

# Treatment patterns of dermatological disorders in the private health care sector of Namibia

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## **PREFACE AND ACKNOWLEDGEMENTS**

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- To all the patients that gave a few minutes of their time to complete questionnaires for the data-collection process.

## SUMMARY

Title: Treatment patterns of dermatological disorders in the private healthcare sector of Namibia.

Keywords: Dermatological disease; prevalence; treatment patterns; private healthcare sector; Namibia.

Many patients suffer from dermatological diseases throughout the world. Literature about this problem is emphasizing that it is getting worse. Factors such as poor hygiene, poverty and diseases such as HIV/AIDS, have increased the prevalence of dermatological diseases in developing countries such as Namibia. Understanding the different dermatological diseases and studying their prevalence will aid in ensuring patients better quality of life.

The aim of the study was to investigate the prevalence and medicinal treatment patterns of dermatological diseases in the private healthcare sector of Namibia, with special reference to Windhoek.

The research methodology was divided into two sections, namely a literature analysis and an empirical study. The literature analysis has been done to ensure knowledge about dermatological conditions before the empirical study was started.

The empirical study was divided into two phases and data were collected from the community pharmacy environment (Phase 1) and a dermatologist (Phase 2). A total number of 507 patients participated in this study.

In the community pharmacy environment, data were gathered from dermatological prescriptions of general practitioners (Phase 1A) and from pharmacist-initiated therapy prescriptions (Phase 1B). The data collected from the dermatologist (Phase 2), were collected from patients files at the dermatologist's practise.

Phase 1A indicated that urticaria (n=36) had the highest prevalence followed by eczema (n=28) and contact dermatitis (n=28).

49% of the patients that participated in this phase were seeking treatment for the same condition the second time. In Phase 1B, contact dermatitis (n=15) showed the highest prevalence with eczema (n=14) and urticaria (n=8) second and third respectively.

77% of the patients participating in this phase of the research study did not have a family history of the same dermatological diseases.

Phase 2 indicated that the highest prevalence of dermatological diseases was acne vulgaris (n=30) and melasma (n=19). The treatment duration that occurred most often in this phase was 180 days.

Over all, the data indicated that eczema was the dermatological disease with the highest incidence of 11.2% (n=57). Other diseases that played a significant part were acne vulgaris (10.5%), urticaria (9.0%), contact dermatitis (8.6%) and melasma (7.1%). Rare dermatological diseases such as Kaposi sarcoma showed relatively high prevalence (n=9). It was concluded that this could be due to the fact that the dermatologist consulted, had been the only dermatologist claiming directly from the government medical aid, and that most of the patients diagnosed with Kaposi sarcoma during this research study were government employees.

Many dermatological diseases were not specifically defined or diagnosed, but still treated with topical corticosteroids which may suggest that the term eczema is undefined and easily used by different healthcare practitioners for dry-skin related conditions.

It is concluded in this research study that the three most prevalent dermatological diseases in the private healthcare sector of Namibia are eczema, acne vulgaris and urticaria. These conditions are not considered to be life-threatening, but they do have a significant effect on the quality of life of patients.

## **OPSOMMING**

Titel: Behandelingspatrone van dermatologiese probleme in die privaat gesondheidsorgsektor van Namibië.

Sleutelwoorde: Dermatologiese siekte; voorkoms; behandeling patrone; privaat gesondheidsorg; Namibië.

Baie pasiënte wêreld wyd ly aan dermatologiese siektes. Literatuur oor hierdie probleem beklemtoon dat dit vererger. Faktore soos swak higiëne, armoede en siektes soos MIV/Vigs, het die voorkoms van dermatologiese siektes in ontwikkelende lande, soos Namibië, laat toeneem. Kennis oor die verskillende dermatologiese siektes en die bestudering van hul voorkoms sal help om pasiënte se kwaliteit van lewe te verbeter.

Die doel van die studie was om die voorkoms en medisinale behandelingspatrone van dermatologiese siektes in die privaat gesondheid sektor van Namibië, met spesiale verwysing na Windhoek, te ondersoek.

Die navorsingsmetodologie is verdeel in twee afdelings, naamlik 'n literatuur studie en 'n empiriese studie. Die literatuur studie is gedoen om kennis oor dermatologiese toestande te verseker voordat die empiriese studie begin.

Die studie was in twee fases opgedeel. In die eerste fase was data vanuit die gemeenskaps apteek omgewing verkry en die tweede fase vanaf 'n dermatoloog se praktyk.

In die gemeenskaps apteek omgewing, was data wat uit dermatologiese voorskrifte van algemene praktisyns (Fase 1A) en van apteker-geïnisieerde terapie (Fase 2A), versamel. Die data vanaf die dermatoloog (Fase 2) was versamel vanuit pasientlêrs.

'n Totaal van 507 pasiënte het aan hierdie studie deelgeneem.

Fase 1A het aangetoon dat urtikarie (n = 36) die hoogste voorkoms het, gevolg deur ekseem (n = 28) en kontak dermatitis (n = 28). 49 % van die pasiënte wat deelgeneem het in hierdie fase is op soek na behandeling vir dieselfde toestand vir die tweede keer.

In Fase 1B, het kontak dermatitis (n = 15) die hoogste voorkoms met ekseem (n = 14) en urtikarie (n = 8) tweede en derde onderskeidelik. 77 % van die pasiënte wat deelneem het in hierdie fase van die navorsing het nie 'n familie geskiedenis van dieselfde dermatologiese siektes gehad nie.

Fase 2 het aangedui dat die hoogste voorkoms van dermatologiese siektes aknee vulgaris (n = 30) en melasma (n = 19) is. Die behandelings tydperk in meeste van die gevalle in hierdie fase, was 180 dae.

Die totale data vanuit die twee fases het aangedui dat ekseem die dermatologiese siekte met die hoogste voorkoms van 11,2 % (n = 57) was. Ander siektes wat 'n belangrike deel bygedra het was aknee vulgaris (10,5 %), urtikarie (9,0 %), kontak dermatitis (8,6 %) en melasma (7,1 %). Skaars dermatologiese siektes soos Kaposi sarkoom het relatief hoë voorkoms (n = 9) aangedui. Dit was grootendeels omdat die pasiënte wat daarmee gediagnoseer was, staatsdienswerknemers was en dat die spesifieke dermatoloog die enigste een was wat direk by die staatsmediese fonds geeis het.

Die gevolgtrekking in hierdie studie is dat die drie mees algemene dermatologiese siektes, in die privaat gesondheidsorg sektor van Namibië, ekseem, aknee vulgaris en urtikarie is. Baie dermatologiese siektes was nie spesifiek omskryf of gediagnoseer is nie, maar wel behandel met kortikosteroïede. Dermatologiese toestande soos ekseem is ongedefinieerd en word maklik gebruik om droë vel, en ander verwante toestande, te beskryf. Hierdie toestande word wel nie beskou as lewensgevaarlik nie, maar dit het 'n beduidende effek op die kwaliteit van lewe van pasiënte.

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## LIST OF ABBREVIATIONS

**HIV/AIDS** - Human immunodeficiency virus/ acquired immunodeficiency syndrome

**GDP** - Gross domestic product

**WHO** - World Health Organization

**TB** - *Tuberculosis* Bacterium

**ACTH** - Adrenocorticotrophic hormone

**GAG** - Glycosaminoglycans

**ACD** - Allergic contact dermatitis

**AD** - Atopic dermatitis

**LSC** - Lichen simplex chronicus

**UV** - Ultra violet

**DNA** - Deoxyribonucleic acid

**MCV** - *Molluscum contagiosum* virus

**VZ** - *Varicella Zoster*

**HPV** - Human papilloma virus

**PUVA** - Psoralen combined with UV-A

**CKD** - Chronic kidney disease

**PUPPP** - Pruritic urticarial papules and plaques of pregnancy

**AB** - Albino black patients

**AC** - Albino coloured patients

**B** - Black patients

**CC** - Caucasian patients

**C** - Coloured patients

**ARV** - Antiretroviral treatment

**ACE** - Angiotensin-converting-enzyme inhibitor

**PAD** - Peripheral artery disease

**USA** - United states of America

## CHAPTER 1: INTRODUCTION

Namibia gained independence in 1990 after a century under German and South African rule. It has a population of 3 million people and extends over approximately 824 000 square kilometers. This population is very unevenly distributed with half of the people living in the northern parts of Namibia.

The life expectancy is 49 years and 40% of the population is under the age of 15. According to the National Planning Commission report of 2003, this is mainly caused by the high incidence of HIV/AIDS (National Planning Commission, 2008:2).

Namibia is one of the wealthiest countries in Africa, with a GDP of N\$10 400 in 2004. Its Gini Coefficient is 0.6, which makes it the most unequal distribution of resources in the world. 28% of the population lives in poverty and 67% in rural areas, most whom rely on subsistence farming (National Planning Commission, 2006:4).

Namibia has 343 hospitals and clinics in both the private and public healthcare sector. The public sector serves 85% of the population and the private sector 15%. The public healthcare sector is extremely understaffed with a doctor to patient ratio of 1:7500. Only 18.6% of the total population is covered by a medical aid scheme. The private healthcare sector has a doctor to patient ratio of 1:810 (Brokmeyer, 2012:1-3).

Namibia is one of the most arid countries in the world, with an average rainfall of just above 300mm per year. According to the City of Windhoek municipality, the current population of Windhoek is 281,300 with a water demand of 22 million m<sup>3</sup> per year. Lahnsteiner *et al* (2007) calculated that this is an average of 60,275m<sup>3</sup> of water per day. However, due to the uncertainty of rainfall and regular droughts, the City of Windhoek estimates the daily use between 60,000 to 100,000m<sup>3</sup> in 2011. Namibia has reclamation plants that purify sewage water. The water available to the public is a combination of purified water, dam water and ground water. This water is classified as hard which indicates a high mineral content or concentration of multivalent cations (Fawell *et al.*, 2003:5).

The burden of skin disease has been increasingly emphasised over the past few years by individuals and organisations alike. Common skin disease and related conditions do not cause any obvious symptoms and patients tend to ignore the condition for long periods before seeking healthcare. Furthermore, the lack of accessibility to dermatological care in Namibia has resulted in the community pharmacist to providing the first-line treatment.

In the first chapter of the dissertation, the processes followed to obtain information and data relevant to this research study are discussed. A brief overview of the chapters that follow will be provided at the end.

## **1.1 BACKGROUND AND PROBLEM STATEMENT**

The prevalence of dermatological disorders is steadily increasing according to the World Health Organization (Mathers *et al.*, 2001:672). In its Global Burden of Disease report of 2001, more than 20 000 mortalities have been associated with skin disorders in Sub-Sahara Africa alone. These rates are comparable to meningitis and hepatitis B in that region (Mathers *et al.*, 2001:672).

Dermatological disorders are common in developing countries due to poor hygiene, underlying causes such as HIV/AIDS, overcrowding in some areas and poverty (Amerson *et al.*, 2010:16). In 2005, the WHO indicated in its annual report on worldwide disease that dermatological disorders are however assumed by the general public to be benign and not life-threatening (World Health Organization report, 2005:11). In 2002, the organisation published a paper on drugs for parasitic infections in which it was estimated that between 21% and 87% of the general population in developing countries suffered from skin disease (World Health Organization report, 2002:24). Another study conducted in south-western Ethiopia concluded that 67% of households were not reporting skin disease (Figuerola *et al.*, 1996:378).

According to a communication by the Health Professionals Council of Namibia secretary (2012), the registration data for 2012 indicate a lack of medical skills in Namibia. This data has not yet been formally published however; it is shown that the



private sector has 667 registered general practitioners and other specialist physicians, more than half of them living in Windhoek. The public sector has one doctor for every 7500 people. Namibia has five registered dermatologists in both these sectors. The three practicing dermatologists in Windhoek visit the city periodically since they are also practicing in South Africa. It has been confirmed that treatment failure rates of dermatological problems are more than 80% in developing countries due to the lack of elementary skills in the management thereof (Figueroa *et al.*, 1996:378).

Another burden on the Namibian healthcare system is the high cost of specialist care. The five dermatologists' practices were contacted to obtain their consultation and administration fees. This information was collected via a communication with the five dermatologists' secretaries (2012). The consultation fees are between N\$600 and N\$1200 in Windhoek. Of the three registered dermatologists in Windhoek, only one of them claims consultation fees directly from medical aid funds. The remaining dermatologists ask for cash ahead and the patient has the option to claim back from his/her medical aid or health insurance.

This lack of accessibility to quality healthcare and the worldwide increasing costs of healthcare (Dehkharghani *et al.*, 2003:592), cause patients to come to the pharmacy for so-called "free consultation". This allows the community pharmacist to provide most of the first-line treatment in dermatological disorders. Skin diseases such as scabies, superficial mycoses, pyoderma, pediculosis, eczema, contact dermatitis, pigmentary anomalies, acne and HIV/AIDS related skin disease are most common in developing countries (Hay *et al.*, 2004:708).

The dominant skin diseases in Namibia, as explained by one of Namibia's dermatologists in an interview, Dr. FJA Smith, are acne vulgaris and melasma. This is mostly due to the majority of ethnic skin, intense solar radiation and long-term use of bleaching agents such as hydroquinone. Eczema, psoriasis and urticaria are also common, according to Dr. Smith. He believes that the mica rock formations in the Khomas Highlands cause a variety of allergic reactions which can be linked to some of these dermatological diseases.

However, no studies specifically relating to the dermatological conditions and the treatments thereof have been conducted in Windhoek, Namibia, up to date.

Research questions covered in this study were:

- What spectrum of dermatological conditions is common in the private health sector of Namibia?
- What is the prevalence of identified dermatological disorders in Namibia?
- Which demographical trends exist regarding the prevalence of the different dermatological problems?
- How are these dermatological conditions treated?
- What significance do HIV/AIDS and other chronic diseases have on the prevalence of skin disease in the private sector of Namibia?
- Which geographical trends exist within the scope of the study?
- Why are the conditions identified in Namibia unique?
- What recommendations can be made to improve the provision of dermatological healthcare in Namibia?

## **1.2 RESEARCH OBJECTIVES**

### **1.2.1 General objectives**

The aim of study is to investigate the prevalence and medicinal treatment patterns of dermatological diseases in the private health sector of Namibia, with special reference to Windhoek.

### **1.2.2 Specific research objectives of the literature study**

The specific objectives of the literature study include the following:

- To describe the anatomy and physiology of the skin;
- state the classification system for skin lesions;
- identify and describe different dermatological conditions with high prevalence in Namibia and define the causes of these conditions;
- describe the treatment regime of these conditions;

- identify the relationship between different dermatological conditions and other contributing factors, such as:
  - Race
  - Age
  - TB and HIV/AIDS
  - Chronic disease
  - Allergies
  - Climate and geographical area
  - Pregnancy
  - Cigarette smoke
  - Genetics.

### **1.2.3 Specific research objectives of the empirical study:**

The specific research objectives of the empirical study are the following:

- To identify the prevalence of dermatological conditions in Windhoek, by identifying patients with dermatological conditions. This includes patients with prescription from their general practitioner, patients who visited the dermatologist and patients coming to the community pharmacy for pharmacist-initiated therapy;
- determine the relationship between the different dermatological conditions and demographical data such as age, race and gender;
- determine the geographical distribution of patients with dermatological diseases;
- investigate the possible differences in the prevalence of dermatological problems among patients with HIV/AIDS;
- formulate recommendations concerning the treatment of dermatological conditions in the private health sector with special reference to Windhoek.

### 1.3 RESEARCH METHODOLOGY

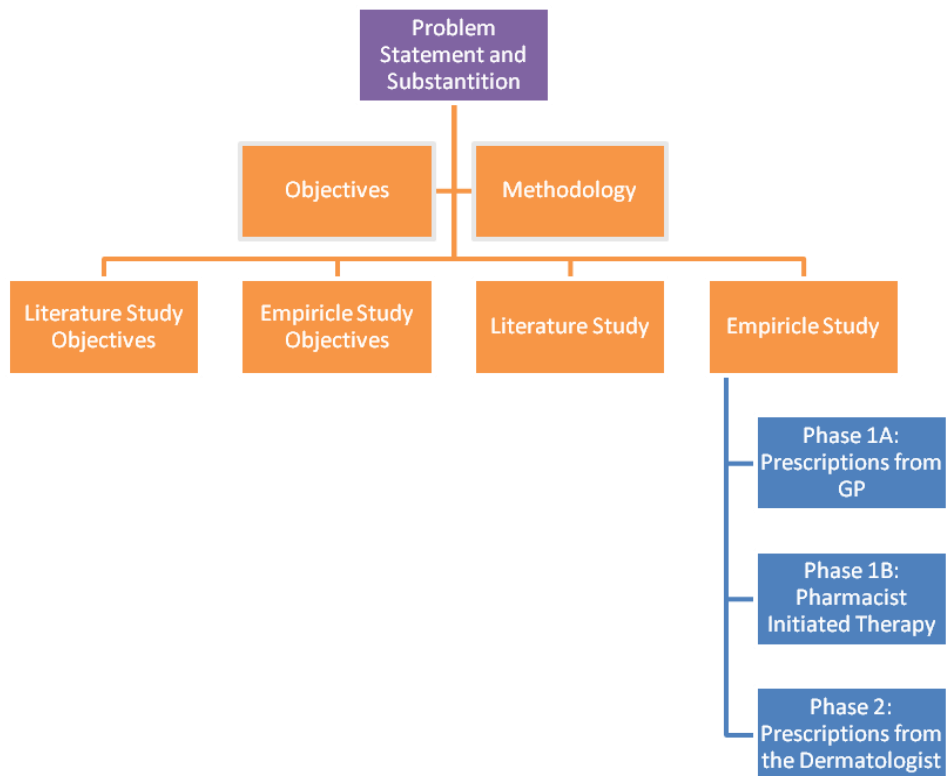


Figure 1: Research Study Plan

The research methodology will be discussed in this section. A brief overview of the literature and empirical study will follow.

#### 1.3.1 Literature Study

The anatomy and physiology of the skin play a significant role in this study and have been studied extensively from literature. The skin lesion classification system was studied from the online Merck Manual of Diagnosis and Treatment for healthcare professionals (MacNeal, 2013). The Merck Manual of Diagnosis and Treatment has also used in other sections of the literature study as it is considered a reliable contemporary reference.

Several books on dermatological diseases and their treatment were identified and studied. Various international published papers and articles on epidemiology and management of skin diseases were also studied. Current treatment regimens were

scrutinized and studied. Literature on how to approach and examine the dermatological patient was also studied. Various studies were also been identified to illustrate the relationship between dermatological disease and contributing factors such as HIV/AIDS, race and socio-economic factors.

### **1.3.2 Empirical Study**

The empirical study took place in two phases; Phase 1 and Phase 2. Phase 1 was done prospectively and Phase 2 retrospectively. Both were done by means of an observational study. Data were collected from three different sources in order to meet the research objectives indicated in 1.2.

#### **1.3.2.1 Phase 1: Community Pharmacy**

This phase was done prospectively by means of an observational study. The target population was patients (N=302) with dermatological prescriptions from general practitioners (thus excluding prescriptions from dermatologists) and patients (N=74) without a prescription, consulting the community pharmacy for pharmacist-initiated therapy or self-medication.

**Phase 1A:** Data were collected from the community pharmacy environment over a period of three months after ethical approval. The dermatological-related prescriptions were collected from two different community pharmacies located in the northern (Katutura) and southern (Suiderhof) of Windhoek. These pharmacies were specifically chosen to ensure a broad spectrum of prescriptions from various ethnic groups.

Every second dermatological prescription received by the community pharmacist was collected. Patients had given permission to participate in the research project in writing by filling in a form of consent. Having given consent (see *Annexure A*), a community pharmacy questionnaire was filled in by the patients (see *Annexure B*) in a provided private area which allowed them to complete the questionnaire without disturbance. The prescription details, including the diagnosis were obtained from the

patient or prescribing doctor; the treatment and treatment duration were recorded on this form by the pharmacist.

On average, the pharmacies received approximately six dermatological-related prescriptions per day. A total of 302 prescriptions were collected over a three-month period after approval from the ethics committee for Phase 1A of the study.

The following data were collected during this phase:

- Demographic data.
- Pregnancy and breastfeeding.
- Smoking.
- Chronic diseases.
- Current treatment for chronic diseases.
- First/second/third line of treatment.
- Dermatological diagnosis.
- Treatment provided for dermatological disorder.
- Period of treatment for dermatological disorder.

The following data were not collected:

- Personal details, for example patient's name and address.
- Family and personal medical history not relevant to dermatology.
- Prescribing doctor's personal details or name.

**Phase 1B:** Patients that consulted the community pharmacy for pharmacist-initiated therapy, thus without prescriptions from a doctor, were examined privately in a consultation area specially prepared for this research study. This data were collected from Auas Valley Pharmacy only.

Every second dermatological case received by the pharmacy was documented after written consent had been obtained from the patient (see *Annexure A*). After written consent, a patient survey form was completed by the pharmacist to obtain patient data (see *Annexure C*). This data were collected over a period of three months.

Skin lesions were classified according to the Merck Manual's skin lesion classification system (Merck Manual, 19<sup>th</sup> Edition, 2011). A dermatological diagnosis

was determined and therapy was provided for the patient. A follow-up consultation took place, by telephone or physical examination, to ensure treatment was successful or to refer the patient to a general practitioner. Some patients were, however, not available for a telephonic or personal follow-up consultation.

The following data were collected during this phase:

- Demographic data (date of birth, gender, race and occupation).
- Contact details for follow-up purposes only.
- Chronic diseases and treatment.
- Description of lesion.
- Possible diagnosis.
- Treatment provided.
- Period of treatment.
- Whether a patient is referred to their general practitioner or not.

The following data were not collected:

- Personal details, for example patient's name and address.
- Family and personal medical history not relevant to dermatology.

### **1.3.2.2 Phase 2: Dermatologist**

This phase was done retrospectively by means of an observational study. The target population was patients (n=131) from a dermatologist practice in Windhoek.

Data were collected retrospectively from patient files, of patients examined by the dermatologist over a period of three months. Only patients that had given their written consent were included in this data (see *Annexure A*).

This data were physically collected and documented in a structure survey form (see *Annexure E*). The dermatologist had given his written consent for the data collection from his practice (see *Annexure D*).

The following data were collected from the patient files:

- Demographic data including age, race, gender and region where person lives in Namibia (geographical location).

- Diagnosis of dermatological disease.
- Recorded observation of type of lesion.
- Treatment provided.
- Duration of treatment.
- The number of patients that are also on anti-retroviral, TB and oncology treatment.
- Follow-up results when available.

The following data were not collected:

- The patient's personal details.
- Family and personal medical history.
- Medical aid information.

#### **1.4 ETHICAL CONSIDERATIONS**

Ethical permission was obtained from and approved by the ethical committee of the North-West University. The reference number is: NWU-00061-12S5.

No personal patient information has been used or documented in this study. All data collected have been kept in a secure office on one personal computer of the primary researcher.

#### **1.5 STATISTICAL ANALYSIS**

Data analysis was performed in SAS Version 9.1.3 (SAS Institute, Cary, NC). All statistical significance was considered with probability of  $p < 0.05$ . The practical significance of the results was computed when the  $p$ -value was statistically significant ( $p \leq 0.05$ ). Chi-square test ( $\chi^2$ ) was used to determine if an association existed between proportions of two or more groups. The Cramer's V statistics was used to test practical significance of this association.



## **1.6 STUY LIMITATIONS**

Data pertaining to this study were collected during a period of just over three months; which did not allow for seasonal changes to be taken into consideration. Other examples of limitations included:

- The patient may have consulted different doctors during Phase 1A. No differentiation in data collection had been done to distinguish between second line treatment from the same doctor or second opion from a new doctor.
- Since the use of ICD-10 codes is not compulsory in Namibia, the prescribers had to be contacted to provide the dermatological diagnosis based on their memory about the specific case.
- In Phase 1B, the type of employment or other daily activities were not recorded which may have provided better information on dermatological conditions.
- Phase 2, some data variables such as demographic information and HIV status were absent in patient files. The dermatologist was then asked to provide this data verbally, relying his memory of the specific case.
- The geographical data collected in this phase indicated where the patient had lived only; not how long the patient had been living in that area or whether the patient had recently travelled.

## **1.7 OVERVIEW OF CHAPTERS TO FOLLOW**

CHAPTER 2: Anatomy of the Skin and Dermatological Diseases

CHAPTER 3: Methodology

CHAPTER 4: Results

CHAPTER 5: Conclusion and Recommendations

Annexure A - F

References

## **1.8 CHAPTER SUMMARY**

Chapter 1 refers to the background and problem statement for this research study. The objectives of the study and the methodology used were briefly discussed. Ethical considerations, statistical analysis and study limitations are pointed out.

## **CHAPTER 2: ANATOMY OF THE SKIN AND DERMATOLOGICAL DISEASES**

### **INTRODUCTION**

An understanding of the anatomy of the skin is essential to ensure the correct interpretation of pathological processes in the skin and to determine the possible aetiology and treatment of the dermatological diseases. The function of the skin, the skin anatomy, physiology, immunology and allergic reactions are examined in this chapter. The most significant dermatological diseases, the treatment thereof and the different factors influencing these diseases are also discussed in this chapter.

### **2.1 ANATOMY**

The skin is in a constant state of change due to external shedding of cells and the replacement by inner cells moving up to the surface (Amirlak, 2011:1). The skin is a metabolically active organ which plays the significant role of protecting the body against injury and foreign penetration. It was commonly believed that the skin protected the body against all penetration of external substances, but the skin is actually quite permeable to many substances like cosmetics, detergents and other chemicals (Casey, 2002:47).

The skin consists of a stratified, cellular epidermis, an underlying dermis of connective tissue and a fatty layer, the subcutis (Amirlak, 2011:1).

#### **2.1.1 Epidermis**

The thickness of the epidermis varies from 0.1 - 1.2mm, depending on the location on the skin. The epidermis can be divided into four layers, namely stratum corneum, stratum granulosum, stratum spinosum and stratum basale. It does not have blood vessels and relies on the dermis for support (Iizuka, 1994:215).

Keratinocytes are most prominent in the epidermis and are found throughout the four layers of the epidermis (Proksch *et al.*, 2008:1063). Their main function is to synthesise keratin. The stratum corneum provides the skin with its prominent barrier

system, preventing penetration of foreign entities and water loss (Proksch *et al.*, 2008:1064).

Melanocytes are found in the basal layer of the epidermis. They produce melanin which protects the skin against harmful ultraviolet rays (Quinn, 2004:1). Skin areas that are constantly exposed to the sun have a ratio of 1:4 melanocytes to keratinocytes. Areas not exposed to the sun may have a ratio as small as 1:3. Pigmentation differentiation between individuals is related to melanocytes size rather than cell number. Melanin production is further stimulated by ACTH (adrenocorticotrophic hormone), oestrogen and progesterone (Amirlak, 2011:1).

Langerhans cells are mostly found in the stratum spinosum layer of the epidermis and are immunologically active. They have the ability to activate T-cells in lymph nodes (Quinn, 2004:1). Merkel cells are the sensory cells of the epidermis and are specialized in the perception of light touch (Polakvicova *et al.*, 2011:80).

### **2.1.2 Dermis**

The main function of the dermis is to support and sustain the epidermis (Powell, 2004:1). Fibroblasts are the most prominent cell type found in the dermis and are responsible for the synthesis of collagen, elastin, GAG (glycosaminoglycans) and other connective tissue. It varies in thickness from 0.6 – 3mm. It contains blood and lymphatic vessels, sweat glands, sebaceous glands and hair follicles (Gawkrödger, 2002:2).

The dermis can be divided into two layers, namely the papillary dermis, which is adjacent to the epidermis, and the reticular dermis, which is the deeper, thicker layer. The papillary layer consists of collagen which is loosely interwoven. The reticular layer consists of more structured form of collagen and elastin connected in bundles (Marks *et al.*, 2006:8). There are 11 different types of collagen in the human skin. Type I collagen is the most common and allows the skin to withstand deformation by providing tensile strength. Type III and Type V are less common, but also play significant roles in stabilising and supporting Type I collagen (De Leo *et al.*, 1998:2).

### **2.1.3 Subcutis**

The subcutaneous layer consists of connective tissue and fat. It is around 2 – 3mm thick (Gawkrodger, 2002:2). Its main function is to connect the skin to the underlying bone and muscle, and to store fat. This fatty hypodermis also serves as padding and insulation to the body. It contains three different types of cells, namely adipose cells, macrophages and fibroblasts (Shimizu, 2007:19).

### **2.1.4 Epidermal Appendages**

#### **2.1.4.1 Hair Follicles**

Hair is found throughout the human skin with exception to areas like palms and soles. The hairs found in genital and auxiliary areas absorb mechanical friction, while eye lashes prevent dirt from entering the eyes (Shimizu, 2007:20).

The base of the hair follicle or derma papilla lies in the inner parts of the dermis. The outer cuticle contains keratinocytes and the inner medulla contains melanocytes which gives hair colour (Amirlak *et al.*, 2011:4-5). The melanins responsible for the pigmentation of hair follicle are eumelanin (lighter hair) and pheomelanin (darker hair) (Thody *et al.*, 1991:340).

The human body has three types of hair, namely the lanugo, vellus and terminal hairs. Lanugo hairs are fine and long and are formed at 20 weeks of gestation of a foetus. These hairs are normally shed before birth. Vellus hairs are short and fine covering most of the body. Terminal hairs are the thicker and darker hairs covering the scalp, pubic areas and auxiliary areas (Krause *et al.*, 2006:2).

The arrector pili muscle is responsible for lifting the hair shaft during temperature changes or emotions such as fear (Paus *et al.*, 1999:491).

The sebaceous gland opens onto the upper root sheath. This gland is discussed in detail under point 2.1.4.2, as it plays a significant role in dermatological diseases.

Caucasian hair follicles are oriented obliquely to the skin surface, whereas the hairs of Asian persons are oriented vertically. A black person's hair follicles are oriented parallel to the skin (Amirlak *et al.*, 2011:4).

#### **2.1.4.2 Sebaceous Glands**

Sebaceous glands are the largest and most concentrated in the face, neck, back and scalp areas with 400 - 900 glands/cm<sup>2</sup> (De Leo *et al.*, 1998:2).

The gland's main function is to produce complex oils that lubricate the skin and prevent moisture loss. These oils consist of triglycerides, wax monoesters, free fatty acids and squalene. A holocrine process takes place when cells release their lipid cytoplasm as they are disintegrated (Smith *et al.*, 2007:271).

Over stimulation or production of sebum causes acne which mostly takes place during puberty when androgen levels change. Acne is a result of sebum and keratin producing a hyperkeratotic plug in the skin pore (Zouboulis *et al.*, 2004:360-366).

#### **2.1.4.3 Sweat Glands**

Sweat glands are most concentrated in hands, feet and the axillae (Amirlak *et al.*, 2011:5). Their main function is the production of sweat that cools the body by evaporation and regulating electrolyte and fluid. Sweat consists mostly of water (98%), but also proteins, steroids, sodium chloride, fatty acids, lactic acid, citric acid, urea, and uric acid (Draelos, 2005:182).

#### **2.1.4.4 Nails**

Nails are made of a dense plate of keratin approximately 0.3 - 0.5mm thick (Gawkrodger, 2002:6). The finger nail grows 2 - 3mm per month and takes around six months to re-grow a complete new nail (Gupta *et al.*, 2005:87).

The nail is made up of three parts: the plate, matrix and bed. The nail plate, which lies on top of the nail bed, is made of dividing keratinocytes (Venus *et al.*, 2011:471). When they mature, they keratinise and form the nail plate. The white half-moon seen

at the base of the nail plate is called white lunula, and is caused by inadequate keratinisation (Shimizu, 2007:21).

## **2.2 PHYSIOLOGY OF THE SKIN**

As already discussed, the skin consists of two main structural layers: the epidermis (outer layer) and the dermis (deep, inner layer).

### **2.2.1 Keratinocyte Life Cycle**

The epidermis consists of various cells which are in different stages of transition as demonstrated by Figure 2. Protein bridges, called desmosomes, connect the different cells to one another (Downing *et al.*, 2000:13). The keratinocyte life cycle takes place in the epidermis of the skin. The basal cells, which are found in the bottom layer of the epidermis, is where reproduction of skin cells starts. Undifferentiated cells from the basal layer divide continuously. The dividing basal cells replicate every 400 hours. It then takes about 14 days for the cell to move to the stratum corneum and around 14 days to be shed as corneocytes by the skin (Gawkrodger, 2002:6).

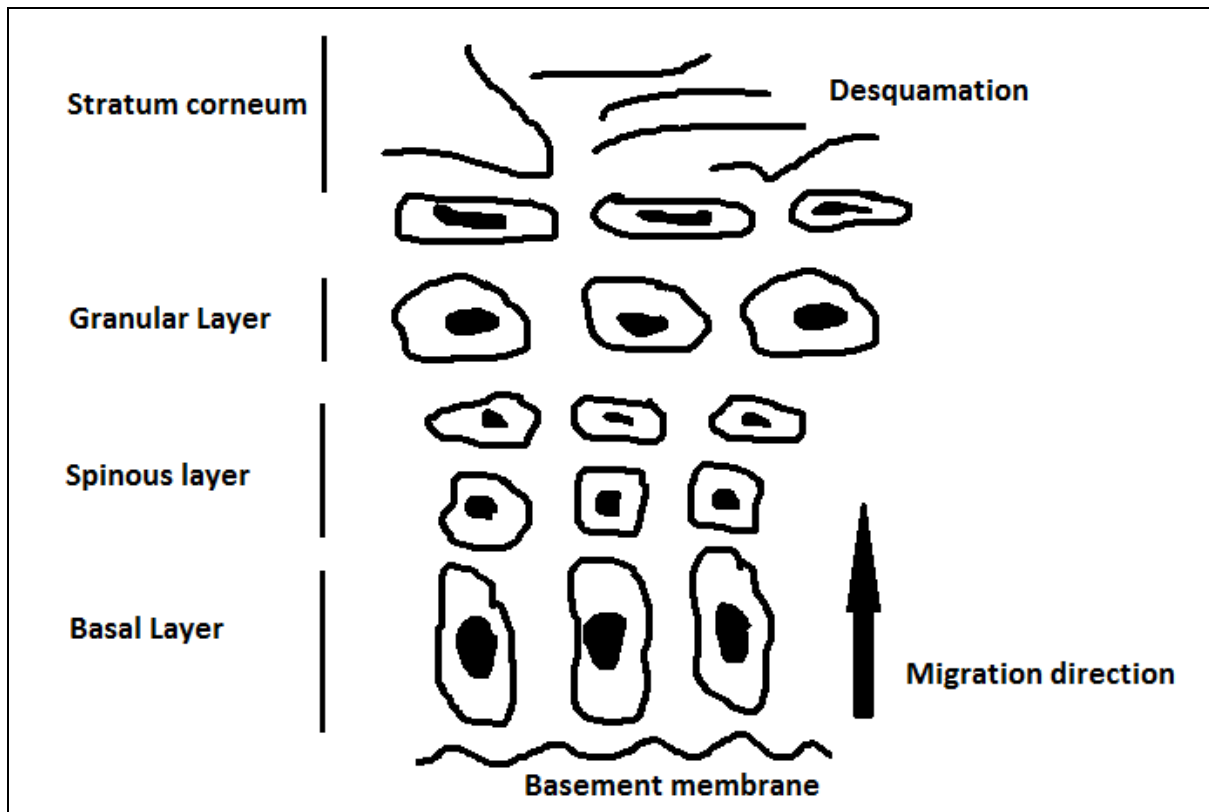


Figure 2: Keratinocyte Maturation Process (adapted from De Leo *et al.*, 1998:3)

In the spinous cell layer, the cells change from columnar to polygonal. The cells then move to the granular layer where enzymes induce degeneration of cell organelles (Gurlek *et al.*, 2002:76).

When the cells reach the top layer of the skin, namely the stratum corneum, the cells are known as corneocytes. These cells lack a nucleus and other cellular structures. Corneocytes are filled with water retaining keratin proteins surrounded by lipids. There are 10 - 30 layers of stacked corneocytes, depending on the area of the skin. These allow the stratum corneum to be flexible and strong (Downing *et al.*, 2000:14).

### 2.2.2 Breakdown of Skin

During the keratinocyte life cycle, as the cells are moving to the stratum corneum, granules, filled with a protein called filaggrin, form in the granular layer (Figure 2). The keratin proteins bind with the filaggrin proteins to form a fillargin-keratin complex; this protects the proteins against proteolytic breakdown as the cell moves upwards, differentiating in every layer (Engelrud, 2000:109).



As the corneocytes move towards the stratum corneum, enzymes start breaking down the filaggrin-keratin complex. Specific proteolytic enzymes further break down the filaggrin protein to amino acids as the moisture contents of the cells in the skin decrease. These processes takes place only if the skin is dry, to control the osmotic pressure of the water in the skin. These amino acids, together with lactic acid, urea and other mineral salts, give the skin its hygroscopic property (Marino, 2006:2).

### 2.2.3 Shedding of Skin (Desquamation)

Desquamation is an enzymatic process by which the desmosomes between the corneocytes are broken down. These specific enzymes are located intracellularly and are activated only if the skin is sufficiently hydrated. If the skin does not contain enough moisture, in other words less than 30%, desquamation does not take place effectively and a scaly skin appearance results. (Engelrud, 2000:110).

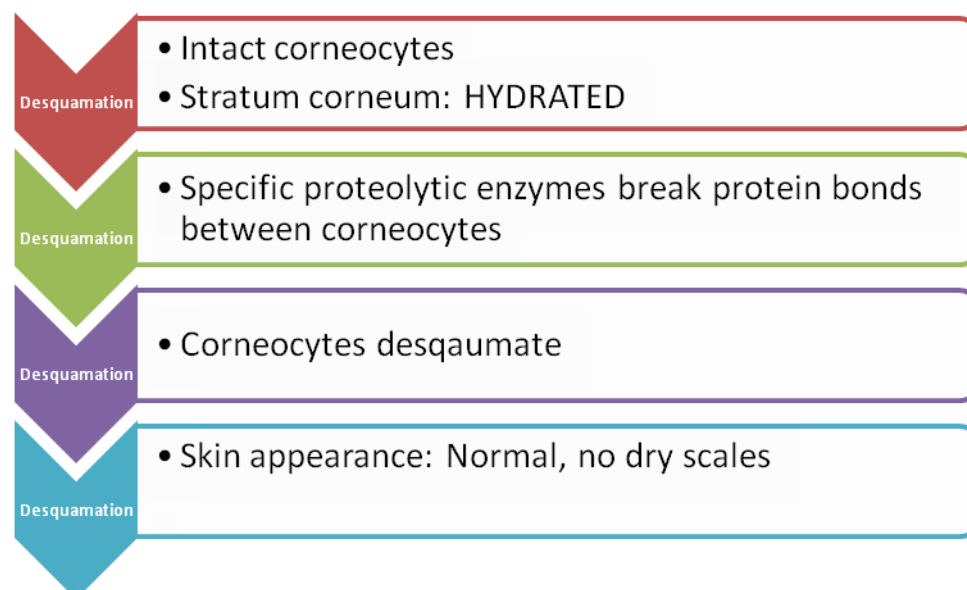


Figure 3: Process of Desquamation (adapted from Marino, 2006:2)

Skin disease often presents itself when the balance between the production of corneocytes and their shedding is altered. For example, increased corneocytes is associated with psoriasis and decreased shedding with ichthyosis (Marino, 2006:2).

### 2.2.4 Intracellular lipids

The intracellular lipids' main function as a skin barrier is to keep the skin supple, flexible and moist (Bouwstra *et al.*, 2003:1). These lipids surround the corneocytes in the stratum corneum and are stacked on one another. Three types of lipids are found in the skin, namely cholesterol, free fatty acids and sphingolipids. These lipids are responsible for trapping water among the corneocytes in the stratum corneum (Marino, 2006:2).

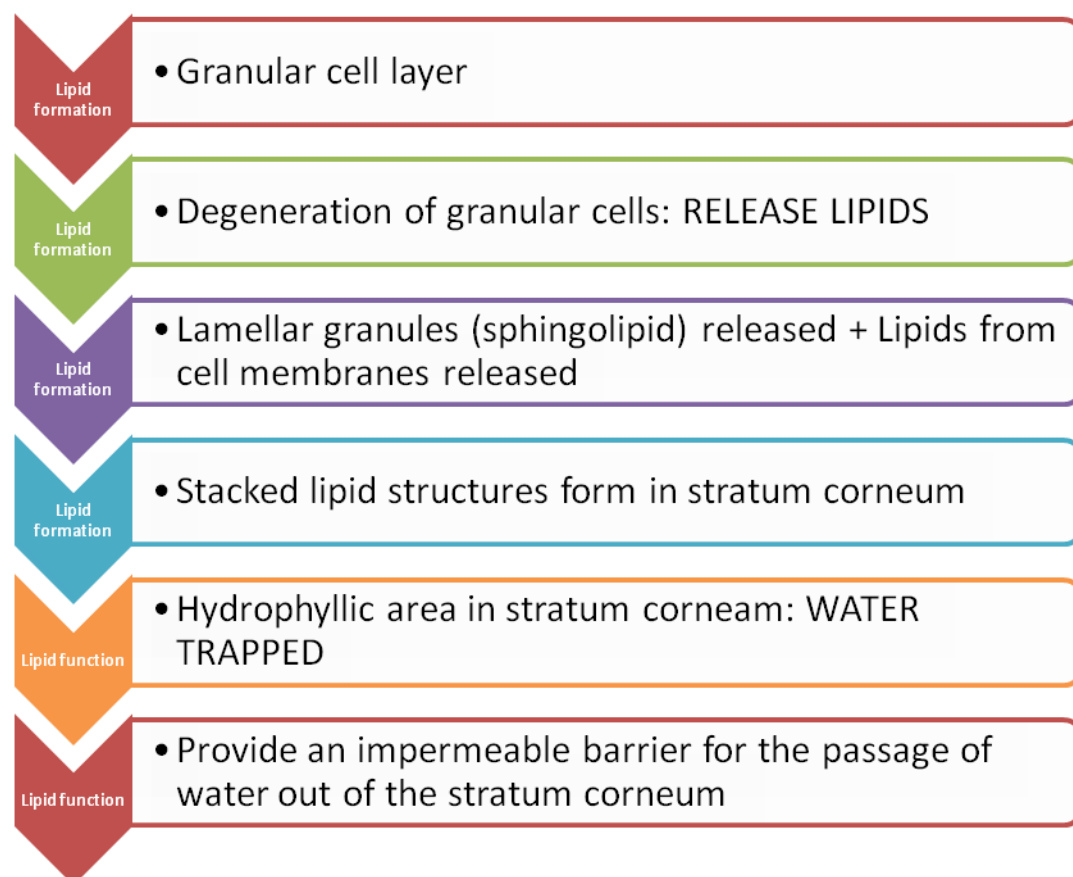


Figure 4: Lipids in the Skin (adapted from Marino, 2006:2)

## 2.3 FUNCTION OF THE SKIN

The skin is the largest organ of the body. It has a total surface area of around 1.8m<sup>2</sup> and weighs 16% of the total body weight. It varies in thickness, from 0.5mm on the eyelids to 4mm on the palms and the soles (Proksch, 2008:1063).

According to the National Health and Nutrition Examination Survey of the United States of America (2004:2), the skin has five mayor functions: protection, temperature regulation, sensation, vitamin D production, electrolyte and fluid regulation.

### **2.3.1 Protection**

The skin is a living, biological barrier that protects internal tissues from exposure to trauma, bacteria, extreme temperature fluctuations, water loss and ultraviolet radiation (Gawkrodger, 2002:2). The stratum corneum layer of the epidermis provides the most significant protection to the body. It contains keratinocytes which are arranged in a scaffold-like lattice, bound together by two different types of protein, namely keratohyalin and involucrin. The robust and waterproof properties of the skin are due to a lipid-rich matrix found in the intercellular spaces (Venus *et al.*, 2011:472).

The skin further provides the body with a first-line immunologic surveillance. Langerhans cells, found in the dermis of the skin, are bone-marrow derived cells which detect and destroy foreign antigens entering the skin (Shimizu, 2007:3).

### **2.3.2 Temperature Regulation**

According to the National Health and Nutrition Examination Survey of the United States of America (2004:2), the skin controls body temperature by dilation and constriction of capillaries in the skin. When the body temperature increases due to exercise or the environment, the capillaries dilate to allow more blood to reach the surface of the skin and allow heat to escape. When the body temperature decreases, the opposite occurs and capillaries constrict to prevent heat loss. The skin also controls temperature by sweat production which cools down the surface of the skin by evaporation. Hairs found on the skin also regulate temperature. When temperature decreases, hairs are erected, trapping air close to the surface of the skin, providing insulation and decreasing heat loss. The opposite occurs if one needs to cool down; hairs lie flat and prevent warm air from being trapped close to the skin.

### **2.3.3 Vitamin-D Production**

Keratinocytes in the epidermis produce Vitamin D from 7-dehydrocholesterol and sunlight (Bikle, 2011:80). Calcium absorption and production can only be maintained if sufficient amounts of Vitamin D are present. This will prevent rickets in children and osteoporosis and osteomalacia in adults. Vitamin D also plays an important role in normal neuromuscular function and anti-inflammatory functions (Pittas *et al.*, 2010:152).

### **2.3.4 Electrolyte and fluid regulation**

The skin, together with other regulatory organs such as the kidneys, regulates electrolyte and fluid homeostasis in the body. Sodium and Potassium are prominent ingredients in sweat and are, amongst others, excreted by the skin. The skin's elasticity and colour also provide us with an indication of the grade of dehydration (Roberts, 2001:369-391).

### **2.3.5 Sensation**

The skin allows sensory perception via the somatosensory system (Gawkrödger, 2002:2). It is connected to a variety of nerve endings that respond to touch, heat, cold, pressure, vibration and pain (Murray, 2000:11).

## **2.4 IMMUNOLOGY OF THE SKIN**

The skin is an important component of the immune system of the body. In many instances it is considered the first-line defence mechanism against pathogens. It produces anti-microbial peptides and activates epidermal T-cells to kill pathogens. The continuous shedding of keratinocytes also assists in preventing the growth of pathogens on the skin (Venus *et al.*, 2011:472).

There are four key cells in cutaneous immunology: keratinocytes, Langerhans cells, mast cells of the skin and dendritic cells. These cells have different immunological mechanisms to prevent pathogenic invasion of the skin. For example, dendritic cells

release cytokines TNF and IL-17 to diminish invading bacteria, whereas mast cells combat infections by releasing antimicrobial peptides (Metz *et al.*, 2009:687).

The skin defense mechanism can be divided into two categories: adaptive and non-adaptive immunity (De Leo *et al.*, 1998:2).

#### **2.4.1 Adaptive Immunity**

This type of immune defense mechanism is antigen-specific and is activated upon re-exposure of an antigen to the immune system. Langerhans cells play the most significant role in this defense system. These cells, which are tennis-racket shaped, are responsible for antigen processing and penetration. Langerhans cells have also been associated with processing and presenting antigens to T-cells in the epidermis (Quinn, 2004:2). Specificity and memory are both major features in the immunity of the skin. Other cells like migratory lymphocytes, keratinocytes and endothelial cells also play significant roles (De Leo *et al.*, 1998:2).

#### **2.4.2 Non-adaptive Immunity**

This barrier function of the skin prevents penetration of bacteria, protozoa, viruses and other pathogens. The stratum corneum is mainly responsible for this function. Its tough and rigid properties allow only water and other selective molecules to move through the epidermis (Venus *et al.*, 2011:472). Antibacterial antibodies are also secreted via the skin by sweat and sebum, thus preventing pathogen growth (De Leo *et al.*, 1998:3).

### **2.5 ALLERGIC SKIN REACTIONS**

Skin allergies can be divided into five categories: Type I, Type II, Type III, Type IV and Type V reaction.

#### **2.5.1 Type I Allergic Reaction**

This type of allergic reaction is described as an immediate response to an antigen. It has an onset of approximately 5 - 15 minutes after antigen exposure. Antigens

include pollen, food, drugs and insects. Mast cells, together with their mediators like histamine, prostaglandins and leucotriene, are responsible for this reaction. Anaphylaxis may occur during this type of allergy reaction. IgE antibody has been linked to allergic type I reactions. This results in urticaria and pruritic symptoms of the skin (Kay, 2000:843).

### **2.5.2 Type II Allergic Reaction**

This type of allergy is also described to have antibody dependant cytotoxicity. The antibodies IgG and IgM bind to antigens on the body's own cell surfaces and cytotoxicity by killer T-cells results. Autoimmune diseases are examples of Type II allergic reactions (Gaffahr, 2010:1).

### **2.5.3 Type III Allergic Reaction**

Immune-complex reactions are defined as Type III allergic reactions. This allergic reaction is induced between 4 - 10 hours after exposure. Antigen-antibody complexes that circulate the blood are deposited in vascular walls. Vascular damage is caused by platelet aggregation and lysosomes enzymes. This type of reaction is noticed in the event of systemic lupus and dermatomyositis (Gawkrödger, 2002:11).

### **2.5.4 Type IV Allergic Reaction**

This allergic reaction is described as cell mediated or delayed reaction. It has an onset of 48 - 72 hours. This reaction is mediated by T-cells and not antibodies. Allergic contact dermatitis is an example of this type of reaction (Lawlor *et al.*, 1995:1).

### **2.5.5 Type V Allergic reaction**

This type of allergic reaction is defined as autoimmune. The type of hypersensitivity is distinguished by the binding of the antibodies, not to the recognised cell surface but rather to its receptors. Hardly any literature clearly distinguishes the difference in hypersensitivity between Type II and Type V. Grave's disease and myasthenia gravis are examples of the Type V allergic reaction (Raja, 2003:376).

Table 1: Types of Allergic Reactions

Type of Allergy	Response	Examples of Dermatological Symptom or Disease	Mediators
Type I	Immediate	Urticaria & pruritus	IgE and IgG4
Type II	Cytotoxicity and anti-body dependant	Autoimmune diseases	IgM or IgG
Type III	Immune complex	Systemic lupus and dermatomyositis	IgG
Type IV	Delayed-type hypersensitivity, cell-mediated immune memory response and antibody-independent	Contact dermatitis	T-cells
Type V	Autoimmune disease, receptor mediated	Autoimmune diseases	IgM or IgG

## 2.6 SKIN LESIONS

Different terminology is used to describe skin lesions. This skin lesion classification is done according to the online Merck Manual of Diagnosis and Therapy for Healthcare Professionals (MacNeal, 2013).

### 2.6.1 Macule

Macules are less than 10mm in diameter and are not raised above the skin's surface. It is recognized by a colour and texture change of the skin. It is a flat lesion, which is non-palpable. Examples: freckles, flat moles, measles and rubella rashes and some allergy reactions.

### 2.6.2 Papule

Papules are smaller than 10mm and are elevated above the skin which can be palpated. Examples: lesions of acne, warts, lichen planus and some insect bites.

### **2.6.3 Nodule**

Nodules are bigger than 5mm and extended deep into the dermis and even subcutis layers of the skin. Examples: cysts, lipomas and fibromas.

### **2.6.4 Plaques**

Plaques are bigger than 10mm and elevated above the skin. This lesion is palpable and plateau-like. Example: psoriasis.

### **2.6.5 Vesicle**

Vesicles are smaller than 10mm. It is a small, blister-like vesicle filled with clear fluid. Examples: herpes infections and acute contact dermatitis.

### **2.6.6 Bullae**

Bullae are bigger than 10mm. It is a bigger blister-like vesicle filled with clear fluid. Examples: burns and acute contact dermatitis.

### **2.6.7 Pustules**

Pustules are lesions which are 2 – 5mm in size. They are elevated and contain pus. These types of lesions are common in bacterial infections. Examples: folliculitis and pustular psoriasis.

### **2.6.8 Urticaria**

Urticaria is described as localised oedema causing elevation in the skin. Skin lesions vary in size. Examples: hypersensitivity to drugs, insect bites and autoimmunity.



### **2.6.9 Ulcer**

An ulcer is characterised by loss of the epidermis and part of the dermis. Examples: physical trauma, infections and fasciitis.

### **2.6.10 Scale**

Scales are described as accumulations of horny epithelium. Examples: psoriasis, seborrheic dermatitis and fungal infections.

### **2.6.11 Crusts**

Crusts are dried serum, blood or pus. Examples: impetigo.

### **2.6.12 Erosions**

Erosions describe the loss of epidermis which causes open areas of skin. Examples: trauma as a result of rubbing and scratching.

### **2.6.13 Petechiae**

Petechiae are non-blanchable hemorrhage caused by platelet abnormalities, vasculitis and other types of infections. Example: rocky mountain spotted fever.

### **2.6.14 Purpura**

Purpura is characterised by large hemorrhage that can be palpable. Example: typhus and meningitis.

### **2.6.15 Atrophy**

Atrophy is the thinning of skin causing the appearance of cigarette paper skin. Examples: long-term use of corticosteroids, lupus erythematosus, chronic sun exposure and aging.

### **2.6.16 Scars**

Scars are described as fibrosis that replaces normal skin after injury.

### **2.6.17 Telangiectasia**

Telangiectasia is characterised by small, permanently dilated, blood vessel. Examples: long term use of fluorinated corticosteroids and diseases like rosacea and other systemic diseases.

## **2.7 DERMATOLOGICAL DISEASES**

### **2.7.1 Eczema (Dermatitis)**

According to a survey done in Johannesburg in 1999, eczema was found the most common dermatological disease accounting for one-third of all dermatological cases examined (Hartshorn *et al.*, 2003:1).

Eczema is commonly defined and described as a red, scaly and itchy rash (Hanifin *et al.*, 2004:391).

Dermatitis can be divided into many categories, such as atopic dermatitis, contact dermatitis, exfoliative dermatitis, hand and foot dermatitis, lichen simplex chronicus, nummular dermatitis, seborrheic dermatitis and status dermatitis (Porter, 2011:954).

#### **2.7.1.1 Atopic Dermatitis (AD)**

##### **Pathophysiology**

According to Kim (2013:1), two main hypotheses exist regarding the development of inflammation that results in AD. Immune dysfunction resulting in IgE sensitisation is described as the first hypothesis and epithelial-barrier disturbance as the second.

## **Aetiology**

Atopic eczema primarily affects children in urban areas. AD is commonly linked to pro-inflammatory T-cell immune response (McKoy, 2012). Eczema also has a definite genetic pre-disposition for hypersensitivity reactions (Van Hees, 2001:11).

## **Symptoms**

AD appears in the folds of elbows and knees, on the wrists and ankles and in the face and neck (Van Hees, 2001:11). The lesions are red, weeping, crusted and lichenification may occur if chronically present (Bershad, 2011:1). Pruritus and a stinging sensation are the primary symptom which may worsen if triggered by sweat, irritants or dryness of skin (Van Hees, 2001:11).

## **Treatment**

The patient should be warned against possible irritants and allergies that may cause dermatitis, such as soaps and moisturizers such as Vaseline®. The condition should be treated with mild corticosteroid like Hydrocortisone 1% twice daily and for more resistant or chronic dermatitis, treat with more potent corticosteroid such as betamethasone (Van Hees, 2001:11). An antihistamine should also be provided to limit pruritus. Nails should be cut short to prevent damage if skin is rubbed or scratched. Systemic corticosteroids may be supplied if patient does not respond to topical treatment. Tacrolimus can also be used as a corticosteroid substitute if no response is obtained from corticosteroid (McKoy, 2012).

### **2.7.1.2 Allergic Contact Dermatitis (ACD)**

## **Pathophysiology**

According to Gaspari (2013:1), ACD is defined as a delayed-type hypersensitivity reaction which is T-cell mediated. It is caused by reoccurring contact to a chemical substance of which an individual has previously been exposed to.

## **Aetiology**

The immune system is not activated in contact dermatitis. 80% of all cases are caused by contact with an irritant (McKoy, 2012). In Africa, we commonly see Vaseline® dermatitis as a result of excessive and repeated application of Vaseline®

(Van Hees, 2001:20). Other irritants such as petrol, diesel, cement, detergents and other industrial chemicals commonly cause contact dermatitis on hands or other exposed skin. ACD is common in the general population and is the most frequent occupational skin disease (Gaspari, 2013:1). According to Handa *et al.* (2011:700), contact dermatitis is also often caused by animal hairs, grass and pollen, which is described as airborne contact dermatitis.

## **Symptoms**

Pruritus is once again the primary symptom of contact dermatitis. Skin lesions, ranging from erythema to blistering and ulceration, are common (McKoy, 2012).

## **Treatment**

It is important to identify the irritant so that it can be avoided. Cool compresses, such as Burow's solution, will effectively manage pruritus. Anti-histamines can also be administered to reduce pruritus (McKoy, 2012).

A mild corticosteroid such as 1% Hydrocortisone should be effective. Second-line treatment includes combining Betamethasone 0.1% and an oral corticosteroid (McKoy, 2012).

### **2.7.1.3 Lichen Simplex Chronicus (LSC)**

#### **Pathophysiology**

The pathophysiology of LSC is not defined. LSC is caused by habitual repetitive scratching or rubbing of the skin (Shimizu, 2007:103). According to Janjou (2006:60), a likely relationship exists between central and peripheral neural tissue, and inflammatory cell response which effects the perception of itch. He also states that interplay may be present among primary lesions, psychic factors, and the intensity of the pruritus.

#### **Aetiology**

Lichen Simplex Chronicus is a chronic type of eczema which is created by repeated scratching or rubbing of the skin (Shimizu, 2007:103). Darker skinned people are especially susceptible (Gawkrodger, 2002:35). This type of eczema is often seen with female patients that suffer from stress or anxiety disorders. Insect bites,

traumatic and post-herpetic scars, acne keloidalis nuchae, xerosis, venous insufficiency and asteototic eczema also form part of this aetiology (Janjou, 2006:60).

### **Symptoms**

Pruritus is the main symptom which is due to the amyloid formation. Plaques are irregular, oval or angular shaped. Lesions are present on easy-to-reach areas such as arms, legs or genital area (McKoy, 2012). Hyper-pigmentation on lichenified areas is common (Janjou, 2006:60). Variable scaling and skin thickening can also occur.

### **Treatment**

Topical corticosteroids can be applied twelve-hourly and oral anti-histamine may be administered to control pruritus (McKoy, 2012). Informing patients of the effects of the rubbing and scratching is also very important. Secondary topical infections can be treated with antibiotics (Janjou, 2006:62).

#### **2.7.1.4 Seborrheic Dermatitis**

##### **Aetiology and Pathophysiology**

The skin inflammation is localised to the areas where sebaceous glands are present (McKoy, 2012). HIV-infected persons and Parkinson's patients commonly develop wide spread seborrheic dermatitis which occasionally becomes a super infection. The pathogenesis of the disease remains controversial (Stefanaki *et al.*, 2010:1).

##### **Symptoms**

Greasy, red scales on sebaceous gland areas, including the face, scalp, back and neck, are common. Pruritus is also common in most cases (Stefanaki, 2010:1). Dandruff may also occur and in severe cases, papules which are yellow-red and found along the hairline (McKoy, 2012).

##### **Treatment**

Selenium sulphide or tar shampoo should be used daily (McKoy, 2012). Imidazole derivatives such as ketoconazole cream should be used daily. Ketoconazole shampoo can be used weekly (Stefanaki *et al.*, 2010:1). Sulphur and salicylic acid

mixtures will decrease scaling. Anti-infective shampoos such as betadine or potassium permanganate solutions are used for widespread lesions (Van Hees, 2001:16).

## **2.7.2 Psoriasis**

### **Pathophysiology**

Psoriasis is a chronic, autoimmune, non-infectious, inflammatory skin disorder, characterised by silvery scales and erythematous plaques (Gelfand *et al.*, 2005:23). Psoriasis is commonly described as a hyper proliferation of epidermal keratinocytes accompanied by inflammation of the epidermis and dermis (Traup *et al.*, 2007:319). According to Traup and Marchall (2007:320), a debate exists whether psoriasis is an autoimmune disorder or a T-helper 1 immune dysfunction. Lowes *et al.* (2007:866), explains that the keratinocytes response is due to the combination in the activation of T-cells, dendritic cells and immune-related cytokines and chemokines.

### **Aetiology**

The aetiology is unknown, but factors such as trauma, infection and certain drugs like beta-blockers, lithium and antimalarias, definitely contribute to its severity (Johnston, 2013:166). Other precipitating factors include the Koebner phenomena, where scar tissue due to operation or injury causes psoriasis (Gawkrodger, 2002:26). A study done on the prevalence of psoriasis on African Americans concludes that Caucasian patients are more at risk of having psoriasis than black patients (Gelfand *et al.*, 2005:23 - 24). Five per cent of patients that are diagnosed with psoriasis also have joint disease. According to Gawkrodger (2002:27), a common trend exists between arthritis and psoriasis.

### **Types of Psoriasis**

Psoriasis can be found in many different forms and types, each with its own characteristics. Usually, more than one form of psoriasis is present in a single patient, although it can also present itself in isolation (Leask *et al.*, 2009:1).

- **Plaque Psoriasis:** The most common type of psoriasis is psoriasis vulgaris. It presents itself as red erythematous plaques with white-

silvery scales. These scales are often itchy and irritating to the patient (Leask *et al.*, 2009:1).

- Guttate Psoriasis: Small, drop-like lesions on the limbs and trunk, are commonly found on children and young adults after a streptococcal infection (Naldi *et al.*, 2001:433).
- Flexural Psoriasis: Also known as inverse or intertriginous psoriasis. Smooth plaques are found on the axillae, natal cleft and sub-mammary areas and more common in the elderly. These plaques are not scaly (Leask *et al.*, 2009:10).
- Pustular Psoriasis: Also known as Palmoplantara pestulosis. Yellowish-brown sterile pustules are found on palms and soles (Leask *et al.*, 2009:2). This type of psoriasis is commonly found in women who smoke (Gawkrodger, 2002:27).
- Scalp Psoriasis: Commonly confused with dandruff, but scales are normally thicker. 50 - 75% of people affected by plaque psoriasis develop scalp psoriasis. It is characterised by single or multiple pruritic, erythematous, flaky scales (Guenther, 2009:1).
- Nail Psoriasis: 20 – 30% of patients with psoriasis vulgaris will suffer from psoriasis of the nails. Symptoms include subungual hyperkeratosis, pitting of the nails, yellow discoloration, thickening of nail plates and, eventually, onycholysis (Yaghoobi *et al.*, 2010:32).

The severity of psoriasis plays an important role with regard to treatment. Normally, psoriasis is classified as follows:

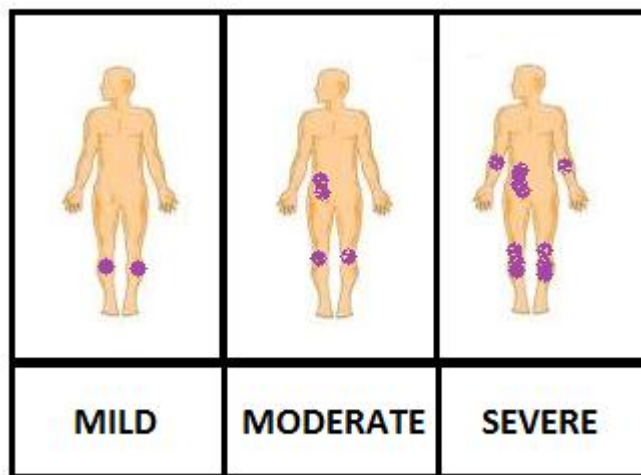


Figure 5: Areas on the body most likely affected by psoriasis (adapted from the National Psoriasis Foundation website).

According to the National Foundation of Psoriasis of the United States of America website, a mild case of psoriasis occurs when the total surface area affected by psoriasis is less than 3%; a moderate case will be between 3 - 10% and a severe case will be more than 10%.

## Symptoms

Lesions are mildly pruritic and often found on the scalp, elbows, knees and buttocks. Psoriasis is commonly known to produce oval erythematous pustules, covered with thick, white-silvery scales (Leask *et al.*, 2009:1).

## Treatment

Psoriasis treatment can generally be divided into three categories, namely topical treatment, systemic treatment and light therapy (Schalock, 2012).

Topical treatment: Corticosteroid is the most common and widely used treatment for psoriasis (Leask *et al.*, 2009:19). Different emollients are used to reduce the scaling of the dermis and epidermis. Salicylic acid is a softening agent which will reduce the



hardness of the scales. Coal tar ointments and solutions are not only anti-inflammatory but decrease keratinocyte hyper proliferation (Schalock, 2012). Calcipotriol ensures normal keratinocyte proliferation by inhibiting T-cell activity (Leask *et al.*, 2009:19).

Systemic treatment: Methotrexate and cyclosporine are the most common and effective means of treatment in severe psoriasis. Systemic retinoid, such as acitretin and isotretinoin, is also effective (Schalock, 2012).

Light Therapy: UV light therapy is used in very severe and extensive psoriasis. The mechanism is unknown, but research has shown that UVB light decreases DNA synthesis (Leask *et al.*, 2009:32).

Treatment during pregnancy and breastfeeding should be carefully monitored since some treatments may cause foetal complications. Drugs to avoid include methotrexate, cyclosporine, oral corticosteroids and retinoids. A study done on pregnant women suffering from psoriasis indicated that about 55% of them experienced improvement of psoriasis during pregnancy (Mowad *et al.*, 1998:257).

### **2.7.3 Fungal Infections**

Fungal infections of the skin are caused by two types of fungi: dermatophyte which is multi-cellular and yeast which is unicellular (Gawkrodger 2002:54).

#### **2.7.3.1 *Tinea Corporis***

*Tinea corporis* is a dermatophyte infection of the body. It is commonly known as body ringworm. Usually, it presents itself in one or two lesions which are easily treatable with a topical anti-fungal; however, occasionally, multiple, resistant lesions are found widespread over the body. In such cases systemic treatment is necessary (Gupta *et al.*, 2003:395).

## **Pathophysiology**

Dermatophyte, causing *Tinea corporis*, usually inhabits dead areas on the skin, including nails, hairs and cornified skin (Leschner, 2012:2). Spores of the fungus will attach themselves to the skin and germinate, producing hyphae. The hyphae then develop and start to spread to the upper layers of the skin. Some fungi release enzymes like keratinases which penetrate deep into the stratum corneum (Brannon, 2010:1). These enzymatic secretions damage the skin. The body reacts by shedding the infected skin, leaving new healthy skin in the central of the lesion and inflammation at the edge. The scaling occurs due to an increased epidermal cell proliferation. Elimination of dermatophytes is eventually achieved by cell-mediated immunity (Leschner, 2012:2).

## **Aetiology**

*Tinea corporis* is caused by dermatophytes *T.mentagrophytes*, *T.rubrum* and *M.canis* (Gupta *et al.*, 2003:395). These fungi survive and thrive in dead keratin in the upper part of the epidermis (Brannon, 2010:1).

## **Symptoms**

*Tinea corporis* typically presents themselves as round, red lesions that expand peripherally and that tend to clear in the centre of the lesion (Aaron, 2013). These lesions enlarge slowly (Gawkrodger, 2002:54). Pruritus is a common symptom which can be accompanied by pain if a secondary infection is present (Gupta *et al.*, 2003:396).

## **Treatment**

For single lesions, topical azoles (ketokonazole, klotrimazole and miconazole) and allylamines (terbinafine and naftifine) are most effective. Treatment needs to be administered for a minimum of 4 weeks and at least 7 days after the lesion has disappeared (Berman, 2008).

For multiple widespread lesions in adults, griseofulvin 500mg is given once daily for 6 weeks. In children, griseofulvin is given at 10mg/kg for 6 weeks. Other treatments for adults include Intraconazole 200mg once daily and flucanazole 200mg weekly (Goldstein, 2013).

### **2.7.3.2 Tinea Capitis**

*Tinea capitis* is a dermatophyte infection of the scalp. The fungus grows in the hair follicle which often causes follicular pustules, resulting in massive purulent secretions. It commonly infects children (Dayle *et al.*, 2004:23). It is more often seen in young boys than girls, but more in adult women than men (Freedberg & Fitzpatrick, 2003:645).

#### **Pathophysiology**

The pathophysiology of *tinea capitis* is the same as *tinea corporis*. The only difference is that the hyphae affect hair follicles and not superficial skin. (Leschner, 2012:2).

#### **Aetiology**

*Tinea capitis* is caused by dermatophytes *M.canis*, *M. audouinii*, *T. schoenleinii*, *T. violaceum* and *T. tonsurans* (Aaron, 2013). In Namibia we often see *Tinea capitis* infection after a child's hair was shaved at the local street barber. These infections are almost always associated with bacterial super infections and continuous re-infection causes scarring in some areas of the scalp.

#### **Symptoms**

Round, dry patches on the scalp with alopecia are the most common symptoms. The term black-dot ringworm is also often used due to *T. tonsurans* infection that causes the hair shafts to break at the scalp's surface, leaving a small, black dot (Aaron, 2013). Occasionally, bacterial super infection manifests itself with purulent lesions and crusting (Van Hees, 2001:25).

#### **Treatment**

Systemic and topical treatment should be administered simultaneously: Griseofulvin 500mg once daily for 12 weeks in adults and 10mg/kg once daily for 12 weeks in children. Selenium sulfide 2.5%, ketoconazole and povidone-iodine shampoos should be used twice a week (Dayle *et al.*, 2004:25). Whitfield's ointment, terbinafine

or ketaconazole cream applied twice daily should be administered (Van Hees 2001:25).

#### **2.7.3.4 *Tinea Unguium***

##### **Pathophysiology**

The pathophysiology of *tinea unguium* is the same as for the other tinea infections. The only difference is that the hyphae affect nails and not superficial skin or hair follicles (Leschner, 2012:2).

Onychomycosis is a common fungal infection that affects mostly adults and the elderly. Toe nails are more commonly affected than finger nails (Gawkrodger, 2002:64).

##### **Aetiology**

*Tinea unguium* is commonly seen in patients that frequently wet their hands, like domestic workers and cleaners (Van Hees, 2001:26). Other contributing factors include poor hygiene, shoes that fit too tightly and beauty salon equipment that are not sterilized effectively. In Namibia, we often see *Tinea unguium* infections in farm workers that wear tight, leather or rubber shoes. Sweat and humid conditions in the shoe together with nail injury, due to tight shoes, are the main causes of the infection. *Tinea pedis* is often also present.

##### **Symptoms**

The nail bed undergoes onycholysis and becomes thick, yellow and crumbly. The process usually starts at the distal nail edge and slowly extends to the whole nail. Often chronic paronychia exists on the skin around the nail (Gawkrodger, 2002:64).

##### **Treatment**

Onychomycosis is normally asymptomatic. Treatment is administered for 4 - 9 months depending on the extent of the infection. Griseofulvin 500mg once daily, together with topical antifungal terbinafine, is given (Goldstein, 2013).

#### **2.7.3.5 *Tinea Pedis***

## **Pathophysiology**

The pathophysiology of *tinea pedis* is identical to the other tinea infections.

Athlete's Foot is common in adults, especially in young men. Fifty per cent of all diagnosed tinea cases are *tinea pedis* infections (Crawford *et al.*, 2001:288).

## **Aetiology**

*Tinea pedis* is commonly caused by *T.rubrum*, *T.mentagrophytes* and *E.floccasum*. Other contributing factors that facilitate the fungus growth are wet, warm weather, sharing of clothes (especially shoes and socks) and communal bathing (Gawkrodger, 2002:54). Secondary bacterial infections, cellulites and lymphangitis are common complications (Aaron, 2013). Patients who suffer from diabetes are especially at risk (Crawford *et al.*, 2001:288).

## **Symptoms**

Athlete's Foot causes white scaling lesions, commonly found between the 4<sup>th</sup> and 5<sup>th</sup> toe. Pruritus and irritation are common (Van Hees, 2001:26).

## **Treatment**

Feet need to be kept dry by using powders and wearing open-toe shoes or sandals. Socks should be changed every day. Medicated foot powders (miconazole and tolnaftate) will help keep the feet dry (Cole, 2013). Allylamines, such as terbinafine, are further recommended. Terbinafine should be applied twice daily for 2 weeks. For extensive infections, oral itraconazole 200mg once daily for 2 – 6 weeks should be administered (Aaron, 2013).

### **2.7.3.6 Pityriasis versicolor**

## **Pathophysiology**

*M.furfur* is part of the normal skin flora. It is described as lipophilic, gram-positive, saprophytic, oval-shaped, yeast. *Malassezia* yeasts do not invade the skin deeper than the cornified epithelium. The yeasts live on free fatty acids which are available in the stratum corneum. They then hydrolyse the triglycerides into free fatty acids. The body reacts by producing a cell-mediated response and activation of the

alternative complement pathway. This causes inflammation in the upper part of the skin (Buckhart, 2012:2).

### **Aetiology**

*Pityriasis versicolor* is a chronic, superficial fungal infection which is caused by a yeast *M.furfur*. This fungal yeast is resident in most of adults and causes infection predominantly when conditions are favourable, like in the rainy, humid season (Shimizu, 2007:25C1).

### **Symptoms**

The lesions are scaly macules that vary in colour from red, yellow, brown to black. Patches are around 1 - 2.5cm in diameter, appearing mostly on the chest and back of young adults. These patches may increase in size if untreated. Scaling and mild pruritus may occur. Re-infection is common (Zenab *et al.*, 2004:36).

### **Treatment**

Topical treatment with Selenium sulphide shampoo, applied as a lotion and left overnight, is effective. Imidazole cream applied twice daily for 2 weeks has shown positive results as well. For more extensive infections, systemic itraconazole 200mg once daily should be administered for 7 days (Goldstein, 2013).

### **2.7.3.7 Candidiasis**

#### **Pathophysiology**

The pathophysiology of *Candida albicans* is very complex (Uwamahoro *et al.*, 2010:16). Candidiasis is common yeast that normally resides on the mucous membranes of the body. An infection occurs only when conditions become favourable for excessive fungal growth (Van Hees, 2001:29). *Candida albicans* is a remarkable pathogen which has the ability to change morphologically. It infects its host either as yeast cells or filaments, or both (Uwamahoro *et al.*, 2010:16). This dimorphism can be achieved by changes observed in the environment such as temperature or pH (Molero *et al.*, 1998:95).

## **Aetiology**

*Candida albicans* is the main causative agent, although 10 other candida species are also found on humans (Shimizu, 2007:25B1). These fungi commonly reside in axillae, groin and gluteal folds, beneath the breast, glans of penis, vulva and in between the toes (Aaron, 2013). Predisposing factors include pregnancy, poor hygiene, HIV/AIDS, diabetes mellitus, antibiotic treatment, humid environment and occupation (Gawkrodger, 2002:55).

According to a study done in Iraq over a period of two years (2003-2005), it was found that 40% of the patients diagnosed with candidiases were between the ages of 12-21 (Hassan *et al.*, 2008:242). This same study also indicated that women (65%) were more likely to get cutaneous candidiases than men (35%) (Hassan *et al.*, 2008:243).

## **Symptoms**

Candidiasis presents itself on the skin as erythema patches with small pustules. It can cause intensive itchiness and even pain (Van Hees, 2001:29). Perianal candidiasis causes a white discharge with severe pruritus. Oropharyngeal candidiasis causes white plaques on the mucous membranes. Candidiasis of the nails causes a yellow, thickening of the nail with onychomycosis (Aaron, 2013).

## **Treatment**

Topical imidazoles can be applied to infected areas daily for 7 days. For nappy rash, nystacid® cream mixed with zinc oxide is effective as treatment. Oral nystatin is given for oropharyngeal candidiasis commonly seen in children (Van Hees, 2001:29).

For more resistant and extensive infections, fluconazole 150mg should be administered for 4 weeks. This dosage is increased to 100mg daily for 2-3 weeks to treat onychomycosis of the nail bed (Aaron, 2013).

## **2.7.4 Viral Infections**

Unlike yeasts and fungi, viruses do not normally reside on the body (Gawkrodger, 2002:48). Many viruses infect the body systemically and then present lesions on the skin (Dinulos, 2012).

#### **2.7.4.1 *Molluscum Contagiosum* (MCV)**

##### **Pathophysiology**

*Molluscum contagiosum* is a self-limiting, harmless infection of the poxvirus, except if a patient immune-suppressed (Sisneros, 2010:169).

According to Smith & Skelton (2002:538), the MCV virus proliferates within the follicular epithelium. It replicates within the cytoplasm of the CD34+ stromal cells where internal organelles are dislocated and suspended in intracytoplasmic matter. This lesion easily ruptures, allowing the MCV infection to spread (Smith & Skelton, 2002:538).

##### **Aetiology**

The poxvirus causing *Molluscum contagiosum* is transmitted by contact and affects mostly children. If infection is contracted by adults, it mostly infects the genital areas (Dinulos, 2012). In healthy adults, the lesions will clear within 2 months (Sisneros, 2010:170); however, they can also remain for 2-3 years (Dinulos, 2012).

##### **Symptoms**

Lesions are dome-shaped papules, with a white, waxy-looking centre (Van Hees, 2001:60). Lesions are either single papules or smooth clusters which can be found anywhere on the body, except the palms and the soles (Dinulos, 2012).

##### **Treatment**

Normally, these lesions are not treated since they spontaneously clear after a few months. If they persist, they can be removed by puncturing the lesions and clearing the waxy, white substance from the centre. The lesion is then cleaned and scrubbed with Iodine solution (Van Hees, 2001:60). Cryotherapy is another common treatment



done usually with pressurised, liquid nitrogen or nitrous oxide (Dinulos, 2012). Imiquimod cream, which is an immune stimulant, can be used (Hanna *et al.*, 2010:575). Paclitaxel, a chemotherapy drug, has shown to be successful in treating *Molluscum Contagiosum*, especially in HIV patients (Sisneros, 2010:16).

### **2.7.5.2 Herpes Simplex**

#### **Pathophysiology**

According to Mark *et al.* (2008:1141), the *Herpes Simplex* virus is defined as a double-stranded DNA virus. It has three unique characteristics, namely neurovirulence, latency in nerve cell ganglion and the reactivation of latent virus. It is the most common viral infection of the skin. Normally, it presents itself as cold sores on the lips (Van Hees, 2001:50).

#### **Aetiology**

*Herpes Simplex* virus I and II that infect humans, belong to the *Herpes-viridae* family. *Herpes Simplex* virus I affects general areas such as the lips, mouth and face. It causes herpes labialis, herpes gingivostomatitis and Kaposi's varicelliform eruption (Shimizu, 2007:23A1). It is spread through contact with an infected person.

*Herpes Simplex* II is a sexually transmitted disease which produces genital sores. The virus produces watery lesions on the skin of the mouth, lips and genital areas. The virus spreads through contact with an infected person (Ryan *et al.*, 2004:556). It is highly contagious and can spread without showing any visible symptoms (Van Hees, 2001:50).

#### **Symptoms**

Watery blisters are normally found on lips, inside of mouth and genitalia (Ryan *et al.*, 2004: 556). A burning sensation on the skin is often experienced before the lesions appear. The patient normally also has a fever, malaise and no appetite. *Herpes Simplex Virus* II is sexually transmitted and care should be taken to prevent further transmission from one partner to another (Van Hees, 2001:50).

### **Treatment**

Acyclovir therapy remains the most effective and least expensive treatment option. For infection of the lips, acyclovir should be applied five times per day for at least 5 days. For more serious systemic infections, including genital herpes, acyclovir 200 - 400mg five times per day, or famciclovir 1g daily and valaciclovir 250mg twice daily, are the preferred treatment (Emmert, 2000: 1697).

For *Herpes Simplex* infection of the lips, patients must also protect infected areas from the sun and use chlorhexidine mouthwash to prevent bacterial super infections (Van Hees, 2001:50).

For *Herpes Simplex* infection of the genitals, patients must sitz-bath in potassium permanganate solutions or wash with iodine daily to prevent bacterial super infections (Van Hees, 2001:50).

### **2.7.5.3 Varicella Zoster**

#### **Pathophysiology**

According to Steiner (2007:1015), *Varicella Zoster* is a double-strained DNA virus which relates to the *Herpes Simplex* virus. In 1892, Bokay was first to suggested a relationship between *Varicella* and *Herpes Zoster*. His theory was confirmed when it was observed that children who had been exposed to adults with *Herpes Zoster*, developed chickenpox. It was concluded that *Herpes Zoster* was a reactivation of a latent *Varicella* virus in the dorsal root ganglia (Arvin, 1996:361).

#### **Aetiology**

The *Varicella Zoster* virus is responsible for two diseases: *Varicella*, also known as chickenpox, and *Herpes Zoster* which is also known as shingles (Arvin, 1996:361). *Varicella* is common in children and highly contagious (Van Hees, 2001:47).

#### **Symptoms**

In chickenpox, children develop red macules that are scattered across the body. Fever, malaise and severe pruritus of the lesions develop (Tynan *et al.*, 2012:212).

The pruritus is the main cause for bacterial super infection. Other complications include pneumonia, encephalitis, Ramsey-Hunt Syndrome and Reye's syndrome (Shimizu, 2007:23C1). In shingles, adults develop itchy papules that quickly change to blisters. The lesions are localised and found on one side of the body only. Fever, pain and irritation often accompany these lesions (Tynan *et al.*, 2012:212).

## **Treatment**

*Varicella* is treated with analgesics, such as ibuprofen and paracetamol. Calamine lotion is often applied to sooth pruritus and anti-histamine may be given. For immune compromised patients, treatment includes acyclovir 200 - 800mg five times per day for 5 - 10 days (Tynan *et al.*, 2012:212). Antibiotic and antiseptic creams can be applied to lesions with super infections (Shimizu, 2007:23A1). In *Herpes Zoster*, treatment is symptomatic with analgesics and agents to decrease itching. In severe cases, acyclovir, valaciclovir and famciclovir should be administered within 24 hours. Immune compromised patients often require intravenous acyclovir (Gawkrödger, 2002:51). Pregabalin is used for pain management (Niv *et al.*, 2005:357).

### **2.7.5.4 Warts**

#### **Pathophysiology**

Warts are caused by the Human *Papilloma* Virus or HPV (Van Hees, 2001:55). The HPV is described as a non-enveloped, double-stranded DNA virus. The genome can encode so-called late and early structural genes that enable viral transcription and replication (Faridi *et al.*, 2011:269). Different types of warts, including common warts, genital warts and plantar warts can result due to a *papilloma* viral infection. A recent study also indicated a possible correlation between people infected with HPV and cardiovascular diseases (Kue *et al.*, 2011:2001).

- **Common warts (*Verruca Vulgaris*)**

#### **Aetiology**

*Verruca vulgaris* is mostly caused by HPV Type 2, 4 and 7 (Sun *et al.*, 2010:579). Infection is due to direct contact with a person infected with the

virus or via auto-inoculation. Normally, no treatment is necessary and the warts will clear within 2 years (Van Hees, 2001:55).

### **Symptoms**

Common warts can be described as flesh-coloured cauliflower-like lesions that occur mostly on hands, feet and knees or anywhere on the body after injury (Marks *et al.*, 1999:20). Lesions usually start out small and then increase to around 5mm in diameter. They can either be found in clusters or as sole lesions (Shimizu, 2007:452).

### **Treatment**

Common treatment includes liquid nitrogen cryotherapy, topical glutaraldehyde application, carbon dioxide gas laser therapy and electrosurgery (Shimizu, 2007:452). Keratolytic therapy with salicylic acid 25% twice a week can be used to remove warts safely (Kwok, 2012).

- **Genital warts (*Condylomata Acuminata*)**

### **Aetiology**

HPV is the most common sexually transmitted disease (Sol dos Ramos Fariasa *et al.*, 2010:96). *Condylomata acuminata* is mostly caused by HPV Type 6 and 11 (Shimizu, 2007:453). It spreads through sexual intercourse and sometimes through infected hands. Transmission can also take place between mother and child during birth (Veldhuijzen *et al.*, 2010:862).

### **Symptoms**

Warts are cauliflower-like lesions that affect the penis, vulva and perianal regions. Warts differ in size (Lacy *et al.*, 2012:1). Genital warts in pregnant woman may show acceleration in growth (Veldhuijzen *et al.*, 2010:862).

### **Treatment**

The traditional treatment for genital warts includes podophyllin 10% - 25% solution applied to the lesions and then washed off after 6 hours. Imiquimod is expensive but very effective treatment for genital warts (Van Hees, 2001:58).

Liquid nitrogen cryotherapy and surgical removal using electrical scalpel or carbon gas laser are also effective (Lacy *et al.*, 2012:1).

- **Plantar warts (*Myrmecia*)**

### **Aetiology**

*Myrmecia* is caused by HPV Type 1 (Shimizu, 2007:453). These warts grow on the soles of feet of adults and children. Because of the pressure of standing, they grow deep into the dermis (Gawkrodger, 2002:48).

### **Symptoms**

Normally, they are asymptomatic but may cause pain and discomfort (James, 2006).

### **Treatment**

Keratolytic therapy with salicylic acid 50% should be applied at night, covered with plastic and left until the morning after which the wart is scraped. The treatment is repeated daily (Van Hees, 2001:56). Warts can also be surgically removed (James, 2006).

## **2.7.5 Pityriasis Rosea**

### **Aetiology**

*Pityriasis rosea* is a common, self-limited, inflammatory skin disease that affects young adults and adolescents (Neoh *et al.*, 2009:300). *Pityriasis rosea* may be associated with the reactivation of human herpes virus 7 and rarely, herpes virus 6 (Drago *et al.*, 1997:1367). However, according to some researchers, these studies are inconclusive (Neoh *et al.*, 2009:300). The exact pathogenesis is not yet known.

### **Symptoms**

The skin lesions associated with Pityriasis Roseas are often described as a herald patch or mother patch. They are characterised by an acute eruption of erythematous

papulo-squamous lesions. Other symptoms experienced include fever, headache, arthralgia or malaise (Neoh *et al.*, 2009:300). The Christmas-tree pattern of lesions is commonly seen on the back of infected patients (Van Hees, 2001:46).

## **Treatment**

Normally, patients do not require any treatment (Drago *et al.*, 1997:1367). It is, however, important to ensure that the lesions are not secondary syphilis which presents themselves quite similarly (Van Hees, 2001:46). Some patients need treatment because of their extensive lesions or due to excessive pruritus. Antihistamines are administered and calamine lotion can be applied to the lesions (Habif, 2004:246).

### **2.7.6 Kaposi's Sarcoma**

#### **Aetiology and Pathophysiology**

Kaposi's sarcoma is a tumour caused by the Human Herpes Virus 8 (Kaposi *et al.*, 1872:265). Moritz Kaposi was the first dermatologist ever to document the condition. Kaposi's sarcoma was then known as a slow-growing malignancy seen in elderly men (Sternbach *et al.*, 1995:671). In the 1980's, Kaposi's sarcoma was linked to HIV/AIDS. It was described as a serious and sometimes fatal skin disorder (Aversaa *et al.*, 2005:253). Kaposi's sarcoma has been found more frequently in HIV positive homosexual men than HIV patients not associated with homosexuality (Schwartz *et al.*, 2008:196).

The pathophysiology of the Human Herpes Virus 8 is complicated by many different complex mechanisms. The most common being the virus that produces molecules critical in the transduction of signals that stimulate cell proliferation and inhibit apoptosis in cells (Sullivan *et al.*, 2008:1209).

## **Symptoms**

The lesions initially develop pink pustule eruptions which change into plaques and nodules (Aversaa *et al.*, 2005:253). Purple-black nodules appear mostly on thighs, but also on face, genitalia, trunk and proximal limbs. When palpated, the lesions feel

hard. Lesions can also be found inside the mouth, especially on the palate and tonsils (Van Hees, 2001:52).

### **Treatment**

Treatment depends on patient HIV viral load and the extent of tumour growth (Van Hees, 2001:52). Radiotherapy has become the most important type of treatment for Kaposi's sarcoma. Intralesional injections of vinblastine, vincristine and interferon-alpha have been reported to be effective (Aversaa *et al.*, 2005:253). Chemotherapy is given to the extensively affected patients (Van Hees, 2001:52).

A study done in 2008 by the New Jersey Medical School found that quinine, the drug used to treat malaria, may serve as an activating agent, changing human Herpes Virus 8 from its latent form to a lytic form which facilitates the process by which Kaposi's sarcoma is formed by the body (Ruocco *et al.*, 2009:436).

### **2.7.7 Bacterial Infections**

Normal, healthy skin has harmless micro-organisms residing on it, including some species of staphylococci. It is only when the skin is broken or the immune system is de-activated that infections occur (Gawkrodger, 2002:44).

#### **2.7.7.1 Impetigo**

##### **Aetiology**

Atopic dermatitis often produces skin lesions which are damaged through scratching. These damaged lesions are very susceptible to micro-organism and thus infections caused by *Staphylococcus aureus* (Adachi *et al.*, 1998:45). Impetigo is highly contagious and mostly infects children. Poor hygiene, hot, humid climate and overcrowding are the main risk factors of the disease (Razmjou *et al.*, 2009:590). Atopic eczema, scabies, psoriasis or even lice infestations can all become infected with *Staphylococcus aureus* and cause impetigo (Gawkrodger, 2002:44).

##### **Pathophysiology**

According to Cole and Gazewood (2007:859), impetigo can only occur if the skin is damaged, exposing epithelial cell receptors called fibronectin. Bacteria like *Staphylococcus aureus* will invade the area where the fibronectin is made available, causing infection.

### **Symptoms**

Superficial pustules or blisters develop which quickly become ruptured erosions with yellow-crusts exudates. Due to this typical appearance impetigo is effortlessly diagnosed (Adachi *et al.*, 1998:46).

### **Treatment**

In adults, impetigo is treated with antibiotics such as cloxacillin 250 - 500mg four times per day for 7 - 10 days. In children, erythromycin 250 - 500mg four times per day also for 7 - 10 days. (Van Hees, 2001:31). Lesions may also be treated with mupirocin ointment applied twice daily (Stulberg *et al.*, 2002:109). Fucidic acid and bacitracin ointments can also be used as topical treatment (Gawkrodger, 2002:44).

#### **2.7.7.2 Cellulitis**

### **Aetiology and Pathophysiology**

Cellulitis is caused by an infection of the subcutaneous tissue (Gawkrodger, 2002:44). It can be caused by normal skin flora or by exogenous bacteria. The bacteria gains entry via a cut or wound in the skin or a microscopic portal of entry. Cellulitis can also occur secondarily after a folliculitis, tinea infection, lymphatic obstruction or obesity (Scheinfeld, 2003:8).

Cellulitis is mostly caused by *Staphylococcus pyogenes*, *Staphylococcus aureus*, but *Group-A  $\beta$ -hemolytic Streptococcus* and *Haemophilus influenzae* are also present at times (Shimizu, 2007:453).

### **Symptoms**

Swelling, redness and localised pain are the main symptoms (Gawkrodger, 2002:47). Other symptoms include fever and elevation in white blood cell count. Cellulitis may occur anywhere on the body (Scheinfeld, 2003:8). Cellulitis may progress to septicaemia if left untreated (Shimizu, 2007:453).



## **Treatment**

Treatment includes resting the infected area, removing the dead tissues and oral antibiotics at times (Vinh *et al.*, 2007:415). Patients suffering from diabetes are treated with beta-lactamase-resistant penicillins, first-generation cephalosporin, amoxicillin-clavulanate, macrolide or fluoroquinolone (Scheinfeld, 2003:8).

### **2.7.7.3 Folliculitis**

Many different types of folliculitis have been described since the 1960's. Folliculitis takes place when hair follicles are damaged and inflammation and infection result (Stulbergh *et al.*, 2002:109).

In the facial area of adolescents, folliculitis is commonly called acne vulgaris. These infections readily develop into a furuncle or carbuncle (Shimizu, 2007:454).

Eosinophilic folliculitis is a distinct dermatosis associated with the advanced stage of HIV (Majors *et al.*, 1997:219).

Folliculitis Keloidalis Nuchae is very common in Namibia and other African countries. This condition is commonly seen in African males after they have shaved their hair. It is a deep folliculitis usually caused by a staphylococci infection, which results in keloid scars to be produced in the deep cutaneous tissue (Van Hees, 2001:33).

Pruritic folliculitis in pregnancy normally occurs after the fourth month of gestation. This dermatosis is known to cause pruritus and erythematous papules. It spontaneously disappears after delivery (Bremmer *et al.*, 2010:32).

## **Aetiology and Pathophysiology**

Classification of folliculitis is defined by the depth of infection and inflammation of the hair follicle. Superficial folliculitis is most common. It causes little pain and normally heals spontaneously. *Staphylococcus aureus* is the most common pathogen to cause superficial folliculitis. In HIV/AIDS patients, yeasts and fungi are also common pathogens (Stulbergh *et al.*, 2002:109). *Staphylococcus epidermidis* is another common pathogen to cause folliculitis (Shimizu, 2007:454).

Deep folliculitis is caused when *Staphylococci* penetrate the hair shaft and infect the deeper tissue. A combination of oral and topical antibiotics is administered in the case of such condition (Stulbergh *et al.*, 2002:109).

Gram negative bacteria and yeast infections like *pitysporum folliculitis* also known as *malassezia folliculitis dermnetnz* are commonly seen in HIV/AIDS patients (Van Hees, 2001:33).

Drug-induced folliculitis is caused by medication such as lithium, iodides, bromides, phenytoin, anabolic steroids, corticosteroids, isoniazid and beta blockers (Kraft *et al.*, 2012:2).

### **Symptoms**

Erythema and pustule occur at the hair follicle. Crusts form on the skin lesions within a few days, but heal without causing scarring. Deep-seated folliculitis normally presents intense inflammatory symptoms (Shimizu, 2007:454). Furuncles and carbuncles may result (Gawkrodger, 2002:44).

Drug-induced folliculitis is identified when monomorphous eruption of follicular pustules and papules is found on the abdomen of patients, without the presence of comedones (Kraft *et al.*, 2012:2).

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*Pityrosporum folliculitis*

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### **Treatment**

Topical and systemic antibiotics are administered. Systemic antibiotics include cloxacillin, doxycycline, flucloxacillin and erythromycin. Topical antibiotics include mupirocin, fusidic acid and neomycin (Dhar, 2013).

Vaseline® application to the skin must be discontinued and better hygiene must be implemented. Pityrospora folliculitis is treated with imidazole topical cream and oral fluconazole if fungal infection is suspected (Cole, 2012).

The treatment of Folliculitis Keloidalis Nuchae is doxycycline twice a day for up to 1 month. Keloids are difficult to remove; however, skin grafting, cryosurgery and laser surgery has been effectively used in the past (Van Hees, 2001:33).

#### **2.7.7.4 Erythrasma**

##### **Aetiology**

Erythrasma is caused by gram positive bacillus *Corynebacterium minutissimum* (Van Hees, 2001:34). This bacterium is thought to be a normal resident of the skin, mostly found around the genital area and under breasts and armpits (Morales-Trujillo *et al.*, 2008:469). Erythrasma is often mistaken for fungal infections (Van Hees, 2001:34).

##### **Pathophysiology**

*Corynebacterium minutissimum* invades the upper third of the stratum corneum which causes it to thicken. The keratin within the cells and that in the intercellular spaces is dissolved, which results in a scaly eruption (Dellion *et al.*, 1996:716).

##### **Symptoms**

Dry, reddish-brown, sometimes scaly eruptions are usually found in skin folds (Gawkrodger, 2002:44). These lesions appear fluorescence red under Wood's light, due to porphyrins that are released by the bacillus when the keratin is dissolved (Morales-Trujillo *et al.*, 2008:469).

##### **Treatment**

Imidazole creams should be applied twice a day for 4 weeks. Erythromycin 2% lotion, and in more severe cases, systemic erythromycin 250mg four times per day for 2 weeks should be administered (Van Hees, 2001:34).

#### **2.7.7.5 Leprosy**

Leprosy is an ancient disease that has been documented since biblical times. Even though the disease is completely treatable today, we still often see patients infected with leprosy.

In 1995, the World Health Organization (1995:269) stated in a report on leprosy that between two and three million people were permanently disabled due to leprosy at the time.

### **Aetiology and Pathophysiology**

Leprosy is a chronic disease caused by the bacterium *Mycobacterium leprae*. It primarily infects the skin and the peripheral nerves (Al-Mutairi *et al.*, 2010:E876).

Leprosy is also known to be caused by another bacterium: *Mycobacterium lepromatosis* (Sasaki *et al.*, 2001:729). It is believed that leprosy is spread through respiratory droplets, but not all people who are exposed to the bacterium develop leprosy. Evidence indicates that certain families are more prone to develop leprosy than others (Alkais *et al.*, 2005:44).

### **Symptoms**

Leprosy symptoms develop slowly. Firstly, the skin and peripheral nerves thicken. Because of the nerve damage, the first symptoms are normally muscle weakness. A red, patchy skin rash follows. This rash is normally on the groin, axilla and scalp. The skin will swell, especially in the face and around the nose. Cartilage in the nose will be damaged. Loss of sensation on extremities may cause the loss of limbs due to injury (Lean *et al.*, 2011:1).

### **Treatment**

Dapsone 50 - 100mg per day is the preferred treatment in leprosy. Rifampicin 600mg once per day is also effective, but expensive. Other treatments include clofazimine and ethionamide (Nardelle, 2012).

### **2.7.8 Pigmentation Anomalies**

Pigmentation disorders are one of the major reasons for seeking dermatological care in Namibia. Pigmentation disorders can cause psychological and emotional distress in patients, and influence their quality of life (Grimes *et al.*, 2009:77).

#### **2.7.8.1 Hyperpigmentation**

Hyperpigmentation is the unusual darkening of the skin, more commonly seen in darker-skinned ethnic groups (Woolery-Lloyd *et al.*, 2011:171). The melanocytes of black people are more easily altered, thus resulting in hyperpigmentation (Grimes *et al.*, 2009:77). Any inflammation or injury to the skin can cause hyperpigmentation (Woolery-Lloyd *et al.*, 2011:171).

- **Melasma**

##### **Aetiology and Pathophysiology**

Melasma is often seen in women and more commonly in those who live in areas with high UV radiation (Gupta *et al.*, 2006:1048). During pregnancy, it is called chloasma gravidarum and usually clears spontaneously several months after delivery (Shimizu, 2007:467).

Melasma is predominately caused by more active melanocytes in the skin and not an increase of melanocytes as previously thought. UV radiation causes an up-regulation of cytokines stimulated by melanocytes. A family history of melasma suggests the significant role of genetics in the aetiology (Sheth & Pandya, 2011:692).

Abnormalities of sex hormones, for example in pregnancy, or the use of oral contraceptives, are also known to contribute to melasma (Shimizu, 2007:467). Phototoxic medications such as minocycline can also cause hyper pigmentation such as melasma (Sheth & Pandya, 2011:692.) Fitzpatrick skin types III and IV are more commonly affected (Sheth & Pandya, 2011:689).

##### **Symptoms**

Melasma is an asymptomatic disease. Symmetrical, light brown, patches form on the face, mostly affecting the forehead, cheeks and the area around the mouth. The patches are irregular in size (Shimizu, 2007:466).

### **Treatment**

Protecting skin from UV radiation with UVB and UVA sunscreens is fundamental in treating melasma. Contraceptives or other hormonal medications should be discontinued (Gupta *et al.*, 2006:1048).

Combination therapy consists of hydroquinone 5%, mixed with isotretinoin and a corticosteroid. Kojic acid and azelaic acid can also be used. Glycolic acid and trichloroacetic acids used to perform chemical peels, have a very high success rate in the treatment of melasma, while laser therapy has proven success to a lesser extend (Rendon *et al.*, 2006:S272).

- **Addison's Disease**

### **Aetiology and Pathophysiology**

Addison's disease is a very rare, autoimmune disease (Brook & Manson, 2005:20).

It is caused by hypo-adrenalism which causes the pituitary gland to cause overstimulation of ACTH. Melanogenesis results due to this overproduction which causes pigmentation (Gawkrödger, 2002:71).

### **Symptoms**

Pigmentation is seen across the entire body, even those areas that are normally not affected like the oral mucosa and tongue (Shimizu, 2007:269).

Nail beds and other areas exposed to sunlight are also affected. Vitiligo is also present in most cases (Brook *et al.*, 2005:21).

### **Treatment**

The most common treatment is corticosteroids taken orally three times per day. Fludrocortisone is administered if androgen is depleted (Brook & Manson, 2005:21).

- **Ephelides**

### **Aetiology and Pathophysiology**

Ephelides are also known as freckles and are more common in patients who have a light skin tone. They are described as a cluster or uneven distribution of melanin in the skin (Kumar *et al.*, 2005:1232). People who are genetically susceptible may have an increase of melanogenesis due to somatic mutations in epidermal melanocytes (Cockerelle *et al.*, 1996:1561).

Ephelides can develop due to intensive UV-A and UV-B exposure which causes melanosomes to enlarge due to an increased dopa reaction (Alai, 2008:2).

### **Symptoms**

Ephelides are flat, light-brown circular lesions on the skin that vary from 2mm to 5mm in diameter. These lesions are randomly scattered on the skin but more concentrated on areas exposed to the sun (Alai, 2008:1).

### **Treatment**

Ephelides are not a skin disease; rather a genetic property of the skin. Treatment is therefore not exactly indicated. The use of sunscreens prevents further melanin concentrations from clustering at one place (Alai, 2008:3). Ephelides are commonly treated with laser therapy, light therapy and chemical peels. In some cases, topical hydroquinone and isotretinoin are also effective (Ortonne *et al.*, 2006:S262).

## **2.7.8.2 Depigmentation**

Skin colour is caused by a mixture of three different types of pigment, namely melanin, oxyhaemoglobin and carotene. If any of these pigmentations are lost, hypo pigmentation results. This loss in pigment can affect a small area only or cover the entire body (Gawkrodger, 2002:70).

- **Vitiligo**

Vitiligo affects around 1% of the American population, but the condition is seen throughout different races worldwide (Gawkrodger, 2002:70). A study done in 2000 in south-eastern Nigeria showed that vitiligo had an incidence of 5.8% in eastern Nigeria (Nnoruka, 2005:125).

### **Aetiology and Pathophysiology**

Vitiligo is an acquired idiopathic skin disease which causes white, patchy areas on the skin (Gawkrodger, 2002:70). It also occurs in traumatised skin and can affect hair follicles, producing white streaks of hair (Van Hees, 2001:71). It is characterised by the selective loss of melanocytes. Approximately half of the patients diagnosed with vitiligo worldwide will develop the disorder before the age of 20 (Alikhan *et al.*, 2011:474).

Vitiligo is believed to be caused by an autoimmune response, but genetics and other external stress factors have been associated with it as well (Halder & Chappell, 2009:86).

### **Symptoms**

Vitiligo is represented by white macules or patches. It can localise to one area only or be distributed across the entire body (Gawkrodger, 2002:70). They are usually symmetrical in size and can increase in size over time. The lesions may cause light pruritus, especially after UV radiation (Alikhan *et al.*, 2011:474).

### **Treatment**

Sunscreen must be worn daily to prevent further degeneration of melanocytes (Gawkrodger, 2002:70). Leukoderma on the face can be concealed with cosmetics to limit the amount of physiological stress. PUVA and topical steroids are still the first-line treatments (Shimizu, 2007:262). PUVA light treatment induces hypertrophy of melanocytes and increases melanocyte growth (Felsten *et al.*, 2011:296).



Topical corticosteroids are used with a fair amount of success. They modulate the immune system and help decrease melanocyte destruction (Felsten *et al.*, 2011:295). Calcipotriol, which is a vitamin D3 analogue, can be used to help the re-pigmentation of the skin. If combined with corticosteroids, the efficacy increases (Felsten *et al.*, 2011:297).

- **Albinism**

Albinism is a name given to a large group of hypopigmentary anomalies. Albinism can be divided into two groups, namely ocular albinism and oculocutaneous albinism (Orlow, 1997:24). Oculocutaneous albinism is the most common autosomal recessive disorder in the southern part of Africa (Manga *et al.*, 1997:1095).

### **Aetiology and Pathophysiology**

Hypopigmentation in albinism can range from complete loss of melanin to slight reduction in colour of skin and hair (Carden *et al.*, 1998:189). The decrease in melanin is caused by immature melanosomes even though the melanocytes are normal and in the right size and numbers (Shimizu, 2007:269). Albinism results from inheriting recessive genes which, among others, cause the decrease of melanin in the skin (Carden *et al.*, 1998:189).

### **Symptoms**

Pigment in skin, hair and eyes are reduced or completely absent. Albinism is also associated with vision defects such as photophobia, nystagmus and astigmatism (Van Hees, 2001:74). Lack of skin pigmentation leaves individuals more susceptible to sunburn and skin cancers, such as squamous cell carcinoma (Shimizu, 2007:259).

### **Treatment**

Albinism is a chronic, irreversible, inherited condition. Treatment thus includes protecting skin from sunburn using sunscreen and protecting eyes from UV radiation wearing sunglasses (Van Hees, 2001:74).



- **Piebaldism**

### **Aetiology and pathophysiology**

According to Spritz (1997:15), Piebaldism is caused by defects in melanocyte development, which cause abnormal distribution of melanocytes during embryogenesis. Piebaldism is a rare, autosomal inherited disorder, mostly due to mutations of the KIT gene (Murakami *et al.*, 2004:30).

### **Symptoms**

Leukoderma is commonly seen on the forehead, abdomen and knees (Murakami *et al.*, 2004:30). Pigmentary anomalies are limited to skin and hair, without any neurological defects. Leukoderma does not change over time and remain stable without re-pigmentation (Passeron *et al.*, 2005:56).

### **Treatment**

Since piebaldism is a cosmetic defect, no treatment is given. Skin grafts of normal pigmented skin have shown some success (Spritz *et al.*, 1997:18).

- **Sutton nevus**

### **Aetiology and Pathophysiology**

Sutton Nevus is also known as *Halo nevi*, *Leukoderma Acquisitum Centrifugum* and *Perinevoid Vitiligo*. Sutton nevus is a rare dermatological condition that occurs when autoimmunisation takes place against melanin in a lentigo (Shimizu, 2007:264). Sutton nevus occurs in 1% of population only and has no correlation to sex or race (Aouthmany *et al.*, 2012:1).

### **Symptoms**

A lentigo becomes prominent due to oval leukoderma that surrounds it. Sutton nevus is commonly found on the trunk, face and neck of patients (Shimizu, 2007:264).

## **Treatment**

The condition is benign and heals spontaneously in most cases. The nevus may be removed or covered cosmetically. Topical steroids and PUVA (Psoralin and UVA) light treatments have shown to be successful to some extent (Shimizu, 2007:262,264).

## **2.7.9 Urticaria and Angioedema**

### **Aetiology, Pathophysiology and Symptoms**

Acute dermal edema takes place as plasma leaks into extra vascular cavities in the skin (Hennino *et al.*, 2006:30). Urticaria is a common dermatological reaction characterised by pruritus and erythema (Van Hees, 2001:88). Angioedema is characterised by the same symptoms but affect a larger area. Urticaria can be caused by immune or non-immune reactions. The lesions are caused by mast cells releasing histamines which cause the edema (Gawkrodger, 2002:72).

Different types of urticaria

- **Acute urticaria:** The sudden onset of urticaria persists for less than 6 weeks (Shimizu, 2007:107). Allergic reaction Type 1, mediated by IgE, is evident; for example, reaction to food, antibiotics, latex, elastoplast, etc. (Gawkrodger, 2002:72).
- **Chronic urticaria:** Defined as urticaria that persists for longer than 6 weeks, with erythema present most days of the week (Shimizu, 2007:107). Chronic urticaria may have many causes but are commonly defined to either be idiopathic or autoimmune (Kaplan *et al.*, 2004:465).
- **Physical urticaria:** Onset is sudden but disappears within 30 - 60 minutes after exposure. Physical urticaria is caused by exposure to hot or cold weather conditions, continuous rubbing of the skin or exposure to water (Shimizu, 2007:107).

- **Contact urticaria:** Urticaria is caused by contact by skin or mucosa with a foreign object (Shimizu, 2007:107).
- **Cholinergic urticaria:** 30% of all physical urticaria is caused by cholinergic urticaria and about 7% of all chronic urticaria (Onn *et al.*, 1996:847). This is commonly known to be caused by sweat when body temperature increases (Shimizu, 2007:107).

### **Treatment**

Treatment for urticaria and angioedema is the same. It is important to prevent further contact with the causative agents. Calamine lotions or topical steroids can be applied to ease pruritus and decrease edema (Van Hees, 2001:88). Histamine-1 antagonist, such as fexofenadine and desloratadine which are non-sedating, is the first-line treatment for both urticaria and angioedema. Oral corticosteroids are also used in severe acute attacks. If anaphylaxis should occur, an adrenaline injection is administered subcutaneously (Gawkrodger, 2002:72).

### **2.7.10 Melanoma**

Malignant melanoma is a tumour of melanocytes in the epidermis (Gawkrodger, 2002:94). Due to its aggressive nature, melanoma is the main cause of death in skin cancer. According to Nagore (2009:205), the increasing incidence of melanoma is the highest among any other type of cancer. According to the World Health Organization Report on Solar Ultraviolet Radiation, 48 000 deaths occur annually due to melanoma (Lucas *et al.*, 2006:20). Caucasians living in South Africa have shown very high incidence of melanoma. Statistics of Cape Town in 2009 has shown an increase of 69 new cases per year per population of 100 000. In Africa, melanoma affects more men than women (Whitaker *et al.*, 2010).

### **Pathophysiology**

Melanocytes are found between the dermis and epidermis of the skin. Early stage melanoma is defined as the period during which the melanocytes grow out of control

but stay in between the layers without contact with blood vessels (HersHKovitz *et al.*, 2010:67). The melanoma will quickly evolve into the radial growth phase, followed by the invasive growth phase. The tumor will become bigger and feed off the blood supply in the skin. In this phase, it will also have the potential to undergo metastases (Friedman *et al.*, 1985:130).

### **Aetiology**

Malignant melanoma has various causes, including excessive exposure to ultraviolet radiation and a family history of melanoma (GawKrodger, 2002:94). Childhood sun exposure plays a significant role in the development of melanoma. Caucasian skin with freckles and moles are at greater risk (Giblin & Thomas, 2007:32).

Melanomas are classified into four groups, namely nodular, superficial spreading, acral lentiginous and lentigo maligna (Shimizu, 2007:419).

### **Symptoms**

All the lesions are asymmetrical with a dark or black colour and a vaguely marked boundary (Shimizu, 2007:420). Lesions will start small and become bigger over time (GawKrodger, 2002:95). In most cases, metastases are lymphatic and will spread to other organs over time if untreated (Shimizu, 2007:421).

### **Treatment**

The first line of treatment is surgical excision. The prognosis is best if tumour is removed while it is still small and thin (GawKrodger, 2002:95).

#### **2.7.11 Epidermal and Follicle Tumours**

Malignant cutaneous tumours are the most common type of cancer. It mostly affects caucasians and seldom causes metastases (GawKrodger, 2002:95).

### **2.7.11.1 Basal Cell Carcinoma**

#### **Aetiology and Pathophysiology**

Basal cell carcinoma is the most common type of malignant tumour. It is mostly found on areas of the skin exposed to the sun, like face and neck (Carra *et al.*, 2007:252). Basal cell carcinoma arises from the basal keratinocytes in the epidermis. It is commonly caused by

- Prolonged UV radiation
- Arsenic ingestion
- Chronic scarring
- Family history of basal cell carcinoma

It affects men and women between the ages of 40 and 60 years and is more commonly seen in men than in women (Shimizu, 2007:390).

#### **Symptoms**

Lesions are small, firm and waxy. In Asians, they have a blackish-brown colour, but in Caucasians they have a pinkish-white colour. Telangiectasia can often be seen in and around the lesions (Shimizu, 2007:390). Lesions grow very slowly, but cause tissue destruction as they grow (Gawkrodger, 2002:95).

#### **Treatment**

Surgical excision is the primary treatment for basal cell carcinoma. Cryotherapy and topical chemotherapy may also be considered alternatively (Shimizu, 2007:390). Other topical treatments include 5-fluorouracil, imiquimod and photodynamic therapy (Carra *et al.*, 2007:252).

### **2.7.11.2 Squamous Cell Carcinoma**

#### **Aetiology and Pathophysiology**

Squamous cell carcinoma usually arises from keratinocytes in the epidermis. Skin exposed to ultraviolet radiation is more prone to develop this type of cancer (Gawkrodger, 2002:95). Squamous cell carcinomas tend to be more aggressive

than basal cell cancers. They are more likely to invade subcutis and spread to undergo metastasis to the lymph nodes and/or distant parts of the body (Leffell *et al.*, 2000:258).

Squamous cell carcinoma occurs mostly in the elderly and four times more frequently in men than in women (Leffell *et al.*, 2000:258).

### **Symptoms**

Lesions appear crusty or scaly and can be described as watery nodules. The lesions vary in size from 3mm - 10mm and are slow-growing (Leffell *et al.*, 2000:258). Secondary infections are common when lesions ulcerate and become infected with bacteria (Shimizu, 2007:393).

### **Treatment**

A biopsy must be made to distinguish squamous cell carcinoma from basal cell carcinoma (Gawkrödger, 2002:95). Surgical excision is the primary treatment for squamous carcinoma. Depending on metastases or the risk thereof, chemotherapy could be included in the treatment (Shimizu, 2007:393).

## **2.7.12 Acne Vulgaris**

### **Aetiology**

*Acne vulgaris* is generally self-limiting and primarily affects teenagers and young adults. It is more common in males than in females during adolescence, but more common in women than in men during adulthood. 85% of all people will suffer from *Acne vulgaris* at some point of their lives (Harper & Fulton, 2007).

### **Pathophysiology**

Acne develops when follicles become blocked, resulting in hyperkeratinisation which forms a keratin plug. The sebum excreted by the sebaceous gland gets trapped and a microcomedo forms. Androgen hormone levels have the most significant effect on sebaceous glands, enlarging them and increasing the production of sebum. The microcomedo forms when this sebum and desquamated keratinocytes cause a



blockage. Bacteria, mostly *Propionibacterium acnes* (*P. acnes*), quickly manifest themselves in the area causing inflammation (McKoy, 2013).

## Symptoms

*Acne vulgaris* affects skin areas with the greatest proportion of sebaceous follicles, which include the face, the upper part of the chest and the back (Harper & Fulton, 2007). It is characterised by papules, pustules, nodules, seborrhea and possible scarring (Adityan *et al.*, 2009:323). Microcomedo can either be an open comedone, commonly known as a blackhead, or a closed comedone, known as a millia (Simpson *et al.*, 2004:43). A millia or whitehead is a flesh-coloured or white lesion approximately 1 - 3mm in diameter. Blackheads have a dark centre and tend to be bigger in size, from 2 - 5mm. The epithelium becomes damaged with the accumulation of white blood cells, which eventually causes it to rupture. The comedone will elicit its contents and an inflammatory reaction results. Deep inflammation produces papules. Nodules are larger, deeper and more solid than papules. Cysts are superlative nodules which when infected with bacteria, can cause an abscess (McCall *et al.*, 2007).

## Treatment

*Acne vulgaris* treatment includes decreasing sebaceous gland activity, decreasing inflammation, inhibiting *P. acnes* bacterial growth and normalisation of follicular keratinisation.

- **Mild Acne**

Mild acne is defined as acne consisting of less than 20 comedones or 15 inflammatory lesions, or less than a total of 30 lesions (McKoy, 2006:954).

Benzoyl peroxide is the first-line treatment for mild acne vulgaris. It is effective against *P. acnes* bacteria and has shown very few side-effects. Sensitivity to the sun is, however, inevitable and the application of sunblock is compulsory. Benzoyl peroxide is almost as effective as antibiotics when applied used in concentrations 2.5 - 10% (Sagransky *et al.*, 2009:2555). Cleansing agents and antibacterial soaps are also good first-line treatment (Davis, 2006:4).

- **Moderate Acne**

Moderate acne is defined as acne consisting of 20 – 100 comedones or 5 – 15 inflammatory lesions or a total of 30 - 125 lesions (comedones and inflammatory) (McKoy, 2006:954).

Antibiotics are the first-line treatment for moderate acne. These include erythromycin, clindamycin and minocycline. Topical clindamycin and erythromycin (with or without zinc) have proven to be very effective. Hormonal treatment is very popular amongst women who suffer from acne. Oral contraceptives containing ethinyl estradiol and norgestimate have demonstrated improvement in acne when compared to a placebo control (Arowojolu *et al.*, 2007).

- **Severe Acne**

Severe acne is defined as more than 5 cysts or a total of 100 comedones or more than 50 inflammatory lesions or more than a total of 125 lesions (McKoy, 2006:954).

Isotretinoin is the gold standard for treating severe cystic acne. This is also the only drug that can provide extended remission. The normal dosage is 1mg/kg per day for 20 weeks. It is very important for the practitioner to explain possible side-effects of the drug and especially warn female patients not to become pregnant while on isotretinoin treatment (Davis, 2006:1).

### **2.7.13 Rosacea**

Rosacea is a chronic cutaneous condition affecting mostly the nose, chin and cheeks. It is defined as facial erythema. Over time, the nose can become lumpy and swollen (Wilkin *et al.*, 2004:905). In the past, rosacea was associated with alcoholism, but there is no clinical evidence of its collaboration.

### **Aetiology and Pathophysiology**

According to the National Rosacea Society, the cause of rosacea is unknown. It mostly affects women between the ages of 30 and 50 years. It is known as the “curse of Celts” in Great Britain. Certain elements like exercise, sunlight, severe sunburn, stress, anxiety, cold wind, medication or food can trigger rosacea. Foods that contain a high percentage of histamine, like red wine and cheese, may cause persistent facial flushing in patients diagnosed with rosacea (Powell, 2005:793).

Rosacea can occur in all racial and ethnic groups but are thought to be more frequently diagnosed in white patients (Powell, 2005:794).

### **Symptoms**

Rosacea is characterised by causing semi-permanent redness or flushing; telangiectasia; red-domed papules and pustules; red gritty eyes; burning sensations and rhinophyma (Wilkin *et al.*, 2004:905). Rosacea is often falsely diagnosed as acne vulgaris (Powell, 2005:793).

### **Treatment**

Oral tetracycline, doxycycline, minocycline and topical antibiotics such as metronidazole are usually the first-line treatment (Dahl *et al.*, 1998:679). Isotretinoin is the first choice after treatment failure with antibiotics (Hoting *et al.*, 1986:660). According to the international rosacea foundation, steroids should never be used to treat rosacea.

## **2.8 DERMATOLOGICAL DISEASES AND CONTRIBUTING FACTORS**

Dermatological disorders and diseases are common in developing countries due to poor hygiene, underlying infections such as HIV/AIDS, trauma, overcrowding in some areas and poverty. These contributing factors can be grouped into internal and external factors. This section will explore the significant contributing factors and define how they can be overcome to prevent the spread of dermatological diseases.

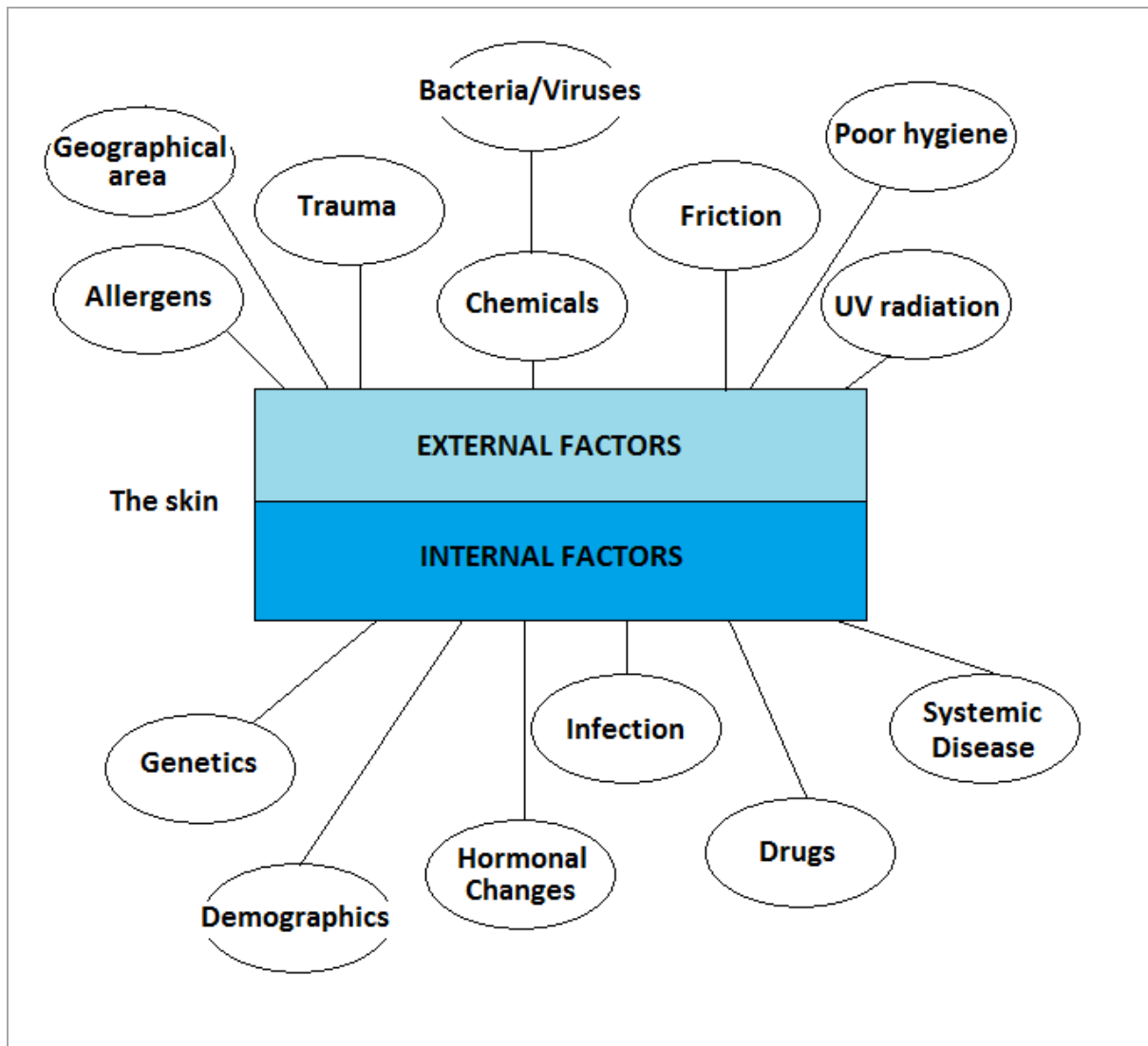


Figure 6: Internal and External Contributing Factors of Dermatological Disease

### 2.8.1 HIV/AIDS

According to the website of Australian College of Dermatologists, 90% of people with HIV/AIDS will develop some kind of dermatological problem or condition during their life. The mere presence of certain skin diseases can be an indication that the patient are HIV positive. (Australian College of Dermatologists, 2001) Other skin diseases can serve as a warning that CD4 counts of the patient is decreasing (Preda & Whitfeld, 2010:56).

Acute retroviral syndrome is defined as a phase between the initial HIV infection and the seroconversion period. Most patients are presented with a rash and

lymphadenopathy. These symptoms are helpful in detecting HIV infection during the window period (Maniar *et al.*, 2008:628).

Patients that are HIV positive can suffer from a variety of dermatological diseases, including oral candidiasis, extensive molluscum contagiosum, eosinophilic pustular folliculitis, cryptococcosis and Kaposi's sarcoma (Amerson & Maurer, 2010:16).

Namibia has one of the highest HIV/AIDS prevalence rates in the world according to the report by the WHO (2009) on Namibia expanding its antiretroviral treatment. According to the 2003 data from USAID, 21.3% of the Namibian population is HIV positive. Data from 2006 suggest that one out of five pregnant women in Namibia is HIV positive (De la Torre *et al.*, 2008: IV).

Opportunistic dermatological diseases can be limited by implementing antiretroviral treatment as soon as possible. Preserving the immune system and keeping the viral load as low as possible will further limit the number of infections (Clay *et al.*, 2001:1).

Antiretroviral drugs can also cause adverse cutaneous reactions, especially at the beginning of high-activity antiretroviral therapy. Dermatological conditions such as hyperpigmentation, erythema multiforme, urticaria and Stevens Johnson syndrome are common under these patients (Radhakrishnan *et al.*, 2010:84).

### **2.8.2 Poor Hygiene and Poverty**

According to Morrone (2008:245), skin disease represents the greatest public healthcare problem in all developing countries. Poor hygiene and poverty play a significant role in dermatological diseases, especially tropical dermatological diseases. Figure 7 indicates the poverty distribution according to race (Adapted from data from the National Review of Poverty and Inequality in Namibia, 2008).

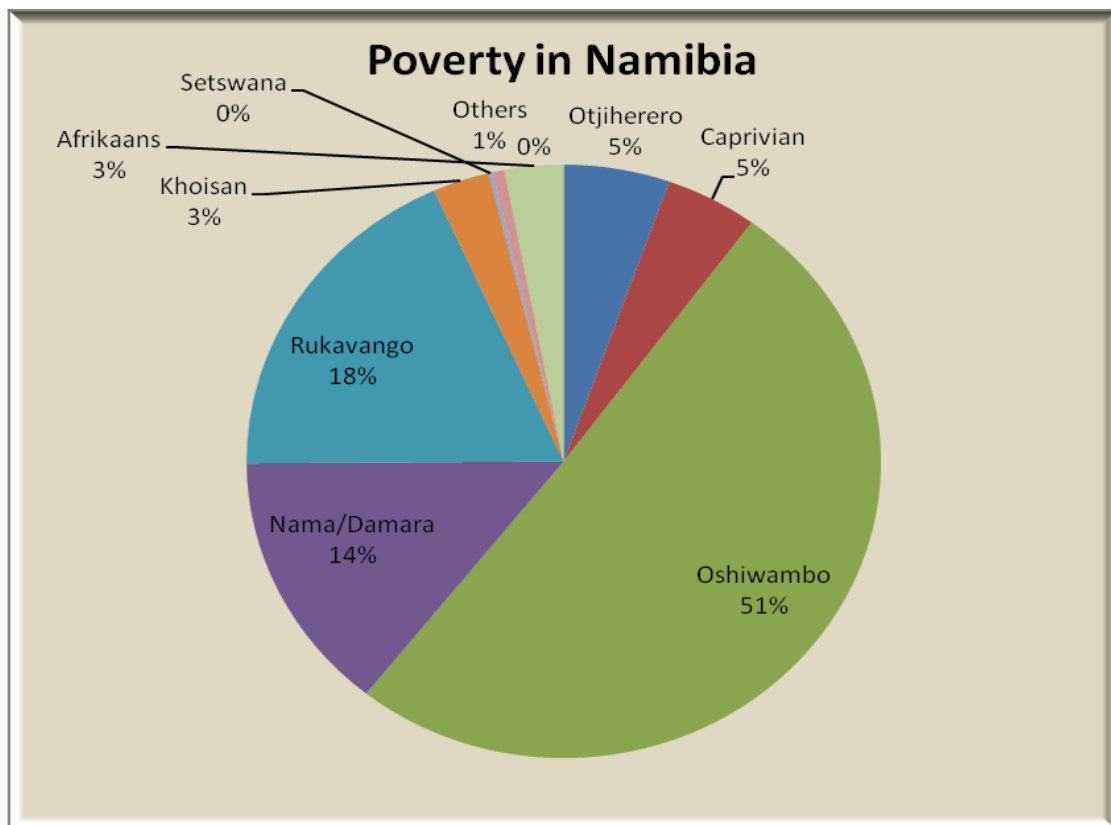


Figure 7: Poverty Distribution in Namibia According to Race

Diseases like scabies, tinea corporis, tinea capitis, cellulitis, folliculitis and impetigo can result from poor hygiene. Different factors like the lack of clean, running water and inability to buy soap have been defined as some of the problems for the high incidence of dermatological diseases (Accorsi *et al.*, 2009:469). People living in poverty have to rely on public health-care services provided by the Ministry of Health.

### 2.8.3 Chronic Diseases

Chronic diseases often present dermatological problems. Chronic kidney disease (CKD) is one of the major chronic diseases that also have a dermatological manifestation. Generally, the skin of patients with chronic kidney disease will appear paler and drier than normal due to uremic pruritus (Chaturvedy, 2012:284).

Many patients with diabetes mellitus are presented with granuloma annulare, necrobiosis lipoidica, xanthoma, bullous disease, neuropathic leg ulceration, lupus

pernio of the nose, erythema nodosum and sarcoidosis. Patients with hyperthyroidism or Grave's disease often also suffer from pretibial myxoedema, diffuse alopecia and palmar erythema. Cushing's syndrome can also cause thinning of the skin, spontaneous bruising, striae, diffuse alopecia, acne and hirsutism (Narayan, 2009:227).

#### **2.8.4 Allergies**

According to Rigoly *et al.* (2011:43), allergic related diseases have increased over the past few years. Many patients are presented with pruritus and other dermatological symptoms due to allergies which, in effect, cause contact dermatitis. The most common seasonal allergy is a reaction to pollen and dust. The most common food allergy is reaction to nuts and fish. Nickel, a metal frequently found in jewellery, is also a common culprit; gold, silver and other metals like cobalt chloride found in antiperspirants and hair-dyes, can cause an allergic reaction. Balsam of Peru, found in many cosmetic lotions and perfumes, also has allergenic potential (De Noon, 2006:1). Sensitivity to formaldehyde, often used as preservative in household cleaners, paints and even medications, is frequently encountered (De Noon, 2006:2).

The building industry in Windhoek is growing at an astounding rate. According to a local newspaper, The Namibian Sun (10 September 2013), the residential and commercial property plans approved for 2013 so far, are 28% higher than for 2012. The growth in the building sector causes more patients to suffer from allergies to cement and other building chemicals, according to an interview with Dr FJA Smith in 2012. He explained that conditions such as urticaria and contact dermatitis were a common occupational hazard for some builders.

The five categories for skin allergy (refer to 2.5) indicate the severity of allergies.

#### **2.8.5 Genetics**

Like in any other disease, genetics play a vital role in some dermatological disorders. Diseases such as melanoma have a 5 - 10% chance of being inherited by family (Leichman *et al.*, 2009:e1).

Atopic eczema has a strong familial correlation. A study was done on monozygotic vs dizygotic twins with eczema; the monozygotic twins had a 72% chance of having or developing eczema whereas the dizygotic twins a 21% only (Forrest *et al.*, 1999:1067). Psoriasis has also shown the same concordance with monozygotic and dizygotic twins (Bowcock *et al.*, 2004:45). Some studies have estimated that at least two-thirds of psoriasis cases are accounted for by genetic factors (Yonghong *et al.*, 2004:318).

Genetic defects in keratine have also been associated with various dermatological disorders, including epidermolytic hyperkeratosis, epidermal nevi, epidermolytic and non-epidermolytic forms of palmoplantar keratoderma, pachyonychia congenital and epidermolysis bullosa simplex (Fuchs, 1966:181).

#### **2.8.6 Climate and Geographical Area**

Dermatological diseases have a strong correlation with geographical area and climate. Diseases such as dermatophyte infections are more prominent in the rainy season or geographical areas where humidity and temperatures are high (Del Boz-Gonzalez, 2012:288). Dermatitis, like atypical dermatitis on the other hand, is the opposite and more prominent in low-humidity climate (Figueroa, 2011:311).

According to Figueroa (2011:311), the thinning of the ozone layer and changes in global climate play a significant role in dermatological disease. Diseases like acne and skin infections caused by gram-positive and negative bacteria, become worse due to the contamination of water. The increase in skin cancer by 2050 is estimated to be 300% by some, due to climate change (Figueroa, 2011:312).

#### **2.8.7 Demographical factors**

According to results of this study, race, age and gender play a significant role in dermatological disease. This will be illustrated in Chapter 4 of the research study.



Since poverty plays such a significant role in the black population (as discussed in 2.8.2), a clear relationship between dermatological disease and race is evident in Africa.

An understanding of racial differences in skin function is important in the prevention and treatment of dermatological disease. According to the report on poverty in Namibia on the Namibian National Planning Commission website (2011), 41.2% of Namibian people live in poverty, 96% of whom is black.

Some dermatological diseases are also age specific. A study has shown that older humans have a decrease in skin immune function. This can cause an increase in bacterial and fungal infections of the skin (Vukmanovic-Stejic *et al.*, 2011:525).

Certain viral infections like that of Varicella occur mostly in children (Van Hees, 2001:47). Acne is also more common during puberty than any other period in a human's life (Harper & Fulton, 2007).

Literature also indicates that certain dermatological diseases, such as melanoma, are more common in men than in women. This statistic is true for Africa, since men still work most of their day in the sun, exposed to harmful UV-rays, however, hyperpigmentation occurs more often in women than in men due to hormones such as progesterone (Whitaker *et al.*, 2009).

### **2.8.8 Cigarette smoke**

Cigarette smoke can cause or increase the chances of developing systemic lupus erythematosus, psoriasis, palmoplantar pustulosis, cutaneous squamous cell carcinoma, hidradenitis suppurativa, candida and genital warts (Thomsen & Sorensen, 2010:4). It also causes various degenerative dermatologic conditions such as skin wrinkling and decreased wound healing (Mottilo *et al.*, 2009:718).

Studies done by Glick *et al.* (2009:6), indicated that contact dermatitis could occur in some patients due to a reaction to cigarette paper, the filter or the tobacco.

Studies suggest that tobacco smoke causes vasoconstriction due to the release of catecholamine (Gottrupp, 2006:61). This decrease in capillary and arteriolar blood flow may cause damage to different skin cells. Fibroblasts, collagen, elastin and connective tissues are all directly affected by cigarette smoke. It is also well known that tobacco smoke induces aging and the appearance of wrinkles (Koh, 2002:21).

Another study suggests that smoke is also phototoxic and will result in more damage to the skin once in contact with UV radiation (Helfrich *et al.*, 2007:397).

### **2.8.9 Pregnancy and breastfeeding**

The most common skin disease during pregnancy is hyperpigmentation or melasma. The increase in melanin causes the condition to appear. Pruritic urticaria papules and plaques (PUPPP) during pregnancy can cause small reds lumps that itch or burn (Tunzi & Gray, 2007:211).

- **Hyperpigmentation**

This is a process by which the skin darkens due to an increase in melanin. The increase in progesterone causes the melanocytes to become more active. The hyperpigmented area lightens after delivery, but hardly ever recovers completely. It can be brown patches around eyes and lips, commonly known as the “mask of pregnancy”, or blemishes on chest, back or abdomen (Tunzi & Gray, 2007:211).

- **Pruritic urticaria papules and plaques of pregnancy (PUPPP)**

Pruritic urticaria papules and plaques of pregnancy is an itching and burning rash which can produce red lesions. Usually, it affects the chest, abdomen or back. If the affected area is large, it will form plaques (Tunzi & Gray, 2007:211).

- **Stretch marks**

This is the process by which the skin does not return to its original state after stretching. Striae develop which change colour from red to white over time (Tunzi & Gray, 2007:211).

- **Skin tags**

A skin tag is a small piece of tissue that develops during pregnancy which looks like a colorless mole. These tags are benign and can usually be easily removed. They appear commonly on the neck, chest, back, under breasts and in the groin area (Tunzi & Gray, 2007:211).

### **2.8.10 Skin colour**

Skin colour has always been a controversial topic, often linked to social economic differences; however many physiological differences have been identified.

Skin colour does not only indicate a difference in outer appearance but also a distinctive difference in physiology and responsiveness of a patient's skin. The significant difference between dark and light skin is that the melanocytes of dark-skinned individuals are larger in size than that of light-skinned individuals. (Cole *et al.*, 2009:1). This contributes to the fact that dark-skinned people are more UV radiation resistant than light-skinned people.

Studies have shown equality in thickness of stratum corneum in dark and light skin, but that dark skin contains more compact layers of corneocytes (Thomson *et al.*, 1955:236). This makes the skin more resistant to external factors. Slower degradation of keratinocytes has also been noticed in dark-skinned people (Cole *et al.*, 2009:1). Increased lipid content, decreased ceramide content and resistance to inflammation have also been noted in darker-skin patients (Weigand *et al.*, 1974:563).

According to Warriar *et al.* (1996:229), dark-skinned individuals have a lower surface pH than light-skinned individuals. Sebum production has also been found to be higher in ethnic skin than caucasian skin.

A study done on the water content of the skin after exposure to UV radiation indicates that dark-skinned people have higher water content after exposure than light-skinned people (Beradesca *et al.*, 1991:89).

According to Cole *et al.* (2009:1), dark-skinned people suffer more frequently from dry, cracked skin which, together with increased sebum production, might cause acne. Hydration of ethnic skin in Africa is commonly achieved by applying Vaseline® that, due to its petroleum content, causes dermatitis (Van Hees, 2001:47).

Hyper- and hypopigmentation, or uneven colouration as some describe it, is a common dermatological complaint among ethnic-skinned patients. The misuse or uninformed use of bleaching agents often causes further skin damage in the form of sensitivity to UV radiation and uneven pigmentation.

Vitiligo, according to literature, not limited to ethnic skin but more noticeable (Lotti *et al.*, 2008:110).

UV radiation damage has a higher incidence in light-skinned individuals which increases their incidence for skin cancer and photo damage (Huncharek *et al.*, 2002:1173).

The difference is, however, not only physiological but also economical and cultural. According to Koghar, (2011:1) white people have 20 times the wealth of black people; therefore white people have better resources to spend on healthcare and sanitation than black people. Dermatological diseases are often caused by the lack of hygiene. If resources are limited, food is considered a higher priority than toiletries.

Cultural differences also play a significant role in dermatological diseases. Some cultures, like the Ovahimba in the north-western parts of Namibia, apply a mixture of animal fat and red powdered rock to their skin. Most of them do not take regular baths or have the luxury of toiletries. Other indigenous tribes living in the northern parts of Namibia, like the Okavango and some sub-tribes of the Oshiwambo, do not

practice daily baths and sanitation. It is assumed that in the caucasian culture in Namibia, daily baths and moisturising of the skin is essential and a priority.

## **2.9 DERMATOLOGICAL TREATMENT WITH CORTICOSTEROIDS**

Dermatological diseases are often treated with topical corticosteroids. Steroids creams, ointments and gels are used and abused on a daily basis. A study done in Pretoria indicates that at least 35% of black African women use corticosteroid containing formulations for skin bleaching (Malangu *et al.*, 2009:1).

Many patients in this research study were treated with corticosteroids. Any condition characterised by inflammation, immunological involvement or hyperproliferation can be treated with a topical steroid (Habif, 1990:27).

Corticosteroid potencies are classified by different systems. The United States of America uses a system that utilises 7 different classes based on the ability of the corticosteroid to constrict capillaries. Most European countries utilize a 4-class classification system.

The table below indicates the different potencies of the various corticosteroids according to the MIMS Drug Therapy Review of 2011 (Ference & Last, 2010:411).

Table 2: Topical corticosteroids potency groups

POTENCY GROUP	GENERIC NAME
Ultra High (I)	Betamethasone dipropionate (>0.05%)
	Clobetasol propionate (>0.05%)
	Diflucortolone valerate ointment (>0.05%)
High (II)	Betamethasone dipropionate 0.05 % ointment
	Fluocinonide acetone 0.05 %
Medium (III)	Betamethasone dipropionate 0.05 % cream
	Fluticasone propionate 0.05 % ointment
	Triamcinolone acetamide 0.5 %
Medium (IV-V)	Betamethasone valerate 0.025 %
	Flucinonide acetone 0.025 %
	Fluticasone propionate 0.05 % cream
	Hydrocortisone butyrate 0.1 % ointment
	Mometasone furoate
	Triamcinolone acetone 0.025 %
Low (VI)	Alclometasone dipropionate 0.05 %
	Flucinolone 0.01 % cream
	Hydrocortisone butyrate 0.1 % cream
Least potent (VII)	Hydrocortisone 1 %

Steroids are available in different vehicles to ensure that the medication delivery is optimised on certain areas of the body. Hydration increases steroid penetration which implies that steroid penetration is best after a shower or bath (Inayat *et al.*, 2009:173). The following table indicates the most effective relation between the condition and vehicle used (Ference & Last, 2010:412).

Table 3: Different Steroid Vehicles used to treat Dermatological Diseases

Steroid Vehicle	Disease/Area
Ointments	Psoriasis. Dry, thick hyperkeratotic lesions
Creams	Hairy areas, facial areas, intertriginous areas
Lotions, Gels	Oozing wounds and hairy areas
Foams, Mousses, Shampoos	Scalp

Dermatological diseases recorded in this study and treatable with topical corticosteroids can be classified as follows according to the MIMS Drug Therapy Review of 2011 (FERENCE & LAST, 2010:412).

Table 4: Strength of Corticosteroid used to treat Dermatological Diseases

Topical Steroid	Dermatological Condition
<b>High Potency Steroid</b> <b>Group I - III</b>	Atopic dermatitis
	Hyperkeratotic eczema
	Nummular eczema
	Psoriasis
	Severe hand eczema
<b>Medium Potency Steroid</b> <b>Group IV-V</b>	Atopic dermatitis
	Scabies
	Seborrhoeic dermatitis
	Severe dermatitis
	Status dermatitis
	Asteatotic eczema
<b>Low potency Steroids</b> <b>Group VI - VII</b>	Dermatitis (nappy)
	Dermatitis (eyelids)
	Dermatitis (face)

It is important that high-potency corticosteroids are not prescribed unnecessarily for diseases that require low-potency corticosteroids. High-potency corticosteroids have a higher incidence in causing skin atrophy and pigmentation side-effects. Sensitive areas like dermatitis around the eyelids or even a nappy rash must be treated with low-potency steroids to prevent irritation in those areas (FERENCE & LAST, 2010:412).

## 2.10 CHAPTER SUMMARY

In this chapter, the anatomy and physiology of the skin has been discussed. The immunology of the skin and allergic skin reaction were also described. The different dermatological diseases and contributing factors were extensively examined. Topical corticosteroid treatment was discussed and its relation to potency groups and dermatological diseases.

## **CHAPTER 3: METHODOLOGY**

This chapter reviews the specific method in which the study was conducted to obtain the necessary data and draw the necessary conclusions. The various study objectives are listed and the statistical methods used to analyse the different phases, are discussed. At the end of this chapter, factors that may have influenced the data, such as data analysis and ethical considerations, are taken into account.

### **3.1 GENERAL OBJECTIVES**

The aim of study is to investigate the prevalence and medicinal treatment patterns of dermatological diseases in the private health sector of Namibia, with special reference to Windhoek.

### **3.2 SPECIFIC RESEARCH OBJECTIVES OF THE EMPIRICAL STUDY**

The specific research objectives of the empirical study are the following:

- To identify the prevalence of dermatological conditions in Windhoek by identifying patients with dermatological conditions. This includes patients with prescription from their general practitioner, patients who visited the dermatologist and patients consulting the community pharmacy for pharmacist-initiated therapy;
- determine the relationship between the different dermatological conditions and demographical data such as age, race and gender;
- determine the geographical distribution of patients with dermatological diseases;
- investigate the possible differences in the prevalence of dermatological problems between patients with HIV/AIDS;
- formulate recommendations regarding the treatment of dermatological conditions in the private health sector with special reference to Windhoek.



### 3.3 RESEARCH METHODOLOGY

The empirical study was done in two phases. Data were collected from three different sources in order to meet the objectives in 3.2.

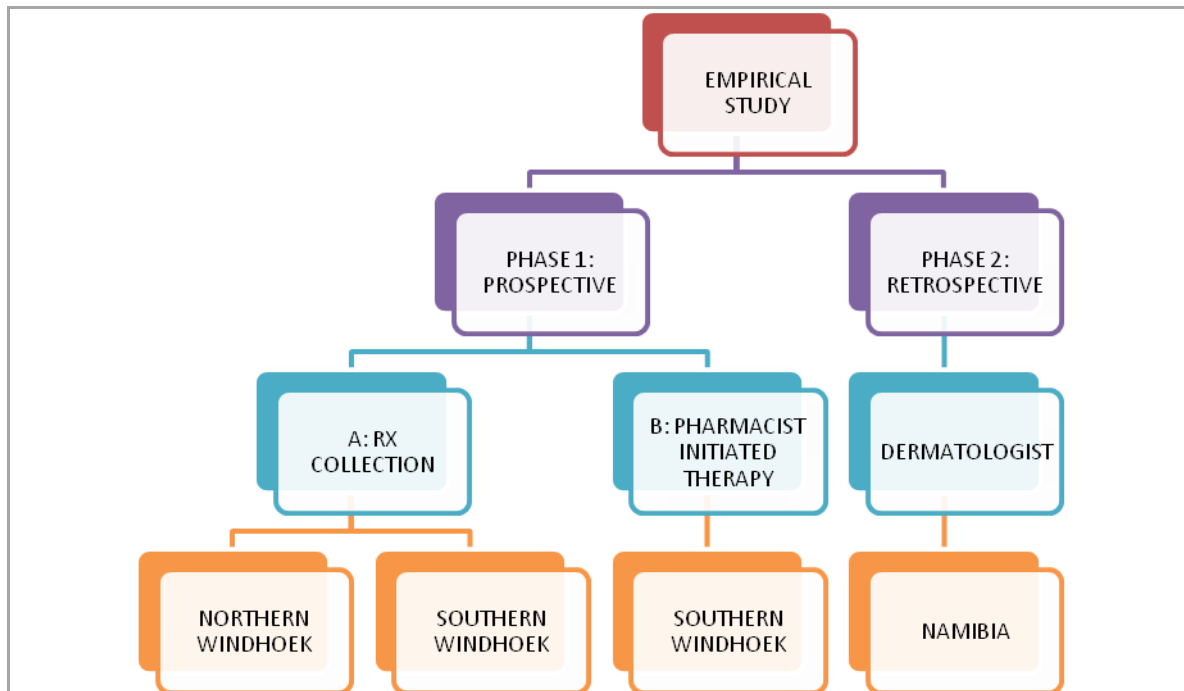


Figure 8: Organogramme of Empirical Study

#### 3.3.1 Phase 1A: Community Pharmacy

This phase was done prospectively by means of an observational study. The target population was patients with dermatological prescriptions only from general practitioners (thus excluding prescriptions from dermatologists) and patients without a prescription, consulting the pharmacy for pharmacist-initiated therapy and self-medication.

##### 3.3.1.1 Phase 1A:

###### (a) Research Design

This phase was done prospectively by means of an observational study to ensure that the data collected are relevant to current dermatological disease trends. The prospective research design was also chosen to ensure that the

dialogue between the pharmacist and patient about their current dermatological diseases was well documented in the questionnaire filled in by the patient.

## (b) Study Population

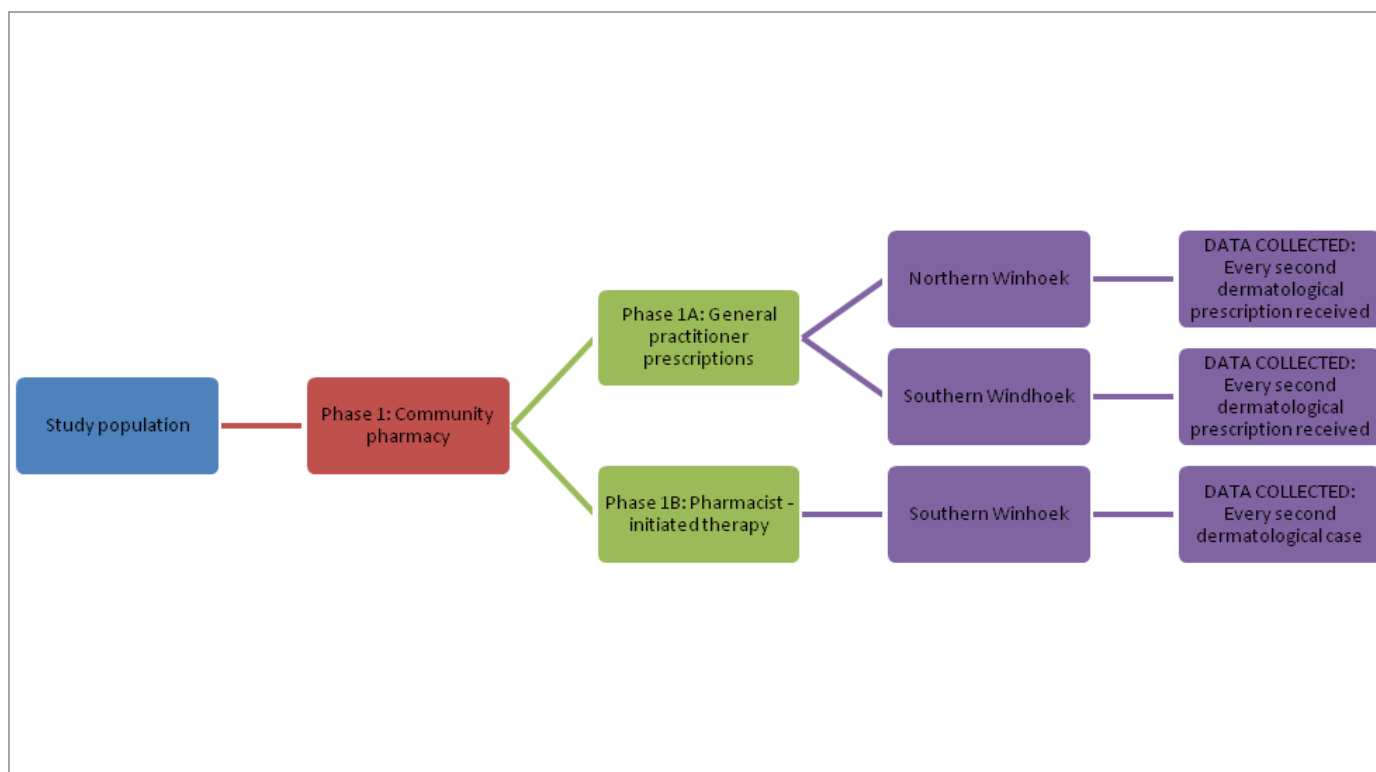


Figure 9: Organogramme of Study Population for Phase 1

Patients were identified based on the specific dermatological condition for which they had a prescription from their general practitioner. The study population was determined by selecting every second patient with a dermatological prescription consulting the pharmacy. This ensured that the data collection was accurate and unbiased. No dermatological prescriptions were seen as exclusions.

## (c) Development of Survey Forms

The data collection for this phase was done by using a questionnaire (*Annexure A & B*). The questionnaires were developed with the following in mind:

- Relevance

- Simplicity
- Accuracy
- Clarity
- Practicability

The questionnaire was designed to be completed by the patient or filled in by the pharmacist while presenting the questions to patients. This was done because some patients that participated in the research study were illiterate. The interview or completion of the questionnaire was done in a private area. The necessary prescriber was then contacted to obtain a diagnosis. The treatment regime prescribed and diagnoses were filled in on the questionnaire by the pharmacist. Care was taken to ensure that all the information for the different variables was accurately recorded.

Reliability of data was ensured by the prospective study design. The pharmacist was in dialogue with the patient before and during the completion of the questionnaire. The most significant data which included the diagnosis, treatment and duration of treatment, were captured by the pharmacist.

Patients were identified based on the specific dermatological condition. The process was explained to the patient and it was ensured that the patient fully understood the process. A pharmacy assistant was asked to explain the process in the patient's mother tongue if it was necessary. Permission was then voluntarily obtained by the signing of a form of consent (*Annexure A*) after which the questionnaire was filled in (*Annexure B*). If the patient was a minor, the parent or legal guardian was asked to give consent.

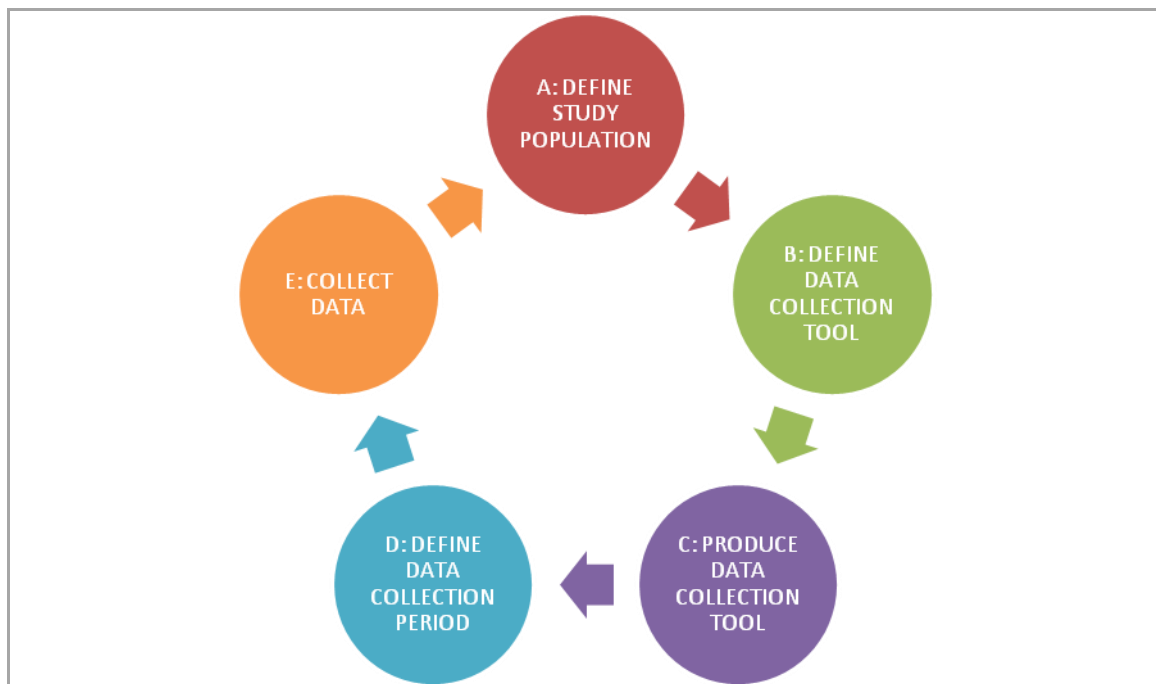


Figure 10: Data Collection Process

Data were collected from the community pharmacy environment over a period of three months after ethical approval. The dermatological-related prescriptions were collected from two different community pharmacies located in the northern and southern parts of Windhoek. These pharmacies were strategically chosen to ensure a broad spectrum of prescriptions from various ethnic groups. A total of 302 prescriptions were collected over the three-month period. Data collected were valid and free from bias. The data were collected with the understanding that patients and prescribers supplied truthful information.

#### (d) Study Variables

The questionnaire was developed to be just over one page in length; this ensured that the most accurate data were captured within the least amount of time, which encouraged patient participation. The following data were captured on the questionnaire:

- Demographic data
  - Gender
  - Date of birth

- Race
- Occupation
- Medical history
  - Chronic disease
  - Chronic medication
  - Other current medical conditions
  - Allergies
  - Immune altering diseases
  - Sensitivity to the sun
  - Procedures done on skin
  - Cosmetic products used
  - Smoker
  - Alcohol use
  - Pregnant or breastfeeding
- Current dermatological disease
  - First-line treatment
  - Family history
  - Period elapsed before seeking healthcare
  - Diagnosis
  - Treatment
  - Treatment duration

The following data were not collected:

- Personal details, for example patient's name and address.
- Family and personal medical history not relevant to dermatology.
- Prescribing doctor's personal details or name.

### **3.3.1.2 Phase 1B:**

#### **(a) Research Design**

This phase was done prospectively by means of an observational study to ensure that the data collected are relevant to current dermatological disease trends.

Data were collected from the community pharmacy environment over a period of three months after the approval of the ethics committee. Every second dermatological case that consulted the pharmacy was documented. In total, 74 dermatological cases were identified over the three-month period of this phase.

### **(b) Study Population**

Patients that came to the pharmacy for pharmacist-initiated therapy, thus without a prescription from a doctor, were examined privately in a consultation area specially prepared for this research study. This data were collected from the southern parts of Windhoek only.

### **(c) Development of Survey Forms**

The data collection for this phase was done by using a questionnaire (*Annexure A & C*).

The questionnaire was designed to be completed by the pharmacist while presenting the questions to the patient. The interview and the physical examination were done in a private area. The process was ensured to be explained and fully understood by the patient. A pharmacy assistant was asked to explain the process in the patient's mother tongue if it was necessary. Written consent was then voluntarily obtained (*Annexure A*) after which the survey form was completed by the pharmacist (*Annexure C*). If the patient was a minor, the parent or legal guardian was asked to give written consent. Care was taken to ensure that all the information for the different variables was accurately recorded. Information provided by the patient was understood to be accurate and trustworthy.

Reliability of data was further ensured by the prospective study design. The pharmacist was in dialogue with the patient during the completion of the questionnaire and the physical examination.

#### **(d) Study Variables**

The survey captured the following data:

- Demographic data
  - Gender
  - Date of birth
  - Race
  - Occupation
  - Contact number for follow-up consultation
- Medical information
  - Smoke
  - Alcohol use
  - Pregnant or breastfeeding
  - Allergies
  - Chronic disease
  - Chronic medication
  - Other current medical conditions
  - Immune altering diseases
  - Sensitivity to the sun
  - Procedures done on skin
  - Cosmetic products used
  - Family history

The physical examination was conducted in a private consultation area. A protocol was developed in order to facilitate this process. The following questions were asked and recorded:

- How long has the skin condition been present?
- Where did it appear the first time?
- Had it spread to other body parts?
- Is the condition consistent or does it appears at intervals?
- Have you ever had this condition before?
- Are the lesions moist or dry?
- Are the dermatological conditions associated with any symptoms like pain, pruritus, oedema etc.?

- What time of day do these symptoms occur?
- Any friends/family that are currently experiencing the same condition?

The physical examination was conducted in the following manner:

The distribution of dermatological disease was determined. This can be classified into four areas (Figure 11):

- Sun-exposed areas: Diseases such as contact dermatitis, photodermatitis and melanoma are often seen in these areas.
- Flexor: Area associated with topic eczema.
- Extensor: Area associated with psoriasis.
- Scalp, eyebrows, sides of nose and central chest: Areas associated with seborrhoeic dermatitis.

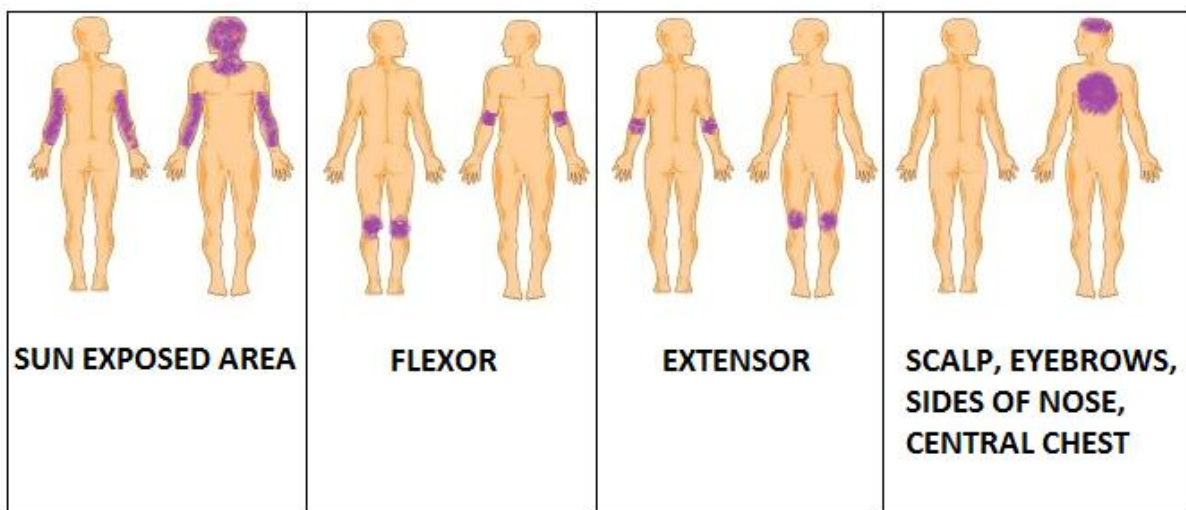


Figure 11: Areas on the Body where Dermatological Problems are most likely to occur (adapted from Narayan, 2009).

- The type of rash or lesion was identified. These morphological characteristics of different diseases help to determine a possible diagnosis.
- An over-all examination of the patient's appearance was conducted.
- A possible diagnosis was determined and treatment was given.
- Follow-up consultation



- Telephonically: The patient was contacted after the treatment duration had elapsed to determine if treatment was successful.
- Physical examination: The patient returned to the pharmacy, upon which a physical examination was done to determine whether treatment provided was successful.
- Referral to general practitioner: If treatment was not successful, the patient was referred.

The following data were not collected in this phase:

- Personal details, for example patient's name and address.
- Family and personal medical history not relevant to dermatology.

### **3.3.2 Phase2: Dermatologist**

This phase was done retrospectively by means of an observational study. The target populations were patients from the dermatologist's consulting rooms in Windhoek. Data were collected from his practice from patient files of those who consulted him over a period of three months. Data were collected only from patients who had given permission to participate in this research project.

#### **(a) Research Design**

This phase was done retrospectively by means of an observational study. Logistically, it was impossible for data to be collected while the patients were in consultation with the dermatologist. Therefore data collection took place after the 3three-month period had elapsed.

### (b) Study Population

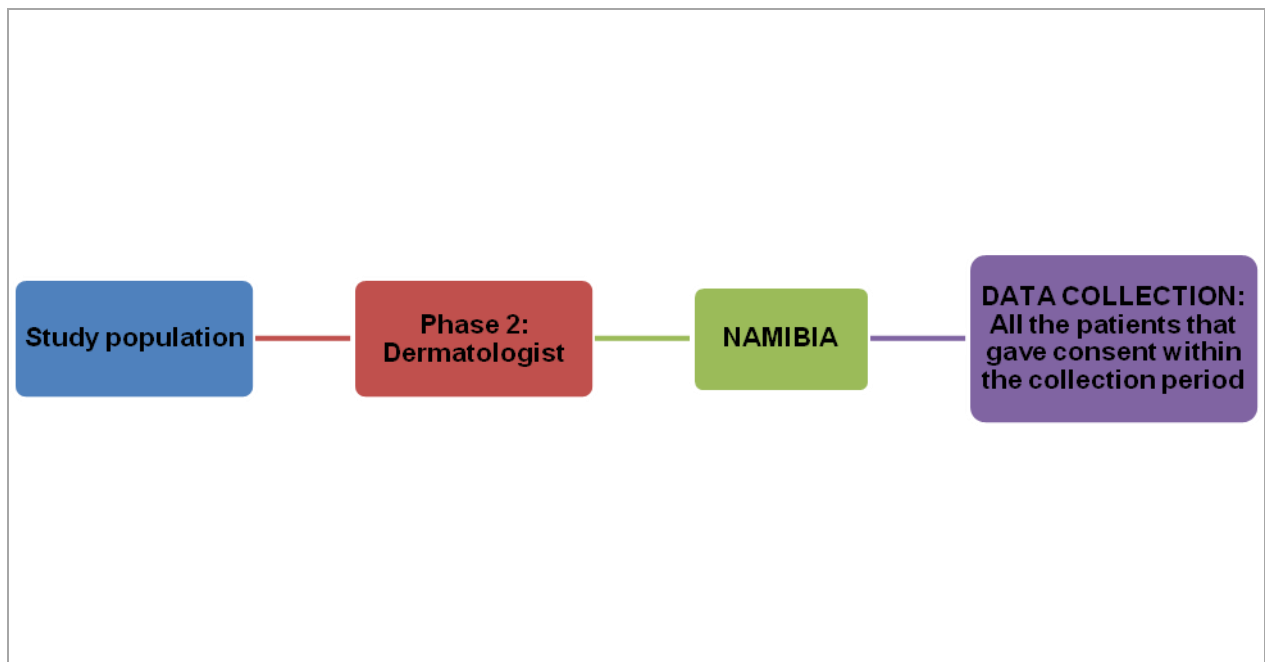


Figure 12: Organogramme of Study Population for Phase 2

The study population included every patient that had given their consent to the dermatologist for data collection at a later stage. This ensured that the data collection was accurate and unbiased. No dermatological cases were seen as exclusions, whether they resulted from internal, systemic or external factors.

### (c) Development of Data Extraction Form

The data collection for this phase was done by using a data extraction form (*Annexure E*). The form was developed with the following in mind:

- Relevance
- Simplicity
- Accuracy
- Clarity
- Practicability

The form was designed to be completed by the pharmacist as information was being obtained from the patient files. Care was taken to ensure that all

information for the different variables was accurately recorded. If anything was unclear, the dermatologist was consulted.

The collection of accurate data was guaranteed since data were physically documented and captured from the original source, namely the patients' files. In addition, reliable data were ensured by the fact that it was collected by the responsible pharmacist and the information was discussed with the specialist dermatologist upon collection.

Patients were identified based on their signing of the form of consent (*Annexure F*). The process was explained to the patient by the dermatologist while the patient was in consultation. If the patient was a minor, the parent or legal guardian was asked to give consent.

Data were collected from the dermatologist practice after a period of three months (upon the approval of the ethics committee). In total, 131 cases were collected after the three-month period of this phase.

#### **(d) Study Variables**

The data extraction form was developed to be one page in length; this ensured that the most accurate data were captured within the least amount of time. The following data was captured on this form:

- Demographic data
  - Gender
  - Date of birth
  - Race
  - Geographical region
- Medical history
  - Chronic disease including, TB, HIV/AIDS or chemotherapy
- Current dermatological disease
  - Diagnosis
  - Observation of skin lesion
  - Treatment

- Treatment duration
- Follow-up results

The following data were not collected:

- Personal details, for example patient's name and address.
- Family and personal medical history not relevant to dermatology.
- Prescribing doctor's personal details or name.

### 3.4 DATA ANALYSIS

The data during this phase of the research study were processed using a variety of statistical tools and calculations. The data were captured in Microsoft Windows Excel 2010 version. Data analysis was performed in SAS Version 9.1.3 (SAS Institute, Cary, NC).

All statistical significance was considered with probability of  $p < 0.05$ . The practical significance of the results was computed when the  $p$ -value was statistically significant ( $p \leq 0.05$ ). Chi-square test ( $\chi^2$ ) was used to determine if an association existed between proportions of two or more groups. The Cramer's V statistics was used to test practical significance of this association.

#### Mean:

The average of various variables will be calculated within the different data sets. The average is obtained by dividing the sum of observed values by the number of observations, which is  $n$  (Kutner *et al.*, 2005:56).

$$\bar{X} = \frac{\sum_{i=1}^{i=n} X_i}{n}$$

#### Median:

The median is defined as the middle value of a set of data containing an odd number of values, or the average of the two middle values of a set of data with an even number of values (Kutner *et al.*, 2005:440).

**Chi-Square:**

A Chi-Squared test can be defined as a test that provides an estimate on the agreement between a set of observed data and a random set of data of which the measurements are expected to fit. It is calculated by summing the distances between the observed and random data. This distance depends on the magnitude of the values which is normalised by dividing them by the random value, as the equation shows. The chi-square can be interpreted as statistically significant from the random if the value is smaller than 0.05 (Johnson *et al.*, 2007:184; Steyn *et al.*, 2000:549).

$$\chi^2 = \sum_{k=1}^N \frac{(\text{observed} - \text{random})^2}{\text{random}}$$

**Cramer's V Coefficient:**

Cramer's V coefficient is useful for comparing multiple  $\chi^2$  test statistics and is used across contingency tables of varying size; hence the test is not affected by sample size. It is further defined as a measure of the relative strength in the association between two variables. The coefficient ranges from 0 to 1, which is a perfect association.

$$\phi_c = \sqrt{\frac{\varphi^2}{(k-1)}} = \sqrt{\frac{\chi^2}{N(k-1)}}$$

The strength of the association is measured as follows:

- >0.5            = strong association
- 0.3 - 0.5       = moderate association
- 0.1 - 0.3       = low association
- <0.1            = no association

### **3.5 ETHICAL CONSIDERATIONS**

This research study had to be accepted for ethical approval by the NWU ethical committee. Approval was granted. The reference number is: NWU-00061-12S5.

In this research project, human participants contributed to Phase 1A of the data collection. No physical danger or risks were associated with this project since the study design was mostly observational.

All information and data collected were done with permission of the participants. No legal authorisation was needed from authoritarian bodies for this specific phase of the research study. No permission or consent was needed from any group representatives for this specific phase of the research study as patients gave consent themselves. The data were collected in manner which prevented any individual to be specifically identified.

Data were captured from the different sources by use of a personal portable computer. The compiled data set was then sent to the research-entity, Medicine Usage in South Africa, Faculty of Health Science, North-West University, for statistical analysis in collaboration with Statistical Consultation Services at the same University.

All data collected were locked in a secure office until processed for statistical purposes. The original data were transferred onto an external disc and stored in a safe of the secure office at the North-West University.

### **3.6 BENEFITS TO THE PATIENT**

Patients with chronic skin disease will have the advantage of a better understanding of their condition and available therapies. In addition, the study will provide valuable information to other healthcare professionals regarding skin conditions and their treatment in the private healthcare sector of Namibia.

### **3.7 CHAPTER SUMMARY**

This chapter discussed the research methodology followed to achieve the stated empirical objectives were discussed. The various objectives were listed and the methods and rules used to analyse the different phases were discussed. At the end of this chapter, other factors that may have influenced the data, such as method of data analysis, ethical considerations and the benefits to the patient are taken into account.

## CHAPTER 4: RESULTS AND DISCUSSION

### 4.1 INTRODUCTION

The results of the empirical study that investigated the prevalence and treatment regimens of dermatological diseases and explored geographical and demographical trends are discussed in this chapter.

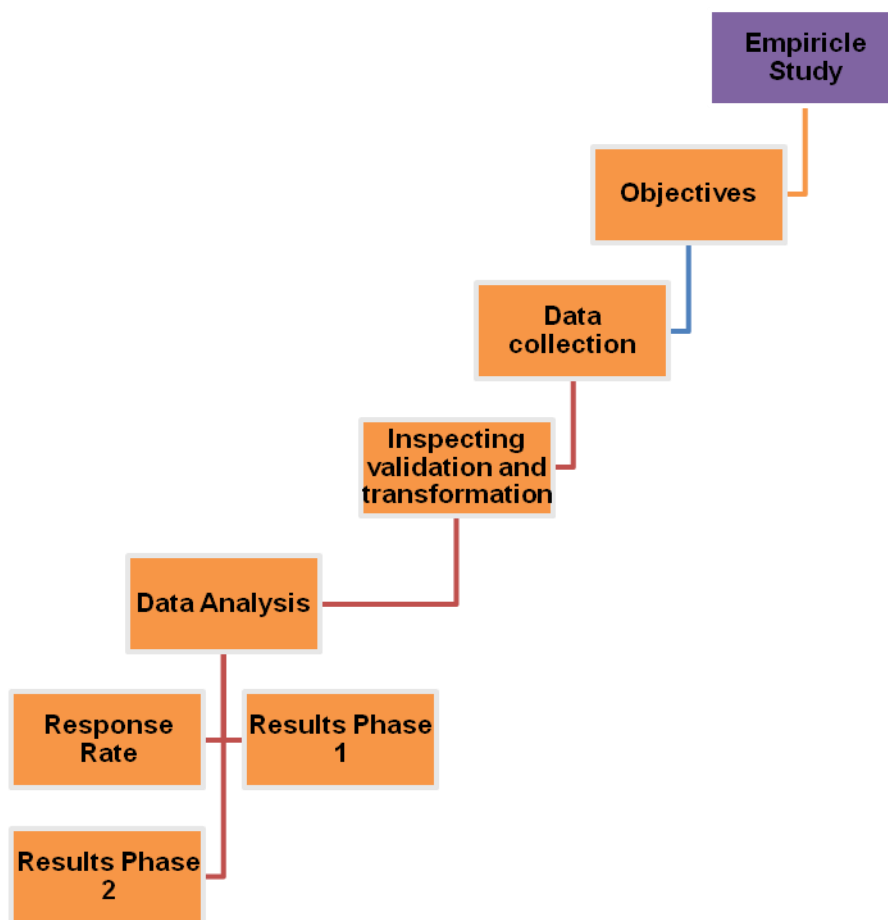


Figure 13: Empirical Study Process

#### 4.1.1 Response Rate

The research study was divided into two phases. A total number of 507 patients participated in this research study: 302 patients were included in Phase 1A, 74 patients in Phase 1B and 131 patients in Phase 2. The results of each phase are discussed and then integrated to draw a final conclusion in Chapter 5.



#### 4.1.2 Results of Phase 1A

The dermatological diagnosis was determined by the patient's general practitioner in each case during this phase of the study. Urticaria (n=36) showed the biggest contribution, while eczema and contact dermatitis had the same prevalence of 9.27% (n=28). The following chart summarises the different dermatological diseases for Phase 1A.

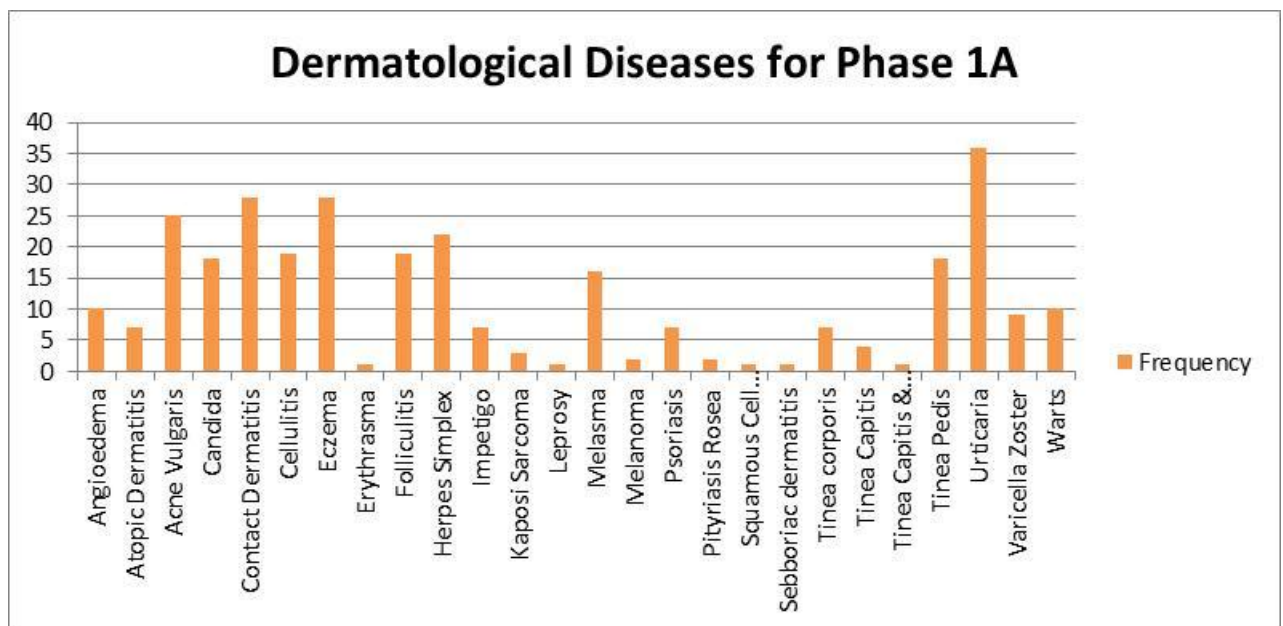


Figure 14: Dermatological Diseases in Phase 1A

Table 5: Dermatological Diseases in Phase 1A

Dermatological Disease	n	%
Acne vulgaris	25	8.28%
Angiodema	10	3.31%
Atopic dermatitis	7	2.32%
Candida	18	5.96%
Cellulitis	19	6.29%
Contact dermatitis	28	9.27%
Eczema	28	9.27%
Eythrasma	1	0.33%
Folliculitis	19	6.29%
Herpes simplex	22	7.28%
Impetigo	7	2.32%
Kaposi sarcoma	3	0.99%
Leprosy	1	0.33%
Melanoma	2	0.66%
Melasma	16	5.30%
Pityriasis rosea	2	0.66%
Psoriasis	7	2.32%
Squamous cell carcinoma	1	0.33%
Seborrheic dermatitis	1	0.33%
Tinea capitis	7	2.32%
Tinea capitis & eczema	1	0.33%
Tinea corporis	4	1.32%
Tinea pedis	18	5.96%
Urticaria	36	11.92%
Varicella zoster	9	2.98%
Warts	10	3.31%

It can be suggested from this graph that Windhoek does not have the typical African pattern of infective bacterial and fungal skin disease (Van Hees, 2001:2). Urticaria plays the most significant role in this section of the research study. Urticaria is more a description of a symptom than a disease in itself. It can be caused by internal factors such as bacterial infections, medication or food. External factors include injury (rubbing, scratching or slapping), exposure to chemicals and poisonous plants (Ramos-Romey *et al.*, 2008:32).

The growing building industry and development in Windhoek is considered to be a contributing factor to the incidence of contact dermatitis and urticaria (refer 2.8.4). Eczema, on the other hand, is a very common skin disease. Eczema is worsened by excessive dry conditions (Proksch *et al.*, 2006:159). As already mentioned, some studies also consider the use of Vaseline® as moisturiser, a causative agent for eczema (Van Hees, 2001:11).

#### 4.1.2.1 Demographic results

Of the 302 patients that participated, 68.21% (n = 206) were from the southern parts of Windhoek and 31.79% (n=96) were from the northern parts of Windhoek.

##### (a) Age Groups

The prevalence of patients in the different age groups was as follows

Table 6: Age Group Distribution for Phase 1A.

Age Group	n	%
≤2 years	3	0.99
>2 and ≤ 6 years	7	2.31
>6 and ≤ 12 years	26	8.60
>12 and ≤ 60 years	218	72.18
>60 years	48	15.89

The value for the Chi-square test ( $\chi^2$ ) was 170.97 with a *p*-value of <0001. This indicates a statistical significant association between the variables. The value for Cramer's V was 0.3762, which shows moderate association (moderate practical significance) between variables.

Table 7: Dermatological Diseases according to the Age Groups for Phase 1A.

Dermatological Disease	≤2 years	>2 - ≤ 6 years	>6 - ≤ 12 years	>12 - ≤ 60 years	>60
Acne vulgaris			6 (23.07%)	19 (8.71%)	
Angiodema				9 (4.12%)	1 (2.08%)
Atopic dermatitis				7 (3.21%)	
Candida				15 (6.88%)	3 (6.25%)
Cellulitis				16 (7.33%)	3 (6.25%)
Contact dermatitis			3 (11.53%)	21 (9.63%)	4 (8.33%)
Eczema	3 (100%)	4 (57.14%)	7 (26.92%)	10 (4.58%)	4 (8.33%)
Erythrasma				1 (0.45%)	
Folliculitis			2 (7.69%)	14 (6.42%)	3 (6.25%)
Herpes simplex			1 (3.84%)	18 (8.25%)	3 (6.25%)
Impetigo		2 (28.57%)	2 (7.69%)	3 (1.37%)	
Kaposi sarcoma				2 (0.91%)	1 (2.08%)
Leprosy				1 (0.45%)	1 (2.08%)
Melanoma				1 (0.45%)	1 (2.08%)
Melasma				12 (5.50%)	4 (8.33%)
Pityriasis rosea				1 (0.45%)	1 (2.08%)
Psoriasis				5 (2.29%)	2 (4.16%)
Seborrheic dermatitis				1 (0.45%)	
Squamous cell carcinoma				1 (0.45%)	
Tinea capitis			1 (3.84%)	5 (2.29%)	1 (2.08%)
Tinea capitis & eczema			1 (3.84%)		
Tinea corporis		1 (14.28%)	2 (7.69%)		1 (2.08%)
Tinea pedis				16 (7.33%)	2 (4.16%)
Urticaria				26 (11.92%)	10 (20.83%)
Varicella zoster				6 (2.75%)	3 (6.25%)
Warts			1 (3.84%)	9 (4.12%)	

**Age group: ≤2 years**

Atopic eczema is the only dermatological disease in this age group. Atopic eczema is common in infants and mostly atopic. Literature describes eczema to almost always initiate before the age 5 years (Möhrenschlager *et al.*, 2006:503).

**Age group: >2 and ≤ 6 years**

Atopic eczema is also the most common dermatological diseases in this sector. At this age, atopic eczema can be linked to allergens like different food types, cosmetic chemicals and dust (Möhrenschlager *et al.*, 2006:503).

**Age group: >6 and ≤ 12 years**

Atopic eczema is again the most common dermatological diseases in this sector. Contact dermatitis is the second most significant disease in this age group. This is probably due to the fact that children at this age explore the outdoors more often and are more likely to come into contact with unfamiliar substances.

**Age group: >12 and ≤ 60 years**

Urticaria showed the highest prevalence in this age group. Contact dermatitis and Acne vulgaris followed. This is the biggest age group and classifies the working-class adult. Urticaria and contact dermatitis are often a result of exposure to chemicals and other harmful substances. According to Taylor & Amando (2010:1), 4350 of the 85 000 harmful chemicals in our environment can cause contact dermatitis and urticaria. But according to a study done by the University of Puerto Rico, 75 - 90% of the etiology of urticaria is unknown (Romey-Ramos *et al.*, 2008:100).

**Age group: >60 years**

Urticaria is once again the dermatological disease with the highest prevalence. A study done in Italy concluded that urticaria is mainly caused by the high number of drugs taken by the elderly, followed by internal pathologies typically seen in elderly patients due to the changes in immunity and metabolism (Ventura *et al.*, 2012:530).

It can be concluded that the children who participated in this phase suffered mostly from atopic eczema. Urticaria showed the highest prevalence among adults and elderly patients.

**(b) Gender**

The number of females who participated in this phase were slightly less at 49.34% (n= 149) compared to the number of males (n=153; 50.66%).

The following table summarises the relationship between the different dermatological diseases and gender. The most significant relationships are discussed. The value for the Chi-square test ( $\chi^2$ ) for this data was 47.76 with a p-value of 0.004. This association was statistically significant. The value for Cramer's V was 0.3977, which indicates a moderate practical significant association between the gender of the patient and dermatological disorders.

Table 8: Dermatological Diseases according to Gender for Phase 1A

Dermatological Disease	Female	%	Male	%
Acne vulgaris	14	9.40	11	7.19
Angiodema	2	1.34	8	5.23
Candida	14	9.40	4	2.61
Cellulitis	8	5.37	11	7.19
Contact dermatitis	14	9.40	14	9.15
Eczema	15	10.07	20	13.07
Folliculitis	11	7.38	8	5.23
Herpes simplex	10	6.71	12	7.84
Impetigo	4	2.68	3	1.96
Kaposi sarcoma	1	0.67	2	1.31
Leprosy	0	0.00	1	0.65
Melasma	15	10.07	1	0.65
Melanoma	0	0.00	2	1.31
Pityriasis rosea	2	1.34	0	0.00
Psoriasis	1	0.67	6	3.92
Seborrheic dermatitis	0	0.00	1	0.65
Tinea capitis	4	2.68	3	1.96
Tinea capitis & eczema	1	0.67	0	0.00
Tinea corporis	0	0.00	4	2.61
Tinea pedis	6	4.03	12	7.84
Urticaria	20	13.42	16	10.46
Varicella zoster	4	2.68	5	3.27
Warts	3	2.01	7	4.58
Erythrasma	0	0.00	1	0.65
Squamous cell carcinoma	0	0.00	1	0.65

## Females

Urticaria showed the highest prevalence with 13.42% in this gender group. Deacock (2008:151) supports this find in his study which indicates that women are more likely to suffer from urticaria than men. Only 10.46% of men suffered from urticaria.

Melasma also indicated a high incidence among women with 10.07%. Melasma is more prevalent in women due to changes in hormones, such as progesterone during pregnancy (Barankin *et al.*, 2002:236).

Eczema showed the same high prevalence among women as melasma (10.07%), but in this phase of the research study, men were more prone to have eczema (13.07%) than women. Literature explains that certain types of eczema are more prone in men or women. Women are more likely to get eczema on their hands, and men to develop discoid eczema (Agner *et al.*, 2012:2 & Kubeyinje, 1995:416).

Seventy-seven per cent (n=14) of the patients diagnosed with cutaneous candida were women (refer to 2.7.3.7). The candida fungus is naturally present on the skin. The immunological system of the skin keeps the fungus growth limited. Candida overgrowth takes place if the immunological system is not properly functioning, like in the case HIV/AIDS (Montejo, 2012:1). Literature does suggest women get candida infection more often due to hormonal changes that alternate the body's flora growth, oxidation rate and copper balance (Wilson, 2012:1).

## **Males**

Eighty per cent (N=8) of the patients diagnosed with angioedema were men which reveals, a high incidence among men. According to Cole, (2013:1) hives are more common in women than in men. Although the etiology is unknown, statistics in a study done by Brostoff concludes that men show a higher prevalence (Brostoff *et al.*, 2011:2).

Eighty-five per cent of patients (n=6) diagnosed with psoriasis were men. Literature, however, suggests that psoriasis is equally distributed among men and women (Traup, 2007:319). A study one by Hagg (2013:1) suggests that men get more severe psoriasis than women. It can only be speculated that the extreme dry conditions in Namibia plays a role in men having a higher prevalence.

Sixty-six per cent (n=12) of patients with tinea pedis were men. It is thought that due to their work, men are more likely to wear tight, closed, fitted shoes than women; thus, the fact that men have a higher prevalence is not due to their gender but to the nature of their work. The same can be said about the high incidence of melanoma (1.3%) among men. According to Tuong *et al.* (2012:113), melanoma is more common in men than woman and this difference in ration is increasing annully. The



commercial farmer in Namibia – mostly men - spends many hours outside in the sun. The extreme UV radiation is thought to be the cause of the high incidence in melanoma.

Tinea corporis also indicates a high incidence among men (2.61%) compared to women. According to Hainer (2003:2), men are more prone to get dermatophyte infections in general than women. It is thought those women are more hygienic and spend more time daily on personal hygiene, than men.

### (c) Race

The results reveal that the majority of patients, 57.28% (n=173), consulting the community pharmacy with dermatological conditions, were black, 25.82% (n=78) were white, 14.23% (n=43) colored; 2.31% (n=7) of patients black albino and 0.33% (n=1) coloured albino.

Table 9: Race Frequency for Phase 1A

RACE	n	%
Black	173	57.28
Caucasian	78	25.82
Coloured	43	14.23
Albino-black	7	2.31
Albino-coloured	1	0.33

Literature has indicated the difference between Caucasian and ethnic skin types - these skin types do not only differ in colour, but also in physiology and responsiveness (refer to 2.8.10). Culture practices and habits play a further significant role in dermatological diseases (Gawgrodger, 2012:215).

The following tables summarise the different races and the frequency in which each type of dermatological disease has occurred in this phase of the research study.

Table 10: Dermatological Diseases in Patients of Phase 1A

Dermatological Disease	Albino Black	%	Albino Coloured	%	Black	%	Coloured	%	Caucasian	%
Angioedema					9	5.20	1	1.28		0.00
Atopic dermatitis					4	2.31	3	3.85		0.00
Acne vulgaris					16	9.25	5	6.41	4	9.30
Candida	2	28.57			10	5.78	2	2.56	4	9.30
Cellulitis					15	8.67	3	3.85	1	2.33
Eczema	1	14.28			7	4.05	14	17.95	6	13.95
Erythrasma					1	0.58		0.00		0.00
Folliculitis			1	100	10	5.78	5	6.41	3	6.98
Herpes simplex					13	7.51	4	5.13	5	11.63
Impetigo					4	2.31	1	1.28	2	4.65
Kaposi sarcoma					3	1.73		0.00		0.00
Leprosy					1	0.58		0.00		0.00
Melasma	1	14.28			9	5.20	3	3.85	3	6.98
Melanoma					1	0.58	1	1.28		0.00
Psoriasis					1	0.58	5	6.41	1	2.33
Pityriasis rosea	1	14.28			1	0.58		0.00		0.00
Squamous cell carcinoma					1	0.58		0.00		0.00
Seborrheic dermatitis						0.00	1	1.28		0.00
Tinea corporis					7	4.05		0.00		0.00
Tinea capitis					4	2.31		0.00		0.00
Tinea capitis & eczema						0.00		0.00	1	2.33

<b>Tinea pedis</b>					10	5.78	4	5.13	4	9.30
<b>Urticaria</b>					17	9.83	15	19.23	4	9.30
<b>Varicella zoster</b>	1	14.28			5	2.89	2	2.56	1	2.33
<b>Warts</b>	1	14.28			7	4.05	2	2.56		0.00

The value for the Chi-square for this data was 118 with a p-value of 0.09. The value for Cramer's V was 0.3134, which indicates moderate association between variables.

### **Albino black patients**

Candida has shown to have the biggest contribution in this part of the research study. Literature, however, does not indicate that there exist a correlation between albinism and candidiasis.

Other dermatological diseases documented in this section were warts, Varicella zoster infection, eczema, Pityriasis rosea and melasma, of which the latter is surely an error. Albino is defined as the lack of melanin due to the absence of the copper containing enzyme tyrosine which is necessary to produce melanin (Miyamura *et al.*, 2005:397). Because the patients do not have any melanin, they cannot suffer from melasma. It can be concluded that the diagnosis of the general practitioner was either wrong, or that the patient was not completely albino, rather suffered from vitiligo.

### **Albino coloured patients**

Only one patient in this research study has been defined as an albino coloured. This patient was suffering from folliculitis. She was 32 years of age without any prior occurrence of folliculitis. She was unemployed at the time. She is not HIV positive or has any other chronic disease. It can be concluded that the folliculitis was caused by irritation to the follicles, most likely due to shaving or the wearing of tight clothes. No correlation between albinism and folliculitis was found.

### **Black patients**

Urticaria and contact dermatitis have occurred most often in the black population in this phase of the research study. People who can afford private doctors' consultation are mostly the working middle class. Of the black patients diagnosed with contact dermatitis (n=17), 58% were men (n = 9) and only 42% were women (n=7). The exact opposite was found in the black patients diagnosed with urticaria (n=17): 58% (n=10) were women and 42% (n=7) men.

Literature explains that urticaria and contact dermatitis have a strong correlation with chemical substances used in the industrial work place (Brehler, 1997:125). Urticaria, which naturally is more descriptive of a symptom than a disease can, however, be

caused by many different external and internal factors, like detergents and medications. Allergies are a common cause of urticaria (Soter, 1991:146).

### **Caucasian patients**

Eczema and urticaria are the most common cause of dermatological disease in Caucasian patients. In Caucasian patients, 17.95% had eczema compared to 4.05% of black patients. Urticaria in caucasians peaked at 19.23% compared to black patients 9.83%.

Urticaria has a much undefined aetiology, as already discussed. Solar urticaria is common under Caucasian and coloured people. This is a condition where sensitivity to UV radiation causes urticaria (Lucovic *et al.*, 2008:153). Literature does, however, support that caucasians are more likely to be diagnosed with urticaria, than other races (Kerr, 227:638).

Like urticaria, eczema is caused by various factors, including allergens, weather changes, exposure to certain substances and even stress (Engin *et al.*, 2007:36). Literature indicates that black patients are more prone to dry skin than white patients. This is due to the fast rate of spontaneous desquamation in black patients; it is 2.5 times faster than Caucasian skin (Gawkrodger, 2002:11).

### **Coloured patients**

In this race group, eczema (13.95%) and herpes simplex (11.63%) have shown the highest prevalence. As mentioned, literature supports that black patients have a higher incidence of eczema than lighter-skinned people. Herpes simplex is caused by a virus which is highly contagious. No significant correlation between herpes simplex and coloured skin type has been found in this study or in literature.

#### 4.1.2.2 Medical conditions contributing to dermatological problems:

##### (a) Allergies

Most patients who participated in this phase, 66.23% (n= 200) were not allergic to anything. Allergies were, however, present for the following variables:

Table 11: Frequency for Allergies for Phase 1A

TYPE OF ALLERGY	n	%
Bee sting	4	3.92%
Cephalosporin's	1	0.98%
Dog/Cat hairs	7	6.86%
Dust	19	18.62%
Eggs	1	0.98%
Grass	2	1.96%
Grass, dust	15	14.07%
Grass, dust, dog/cat hairs	7	6.86%
Lactose	1	0.98%
Morphine	3	2.94%
Non-steroid anti-inflammatories	6	5.88%
Peanuts	2	1.96%
Penicillin	19	18.62%
Preservatives	2	1.96%
Seafood	7	6.86%
Seafood, Bee sting	1	0.98%
Sulphonamides	5	4.90%

These results reveal that the most common allergies were those to dust, grass and penicillin (n=53).

An allergic reaction is a process by which the immune system is activated. The skin is the largest organ of the immune system (Blauvelt *et al.*, 2003:560). As already discussed in Chapter 2.4, the skin has both adaptive and non-adaptive immune systems. Literature suggests that the skin will present itself with various symptoms such as urticaria and pruritus if an immune response, in the form of an allergy, is

stimulated (Blauvelt *et al.*, 2003:560). It is not clear from these results if a correlation exists between dermatological diseases and the tree major allergens, but such correlation is expected.

It is well known that the skin is the largest organ of the body. Exposure to different allergens often presents symptoms visible on the skin. Many correlations can thus be made between different allergens and dermatological disease. The table below summarises the data for Phase 1A.

Table 12: Table demonstrating allergies vs dermatological disease

Dermatological Condition	Food	Grass, dust, animal hairs	Drugs	Preservatives	Bee sting
Angioedema	1	1	0	0	0
Atopic dermatitis	0	2	0	0	0
Acne vulgaris	0	3	2	0	0
Candida	1	3	0	0	0
Contact dermatitis	1	4	4	0	2
Cellulitis	1	5	0	0	0
Eczema	0	4	3	0	1
Erythrasma	0	1	0	0	0
Folliculitis	1	3	3	1	0
Herpes simplex	0	6	3	0	0
Impetigo	0	1	2	0	0
Kaposi sarcoma	0	0	0	0	0
Leprosy	0	0	0	0	0
Melasma	1	4	2	0	0
Melanoma	0	0	0	0	0
Psoriasis	0	0	0	0	0
Pityriasis rosea	0	0	0	1	0
Squamous cell carcinoma	0	1	0	0	0
Seborrheic dermatitis	0	0	0	0	0
Tinea corporis	0	2	1	0	0
Tinea capitis	0	0	2	0	0
Tinea capitis & eczema	0	0	0	0	1
Tinea pedis	2	0	7	0	1
Urticaria	1	5	4	0	0
Varicella zoster	2	1	1	0	0
Warts	0	4	0	0	0

Contact dermatitis showed the highest incidence in allergies (n=11). These allergies included dog and cat hairs, dust, grass, penicillin and sulphonamides, and even bee sting. Allergic contact dermatitis has been extensively described in the literature study of this dissertation. It can be concluded that the allergies like those to animal hairs and grass have a correlation to contact dermatitis (refer to 2.7.1.2).

Urticaria also showed a prevalence (n=10) as literature predicts. The allergies in Phase 1A include those to dust, grass, seafood and medication such as NSAIM's and Penicillin. Chronic urticaria has been defined as urticaria when sustained for period longer than 6 weeks (Maurer, 2013:924). This occurs most frequent as a result of the continuous exposure to an allergen. It can therefore be concluded that urticaria can have a relation to exposure to allergens.

#### **(b) Cigarette smoke**

Thirty-three per cent (n=69) of the patients who participated in this phase of the research study smoked cigarettes.

The following table provides a summary of dermatological diseases which are prevalent in cigarette smokers.



Table 13: Dermatological Diseases in Smokers of Phase 1A

Dermatological Disease	n	%
Angioedema	3	3.03
Atopic dermatitis	1	1.01
Acne vulgaris	1	1.01
Candida	10	10.10
Contact dermatitis	9	9.09
Cellulitis	6	6.06
Eczema	2	2.02
Erythrasma	1	1.01
Folliculitis	8	8.08
Herpes simplex	5	5.05
Impetigo	1	1.01
Kaposi sarcoma	2	2.02
Melasma	6	6.06
Melanoma	1	1.01
Psoriasis	3	3.03
Pityriasis rosea	1	1.01
Seborrheic dermatitis	1	1.01
Tinea capitis	1	1.01
Tinea pedis	13	13.13
Urticaria	15	15.15
Varicella zoster	4	4.04
Warts	5	5.05

Thirteen per cent (n=13) of patients who smoked also experienced occurrence of *tinea pedis* and *candida albicans*, 10.10% (n=10). Literature suggests that smoking does have a negative effect on anti-bodies and the immune system in general, because cigarette smoke depletes vitamin C in the body (Schechtman *et al.*, 1989:158). It is safe to conclude that people who smoke will get more infections in general. The literature does not suggest a direct correlation between smoking and *tinea pedis*, *candida albicans* or folliculitis. Some smokers do, however, develop contact dermatitis from cigarette smoking. The allergic reaction usually takes place on their hands or around the mouth area (refer to 2.8.8).

Urticaria indicated the highest prevalence in smokers, but the literature does not predict any correlation. Urticaria has shown high prevalence throughout the study. Cigarette smoke is not considered part of the etiology of urticaria.

### **(c) Pregnancy and Breastfeeding**

Many women experience dermatological problems during pregnancy (n=24), this is mainly due to increasing levels of progesterone. Twenty-nine per cent (n=7) of the pregnant women who participated in this research study suffered from melasma. Folliculitis, urticaria and cellulitis all showed a high prevalence of 12.5% (n=3). Pruritic folliculitis in pregnancy has been well described in literature (refer to 2.8.9). This condition is pregnancy-specific and will clear after delivery. The literature indicates a strong relationship between melasma and folliculitis in pregnancy which is supported by the data.

Only 7.95% (n=24) of the patients were breastfeeding.

Table 14: Dermatological Diseases in Pregnant Women in Phase 1A

<b>Dermatological Diseases for Phase 1A</b>	<b>Pregnancy (n=24)</b>	<b>%</b>
<b>Candida</b>	2	8.33
<b>Contact dermatitis</b>	2	8.33
<b>Cellulitis</b>	3	12.50
<b>Eczema</b>	2	8.33
<b>Folliculitis</b>	3	12.50
<b>Melasma</b>	7	29.16
<b>Tinea corporis</b>	1	4.16
<b>Urticaria</b>	3	12.50
<b>Warts</b>	1	4.16

The value for the Chi-square for this data was 42.42 with a probability value of 0.0162. The value for Cramer's V was 0.3748.

### **(d) HIV/AIDS**

The results reveal that 16.98% of the patients were HIV positive.

Human Immunodeficiency Virus decreases the amount of active CD4 – T cells in the body. The decrease of these lymphocytes results in opportunistic diseases to cause HIV/AIDS (Weiss, 1993:1273). The table below compares the number of HIV positive patients with the different dermatological diseases. The value for the chi-square for this data was 57.44 with a probability value of 0.0002, which indicates that the association between the HIV/AIDS statuses and the type of dermatological disorders was statistically significant. The value for Cramer's V was 0.4361 indicating a practical significant association between patients with HIV/AIDS and the type of dermatological disease.

Table 15: Prevalence of Dermatological Disorders in HIV Positive Patients: Phase 1A

<b>Dermatological Disease</b>	<b>Number of HIV positive patients</b>	<b>%</b>
<b>Angioedema</b>	2	3.92
<b>Acne vulgaris</b>	2	3.92
<b>Candida</b>	4	7.84
<b>Contact dermatitis</b>	2	3.92
<b>Cellulitis</b>	3	5.88
<b>Eczema</b>	1	1.96
<b>Folliculitis</b>	1	1.96
<b>Herpes simplex</b>	8	15.69
<b>Impetigo</b>	4	7.84
<b>Kaposi sarcoma</b>	3	5.88
<b>Leprosy</b>	1	1.96
<b>Melasma</b>	4	7.84
<b>Tinea corporis</b>	1	1.96
<b>Tinea capitis</b>	1	1.96
<b>Tinea capitis &amp; eczema</b>	1	1.96
<b>Tinea pedis</b>	2	3.92
<b>Urticaria</b>	6	11.76
<b>Varicella zoster</b>	1	1.96
<b>Warts</b>	4	7.84

*Herpes simplex* showed the highest prevalence in HIV positive patients (n=8). *Herpes simplex* is a common opportunistic disease since it is very contagious and remains life-long in its host (Strick *et al.*, 2006:347). The results support this finding. Urticaria also showed high prevalence under HIV patients (n=6). Literature does confirmed that HIV positive patients do develop urticaria quite commonly (refer 2.8.1). Literature also suggests that patients on antiretroviral therapy may develop urticaria as adverse drug reaction (Lin *et al.*, 2006:465).

Dermatological conditions like warts, impetigo and candida are evidence of opportunistic diseases in HIV/AIDS patients. These diseases have a possible association with the fact that the patients are HIV positive (Knott, 2011:1).

#### **(e) Chronic disease**

Twenty-six per cent (n=122) of the patients who participated in this phase were suffering from chronic diseases.

Table 16: Frequency of Chronic Diseases in Phase 1A

VARIABLE	n	%
Addison's disease	1	1.19
Asthma	8	9.52
Crohn's disease	1	1.19
Depression	2	2.38
Diabetes mellitus	7	8.33
Epilepsy	1	1.19
Glaucoma	2	2.38
Hypertension	18	21.43
Hypertension, glaucoma	1	1.19
Hypertension, cardiac failure, hyperlipidaemia	1	1.19
Hypertension, hyperlipidaemia	27	32.14
Hypertension, hyperlipidaemia, diabetes	2	2.38
Hypertension, hyperlipidaemia, hypothyroidism	1	1.19
Hypothyroidism	1	1.19
Osteoporosis	3	3.57
Parkinson's	1	1.19
Rheumatoid arthritis	2	2.38

Chronic medication data were also collected to measure the association between different dermatological diseases and the chronic medication taken by patients. Patients with chronic diseases mostly experienced the following dermatological problems: cellulitis, folliculitis, herpes simplex, tinea pedis and urticaria.

### Cellulitis

Three-and-a-half per cent of the patients who participated in this phase of the research project and used chronic medication, were diagnosed with cellulitis. The table below indicates the frequency of cellulitis patients on chronic medication.

Table 17: Chronic Medication in Patients Diagnosed with Cellulitis

Chronic medication	n	%
Hypotensive lipids	1	4.00
Statin	4	16.00
Cardio aspirin	3	12.00
Serotonin re-uptake inhibitor	1	4.00
Metformin	1	4.00
Sulphonylureas	1	4.00
Insulin	1	4.00
Diuretics	1	4.00
Antiretroviral drugs	2	8.00
Anti-epileptic drugs	1	4.00
Calcium channel blockers	1	4.00
Thionaimde	2	8.00
Calcium suppliment	1	4.00
ACE-Inhibitors	5	20.00

Medication that will suppress the immune system forms part of the etiology of cellulitis. Antiretroviral drugs in this case can be considered a causative agent according to literature (Radhakrishnan *et al.*, 2010:84).

Patients with peripheral artery disease (PAD) are at larger risk to develop cellulitis. The list of chronic medication above gives a clear indication that many patients are suffering from or will be potential candidates for PAD, hence the combination of statins and cardio aspirin (Vinh, 2007:418).

### Folliculitis

Almost three-and-a-half per cent of the patients who participated in this phase of the research study used various chronic medications and were diagnosed with folliculitis.

Table 18: Chronic Medication in Patients Diagnosed with Folliculitis.

Chronic medication	n	%
ACE-Inhibitors	4	17.39
Beta blockers	1	4.35
Statins	3	13.04
Proton pump inhibitors	1	4.35
Corticosteroids	1	4.35
Metformin	2	8.70
Sulphonylureas	2	8.70
Insulin	1	4.35
Multivitamin	1	4.35
Antiretroviral drugs	2	8.70
Oral contraceptives	1	4.35
Calcium channel blockers	2	8.70
Alpha agonists	1	4.35
NSAIM's	1	4.35

Drug-induced folliculitis is well-defined in literature (refer to 2.7.7.3). In this phase of the research study, it can be concluded that those patients on beta-blockers, androgenic contraceptives and corticosteroid treatment might have drug induced folliculitis and not bacterial. The diagnostic factor will be the absence of comedones and this is unfortunately not captured in the data.

Chronic medications such as ACE-inhibitors and statins do show the highest prevalence, but there has been no studies indicating folliculitis. Literature does not suggest any other correlations between folliculitis and other types of chronic medication neither do the results.

### ***Herpes simplex***

Five per cent of the patients that participated in this part of the research study and used chronic medication were diagnosed with *Herpes simplex*.

The table below summarises the chronic medication used by those people diagnosed with *Herpes simplex*.

Table 19: Chronic medication for patients diagnosed with *Herpes simplex*

Chronic medication	n	%
ACE-Inhibitors	2	4.35
Hormone replacement therapy	1	21.74
Statins	5	4.35
Diuretics	1	4.35
Calcium supplements	1	8.70
Metformin	2	13.04
Cardio Aspirin	3	4.35
Insulin	1	8.70%
Calcium channel blocker	2	30.43%
Antiretroviral drugs	7	4.35%

It is well known that *Herpes simplex* is one of the first opportunistic diseases targeting HIV positive patients, hence the ARV treatment's prevalence in the above table. A study by Sun and Singh suggests that statins have an anti-viral effect on common viruses; thus the use of statins will not cause *Herpes simplex*, but can be used to treat long-term viral infections (Sun *et al.*, 2009:745). Neither literature nor data suggest that *Herpes simplex* shows a correlation with any of the above chronic medications.

### ***Tinea Pedis***

Almost four per cent of patients who participated in this research study and used chronic medication were diagnosed with *tinea pedis*. The table below summarises the amount of patients on chronic medications.



Table 20: Chronic Medication of Patients Diagnosed with *Tinea pedis*

Chronic medication	n	%
ACE-Inhibitors	3	13.04
Hormone replacement therapy	1	4.35
Statins	3	13.04
Diuretics	2	8.70
Calcium supplements	1	4.35
NSAIM's	1	4.35
Cardio Aspirin	2	8.70
Antiresorptives	1	4.35
Vitamin D	1	4.35
Proton-Pump inhibitors	1	4.35
Calcium channel blockers	2	8.70
Antitetroviral drugs	2	8.70
Sulphonylureas	1	4.35
Beta Blocker	1	4.35
Multivitamin	1	4.35

Literature does not suggest that *tinea pedis* is caused by any medication such as ACE-inhibitors or statins. The data do show correlation, but this is more likely due to the high prevalence of ACE-inhibitors and statins found the overall data.

## Urticaria

Four per cent of patients who participated in this research study and used chronic medication, were diagnosed with urticaria. The table below summarises the number of patients on various chronic medications.

Table 21: Chronic Medication of Patients Diagnosed with Urticaria

Chronic medication	n	%
ACE-inhibitors	2	9.52
Insulin	1	4.76
Statins	2	9.52
Diuretics	2	9.52
Beta blockers	1	4.76
NSAIM's	1	4.76
Corticosteroids	2	9.52
Antiretroviral drugs	4	19.05
Thionamide	1	4.76

The data suggest a relationship between urticaria and antiretroviral drug therapy. The literature explains that cutaneous adverse drug reactions are fairly common in patients receiving first-time ARV treatment or who are initiated on high activity drug treatment (Radhakrishnan *et al.*, 2010:84). Hypersensitivity towards medication is a common cause for a urticaria reaction to occur.

#### 4.1.2.3 Dermatological Diagnosis, Treatment, Treatment Durations

The dermatological diagnosis was determined by the prescribing physicians. The frequency is the number of patients in this phase that had the specific condition. The treatment was the one treatment that was prescribed most often (the mode average) by the different physicians for the specific condition. The duration is the mode period for which the treatment was prescribed, and the period waited, is the mode time before the patient consulted his/her prescribing physician. The table below provides a summary on dermatological diseases in Phase 1A:

The treatment prescribed most often by the general practitioners was a medium strength topical corticosteroid (n=38). The item prescribed second most was topical anti-bacterial ointment (n=32). In 90% of the cases, mupirocin was prescribed and in 10% cases, fucidic acid was prescribed.

Duration before seeing a healthcare professional varied depending on the type of dermatological disease; however, most of the patients had waited 2 days before seeing the general practitioner (n=96). Literature suggests that patients ignore dermatological diseases for a long period before seeking healthcare. In Windhoek, however, the contrary is evident; patients consulted their general practitioner within 2 days after noticing the dermatological condition.

Treatment duration depended solely on the diagnosis. The average treatment duration, independently of the dermatological problem for phase 1A, was 14 days (n=68). Almost fifty-one per cent of the patients visited their general practitioner for the first time for their dermatological problem. In other words, 49.2% of patients are consulted the general practitioner for second-line treatment

Table 22: Summary of dermatological diseases, frequency (f), treatment, duration (days waited) before seeking healthcare and duration of treatment thereof.

<b>Dermatological Diagnosis</b>	<b>f</b>	<b>Treatment</b>	<b>Treatment duration</b>	<b>Days waited</b>
<b>Angioedema</b>	10	Topical corticosteroid: Medium IV	5	2
<b>Atopic dermatitis</b>	7	Topical corticosteroid: Medium IV	14	4.5
<b>Acne vulgaris</b>	25	Oral retinoid + topical macrolide/zinc combination	90	30
<b>Candida</b>	18	Anti-fungal vaginal cream	7	2
<b>Contact dermatitis</b>	28	Topical corticosteroid: High III	14	2
<b>Cellulitis</b>	19	Penicillin + topical anti-bacterial + anti-septic solution	14	7
<b>Eczema</b>	28	Topical corticosteroid: Medium IV	14	10
<b>Erythrasma</b>	1	Topical lincosamides + Whitfield's ointment	30	2
<b>Folliculitis</b>	19	Topical antibiotic + combination Penicillin	14	10
<b>Herpes simplex</b>	22	Topical anti-viral	5	2
<b>Impetigo</b>	7	Penicillin + topical anti-bacterium + 4th generation antihistamine + NSAID's	14	2
<b>Kaposi sarcoma</b>	3	Analgesic	75	10
<b>Leprosy</b>	1	Dapsone + anti-mycobacterium	60	30
<b>Melasma</b>	16	Topical retinoid + 5% hydroquinone + topical corticosteroid: Medium IV	90	8
<b>Melanoma</b>	2	Analgesic	60	8.5
<b>Psoriasis</b>	7	Topical corticosteroid: High II + salicylic acid + oral corticosteroid	30	8.5
<b>Pityriasis rosea</b>	2	Topical corticosteroid: High III + topical Macrolide	12	7.5
<b>Squamous cell carcinoma</b>	1	Analgesic + sunscreen	14	10
<b>Seborrheic dermatitis</b>	1	Anti-Septic shampoo + topical anti-bacterium	7	1
<b>Tinea corporis</b>	7	Topical anti-fungal	14	2.5
<b>Tinea capitis</b>	4	Anti-fungal shampoo + topical anti-fungal	21	5
<b>Tinea capitis &amp; eczema</b>	1	Epi-zone E + topical corticosteroid: High II	30	10

<b>Tinea pedis</b>	18	Topical fungicide	7	2
<b>Urticaria</b>	36	Topical corticosteroid: Low VI	5	2
<b>Varicella zoster</b>	9	Oral anti-viral + topical anti-Viral + analgesic	14	4.5
<b>Warts</b>	10	Aldara	11	2

The three most significant dermatological diseases are now be discussed, namely urticaria, contact dermatitis and eczema:

### **Urticaria**

Almost twelve per cent of the patients were diagnosed with urticaria. This disease was treated with low potency topical corticosteroid over a period of 5 days. Most patients diagnosed with urticaria had waited two days before seeking healthcare. Urticaria persisting longer than 6 weeks is considered chronic urticaria (refer to 2.7.9). Acute urticaria on the other hand, normally clears within 2-3 days after corticosteroid treatment. Low potency corticosteroid is normally used when the cause of the urticaria is minor. The data does, however, do show that 52% of the patients did not visit the doctor for the first time about their condition. The urticaria could have been reoccurring (due to exposure to allergens) or even defined as chronic. This exact data were however not collected, nor was the fact whether the patient visited the same doctor every time or tried to get a second opinion.

### **Contact Dermatitis**

Nine per cent of the patients were diagnosed with contact dermatitis. The disease was treated with a high potency corticosteroid over a period of 14 days. Most of the patients had waited 2 days before seeking healthcare. Contact dermatitis is defined as a condition when the skin reacts due to exposure to foreign antigen (refer to 2.7.1.2). Certain plants, soaps, detergents and even jewellery are examples of common antigens. The treatment period for contact dermatitis was fairly long compared to urticaria. It is unclear why the treatment period was 14 days. According to literature, it is treated with corticosteroid only until the reaction disappears and the causative agent is identified and avoided. 42% of the patients consulted their doctor previously for the first time condition.

## **Eczema**

Nine per cent of patients were diagnosed with eczema. The disease was mostly treated with medium strength corticosteroids over a period of 14 days. Most patients had waited 2 days before seeking healthcare.

In an interview with the dermatologist, it became evident that eczema is common in Namibia and especially Windhoek. The mica rock, which is shimmering rock formations in the mountains around Windhoek, as well as dry weather conditions, contribute to eczema.

Forty-six per cent of the patients did not see the doctor for first-line treatment.

In all three these significant conditions it is clear that many patients went to the doctor for a second opinion or second-line treatment regime. The most common differential diagnosis for eczema is psoriasis, tinea infections, pityriasis rosea and pityriasis vesicolour (Wedi & Kapp, 2007:100). The differential diagnosis for contact dermatitis is asteatotic eczema, drug-Induced bullous disorders and photosensitivity, onycholysis, seborrheic dermatitis and tinea corporis (Taylor & Amando, 2010:1). The differential diagnosis for urticaria is angioedema, contact dermatitis, thrombophlebitis, drug allergies and serum sickness (Peroni *et al.*, 2005:541).

For chronic conditions such as the eczema, a corticosteroid was prescribed, which will definitely relieve the symptoms, but reoccurrence is inevitable if treatment is stopped. The corticosteroids are prescribed for unusually long period, which is unexplained. It can therefore be concluded that the main reason for the high percentage of patients receiving second line therapy for the same conditions, is the use of corticosteroid as a broad spectrum treatment without proper and effective diagnosis or referral to specialist dermatologist.

### 4.1.3 Results of phase 1B

The dermatological diagnosis was determined for each patient that participated in the phase. Contact dermatitis (n=15) and eczema (n=14) were the most common among the patients and accounted for 39.7% of the identified dermatological problems. Urticaria and psoriasis also indicated a high prevalence. The following table summarises on the different dermatological diseases for Phase 1B.

Table 23: Dermatological Diseases Identified in Phase1B

Dermatological disease	n	%
Angiodema	1	1.35
Candida	4	5.41
Cellulitis	2	2.70
Contact dermatitis	15	20.27
Eczema	14	18.92
Folliculitis	4	5.41
Melasma	1	1.35
Psoriasis	6	8.11
Tinea capitis	3	4.05
Tinea corporis	4	5.41
Tinea pedis	4	5.41
Urticaria	8	10.81
Varicella zoster	3	4.05
Warts	5	6.76

Figure 15 represents the percentage of patients suffering from certain dermatological diseases. Angioedema and melasma showed the lowest prevalence in this phase.

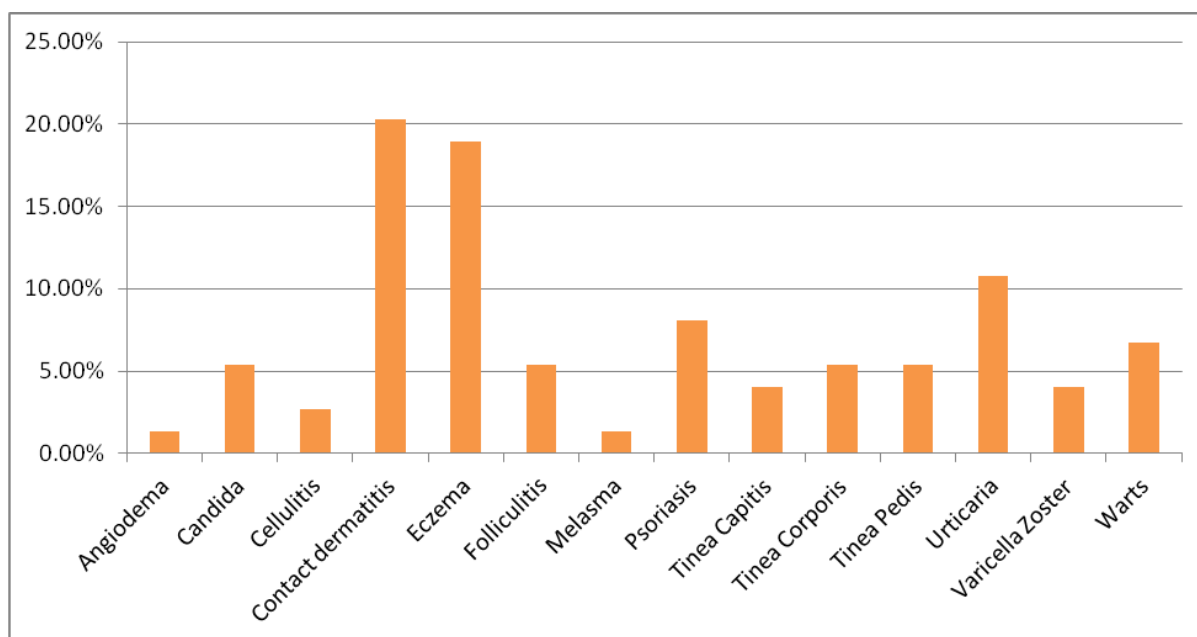


Figure 15: Percentage of Patients Suffering from Dermatological Diseases in Phase 1B.

#### 4.1.3.1 Demographic Results

A total number of 74 patients participated in this phase. Data were collected from patients that consulted the pharmacy for pharmacist-initiated therapy or self-treatment of their dermatological problems.

##### (a) Age

The table presents the age of the different patients that participated in this phase of the research study.

Table 24: Age Groups for Phase 1B

AGE	n	%
≤2 years	0	0.00
>2 and ≤ 6 years	3	4.05
>6 and ≤ 12 years	5	6.75
>12 and ≤ 60 years	54	72.97
>60 years	12	16.21



The following table summarises the relationship between the age of the patient and the dermatological condition. The most significant relationships within each age group are discussed.

Table 25: Age Groups vs Dermatological Diseases Phase 1B

Dermatological Disease	≤2 years	>2 and ≤ 6 years	>6 and ≤ 12 years	>12 and ≤ 60 years	>60 years
Angioedema	0	0	0	1(1.85%)	0
Candida	0	0	0	3 (5.56%)	1(8.33%)
Contact dermatitis	0	0	0	13 (24.07%)	2 (16.67%)
Cellulitis	0	0	0	1(1.85%)	1(8.33%)
Eczema	0	2 (66.66%)	0	9 (16.67%)	3 (25.00%)
Folliculitis	0	0	1 (20.00%)	3(5.56%)	0
Melasma	0	0	0	1(1.85%)	0
Psoriasis	0	0	0	4 (7.41%)	2 (16.67%)
Tinea pedis	0	1 (33.33%)	0	2 (3.70%)	0
Tinea corporis	0	0	1(20.00%)	2 (3.70%)	1(8.33%)
Tinea capitis	0	0	1(20.00%)	3(5.56%)	0
Urticaria	0	0	0	7 (12.96%)	1(8.33%)
Varicella zoster	0	0	2 (40.00%)	1(1.85%)	0
Warts	0	0	0	4 (7.41%)	1(8.33%)

### Age group: ≤2 years

No cases have been documented for the first age group in this phase of the research study. This is most probably due to the fact that parents will take infants to the doctor immediately rather than self-medicating.

### Age group: >2 and ≤ 6 years

Two cases of eczema have been documented and one case of *tinea pedis*. Eczema is fairly common in children and occurs often in this age group. Literature indicates that 1 out of 10 children will develop eczema, and it will happen before the age of 5

years (Van Hees, 2001:11). An association between eczema and several allergies have been documented (McKoy, 2012).

The specific case of *Tinea pedis*, involved a child that had a fractured ankle and the cast provided the perfect conditions for fungus growth.

#### **Age Group: >6 and ≤ 12 years**

*Varicella zoster* (VZ) showed the highest prevalence (n=2) in this age group. VZ is a viral infection also known as chickenpox. This type of infection flares up once or twice a year and easily spreads under children (Van Hees, 2001:47). Other conditions that were also documented included folliculitis and tinea infections.

#### **Age Group: >12 and ≤ 60 years**

Contact dermatitis (n=13) was the most prevalent in this age group. The pharmacy from where this data were collected is located in the southern parts of Windhoek near an industrial area. It can be speculated that the high prevalence of contact dermatitis is due to chemical exposure to different substances. Urticaria (n=7) is often a result of exposure to or contact with chemical substances, but urticaria can also be caused by injury like friction or bruising.

#### **Age Group: >60 years**

Eczema (n=3) once again showed the highest prevalence in this age group. Contact dermatitis and psoriasis were also significantly documented.

It is evident from the data that eczema is the most prominent dermatological disease throughout the different age groups. It accounts for a 13.5% of all the cases in this phase of the research study.

Dr. Robert Hopp, dermatologist in the United States of America, explains on his website that eczema is an inherited tendency of the skin to be easily irritated and dried out. He also states that eczema will most probably worsen in dry, hot climate and that eczema is more prominent in the winter months than summer (Hopp, 2012).

The Namibian climate thus plays a significant role in the high eczema prevalence. In winter, temperatures drop during the night but during day time, temperatures easily reach 26°C. Namibia does not receive any rainfall during winter, which makes the climate very dry.

Furthermore, although eczema is not caused by allergies, it can be aggravated by factors like dust and pollen (MacKoy, 2012). Windhoek also has mica rock formations which cause mica dust. In late winter the mica dust is lifted due to an increase in wind. An influx of patients consulting the pharmacy with eczema is noted during this time of the year.

### **(b) Gender**

The same numbers of females (n=37) and males (n=37) were included in this phase which indicates that not only women are focused on skin appearance.

Table 26 summarises the relationship between the different dermatological diseases and gender. The most significant relationships are discussed. The value for the Chi-square for this data was 12.05 with a probability value of 0.5234. Data are thus statistically insignificant.

Table 26: Dermatological Disease vs Gender for Phase 1B

<b>Dermatological Disease</b>	<b>Female</b>	<b>%</b>	<b>Male</b>	<b>%</b>
<b>Angioedema</b>	1	2.70	0	0.00
<b>Candida</b>	4	10.81	0	0.00
<b>Contact dermatitis</b>	7	18.92	8	21.62
<b>Cellulitis</b>	1	2.70	1	2.70
<b>Eczema</b>	6	16.22	8	21.62
<b>Folliculitis</b>	2	5.41	2	5.41
<b>Melasma</b>	0	0.00	1	2.70
<b>Psoriasis</b>	4	10.81	2	5.41
<b>Tinea pedis</b>	1	2.70	2	5.41
<b>Tinea corporis</b>	2	5.41	2	5.41
<b>Tinea capitis</b>	1	2.70	3	8.11
<b>Urticaria</b>	5	13.51	3	8.11

Varicella zoster	0	0.00	3	8.11
Warts	3	8.11	2	5.41

## Females

In this category, females had the highest prevalence in contact dermatitis (18.92%), eczema (16.22%) and urticaria (13.51%).

*Candida albicans* showed a prominent difference between male (n=0) and female (n=4). Literature shows that pH plays a vital role in fungus growth. The pH on the skin is largely affected by hormonal secretion, for example sebum (Toddy, 1977:413). As mentioned before, candidiases are more prone to affect women than men (refer to 2.7.3.7).

In Phase 1B, the data indicate that psoriasis is more prominent in females than males. Phase 1A showed the contrary, which is also supported by literature. In Phase 1B, the patients received pharmacist-initiated therapy and since pharmacists are not experts in diagnoses, it can be expected that not all diagnoses in this phase were accurate.

Female urticaria (13.51%) had once again a higher prevalence than male urticaria (8.11%). As already mentioned (refer to 4.1.2.1 (b)) urticaria is more likely in females than males.

## Males

In this category, males had the highest prevalence in contact dermatitis (21.62%) and eczema (21.62%).

The results of Phase 1A reveal that *Tinea pedis* were more prevalent in men than in woman, possibly due to men more likely to wear closed, tight-fitting shoes. *Tinea capitis* also showed a higher prevalence in men. All the patients with a *Tinea capitis* infection were black. It is custom for black men to shave their hair at local barbour shops on the streets; *Tinea capitis* infections are most probably the result of transmission of the fungus via the blades (Higgins *et al.*, 2000:53).

Eight per cent of males were diagnosed with *Varicella zoster* and no females. A study done in Louisiana compared data over a period of 14 years which indicates that males had a higher risk of developing a *Varicella zoster* infection than women (Baxter *et al.*, 2013:1). The data from this phase of the study also conclude that men have a higher incidence of *Varicella zoster* than the female patients.

### **(c) Race**

Table 27 summarises the different ethnic groups relevant to this research study and the frequency in which each type of dermatological disease has occurred in these groups.

The value for the Chi-square for this data was 49.37 with a probability value of 0.1235. Data are thus statistically insignificant.

Table 27: Dermatological Disease and Race

<b>Dermatological Disease</b>	<b>Albino black (n=1)</b>	<b>%</b>	<b>Black (n=46)</b>	<b>%</b>	<b>Coloured (n=13)</b>	<b>%</b>	<b>Caucasian (n=14)</b>	<b>%</b>
<b>Angioedema</b>	0	0.00	0	0.00	1	7.69	0	0.00
<b>Candida</b>	0	0.00	1	2.17	1	7.69	2	14.29
<b>Contact dermatitis</b>	0	0.00	10	21.74	1	7.69	4	28.57
<b>Cellulitis</b>	0	0.00	1	2.17	1	7.69	0	0.00
<b>Eczema</b>	0	0.00	9	19.57	3	23.08	2	14.29
<b>Folliculitis</b>	0	0.00	2	4.35	1	7.69	1	7.14
<b>Melasma</b>	0	0.00	0	0.00	1	7.69	0	0.00
<b>Psoriasis</b>	0	0.00	5	10.87	1	7.69	0	0.00
<b>Tinea pedis</b>	1	100.00	0	0.00	1	7.69	1	7.14
<b>Tinea corporis</b>	0	0.00	3	6.52	0	0.00	1	7.14
<b>Tinea capitis</b>	0	0.00	4	8.70	0	0.00	0	0.00
<b>Urticaria</b>	0	0.00	5	10.87	1	7.69	2	14.29
<b>Varicella zoster</b>	0	0.00	3	6.52	0	0.00	0	0.00
<b>Warts</b>	0	0.00	3	6.52	1	7.69	1	7.14

### **Albino Black Patients**

Only one case was reported under this classification. *Tinea pedis* is a common fungus infection of the feet. No literature was found to support any association between *Tinea pedis* and race.

### **Black Patients**

Contact dermatitis and eczema were the most prominent diseases in this classification. No literature was found to support any association between contact dermatitis and black patients. It is, however, probable that most of these patients were workforce from the surrounding industrial area. These patients are more likely to be exposed to hazardous chemicals and substance as already mentioned in Phase 1A and literature, which might have triggered contact dermatitis (refer to 2.8.4 & 4.1.2.1 (c)).

In this phase of the research study, it can be stated that 18.9% of all dermatological cases in this phase were of eczema and 64% of the patients with eczema were black.

### **Coloured Patients**

Eczema had the highest prevalence in this category. A study done in the USA in 1999, suggests that black patients are more likely to get eczema than white patients (Baker, 1999:31). Although eczema shows a high prevalence in this category (n=4), it is still much lower than that of black patients (n=9). 21.4% of the patients with eczema were coloured.

### **Caucasian Patients**

Contact dermatitis showed the highest prevalence in this category (n=4). Although it had the highest frequency under Caucasian patients, it had only 26.6% over-all prevalence in patients diagnosed with contact dermatitis. Black patients had the highest prevalence. It can therefore not be concluded that Caucasian patients are more prone to contact dermatitis.

Table 28 below indicates the gender vs. the race of patients with dermatological disease. From this table it can be conclude that patients with dermatological diseases who consulted the pharmacists most often, were black men (n=27).

Table 28: Gender vs Race Phase 1B

Race	Male	% of total	Female	% of total
Black	27	72.97	19	51.35
Coloured	6	16.21	7	18.91
Caucasian	4	10.81	10	27.02
Albino	0	0	1	0.73

#### (d) Geographical Distribution

Patient who that participated in this phase were residence from Windhoek (N=63). Table 23 summarises the geographical distribution of dermatological diseases in Phase 1B.

Table 29: Geographical Distribution of Dermatologic Diseases in Phase 1B

Dermatological Diseases	GOB	KAT	OKA	OSH	REH	SWA	WIN
Angioedema							1
Candida					1		3
Contact Dermatitis			1		2		12
Cellulitis			1				1
Eczema					1	1	12
Folliculitis							4
Melasma							1
Psoriasis				1			5
Tinea Pedis							3
Tinea Corporis							4
Tinea Capitis							4
Urticaria	1	1					6
Varicella Zoster							3
Warts			1				4

Fifteen per cent of the patients who visited the pharmacy were not from Windhoek. 54% of the patients not from Windhoek were from the neighbouring towns. If this



data are studies in isolation, it does not suggest any association between the geographical area and the specific dermatological disease, but when compared with data from the other phases, some trends are noticeable. This is illustrated later.

#### **4.1.3.2 Medical conditions, habits and physical state contributing to dermatological problems**

##### **(a) Cigarette smoke**

In this phase of the research study, only 24.3% of the patients smoked (n=18).

Smoking cigarettes has been known to reduce oxygen perfusion in cells. A study has shown that smoking one cigarette will reduce the oxygen cell perfusion by as much as 30% (Gotrupp, 2006:61). This is mainly due to carbon monoxide. Nicotine, the main ingredient in cigarette smoke, releases catecholamine which results in peripheral vascular constriction.

A study done on rat skin and human breast skin indicated that the addition of catecholamine to the skin accelerates skin death within 6 hours (Burck *et al.*, 1990:92).

In this study, as mentioned, only 24.3% of the patients who participated in this section of the research study smoked. The value for the Chi-square for this data was 13.16 with a probability value of 0.4354. Data are thus statistically insignificant.

Table 30: Dermatological Diseases and Smoking Cigarettes in Phase 1B

Dermatological Diseases	No	%	Yes	%
Angioedema	1	1.79	0	0.00
Candida	1	1.79	3	16.67
Contact dermatitis	11	19.64	4	22.22
Cellulitis	2	3.57	0	0.00
Eczema	11	19.64	3	16.67
Folliculitis	3	5.36	1	5.56
Melasma	1	1.79	0	0.00
Psoriasis	5	8.93	1	5.56
Tinea pedis	3	5.36	0	0.00
Tinea corporis	3	5.36	1	5.56
Tinea capitis	3	5.36	1	5.56
Urticaria	7	12.50	1	5.56
Varicella zoster	3	5.36	0	0.00
Warts	2	3.57	3	16.67

Three dermatological diseases showed a high prevalence in smokers namely contact dermatitis, eczema, candida and warts. Contact dermatitis and eczema showed a high prevalence throughout the study. Twenty-six per cent of the patients with contact dermatitis smoked and 21% of smoking patients had eczema. Smoking cannot be implicated in contact dermatitis and eczema, but will definitely worsen the condition, as this study shows. Conditions like warts and candida, however, can result indirectly from cigarette smoke. Seventy-five per cent of the patients diagnosed with candida smoked. This high prevalence is explained in the literature as the result of a change in pH on the surface of the skin and the decrease of oxygen supply (Khaled *et al.*, 2006:1). Candida is an anaerobic fungus and does not need oxygen for growth.

Sixty per cent of the patients who were presented with warts were cigarette smokers. Studies have shown that cigarette smoke increases the human papilloma-virus load in the body (Xi., *et al.*, 2009:3490). Due to the vasoconstriction effect of nicotine, circulating blood cells have limited access to the skin to prevent infection. It also

decreases the amount of vitamin C in the circulation, which further impairs the immune system (Preston *et al.*, 2000:167).

### **(b) Pregnancy and breastfeeding**

Pregnancy causes hormonal changes to occur which directly affects the skin. In this research study, 9% of the patients who participated were pregnant. The most significant relationships are now discussed.

Table 31: Dermatological Diseases and Pregnancy for Phase 1B

Dermatological Diseases	No	%	Yes	%
Angioedema	1	1.49	0	0.00
Candida	4	5.97	0	0.00
Contact dermatitis	14	20.90	1	14.29
Cellulitis	2	2.99	0	0.00
Eczema	13	19.40	1	14.29
Folliculitis	4	5.97	0	0.00
Melasma	1	1.49	0	0.00
Psoriasis	3	4.48	3	42.86
Tinea pedis	3	4.48	0	0.00
Tinea corporis	4	5.97	0	0.00
Tinea capitis	4	5.97	0	0.00
Urticaria	6	8.96	2	28.57
Varicella zoster	3	4.48	0	0.00
Warts	5	7.46	0	0.00

Psoriasis had the highest prevalence in this section. According to literature psoriasis normally improves during pregnancy, due to high progesterone levels (Murase, *et al.*, 2005:601). The data suggest the contrary since 42% of the patients that were pregnant had psoriasis (n=3). According to the national psoriasis foundation of the USA website, psoriasis during pregnancy is strictly genetic and unpredictable.

### (c) Allergies

In Phase 1B 68% (n=50) of the patients were not allergic to anything. Allergies to animal hairs, grass and pollen showed the highest prevalence in the patients that were allergic.

Table 32: Frequency of Allergies in Phase 1B

Allergy	n	%
Bee sting	1	4.16
Dog or cat hairs	4	16.66
Dust	6	25.00
Grass, pollen	5	20.00
Grass, dust, pollen	1	4.16
Morphine	1	4.16
NSAIM's, Penicillin	1	4.16
Peanuts	2	8.33
Penicillin	1	4.16
Preservatives	1	4.16
Seafood	1	4.16

### (d) Family History of Dermatological Disease

In this phase, 77.3% (n=57) of patients who participated did not show a family history for dermatological disease; only 22.7% did.

Genetics plays a significant role in dermatological diseases since it not only determines the anatomy of the skin but also the internal factors such as immunology of the individual (refer to 2.8.5).

Table 33: Dermatological Diseases and Family History for the Disease in Phase 1B

Dermatological Diseases	No	%	Yes	%
Angioedema	1	1.75	0	0.00
Candida	3	5.26	1	5.88
Contact dermatitis	12	21.05	3	17.65
Cellulitis	2	3.51	0	0.00
Eczema	8	14.04	6	35.29
Folliculitis	4	7.02	0	0.00
Melasma	1	1.75	0	0.00
Psoriasis	6	10.53	0	0.00
Tinea pedis	3	5.26	0	0.00
Tinea corporis	3	5.26	1	5.88
Tinea capitis	3	5.26	1	5.88
Urticaria	6	10.53	2	11.76
Varicella zoster	1	1.75	2	11.76
Warts	4	7.02	1	5.88

Twenty-three per cent of patients who participated in this phase of the research study showed an association with a family history of the same disease. Eczema had the highest prevalence with 35.29% of family history of the disease. Since the etiology of eczema is not completely understood, genetics is thought to contribute substantially. This data suggest a high correlation between eczema and a family history of the disease.

Contact dermatitis also showed a high prevalence, but this is most likely due to the high over-all prevalence in the study. It can, however, be said that sensitivity towards certain external factors is attributed to the genetics of the specific individual. As already discussed, literature suggests that the immune response to the relevant antigen is strongly genetic.

It can therefore be concluded that the family history of patients plays a significant role in eczema and contact dermatitis.

### **(e) Chronic Medication**

Fifty-six per cent of the patients in this phase were not using any chronic medication; 9% of the patients were on an oral contraceptive which accounts for the most chronic medication used.

### **Contact Dermatitis**

Contact dermatitis showed the highest prevalence in this phase and 33.3% of the patients with contact dermatitis were also on chronic medication. ACE-inhibitors, oral contraceptives and multivitamins were medication shared by the different patients. No literature was found to suggest any association between these medications and contact dermatitis. The prevalence of medications used by patients is also not sufficient evidence to suggest otherwise.

Table 34: Chronic Medication of Patients with Contact Dermatitis

Chronic medication	n	%
ACE-inhibitor, omega 3 and cardio aspirin	1	20.00
Insulin + ACE-inhibitor + Sulfonylureas + Metformin	1	20.00
Multivitamin	1	20.00
Oral contraceptive	1	20.00
Oral contraceptive & multivitamin	1	20.00

### **Eczema**

Almost forty-three per cent of the patients diagnosed with eczema were using chronic medication. Anti-histamine and corticosteroid nasal spray showed a high prevalence. This is expected since allergies have illustrated to have a significant correlation with eczema. Multivitamins also have a high prevalence. Unfortunately, it is not specified which vitamins exactly the patients were using, thus no conclusion can effectively be drawn from this.

Table 35: Chronic Medication of Patients with Eczema

Chronic medication	n	%
ACE-inhibitor	1	16.66
Anti-histamine & corticosteroid nasal spray	2	33.33
Multivitamin	1	16.66
Multivitamin and calcium channel blocker	1	16.66
Multivitamin & omega 3	1	16.66

## Urticaria

Oral contraceptives are showing the highest prevalence in this section of Phase 1B. 50% of the patients with urticaria were on chronic medication. 75% of these patients were on oral contraceptives. Literature does suggest cyclic urticaria to appear in some patients. Progesterone in oral contraceptives can cause urticaria to appear in the second parts of the menstrual cycle (MacNeal, 2013). A possible relationship between urticaria in females on oral contraceptives is expected.

Table 36: Chronic Medication of Patients with Urticaria

Chronic medication	n	%
Multivitamin & Oral Contraceptive	1	25.00
Multivitamin	1	25.00
Oral Contraceptive	2	50.00

## Psoriasis

Eighty-three per cent of the patients diagnosed with psoriasis were on chronic medication. Once again, multivitamins showed the highest correlation, but the data are incomplete and conclusion cannot be suggested from it.

Psoriasis cannot be caused by vitamins, however, literature does suggest that Vitamin A, Vitamin C and Vitamin B3, which is niacin, can cause psoriasis to flairup. These three vitamins are normally formulated in multivitamins. It can therefore be concluded that vitamins may worsen, but not cause psoriasis.

Table 37: Chronic Medication in Patients with Psoriasis

Chronic medication	n	%
ACE-Inhibitor, calcium channel blocker, cardio aspirin, statin and Q10 co-enzyme supplement	1	20.00
Eltroxin	1	20.00
Multivitamin	1	20.00
Multivitamin and calcium channel blocker	1	20.00
Multivitamin + Folic acid	1	20.00

#### (f) Patients Referred to the General Practitioner

In this phase of the research study, the pharmacist applied pharmacist-initiated therapy. However, some of the diagnosis that was made was not treatable by the pharmacist and the patient was referred to his/her general practitioner, as indicated by table 37.

The value for the Chi-square for this data was 54.67 with a probability value of <0.0001. Data are thus statistically significant. The value for Cramer's V was 0.8595 which indicates a high association between variables.



Table 38: Dermatological Diseases and Referral to General Practitioner in Phase 1B

Dermatological Diseases	No	%	Yes	%
Angioedema	1	1.85	0	0.00
Candida	4	7.41	0	0.00
Contact dermatitis	14	25.93	1	5.00
Cellulitis	0	0.00	2	10.00
Eczema	13	24.07	1	5.00
Folliculitis	1	1.85	3	15.00
Melasma	0	0.00	1	5.00
Psoriasis	0	0.00	6	30.00
Tinea pedis	3	5.56	0	0.00
Tinea corporis	4	7.41	0	0.00
Tinea capitis	4	7.41	0	0.00
Urticaria	8	14.81	0	0.00
Varicella zoster	0	0.00	3	15.00
Warts	2	3.70	3	15.00

27.20% of the patients who participated in this phase of the research study were referred to their general practitioner.

Disease such as cellulitis, melasma, psoriasis and *Varicella zoster* were not treated by the pharmacist since the medication required treating these diseases are scheduled and not dispensed without a prescription from a general practitioner.

One case of contact dermatitis and eczema was also referred due to secondary infection that required systemic antibiotics.

It can be concluded that the 73.00% of patients who visited the community pharmacy with a dermatological condition could be treated by the pharmacist. This data conclude the initial problem statement that has led to the research for this study. The limited access to general practitioners and dermatologists in Namibia has caused the pharmacist to become the first-line healthcare practitioner in the treatment of skin disease.

### 4.1.3.3 Treatments and Treatment Duration

A variety of treatments was used in this phase of the research study. Hydrocortisone 1% was used most often (n=20). Topical terbinafine, Oilatum® bath emollient, Selsun® shampoo, Bactroban® and a variety of mixtures prepared by the pharmacist were used. The different mixtures were assigned different numbers, in order to facilitate the data-collection process. The following table demonstrates the ingredients found in the mixtures.

Table 39: Mixtures used in Phase 1B

Ingredients	Mixture
1% Hydrocortisone and Epi-Max®	MIX 6
2% SSA and Miracle comfrey®	MIX 7
10% Ketokonazole + 50% Selsun® Shampoo + 10% Betadine + add 100ml sterile water (spray lotion)	MIX 8
10% Hydrocortisone + Epi-Max®	MIX 9
50% Hydrocortisone + Epi-Max®	MIX 10

#### **Mixture 6: 1% Hydrocortisone and Epi-Max®**

This is a mixture containing 1% Hydrocortisone mixed with Epi-max® in a ration 1:5. The mixture dilutes the concentration of the hydrocortisone and adds moisture to the mixture. This mixture is often use to dermatological diseases such as eczema, psoriasis and urticaria on sensitive areas. This mixture was dispensed most often (n=11).

#### **Mixture 7: 2% SSA and Miracle comfrey®**

This mixture contains salicylic acid at 2% mixed with Miracle comfrey ointment which is a homeopathic ointment produced in South Africa. This mixture is often used to soften very thick skin. It is also used to treat plantar warts. This mixture was only used once.

**Mixture 8: 10% Ketokonazole + 50% Selsun® Shampoo + 10% Betadine + add 100ml sterile water (spray lotion)**

This mixture contains several ingredients, namely ketokonazole, selsun shampoo and betadine solution. This mixture is an antiseptic and antifungal treatment spray that effectively treats tinea infections. This mixture was dispensed only twice.

**Mixture 9 and 10: 10% or 50% hydrocortisone + Epi-Max®**

These mixtures contain hydrocortisone and Epi-max®. The hydrocortisone concentration is increased or decreased according to the requirements. Epi-max® serves as an intensive moisturiser.

Treatment duration for above-mentioned dermatological diseases averaged at 14 days. Sixty five percent of patients did not return for a follow up consultation, while 34.3% of the patients did.

Twenty-seven per cent (n=20) of the patients were referred to a general practitioner since they could not be treated by the pharmacist. Seventy-two per cent of the patients in this phase did, however, receive pharmacist-initiated therapy.

#### 4.1.4 Results for Phase 2

In this phase of the research study, the dermatologist often assigned more than one dermatological diseases to one patient. The chart below summarises the number of cases of each of the following dermatological diseases. One patient might be listed twice since the patient was diagnosed with two or more diseases.

Table 40: Dermatological Diseases in Phase 2

Dermatological Disease	n	%
Angioedema	1	0.66
Acne vulgaris	30	19.87
Basal cell carcinoma	1	0.66
Candida	12	7.95
Conatct dermatitis	2	1.32
Cellulitis	8	5.30
Eczema	7	4.64
Erythrasma	2	1.32
Folliculitis	8	5.30
Impetigo	9	5.96
Kaposi sarcoma	7	4.64
Leprosy	1	0.66
Melasma	19	12.58
Melanoma	2	1.32
Psoriasis	5	3.31
Pityriasis rosea	6	3.97
Pityriasis vesicolour	2	1.32
Squamous cell carcinoma	3	1.99
Sebborheic dermatitis	4	2.65
TB	4	2.65
Tinea corporis	11	7.28
Tinea capitis	4	2.65
Urticaria	2	1.32
Vitiligo	1	0.66

From the chart it can be concluded that *Acne vulgaris* is the most common dermatological disease with a prevalence of 19.87% (n=30). Melasma is the second most prevalent with 12.58% (n=19). This data reflect what the dermatologist predicted in the interview explained in Chapter 1 of this research study. Both these

conditions mainly occur on the face and can be described as conditions which have a negative effect on self-esteem. Patients are therefore more likely to seek healthcare quickly compared to other dermatological diseases.

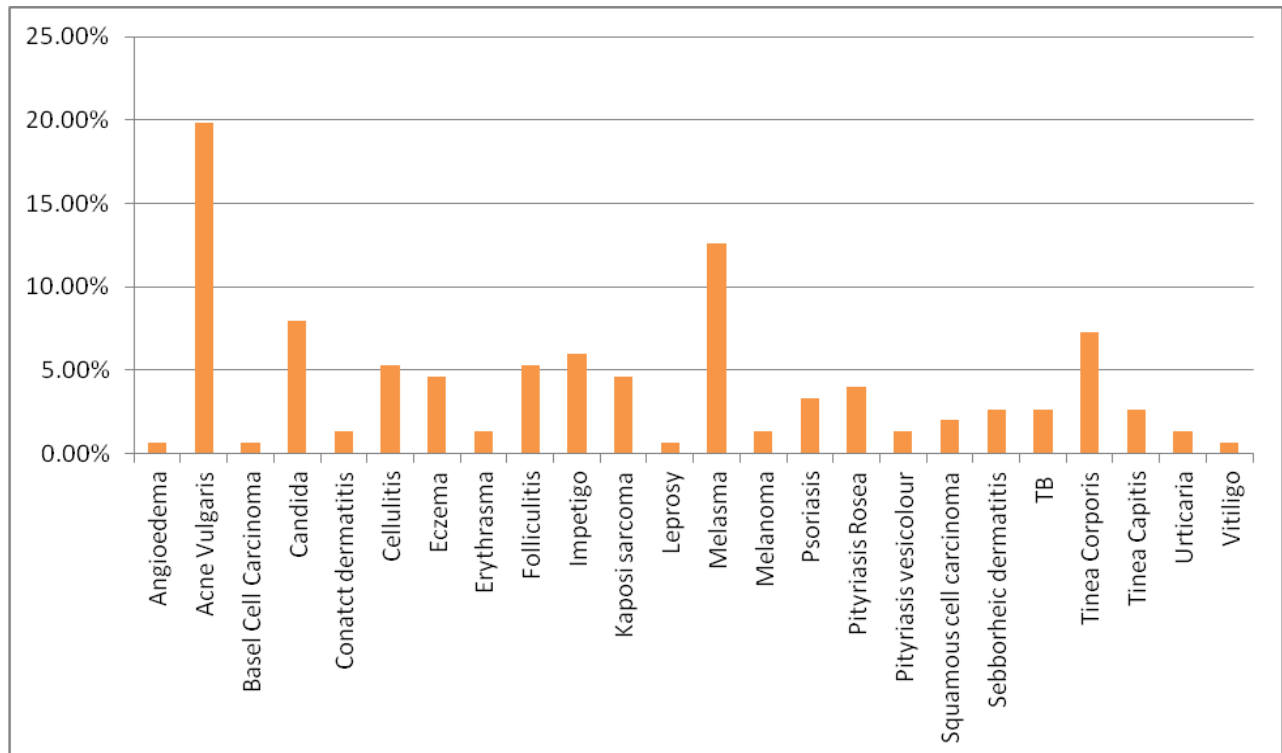


Figure 16: Dermatological Diseases in Phase 2

*Candida* and *tinea corporis* also show high prevalence in this phase. According to literature fungal infections prefer a wet and humid environment to thrive (Gawkrödger, 2002:54). Namibia's climate is hardly that, which makes this data unique.

#### 4.1.4.1 Demographic results

##### (a) Age

One-hundred-and-thirty-one patients participated in this phase of the research study. Table 41 indicates the different age groups of patients in this phase.

Table 41: Age Groups for Phase 2

AGE	n	%
≤2 years	1	0.76
>2 and ≤ 6 years	5	3.82
>6 and ≤ 12 years	9	6.87
>12 and ≤ 60 years	109	83.21
>60 years	7	5.34

Table 41 reveals the relationship between the age of the patient and the dermatological conditions. The most significant relationships within each age group are discussed. No statistical significant associations were found between the age group and prevalence of dermatological disorders ( $p = 0.607$ ).

Table 42: Dermatological Disease according to Age in Phase1B

Dermatological Disease	≤2 years	>2 and ≤ 6 years	>6 and ≤ 12 years	>12 and ≤ 60 years	>60 years
Angioedema	0	0	0	1(0.91%)	0
Acne vulgaris	0	0	0	26 (23.85%)	0
Basal cell carcinoma	0	0	0	1(0.91%)	0
Candida	0	0	2 (22.22%)	9 (8.25%)	1 (14.28%)
Contact dermatitis	0	1(20.00%)	0	1(0.91%)	0
Cellulitis	0	0	1 (11.11%)	6 (5.50%)	1(14.28%)
Eczema	1 (100%)	1(20.00%)	2 (22.22%)	3 (2.75%)	0
Erythrasma	0	0	1(11.11%)	1 (0.91%)	0
Folliculitis	0	0	0	7 (6.42%)	1(14.28%)
Impetigo	0	1 (20.00%)	1(11.11%)	5 (4.58%)	1(14.28%)
Kaposi sarcoma	0	0	1(11.11%)	6 (5.50%)	0
Leprosy	0	0	1(11.11%)	1 (0.91%)	0
Melasma	0	1(20.00%)	0	18 (16.51%)	1(14.28%)
Melanoma	0	0	0	3 (2.75%)	0
Psoriasis	0	0	0	8 (7.33%)	0
Pityriasis rosea	0	0	0	2 (1.83%)	0
Pityriasis vesicolour	0	0	0	3 (2.75%)	0
Squamous cell carcinoma	0	0	0	0	1(14.28%)
Seborrheic dermatitis	0	0	0	3 (2.75%)	0
Tuberculosis	0	1(20.00%)	0	3 (2.75%)	1(14.28%)
Tinea pedis	0	0	1(11.11%)	8(7.33%)	0
Tinea corporis	0	0	0	1(0.91%)	0
Tinea capitis	0	0	0	4 (3.66%)	0
Urticaria	0	0	0	3 (2.75%)	0
Vitiligo	0	0	0	1(0.91%)	0

**Age group: ≤2 years**

Eczema is the only dermatological diseases in this phase that occurred in this age group. Eczema is common in infants and mostly atopic. This subject has already been discussed extensively in the literature study and other phases. It can be concluded that eczema is common in children under the age of 2 years.

**Age Group: >2 and ≤ 6 years**

All five conditions showed the same prevalence. Impetigo, eczema and contact dermatitis are common dermatological diseases in children; melasma, however, is not. This child was an epileptic and the seizure medication was said to be the cause of the melasma. The dermatologist explained that the medication increased the sensitivity of the melanocytes in the skin, causing melasma to occur more frequently than in patients not on anti-seizure medication.

Impetigo accounts for approximately 10% of skin problems observed in pediatric clinics in the United States of America (Cole *et al.*, 2007:859). It is the most common bacterial skin infection and the third most common skin disease among children (Cole *et al.*, 2007:859).

**Age Group: >6 and ≤ 12 years**

Eczema and candida had equal prevalence in this category. Eczema is fairly common in children as already mentioned. Sixteen per cent of the candida and eczema in this phase occurred in children between the ages of 6 and 12 years. Cutaneous candida is more prevalent in the age group 12-21 years (refer to 2.7.3.7). It is more prevalent in patients that are HIV positive (Montejo, 2012:1). In this study, 50% of the children diagnosed with candida were HIV positive.

**Age Group: >12 and ≤ 60 years**

Acne Vulgaris showed the highest prevalence at 20.90% of the total of patients in this phase and 23.85% of cases within this age group. This age group is the biggest and classifies patients from teenagers to the working-class adult. *Acne vulgaris* is common in puberty due to the increase in androgen levels. According to Dawson & Dellavalle (2013:346), 90% of teenagers will have acne at some point in time and 50% of these teenagers will continue to experience acne as adults. This study agrees that *Acne vulgaris* is the most common skin disease documented by the specialist physician.



**Age Group: >60 years**

No specific dermatological disease was presented in this phase within the age group. Literature however explains that the skin renewal process slows down with age which causes skin to become thinner and easily bruised (Jung *et al.*, 2006:175). Skin conditions like senile purpura, eczema, melasma and melanoma are due to years of UV radiation exposure.

**(b) Gender**

One-hundred-and-thirty-one patients participated in this phase of the study. 56.5% (n=74) of the patients were female and 43.5% (n=57) were male.

The following table summarises the relationship between the different dermatological diseases and gender. Some of the patients had more than one condition; therefore the total of cases will not reflect the number of patients who participated.

The value for the Chi-square test ( $\chi^2$ ) was 70.56 with a *p*-value of <0001. This indicates a statistical significant association between the variables. The value for Cramer's V was 0.7340 which shows good association (moderate practical significance) between variables.

Table 43: Dermatological Diseases by Gender in Phase 2

Dermatological Diseases	Female	%	Male	%
Angioedema	0	0.00	1	1.49
Acne vulgaris	13	16.05	13	19.40
Basal cell carcinoma	0	0.00	1	1.49
Candida	10	12.35	2	2.99
Contact dermatitis	2	2.47	0	0.00
Cellulitis	2	2.47	6	8.96
Eczema	5	6.17	2	2.99
Erythrasma	1	1.23	1	1.49
Folliculitis	5	6.17	3	4.48
Impetigo	3	3.70	7	10.45
Kaposi sarcoma	1	1.23	5	7.46
Leprosy	2	2.47	0	0.00
Melasma	19	23.46	1	1.49
Melanoma	2	2.47	1	1.49
Psoriasis	1	1.23	7	10.45
Pityriasis rosea	0	0.00	2	2.99
Pityriasis vesicolour	0	0.00	3	4.48
Squamous cell carcinoma	0	0.00	1	1.49
Seborrheic dermatitis	2	2.47	2	2.99
Turbiculosis	3	3.70	2	2.99
Tinea pedis	1	1.23	0	0.00
Tinea corporis	4	4.94	4	5.97
Tinea capitis	4	4.94	0	0.00
Urticaria	0	0.00	3	4.48
Vitiligo	1	1.23	0	0.00

## Female

In this phase, women had the highest prevalence in *acne vulgaris* (n=13), *Candida* (n=10) and *melasma* (n=19).

*Candida* was responsible for 8% (n=12) of the cases in Phase 2. Eighty-three per cent of the cases were female and only 17% male. The data suggest that women are

more likely to get cutaneous candida than men. A study done in Argentina collected 2073 samples of nail, hair and skin. 55.7% of these samples were positive for candida, and 63% of the samples were retrieved from women (Nardin *et al.*, 2006:25).

Melasma was responsible for 13.5% of the cases of Phase 2. Ninety-five per cent of the patients were female and only 5% male. As already mentioned, the aetiology for melasma includes female hormones and exposure to sunlight (refer to 2.7.8). Dark-skinned individuals are also at greater risk than lighter-skinned individuals (Sheth *et al.*, 2011:689).

Eczema had a higher prevalence in women (6.17%) than in men (2.99%). As already mentioned in phase 1A, no literature was found to indicate that eczema is strongly gender specific.

### **Male**

Cellulitis had a higher prevalence in males (n=6) than in females (n=2). According to literature, cellulitis is not gender specific; however, it can be said that men are more likely than a women to sustain an injury that might develop into cellulitis due to their type of work.

Impetigo was responsible for 6% of the cases in Phase 1B. 70% of these cases were men and only 30% women. Children are more likely to have impetigo than adults; however, when an adult is affected, it is more likely to be a man than a woman (Lewis *et al.*, 2013:4). Thirty per cent of the patients diagnosed with impetigo were toddlers between the ages of 2-6 years. 66.6% were male and 33.3% female.

Psoriasis indicated a clear gender distinction with men having a higher prevalence. Literature, however, indicates that psoriasis is not gender specific (refer to 4.1.2 9(b)).

Urticaria is also indicating a higher prevalence in men (4.5%) than in women (0.00%). Literature does not indicate a relationship between gender and urticaria other than the possibility of occupation playing a significant role. Men are more likely

to have an occupation exposing them to chemicals, dust and other antigens than women (Eng *et al.*, 2011:888).

### **(c) Race**

In this phase, around 75.6% (n=99) of patients with dermatological conditions were black, 11.45% (n=15) of patients white, 11.45% (n=15) colored and 1.5% (n=2) black-albino.

Table 44: Race Distribution in Phase 2

RACE	n	%
Black	99	75.57
Caucasian	15	11.45
Coloured	15	11.45
Albino-black	2	1.53
Albino-caucasian	0	0.00

The following tables summarise the relation between the different ethnic groups relevant to this research study and the frequency in which each type of dermatological disease has occurred in each group.

Some of the patients had more than dermatological condition. These patients' data were divided into the necessary categories. The total of cases will not reflect the number of patients who participated.

Table 45: Dermatological Disease according to Race in Phase 2

<b>Dermatological Disease</b>	<b>Albino Black</b>	<b>%</b>	<b>Black</b>	<b>%</b>	<b>Coloured</b>	<b>%</b>	<b>Caucasian</b>	<b>%</b>
<b>Angioedema</b>	0	0.00	2	1.72	0	0.00	0	0.00
<b>Acne vulgaris</b>	0	0.00	22	18.97	2	13.33	1	6.25
<b>Basal cell carcinoma</b>	0	0.00	0	0.00	1	6.67	0	0.00
<b>Candida</b>	0	0.00	10	8.62	2	13.3	0	0.00
<b>Contact dermatitis</b>	0	0.00	1	0.86	0	0.00	1	6.25
<b>Cellulitis</b>	0	0.00	7	6.03	0	0.00	1	6.25
<b>Eczema</b>	0	0.00	7	6.03	0	0.00	0	0.00
<b>Erythrasma</b>	0	0.00	1	0.86	0	0.00	1	6.25
<b>Folliculitis</b>	1	50.00	5	4.31	0	0.00	2	12.50
<b>Impetigo</b>	0	0.00	6	5.17	1	6.67	2	12.50
<b>Kaposi sarcoma</b>	0	0.00	6	5.17	0	0.00	1	6.25
<b>Leprosy</b>	0	0.00	2	1.72	0	0.00	0	0.00
<b>Melasma</b>	1	50.00	15	12.93	1	6.67	3	18.75
<b>Melanoma</b>	0	0.00	0	0.00	3	20.00	0	0.00
<b>Psoriasis</b>	0	0.00	5	4.31	2	13.33	1	6.25
<b>Pityriasis rosea</b>	0	0.00	2	1.72	0	0.00	0	0.00
<b>Pityriasis vesicolour</b>	0	0.00	2	1.72	0	0.00	1	6.25
<b>Squamous cell carcinoma</b>	0	0.00	0	0.00	1	6.67	0	0.00
<b>Seborrheic dermatitis</b>	0	0.00	4	3.45	0	0.00	0	0.00
<b>Turbiculosis</b>	0	0.00	3	2.59	1	6.67	1	6.25

<b>Tinea pedis</b>	0	0.00	7	6.03	0	0.00	1	6.25
<b>Tinea corporis</b>	0	0.00	2	1.72	0	0.00	0	0.00
<b>Tinea capitis</b>	0	0.00	4	3.45	0	0.00	0	0.00
<b>Urticaria</b>	0	0.00	3	2.59	0	0.00	0	0.00
<b>Vitiligo</b>	0	0.00	0	0.00	1	6.67	0	0.00

### **Albino Black (AB)**

Only two cases were documented in this category, those of folliculitis and melasma. Literature does not indicate any relationship between folliculitis and albino patients. Data, however, show two patients, one in Phase 1A and one in this phase, that are suffering from folliculitis. The same pattern was identified with melasma documented in the data of Phase 1A. By definition, a person suffering from albinism does not have any melanocytes which make melasma an impossible condition from which to suffer from. It could be that the patient had partial vitiligo and that other non-affected areas were diagnosed with melasma.

### **Black (B)**

Almost seventy-eight per cent (n=116) of the cases in this phase were of black patients. *Acne vulgaris* and melasma were the two most prominent diseases in this phase. *Acne vulgaris* was responsible for 18.5% (n=22) of the cases and melasma 12.9%, (n=15). Literature, however, suggests that acne is more common in Caucasians than in Blacks. When compared with the amount of Caucasians that had *Acne vulgaris*, 13.3% (n=2) this seems inaccurate. It must however be kept in mind that  $\frac{3}{4}$  of the total cases were that of Blacks, thus the data does not provide a broad enough spectrum to disprove literature.

### **Caucasian (CC)**

The condition with the highest prevalence in this category was melanoma (N=3). Melanoma is more common in Caucasians than in any other race, according to literature. Melanoma is caused when a tumour develops in the skin due to exposure to UV radiation. The UV radiation causes damage to skin cells, resulting in abnormal growth. Melanin produced by melanocytes protects the skin against UV radiation, but the lighter the skin, the less melanin it has. It can therefore be concluded from the data that melanoma has a direct correlation with Caucasian skin colour.

### **Coloured (C)**

Melasma is, once again, the highest prevalence in this category of Phase 2. These patients are all women and older than 12 years of age. As already mentioned

melasma plays a vital role in this research study, while high UV radiation and hot and dry climate highly impact on this dermatological disease

#### (d) Geographical Distribution

One of the variables that were documented was the geographical area in which the patients live. This data should provide us with information indicating a possible correlation between the different dermatological diseases and the climate, culture, race, traditions etc. from each geographical area.

Table 46: Geographical Distribution of Phase 2

Geographical Area	n	%
Windhoek	74	56.49
Oshakati	22	16.79
Tsumeb	7	5.3
Okahandja	4	3.05
Rehoboth	2	1.53
Katima Malilo	4	3.05
Gobabis	2	1.53
Grootfontein	2	1.53
Keetmanshoop	2	1.53
Ondagwa	3	2.29
Swakopmund	4	3.05
Walvisbay	1	0.76
Oranjemund	3	2.29
Mariental	1	0.76

Most of the patients who consulted the dermatologist were from Windhoek (56%). The second highest frequency was Oshakati, which is in the northern parts of Namibia. Oshakati has a large Oshiwambo population. The table that follows shows the distribution of patients in the various geographical areas.

The value for the Chi-square for this data was 636.16 with a probability value of 0.0282, which indicates that data is moderately significant.



Table 47: Dermatological Disease in Different Geographical Regions

Dermatological disease	GO	GFT	KM	K	M	OH	O	OM	OS	RH	SM	TS	WB	WHK
Angioedema												1		
Acne vulgaris						1		1	3		2	1		18
Basal cell carcinoma														1
Candida			1		1		1		3			1		5
Contact dermatitis						1			1					
Cellulitis		1							2					5
Eczema									2		1			4
Erythrasma						1				1				0
Folliculitis											1			7
Impetigo			1						4			1		3
Kaposi sarcoma			1						3					3
Leprosy									2					
Melasma			1			1	1						1	16
Melanoma	1			1								1		
Psoriasis		1						1	1					5
Pityriasis rosea									1			1		
Pityriasis vesicolour									1	1				1
Squamous cell carcinoma	1													
Seborrheic dermatitis								1						2
Tuberculosis							1		2			1		1



## Northern Namibia

Grootfontein, which is in the north-eastern parts of Namibia, had only two cases, namely cellulitis and psoriasis. Cellulitis has a higher incidence in warm, humid climate. Psoriasis is not affected by climate, but an increase in sun exposure might reduce psoriasis symptoms.

Katima Mulilo is a small town at the far end of the Caprivi. Candida, Impetigo, Kaposi sarcoma and melasma were all indicated in this region. Katima has very humid and hot climate and receives the highest rainfall in Namibia. Conditions such as Impetigo and candida might be directly related to the climate.

Ondangwa is in the north-western parts of Namibia. Candida, melasma and cutaneous tuberculosis were found in this area. Ondangwa is a densely populated area where diseases like candida and tuberculosis are easily spread. Poverty and poor sanitation are also contributing factors.

Oshakati is in the same area, and indicated a overall high incidence of dermatological diseases. Impetigo, Kaposi sarcoma and candida infections are all typical opportunistic infections due to HIV. According to the website Namibweb.com, Dr. Tara Lumpkin explains that Oshakati has one of the highest HIV/AIDS prevalence in the world, with an average of 34% of all pregnant women. It can therefore be concluded that the high incidence of Oshakati residents consulting a private dermatologist in Windhoek, is most probably due to the high HIV/AIDS rate of the area and not necessarily the climate.

Tsumeb is in the northern parts of Namibia. This area receives above-average rainfall and is known to have hot, humid climate. Tsumeb also indicated a high incidence of dermatological diseases; these included tinea capitis, candida, impetigo, *Acne vulgaris*, Tuberculoses, *Pityriasis rosea*, angioedema and melasma. The diseases listed were possibly prompted by the climate in that area. Melasma is known to be caused by high UV radiation, whereas pityriasis rosea is a self-limiting skin rash believed to have viral influence. Angioedema's prevalence does not show any direct relationship with climate.

### **Eastern Namibia**

Gobabis, which is in the eastern parts of Namibia, had two cases, namely melanoma and squamous cell carcinoma. The Gobabis area is well-known for its cattle farmers who are mostly Caucasian. The high prevalence for carcinoma and melanoma is evidence of high UV radiation on light-skinned patients.

### **Southern Namibia**

Keetmanshoop had one case, that of melanoma. High UV radiation in this area is the main probable cause.

Mariental had one case of cutaneous candida. Mariental is a town in the southern parts of Windhoek with very hot and dry conditions.

### **Western Namibia**

Swakopmund and Walvisbay are coastal towns in the Namib Desert. These areas experience extreme temperatures, from very hot and dry, to wet, cold and humid. Dermatological diseases included *Acne vulgaris*, eczema, folliculitis and melasma. Melasma is an obviously result of high UV radiation; whereas *Acne vulgaris* and eczema are worsened by the climate. Studies have shown that sea sand, water and sun screen lotion can worsen or even cause folliculitis.

### **Central Namibia**

Okahandja is a town very close to Windhoek. The dermatological diseases affecting Okahandja are most probably random since no association with the climate in this area could be found.

Rehoboth had only two cases, namely folliculitis and erythrasma, none of which is caused by the specific region's climate. Erythrasma is caused by a bacterial infection which will shows a higher incidence in warm, humid areas. Rehoboth is close to Windhoek, with warm, dry climate.

Windhoek had the overall highest prevalence when compared to other geographical areas. *Acne vulgaris* and melasma were the most prominent in this region. Melasma has a direct correlation with the amount of UV radiation in the central parts of

Namibia. *Acne vulgaris* has an overall high prevalence; data indicate Windhoek to have had the highest number of *Acne vulgaris* (N=82) cases.

#### **4.1.4.2 Medical Conditions Contributing to Dermatological Conditions**

##### **(a) HIV/AIDS and TB**

19% (n=25) of the patients who participated in this phase were HIV positive and 4% (n=6) had active TB.

As already mentioned in the literature study, HIV decreases the number of active CD4 – T cells in the body. The decrease of these lymphocytes results in opportunistic diseases to cause AIDS.

The table below indicates the relation between the number of HIV positive and TB patients and different dermatological diseases for Phase 2. The value for the Chi-square for this data was 31.00 with a probability value of 0.0404 which indicates that data is moderately statistically significant.

Table 48: HIV and TB Prevalence in Phase 2

Dermatological disease	HIV	%	TB	%
Acne vulgaris	5	16.13	0	0.00
Candida	2	6.45	1	12.50
Eczema	1	3.23	0	0.00
Erythrasma	1	3.23	0	0.00
Folliculitis	1	3.23	0	0.00
Impetigo	5	16.13	2	25.00
Kaposi sarcoma	6	19.35	0	0.00
Leprosy	2	6.45	0	0.00
Melasma	3	9.68	0	0.00
Psoriasis	2	6.45	0	0.00
Pityriasis rosea	1	3.23	0	0.00
Pityriasis vesicolour	1	3.23	0	0.00
Seborrheic dermatitis	1	3.23	0	0.00
Tuberculosis	0	16.13	5	62.5
Tinea corporis	0	6.45	1	0.00
Tinea capitis	2	3.23	0	12.50
Urticaria	1	3.23	0	0.00

The condition with the highest prevalence in HIV patients is Kaposi Sarcoma (N=6). This dermatological tumour is synonym with HIV/AIDS. The incidence of Kaposi Sarcoma in Windhoek is fairly high when compared to Zimbabwe (3:100 000), but it must be kept in mind that the dermatologist treating these patients was one of three available in the country. This dermatologist is also the only one claiming directly from the government medical aid fund, whereas the other dermatologists only accept cash. The likeliness of the patients suffering from Kaposi Sarcoma to be treated by this dermatologist is very high.

Impetigo also showed a very high incidence in HIV positive patients. This bacterial infection is usually staphylococcus related, which may imply that this is a case of an opportunistic disease. Impetigo is also common in developing countries with poor sanitation.

Tuberculosis of the skin also has a high incidence in this research study (n=5). Studies have shown that cutaneous TB usually accounts for around 1 - 2% of the total TB cases (Veras, *et al.*, 2012:493; Bravo & Gutizzo, 2007:173). Unfortunately, no data on cutaneous TB is available with regard to Windhoek, but TB in general is rated at 603:100 000 according to the World Bank Report in 2012. It can therefore be estimated that approximately 120 people in Namibia suffer from cutaneous TB.

### **(b) Patients receiving oncology treatment**

Four per cent (n=6) of the patients who participated were patients receiving cancer treatment.

The data collected also included information regarding patients' history of cancer, whenever applicable. The following table summarises the findings:

Table 49: Dermatological Disease in Patients with a History of Cancer

<b>Dermatological Disease</b>	<b>n</b>	<b>%</b>
<b>Basal cell carcinoma</b>	1	16.66
<b>Acne vulgaris</b>	1	16.66
<b>Melanoma</b>	3	50.00
<b>Squamous cell carcinoma</b>	1	16.66

Patients diagnosed with melanoma showed a significant history of cancer. Melanoma is the cancer of skin responsible for most dermatological-related deaths. As explained in literature genetics plays a vital role in melanoma (refer to 2.7.10).

#### **4.1.4.3 Treatment for Dermatological Diseases**

The dermatologist had five standard mixtures which he prescribed regularly. These mixtures can be summarised as follows:

Table 50: Ingredients for Dermatologist Mixtures

Ingredients	Mixture
12.5% Mometasone + 10% Retinoid + 0.5% Ascorbic Acid + 5% Hydroquinone + add 30g Aqueous Cream	MIX 1
10% Mometasone + 0.5% Ascorbic Acid + 5% Hydroquinone + add 30g Aqueous Cream	MIX 2
10% Glycerine + 5% Urea + add 500g Aqueous Cream	MIX 3
20g Methylprednisolone Aceponate + 400g Epi-Max	MIX 4
5% SSA + 20g Betametasone/Gentamicin	MIX 5

The above mixtures were determined by the dermatologist's 40 years of practical experience. These are not standard mixture per say, rather formulae invented over years.

The treatment that was prescribed most often in this phase of the study was Mixture 1 with sunscreen (n=19). The combination of Mixture 1, oral isotretinoin and topical lincosamide & macrolide was prescribed second most often (n= 13).

In this phase, 63.7% (n=83) of the patients did not return for a follow-up consultation, 36.6% did.

The treatment duration for different treatments varied from 180 days to 1 day of treatment. The treatment duration for most of the patients was 180 days. Treatment duration is summarised below:



Table 51: Duration of Treatment for Phase 2

<b>DURATION in days</b>	<b>n</b>	<b>%</b>
<b>1</b>	6	4.58
<b>7</b>	16	12.21
<b>10</b>	1	0.76
<b>14</b>	20	15.27
<b>21</b>	7	5.34
<b>30</b>	19	14.50
<b>60</b>	5	3.82
<b>90</b>	24	18.32
<b>180</b>	33	25.19

4.1.4.5 Following is a summary of treatment regimens for the relevant dermatological diseases in Phase 2.

Table 52: Summary of Treatment Regimens for the different Dermatological Diseases

Disease	Treatment	n of patients	Duration	Adjustment to treatment
<b>Angioedema</b>	Corticosteroid and anti-histamine	1	7	no
<b>Acne vulgaris</b>	Mix 1, oral isotretinoin, topical lincosamide	3	90	repeated
	Mix 1, oral isotretinoin, topical lincosamide, sunscreen	1	180	no
	Mix 1, oral isotretinoin, topical lincosamide, topical macrolide/zinc combination	12	90	repeated + tetracycline/ minocycline added
	Mix 1, oral isotretinoin, sunscreen, multivitamin	1	90	no
	Mix 1, oral tetracycline, topical lincosamide	2	135	repeated
	Mix 1, oral tetracycline, topical lincosamide, sunscreen	2	90	no
	oral tetracycline, topical isotretinoin , topical lincosamide	1	180	no
	oral tetracycline, topical isotretinoin , topical lincosamide, sunscreen	1	90	no
<b>Acne vulgaris &amp; melasma</b>	Mix 1, oral isotretinoin, topical lincosamide	1	90	no

<b>Acne vulgaris, psoriasis and urticaria</b>	Mix 1, oral isotretinoin, topical lincosamide, topical macrolide/zinc combination	1	30	no
<b>Basel cell carcinoma</b>	Surgery	1	-	yes
<b>Candida</b>	Topical anti-fungal	1	7	no
	Topical anti-fungal, oral anti-fungal	9	7	no
<b>Candida &amp; impetigo</b>	Topical anti-fungal, oral anti-fungal and oral penicillin	1	14	no
<b>Conatct dermatitis</b>	Topical corticosteroid: High II, anti-histamine	1	7	no
<b>Contact dermatitis &amp; impetigo</b>	Oral penicillin, topical corticosteroid: Medium IV	1	14	added oral corticosteroid + anti-histamine
<b>Cellulitis</b>	Oral penicillin, topical anti-bacterium and anti-septic wash	1	1	added lincosamide
<b>Eczema</b>	Mix 4, Cetaphyl® wash	1	30	no
	Oral corticosteroids, topical corticosteroid: High II	1	30	no
	Topical corticosteroid: Medium IV, anti-histamine	2	60	no
	Topical corticosteroid: Medium IV, EPI-MAX®, EPI-WASH®	1	30	no
	Topical corticosteroid: Medium V, EPI-MAX®, anti-histamine	1	30	no

<b>Eczema &amp; impetigo</b>	Mix 4, Oral penicillin, topical anti-bacterium	1	14	no
<b>Erythrasma</b>	Oral penicillin, topical anti-bacterium and anti-septic wash	1	14	no
	Oral penicillin, WHITFIELD'S® ointment	1	21	no
<b>Folliculitis</b>	Oral penicillin, topical anti-bacterium	5	60	added lincosamide or repeated
	Surgery, oral penicillin, topical anti-bacterium	1	14	added lincosamide
<b>Impetigo</b>	Oral penicillin, topical anti-bacterium, anti-histamine	1	14	no
	Oral penicillin, topical anti-bacterium	1	7	no
	Oral penicillin, topical anti-bacterium and anti-septic wash	1	14	no
	Oral penicillin, topical anti-bacterium, multivitamin	1	14	no
<b>Kaposi sarcoma</b>	Chemotherapy	5	90	repeated
<b>Kaposi sarcoma &amp; folliculitis</b>	Chemotherapy, oral penicillin, Topical corticosteroid: Medium V	1	21	chemotherapy repeated
<b>Kaposi sarcoma, impetigo and psoriasis</b>	Chemotherapy, oral penicillin, Topical corticosteroid: Medium V	1	30	tar, clobetasol
<b>Leprosy</b>	Dapsone® and topical anti-bacterium	1	60	no

	Dapsone® and topical anti-bacterium	1	90	no
<b>Melasma</b>	Mix 1, sunscreen	19	90	repeated
<b>Melanoma</b>	Surgery	2	-	chemotherapy
	Surgery, chemotherapy	1	30	no
<b>Psoriasis</b>	Mix 5, Oral corticosteroids, Methotrexate®	4	30	repeated
	Oral corticosteroids, topical corticosteroid: High II	1	60	no
	Topical corticosteroid: High II, oral corticosteroids, Methotrexate®	1	30	no
<b>Pityriasis rosea</b>	Oral penicillin, topical anti-bacterium	1	7	no
	Topical corticosteroid: Medium V, oral penicillin	1	7	no
<b>Pityriasis vesicolour</b>	Topical anti-fungal, oral anti-fungal	1	30	no
	Topical anti-fungal, oral anti-fungal	2	60	no
<b>Squamous cell carcinoma</b>	Chemotherapy	1	30	no
<b>Seborrheic dermatitis</b>	Oral penicillin, topical anti-bacterium	1	14	no
<b>TB</b>	RIFAMPICIN®, oral penicillin, topical anti-bacterium	1	180	no
	RIFAMPICIN®, topical anti-bacterium	1	90	no
	RIFAMPICIN®, topical anti-bacterium, multivitamin	1	90	no

<b>TB &amp; Impetigo</b>	RIFAMPICIN®, topical anti-bacterium,	1	180	no
	RIFAMPICIN®, topical anti-bacterium, anti-septic wash	1	180	Fluoro-quinolone
<b>Tinea corporis</b>	Anti-fungal shampoo, topical anti-fungal	1	21	no
	Anti-fungal shampoo, topical anti-fungal, multivitamin	3	90	no
<b>Tinea corporis &amp; Acne vulgaris</b>	Mix 2, sunscreen, anti-fungal shampoo	1	180	no
<b>Tinea corporis &amp; candida</b>	Anti-fungal shampoo, topical anti-fungal	1	180	no
<b>Tinea corporis &amp; Seborrheic dermatitis</b>	Anti-fungal shampoo, topical anti-fungal, multivitamin, topical anti-bacterium	1	14	no
<b>Tinea corporis</b>	Mix 5, oral anti-fungal	1	14	no
<b>Tinea capitis</b>	Anti-fungal shampoo, topical anti-fungal	1	14	no
<b>Tinea capitis &amp; folliculitis</b>	Oral penicillin, topical anti-bacterium, topical anti-fungal	1	14	no
<b>Tinea capitis &amp; seborrheic dermatitis</b>	Anti-fungal shampoo, topical anti-fungal, topical anti-bacterium	1	14	no
<b>Urticaria</b>	Topical corticosteroid: High II, anti-histamine	1	7	no
	Topical corticosteroid: Medium V, anti-histamine	1	7	no
<b>Vitiligo</b>	Topical corticosteroid: High II, anti-histamine	1	30	no

#### **4.1.5 Phases Combined**

In this section, the phases of the study are compared with one another. Only four variables were constant throughout the different phases; these variables will be compared.

##### **4.1.5.1 Dermatological Diagnoses**

Eczema showed the overall highest prevalence throughout the three phases. It was responsible for 11.4% of all cases documented. Acne vulgaris had the second highest prevalence with a total of 10.05%. Urticaria and contact dermatitis had the third and fourth highest prevalence respectively and melasma the fifth.

Eczema is a term readily used by doctors and pharmacists alike. A study done in 1996 on the accuracy and appropriateness of general practitioner's diagnoses in dermatological cases indicated that only 47% of the dermatological diagnoses that has been made were correct; it also indicated eczema to be the condition diagnosed the most (Basarab *et al.*, 1996:70).

Eczema has a long list of differential diagnoses ranging from psoriasis to zinc deficiency (refer to 2.7.1). All these patients that were diagnosed with eczema in this research study received treatment with topical corticosteroids.

The following table summarises the prevalence of dermatological diseases relevant to all phases of this research study:

Table 53: Overall Prevalence of Dermatological Diseases in this Research Study

Dermatological Disease	n	%
Eczema	55	10.85
Acne vulgaris	48	9.47
Urticaria	46	9.07
Contact dermatitis	44	8.68
Melasma	36	7.10
Candida	32	6.31
Cellulitis	29	5.72
Folliculitis	29	5.72
Herpes simplex	22	4.34
Tinea pedis	21	4.14
Psoriasis	19	3.75
Tinea corporis	16	3.16
Warts	15	2.96
Angioedema	12	2.37
Varicella zoster	12	2.37
Impetigo	11	2.17
Tinea capitis	9	1.78
Kaposi sarcoma	8	1.58
Melanoma	5	0.99
Pityriasis rosea	4	0.79
Erythrasma	3	0.59
Leprosy	3	0.59
Pityriasis vesicolour	3	0.59
TB	3	0.59
Seborrheic dermatitis	2	0.39
Squamous cell carcinoma	2	0.39
TB & impetigo	2	0.39
Tinea capitis & seborrheic dermatitis	2	0.39
Acne vulgaris & melasma	1	0.20
Acne vulgaris, psoriasis and urticaria	1	0.20
Basal cell carcinoma	1	0.20
Candida & impetigo	1	0.20
Contact dermatitis & impetigo	1	0.20
Eczema & impetigo	1	0.20
Kaposi sarcoma & folliculitis	1	0.20



<b>Kaposi sarcoma, impetigo, psoriasis</b>	1	0.20
<b>Tinea capitis &amp; eczema</b>	1	0.20
<b>Tinea capitis &amp; folliculitis</b>	1	0.20
<b>Tinea corporis &amp; acne vulgaris</b>	1	0.20
<b>Tinea corporis &amp; candida</b>	1	0.20
<b>Tinea corporis &amp; seborrheic dermatitis</b>	1	0.20
<b>Vitiligo</b>	1	0.20

This research study suggests that approximately half of the patients (49%) that consulted the general practitioner or dermatologist did so for the second time as they were receiving second-line treatment. It can be concluded that only half of the possible dermatological diagnoses that has been made were accurate and treatable the first time round.

Corticosteroids were prescribed at least four of the five most common dermatological diseases. Frequently, corticosteroids were prescribed without a diagnosis. It is implied from this data that since dermatological diseases are mostly not life-threatening they will not receive the high-quality and urgent healthcare as with life-threatening diseases such as diabetes or hypertension.

#### 4.1.5.1 Age Groups

Table 54: Dermatological Diseases According to Age – Relevant to all Phases of the Study

Dermatological Disease	≤2 years	>2 and ≤ 6 years	>6 and ≤ 12 years	>12 and ≤ 60 years	>60 years
Acne vulgaris	0	0	6	45	0
Angioedema	0	0	0	11	1
Atopic dermatitis	0	0	0	7	0
Basal cell carcinoma	0	0	0	1	0
Candida	0	0	2	27	5
Cellulitis	0	0	1	23	5
Contact dermatitis	0	1	3	35	6
Eczema	4	7	10	22	7
Erythrasma	0	0	1	2	0
Folliculitis	0	0	3	24	4
Herpes simplex	0	0	1	18	3
Impetigo	0	5	3	8	1
Kaposi sarcoma	0	0	1	8	1
Leprosy	0	0	1	2	1
Melanoma	0	0	0	4	1
Melasma	0	1	0	31	5
Pityriasis rosea	0	0	0	3	1
Pityriasis vesicolour	0	0	0	3	0
Psoriasis	0	0	0	17	4
Seborrheic dermatitis	0	0	0	4	0
Squamous cell carcinoma	0	0	0	1	1
Tinea capitis	0	0	3	12	1
Tinea corporis	0	1	3	3	2
Tinea pedis	0	1	1	26	2
Tuberculosis	0	1	0	3	1
Urticaria	0	0	0	36	11
Varicella zoster	0	0	2	7	3
Vitiligo	0	0	0	1	0
Warts	0	0	1	13	1

**Infant:  $\leq 2$  years**

Eczema was the only documented dermatological diseases in this age group. It can therefore be concluded that eczema has been found to have a distinct association with infants. Other studies have shown similar high prevalence of eczema in infants (Möhrenschlager *et al.*, 2006:503 & Van Hees, 2001:11). It must, however, be noted that in this research study, infants did not show the highest overall prevalence of eczema; should the diagnosis be correct, adults above 12 years showed the highest prevalence.

**>2 and  $\leq 6$  years**

Eczema showed the highest prevalence in this age group and Impetigo the second highest. Other studies, already mentioned, have shown us that impetigo is likely among toddlers. Like with eczema, impetigo's highest prevalence were among adults between the ages of 12 and 60 years.

**>6 and  $\leq 12$  years**

Eczema and *Acne vulgaris* had the highest prevalence in this age group. Eczema, as already mentioned, is common among children. 40.8% of all cases of eczema involved children up to 12 years of age. *Acne vulgaris* showed the second highest prevalence. It is well known that *Acne vulgaris* is stimulated in early puberty due to hormonal changes.

**>12 and  $\leq 60$  years**

*Acne vulgaris* (n=45), contact dermatitis (n=35) and urticaria (n=36) showed the highest overall prevalence in this age group. This is the biggest age group and classifies the patients as adults. As mentioned, *Acne vulgaris* is commonly diagnosed in patients around puberty. Contact dermatitis and urticaria are more likely to be found in the working-class adult than in children, due to higher risk of exposure to causative environmental factors.

Urticaria is more a description of symptoms than a disease; however, in this research study it was used as a diagnosis for a dermatological disease. The

causative agent for urticaria was not mentioned. Urticaria can be drug-induced; pressure-induced; cholinergic; related to stress; hot or cold weather; food; UV radiation etc. (refer to 2.7.9). This data was, however, not captured.

### **>60 years**

Urticaria and contact dermatitis once again showed the highest prevalence in this category. The conditions are not directly associated with age, but showed a high overall prevalence.

#### **4.1.5.1 Gender**

Gender has been found to have a direct association with some of the dermatological diseases. Candida, melasma and *Tinea pedis* were some of the diseases which indicated the highest difference between male and female. The reasons for these differences are well known and already extensively discussed in the other sections. Other diseases such as cellulitis, angioedema, folliculitis, Kaposi sarcoma and folliculitis also indicated a possible gender relationship.

Table 55: Dermatological Diseases According to Gender – Related to all Phases

Dermatological disease	Female	%	Male	%
Acne vulgaris	25	9.58	23	9.35
Acne vulgaris & melasma	1	0.38	0	0.00
Acne vulgaris, psoriasis and urticaria	0	0.00	1	0.41
Angioedema	3	1.15	9	3.66
Basel cell carcinoma	0	0.00	1	0.41
Candida	28	10.73	4	1.63
Candida & impetigo	0	0.00	1	0.41
Cellulitis	11	4.21	18	7.32
Conatct dermatitis	22	8.43	22	8.94
Contact dermatitis & impetigo	1	0.38	0	0.00
Eczema	26	9.96	29	11.79
Eczema & impetigo	0	0.00	1	0.41
Erythrasma	1	0.38	2	0.81
Folliculitis	17	6.51	12	4.88
Herpes simplex	10	3.83	12	4.88
Impetigo	5	1.92	6	2.44
Kaposi sarcoma	3	1.15	5	2.03
Kaposi sarcoma & folliculitis	0	0.00	1	0.41
Kaposi sarcoma, impetigo, psoriasis	0	0.00	1	0.41
Leprosy	2	0.77	1	0.41
Melanoma	2	0.77	3	1.22
Melasma	33	12.64	3	1.22
Pityriasis rosea	2	0.77	2	0.81
Pityriasis vesicolour	0	0.00	3	1.22
Psoriasis	6	2.30	13	5.28
Seborrheic dermatitis	0	0.00	2	0.81
Squamous cell carcinoma	0	0.00	2	0.81
TB	3	1.15	0	0.00
TB & impetigo	0	0.00	2	0.81
Tinea capitis	3	1.15	6	2.44
Tinea capitis & eczema	1	0.38	0	0.00
Tinea capitis & folliculitis	1	0.38	0	0.00
Tinea capitis & seborrheic dermatitis	2	0.77	0	0.00
Tinea corporis	8	3.07	8	3.25
Tinea corporis & acne vulgaris	1	0.38	0	0.00

<b>Tinea corporis &amp; candida</b>	0	0.00	1	0.41
<b>Tinea corporis &amp; Tinea pedis</b>	1	0.38	0	0.00
<b>Tinea pedis</b>	7	2.68	14	5.69
<b>Urticaria</b>	25	9.58	21	8.54
<b>Varicella zoster</b>	4	1.53	8	3.25
<b>Vitiligo</b>	1	0.38	0	0.00
<b>Warts</b>	6	2.30	9	3.66

#### 4.1.5.3 Race

Many studies have shown that race plays a significant role in dermatological diseases (Gawkrodger, 2002:11 & Kerr, 227:638).

Not only because of an anatomical difference in the different skin types, but also because of diverse cultures, living conditions, beliefs etc. (refer to 2.8.10).

Sixty-three per cent of the patients who participated in this research study were black, 20% white, 13.9% of the patients were coloured, 1.8% albino-black and less than 1% albino-coloured. This data reflect a fairly accurate race distribution in Namibia when compared with the census data for 2012, discussed in Chapter 1.

Diseases such as candida, folliculitis, impetigo, tinea infections etc. are directly correlated to unhygienic living conditions (refer to 2.8.2). The black patients showed the highest prevalence in the category of infective dermatological diseases, mainly due to their way of living and socio-economic conditions. Some of these infective diseases are also due to HIV/AIDS (refer to 2.8.1). In this research study, HIV positive patients were either black or coloured. This also explains the high rate of infective skin diseases and conditions such as Kaposi sarcoma.

Melanoma was the only skin disease more common in white than black patients. Eczema was common throughout the different races.

Melasma, one of the highest prevalent conditions in this research study, is more common in black patients than in any other race.

Albino patients showed the highest prevalence in infective skin conditions. The fact that these patients were albino was not the cause of their disease; rather, as already discussed, living conditions and financial status played a significant role (refer to 2.8.2). Coloured patients had the highest prevalence in contact dermatitis and eczema, while infective dermatological diseases such as folliculitis, *Candida albicans*, *Herpes simplex* etc. also had a significant prevalence.

Table 56: Dermatological Diseases According to Race – Relevant to all Phases

Dermatological Disease	Albino black	%	Albino coloured	%	Black	%	Coloured	%	Caucasian	%
Acne vulgaris	0	0.00	0	0.00	38	11.34	7	6.60	5	6.85
Angioedema	0	0.00	0	0.00	11	3.28	2	1.89	0	0.00
Atopic dermatitis	0	0.00	0	0.00	4	1.19	3	2.83	0	0.00
Basal cell carcinoma	0	0.00	0	0.00	0	0.00	1	0.94	0	0.00
Candida	2	20.00	0	0.00	21	6.27	5	4.72	6	8.22
Cellulitis	0	0.00%	0	0.00%	23	6.87	4	3.77	2	2.74
Contact dermatitis	0	0.00	0	0.00	28	8.36	8	7.55	9	12.33
Eczema	1	10.00	0	0.00	23	6.87	17	16.04	8	10.96
Erythrasma	0	0.00	0	0.00	2	0.60	0	0.00	1	1.37
Folliculitis	1	10.00	1	100.00	17	5.07	6	5.66	6	8.22
Herpes simplex	0	0.00	0	0.00	13	3.88	4	3.77	5	6.85
Impetigo	0	0.00	0	0.00	10	2.99	2	1.89	4	5.48
Kaposi sarcoma	0	0.00	0	0.00	9	2.69	0	0.00	1	1.37
Leprosy	0	0.00	0	0.00	3	0.90	0	0.00	0	0.00
Melanoma	0	0.00	0	0.00	1	0.30	4	3.77	0	0.00
Melasma	2	20.00	0	0.00	24	7.16	5	4.72	6	8.22
Pityriasis rosea	1	10.00	0	0.00	3	0.90	0	0.00	0	0.00
Pityriasis vesicolour	0	0.00	0	0.00	2	0.60	0	0.00	1	1.37
Psoriasis	0	0.00	0	0.00	11	3.28	8	7.55	2	2.74
Seborrheic dermatitis	0	0.00	0	0.00	4	1.19	1	0.94	0	0.00



<b>Squamous cell carcinoma</b>	0	0.00	0	0.00	1	0.30	1	0.94	0	0.00
<b>Tinea capitis</b>	0	0.00	0	0.00	12	3.58	0	0.00	0	0.00
<b>Tinea capitis &amp; eczema</b>	0	0.00	0	0.00	0	0.00	0	0.00	1	1.37
<b>Tinea corporis</b>	0	0.00	0	0.00	12	3.58	0	0.00	1	1.37
<b>Tinea pedis</b>	1	10.00	0	0.00	17	5.07	5	4.72	6	8.22
<b>Tuberculosis</b>	0	0.00	0	0.00	3	0.90	1	0.94	1	1.37
<b>Urticaria</b>	0	0.00	0	0.00	25	7.46	16	15.09	6	8.22
<b>Varicella zoster</b>	1	10.00	0	0.00	8	2.39	2	1.89	1	1.37
<b>Vitiligo</b>	0	0.00	0	0.00	0	0.00	1	0.94	0	0.00
<b>Warts</b>	1	10.00	0	0.00	10	2.99	3	2.83	1	1.37

## **Chapter Summary**

At the beginning of this chapter, the empirical study process has been discussed; the response rate defined and the results for each phase stated.

The results from Phase 1 and 2 have been analysed and discussed; whereafter the results have been combined to conclude which dermatological diseases indicate the highest prevalence.

## CHAPTER 5: CONCLUSION AND RECOMMENDATIONS

In this chapter, the relevant conclusions are discussed and recommendations are made accordingly. This discussion follows the specific objectives stated in Chapter 1.

### 5.1 LITERATURE STUDY

The literature study has been conducted before the empirical study was initiated. This was done to ensure a broad spectrum of knowledge about dermatology as a subject before data were collected and analysed.

The following objectives were explored in literature study:

*5.1.1 To describe to anatomy and physiology of the skin and classification system for skin lesions:*

The anatomy and physiology of the skin were studied extensively to ensure that sufficient knowledge was available before the empirical study was initiated. This knowledge was especially applied in Phase 1B of the research study. Pharmacist-initiate therapy was positively welcomed by the patients who participated in this phase. The knowledge gained by studying the anatomy of the skin allowed the pharmacist to better explain the current dermatological condition to the patient. Treatment compliance and follow-up consultation were increased by this action.

It can be concluded that many practitioners lack sufficient knowledge of the anatomy and dermatology of skin disorders as a whole. Although dermatological diseases have a high prevalence in Namibia, most healthcare practitioners are not especially concerned about providing accurate dermatological diagnoses since most of the conditions are not life-threatening. As already mentioned in Chapter 1, Namibia has a shortage of general practitioners. The practising general practitioners, therefore, focus their time and effort on life-threatening diseases and may neglected patients with dermatological diseases.

5.1.2 *To identify and describe different dermatological conditions with high prevalence in Namibia and define the causes of these conditions:*

The most common dermatological diseases were identified from various studies done on dermatology in Africa. These conditions included various dermatophyte infections, dermatitis, psoriasis, urticaria and bacterial infections. The following conditions were identified in Namibia: Eczema, *Acne vulgaris*, urticaria, melasma, cutaneous *Candida albicans*, cellulitis, folliculitis and *Herpes simplex*.

The aetiology for each condition was identified in literature. By describing these conditions, the data could be interpreted more effectively. Unfortunately, studies that described dermatological conditions in Namibia were not available at the time.

Five-hundred-and-seven cases were documented within the three-month period of data collection, which indicates that 0.02% of the total population of Namibia suffered from dermatological diseases during that three-month period. This reveals the relevance of dermatological diseases in Namibia.

5.1.3 *To describe the treatment regime of these conditions:*

Treatment regimes for each dermatological condition were described in literature study. These regimes were not studied to scrutinise prescription obtained in Phase 1A and 2, but rather explore the variety of treatment available.

5.1.4 *To identify the relationship between different dermatological conditions and other contributing factors:*

Literature was studied to identify relationships among factors such as demographics, underlying HIV/AIDS, chronic diseases, allergies, climate in a geographical area and dermatological conditions. It has been concluded from that certain contributing factors have direct relationships with dermatological diseases.

(a) Age

Dermatological diseases such as eczema have been found to have a higher prevalence in children under the age of 2 years. Urticaria, fungal infections and contact dermatitis have been found more prevalent in the working-class adult, and the elderly mostly suffering from atypical eczema and contact dermatitis (McKoy, 2012).

(b) Gender

Dermatological diseases like *Tinea pedis* and contact dermatitis have been described to be more frequent in males than females. Melasma and candida infections are more likely to be diagnosed in females than males (Shimizu, 2007:467).

(c) Race

Anatomical difference in skin types indicates a difference in the type of dermatological diseases. Other cultural factors, such as poor hygiene and poverty are also defined as have a significant part in the prevalence of dermatological diseases (Accorsi *et al.*, 2009:469).

(d) HIV/AIDS

Ninety per cent of patients with HIV/AIDS will develop some kind of dermatological condition during their lifetime (Australian dermatologists, 2001). Patients with HIV/AIDS often suffer from diseases such as candidiasis, molluscum contagiosum, folliculitis, Cryptococcus and Kaposi sarcoma (Amerson *et al.*, 2010:16).

(e) Chronic Disease

Chronic conditions such as kidney diseases can cause uremic pruritus (Chaturvedy, 2012:284). Many patients with diabetes mellitus are present with granuloma annulare, necrobiosis lipoidica, xanthoma, bullous disease, neuropathic leg ulceration, lupus pernio of the nose, erythema nodosum and sarcoidosis. Patients with hyperthyroidism or Grave's Disease often also suffer from pretibial myxoedema, diffuse alopecia and palmar erythema. Cushing's Syndrome can cause thinning of the skin, spontaneous bruising, striae, diffuse alopecia, acne and hirsutism (Narayan, 2009:227).

*(f) Allergies*

Many patients are presented with pruritus and other dermatological symptoms due to allergies which, in effect, cause contact dermatitis. Nickel, a metal frequently found in jewellery, is probably the most common cause. Gold, silver and other metals like cobalt chloride found in antiperspirant and hair-dye can cause an allergic reaction (De Noon, 2006:1).

*(g) Climate and Geographical Area*

Diseases such as dermatophyte infections are more prominent in the rainy season or geographical areas where humidity and temperatures are high (Del Boz-Gonzalez., 2012:288). Dermatitis, like atypical dermatitis, is the opposite and more prominent in low humidity climate (Figueroa, 2011:311).

According to Figueroa (2011:311), the thinning of the ozone layer and changes in global climate play a significant role in dermatological disease. Diseases like acne and skin infections caused by gram-positive and negative bacteria are being worsened by the contamination of water. The increase in skin cancer by 2050 is estimated to be 300% by some, due to climate change (Figueroa, 2011:312).

*(h) Pregnancy*

The most common skin disease during pregnancy is hyperpigmentation and melasma. The increase in melanin causes the condition to appear. Pruritic urticaria papules and plaques of pregnancy can cause small reds lumps that itch or burn (Tunzi *et al.*, 2007:211).

*(i) Cigarette Smoke*

Cigarette smoke can cause or increase the chances of developing systemic lupus erythematosus, psoriasis, palmoplantar pustulosis, cutaneous squamous cell carcinoma, hidradenitis suppurativa, candida and genital warts (Thomson *et al.*, 2010:4). It also causes various degenerative dermatologic conditions such as skin wrinkling and decreased wound healing (Mottilo *et al.*, 2009:718).

### (j) Genetics

Like in any other disease, genetics plays a vital role in some dermatological disorders. Diseases such as melanoma has a 5 - 10% chance of being inherited by family (Leichman *et al.*, 2009:e1). Atopic eczema also has a strong familial correlation. A study done on monozygotic vs dizygotic twins with eczema indicated that the monozygotic twins had a 72% probability of having or developing eczema whereas the dizygotic twins only had only 21% probability (Forest *et al.*, 1999:1067). Psoriasis had also shown the same concordance with monozygotic and dizygotic twins (Bowcock *et al.*, 2004:45). Some studies have estimated that at least two-thirds of psoriasis cases are accounted for by genetic factors (Yonghong *et al.*, 2004:318).

## 5.2 EMPIRICAL STUDY

After studying the data intensively, it has been concluded that the dermatological diseases in Namibia are not typically the diseases found in the rest of Africa. The spectrum of dermatological diseases includes eczema, urticaria, contact dermatitis, *Acne vulgaris*, *Candida albicans*, melasma, cellulitis and folliculitis.

The following objectives were explored in the empirical study:

*5.2.1 To identify the prevalence of dermatological conditions in Windhoek by identifying patients with dermatological conditions. This includes patients with prescription from their general practitioner, patients who consulted the dermatologist and patients who consulted to the community pharmacy for pharmacist-initiated therapy:*

In 4.1.5.4 the results indicates that eczema (n=55) showed the highest overall prevalence. Acne vulgaris (n=48) had the second highest prevalence, urticaria (n=46), contact dermatitis (n=44) and melasma (n=36) followed.

During the data collection process, the term eczema was used extensively by practitioners and patients alike. Most patients were partially informed about what the condition entailed. In Phase 1A when practitioners were contacted to receive information about the current condition, eczema was often found to be the cause.

It can be concluded that environmental factors such as the hardness of the drinking water and mica dust from the surrounding Khomas Mountains are among the main causes of the high prevalence of eczema. According to Van Hees (2001:11), the use of petroleum jelly plays a significant role in eczema. It is recommended that these factors should be further investigated.

Although this study has not focused on scrutinising the diagnosis obtained by the patient, the fact that many patients simply suffered from dry skin, and not eczema, cannot be ignored. Many patients diagnosed with eczema actually did have a fungal infection. This data were unfortunately not captured and are defined as a limitation to this study.

*Acne vulgaris* showed the second highest prevalence in this study. It showed high prevalence in data collected in Phase 1A & 2, but not in Phase 1B. It can therefore be concluded that patients with *Acne vulgaris* seek healthcare from medical practitioners rather than consulting the pharmacy for pharmacist-initiated therapy. This is most likely due to fact that *Acne vulgaris* is mostly found on the face which affects self-esteem and social standing.

Urticaria and contact dermatitis also showed high prevalence throughout this study. These dermatological conditions are associated with exposure to chemicals or other substances in the workplace. Most of the data were collected in the vicinity of industrial areas.

#### *5.2.2 To determine the relationship between the different dermatological conditions and demographical data such as age, race and gender:*

In this research study, demographic data such as age, race and gender were collected in all phases. This data indicate the relationship between different dermatological conditions and groups of patients that are mostly at risk.

Different demographical trends have also been noticed. It is concluded that babies and young toddlers are more prone to suffer from eczema. Children over 12 years of age commonly suffer from *Acne vulgaris* and the elderly suffered from urticaria and



cutaneous fungal infections. It was further concluded that male patients suffer from contact dermatitis and *Tinea pedis*, while female patients suffer from *Candida albicans* and melasma. Disease like eczema does not show any difference in prevalence in male and female.

Black patients show high prevalence in *Acne vulgaris*, eczema, melasma, contact dermatitis, and other infective dermatological diseases. A study done in 2011 compared the prevalence of acne among African American, Hispanics, Indian, Caucasian and Asian women. The African American women showed the highest prevalence (Perkins *et al.* ,2001:1054). In this study, 79.5% of the patients diagnosed with *Acne vulgaris* are black. Caucasian patients showed the highest prevalence in eczema and urticaria. Coloured patients have high prevalence in infective dermatological diseases.

### *5.2.3 To determine the geographical distribution of patients with dermatological diseases in Phase 2:*

Geographical trends were explored in Phase 2 only since the patients who participated in the other phases were all from Windhoek. The data from the dermatologist offered a broad spectrum of patients since he was one of five dermatologists practising in Namibia, and patients came for consultation from all over the country.

It can be concluded that specific regions have higher prevalence due to differences in climate.

The northern parts of Namibia show high prevalence in a variety of dermatological diseases. The diseases with the highest prevalence include melasma, candida and other infective dermatological diseases. The northern parts of Namibia receive more rainfall than other parts of the country, making the climate more favourable for bacterial and fungal infections. Furthermore, the northern parts of Namibia have the highest population density in the country. These contagious infective diseases easily spread in populated areas due to overcrowding.

Dr. Tara Lumpkin (refer to 4.1.4.1) indicate that towns like Oshakati in the far north of Namibia, has one of the highest HIV/AIDS rates in the World. It can be concluded that HIV/AIDS definitely impacts on the high prevalence of dermatological diseases in this area.

*5.2.4 To investigate the possible differences in the prevalence of dermatological conditions between HIV positive and HIV negative patients in Namibia.*

In Phase 1A, 16.98% of the patients who participated were HIV positive and in Phase 2, 19% were HIV positive. The four dermatological diseases with the highest prevalence in HIV/AIDS patients were *Herpes simplex* (n=8), Kaposi sarcoma (n=6), impetigo (n=5) and *Acne vulgaris* (n=5).

In Phase 1A it can be concluded that of the total 23% of patients with *Herpes simplex*, 17% of them were HIV positive, which confirms an association between *Herpes simplex* and HIV/AIDS. *Herpes Simplex* is a common opportunistic disease in AIDS, because it is very contagious and remains life-long in its host (Strick *et al.*, 2006:347).

In Phase 2, the patients that were referred to the dermatologist by their general practitioner, were diagnosed with Kaposi sarcoma. As already mentioned, the prevalence of this dermatological disease is seemingly high compared to data from literature. It can be concluded that this is due to the fact that the dermatologist is one of five available in the country and the only one claiming directly from the government and private medical aid funds.

Impetigo and *Acne vulgaris* are defined as common opportunistic diseases due to AIDS. Immune reconstitution diseases (IRD) are being defined as diseases that appear after ARV have been initiated. *Acne vulgaris* has recently been described as such as disease (Scott *et al.*, 2003:493). Most of the above mentioned, conditions can, however, be present in the absence of HIV/AIDS.

### 5.2.5 To formulate recommendations on the treatment regarding dermatological conditions in the private healthcare sector of Windhoek:

The three dermatological diseases with the highest prevalence are discussed, namely eczema, *Acne vulgaris* and urticaria.

The following protocol for eczema is recommended:

- Discontinue the use of any type of soap and use only aqueous cream as soap for bathing.
- Use moisturising bath emollients in bath water, such as Epi-bath®
- Use moisturisers free from perfumes and artificial colourant. Moisturise twice a day using, for example, Epi-max®.
- If eczema breakout does occur use either calcineurin inhibitors, tacrolimus, pimecrolimus, or a low dose glucocorticosteroids. Only increase glucocorticosteroids if no result is obtained within 7 days of treatment. It is further suggested that the lowest strength corticosteroid must first be initiated before stronger corticosteroids are prescribed.
- Inform the patient about possible eczema triggers like potentially allergic foods, washing powders, seasonal changes, chemicals in water and other environmental factors.
- If no change occurs, refer the patient to a dermatologist.

The following protocol for *Acne vulgaris* is recommended:

- Discontinue the use of strong, soapy facial washes and scrubs.
- Educate patient on possible triggers for acne breakout and the avoidance of picking, rubbing, scratching and damaging pimples.
- Initiate oral zinc and selenium containing tablets and facial wash.
- Initiate water-based sunscreen.

If no change occurs:

- Initiate topical anti-microbial therapy.
- Initiate hormonal therapy if the patient is female.
- Initiate oral anti-microbial therapy if the patient is male.

If no change occurs:

- Initiate topical and oral isotretinoin therapy.

If no change occurs:

- Refer the patient to a dermatologist.

The following protocol for urticaria is recommended:

- Identify a possible cause of the urticaria and avoid it.
- Initiate a non-sedating antihistamine

If no change occurs:

- Increase the dose of the non-sedating antihistamine
- Initiate topical antihistamine such as mepyramine maleate.

If no change occurs, or in severe cases:

- Initiate a topical and oral glucocorticosteroids if required.

For chronic urticaria:

- Initiate a non-sedating antihistamine

If no change occurs:

- Increase the dose of the non-sedating antihistamine

If no change occurs:

- Combine two different non-sedating antihistamine

In severe cases:

- Initiate a topical and oral glucocorticosteroids if required.

### 5.3 RECOMMENDATIONS

Based on the results obtained in Chapter 4 and the conclusions listed above, the following recommendations are made:

- Eczema has the highest prevalence in this study; it is recommended that differential diagnosis of eczema is explored more often.
- Melasma shows a high prevalence throughout this study. The high solar radiation in Namibia is responsible for this condition. It is recommended that the public is made more aware of the intense solar radiation in Namibia and

that the use of sun screens, hats and umbrellas throughout the year is emphasised.

- Topical corticosteroids were prescribed most often during the course of this research study. It is recommended that healthcare practitioners are made more aware of other non-steroidal treatments available. The community pharmacists also have a variety of homeopathic remedies at their disposal. It is recommended that these alternative medicines are explored more frequently in the treatment of dermatological diseases. Older treatment regimes such as coal-tar mixtures are also available over the counter.
- It is recommended that public awareness and knowledge on dermatological diseases are increased. The northern parts of Namibia have multiple clinics spread across the countryside which can be used as a base to provide education to the public on basic hygiene and skin-care. The dangers of using Vaseline® as a primary moisturiser should also be emphasised.
- It is recommended that patients and practitioners been made aware of opportunistic infections such as *Herpes simplex*. HIV/AIDS patients should be informed on the prevention of *Herpes simplex*, for example staying out of the sun and starting treatment for a *Herpes simplex* infection as soon as the lesion appears. The same protocol for *Acne vulgaris* and impetigo should be followed. It is further recommended that practitioners should be more alert to HIV/AIDS patients being presented with dermatological diseases, as it can provide insight into their current CD-4 count.
- It is recommended that future studies in Namibia regarding dermatology are undertaken.
- It is recommended that the relationship between the community pharmacist and general practitioner is improved, ensuring best possible treatment for patients with dermatological diseases.

## 5.4 LIMITATIONS

A variety of limitations were encountered throughout the course of this research study. These limitations could have affected the data and should be taken in consideration when results are interpreted.

- The data collection was conducted within a three-month period. Seasonal changes during this period were not taken into consideration.
- Differential diagnoses were not documented in any of the three phases of this study.
- In some cases, patients had more than one dermatological condition; this affected the percentage of patients suffering from dermatological diseases. These percentages are an indication of the number of cases of dermatological diseases, rather than patients.
- In Phase 1A, the prescribing practitioner was contacted to retrieve dermatological diagnoses; this data was captured based on the verbal information given by the practitioner.
- In Phase 1A, the dermatological diagnoses were provided based on the memory of the practitioner. On many occasions, the practitioner was contacted while he/she was in consultation with another patient.
- In phase 1B, the treatment success or failure was not documented, unless a patient returned for a follow-up consultation.
- In Phase 1B, the data do not indicate whether a patient visited the same doctor twice for the same condition– only the patient received second-line treatment.

- In Phase 2, some of the variables were not available from the patient's files, and the data were collected from the dermatologist himself, based on his memory of the particular case.

## **5.5 CHAPTER SUMMARY**

In this chapter the specific objectives are concluded and recommendations and limitation to the study are described.

## **5.6 FINAL WORD**

There are many ways in which skin disease can adversely affect the quality of an individual's life. It is important that doctors and pharmacists alike treat dermatological diseases as any other life-threatening disease, with proper investigation, diagnoses and treatment. Effective dermatological treatment has been neglected in the private healthcare sector of Namibia. The community pharmacist is the most accessible and affordable healthcare professional to treat most dermatological conditions.



**ANNEXURE: A**



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**Medicine Usage in South Africa**

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Fax: 018 2994244

Email: martie.lubbe@nwu.ac.za

***Dear Patient***

You are invited to participate in a study conducted by the Research entity, Medicine Usage in South Africa at the School of Pharmacy from the North-West University in South Africa.

We hope to learn more about skin diseases in Namibia's private healthcare system. You were selected as a possible participant in this study because of your current symptoms.

If you decide to participate, I Ronja King, will document your specific skin condition and/or your doctor's prescription. Your medical history will also be documented in a short questionnaire. This process will take less than 15 minutes of your time. This research project is an observational study only and no tissue, blood or any other samples will be collected.

Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission or as required by law.

If you have any questions, please ask me. If you have any additional questions later, you can also phone me at 081 612 3735 or 061 239 241.

YOUR SIGNATURE INDICATES THAT YOU HAVE DECIDED TO PARTICIPATE, HAVING READ THE INFORMATION PROVIDED ABOVE.

You will be provided with a copy of this form.

Thank you.

A handwritten signature in purple ink that reads 'King'.

.....  
Ronja King

.....  
Patient signature

.....  
Date

## ANNEXURE: B - Community Pharmacy Questionnaire Phase 1A

### A: Demographic Information:

1. Gender: \_\_\_\_\_
2. Date of birth: \_\_\_\_\_
3. Race: \_\_\_\_\_
4. Occupation: \_\_\_\_\_

### B: Medical Information:

1. Do you have any chronic diseases like blood pressure, diabetes, heart disease etc.?  
\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_
2. Are you taking any chronic medications, non-prescription drugs like vitamins or herbal supplements?  
If yes, please list.  
\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_
3. Are you being treated for any other medical conditions at present? If yes, please state for what you are being treated.  
\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_
4. Do you have any allergies? If yes, please state to what you are allergic?  
\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_
5. Do you have any conditions or receive any therapies that could affect your immune system, e.g. leukemia, HIV/AIDS, radiotherapy, chemotherapy?  
\_\_\_\_\_
6. Have you discovered any new moles on your skin or any other abnormal skin growths?  
\_\_\_\_\_
7. Do you easily burn in the sun? \_\_\_\_\_
8. Do you daily wear sun screen? \_\_\_\_\_
9. Have you ever had something removed on/from your skin, e.g. moles, warts, skin growths, cancer?  
\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

10. What cosmetic skin care products do you use?

---

---

---

---

11. Do you smoke? \_\_\_\_\_

12. Do you drink alcohol? \_\_\_\_\_

13. Are you pregnant or breast-feeding? \_\_\_\_\_

**D: Dermatological condition/disease:**

1. Is this the first time you are receiving treatment for this skin condition?

---

2. Does any person in your direct family have the same skin condition? \_\_\_\_\_

3. How long have you had this condition before seeking healthcare?

---

**Thank you for filling in this patient information sheet. Your time is appreciated.**

*To be filled in by the PHARMACIST:*

Diagnosis: \_\_\_\_\_

Treatment: \_\_\_\_\_

---

---

Treatment duration: \_\_\_\_\_

## ANNEXURE: C - Community Pharmacy Questionnaire Phase 1B

\*To be completed by the PHARMACIST

### A: Demographic Information:

1. Gender: \_\_\_\_\_
2. Date of birth: \_\_\_\_\_
3. Race: \_\_\_\_\_
4. Occupation: \_\_\_\_\_
5. Contact number for follow-up: \_\_\_\_\_

### B: Medical Information:

6. Do you smoke? \_\_\_\_\_
7. Do you drink alcohol? \_\_\_\_\_
8. Are you pregnant or breast-feeding? \_\_\_\_\_
9. Do you have any allergies? If yes, please state to what you are allergic?  
\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_
10. Are you being treated for any medical condition at present? If yes, please state for what you are being treated. \_\_\_\_\_  
\_\_\_\_\_
11. Are you taking any medications, non-prescription drugs or herbal supplements? If yes, please list.  
\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_
12. Do you have any conditions or therapies that could affect your immune system, e.g. leukemia, HIV/AIDS, radiotherapy, chemotherapy?  
\_\_\_\_\_
13. Do you have a bleeding disorder?  
\_\_\_\_\_
14. Do you suffer from any chronic dermatological diseases?  
\_\_\_\_\_  
\_\_\_\_\_
15. Does anybody in your direct family suffer from any dermatological diseases?  
\_\_\_\_\_

---

---

16. Have you discovered any new moles on your skin or any other abnormal skin growths?

---

17. Do you easily burn in the sun? \_\_\_\_\_

18. Do you daily wear sun screen? \_\_\_\_\_

19. Have you ever had something removed on/from your skin, (e.g. moles, warts, skin growths, cancer)?

---

---

20. What cosmetic skin care products do you use?

---

**C: Physical examination:**

21. How long has this condition lasted? \_\_\_\_\_

22. Where on the body did it start? \_\_\_\_\_

23. Did it spread to other parts of the body and over what period?

---

---

24. Is it present continuously or does it come and go over time?

---

25. Have you ever had this condition before? \_\_\_\_\_

26. Are the lesions moist or dry?

---

27. Does it itch? \_\_\_\_\_

28. Does the itching become worse during day/night? \_\_\_\_\_

29. Does the lesion swell or form nodules/pustules?

---

30. Do any of your friends or colleagues have the same condition? \_\_\_\_\_

31. Describe the type of lesion:

---

---

---

**D: Diagnosis and treatment:**

32. Possible diagnosis: \_\_\_\_\_

33. Treatment: \_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

34. Treatment duration: \_\_\_\_\_

**E: Follow-up:**

35. Telephonic description of lesion after treatment

\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

36. Physical examination of lesion after treatment

\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

37. Refer to medical practitioner? \_\_\_\_\_

**ANNEXURE: D**



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**Medicine Usage in South Africa**

Tel: 018 2992288

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Email: martie.lubbe@nwu.ac.za

**TO WHOM IT MAY CONSERN**

I, Dr. FJA Smith, hereby give consent for the data collection that will be done by Mrs. Ronja King from my practice for a study conducted by the Research entity, Medicine Usage in South Africa at the School of Pharmacy from the North-West University in South Africa.

**Dermatological disorders in Namibia, with special reference to the private healthcare sector in Namibia.**

The following data will be collected:

- Demographic data including age, race, gender and region where person lives in Namibia (geographical location)
- Diagnosis of dermatological disease
- Recorded observation of type of lesion
- Treatment provided
- Duration of treatment
- The number of patients that are also on anti-retroviral, TB and oncology treatment
- Follow-up results when available

No personal patient information will be used in this study, recorded or taken into consideration. Data will only be used for research purposes and no information collected will be traceable to individual patients. Doctor-patient confidentiality will be kept at all times.

A handwritten signature in blue ink, appearing to read 'FJA Smith', written over a dotted line.

Practitioner

## ANNEXURE E:

A: Demographic data of patient:

Date of birth	
Race	
Gender	
Geographical Region	

B: Diagnosis of Dermatological Disease:

--

C: Observation of skin lesion:

--

D: Treatment of dermatological problem and duration of treatment:


E: Patient receiving treatment for:

ARV treatment	
TB treatment	
Oncology treatment	

F: Follow-up results & treatment adjustments:




**ANNEXURE: F**



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***Dear Patient***

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We hope to learn more about skin diseases in Namibia's private healthcare system. You were selected as a possible participant in this study because of your current symptoms.

If you decide to participate, I Ronja King, will document your specific skin condition from your file at Dr. Smith's practice. Your medical history and the doctor's notes will be documented. All your information regarding treatment will also be documented.

This research project is an observational study only and no tissue, blood or any other samples will be collected.

Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission or as required by law.

If you have any questions, please ask me. If you have any additional questions later, you can also phone me at 081 612 3735 or 061 239 241.

YOUR SIGNATURE INDICATES THAT YOU HAVE DECIDED TO PARTICIPATE, HAVING READ THE INFORMATION PROVIDED ABOVE.

You will be provided with a copy of this form.

Thank you.

A handwritten signature in cursive script that reads 'King'.

.....  
Ronja King

.....  
Patient Signature

.....  
Date

## ANNEXURE: G

# Prevalence of dermatological disorders among patients in an urban area in Namibia

Ronja King, Martie Lubbe, Jan Gerber, Jesslee du Plessis

Niche area: Medicine Usage in South Africa (MUSA), North-West University (Potchefstroom campus), Potchefstroom, South Africa



## Introduction

The burden of skin diseases has been increasingly emphasised over the past few years. Dermatological disorders and diseases are common in developing countries due to underlying causes such as HIV/AIDS, poor hygiene, overcrowding and poverty in some areas<sup>1</sup>. Skin diseases such as scabies, superficial mycoses, pyoderma, pediculosis, eczema, dermatitis, pigmentary abnormalities, acne and HIV/AIDS related skin diseases are most common in developing countries<sup>2</sup>.



## Objective

To establish the prevalence of dermatological disorders among general practitioners' patients in an urban area in Namibia.

## Methods

### Research design:

Descriptive, cross-sectional analysis.

### Study setting:

- Two community pharmacies located in the Northern and Southern parts of Windhoek, Namibia.
- Community pharmacies were strategically chosen to ensure a broad spectrum of prescriptions from various ethnic groups in an urban area.

### Study population:

- 302 dermatological prescriptions obtained over a three month period.

### Inclusion criteria:

- Every second patient with a dermatological prescription.
- Only general practitioners' prescriptions.

### Data obtained:

The following patient and prescription details were recorded:

- Patient's gender, age and race.
- Diagnosis as obtained from the patient or general practitioner (telephonically).
- Medical treatment and treatment duration.
- HIV-status.

### Statistical analysis

- Statistical analyses were performed using SAS Software, version 9.3 (SAS, Cary, NC).
- Association between dermatological disorders and gender, age group, race, and HIV-status were investigated using the Chi-square test.
- P-values < 0.05 was considered statistically significant.
- Cramer's V statistics was used to test for practical significance of associations.

### Ethical considerations

- Ethical permission from the Ethical Committee North-West University: NWU-00061-12-5.
- Written consent to participate in the study was obtained from every patient.

## Results

Approximately 57.3% of patients with dermatological conditions were black (Fig 1), 50.7% (n=153) were male, and 72.2% (n=218) were in the age group older than 12 and younger than 60 years (Fig 2).



Fig 1 Dermatological problems according to race

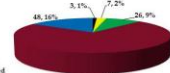


Fig 2 Dermatological problems according to age group



- Urticaria was the most common skin disease identified, affecting 11.9% of all patients (Table 1).
- Other conditions included contact dermatitis (9.3%), eczema (9.3%), Acne Vulgaris (8.3%), Herpes Simplex (7.3%), cellulitis (6.3%) and folliculitis (6.3%) (Table 1).
- Statistical and practical significant association were found between the type of dermatological disorder and
  - gender ( $P = 0.0040$ , Cramer's  $V = 0.3977$ ),
  - age group ( $P < 0.0001$ , Cramer's  $V = 0.3762$ ) and
  - HIV-status ( $P = 0.0002$ , Cramer's  $V = 0.4361$ ).
- No statistical and practical significant association were found between the race of the patient and the type of dermatological disorder ( $P > 0.05$ ).

### Prevalence of dermatological disorders: Gender spreading

- Male patients experienced predominantly problems with Tinea Pedis (n=12), Herpes Simplex (n=12), cellulitis (n=11), angioedema (n=8), psoriasis (n=6) and atopic dermatitis (n=6).
- Female patients were mostly be subjected to Acne Vulgaris (n=14), candida infections (n=14), melasma (n=15), contact dermatitis (n=14), folliculitis (n=11).

### Prevalence of dermatological disorders: Age group spreading

- Urticaria was predominant in the age groups > 6 to ≤ 12 years (n=24) and > 12 to ≤ 60 years (n=10).
- Contact dermatitis was more prevalent in the possible employed age group > 12 to ≤ 60 year (n=21) than in the other age groups.
- Patients ≤ 12 years experienced mostly eczema (n=14) and impetigo (n=4).
- Acne Vulgaris was more common in the age groups > 6 to ≤ 12 years (n=6) and > 12 to ≤ 60 years (n=19).
- Herpes Simplex (n=18), cellulitis (n=15) and folliculitis (n=14) were prominent in the age group > 12 to ≤ 60 years.

### Prevalence of dermatological disorders in HIV-positive patients

- 51 patients (16.9%) were HIV-positive.
- Experienced mostly problems with Herpes Simplex (n=8), urticaria (n=6), impetigo (n=4), warts (n=4) and melasma (n=4).

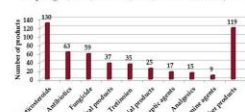


Fig 3 Medical treatment of dermatological disorder

Table 1 Prevalence of dermatological disorders

Dermatological Disorders	N	%
Angioedema	10	3.3
Atopic Dermatitis	7	2.3
Acne Vulgaris	25	8.3
Candida	18	6.0
Contact Dermatitis	28	9.3
Cellulitis	19	6.3
Eczema	28	9.3
Erythrasma	1	0.3
Folliculitis	19	6.3
Herpes Simplex	22	7.3
Impetigo	7	2.3
Kaposi Sarcoma	3	1.0
Leprosy	1	0.3
Melasma	16	5.3
Melanoma	2	0.7
Psoriasis	7	2.3
Pityriasis Rosea	2	0.7
Squamous Cell Carcinoma	1	0.3
Seborrheic Dermatitis	1	0.3
Tinea Corporis	7	2.3
Tinea Capitis	5	1.3
Tinea Pedis	18	6.0
Urticaria	36	11.9
Varicella Zoster	9	3.0
Warts	10	3.3

### Medical treatment of dermatological disorders

- Patients received on average 1.69 [95% CI: 1.59-1.77] dermatological items per prescription, with 49.0% of patients receiving only one, 34.4% received two and 16.6% received more than two items.
- The most common dermatological items prescribed were corticosteroids (25.5%), other products (23.4%), antibiotics (12.4%) and fungicides (11.6%) (Fig 3).
- The corticosteroids were predominantly prescribed for urticaria, eczema, and contact dermatitis.
- Antibiotics were prescribed for Acne Vulgaris, cellulitis and folliculitis.
- Fungicides were prescribed for candida, Tinea Corporis, Tinea Capitis and Tinea Pedis.

## Conclusions

The results reveal the prevalence of dermatological disorders and the treatment thereof in the private health system of Namibia. It will make health professionals in Namibia more aware of the significance of dermatological diseases in their region.



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## Disclosure

The authors declare no conflict of interest

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## ANNEXURE: H

## ANNEXURE: I

### Prevalence of dermatological disorders among patients in an urban area in Namibia

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**Background** The burden of skin diseases has been increasingly emphasised over the past few years. Dermatological disorders and diseases are common in developing countries due to poor hygiene, underlying causes such as HIV/AIDS, overcrowding in some areas and poverty. Skin diseases such as scabies, superficial mycoses, pyoderma, pediculosis, eczema, dermatitis, pigmentary anomalies, acne and HIV/AIDS related skin disease are most common in developing countries.

**Objectives** To establish the prevalence of skin diseases among patients in an urban area in Namibia.

**Methods** A cross-sectional study was done on 302 dermatological prescriptions obtained over a three month period, from two community pharmacies, located in the Northern and Southern parts of Windhoek in Namibia. These pharmacies were strategically chosen to ensure a broad spectrum of prescriptions from various ethnic groups. Only general practitioners' prescriptions were included, Prescriptions from dermatologists were excluded. Written consent to participate in the study was obtained from every second patient with a dermatological prescription. The following prescription details were recorded: Patient gender, age, race, the diagnosis as obtained from the patient or general practitioner (telephonically); medical treatment and treatment duration, smoking, pregnancy, HIV status and allergy information. Association between dermatological disorders and gender, age group, race, and HIV-status were investigated using the chi-square test. P-values less than 0.05 was considered statistical significant.

**Results** Approximately 57.3% of patients with dermatological conditions were black, 50.7% were male, and 72.2% were in the age group older than 12 and younger than 60 years. Urticaria was the most common skin disease identified, affecting 11.9% of all patients. Other conditions included contact dermatitis (9.3 %), eczema (9.3%), acne vulgaris (8.3%), Herpes Simplex (7.3%), cellulitis (6.3%) and folliculitis (6.3%). Patients received on average 1.69 [95% CI, 1.59-1.77] dermatological items per prescription, with 49% of patients receiving only one. The most common dermatological items prescribed were corticosteroids (25.5%), antibiotics (12.4%), and fungicides (11.6%). Statistical significant association were found between the type of dermatological disorder and/ gender ( $p = 0.004$ ), age group ( $p < 0.0001$ ) and HIV-status ( $p = 0.0002$ ). 51 patients (16.9%) were HIV-positive and experienced mostly problems with urticaria, melasma, and warts

**Conclusions** The results reveal the prevalence of dermatological disorders and the treatment thereof in the private health system of Namibia. It will make health professionals in Namibia more aware of the significance of dermatological diseases in their region.

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