Chapter 1

Introduction

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Vertebrate body contains both soft and hard tissues to carry out life processes. Among the hard tissues horn, hoof, feather, claw and nail are the derivatives of the skin. Generally skin is known as integument. However, the hard tissues, which serve as exoskeleton, can also be referred as integuments as they are highly useful in protecting the body from the environmental influences, acting as thermo- insulators and thermo – regulators.

In nail, the dorsal unguis is brought and flattened while the sub unguis is reduced to a small remnant, which lies under the tip of the nail. The root of the nail is the place of growth of the unguis, lies embedded in a pocket under the skin called the nail groove or sulcus unguis. The nail bed lies beneath both the nail and its root. The proximal part or matrix is the most important and is concerned in the formation of the nail. Its anterior portion in man may be seen through the base of the

transparent thumbnail and forms the whitish lunula, the crescent shaped area. The stratum germinativum above the base of the nail forms a rather rough margin where the nail emerges from the sulcus unguis.

As far as organic matter is concerned, the major portion of the nail is made up of albuminoid proteins called keratins. These fibrous proteins are characterized by their very high insolubility in usual protein solvents, because of high sulphur content, which is in the form of cystine.

Indeed, biologists have developed specific areas – anatomy, physiology and biochemistry of the calcified tissues, and valuable treatises have been presented as a result of these studies. But no attention is paid to make systematic investigations on physical properties of nail.

In view of the above facts it is proposed to study molecular composition and structure; growth, mechanical, electrical and electronic properties of keratinous and calcified hard tissue the human nail.

1.1 Integumentary system

Integumentary system consists of skin and its derivatives (Sweat, oil glands, hairs and nails). All structures of the integumentary system developed from the ectodermal and mesoderm germ layers. The integumentary system, as a body covering develops during the early embryonic period. Other integumentary structures, such as hair, nails, and glands, develop throughout the prenatal period. The system is an important boundary layer that separates the organism form the outside world. It is the largest organ of the human body.

1.2 Types of integument

1.2.1 Skin

The skin consists of three structural and functional layers, the epidermis dermis and hypodermis. The epidermis is the outer a vascular epithelial layer. It consists of a keratinised stratified squamous epithelium that provides skin's barrier function. This layer is constantly being replaced by a system of proliferation and desquamation.

The dermis is the inner layer and consists of a dense irregular connective tissue. It provides the skin's mechanical supports and strength. The glands and hair follicles mostly reside here.

The hypodermis is a transitional layer of loose connective and adipose tissue between the dermis and underlying structures, permitting movement between the two. The hypodermis is not considered to be part of the skin proper; in gross anatomy it is referred to as the superficial fascia.

Skin is classified as thick or thin depending upon the thickness of the epidermis. Thick skin covers the palms (vola) and soles (planta), areas subject to abrasion. It is typically glabrous (hairless). Thin skin covers basically everywhere else and is typically pileous (haired).

The epidermis is composed of a keratinized stratified squamous epithelium. Its cells, keratinocytes, are arranged into 4 (thin skin) or 5 (thick skin) layers. The outermost layer is always composed of non-living, highly keratinized (conrnified) cells that function to reduce water loss. As keratinocytes leave the germinal layer, they accumulate specific products

to the exclusion of all other products and organelles, eventually resulting in their demise.

From basal to apical, the layers or strata of the epidermis are as follows:

I. Stratum basale: This is the proliferative layer resting on the basement membrane. It consists of a single layer of cuboidal cells with spars, basophilic cytoplasm and variable amount of melanin. These cells are connected to one another by desmosomes and to the basal lamina by hemidesmosomes.

II. Stratum spinosum : This stratum is typically several cells thick but does vary with location. Its polygonal cells have a central nucleus and exhibit numerous cytoplastmic processes (Spines) which attach to reciprocating processes of adjacent cells by desmosomes.

III. Stratum granulosum: Usually 1-3 cells layers thick, the keratinocytes in these layers are flattened, nucleated cells containing basophilic keratohyalin granules.

IV. Stratum lucidum : Characteristic of only thick skin, this thin layer of consists of flat, eosinophilic , enucleated cells.

V. Stratum corneum : The cells of this layer are filled with keratin and devoid of all organelles. Their plasma membranes are coated with an extra-cellular lipid layer. The thickness of this stratum is variable, being thin in thin skin, thicker in thick skin and hypertrophied in celluses.

1.2.2 Dermis

The dermal-epidermal interface is not planar but marked by elaborate interdigitations between the two layers that strengthen their connection. The projections of the dermis into the epidermis are called dermal papillae and the complementary projections of the epithelium are the rete ridges. On the volar (palmar) and plantar surfaces these interdigitations

are elaborated and organizes to produce the external epidermal ridges (cristae cutis) that provide increased traction (and identification).

The dermis is comprised of two layers: (1) papillary and (2) reticular. The superficial papillary layer is thinner, more cellular and contains small diameter reticular and type I collagen fibers. It is richly vascularized (for thermoregulation) and innervated. The reticular layer lies deep to the papillary layer and is typically thicker and less cellular. It has larger diameter type I collagen and elastic fibers. In the areolae and perineum, this layer contains smooth muscle. Cells of the dermis are typically of collagenous connective tissues: fibroblasts, lymphocytes, macrophages and mast cells.

1.2.3 Hypodermis

The hypodermis is comprised of adipose and loose connective tissues. Its thickness varies with location on the body, sex and nutritional status. It is the principal area of fat storage, providing both energy reserves and insulation. Its cells include adipocytes, fibroblasts, lymphocytes, macrophages, and mast cells. In human a thin layer of skeletal muscle is present in the hypodermis of the face and neck (muscles of facial expression).

1.2.4 Epidermal cells

Four types of cells are found within the epidermis: keratinocytes, Langerhans'cells, melanocytes and sensory epithelial (Merkel) cells. Keratinocytes are the most abundant cells of the epidermis. They are attached to one another by desmosomes and to the basal lamina by hemidesmosomes. Their shape transforms from cuboidal to polygonal to squamous during their apical migration and differentiation. They serve

two major functions: (1) keratin production and (2) formation of the epidermal water barrier.

Keratin is a protein family of some 11+ members. Keratin filaments (a type of intermediate filament) are first synthesized in the stratum basale where they are termed tonofilaments. As the keratinocytes migrate apically, keratin filaments in the cell continue to accumulate. In the strata spinosum and granulosum keratohyalin granules are synthesized. These granules are rich in filaggrin and trichohyalin which when released to the cytoplasm cause the tonofilaments to aggregate into tonofibrila (= keratin fibrils). In the stratum corneum the keratin fibrils (tonofibrils) completely replace the cytoplasmic contents of the cell.

The epidermal water barrier of the epidermis is formed by two components found in the differentiating keratinocytes: (1) the cell envelope and (2) the lipid envelope. The cell envelope consists of a 15 nm thick layer of insoluble protein attached to the inner surface of the plasma membrane; its major protein is loricrin. The lipid envelope is a 5 nm thick layer of lipid attached to the outer surface of the plasma membrane. This lipid layer is produced by exocytosis of lamellar bodies in the keratinocytes in the strata spinosum and granulosum. These lamellar bodies contain glycosphingolipds, phospholipids and ceramides.

The life cycle of a keratinocyte begins by mitosis in the stratum basale. Once contact with the basal lamina is severed the cells are post-mitotic and differentiation begins. As differentiation progresses, cells are pushed towards the surface by basal cell division and growth. Differentiation includes the accumulation of keratin filaments (tonofilaments) and their aggregation into tonofibrils, and the synthesis of lamellar bodies. In the outer layer of the stratum granulosum anoikis begins as nuclei begin to degenerate and organelles are degraded by

lysosomal enzymes. The process is complete in the stratum corneum which consists of completely keratinised “cells “that are continuously desquamated.

Melanocytes are derives form neural cells and account for about 5% of the living cells in the epidermis. Found near the base of the epidermis, they are attached to the basal lamina by hemidesmosomes but to not attach to neighboring keratinocytes. Their dendritic process extend into the stratum spinosum. Melanocytes synthesize melanin and distribute it to the keratinocytes to protect them from the mutagenic effect of ultraviolet (UV) radiation. Melanin comes in two different forms, (1) eumalenin (brown pigment) and pheomelanin (red pigment) and is produces in membrane-bound vesicles called melanosomes by a series of enzymatic reactions. This enzymatic process occurs while the melanosomes move apically thought the dendrites. The melanosomes and their contents are transferred (“pigment donation”) by phagocytosis of the tips of the melanocytes by the keratinocytes.

Langerhans’ cells account for 2-3 % of the living epidermal cells. Unlike melanocytes, these immune system cells can be found through out the epidermis. These are antigen-presenting cells, part of the mononuclear phagocytotic system (MPS), derived from stem cells in the bone marrow, they migrate into the epithelium. Antigens entering through the skin are taken-up and transported via lymph channels to local lymph nodes for presentation to T- cells. These cells are important in contact (allergic) dermatitis and other cell-mediated immune responses in the skin.

Sensory epithelial cells (Merkel cells) are relatively rare (<1% of the living epidermal cells) and are most abundant in areas with acute sensory perception (e.g., finger tips, lips). They are a modified

keratinocyte found in the stratum basale. While containing tonofilaments and desmosomes, these cells also contain small dense granules characteristic of neuroendocrine cells. In combination with lens-shaped sensory neuron endings in the basal lamina, sensory epithelial cells form a mechanoreceptor called a tactile meniscus (Merkel's corpuscle).

1.3 Nail

Nails and their relatives (claws and hooves) are basically localized growths of hard keratin in the epidermis. The nail plate consists of hard keratin and corresponds to the stratum corneum of the epidermis. Beneath the exposed nail plate is the non-proliferative nail bed over which the newly formed nail moves distally. The nail root is the unexposed, proximal end of the nail plate that is surrounded by nail matrix. Within the matrix keratinocytes proliferate, grow, synthnd die to form the nail plate. Note that the growth of nail is perpendicular to the growth of the adjacent epidermis.

The root of the nail is overlain by a fold of skin called the eponychium; distally the hyponychium marks the junction of the skin and the free edge of the nail plate. The moon-shaped light region found at the base of the nail plates (produced by the newly formed immature hard keratin) is called the lunula.

1.3.1 Evolution and development of nail

In higher primates and man, nails have developed in conjunction with the acquisition of manual dexterity; other mammals do not possess such flattened claws. Close inspection of the evolutionary 'ladder' shows that nails have evolved from claws. The lowest evolutionary at which claws

are present is in amphibian (Biedermann, 1926). As will be seen later, in man the intermediate matrix contributes the greatest mass to nail plate, whereas claws are mainly a product of the dorsal matrix (Thorngyke, 1966).

The nail apparatus is formed from an invagination of the primitive epidermis on the dorsum of the terminal phalanges. In this respect it is similar to the hair follicle. This first appears during the 9th week of gestation. By the 13th week the nail bed and nail fold possess a granular layer with keratohyalin granules which disappear when the hard nail plate is formed (Lewis,1954), though the ventral part of the root, which subsequently gives rise to the intermediate nail matrix, never forms a granular layer. By 24 weeks, the free nail plate is visible to the naked eye and at full – term the free edge of the nail plate extends over the hyponychium (Lewis, 1954 and Zaias, 1963). It is presumably this embryological Association with the integumentary epidermis which predisposes the nail apparatus to those diseases which primarily affect the epidermis.

Nail matrix and plate structure the nail plate is formed from the nail matrix. There is still controversy regarding which part of the differentiated epidermis produces the definitive hard nail plate. The fact that he delineated three matrix areas fits the evolutionary facts (Thorngyke, 1966 and Spearman, 1978) and the little cell kinetics work carried out thus for in humans. The sohlenhorn ('solehorn') of Boas (Pinkus,1927), the most distal part of the nail bed, takes no part in the formation of the hard nail plate; however, in view of its importance in producing horny subungual tissue in claws and hooves, it has been suggested as the site of pityriasis rubra pilaris.

The nail matrix epidermis has a distinct basal layer whose cells interdigitate (Roth, 1967). As with the interfollicular epidermis, adjacent basal cells possess desmosomal contact while the cell surfaces in contact with the basement membrane have hemidesmosomes. Differentiation of keratinocytes in the transitional area takes place over several cell layers (Jarrett and Spearna,1966) Despite the short distance from the germinal to the fully keratinised layer, transmission electron microscope studies have shown little cytoplasmic endoplastic reticulum and only small Golgi apparatus. However, differentiating cells in the 'prickle cell' area have more RNA, ribosomes and polysomes than equivalent epidermal cells. No keratohyalin granules are formed during nail matrix keratinisation; in this respect it is similar to hair formation (Breathnach, 1971 and Baden,1970). Microfibrils are manufactured during differentiation having a diameter of approximately 75/~; these become orientated vertically relative to the axis of growth of the hardened nail plate (Baden, 1970) in contents to the longitudinal orientation of the equivalent microfibrils in the hair context. Lysosomal cytoplasmic organelles, the membrane-coating granules, discharge their contents into the intercellular spaces in the transitional zone (Zelikson, 1967). The exact function of this organelle is not known. In the epidermis it is currently thought to play some part in desquamation but evidently this cannot be the function in the nail apparatus in view of the firm adhesion between nail plate cells (Spearman, 1973). Ribosomes containing RNA are still present in transitional cells until the stage of plasma membrane thickening; mitcochondria have become degraded by this stage. Such retention of ribosomes and also nucleoli to a late phase of keratinisation differ from hair development but is similar to feather formation in birds (Cane and Spearman, 1967). In general, cells forming the dorsal, and ventral nail plate lose cytoplasmic and nuclear structures at an earlier stage of keratinisation; nuclear and nucleolar remnants are frequently seen in the mature intermediate nail plate. Since distinct differences are evident

in the structure of the dorsal, intermediate and ventral nail plate (Fig.1.1.) and the corresponding transitional areas it is useful to describe these separately.

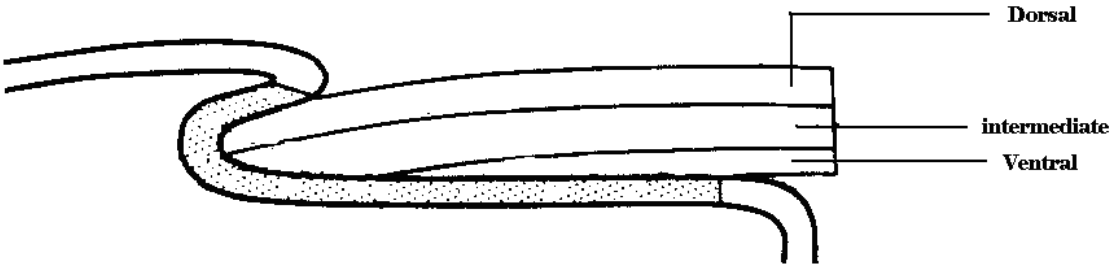

Fig.1.1. Three layers of nail

1. Dorsal nail plate

This stains very poorly with routine stains, such as eosin. Individual cells are very flat and are closely apposed by many gap junctions (Forslind, 1970); the shininess and smoothness of the nail surface is probably related to this characteristic. In the transitional area there is a strong reaction for bound cysteine (SH) and phospholipids are present. Cells about to keratinise take up acid phosphatase stains associated with the presence of nucleotides released from RNA and DNA. The mature dorsal nail plate does not contain these nucleic acids.

2. Intermediate nail plate

A much broader transitional zone is present in this area. Nuclear remnants are retained and stain yellow with thioflavine T. Membrane thickening and disulphide bonding of keratin occur earlier than in the dorsal zone and there is also less bound phospholipids release. Bound cysteine is found in highest concentration in the upper part of the intermediate region. Acid phosphatase activity can be demonstrated in

both the transitional zone and nail plate. In contrast to the dorsal and ventral matrix, non-specific esterase activity is present in the transitional area (and the earlier stage of keratinisation); this is absent from the fully hardened nail. Fully keratinised cells are less flat than those of the dorsal plate and contain many vacuoles. The intermediate plate comprises the main bulk of the nail plate, analogous to the cortex of the hair shaft; unlike the latter, however, transmission electron-microscopic studies reveal irregular staining suggestive of uneven keratinisation and retention of non-keratinised in the fully formed intermediate plate. The non-fibrillary keratin demonstrates a granular structure similar to hair cuticle cells. The darker staining of the nail plate, non-fibrillary keratin compared to the microfibrils is in general similar to that seen in the hair cortex where the matrix protein is relatively rich in sulphur. The fibrillary protein is arranged in a dorso-ventral orientation.

3. Ventral nail plate (from nail bed matrix)

This is the thinnest layer being only one or two cells thick; nuclear remnants are visible in keratinized cells. Bound phospholipids are esterase are absent (the dorsal and intermediate zones), whilst bound cysteine can be detected in the transitional region. The histochemical characteristics of this layer suggest the occurrence of pressure keratinisation (Jarrett, Spearman and Hardy, 1959).

1.3.2 Biochemistry of Nail Plate

Keratin

Jarrett and Spearman (1966) have shown by histochemical methods that cystine, containing stable disulphide bonds, is concentrated particularly in the intermediate nail plate at the periphery of individual cells; the lowest concentration is found in the dorsal plate. The reverse position applies with regards to bound sulphydryl groups, the highest

concentration being present in the dorsal nail plate. Total sulphur concentration is similar in the dorsal and intermediate plates (Forslind, Wroblewski and Afzelius, 1976). Nail keratin analysis has revealed essentially the same fraction present in nail as in hair:

(i) e-fibrillar, low sulphur protein.

(ii) Globular high sulphur matrix protein.

(iii) High glycine-tyrosine matrix protein.

Amino-acid analytical studies on nail keratin (Gillespie and Frenkel, 1974) show higher cystein, glutamic acid, and serine and less tyrosine in nail compared to wool. Most of the cysteine is probably in the intermediate plate (Jarrett and Spearna 1966). Praline and threonine are in greatest concentrations in the high sulphur protein of the keratins; moving boundary electrophorasis, used to compare high sulphur fractions of hair and nail has shown that there are marked differences, suggesting that a different mixture of protein comprises the high sulphur fraction of hair and nail (Gillespie and Inglis, 1965).

Calcium

This is found both as the phosphate in hydroxyapatite crystal in the cytoplasm and bound to phospholipids particularly in the dorsal and ventral nail plates (Pautard, 1964). Using alizarin red staining, Cane and Spearman (1976) showed calcium to be the chief metal in nail. The concentration of calcium is approximately 0.11~ by weight, i.e., ten times greater than in hair (Pautard, 1963). It has been suggested that nail calcium is not part of the intrinsic structure (Forslind, Wroblewski and Afzelius, 1970) but absorbed into the nail from extrinsic sources such as soaps; nail is relatively porous and calcium could enter as ionic calcium or bound to fatty acid. In support of this theory is the finding of significantly grater quantities of calcium in the terminal free edge of ventral plate. Pautard (1963, 1964) believes that calcium is a constituent part of the nail structure; supporting this is the presence of the same

calcium/magnesium ration (4.5:1) in the nail as in blood (Vellar, 1970). Both ideas are probably relevant. The author agrees with Forslind(1970) that calcium content contributes little if anything to the hardness of the nail plate. Phospholipids the nail plate contains significant amounts of phospholipids particularly in the dorsal and ventral layer (Spearman, 1976). Free fats and long chain fatty acids are detectable but like calcium it has been suggested that such constituents are of extrinsic origin.

1.3.3 Structure of nail.

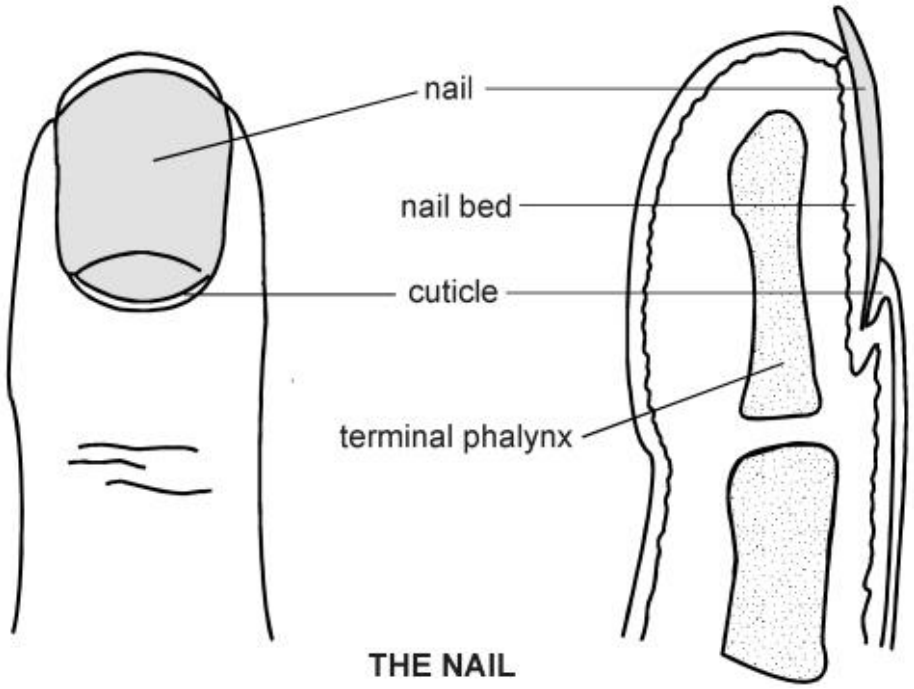

Fig.1.2. Different parts of nail

Human nails are formed by living skin cells in fingers. They consist of primarily of keratins. In skin, hard protein is also found. The different parts of finger nail are shown in Fig.1.2. The upper part of nail which is visible is known as nail plate. The skin below the nail is called as nail bed. Matrix is the region below the cuticle, the unseen area, where growth takes place. Finger nail base, which looks like half moon whitish region, is called lunula. Tissue which holds the nail plate and nail base

rim is known as cuticle. The skin that holds the nail three sides is said to be nail folds. Similar to hair, nail also develops from matrix. The old cells develop and becomes hard their place will occupied by new cells.

1.3.4 Chemical composition of nail

The nail plate is made up of keratins which contain low sulphur packed in an amorphous interfilametous matrix rich in sulphur proteins and also glycine or tyrosine proteins (Baran and Dawber, 1994). Keratins are consists of eith a 7 to 11 nm diameter intermediate filamentous proteins. These proteins are structurally safeguard alpha-helical rod domain present on the side of tissue specific tail domains and non-helical head. Cytoskeleton of epithelial cells is formed by keratins. Keratin filaments activity in the human nail is to give mechanical, stability in order to oppose the physical stress and trauma. These are classified into type-I keratins (acidic protein) and type-II keratin (basic protein) and are stated as heterodimers of type-I and type-II keratins (McGowan et al, 2000). Division of any protein of a keratin may cause the disease phenotypes (Smith et al, 1999). The K9-K20 epithelial, Ha1-Ha5 hair and HRal are acidic type-I keratins. K1-K8 epithelial, Hb1, Hb3, Hb5 hair keratins and Hb6 are type-II keratins. On chromosomes 17 type-I keratin genes are grouped, while on chromosome 12 type-II keratin genes are grouped together (Rogers et al, 1995). In various stages of development various keratin pairs are observed in different epithelial tissues (De Berker et al, 2000). Human nail keratin consists of 80 to 90 percent hard keratins “hair type” and10 to 20 percent soft keratins “epithelial type”. Heid et al (1988) identified that nail material has 8 major Ha1-4 and Hb1-4 trichocytic hard cytokeratins along with two minor cytokeratin polypetides Hbx and Hax. Along this they isolate consideriable amount of K5, K6, K14, K16 and K17 epithelial keratins. In featal nails, epithelial K19 cytokeratin in cells of nail bed and fold are localised. De Berker et al

(2000) found that ventral dorsal nail fold has K1 and K10 on digit pulp. As in the hair follicle, nail bed, sebaceous glands and sweat glands keratin 17 is present in the basal cells of complex epithelia. The base of primary epidermal ridges of palmoplabtar K17 is located (Swensson et al, 1998). K6b is paired with K17. Keratin 6a form pairs with K16. Kba/k16 present in palmoplantar skin (Swensson et al,1998), proximal nail fold, nail bed and digit pulp along with oral mucosa and epidermal appendages (De Berker et al, 2000 and Irvine et al, 1999). As in wound healing or psoriasis K6, K16 and K17 express in hyperproliferative epidermis (Leigh et al, 1995).

Lipids, water and minerals are constituents of human nail. Nail plate contains about 18 % of water. Nail plate contains 0.1 to 1 % of lipid. Sulphur, aluminum, calcium, iron and copper are identified as trace minerals in nail.

1.4 Survey of Literature

Scoble and Litman (1978) found a method of washing of human hair and nail samples to examine by neutron activation and gamma - ray analysis. The amounts of Na, K, Br, Au, Zn and La that are removed by successive washings determine the optimum number of washing for removing these trace elements as surface contaminants. A total solution contact time with the nails is 5 min, and leaching effects are observed after 6 washings.

Samman (1977) focused on the physical or chemical damage of human nails. Chemical damage is rather uncommon but frequent contact with H_2O or soap and H_2O is responsible for much damage to nails.

Greaves and Moll (1976) studied the amino-acid composition of human nail as measured by gas liquid chromatography. A quantitative method for determination of histidine and 16 other amino acids in a nail hydrolysate is described, based on the formation of trifluoroacetyl amino acid methyl esters and GLC analysis with use of 2 columns of mixed stationary phases. Histidine is determined as a triple derivative Na-trifluoroacetyl-Nim-carbethoxy methyl histidinate.

Aantonov et al (1981) studied the presence of potassium and sodium levels in nail plates with flame photometry as a screening test for diagnosis of muco viscidosis and may be recommended as a screening test in diagnosis of mucoviscidosis.

Brans and Shannon (1981) showed finger nail nitrogen content in infants of diabetic mothers and in macrosomic neo nates. Macrosomic infants of nondiabetic mothers had higher mean FNC than their normally grown peers. Normally grown and macrosomic infants of diabetic mothers had higher mean FNC than normally grown infants of diabetic mothers. Fingernail N content (FNC; an indicator of total body protein).

Walters et al (1981) studied the diffusion of water, methanol and ethanol at constant temperature (37.degree. C) was examined over periods up to 4 h. Rates of diffusion across the nail were inversely proportional to nail thickness. Based on methanol data, nail plate barrier property appears stable for long periods of aqueous immersion.

Marshall (1980) made a survey of the proteins from human nail, genetic variation has been observed in both the low-sulfur and high-sulfur protein fractions. 69 % showed a characteristic pattern of 3 major low-sulfur and 5 major high-sulfur proteins. The remaining samples

showed an additional high-sulfur band, and about 1/2 of these samples also manifested an additional major low-sulfur band. In the high-sulfur protein fractions, over 30 proteins were observed in the 2-dimensional electrophoretic patterns and there were at least 3 additional components in the variant fraction.

Katayama et al (1987) studied the concentration of antimony in nail and hair by thermal neutron activation analysis. The concentration of Sb in nail before washing was 730 ppm for the workers, 2.46 ppm for habitants near the refinery, and 0.19 ppm for the control; after washing, it became 230 ppm for the workers, 0.63 ppm for habitants, and 0.09 ppm for the control. The concentration of Sb in hair before and after washing was 222 ppm and 196 ppm for the workers, and 0.21 ppm and 0.15 ppm for the control, respectively.

Acidic and basic hair-nail hard keratins their colocalization in upper cortical and cuticle cells of the human hair follicle and their relationship to soft keratins was studied by Lynch et al (1986). Bakan et.al (1986) studied the glycosylation of nail in diabetic patients. In nondiabetics, the protein glycosylation in nail and glycosylated hemoglobin were found 8.35 .+. 2.70 nmol fructosamine per mg nail and 2.24 .+. 0.45 micromal fructosamine per g Hb, respectively. In diabetics, however, there was an extremely high glycosylation in both nail protein and hemoglobin: 16.00 .+. 7.35 nmol fructosamine per mg nail and 5.17 .+. 1.17 micromol fructosamine per g Hb.

Marshall (1983) studied the Low-sulfur and high-sulfur proteins from human hair and nail were characterized by 2-dimensional polyacrylamide gel electrophoresis.

Walters et al (1985) made studies on physicochemical characterization of the human nail permeation pattern for water and the homologous alcohols and differences with respect to the stratum corneum.

Mercury in hair and nails was determined by Suzuki et al (1989) with speciation of chemical forms of mercury. Total mercury (THg) and inorganic mercury (I Hg) were determined. No sex-related differences were found in mercury levels in hair and nails.

Tazawa - Toshio et al (1997) investigated the fetal development of the nail epithelium, the expression of hard keratin and epidermal keratin in human fetal skin at various stages of gestation by immunofluorescence microscopy using anti-hair keratin and anti-epidermal keratin 10 monoclonal antibodies. In conclusion, the coexpression of hard and epidermal keratins is one biological characteristic of the nail epithelium distinguishable from those of the epidermis and hair apparatus during human fetal development of keratinising epithelia of the skin.

Kitahara et al (1991) investigated nail keratins obtained by sequential extraction with increasing reducing agent concentrations (50 mM and 200 mM 2-mercaptoethanol) and examined each of the extracted keratins by gel electrophoresis followed by immunoblotting. It was found that nail contained epidermal and hair keratins. Results may indicate that nail has unique properties with respect to its keratin compositions. The difference in durabilities of epidermal and hair keratins in nail against 2-mercaptoethanol suggests that two different types of the keratin filaments which are composed of epidermal or hair keratin polypeptides may have differing structural features, despite of co-existence in nail.

Kitahara Takashi et al (1993) investigated the differentiation of nail, this study examined the composition and expression patterns of nail keratin by using monoclonal antibodies specific for keratins characteristic of skin or hair differentiation results indicate that the nail matrix consists of both skin-type and hair-type differentiating cells and, additionally, intermediate keratinocytes that may be progressing the pathways of both skin and hair differentiation.

Hobson Keith et al (1999) studied the influence of drinking water and diet on the stable-hydrogen. Showed that both dietary and drinking water hydrogen are incorporated into non exchangeable hydrogen in both metabolically active (i.e., muscle, liver, blood, fat) and inactive (i.e., feather, nail) tissues. Approximately 20% of hydrogen in metabolically active quail tissues and 26-32% of feathers and nail was derived from drinking water.

Soligo Christophe et al (1999) investigated on Nails and claws in primate evolution. The combined new and old data indicate that the last common ancestor of the extant primates had lost the typical mammalian claws of its ancestors and developed nails on all pedal digits except digit II, which bore a toilet-claw. All nails as well as the toilet-claw originally consisted of two layers. They present a new hypothesis regarding the adaptational significance of these changes.

A perusal of literature reveals that investigations have been done on inorganic composition: organic content: physico chemical properties and characterization of human and animal nail. But systematic studies have not been done on biophysical aspects of human nail. In view of this, in the present investigation, an attempt is made to study physical, electrical and electronic properties. The morphology and inorganic composition

have been studied by employing the techniques of electron microscopy, IR spectroscopy and X-ray diffraction.

REFERENCES

Antonov MP and Petrukhin IS	1981	Laboratornoe-Delo. (9): 532-534
Baden HP	1970	J Invest Dermatol 55:115
Bakan E , Bakan N and Agbas A	1986	DOGA-Bilim-Dergisi-Seri-C-Tip. 10(1): 8-11
Baran R, Dawber R.P.R.	1994	Blackwell Science, Second ed.
Biedermann W	1926	Ergeb Biol 2:1
Brans and Shannon	1981	Biology-of-the-Neonate. 40(5-6): 237-239
Breathnach AS	1971	An atlas of the ultra structure of the human skin.
Cane AK, Spearman RIC	1967	A histochemical study of keratinisation in the domestic fowl.J Zool (Lond) 153:337
De Berker D, Wojnarowska F, Sviland L, Westgate GE, Dawber RPR, Leigh IM.	2000	Br J Dermatol ; 142: 89-96.
Forslind B	1970	Acta Dermatol Venereol (Stockh) 50:161
Forslind B, Wroblewski R, Afzelius BA	1976	J Invest Dermatol 67:273

Gillespie JM, Frenkel MJ	1974	Compar Biochem physiol 47B:339
Gillespie JM, Inglis AS	1965	Compar Biochem Physiol 15:175
Greaves MS	1976	Clinical-Chemistry. 22(10): 1608-1613
Heid HW, Moll I, Franke WW.	1988	Differentiation; 37: 215-30.
Hobson, Keith A , Atwell,-Lisa and Wassenaar, Leonard I.	1999	Proceedings-of-the-National-Academy-of-Sciences-of-the-United-States-of-America. 96(14): 8003-8006
Irvine AD, Mclean WHI.	1999	Br J Dermatol ; 140: 815-28.
Jarrett A, Spearman RIC, Hardy JA	1959	Br Dermatol 71:277
Jarrett A, Spearna RIG	1966	Arch Dermatol 94:652
Katayama Y and Ishida-N	1987	Radioisotopes-. 36(3): 103-107
Kitahara, Takashi and Ogawa,-Hideoki.	1993	Journal-of-Investigative-Dermatology. 100(2): 171-175
Kitahara T and Ogawa H	1991	Journal-of-Dermatological-Science. 2(6): 402-406

Leigh IM, Navsaria H, Purkis PE, McKay IA, Bowden PE, Riddle PN.	1995	Br J Dermatol ; 133: 501-11.
Lewis BL	1954	Arch Dermatol 70:732
Lynch MH, O guin WM, Hardy C, Mak L and Sun T T	1986	Journal-of-Cell-Biology. 103(6 PART 2): 2593-2606
Marshall RC	1980	Journal-of-Investigative-Dermatology. 75(3): 264-269
Marshall RC	1983	Journal-of-Investigative-Dermatology. 80(6): 519-524
McGowan KM, Coulombe PA.	2000	J Invest Dermatol114: 1101-7.
Pautard FGE	1963	Nature (Lond) 199:561
Pautard FGE	1964	Rook A, Champion Rh (eds) Progress in the biological sciences in relation to dermatology, vol 2. P 227
Rogers MA, Nischt R, Korge B, Krieg T, Fink TM, Lichter P, Winter H, Schweizer J	1995	Exp Cell Res ; 220: 357-62.
Roth SI	1967	In Zelickson AS (ed) p 105
Samman PD	1977	Journal-of-the-Society-of-Cosmetic-Chemists. 28(6): 351-356

Scoble H and Litman	1978	Analytical-Letters. 11(2): 183-190
Smith FJD, Del Monaco M, Steijlen PM, Munro CS, Morvay M, Coleman CM, Rietveld FJR, Uitto J, Mclean WHI.	1999	Br J Dermatol 1999; 141
Soligo Christophe and Muller, Alexandra E	1999	Journal-of-Human-Evolution. 1999; 36(1): 97-114
Spearman RIC	1973	A text book of skin biology
Spearman RIC	1976	Comparative biology of skin.Symposium of the Zoological Soc. p 356
Spearman RIG	1978	Jarrett A (ed) vol 5, chapt. 59.
Suzuki T , Watanabe S and Matsuo N	1989	Japanese-Journal-of-Industrial-Health. 31(4): 235-238
Swensson O, Langbein L, McMillan JR, Stevens HP, Leigh IM, Mclean WHI, Lane EB, Eady RAJ.	1998	Br J Dermatol 139: 767-75.
Tazawa, Toshio, Fujiwara, Hiroshi , Ito, Kaoru, Ito, Masaaki and Iwanaga, Toshihiko.	1997	Acta-Medica-et-Biologica. 45(1): 27-32

Thorngyke EE	1966	am J Phys Anthropol NS 28:247
Vellar OD	1970	Am J Clin Nutrit 23:1271
Walters KA , Flynn GL and Marvel JR	1981	Journal-of-Investigative-Dermatology. 76(2): 76-79
Walters KA , Flynn GL and Marvel JR	1985	Journal-of-Pharmacy-and-Pharmacology. 1985; 37(11): 771-775
Zaias N	1963	Arch Dermatol 87:37
Zelikson As	1967	Ultrastructure of normal and ubnormal skin.