.9.1 Procedure 1

Initially intra-group analyses were conducted to assess the effects of each treatment individually. For ordinal data the intra-group comparison between pre and post treatment was achieved using Wilcoxon-signed ranks tests.

3.9.2 Procedure 2

Intra-group analyses were conducted to compare the effect of each treatment with the placebo and with each other treatment. The change in the number of cigarettes smoked was recorded between baseline and end of study. This was compared between the four groups using a non parametric Kruskal-Wallis test.

3.9.3 Procedure 3

The binary and categorical outcome variables, such as self reported assessment of the effect of the medication, was compared between treatment groups using Pearson's chi square tests and Kruskal-Wallis tests as appropriate.

3.9.4 Procedure 4

The proportion actually quitting smoking in the study period was compared between the treatment groups using chi square tests.

3.9.5 Procedure 5

For ordinal variables such as the tolerance dependence questionnaire, the changes between pre and post treatment were computed, and compared between the treatment groups using Kruskal-Wallis tests.

The results were measured according to the information obtained from the tolerance dependence questionnaire, (See Appendix D) which was completed both before, and after the study and a perceptions questionnaire completed at the final consultation.

3.10 Ethics

The research proposal was approved by the DUT Faculty of Health Sciences Research & Ethics Committee. Participation was voluntary, no vulnerable groups utilized and informed consent was obtained from all participants who were free to

withdraw from the study at any stage. Although a placebo control was utilized the condition being treated was not considered to be of immediate high risk to the participant and the duration of the study was only two weeks; participants were informed that there was a 25% chance they would receive placebo before considering to participate and those who were allocated to the placebo group were offered free treatment once the study was un-blinded.

Chapter Four

Results

4.1 Criteria governing admissibility of the data

Data was obtained from the Tolerance Dependence Questionnaire, completed at the start and end of the study, the Perceptions Of Treatment Questionnaire completed at the end of the study and from the smoking history and types of smokers questionnaire completed at the start of the study.

4.2 Demographics

4.2.1 Gender

Forty participants were randomly allocated into four treatment groups. The gender distribution per-group is detailed in Table 4.1. There was no significant difference between the proportions of each gender between the four groups (p=0.347) and all groups had a higher proportion of males than females.

Table 4.1: Participants by gender and treatment group

		Group									
		Homeopathic complex only		Placebo		Complex and tautopathic preparation		Tautopathic preparation only		Total	
		Count	%	Count	%	Count	%	Count	%	Count	%
Sex	Female	2	20.0%	1	11.1%	5	45.5%	3	30.0%	11	27.5%
	Male	8	80.0%	8	88.9%	6	54.5%	7	70.0%	29	72.5%

Pearson's chi square = 3.305, p=0.347

4.2.2 Age and smoking history

There was no significant difference between the treatment groups with regard to age, age started smoking and number of cigarettes smoked per day (Table 2).

Table 4. 2: Mean (SD) age, age started smoking and number of cigarettes per day per treatment group

	Group					P value
	Homeopathic complex only	Placebo	Complex and tautopathic preparation	Tautopathic preparation only	Total	
Age	43.6 (11.9)	39.3 (15.5)	43.6 (10.3)	41.6 (14.4)	42.2 (12.7)	0.872
age started smoking	14.9 (3.8)	15.4 (2.5)	17.5 (6.1)	18.4 (2.2)	16.6 (4.2)	0.195
number cig smoked per day	22.3 (8.7)	17.7 (3.6)	19.4 (6.5)	20.4 (11.9)	20.0 (8.1)	0.668

4.2.3 Occupation

Table 4.3 shows the various occupations of the study participants by group.

Table 4.3: Occupations of the participants by group

		Group							
		Homeopathic complex only		Placebo		Complex and tautopathic preparation		Tautopathic preparation only	
		Count	%	Count	%	Count	%	Count	%
Occupation	Sales	3	30.0%	0	.0%	1	9.1%	0	.0%
	Manager	1	10.0%	1	11.1%	2	18.2%	0	.0%
	Self employed	3	30.0%	3	33.3%	3	27.3%	3	30.0%
	Service	1	10.0%	1	11.1%	1	9.1%	1	10.0%
	Housewife	0	.0%	1	11.1%	0	.0%	1	10.0%
	Unemployed	1	10.0%	2	22.2%	1	9.1%	0	.0%
	Security	0	.0%	0	.0%	0	.0%	1	10.0%
	Admin	1	10.0%	0	.0%	2	18.2%	2	20.0%
	Student	0	.0%	1	11.1%	1	9.1%	2	20.0%

4.2.4 Cigarette brands

Table 4.4 shows the cigarette brands smoked by study participants by group; the most popular brand of cigarette smoked was Peter Styvesant Red (33% of all participants) followed by Peter Styvesant Blue (23% of all participants).

Table 4.4: Cigarette brands smoked by the participants by group

		Group							
		Homeopathic complex only		Placebo		Complex and tautopathic preparation		Tautopathic preparation only	
		Count	%	Count	%	Count	%	Count	%
brand smoked	Styvesant red	6	60.0%	2	22.2%	1	9.1%	4	40.0%
	Styvesant blue	1	10.0%	3	33.3%	3	27.3%	2	20.0%
	Royals red	0	.0%	0	.0%	1	9.1%	1	10.0%
	Kent special	1	10.0%	1	11.1%	1	9.1%	1	10.0%
	Dunhill lights	1	10.0%	0	.0%	1	9.1%	1	10.0%
	Craven	0	.0%	0	.0%	1	9.1%	0	.0%
	Rothmans red	0	.0%	1	11.1%	2	18.2%	0	.0%
	Camel filter	0	.0%	0	.0%	0	.0%	1	10.0%
	Camel light	1	10.0%	0	.0%	0	.0%	0	.0%
	Ransom	0	.0%	1	11.1%	1	9.1%	0	.0%
	Marlboro	0	.0%	1	11.1%	0	.0%	0	.0%

4.3 The effect of the treatment interventions

4.3.1 Intra-group analyses-Tautopathic Group

The effectiveness of the tautopathic preparation in the treatment of nicotine withdrawal syndrome as defined by the tolerance dependence questionnaire and questionnaire on perception of results-was determined as follows:

Table 4.5 shows that there were statistically significant improvements in the responses of this group to 6 of the 9 the tolerance dependence questions.

Table 4.5: Comparison of pre and post responses to the Tolerance dependence questionnaire in the Tautopathic preparation group

		N	Mean Rank	Sum of Ranks	P value
How many cigarettes do you normally smoke per day? (post) - How many cigarettes do you normally smoke per day?	Negative Ranks	0	.00	.00	0.009
	Positive Ranks	8	4.50	36.00	
	Ties	2			
	Total	10			
When stressed how many cigarettes do you smoke? (post) - When stressed how many cigarettes do you smoke?	Negative Ranks	0	.00	.00	0.003
	Positive Ranks	10	5.50	55.00	
	Ties	0			
	Total	10			
Normally how often do you smoke? (post) - Normally how often do you smoke?	Negative Ranks	0	.00	.00	0.005
	Positive Ranks	10	5.50	55.00	
	Ties	0			
	Total	10			
Do you ever smoke one cigarette immediately after another? (post) - Do you ever smoke one cigarette immediately after another?	Negative Ranks	1	1.50	1.50	0.031
	Positive Ranks	6	4.42	26.50	
	Ties	3			
	Total	10			
Which cigarette of the day is most difficult to give up? (post) - Which cigarette of the day is most difficult to give up?	Negative Ranks	0	.00	.00	0.180
	Positive Ranks	2	1.50	3.00	
	Ties	8			
	Total	10			
How soon after you wake up do you smoke? (post) - How soon after you wake up do you smoke?	Negative Ranks	0	.00	.00	0.063
	Positive	4	2.50	10.00	

	Ranks				
	Ties	6			
	Total	10			
Do you smoke more frequently in the first hours after waking than during the rest of the day? (post) -Do you smoke more frequently in the first hours after waking than during the rest of the day?	Negative Ranks	3	3.00	9.00	0.103
	Positive Ranks	6	6.00	36.00	
	Ties	1			
	Total	10			
Do you find it difficult to refrain from smoking in public places where smoking is forbidden? (post) -Do you find it difficult to refrain from smoking in public places where smoking is forbidden?	Negative Ranks	0	.00	.00	0.027
	Positive Ranks	6	3.50	21.00	
	Ties	4			
	Total	10			
How badly do you want to quit? (post) -How badly do you want to quit?	Negative Ranks	4	2.50	10.00	0.046
	Positive Ranks	0	.00	.00	
	Ties	6			
	Total	10			

Negative ranks = post < pre

Positive ranks = post > pre

Ties= post = pre

Table 4.6 shows that the mean reduction in cigarettes was 11.1 (SD 6.5) with a range from 3 to 25 per day reduction. Thus all participants in this group showed a reduction in their cigarette consumption.

Table 4.6: Reduction in cigarettes in the Tautopathic group

N	Valid	10
	Missing	0
Mean		11.1000
Std. Deviation		6.45411
Minimum		3.00
Maximum		25.00

Table 4.7 shows that 80% of this group thought that the medication had assisted them to quit or cut down, 90% thought it reduced their cravings, 90% would continue to use it and 70% had previous attempts to quit.

Table 4.7: Responses of the tautopathic treatment group to the questionnaire on perception of results

	yes		No	
	Count	Row N %	Count	Row N %
Do you think the medication has assisted you to quit or cut down?	8	80.0%	2	20.0%
Do you think the medication has assisted by reducing your cravings?	9	90.0%	1	10.0%
Would you continue to use the medication to control your nicotine cravings?	9	90.0%	1	10.0%
Previous attempts to stop smoking	7	70.0%	3	30.0%

4.3.2 Intra group analysis – Homoeopathic Complex

The effectiveness of a homoeopathic complex (Caladium seguinum 30CH, Nux vomica 30CH, Staphysagria delphinium 30 CH) in the treatment of nicotine withdrawal syndrome as determined by the tolerance dependence questionnaire and questionnaire on perception of results was determined as follows:

Table 4.8 shows that there were statistically significant improvements in the responses of this group to 6 of the 9 the tolerance dependence questions.

Table 4.8: Comparison of pre and post responses to the Tolerance dependence questionnaire in the Homeopathic complex group

		N	Mean Rank	Sum of Ranks	P value
How many cigarettes do you normally smoke per day? (post) - How many cigarettes do you normally smoke per day?	Negative Ranks	0	.00	.00	0.010
	Positive Ranks	8	4.50	36.00	
	Ties	2			

	Total	10			
When stressed how many cigarettes do you smoke? (post) - When stressed how many cigarettes do you smoke?	Negative Ranks	0	.00	.00	0.011
	Positive Ranks	8	4.50	36.00	
	Ties	2			
	Total	10			
Normally how often do you smoke? (post) - Normally how often do you smoke?	Negative Ranks	0	.00	.00	0.010
	Positive Ranks	8	4.50	36.00	
	Ties	2			
	Total	10			
Do you ever smoke one cigarette immediately after another? (post) - Do you ever smoke one cigarette immediately after another?	Negative Ranks	0	.00	.00	0.027
	Positive Ranks	6	3.50	21.00	
	Ties	4			
	Total	10			
Which cigarette of the day is most difficult to give up? (post) - Which cigarette of the day is most difficult to give up?	Negative Ranks	2	1.50	3.00	1.000
	Positive Ranks	1	3.00	3.00	
	Ties	7			
	Total	10			
How soon after you wake up do you smoke? (post) - How soon after you wake up do you smoke?	Negative Ranks	0	.00	.00	0.083
	Positive Ranks	3	2.00	6.00	
	Ties	7			
	Total	10			
Do you smoke more frequently in the first hours after waking than during the rest of the day? (post) - Do you smoke more frequently in the first hours after waking than during the rest of the day?	Negative Ranks	0	.00	.00	0.038
	Positive Ranks	5	3.00	15.00	
	Ties	5			
	Total	10			
Do you find it difficult to refrain from smoking in public places where smoking is forbidden? (post) - Do you find it difficult to refrain from smoking in public places where smoking is forbidden?	Negative Ranks	0	.00	.00	0.024
	Positive Ranks	6	3.50	21.00	
	Ties	4			
	Total	10			

How badly do you want to quit? (post) - How badly do you want to quit?	Negative Ranks	1	3.00	3.00	0.461
	Positive Ranks	3	2.33	7.00	
	Ties	6			
	Total	10			

Negative ranks = post < pre

Positive ranks = post > pre

Ties= post = pre

Table 4.9 shows that the mean reduction in cigarettes was 10.6 (SD 9.4) with a range from 0 to 27 per day reduction. Thus not all participants in this group showed a reduction in their cigarette consumption but there was a wide range in the effect.

Table 4.9: Reduction in cigarettes in the Homeopathic complex group

N	Valid	10
	Missing	0
Mean		10.6000
Std. Deviation		9.44222
Minimum		.00
Maximum		27.00

Table 4.10 shows that 50% of this group thought that the medication had assisted them to quit or cut down, 40% thought it reduced their cravings, 70% would continue to use it and 100% had previous attempts to quit.

Table 4.10: Responses of the Homeopathic complex group to the questionnaire on perception of results

	yes		No	
	Count	Row N %	Count	Row N %
Do you think the medication has assisted you to quit or cut down?	5	50.0%	5	50.0%
Do you think the medication has assisted by reducing your cravings?	4	40.0%	6	60.0%
Would you continue to use the medication to control your nicotine cravings?	7	70.0%	3	30.0%

previous attempts to stop smoking	10	100.0%	0	.0%

4.3.4 Intra group analysis – Combination of Tautopathy and Homoeopathic Complex

The combined effect of a tautopathic preparation and homoeopathic complex (Caladium seguinum 30 CH, Nux vomica 30 CH and Staphysagria delphinium 30 CH) in the treatment of nicotine withdrawal syndrome as determined by the tolerance dependence questionnaire and questionnaire on perception of results was determined as follows:

Table 4.11 shows that there were statistically significant improvements in the responses of this group to 4 of the 9 the tolerance dependence questions.

Table 4.11: Comparison of pre and post responses to the Tolerance dependence questionnaire in the combined group

		N	Mean Rank	Sum of Ranks	P value
How many cigarettes do you normally smoke per day? (post) - How many cigarettes do you normally smoke per day?	Negative Ranks	0	.00	.00	0.015
	Positive Ranks	7	4.00	28.00	
	Ties	4			
	Total	11			
When stressed how many cigarettes do you smoke? (post) - When stressed how many cigarettes do you smoke?	Negative Ranks	0	.00	.00	0.010
	Positive Ranks	8	4.50	36.00	
	Ties	3			
	Total	11			
Normally how often do you smoke? (post) - Normally how often do you smoke?	Negative Ranks	0	.00	.00	0.010
	Positive Ranks	8	4.50	36.00	
	Ties	3			
	Total	11			
		0	.00	.00	0.059

Do you ever smoke one cigarette immediately after another? (post) - Do you ever smoke one cigarette immediately after another?	Negative Ranks				
	Positive Ranks	4	2.50	10.00	
	Ties	7			
	Total	11			
Which cigarette of the day is most difficult to give up? (post) - Which cigarette of the day is most difficult to give up?	Negative Ranks	1	1.00	1.00	0.317
	Positive Ranks	0	.00	.00	
	Ties	10			
	Total	11			
How soon after you wake up do you smoke? (post) - How soon after you wake up do you smoke?	Negative Ranks	0	.00	.00	0.102
	Positive Ranks	3	2.00	6.00	
	Ties	8			
	Total	11			
Do you smoke more frequently in the first hours after waking than during the rest of the day? (post) - Do you smoke more frequently in the first hours after waking than during the rest of the day?	Negative Ranks	0	.00	.00	0.063
	Positive Ranks	4	2.50	10.00	
	Ties	7			
	Total	11			
Do you find it difficult to refrain from smoking in public places where smoking is forbidden? (post) - Do you find it difficult to refrain from smoking in public places where smoking is forbidden?	Negative Ranks	0	.00	.00	0.017
	Positive Ranks	7	4.00	28.00	
	Ties	4			
	Total	11			
How badly do you want to quit? (post) - How badly do you want to quit?	Negative Ranks	2	2.25	4.50	0.414
	Positive Ranks	1	1.50	1.50	
	Ties	8			
	Total	11			

Negative ranks = post < pre

Positive ranks = post > pre

Ties= post = pre

Table 4.12 shows that the mean reduction in cigarettes was 8.73 (SD 6.10) with a range from -1 to 17 per day reduction. Thus not all participants in this group showed a reduction in their cigarette consumption.

Table 4.12: Reduction in cigarettes in the combined group

Reduction in cigarettes

N	Valid	11
	Missing	0
Mean		8.7273
Std. Deviation		6.10067
Minimum		-1.00
Maximum		17.00

Table 4.13 shows that 73% of this group thought that the medication had assisted them to quit or cut down, 73% thought it reduced their cravings, 73% would continue to use it and 100% had previous attempts to quit.

Table 4.13: Responses of the combined treatment group to the questionnaire on perception of results

	yes		No	
	Count	Row N %	Count	Row N %
Do you think the medication has assisted you to quit or cut down?	8	72.7%	3	27.3%
Do you think the medication has assisted by reducing your cravings?	8	72.7%	3	27.3%
Would you continue to use the medication to control your nicotine cravings?	8	72.7%	3	27.3%
previous attempts to stop smoking	11	100.0%	0	.0%

4.3.5 Intra group analysis – Placebo Group

The effectiveness of placebo in the treatment of nicotine withdrawal syndrome as determined by the tolerance dependence questionnaire and questionnaire of perception of results was determined as follows:

Table 4.14 illustrates that there were statistically significant improvements in the responses of the placebo group to 5 of the 9 the tolerance dependence questions.

Table 4.14: Comparison of pre and post responses to the Tolerance dependence questionnaire in the placebo group

		N	Mean Rank	Sum of Ranks	P value
How many cigarettes do you normally smoke per day? (post) - How many cigarettes do you normally smoke per day?	Negative Ranks	0	.00	.00	0.009
	Positive Ranks	8	4.50	36.00	
	Ties	1			
	Total	9			
When stressed how many cigarettes do you smoke? (post) - When stressed how many cigarettes do you smoke?	Negative Ranks	0	.00	.00	0.011
	Positive Ranks	8	4.50	36.00	
	Ties	1			
	Total	9			
Normally how often do you smoke? (post) - Normally how often do you smoke?	Negative Ranks	0	.00	.00	0.017
	Positive Ranks	7	4.00	28.00	
	Ties	2			
	Total	9			
Do you ever smoke one cigarette immediately after another? (post) - Do you ever smoke one cigarette immediately after another?	Negative Ranks	0	.00	.00	0.010
	Positive Ranks	8	4.50	36.00	
	Ties	1			
	Total	9			
Which cigarette of the day is most difficult to give up? (post) - Which cigarette of the day is most difficult to give up?	Negative Ranks	1	1.00	1.00	0.077
	Positive Ranks	4	3.50	14.00	
	Ties	4			
	Total	9			
How soon after you wake up do you smoke? (post) - How soon after you wake up do you smoke?	Negative Ranks	2	1.50	3.00	0.113
	Positive	4	4.50	18.00	

	Ranks				
	Ties	3			
	Total	9			
Do you smoke more frequently in the first hours after waking than during the rest of the day? (post) - Do you smoke more frequently in the first hours after waking than during the rest of the day?	Negative Ranks	1	1.00	1.00	0.078
	Positive Ranks	4	3.50	14.00	
	Ties	4			
	Total	9			
Do you find it difficult to refrain from smoking in public places where smoking is forbidden? (post) - Do you find it difficult to refrain from smoking in public places where smoking is forbidden?	Negative Ranks	0	.00	.00	0.010
	Positive Ranks	8	4.50	36.00	
	Ties	1			
	Total	9			
How badly do you want to quit? (post) - How badly do you want to quit?	Negative Ranks	1	1.00	1.00	0.141
	Positive Ranks	3	3.00	9.00	
	Ties	5			
	Total	9			

Negative ranks = post < pre

Positive ranks = post > pre

Ties= post = pre

Table 4.15 shows that the mean reduction in cigarettes was 11.3 (SD 6.3) with a range from 2 to 20 per day reduction. Thus all participants in this group showed a reduction in their cigarette consumption.

Table 4.15: Reduction in cigarettes in the placebo group

N	Valid	9
	Missing	0
Mean		11.3333
Std. Deviation		6.28490
Minimum		2.00
Maximum		20.00

Table 4.16 shows that 89% of this group thought that the medication had assisted them to quit or cut down, 89% thought it reduced their cravings, 89% would continue to use it and 89% had previous attempts to quit.

Table 4.16: Responses of the placebo group to the questionnaire on perception of results

	yes		No	
	Count	Row N %	Count	Row N %
Do you think the medication has assisted you to quit or cut down?	8	88.9%	1	11.1%
Do you think the medication has assisted by reducing your cravings?	8	88.9%	1	11.1%
Would you continue to use the medication to control your nicotine cravings?	8	88.9%	1	11.1%
previous attempts to stop smoking	8	88.9%	1	11.1%

4.4 The effect of the treatment interventions -Inter-group analyse

The effectiveness of the three interventions (tautopathic preparation, homoeopathic complex and a combination of tautopathic and homoeopathic complex) as determined by the Tolerance Dependence Questionnaire and Perception Of Results were compared:

In terms of the median number of cigarettes reduced there was no statistically significant difference between the groups (p=0.873). Although the tautopathic group showed the largest reduction, this difference could have arisen by chance as the standard deviations and ranges are wide. Therefore all treatments are equally effective in terms of reducing actual number of cigarettes smoked.

Table 4.17: Reduction in cigarettes smoked by treatment group

Group	Mean	Std. Deviation	Median	Range
Homeopathic complex only	10.6000	9.44222	8.0000	27.00
Placebo	11.3333	6.28490	10.0000	18.00
Complex and tautopathic preparation	8.7273	6.10067	8.0000	18.00
Tautopathic preparation only	11.1000	6.45411	11.0000	22.00
Total	10.3750	6.99702	9.5000	28.00

P=0.873

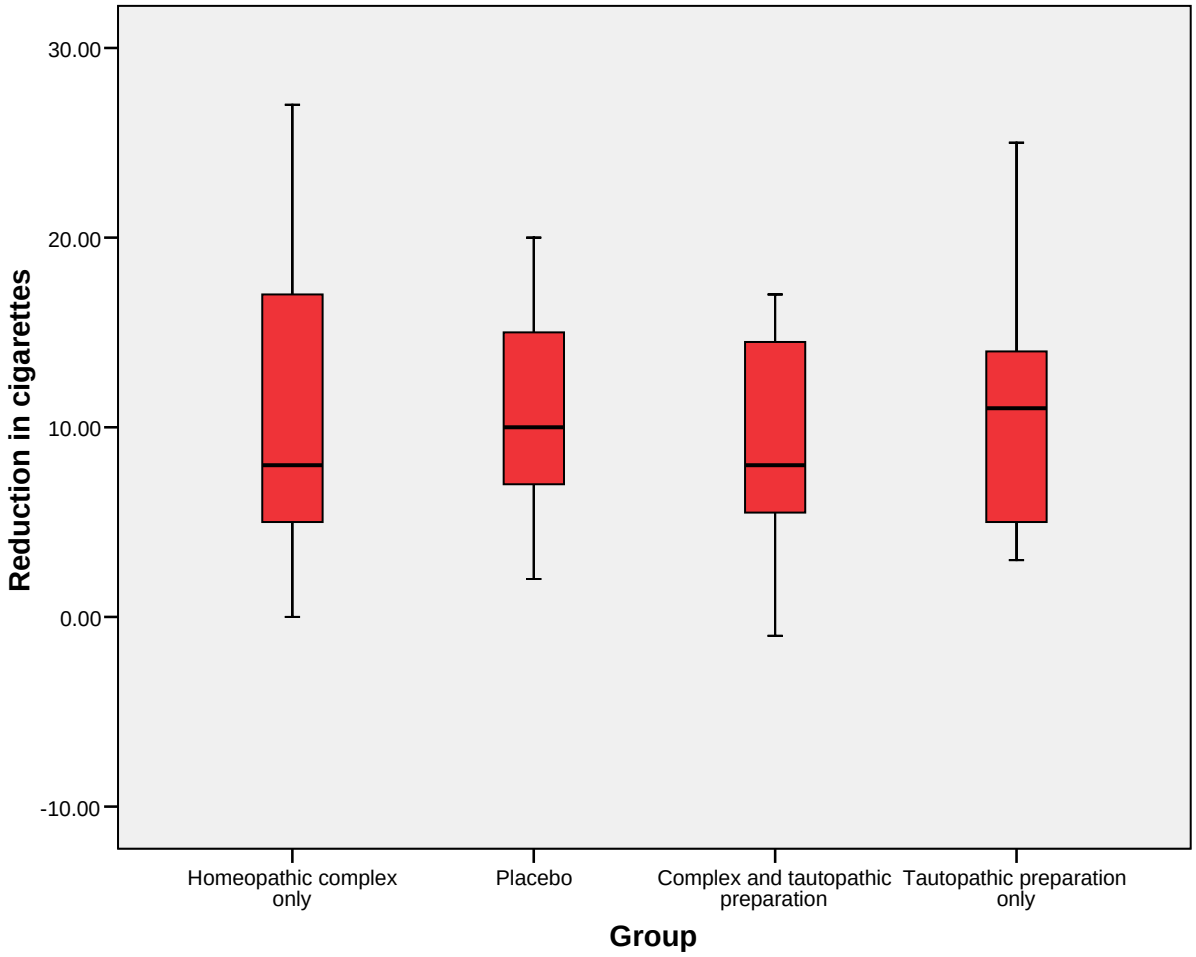

Figure 4.1: Box and whisker plot of reduction in number of cigarettes by group

Additionally, when the percentage of cigarettes they are currently smoking compared to their baseline was compared between the groups, there was no significant difference between the groups (p=0.610). The placebo group was smoking on average one third of their starting amount, while the other groups were smoking

between 46% and 56% of their baseline amount. The differences could have been due to chance since there was no statistical significance between the groups.

Table 4.18: Percentage of baseline currently smoked by treatment group

Group	Mean	Std. Deviation	Median	Range
Homeopathic complex only	50.3860	33.39991	49.6491	90.00
Placebo	34.7531	32.23313	33.3333	90.00
Complex and tautopathic preparation	55.5023	29.78604	57.1429	90.88
Tautopathic preparation only	45.9306	18.04250	50.0000	51.25
Total	47.1617	28.83363	50.0000	105.88

P=0.610

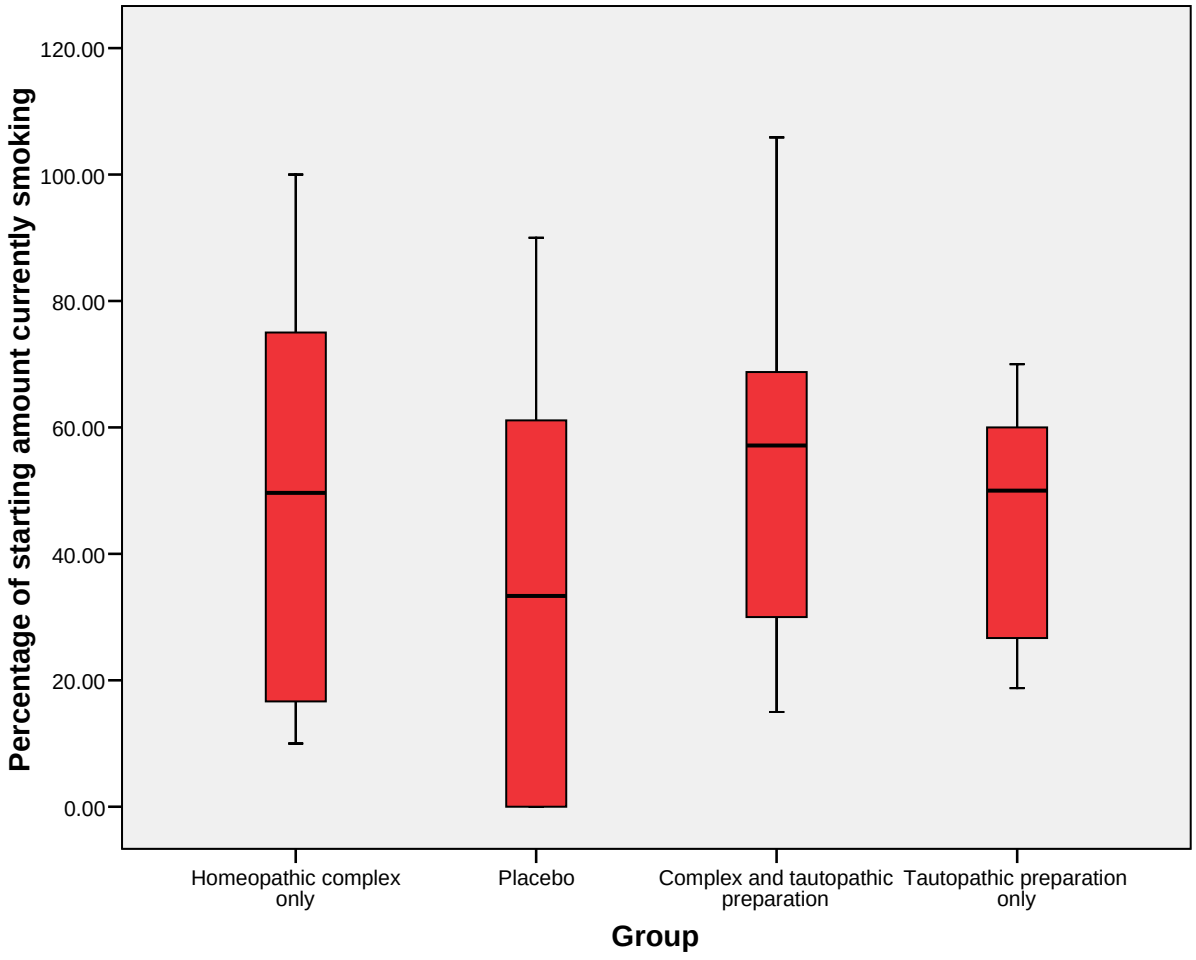

Figure 4.2: Box and whisker plot of percentage of baseline cigarettes currently smoked by group

Only three participants quit smoking (currently reported smoking 0% of their baseline). All three were in the placebo group. Table 4.19 shows the results by group. A Kruskal-Wallis test showed that there was a statistically significant difference in results between the four groups overall (p=0.022). The groups which differed from each other were placebo and combined group (p= 0.024), placebo and homeopathic group (p=0.026). Thus the placebo group had significantly better results than each of these two groups.

Table 4.19: Cross tabulation of results of the trial by group

			quit				Total
			Quit	Reduced	No change	Increased	
Group	Homeopathic complex only	Count	0	8	2	0	10
		% within Group	.0%	80.0%	20.0%	.0%	100.0%
	Placebo	Count	3	6	0	0	9
		% within Group	33.3%	66.7%	.0%	.0%	100.0%
	Complex and tautopathic preparation	Count	0	9	1	1	11
		% within Group	.0%	81.8%	9.1%	9.1%	100.0%
	Tautopathic preparation only	Count	0	10	0	0	10
		% within Group	.0%	100.0%	.0%	.0%	100.0%
Total		Count	3	33	3	1	40
		% within Group	7.5%	82.5%	7.5%	2.5%	100.0%

Table 4.20 shows the chi square tests for comparison of the perception of results by group. Only the question on reduction of cravings was different between the groups (p=0.045) and this should be interpreted with caution since the assumptions of the chi square test was not met (50% of cells had expected counts less than 5). However, it is clear that the tautopathic group had the most favourable response to

this question, followed by the placebo group, the combined group and the homeopathic group had the least percentage of favourable responses.

Table 4.20: Cross tabulation of perception of results by group

		Group								P value
		Homeopathic complex only		Placebo		Complex and tautopathic preparation		Tautopathic preparation only		
		Count	%	Count	%	Count	%	Count	%	
Do you think the medication has assisted you to quit or cut down?	yes	5	50.0%	8	88.9%	8	72.7%	8	80.0%	0.258
	no	5	50.0%	1	11.1%	3	27.3%	2	20.0%	
Do you think the medication has assisted by reducing your cravings?	yes	4	40.0%	8	88.9%	8	72.7%	9	90.0%	0.045
	no	6	60.0%	1	11.1%	3	27.3%	1	10.0%	
Would you continue to use the medication to control your nicotine cravings?	yes	7	70.0%	8	88.9%	8	72.7%	9	90.0%	0.560
	no	3	30.0%	1	11.1%	3	27.3%	1	10.0%	
previous attempts to stop smoking	yes	10	100.0%	8	88.9%	11	100.0%	7	70.0%	0.079
	no	0	.0%	1	11.1%	0	.0%	3	30.0%	

The variables of the TDQ were treated as ordinal (except for question 5) and the difference between pre and post treatment was computed (post minus pre). This difference was compared between the treatment groups using Kruskal-Wallis tests. An increase meant an improvement in all questions except for question 9 where the scoring was opposite to that of the other questions. For question 9 a decrease meant improvement. None of the differences were significantly different between the four treatment groups, as shown in Table 4.21.

Table 4.21: Median change in TDQ questions by group

	Group				P value
	Homeopathic complex only	Placebo	Complex and tautopathic preparation	Tautopathic preparation only	
Change in TDQ1	1.00	1.00	1.00	1.00	0.760
Change in TDQ2	1.00	1.00	1.00	1.00	0.696
Change in TDQ3	1.00	2.00	2.00	2.00	0.584
Change in TDQ4	1.00	2.00	.00	1.50	0.119
Change in TDQ5	.00	.00	.00	.00	0.123
Change in TDQ6	.00	.00	.00	.00	0.876
Change in TDQ7	.50	.00	.00	1.00	0.912
Change in TDQ8	1.00	4.00	2.00	1.00	0.244
Change in TDQ9	.00	.00	.00	.00	0.101

4.5 Summary of results

All treatments showed an average reduction in number of cigarettes smoked in the study period, generally favourable perception of results, and improved tolerance. However, when results were compared between treatment groups, there was not enough statistical evidence to favour one treatment over the other in terms of tolerance to nicotine, and perception of results. In addition, there was no statistically significant difference between the groups in terms of absolute number of cigarettes reduced or percentage of baseline currently smoked, however, when results were categorised into quit, reduced, no change and increased, there was a statistically significantly advantage for the placebo group compared with the homeopathic group and the combined group but not compared with the tautopathic group. Therefore there was a large placebo effect in this study, but also a suggestive beneficial effect

of the tautopathic treatment which warrants further study. The homeopathic treatment on its own or combined with the tautopathic treatment was worse than or equivalent to the placebo.

Chapter five

Discussion

5.1 Introduction

All four research groups experienced a statistically significant reduction in the amount of cigarettes smoked, favourable perceptions of their response to treatment and improved tolerance. Statistically however when the groups were compared with each other they were similar with respect to their tolerance to nicotine, perception of response to treatment and reduction in amount smoked.

5.2 Tautopathic group

All participants in the tautopathic group experienced a statistically significant reduction in their cigarette consumption; on average they achieved a mean reduction of 11.1 cigarettes smoked per day (Table 4.6) (median reduction of 1.1).

Eighty percent (Table 4.7) of participants in this group reported that the intervention had assisted them in quitting or reducing the amount of cigarettes smoked, ninety percent (Table 4.7) of the participants in this group reported that the intervention assisted in reducing cravings for cigarettes and ninety percent (Table 4.7) would continue using the intervention to control their cravings.

Furthermore this group experienced statistically significant improvements in six of the nine questions in the Tolerance Dependence Questionnaire.

5.3 Homoeopathic complex group

Although a statistically significant reduction in the number of cigarettes smoked was achieved (mean reduction of 10.6 cigarettes a day) (median reduction of 8) only fifty

percent (Table 4.10) of this group reported that the intervention assisted them in quitting or reducing the amount smoked and only forty percent (Table 4.10) believed that the intervention assisted in reducing craving for cigarettes with seventy percent (Table 4.10) being prepared to continue using the intervention to control cravings.

Statistically significant improvements in six of the nine questions of the Tolerance Dependence Questionnaire were noted for this group.

5.4 Combined Tautopathic and Homoeopathic complex group

Although statistically significant, participants in this group only achieved on average reduction of 8.73 cigarettes smoked (Table 4.12) (median reduction of 8).

Seventy three percent (Table 4.12) of the participants reported that the intervention had assisted them to quit or reduce their number of cigarettes smoked and, seventy three percent (Table 4.12) experienced a reduction in cravings and would also continue to use the medication to control their nicotine cravings respectively.

This group showed less favourable results with respect to the Tolerance Dependence Questionnaire, with only four of the questions showing statistically significant improvement in contrast with that of the tautopathic and homoeopathic complex groups.

5.5 Placebo group

Those taking placebo achieved a mean reduction in number of cigarettes smoked of 11.3 per day (Table 4.15) (median reduction of 10).

Eighty nine percent of participants reporting that the intervention assisted them to reduce their cigarette consumption assisted in reducing their craving for nicotine and would continue to use the intervention to control their nicotine cravings respectively.

In terms of responses to the Tolerance Dependence Questionnaire this group experienced statistically significant improvements in five of the nine questions i.e. more favourable than the combined group (4/9) but less than that of the tautopathic and homoeopathic complex group (6/9) respectively.

5.6 Comparison of the four interventions

Although all groups achieved a statistically significant reduction in the number of cigarettes smoked per day, improved tolerance and favourable perception of results, none of the groups proved statistically superior in this regard.

The data analysis does suggest however that tautopathy as an intervention may warrant further investigation as there is some suggestion of its efficacy; the tautopathic group having the highest reduction in cigarettes smoked (median) of all four groups in addition the tautopathic group being the only group which was not shown to be inferior to placebo with respect to current number of cigarettes smoked in relation to baseline amount (percentage) and the only group not inferior to placebo when categorising results into ‘quit’, ‘reduced’, ‘no change’ and ‘increased’. The tautopathic group also experienced the most favourable response to the question ‘Do you think the medication has assisted you by reducing your cravings’. The apparent efficacy of the tautopathic intervention is however overshadowed by the undeniable influence of the placebo effect in this study.

Table 5.1 – Comparison of treatment interventions

Group	Mean & (median) reduction in smoking	% had reduced cravings	% who would continue using intervention	% assisted in reducing quitting	No. Of Questions improved in TDQ
Tautopathic	11.1 (11)	90%	90%	80%	6/9
Complex	10.6 (8)	40%	70%	50%	6/9

Combined	**8.73 (8)**	**73%**	**73%**	**73%**	**4/9**
Placebo	**11.3 (10)**	**89%**	**89%**	**89%**	**5/9**

Based on the findings above, hypotheses 1, 2 and 3 were proven (accepted) i.e. the tautopathic, homoeopathic complex and combined approach were effective in the reducing the number of cigarettes smoked daily and the effects of nicotine withdrawal syndrome. However hypothesis 4 was disproven (rejected) i.e. none of the 'active' interventions proved to be superior to placebo in this regard. Hypothesis 5 was also disproved (rejected); the effectiveness of the combined group proving to be statistically no different from that of the other two interventions (or that of placebo), furthermore although not statistically significant the data is clearly suggestive that the combined approach is in fact the least effective of the four interventions.

5.7 Comparison of findings with related research

Lachman-Maharaj (2002) used a homoeopathic complex (the same complex used in this study) as well as an individually selected homoeopathic similimum and placebo; the results obtained were comparable to this study in that all three groups achieved a statistically significant reduction in the number of cigarettes smoked but no statistically significant difference between the groups. However in the Lachman-Maharaj study (2002) the group which achieved the greatest reduction was the homoeopathic complex with a mean reduction of 4.65 cigarettes per day. The present study achieved a mean reduction of 10.6 cigarettes per day (using exactly the same Homoeopathic Complex), generally all four groups in this study achieved significantly higher reductions in the number of cigarettes smoked than that of the Lachman-Maharaj (2002) study using the same measurement tools. This phenomenon is suggestive of the fact that the novel method of administration used in this study (oral spray) may have been a contributing factor to the generally superior results achieved.

Hellberg (2001) completed a study using a dissimilar homoeopathic complex and a placebo but, applied similar measurement tools to the current study. The findings were similar to that of the current study in that a statistically significant improvement

was achieved by both groups but neither group was shown to be superior in this regard.

Pautz (1998) used a combination of isotherapy and homoeopathic similimum and a placebo group; her study demonstrated superior positive results with a mean reduction of cigarettes of 19.36 and 11.5 per day respectively using interventions in pill and granular forms. Pautz conducted her study over a three month period however, it could thus be argued that had the current study been extended from two weeks to three months superior results may have been achieved i.e. allowing the participants more time to further reduce their cigarette consumption.

De la Rouviere (1996) used acupuncture and homoeopathic treatment to help people stop smoking, no placebo group was used. Both treatment groups were found to be effective in helping people stop smoking over a three month period, with the acupuncture group demonstrating a quit rate of 33% versus the homoeopathic group which demonstrated a quit rate of 40%. De la Rouviere used a type of hetero-isopathy, whereby a mother tincture was manufactured from the un-combusted contents (without filter or paper) of the same brand and strength of cigarette that each participant smoked; this is dissimilar to the current study which made use of the combusted cigarette including the paper and parts of the used filter of the participants make and strength of cigarette to manufacture a trituration in lactose that was infused with the combusted cigarettes' main and side stream smoke.

The use of the combusted cigarette along with infusing the lactose of the trituration incorporated all aspects and chemical changes that occur in the act of smoking into the preparation used; the smoke (main stream and side stream), the added components of the cigarette paper and filter along with the chemical changes that occur within a cigarette as it combusts. The trituration process used in the current study is considered to be superior to the manufacture of a tincture as it incorporates both the water soluble and insoluble components into the preparation. This method of preparation can be argued as being superior to the preparation used in the De la Rouviere (1996) study as it is a more complex and complete representation of the smoking process in its chemical entirety. This could be a contributing factor to the slightly more positive outcome of the current study which demonstrated rapid results within the two week time frame with a mean reduction of 11.1 per day, improvements

in 6/9 questions of the Tolerance Dependence Questionnaire, 90% of participants reporting reduced cravings, 80% of participants reporting the medication helped them to reduce their daily cigarettes and 90% of participants reported they would continue to use the medication to further reduce their daily cigarette use.

5.8 Placebo effect and other variables contributing to positive outcome

The strong influence of the placebo effect in homoeopathic trials on nicotine withdrawal syndrome is well documented in the literature; all three interventions applied by Lachman-Maharaj (2002)(homoeopathic similimum, homoeopathic complex and placebo) resulted in statistically significant improvements in tobacco addiction, none of the interventions proving superior to another, similarly Hellberg (2001) found both a homoeopathic complex and placebo to be significantly effective in the management of cigarette addiction, neither group being superior in doing so. Although Pautz (1998) demonstrated that Isotherapy and a homoeopathic intervention to be superior to placebo in managing cigarette addiction, the placebo group alone did too demonstrate a statistically significant effect in this regard. The strong placebo effect encountered in this study is thus well described and confirmed in multiple clinical trials on tobacco addiction.

One of the prerequisites of this study was that the participants had to want to stop smoking; each participant demonstrated this desire and commitment by taking the time to make the appointment with the researcher fill in all the documentation and conform to the study requirements. The influence of willpower and determination to quit may have significantly influenced the outcomes of all four groups.

A factor that could explain the marked placebo effect more than will power alone is that this study differed significantly from that of the related studies; the method of administration of the interventions being in oral spray format which was administrated three times daily and upon demand should it be required with the onset of nicotine cravings. It could be argued that this mode of administration may also have satisfied the participants need to be doing something with their hands instead of handling a cigarette, it could also be argued that the spray distracted the individual from thinking of having a cigarette and provided an immediate substitute for a habitual process. This combination of substituting the physical act of smoking, the use of the hands and the immediate ability to substitute the habitual action of

smoking with an oral spray may have contributed to the evidently strong placebo effect noted in this study. The possible influence of this mode of administration is supported by the comparison with the Lachman-Maharaj (2002) study which using the same measurement tools and the identical homoeopathic complex achieved a much smaller reduction in the number of cigarettes smoked i.e. 4.65 versus 10.6 for the respective homoeopathic complex groups.

Although the greatest amount of care was taken not to provide counselling or a sense of accountability to the researcher so as to not influence the outcome of the intervention it is possible that through the consultation process the participants may have felt a desire to be helpful or even a sense of accountability knowing they would have to have a final consultation with the researcher at which stage their smoking would be quantified.

This can be explained as the Hawthorne effect which is described as the situation where the participant would alter their behaviour as a result of being observed. The difference between a placebo effect and the Hawthorne effect is that a placebo response would have long lasting effect whilst the Hawthorne response is short lived and diminishes as soon as the participant is no longer being observed (Leonard, 2008) such a distinction would only have been possible in a study with a longer observation period; the current study being only 2 weeks in duration made this distinction impossible.

5.9 Critical reflection

There were a number of positive outcomes in this study, with a majority of the participants finding their treatment to be helpful and many reporting they would continue to use the intervention to help them reduce their cravings, an outcome however that could also be due to the unique mode of administration used.

The measurement tools used in this study where similar to those used in previous studies and were effective in quantifying and determining effectiveness in keeping with the main aim of the study, namely reducing the number of cigarettes smoked, but perhaps more emphasis could have been placed on some of the lesser benefits of the study such as the reduction of each specific anticipated symptom of nicotine

withdrawal syndrome as outlined in the DSM IV (1995). In hind sight it would have been beneficial to ask more specific questions especially describing each participant's withdrawal symptoms or lack thereof and details of their nicotine craving which would provide valuable insight to future studies particularly homoeopathic studies. The use of carbon monoxide concentrations on expired air or salivary cotinine levels would provide a much improved objective measurement which would rule out any chance of dishonest responses from the participants, such objective measures should be considered in future studies.

Tautopathic treatment in particular warrants further study, the concept of tautopathy itself, whereby a toxic substance such as nicotine or tobacco can be manufactured in a specific way (by means of homoeopathic principles) and be used to treat years of abuse of the substance such as in the case of smoking, particularly in light of the positive outcome of this study warrants further exploration. The eliminatory effect of Tautopathy or the undoing of symptoms caused by substance abuse, provide many questions which would benefit from informative studies or clinical trials.

The use of the Tautopathic preparation including the chemicals found in the cigarette paper and filter as well as using the smoke infused lactose and combusted portion of the cigarette along with the mode of administration (oral spray) have produced statistically superior results and this warrants further studies.

Chapter Six

6.1 Conclusion

The study concludes that each of the four subject groups proved to be marginally successful in aiding the participant to cease smoking, with the results showing a significantly positive perception of the participants to the interventions used. This could be largely due to the mode of application of the interventions which would serve the participants need to be doing something with their hands and also doing something with their mouths, both of which would normally be functioning whilst smoking.

The study had a pre requisite that each individual had too have the express desire to want to stop smoking; this could have greatly influenced the positive response to treatment.

6.2 Recommendations

- The mode of administration was found to be most useful, further trials conducted to treat nicotine withdrawal syndrome would derive benefit from using this form of administering a test substance.
- The statistical analysis would be more accurate if a larger test group were utilised, thus it is recommended to increase the sample size of participants.
- The study should be repeated using more participants over a longer time period of 3 months with weekly follow ups.
- Salivary cotinine levels and carbon monoxide concentrations in expired air would be an extremely valuable tool of measurement for any future and similar studies as this would eliminate subjective data and offer better quantitative data of results.

- With respect to future tautopathic studies researchers should consider the merit of exploring potencies other than that of 6CH used in this study.
- A more comprehensive approach is needed when dealing with any form of addiction, a more positive response may have been achieved if the participants were also given behavioural and cognitive therapy.
- Future studies involving the aiding of smoking cessation should include weekly counselling and follow up sessions in order for the treatment to be more successful.
- Similar studies should be conducted with pregnant women in mind, as there has been a general interest expressed by pregnant and smoking women who cannot use orthodox forms of NRT during pregnancy, but who would benefit hugely from the safer effects of homoeopathic treatment to help them stop smoking and reduce their nicotine withdrawal syndrome.

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Appendix A:

Research patient details

Private and confidential

Title: _____ Name: _____ Surname: _____

Date of Birth: _____ Gender: ___ Occupation:_____

Residential address: _____

_____ Code: _____

Postal address: _____

_____ Code: _____

Tel: (H) _____ (W) _____ Cell _____

Medical history

Operations:

_____

_____

_____

Serious illnesses:

_____

_____

_____

Current medication:

_____

_____

_____

Other medication (supplements, vitamins, contraception etc.)

_____

_____

Appendix B:

INFORMED CONSENT FORM:

(To be completed in duplicate by participant)

TITLE OF THE RESEARCH PROJECT:

The effectiveness of a homoeopathic complex (Caladium seguinum 30CH, Nux vomica 30CH and Staphysagria delphinium 30CH) compared to a tautopathic preparation of the cigarette smoked in the management of nicotine withdrawal syndrome.

NAME OF SUPERVISOR:

Dr David Naude Tel: (031) 3732541

NAME OF RESEARCH STUDENT:

Catherine Riggien 0722734417

DATE:

PLEASE CIRCLE THE APPROPRIATE ANSWER:

1. Have you read the research information sheet? **YES/NO**
2. Have you had opportunity to ask questions regarding this study? **YES/NO**
3. Have you received satisfactory answers to your questions? **YES/NO**
4. Have you had an opportunity to discuss this study? **YES/NO**
5. Who have you spoken to? _____
6. Have you received enough information about this study? **YES/NO**
7. Do you fully understand the implication of your involvement in this study? **YES/NO**
8. Do you understand that you are free to withdraw from this study:

- At any time, **YES/NO**
- Without having to give reason for withdrawing, and
- Without affecting your future healthcare? **YES/NO**

9. Do you agree to voluntarily participate in this study? **YES/NO**

10. Do you agree not to discuss any of the particulars of your treatment with any other study Participants? **YES/NO**

11. There is no expense to the participant for participating in the study and no remuneration is offered to the participant.

12. Every participant is given the names and telephone numbers of the research student and supervisor of the study if problems or questions arise.

Research student:	Cell Number:	Homoeopathic Day Clinic:
Catherine Riggien	072 273 4417	031 3732041

If you have answered NO to any of the above, please obtain the information before signing.

Participant *NAME:* _____ ***SIGNATURE:*** _____

WITNESS NAME: _____ **SIGNATURE:** _____

RESEARCH

STUDENT NAME: Catherine Riggien **SIGNATURE:** _____

This appendix has been adapted from Webster, H. 2002. A Homoeopathic Drug Proving of Sutherlandia frutescens. M. Tech. Hom. Dissertation, Durban Institute of Technology.

Appendix C:

Questionnaire on health hazards of smoking

Goldstein 1988:2-1-2-3)

Part A

1. If you quit smoking right away, at what age (barring unforeseen accidents) might you honestly predict you would die?

A1_____

2. If you continue to smoke (and barring unforeseen accidents), at what age might you honestly predict you would die?

A2_____

Part B

Directions: Below are some statements, which are frequently given as reasons why a person continues to smoke. Please tick the ones that you could endorse or go along with.

1. The relationship between smoking and cancer has not really been proven._____

2. Smoking probably won't shorten my life by more than five years, and its better to enjoy life now than to live five years longer and be unhappy. _____

3. I am truly addicted and therefore unable to stop. _____

4. We do not stop the use of alcohol or automobiles, yet they are more dangerous than cigarettes. _____

5. I have to smoke to relieve my nerves _____

6. I smoke filter tips; the harmful material has largely been removed. _____

7. When I stop smoking I gain weight and that is just as bad. _____

8. Anything (including cigarettes) is good in moderation and bad in excess. _____

9. I personally know of at least one very old person who has smoked most of his life yet continues to be in fine health. _____

10. Cancer comes with age and heredity. There is no cancer in my family so therefore I need not worry much about it. _____

11. Hydrogen bombs, highway accidents, murders, alcoholism, suicide

there is no safety anywhere, so why worry? _____

12. The pleasure I get, which is certain, outweighs the health hazard, which is uncertain. _____

13. The emotional effects of my going without cigarettes are more hazardous to me than smoking. _____

14. Scientific research will develop a “safe” cigarette before too long, and the effects of my smoking between now and then are probably insignificant. _____

15. Under present conditions, who wants to live long? _____

16. God would not have put the tobacco plant on earth if He did not have some non-harmful purpose in mind. _____

17. So smoking proves I am weak-willed? Everybody is entitled to one’s weaknesses.

Appendix D:

TOLERANCE DEPENDENCE QUESTIONNAIRE:

(Adapted from Goldstein 1988: 11 – 7)

DIRECTIONS: Tick the box of the answer that you find most appropriate for each question.

1. How many cigarettes do you normally smoke per day?
   - ❑ 37 or more
   - ❑ 27 - 36
   - ❑ 17 - 26
   - ❑ 7 - 16
   - ❑ 6 or less

2. When stressed how many cigarettes do you smoke per day?
   - ❑ 37 or more
   - ❑ 27 - 36
   - ❑ 17 - 26
   - ❑ 7 - 16
   - ❑ 6 or less

3. Normally, how often do you have to smoke?
   - ❑ Every 10 minutes or less
   - ❑ Every 15 minutes
   - ❑ Every half hour
   - ❑ Every 1 hour to 1½ hours
   - ❑ Every 2 – 3 hours

- ❑ Every 4 hours / more

4. Do you ever smoke one cigarette immediately after another?
   - ❑ Always
   - ❑ Frequently
   - ❑ Occasionally
   - ❑ Seldom
   - ❑ Never

5. Which cigarette of the day is the most difficult to give up?
   - ❑ The cigarette of the morning
   - ❑ Midmorning
   - ❑ Midday
   - ❑ Mid-afternoon
   - ❑ Night
   - ❑ Any other

6. How soon after you wake up do you smoke your first cigarette?
   - ❑ Within 5 minutes
   - ❑ 6-30 minutes
   - ❑ 31-60 minutes
   - ❑ 61- or more

7. Do you smoke more frequently during the first hours after waking than during the rest of the day?
   - ❑ Always
   - ❑ Frequently
   - ❑ Occasionally
   - ❑ Seldom
   - ❑ Never

8. Do you find it difficult to refrain from smoking in public places where it is forbidden (for example in church, cinema, library etc.)?
    - ❑ Always
    - ❑ Frequently
    - ❑ Occasionally
    - ❑ Seldom
    - ❑ Never

9. How badly do you want to quit smoking?
    - ❑ Desperately
    - ❑ Very keen
    - ❑ Moderately keen
    - ❑ Not so serious
    - ❑ Do not wish to quit

Appendix E:

Questionnaire on types of smoking

(Goldstein 1988:11-9-11-10)

Directions: Write down the number allocated to the answer that you find most appropriate to the question. The scoring is as follows:

SCORE:	Always	Frequently	Occasionally	Seldom	Never
	5	4	3	2	1

1. I smoke cigarettes to stimulate me, perk myself up. _____

2. I have found a cigarette in my mouth and did not remember putting it there._____

3. When I am trying to solve a problem, I light up a cigarette. _____

4. When I am smoking a cigarette, part of the enjoyment is watching the smoke as I exhale it. _____

5. I am very much aware of the fact when I am not smoking a cigarette. _____

6. Part of the enjoyment of smoking a cigarette comes from the steps I take to light it up. _____

7. When I feel “blue” or want take my mind off cares and worries, I smoke cigarettes. _____

8. I smoke cigarettes automatically even without being aware of it. _____

9. I smoke cigarettes in order to keep myself from slowing down. _____

10. I get a real gnawing hunger for a cigarette if I have not smoked for a while.____

11. When I feel uncomfortable or upset about something I light up a cigarette._____

12. Handling a cigarette is part of the enjoyment of smoking it. _____

13. Between cigarettes, I get a craving that only a cigarette can satisfy. _____

14. I light up a cigarette when if feel angry about something. _____

15. I light up a cigarette without even realising I still have one burning in the ashtray. _____

16. I find cigarettes pleasurable. _____

17. When I run out of cigarettes I find it almost unbearable until I can get them. _____

18. When I feel embarrassed or ashamed about something I light up a cigarette.___

19. Few things help better than a cigarette when I am upset. _____

20. I smoke cigarettes just from habit, without even really wanting the one I am smoking. _____

21. Smoking cigarettes is pleasant and relaxing. _____

22. I do not feel content for long unless I am smoking a cigarette. _____

23. I smoke cigarettes to give me a “lift”. _____

Appendix F:

Methods of scoring for the questionnaire on types of smoking

(Goldstein 1988:11-10)

Add scores for items and divide as indicated for AVERAGE SCORE

	HABITUAL-ADDICTIVE	REDUCTION OF NEGATIVE AFFECT	POSSITIVE AFFECT
	2._____ 5._____ 8._____ 10._____ 13._____ 15._____ 18._____ 20._____ 22._____	3._____ 7._____ 11._____ 14._____ 17._____ 19._____	1._____ 4._____ 6._____ 9._____ 12._____ 16._____ 21._____ 23._____
TOTAL	Divide by 9	Divide by 6	Divide by 8

AVERAGE SCORE	=	=	=

Appendix G:

Smoking history

When did you start smoking?

_____

_____

How many cigarettes do you smoke in a day?

_____

_____

What brand of cigarette do you currently smoke?

_____

_____

Have you tried to stop smoking before?

_____

_____

Appendix H:

Questionnaire on perception of results

1. How many cigarettes are you currently smoking per day?

   _____

2. How many cigarettes were you smoking at the start of the study?

   _____

3. Do you think the medication has assisted you in quitting smoking or reducing the quantity of cigarettes you smoke? YES / NO

4. Do you think the medication has assisted you by reducing your nicotine cravings? YES / NO

5. Would you continue using this medication in order to control your nicotine cravings or to quit smoking? YES / NO

Appendix I:

Participant information sheet

Title of research study:

Study to test the effectiveness of a homoeopathic complex and a tautopathic preparation in managing tobacco addiction.

Name of supervisor: Dr. David Naude (M.Tech:Hom) Tel: (031) 3732514

Name of research student: Catherine Riggien Tel: 072 273 4417

Dear Participant

According to the Cancer Journal for Clinicians (which can also be viewed online).

- People who quit smoking, regardless of their age, live longer than those people of the same age who continue to smoke.
- By quitting smoking, you reduce the risk for developing cancer of the lung, mouth, nasal cavities, throat, stomach, pancreas, liver, kidney, bladder, cervix and some types of leukaemia.
- Nicotine found naturally in tobacco is a drug and it is addictive, just like heroin or cocaine.

Quitting smoking can often result in nicotine withdrawal syndrome, which may result in any of the following symptoms.

- Depression
- Feelings of frustration or anger
- Irritability
- Trouble sleeping
- Difficulty concentrating
- Restlessness
- Headache
- Tiredness
- Increased appetite
- Coughing
- Cravings
- Mood swings
- Dizziness
- Nervousness

All these will be short lived and the positive effects of quitting, such as a reduction of your blood pressure, greater lung capacity and a great deal of money saved, will soon be enjoyed.

The medicines being tested in this study are designed to control or reduce the above symptoms to facilitate smoking cessation.

The study

This study will take two weeks, and in that time you will be expected to take the medication that will be given to you.

This study will be made up of four groups of ten participants each; the first group of participants will receive a tautopathic preparation made of the individual's own make and strength of cigarette which will include the combusted portion and smoke of the sample.

The second group will receive a homoeopathic complex chosen specifically to reduce the expected withdrawal effects of nicotine reduction.

The third group will receive a combination of the tautopathic and homoeopathic complex and the fourth group will receive a placebo medication that will resemble the medication given to all the other groups.

Please note that there is thus a 25% chance that you could be in the placebo group but be assured that should this be the case you will receive the medication in question at no charge at the end of the study.

Participant requirements

As a participant of this study it is imperative that you understand that should you take part in the study you are not obligated to stop smoking.

Your most important and only obligations are to take the medication for the duration of the study and commit to two consultations (one at the beginning of the study and one at the end of the study) where your data will be collected and once the study is complete, the nature your medication revealed. Should you not be able to fulfil these requirements please inform Catherine Riggien immediately so that the study will not be compromised.

Risks

The medications that will be used in this study are considered safe; they themselves cannot cause any side effects.

Some uncomfortable effects may be experienced due to the nicotine withdrawal should you manage to reduce your intake of tobacco, although none of these effects are long-lasting or dangerous.

Confidentiality

Your details and privacy will be respected as a participant of this study; my supervisor and I will have access to these details and they will be held in the strictest confidence.

Thank you for your co-operation. If you have any questions please do not hesitate to contact me within business hours.

Contact numbers

Catherine Riggien 072 273 4417

APPENDIX J:

Do you smoke more than ten cigarettes a day and have done so for at least one year?
Are you between the ages of 18 and 60?

Do you want to stop smoking?

Make an appointment with Catherine
072 273 4417
To see if you qualify.

Free treatment is available should you qualify for the study.

Appendix K:

Production of tautopathic preparation

I will be following the method used in the German Homoeopathic Pharmacopoeia with the addition of one extra step.

The combusted portion of the cigarette and a piece of the tar filled filter will be triturated up to a 3CH using a 1:100 ratio then converted into a liquid potency and potentised and sucussed up to a 6CH.

Method

Take combusted portion of cigarette and used filter, remove filter paper and cut into tiny pieces. Weigh out 0.1g

Weigh out 3x 3.3g of lactose

Place first 3.3g amount of lactose in a clean conical flask.

Place a lit cigarette of the same brand and strength at the neck of the flask allowing the smoke to move into the flask. Place a tissue in the opening to trap the smoke and shake the lactose so that it becomes infused with the smoke.

Allow this lactose to dry and cool in a mortar.

To this add the 0.1g of cigarette sample

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Add second amount of 3.3g of lactose

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Add last amount of 3.3g of lactose

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Remove from mortar, place in a number ten vial

Label as sample _____ 1CH

Weigh out 0.1g sample 1CH

Weigh out 3x 3.3g lactose

Place the 0.1g 1CH sample and one 3.3g amount of lactose in a clean mortar

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Add second 3.3g amount of lactose

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Add third amount of 3.3g lactose

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Remove from mortar, place in a number ten vial

Label as sample _____ 2CH

Weigh out 0.1g sample 2CH

Weigh out 3x 3.3g lactose

Place the 0.1g 2CH sample and one 3.3g amount of lactose in a clean mortar

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Add second 3.3g amount of lactose

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Add third amount of 3.3g lactose

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Triturate for 6 minutes

Scrape for 3 minutes

Mix for 1 minute

Remove from mortar, place in a number ten vial

Label as sample _____ 3CH

Begin with triturate to liquid potency (m/v) ratio 1:100

Weigh 0.1g of sample 3CH place in a 25ml amber glass bottle

Add 5ml of aq dist (distilled water)

Swirl until lactose completely dissolved

Add 5ml 96% svr (alcohol)

Sucuss ten times

Label as sample 4CH

Place 0.3ml of 4CH in a 50ml amber glass bottle

Add 29.7ml of 96% svr (alcohol)

Sucuss ten times

Label as sample 5CH

Place 3ml of sample 5Ch in a 500ml amber glass bottle

Add 297ml of 30% svr (alcohol)

Sucuss ten times

Label as sample 6CH

Dispensing stock

(Repeat this final step 5 times to obtain 1500ml of final product)

Decant 50ml of the final tautopathic preparation into a 50ml spray bottle

Appendix L

Method of manufacture of homoeopathic complex

Stock potencies of Caladium seguinum 29CH, Nux vomica 29CH and Staphysagria delphinium 29CH, were obtained from Natura Laboratories.

Place 1ml of each of the homoeopathic 29CH stock potencies into a 500ml amber glass bottle, add 297ml of 30%svr (alcohol)

Success ten times

Label as homoeopathic complex 30CH.

(Repeat this process 5 times to obtain 1500ml of final product)

Decant 50ml of the final product into 50ml spray bottles

Appendix M

Method of manufacture of Tautopathic and Homeopathic complex

The homoeopathic complex was manufactured (see Appendix L) and 25ml of this complex was added to 25ml of the tautopathic preparation (see Appendix K) corresponding to the individuals own make and strength of cigarette, in a 50ml spray bottle identical to the other groups.

Appendix N

Method of manufacture of Placebo

A solution of 20% alcohol was prepared and decanted into 50ml spray bottles, identical to those used by the other groups.

Appendix O:

15 cigarette samples which will be made into tautopathic preparations

- Styvesant red
- Styvesant blue
- Kent special
- Kent ultra
- Craven A menthol
- Courtley
- Rothmans
- Royal red
- Prinston
- Camel filter
- Yes
- Marlboro light
- Dunhill light
- Dunhill infinite light
- Camel light