An assessment of chiropractic adjustment beds as reservoirs for normal flora and infectious bacterial pathogens, at a chiropractic teaching clinic

by

Jana Logtenberg

A dissertation submitted to the Faculty of Health Sciences, in partial compliance with the requirements for a Master Degree in Technology: Chiropractic at the Durban University of Technology.

I, Jana Logtenberg, do hereby declare that this dissertation represents my own work in both conception and execution, except where specific assistance is sought and duly acknowledged.

J Logtenberg
Jana Logtenberg

30 July 2009
Date

Approved for final submission:

B Odhav.
Prof B. Odhav (Phd)

30/7/2009
Date

Dr A. Docrat (M.Tech: Chiropractic, CCFC (TN))

30-7-2009
Date

DEDICATION

I would like to dedicate this dissertation to:

My parents, Marius and Santa Logtenberg:

Poups and Moekie, I want to thank you for your endless love, support, assistance and motivation. I really appreciate that you have always been there for me through thick and thin. Without you it would have been impossible to study all these years and complete this research dissertation.

My sister, Johmari Logtenberg:

Johmi, I want to thank you for your endless love, support, motivation and understanding. I really appreciate all that you have done for me.

During my research journey I found these little gems that each of us can draw on as sources of inspiration:

"Apparent failure may hold in its rough shell the germs of success that will blossom in time, and bear fruit throughout eternity".

By Frances Ellen Watkins Harper, 1875

"Failure is just another way to learn to do something right".

By Marian Wright Edelman, 1987

ACKNOWLEDGEMENTS

I would like to thank the following people:

Daniel Anstey: I cannot say thank you enough for always supporting and loving me unconditionally, and being there for me at all times. You always knew what to say and always understood. You mean the world to me!

Prof Odhav: Thank you for all your advice, patience, assistance and contributions to this research project, it is greatly appreciated! I learned a great deal from you and I am glad that our life paths have crossed.

Dr Docrat: Thank you for all your advice, cooperation and input on this research project. I really appreciate it!

Dr Korporaal: Thank you for always making time available and being willing to help me. We can all learn from you about time management!

Marlise Roodt, Angela Pastellides and Mandy Higgs:
Thank you for all your support, advice, assistance and enduring the hardships with me through the years. We have finally made it!

Pat van den Berg and Linda Twiggs:
Thank you for all your help, advice and patience!

Tonya Esterhuizen: Thank you for the statistical analyses of this research study.

Tasneem Paulus: Thank you for all your hard work doing the proofreading of this research study.

Staff at the Chiropractic & Somatology and Biotechnology & Food technology departments:
Thank you for all your help, it is much appreciated!

ABSTRACT

Background: Research has indicated the majority of bacteria on chiropractic adjustment beds (beds), can persist on dry inanimate surfaces for months. Thus, insufficient disinfection procedures create continuous sources of pathogens endangering patients and healthcare workers alike. This research study aimed to assess the beds as reservoirs for micro-organisms, at a chiropractic teaching clinic (clinic) in South Africa.

Method: A selection of samples obtained from the headrests and armrests of the beds were serially diluted, plated in duplicate (using the spread plate technique) and incubated for 24-48 hours at 37°C. After inspection for the presence of micro-organisms, those present were enumerated to determine their quantities, the microbial build-up throughout the day, as well as the degree of the transmission from the patients to the beds during treatment. The incidence of the micro-organisms was established, along with their identities, using microscopic and macroscopic characteristics. These micro-organisms were also used to assess the efficacy of the disinfectant currently in use by the clinic.

Results: Microbial growth was present on 89.4% of the beds sampled. The quantities of the micro-organisms increased significantly (p=0,027) from 7:30 am to 16:30 pm, with the median increasing from 25 colony forming units (cfu) / cm^2 to 714 792 cfu/ cm^2. The microbial build-up was highly significant (p<0.001), with a median of 346 cfu/ cm^2 at 7:30 am and 10:30 am; increasing to 162 291 cfu/ cm^2 by 13:30 pm and 250 million cfu/ cm^2 by 16:30 pm. There was also a significant increase (p<0.001) in the quantity of micro-organisms during treatment with a median of 0 cfu/ cm^2 before treatment that rose to 23 479 cfu/cm^2 after treatment, indicating that the micro-organisms present on the beds were being deposited by the patient`s skin during the treatment. The most prevalent micro-organisms identified were Staphylococci and Serratia, with an average of 59% and 40% of colonies; while Micrococci and Bacilli were relatively uncommon. No growth was evident after 5 minutes of exposure to the disinfectant during the growth inhibition test. For the Kirby Bauer test, the average size of the zone of inhibition increased as the dilution decreased. The disinfectant is effective but more so against the Gram-positive than the Gram-negative bacteria. The disinfectant was 5,0, 5,5 and 5,6 times more effective than phenol in eradicating Staphylococci, Serratia and Bacilli, respectively.

Conclusions and Recommendations: This study showed that micro-organisms were present on the beds. Staphylococci and Serratia have been implicated in many healthcare associated infections. The present disinfectant is effective, but should be used in between every patient. A different or additional disinfectant that is more effective against the Gram-negative bacteria should be considered for future use.

CONTENTS

TABLE OF CONTENTS

Page

Page

CHAPTER 3 : METHODOLOGY

Page

CHAPTER 4 : RESULTS AND DISCUSSION

Page

Page

LIST OF TABLES

Chapter 2:

Page

Chapter 4:

Page

Page

Page

Page

LIST OF FIGURES

Chapter 2:

Chapter 4:

Page

LIST OF ANNEXURES

LIST OF ABBREVIATIONS

AIDS: Acquired immunodeficiency syndrome

AL: Left armrest

AR: Right armrest

CFU: Colony forming units

HAI: Healthcare associated infection

HIV: Human immunodeficiency syndrome

HL: Left headpiece

HM: Middle portion of headpiece

HR: Right headpiece

SA: South Africa

TB: Tuberculosis

WFC: World federation of Chiropractic

WHO: World Health Organization

GLOSSARY

Acquired immune deficiency syndrome (AIDS): A serious viral disease caused by human immunodeficiency virus (HIV), in which the T-lymphocytes are destroyed and opportunistic infections occur in the patient (Alcamo, 1994).

Active immunity: An immune state achieved by self-production of antibodies (Madigan, Martinko and Parker, 2000).

Acute: In reference to infections; short-term, usually characterized by dramatic onset and rapid recovery (Madigan, Martinko and Parker, 2000).

Agar: A derivative of marine seaweed used as a solidifying agent in many microbiological media (Alcamo, 1994).

Amoxicillin: A semisynthetic penicillin antibiotic that is related to ampicillin (Alcamo, 1994).

Ampicillin: A semisynthetic penicillin derivative active against Gram-positive bacteria and certain Gram-negative bacteria (Alcamo, 1994).

Anorexia: Loss of appetite (Alcamo, 1994).

Antibiotic: A chemical agent produced by one organism that is harmful to other organisms (Madigan, Martinko and Parker, 2000).

Antibiotic resistance: The acquired ability of a micro-organism to grow in the presence of an antibiotic, to which the micro-organism is usually sensitive (Madigan, Martinko and Parker, 2000).

Antimicrobial: Harmful to micro-organisms by either killing or inhibiting growth (Madigan, Martinko and Parker, 2000).

Antimicrobial agent: A chemical that kills or inhibits the growth of micro-organisms (Madigan, Martinko and Parker, 2000).

Antiseptic: An agent that kills or inhibits the microbial growth but is not harmful to human tissue (Madigan, Martinko and Parker, 2000).

Aseptic technique: Manipulation of sterile instruments or culture media in such a way, as to maintain sterility (Madigan, Martinko and Parker, 2000).

Autoclave: A steriliser that destroys micro-organisms by high temperature using steam under pressure (Madigan, Martinko and Parker, 2000).

Autolysis: The lysis of a cell brought about by the activity of the cell itself (Madigan, Martinko and Parker, 2000).

Bacillus: A bacterial rod (Alcamo, 1994).

Bacteremia: The transient appearance of bacteria in the blood (Madigan, Martinko and Parker, 2000).

Bacteria: All prokaryotes that are not members of the domain Archaea (Madigan, Martinko and Parker, 2000).

Bacteriocidal: Capable of killing bacteria (Madigan, Martinko and Parker, 2000).

Bacteriostatic: Capable of inhibiting bacterial growth without killing (Madigan, Martinko and Parker, 2000).

Biofilm: Microbial colonies encased in an adhesive, usually polysaccharide material, and attached to a surface (Madigan, Martinko and Parker, 2000).

Capsule: Dense, well-defined polysaccharide or protein layer closely surrounding a cell (Madigan, Martinko and Parker, 2000).

Carrier: An individual that harbours infectious organisms but does not show symptoms of disease (Madigan, Martinko and Parker, 2000).

CD4 cells: T helper cells. They are targets for HIV infection (Madigan, Martinko and Parker, 2000).

Cell: The fundamental unit of life (Madigan, Martinko and Parker, 2000).

Cell mediated immunity: An immune response generated by the activities of non-antibody-producing cells, such as T cells (Madigan, Martinko and Parker, 2000).

Chemotherapeutic agent: An antimicrobial agent that can be used internally (Madigan, Martinko and Parker, 2000).

Chemotherapy: Treatment of infectious disease with chemicals or antibiotics (Madigan, Martinko and Parker, 2000).

Cidal: Lethal or killing (Madigan, Martinko and Parker, 2000).

Coccus: A spherical bacterium (Madigan, Martinko and Parker, 2000).

Colony: A macroscopically visible population of cells growing on solid medium, arising from a single cell (Madigan, Martinko and Parker, 2000).

Communicable disease: A disease that is transmissible among various hosts (Alcamo, 1994).

Contagious: Transmissible (Madigan, Martinko and Parker, 2000).

Contagious disease: A communicable disease whose agent passes with particular ease among hosts (Alcamo, 1994).

Culture: A particular strain or kind of organism growing in a laboratory medium (Madigan, Martinko and Parker, 2000).

Culture medium: An aqueous solution of various nutrients suitable for the micro-organisms (Madigan, Martinko and Parker, 2000).

Cutaneous: Relating to the skin (Madigan, Martinko and Parker, 2000).

Cytoplasmic membrane: The permeability barrier of the cell, separating the cytoplasm from the environment (Madigan, Martinko and Parker, 2000).

Decontamination: Treatment that renders an object or inanimate surface safe to handle (Madigan, Martinko and Parker, 2000).

Denaturation: Irreversible destruction of a macromolecule, as for example, the destruction of a protein by heat (Madigan, Martinko and Parker, 2000).

Deoxyribonucleic acid (DNA): A polymer of nucleotides connected via a phosphate-deoxyribose sugar backbone; the genetic material of cells and some viruses (Madigan, Martinko and Parker, 2000).

Disease: Injury to the host that impairs host function (Madigan, Martinko and Parker, 2000).

Disinfectant: An agent that kills micro-organisms but may also be harmful to human tissue (Madigan, Martinko and Parker, 2000).

Disinfection: The process of eliminating nearly all pathogens, but not all micro-organisms, from inanimate objects or surfaces (Madigan, Martinko and Parker, 2000).

Endemic: A disease that is constantly present in low numbers in a population (Madigan, Martinko and Parker, 2000).

Endogenous disease: A disease caused by organisms commonly found within the body (Alcamo, 1994).

Endospore: A differential cell formed within the cells of certain gram-positive bacteria that are extremely resistant to heat, as well as to other harmful agents (Madigan, Martinko and Parker, 2000).

Enzyme: A catalyst, usually composed of protein, that promotes specific reactions or groups of reactions (Madigan, Martinko and Parker, 2000).

Fomites: Inanimate objects that, when contaminated with a viable pathogen, can transfer the pathogen to a host (Madigan, Martinko and Parker, 2000).

Fungi: Nonphototrophic eukaryotic micro-organisms that contain rigid cell walls (Madigan, Martinko and Parker, 2000).

Genus: A taxonomic group of related species (Madigan, Martinko and Parker, 2000).

Germicide: A substance that inhibits or kills micro-organisms (Madigan, Martinko and Parker, 2000).

Gram-negative cell: A prokaryotic cell whose cell wall contains relatively little peptidoglycan but has an outer membrane composed of lipopolysaccharide, lipoprotein, and other complex macromolecules (Madigan, Martinko and Parker, 2000).

Gram-positive cell: A prokaryotic cell whose cell wall consists chiefly of peptidoglycan and lacks the outer membrane of gram-negative cells (Madigan, Martinko and Parker, 2000).

Host: An organism capable of supporting the growth of a virus or other parasite (Madigan, Martinko and Parker, 2000).

Immune: Able tot resist infectious disease (Madigan, Martinko and Parker, 2000).

Immunity: The ability of an organism to resist infection (Madigan, Martinko and Parker, 2000).

Immunodeficiency: Having a dysfunctional or completely non-functional immune system (Madigan, Martinko and Parker, 2000).

Innate immunity: An inborn capacity for resisting disease (Alcamo, 1994).

Incidence: In reference to disease transmission, the number of cases of the disease in a specific subset of the population (Madigan, Martinko and Parker, 2000).

Infection: Growth of an organism within the body (Madigan, Martinko and Parker, 2000).

Inoculum: Material used to initiate a microbial culture (Madigan, Martinko and Parker, 2000).

Kirby Bauer test: An agar diffusion test used to determine the antibiotic concentration effective against a test organism (Alcamo, 1994).

Lysis: Rupture of a cell, resulting in a loss of cell contents (Madigan, Martinko and Parker, 2000).

Medium: In microbiology, the nutrient solution(s) used to grow micro-organisms (Madigan, Martinko and Parker, 2000).

Microbe: An alternative expression for micro-organism (Alcamo, 1994).

Micrometer: One-millionth of a meter, or 10^{-6} m (abbreviated µm); the unit used for measuring micro-organisms (Madigan, Martinko and Parker, 2000).

Micro-organism: A microscopic organism consisting of a single cell or cell cluster, also including the viruses (Madigan, Martinko and Parker, 2000).

Morbidity: Incidence of disease in a population, including both fatal and non-fatal cases (Madigan, Martinko and Parker, 2000).

Mortality: Incidence of death in a population (Madigan, Martinko and Parker, 2000).

Nanometer: A unit of measurement equivalent to one billionth of a meter; the unit is designated as nm and is often used in measuring viruses and the wave-length of energy (Alcamo, 1994).

Non-communicable disease: A disease whose causative agent is acquired from the environment and is not easily transmitted to the next host (Alcamo, 1994).

Normal flora: Micro-organisms that are usually found associated with healthy body tissue (Madigan, Martinko and Parker, 2000).

Nosocomial infection: Hospital-acquired infection (Madigan, Martinko and Parker, 2000).

Nutrient agar: A common bacteriological growth medium consisting of beef extract, peptone, water and agar (Alcamo, 1994).

Pandemic: A worldwide epidemic (Madigan, Martinko and Parker, 2000).

Passive immunity: Immunity resulting from transfer of antibodies or immune cells from an immune to a non-immune individual (Madigan, Martinko and Parker, 2000).

Pasteurization: A heating process that destroys pathogenic bacteria in a fluid, such as milk, and lowers the overall number of bacteria in the fluid (Alcamo, 1994).

Pathogen: An organism able to inflict damage on a host it infects (Madigan, Martinko and Parker, 2000).

Pathogenicity: The ability of a parasite to inflict damage on to the host (Madigan, Martinko and Parker, 2000).

Penicillin: Any of a group of antibiotics derived from *Penicillium* species or produced synthetically; the antibiotics are effective for Gram-positive bacteria and several Gram-negative bacteria; they interfere with cell wall synthesis (Alcamo, 1994).

Prokaryote: A cell or organism lacking a nucleus and other membrane-enclosed organelles, usually having its DNA in a single circular molecule (Madigan, Martinko and Parker, 2000).

Pure culture: A culture containing a single kind of micro-organism (Madigan, Martinko and Parker, 2000).

Sexually transmitted disease (STD): A disease whose usual means of transmission is by sexual contact (Madigan, Martinko and Parker, 2000).

Species: Of prokaryotes, a collection of closely related (>97% 165 rRNA sequence homology and >70% genomic hybridization) strains sufficiently different from all other strains, to be recognized as a distinct unit (Madigan, Martinko and Parker, 2000).

Spore: A general term for resistant resting structures formed by many prokaryotes and fungi (Madigan, Martinko and Parker, 2000).

Sporicidal agent: An agent that kills bacterial spores (Alcamo, 1994).

Staphylococcus: A form of bacteria characterized by spheres in a grapelike cluster (Alcamo, 1994).

Sterile: Free of living organisms and viruses (Madigan, Martinko and Parker, 2000).

Sterilization: Treatment resulting in death of all living organisms and viruses in a material (Madigan, Martinko and Parker, 2000).

Strain: A population of cells of a single species all descended from a single cell; a clone (Madigan, Martinko and Parker, 2000).

Vehicle: Non-living source of pathogens that infect large numbers of individuals; common vehicles are food and water (Madigan, Martinko and Parker, 2000).

Viable count: The number of living (viable cells) (Baveja, 2005).

Virucidal agent: An agent that inactivates viruses (Alcamo, 1994).

Virulence: Degree of pathogenicity of a parasite (Madigan, Martinko and Parker, 2000).

CHAPTER 1 : INTRODUCTION

1.1 INTRODUCTION

A healthcare-associated infection is an infection that occurs in a patient, within a health care facility, in whom the infection is not present or incubating at the time of admission or that is acquired during the stay in the health care facility, but arises within 48 hours after discharge. Infections contracted by staff or visitors to the facility are also considered as a healthcare-associated infection (WHO, 2002; Moor and Ferguson, 2006).

Globally, more than 1,4 million patients in developed, transitional or developing countries have a healthcare-associated infection at any point in time. In developed countries the infection rate is 5 – 10% and in developing countries it exceeds 25% (Ma *et al*., 2003; Lynch *et al*., 2007; Pittet *et al*., 2008). In South Africa, the conservative infection rate is 15% with an associated estimated mortality rate of 5% (Duse, 2005).

Escherichia coli and *Staphylococcus aureus*, the most commonly implicated bacteria in the majority of healthcare-associated infections, are in actual fact constituents of the body's normal flora (Struelens, Denis and Rodriquez-Villalobos, 2004). Under normal circumstances they protect the host from colonisation by pathogenic flora; but when the host's immune status becomes compromised, inappropriate antibiotic therapy allows overgrowth or transmission occurs to sites outside the natural habitat, which makes it possible for infection to occur (WHO, 2002).

The most prevalent means of transmission of pathogens is through direct contact between a susceptible host and an infected person (Borkow and Gabbay, 2007). A host can also be infected indirectly, via intermediate inanimate objects that have become contaminated through direct contact with skin or bodily secretions, soiled hands, aerosolized pathogens or airborne pathogens that have settled after the disturbance of a contaminated object (Boone and Gerba, 2007; Borkow and Gabbay, 2007). Thus, since humans liberate roughly 3×10^8 squamae per day, it is to be expected that intermediate inanimate objects or equipment in the patient's immediate environment, such as chiropractic adjustment beds (beds), will get contaminated (Borkow and Gabbay, 2007).

Bifero, Prakash and Bergin (2006) and Evans *et al*. (2007), previously investigated the role of vinyl-covered beds as reservoirs for microbial diseases and their results indicated the presence of fungi and a wide variety of Gram-positive and Gram-negative bacteria, including Methicillin-resistant *Staphylococcus aureus*. The latest study by Evans *et al*. (2008), found that cloth-covered chiropractic adjustment were not only sources of pathogenic microbes but also of allergens.

This concurs with the estimated 21,1% of healthcare-associated infection outbreaks that are attributed to contaminated surfaces, and research has confirmed that most gram-positive and many gram-negative bacteria can persist on dry inanimate surfaces for months (Kramer, Schwebke and Kampf, 2006; Borkow and Gabbay, 2007). Neglecting to disinfect the beds will, thus, result in the creation of continuous sources of pathogens endangering patients and healthcare workers (chiropractors) alike (Boone and Gerba, 2007).

Considering that chiropractors are amongst the most popular complementary care practitioners (Meeker and Haldeman, 2002; Menke, 2003; Redwood and Cleveland, 2003; Haldeman, 2005; Morgan, 2005), it is of paramount importance to take every precaution and to implement efficient and integrated infection control programmes that are re-assessed frequently and adjusted according to the assessment (Meeker and Haldeman, 2002; Haldeman, 2005; Evans *et al*., 2007). It has been the experience in developed countries, that when unassailable infection control programmes are in place, there will be a decline in the incidence of healthcare-associated infections (Duse, 2005). According to Duse (2005), scrupulous systems to identify healthcare-associated infections are lacking in South Africa, and the first step towards establishing infection control programmes is to determine the extent of the problem, by amongst others, identifying the prevalent types of micro-organisms. South Africa is also currently struggling to cope with HIV/ AIDS and related diseases, such as extreme drug resistant tuberculosis, which renders patients immuno-compromised and thus, susceptible to even normal flora (Johansson, 2007; Kapp, 2007; Harling, Ehrlich and Myer, 2008).

Therefore, this research study aimed to evaluate the current infection control parameters at a chiropractic teaching clinic (clinic) and determine the extent of the problem. It aimed to provide information on the microbial flora and infectious bacterial pathogens specifically encountered in the clinic, in a South African setting, and also verified the efficacy of the current disinfectant in use by the clinic.

1.2 AIM

The aim of the study was to assess the beds as reservoirs for normal microbial flora and infectious bacterial pathogens at a clinic in South Africa.

1.3 OBJECTIVES AND HYPOTHESES

Objective 1: To determine the presence of and enumerate the micro-organisms from the incubated samples obtained from the beds in the clinic.

> **Hypothesis one:** The null hypothesis (Ho) states that there will be no micro-organisms present on the beds in the clinic. The alternate hypothesis (Ha) states that there will be micro-organisms present on the beds in the clinic.

Objective 2: To determine the microbial build-up on the beds in the clinic at regular intervals over a 12 hour period.

> **Hypothesis two:** The null hypothesis (Ho) states that there will not be a microbial build-up on the beds in the clinic over a 12 hour period. The alternate hypothesis (Ha) states that there will be a microbial build-up on the beds in the clinic over a 12 hour period.

Objective 3: To identify and determine the incidence of normal human microbial flora and the infectious bacterial pathogens found on the beds in the clinic.

Objective 4: To assess the efficacy of the disinfectant currently in use by the clinic.

> **Hypothesis three:** The null hypothesis (Ho) states that the disinfectant currently in use by the clinic is not an effective disinfectant. The alternate hypothesis (Ha) states that the disinfectant currently in use by the clinic is an effective disinfectant.

Objective 5: To determine the degree of the transmission of normal human microbial flora and the infectious bacterial pathogens from the patients to the beds in the clinic during treatment.

Hypothesis four: The null hypothesis (Ho) states that there will be no transmission of normal human microbial flora and infectious bacterial pathogens from the patients to the beds in the clinic during treatment. The alternate hypothesis (Ha) states that there will be a transmission of normal human microbial flora and infectious bacterial pathogens from the patients to the beds in the clinic during treatment.

1.4 RATIONALE

- Chiropractic care has been shown to be a cost effective alternative for the management of neuro-musculoskeletal conditions (Manga, 2000; Chapman-Smith, 2008), which is even more the case at the clinic, since patients are charged with fees lower than medical aid rates and further fee reductions are granted to those individuals unable to afford even the normal clinic fees (Korporaal, 2009). The majority of patients seek chiropractic care for musculoskeletal conditions and have been shown to have higher levels of education, household income and permanent employment (Lawrence and Meeker, 2007; Barnes *et al*., 2004; Leboeuf-Yde *et al*., 2005). Due to the above mentioned reduced fees, chiropractic care is accessible and available at the clinic to patients of all socio-economic backgrounds (Drews, 1994; Benjamin, 2007; Kandhai, 2007 and Venketsamy, 2007), which exposes the clinic to a wide range of pathogens. This research will address this concern by determining if infectious pathogens are present on the beds and subsequently, if the risk of transmission between patients, students and the beds exists.

- South Africa is a developing country struggling to cope with HIV/ AIDS and related diseases, such as extreme drug resistant tuberculosis, which renders patients immuno-compromised and thus, susceptible to even normal flora (Johansson, 2007; Kapp, 2007; Harling, Ehrlich and Myer, 2008). No such research has been previously performed on beds in a South African setting. Doing so will incorporate all the factors unique to South Africa and make the results relevant to South Africa, so that the exact extent of the situation will become known.

- Results obtained from this research will verify if the current disinfectant used by the clinic is effective, should infectious pathogens be found on the beds, and whether an alternative should be considered.

1.5 LIMITATIONS

Of the 22 beds available for the treatment of patients, only 14 were randomly selected for sampling. However, of these 14 beds, 6 were sampled more than once but on different occasions.

According to the methodology, samples were expected to be obtained from the left and right armrest, the middle part of the headrest and then either the left or right part of the headrest. However, some beds had no middle part of the headrest. In these cases both the right and left parts of the headrest were sampled.

To determine the transmission of micro-organisms from the patients to the beds during treatment, beds on which two new and two follow up patients were going to be treated were randomly selected. For the two new patients, only those who have not had prior treatment at the clinic or those who have not had treatment in the last 6 months were considered. This was so that the patients would spend the maximum amount of time possible on the beds during treatment.

Due to safety reasons, the samples could not be obtained before or after clinic operation hours so some of the students, staff and clinicians, thus, knew the study was being conducted. This could mean that the students, staff or clinicians could have practiced better hand and bed hygiene that would have had an effect on the results.

The bacteria present on the beds were identified to the genus level, only since the researcher has not had formal training in microbiological techniques, and any bacteria can result in an infection in immuno-compromised patients.

When enumerating agar plates, for reliability and validity reasons, only those with colony forming units between 30 and 300 were used. When there was less than 30 colony forming units it is termed as being “too few to count” (TFTC) while if there were more than 300 it was termed “too numerous to count” (TNTC). In order to complete statistical analysis, the minimum and maximum of 30 and 300 were used as mean number of colonies for these plates when calculating the total viable number of colony forming units, by taking the volume and dilution of the inoculum into account.

1.6 ASSUMPTIONS

The researcher assumed that the appointments booked by the patients for treatment in the clinic, on the beds selected for sampling on the relevant days, were not cancelled before or after the swabs had been taken and that no additional appointments were made before or after the swabs had been taken.

The assumption was made that the new and follow-up patients will spend the majority of the time allocated for the appointments (2,5 to 3 hours and 1 hour respectively), on the beds.

1.7 THESIS LAYOUT

This chapter has briefly summarised the literature, highlighting the relevant areas of interest in this research study, and presented the objectives and hypotheses along with the rationale behind the study, as well as the inherent limitations and assumptions of this study. In Chapter Two, a comprehensive review of the body of literature pertaining to the research study will be presented. This will be followed by the delineation of the materials and methods utilised to structure the design of this research study in Chapter Three. The results obtained and the discussion thereof, within the context of the literature, will be featured in Chapter Four. In Chapter Five, conclusions will be drawn with the recommendations based on the study outcome thereafter, thereby, concluding the research study.

CHAPTER 2 : LITERATURE REVIEW

2.1 INTRODUCTION

This literature review covers the background of the study on the assessment of the beds as reservoirs for normal flora and infectious bacterial pathogens at a clinic. It includes a full overview of the chiropractic profession, healthcare-associated infections (HAI) and the intervention strategies employed.

2.2 THE CHIROPRACTIC PROFESSION

2.2.1 Definition

The World Health Organization (WHO, 2005) recognizes chiropractors as primary contact healthcare providers and defines chiropractic as, “a health care profession concerned with the diagnosis, treatment and prevention of disorders of the neuromusculoskeletal system and the effects of these disorders on general health. There is an emphasis on manual techniques, including joint adjustment and/ or manipulation with a particular focus on subluxations”. This is in accordance with the definitions that have been issued by the World Federation of Chiropractic (WFC, 2001), the Chiropractic Association of South Africa (CASA, 2009b) and The Allied Health Professions Council of South Africa (AHPCSA, 2009; Act 63 of 1982 (as amended)).

2.2.2 Chiropractic and its principles and practices

The chiropractic profession acknowledges the relationship between structure of the musculoskeletal system, especially the spine, and function, as coordinated by the nervous system. Anything that impacts on this relationship, has been associated with adverse repercussions on health, and is termed a subluxation (Meeker and Haldeman, 2002; Chapman-Smith, 2008; Ernst, 2008). Healing is the process of becoming whole through the curing disease and / or dysfunction, as facilitated by the physician or healer (Davis and Bove, 2008). The chiropractic profession adheres to the biopsychosocial philosophy of health by recognising the

inherent ability of the body to heal itself without the use of drugs or surgery (Kremer, Duenas and McGuckin, 2002; Meeker and Haldeman, 2002; Chapman-Smith, 2008; CASA, 2009a). It, thus, offers non-invasive treatment in the form of a spinal adjustment (Schafer and Faye, 1990; Bergman, Peterson and Lawrence, 1993; Haldeman, 2005; CASA, 2009a) directed at correcting the subluxation, which is considered to be a sprained or strained joint, with subsequent hypomobility, malalignment, local and/or referred pain, inflammation and muscle tension (Meeker and Haldeman, 2002; Chapman-Smith, 2008). Subluxation is believed, by 88% of chiropractors in the United States, to contribute to over 60% of all visceral ailments and 90%, therefore, think that chiropractic care should not be limited to neuromusculoskeletal conditions only (Ernst, 2008).

2.2.3 The role of chiropractic in the health care system

2.2.3.1 Chiropractic as complementary and alternative medicine (CAM) and/ or primary health care practitioner

The phrase complementary and alternative medicine refers to the broad domain of healing resources that encompasses all health systems, modalities, and practices and their accompanying theories and beliefs, other than those intrinsic to the politically dominant health system of a particular society or culture in a given historical period. It includes all such practices and ideas, self-defined by their users, as preventing or treating illness or promoting well-being and that view health and disease in the context of the human totality of body, mind, and spirit (Barnes *et al*., 2004; Duke, 2005; Ben-Arye *et al*., 2008; Nahin *et al*., 2007). Chiropractic has been classified as a complementary and alternative therapy and currently it is the best recognised and most popular (Meeker and Haldeman, 2002; Menke, 2003; Redwood and Cleveland, 2003; Haldeman, 2005; Morgan, 2005). Up to 80% of the population in Africa uses CAM (Frenkel *et al*., 2008), while chiropractic care is selected by 30% of the people seeking CAM in the United States (Meeker and Haldeman, 2002) and approximately 3 – 18% of the general population (Ernst, 2008).

Many chiropractors do not want to be termed CAM practitioners and since they have many attributes of primary care providers, they often describe themselves as such (Meeker and Haldeman, 2002). Others view chiropractic as being a limited medical profession or specialty (Meeker and Haldeman, 2002) and according to Ernst (2008); some chiropractors do view themselves as an “alternative” form of healthcare. The author goes on to say that in most

countries chiropractic is only seen as an adjunct rather than a replacement of healthcare. Gaumer, Koren and Gemmen (2002) also state that not all chiropractors perceive themselves to be actual or potential primary health care practitioners. These chiropractors prefer to limit their scope of practices to neuromusculoskeletal conditions due to training and lack of hospital privileges, which does not allow for access to all aspects of primary care or legal limitations (Gaumer, Koren and Gemmen, 2002; Kremer, Duenas and McGuckin, 2002; Maharaj, 2009).

2.2.3.2 Legal requirements and limits with regards to the chiropractic profession

In the United States of America (USA), each state has a scope of practice law that governs chiropractic practice and imposes legal limits on the kinds of diagnostic and treatment procedures performed by chiropractors, in terms of matters related to public health and safety (Gaumer, Koren and Gemmen, 2002). The effects of these laws on the provision of primary health care by chiropractors appear inconsistent and limited; and although they prevent chiropractors from performing certain activities, they are silent, ambiguous or inconsistent concerning most activities (Gaumer, Koren and Gemmen, 2002). Common features in all national jurisdictions, concerning the scope of chiropractic practice, are that primary care and the right and duty to diagnose, including the right to perform and/or order diagnostic imaging, is recognized and legal (Chapman-Smith, 2008). Evidence indicates chiropractors do not routinely provide primary care services in practice (Mootz *et al*., 2006), even though many primary care activities are widely permitted (Gaumer, Koren and Gemmen, 2002).

The Allied Health Professions Council of South Africa is a statutory body controlling all allied health professions, including chiropractic, in South Africa. The Allied Health Professions Act of 63 of 1982 (as amended) stipulates that South African chiropractors are primary healthcare practitioners (AHPCSA, 2009) as in agreement with WHO (2005) and the Councils on Chiropractic Education International (CCEI, 2005). In order to interpret the International chiropractic accreditation standards, one has to understand the following notion of a chiropractor as laid out by the CCEI (2005): “The chiropractor, as a practitioner of the healing arts, is concerned with the health needs of the public. He/ she gives particular attention to the relationship of the structural and neurological aspects of the body in health and disease; he/ she is educated in basic and clinical sciences as well as in related health subjects. The purpose of his/ her professional education is to prepare the chiropractor as a primary health care provider.

As a portal of entry to the health delivery system, the chiropractor must be well educated to diagnose, to care for the human body in health and disease and to consult with, or refer to, other health care providers when appropriate for the best interest of the patient". The institution, whose clinic is involved in this research study, is currently in the process of seeking accreditation for its chiropractic education programme from the ECCE, an agent of the CCEI (Korporaal, 2009). Chiropractic students at this institution are, thus, schooled in a broadly based curriculum adhering to the minimum norms of the CCEI, statutory regulations (Allied Health Professions Act 63 of 1982 (as amended)) as well as the minimum health care norms (CASA, 2009a).

2.2.3.3 Primary health care and adequacy of chiropractic curricula or education programmes

The latest and most comprehensive definition of primary health care is that provided by the Institute of Medicine in 1996 which reads: "primary health care is the provision of integrated, accessible health care services by clinicians who are accountable for addressing a large majority of the personal health care needs, developing a sustainable partnership with patients, and practicing in the context of family and community" (Duenas, 2002; Kremer, Duenas and McGuckin, 2002; Duenas et al., 2003). Despite not participating in the formation of this definition, the broad scope of chiropractic practice appears to meet the criteria for primary care, although some may argue that the criteria could not be achieved with chiropractic practice, seeing that it has limitations in terms of the provision of care for organic disorders (Duenas, 2002). In response, Duenas (2002) argued that:

- "Chiropractic education requires the student to be prepared in whole-body diagnosis for all ages and recognize the need for appropriate treatment of the patient`s condition."
- "The primary care provider need not render a broad range of services, but should rather recognize problems presented by the patient and arrange for appropriate patient care."
- "The effects of chiropractic adjustments are not limited to the musculoskeletal or neuromusculoskeletal systems, but also include visceral and constitutional effects."
- "Chiropractic care not only comprises of spinal adjustments but also includes the provision of nutrient, dietary, hygiene, psychosocial counsel, public health, ergonomic and exercise services."

Gaumer, Walker and Su (2001) performed a study in which two panels were asked to identify tasks performed by healthcare providers that were considered to be primary health care activities, as well as to consider the potential role of chiropractors to provide primary health care. The first panel was interdisciplinary, consisting of six allopathic physicians, two chiropractors (of which one was an allopathic physician as well), one nurse practitioner and one physician assistant. The other panel was chiropractic, consisting of nine chiropractors, one allopathic physician (a member of the faculty in a chiropractic college) and one jointly trained chiropractor and allopathic physician. Major groups of activities and component activities were compiled by both panels but comparisons can only be made on 172 of the activities rated by both the panels. The results of the study can be synopsed as follows:

- No real differences were found between the panels regarding the scope and importance of activities for good health.
- Approximately 40% of activities were performed more frequently in the typical primary medical care practices than in typical chiropractic practices.
- Of the 53 daily occurring activities, as compiled by the interdisciplinary panel, chiropractors were unable to diagnose only approximately 7,5% of these but were able to make therapeutic contributions in about 55%.
- In approximately two thirds of the above 53 daily occurring activities, the chiropractic panel believed chiropractors could provide patient care up through the level of making some diagnostic contributions.
- The chiropractic panel perceived the necessity of allopathic physician involvement more frequently (15%) than by the interdisciplinary panel.

The curricula of chiropractors, in the United states, has an average of 4820 classroom and clinical hours, with 30% spent in the basic sciences and the remaining 70% in clinical sciences and internship. On the other hand, medical school curricula averaged about 4670 hours with a similar breakdown. In comparison, chiropractic students spend more hours in anatomy and physiology, fewer hours in public health but similar hours in biochemistry, microbiology and pathology. Chiropractic curricula supplies modest instruction in pharmacology, critical care and surgery but emphasize biomechanics, musculoskeletal function and manual treatment methods. Medical curricula has more than double the hours in clinical experience (Meeker and Haldeman, 2002).

It seems that allopathic physicians and chiropractors agree on what constitutes primary care activities and the importance of these (Gaumer, Walker and Su, 2001). Studies have shown that 60 – 90% of diagnoses made in outpatient primary care settings can be handled by certain alternative healthcare providers (Gaumer, Walker and Su, 2001). The knowledge of primary care activities were assessed and compared between allopathic and chiropractic students in a study performed by Sandefur, Febbo and Rupert (2004). Results indicated that chiropractic students marginally scored below their allopathic counterparts. This was considered noteworthy considering the ambiguity with which chiropractic has approached primary care, according to Sandefur, Febbo and Rupert (2004). One can then deduce that the chiropractic curricula are adequate in preparing chiropractors for their role as primary healthcare providers.

2.2.3.4 Integrative care and its barriers

Integrative care is defined as, “practicing medicine in a way that selectively incorporates elements of CAM into comprehensive treatment plans alongside solidly orthodox methods of diagnosis and treatment (Menke, 2003).” Integrative chiropractic care is, thus, the integration of chiropractic care with the aforementioned “orthodox methods of diagnosis and treatment.”

The integration process has multiple barriers and obstacles, which include: financial disincentives; patient fear or concern with legal issues; communication gaps between CAM providers, conventional healthcare workers, and physicians; lack of access to proper education about and experiences with CAM; resistance from conventional healthcare system; differences in beliefs about healing; limited information on clinical outcomes; and lack of experience and knowledge about how to overcome these and other obstacles (Frenkel *et al*., 2008). The issues regarding chiropractic education and the legal matters have been discussed already. A discussion of the main issues not yet addressed will now be discussed in short.

The coordination of care provided by various health care professionals can be impeded by insufficient inter-professional relationships, lack of knowledge about the services provided by one another and resistance to refer patients to others (Gaumer, Koren and Gemmen, 2002). According to Gaumer, Koren and Gemmen (2002), inter-professional referral patterns have been shown to be adequate with evidence of in-referral and out-referral. A survey found that 66,6% family physicians in North Carolina felt moderately or very informed about chiropractic and 65% have referred patients to chiropractors, while 98% chiropractors made routine referrals to the physicians (Meeker and Haldeman, 2002).

Integration of chiropractic and mainstream medicine has been established in the United States, Denmark and Switzerland with the United States being the only country with chiropractors on the sports medicine teams for the Beijing Olympic Games (Chapman-Smith, 2008). In South Africa, chiropractic services are not offered in hospital settings as is the case overseas. Thus far, the closest example of integrative medicine in South Africa is that of the separate chiropractic clinic established on the premises of the Kimberley Hospital complex (Higgs, 2009; Maharaj, 2009).

Consumer preferences are a major driving force in the demand for chiropractic services and if chiropractors want to serve as primary health care providers, they must overcome the impression that they mostly treat neuromusculoskeletal conditions. A survey by Cambron, Cramer and Winterstein (2007) at two chiropractic teaching clinics, revealed that only 19% of the patients saw their chiropractor as their primary healthcare provider. In another study, 66% of the participants indicated that they used CAM in the previous year, 42,6% expressed interest in using CAM over the next year and 93,6% believed that natural practitioners, including chiropractors, should be part of a family practice clinic. In addition, 74,9% of patients were willing to pay out of their own pocket for some of the services (Frenkel *et al*., 2008).

Convincing consumers poses to be a difficult task since some chiropractors don't even perceive themselves as actual or potential primary health care practitioners (Gaumer, Koren and Gemmen, 2002). They seem to be in the minority as is evident in a survey. Most respondents (63,3%) believed that chiropractors should be portal-of-entry primary healthcare providers; 25,7% believed chiropractors should be portal-of-entry for musculoskeletal conditions but not primary healthcare and only 4,1% believed chiropractors should be musculoskeletal specialists and not portal-of-entry practitioners (Gaumer, Koren and Gemmen, 2002).

2.2.3.5 Advantages of integrative chiropractic care

Patients can only benefit from integrated health care since conservative measures decrease the need and reduce the side effects of surgery, resulting in better outcomes. In the end, treatment should be centered on the best interests of the patients and whatever is best for the patient should guide treatment (Menke, 2003). A varying amount of CAM consumers actually report their use to CAM or to their orthodox health care providers (Hori *et al*., 2008; Peltzer *et al*., 2008; Sirois, 2008) and many use a combination of both concurrently (MacIntyre, Holzemer and Philippek, 1997; Sharma, Haas and Stano, 2003; Shmueli and Shuval, 2006).

The chiropractic profession will benefit by having access to better resources and diagnostics, a broader base of patients who could benefit from chiropractic treatment and management, and establishing reputation and stature as a community health resource (Menke, 2003).

Integrative medicine that includes chiropractic holds the promise of improving the health outcomes of patients through the collaboration of treatment and management of difficult and chronic conditions. Chiropractors will contribute to public health system through prevention and health promotion, as well as through its cost-effectiveness (Manga, 2000). The majority of patients seek out chiropractic care for the treatment of musculoskeletal conditions or other conditions associated with chronic or recurring pain (Drews, 1994; Meeker and Haldeman, 2002; Barnes *et al*., 2004; Benjamin, 2007; Jaman, 2007; Khandai, 2007; Mahomed, 2007; Venketsamy, 2007; Ernst, 2008; Evans, Williams and Perko, 2008). Approximately 66,6% of all patients seeking care for low back pain, consult chiropractors in a primary health care role and half of these patients seeking chiropractic care, suffer from chronic symptoms (Meeker and Haldeman, 2002). It has been found that many low back pain sufferers are dissatisfied with orthodox medicine, especially when compared to CAM (Sherman *et al*., 2004).

2.2.3.6 Consumers of chiropractic care and/ or integrative chiropractic and/ or CAM care

A comprehensive review of the consumers is provided in terms of demographic profiles; characteristics, health behaviours and risk factors; health status, healthcare access and utilisation; and the primary conditions needing treatment. In order to provide a cohesive synopsis that is easy to read, the information is provided in bullet form. Where information is contradictory, it will be discussed in more detail to provide possible explanations.

2.2.3.6.1 Demographics

Demographic profiles indicate that consumers are most likely to be:

- **Caucasian** (Fleming *et al*., 2007; Thoresen, 2006; Mahomed, 2007).
- **Younger** (Drews, 1994; Sharma, Haas and Stano, 2003; Barnes *et al*., 2004; Nahin *et al*., 2007).
- **Female** (Al-Windi, 2004; Barnes *et al*., 2004; Shmueli and Shuval, 2006; Fleming *et al*., 2007; Mahomed, 2007; Nahin *et al*., 2007). According to Arcury *et al*. (2006), females are more regular users of CAM, but not among older adults. This is possibly due to females having greater health knowledge and more health concerns (Arcury *et al*., 2006). Results from a study by Al-Windi (2004), indicated that females used CAM more often than males. Local research by Drews (1994), found that private chiropractic practitioners saw more female patients compared to male patients than chiropractic students at the clinic.
- Have **higher education** (Barnes *et al*., 2004; Shmueli and Shuval, 2006; Fleming *et al*., 2007; Nahin *et al*., 2007).
- Are **currently employed** (Singh, Raidoo and Harries, 2004; Thoresen, 2006; Benjamin, 2007; Fleming *et al*., 2007; Jaman, 2007; Khandai, 2007; Mahomed, 2007; Nahin *et al*., 2007, Venketsamy, 2007). A greater majority of patients presenting to teaching clinics are students than when compared to private practices, seeing that teaching clinics are located on the grounds of education facilities.
- Have a **higher monthly income** than non-users (Fleming *et al*., 2007; Mahomed, 2007; Nahin *et al*., 2007).
- Have a **higher socio-economic status** (Shmueli and Shuval, 2006; Nahin *et al*., 2007).

Personal resources either facilitate or impede the use of health care services. Education may be an indicator for socio-economic status, with those people of higher socio-economic status being in a better position to purchase CAM therapies. It can also be an indicator to the access of information resulting in more knowledge about CAM. However, lack of education and lower socio-economic status could result in less access to orthodox care leading to greater use of CAM-home remedies (Arcury *et al*., 2006). Financial resources, such as socio-economic status and private healthcare insurance affect the use of healthcare services in a similar way than personal resources. Many CAM therapies are either partially subsidised or not at all, meaning that patients have to pay for CAM care out of their own pockets (Sharma, Haas and Stano, 2004; Arcury *et al*., 2006).

2.2.3.6.2 The characteristics, health behaviours and risk factors

The characteristics, health behaviours and risk factors show that consumers are

- More likely to take an **active role in maintaining their health** (Barnes *et al*., 2004; Nahin *et al*., 2007).
- Engaging in **regular exercise** (Nahin *et al*., 2007).
- More likely to be **former smokers** (Barnes *et al*., 2004; Nahin *et al*., 2007). Interestingly, no differences were found in CAM use between current smokers and those who have never smoked (Nahin *et al*., 2007).
- More likely to be **current or former drinkers** than lifetime abstainers (Nahin *et al*., 2007). Among current drinkers, infrequent drinkers had the highest use of CAM and heavy drinkers had the lowest (Nahin *et al*., 2007).
- More likely to be of **normal weight** (Nahin *et al*., 2007).
- Presenting to CAM providers due to **self-referral** (Shmueli and Shuval, 2006).

Findings by Nahin *et al*. (2007), showed strong associations between the use of CAM and a number of factors indicative of poorer health and conversely, that CAM use were also associated with improved health status and increased use of self-care indicators such as regular exercise. They concluded that a subset of CAM users may be healthier or more health-conscious than those who did not use CAM (Nahin *et al*., 2007). Al-Windi (2004) also found that CAM use was associated with poor physical fitness.

2.2.3.6.3 The health status, healthcare access and utilisation

In terms of health status, healthcare access and utilisation, CAM and chiropractic patients:

- Have a **lower quality of life**, **worse health status** or **poor perception of health** (Al-Windi, 2004; Shmueli and Shuval, 2006; Fleming *et al*., 2007).
- Suffer from **life threatening diseases** such as cancer, HIV/AIDS and diabetes (Barnes *et al*., 2004; Littlewood and Vanable, 2008; Ceylan et al., 2009).
- Have a **functional limitation or disability** (Nahin *et al*., 2007).
- Have **one or more health condition(s)** (Fleming *et al*., 2007; Jaman, 2007; Khandai, 2007: Nahin *et al*., 2007; Venketsamy, 2007).
- **Delayed conventional care** due to cost or for reasons other than cost (Nahin *et al*., 2007).
- Have **access to private health insurance** (Sharma, Haas and Stano, 2004; Thoresen, 2006).
- **Use orthodox medicine and CAM concurrently** (Sharma, Haas and Stano, 2004; Shmueli and Shuval, 2006).
- Are **dissatisfied with orthodox medicine** (Barnes *et al*., 2004).
- Think **CAM is interesting** (Barnes *et al*., 2004).
- **Use CAM** to **treat HIV related concerns** (Littlewood and Vanable, 2008).
- **Use CAM** as **complementary or additional therapy** to orthodox medicine (Al-Windi, 2004).
- **Trust chiropractic care** and are **opposed to prescription drugs** (Sharma, Haas and Stano, 2004).
- Use **CAM,** seeing that it is **safe** and that **orthodox medicine** either **carries a risk** of **unwanted side** effects or that unwanted side effects have been experienced (Singh, Raidoo and Harries, 2004).
- Have experienced **improvement** in their conditions, **since using CAM** (Singh, Raidoo and Harries, 2004).

Health status is indicative of the need for health care and, therefore; individuals suffering from certain disabilities, more than one condition and chronic conditions; will utilize health care more. At the same time, the level of disabilities of the patient could also limit CAM use (Arcury *et al*., 2006). Sharma, Haas and Stano (2004) found that patients with less disability were more likely to select chiropractors, which is concurrent with findings from Arcury *et al*. (2006), which state that disabilities could limit the use of CAM.

2.2.3.6.4 Conditions treated by CAM and/ or chiropractic care providers

Conditions patients seek **chiropractic care** for, are the following:

- **Musculoskeletal conditions** (majority) with approximately 60% being for low back pain; 20% being neck, shoulder, extremity and arthritic pain; 10% being headaches, including migraine; and 10% being a wide variety of conditions caused, aggravated or mimicked by neuromusculoskeletal disorders (Meeker and Haldeman, 2002; Ernst, 2008; Evans, Williams and Perko, 2008). Local studies performed at the clinic involved in this research study, support these findings (Drews, 1994; Thoresen, 2006; Benjamin, 2007; Jaman, 2007; Khandai, 2007; Mahomed, 2007; Venketsamy, 2007).
- **Non-musculoskeletal conditions** (estimated 11- 19%) (Meeker and Haldeman, 2002; Ernst, 2008; Evans, Williams and Perko, 2008).
- Majority of conditions are **chronic** in nature (Drews, 1994; Mahomed, 2007).

The list of conditions that patients mainly seek **CAM care** for, includes

- **Musculoskeletal conditions** such as low back pain, neck pain and joint pain (Al-Windi, 2004; Barnes *et al*., 2004; Singh, Raidoo and Harries, 2004; Shmueli and Shuval, 2006).
- **Diabetes mellitus** (Singh, Raidoo and Harries, 2004).
- **Headaches** (Singh, Raidoo and Harries, 2004).
- **Hypertension** (Singh, Raidoo and Harries, 2004).

2.2.3.6.5 Conditions treated by chiropractic students at the clinic

Four retrospective cross-sectional surveys were performed at the clinic recently on the cervical, thoracic, lumbo-sacral and extremity cases on record from 1995 to 2005. Each of these will be briefly discussed according to the overall prevalence, mean age, gender, pre-existing conditions, etiology and main complaint. The purpose is to get an indication of the patients and their conditions that present to the clinic.

a. Cervical cases (Venketsamy, 2007)

- Overall prevalence: 17,92%. This was comparable with the study done by Drews (1994) on the same population which found that 16,7% of patients presenting to the clinic suffered from neck pain.
- Mean age: 36,89 years with the majority (30,2%) being in the 20 – 29 year age group
- Gender: Female predominance (59, 6%). This was comparable with the study done by Drews (1994) on the same population, which found that 52,2% of patients presenting to the clinic were female. Results by Thoresen (2006) also indicated a female predominance with 53,3%.
- Pre-existing conditions: 31%.
- Etiology: Unknown in 61,8%.
- Main complaint: Neck pain was predominant with 91,2%.

b. Thoracic cases (Benjamin, 2007)

- Overall prevalence: 3,5%. This is not a true reflection of the general population, as only 30% of cases recorded of patients presenting to the clinic was used.
- Mean age: 33,3 years.
- Gender: Female predominance (54,8%). This differed from the literature which indicated males predominantly suffer from thoracic spine pain.
- Etiology: Unknown in 61,4%.
- Main complaint: Mid-back pain (T1 – T12) was predominant with 41,4%.

c. Lumbo-sacral cases (Jaman, 2007)

- Overall prevalence: 17,3%. This differs from findings at other teaching clinics but this is not a true reflection of the general population, as only 30% of cases recorded of patients presenting to the clinic was used.
- Mean age: 39,47 years with 28,5% in the age group 20 – 29 years.
- Gender: Male predominance (55,3%). This differs from the literature but suggests males more likely to seek care for their lumbo-sacral complaints at the clinic.
- Etiology: Unknown in 57,2%.
- Main complaint: Low back pain was predominant with 89,8%.

d. Extremity cases (Khandai, 2007)

- Overall prevalence: 9,38% with upper extremity cases being more frequent than lower extremity cases. It has been noted that extremity conditions are the most common presenting condition to chiropractors after low back and neck pain.
- Mean age: 39,6 years with 30,9% in the age group 20 – 29 years.
- Gender: Male predominance (62,1%).
- Pre-existing conditions: 26,7%.
- Etiology: Unknown in 53,9%.
- Main complaint: Shoulder pain (54,9%) for upper extremity cases and knee pain (44,3%) for lower extremity cases.

It seems that patients seeking treatment at the clinic present with predominantly low back or neck pain of unknown etiology, are usually in the age group 20 – 29 years and have pre-existing conditions. The gender appears to vary according to the main complaint and/ or condition the patient presents with.

2.2.4 The chiropractic clinical encounter

2.2.4.1 Diagnosis of disorders

Chiropractors use the information gained from the presenting complaint(s), case history and physical examination as a guide to perform the relevant neurologic and orthopaedic examinations and additional studies (if needed) to reach a clinical impression or diagnosis (Kirkaldy-Willis and Bernard, 1999; Munro and Campbell, 2000; Meeker and Haldeman, 2002; Bickley and Szilagyi, 2003; Haldeman, 2005; Magee, 2006). The features identified in the history should lead to a strong suspicion of the likely diagnosis, or at least a differential diagnosis, which is either confirmed by the physical examination or a hierarchy is created of the most probable to the least probable differential diagnosis (Kirkaldy-Willis and Bernard, 1999; Munro and Campbell, 2000; Meeker and Haldeman, 2002; Bickley and Szilagyi, 2003; Haldeman, 2005; Magee, 2006). The neurological and orthopaedic examinations are then used to either confirm or eliminate the diagnosis or differential diagnoses. Once the diagnosis is determined, the chiropractor will choose a treatment plan and approximate the prognosis (Kirkaldy-Willis and Bernard, 1999; Munro and Campbell, 2000; Meeker and Haldeman, 2002; Bickley and Szilagyi, 2003; Haldeman, 2005; Magee, 2006). The prime reason for chiropractic care and, thus, spinal manipulation is always the diagnosis of a spinal functional disorder, not a visceral disorder. Patients with diagnoses not amenable to chiropractic care, such as visceral disorders, will be referred for co-management or to an appropriate specialist (Meeker and Haldeman, 2002; Haldeman, 2005).

2.2.4.2 Subluxations and treatment

The subluxation or joint dysfunction is essentially a mechanical event or behaviour of the joint components that have both local and remote influences on health and symptoms (Gatterman, 1995; Haldeman, 2005). It is a term given to an entity with abnormal function or movement in a spinal joint with neurological and vascular involvement and often, but not necessarily, structural displacement of a vertebra (Chapman-Smith, 2008). Subluxations cannot be seen on plain film radiographs, as it is a functional not an anatomical lesion (Meeker and Haldeman, 2002).

The crux of the clinical action by a chiropractor is directed at correcting the subluxation through spinal adjustment, which is the application of a load or force to specific body tissues with therapeutic intent (Meeker and Haldeman, 2002; Ernst, 2008). This load or force varies in

velocity, amplitude, duration, frequency, anatomic location, choice of levers and direction (Schafer and Faye, 1990; Bergmann, Peterson and Lawrence, 1993). Vertebrae are moved beyond their physiological range of motion into the paraphysiological space but not far enough to damage or destroy joint structures (Redwood and Cleveland, 2003; Ernst, 2008). Some chiropractors also use other therapies and modalities such as heat, cold, myofascial and electrotherapy; and give advice on or suggest supplements, therapeutic exercises, general fitness and a healthy lifestyle (Meeker and Haldeman, 2002).

Spinal adjustments and some of the examinations are performed on the beds which are designed specifically to suit the needs of the patients and the chiropractor (see Figures 2.1 and 2.2). It is composed of cushioned head, thoracic, lumbar/pelvic pieces and ankle and hand rests; all covered in leather, vinyl or cloth, and mounted on a steel frame. The cushioned headrest accommodates the patient`s nose when lying prone and the hand-rests are positioned in such a way that the patient relaxes as much as possible and ensures that the spine is in a neutral position; all of which is needed when spinal adjustments are being executed. There is also usually a changeable paper roll next to the headrest so that patients place their face on fresh paper rather than directly on the adjustment bed. If not, the onus lies on the chiropractor to provide fresh paper squares for each patient (Meeker and Haldeman, 2002).

Figure 2.1 Chiropractic adjustment bed in the clinic with the middle portion of the headrest

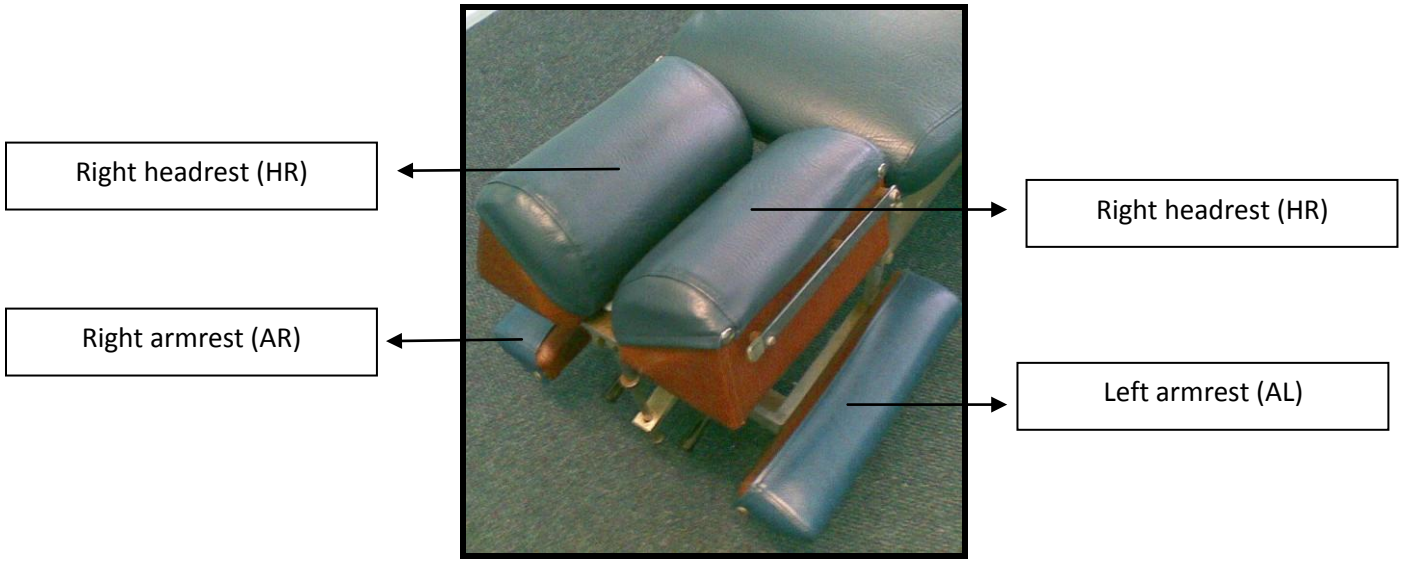


Figure 2.2 Chiropractic adjustment bed in the clinic without the middle portion of the headrest

The chiropractic clinical encounter is a high-touch, low technology health model with concern for the person rather than the disease (Meeker and Haldeman, 2002). Care which involves touching as part of the healing process, requires a certain level of trust between the physician and the patient; and the hands-on treatment and positive attitude of the typical chiropractor is reassuring and immediately satisfying. This characteristic patient and touch-orientated care has been what historically distinguished chiropractors from mainstream medicine, and has resulted in higher patient satisfaction with boosted clinical results (Meeker and Haldeman, 2002; Davis and Bove, 2008).

2.2.5 The role of chiropractors in the South African primary healthcare system

Inequity is present in the progress of health with convergence towards improved health in a large part of the world, but at the same time, with a considerable number of countries increasingly lagging behind or losing ground (WHO, 2008). South Africa is just one example of many of the developing countries that is being referred to here by the WHO.

Health care in South Africa varies from under-sourced and over-used basic public health care (delivered by the government free of charge to 80% of the population) to highly specialized private health catering for the middle- to high-income earners that can afford it and tend to belong to medical schemes - 18% of the population (About South Africa, 2008b). The government contributes 40% of all expenditure on health. This equates to approximately R33,2 billion for 38 million people, while the private sector spends approximately R43-billion on 7 million people (About South Africa, 2005).

Over the past few years there have been some improvements in the South African society:

- The proportion of households living in informal dwellings has decreased from 16,4% in 2001 to 14,4% in 2007 (About South Africa, 2008a).
- The percentages of households that have access to piped water has increased from 72,1% in 2001 to 74,4% in 2007 (About South Africa, 2008a).
- There has been a reduction in both the absolute income poverty (income of poor people) and relative income poverty (gap between average income of poor and those on poverty line) (About South Africa, 2008a).
- The social grant system is the largest form of government support to the poor and the amount of beneficiaries has increased from 2,5 million in 1999 to 12 million in 2007. Of these grants, 62% went to the poorest 40% households, while the rest went to the other 60% of poor households (About South Africa, 2008a).

According to WHO (2008), the effects of ill-managed urbanisation and globalisation accelerates the worldwide transmission of communicable diseases, and increases the burden of chronic and non-communicable disorders on the health care systems. In South Africa, an estimated 59,3% of the population is living in urban areas, which is good since evidence indicates that urbanisation is good for economic productivity and income levels (Naude, Rossouw and Krugell, 2009). According to Sanders and Chopra (2006), infectious (communicable) diseases increasingly affect the poor and chronic, whilst non-communicable affect both the rich and poor.

In light of the current situation in SA and despite improvements, the inequality gap in the healthcare keeps widening. As primary care providers, chiropractors can help fight the inequalities worsened by a shortage of primary health care providers (Gaumer, Koren and Gemmen, 2002).

2.3 HEALTHCARE-ASSOCIATED INFECTIONS

2.3.1 Definition, incidence and impact

A healthcare-associated infection is an infection that occurs in a patient in a health care facility, in whom the infection is not present or incubating at the time of admission or that is acquired during the stay in the health care facility, but arises within 48 hours after discharge. Infections contracted by staff or visitors to the facility are also considered as a healthcare-associated infection (WHO, 2002; Moor and Ferguson, 2006).

Globally, more than 1,4 million patients in developed, transitional or developing countries have a healthcare-associated infection at any point in time. In developed countries the infection rate is 5 – 10% and in developing countries it exceeds 25% (Ma *et al*., 2003; Lynch *et al*., 2007; Pittet *et al*., 2008). In South Africa, the conservative infection rate is 15% with an associated estimated mortality rate of 5% (Duse, 2005). According to Brink *et al*. (2006), approximately 1 in 7 patients entering South African hospitals, are at high risk of acquiring a healthcare-associated infection.

Healthcare-associated infections pose substantial risks to patients in terms of morbidity and mortality, as well as financially, through direct costs related to prolonged stay in the healthcare facility and indirectly due to loss of income (Moor and Ferguson, 2006; Lynch *et al*., 2007). Since resources are more limited, the economic impact of these infections is far greater in developing countries (Duse, 2005).

2.3.2 Factors influencing healthcare-associated infections

The susceptibility to acquire a healthcare-associated infection depends on the so-called “pathogenic triad” comprised of host, microbial and environmental factors. These factors will determine the distribution and development of healthcare-associated infections in any healthcare facility and can either operate systemically or locally (WHO, 2002).

The risk factors of the patients presenting to the clinic will also be briefly discussed and mainly concerns the concomitant or pre-existing diseases that they present with. All of these should be taken into account when treating patients.

2.3.2.1 Host factors

For any infection to occur, there are two required components. The first being a host and the second being that the microorganism that has to penetrate the host's barriers (innate immune system), overcomes or evades the specific / adaptive immune system and multiplies rapidly before being removed or eliminated (Talaro and Talaro, 1993; Gennery and Cant, 2006).

2.3.2.1.1 Age

Neonates, especially those born prematurely, and pediatrics have immature immune systems that are unable to mount effective immune responses against infections (Breathnach, 2005; Srivastava and Shetty, 2007). The percentage of the population in KwaZulu-Natal (KZN) from newborn to the age of 4 years, makes up 10,7% of the population (Statistics South Africa, 2006).

At the other end of the spectrum is the geriatrics that are also considered as compromised with respect to infection, as ageing is associated with impaired capacity of the immune system (Van den Biggelaar *et al*., 2004). In South Africa (SA) geriatrics are even worse off, seeing that being older is a negative determinant of health (Charasse-Pouélé and Fournier, 2006). Only 6,9% of the population in KZN is 60 years and older (Statistics South Africa, 2006). Very few geriatrics present to the clinic (Drews, 1994; Benjamin, 2007; Jaman, 2007; Khandai, 2007; Venketsamy, 2007).

2.3.2.1.2 Gender

Being male is a positive determinant of health status in South Africa (Charasse-Pouélé and Fournier, 2006). For every 100 females in KZN, there are 87,9 males, which explains why the majority patients presenting to the clinic are usually female.

2.3.2.1.3 Ethnicity

Africans make up the majority of the population in KZN (84,9%), while Caucasians only make up 5,1%. Being African is a negative determinant of health in SA, with 60% of them being poor and only 8% having access to medical aid (Charasse-Pouélé and Fournier, 2006). Despite the fact that fees at the clinic are below medical aid rates and further reductions can be negotiated, the majority of patients presenting to the clinic is still Caucasian (Drews, 1994; Benjamin, 2007; Jaman, 2007; Khandai, 2007; Venketsamy, 2007; Korporaal, 2009).

2.3.2.1.4 Health status

Obesity, malnutrition, heavy smoking and alcoholism have been shown to contribute to the increased rates of infections (Heinzelmann, Scott and Lam, 2002; Moor and Ferguson, 2006).

Trauma of any kind, including surgery, can increase the risk of developing a healthcare associated infection by resulting in the suppression of the immune system. The more invasive the surgery, the greater the suppressive effect on the immune system (Budd and Shipton, 2004).

Patients with other underlying diseases, especially when chronic, are also more prone to healthcare associated infections (WHO, 2002; Moor and Ferguson, 2006).

Cancer patients are predisposed to infections due to their decreased supply and/ or function of lymphocytes and granulocytes, wounds following invasive surgery, and malnutrition (Friese, 2007). The suppressive effects on the immune systems of these patients are much greater than for normal patients. Irradiation therapy compromises the immune system even further (WHO, 2002; Budd and Shipton, 2004).

Diabetes mellitus has been implicated in delayed wound healing, as result of disruption in collagen matrix proteins and in neutrophil chemotaxis, phagocytosis and intracellular killing. Keto-acidosis also delays cellular responses to inflammation and also contributes to delayed wound healing (Heinzelmann, Scott and Lam, 2002). It is well known that Indians are prone to having diabetes mellitus. A study by Singh, Raidoo and Harries (2004) on the prevalence and patterns of usage of CAM in the Indian community in Chatsworth, showed that 38,5% used CAM, 46,8% used CAM alone and 22% had diabetes mellitus. After Caucasians, Indians are the

largest ethnic group to receive treatment at the clinic (Drews, 1994; Benjamin, 2007; Jaman, 2007; Khandai, 2007; Venketsamy, 2007).

The symptoms of HIV depend on age, sex, culture, poverty, genetics and available therapies. Some of the symptoms found most prevalent are similar to the main complaints and symptoms from patients presenting to the clinic and some are so common they will not raise the suspicion of possible underlying serious illnesses (Makoae *et al*., 2005). These were severe headache (39,3%), sinusitis (34,8%) and dysthesias (48,9%), cough/ dyspnea (30,4%), rash (24,3%) and oral pain (24,1%). KZN has the highest rates of HIV+ people (27%) and it was estimated that by the end of 2003 the prevalence was 21,5% in adults (ages 15 – 49) (Makoae *et al*., 2005). It is to be expected that patients receiving treatment at the clinic could be HIV+ and, therefore, students should take every precaution to prevent transmission via needle-stick injuries while performing myofascial therapy, as well as to prevent patients from being exposed to possible pathogens present on the beds (Makoae *et al*., 2005).

South Africa had the third highest rate of tuberculosis (TB) nationwide in 2005, with 600 cases per 100 000 persons (Harling, Ehrlich and Myer, 2007). Poverty and inadequate healthcare systems are contributing to the global TB situation. In South Africa, other major contributors are the lack of infection control in healthcare facilities and HIV/ AIDS (Singh, Upshur and Padayatchi, 2007). Those who are HIV+ and have been infected with TB, are 30 times more likely to develop active TB than those who are negative (Broxmeyer and Cantwell, 2008). Tuberculosis was ranked the top cause of the majority of deaths in SA (14,6%) in the period of 1997 to 2003 (Statistics South Africa, 2006) and contributes to a majority of deaths in HIV/AIDS sufferers (Broxmeyer and Cantwell, 2008).

2.3.2.1.5 Nutritional status

Obesity contributes to increased rates of infections (Heinzelmann, Scott and Lam, 2002). In 2003, 56,2% of the population in South Africa was overweight or obese, with the highest prevalence seen in females (23,3%) (Schönfeldt and Gibson, 2008). Obesity increases the risk of low back pain, in that it is suspected to increase the mechanical demands on the spine (Haldemann, 2005). It is also strongly associated with hypertension, hypercholesterolaemia, diabetes, coronary heart disease and, thus, increased morbidity and mortality (Samartin and Chandra, 2001).

Eating disorders, such as anorexia nervosa and bulimia nervosa, are becoming increasingly common. Impaired immune-competence has been shown to be the cause of the increased susceptibility of these individuals to infection (Marcos, 1997).

A study on Australian chiropractic patients, found that patients consumed less fruit and vegetables than the standard recommendations, despite usually opting for healthy lifestyle choices in terms of smoking, alcohol, diet and exercise (Jamison, 2003). In 2006, 43,2% of South Africans were living in poverty, which restricts the variety of foods available in the home to mainly staples (Schönfeldt and Gibson, 2008).

2.3.2.1.6 Emotional status and Lifestyle

Physical or psychological stress can result in clinically relevant immune-suppression (Pruett, 2003; Cao, *et al*., 2004; Freestone *et al*., 2008), and the latter has been shown to diminish the efficacy of vaccines (Pruett, 2003).

Smoking has been associated with higher rates of neoplastic disease and infection, as it affects several components of the immune system. It is also suggested that it increases the rate of infection after exposure to HIV and accelerates the progression to AIDS (McAllister-Sistilli, Caggiula and Knopf, 1998). The risk of contracting low back pain is higher when smoking, due to the following reasons: it leads to coughing which increases intra-abdominal press, which in turn increases intradiscal press; it diminishes bone-mineral content leading to osteoporosis and microfractures of trabeculae and vertebral bodies; and reduces the blood flow to the vertebral bodies, leading to altered metabolic activity in vertebral endplates, thereby affecting nutrition of the intervertebral disc (Kirkaldy-Willis and Bernard, 1999; Haldemann, 2005)

Alcohol and drug abuse is associated with HIV-seropositivity and sexually transmitted diseases, in that it impairs judgement and decision-making which leads to risky behavior (Wechsberg *et al*., 2008). It has been found that alcohol abuse increases the risk of contracting infectious diseases, including TB (Donahoe and Klein, 1996). Alcohol is also often linked to major depression and can cause exacerbations of depressive disorders (Sher, 2003). Drinkers in South Africa are the top consumers of alcohol, with 16,6 litres of pure alcohol per drinker and a high prevalence of heavy drinking was reported in school surveys of Durban (Rataemane and Ratamaene, 2006).

Delta-9-tetrahydrocannibinol, the major psychoactive component in marijuana, has been shown to suppress the immune system and, thus, decrease the body`s defences against infectious pathogens (Cabral and Dove Pettit, 1998).

2.3.2.1.7 Socio-economic status

In 2006, 43,2% of the South African population was living in poverty, which increases sanitation and infection risks (Charasse-Pouélé and Fournier, 2006; Schönfeldt and Gibson, 2008). There is also a growing recognition of the link between HIV/AIDS and poverty. Income is a significant determinant of the presentation of HIV-related signs and symptoms (Makoae et al., 2005)

2.3.2.1.8 Therapeutic regimes, medication and drugs

Prior antimicrobial therapy has been identified as a risk factor for the acquisition of infection by pathogens resistant to the previous antibiotics, since the normal flora has been eliminated, modified or given the opportunity to turn opportunistic (Vincent, 2003; Roghmann and McGrail, 2006).

Corticosteroids, glucocorticoids, catecholamines, sedatives and opioids; as well as irradiation therapy, alters the immune system, thereby increasing the risk of infections (WHO, 2002; Budd and Shipton, 2004).

2.3.2.2 Microbial factors

2.3.2.2.1 Portal of entry

The portal of entry is usually specific to each organism, as they have adapted to this habitat in order to attain maximum escalation in numbers and invasion of the host. These habitats are the same anatomical areas (skin, gastrointestinal tract, respiratory tract and urogenital tract) that also support the resident flora. In cases where the pathogen enters the wrong site, they will not be infectious but some have more than one portal of entry (Talaro and Talaro, 1993; Alcamo, 1994).

2.3.2.2.2 Mode of transmission

In order to survive, some pathogens must continuously be transmitted from one host to the next and others have adapted morphologically and physiologically to survive in an environment outside a host. This transmission is restricted via the portals of entry and is typically related to the habitats inside the host and occurs either through exogenous or endogenous means (Talaro and Talaro, 1993; Atlas, 1997; Caraco and Wang, 2008).

Direct contact between a susceptible host and an infected person (fellow patient or a healthcare worker) and indirect contact via intermediate contaminated inanimate objects, are two exogenous ways by which transmission can occur (Boone and Gerba, 2007; Borkow and Gabbay, 2007). Other indirect exogenous routes include consumption or inhalation of contaminated air or water supplies (Boone and Gerba, 2007; Borkow and Gabbay, 2007).

Endogenous transmission is also a form of contact spread and occurs when the patient`s own microbial flora is transmitted from one part of the body to another (Borkow and Gabbay, 2007). The main endogenous sources of micro-organisms are the oropharynx, gastrointestinal tract and the genitor-urinary tract (Moor and Ferguson, 2006).

2.3.2.2.3 Pathogenicity and Virulence of pathogens and the Infectious dose

The qualitative measure of the actual and comparative potential of a pathogen to incite an infection is called the pathogenicity; while virulence is the quantitative measure of the ability to cause disease, microbial invasiveness and toxigenicity. There are different virulence factors that act alone or together at various stages of the infection process to contribute to the pathogen's ability of inciting infection (Talaro and Talaro, 1993; Printzen, 1996; Atlas, 1997; Wu, Wang and Jennings, 2008). They are usually implicated in direct interactions with the host tissues or in obscuring the bacterial surface from the host's defence mechanisms (Wu, Wang and Jennings, 2008).

The minimum amount of pathogens required to incite an infection is called the infectious dose and it varies depending on the pathogen. When dealing with the most pathogenic and highly virulent bacteria, quantities of only 10 to 100 are all that is needed to incite infection. Should the virulence of the particular pathogen be low but the quantity is in excess of the infectious dose or

the body's immunological defences are compromised in any way, infections can still be generated (Talaro and Talaro, 1993; Alcamo, 1994; Printzen, 1996; WHO, 2002).

2.3.2.2.4 Infection with resistant micro-organisms

Microbial resistance started rearing its head not long after the first antibiotics were used and is driven by inexorable evolutionary demands (Wright, 2003). Prolonged stay in healthcare facilities can lead to consecutive infections in patients and they are more likely to have HAI due to resistant pathogens (Vincent, 2003).

According to French (2005), microbial resistance leads to inapt experimental therapy, postponement of initialising effective treatment and the use of less efficient, more toxic and more costly and often less readily accessible drugs. The chances of treatment failure, extended or additional hospitalisation, amplified expenses and increased mortality are twice as great for patients infected with resistant micro-organisms, than for those infected with susceptible strains of the same species (Graf and Martin, 2000; Far *et al*., 2001; Byarugaba, 2004; French, 2005).

In a recent survey by Brink *et al*. (2007) on the susceptibility profiles of bacteraemic pathogens, it became evident that there is a widespread problem of antibiotic resistance in these pathogens, in the private institutions in South Africa at which the study was conducted. Resistance to ampicillin and fluoroquinolones were 84% and 20% for *E. Coli* and extended-spectrum β-lactamase production was 5%. For *Klebsiella pneumoniae* resistance rates for ceftriaxone and/or cefotaxime varied between 39 to 87% and extended-spectrum β-lactamase production was evident in 26%. The most active agents in the *Enterobacter* species were imipenem/meropenem, ertapenem, ciprofloxacin, levofloxacin and cefepime; with 100%, 94%, 88%, 87% and 80% susceptibility, respectively, and extended-spectrum β-lactamase production was 12%. Carbapenem resistance in *Pseudomonas aeruginosa* varied between 42% and 45%. Resistance of *Acinetobacter baumannii* varied between 32% and 33% for meropenem and imipenem, respectively. The incidence of oxacillin resistance for *Staphylococcus aureus* was 36%. It is to be expected that the problem of resistance is even greater in public hospitals which are under-resourced and over-used by 80% of the South African population (About South Africa, 2005). A considerate burden is placed on the public health sector by HIV, in that 8-22% of HIV+ patients access it for HIV-related care (Parikh and Veenstra, 2008).

2.3.2.3 Environmental factors

2.3.2.3.1 Location and type of healthcare facility and type of ward within facility

At health care facilities there is a congregation of people who are susceptible to infections and those who are potential sources of infection in the form of individuals already infected and/ or individuals who are carriers of pathogenic micro-organisms (WHO, 2002). Infection rates vary among different healthcare facilities and among different departments within the same healthcare facility. This is determined by the patients admitted to that facility and the definition used to define a HAI (Shook, 1995; Vincent, 2003).

Patients in developing countries have a 2 – 20 times bigger chance of contracting healthcare-associated infections due to overcrowding, understaffing, financial constraints, inadequate and rudimentary medical and medicinal resources and deficient infrastructures (Lynch *et al.*, 2007; Pittet *et al*., 2008). Another burden that developing countries often face, is infections by multi-resistant pathogens (Lynch *et al.*, 2007; Pittet *et al*., 2008). According to Macías and Ponce-de-León (2005), these health care facilities can actually serve as reservoirs for the dissemination of these resistant pathogens to the community. When taking the above mentioned results from the study by Brink *et al*. (2007) into consideration, it is clear that something drastic has to be done to curb the trends of antibiotic resistance in South African hospitals, especially those in the public health sector.

2.3.2.3.2 Antimicrobials

Antimicrobials are capable of destroying or inhibiting micro-organisms and their scope of activity ranges from a broad-spectrum where it is effective against a wide range of bacteria, including Gram-positive and Gram-negative types, to a narrow-spectrum that is effective against a specific bacteria or bacterial species (Talaro and Talaro, 1993; Atlas, 1997).

Only after identification of a definite infectious process, should antimicrobial therapy commence (Vincent, 2003). Successful treatment depends on preliminary use of the correct antimicrobial agent at the most suitable dose, in order to maximize the chances of success and curtail drug-related toxicities (Masterton *et al*., 2003). In cases where diagnosis is not clear-cut due to the identity of the infecting organism or its sensitivity not yet being ascertained, the most appropriate broad spectrum antimicrobial should be used and then adjusted as needed (Vincent, 2003;

Roghmann and McGrail, 2006). Even when the patient responds well, the spectrum must be narrowed as soon as possible in order to prevent resistance (Vincent, 2003).

Appropriate antimicrobial therapy will reduce mortality rates, decrease duration of stay in healthcare facilities and lower the overall costs of healthcare-associated infections (Masterton *et al*., 2003). Since antimicrobials affect pathogens and normal flora, inappropriate therapy such as the continued use of antimicrobials - to which the pathogens are resistant - will result in these resistant pathogens gaining the upper hand over sensitive bacteria and normal flora. This advantage will result in the resistant pathogens increasing in massive quantities, thereby forming reservoirs for resistant genes, increasing the potential for cross-contamination and the chances of infections occurring (Ižman,2003; Roghmann and McGrail, 2006).

2.3.2.3.3 Diagnostic and therapeutic interventions

Healthcare devices, such as interferential therapy and ultrasound machines used by chiropractors, may become contaminated with pathogens and form a nidus for colonisation. Subsequently, these come in contact with susceptible patients or susceptible sites on a patient and, thus, result in infection (WHO, 2002; Moor and Ferguson, 2006). Studies have shown that micro-organisms can be transferred from the patient`s skin to suction cups and sponges of interferential therapy machines, as well as to ultrasound probes (Ohara, Itoh and Itoh, 1998; Lambert *et al*., 2000)

Immunosuppressive therapy allows even low virulence pathogens and normal commensal flora the chance of becoming pathogenic (Breathnach, 2005; Moor and Ferguson, 2006). Bacterial colonisation of patients has been strongly associated with stays in healthcare facilities and prior antimicrobial therapy, with the latter being by pathogens resistant to the antimicrobials previously prescribed (Masterton *et al*., 2003; Vincent, 2003; Moor and Ferguson, 2006). These patients then act as carriers or reservoirs of infection or become infected themselves.

2.3.3 Transmission routes

Infectious diseases can be classified as either communicable or non-communicable according to the manner in which they were acquired. When an infected host transmits the pathogens to another susceptible host and an infection is established, it is termed communicable. These can be further subdivided into horizontal, meaning spread from one person to another; or vertical, with transmission from parent to offspring via ovum, placenta, sperm or milk, and can either occur directly or indirectly (Talaro and Talaro, 1993).

Non-communicable diseases on the other hand, are those acquired through endogenous transmission and inadvertent contact with a free-living pathogen in a non-living reservoir, such as soil (Talaro and Talaro, 1993).

2.3.3.1 Contact transmission

2.3.3.1.1 Direct contact transmission

The most imperative and frequent means of transmission of healthcare-associated infections is through direct contact between the skin or mucous membranes of a susceptible host and an infected person or a carrier (Borkow and Gabbay, 2007). This infection route is principally accredited to the contaminated hands of healthcare workers who fail to practice sufficient hand hygiene (Talaro and Talaro, 1993; Kampf and Kramer, 2004; Borkow and Gabbay, 2007).

According to Pittet *et al*. (2006), transmission can occur via the hands of healthcare workers and requires the following five steps, in this order:

- Presence of pathogens on patient's skin or have been shed onto inanimate objects or equipment in the immediate area surrounding the patient.
- Transfer of pathogens to the hands of healthcare workers.
- Survival of pathogens on the hands of healthcare workers for at least some time.
- Insufficient agent used for hand hygiene or suboptimal or absent hand hygiene practices by health care worker.
- Direct contact between contaminated hands of healthcare worker with another patient or with inanimate objects that will come into direct contact with that other patient.

Stay in healthcare facilities is linked to bacterial colonisation and pathogens, like *Staphylococcus aureus*, *Proteus mirabilis*, *Klebsiella* spp and *Acinetobacter* spp, which are readily found on the normal and intact skin of patients within the ranges of 100 to 10^6 (Vincent, 2003; Pittet *et al*., 2006). Results of a systematic review illustrated that single hand contact resulted in variable degrees of pathogen transfer but was most successful with *Escherichia coli*, *Salmonella* spp., *Staphylococcus aureus* (all 100%), *Candida albicans* (90%), *rhino* virus (61%), hepatitis A virus (22- 33%) and *rota* virus (16%) (Kramer, Schwebke and Kampf, 2006).

Data on which patient-care activities are most likely to result in the transmission of pathogens to the hands of healthcare workers, are scarce but results have indicated that “clean” procedures and direct contact with the intact skin of patients, leads to contamination. Research has shown that healthcare workers` hands were as contaminated after five minutes of direct contact with a patient`s skin as after one minute of contact with respiratory secretions during respiratory care (Banfield and Kerr, 2005). With time, the contamination increases linearly and given that these pathogens can survive on unwashed hands for significant periods of time, effective hand hygiene practices are of utmost importance, otherwise cross-transmission between patients can occur (Banfield and Kerr, 2005; Pittet *et al*., 2006).

Of equal and underappreciated concern, is the hand hygiene practice of visitors to the healthcare facilities, as well as those of the patients themselves. Surveys of the hands of healthy adults in the general population, showed marginally higher bacterial colony counts when collated to healthcare workers. In addition, upon comparison of the antimicrobial susceptibility of isolated colonies of healthy adults in the community with those of patients admitted to a healthcare facility, the community *P. aeroginosa* isolate was significantly more resistant than that of the inpatients for four of the ten agents tested. Few studies have investigated the role that patients’ hands play in healthcare-associated infections, but results indicate that they can also contribute to the transmission of pathogens to other parts of their own body (endogenous transmission), to fellow patients and to healthcare workers; resulting in healthcare-associated infections (Banfield and Kerr, 2005; Borkow and Gabbay, 2007).

2.3.3.1.2 Indirect contact transmission

Hosts can be infected indirectly during direct contact between their skin or mucous membranes and intermediate inanimate objects that have been contaminated through direct contact with bodily secretions, contact with soiled hands, contact with aerosolised pathogens or contact with airborne pathogens that have settled after disturbance of a contaminated object (Boone and Gerba, 2007; Borkow and Gabbay, 2007). Gastmeier *et al*. (2006) state that 2 – 5% of all healthcare-associated infections are outbreaks and their systematic analysis of data on 1561 outbreaks identified healthcare equipment and devices as sources of infection in 21,1% of cases.

Seeing that normal skin liberates roughly 3×10^8 squamae per day, and that each squame can contain more than a 100 viable pathogens and/or commensal flora, it's to be expected that intermediate inanimate objects in the patient's immediate environment or equipment, such as beds, will get contaminated (Borkow and Gabbay, 2007; Beggs *et al*., 2008). Strands of methicillin-resistant *Staphylococcus aureus* isolated from the mattresses and bed frames in a study conducted in isolation rooms, were similar to those isolated from the patients, therefore, providing evidence of transmission from the patients to the beds (Creamer and Humphreys, 2008).

A systematic review with the aim of determining the degree of contamination of healthcare equipment, amongst others, reported contamination of more than 70% in twenty of the twenty three studies. The number of cfu ranged from zero to greater than 500 and the equipment sampled included stethoscope membranes, diagnostic ultrasound, stethoscope ear tips, otoscopes, auriscopes and interferential therapy. The majority of bacterial species were skin and environmental flora, with coagulase negative staphylococci being the most common and 27% being potentially pathogenic *Staphylococcus aureus*, with 15% of these being multidrug-resistant. Other potential pathogens identified were *Pseudomonas* spp, *Acinetobacter* spp and *Pasteurella* spp (Schabrun and Chipcase, 2006).

A survey of multi-resistant bacteria in a healthcare environment, showed that 4,9% of Gram-negative and 24,7% of Gram-positive isolates were found close to carrier patients (Hakaletho, 2006). Another study investigating the role of contaminated environmental surfaces as reservoirs for methicillin-resistant *Staphylococcus aureus*, revealed that contamination occurred in the rooms of 73% of infected patients and 69% of colonised patients. The gloves of 42% of healthcare workers that had had no contact with patients became contaminated with methicillin-resistant *Staphylococcus aureus* through contact with contaminated surfaces (Cozad and Jones, 2003).

The risk of acquiring healthcare-associated infections from these contaminated inanimate surfaces not only derives from their presence but also from their persistence on these surfaces (Cozad and Jones, 2003). Once contaminated, transmission of pathogens can readily occur between animate and inanimate objects and vice versa and this will continue for as long as the pathogens remain viable (Boone and Gerba, 2007).

Lankford *et al*. (2006) investigated the recovery and transmission of vancomycin resistant enterococci and *Pseudomonas aeruginosa* on upholstery, walls and flooring in hospitals after a period of time had elapsed and after following the cleaning protocols as recommended by the manufacturers. Altogether, 14 materials were tested and these included: fabric upholstery, polyester and acrylic blend upholstery, 100% polyester upholstery, vinyl upholstery, synthetic-backed carpet, vinyl-backed carpet, vinyl composition tile, linoleum, vinyl sheet goods flooring, rubber tile flooring, latex paint with eggshell finish, vinyl wall covering with non-woven backing, microvented perforated vinyl floor covering and paper-backed wall covering. The materials were inoculated with 10^5 colony forming units of multidrug-resistant PSAE and enterococci resistant to vancomycin, gentamicin and ampicillin. All surfaces had persistent vancomycin resistant enterococci but *Pseudomonas aeruginosa* had an overall lower growth rate after 24 hours, 72 hours and 7 days after inoculation. The vinyl upholstery needs mentioning, as chiropractic beds are often covered in this material. After five minutes it had confluent growth of both micro-organisms, with non-confluent growth for vancomycin resistant enterococci up to 7 days and *Pseudomonas aeruginosa* at 24 hours and no growth from there onwards. Cleaning with anionic surfactants, quaternary ammonium compounds, 70% alcohol and vinegar successfully eliminated vancomycin resistant enterococci from 50% of the surfaces. Of the surfaces contaminated with *Pseudomonas aeruginosa*, 64,3% were successfully cleaned but the rest had persistent growth. The vinyl upholstery had no growth and was successfully cleaned. Lankford *et*

al. (2006) also demonstrated the potential for healthcare provider transmission. After hand washing, participants touched the contaminated surfaces 5 minutes after inoculation, for 5 seconds. Agar impression plates were taken, which showed the presence of confluent growth of the vancomycin resistant enterococci.

Kramer, Schwebke and Kampf`s (2006) systematic review, ascertained that most Gram-positive bacteria such as Enterococcus spp (including vancomycin resistant enterococci), *Staphylococcus aureus* (including methicillin resistant *Staphylococcus aureus*) and *Streptococcus pyogenes*, can survive for months on dry surfaces. As does many Gram-negative species like *Acinetobacter* spp., *Escherichia coli*, *Klebsiella* spp., *Pseudomonas aeruginosa*, *Serratia marcescens* and *Shigella* spp with the exception of *Bordetella pertussis*, *Haemophilus influenzae*, *Proteus vulgaris* and *Vibrio cholerae* that only persist for a few days. Mycobacteria, including *Mycobacterium tuberculosis*, and spore-forming bacteria, including *Clostridium difficile*, can also last for months. *Candida albicans* and *Torulopsis glabrata* subsisted 4 and 5 months, respectively, whereas other yeasts similar to *Candida parapsilosis* only lasted 2 weeks. Respiratory tract viruses, for example Hepatitis A virus, *astro*-, *polio* and *rota* virus persisted for roughly 2 months.

The frequency with which high touch surfaces are contaminated depends on the body sites at which patients are colonized or infected (Boyce, 2007). The normal skin flora, with colony counts of up to 10^7 bacteria per cm^2, can be found in areas with high moisture content such as the axilla, groin and toe web spaces; whereas areas like the forearm, sternum and upper back - with lower moisture content - have 10^4 or fewer bacteria per cm^2, which also has to be kept in mind (Fredericks, 2001; Larson *et al*., 2000). All this and whether the levels of contamination are sufficient enough to result in transmission, are just some of the factors that influence the potential of contaminated environmental surfaces to transmit healthcare associated pathogens (Boyce, 2007). Another very important factor is the ability of pathogens to remain viable on a variety of dry environmental surfaces, which in turn is subject to the ambient temperature, relative humidity, concentration of the initial inoculum and the surface substrate (Boyce, 2007; Bartley and Olmsted, 2008). Results from the study by Kramer, Schwebke and Kampf (2006), revealed that low temperatures (4ºC or 6ºC), higher inocula, presence of protein, serum, sputum, absence of dust and humid conditions (> 70%) improved persistence of most bacteria but the type of test material and the type of suspension medium yielded inconsistent results.

Healthcare-associated infections have been linked to many inanimate objects and equipment in the immediate environment surrounding patients but the debate on whether they have an impact on healthcare-associated infections is still controversial. Boyce (2007) states that the causal role that they play is supported by evidence that cleaning and/ or disinfection reduces the incidence of healthcare-associated infections, whereas Bartley and Olmsted (2008) are of the opinion that more direct evidence that demonstrates the direct correlation between enhanced removal of environmental reservoirs and the incidences of healthcare-associated infections are still needed. Al-Hamad and Maxwell (2008) support this by saying that as of yet, no direct proof that the environment acts as secondary reservoirs exists.

It is important to note that each researcher has their own ideas on what constitutes environmental contamination. Some limit it to the floors and walls in healthcare facilities, whereas others perceive it as inanimate objects in the immediate environment such as bedframes, case notes, blood pressure cuffs, pens and then some make no distinction between these.

2.3.3.2 Airborne and droplet transmission

This mode of transmission generally involves the dissemination of infectious aerosols from the nose or mouth of an infected host to another person or to inanimate objects in the immediate environment (Tang *et al*., 2006; Bolashikov and Melikov, 2008). Aerosols are formed when the passage of an air-stream at sufficiently high speeds, creates tongue- or wave-like disturbances in the films of fluid lining the airways, that are pulled into thin columns that are split into aerosols (Fiegel, Clarke and Edwards, 2006 ; Morawska *et al*., 2008).

If the respiratory tract is a site for replication and/or colonisation of a pathogen, it is likely to be present in the aerosols generated and released in the surrounding air during expiratory activities (Tang *et al*., 2006). The nose, oral cavity, throat and lungs provide four different habitats to which pathogens have adapted (Bolashikov and Melikov, 2008). Deep lung pathogens are dispersed into the air through coughing, while talking and sneezing will disperse pathogens located mainly in the mouth, nose or throat (Bolashikov and Melikov, 2008). The following bacteria are common causes of healthcare-associated infections and even though contact transmission is their main route of infection, airborne transmission can also occur:

Staphylococcus aureus, methicillin-resistant *Staphylococcus aureus*, *Mycobacterium tuberculosis*, *Acinetobacter* spp., *Aspergillus* spp., *Pseudomonas* spp. and *Legionella* spp (Tang *et al*., 2006).

When coughing, sneezing, talking or breathing; different sizes, quantities and concentrations of particles are generated for each individual making it difficult to determine, especially under experimental conditions. A sneeze generates over 40 000 droplets and a single cough about 3000 droplet nuclei, the same amount as five minutes of talking (Tang *et al*., 2006). Zhu, Kato and Yang (2006) concluded that in each cough the summative amount of saliva expelled was more than 6,7 milligrams at a maximum velocity of 22 meters per second. During visualisation of the dispersion of the droplets, they found that particles expelled during a cough travelled at high speed over a distance of 30 centimetres or more, then steadily disintegrated and dispersed in the air while its velocity reduced. The tracer used travelled more than 2 metres, indicating that droplets with diameters less than that of the tracer could spread even further.

According to Bolashikov and Melikov (2008), 71% of all aerosols dispersed by coughing are small and the rest are large. According to Fiegel, Clarke and Edwards (2006), the highest number of droplets less than one (< 1) micrometer in size is produced by mouth breathing and Morawska *et al*. (2008) states that 80 – 90% of aerosols from human expiratory activities (breathing, talking, sneezing, coughing and laughing) are smaller than one micrometer.

The size of the aerosols impacts hugely on the disease transmission process. Droplet transmission occurs when infection occurs due to droplets that have come into contact with the mucous membranes or conjunctiva of another individual. Large droplets of > 60 micrometers and small droplets of ≤ 60 micrometers are implicated in this infection route since they tend to be produced by coughing and sneezing at high initial air velocities. This causes them to evaporate, settle out of the air quicker due to gravity and deposit on the nearest environmental surface. Airborne transmission refers to the inhalation of droplet nuclei sized <10 micrometers that's travelling at slower initial air speeds (generated by activities such as breathing). These are dispersed widely and are capable of remaining airborne for extended periods. Small droplets can also participate in airborne transmission as they can evaporate rapidly and become droplet nuclei themselves (Tang *et al*., 2006; Bolashikov and Melikov, 2008; Chao *et al*., 2008). Due to their size, larger droplets have a higher capacity to transmit pathogens, but due to the effective filtering mechanisms, only small percentages of droplets greater than 10 micrometers can reach

the alveolar or pulmonary regions during moderate breathing conditions. Thus, depending on the pathogen and the target infection site, smaller droplets can be more infectious (Longest and Kleinstreuer, 2004; Chao *et al*., 2008).

It should also be kept in mind that air can become contaminated with infectious aerosols through vomiting, flushing of toilets and sewage removal (Bolashikov and Melikov, 2008; Morawska *et al*., 2008). Dust and skin squamae carrying pathogens that are daily shed into the environment by healthy or infected individuals and those acting as carriers are transmitted in a similar manner to aerosols (Borkow and Gabbay, 2007).

2.3.4 Pathogens commonly implicated in Healthcare-associated infections

The range of pathogens accountable for healthcare-associated infections has shifted from Gram-negative bacilli to Gram-positive cocci, such as staphylococci and enterococci (Struelens, Denis and Rodriquez-Villalobos, 2004; Moor and Ferguson, 2006). *Escherichia coli* and *Staphylococcus aureus*, the most commonly implicated bacteria in the majority of healthcare-associated infections, are in actual fact constituents of the body's normal flora (Struelens, Denis and Rodriquez-Villalobos, 2004). Under normal circumstances they protect the host from colonisation by pathogenic flora but when the host's immune status becomes compromised, inappropriate antibiotic therapy allows overgrowth or if there is a transmission to sites outside the natural habitat, infections can occur (WHO, 2002).

In Table 2.1 some of the micro-organisms implicated in healthcare-associated infections can be seen along with the inanimate objects they tend to contaminate and their survival thereupon.

Table 2.1: Summary of healthcare-associated pathogens (Hota, 2004)

Pathogen	Types of environmental contamination	Length of survival of organism	Evidence of transmission
Influenza virus	Aerosolisation after sweeping Survival on fomites	24–48 hours on non-porous surfaces	Fomite to hands of health care workers
Parainfluenza virus	Survival on clothing and non-porous surfaces	10 hrs on non-porous surfaces 6hrs on clothing	Not proven
Noroviruses	Persistent outbreaks on ships Extensive environmental contamination Possible aerosolisation	≤14 days in stool samples ≤12 days on carpets	Not proven
Hepatitis B virus	Environmental contamination with blood	7 days	Lancets, electroencephalographic electrodes in outbreaks Nosocomial transmission to health care workers
Severe acute respiratory syndrome -associated coronavirus	Positive results of cultures of samples from an emergency department environment High–secondary attack rate events (i.e., super spreading events)	24–72 hours on fomites and in stool samples	Not proven but suspected Clothing not clearly affected
Candida species	Contamination of fomites	3 days for *Candida albicans* and 14 days for *Candida parapsilosis*	Suggested by molecular epidemiologic findings
Clostridium difficile	Extensive environmental contamination	5 months on hospital floors	Correlation between degree of environmental contamination and health care worker hand contamination
Pseudomonas aeruginosa	Contamination of sink drains	7 hours on glass slide	Multiple types in environment that do not correlate with acquisition Most acquisition is endogenous
Acinetobacter baumannii	Extensive environmental contamination	33 days on plastic laminate surfaces	Multiple types in environment that do not correlate with acquisition
Methicillin-resistant *Staphylococcus aureus*	Burn units extensively contaminated	≤ 9 weeks after drying 2 days on plastic laminate surfaces	Evidence of spread from environment to healthcare worker Phage types in environment discordant with patient phage types
Vancomycin-resistant enterococci	Extensive environmental contamination	≤ 58 days on countertops	Evidence of spread from environment to healthcare worker High risk of acquisition by patients in contaminated rooms

2.4 INTERVENTION STRATEGIES

2.4.1 Decontamination

Contamination is the tarnishing of articles (such as healthcare equipment) with potentially infectious and unwanted material varying from organic substances and micro-organisms to inorganic substances such as dust or chemical residues (De Zoysa and Morecroft, 2007).
Equipment can be divided into three categories based on the risk of acquiring an infection, according to the Spalding classification, as in the Practical guidelines for infection control in healthcare facilities (WHO, 2004) and as used by De Zoysa and Morecroft (2007):

- Critical equipment penetrates sterile tissues, cavities or the bloodstream and must be sterilised before use.
- Semi-critical equipment come into contact with the intact non-sterile mucosa or non-intact skin and, thus, poses an intermediate risk. They call for high- level disinfection and sterilisation can be considered.
- Non-critical equipment comes into contact with intact skin or doesn't come into contact with the patient at all and only requires cleaning after use.

In order to ensure the equipment is safe for use or to discard, the microbial growth is brought under control through decontamination which involves the removal, inactivation or obliteration of micro-organisms with cleaning followed by disinfection and / or sterilisation (De Zoysa and Morecroft, 2007).

2.4.2 Cleaning

Cleaning is the physical elimination of foreign or organic material and biofilms on which pathogenic micro-organisms flourish from a surface or an article through manual or mechanical means. There are three kinds of solutions available for use during cleaning and these are detergents (remove organic material and suspend oily substances), disinfectants (rapidly destroy or inactivate micro-organisms) and detergent-disinfectants (Hota, 2004; De Zoysa and Morecroft, 2007).

Manual cleaning is performed by immersing the article in a non-corrosive, non-abrasive and free-rinsing solution at 35°C, removing the visible contaminants, rinsing it with clean hot water and drying it by hand. In certain cases, such as with electrical equipment, the articles are wiped in a cloth soaked in the detergent solution until all visible contaminants are removed, then hand-

dried to prevent damage (De Zoysa and Morecroft, 2007). Manual cleaning is used when mechanical or automated cleaning is inappropriate or unavailable. Mechanical cleaning often incorporates disinfection and involves thermal and chemical washer disinfectors, as well as ultrasonic cleaners (De Zoysa and Morecroft, 2007).

Cleaning only reduces the microbial load and doesn't destroy the pathogenic micro-organisms, but is necessary since the organic material or biofilms could avert disinfectants or sterilants from having contact with the equipment and may also alter the chemical action of the disinfectant (WHO, 2004; De Zoysa and Morecroft, 2007). When used in conjunction with disinfectants, it can also significantly reduce the microbial load in a much shorter time span (Hota, 2004).

2.4.3 Disinfection and Sterilization

The eradication of numerous or all pathogenic micro-organisms, except for the bacterial spores, is the process called disinfection which is used on delicate or heat-sensitive articles. Various levels of disinfection can be attained, depending on the techniques used but all of these are best suited for the use on semi-critical and non-critical articles (WHO, 2004; De Zoysa and Morecroft, 2007).

Sterilization is the process that destroys roughly all forms of microbial life, even the bacterial spores and viruses. A sterile article has a sterility assurance level of 10^{-6}, which means that the chance of an organism being present on it is one in a million. It is for this reason that all critical equipment requires sterilisation before use.

2.4.3.1 Parameters influencing disinfection and sterilisation

A term describing the disinfective or sterilisation processes with the root "-cide" indicates that it is capable of killing the target organisms, while if the root is "-static" it means that the agent only inhibits growth of the target organisms (Talaro and Talaro, 1993).

Talaro and Talaro (1993), Bessems (1998), Maillard (2005) and Rutala and Weber (2008) state that various factors inherent to the disinfective or sterilising process and the microbial cells in question, have an effect on the activity and thus the efficacy, as well as in determining whether it is "-cidal" or "-static". These factors are listed in order of importance:

- Factors inherent to the process
    - Concentration: A chemical agent can be applied for use as a disinfectant, an antiseptic and as a preservative depending on which concentration is being used. The strength or concentration of a chemical agent is often misunderstood as it is expressed in more than one way. Usually in dilutions, a small part of the chemical solvent is diluted in large amounts of water to attain a certain ratio but some are effective in very high concentrations and these are expressed as parts per million. Percent dilutions involve the addition of the chemical solvent according to weight or volume to achieve a certain percentage in the solution. Solutions of low dilution or high percentage contain more of the active chemicals and are, thus, more effective but a balance should be kept between efficacy and toxicity.
    - Contact / Exposure time: Time is needed so as to penetrate and act on the micro-organisms. A longer contact time is, thus, associated with increased activity and the eradication of more micro-organisms.
    - Organic matter, solvents or inhibitors: These interfere by protecting the micro-organisms and hindering the penetration of heat or chemical agents and can even inactivate certain chemical agents.
    - Temperature: Some processes depend specifically on the temperature, while some chemical agents rely upon the combination of chemical inactivation and elevated temperatures in order to be effective.
    - pH: Affects the stability and ionisation of chemical agents, as well as the growth of micro-organisms and their surface charge which augments the activity of cationic chemical agents.
    - Composition of surface: Micro-organisms are destroyed more readily on smooth, solid objects than porous objects or those with spaces that can trap soil.
- Factors inherent to the relevant microbial cell
    - Presence of biofilm: Interferes with disinfection by protecting the micro-organisms from the chemical agents.
    - Type of micro-organism: The most resistant micro-organisms are bacterial spores then mycobacteria, Gram-negative bacteria, Gram-positive bacteria and fungi. The resistance of viruses depends on their structure, as well as the presence of an envelope. Those with envelopes tend to be more sensitive to chemical agents.
    - Number of micro-organisms: The more micro-organisms present the more difficult it is to destroy them and the more time is needed.

Knowledge of these factors will assist in the selection of an appropriate course of action but according to Maillard (2005), one has to keep in mind that it is not always possible to foresee which micro-organisms will be needed to be dealt with. He does, however, add that the load of organic matter, the extent of microbial contamination and the presence or absence of a biofilm can be predicted.

2.4.3.2 Effects of disinfection and sterilisation

According to Denyer and Stewart (1998), disinfection and sterilisation incurs damage to micro-organisms. These have been listed in order of increasing severity, ranging from bacteriostatic to bacteriocidal action:

- Disruption of transmembrane proton motive force, resulting in uncoupling of oxidative phosphorylation and inhibition of active transport across the membrane.
- Inhibition of respiration or catabolic/ anabolic reactions.
- Disruption of replication.
- Loss of membrane integrity leading to leakage of intracellular constituents such as potassium cations, inorganic phosphate, pentoses, nucleotides and nucleosides and proteins.
- Lysis.
- Coagulation of intracellular material.

2.4.3.3 Physical methods of disinfection and sterilisation

There are various physical methods than can be used for the disinfection or sterilisation of articles such as radiation, sound waves, filtration, heat and cold. Only the latter two will be discussed as they have most relevance on this research study.

2.4.3.3.1 Heat

Micro-organisms grow over a large range of temperatures but growth rates lessen above and below the optimal temperature and generally death rates exceed the growth rate. Elevation above the maximum temperature is microbicidal by denaturing proteins and dehydrating the micro-organisms, while temperatures below the minimum tend to have static effects. Thermal death time is the time needed for killing a specific micro-organism at a given temperature. Each micro-organism also has a thermal death point, which refers to the temperature at which it dies in a given time (Talaro and Talaro, 1993; Alcamo, 1994; Atlas, 1997).

The two physical states of heat used in microbial control is moist heat, which consists of boiling or vaporized water (steam) with temperatures ranging from 60°C to 135°C; and the air with low moisture contents, heated by a flame or electric coil to temperatures between 160°C and several thousand degrees, are referred to as dry heat. It works slower than moist heat and when the length of exposure is identical, moist heat can kill at lower temperatures than dry heat, since it coagulates and denatures cytoplasmic proteins which occur faster and at lower temperatures in the presence of moisture. Moist heat also damages cell membranes, ribosomes, DNA and RNA. Through dry heat cells become oxidised, the protoplasm gets concentrated through dehydration and at very high temperatures the cells can also be incinerated. It does denature proteins and DNA but since proteins are more stable at higher temperatures, it takes longer and higher temperatures for this to occur (Talaro and Talaro, 1993; Alcamo, 1994).

a. Moist heat methods employed for microbial control

Articles or equipment are sterilised or disinfected through moist heat by means of steam under pressure, non-pressurised steam, boiling water and pasteurisation.

Pressurised steam is the most reliable method of sterilisation but since 100°C is the highest temperature that steam can reach under normal atmospheric pressure, the use of an autoclave is needed. This device can subject pure steam to pressures higher than one atmosphere, resulting in the higher temperatures required to kill the micro-organisms. Sterilisation is attained when the steam condenses against the surfaces of the articles and gradually raises them to its temperature. Through experience, the optimal pressure of two atmospheres resulting in a temperature of 121°C has been found to be the best temperature/ pressure combination and

increasing the pressure any higher won't reduce the exposure time and can only cause damage to the articles. Exposure time varies from 10 – 40 minutes depending on the bulk of the articles inside the autoclave chamber (Talaro and Talaro, 1993; Alcamo, 1994).

Articles or materials that are unable to withstand the higher temperatures are subjected to free-flowing non-pressurised steam for 30 to 60 minutes. This method is called intermittent sterilisation but the temperatures reached are not adequate to reliably kill spores. After exposure to the steam the articles are incubated for 24 hours and the cycle is then repeated twice so that the articles get exposed to the steam a total of three times. Since the temperature never exceeds 100°C, the highly resistant spores that have not germinated upon incubation, can still be viable (Talaro and Talaro, 1993; Alcamo, 1994).

Boiling water is the easiest and least toxic method of decontamination for use at home or in the clinic and since the temperatures don't exceed 100°C again, it is technically for disinfection purposes as sterilisation cannot be assured. The articles must be cleaned first and should be exposed to the boiling water for at least 30 minutes, as then the resistant non-spore-forming pathogens should be destroyed. At high altitudes the time of exposure should be increased to compensate for the lower boiling point of water (Talaro and Talaro, 1993; Alcamo, 1994).

Pasteurisation is when heat is applied to fresh beverages (milk, fruit juice, beer and wine) to eradicate the micro-organisms that have the potential to spoil these foods and cause disease but while still retaining the flavour and nutrient values. Temperatures of 63- 66°C for 30 minutes or 71,6°C for 15 seconds are used with the latter being preferred, as it is less likely to change the flavour while being more effective against the non-spore-forming pathogens and some of the resistant species. Endospores and thermoduric species such as lactobacilli, micrococci and yeasts will not be killed (Talaro and Talaro, 1993; Alcamo, 1994).

b. Dry heat methods employed for microbial control

The most rapid method of sterilisation is by utilising a direct flame either by way of a Bunsen burner or a furnace. The flame of a Bunsen burner can reach a temperature of 1870°C at its hottest point and is employed to sterilise a bacteriological loop before working with inoculum. The temperatures of furnaces range from 800° to 6500°C and is used for the incineration of disposable objects such as gowns and certain plastic appliances. The intense heat ignites and reduces the micro-organisms to ashes and dust (Talaro and Talaro, 1993; Alcamo, 1994).

Sterilisation can also be achieved by placing articles inside a dry oven where heat is transferred to the articles as the hot air circulates through the enclosed compartment, heating and killing the micro-organisms and spores. Dry heat doesn't penetrate materials easily and, therefore, two to four hours at temperatures of 150°C to 180°C is necessary. This method is ideal for dry powders, water-free oily substances, glassware and metallic instruments that may be corroded by steam (Talaro and Talaro, 1993; Alcamo, 1994).

2.4.3.3.2 Cold and dessication

Cold may kill some micro-organisms but merely retards the majority as the gradual cooling, long-term refrigeration and deep-freezing have no adverse effects. Temperatures of -70°C to -135°C actually provide an environment for the preservation of certain bacteria, yeasts, moulds and viruses and others (psychrophiles) continue growing and secreting toxins (Talaro and Talaro, 1993; Alcamo, 1994).

Exposure to normal room air leads to the gradual dehydration or dessication of vegetative cells, depending on the substrate, the length of exposure and the relevant micro-organism (Talaro and Talaro, 1993; Alcamo, 1994).

2.4.3.4 Chemical methods of disinfection and sterilisation

Chemical disinfection involves the use of germicides (also called microbicides) which are compounds or mixtures that exterminate micro-organisms through the use of chemical or physiochemical processes (Talaro and Talaro, 1993). Antiseptics are the chemical agents referred to that are applied directly to skin or mucous membranes, wounds or surgical incisions and those used on inanimate surfaces or objects are called disinfectants (Talaro and Talaro, 1993; McDonnell and Russel, 1999).

2.4.3.4.1 Choosing chemical agents

Certain qualities have been identified as being desirable in a chemical agent but no chemical agents have thus far had all the qualities. These qualities are listed by Talaro and Talaro (1993) as:

- Rapid action even when in high dilutions (in other words, in low concentration).
- Solubility in water or alcohol and with long-term stability.
- Broad spectrum microbicidal action without toxic negative side effects to humans or animals.
- Penetration into inanimate surfaces to sustain a cumulative or persistent action.
- Resistance to become inactivated by organic matter.
- Non-corrosive or non-staining.
- Sanitising and deodorising.
- Readily available and inexpensive.

2.4.3.4.2 Mechanism of action of chemical agents on general cellular targets

The main cellular targets of the chemical agents are the cell wall, the cytoplasmic membrane and the cytoplasm. Table 2.2 depicts the mechanisms of action on these general cellular targets along with the consequences.

Table 2.2: Mechanism of action on general cellular targets (Denyer, 1995)

Target region	Damaging event	Consequences
Cell wall	Structural/functional changes Release of wall components Initiation of autolysis	Abnormal morphology and construction Non-specific increase in cell permeability Lysis
Cytoplasmic membrane	Loss of structural organisation and integrity	Progressive leakage of intracellular material (e.g. potassium ions, inorganic phosphate, amino acids, pentoses, nucleotides, protein) Initiation of autolysis
	Selective increase in permeability to protons and other ions	Dissipation of proton motive force Uncoupling of oxidative phosphorylation Inhibition of active transport Loss of metabolic pools
	Inhibition of membrane-bound enzymes	Inhibition of respiration and energy transfer Inhibition of ATP synthesis Inhibition of substrate oxidation Inhibition of transport processes
Cytoplasm	Inhibition of cytoplasmic enzymes Interaction with functional biomolecules (e.g. DNA, RNA)	Inhibition of catabolic and anabolic processes
	Coagulation and precipitation of Cytoplasmic constituents (usually at high biocide concentrations)	Denaturation of enzymes Destruction of biomolecules

2.4.3.4.3 Categories of chemical agents

There are many general groups of chemical compounds into which chemical agents can be divided, each with its own spectrum of activity on the various micro-organisms, as depicted in Table 2.3. This will influence the choice of disinfectant chosen but the level of toxicity and level of disinfection required has to be taken into consideration as well.

Table 2.3: Characteristics of main disinfectant groups

Chemical agent	Bactericidal activity	Tuberculocidal activity	Fungicidal activity	Virucidal activity	Sporicidal activity	Human toxicity	Level of disinfection	Mode of action
Alcohols (ethanol, isopropanolol, n-propanol)	+++	+++	+++	+++	-	Moderate	Intermediate	Penetrating agents → loss of cellular membrane function → release of intracellular components, denaturing proteins & inhibition of DNA, RNA, protein & peptidoglycan synthesis
Aldehydes Formaldehyde	+++	+++	+++	+++	+	High	High	Cross-linking agents → interact with unprotected amines in outer cell wall → loss of cell wall function.
Gluteraldehyde	+++	+++	+++	+++	+++	High	High	Cross-linking of thiol, sulphydryl & amino groups → inhibition of protein, DNA & RNA synthesis.
Biguanides Chlorhexidine	+ against Gram-negative bacilli	-	+	-	-	Low		Damage cell wall & outer membrane → collapse of membrane potential & intracellular leakage. Enhanced passive diffusion mediates further uptake → coagulation of cytosol
Halogen-releasing agents Hypochlorites & chloramines	+++	++	++	+++	+	Moderate	High	Highly active oxidising agents → destroy cellular activity of proteins. Disrupts oxidative phosphorylation & membrane-associated activities
Iodophores	++	++	+	++	-	Moderate		Iodine reacts with cysteine & methionine thiol groups, nucleotides & fatty acids → resulting in cell death
Peroxygens Hydrogen peroxide	+ against staphylococci & enterococci	++	++	++	++	Low	High	Hydrogen peroxide produces hydroxyl free radicals → function as oxidants → react with lipids, proteins & DNA. Sulphydryl groups & double bonds targeted especially → increasing cell permeability
Peracetic acid	+++	++	++	++	++	High	High	
Phenolic compounds Phenol	+++	+++	+++	+	-	High	Intermediate	Increase cytoplasmic membrane permeability → progressive leakage of intracellular constituents. Permeable to protons → dissipate proton motive force & uncouple oxidative phosphprylation, coagulation of cytoplasm → cell lysis
Quaternary ammonium compounds # (Benzalkonium chloride & cetrimide)	+ against Gram-negative bacilli	-	+	+	-	Low	Low	Damage cell wall & cytoplasmic membrane mediated by binding to phospholipids → loss structural integrity of cytoplasmic membrane. Enhances further uptake → induces leakage of intracellular

+++ very active ; ++ active ; + less active ; - not active

(WHO, nd; Sheldon, 2005; WHO, 2002)

- main component of the disinfectant used at clinic is a Quaternary ammonium compound

2.4.3.4.4 Selectiveness of chemical agents

Not only can chemical agents be categorized according to the general chemical group they belong to, but also by their selectiveness. This ranges from the least selective, that inflict severe damage on many cell parts, to moderate selective agents with intermediate specificity, and lastly to more selective agents with specific cell structures or functions as targets usually effective only on certain micro-organisms. More than one target can be affected by chemical agents and their use may result in both primary and secondary damage that sooner or later ends in cell death (Talaro and Talaro, 1993).

Table 2.4 has been adapted from the work by Denyer and Stewart (1998) and depicts the prospective specific cellular targets and the mechanism in which it affects these. It also provides examples of the chemical agents along with their antimicrobial effects.

Table 2.4: Mechanism of interaction with specific cellular targets (Denyer and Stewart, 1998)

Mechanism of interaction	Examples of biocides	Specific targets	Antimicrobial effects
Chemical reactions			
Oxidation of predominantly thiol groups	Heavy metals Hypochlorites	Thiol-containing cytoplasmic and membrane-bound enzymes	Metabolic inhibition
General alkylation reactions	Gluteraldehyde Formaldehyde	Biomolecules containing amino, imino, amide, carboxyl and thiol groups. Intermolecular cross-linking can also occur.	Metabolic and replicative inhibition and possibly some cell wall damage
Halogenation	Hypochlorites Chlorine-releasing agents	Amino groups in proteins	Metabolic inhibition
Free radical oxidation	Hydrogen peroxide Peracetic acid	Enzyme and protein thiol groups	Metabolic inhibition
Ionic reaction			
Electrostatic interaction with phospholipids	Quaternary ammonium coumpounds Chlorhexidine Biguanides	Cytoplasmic membrane integrity Membrane-bound enzyme environment and function	Leakage Respiratory inhibition Intracellular coagulation
Physical reactions			
Penetration/ separation into phospholipid bilayer Possible displacement of phospholipid molecules Intramembrane molecular cycling	Phenols Weak acids	Transmembrane pH gradient Membrane integrity	Leakage Disruption of transport, respiratory and energy coupling processes
Solution of phospholipids	Alipathic alcohols	Membrane integrity	Leakage
Membrane-protein solubilisation	Anionic surfactants	Cytoplasmic membrane integrity Membrane bound enzyme environment and function	Leakage Uncoupling of energy processes Lysis

2.4.4 Proposed standards for assessment of surface hygiene

The proposed standards are based on standards applied in the food industry but are tailored to reflect the differences between risk management in food preparation and the risk for acquiring an infection in hospital (Dancer, 2004). Dancer (2004) proposes that surfaces should be inspected for certain indicator organisms, as well as for the total amount of micro-organisms present in that specified area. Indicator organisms are considered to be of high risk to any patient in any amount. They include *Staphylococcus aureus*, *Clostridium difficile*, multiply resistant Gram-negative bacilli, vancomycin-resistant enterococci and *Salmonella* spp. Sufficient quantities of any type of micro-organism could be of equal risk to patients in terms of acquiring an infection when concentrated in a specified area. The presence of indicator micro-organisms and a total aerobic colony count in quantities of more than 1 cfu/ cm^2 and 5 cfu/ cm^2, respectively, should generate immediate attention towards the quality and quantity of the disinfection techniques employed (Dancer, 2004).

2.4.5 *Taski Sani Des J-flex*

Taski Sani Des J-flex is the product currently being used by the clinic for the cleaning and disinfection of the beds. It is classified as a cleaner/ disinfectant for professional use and is described as a mixture of non-hazardous substances and the more dangerous components are 5 – 15% alkyl dimethyl benzylammonium chloride (quaternary ammonium compound), 5 – 15% alkyl alcohol ethoxylate (non-ionic surfactant), less than 5% sodium carbonate and less than 1% benzyl salicate.

The instructions for use read:

- Apply product on surface and leave on for 5 minutes.
- Remove the soiling with a damp cloth or scourer.
- Rinse with clean water.

2.4.6 Chiropractic guidelines on hand and adjustment bed sanitising

2.4.6.1 Hand antisepsis

In order to disinfect hands, use an alcohol based gel containing at least 60% alcohol and emollients for skin sensitivity. It should also be available in all areas where skin-to-skin contact by hand occurs, for example in each clinic room, in the clinician`s office and in the classroom utilised for practicing adjustment techniques (Evans *et al*., 2009).

When hands are visibly soiled, wash hands as recommended by Evans *et al*. (2009) before using the alcohol-based hand hygiene product:

- Jewellery and watches should be removed. Clothing should not touch the sink.
- Water should be turned on and adjusted to warm.
- Hands should be wet to above the wrists, lathered up with soap and kept below elbows throughout the hand washing.
- All areas of hands and wrists should be washed for at least 20 seconds. In order to scrub between fingers, they should be interlaced.
- If hands were exposed to infectious material, scrub beneath fingernails with a nail brush or nail stick.
- Rinse wrists and hands well and repeat the procedure if hands were exposed to infectious material.
- Dry hands thoroughly with paper towel, and dispose of it properly.
- A dry paper towel should be used to turn off the water faucet and open the door.

When using an alcohol-based hand hygiene product, Evans *et al*. (2009) advises:

- Dispense a portion of gel the size of a dime or R1 coin on the palm of hand and rub over hands and wrists including each finger to the ends
- After making sure all surface areas of the hand has been covered, hands should be allowed to air dry. Excess should not be wiped off.

At least one of these two measures should be performed between each patient contact, whether in clinic or when practicing adjustment techniques. In cases where either the doctor or patient has a visible skin lesion, gloves should be worn during the examination and treatment, after which the gloves should be exposed off appropriately and hand sanitising should follow immediately (Evans *et al*., 2009).

2.4.6.2 Adjustment bed disinfection

Evans *et al*. (2009) suggests the use of a disinfecting wipe or solution for the cleaning of adjustment beds in the following manner:

- Soiled face paper should be removed from facial / head piece portion of bed so that head piece is exposed.
- Solution or disinfectant wipe should be applied evenly to cover the surface of face/ head piece, including face paper bar, chest piece of table and hand rests.
- If spray is used, the entire surface must be covered by spreading solution with paper towel.
- The beds should be allowed to air dry at least 45 seconds.
- New face paper should be placed on the table.

This procedure should be followed between each patient and when indicated additional or all surfaces of the adjustment beds should be disinfected. When blood or body secretions have come into contact with the adjustment beds, a stronger chemical agent is advised (Evans *et al*., 2009).

2.5 PREVIOUS STUDIES

Bifero, Prakash and Bergin (2006) performed a study to investigate the role of chiropractic adjusting tables as reservoirs for microbial diseases. They obtained samples from nine vinyl-covered chiropractic adjusting tables by swabbing the head, arm, thoracic and abdominal pieces twice. The swabs were processed and incubated at 25°C for 24 hours for the fastidious and non-fastidious bacteria and at 30°C for up to 7 days for yeast. Tables 2.5 and 2.6 show the bacteria identified on each bed and the bacterial load on the different sections of the beds.

Table 2.5: Bacterial isolates identified on the various sections of beds (Bifero, Prakash and Bergin, 2006)

Bacterial isolate	Number of isolates	Number of rooms and percentages containing isolate	Predominant area of growth:
Coagulase-negative staphylococci	25	9 (100%)	Headrest, armrest and thorax
Staphylococcus aureus	14	7 (78%)	Armrest
Micrococci	7	3 (33%)	Headrest and thorax
Moraxella spp	3	2 (22%)	Headrest and armrest
Methylobacter Acinetobacter	3	1 (11%)	Headrest and armrest
Acinetobacter spp	2	2 (22%)	Headrest
Bacillus spp	2	2 (22%)	Headrest and armrest
Methicillin resistant *Staphylococcus aureus*	2	2 (22%)	Armrest
Serratia marcescens	2	1 (11%)	Headrest and thorax
Alcaligenes spp	1	2 (22%)	Armrest
Escherichia coli	1	1 (11%)	Thorax
Pasteurella multocida	1	1 (11%)	Armrest
Propionibacterium	1	1 (11%)	Armrest
Pseudomonas aeruginosa	1	1 (11%)	Armrest

Table 2.6: Average bacterial load on the various sections of the beds (Bifero, Prakash and Bergin, 2006)

Areas sampled	Average colony-forming units	Predominant species
Headrest	40	Coagulase-negative staphylococci, *Staphylococcus aureus*, *Acinetobacter, Moraxella*
Thorax	24	Coagulase-negative staphylococci, *Staphylococcus aureus*
Armrest	42	*Staphylococcus aureus*, Methicillin resistant *Staphylococcus aureus*

In a similar study by Evans *et al*. (2007), samples were obtained from 10 vinyl-covered beds, processed and incubated at 35°C for up to 48 hours. Unfortunately, they didn't quantify the bacterial counts but identified several Gram-positive and Gram-negative bacteria. Two of the tables contained Gram-negative bacteria, while all the tables contained Gram-positive bacteria; including *Staphylococcus epidermidis*, *Staphylococcus saprophyticus*, *Staphylococcus aureus* and Methicillin resistant *Staphylococcus aureus*.

The last study, done by Evans *et al*. (2008), examined cloth-covered beds as sources of allergens and pathogenic microbes. Organisms present on the beds included mould spores (possibly *Candida* colonies) and the following bacteria: *Micrococcus luteus, Propionibacterium acnes, Staphylococcus epidermidis, Staphylococcus aureus, and Bacillus subtilis.* In this study the bacterial counts were also not quantified.

These research studies show that beds harbour micro-organisms and could, thus, serve as potential reservoirs for infectious bacterial pathogens.

The aim of this research study was to assess the beds as reservoirs for normal microbial flora and infectious bacterial pathogens, at a clinic in South Africa.

CHAPTER 3 : METHODOLOGY

3.1 STUDY DESIGN

This study was a quantitative clinical investigation that has been subdivided into four major parts.

Part 1 was further subdivided so that it comprised of three parts. Part 1.1 consisted of verifying the presence of the micro-organisms from incubated samples obtained from the headrests and armrests of the beds. If any were found residing on the beds, these were enumerated (Part 1.2) and the microbial build-up (Part 1.3) determined.

Part 2 was to determine the incidence of normal human microbial flora and the infectious bacterial pathogens found on the headrests and armrests of the beds in a clinic, subsequent to their identification to a genus level.

Part 3 involved assessing the efficacy of the disinfectant in use by the clinic at the time of the study. This was done by performing the growth inhibition and modified Kirby Bauer tests and by determining the phenol coefficient.

Part 4 was to determine the degree of the transmission of normal human microbial flora and infectious bacterial pathogens from the patients to the beds in a clinic.

3.2 SAMPLING PROTOCOL

The Clinic Director granted permission for the use of the clinic`s facilities and to obtain swabs from the beds in the clinic in order to conduct the study (see Annexure D page 141).

3.2.1 Sample groups

There was one sample group that comprised of the beds in the clinic.

The beds were divided into three categories based on their frequency of use:

- High: Refers to beds on which 4 or more patients have been treated.
- Intermediate: Refers to beds on which 2 – 3 patients have been treated.
- Low to none: Refers to beds on which 0 – 1 patient has been treated.

The patients were divided into the two following groups:

- New: Refers to those patients who have not received prior treatment. at the clinic or have not received treatment for at least 6 months.
- Follow up: Refers to those patients who have had prior treatment at the clinic.

In Part 1.1, three beds, which included a bed from each of the three categories, were randomly selected for the collection of samples.

In Part 1.2, ten beds, which included beds from all three categories (four high, four intermediate and two low to none), were randomly selected for the collection of samples.

In Part 1.3 and 2, a total of four beds, which consisted of two beds, each from the high and intermediate categories, were randomly selected for the collection of samples.

Part 3 did not involve any sample groups as the processing and analysis had been performed in the microbiology laboratory.

In Part 4, a total of four beds, which consisted of two beds, each upon which new and follow up patients were examined and treated, were randomly selected for the collection of samples.

3.2.2 Sample group characteristics

Inclusion criteria:

- All beds had to be covered in vinyl.
- Beds were sampled depending on the amount of patients who were to be treated on that bed on the day the samples were to be obtained, in other words, depending on its category based on frequency of use. When there were more beds available than needed to be sampled, the name was written on a piece of paper and the chosen bed(s) were drawn out of a hat.

Exclusion criteria:

- Beds that had less or more patients to be treated on it on the day the samples were to be obtained, resulting in it not being included in any of the categories, were excluded from sampling.

3.2.3 Specimen collection times

Specimen collection times were specifically adapted to each objective:

To facilitate the verification of the presence of the micro-organisms located on the headrests and armrests of the beds for Part 1.1, three beds in the clinic (which included one bed from each of the three categories) were swabbed at 7:30 am, after cleaning, and at 16:30 pm, before cleaning, for one day during the first week of data collection.

To facilitate the verification of the presence and the enumeration of the micro-organisms located on the headrests and armrests of the beds for Part 1.2, ten beds in the clinic, which included beds from each of the three categories (four high, four intermediate and two low to none), were swabbed at 7:30 am, after cleaning, and at 16:30 pm, before cleaning, for two consecutive days during the second week of data collection.

To determine the microbial build-up of the micro-organisms found on the headrests and armrests of the beds, a total of four beds in the clinic, which included two beds each from the high and intermediate categories, were swabbed four times at 7:30 am, 10:30 am, 13:30 pm and 16:30 pm for one day during the third week of data collection.

To determine the degree of the transmission of normal human microbial flora and infectious bacterial pathogens from the patients to the headrests and armrests of the beds, a total of four beds, consisting of two beds each upon which new and follow patients were examined and treated, were cleaned with ethanol then swabbed before and after the treatment of the patients during the fourth week of data collection.

3.2.4 Sampling procedure

The selected beds were swabbed according to the procedures described by researchers (Maier, Pepper and Gerba, 2000; Bifero, Prakash and Bergin, 2006; Estridge and Reynolds, 2007; Evans *et al*., 2007).

The sterile polyester swab moistened with a phosphate buffer solution was rubbed over a 6 centimeters by 6 centimeters area demarcated by a flexible template, placed on the appropriate parts of the head- and armrests as these areas were most likely to harbour the highest average of colony forming units (Bifero, Prakash and Bergin, 2006). The template was placed 10 centimeters from the most caudal part of the headpiece and 2 centimeters from the most cephalad part of the armrests in the midline, so that the swab was taken in exactly the same area every time.

Two samples were obtained from the headrest. The first was either the left or right part upon which the patient's cheeks rested. This was determined by drawing the piece of paper with the respective name on it from a hat. The second was the middle part upon which the patient breathed. Two samples were also taken from the hand rests, one each for the right and left hand. A total of four samples were thus taken at each specified time from each of the beds.

Each swab, containing the specimen, was then placed into its individual test tube containing 3 milliliters of tryptic soy broth and shaken well to disperse all microbial flora and infectious bacterial pathogens evenly into the tryptic soy broth. Thereafter, they were transported to the microbiology laboratory where it was either used immediately or stored in the refrigerator at 4°C for a maximum period of three days (Forbes, Sahm, Weissfeld, 2007).

3.2.5 Data collection sheets

All data obtained at the clinic and in the microbiology laboratory were recorded on data collection sheets as follows:

- Upon the collection of samples the date, time, room code, area from where the sample was obtained, and for which part of the study the sample was obtained, was recorded on data collection sheet 1 (see Annexure A page 137).
- During the enumeration of samples for Part 1.2, 1.3 and 4 the rest of the data was recorded on data collection sheet 1 (see Annexure A page 137).
- In Part 2 of the study, the samples of Part 1.3 were utilised and the individual bacteria enumerated as for Part 1.2. For each individual colony, a new data collection sheet 1 (see Annexure A page 137) was used and when these were identified, data collection sheet 2 (see Annexure B page 138) was used.
- Data obtained from testing the efficacy of the disinfectant was recorded on data collection sheet 3 (see Annexure C page 139).

In order to correlate the clinic rooms, data collection sheets and samples obtained, each room was randomly assigned a code consisting of a capital letter of the alphabet. This code did not in any way correspond to the actual clinic room numbers. Each sample also had a label that consisted of the code for the clinic room, the time the sample was obtained, the area from which the sample was obtained and the date the sample was obtained. Both these codes and labels were recorded on the data collection sheets. In Part 2 of the study, an extra number in brackets was added to the labels to identify the individual bacterial colonies isolated from the samples in Part 1.3.

3.3 MICROBIOLOGICAL ANALYSIS

The Head of Department of Biotechnology and Food Technology granted permission for the use of the department`s facilities where the microbiological analyses were performed (see Annexure E page 142).

3.3.1 Verification of presence and enumeration of bacteria

Specimen collection

As previously discussed under the heading “Sampling procedure” page 63.

Serial dilution

For each specimen, six test tubes containing 9 milliliters of saline were labeled 10^{-1} to 10^{-6}, along with the code and the date. Using a pipette, 1 milliliter of the 3 milliliters of tryptic soy broth containing the specimen, was aseptically taken up and dispensed into the test tube labeled 10^{-1}. The used pipette tip was then discarded into disinfectant and replaced with a new tip. The contents of the test tube labeled 10^{-1} were mixed well and 1 milliliter taken up with the pipette and dispensed into the test tube labeled 10^{-2}, containing 9 milliliters saline. The used pipette tip was then discarded into disinfectant and replaced with a new tip. The procedure was then repeated until the last test tube contained a dilution of 10^{-6}.

Spread plate technique

For each of the 6 serial dilutions, twelve tryptic soy agar plates that have been made in advance, were labeled in duplicate 10^{-1} to 10^{-6}, along with the code and the date. A volume of 0,1 milliliters of the serial dilution 10^{-1} (called the inoculum) was dispensed onto each of the two tryptic soy agar plates labeled 10^{-1}, using the same pipette tip, and spread across the entire surface using a sterile swab. The plates were then rotated 60º, 90º and 120º and the inoculum spread across the entire surface, as described above. Then the swab was run round the edges of the plates twice to ensure that no sections were left untouched by the inoculum. The procedure was then repeated until 0,1 milliliters of each serially diluted sample was dispensed onto their two respective agar plates. A new pipette tip was used in between each of the different serial dilutions. The agar plates were then incubated at 37ºC for 24 to 48 hours in an inverted (agar side up) position.

Verification of presence of bacteria

Any evidence of the growth of bacterial colonies on the tryptic soy agar served as verification of the presence of bacteria on the arm- and headrests of the beds.

Enumeration and calculation of viable count of bacteria present

The average or mean quantity of colony forming units (cfu) enumerated from the two tryptic soy agar plates for each of the 6 dilutions was determined. For maximum reliability, only plate counts with between 30 and 300 colony forming units were used. Plates with colony forming units below or above that were excluded and subsequently, that dilution was eliminated since a reliable average could not be calculated. The optimum counting dilution was, thus, the highest dilution with an average between 30 and 300 cfu.

The following calculation based on research by Penn (1991), Pollack *et al*. (2002), Reasoner (2003) and Harley (2008) was used:

Viable count (cfu/ ml) = (average or mean number of cfu) ÷ (volume in ml × optimum counting dilution).

3.3.2 Microbial build-up of the bacteria

The specimen collection times, serial dilution, spread plate technique and the enumeration and calculation of viable count of bacteria present are as discussed on pages 62 to 66.

3.3.3 Incidence of the different types of bacteria

The specimen collection times, serial dilution, spread plate technique and the enumeration and calculation of viable count of bacteria present are as discussed on pages 62 to 66.

Isolation of bacteria

The petri dish containing the tryptic soy agar was placed upside down (agar side up with lid down) on table. The loop was flamed to redness, allowed to cool and a sample of each of the already enumerated bacterial colonies was taken. Then the bottom of the plate was picked up in the palm of the hand and held so that light from above shined off the surface of the agar. The loop was placed on the edge of the plate at a 30° to 45º angle and drawn across the surface of the agar in a zig-zag pattern, in such a way as to avoid overlapping the previous streak. Approximately one-fourth of the plate was covered with 10 to 20 streak marks and the previous streaks were not overlapped. After flaming the loop and allowing it to cool, it was drawn diagonally across the first group of streak marks once. Then the second one-fourth to one-third of the agar plate was covered using the same technique as described above. The loop was flamed again and the process repeated until 3 to 4 sections of the plate were covered with streak marks. The bottom of the plate was replaced back in the cover and the loop flamed. All the petri dishes were then incubated at 37ºC for 24 hours (Penn, 1991; Pollack *et al*., 2002).

Identification of bacteria

Discrete isolated colonies attained from the streaking procedure were incubated then used as specimens during the process of identification using colonial morphology.

The isolated colonies were observed, noting the following macroscopic characteristics as described by Bhatia and Ichpujani (2004), Baveja (2005) and Forbes, Sahm and Weissdeld (2007):

- Size measured in millimeters.
- Colour → colourless, white, yellow, black, pink.
- Shape → circular, filamentous, irregular, rhizoid, curled or punctiform.
- Elevation → flat, raised, low convex, convex, umbonate, papillate or pulvunate margin → eroded, lobate, curled, entire, undulate or filamentous.
- Surface appearance → concentric rings, smooth, radiating ridges or matt.
- Consistency → butyrous, brittle.
- Odour eg. *Proteus* smells like burnt chocolate and *Pseudomonas* like grape juice.
- Adherence to medium.
- Changes in medium resulting from bacterial growth.

The Gram stain was performed as described by Pollack *et al*. (2002) and Forbes, Sahm and Weissdeld (2007): The slide was placed on the staining tray, covered with crystal violet for approximately 1 minute, and then rinsed by gently pouring water over it. The smear was then covered with gram`s iodine, rotated and tilted to allow the iodine to drain, and then re-covered with iodine for another minute. The slide was rinsed with water and several drops of alcohol were placed evenly over the smears. The slides were rotated and tilted until most of the excess stain was removed and the alcohol running from the slide appeared clear. Then the slides were immediately rinsed with water to stop the decolourisation process. The slide was flooded with the counterstain, safranin, for 30 seconds then rinsed and shaken to get excess water off and allowed to air dry; after which it was viewed under a Nikon light microscope at 100× magnification and the microscopic characteristics noted.

The bacteria were then identified according to their macroscopic and microscopic characteristics.

3.3.4 Efficacy of Disinfectant

Growth inhibition

Staphylococci, Micrococci, Bacilli and Serratia all obtained from the plates of Part 2 were used to ensure the test was most relevant to the clinic in question. A sample of the disinfectant in its use-dilution was obtained from the spray bottles used to clean the clinic. A quantity of 5 milliliters of the disinfectant was placed into four sterile tubes each and 0,05 milliliters of each bacteria added. After labeling each test tube with the name of the disinfectant and the name of the bacteria that it contains, the contents of the tubes were mixed in order to obtain a homogenous suspension. At intervals of 1, 2, 5, 10 and 15 minutes; 0,1 milliliters of the contents of each test tube was transferred to individual tubes of tryptic soy broth. Four tubes of tryptic soy broth were also inoculated with 0,1 milliliters of each bacteria and these tubes were labeled as the controls. After incubation for 48 hours at 35ºC each test tube was shaken and observed for growth (Harley, 2008). The findings were recorded on the data collection sheet.

Phenol coefficient (PC)

Phenol was diluted with sterile distilled water 1/80, 1/90 and 1/100 and the disinfectant to 1/400, 1/450 and 1/500 so that the final volume in each tube was 5 milliliters. Tryptic soy broths were labeled with the name and dilution of the disinfectant and the time interval of the subculture. Each dilution was tested after 5, 10 and 15 minute incubations. One test tube of each of the phenol and disinfectant dilutions was placed in order in the test tube rack, for each time interval. A quantity of 0,5 milliliters of Staphylococcus was added to each tube and the time noted. The contents of each tube was mixed in order to obtain a homogenous suspension and to allow the disinfectant to come into contact with the bacteria. At intervals of 5, 10 and 15 minutes, a loopful from each tube was transferred into the appropriately labeled tryptic soy broth tubes, using the aseptic technique. After incubation for 48 hours at 35°C, all tryptic soy broth cultures were shaken and observed for presence (+) or absence (-) of growth and the results recorded. The experiment was then repeated with the Micrococcus, Bacillus and Serratia. From the data, the phenol co-efficient was determined for the disinfectant (Harley, 2008).

For example: Assume the 1/20 dilution of phenol killed Staphylococci in 10 minutes and a 1/300 dilution of the disinfectant also killed Staphylococcus within 10 minutes.

- Phenol coefficient = (300 ÷ 20) or ((1 ÷ 20) ÷ (1 ÷ 300)) = 15

The disinfectant was, thus, 15 times more effective than phenol in killing Staphylococcus

Modified Kirby Bauer technique

The modified Kirby Bauer technique, using the disk diffusion method as described by Pollack *et al*. (2002) was used.

Agar plate preparation

Two tryptic soy agar plates each were labeled with the name of one of each of the four bacteria. Staphylococcus inoculum was removed, using a sterile cotton swab, and spread across the entire surface of the first tryptic soy agar plate with the swab making sure the entire surface of the agar was covered. The plate was rotated 60º to 90º and the previous step repeated. Then the plate was rotated again and the steps repeated until the plate had been completely covered three times with inoculum. The swab was run around the rim of the plate once or twice to ensure no sections were untouched by the inoculum, then discarded in a container of disinfectant. The procedure was then repeated to prepare the second plate with Staphylococcus. Thereafter, plates were prepared with Micrococci, Bacilli and Serratia inoculi using the same procedure as above.

Disinfectant preparation

Samples of the use dilution and the concentrate of the disinfectant were obtained from the cleaners of the clinic. (The use dilution is the dilution used for disinfection of the beds in the clinic). A quantity of 10 milliliters of each was transferred to individual agar plates. Then 5 milliliters of the concentrate were mixed with 5 milliliters sterile distilled water, in order to make a 50% dilution, which was then transferred to an agar plate.

Placement of disinfectant on prepared agar plates

The bottom of the eight tryptic soy agar plates was subdivided into three sections. The tip of the forceps was dipped into the alcohol solution and the alcohol was allowed to evaporate. Using the forceps, the filter paper disk was removed from the container, dipped halfway once into one of the dilutions of the disinfectant and tapped on the side of the container to remove excess solution. The saturated disk was placed in the center of one of the three sections on the plate and pressed down lightly with the forceps. The bottom of the plate was labeled to indicate which dilution of disinfectant was used on each disk. This was repeated with the other dilutions. Once completed, each plate had three disks of different solutions of the disinfectant diffusing into the growing bacteria. The plates were then placed in the incubation tray, upside down and incubated for 24 hours.

Measurement of the zone of inhibition

The diameter of all three zones of inhibition was measured in millimeters for each of the three dilutions, on all eight plates. The average zone of inhibition was then calculated for each of the three dilutions and for each of the bacteria.

3.3.5 Transmission of normal human microbial flora and infectious bacterial pathogens

The specimen collection times, serial dilution, spread plate technique and the enumeration and calculation of viable count of bacteria present are as discussed on pages 62 to 66.

3.4 STATISTICS

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. Paired non parametric Wilcoxon signed ranks tests were used to compare the number of viable micro-organisms at the start of the day to the number at the end of the day. The Friedman test (also a paired non parametric test used when there are more than two paired values) was used to compare viable micro-organisms between four time points during the day. A variable calculating the change in the number of micro-organisms on a daily basis was created by subtracting the quantity of micro-organisms at the beginning of the day from the quantity at the end of the day. If this figure was negative, this indicated that the quantity of micro-organisms had decreased during the day and if it was positive then the micro-organisms had increased. This variable was compared between factors such as different beds, different areas on the same bed and the different categories of beds using non parametric Kruskal-Wallis tests, since there were more than 2 independent categories to compare. When there were two categories to compare, the Mann-Whitney test was used. ANOVA testing, with Bonferroni post-hoc tests, was used to compare means between three groups where data were normally distributed.

CHAPTER 4 : RESULTS AND DISCUSSION

4.1 INTRODUCTION

In this study the aim was to assess the beds as reservoirs for normal microbial flora and infectious bacterial pathogens at a clinic. In this chapter the results are presented and discussed according to the five study objectives:

Objective 1: To determine the presence of and enumerate the micro-organisms from the incubated samples obtained from the beds in the clinic.

Objective 2: To determine the microbial build-up on the beds in the clinic at regular intervals over a 12 hour period.

Objective 3: To identify and determine the incidence of normal human microbial flora and the infectious bacterial pathogens found on the beds in the clinic.

Objective 4: To assess the efficacy of the disinfectant currently in use by the clinic.

Objective 5: To determine the degree of the transmission of normal human microbial flora and the infectious bacterial pathogens from the patients to the beds in the clinic, during treatment.

4.2 DATA

4.2.1 Primary data

The primary data was obtained from the microbiological analyses of the samples taken on the beds and recorded on: Data collection sheets 1, 2 and 3.

4.2.2 Secondary data

Secondary data was obtained from various sources of related literature, journal articles, textbooks and the internet.

4.3 ABBREVIATIONS PERTINENT TO THE CHAPTER

%: Percentage.

<: Refers to a figure less than the figure reported.

>: Refers to a figure more than the figure reported.

=: Implies equals to.

+, **++**, **+++**: Represents varying amounts of growth.

- : Represents absence of growth.

AR and **AL**: Refers to the right and left armrest.

Beds C, E, Y, K… Refers to the different beds from which the samples were obtained. Each clinic room and, thus, each bed was randomly assigned a code consisting of a capital letter of the alphabet.

df: Refers to the degrees of freedom, which measures the amount of information available in the data that can be used to estimate the standard deviation (Pagano and Gauvreau, 2002).

HAI: Healthcare associated infection.

HL and **HM** and **HR**: Refers to the left, right and middle portion of headrest.

Mann-Whitney U: The number of all the possible pairs of observations comprising one from each sample, for which the value in the first group precedes a value in the second group (when using Mann-Whitney test) (Campbell, Machin and Walters, 2007).

n: Refers to the sample size.

p: Refers to the *p*-value, which is the probability of getting the output observed (Bowers, 2002).

Wilcoxon W: Sum of the ranks in the smaller group (when using Mann-Whitney test) (Campbell, Machin and Walters, 2007).

Z: Indicates the distance and the direction of a point from the mean in terms of standard units. The deviations are standardised or placed on a common scale (Blair and Taylor, 2008).

4.4 STATISTICAL CONCEPTS AND CALCULATIONS EXPLAINED

1-sided/tailed and 2-sided/tailed *p*-value: The *p*-value is the probability of obtaining a result as observed when the null hypothesis is true and such results can occur by chance equally often in either direction. To allow for this, a two-sided or two-tailed *p*-value is calculated (Campbell, Machin and Walters, 2007).

Count data: The data is often counts per unit of time for example the number of asthma attacks a person has in one month (Campbell, Machin and Walters, 2007).

Gaussian curve: Means that the data is distributed according to a normal, bell-shaped curve on a graph.

Hypothesis testing: Hypotheses are constructed to describe the possible responses to the research questions and thus, the objectives. The null hypothesis is a statement that expresses no difference or change in the parameters of interest from previous knowledge, while the alternate hypothesis describes that there is a change or difference in the parameters of interest from previous knowledge. In order to accept or reject the hypotheses, a significance level is decided on before commencing the research study (Gardiner, 1997; Campbell, Machin and Walters, 2007; Blair and Taylor, 2008).

Inter-quartile range: Data is divided into 4 equal parts by the lower quartile, the median and the upper quartile. The inter-quartile range is the difference of the lower and upper quartiles and is not vulnerable to outliers, so whatever the distribution of data, 50% of the data will lie within the inter-quartile range.

Mean or average: It is calculated by dividing the sum of the values by the number of values. In contrast to the median, every sample value is used to calculate the mean and it can, thus, be influenced by extreme values called outliers. This may produce a mean which is not very representative of the general mass of data (Bowers, 2002).

Measured or numerical continuous data: In theory this data can take any value within a given range and are the ones used most commonly for stastistics. For simplicity, however, this data is dichotomized in medicine to make nominal data. Diastolic blood pressure, which is continuous, is converted into hypertension and normotension. This does lead to a loss of information but makes it easier to discuss a population by the proportion affected and to decide whether this person needs treatment (Campbell, Machin and Walters, 2007).

Median: It is the middle value when data has been arranged in ascending order of size where there are an odd number of values. In cases where there is an even number of values, the average of the two middle values is used (Bowers, 2002; Campbell, Machin and Walters, 2007).

Non-parametric significance tests: The data collected is recoded into ranks and these are used as the basis of the test procedure. They are based on less stringent assumptions about the distribution of the measured response and can assess data which are not continuous. Examples include the Kruskal Wallis, Wilcoxon signed ranks and Mann-Whitney tests.

Nominal data: Data that one can name and put into unordered categories, for example male or female and blood group O, A, B or AB (Bowers, 2002 Pagano and Gauvreau, 2002).

Ordinal data: If there are more than two categories and the order of the categories become income important, it may be possible to order them in some way; for example after treatment a patient can either be the same, improved or worse (Pagano and Gauvreau, 2002; Campbell, Machin and Walters, 2007). Ordinal values are like rungs of a ladder whose rungs are not necessarily equal distances apart. Anyone standing on the fourth rung is higher than someone on the third rung, who in turn is higher than somebody on the second rung, but the actual differences between rungs 2 and 3 and between 3 and 4, are not necessarily the same. In other words, the person standing on rung 4 is not necessarily twice as high as the person standing on rung 2 (Bowers, 2002).

Parametric significance tests: These tests use collected data, usually continuous data, as they have been collected. They are based on the underlying assumption that the measured response, or mean of responses, conform to the normal (Gaussian) distribution. Examples include the ANOVA test.

Percentiles: A percentile is a point on the scale of measurement below which a specified percentage of the observations or values are located. The median is, thus, also called the 50^{th} percentile (Blair and Taylor, 2008).

***p*-value**: This *p*-value is compared to the pre-determined significance level (0,05) and serves as a measure of the weight or strength of evidence for rejection of the null hypothesis, with large values implying no evidence and small values indicating strong evidence. If the *p* is less than or equal to 0,05 the data is considered highly significant and the null hypothesis is rejected. If the p-value is greater than 0,05; there is not enough evidence to reject the null hypothesis (Gardiner, 1997; Campbell, Machin and Walters, 2007).

Range: Is the distance between the minimum and the maximum data values but if outliers are present, it may give a distorted impression of the variability of the data since only two of the data points are included in making the estimate (Bowers, 2002; Campbell, Machin and Walters, 2007).

Rank: Ordinal or metric data can be ranked, which means in a set of values, the largest value is given the rank of 1, the next largest value is given the rank of 2, and so on. When metric data is ranked, the ranked data becomes ordinal. (You know that the rank of 1 is higher than 2 but you do not know by exactly how much) (Bowers, 2002).

Significance level of a statistical test: The significance level provides a measure of the probability of rejecting the null hypotheses when it should have been accepted and described the level at which the specified hypotheses should be statistically tested. The significance level of this research study was 0,05 or 5%, which means that there was only a 5% probability that the collected data could have provided the evidence for the rejection of the null hypotheses by chance alone (Gardiner, 1997).

Standard deviation: The range is too sensitive to outliers and the inter-quartile range is limited in that it does not use all the data, with the top and bottom quarter being lost. The standard deviation is the average distance of all data values from the mean value (Bowers, 2002; Campbell, Machin and Walters, 2007).

Type I error: This is a false positive and occurs when the null hypothesis is rejected when in fact it is true. The probability of committing a Type I error is the same as the significance level of the test (Gardiner, 1997; Bowers, 2002; Campbell, Machin and Walters, 2007).

Type II error: This is a false negative and occurs when the null hypothesis is accepted when in fact it is false. The probability of a correct decision, in other words to reject a false the null hypothesis, is termed power. Power increases as the level of significance increases and as the sample size increases (Gardiner, 1997; Campbell, Machin and Walters, 2007; Blair and Taylor, 2008).

Variable: Label given to something whose value can vary, as for example age (Bowers, 2002).

4.5 RESULTS

The quantities of the colony forming units (cfu) were calculated as follows:

- Viable count (cfu/ ml) = (average or mean number of cfu) ÷ (volume in milliliters × optimum counting dilution).

All the figures used in the tables are the viable count (cfu/ ml), so for discussion purposes it was converted into the number of colony forming units per $cm^{2,}$ by using the following calculation:

- Viable count (cfu/ ml) × 3 ml (original sample) ÷ 36 cm^2 (surface area swabbed)

4.5.1 Objective One: To determine the presence of and enumerate the micro-organisms in the samples obtained from the beds in the clinic, during the first two weeks

4.5.1.1 The presence of micro-organisms on the chiropractic adjustment beds

Microbial growth was evident on **89,4%** of the beds sampled during the first 2 weeks of data collection, as can be seen in Table 4.1. This is consistent with findings of Bifero, Prakash and Bergin (2006) and Evans *et al*. (2007), that confirmed the presence of micro-organisms on vinyl-covered beds in clinics and in addition, Evans *et al*. (2008) also confirmed the presence of allergens on cloth-covered beds. They concluded that the main source of these micro-organisms were the normal flora present on patient`s skin that was being deposited on the beds during treatment.

It was anticipated to have growth present on only approximately 50% of the beds, since samples were obtained after the cleaners had cleaned the clinic, before the commencement of any treatment for the day and then again at the end of the day. The assumption was made that micro-organisms would only be present after the treatment of the patients had commenced but these results only indicate the presence and do not quantify how many micro-organisms were present.

Table 4.1: Presence of growth in samples obtained during week one and two

	Frequency	Percentage
Growth absent	11	10.6
Growth present	93	89.4
Total	104	100

4.5.1.2 Comparison of the viable count of micro-organisms at the start and end of the day

Using data from week one and two, fifty-two individual samples representing unique combinations of the beds, weeks, days and sections, were analysed. The two time points compared were at 7:30 am and 16:30 pm. These were paired measurements so paired tests were used to compare the quantities of the micro-organisms. The data were highly skewed since there was a large number with low counts and few with extremely high counts, thus, the distribution did not follow a Gaussian curve and non-parametric tests had to be used. Similarly, the median and inter-quartile range was used to describe the distribution of the data, rather than mean and standard deviation.

There was a large increase in the viable count of micro-organisms during the day. Table 4.2 shows that the median viable count at the start of the day was 300 cfu/ ml (**25 cfu/ cm^2**). This increased to a median viable count of 8 577 500 cfu/ ml (**714 792 cfu/ cm^2**) at the end of the day. The increase was significant with ***p*=0,027** (Table 4.3a and 4.3b). The assumption is made that the viable count is related to the number of patients who have been treated on the bed, due to deposition of micro-organisms from the patients` skin to the beds and/ or in that the patients contribute to a favourable environment for the growth of the micro-organisms.

Bifero, Prakash and Bergin (2006) also collected samples towards the end of the day and found an average of 40 cfu, 24 cfu and 42 cfu on the headrests, thorax and armrests, respectively, on the 9 beds sampled. Unfortunately, no indication is given whether it is per cm^2 or of the entire surface swabbed, so direct comparisons cannot be made. In the two other studies, the microbes found were not quantified, as the researchers felt that the presence of any microbes indicated the need for disinfection protocols (Evans *et al.*, 2007; Evans *et al.*, 2008).

These results indicate that after disinfection and before the treatment of patients even began, 25 cfu/ cm^2 were present on the beds. This is 5 times higher than the proposed standard of 5 cfu/ cm^2 for the total aerobic colony count, which indicates that either the quality of the disinfection process or the disinfectant used is insufficient (Dancer, 2004). It is also apparent that the cleaning of the clinic only once a day is inadequate.

Table 4.2: The viable count at the start and at the end of the day

		Viable count at the start of the day	Viable count at the end of the day
n	Valid	52	52
	Missing	0	0
Median		300	8577500
Minimum		0	0
Maximum		3×10^9	3×10^9
Percentiles	25	300	300
	50	300	8577500
	75	25087.5	37×10^7

The increase in the viable count was significant with ***p* = 0,027** (Table 4.3a and b).

Table 4.3a: Comparison of the viable count at the end and at the start of the day

		n	Mean Rank	Sum of Ranks
Viable count at end of the day - Viable count at start of the day	Negative Ranks	15(a)	21.4	321
	Positive Ranks	30(b)	23.8	714
	Ties	7(c)		
	Total	52		

a) Viable count at end of day < Viable count at start of day

b) Viable count at end of day > Viable count at start of day

c) Viable count at end of day = Viable count at start of day

Table 4.3b: Wilcoxon signed ranks test

	Viable count at end of the day - Viable count at start of the day
Z	-2.218(a)
p value. (2-tailed)	0.027

a) based on the negative ranks

4.5.1.3 Comparison of the change in the viable count of the micro-organisms from the start to the end of the day between the different beds

Samples were obtained from ten individual beds and these were represented by unique alphabetic letters that were randomly allocated to each bed. Beds E and T were sampled during both week one and two and Bed C was sampled on both days of week two.

Table 4.4 shows the median change in the viable count of the micro-organisms per bed from the beginning of the day to end of the day. A positive median indicates an increase, while a negative median indicates a decrease. All the beds, except for M, T and Y, showed an increase in micro-organisms. These three beds were all in the “low to none” category, meaning that either none or 1 patient was treated on these beds during the day the samples were taken.

It can then be deduced that the viable count is related to the number of patients who have been treated on the bed, due to deposition of micro-organisms from the patients` skin to the beds and/ or in that the patients contribute to a favourable environment for the growth of the micro-organisms.

Table 4.4: The viable count from the start of the day to end of the day on the different beds

Bed	Median	Minimum	Maximum	*n*
Y	-11475.0000	-179700.00	890.00	4
T	-266501500.0000	-2648445000.00	2999999700.00	8
S	14475150.0000	.00	29999700.00	4
R	8507975.0000	300.00	285499700.00	4
M	-37.5000	-300.00	.00	4
L	1217500000.0000	-300.00	2879997000.00	4
K	3782.5000	-513735.00	5195.00	4
G	339997025.0000	-2570000000.00	374999700.00	4
E	1587.5000	-4350000.00	2590750000.00	8
C	177674850	-1374723500	2559999700.00	8
Total	1945.0000	-2648445000.00	2999999700.00	52

Figure 4.1 graphically shows these median changes in the viable counts. The highest increase in the quantity of micro-organisms was on Bed L, which was in the “intermediate” category. This could possibly be due to more new patients being treated on these beds, thereby resulting in longer contact times with the beds and in turn, allowing for more deposition. There would, thus, be fewer patients being treated on the bed for the day due to time constrains, making it fall into the “intermediate” category. Bed T had the largest decrease in the viable count.

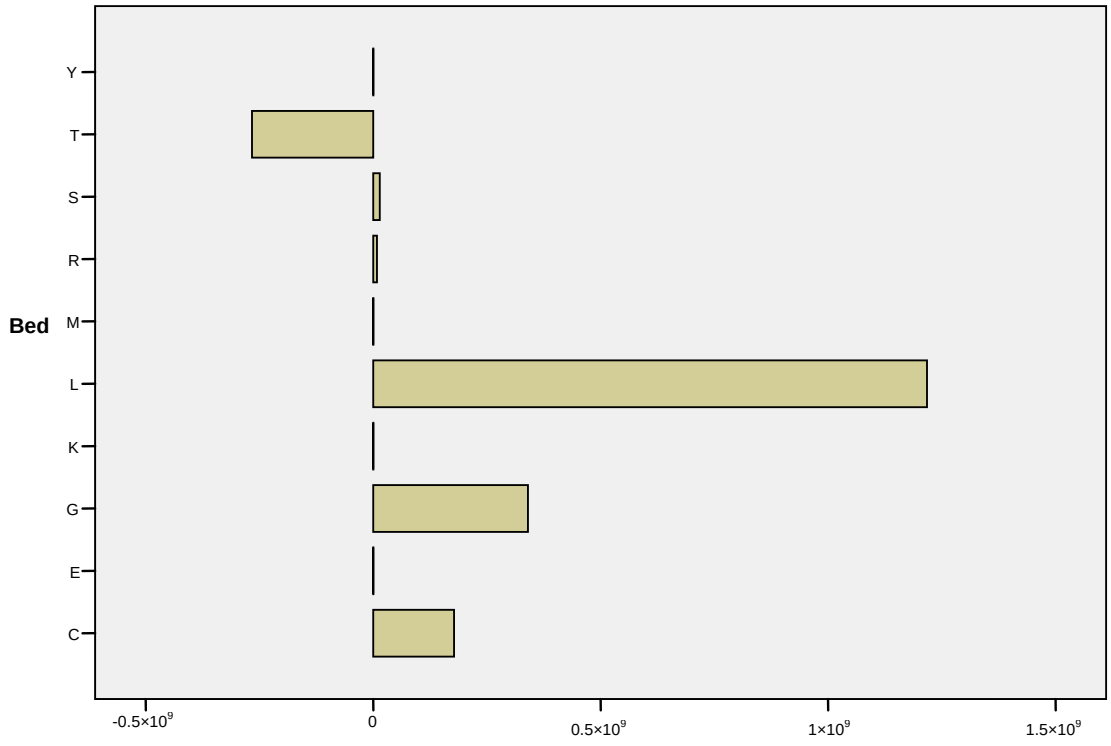


Figure 4.1: Median change in the viable count of micro-organisms on the different beds

The Kruskal-Wallis test was used to compare this change between the ten different beds. There was no significant difference between the beds (***p*=0.831**) (Table 4.5a and b). However, the sample sizes in the individual beds were low and could have accounted for the non-significant findings when the actual observed differences were high. Thus, this could be a Type II error.

Table 4.5a: Comparison of the changes in the viable count of micro-organisms between the beds

	Bed	*n*	Mean Rank
Change in viable count from the start to the end of the day	C	8	29.75
	E	8	26.75
	G	4	31.00
	K	4	23.75
	L	4	32.38
	M	4	16.63
	R	4	31.63
	S	4	28.38
	T	8	24.88
	Y	4	18.00
	Total	52	

Table 4.5b: Kruskal-Wallis test with the grouping variable being the beds

	Change in viable count from the start to the end of the day
Chi-Square	5.036
df	9
p value	0.831

4.5.1.4 Comparison of the change in the viable count of the micro-organisms from the start to the end of the day between the different sections of the beds

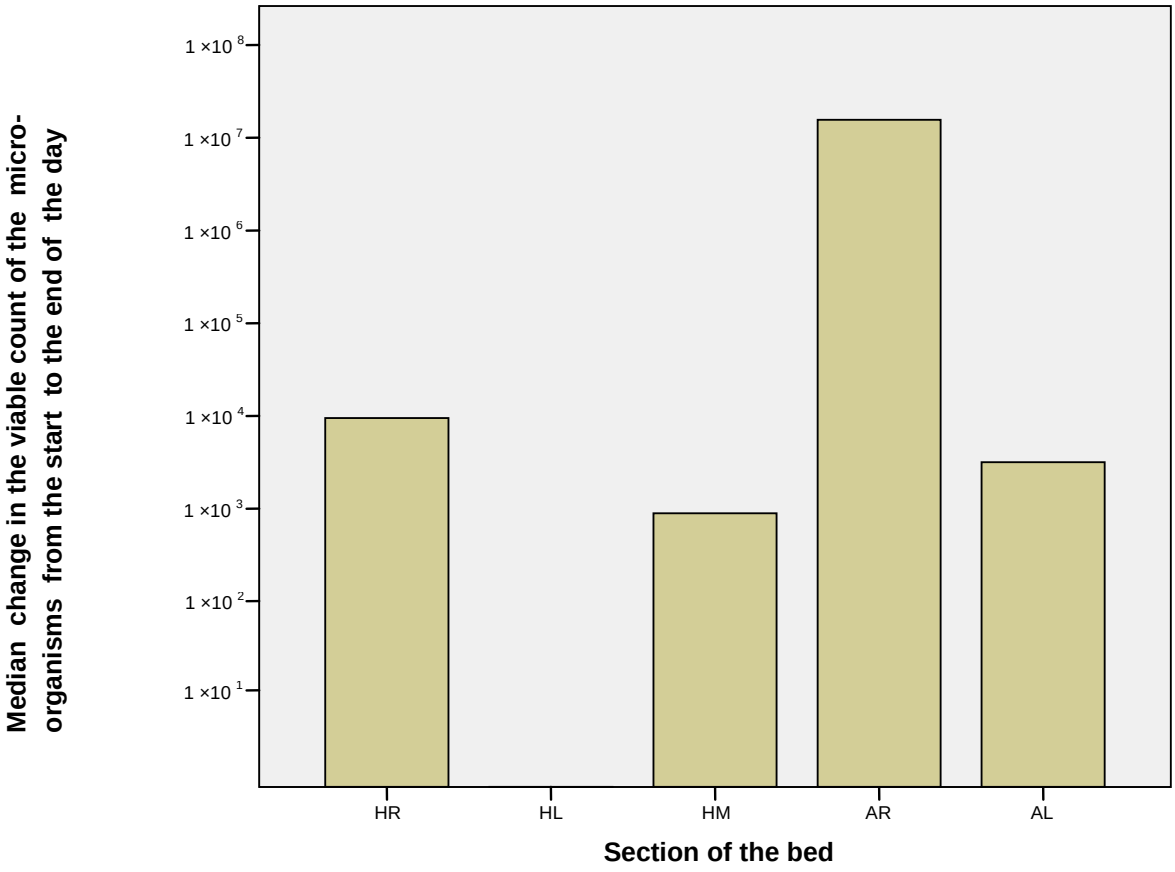
Table 4.6 shows that there was no change in the viable count of micro-organisms enumerated on average during the day on the left headrest sections of the chiropractic adjusting beds. No sections had a decrease in the viable count, with the right armrest sections having the highest increase; possibly related to the majority of the population being right-handed (Vuoksimaa, *et al*., 2008).

Table 4.6: Change in the viable count of the micro-organisms from start of the day to end of the day between the different sections of the beds

Section	Median	Minimum	Maximum	*n*
HR	9475.0000	.00	2999999700.00	6
HL	.0000	-2648445000.00	329994350.00	9
HM	890.0000	-4350000.00	429999700.00	11
AR	15594500.0000	-2500000000.00	2879997000.00	13
AL	3175.0000	-2570000000.00	2999999700.00	13
Total	1945.0000	-2648445000.00	2999999700.00	52

HR = right headrest, HL = left headrest, HM = middle of headrest, AR = right armrest and AL = left armrest

The changes in the viable count of the micro-organisms from the start to the end of the day is illustrated graphically in Figure 4.2 (NB the y axis is on a log scale in order to show the smaller changes, as well as the large changes).



HR = right headrest, HL = left headrest, HM = middle of headrest, AR = right armrest and AL = left armrest

Figure 4.2: Median change in the viable count of the micro-organisms by section

There was no significant difference between the sections in terms of a change in the viable count of the micro-organisms (***p* = 0.794**), as seen in Table 4.7a and b. This may be due to small sample sizes in the various sections, leading to a Type II error.

Table 4.7a: Comparison of the change in the viable count of the micro-organisms between the sections

	Section	*n*	Mean Rank
Change in the viable count of the micro-organisms from the start to the end of the day	HR	6	31.92
	HL	9	21.72
	HM	11	26.59
	AR	13	26.77
	AL	13	26.96
	Total	52	

HR = right headrest, HL = left headrest, HM = middle of headrest, AR = right armrest and AL = left armrest

Table 4.7b: Kruskal-Wallis test with the grouping variable being the sections of the beds

	Change in the viable count of the micro-organisms from the start to the end of the day
Chi-Square	1.682
df	4
p value	0.794

4.5.1.5 Comparison of the change in the viable count of the micro-organisms from the start to the end of the day between the different sections of the same beds

Since there was only one measurement of change in the viable count of the micro-organisms per section, per bed, per week, per day; it was not possible to compare statistically the differences between sections on the same bed. Therefore, Table 4.8 shows descriptively the actual changes in the viable counts. A lot of variability can be seen between the different sections of the same beds.

Table 4.8: Changes in the viable count of the micro-organisms per bed, per section, per week and per day (continued on the next page)

Bed	Section	Week	Day	Change in the viable count of the micro-organisms from the start to the end of the day
C	HL	2	1	.00
	HM	2	1	350000.00
	AR	2	1	-1374723500.00
	AL	2	1	-10500000.00
	HR	2	2	399999700.00
	HM	2	2	369999700.00
	AR	2	2	354999700.00
	AL	2	2	2559999700.00
E	HR	1	1	.00
	HM	1	1	-650.00
	AR	1	1	15594500.00
	AL	1	1	3175.00
	HL	2	2	.00
	HM	2	2	-4350000.00
	AR	2	2	1559999700.00
	AL	2	2	2590750000.00
G	HL	2	2	329994350.00
	HM	2	2	374999700.00
	AR	2	2	349999700.00
	AL	2	2	-2570000000.00
K	HR	2	2	3000.00
	HL	2	2	5195.00
	AR	2	2	-513735.00
	AL	2	2	4565.00
L	HL	2	1	.00
	HM	2	1	-300.00
	AR	2	1	2879997000.00
	AL	2	1	2435000000.00
M	HR	2	1	.00
	HM	2	1	-75.00
	AR	2	1	.00
	AL	2	1	-300.00
R	HR	2	1	15950.00
	HL	2	1	300.00
	AR	2	1	17000000.00
	AL	2	1	285499700.00
S	HL	2	1	28950000.00
	HM	2	1	.00
	AR	2	1	29999700.00
	AL	2	1	300.00

Table 4.8: Continued from the previous page

Bed	Section	Week	Day	Change in the viable count of the micro-organisms from the start to the end of the day
T	HR	1	1	2999999700.00
	HM	1	1	176997000.00
	AR	1	1	-710000000.00
	AL	1	1	2999999700.00
	HL	2	2	-2648445000.00
	HM	2	2	429999700.00
	AR	2	2	-2500000000.00
	AL	2	2	-2505000000.00
Y	HL	1	1	-179700.00
	HM	1	1	890.00
	AR	1	1	750.00
	AL	1	1	-23700.00

HR = right headrest, HL = left headrest, HM = middle of headrest, AR = right armrest and AL = left armrest

4.5.1.6 Comparison of the change in the viable count of the micro-organisms from the start to the end of the day between the different categories of beds

Table 4.9 shows that the beds in the “low to none” category did not show any change in the viable count on average during the day. The beds in the “intermediate” category showed the smallest increase and the beds in the “high” category showed the greatest increase. This is shown graphically in Figure 4.3 (NB the y axis is on a log scale in order to show the smaller changes, as well as the large changes).

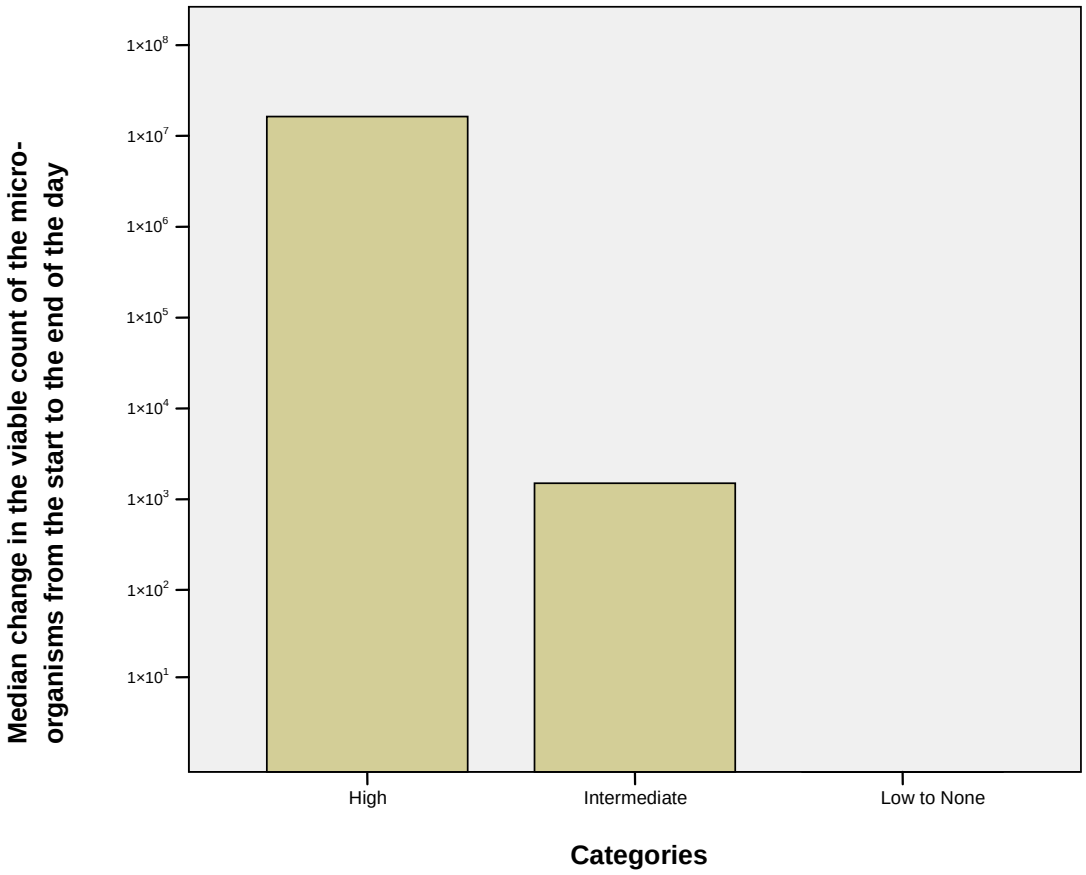
Table 4.9: Summary of the statistics of the changes in the viable count of the micro-organisms per category of bed

Category	Median	Minimum	Maximum	*n*
High	16297250.0000	-2648445000.00	2999999700.00	28
Intermediate	1500.0000	-1374723500.00	2879997000.00	12
None	.0000	-2570000000.00	374999700.00	12
Total	1945.0000	-2648445000.00	2999999700.00	52

Low to none category = 0 – 1 patient

Intermediate category = 2 – 3 patients

High category = 4 patients and more



L

ow to none category = 0 – 1 patient

Intermediate category = 2 – 3 patients

High category = 4 patients and more

Figure 4.3: Change in the viable count of the micro-organisms by category

The assumption was made that with an increase in the quantity of patients who were treated on the beds, the viable count would also increase, but Table 4.10a and b show that there was no significant difference between the categories in terms of the change in the viable count (***p* = 0.271**). This may be due to the small sample sizes in the various categories, leading to a Type II error.

Table 4.10a: Comparison of the change in the viable count of the micro-organisms between the categories

	Category	*n*	Mean Rank
Change in viable count of the micro-organisms from the start to the end of the day	High	28	29.57
	Intermediate	12	23.96
	Low to None	12	21.88
	Total	52	

Low to none category = 0 – 1 patient

Intermediate category = 2 – 3 patients

High category = 4 patients and more

Table 4.10b: Kruskal-Wallis test with the grouping variable being the categories of the beds

	Change in viable count of the micro-organisms from the start to the end of the day
Chi-Square	2.612
df	2
p value	0.271

4.5.1.7 Comparison of the change in the viable count of the micro-organisms from the start to the end of the day between the different beds in the same category

Table 4.11 shows that there was variability between the beds of the same categories. Bed T was in the "high" category, yet showed a median decrease. Bed L and G were in the "intermediate" and "low to none" categories, respectively, but showed large increases. This could be due to an alteration in the patients treated on the beds, due to cancellations or the making of new appointments. The beds were randomly selected the afternoon prior to the day the samples were to be obtained, so any changes were taken into consideration where possible. It is also possible that these clinic rooms were used by students for practice purposes that the researcher did not know about.

Table 4.11: Median change in the viable count of the micro-organisms between the different beds in the same category

				Change in the viable count of the micro-organisms from the start to the end of the day
				Median
Category	High	Bed	C	384999700.00
			E	1587.50
			R	8507975.00
			S	14475150.00
			T	-266501500.00
	Intermediate	Bed	C	-5250000.00
			K	3782.50
			L	1217500000.00
	Low to None	Bed	G	339997025.00
			M	-37.50
			Y	-11475.00

Low to none category = 0 – 1 patient

Intermediate category = 2 – 3 patients

High category = 4 patients and more

Figure 4.4 is a graphic depiction of the median change in the viable count of micro-organisms with the x-axis being on a log scale in order to show both the smaller and larger changes.

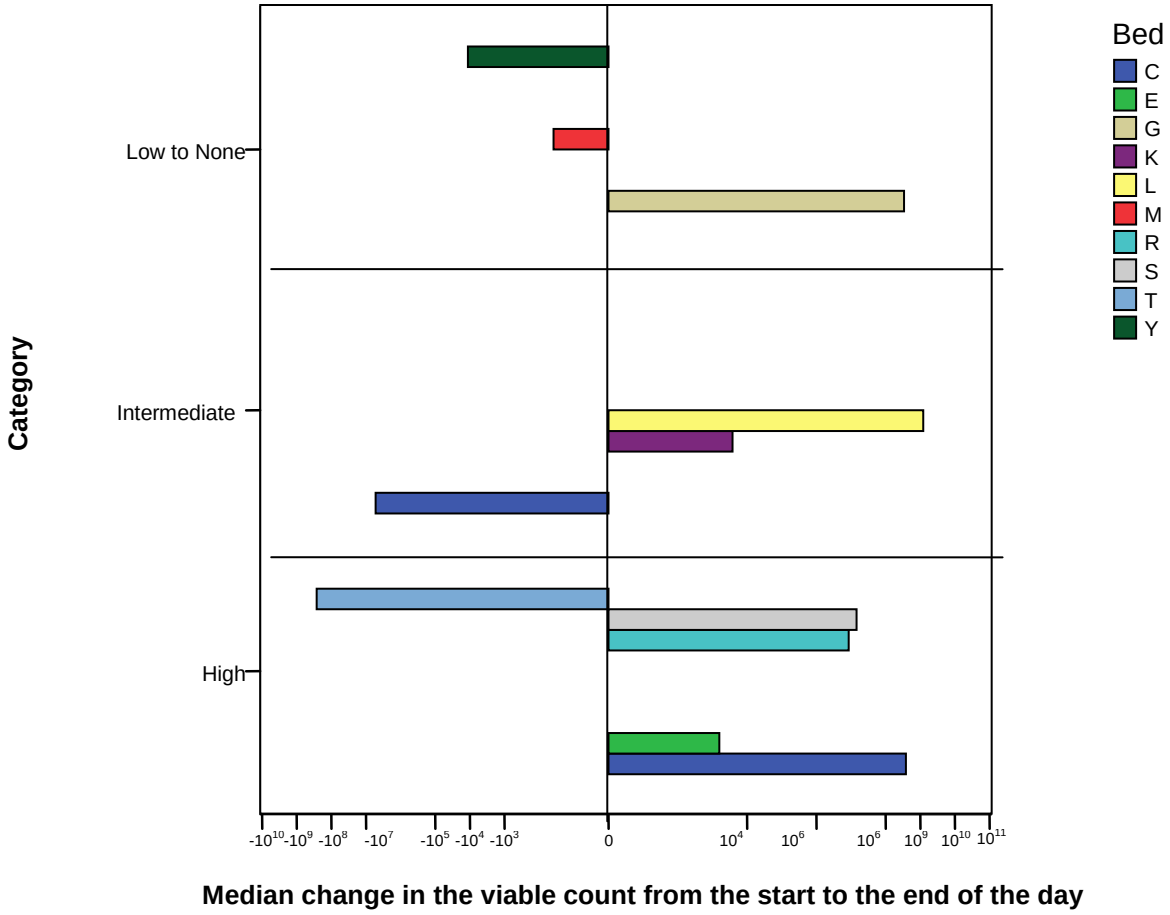


Figure 4.4: Median change in the viable count of the micro-organisms between the different beds in the same category

Tables 4.12a to 4.14b compare the median change in the viable count of the micro-organisms between the different beds in the respective categories. There are no significant differences between the beds of the respective categories (***p*=0.408**, ***p*=0.383** and ***p*=0.396**, respectively).

Table 4.12a: Comparison of the change in the viable count of the micro-organisms between the beds in the high category

	Bed	*n*	Mean Rank
Change in the viable count of the micro-organisms from the start to the end of the day	C	4	22.00
	E	8	12.88
	R	4	14.38
	S	4	12.88
	T	8	13.25
	Total	28	

High category = 4 patients and more

Table 4.12b: Kruskal-Wallis test with the grouping variable being the beds

	Change in the viable count of the micro-organisms from the start to the end of the day
Chi-Square	3.986
df	4
p value	0.408

Table 4.13a: Comparison of the change in the viable count of the micro-organisms between the beds in the intermediate category

	Bed	*n*	Mean Rank
Change in the viable count of the micro-organisms from the start to the end of the day	C	4	4.63
	K	4	6.75
	L	4	8.13
	Total	12	

Intermediate category = 2 – 3 patients

Table 4.13b: Kruskal-Wallis test with the grouping variable being the beds

	Change in the viable count of the micro-organisms from the start to the end of the day
Chi-Square	1.920
df	2
p value	0.383

Table 4.14a: Comparison of the change in the viable count of the micro-organisms between the beds in low to none category

	Bed	*n*	Mean Rank
Change in the viable count of the micro-organisms from the start to the end of the day	G	4	8.50
	M	4	5.50
	Y	4	5.50
	Total	12	

Low to none category = 0 – 1 patient

Table 4.14b: Kruskal-Wallis test with the grouping variable being the beds

	Change in the viable count of the micro-organisms from the start to the end of the day
Chi-Square	1.853
df	2
p value	0.396

4.5.1.8 Comparison of the change in the viable count of the micro-organisms from the start to the end of the day between the two days of week two

Table 4.15 shows that day one experienced on average a much smaller increase in the quantity of micro-organisms during the day than day two. However, when one considers the range of values in the distribution around this median, as shown in Figure 4.5, it is not surprising that this difference was not found to be statistically significant (***p*=0.463**) (Table 4.16a and b). Patients rarely receive treatment on two consecutive days. These results could indicate that the quantities of micro-organisms deposited on the beds vary according to the different patients who receive treatment each day. Patients could differ by being in more pain, suffering from hyperhydrosis, having a disability and not having access to proper sanitation, to mention but a few examples. Another possibility is the influence of the weather conditions on the day, in terms of humidity and temperatures influencing whether the air-conditioning systems or ceiling fans are used or not.

Table 4.15: Summary statistics of changes in the viable count of the micro-organisms per day

Day	Median	Minimum	Maximum	*n*
1	300.0000	-1374723500.00	2999999700.00	32
2	164999772.5000	-2648445000.00	2590750000.00	20
Total	1945.0000	-2648445000.00	2999999700.00	52

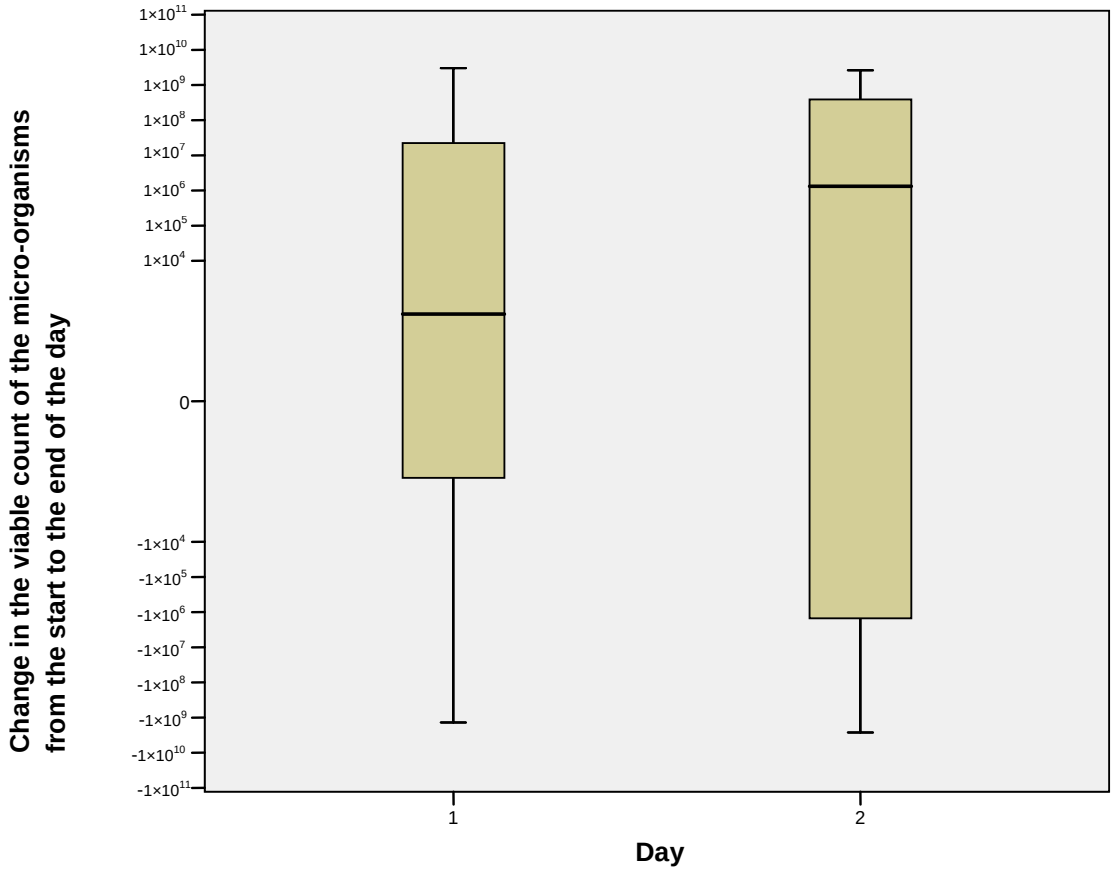


Figure 4.5: Box plot of the change in the viable count of the micro-organisms by day

Table 4.16a: Comparison of the change in the viable count of the micro-organisms between the two days

	Day	*n*	Mean Rank	Sum of Ranks
Change in the viable count of the micro-organisms from the start to the end of the day	1	32	25.28	809.00
	2	20	28.45	569.00
	Total	52		

Table 4.16b: Mann-Whitney test with the grouping variable being the different days

	Change in the viable count from the start to the end of the day
Mann-Whitney U	281.000
Wilcoxon W	809.000
Z	-.734
p value (2-tailed)	0.463

4.5.2 Objective Two:

To determine the microbial build-up on the chiropractic adjustment beds in the clinic at regular intervals over a twelve hour period.

4.5.2.1 Comparison of the microbial build-up measured at four time points during the day.

Using data from the third week, sixteen individual samples representing unique combinations of the beds and sections were analysed. The four time points compared were at 7:30 am, 10:30 am, 13:30 pm and 17:30 pm. These were paired measurements from the same bed and section, thus, paired tests were used to compare the viable micro-organisms. The data were highly skewed since there were a large number with low counts and few with extremely high counts, thus, the distribution did not follow a Gaussian curve and non parametric tests had to be used. Similarly, the median and inter quartile range was used to describe the distribution of the data rather than mean and standard deviation.

Table 4.17 shows that the median viable count at the start of the day was 3000 cfu/ ml (**346 cfu/ cm^2**), at 10:30 am the median was still 3000 cfu/ ml (**346 cfu/ cm^2**), and by 13:30 pm this had increased to 1 947 500 cfu/ ml (**162 291 cfu/ cm^2**), and at the end of the day it had increased even further to 3 billion cfu/ ml (**250 million cfu/ cm^2**). There was, thus, a large increase in the quantity of the micro-organisms observed throughout the day, especially in the afternoon, seen graphically in Figure 4.6 (note that the y-axis is on a log scale). A possible explanation is that when micro-organisms are deposited onto a new surface they are usually depleted of various essential constituents and time is needed for re-synthesis (Madigan, Martinko and Parker, 2000). Conditions could also have been unfavourable until the afternoon, allowing for optimal growth conditions and, thus, exponential growth.

Table 4.17: The viable count of the micro-organisms at four time points during the day in week three

		7:30 am	10:30 am	13:30 pm	17:30 pm
n	Valid	16	16	16	16
	Missing	0	0	0	0
Median		3000	3000	1947500	3000000000
Minimum		0	3000	3450	8000000
Maximum		300000	3000000000	3000000000	3000000000
Percentiles	25	3000	3000	313750	119250000
	50	3000	3000	1947500	3000000000
	75	3000	3000	29662500	3000000000

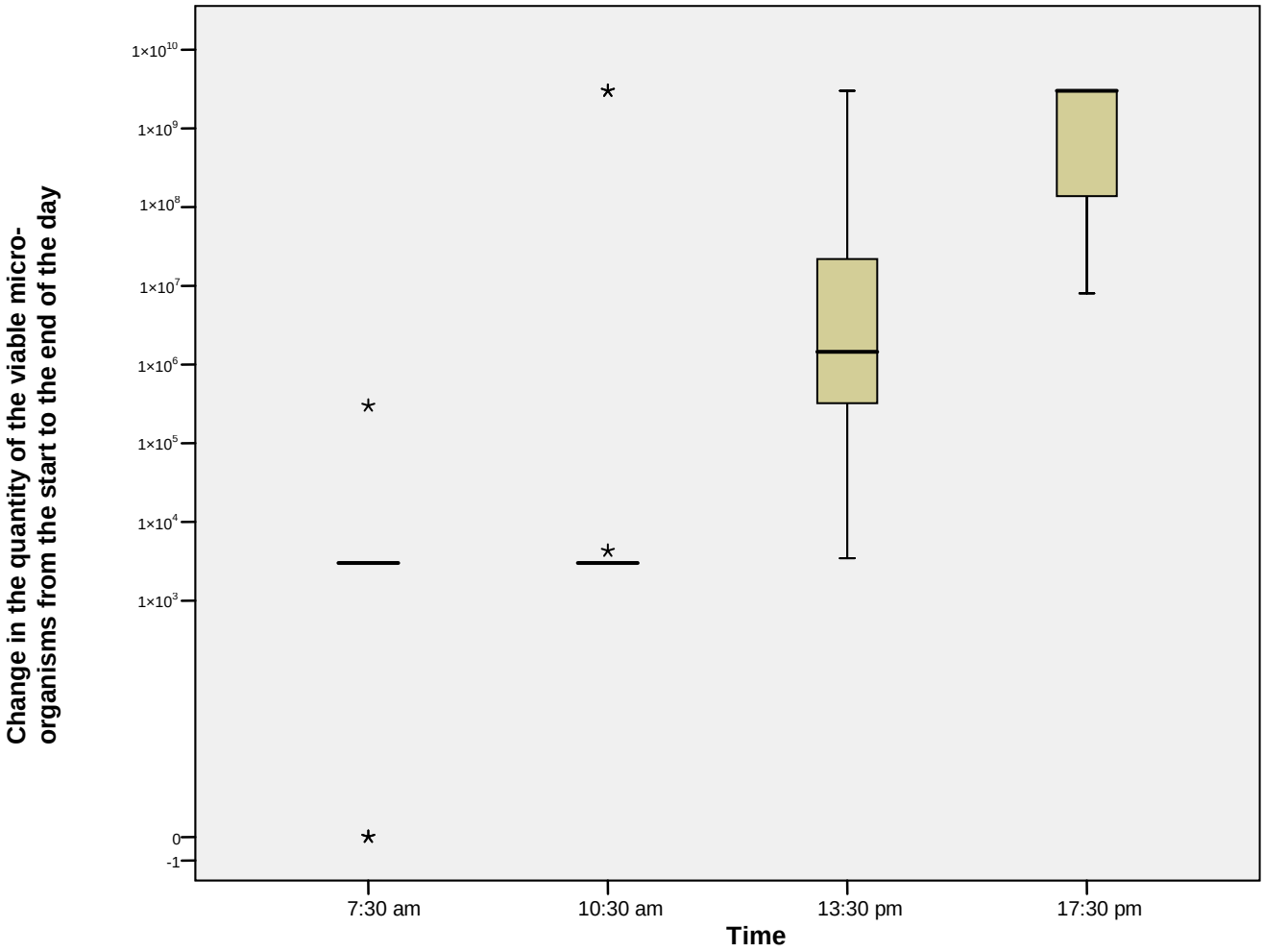


Figure 4.6: Box plot of the viable count at the four time points during the day

There was a highly statistically significant increase in the number of micro-organisms throughout the day (***p* < 0.001**), as can be seen in Table 4.18a and b.

Table 4.18a: Comparison of the viable count of micro-organisms at the four time points throughout the day

	Mean Rank
Viable count at 7:30 am	1.38
Viable count at 10:30 am	1.88
Viable count at 13:30 pm	3.00
Viable count at 17:30 pm	3.75

Table 4.18b: Friedman test

n	16
Chi-Square	36.000
df	3
p value	<0.001

4.5.3 Objective Three: To identify and determine the incidence of normal human microbial flora and the infectious bacterial pathogens found on the beds in the clinic

4.5.3.1 To determine the identity of the micro-organisms overall

The identity of the four micro-organisms was determined by using data obtained from 14 of the microbial build-up agar plates from week three.

The macroscopic colonial morphology of the first type of colony can be seen in Figure 4.7 as being small, white and round colonies. The microscopic characteristics, elicited by the simple and Gram stains, showed that the bacteria were Gram-positive cocci arranged in clusters. This indicated that the bacteria were most likely from the genus Staphylococci, which can be divided into coagulase-positive *Staphylococcus aureus* and coagulase negative species. There is persistent nasal carriage of *Staphylococcus aureus* in 20- 40% of adults and also in the perineal area of 20% of healthy people. The majority of the normal skin flora is made up of coagulase negative cocci, of which S*taphylococcis epidermidis* constitutes more than 50% (Oumeish, Oumeish and Bataineh, 2000).

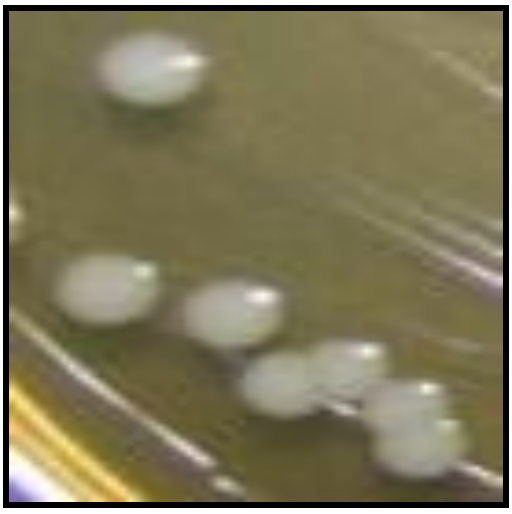


Figure 4.7: Staphylococci

The second colony was variable ranging from pink to red in colour, as illustrated in Figures 4.8 and 4.9. The simple and Gram stain indicated the bacteria as being Gram-negative rods. These bacteria were of the genus Serratia, which sometimes produces a red pigment, prodigiosin, in the presence of oxygen and at suitable temperatures. It is most probably *Serratia marcescens*, the most common of all the Serratia species (Koneman *et al*., 1997; Baveja, 2005; Greenwood *et al*., 2007). *Serratia marcescens* is widely distributed in nature and is found in soil, water and food products (Baveja, 2005; Greenwood *et al*., 2007).

Figure 4.8: Serratia

Figure 4.9: Serratia

Figure 4.10 depicts the third type of colony that is large, spreading, and grey-white with irregular margins. On simple and Gram stain it became evident that the bacteria are large Gram-positive bacilli. It is perhaps *Bacillus cereus*, a bacteria widely distributed in soil, vegetables, cereals and meat that were transferred to the beds from the skin of patients who had been contaminated after contact and/ or exposure to the abovementioned articles (Koneman *et al*., 1997; Baveja, 2005; Greenwood *et al*., 2007).

Figure 4.10 Bacilli

The fourth type of colony was yellow and extremely small (Figure 4.11). The bacteria were also Gram-positive rods, like the Staphylococci, but are thought to be Micrococci due to the colonies being smaller in size than those of the staphylococci after the same incubation period (Koneman, 1997). The only manner in which to distinguish accurately between Staphylococci and Micrococci is to perform the oxidation-fermentation test (Koneman et al., 1997; Madigan, Martinko and Parker, 2000). Micrococci form part of the normal skin flora but in small amounts (Cove and Eady, 1998; Larson *et al*., 2000).

Figure 4.11: Micrococci

The percentages of the colonies of each of the species are shown in Table 4.19. **Staphylococci** were the most prevalent organism at an average of **59%** of the colonies, followed by **Serratia** with **40%**. **Micrococci** and **Bacilli** were relatively **uncommon**.

Research by Bifero, Prakash and Bergin (2006) in vinyl-covered beds had similar results with coagulase negative staphylococci and *Staphylococcus aureus* present on 100% and 78% of the beds, respectively, and 22% methicillin-resistant *Staphylococcus aureus*. Micrococci were present on 33% and bacilli on 22% of the beds, with Serratia being even more uncommon (11%). Evans *et al*. (2007) did not quantify bacterial counts but determined that Gram-negative bacteria were present on 2 of the 10 vinyl-covered beds, while all of the beds contained at least some of the Gram-positive bacteria which included *Staphylococcus aureus, Staphylococcus epiderrmidis* and *Staphylococcus saprophyticus*. Further tests designated the *Staphylococcus aureus* as being methicillin-resistant. Results from a study on cloth-covered chiropractic adjustment tables found common mould spores and Gram-positive bacteria which consisted of *Micrococcus luteus, Propionibacterium acnes, Staphylococcus epidermidis, Staphylococcus aureus* and *Bacillus subtilis*.

The predominance of staphylococci is consistent with the literature in that this genus is the most frequently isolated micro-organisms in medical microbiology (Becker et al., 2007). The presence of staphylococci in such high quantities is of great concern since they are commonly implicated in HAI. Staphylococcus aureus has a broad pathogenic potential causing a wide range of infections such as pneumonia, meningitis, septicaemia, osteomyelitis, endocarditis and abscesses (Becker et al., 2007; Greenwood *et al*., 2007). Should the Staphylococci be *Staphylococcus epidermidis*, there would be less of a concern except in immunocompromised patients (Becker et al., 2007; Greenwood *et al*., 2007).

It has been demonstrated that some strains of Staphylococci can be lytic to Micrococci, explaining why it was uncommon (Larson, et al., 2000). They are rarely involved in HAI (Baveja, 2005).

The presence of the bacilli can be explained as being transient flora present on the skin at the time of deposition. It has been implicated in food poisoning but is not of great concern in such small quantities (Baveja, 2005; Greenwood *et al*., 2007).

The finding of 40% Serratia, is serious in that it can lead to serious healthcare associated infections including meningitis, septicaemia, endocarditis, urinary and respiratory tract infections (Baveja, 2005; Greenwood *et al*., 2007). It grows at ambient temperatures with minimal nutrients and can become established endemically in healthcare facilities (Greenwood *et al*., 2007).

Table 4.19: Percentages of each of the four micro-organisms

		Staphylococci percentage	Micrococci percentage	Bacilli percentage	Serratia percentage
n	Valid	14	13	13	14
	Missing	0	1	1	0
Median		58.5784	.0000	.0000	40.0327
Minimum		13.33	.00	.00	5.43
Maximum		90.94	7.14	3.62	86.11
Percentiles	25	49.1385	.0000	.0000	21.2339
	50	58.5784	.0000	.0000	40.0327
	75	78.7661	2.7652	.0000	50.2365

4.5.3.2 To determine the identity of the micro-organisms on the different beds

Table 4.20 shows that the 16 agar plates chosen from which the micro-organisms were identified, came from beds B, F, L, and S. Staphylococci were most predominant on all beds, but especially on bed F, while Serratia were highest on Bed L.

Table 4.20: Median percentages of the micro-organisms by bed

		Staphylococci percentage	Micrococci percentage	Bacilli percentage	Serratia percentage
Bed	B	63.52	.00	.00	36.48
	F	88.00	.00	.00	12.00
	L	53.01	3.18	.00	44.74
	S	58.33	.00	.00	41.18

Only Micrococci differed significantly by bed (***p*=0.025**). Bed L showed the highest median percentage of Micrococci. No other organism was significantly differently distributed between the beds (***p*=0.247**, ***p*=0.599** and ***p*=0.249**) (Table 4.21a and b).

Table 4.21a: Comparison of the median percentage of each type of organism between the four beds

	Bed	n	Mean Rank
Staphylococci percentage	F	5	10.40
	S	3	5.67
	L	4	5.25
	B	2	7.50
	Total	14	
Micrococci percentage	F	4	5.88
	S	3	4.50
	L	4	11.25
	B	2	4.50
	Total	13	
Bacilli percentage	F	4	7.75
	S	3	8.00
	L	4	6.00
	B	2	6.00
	Total	13	
Serratia percentage	F	5	4.60
	S	3	9.67
	L	4	9.50
	B	2	7.50
	Total	14	

Table 4.21b: Kruskal-Wallis test with the grouping variable being the different beds

	Staphylococci percentage	Micrococci percentage	Bacilli percentage	Serratia percentage
Chi-Square	4.136	9.305	1.875	4.122
df	3	3	3	3
p value	0.247	0.025	0.599	0.249

4.5.3.3 To determine the identity of the micro-organisms on different sections of the beds

The sample sizes within each section were too small to make valid comparisons, but Table 4.22 gives an idea of the percentages of the different micro-organisms found on each section. Staphylococci were found in high quantities in the nares and on the forehead, explaining the finding that the middle and left headrest sections had the highest percentages of Staphylococci. Micrococci and Serratia were highest on the right headrest sections.

Table 4.22: Percentages of each organism according to the different sections of the beds

Section		Staphylococci percentage	Micrococci percentage	Bacilli percentage	Serratia percentage
HR	Median	13.3333	3.3333	.0000	83.3333
	Minimum	13.33	3.33	.00	83.33
	Maximum	13.33	3.33	.00	83.33
	n	1	1	1	1
HL	Median	69.9614	.0000	.0000	30.0386
	Minimum	51.35	.00	.00	11.43
	Maximum	88.57	.00	.00	48.65
	n	2	2	2	2
HM	Median	69.9869	.0000	.0000	26.4417
	Minimum	58.33	.00	.00	12.00
	Maximum	88.00	7.14	2.78	38.89
	n	4	4	4	4
AR	Median	58.8235	1.5152	.0000	41.1765
	Minimum	54.55	.00	.00	27.78
	Maximum	72.22	3.03	.00	42.42
	n	3	2	2	3
AL	Median	46.9853	.7353	.0000	51.0294
	Minimum	13.89	.00	.00	5.43
	Maximum	90.94	2.50	3.62	86.11
	n	4	4	4	4

HR = right headrest, HL = left headrest, HM = middle of headrest, AR = right armrest and AL = left armrest

4.5.3.4 To determine the identity of the micro-organisms in the different categories of beds

Table 4.23 shows that the beds in the high category tended to have higher percentages of Staphylococci than those in the intermediate category, which was borderline non-significantly different (***p* = 0.064**). Micrococci and Bacilli were not significantly different between the categories of beds, and Serratia were almost significantly higher in the beds of the Intermediate category (***p* = 0.064**) (Table 4.24a and b).

Table 4.23: Percentages of each organism according to the different categories of the beds

Category		Staphylococci percentage	Micrococci percentage	Bacilli percentage	Serratia percentage
High	Median	75.6881	.0000	.0000	24.3119
	Minimum	42.50	.00	.00	5.43
	Maximum	90.94	2.50	3.62	55.00
	n	7	6	6	7
Intermediate	Median	54.5455	1.4706	.0000	42.4242
	Minimum	13.33	.00	.00	28.57
	Maximum	64.29	7.14	2.78	86.11
	n	7	7	7	7

Intermediate category = 2 – 3 patients

High category = 4 patients and more

Table 4.24a: Comparison of the percentages of each organism by category of bed

	Category	n	Mean Rank	Sum of Ranks
Staphylococci percentage	High	7	9.57	67.00
	Intermediate	7	5.43	38.00
	Total	14		
Micrococci percentage	High	6	5.42	32.50
	Intermediate	7	8.36	58.50
	Total	13		
Bacilli percentage	High	6	7.17	43.00
	Intermediate	7	6.86	48.00
	Total	13		
Serratia percentage	High	7	5.43	38.00
	Intermediate	7	9.57	67.00
	Total	14		

Table 4.24b: Mann-Whitney test with the grouping variable being the different categories

	Staphylococci percentage	Micrococci percentage	Bacilli percentage	Serratia percentage
Mann-Whitney U	10.000	11.500	20.000	10.000
Wilcoxon W	38.000	32.500	48.000	38.000
Z	-1.853	-1.547	-.227	-1.853
p value (2-tailed)	0.064	0.122	0.820	0.064

4.5.3.5 To determine the identity of the micro-organisms at the different times of the day

Only the plates from 13:30 pm and 16:30 pm were used, since very few micro-organisms were present on the plates from 7:30 am and 10:30 am. No plates were measured sequentially from the same section of the same bed at both time points, thus, the measurements were not considered as paired and the two time points were analysed as independent groups.

At 13:30 pm Staphylococci and Serratia were present on **54,5%** and **42%** of the beds, respectively, as can be seen in Table 4.25. By 16:30 pm, the Staphylococci had increased further to **75,6%**, while the Serratia had decreased to **24%**.

Table 4.25: The percentages of each organism according to the time of day

Time		Staphylococci percentage	Micrococci percentage	Bacilli percentage	Serratia percentage
13:30 pm	Median	54.5455	.0000	.0000	42.4242
	Minimum	13.33	.00	.00	5.43
	Maximum	90.94	7.14	3.62	86.11
	n	9	9	9	9
16:30 pm	Median	75.6881	.0000	.0000	24.3119
	Minimum	42.50	.00	.00	11.43
	Maximum	88.57	2.50	.00	55.00
	n	5	4	4	5

Table 4.26 shows that Staphylococci increased non-significantly over time (***p*=0.125**) and Serratia decreased non-significantly over time (***p*=0.125**). There was no change in the percentages of Micrococcus and Bacillus over time.

Table 4.26a: Comparison of the percentages of each organism by time of day

	Time	n	Mean Rank	Sum of Ranks
Staphylococci percentage	13:30 pm	9	6.22	56.00
	16:30 pm	5	9.80	49.00
	Total	14		
Micrococci percentage	13:30 pm	9	7.50	67.50
	16:30 pm	4	5.88	23.50
	Total	13		
Bacilli percentage	13:30 pm	9	7.44	67.00
	16:30 pm	4	6.00	24.00
	Total	13		
Serratia percentage	13:30 pm	9	8.78	79.00
	16:30 pm	5	5.20	26.00
	Total	14		

Table 4.26b: Mann-Whitney test with the grouping variable being the time

	Staphylococci percentage	Micrococci percentage	Bacilli percentage	Serratia percentage
Mann-Whitney U	11.000	13.500	14.000	11.000
Wilcoxon W	56.000	23.500	24.000	26.000
Z	-1.533	-.792	-.981	-1.533
p value (2-tailed)	0.125	0.429	0.326	0.125

4.5.4 Objective Four: To assess the efficacy of the disinfectant currently in use by the clinic

The disinfectant in use by the clinic, at the time the research was conducted, was *Taski Sani Des J-flex*. Its efficacy was tested by performing the growth inhibition, modified Kirby Bauer and phenol coefficient tests.

4.5.4.1 Testing the efficacy of the disinfectant using the growth inhibition test

This test was done in replicates for each organism but was done only once for each dilution, thus, statistical analysis was not possible. This test was done to determine the amount of time it took for the disinfectant to destroy all the micro-organisms. Table 4.27 shows that all micro-organisms were destroyed 5 minutes after exposure to the *Sani Des J-flex* disinfectant, which is consistent with and emphasizes the instructions from the manufacturers to leave the disinfectant on for 5 minutes before rinsing it off.

Table 4.27: The growth inhibition test

Organism	Control	1 minute	2 minutes	5 minutes	10 minutes	15 minutes
Staphylococcus	+++	++	++	-	-	-
Micrococcus	+++	-	-	-	-	-
Bacillus	+++	-	-	-	-	-
Serratia	+++	++	+	-	-	-

Absence of growth = "-"

Varying amounts of growth = "+", "++" or "+++"

In Figure 4.12 examples of the varying amounts of growth can be seen. The left and middle tube is murky, indicating growth of micro-organisms, while the tube on the right is clear, indicating no growth.

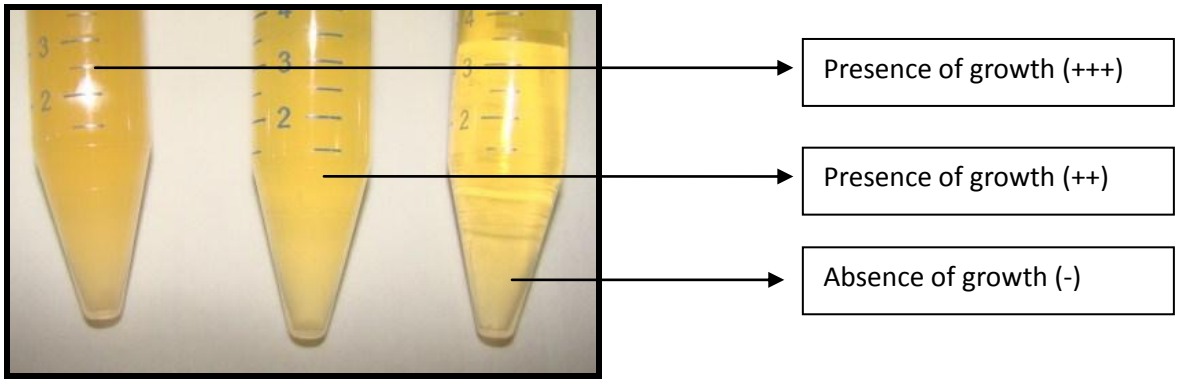


Figure 4.12: The varying amounts of growth in the growth inhibition test

4.5.4.2 Testing the efficacy of the disinfectant using the modified Kirby Bauer test

This test was done in replicates for each organism but was done only once for each dilution, thus, statistical analysis was not possible at the level of micro-organism and dilution. Therefore, statistical comparison was done on all micro-organisms combined at the three levels of dilution.

The Kirby Bauer test was designed for determining the resistance of micro-organisms to antibiotics but can also be used to test disinfectants. It is a cheap, quick and easy way to determine the efficacy of disinfectants with a very graphic portrayal (Jones *et al*., 2001; Pollack, 2002). Pre-standardised antimicrobial disks are used and results interpreted as follows: a large zone of inhibition indicates the micro-organisms are sensitive to the antibiotic and a small zone indicates it is resistant with an intermediate zone being in between the two.

Table 4.28 shows that for each organism there was an increase in the average size of the zone of inhibition as the dilution was decreased. In other words, the more concentrated the disinfectant solution, the more effective it was against the bacteria. The disinfectant was most effective in inhibiting the Gram-positive Staphylococci, Micrococci and Bacilli than the Gram-negative Serratia (Figures 4.13 and 4.14). The amount of disinfectant penetrating into the cells is limited by the extra lipopolysaccharide layer (Denyer and Stewart, 1998). The sizes of the zones of inhibition are comparable to that found in antibiotic testing (Jones *et al*., 2001).

Table 4.28: Average inhibition zone size by organism and dilution

Organism	Dilution	Average size of zone of inhibition in millimeters
Staphylococci	Use dilution	15.0
	50%	26.5
	100%	27.0
Micrococci	Use dilution	16.5
	50%	23.0
	100%	24.5
Bacilli	Use dilution	16.5
	50%	24.5
	100%	25.0
Serratia	Use dilution	6.0
	50%	14.5
	100%	15.0

Use dilution = dilution of the disinfectant that is used in the clinic to clean the adjustment beds

50% = a sample of the concentrate of the disinfectant is mixed with the same amount of sterile water

100% = the concentrate of the disinfectant is used as it is

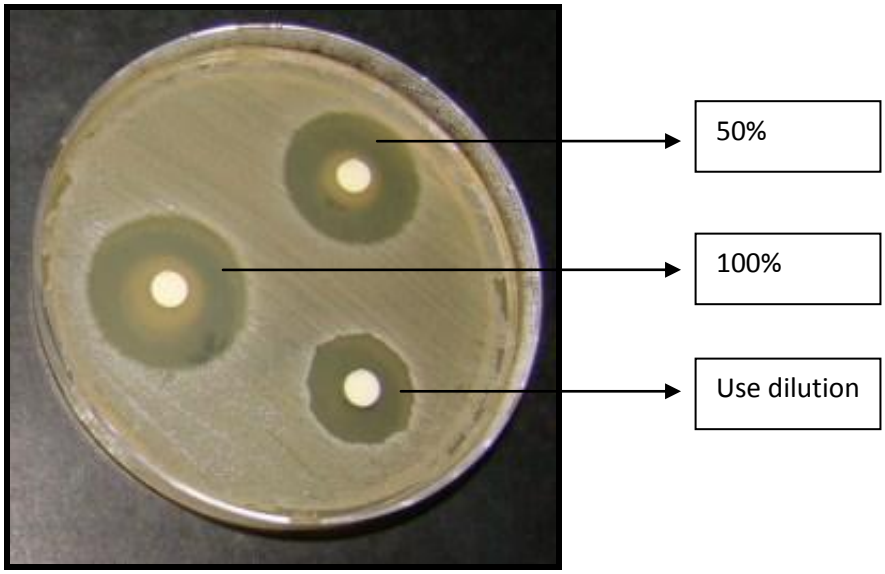


Figure 4.13: Modified Kirby Bauer test using Micrococci

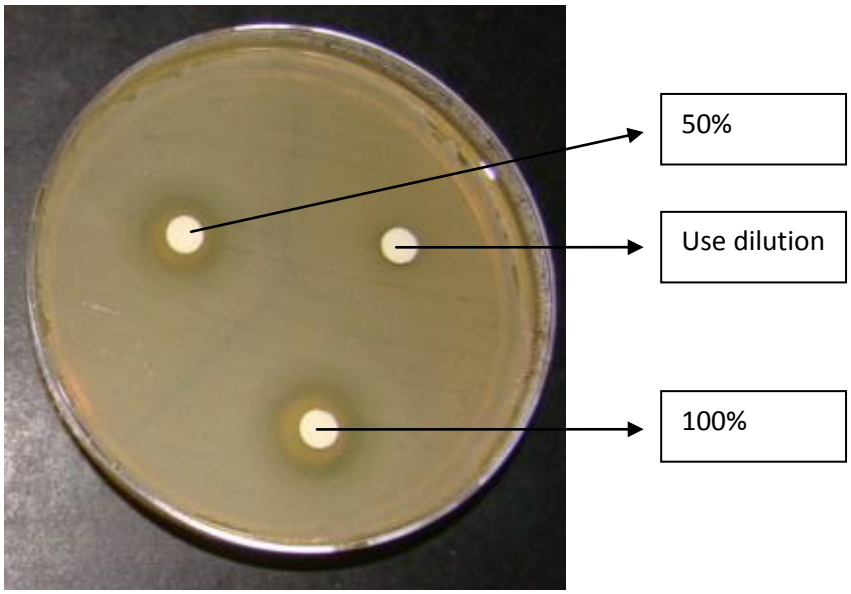


Figure 4.14: Modified Kirby Bauer test using Serratia

Figure 4.15 shows that for all micro-organisms combined, the mean inhibition zone size increased as dilution decreased, however, between the 50% and the 100% dilution the increase was minimal. The difference between the means of the three dilutions was not quite statistically significant (***p*=0.058**). Bonferroni post-hoc tests showed that the largest, although non-significant difference, was found between the use dilution and the 100% (***p*=0.096**), while there was a non-significant difference between the use dilution and the 50% (***p*=0.137**) and no difference between the 50% and 100% dilutions (***p* =1.00**).

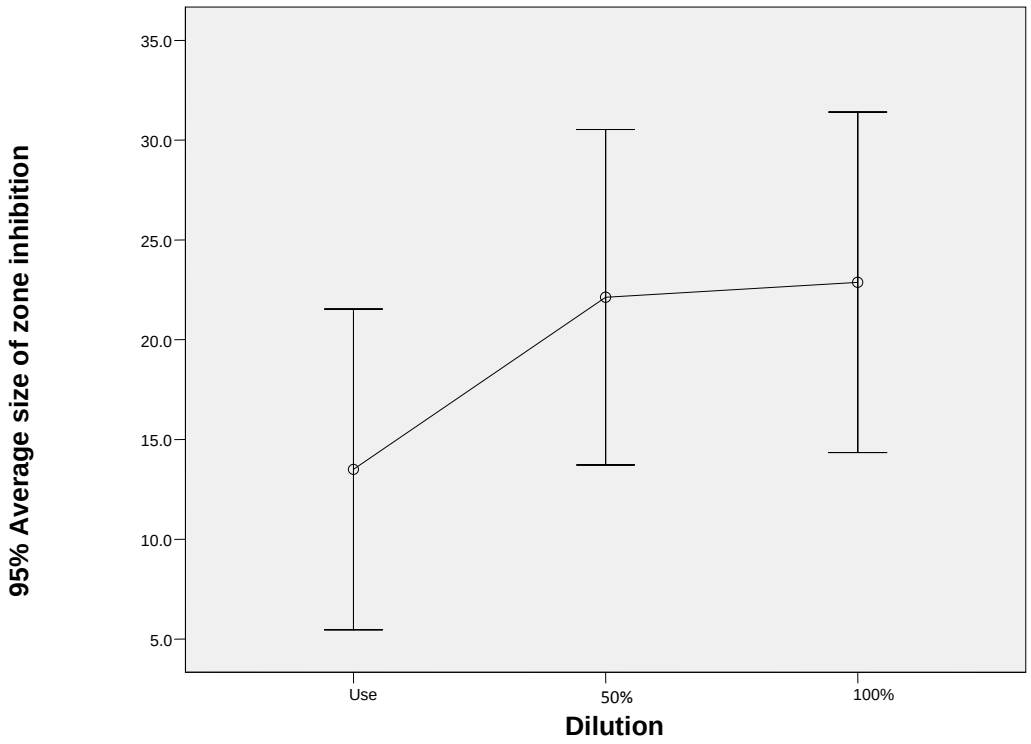


Figure 4.15: Mean and 95% confidence interval for zone inhibition size at three different dilutions in the Kirby- Bauer test

4.5.4.3 Testing the efficacy of the disinfectant using the Phenol test

Data from Table 4.29 were used to calculate the efficacy of *Taski Sani Des J-flex* versus that of Phenol. This test is used when comparisons are made between phenol and phenol-like compounds (Harley, 2008). *Taski Sani Des J-flex* is not a phenol-like compound but due to a lack of any other substances to compare it with, it was decided upon phenol which has been known to be the conventional standard (Harley, 2008).

The calculations (seen underneath Table 4.29) show that *Taski Sani Des J-flex* was 5 times more effective than phenol in destroying Staphylococci, 5,5 times more effective than phenol in destroying Serratia and 5,6 times more effective than phenol in destroying Bacilli.

Table 4.29: Summary of Taski Sani Des J-flex and phenol results according to the micro-organisms, dilutions and time

Disinfectant	Organism	Dilution	5 minutes	10 minutes	15 minutes
Phenol	Staphylococci	1/80	+	-	+
		1/90	+	++	+
		1/100	+	++	+
	Micrococci	1/80	++	+	++
		1/90	+	++	+
		1/100	+	++	+
	Bacilli	1/80	+	+	-
		1/90	+	+	-
		1/100	+	++	-
	Serratia	1/80	++	++	++
		1/90	-	-	-
		1/100	-	-	-
Sani Des	Staphylococci	1/400	-	-	-
		1/450	++	-	-
		1/500	++	-	++
	Micrococci	1/400	+	-	+
		1/450	+	-	++
		1/500	++	-	+++
	Bacilli	1/400	+	+	++
		1/450	+	+	-
		1/500	++	++	++
	Serratia	1/400	-	++	-
		1/450	++	++	++
		1/500	+++	-	++

Absence of growth = "-"

Varying amounts of growth = "+", "++" or "+++"

1/400 dilution *Taski Sani Des J-flex* and 1/80 dilution Phenol killed Staphylococci in 10 minutes.

The Phenol coefficient = 400 ÷ 80 = 5

The disinfectant is thus 5 times more effective than phenol in killing Staphylococci

1/500 dilution *Taski Sani Des J-flex* and 1/90 dilution phenol killed Serratia in 10 minutes

The Phenol coefficient = 500 ÷ 90 = 5,55

The disinfectant is thus 5,55 times more effective than phenol in killing Serratia

1/450 dilution *Taski Sani Des J-flex* and1/80 dilution phenol killed Bacilli in 15 minutes

The Phenol coefficient = 540 ÷ 80 = 5,625

The disinfectant is thus 5,625 times more effective than phenol in killing Bacilli

4.5.5 Objective Five:

To determine the degree of the transmission of normal human microbial flora and the infectious bacterial pathogens from the patients to the beds in the clinic during treatment

Data from week four was used, resulting in sixteen individual paired samples analysed from four different beds at two time points.

4.5.5.1 Comparison of the difference in the viable count of the micro-organisms before and after patient treatment

The median viable count of micro-organisms increased from a median of **0** (ml^{-1}) before treatment to a median of 281 750 (ml^{-1}) (**23 479 cfu/cm^2**) after treatment (Table 4.30). There was a significant increase in the viable count of micro-organisms from before to after treatment (***p*<0.001**), as can be seen in Tables 4.31a and b. Bifero, Prakash and Bergin (2006) assumed that the patients` collective flora deposited on the surfaces of the beds, but results from this study provides evidence that there is indeed a correlation between the quantity of micro-organisms on the beds and the number of patients who have received treatment upon it. These results also indicate the necessity of disinfecting the beds after each patient, as proposed by Evans et al. (2009).

Table 4.30: The viable count of micro-organisms before and after patient treatment

		Viable count before patient treatment	Viable count after patient treatment
n	Valid	16	16
	Missing	0	0
Median		.00000	281750.00000
Minimum		.000	168000.000
Maximum		2680.000	2800000.000
Percentiles	25	.00000	247000.00000
	50	.00000	281750.00000
	75	.00000	1457375.00000

Table 4.31a: Comparison of the median viable count of micro-organisms before and after patient treatment

		n	Mean Rank	Sum of Ranks
Viable count after patient treatment - Viable count before patient treatment	Negative Ranks	0(a)	.00	.00
	Positive Ranks	16(b)	8.50	136.00
	Ties	0(c)		
	Total	16		

a) Viable count after patient treatment < Viable count before patient treatment
b) Viable count after patient treatment > Viable count before patient treatment
c) Viable count after patient treatment = Viable count before patient treatment

Table 4.31b: Wilcoxon signed ranks test based on the negative ranks

	Viable count after patient treatment - Viable count before patient treatment
Z	-3.516(a)
p value(2-tailed)	<0.001

a) based on the negative ranks

4.5.5.2 Comparison of the change in the viable count of the micro-organisms before and after patient treatment between the different beds

The change in the viable counts of the micro-organisms between the different beds was statistically significant (***p*=0.020**). The change was the highest in bed W (Table 4.32 – 4.33b), suggesting that there is either an association between the time that the patient spends on the bed and the quantity of micro-organisms transferred; or it could be related to the different patients being treated on the beds.

Table 4.32: The change in the viable count of micro-organisms between the different beds

Bed	Median	Minimum	Maximum	*n*
G	282000.0000	269000.00	294500.00	4
H	248000.0000	168000.00	294000.00	4
J	260500.0000	236000.00	286000.00	4
W	2270000.0000	1842320.00	2800000.00	4
Total	281750.0000	168000.00	2800000.00	16

Bed H and W were new patients
Bed G and J were follow up patients

Table 4.33a: Kruskal-Wallis test to compare the viable count of the micro-organisms between the different beds

	Bed	*n*	Mean Rank
Change in total micro-organisms	G	4	8.75
	H	4	5.25
	J	4	5.50
	W	4	14.50
	Total	16	

Bed H and W were new patients
Bed G and J were follow up patients

Table 4.33b: Kruskal-Wallis test with the grouping variable being the different beds

	Change in total micro-organisms
Chi-Square	9.816
df	3
p value	0.020

4.5.5.3 Comparison of the change in the viable count of the micro-organisms before and after patient treatment between the different sections of the beds

Table 4.34 – 4.35b shows that there was no significant difference between the different sections (**p=0.287**).

Table 4.34: The change in the viable count of the micro-organisms between the different sections of the beds

Section	Median	Minimum	Maximum	*n*
HR	276000.0000	243500.00	1842320.00	3
HL	1009000.0000	168000.00	1850000.00	2
HM	246000.0000	236000.00	269000.00	3
AR	287000.0000	250000.00	2690000.00	4
AL	294250.0000	277500.00	2800000.00	4
Total	281750.0000	168000.00	2800000.00	16

HR = right headrest, HL = left headrest, HM = middle of headrest, AR = right armrest and AL = left armrest

Table 4.35a: Comparison of the viable count of the micro-organisms between the different sections of the beds

	Section	*n*	Mean Rank
Change in total viable count	HR	3	7.67
	HL	2	7.50
	HM	3	4.00
	AR	4	9.75
	AL	4	11.75
	Total	16	

HR = right headrest, HL = left headrest, HM = middle of headrest, AR = right armrest and AL = left armrest

Table 4.35b: Kruskal-Wallis test with the grouping variable being the different sections of the beds

	Change in viable count
Chi-Square	5.000
df	4
p value.	0.287

4.5.5.4 Comparison of the change in the viable count of the micro-organisms before and after patient treatment between the different beds in the different categories of patients

There was no significant difference between the categories of patients (**p=0.248**) although there was a trend showing that new patients had a greater increase in number of micro-organisms than follow up patients (Tables 4.36 – 4.37b).

Table 4.36: The change in the viable count of micro-organisms between the different categories

Category	Median	Minimum	Maximum	*n*
New patient	1068160.0000	168000.00	2800000.00	8
Follow up patient	276750.0000	236000.00	294500.00	8
Total	281750.0000	168000.00	2800000.00	16

Table 4.37a: Comparison of the viable count of micro-organisms between the different categories

	Category	*n*	Mean Rank	Sum of Ranks
Change in total micro-organisms	New patient	8	9.88	79.00
	Follow up patient	8	7.13	57.00
	Total	16		

Table 4.37b: Mann-Whitney test with the grouping variable being the different categories

	Change in viable count
Mann-Whitney U	21.000
Wilcoxon W	57.000
Z	-1.155
p value (2-tailed)	0.248

4.6 HYPOTHESES

Therefore, with respect to the hypotheses made in chapter one, the following is applicable:

Hypothesis one: The null hypothesis (Ho) states that there will be no micro-organisms present on the beds in the clinic. The alternate hypothesis (Ha) states that there will be micro-organisms present on the beds in the clinic.

The null hypothesis (Ho) is then rejected and the alternate hypothesis (Ha) accepted.

Hypothesis two: The null hypothesis (Ho) states that there will not be a microbial build-up on the beds in the clinic over a 12 hour period. The alternate hypothesis (Ha) states that there will be a microbial build-up on the beds in the clinic over a 12 hour period.

The null hypothesis (Ho) is then rejected and the alternate hypothesis (Ha) accepted.

Hypothesis three: The null hypothesis (Ho) states that the disinfectant currently in use by the clinic is not an effective disinfectant. The alternate hypothesis (Ha) states that the disinfectant currently in use by the clinic is an effective disinfectant.

The null hypothesis (Ho) is then rejected and the alternate hypothesis (Ha) accepted.

Hypothesis four: The null hypothesis (Ho) states that there will be no transmission of normal human microbial flora and infectious bacterial pathogens from the patients to the beds in the clinic during treatment. The alternate hypothesis (Ha) states that there will be transmission of normal human microbial flora and infectious bacterial pathogens from the patients to the beds in the clinic during treatment.

The null hypothesis (Ho) is then rejected and the alternate hypothesis (Ha) accepted.

CHAPTER 5 : CONCLUSIONS AND RECOMMENDATIONS

5.1 CONCLUSIONS

The main aim of this study was to assess the beds as reservoirs for normal microbial flora and infectious bacterial pathogens, at a clinic in South Africa. The following conclusions were made:

- Micro-organisms are present on the beds in the clinic
- The viable amount of micro-organisms at the start of the day, before the commencement of treatment is already 5 times higher than the proposed standards.
- The viable amounts increase significantly throughout the day.
- The micro-organisms present were Staphylococci, Micrococci, Bacilli and Serratia. Of these, the Staphylococci and Serratia are of most concern, seeing that even in low quantities they could result in HAI.
- The disinfectant currently in use by the clinic is effective, which indicates that the quality of the disinfective process is insufficient.
- The micro-organisms are transferred to the beds during treatment from the skin of the patients.
- It seems that the quantity of micro-organisms transferred depends on the length of time that the patient spends on the beds and the different patients.

5.2 RECOMMENDATIONS

- Larger sample groups should be used in future studies to avoid Type II errors.
- The quality of the disinfective process employed by the cleaners needs to be reviewed.
- Although the disinfectant has been proven to be effective, it is less effective against Gram-negative bacteria at the current dilution used. A lower dilution (i.e. higher concentration) or another disinfectant should be considered.
- The disinfection of the beds in between patients is recommended to minimise infection risks.
- Research needs to be done to assess the compliance and adherence of students and staff with hand sanitising practices.
- The staff and students working in the clinic should be educated on the role of hand and bed hygiene practices in the prevention of HAI.

REFERENCES

About South Africa. 2005. Charter to improve healthcare. [online]. Available at: http://www.southafrica.info/ess info/about/health/health-charter-120705.htm. [Accessed 22 May 2009]

About South Africa. nd. Healthcare in South Africa. [online]. Available at: http://www.southafrica.info/ess info/about/health/health.htm. [Accessed 22 May 2009]

About South Africa. 2008a. More houses, more with running water . [online]. Available at: http://www.southafrica.info/ess info/about/health/health-charter-120705.htm. [Accessed 22 May 2009]

About South Africa. 2008b. Poverty in South Africa "is declining". [online]. Available at: http://www.southafrica.info/ess info/about/health/health-charter-120705.htm

Adato, M., Lund F. and Mhlongo, P. 2007. Methodological innovations in research on the dynamics of poverty: a longitudinal study in KwaZulu-Natal, South Africa. World development. 35(2):247 – 263.

Alcamo, I. E. 1994. *Fundamentals of Microbiology*. 4th edition. United States of America. The Benjamin/Cummings Publishing Company Inc.

Al-Hamad, A. and Maxwell, S. 2008. How clean is clean? Proposed methods for hospital cleaning assessment. *Journal of Hospital Infection*. 70(4):328-334

Allied Health Professions Council of South Africa (AHPCSA) [online]. 2009. Available: http://www.ahpcsa.co.za/pb_pbco_chiropractic.htm. [Accessed 22 May 2009].

Al-Windi, A. 2004. Determinants of Complementary and alternative medicine (CAM) use. *Complementary therapies in medicine*. 12(2-3):99-111.

Arcury T. A., Bell, R. A., Sively, B. M., Smith, S. L., Skelly, A. H., Wetmore, L.K. and Quandt, S. A. 2006. Complementary and alternative medicine use as health self-management: Rural older adults with diabetes*. Journal of Gerontology: Psychological sciences.*61(2): S62-S70.

Atlas, R. M. 1997. *Principles of Microbiology*. 2nd edition. United States of America. Wm. C. Brown Publishers.

Banfield, K. R. and Kerr, K. G. 2005. Could hospital patients' hands constitute a missing link? *Journal of Hospital Infection*. 61(3):183 - 188.

Barnes, P. M., Powell-Griner, E., McFann, K. and Nahin, R. L. 2004. Complementary and alternative medicine use among adults: United States, 2002. *Seminars in integrative medicine*. 2(2):54- 71.

Bartley, J. M. and Olmsted, R. N. 2008. Reservoirs of pathogens causing healthcare associated infections in the 21st century: Is renewed attention to inanimate surfaces warranted? *Clinical Microbiology Newsletter*. 30(15):113 - 117.

Baveja, C. P. 2005. Textbook of microbiology. 2nd edition. India. Arya publications (Avichal publishing company).

Becker, K., Bierbaum, G., von Eiff, C., Engelmann, S., Götz, F.,Hacker, J., Hecker, M., Peters, G., Rosenstein, R. and Ziebuhr, W. 2007. Understanding the physiology and adaptation of staphylococci: A post-genomic approach. *International Journal of Medical Microbiology.* 297:483–501.

Beggs, C. B., Kerr, K. G., Noakes, C. J., Hathway, E. A. and Sleigh, P. A. 2008. The ventilation of multiple-bed hospital wards: Review and analysis. *American Journal of Infection Control*. 36(4): 250-259

Ben-Arye, E., Frenkel, M., Klein, A. and Scharf, M. 2008. Attitudes toward integration of complementary and alternative medicine in primary care: Perspectives of patients, physicians and complementary practitioners. *Patient education and counseling*. 70(3):395-402.

Benjamin, R. L. 2007. *A retrospective cross sectional survey of thoracic cases on record at Durban University of Technology Chiropractic day clinic*. M.Tech. Chiropractic dissertation, Durban University of Technology.

Bergmann, T. F., Peterson, D. H. and Lawrence, D. J. 1993. Chiropractic technique. Principles and Procedures. Churchill livingstone.

Bessems, E. 1998. The effect of practical conditions on the efficacy of disinfectants. *International Biodeterioration & Biodegradation*. 41(1998):177 - 183.

Bhatia, R. and Ichpujani, R. L. 2004. *Essentials of Medical Microbiology*. 3rd edition. Jaypee Brothers medical publishers. New Delhi. Page 55.

Bifero, A. E., Prakash, J. and Bergin, J. 2006. The role of chiropractic adjusting tables as reservoirs for microbial diseases. *American journal of Infection Control*. 369(9563):729.

Bickley, L. S. and Szilagyi, P. G. 2003. Bates` guide to physical examination and history taking. 8th edition. United States of America. Lippincott Williams and Wilkins.

Blair, R. C. and Taylor, R. A. 2008. *Biostatistics for the health sciences*. United States of America. Pearson Education Inc.

Bolashikov, Z. D and Melikov, A. K. 2008. Methods for air cleaning and protection of building occupants from airborne pathogens. *Building and Environment*. 44(7): 1378-1385 .

Boone, S. A. and Gerba, P. C. 2007. Significance of fomites in spread of respiratory and Enteric viral disease. *Applied Environmental Microbiology*. 73(6):1687-1696.

Borkow, G. and Gabbay, J. 2007. Biocidal textiles can help fight nosocomial infections. *Medical Hypotheses*. 70(5):990-994.

Bowers, D. 2002. *Medical statistics from scratch*. United Kingdome. John Wiley & Sons Ltd.

Boyce, J. M. 2007. Environmental contamination makes and important contribution to hospital infection. *Journal of Hospital Infection*. 65(S2):50 - 54.

Breathnach. A. S. 2005. Nosocomial infections. *Medicine*. 33(3):22- 26.

Brink, A., Feldman, C., Duse, A., Gopalan, D., Grolman, D., Mer, M., Naicker, S., Paget, G., Perovic, O. and Richards, G. 2006. Guideline for management of Nosocomial Infections in South Africa. *South African Medical Journal*. 96(7):642 – 651.

Brink, A., Moolman, J., da Silva, M. C. and Botha, M. 2007. Antimicrobial susceptibility profile of selected bacteraemic pathogens from private institutions in South Africa. *South African Medical Journal*. 97:273-279

Broxmeyer, L. and Cantwell, A.2008. AIDS: "It`s bacteria, stupid!". Medical hypotheses. 71(5):741-748

Budd, K. and Shipton, E. A. 2004. Acute pain, the immune system and opio-immunosupression. *Acute pain*. 6(3-4):123-135.

Byarugaba, D. K. 2004. Antimicrobial resistance in developing countries and responsible risk factors. *International Journal or Antimicrobial Agents*. 24(2):105 - 110.

Cabral, G. A. and Dove Pettit, D. A. 1998. Drugs and immunity: cannibinoids and their role in decreased resistance to infectious disease. Journal of neuroimmunology. 83(1-2):116 – 123.

Cambron, J.A., Cramer, G. D. and Winterstein, J. 2007. Patient perceptions of chiropractic treatment for primary care disorders. *Journal of Manipulative and Physiological Therapeutics*. 30(1):11 -16.

Campbell, M. J., Machin, D. and Walters, S. J. 2007. *Medical statistics: A textbook for the health sciences*. 4th edition. United Kingdom. John Wiley & Sons Ltd.

Cao, L., Martin, A., Polakos, N. and Moynihan, J. A. 2004. Stress causes a further decrease in immunity to herpes simplex virus-1 in immunocompromised hosts. *Journal of Neuroimmunology*. 156(1-2):21 – 30.

Caraco, T. and Wang, I. 2008. Free-living pathogens: Life-history constraints and strain competition. *Journal of Theoretical Biology*. 250(3):569-579

Ceylan, S., Azal, O., Taşlipinar, A., Türker, T., Açikel, C. H. and Gulec, M. 2009. Complementary and alternative medicine use among Turkish diabetes patients. *Complementary therapies in medicine*. 17(2):78-83.

Chiropractic Association of South Africa (CASA). 2009a. President`s desk. [online] Available at: www.chirpractic.co.za/index.html?lf=1;pg=7. [Accessed 23 May 2009]

Chiropractic Association of South Africa (CASA). 2009b. Chiropractic in South Africa. [online] Available at: www.chirpractic.co.za/index.html?lf=1;pg=9. [Accessed 23 May 2009]

Chao, C. Y. H., Wan, M. P., Morawska, L., Johnson, G. R., Ristovski, Z. D., Hargreaves, M., Mengersen, K., Corbett, S., Li, Y., Xie, X. and Katoshevski, D. 2008. Characterization of expiration air jets and droplet size distributions immediately at mouth opening. 40(2):122 - 133 .

Chapmin-Smith, D. 2008. The Chiropractic profession. *The chiropractic report*. 22(5):1 - 8.

Charasse-Pouélé, C. and Fournier, M. 2006. Health disparities between racial groups in Sout Africa: a decomposition analysis. Social science & medicine. 62(11):2897-2914

Council on chiropractic education international (CCEI). 2005.International chiropractic standards. [online]. Available at: www.cceintl.org. [Accessed 1 June 2009]

Cove, J. H. and Eady, E. A. 1998. Cutaneous antimicrobial defense. Clinics in Dermatology. 16:141-147.

Cozad, A. and Jones, R. D. 2003. Disinfection and the prevention of infectious disease. *American Journal of Infectious Diseases*. 31(4):243 - 254.

Creamer, E. and Humphreys, H. 2008. The contribution of beds to healthcare associated infection: the importance of adequate decontamination. *Journal of Hospital Infection*. 69(1):8 - 23.

Dancer, S. J. 2004. How do we assess hospital cleaning? A proposal for microbiological standards for surface hygiene in hospitals. *Journal of Hospital infection*. 56:10-15.

Davis, M. A. and Bove, G. M. 2008. The chiropractic healer. *Journal of Manipulative and Physiological Therapeutics*. 31(4):323 - 327.

Denyer, S. P. 1995. Mechanisms of Action of Antibacterial Biocides. *International Biodeterioration & Biodegradation*. (1995):227 - 245.

Denyer, S. P. and Stewart, G. S. A. B. 1998. Mechanisms of action of disinfectants. *International Biodeterioration & Biodegradation*. 41(1998):261 - 268.

De Zoysa, H. and Morecroft, E. 2007. Cleaning, disinfection and sterilization of equipment. *Anaesthesia & intensive care medicine*. 8(11): 453- 456.

Donahoe, R. and Klein, T. W. 1996. Cocaine, alcohol, and host defenses. *Journal of Neuroimmunology*. 69(1-2):53-56

Drews, E.R.1994. *A study of demographic and epidemiological factors of private practices and a chiropractic clinic.* M.Tech. chiropractic dissertation, Natal Technikon (now the Durban University of Technology).

Duenas, R. 2002. United states of chiropractic practice acts and the institute of medicine defined primary care practice. *Journal of Chiropractic Medicine*. 1(4):155-169.

Duenas, R., Carucci, G. M., Funk, M. F. and Gurney, M. W. 2003. Chriopractic – Primary care, neuromusculoskeletal care, or musculoskeletal care? Results of a survey of chiropractic college presidents, chiropractic organization leaders, and Connecticut-licensed doctors of chiropractic. *Journal of Manipulative and Physiological Therapeutics*. 26(8):510 – 522.

Duke, K. 2005. A century of CAM in New Zealand: a struggle for recognition. *Complementary Therapies in Clinical Practice.* 11(1):11- 16.

Duse, A.G. 2005. Infection control in developing countries with particular emphasis on South Africa. *The Southern African Journal of Epidemiology and Infection*. 20(2): 37-71.

Ernst, E. 2008. Chiropractic: the critical evaluation. *Journal of Pain and Symptom Management.* 35(5):544- 562.

Estridge, B. H. and Reynolds, A. P. 2007. *Basic clinical laboratory techniques*. 5th edition. USA. Thomson Delmar learning.

Evans, M. W., Breshears, J., Campbell, A., Husbands, C. and Rupert, R. 2007. Assessment and risk reduction of infectious pathogens on chiropractic treatment tables. *Chiropractic and osteopathy*. 15:8.

Evans, M. W., Campbell, A., Husbands, C., Breshears, J., Ndetan, H. and Rupert, R. 2008. Cloth-covered chiropractic treatment tables as source of allergens and pathogenic microbes. *Journal of Chiropractic medicine*. 7(1):34-38.

Evans, M. W., Williams, R. D. and Perko, M. 2008. Public health advocacy and chiropractic: a guide to helping your community reach its health objectives. *Journal of Chiropractic Medicine*. 7(2):71- 77.

Evans, M. W., Ramcharan, M., Floyd, R., Globe, G., Ndetan, H., Williams, R. and Ivie, R. 2009. A proposed protocol for hand and table sanitizing in chiropractic clinics and education institutions. *Journal of Chiropractic Medicine*. 8(1):38- 47.

Far, B. M., Salgado, C. D., Karchmer, T. B. and Sherertz, R. J. 2001. Can antibiotic resistant nosocomial infections be controlled? *The Lancet Infectious Diseases*. 1(1):38- 45.

Fiegel, J., Clarke, R. and Edwards, D. A. 2006. Airborne infectious disease and the suppression of pulmonary bioaerosols. *Drug Discovery Today*. 11(1-2):51-57

Fleming, S., Rabago, D. P., Mundt, M. P. and Fleming, M. F. 2007. CAM therapies among primary care patients using opioid therapy for chronic pain. *BMC Complementary and alternative medicine*. 7:15

Forbes, B. A., Sahm, D. F. and Weissfeld, A. S. 2007. *Bailey & Scott`s Diagnostic Microbiology*. 12th edition. Mosby Elsevier. Missouri. Pages 63 and 103 – 109.

Fredericks, D. 2001. Microbial ecology of human skin in health and disease. *Journal of Investigative Dermatology Symposium Proceedings*. 6: 167- 169.

Freestone, P. P. E., Sandrini, S. M., Haigh, R. D. and Lyte, M. 2008. Microbial endocrinology: how stress influences susceptibility to infection. *Trends in microbiology*. 16(2):55- 64.

French, G. L. 2005. Clinical impact and relevance of antibiotic resistance. *Advanced Drug Delivery Reviews*. 57(10):1514- 1527.

Frenkel, M., Arye, E. B., Carlson, C. and Sierphina, V. 2008. Incorporating Complementary and altrnative medicine into conventional primary care: the patient perspective. *EXPLORE: The Journal of Science and Healing*. 4(3):178-186.
Friese, C. R. 2007. Prevention of infection in patients with cancer. *Seminars in Oncology Nursing*. 23(3):174- 183.

Gardiner, W. P. 1997. Statistics for the Biosciences. A Pearson print on demand edition. United Kingdom. Pearson Education.

Gastmeier, P., Stamm-Balderjahn, S., Hansen, S., Zuischneid, I., Sohr, D., Behnke, M., Vonberg, R. and Rüben, H. 2006. Where should one search when confronted with outbreaks of nosocomial infection? *American Journal of Infection Control*. 1(1):38- 45.

Gatterman, M. I. 1995. Foundations of chiropractic: Subluxation. United States of America. Mosby, Inc.

Gaumer, G. L., Walker, A. and Su, S. 2001. Chiropractic and a new taxonomy of primary care activities. *Journal of Manipulative and Physiological Therapeutics,* 24(4): 239-259.

Gaumer, G., Koren, A. and Gemmen, E. 2002. Barriers to expanding primary care roles for chiropractors: The role of chiropractic as primary care gatekeeper. *Journal of Manipulative and Physiological Therapeutics,* 25(7): 427-449.

Gennery, A. R. and Cant, A. J. 2006. Applied physiology: Immune competence. *Current Pediatrics*. 16(6):447- 452.

Graf, B. M. and Martin, E. 2000. The intensive care physician and control of antimicrobial resistance. *International Journal of Antimicrobial Agents*. 16(4):511 - 514.

Greenwood, D., Slack, D., Peutherer, J. and Barer, M. 2007. Medical microbiology. A guide to microbial infections: pathogenesis, immunity, laboratory diagnosis and control. 17th edition. United States of America. Churchill Livingstone.

Hakaletho, E. Semmelweis` present day follow-up: Updating bacterial sampling and enrichment in clinical hygiene. *Pathophysiology*. 13(4): 257-267.

Haldeman, S (ed). 2005. *Principles and Practice of Chiropractic*. 3rd edition. United States of America. The McGraw-Hill Companies, Inc.

Harley, J. P. 2008. Laboratory exercises in Microbiology. 7th edition. New York. Mc Graw Hill Higher education. Pages 107 – 109, 338, 255 – 259.

Harling, G., Ehrlich, R. and Myer, L. 2008. The social epidemiology of tuberculosis in South Africa: A multilevel analysis. *Social sciences & Medicine*. 66(2):492-505.

Heinzelmann, M., Scott, M. and Lam, T. 2002. Factors predisposing to bacterial invasion and infection. *The American Journal of Surgery*. 183(2):179 - 190.

Higgs, M. 2009. A demographic and descriptive survey of Chiropractic patients at the Chiropractic Clinic at Kimberley Hospital Complex. M. Tech. Chiropractic Thesis. Durban University of Technology, Durban.

Hori, S., Mihaylov, I., Vasconcelos, J. C. and McCoubrie, M. 2008. Patterns of complementary and alternative medicine use amongst outpatients in Tokyo, Japan. BMC complementary and Alternative medicine.

Hota, B. 2004. Contamination, Disinfection and Cross-colonization: Are hospital surfaces reservoirs for nosocomial infection? Clinical infectious diseases CID 39(8)1182 - 1189.

Ižman, M. Č. 2003. The use and resistance to antibiotics in the community. *International Journal of Antimicrobial Agents*. 21(4):297 - 307.

Jaman, R. 2007. *A retrospective cross-sectional survey of lumbo-sacral cases recorded at the D.U.T chiropractic day clinic (1995-2005)*. M.Tech. Chiropractic dissertation, Durban University of Technology.

Jamison, R. Dietary diversity: a case study of fruit and vegetable consumption by chiropractic patients. *Journal of Manipulative and Physiological Therapeutics*. 26(6):383-389.

Johansson, L. M. 2007. Fiscal implications of AIDS in South Africa. *European Economic review*. [online] 51(7):1614-1640.

Jones, R. N., Ballow, C. H., Biedenbach, D. J. and the ZAPS Study Group Medical Centers. 2001. *Diagnostic Microbiology and Infectious Disease*.40: 59–66.

Kampf, G. and Kramer, A. 2004. Epidemiologic background of hand hygiene and evaluation of the most important agents for scrubs and rubs. *Clinical Microbiology Rev*iews. 17(4):863 - 893.

Kapp, C. 2007. South Africa unveils new 5-year HIV/AIDS plan. *The Lancet*. 369(9573):1589-1590.

Khandai, S. 2007. *A retrospective cross sectional survey of extremity cases on record at the Durban University of Technology Chiropractic Day Clinic (1995-2005)*. M.Tech. Chiropractic dissertation, Durban University of Technology.

Kirkaldy-Willis, W. H. and Bernard, T. N. 1999. Managing low back pain. 4th edition.United states of America. Churchill Livingstone.

Koneman, E., Allen, S. D., Janda, W. M., Schreckenberger, P. C. and Winn, W. C. 1997. Color atlas and textbook of diagnostic microbiology. 5th edition. United States of America. Lippincott-Raven Publishers.

Korporaal, C. 2009. Interviewed by Jana Logtenberg. Durban University of Technology. 22 May 2009.

Kramer, A., Schwebke, I. and Kampf, G. 2006. How long do nosocomial pathogens persist on inanimate surface? A systematic review. *Infectious diseases*. 6:130.

Kremer, R. G., Duenas, R. and McGuckin, B. 2002. Defining primary care and the chiropractic physician`s role in the evolving health care system. *Journal of Chiropractic medicine*. 1(1):3-8.

Lambert, I., Tebbs, S. E., Hill, D., Moss, H. A., Davies, A. J. and Elliott, T. S. J. 2000. Interferential therapy machines as possible vehicles for cross-infection. *Journal of Hospital infection*. 44:59-64.

Lankford, M. G., Collins, S., Youngberg, L., Rooney, D. M., Warren, J. R. and Noskin, G. A. 2006. Assessment of materials commonly utilized in health care: Implications for bacterial survival and transmission. *American Journal of Infection Control*. 34(5)258-263.

Larson, E. L., Cronquist, A. B.,Whittier, S., Lai, L., Lyle, C. T., Della Latta, P. 2000. Differences in skin flora between inpatients and chronically ill outpatients. *Heart & Lung: The Journal of Acute and Critical Care.* 1(4):307 - 320.

Lawrence, D. J. and Meeker, W. C. 2007. Chiropractic and CAM utilization: A descriptive review. Chiropractic and Osteopathy. 15:2 .

Leboeuf-Yde, C., Pedersen, E. N., Bryner, P.,Cosman, D., Hayek, R., Meeker, W. C., Shaik, J., Terrazas, O., Tucker, J. and Walsh, M. 2005. Self-reported Nonmusculoskeletal Responses to Chiropractic Intervention: A Multination Survey. *Journal of Manipulative and Physiological Therapeutics* 28(5):294-302

Littlewood, R. A. and Vanable, P. A. 2008. Complementary and alternative medicine use among HIV+ people: research synthesis and implications for HIV care. *AIDS care* 20(8): 1002-1018.

Longest, P. W. and Kleinstrauer, C. 2004. Interacting effects of uniform flow, plane shear, and near-wall proximity on the heat and mass transfer of respiratory aerosols. *International Journal of Heat and Mass Transfer*.47(22):4745 - 4759.

Lynch, P., Pittet, D., Borg. M. A. and Mehtar, S. 2007. Infection control in countries with limited resources. *Journal of Hospital Infection*. 65(S2):148- 150.

Ma, L., Tsui, F., Hogan, W., Wagner, M. and Ma, H. 2003. A Framework for Infection Control Surveillance Using Association Rules. *AMIA Annual symposium proceedings archive*. 2003: 419 - 414.

Macías, A. E. and Ponce-de-León, S. 2005. Infection control: Old problems and New challenges. *Archives of medical research*. 36(6):637- 645.

MacIntyre, R. C., Holzemer, W. L. and Philippek, M. 1997. Complementary and alternative medicine and HIV/AIDS. Part 1 Issues and Context. *Journal of the association of nurses in AIDS care*. 8(1):23-31.

Madigan, M. T., Martinko, J. M. and Parker, J. 2000. Brock. Biology of micro-organisms. 9th edition. United States of America. Prentice Hall.

Magee. D. J. 2006. Orthopedic physical assessment. Enhanced edition. United States of America. Elsevier.

Maharaj, P. 2009. The knowledge and perceptions of the provincial and national Health Portfolio Committee members of South Africa regarding the chiropractic profession. M.Tech. Chiropractic dissertation, Durban University of Technology.

Mahomed, F. 2007. *Chiropractic patients in South Africa: A Demographic and Descriptive Profile*. M.Tech. Chiropractic dissertation, Durban University of Technology.

Maier, R. M., Pepper, I. L. and Gerba, C. P. 2000. *Environmental microbiology*. USA. Academic Press.

Maillard, J. 2005. Antimicrobial biocides. *Therapeutics and Clinical Risk Management*.1(4):307-320.

Makoae, L. N., Seboni, N. M., Molosiwa, K., Moleko, M., Human, S., Sukati, N. A. and Holzemer, W. L. 2005. The symptom experience of people living with HIV/AIDS in Southern Africa. *Journal of the association of nurses in AIDS care*. 16(3):22-32.

Manga, P. 2000. Economic case for the integration of chiropractic services into the health care system. *Journal of Manipulative and Physiological Therapeutics.* 23(2):118-122 .

Marcos, A. 1997. The immune system in eating disorders: An overview. Nutrition. 13(10):853-862.

Masterton, R., Drusano, G., Paterson, D. L. and Park, G. 2003. Appropriate antimicrobial treatment in nosocomial infections – the clinical challenges. *Journal of Hospital Infection*. 55(1):1 - 12.

Meeker, W. C. and Haldeman, S. 2002. Chiropractic: A profession at the crossroads of mainstream and alternative medicine. *Annals of Internal medicine*. 136(3):216 - 223.

Menke, J. M. 2003. Principles in integrative chiropractic. *Journal of Manipulative and Physiological Therapeutics.* 26(4):254-272 .

McAllister-Sistilli, C. G., Caggiula, A. R. and Knopf, S. 1998. The effects of nicotine on the immune system. *Psychoneuroendocrinology*. 23(2):175 - 187.

McDonnell, G. and Russell, A. D. 1999. *Antiseptics and Disinfectants: Activity, Action and Resistance*. 12(1):147 - 179.

Moor, P. and Ferguson, S. 2006. Nosocomial infections in critically ill. *Anaesthesia and intensive care medicine*. 7(5):148- 151.

Mootz, R. D., Hansen D. T., Breen, A., Killinger, L. Z. and Nelson, C. 2006. Health Services Research Related to Chiropractic: Review and Recommendations for Research Prioritization by the Chiropractic Profession. *Journal of Manipulative and Physiological Therapeutics.* 29(9):707-725 .

Morawska, L., Johnson, G. R., Ristovski, Z. D., Hargreaves, M., Mengersen, K., Corbett, S., Chao, C. Y. H., Li, Y. and Katoshevski, D. 2008. Size distribution and sites of origin of droplets expelled from the human respiratory tract during expiratory activities. *Journal of Aerosol Science*.

Morgan, I. 2005. Is chiropractic a CAM therapy or is it a separate profession? *Journal of Canadian Chiropractic Association*. 49(3):133-136.

Munro, J. F. (ed) and Campbell, I. W. (ed) 2000. Macleod`s Clinical examination. 10th edition. United Kingdom. Churchill Livingstone.

Nahin, R. L., Dahlhamer, J. M., Taylor, B. L., Barnes, P. M., Stussman, B. J., Simile, C. M., Blackman, M. R., Chesney, M. A., Jackson, M., Miller, H. and McFan, K. K. 2007. Health behaviours and risk factors in those who use complementary and alternative medicine. BMC Journal or Public Health. 7:127.

Naude, W., Rossouw, S. and Krugell, W. 2009. The non-monetary quality of city life in South Africa. *Habitat International*. 33(4)319-326.

Ohara, T., Itoh, Y. and Itoh, K. 1998. Ultrasound instruments as possible vectors of staphylococcal infection. *Journal of Hospital Infection*. 40:73-77.

Oumeish, I., Oumeish, O. I. and Bataineh, O. 2000. Acute bacterial skin infections in children. *Clinics in dermatology*. 18(6):667-678.

Owens, R. C. 2008. Antimicrobial stewardship: concepts and strategies in the 21st century. *Diagnostic Microbiology and Infectious Diseases*. 61(1):110 - 128.

Pagano, M. and Gauvreau, K. 2000. *The principles of Biostatistics*. 2nd edition. United States of America. Duxbury Thomson Learning.

Parikh, A. and Veenstra, N. 2008. The evolving impact of HIV/AIDS on outpatient services in KwaZulu-Natal, South Africa. *South African Medical Journal*. 98(6): 468-472

Peltzer, K., Friend-du Preez, N., Ramlagan, S. and Fomundam, H. 2008. Use of traditional complementary and alternative medicine for HIV patients in Kwa-Zulu-Natal, South Africa. *BMC Public Health*. 8:255.

Penn, C. 1991. *Handling laboratory microorganisms*. Philadelphia. Open university press.

Pittet, D., Allegranzi, B., Sax, H., Dharan, S., Pessoa-Silva, C. L., Donaldson, L., Boyce, J. M. and on behalf of the WHO Global Patient Safety Challenge, World Alliance for Patient Safety. 2006. Evidence-based model for hand transmission during patient care and the role of improved practices. *The Lancet Infectious Diseases*. 6(10):641- 652.

Pittet, D., Allegranzi, B., Storr, A., Bagheri Nejad, S., Dziekan, G., Leotsakos, A. and Donaldson, L. 2008. Infection control as a major World Health Organization priority for developing countries. *Journal of Hospital infection*. 68(4):285 - 292.

Pollack, R. A., Findlay, L., Mondschein, W. and Modesto, R. R. 2002. *Laboratory exercises in Microbiology*. John Wiley & Sons, Inc. USA. Pages vii, viii, 4 - 5, 16 – 21, 47 – 50, 56 – 57, 91 – 96, 101 – 105, 113, 134 and 151.

Printzen, G.1996. Relevance, pathogenicity and virulence of microorganisms in implant related infections. *Injury* 27(3):S-C9 – S-C15.

Pruett, S. B. 2003. Stress and the immune system. *Pathophysiology*. 9(3):133-153

Rataemane, S. and Rataemane, L. 2006. Alcohol in South Africa. *International Journal of Drug Policy*. 17(4):373-375

Reasoner, D. J. 2003. Heterotrophic plate count methodology in the US. *International Journal of Food Microbiology*. 92(3):307 – 315.

Redwood, D. and Cleveland, C. S. 2003. Fundamentals of Chiropractic. United States of America.Mosby, Inc.

Roghmann, M. and McGrail, L. 2006. Novel ways of preventing antibiotic-resistant infections: What might the future hold? *American Journal of Infection Control*. 34(8):469 - 475.

Rutala, W. A. and Weber, D. J. 2004. *Disinfection and Sterilization in Healthcare facilities: What clinicians need to know*. Clinical infectious diseases CID 39(5)702 - 709.

Rutala, W. A., Weber, D. J. and Healthcare Infection Control Practices Advisory Committee (HICPAC). 2008. Guideline for disinfection and Sterilization in healthcare facilities. [online] Available at: www.cdc.gov/ncidod/ahqp/pdf/guidelines/disinfection_Nov_2008.pdf [Accessed September, 2008]

Samartin, S. and Chandra, R. K. 2001. Obesity, overnutrition and the immune system. *Nutrition research*. 21(1-2):243-262.

Sandefur, R., Febbo, T.A. and Rupert, R. L. 2005. Assessment of the knowledge of primary care activities in a sample of medical and chiropractic students. *Journal of Manipulative and Physiological Therapeutics,* 28(5): 336-344.

Sanders, D. and Chopra, M. 2006. Key challenges to achieving health for all in an inequitable society: The case of South Africa. *American Journal of Public Health*. 96(1):73-78

Schabrun, S. and Chipcase, L. 2006. Healthcare equipment as a source of nosocomial infection: a systematic review. *Journal of Hospital infection*. 63(3):239 - 245.

Schafer, R. C. and Faye, L. J. 1990. Motion palpation and Chiropractic technic. Principles of dynamic chiropractic. USA. The motion palpation institute.

Schönfeldt, H. C. and Gibson, N. 2008. Healthy eating guidelines in the South African context. *Journal of food composition and analysis*. Manuscript in press, accepted for publishment.

Sharma, R., Haas, M. and Stano, M. 2003. Patient attitudes, insurance and other determinants of self-referral to medical and chiropractic physicians. *American Journal of Public Health*. 93(12):2111-2117.

Sheldon, A. T. 2005. Antiseptic “resistance”: real or perceived threat? Clinical infectious Diseases. 40(1 June):1651 - 1656.

Shook, J. E. 1995. Infection control in the emergency department. *Seminars in Pediatric Infectious Diseases*. 6(4):265 – 272

Sher, L. 2003. Effects of heavy alcohol consumption on the cardiovascular system may be mediated in part by the influence of alcohol-induced depression on the immune system. *Medical hypotheses*. 60(5):702 – 706.

Sherman, K.J., Cherkin, D.C., Connelly, M.T., Erro, J., Savetsky, J.B., Dais, R.B. and Eisenberg, D.M. 2004. Complementary and alternative medical therapies for chronic low back pain: What treatments are patients willing to try? *BMC Complement Alternative Medicine*, 4(9): 134–153.

Shmueli, A. and Shuval, J. 2006. Complementary and alternative medicine: Beyond users and nonusers. *Complementary therapies in medicine*. 14:4):261-267.

Singh, V., Raidoo, D. M. and Harries, C. S. 2004. The prevalence, patterns of usage and people's attitude towards complementary and alternative medicine (CAM) among the Indian community in Chatsworth, South Africa. *BioMed Central Complementary and Alternative medicine.* 4:3.

Singh, J. A., Upshur, R. and Padayatchi, N. 2007. XDR-TB in South Africa: No Time for Denial or Complacency. PLos Medicine. 4(1): e50.

Sirois, F. M. 2008. Provider based complementary and alternative medicine use among three chronic illness groups: Associations with psychosocial factors and concurrent use of conventional healthcare services. *Complementary therapies in medicine*. 16(2): 73-80.

Srivastava, S. and Shetty, N. 2007. Healthcare associated infections in neonatal units: lessons from contrasting worlds. *Journal of Hospital infection*. 65(4):292 - 306.

Statistics South Africa. 2006. Provincial profile Kwa-Zulu Natal South Africa. [online] Available at: www.statssa.gov.co.za/Publications/Report-00-91-05/Report-00-91-052004.pdf. [Accessed: 27 January 2009]

Struelens, M. J., Denis, O. and Rodriquez-Villalobos, H. 2004. Microbiology of nosocomial infections: progress and challenges. *Microbes and Infection*. 6(11):1043-1048.

Talaro, K. and Talaro, A. 1993. *Foundations in Microbiology*. United States of America. Wm. C. Brown Publishers.

Tang, J. W., Li, Y., Eames, I., Chan, P. K. S. and Ridgway, G. L. 2006. Factors involved in the aerosol transmission of infection and control of ventilation in healthcare premises. *Journal of Hospital Infection*. 64(2):100-114.

Thoresen, B. 2006. P*atient satisfaction at the Durban University of Technology chiropractic day clinic.* M.Tech. Chiropractic dissertation, Durban University of Technology.

Van den Biggelaar, A. H. J., Huizinga, T. W. J., de Craen, A. J. M., Gussekloo, J. Heijmans, B. T., Frölich, M. and Westendorp, R. G. J. 2004. Impaired innate immunity predicts frailty in old age: The Leiden 85-plus study. *Experimental Gerontology*. 39(9):1407- 1414.

Venketsamy, Y. 2007. *A retrospective cross-sectional survey of cervical cases recorded at the Durban University of Technology (D.U.T) chiropractic day clinic (1995-2005).* M.Tech. Chiropractic dissertation, Durban University of Technology.

Vincent, J. 2003. Nosocomial infections in adult intensive-care units. *The Lancet*. 361(9374):2068- 2077.

Vuoksimaa, E., Koskenvuoa, M., Rosea, R. J. and Kaprio, J. 2008. Origins of handedness: A nationwide study of 30 161 adults. Neurophysiologica. 47:1294–1301

Wechsberg, W. M., Luseno, W. K., Karg, R. S., Young, S., Rodman, N., Myers, B. and Parry, C. D. H. 2008. Alcohol, cannabis, and methamphetamine use and other risk behaviours among Black and Coloured South African women: A small randomized trial in the Western Cape. *International Journal of Drug Policy.* 19(2):130-139

Weber, D. J., Rutala, W. A. and Sickbert-Bennett. 2007. Outbreaks associated with contaminated antiseptics and disinfectants. Antimicrobial agents, and Chemotherapy. 51(12):4217-4224.

World Health Organization (WHO). 2002. Prevention of Health-care associated infections [online]. Available at http://www.who.int/csr/resources/publications/drugresist/en/whocdscsreph2002/2.pdf [Accessed 30 March 2008]

World Health Organization (WHO). 2004. Practical guidelines for infection control in healthcare facilities. Available at www.wpro.who.int/NR/rdonyres/006EF250_6B11_42B4_BA17_C98D413BE8BB/0/practical_guidelines_infection_control.pdf [Accessed 30 March 2008]

World Health Organization (WHO). 2005. WHO guidelines on basic training and safety in chiropractic. [online]. Available at http://aps.who.int/medicinedocs/en/m/abstract/Js14076e.pdf [Accessed 30 March 2008]

World Health Organization (WHO). 2008. World health primary report. Primary health care. Now more than ever. [online]. Available at http://www.who.int/whr/2008/en/index.html [Accessed 30 March 2008]

World Health Organization, nd. Hospital hygiene and infection control. Available at www.who.int/water_sanitation_health/medicalwaste/148to158.pdf [Accessed 12 December 2008]

World federation of Chiropractic (WFC). 2001. Definition of Chiropractic. Available at www.wfc.org. [Accessed 10 May 2009]

Wright, G. D. 2003. Mechanisms of resistance to antibiotics. *Current Opinion in Chemical Biology*. 7(5):563- 569.

Wu, H., Wang, A. H. and Jennings, M. P. 2008. Discovery of virulence factors of pathogenic bacteria. *Current opinion in Chemical Biology*. 12(1):93-101.

Zhu, S., Kato, S. and Yang, J. 2006. Study on transport characteristics of saliva droplets produced by coughing in a calm indoor environment. *Building and Environment*. 41(12):1691-1702.

Annexure A – Data Collection sheet 1

Label:

Part of study:	1.1	1.2	1.3	2	4

Date:	Time:	Code:	Specimen: Hr, Hl, Hm, Ar, Al

Serial dilution concentration:	-1	-2	-3	-4	-5	-6
Nr of colonies in 1st set:						
Nr of colonies in 2nd set:						
Average:						

Viable count count (ml^{-1})

=(Average cfu) ÷ (volume in ml × optimum counting dilution)

=( ) ÷ (0,1ml ×)

=( ) ÷ ( )

=

Annexure B – Data Collection sheet 2

Average nr of colonies enumerated:	Label:

Date:	Time:	Code:	Specimen: Hr, Hl, Hm, Ar, Al

Serial dilution concentration:	-1	-2	-3	-4	-5	-6

Macroscopic colony morphology:			
	Size:	Margin:	
	Shape:	Surface appearance:	
	Colour:	Consistency:	
	Elevation:	Changes in medium:	
Microscopic morphology:			
	Simple stain	Gram stain	
	Morphological shape	Positive	
	Arrangement	Negative	

Annexure C – Data Collection sheet 3

Growth inhibition

			Degree of growth				
			Times of exposure in minutes				
Bacteria	Use dilution	Control	1	2	5	10	15
Staphylococcus							
Micrococcus							
Bacillus							
Serratia							

Kirby Bauer

Bacteria	Dilution	Zone size plate 1	Zone size plate 2	Average zone size
Staphylococcus	Use			
	50%			
	100%			
Micrococcus	Use			
	50%			
	100%			
Bacillus	Use			
	50%			
	100%			
Serratia	Use			
	50%			
	100%			

Annexure C – Data Collection sheet 3 (continued)

Phenol coefficient

Bacteria	Solution	Dilution	Degree of growth		
			Time of exposure in minutes		
			5	10	15
Staphylococci	Phenol	1/80			
		1/90			
		1/100			
	Disinfectant	1/400			
		1/450			
		1/500			
Micrococci	Phenol	1/80			
		1/90			
		1/100			
	Disinfectant	1/400			
		1/450			
		1/500			
Bacilli	Phenol	1/80			
		1/90			
		1/100			
	Disinfectant	1/400			
		1/450			
		1/500			
Serratia	Phenol	1/80			
		1/90			
		1/100			
	Disinfectant	1/400			
		1/450			
		1/500			

Calculation of phenol coefficient:

= (1 ÷) ÷ (1 ÷)

= ( ) ÷ ( )

=

Annexure D – Letter of request to Clinic Director for permission to conduct research study at Durban University of Technology Clinic

DURBAN UNIVERSITY of TECHNOLOGY

To whom it may concern

My name is Jana Logtenberg, I'm a current sixth year student pursuing my M. Tech Chiropractic qualification at the Durban University of Technology (DUT). I would like to conduct my research at the Durban University of Technology Chiropractic Day Clinic.

Studies have revealed the presence of many infectious bacterial pathogens on Chiropractic adjustment beds at teaching institutions with the highest average of colony forming units on the armrest (42) then the headrest (40) and lastly the thoracic portion (24). My research will be a quantitative clinical investigation with the aim of assessing the Chiropractic adjustment beds as reservoirs for normal microbial flora and infectious bacterial pathogens at the Durban University of Technology Chiropractic Day Clinic.

My objectives will be to verify the presence, enumerate, isolate, identify and establish the incidence of individual skin and throat microbial flora and the infectious bacterial pathogens. I will also be determining the microbial build-up over a 12hr period, assessing the efficacy of the disinfectant (Nupine) currently in use as well as illustrate and ascertain the degree of the transmission of flora and pathogens from the patients to the Chiropractic adjustment beds during treatment.

This research study will proceed without the knowledge of the students, cleaning staff and patients. All the Chiropractic Adjustment beds will be sampled by rubbing a swab over the head- and armrest at specific times depending on the objective in question. Microbiological processing and analysis of these samples will take place in the Microbiology Laboratory located on the Steve Biko Campus with the help of a laboratory technician. All data obtained will be recorded on data collection sheets which will have randomly assigned codes and labels that will allow for easy identification and correlation and will ensure unbiased results. All data will be confidential and only viewed by my co-supervisor Dr A. Docrat, supervisor Prof B. Odhav and myself.

For purposes of Ethics approval, permission is required from the Clinic Director to perform this research. The Faculty of Health Sciences Research and Ethics Committee requires acknowledgement from the Clinic Director in the form of a signature, indicating that in principle the Clinic Director will grant approval of the proposed research pending its final approval by the same committee.

Thank you for your co-operation.

Yours sincerely,
Logtenberg
Miss Jana Logtenberg
Student number: 20302099

B. Odhav.
Prof B. Odhav
Supervisor

Docrat
Dr A. Docrat
Co-supervisor

Approved

24/7/08

HOD: Chiropractic + Somatology

Annexure E – Letter of request to Head of Biotechnology and Food technology Department for permission to conduct study at Durban University of Technology Clinic

DURBAN UNIVERSITY of TECHNOLOGY

To whom it may concern

My name is Jana Logtenberg, I'm a current sixth year student pursuing my M. Tech Chiropractic qualification at the Durban University of Technology (DUT). I would like to conduct my research at the Durban University of Technology Chiropractic Day Clinic.

Studies have revealed the presence of many infectious bacterial pathogens on Chiropractic adjustment beds at teaching institutions with the highest average of colony forming units on the armrest (42) then the headrest (40) and lastly the thoracic portion (24). My research will be a quantitative clinical investigation with the aim of assessing the Chiropractic adjustment beds as reservoirs for normal microbial flora and infectious bacterial pathogens at the Durban University of Technology Chiropractic Day Clinic.

My objectives will be to verify the presence, enumerate, isolate, identify and establish the incidence of individual skin and throat microbial flora and the infectious bacterial pathogens. I will also be determining the microbial build-up over a 12hr period, assessing the efficacy of the disinfectant (Nupine) currently in use as well as illustrate and ascertain the degree of the transmission of flora and pathogens from the patients to the Chiropractic adjustment beds during treatment.

This research study will proceed without the knowledge of the students, cleaning staff and patients. All the Chiropractic Adjustment beds will be sampled by rubbing a swab over the head- and armrest at specific times depending on the objective in question. Microbiological processing and analysis of these samples will take place in the Microbiology Laboratory located on the Steve Biko Campus with the help of a laboratory technician. All data obtained will be recorded on data collection sheets which will have randomly assigned codes and labels that will allow for easy identification and correlation and will ensure unbiased results. All data will be confidential and only viewed by my co-supervisor Dr A. Docrat, supervisor Prof B. Odhav and myself.

For purposes of Ethics approval, permission is required from the Head of the Biotechnology Department to perform this research. The Faculty of Health Sciences Research and Ethics Committee requires acknowledgement from the Head of the Biotechnology Department in the form of a signature, indicating that in principle the Head of the Biotechnology Department will grant approval of the proposed research pending its final approval by the same committee.

Thank you for your co-operation.

Yours sincerely,

Logtenberg

Miss Jana Logtenberg
Student number: 20302099

B. Odhav.

Proff B. Odhav
Supervisor

Dr. A. Docrat
Co-supervisor

APPROVED.

30/07/2008.

Head of the Biotechnology Department

Annexure F – Ethics clearance certificate

DURBAN UNIVERSITY of TECHNOLOGY

Faculty of Health Sciences

ETHICS CLEARANCE CERTIFICATE

Student Name	**Jana Logtenberg**	**Student No**	**20302099**
Ethics Reference Number	**FHSEC** 035 | 08	**Date of FRC Approval**	
Research Title:	**An assessment of Chiropractic adjustment beds as reservoirs for normal microbial flora and infectious bacterial pathogens at a Chiropractic Day Clinic.**		

In terms of the ethical considerations for the conduct of research in the Faculty of Health Sciences, Durban University of Technology, this proposal meets with Institutional requirements and confirms the following ethical obligations:

1. The researcher has read and understood the research ethics policy and procedures as endorsed by the Durban University of Technology, has sufficiently answered all questions pertaining to ethics in the DUT 186 and agrees to comply with them.
2. The researcher will report any serious adverse events pertaining to the research to the Faculty of Health Sciences Research Ethics Committee.
3. The researcher will submit any major additions or changes to the research proposal after approval has been granted to the Faculty of Health Sciences Research Committee for consideration.
4. The researcher, with the supervisor and co-researchers will take full responsibility in ensuring that the protocol is adhered to.
5. ***The following section must be completed if the research involves human participants:***

	YES	NO	N/A
❖ Provision has been made to obtain informed consent of the participants			X
❖ Potential psychological and physical risks have been considered and minimised			X
❖ Provision has been made to avoid undue intrusion with regard to participants and community	X		
❖ Rights of participants will be safe-guarded in relation to: - Measures for the protection of anonymity and the maintenance of Confidentiality.	X		
- Access to research information and findings.	X		
- Termination of involvement without compromise			X
- Misleading promises regarding benefits of the research			X

Logtenberg — 21/8/2008
SIGNATURE OF STUDENT/RESEARCHER — DATE

21/8/2008
SIGNATURE OF SUPERVISOR/S — DATE

29/9/08
SIGNATURE OF HEAD OF DEPARTMENT — DATE

1/10/08
SIGNATURE: CHAIRPERSON OF RESEARCH ETHICS COMMITTEE — DATE