A Survey on Drug's Side Effects Due to Using Skin Drugs Without Prescription in Lower Class People in Bangladesh

A Dissertation submitted to the Department of Pharmacy, East West University, Bangladesh, in partial fulfillment of the requirements for the Degree of Bachelor of Pharmacy

Submitted by

Md Yusuf Madbar

ID: 2013-3-70-064

Department of Pharmacy

East West University

Declaration by the Research Candidate

I, Md. Yusuf Madbar (ID # 2013 – 3 – 70 – 064) , hereby declare that the Research entitled "**Drug's side effects due to using skin drugs without prescription in lower class people in Bangladesh**" , submitted by me to the Department of Pharmacy, East West University, Aftabnagar, Dhaka, Bangladesh in the partial fulfillment of the requirement for the award of the degree of Bachelor of Pharmacy is a bonafide record of original Project work carried out by me during 2013 under the supervision and guidance of Mrs. Nazia Hoque, Assistant Professor, Department of Pharmacy, East West University, Aftabnagar, Dhaka, Bangladesh and it has not formed the basis for the award of any other Degree/Diploma/Fellowship or other similar title to any candidate of any University.

Md. Yusuf Madbar
ID: 2013-3-70-064

Certificate by the Supervisor

This is to certify that the Research entitled "**Drug's side effects due to using skin drugs without prescription in lower class people in Bangladesh**" submitted to the Department of Pharmacy, East West University, Aftabnagar, Dhaka in partial fulfillment of the requirements of the Degree of Bachelor of Pharmacy was carried out by Md. Yusuf Madbar (ID: 2013-3-70-064) under our guidance and supervision and that no part of the project has been submitted for any other degree. We further certify that all the sources of information and facilities availed of in this connection duly acknowledged.

Ms. Nazia Hoque

Assistant professor

Department of Pharmacy

East West University

Certificate by the Chairperson

This is to certify that the Research entitled "**Drug's side effects due to using GIT drugs without prescription in lower class people in Bangladesh**" submitted to the Department of Pharmacy, East West University, Aftabnagar, Dhaka in partial fulfillment of the requirements of the Degree of Bachelor of Pharmacy was carried out by Md. Yusuf Madbar (ID: 2013-3-70-064). We further indorse that all the sources of information and facilities availed of in this connection duly acknowledged.

_____

Dr. Chowdhury Faiz Hossain
Chairperson and Professor
Department of Pharmacy
East West University

Acknowledgement

At first, I would like to thanks the almighty “ALLAH” the most gracious and merciful for enabling me to successfully completing my research work soundly and orderly.

I wish to express my thanks and gratitude towards my supervisor **Mst. Marium Begum,** Senior Lecturer, Department of Pharmacy, East West University, for her constant encouragement, helpful discussions and able guidance throughout this study. Her generosity with time and constructive comments were of vital support and were above the call of duty.

I am indebted to **Dr. Chowdhury Faiz Hossain**, Chairperson and Professor, East West University, for his kind support and the permission to use the facilities available at the Department of Pharmacy, East West University, as well as for his encouragement to carry out research.

I am indebted to **Dr. Shamsun Nahar Khan**, Associate Professor, East West University, for his kind support and the permission to use the facilities available at the Department of Pharmacy, East West University, as well as for his encouragement to carry out research.

I would also like to thank first my dear friends **Shamim Samad, Salman, Yusuf, Noor Islam** and my dear sisters **Sarah, Faraha** lastly and up most importantly my dear husband **Md. Joynal** who helped me instantaneously when I needed the most while working on this research paper.

And at the end, I would like to thank my family for their endless inspiration, support and care during my research work.

Dedication

This Research Paper is dedicated to my beloved Parents and my friends.

Table of contents

List of Figures

Abstract

Multiple environmental exposures may derange the regulatory and repair mechanisms of the skin and lead to dermatological disease. Skin problem is one of the major diseases for our country. But there has no actual document or data about this. In this study only lower class people of Bangladesh are included and seen the past history of skin patient. Most of them (94.33%) are bought medicine without prescription and taking those without any consultation. Resulting, it shocked because they don't know, every medicine is poisons if it doesn't use properly. The skin is one of the largest immunologic organs and is affected by both external and internal factors, as well as innate and adaptive immune responses. This analytic cross-sectional study was conducted on a total of 300 patients in which 69% were male and 31% female. On the study it was seen that the highest percent of respondents were suffering from itching (51%) and allergy (44.33%) at ring worm disease, blurred vision (69%) and dry mouth (43%) at eczema diseases, muscle weakness (71%) and urinary difficulties (53%) at scabies disease which is alarming. The majority portion of the respondents was facing different types of side effects when they took a certain drug's due to lack of knowledge. Using a drug without prescription was increasing the chances of misusing a drug which result ultimately prompted the side effect, drug resistant and even increase the drug -drug interaction. So, need more awareness program to abolish this condition by the healthcare professional.

Keywords: skin diseases, side effect of drug, ringworm, eczema, scabies, Allergy.

CHAPTER ONE

INTRODUCTION

1.1 Skin Disease:

Skin diseases are most common form of infections occurring in people of all ages. Skin disorders due to its ugliness and associated hardships are one of the hardest ailments to get accustomed to especially when it is located in a place that is difficult to conceal like the face, even with makeup. Most of the skin infections treatment takes long time to show their effects. The problem becomes more worrisome if the ailment does not respond to skin disorder treatments. There are not many statistics to prove the exact frequency of skin diseases in this country, but general impression is 10-20 percent of patients seeking medical advice suffer from skin diseases. The skin conditions are prevalent across all parts of the world. Sun is one of the most prominent sources of skin cancer and related traumas. Diseases of the skin account for a great deal of misery, suffering, incapacity and economic loss. Besides this, they are a great handicap in the society, because they are visible. Fortunately, however, due to recent advances, cutaenous scars can be successfully removed by plastic planning, laser therapy and skin grafting. There are main types of Skin disease: Skin infection, Bacterial infection, Skin tumors, fungal infection and Virus infection. (MedicineNet, 2017).

1.2 Skin Physiology:

The skin is the largest organ of the body, accounting for about 15% of the total adult body weight. It performs many vital functions, including protection against external physical, chemical, and biologic assailants, as well as prevention of excess water loss from the body and a role in thermoregulation. The skin is continuous, with the mucous membranes lining the body's surface (Kanitakis, 2002).

The integumentary system is formed by the skin and its derivative structures. The skin is composed of three layers: the epidermis, the dermis, and subcutaneous tissue. The outer most level, the epidermis, consists of a specific constellation of cells known as keratinocytes, which function to synthesize keratin, a long, threadlike protein with a protective role. The middle layer, the dermis, is fundamentally made up of the fibrillar structural protein known as collagen. The dermis lies on the subcutaneous tissue, or panniculus, which contains small lobes of fat cells known as lipocytes. The thickness of these layers varies considerably, depending on the geographic location on the anatomy of the body. The eyelid, for example, has the thinnest layer of the epidermis, measuring less than 0.1 mm, whereas the palms and soles of the feet have the thickest epidermal

layer, measuring approximately 1.5 mm. The dermis is thickest on the back, where it is 30-40 times as thick as the overlying epidermis (james et al.,1997)

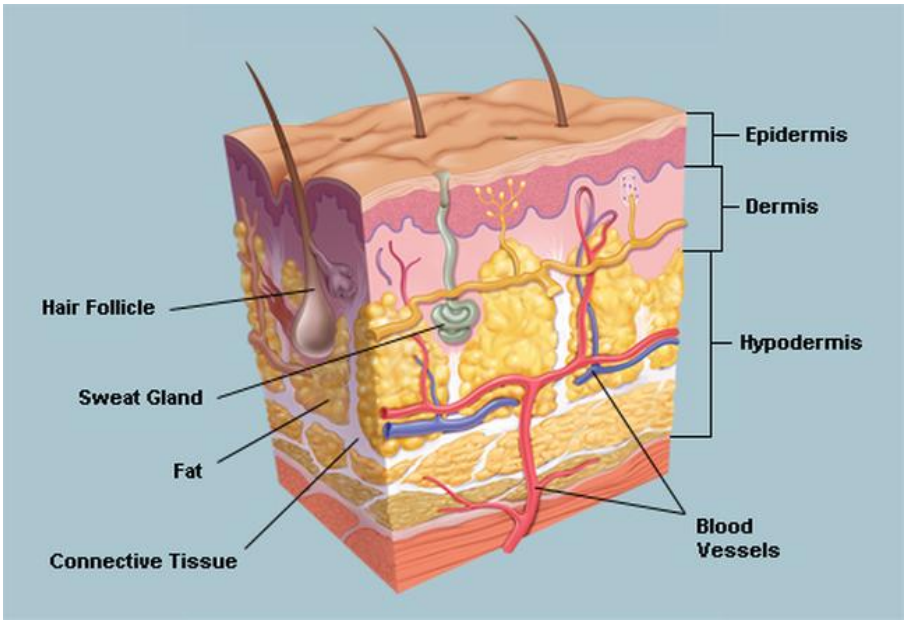


Figure 1.1 Skin Layer

Here epidermis is a stratified, squamous epithelium layer that is composed primarily of two types of cells: keratinocytes and dendritic cells. The keratinocytes differ from the "clear" dendritic cells by possessing intercellular bridges and ample amounts of stainable cytoplasm. The epidermis harbors a number of other cell populations, such as melanocytes, Langerhans cells, and Merkel cells, but the keratinocyte cell type comprises the majority of the cells by far. The epidermis commonly is divided into four layers according to keratinocyte morphology and position as they differentiate into horny cells, including the basal cell layer (stratum germinativum), the squamous cell layer (stratum spinosum), the granular cell layer (stratum granulosum), and the cornified or horny cell layer (stratum corneum).The lower three layers that constitute the living, nucleated cells of the epidermis are sometimes referred to as the stratum malpighii and rete malpighii (Murphy,1997).

The epidermis is a continually renewing layer and gives rise to derivative structures, such as pilosebaceous apparatuses, nails, and sweat glands. The basal cells of the epidermis undergo proliferation cycles that provide for the renewal of the outer epidermis. The epidermis is a dynamic tissue in which cells are constantly in unsynchronized motion, as differing individual cell populations pass not only one .Beautiful, healthy skin is

determined by the healthy structure and proper function of components within the skin. To maintain beautiful skin, and slow the rate at which it ages, the structures and functions of the skin must be supplemented and protected. In order to know how to supplement and protect the skin, it's important to know more about the skin's basic anatomy and composition.

There are three major components of the skin. First is the hypodermis, which is subcutaneous (just beneath the skin) fat that functions as insulation and padding for the body. Next is the dermis, which provides structure and support. Last is the epidermis, which functions as a protective shield for the body (Chu, 2008).

1.3 Keratinocyte:

At least 80% of cells in the epidermis are the ectodermally derived keratinocytes. The differentiation process that occurs as the cells migrate from the basal layer to the surface of the skin results in keratinization, a process in which the keratinocyte first passes through a synthetic and then a degradative phase. In the synthetic phase, the cell builds up a cytoplasmic supply of keratin, a fibrous intermediate filament arranged in an alpha-helical coil pattern that serves as part of the cell's cytoskeleton. Bundles of these keratin filaments converge on and terminate at the plasma membrane forming the intercellular attachment plates known as desmosomes. During the degradative phase of keratinization, cellular organelles are lost, the contents of the cell are consolidated into a mixture of filaments and amorphous cell envelopes, and the cell finally is known as a horny cell or corneocyte. The process of maturation resulting in cell death is known as terminal differentiation (James, Berger & Elston, 2006).

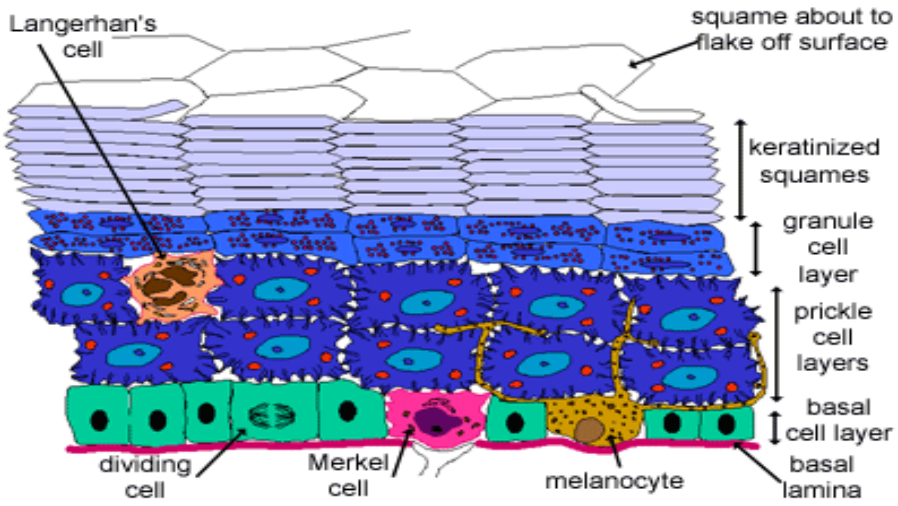


Figure 1.2 Layer of Epidermis

1.3.1 Basal Layer:

The basal layer, also known as the stratum germinatiaum, contains column-shaped keratinocytes that attach to the basement membrane zone with their long axis perpendicular to the dermis. These basal cells form a single layer and adhere to one another as well as to more superficial squamous cells through desmosomal junctions Other distinguishing features of the basal cells are their dark-staining oval or elongated nuclei and the presence of melanin pigment transferred from adjoining melanocytes (Murphy, 1997).

The basal layer is the primary location of mitotically active cells in the epidermis that give rise to cells of the outer epidermal layers. However, not all basal cells have the potential to divide. Epidermal stem cells in the basal layer are clonogenic cells with a long lifespan that progress through the cell cycle very slowly under normal conditions. Hyperplasiogenic conditions, such as wounding, can increase the number of cycling cells in the epidermis by stimulating division of stem cells. DNA damage caused by carcinogenic agents may mutate cell proliferation machinery and can also affect the rate of cellular division. Migration of a basal cell from the basal layer to the cornified layer in humans takes at least 14 days, and the transit through the cornified layer to the outermost epidermis requires another 14 days (Jones, 1996; Lavker & Sun, 1982)

Overlying the basal cell layer is a layer of the epidermis that is 5-10 cells thick and known as the squamous cell layer or stratum spinosum The squamous layer is composed of a variety of cells that differ in shape, structure, and subcellular properties depending on their location. Suprabasal spinous cells, for example, are polyhedral in shape and have a rounded nucleus, whereas cells of the upper spinous layers are generally larger in size, become flatter as they are pushed toward the surface of the skin, and contain lamellar granules Lamellar granules are membrane-bound organelles containing glycoproteins, glycolipids, phospholipids, free sterols, and a number of acid hydrolases, including lipases, proteases, acid phosphatases, and glycosidases. The lack of hydrolytic enzymes indicates that the lamellar granules are a type of lysosome. Although the lamellar granules primarily are active in cells at the interface between the granular and cornified layers, they also function in cells of the upper spinous layer to deliver precursors of stratum corneum lipids into the intercellular space (Haake & Hollbrook, 1999).

Intercellular spaces between spinous cells are bridged by abundant desmosomes that promote mechanical coupling between cells of the epidermis and provide resistance to physical stresses. Organized concentrically around the nucleus, keratin filaments in the cytoplasm are bound to desmosomal plaques at one end and remain free at the end closer to the nucleus The desmosomal plaques are composed of six polypeptides found on the cytoplasmic side of the cell membrane that are importan). The spine-like appearance of the numerous desmosomes along cell margins is where the stratum spinosum derives its name.

Gap junctions are another type of connection between epidermal cells. Essentially forming an intercellular pore, these junctions allow for physiologic communication via chemical signals that is vital in the regulation of cell metabolism, growth, and differentiation (Caputo & Peluchetti, 1977).

1.3.2 Granular Layer:

In proportion to that of the overlying horny cell layer, For example, under thin cornified layer areas, the granular The most superficial layer of the epidermis containing living cells, the granular layer or stratum granulosum, is composed of flattened cells holding abundant keratohyaline granules in their cytoplasm. These cells are responsible for further synthesis and modification of proteins involved in keratinization The granular layer varies in thickness layer may be only 1-3 cell layers in thickness, whereas under the palms of the

hands and soles of the feet the granular layer may be 10 times this thickness. A very thin or absent granular layer can lead to extensive parakeratosis in which the nuclei of keratinocytes persist as the cells move into the stratum corneum, resulting in psoriasis.

The keratohyaline granules are deeply basophilic and irregular in shape and size, and they are necessary in the formation of both the interfibrillary matrix that holds keratin filaments together and the inner lining of the horny cells. Enzymatic action of the keratohyaline granules results in the production of "soft" keratin in the epidermis by providing periodic cutting of keratin filaments. In contrast, the hair and nails do not contain keratohyaline granules, and the tonofibril filaments traversing the cell cytoplasm will harden because of the incorporation of disulfide bonds, producing "hard" keratin in those structures.

Lysosomal enzymes present only in small amounts in the stratum basalis and stratum spinosum are found at high levels in the stratum granulosum because the granular layer is a keratogenous zone of the epidermis. Here, the dissolution of cellular organelles is prepared as the cells of the granular layer undergo the abrupt terminal differentiation process to a horny cell of the cornified layer (Matoltsy, 1976; Schwarz, 1979).

1.3.3 Cornified Layer:

Horny cells (corneocytes) of the cornified layer provide mechanical protection to the underlying epidermis and a barrier to prevent water loss and invasion by foreign substancesThe corneocytes, which are rich in protein and low in lipid content, are surrounded by a continuous extracellular lipid matrix The large, flat, polyhedral-shaped horny cells have lost their nuclei during terminal differentiation and technically are considered to be dead The physical and biochemical properties of cells in the cornified layer vary in accordance with position in order to promote desquamation moving outward. For instance, cells in the middle have a much higher capacity for water-binding than the deeper layers because of the high concentration of free amino acids found in the cytoplasm of middle layer cells. The deep cells also are more densely compact and display a greater array of intercellular attachments than the more superficial layers. Desmosomes undergo proteolytic degradation as the cells progress outward, contributing to the shedding of corneocytes during desquamation (Haake & Hollbrook, 1999).

1.4.3 The Regulation of Epidermal Proliferation and differentiation:

As a perpetually regenerating tissue, the epidermis must maintain a relatively constant number of cells as well as regulate the interactions and junctions between epidermal cells. Adhesions between keratinocytes, the interactions of keratinocytes and immigrant cells, the adhesion between the basal lamina and the underlying dermis, and the process of terminal differentiation to produce corneocytes must be regulated as cells relocate during development as well as throughout life. Epidermal morphogenesis and differentiation is regulated in part by the underlying dermis, which also plays a critical role in the maintenance of postnatal structure and function. The epidermal-dermal interface is also a key site in the development of epidermal appendages.

The maintenance of a constant epidermal thickness depends also on intrinsic properties of epidermal cells, such as the ability to undergo apoptosis, programmed cell death. Apoptosis follows an orderly pattern of morphologic and biochemical changes resulting in cell death without injury to neighboring cells, as is often the case in necrosis. This major homeostatic mechanism is regulated by a number of cellular signaling molecules including hormones, growth factors, and cytokines. In the skin, apoptosis is important in developmental remodeling, regulation of cell numbers, and defense against mutated, virus-infected, or otherwise damaged cells. Terminal differentiation is a type of apoptosis evolved to convert the keratinocyte into the protective corneocyte The disruption of dynamic equilibrium maintaining constant epidermal thickness can result in conditions such as psoriasis, whereas the dysregulation of apoptosis is often seen in tumors of the skin (Kerr, Wyllie, & Currie, 1972).

1.4 Non Keratinocyte Cell of Epidermis:

1.4.1 Melanocyte:

The melanocyte is a dendritic, pigment-synthesizing cell derived from the neural crest and confined in the skin pre-dominantly to the basal layer .Branching into more superficial layers, extensions of the melanocyte come into contact with keratinocytes but do not form cellular junctions. Melanocytes are responsible for the production of the pigment melanin and its transfer to keratinocytes. Melanin is produced in a rounded, membrane-bound organelle known as the melanosome via a series of receptor-mediated, hormone-stimulated, enzyme-catalyzed reactions.

Melanosomes are moved to the end of the melanocyte processes that lie closest to the skin surface and are transferred to keratinocytes). In white skin, these melanosomes are aggregated into membrane-bound melanosome complexes containing two or three melanosomes, whereas melanosomes tend to be removed from these complexes more rapidly in keratinocytes of individuals with dark skin. Heavily pigmented skin can be attributed to the greater production of melanosomes in melanocytes, the higher degree of melanization in each melanosome, the larger size of melanosomes, the greater amount of dispersion of melanosomes in keratinocytes, and the slower rate of melanosome degradation in comparison to fair skin (Haake & Hollbrook, 1999)

1.4.2 Merkel Cells:

Merkel cells are oval-shaped, slow-adapting, type I mechanoreceptors located in sites of high tactile sensitivity that are attached to basal keratinocytes by desmosomal junctions. Merkel cells are found in the digits, lips, regions of the oral cavity, and outer root sheath of the hair follicle and are sometimes assembled into specialized structures known as tactile discs or touch domes.Relatively small deformations of adjoining keratinocytes are stimulus enough to cause Merkel cells to secrete a chemical signal that generates an action potential in the adjoining afferent neuron, which relays the signal to the brain. The high concentration of Merkel cells in certain regions such as the fingertips results in smaller and more densely packed receptive fields and thus higher tactile resolution and sensitivity (Moll, 1994).

1.4.3 Langerhans Cells:

Langerhans cells are involved in a variety of T-cell responses. Derived from the bone marrow, these cells migrate to a suprabasal position in the epidermis early in embryonic development and continue to circulate and repopulate the epidermis throughout life. The cells are dendritic and do not form cellular junctions with neighboring cells. Langerhans cells constitute 2%-8% of the total epidermal cell population and maintain nearly constant numbers and distributions in a particular area of the body. In the epidermis, the cells mainly are distributed among the squamous and granular layers with fewer cells in the basal layer. They are found in other squamous epithelia in addition to the epidermis, including the oral cavity, esophagus, and vagina, as well as in lymphoid organs and in the normal dermis

Langerhans cells must recognize and process soluble antigens found in epidermal tissue. When a membrane-bound antigen is ingested via endocytosis, cell granules are formed. The contents of these granules are delivered to phagolysosomes in the cytoplasm containing hydrolytic enzymes similar to those found in macrophages. In the first stage of life, the Langerhans cells are weak stimulators of unprimed T cells but are able to ingest and process antigens. Later, once the cell has become an effective activator of naïve T cells, activation via contact with the antigen will not trigger phagocytosis but rather will stimulate cell migration (Udey, 1997).

1.4.4 Eccrine Sweat Glands:

Eccrine sweat glands are involved in the regulation of heat and are most abundant on the soles of the feet and least plentiful on the back .The sweat glands originate as a band of epithelial cells growing downward from the epidermal ridge. This tubular, or ductal, structure is modified during development to generate the three composite parts

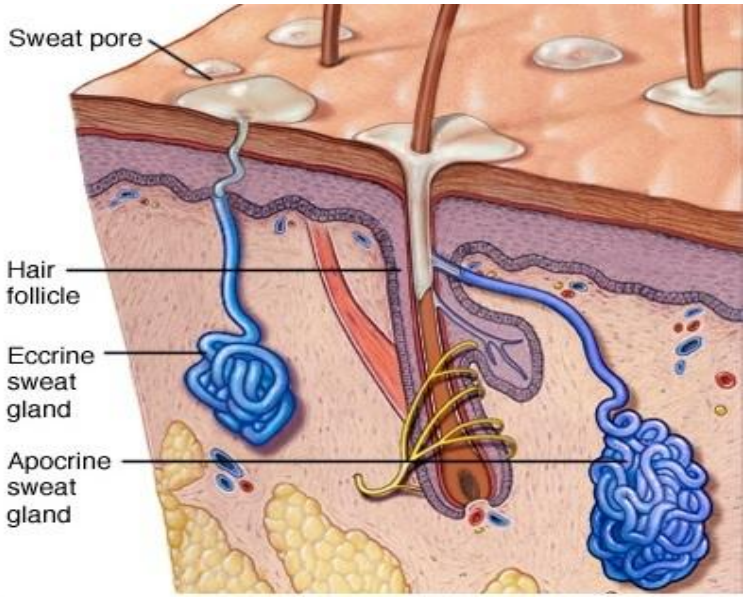


Figure 1.3 Eccrine Sweat Glands

of the eccrine sweat unit, which are the intraepidermal spiral duct, the straight dermal portion, and the coiled secretory duct .The spiral duct opens onto the skin surface and is composed of dermal duct cells that have migrated upward. Cells undergo cornification within the duct, and the corneocytes produced ultimately will become part of the cornified layer. The straight dermal segment connects the superficial spiral duct to the inner secretory portion of the gland.

The secretory coil of the eccrine unit lies deep in the dermis or within the superficial panniculus and is composed of glycogen-rich clear secretory cells, dark mucoidal cells, and myoepithelial cells specialized in contractile properties Clear cells rest either on the basement membrane or on the myo epithelial cells and form intercellular canaliculi where two clear cells adjoin. The canaliculi open directly into the lumen of the gland (Mauro & Goldsmith). Large, glycogen-rich inner epithelial cells initiate the formation of sweat in response to a thermal stimulus. Initially an isotonic solution, the darker mucoidal cells in the secretory coil and in the dermal duct actively reabsorb sodium from sweat in the duct, thereby resulting in the extremely hypotonic solution that is emitted onto skin surface through the intra epidermal spiral duct. This response promotes cooling while conserving sodium (James, Berger & Elston, 2006).

1.4.5 Apocrine Sweat Glands:

Whereas eccrine glands are primarily involved in thermal regulation, apocrine glands are involved in scent release .Apocrine sweat glands in humans are confined mainly to the regions of the axillae and perineum, and unlike eccrine and apoeccrine glands, they do not open directly to the skin surface. Instead, the intraepithelial duct opens into sebaceous follicles, entering in the infundibulum above the sebaceous duct. The basal secretory coil of apocrine glands, which is normally located entirely in subcutaneous fat, differs from that of eccrine glands in that it is composed exclusively of secretory cells; no ductal cells are presents.

Apocrine sweat glands develop their secretory portions and become active just before puberty, a response induced presumably by hormonal signals. The proteinaceous, viscous secretion has distinct odor and can function as a territorial marker, warning signal, and sexual attractant, but its sexual functions may now be vestigial in humans. Difficulties in acquiring pure samples of apocrine sweat have made it impossible to determine the exact chemical composition of the secretion (Mauro & Goldsmith, 2008)

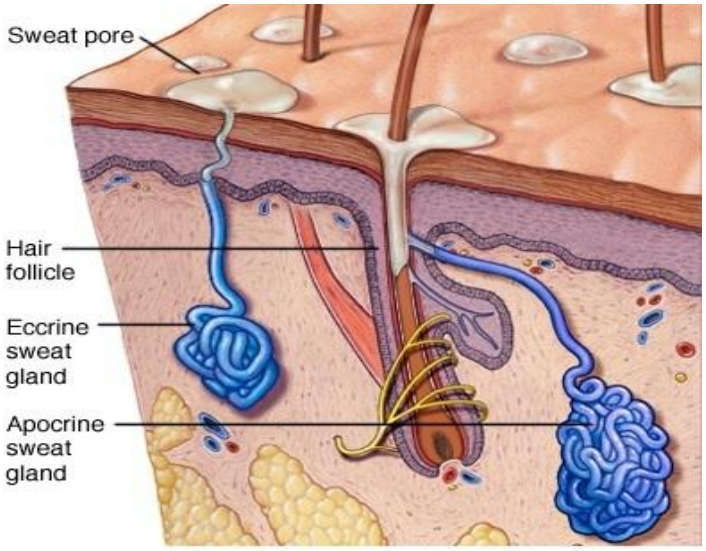


Figure 1.4 Apocrine Sweat Glands

The apoeccrine sweat gland (AEG) develops during puberty from eccrine-like precursors, opening directly unto the skin. Discovered during the isolation of human axillary sweat from patients with axillary hyperhidrosis, a condition characterized by abnormally increased rates of perspiration, the AEG is found in the adult axillae; its relative frequency varies from person to person. Like eccrine glands, the AEG opens directly to the skin surface. The AEG has a secretory rate as much as 10 times that of the eccrine gland and is therefore thought to contribute to axillary hyperhidrosis (Mauro & Goldsmith, 2008).

1.5 Hypodermis:

The hypodermis also called the subcutaneous layer is a layer directly below the dermis and serves to connect the skin to the underlying fascia (fibrous tissue) of the bones and muscles. It is not strictly a part of the skin, although the border between the hypodermis and dermis can be difficult to distinguish. The hypodermis consists of well-vascularized, loose, areolar connective tissue and adipose tissue, which functions as a mode of fat storage and provides insulation and cushioning for the integument.

Lipid Storage The hypodermis is home to most of the fat that concerns people when they are trying to keep their weight under control. Adipose tissue present in the hypodermis consists of fat-storing cells called adipocytes. This stored fat can serve as an energy reserve, insulate the body to prevent heat loss, and act as a cushion to protect underlying structures from trauma.

Where the fat is deposited and accumulates within the hypodermis depends on hormones (testosterone, estrogen, insulin, glucagon, leptin, and others), as well as genetic factors. Fat distribution changes as our bodies mature and age. Men tend to accumulate fat in different areas (neck, arms, lower back, and abdomen) than do women (breasts, hips, thighs, and buttocks). The body mass index (BMI) is often used as a measure of fat, although this measure is, in fact, derived from a mathematical formula that compares body weight (mass) to height. Therefore, its accuracy as a health indicator can be called into question in individuals who are extremely physically fit.

In many animals, there is a pattern of storing excess calories as fat to be used in times when food is not readily available. In much of the developed world, insufficient exercise coupled with the ready availability and consumption of high-calorie foods have resulted in unwanted accumulations of adipose tissue in many people. Although periodic accumulation of excess fat may have provided an evolutionary advantage to our ancestors, who experienced unpredictable bouts of famine, it is now becoming chronic and considered a major health threat. Recent studies indicate that a distressing percentage of our population is overweight and/or clinically obese. Not only is this a problem for the individuals affected, but it also has a severe impact on our healthcare system. Changes in lifestyle, specifically in diet and exercise, are the best ways to control body fat accumulation, especially when it reaches levels that increase the risk of heart disease and diabete (Aumailley & Krieg T,1996).

1.6 Pigmentation:

The color of skin is influenced by a number of pigments, including melanin, carotene, and hemoglobin. Recall that melanin is produced by cells called melanocytes, which are found scattered throughout the stratum basale of the epidermis. The melanin is transferred into the keratinocytes via a cellular vesicle called a melanosome.

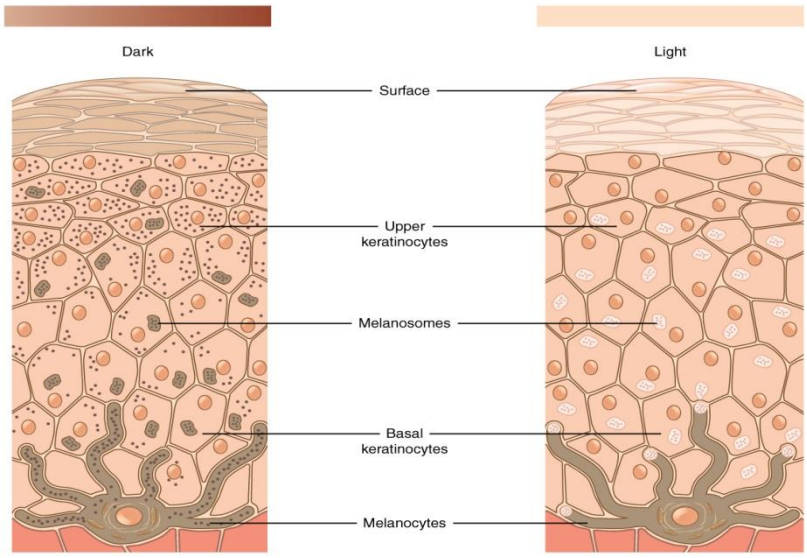


Figure 1.5 the relative coloration of the skin depends of the amount of melanin produced by melanocytes

Melanin occurs in two primary forms. Eumelanin exists as black and brown, whereas pheomelanin provides a red color. Dark-skinned individuals produce more melanin than those with pale skin. Exposure to the UV rays of the sun or a tanning salon causes melanin to be manufactured and built up in keratinocytes, as sun exposure stimulates keratinocytes to secrete chemicals that stimulate melanocytes. The accumulation of melanin in keratinocytes results in the darkening of the skin, or a tan. This increased melanin accumulation protects the DNA of epidermal cells from UV ray damage and the breakdown of folic acid, a nutrient necessary for our health and well-being. In contrast, too much melanin can interfere with the production of vitamin D, an important nutrient involved in calcium absorption. Thus, the amount of melanin present in our skin is dependent on a balance between available sunlight and folic acid destruction, and protection from UV radiation and vitamin D production.

It requires about 10 days after initial sun exposure for melanin synthesis to peak, which is why pale-skinned individuals tend to suffer sunburns of the epidermis initially. Dark-skinned individuals can also get sunburns, but are more protected than are pale-skinned individuals. Melanosomes are temporary structures that are eventually destroyed by fusion with lysosomes; this fact, along with melanin-filled keratinocytes in the stratum corneum sloughing off, makes tanning impermanent.

The hypodermis is the deepest section of the skin. The hypodermis refers to the fat tissue below the dermis that insulates the body from cold temperatures and provides shock absorption. Fat cells of the hypodermis also store nutrients and energy. The hypodermis is the thickest in the buttocks, palms of the hands, and soles of the feet. As we age, the hypodermis begins to atrophy, contributing to the thinning of aging skin.

1.7 Dermis:

The dermis might be considered the "core" of the integumentary system as distinct from the epidermis ("over") and hypodermis ("below"). It contains blood and lymph vessels, nerves, and other structures, such as hair follicles and sweat glands. The dermis is made of two layers of connective tissue that compose an interconnected mesh of elastin and collagenous fibers, produced by fibroblasts.

Layers of the Dermis

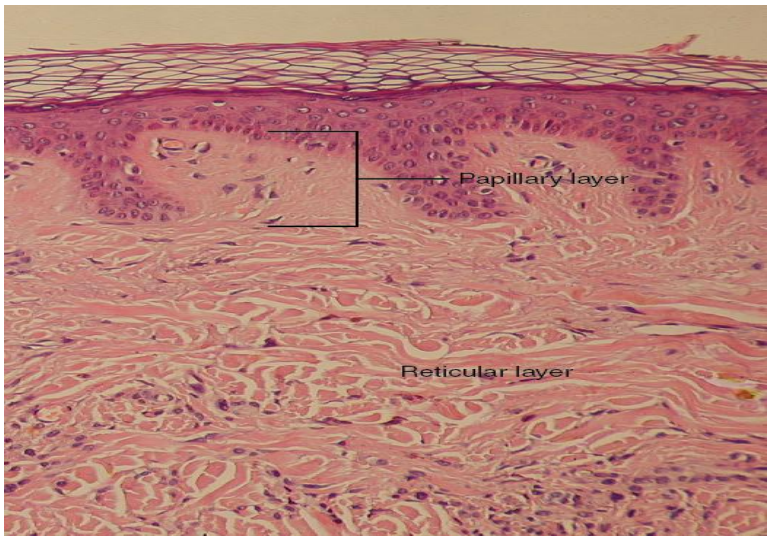


Figure 1.6 Layer of Dermis

1.7.1 Papillary Layer:

The papillary layer is made of loose, areolar connective tissue, which means the collagen and elastin fibers of this layer form a loose mesh. This superficial layer of the dermis projects into the stratum basale of the epidermis to form finger-like dermal papillae .Within the papillary layer are fibroblasts, a small number of fat cells (adipocytes), and an abundance of small blood vessels. In addition, the papillary layer contains phagocytes, defensive cells that help fight bacteria or other infections that have breached the skin.

This layer also contains lymphatic capillaries, nerve fibers, and touch receptors called the Meissner corpuscles.

1.7.2 Reticular layer:

Underlying the papillary layer is the much thicker reticular layer, composed of dense, irregular connective tissue. This layer is well vascularized and has a rich sensory and sympathetic nerve supply. The reticular layer appears reticulated due to a tight meshwork of fibers. Elastin fibers provide some elasticity to the skin, enabling movement. Collagen fibers provide structure and tensile strength, with strands of collagen extending into both the papillary layer and the hypodermis. In addition, collagen binds water to keep the skin hydrated. Collagen injections and Retin-A creams help restore skin turgor by either introducing collagen externally or stimulating blood flow and repair of the dermis, respectively.

The dermis is located between the hypodermis and the epidermis. It is a fibrous network of tissue that provides structure and resilience to the skin. While dermal thickness varies, it is on average about 2 mm thick.

The major components of the dermis work together as a network. This mesh-like network is composed of structural proteins (collagen and elastin), blood and lymph vessels, and specialized cells called mast cells and fibroblasts. These are surrounded by a gel-like substance called the ground substance, composed mostly of glycosaminoglycans. The ground substance plays a critical role in the hydration and moisture levels within the skin.

The most common structural component within the dermis is the protein collagen. It forms a mesh-like framework that gives the skin strength and flexibility. The glycosaminoglycans moisture binding molecules enable collagen fibers to retain water and provide moisture to the epidermis. Another protein found throughout the dermis is the coil-like protein, elastin, which gives the skin its ability to return to its original shape after stretching. In other words, elastin provides the skin with its elasticity. Intertwined throughout the dermis are blood vessels, lymph vessels, nerves, and mast cells. Mast cells are specialized cells that play an important role in triggering the skin's inflammatory response to invading microorganisms, allergens, and physical injury.

The blood vessels in the dermis help in thermoregulation of the body by constricting or dilating to conserve or release heat. They also aid in immune function and provide oxygen and nutrients to the lower layers of the epidermis. These blood vessels do not extend into the epidermis. Nourishment that diffuses into the epidermis only reaches the very bottom layers. The cells in the upper layers of the epidermis are dead because they do not receive oxygen and nutrients.

The junction between the dermis and epidermis is a wave-like border that provides an increased surface area for the exchange of oxygen and nutrients between the two sections. Along this junction are projections called dermal papillae. As you age, your dermal papillae tend to flatten, decreasing the flow of oxygen and nutrients to the epidermis (Sawaya, 1994).

1.8 Epidermis:

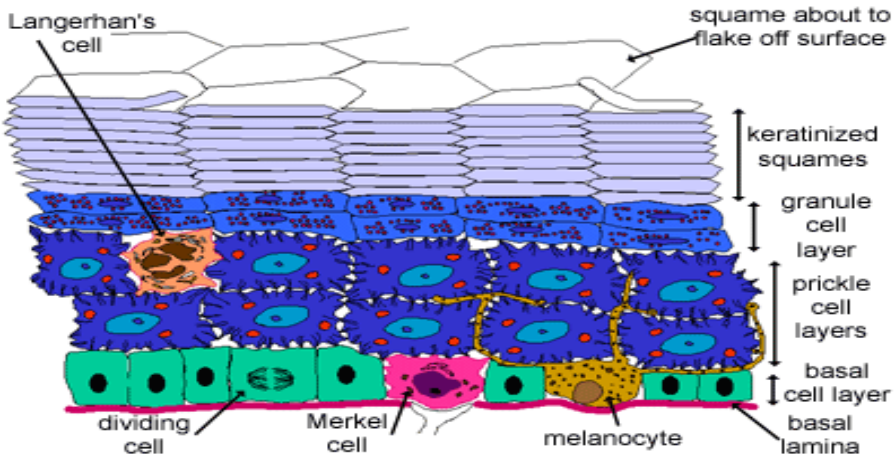


Figure 1.7 Layers of Epidermis

The epidermis is the outermost layer of the skin. Categorized into five horizontal layers, the epidermis actually consists of anywhere between 50 cell layers (in thin areas) to 100 cell layers (in thick areas). The average epidermal thickness is 0.1 millimeters, which is about the thickness of one sheet of paper. The epidermis acts as a protective shield for the body and totally renews itself approximately every 28 days.

The first layer of the epidermis is the stratum basale. This is the deepest layer of the epidermis and sits directly on top of the dermis. It is a single layer of cube-shaped cells. New epidermal skin cells, called keratinocytes, are formed in this layer through cell division to replace those shed continuously from the upper layers of the epidermis. This regenerative process is called skin cell renewal. As we age, the rate of cell renewal decreases. Melanocytes, found in the stratum basale, are responsible for the production of skin pigment, or melanin. Melanocytes transfer the melanin to nearby keratinocytes that will eventually migrate to the surface of the skin. Melanin is photoprotective: it helps protect the skin against ultraviolet radiation (sun exposure).

The second layer of the epidermis is the stratum spinosum, or the prickle-cell layer. The stratum spinosum is composed of 8-10 layers of polygonal (many sided) keratinocytes. In this layer, keratinocytes are beginning to become somewhat flattened.

The third layer is called the stratum granulosum, or the granular layer. It is composed of 3-5 layers of flattened keratin—a tough, fibrous protein that gives skin its protective properties. Cells in this layer are too far from the dermis to receive nutrients through diffusion, so they begin to die.

The fourth layer in the epidermis is called the stratum lucidum, or the clear layer. This layer is present only in the fingertips, palms, and soles of the feet. It is 3-5 layers of extremely flattened cells.

The fifth layer, or horny layer, is called the stratum corneum. This is the top, outermost layer of the epidermis and is 25-30 layers of flattened, dead keratinocytes. This layer is the real protective layer of the skin. Keratinocytes in the stratum corneum are continuously shed by friction and replaced by the cells formed in the deeper sections of the epidermis. In between the keratinocytes in the stratum corneum are epidermal lipids (ceramides, fatty acids, and lipids) that act as a cement (or mortar) between the skin cells (bricks). This combination of keratinocytes with interspersed epidermal lipids (brick and mortar) forms a waterproof moisture barrier that minimizes transepidermal water loss (TEWL) to keep moisture in the skin. This moisture barrier protects against invading microorganisms, chemical irritants, and allergens. If the integrity of the moisture barrier is compromised, the skin will become vulnerable to dryness, itching, redness, stinging, and other skin care concerns.

In the very outer layers of the stratum corneum, the moisture barrier has a slightly acidic pH (4.5 to 6.5). These slightly acidic layers of the moisture barrier are called the acid mantle. The acidity is due to a combination of secretions from the sebaceous and sweat glands. The acid mantle functions to inhibit the growth of harmful bacteria and fungi. The acidity also helps maintain the hardness of keratin proteins, keeping them tightly bound together. If the skin's surface is alkaline, keratin fibers loosen and soften, losing their protective properties. When the pH of the acid mantle is disrupted (becomes alkaline)—a side effect of common soaps—the skin becomes prone to infection, dehydration, roughness, irritation, and noticeable flaking.

A number of components are common to both the dermis and epidermis. These are: pores, hair, sebaceous glands, and sweat glands.

Pores are formed by a folding-in of the epidermis into the dermis. The skin cells that line the pore (keratinocytes) are continuously shed, just like the cells of the epidermis at the top of the skin. The keratinocytes being shed from the lining of the pore can mix with sebum and clog the pore. This is the precursor to acne. If oil builds up inside pores, or if tissue surrounding the pore becomes agitated, pores may appear larger.Hair grows out of the pores and is composed of dead cells filled with keratin proteins. At the base of each hair is a bulb-like follicle that divides to produce new cells. The follicle is nourished by tiny blood vessels and glands. Hair prevents heat loss and helps protect the epidermis from minor abrasions and exposure to the sun's rays.

Sebaceous glands are usually connected to hair follicles and secrete sebum to help lubricate the follicle as it grows. Sebum also contributes to the lipids and fatty acids within the moisture barrier. Oil production within the sebaceous gland is regulated by androgen levels (hormones such as testosterone).

Sweat glands are long, coiled, hollow tubes of cells. The coiled section is where sweat is produced, and the long portion is a duct that connects the gland to the pore opening on the skin's surface. Perspiration excreted by the sweat glands helps cool the body, hydrate the skin, eliminate some toxins (Caputo & Peluchetti, 1977).

1.9 Scabies:

Human scabies is an intensely pruritic skin infestation caused by the host-specific mite Sarcoptes scabiei hominis. Approximately 300 million cases of scabies (see the image below) are reported worldwide each year.

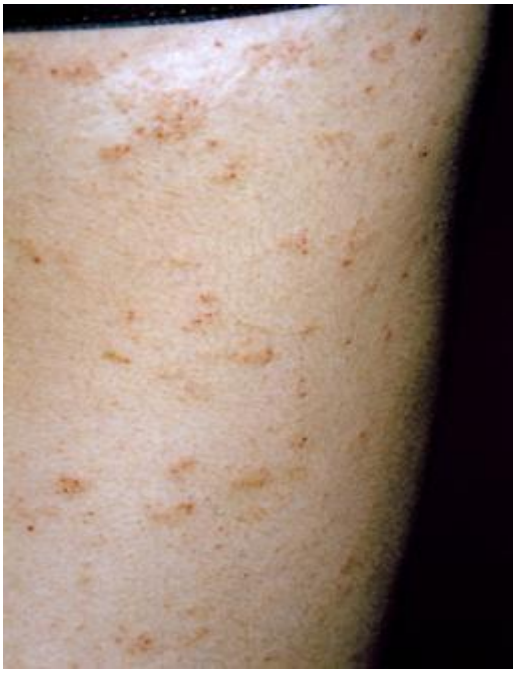


Figure 1.8 Scabies. Erythematous vesicles and papules are present on torso extremities.

1.9.1 Signs and symptoms:

Burrows are a pathognomonic sign and represent the intraepidermal tunnel created by the moving female mite. They appear as serpiginous, grayish, threadlike elevations in the superficial epidermis, ranging from 2-10 mm long. High-yield locations for burrows include the following:

- Webbed spaces of the fingers
- Flexor surfaces of the wrists
- Elbows
- Axillae
- Belt line
- Feet

- Scrotum (men)
- Areolae (women)

In geriatric patients, scabies demonstrates a propensity for the back, often appearing as excoriations. In infants and small children, burrows are commonly located on the palms and soles. One- to 3-mm erythematous papules and vesicles are seen in typical distributions in adults. The vesicles are discrete lesions filled with clear fluid, although the fluid may appear cloudy if the vesicle is more than a few days old.

Nodular scabies

Nodules occur in 7-10% of patients with scabies, particularly young children. In neonates unable to scratch, pinkish brown nodules ranging in size from 2-20 mm in diameter may develop.

Crusted scabies

In crusted scabies, lesions are often hyperkeratotic and crusted and cover large areas. Marked scaling is common, and pruritus may be minimal or absent. Nail dystrophy and scalp lesions may be prominent. The hands and arms are the usual locations for lesions, but all sites are vulnerable.

Secondary lesions

These lesions result from scratching, secondary infection, and/or the host's immune response against the scabies mites and their products. Characteristic findings include the following:

- Excoriations
- Widespread eczematous dermatitis
- Honey-colored crusting
- Postinflammatory hyperpigmentation
- Erythroderma
- Prurigo nodules

- Frank pyoderma

1.9.2 Diagnosis:

The diagnosis of scabies can often be made clinically in patients with a pruritic rash and characteristic linear burrows. The diagnosis is confirmed by light microscopic identification of mites, larvae, ova, or scybala (feces) in skin scrapings.

In rare cases, mites are identified in biopsy specimens obtained to rule out other dermatoses. Characteristic histopathology in the absence of actual mites also may suggest the diagnosis of scabies.

Clinically inapparent infection can be detected by amplification of Sarcoptes DNA in epidermal scale by polymerase chain reaction (PCR) assay.In addition, elevated immunoglobulin E (IgE) titers and eosinophilia may be demonstrated in some patients with scabies.

1.9.3 Management:

Scabies treatment includes administration of a scabicidal agent (eg, permethrin, lindane, or ivermectin), as well as an appropriate antimicrobial agent if a secondary infection has developed.

Pruritus may be partially alleviated with an oral antihistamine, such as hydroxyzine hydrochloride (Atarax), diphenhydramine hydrochloride (Benadryl), or cyproheptadine hydrochloride (Periactin). In rare cases, prednisone may be used to treat severe pruritus.

Because of their heavy mite burden, patients with crusted scabies may require repeated applications of topical scabicides or treatment that simultaneously uses oral ivermectin and a topical agent, such as permethrin (Emedicine.medscape.com. 2017).

1.10 Prednisolone

Prednisolone is a steroid. It prevents the release of substances in the body that cause inflammation.

Prednisolone is used to treat many different conditions such as allergic disorders, skin conditions, ulcerative colitis, arthritis, lupus, psoriasis, or breathing disorders.

Prednisolone provides relief for inflamed areas of the body. It is used to treat a number of different conditions, such as inflammation (swelling), severe allergies, adrenal problems, arthritis, asthma (Drugs . com, 2017).

1.10.1 Precaution:

One should not use this medication if he/she is allergic to prednisolone, or if have a fungal infection anywhere in the body.

Steroid medication can weaken immune system, making it easier for ones to get an infection. Steroids can also worsen an infection already have, or reactivate an infection recently had. Before taking this medication, tell doctor about any illness or infection have had within the past several weeks.

To make sure prednisolone is safe for patient, tell doctor about other medical conditions, especially:

- liver disease (such as cirrhosis);
- kidney disease;
- a thyroid disorder;
- diabetes;
- a history of malaria;
- tuberculosis;
- osteoporosis;
- glaucoma or cataracts;
- herpes infection of the eyes;
- stomach ulcers, ulcerative colitis, or diverticulitis;
- depression or mental illness;
- congestive heart failure; or
- high blood pressure

FDA pregnancy category C. It is not known whether prednisolone will harm an unborn baby. Prednisolone can pass into breast milk and may harm a nursing baby. Do not use this medication without telling permission of doctor if patient are breast-feeding a baby.

1.10.2 Prednisolone side effects

Get emergency medical help if patient have any of these **signs of an allergic reaction**: hives; difficult breathing; swelling of face, lips, tongue, or throat.

Side effect such as:

- problems with your vision;
- swelling, rapid weight gain, feeling short of breath;
- severe depression, unusual thoughts or behavior, seizure (convulsions);
- bloody or tarry stools, coughing up blood;
- pancreatitis (severe pain in your upper stomach spreading to your back, nausea and vomiting, fast heart rate);
- low potassium

Less serious side effects may include:

- sleep problems (insomnia), mood changes;
- acne, dry skin, thinning skin, bruising or discoloration;
- slow wound healing;
- increased sweating;
- headache, dizziness, spinning sensation;
- nausea, stomach pain, bloating;

1.11 Eczema

Eczema (E) (also called atopic dermatitis (AD)) is an inflammatory, chronically relapsing, non-contagious and extremely pruritic skin disease. The discussion about pathogenesis of this disease is mirrored by the different names that it has been given.

Atopy is a strikingly common finding in these patients. The Nomenclature Task Force of the European Academy of Allergology and Clinical Immunology (EAACI) proposed the term the Atopic Eczema/Dermatitis Syndrome (AEDS) for this disease, since IgE is not a prerequisite in all patients. The World Allergy

Organization 2003 Nomenclature Task force recommended that under the umbrella term dermatitis, eczema is now the agreed term to replace the transitional term atopic eczema/dermatitis syndrome (AEDS). Atopic eczema is eczema with demonstrable IgE association.

1.11.1 Epidemiology and Classification:

With a prevalence of 2-5% (in children and young adults approximately 10%), atopic eczema is one of the most commonly seen dermatoses. The "atopic diseases" E, allergic bronchial asthma and allergic rhinoconjunctivitis are familiar. A multifactorial trait, with gene loci on several chromosomes, has been proposed by different groups. The concordance of AE in monzygotic twins is 75-85%, in dizygotic twins 30%. The genetic predisposition exerts its effects in an immunological and organ-specific fashion. In addition environmental factors can work to increase or reduce the development of allergies. The first and most important step is exposure to an allergen. The long and often incomplete progression from genetic susceptibility via IgE-mediated hypersensitivity to hyperrreactivity of skin and mucosa, and finally the manifestations of an allergic disease, is modulated by environmental factors.

Modern molecular genetics has made it possible to couple certain gene loci with different clinical phenotypes. The search for an atopy gene has made it clear that E is inherited in a polygenenic fashion with many genes involved and even provided evidence for genomic imprinting, as maternal influences exceed paternal. Genomic regions linked to E show only limited overlap with asthma, but some coincide with psoriasis susceptibility regions, such a region on chromosome 1 is known as the epidermal differentiation complex, which is a cluster of genes and gene families expressed in the terminally differentiating epithelium. In 2006, a breakthrough in the genetics of E was achieved with the identification of loss-of-function mutations within the EDC gene filaggrin, which cause ichthyosis vulgaris and confer a substantial risk to develop E, especially early-onset with persistent sensitization. Filaggrin is an important protein in the formation of the epidermal barrier through binding to and aggregation of the keratin cytoskeleton. These findings

underline the importance of the skin barrier in preventing allergic responses and give rise to the concept that the primary defect in E is a failure of skin barrier function allowing abnormally enhanced cutaneous presentation of antigens, allergens and chemicals to the immune system.

In many patients with eczema, IgE-mediated allergic reactions play a pathophysiological role. However there are also patients in whom nonspecific factors such as irritants or psychosomatic influence appear to be of major importance. Careful allergy diagnosis is thus mandatory in patients with E. The clinical relevance of a given allergic sensitization should be evaluated in each individual.

1.11.2 Symptoms and Signs, Differential Diagnosis:

Often beginning with the clinical sign known as cradle cap after the first 8-12 weeks of life and the disease spreads to face and extensor sides of arms and legs of toddlers, showing extensive oozing and crusting. Later on, the typical preferential pattern with eczematous skin lesions of flexures, neck and hands develops, accompanied by dry skin, both as a subjective impression and as measurable transepidermal water loss. Lichenification is a result of scratching and rubbing, and in adults this may also result in excoriated nodules, the "prurigo form" of E. New exacerbations often start without obvious symptoms except increased itching (sometimes localized). This is followed by erythema, papules and infiltration. Acute E is histopathologically characterized by acanthosis, hyperkeratosis, parakeratosis, spongiosis, exocytosis and a sparse lymphohistiocytic infiltrate. Chronic lichenified lesions show acanthosis, hyperkeratosis, parakeratosis, dense dermal mononuclear infiltrate, increase in mast cell and capillary numbers, enlargement of capillary walls with endothelial hyperplasia, and fibrosis. However, these features are not specific for E. Accordingly, eczematous rashes of different origin are the main differential diagnosis for E.

As there is no laboratory marker specific for the disease, "stigmata" and minimal manifestations of E have been found to have diagnostic significance. The clinical diagnosis may be established by finding four of the criteria listed.

1.11.3 Causes and Pathophysiology:

E is based upon complex interactions of genetic predispositions, environmental triggers, and immune dysregulation. The clinical hallmarks of E are largely explained by two main Causes and Pathophysiology features:

1.12 Disturbed epidermal barrier:

A profoundly disturbed epidermal barrier leads to dry skin as a consequence of a high transepidermal water loss on the one hand and to enhance penetration of irritative substances and allergens into the skin on the other side. The disruption of the epidermal barrier may be caused by genetic alterations such as null mutations in the gene filaggrin that strongly predispose to development of E or ichthyosis. Filaggrin is expressed in the upper layers of the stratum corneum and is encoded within the epidermal differentiation complex (EDC). Also other genes of the EDC such as hornerin might be associated with the development of E.

Besides genetic determination, the epidermal barrier function also depends on the immune system. It has been demonstrated that Th2 cytokines such as IL-4 inhibit the expression of filaggrin and S100 proteins and thus impair the epidermal barrier. Mechanical (scratching) or physical (hot water, UV exposure, sweating) irritation further weakens the epidermal barrier.

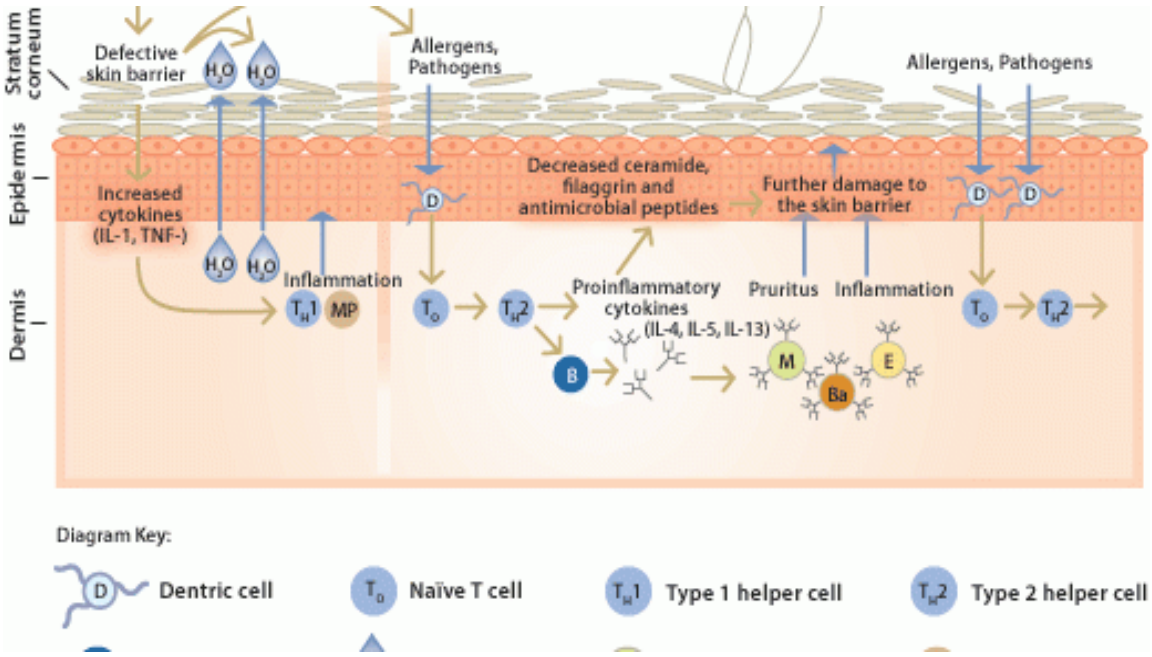


Figure 1.9 Mechanism of Action Occurring Allergy

1.12.1 Deviated immune response, allergy, and impaired innate immunity:

E is typically characterized by a Th2 dominated immune response both in skin and in circulation. This is especially true for atopic E (formerly called extrinsic atopic E), but also for other kinds of E such as non-atopic E (formerly called intrinsic atopic E), allergic contact dermatitis, and nummular or dishydrotic E. The Th2 prevalence is partially based on genetic predisposition (e.g. mutations in the IgE receptor or the Th2 inducer TSLP) and/or on the nature of the antigen causing an immune reaction. Increasing evidence suggests that for example pollen-derived low-molecular weight substances favor a Th2 immune response.

In lymphoid tissue, Th2 cells induce the production of IgE antibodies by plasma cells. Increased total and allergen specific IgE is a typical hallmark of atopic E, even if it is most likely not directly related to the development of E in most cases of adult E. In contrast, type I allergic reactions with food allergen specific IgE regularly cause E exacerbations in young children. The most common allergens are milk, egg, peanut, soy, and cereals. These allergies are typically lost during school age. In adults, a subpopulation of E patients may also react with exacerbation to some allergens, most commonly to aeroallergens such as house dust mite or pollen. The atopy patch test might help to identify those patients.

E patients with known allergy-driven exacerbation should generally avoid or reduce contact to the eliciting substances. In contrast, no evidence suggests so far that non-sensitized E patients take benefit from primary prevention (e.g. a diet during pregnancy or early childhood).

In the skin, Th2 cells induce an inflammatory reaction involving mast cells and eosinophil granulocytes. Importantly, Th2 cytokines such as IL-4, IL-5, and IL-13, inhibit the induction of an adequate innate immune response of epithelial cells. This is why E patients display lower amounts of antimicrobial peptides in the skin than for example psoriasis patients. The reduced innate immunity explains why the skin of almost all E patients is frequently colonized with Staphylococcus aureus. There is a correlation of the number of staphylocci and the severity of E, most likely due to release of exotoxins such as staphylococcus enterotoxin A/B. Also other microorganisms such as the yeast Malassezia furfur (formerly called Pityrosporum ovale or orbiculare) or the Molluscum contagiosum virus are regularly detected on E skin. Primary infections

with herpes simplex virus (E herpeticum) are often severe in atopic E patients and require hospitalization.

Besides reducing the epidermal immunity, Th2 cells also further decrease the epidermal barrier function by inhibition of genes belonging to the EDC (e.g. filaggrin).

While acute E lesions are infiltrated by a vast majority of Th2 cells, more chronic lesions are characterized by a broader immune response of mostly Th1, Th2, and Th22 cells. In line with that observation, the clinical hallmarks of acute versus chronic E are strikingly different, but the reduced epidermal barrier function with dryness of the skin and the skin colonization with extracellular microorganisms are constantly observed throughout all stages of E.

The dry skin with reduced epidermal barrier function causes a non-specific hypersensitivity of the skin towards all kinds of irritant factors. This involves substances with irritative potential (e.g. citrus fruits, rough woolen clothing, tobacco smoke) and physical factors (sweating, cold, heat, extensive washing).

There is no doubt that psychology greatly influences E, and most patients report stress results in aggravation of the disease. The field of psychoneuroimmunology is rapidly evolving and initial theories report a functional and morphological interaction of mast cells, neuropeptides, and nerve fibers. It is also known that most E patients respond less to β-adrenergic and more to α- adrenergic or cholinergic stimuli, which partially explains the white dermographism observed after mechanical provocation of the skin.

It is currently under debate whether psychiatric and/o psychosomatic diseases are associated with E. While initial studies reported an association with attention deficit hyperactivity disorder in children, the current concept assumes concentration problems in children might occur secondary as a consequence of the permanent and agonizing itch and sleep loss. Itching and social stigma are also the two main explanations why E patients suffer from a severely reduced quality of life (Worldallergy.org. 2017).

Increased IgE production, role of allergy:

IgE antibodies and positive Atopy Patch Test have been found in the majority of adult patients with AE. The inflammatory infiltrate in AE lesions mainly consists of CD4+ T lymphocytes, and a correlation with disease activity can be shown by the proportion of

activated and unactivated CD4+ cells. In the early lesions Th2 cells predominate, later in the more chronic phase Th1 cells prevail.

More than in other allergic diseases, E is characterized by increased serum IgE levels. T cells play a major role in regulation of IgE production. The Th2 subtype secreting the cytokines IL-4, IL-5 and IL-13 is most important, working via the MHC-II and T-cell receptor, and with co-stimulatory molecules to induce an isotype switch in B cells to produce IgE. While Th2 reactions are crucial for triggering reactions, in chronic skin lesions Th1 reaction patterns can be observed.

1.12.2 Cell-mediated dysfunction:

As patients with E are prone to develop a variety of infectious diseases of fungal, viral or bacterial origin like candidosis, E herpeticum (Kaposi´s varicelliform eruption), or staphylococcal impetigo, defective cellular immunity has been suspected. However, the hypothesis of a lower prevalence of T cell-mediated contact allergy in E has been questioned. Greater prevalence rates of contact allergy are found in E patients than in a normal population. Rather, these patients show a different contact allergen spectrum compared to individuals lacking the atopic constitution (metal allergy more frequent; lanolin, fragrance, etc. less frequent).

1.12.2.1 Autonomic nervous system dysregulation:

In response to different pharmacological stimuli, a substantial proportion of patients with E shows a decreased α-adrenergic and an increased β-adrenergic or cholinergic reactivity. Clinically, the white dermatographism and some psychosomatic interactions may partially be explained by this imbalance, which also gives rise to enhanced releasability of vasoactive mediators, e.g., histamine and leukotrienes, after appropriate stimulation.

1.12.2.2 Food allergy and other non-allergic food hypersensitivity:

An exacerbation of atopic E by foods in food allergic patients has been repeatedly reported, with the vast majority of cases seen in children. This should be considered in the management of E when there is a history of food allergy, or when conventional treatment measures are ineffective. Whereas the IgE-mediated reactions are the most common ones, a non-immunological hypersensitivity reaction to food additives can also worsen AE in some cases. An appropriate diagnosis of the suspected food allergy should be made.

1.12.2.3 Aeroallergens:

Some patients with atopic E suffer exacerbations of their skin lesions after contact with certain aeroallergens, e.g., house dust mite, pollen or animal dander, and improve after appropriate avoidance strategies have been applied. In certain patients, E skin lesions can be induced by epicutaneous patch testing with aeroallergens, e.g. house dust mite. The term "atopy patch test" (APT) has been proposed for this test procedure.

1.13 Skin barrier:

The clinical appearance of E inflamed lesions emerging on dry, scaling skin is suggestive of an impairment of skin barrier function. An enhanced transepidermal water loss (TEWL) and reduced skin surface water content are physical parameters that directly reflect this impaired barrier function. The barrier function is maintained by the stratum corneum which forms a continuous sheet of alternating squamae, which are protein-enriched corneocytes embedded in an intercellular matrix, consisting mainly of non-polar lipids which have developed as lamellar sheets. Even uninvolved skin of E patients is characterized by distinct differences in skin surface lipid composition, especially in the ceramide fraction.

1.13.1 Microbial colonization:

Profound changes in cutaneous flora occur in some patients with E and the pathogenic importance of microbial organisms is recognized. Among these, Malassezia furfur and Staphylococcus aureus seem to play a major role. S. Aureus is responsible for a known, very often quite dramatic complication of E, named "impetiginised E," requiring systemic antibacterial treatment, and in addition S. aureus may act as a persistent allergen stimulating IgE antibody production, or as an irritant with inflammatory potency when colonizing atopic skin. The inflammatory reaction may be caused by enterotoxin production, possibly with superantigen effects. More than 50% of S. aureus isolates cultured from patients with E have the ability to produce these enterotoxins. It is speculated that staphyloccocal superantigens, when released within the epidermis, cause a marked immune stimulation. The ability of staphylococcal enterotoxin B to elicit dermatitis after application to intact normal or intact atopic skin has been shown. It is likely that reduced innate immune responses, eg, reduced formation of antimicrobial peptides like defensins, give rise to increased microbial colonization.

1.13.2 Psychosomatic factors:

Severity of pruritus in E has been described as directly related to severity of depressive symptoms. Increased itch and sweating in lichenified skin areas, following emotional stimuli, can be recorded by psychophysiological methods. Investigations of parent-child relationships have shown different emotional responses from diseased children when compared to controls. Increased "fear scores" on personality questionnaires of patients with E have been reported by different investigators. It is uncertain whether these findings have any impact on the etiology of E, as they may also result from the prolonged process of coping with chronic disease experienced by the patient and his/her family. Stressful emotional events have been shown to precede the deterioration of E symptoms, not to follow them. Partner conflict situations in the parents are associated with a higher risk for E in the offspring. Further investigations in the field of psychoneuroimmunology may shed light on the reasons for the contradictory results.

Treatment

The concept of patient management of eczema is based on patient education, which aims to achieve a constant cooperation between physician and patient in the treatment of this chronic disease, and to address also the psychological aspects of eczema. "Eczema school" programs have been successfully introduced in many countries.

Symptomatic treatment includes frequent use of emollients to restore disturbed epidermal barrier, oil baths and topical application of moisturizers, e.g., urea. Anti-inflammatory treatment uses topical steroids and calcineurin inhibitors, antiseptics, wet-wrap dressings in acute cases, and oral antihistamines. UV therapy, especially the long-wave UVA modality, has proven helpful in many patients. If indicated, topical antifungal treatment (for head and neck dermatitis) or systemic antibiotics (for treatment of impetiginization) are also given. In severe cases the use of systemic immunosuppressives, eg, cyclosporine is indicated**.**

Prevention

Recommendations for primary prevention in children at risk for atopic diseases propose breast feeding up to 4 months and late introduction of solid foods. Guidelines recommend avoidance of allergens (eg, mites, pets) although this is an area of controversy. In manifest E trigger factors that have been identified should be avoided, or specific allergen

avoidance strategies may be applied (e.g., dietary changes, encasing bedding against house dust mite allergen, removal of pets from the home, climate therapy at sea level or high altitude etc.). Prevention of drying of the skin of predisposed patients by creams and emollients is useful to protect against relapsing disease. Pharmacological prevention by intermittent use of anti-inflammatory topicals (steroids, calcineurin inhibitors) is under discussion and may be an option in the future.

1.14 Ringworm

Ringworm, also known as dermatophytosis or tinea, is a fungal infection of the skin. The name “ringworm” is a misnomer, since the infection is caused by a fungus, not a worm.

Ringworm infection can affect both humans and animals. The infection initially presents with red patches on affected areas of the skin and later spreads to other parts of the body. The infection may affect the skin of the scalp, feet, groin, beard, or other areas.

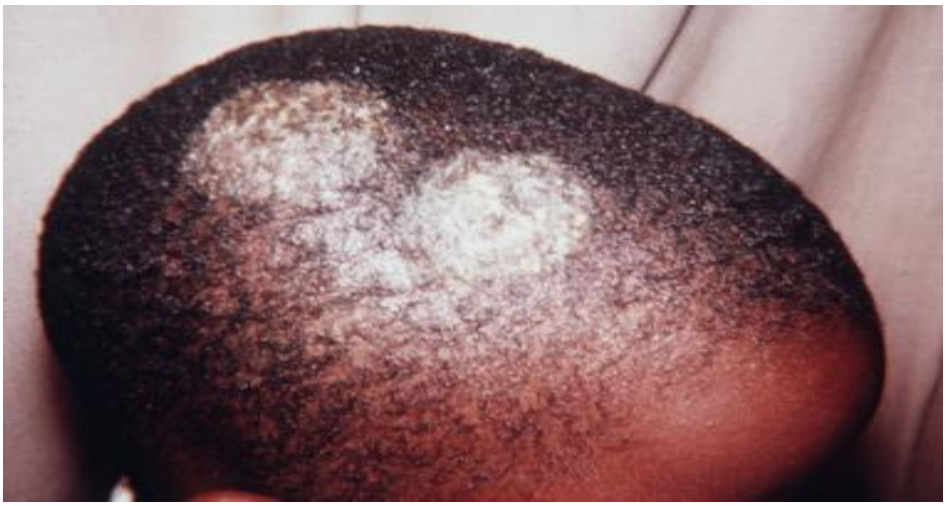


Figure 1.10 Tineasis

1.14.1 Recognizing ringworm:

Symptoms vary depending on where are infected. With a skin infection, individual may experience the following:

- red, itchy, scaly, or raised patches
- patches that develop blisters or begin to ooze
- patches that may be redder on the outside edges or resemble a ring

- patches with edges that are defined and raised

If someone is experiencing dermatophytosis in nails, they may become thicker or discolored, or they may begin to crack. If the scalp is affected, the hair around it may break or fall off, and bald patches may develop.

1.14.2 Types of ringworm:

Ringworm can go by different names depending on the part of the body affected.

- Ringworm of the scalp (tinea capitis) often starts as small sores that develop into itchy, scaly bald patches. It is most common among children.
- Ringworm of the body (tinea corporis) often appears as patches with the characteristic round "ring" shape.
- Jock itch (tinea cruris) refers to ringworm infection of the skin around the groin, inner thighs, and buttocks. It is most common in men and adolescent boys.
- Athlete's foot (tinea pedis) is the common name for ringworm infection of the foot. It is frequently seen in people who go barefoot in public places where the infection can spread, such as locker rooms, showers, and swimming pools.

1.14.3 Causes of ringworm

Three different types of fungi can cause this infection. They are called trichophyton, microsporum, and epidermophyton. It's possible that these fungi may live for an extended period as spores in soil. Humans and animals can contract ringworm after direct contact with this soil. The infection can also spread through contact with infected animals or humans. The infection is commonly spread among children and by sharing items that may not be clean.

1.14.4 Risk for ringworm

Anyone can develop ringworm. However, the infection is very common among children and people who own pet cats. Both cats and dogs can catch ringworm and then pass it on to humans who touch them. Signs to be aware of in pets include:

- hairless patches of skin that appear circular

- crusty or scaly patches
- patches that may not be completely hairless but have brittle or broken hairs
- opaque or whitish areas around the claws

Patient may be more likely to develop dermatophytosis if come into contact with the fungi while wet or if have minor skin injuries or abrasions. Using a public shower or public pool areas may also expose to the infective fungi.

If patient is often barefoot, he may develop ringworm of the feet (athlete's foot). Those who often share items such as hairbrushes or unwashed clothing also have an increased risk of developing the infection.

1.14.5 Diagnosing ringworm

Doctor will diagnose ringworm by examining skin and possibly using a black light to view skin in the affected area. The fungus will fluoresce (glow) under black light. If he/she is infected, the areas of the skin where fungus is located will glow.

Doctor may confirm a suspected diagnosis of ringworm by requesting certain tests:

- If patient is getting either a skin biopsy or fungal culture, doctor will take a sample of skin or discharge from a blister and send it to a lab to test it for the presence of fungus.
- If patient is getting a KOH exam, doctor will scrape off a small area of infected skin and place it in potassium hydroxide (KOH). The KOH destroys normal cells and leaves the fungal cells untouched, so they're easy to see under a microscope.

1.14.6 Treating ringworm

Doctor may recommend both medications and lifestyle adjustments to treat ringworm.

1.14.7 Medication

Doctor may prescribe various medications depending on the severity of ringworm infection. Jock itch, athlete's foot, and ringworm of the body can all be treated with topical medications, such as antifungal creams, ointments, gels, or sprays. Ringworm

of the scalp or nails may require prescription-strength oral medications such as ketoconazole, griseofulvin, or terbinafine.

Over-the-counter medications and antifungal skin creams may be recommended for use as well. Over-the-counter products may contain clotrimazole, miconazole, terbinafine, or other related ingredients.

1.14.8 Home care

In addition to prescription and over-the-counter medication, doctor may recommend that patient care for infection at home by practicing some of the following behaviors:

- avoid clothing that irritates the infected area
- cover it with a bandage if unable to avoid clothing that irritates the area
- wash bedding and clothes daily during an infection to help disinfect your surroundings
- clean and dry skin regularly

If someone've been scratching skin frequently due to the infection, he/she may also develop a staph or strep infection of the skin. Your doctor may prescribe antibiotics to treat this bacterial infection as you continue your treatment for the ringworm.

1.14.9 Preventing ringworm

Someone can prevent ringworm by practicing healthy and hygienic behaviors. Many infections come from contact with animals and lack of proper hygiene. Tips to avoid ringworm include:

Tips

1. Wash hands after interacting with an animal.
2. Disinfect and clean pet living areas.
3. Avoid people or animals with ringworm if someone have a weakened immune system.
4. Shower and shampoo hair regularly.

5. Wear shoes if showering in community areas.
6. Avoid sharing personal items like clothing or hairbrushes with people who might have ringworm.
7. Keep feet clean and dry.

1.15 Griseofulvin

Griseofulvin is used to treat skin infections such as jock itch, and ringworm; and fungal infections of the scalp, fingernails, and toenails. Griseofulvin comes as a tablet, capsule, and liquid to take by mouth. It is usually taken once a day or can be taken two to four times a day. Although symptoms may get better in a few days and will have to take griseofulvin for a long time before the infection is completely gone. It is usually taken for 2 to 4 weeks for skin infections, 4 to 6 weeks for hair and scalp infections, 4 to 8 weeks for foot infections, 3 to 4 months for fingernail infections, and at least 6 months for toenail infections. Follow the directions on your prescription label carefully, and ask your doctor or pharmacist to explain any part you do not understand. Take griseofulvin exactly as directed. Do not take more or less of it or take it more often than prescribed by doctor.

1.15.1 Pharmacodynamics

Griseofulvin is a mycotoxic metabolic product of Penicillium spp. It was the first available oral agent for the treatment of dermatophytoses and has now been used for more than forty years. Griseofulvin is fungistatic with in vitro activity against various species of Microsporum Epidermophyton, and Trichophyton. It has no effect on bacteria or on other genera of fungi. Following oral administration, griseofulvin is deposited in the keratin precursor cells and has a greater affinity for diseased tissue. The drug is tightly bound to the new keratin which becomes highly resistant to fungal invasions. Once the keratin-Griseofulvin complex reaches the skin site of action, it binds to fungal microtubules (tubulin) thus altering fungal mitosis.

1.15.2 Mechanism of Action:

Griseofulvin is fungistatic, however the exact mechanism by which it inhibits the growth of dermatophytes is not clear. It is thought to inhibit fungal cell mitosis and nuclear acid synthesis. It also binds to and interferes with the function of spindle and cytoplasmic microtubules by binding to alpha and beta tubulin. It binds to keratin in human cells, then

once it reaches the fungal site of action, it binds to fungal microtubes thus altering the fungal process of mitosis

1.15.3 Before taking griseofulvin

- Patient should tell doctor and pharmacist if he/she is allergic to griseofulvin, or any other medications.
- Patient should tell doctor and pharmacist what prescription and nonprescription medications are taking, especially anticoagulants ('blood thinners') such as warfarin (Coumadin), oral contraceptives, cyclosporine (Neoral, Sandimmune), phenobarbital (Luminal), and vitamins.
- Patient should tell doctor if he/she have or have ever had liver disease, porphyria, lupus, or a history of alcohol abuse.
- Patient should tell doctor if she is pregnant, plan to become pregnant, or are breast-feeding. If she become pregnant while taking griseofulvin, call the doctor.
- Patient should tell doctor if he drink alcohol.
- Patient should Ones should plan to avoid unnecessary or prolonged exposure to sunlight and to wear protective clothing, sunglasses, and sunscreen. Griseofulvin may make skin sensitive to sunlight.

2.9.7 Griseofulvin may cause side effects.

- upset stomach
- vomiting
- diarrhea or loose stools
- thirst
- fatigue
- dizziness
- faintness

If someone experience any of the following symptoms, call the doctor immediately:

- fever
- sore throat
- skin rash
- mouth soreness or irritation

Periorbital contact eczema is most commonly the result of an allergic contact dermatitis whereas other eczematous skin diseases like atopic eczema or seborrheic eczema occur less frequently. Also, other diseases like autoimmune disorders or rosacea need to be considered. Allergic contact dermatitis is a T-cell-mediated immunological response towards ubiquitous contact allergens. Activated T-cells migrate through the vessels into the skin and produce several inflammatory mediators. Epicutaneous patch testing is an important tool for the diagnosis of contact allergy whereby the allergens are analysed in terms of their ability to induce eczematous skin reaction. Until now the short-term use of corticosteroids are is employed for the treatment of allergic contact eczema. Modern substances with an optimal therapeutic index should rather be used

CHAPTER TWO

LITERATURE REVIEW

The global burden of disease (GBD) Study 2010 estimated the GBD attributable to 15 categories of skin disease from 1990 to 2010 for 187 countries. For each of the following diseases, we performed systematic literature reviews and analyzed resulting data: eczema, psoriasis, acne vulgaris, pruritus, alopecia areata, decubitus ulcer, urticaria, scabies, fungal skin diseases, impetigo, abscess, and other bacterial skin diseases, cellulitis, viral warts, molluscum contagiosum, and non-melanoma skin cancer. We used disability estimates to determine nonfatal burden. Three skin conditions, fungal skin diseases, other skin and subcutaneous diseases, and acne were in the top 10 most prevalent diseases worldwide in 2010, and eight fell into the top 50; these additional five skin problems were pruritus, eczema, impetigo, scabies, and molluscum contagiosum. Collectively, skin conditions ranged from the 2nd to 11th leading cause of years lived with disability at the country level. At the global level, skin conditions were the fourth leading cause of nonfatal disease burden. Using more data than has been used previously, the burden due to these diseases is enormous in both high- and low-income countries. These results argue strongly to include skin disease prevention and treatment in future global health strategies as a matter of urgency. (Hay *et al.,* 2014)

Many skin diseases in developing countries are associated with socioeconomic factors. It is generally agreed that a public health approach to dermatology in this setting is particularly appropriate; but, there has been little epidemiologic research done to examine which particular socioeconomic factors are important determinants of the prevalence of skin disease. This is especially true in sub-Saharan Africa. A survey of two villages in Ngara district, North West Tanzania, involved 254 randomly selected households using a questionnaire, measurements of houses and water quality, and examination of the skin of 1114 household members. Significant skin disease was encountered in 300 individuals (26.9%). Transmissible diseases comprised the bulk of skin disease (73.9%) with younger age groups being affected most. Socioeconomic conditions were poor, with low quality, crowded housing, low levels of literacy, unsatisfactory water sources, and few households with a regular cash income. Household density was significantly associated with transmissible skin disease. Other indicators of poverty (e.g., no regular cash income and illiteracy) did not correlate with the prevalence of skin disease. Skin disease as highly prevalent in the villages surveyed, especially transmissible diseases in the younger age groups. Household density was the only socioeconomic factor significantly associated with skin disease. Reduction of household density is an attainable intervention that could

reduce the prevalence of skin disease in rural African populations. Simple studies like this one should be the first step in community dermatology, assessing the burden of skin disease in communities and looking for particular factors with which public health interventions could effectively reduce the prevalence of skin disease. (Gibbs, 2017).

An international collaboration lead by Nagoya University researchers has drawn on their experience helping patients with serious skin conditions to define a whole new category of genetic skin diseases. Keratinization is the process that forms the outer layer of our skin, based on tough and water-resistant proteins, including keratin and lipids, such as ceramides. However, any number of problems can stop keratinization from working properly, leading to familiar skin problems. For example, immune or allergic reactions and many types of eczema involve an inflammatory response, where the body's own immune system attacks itself, overreacting to an irritant. In their recent perspective article, published in the Journal of Allergy and Clinical Immunology, the Nagoya group argues that some skin conditions are actually an "autoinflammatory" response with a genetic basis. These conditions reflect a more uncontrolled primitive response of the body's deeper immune system, which does not necessarily depend on exposure to an irritant. (Alphagalileo.org., 2017).

It is generally agreed that the pattern of skin diseases differs in different countries, and within various regions of a country depending on social, economic, racial and environmental factors. Many workers have reported various patterns of skin diseases in different countries. So far, no such report is available in our country for Faridpur region. To fill the lacunae we decided to undertake a retrospective study of the skin disease pattern in this tertiary hospital of Faridpur, Bangladesh. All the newly diagnosed cases attending the OPD of Dermatology and Venereology, Faridpur Medical College Hospital, during the period of one year starting from 1st July 2007 to 30th June, 2008 were included in the study. Diagnosis was done on clinical grounds and laboratory investigations were done whenever required. Eczema (19.2%), fungal infections (17.26%), scabies (15.16%) and pyodermas (7.59%) were the major skin diseases. STD's accounted for (0.73%) of the cases. Genodermatoses (0.01%) formed the minimal number of cases. Eczema was the commonest group of disorders. Out of the infective skin disorders fungal infections were the commonest group. Genodermatoses formed the least number of cases. (Sarkar *et al.,* 2011)

A cross sectional study was carried out in the Dermatology & Venereology OPD of NIMCH, Dhanmondi, Dhaka, Bangladesh, during the period of January 2012 to December 2012. A total of 1169 patients (511male and 658 female) aged 10-44 years, attended in the OPD in which 969 patients (450 male and 519 female) were found to have dermatological problems. Data were analyzed by using SPSS soft-ware version 18. The pattern of skin diseases was higher in females (53.6%) than in males (46.4%). The most common disorders seen in outpatient department is eczema (23.7%) followed by fungal infection (15.5%), scabies (13.4%) and acne vulgaris (11.9%). A remarkable part (41.4%) of the patients was illiterate. Majority of the patients (67.6%) belonged to poor socio-economic class. 61.4 % patients were residing in overcrowded homes and 53% patients were not maintained personal hygienic standards. (Yousuf *et al.*, 2014)

The research hypothesized that the disease burden from inadequate water, sanitation, and hygiene practices in selected unions of Mymensingh, Madhupur and Durgapur Upazilas of Bangladesh would be the infection of various diseases, principally diarrhea and skin diseases, and usually caused in conjunction by other risk factors or diseases. Inthis study, the disease burden was primarily finding out from the baseline survey and then it was justified with some economic and demographic characteristics to understand the dynamics of the diseases. For that purpose, two models has been drawn up: the baseline survey model to predict the water borne diseases and the follow up survey or risk assessment model to find the association with different water uses in the household. For the assessment, some field-based scenarios have been developed to identify risk factors, obtained mainly from the follow up survey. We hypothesized that the disease burden from inadequate water, sanitation, and hygiene resulting in diarrhea and skin diseases occurred widely in the study area and that this burden is largely preventable. Other water and sanitation related diseases remain to be evaluated. (Haque , 2017)

Significance of the Study

Most of the skin infections treatment takes long time to show their effects. But general impression is 10-20 percent of patients seeking medical advice suffer from skin diseases. The skin conditions are prevalent across all parts of the world. It is generally agreed that the pattern of skin diseases differs in different countries, and within various regions of a country depending on social, economic, racial and environmental factors. Many workers have reported various patterns of skin diseases in different countries. Skin disease is an important problem in Bangladesh especially for lower class people because of unhealthy life style and unhealthy environment of the lower class people in Bangladesh.

From the present study among the 300 lower class people in Bangladesh it has been seen that among 300 people 13% were within (18-28) years, about 15 were within (28-38) years, approximately 19 were within (38-48) years, 31% were (48-58) years, and 19% were (58-70) years old.

From the present study among the 300 lower class people in Bangladesh it was seen that among 300 people 31.25% were rickshaw puller, 4% were sweeper, 6.25% were worker, 21% were hawker, 25.25% fisher man and 14 were other types of occupation.

A study mentioned that on skin disease diagnosis was done on clinical grounds and laboratory investigations were done whenever required. Eczema (19.2%), fungal infections (17.26%), scabies (15.16%) and pyodermas (7.59%) were the major skin diseases. STD's accounted for (0.73%) of the cases. Genodermatoses (0.01%) formed the minimal number of cases. Eczema was the commonest group of disorders. Out of the infective skin disorders fungal infections were the commonest group. Genodermatoses formed the least number of cases. (Sarkar *et al.,* 2011)

Another study showed that a total of 1169 patients (511male and 658 female) aged 10-44 years, attended in the OPD in which 969 patients (450 male and 519 female) were found to have dermatological problems in the Dermatology & Venereology OPD of NIMCH, Dhanmondi, Dhaka, Bangladesh in where The pattern of skin diseases was higher in females (53.6%) than in males (46.4%). The most common disorders seen in outpatient department is eczema (23.7%) followed by fungal infection (15.5%), scabies (13.4%) and acne vulgaris (11.9%). A remarkable part (41.4%) of the patients was illiterate. Majority of the patients (67.6%) belonged to poor socio-economic class. 61.4 % patients were

residing in overcrowded homes and 53% patients were not maintained personal hygienic standards. (Yousuf *et al.*, 2014)

Aims and Objective of the Study

The main objectives of the study are –

- To determine prevalence estimation of skin diseases risk factors of mass people.
- To determine the Behavioral factors associated with skin disease.
- To determine the Biological factors associated with skin disease.
- To determine the knowledge and awareness regarding the Risk factors.

CHAPTER THREE

METHODOLOGY

Methodology:

3.1 Type of the Study

It was a survey based study.

3.2 Study Area

- Rampura
- Munshiganj
- Banasree
- Moduberg
- Kamalapur
- Dhakkin khan
- Abdullah pur
- Kamar para
- Uttarkhan

3.3 Inclusion Criteria

- Lower class people
- Both males and females

3.4 Exclusion Criteria

The respondents who are unwilling to conduct the survey were excluded.

3.5 Study Population

In this study, a total number of 300 respondents out of mass people were surveyed.

3.6 Study Period

The duration of the study was about one years starting from July,2016 to June, 2017.

3.7 Questionnaire Development

The pre-tested questionnaire was specially designed to collect the simple background data and the needed information. The questionnaire was written in simple English in order to avoid unnecessary semantic misunderstanding. The questionnaire was pilot tested to ensure it was understandable by the participants to assess the awareness and knowledge about prevalence and risk factors of skin diseases.

3.8 Sampling Technique

In this study convenient sampling technique was followed.

3.9 Data Collection and Analysis

Informed consent was obtained from the eligible participants before interviewed and participants who agreed to join the study provided the required information for the studies. After collecting, the data were checked and analyzed with the help of Microsoft Excel 2010. The result was shown in bar, pie and column chart and calculated the percentage of the awareness and disease regarding skin diseases among the peoples.

CHAPTER FOUR

RESULT

4.1 Age of the respondents

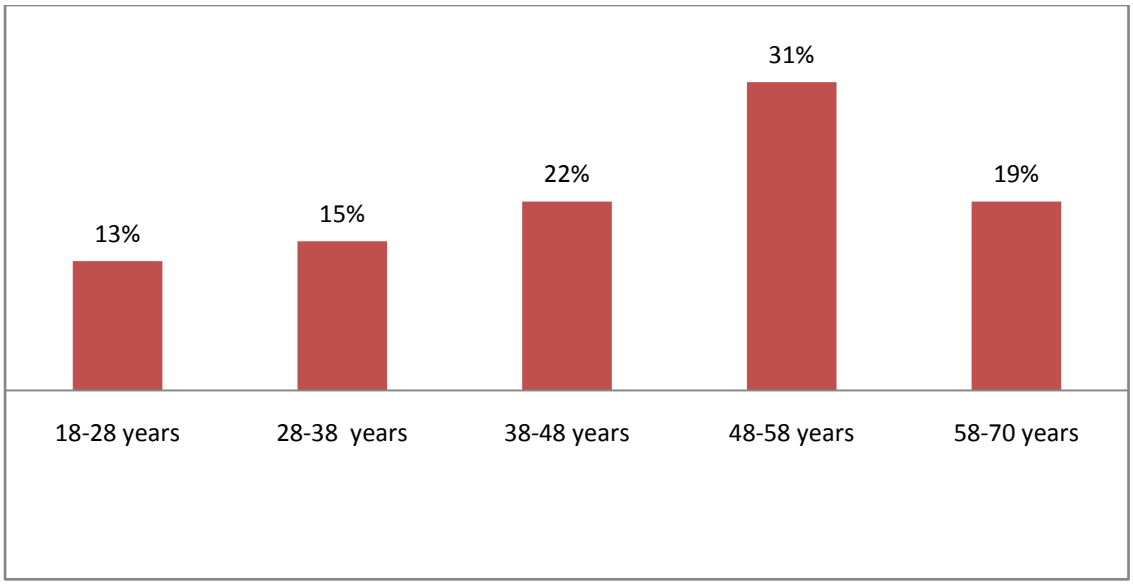
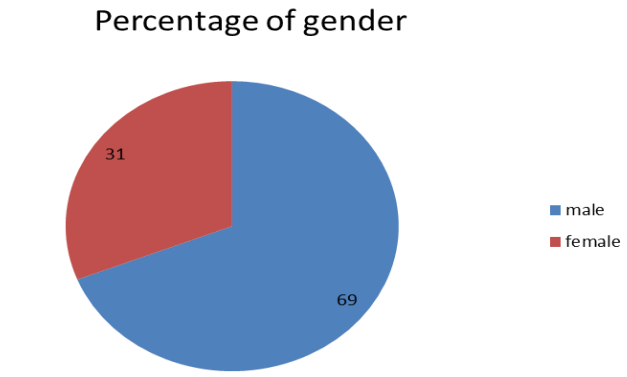


Figure 4.1 Age of the respondents

In this study within 300 people, 13% people was being between 18-28 years,15% being 28-38 years, 22% being 38-48 years,31% being 48-58 years and rest 19% was 58-70 years.

4.2 Gender of the respondents



4.2 Figure: Percentage of gender

In study, 69% were male and 31% were female among the 300 respondents.

4.3 Occupation of the respondents

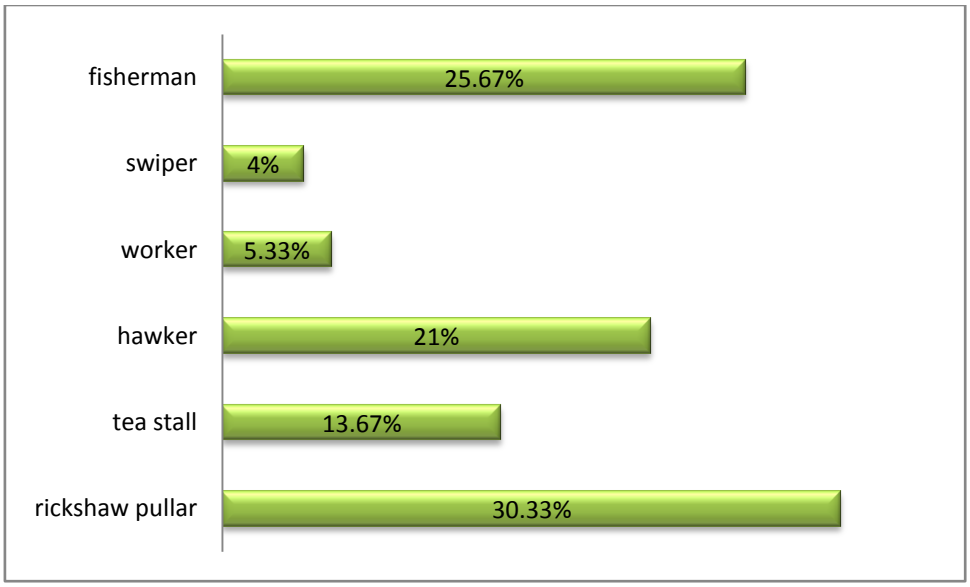


Figure 4.3 Occupation of the respondents

In this survey, 30.33% were rickshaw puller, 25.75 were fisherman, 21% were hawker, 13.75% were tea stall, 5.25 were worker and 4% were sweeper.

4.4 Place of going for treatment

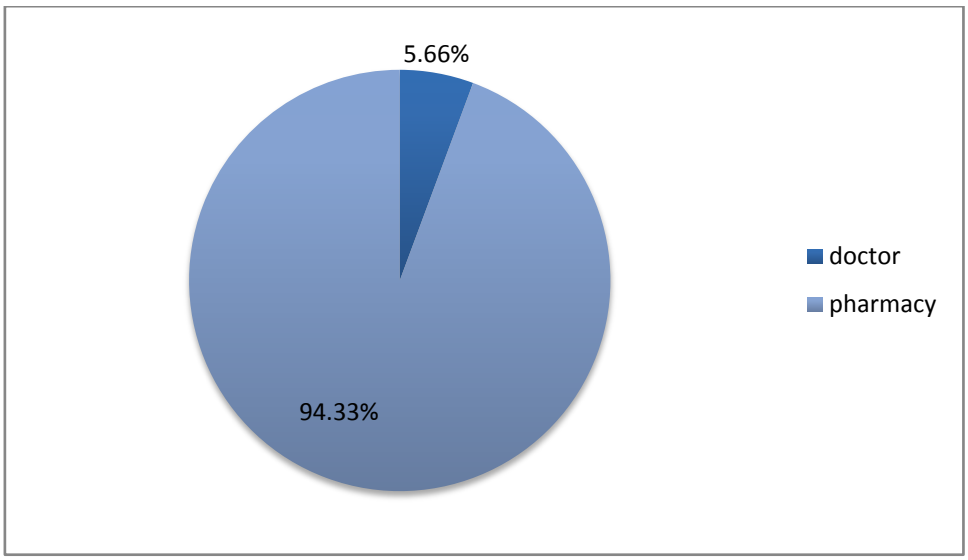


Figure 4.4 place of going for treatment

In this survey among 300 respondents, 94.25% were gone to pharmacy and 5.75% were gone to doctor.

4.5 Patient with ring worm/ Tineasis

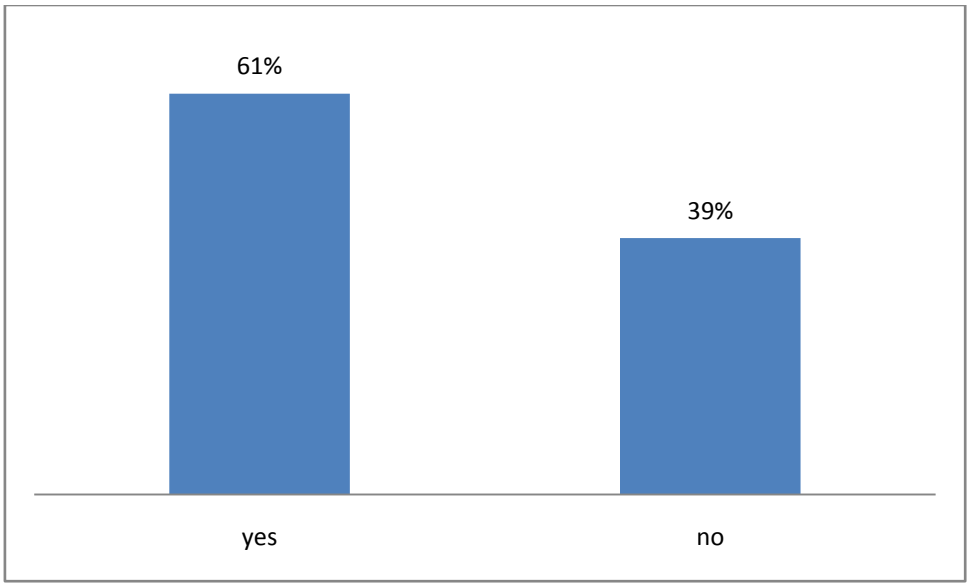


Figure 4.5 Patient with ring worm/Tineasis

In this study, patients with ringworm were 61% and rest of the respondents gives negative answer.

4.6 Drug taking percentage for ring worm disease

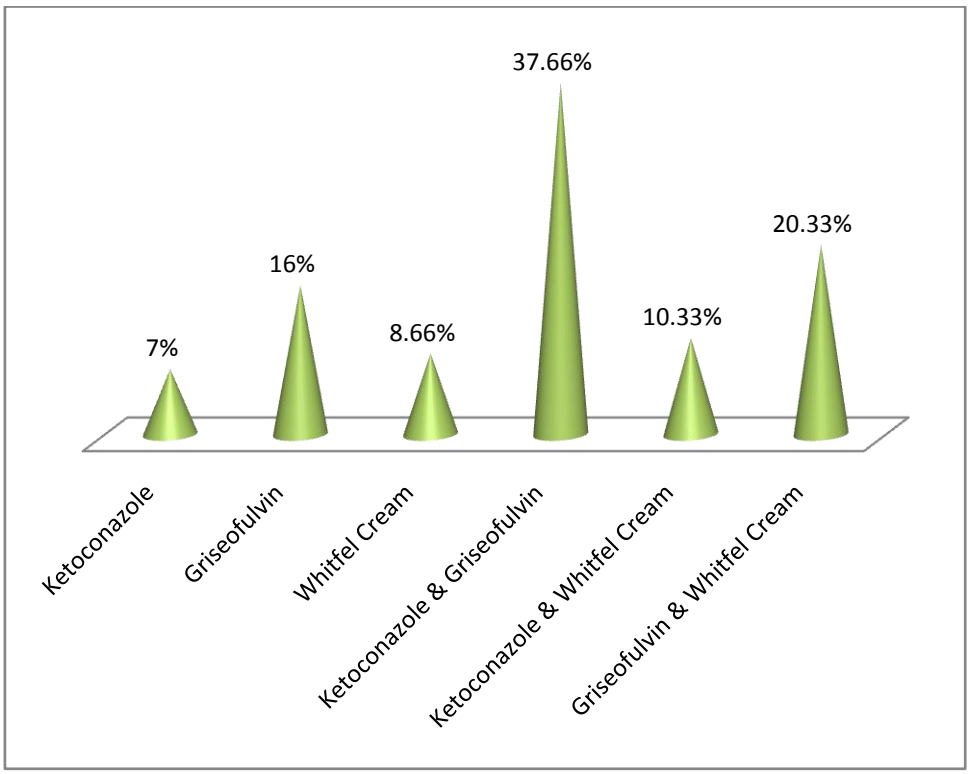


Figure 4.6 Percentage of drug taking for ringworm disease

Patients were highly used Ketoconazole & Griseofulvin combination (37.66%). Other were used ketoconazole (7%), Griseofulvin (16%), Whitfel Cream (8.66%), Ketoconazole & Whitfel Cream (10.33%) and Griseofulvin & Whitfel Cream (20.33%), respectively.

4.7 Percentage of side effect of ringworm drug

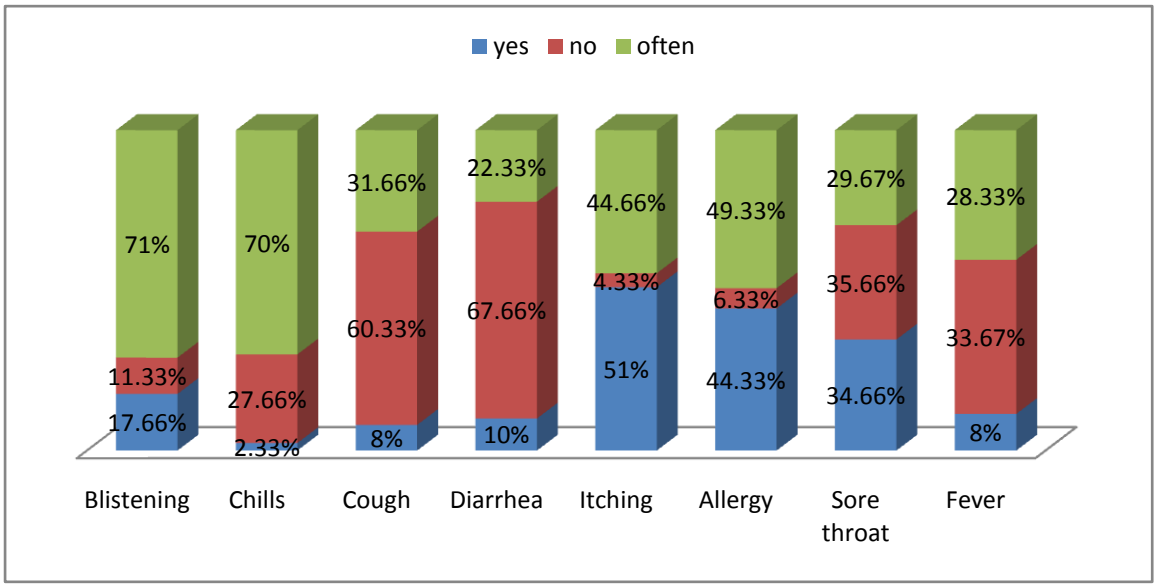


Figure 4.7 Percentage of side effect of ringworm drug

In this study, highest percent of respondents were suffering from itching (51%) and allergy (44.33%). Rest of the respondents was facing the other side effects of ringworm drug.

4.8 Patients with Eczema

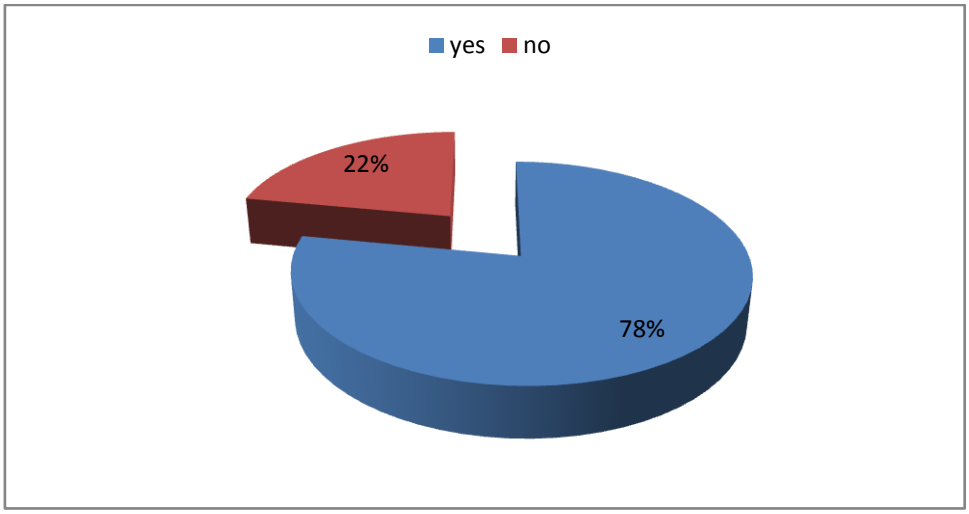


Figure 4.8 Patients with Eczema

Patients with eczema were 78% and others (22%) were not suffering from any kind of eczema.

4.9 Percentage of patient who is taking medicine for Eczema

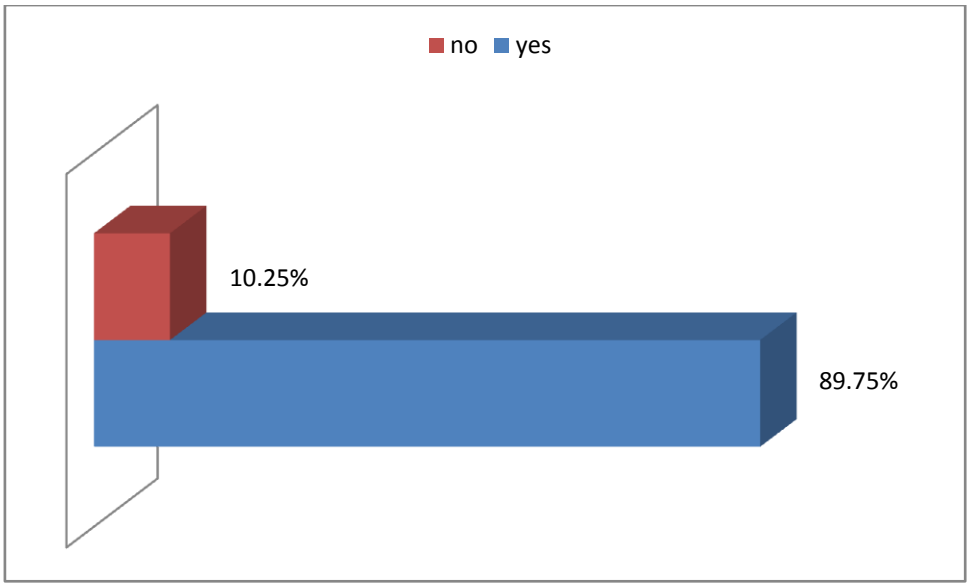


Figure 4.9 Percentage of patient who is taking medicine for that disease

For the eczema disease 89.75% respondents were taking drug to prevent from this condition.

4.10 Percentage of various drugs taking for Eczema

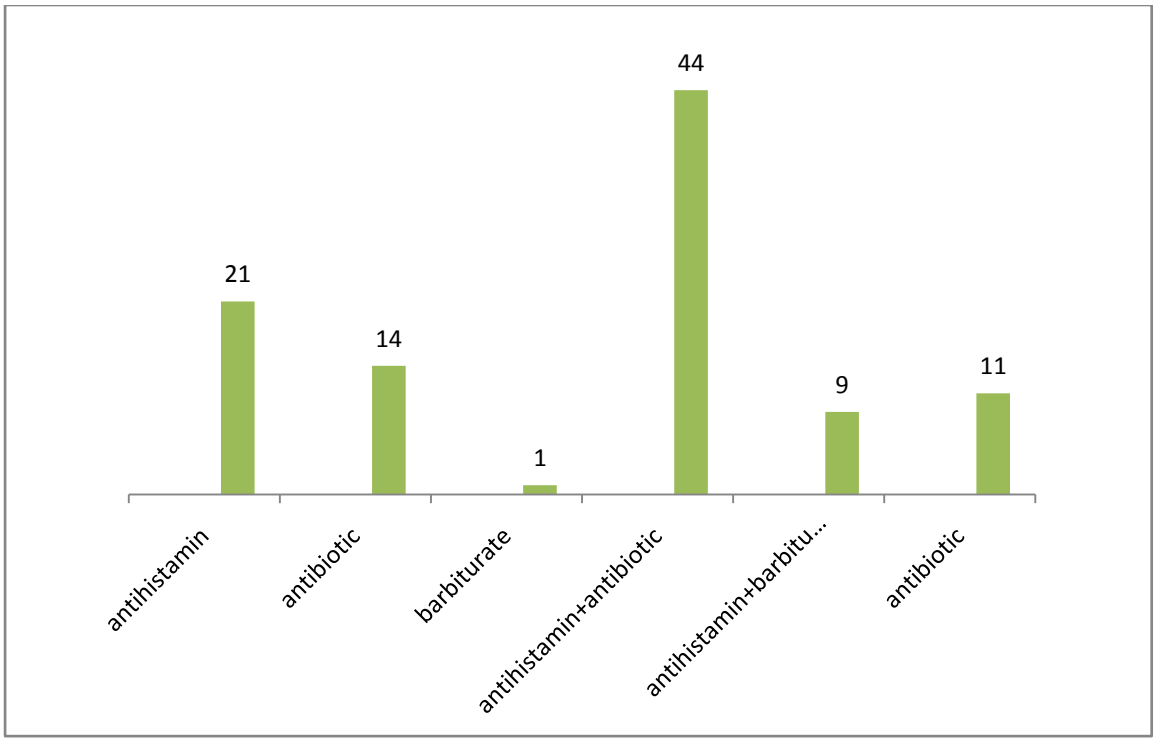


Figure 4.10 Percentage of various drugs taking for Eczema

In this study 44% of the respondent's taking antihistamine and barbiturates combination drug. Rest of the respondent's taking antihistamine (21%), antibiotic (14%), barbiturates (1%), antihistamine and barbiturates combination (9%) and antibiotic (11%), respectively.

4.11 Side effect of Eczema drug

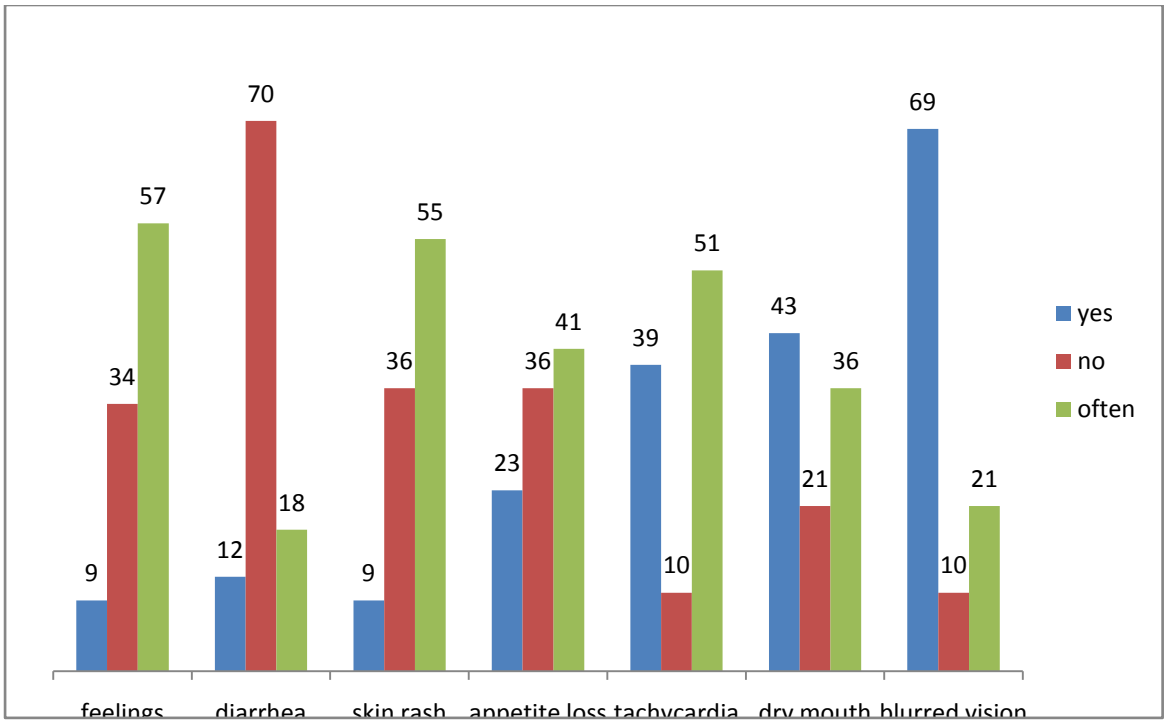


Figure 4.11 Side effect of eczema drug

In this study, highest percent of the respondents were suffering from blurred vision (69%) and dry mouth (43%). Rest of the respondents was facing the other side effects of eczema drug.

4.12 Percentage of people who are suffering from scabies skin disease

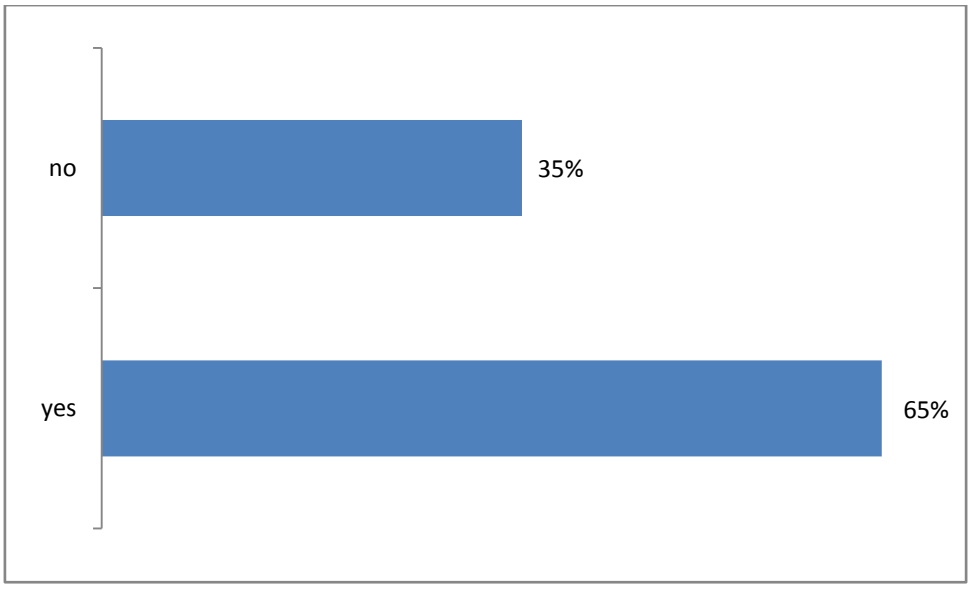


Figure 4.12 Percentage of people who are suffering from scabies skin disease

In this study 65% of the respondents were suffering from scabies diseases and others gave the negative answer.

4.13 Taking medication for scabies disease

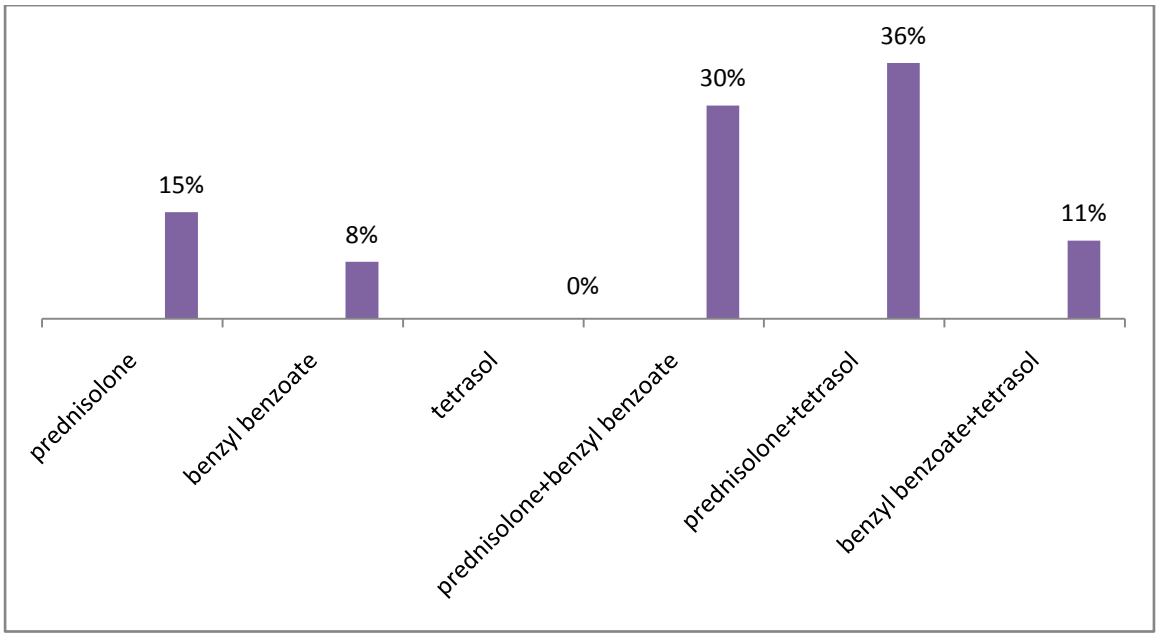


Figure 4.13 taking medication for scabies disease

In this study 36% of the respondent's taking prednisolone tetrasol combination drug. Rest of the respondent's taking prednisolone (15%), benzyl benzoate (8%), prednisolone and benzyl benzoate combination (30%) and benzyl benzoate tetrasol combination (11%) respectively.

4.14 Side effect of scabies drug

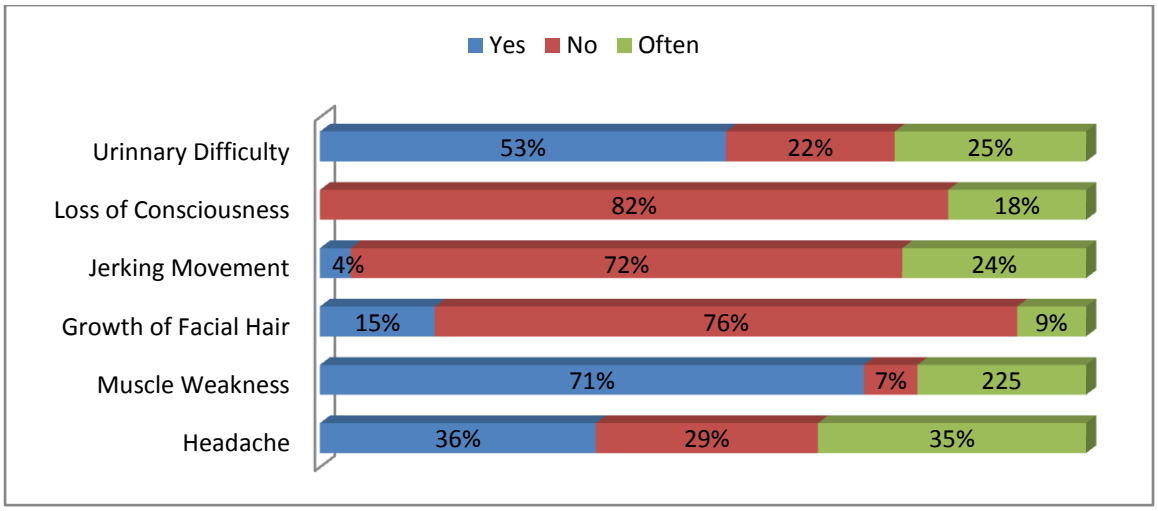


Figure 4.14 Side effect of scabies drug

In this study, highest percent of the respondents were suffering from muscle weakness (71%) and urinary difficulties (53%). Rest of the respondents was facing the other side effects of scabies drug.

CHAPTER FIVE

DISCUSSION AND CONCLUSION

5.1 Discussion:

Skin disease is an important problem in Bangladesh especially for lower class people because of unhealthy life style and unhealthy environment of the lower class people in Bangladesh.

The objective of this study is to get a picture of overall condition of the skin disease, the drug's side effects profile which are using for skin disease without prescription. And thereby get more accurate measures to minimize the occurrence. The result are obtained in this study are correlated with each other and by establishing a relationship among these give a better picture to make a suitable decision with lower class people in Bangladesh. The results were from the present study among the300 lower class people in Bangladesh it was seen that among 300 people about 31% people were female and 70% people were male.

From the present study among the 300 lower class people in Bangladesh it has been seen that among 300 people 13% were within (18-28) years, about 15 were within (28-38) years, approximately 19 were within (38-48) years, 31% were (48-58) years, and 19% were (58-70) years old.

From the present study among the 300 lower class people in Bangladesh it was seen that among 300 people 31.25% were rickshaw puller, 4% were sweeper, 6.25% were worker, 21% were hawker, 25.25% fisher man and 14 were other types of occupation.

From the present study among the 300 lower class people in Bangladesh it was seen that among 300 people 5.75% were go to the doctor and 94.25% people were go to pharmacy shop for the treatment.

In this study within 300 people,13% people was being between 18-28 years,15% being 28-38 years, 22% being 38-48 years,31% being 48-58 years and rest 19% was 58-70 years. In study, 69% were male and 31% were female among the 300 respondents.

In this study, patients with ringworm were 61% and rest of the respondents gives negative answer. Patients were highly used Ketoconazole & Griseofulvin combination (37.66%). Other were used ketoconazole (7%), Griseofulvin (16%), Whitfel Cream (8.66%), Ketoconazole & Whitfel Cream (10.33%) and Griseofulvin & Whitfel Cream (20.33%), respectively. highest

percents of respondents were suffering from itching (51%) and allergy (44.33%). Rest of the respondents was facing the other side effects of ringworm drug.

Patients with eczema were 78% and others (22%) were not suffering from any kind of eczema. For the eczema disease 89.75% respondents were taking drug to prevent from this condition. For the treatment of this these disease 44% of the respondent's taking antihistamine and barbiturates combination drug. Rest of the respondent's taking antihistamine (21%), antibiotic (14%), barbiturates (1%), antihistamine and barbiturates combination (9%) and antibiotic (11%), respectively. The highest percent of the respondents were suffering from blurred vision (69%) and dry mouth (43%). Rest of the respondents was facing the other side effects of eczema drug.

In this study, about 65% of the respondents were suffering from scabies diseases and others gave the negative answer. About 36% of the respondent's taking prednisolone tetrasol combination drug for the prevention of this disease. Rest of the respondents was taking prednisolone (15%), benzyl benzoate (8%), prednisolone and benzyl benzoate combination (30%) and benzyl benzoate tetrasol combination (11%) respectively. In this study, highest percent of the respondents were suffering from muscle weakness (71%) and urinary difficulties (53%). Rest of the respondents was facing the other side effects of scabies drug.

5.2 Conclusion:

Skin disease are numerous and a frequently occurring health problem affecting all ages from the neonates to the elderly and cause harm in number of ways. Maintaining healthy skin is important for a healthy body. Many people may develop skin diseases that affect the skin, including cancer, herpes and cellulitis .Various factors are associated with this like as irritation, chemicals, infection, UV-radiation, temperature. WHO has suggested about the prevalence and cost of skin disease in the United States are increasing and will continue to do so as the population ages, according to an analysis of claims data presented here at the American Academy of Dermatology (AAD) Annual Meeting (Skin Diseases Affect One in Four Americans). Americans older than 65 years were more likely to make a claim for skin disease than those 18 to 44 years (49.4% vs 34.0%). And, on average, people in the older age group had 2.2 skin diseases. Some wild plants and their parts are frequently used to treat these diseases. The use of plants is as old as the mankind. Natural treatment is cheap and claimed to be safe. It is also suitable raw material for production of new synthetic agents. A review of some plants for the treatment of skin diseases is provided that Skin disease is most leading disease in the perspective of Bangladesh. Skin diseases are summarizes the recent technical advancements that have taken place in this area during the past 17 years.

CHAPTER SIX

REFERENCES

Alphagalileo.org. (2017). Nagoya Medical Researchers Propose New Disease Category of Skin Disorders. *Journal of Investigative Dermatology* [online] Available at: http://www.alphagalileo.org/ViewItem.aspx?ItemId=177320&CultureCode=en [Accessed 13 Jul. 2017].

Aumailley, M., & Krieg, T. (1996). Laminins: A family of diverse multifunctional molecules of basement membranes. *Journal of Investigative Dermatology*, *106*(2), 209-214

Caputo, R., & Peluchetti, D. (1977). The junctions of normal human epidermis: A freeze-fracture study. Journal of Ultrastructure Research, 61(1), 44-61.

Chu, D. H. (2008). Overview of biology, development, and structure of skin. In K. Wolff, L. A. Goldsmith, S. I. Katz, B. A. Gilchrest, A. S. Paller, & D. J. Leffell (Eds.), *Fitzpatrick's dermatology in general medicine* (7th ed., pp. 57-73). New York: McGraw-Hill.

Drugs.com. (2017). Prednisolone Uses, Side Effects & Warnings - Drugs.com. [online] Available at: https://www.drugs.com/mtm/prednisolone.html [Accessed 11 Jul. 2017].

Emedicine.medscape.com. (2017). Scabies: Practice Essentials, Background, Pathophysiology. [online] Available at: http://emedicine.medscape.com/article/1109204-overview [Accessed 11 Jul. 2017].

Fitz Gerald, D., Grainger, R. and Reid, A. (2017). Interventions for preventing the spread of infestation in close contacts of people with scabies.

Gibbs, S. (2017). Skin disease and socioeconomic conditions in rural Africa: Tanzania. *Journal of Investigation Dermatology*. [online] 35(9):633-9. Available at: https://www.ncbi.nlm.nih.gov/pubmed/8876289 [Accessed 13 Jul. 2017].

Haake, A. R., & Hollbrook, K. (1999). The structure and development of skin. In I. Freedberg, A. Eisen, K. Wolff, K. Austen, L. Goldsmith, S. Katz, et al. (Eds.), Fitzpatrick's dermatology in *general medicine* (5th ed., pp. 70-111). New York: McGraw-Hill.

Haque, M. (2017). [online]. Research Paper On Water borne Diseases Caused by Using Surface Water, *Knowledge for Health* [online] Available at:

https://www.k4health.org/sites/default/files/research_report_water_borne_disease_caused_by_using_surface_water.pdf [Accessed 13 Jul. 2017].

Hay, R., Johns, N., Williams, H., Bolliger, I., Dellavalle, R., Margolis, D., Marks, R., Naldi, L., Weinstock, M., Wulf, S., Michaud, C., Murray, C. and Naghavi, M. (2014). The global burden of skin disease in 2010: an analysis of the prevalence and impact of skin conditions. *Journal of Investigation Dermatology*. 134(6):1527-34. doi: 10.1038/jid.2013.446

Jackson, S. M., Williams, M. L., Feingold, K. R., & Elias, P. M. (1993). Pathobiology of the stratum corneum. *Western Journal of Medicine, 158*(3), 279-285.

James, W. D., Berger, T. G., & Elston, D. M. (2006). *Andrews' diseases of the skin: Clinical dermatology* (10th ed.). Philadelphia: Elsevier Saunders.

Kanitakis, J. (2002). Anatomy, histology and immunohistochemistry of normal human skin. *European Journal of Dermatology*, *12*(4), 390-401.

Kerr, J. F., Wyllie, A. H., & Currie, A. R. (1972). Apoptosis: A basic biological phenomenon with wide-ranging implication in tissue kinetics. *British Journal of Cancer*, *26*(4), 239-257.

Kolarsick, P., Kolarsick, M. and Goodwin, C. (2017). Anatomy and Physiology of the Skin.

Matoltsy, A. G. (1976). Keratinization. Journal of Investigative Dermatology, 67(1), 20-25.

Mauro, T., & Goldsmith, L. (2008). Biology of eccrine, apocrine, and apoeccrine sweat glands. In K. Wolff, L. A. Goldsmith, S. I. Katz, B. A. Gilchrest, A. S. Paller, & D. J. Leffell (Eds.), *Fitzpatrick's dermatology in general medicine* (7th ed., pp. 713-720). New York: McGraw-Hill

MedicineNet. (2017). Medical Definition of Skin Diseases, National Institute of Arthritis & Musculoskeletal &. [online] Available at: http://www.medicinenet.com/script/main/art.asp?articlekey=7062 [Accessed 15 Jul. 2017].

Medlineplus.gov. (2017). Tinea infections | Ringworm | Jock itch | MedlinePlus. [online] Available at: https://medlineplus.gov/tineainfections.html [Accessed 12 Jul. 2017].

Moll, I. (1994). Merkel cell distribution in human hair follicles of the fetal and adult scalp. Cell and Tissue Research, 277(1), 131-138.

Murphy, G. F. (1997). Histology of the skin. In D. Elder, R. Elenitsas, C. Jaworsky, & B. Johnson Jr. (Eds.), *Lever's histopathology of the skin* (8th ed., pp. 5-45). Philadelphia: Lippincott Williams & Wilkins.

Paus, R., Foitzik, K., Welker, P., Bulfone-Paus, S., & Eichmuller, S. (1997). Transforming growth factor beta receptor type I and type II expression during murine hair follicle development and cycling. Journal of Investigative Dermatology, 109(4), 518-526.

Sarkar, S., Islam, A., Sen, K. and Ahmed, A. (2011). Pattern Of Skin Diseases In Patients Attending OPD Of Dermatology Department At Faridpur Medical College Hospital, Bangladesh. *Faridpur Medical College Journal*, [online] 5(1). Available at: http://www.banglajol.info/index.php/FMCJ/article/view/6807 [Accessed 13 Jul. 2017]

Sawaya, M. E. (1994). Biochemical mechanisms regulating human hair growth. Skin Pharmacology, 7(1-2), 5-7.

Schwarz, E. (1979). Biochemie der epidermalen keratinisation. In A. Marchionini (Ed.), *Handbuch der hautund geschlectskrankheiten* (Vol. I). Berlin: Springer-Verlag.

Stepp, M. A., Spurr-Michaud, S., Tisdale, A., Elwell, J., & Gipson, I. K. (1990). Alpha 6 beta 4 integrin heterodimer is a component of hemidesmosomes. Proceedings of the National Academy of Sciences of the United States of America, 87(22), 8970-8974.

Udey, M. C. (1997). Cadherins and Langerhans cell immunobiology. *Clinical and Experimental Immunology*, *107*(Suppl. 1), 6-8.

Worldallergy.org. (2017). Eczema Pathophysiology | World Allergy Organization. [online] Available at: http://www.worldallergy.org/education-and-programs/education/allergic-disease-resource-center/professionals/eczema-pathophysiology [Accessed 11 Jul. 2017].

Worm, M. & Sterry, W. (2017). [Periorbital contact eczema]. - PubMed - NCBI. [online] Ncbi.nlm.nih.gov. Available at:

https://www.ncbi.nlm.nih.gov/pubmed/?term=the+skin+disease+like+eczema+%2Cring+worm [Accessed 12 Jul. 2017].

Yousuf, A., Joarder, Y., Aktar, A., Hoq, M., Rahman, M., Akter, F., Begum, H., Akhtar, M. and Rahman, A. (2014). Pattern of skin diseases in patients attended OPD of Dermatology and Venereology in a tertiary care teaching hospital in Bangladesh. *Bangladesh Journal of Physiology and Pharmacology*, 29(1-2). DOI: http://dx.doi.org/10.3329/bjpp.v29i1-2.20061