2 REVIEW OF LITERATURE

Introduction

Friction ridge skin impressions were used as proof of a person’s identity in China perhaps as early as 300 B.C., in Japan as early as A.D. 702, and in the United States since 1902.

2.1 Ancient History

Earthenware estimated to be 6000 years old was discovered at an archaeological site in northwest China and found to bear clearly discernible friction ridge impressions. These prints are considered the oldest friction ridge skin impressions found to date; however, it is unknown whether they were deposited by accident or with specific intent, so as to create decorative patterns or symbols (Xiang-Xin and Chun-Ge, 1988). In this same Neolithic period, friction ridges were in other ancient materials by builders (Ashbaugh, 1999). Just as someone today might leave impressions in cement, early builders left impressions in the clay used to make bricks (Berry and Stoney, 2001).

221 B.C. to A.D. 1637

The Chinese culture was the first known to have used friction ridge impressions as a means of identification. The earliest example comes from a Chinese document entitled “The Volume of Crime Scene Investigation-Burglary”, from the Qin Dynasty (221 to 206 B.C.). The document contains a description of how handprints were used as a type of evidence (Xiang-Xin and Chun-Ge 1988).
After the invention of paper by the Chinese in A.D. 105, it became common to sign documents using friction ridge skin. It was a standard practice in China to place an impression either palmprints, phalangeal (lower finger joint) marks, or fingerprints on all contract-type and possibly other nations prior to European discovery. (Xiang-Xin and Chun-Ge 1988).

It is said that the use of prints on important documents was adopted from the Chinese, where it was used generally, but in India it was mainly reserved for royalty (Sodhi and Kaur 2003). The use of friction ridge skin as a signature in China, Japan, India and possibly other nations prior to European discovery is thus well documented.

17th and 18th Centuries

In the late 17th century, European scientists began publishing their observations of human skin. Friction ridge skin was first described in detail by Dr. Nehemiah Grew in the 1684 paper Philosophical Transactions of the Royal Society of London. Dr. Grew’s description marked the beginning in the Western Hemisphere of friction ridge skin observations and characterizations (Lambourne 1984, Ashbaugh 1999). In 1685, Govard Bidloo, a Dutch anatomist, published Anatomy of the Human Body, which included details of the skin and the papillary ridges of the thumb but failed to address individualization or permanence (Felsher 1962, Ashbaugh 1999). In 1687, the Italian physiologist Marcello Malpighi published concerning the External Tactile Organs, in which the function, form, and structure of friction ridge skin was discussed. Malpighi is credited with being the first to use the newly invented microscope for medical studies. Although friction ridge skin had been studied for a number of years, it would
be 1788 before the uniqueness of this skin was recognized in Europe. Mayer was the first to write that friction ridge skin is unique.

19th Century

In his 1823 thesis titled “Commentary on the Physiological Examination of the Organs of Vision and the Cutaneous System”, Dr. Johannes E. Purkinje (1787–1869), professor at the University of Breslau in Germany, classified fingerprint patterns into nine categories and gave each a name (Galton 1892, Lambourne 1984). Although Dr. Purkinje went no further than naming the patterns, his contribution is significant because his nine pattern types were the precursor to the Henry classification system (Galton 1892, Herschel 1916).

German anthropologist Hermann Welcker (1822–1898) of the University of Halle led the way in the study of friction ridge skin permanence. Welcker began by printing his own right hand in 1856 and then again in 1897, thus gaining credit as the first person to start a permanence study.

Generally, the credit for being the first person to study the persistence of friction ridge skin goes to Sir William James Herschel (Faulds 1880). Faulds was the first person to publish in a journal the value of friction ridge skin for individualization, especially its use as evidence. Kollman was the first to identify the presence and locations of the volar pads on the hands and feet (Hale 1952, Ashbaugh 1999). As the author of the first book on fingerprints (Finger Prints 1892), Galton established that friction ridge skin was unique and persistent. Because Galton was the first to define and name specific print minutiae, the minutiae became known as
Galton details. Galton’s (bifurcation), the end or beginning of a ridge (ending ridges), a short island (short ridge), and an enclosure (two bifurcations facing each other) (Galton 1892). Vucetich, having studied Galton’s research, began to experiment with fingerprints in 1891. He started recording the fingerprints of criminals and devised his own classification system (Lambourne 1984). Vucetich’s classification system and individualization of prisoners through the use of fingerprints were the first practical uses of the fingerprint science by law enforcement personnel. Wilder was the first to suggest that the centers of disturbance of primate friction ridge formations actually represented the locations of the volar pads. He also developed the hypothesis of a relationship between primate friction ridge patterns and volar pads.

20th Century

Claughry (1904) began fingerprinting all inmates at the Leavenworth, KS, federal prison. These fingerprint records became the beginning of the U.S. Government’s fingerprint collection (Wilder and Wentworth 1918, Myers 1938). This was the first scientific research supporting third level detail as permanent and unique. Several years later, Dr. Harold Cummins (1893–1976) of Tulane University in New Orleans, LA, conducted a great deal of research on friction ridge skin. By examining fetuses in various stages of growth and health, Cummins made many contributions to the modern understanding of friction ridge skin. Cummins’s book *Fingerprints, Palms, and Soles - An Introduction to Dermatoglyphics* (Cummins and Midlo 1943) describes the formation and development of volar pads on the human fetus. Cummins notes that volar pad regression takes place almost
concurrently with the beginning of friction ridge development; that the size, location, growth, and configuration of the volar pad affects the friction ridge patterns; and that disease or birth defects have an effect on the growth of volar pads (Cummins and Midlo 1943).

In 1952, Dr. Alfred R. Hale, also of Tulane University, published a thesis titled “Morphogenesis of the Volar Skin in the Human Fetus”. By studying cross sections of fetal skin, Hale was able to describe the formation of friction ridges during fetal development and the differential growth of friction ridges, which is the major premise of friction ridge identification (Ashbaugh 1999).

Salil Kumar Chatterjee (1905–1988) of Calcutta, India, published the book *Finger, Palm, and Sole Prints* in 1953, but Chatterjee is best known for his 1962 article “Edgeoscopy” (Chatterjee, 1962), in which he described his theory of using specific ridge-edge shapes to supplement fingerprint individualization. He defined ridge shapes including straight, convex, peak, table, pocket, concave, and angle. Chatterjee believed that these edge shapes could be used to assist in making individualizations (Ashbaugh, 1999). In 1976, Dr. Michio Okajima of Japan published the paper “Dermal and Epidermal Structures of the Volar Skin”. The main contribution from his work is the study of incipient ridges, which appear as smaller ridges in friction ridge impressions (Ashbaugh 1999).

Dr. William Babler of Marquette University in Milwaukee, WI, published “Embryological Development of Epidermal Ridges and Their Configurations” in 1991. That paper reviewed prior work by other scientists and the research Babler performed relative to the “prenatal relationship between epidermal ridge dimension and bone dimension of the hand” (Babler, 1991).

2.2 Embryology & Development: Introduction to Embryology

The uniqueness of friction ridge skin comes under the larger umbrella of biological individualities. In any living organism no two portions are exactly alike. The intrinsic and extrinsic factors that affect the development of any individual organ, such as human skin, are impossible to duplicate, even in very small areas. The uniqueness of skin can be traced back to the late embryological and early fetal development periods.

Early Embryological Development: 0–2 Weeks EGA (Raven and Johnson, 1992)

Embryological development begins with fertilization and continues through a period of rapid cell division called “cleavage”. In mammalian eggs, an inner cell mass is concentrated at one pole, causing patterned alterations during cleavage. Although egg cells contain many different substances that act as genetic signals during early embryological development, these substances are not distributed uniformly. Instead, different substances tend to be clustered at specific sites within the growing embryo. During growth, signal substances are partitioned into different daughter cells, endowing them with distinct developmental instructions. In
this manner, the embryo is pre-patterned to continue developing with unique cell orientation.

**Late Embryological Development: 3–8 Weeks EGA (Raven and Johnson, 1992)**

The first visible results of pre-patterning can be observed immediately after completion of the cleavage divisions as different genes are activated. Certain groups of cells move inward toward the center of the sphere in a carefully orchestrated migration called “gastrulation”. This process forms the primary tissue distinctions between ectoderm, endoderm and mesoderm. The ectoderm will go on to form epidermis, including friction ridge skin; the mesoderm will form the connective tissue of the dermis, as well as muscle and elements of the vascular system and the endoderm goes on to form the organs.

Once specialized, the three primary cell types begin their development into tissue and organs. The process of tissue differentiation begins with neurulation, or the formation of the notochord (the precursor to the spinal cord and brain) as well as the neural crest (the precursor to much of the embryo’s nervous system). Segmented blocks of tissue that become muscles, vertebrae, and connective tissue form on either side of the notochord. The remainder of the mesoderm moves out and around the inner endoderm, forming a hollow chamber that will ultimately become the lining of the stomach and intestines.

During late embryological development, the embryo undergoes “morphogenesis”, or the formation of shape. Limbs rapidly develop from about 4 weeks EGA, and the arms, legs, knees, elbows, fingers, and toes
can all be seen in the second month. During this time, the hand changes from a paddlelike form to an adult form, including the formation of the fingers and rotation of the thumb. Also during this time, swellings of mesenchyme called "volar pads" appear on the palms of the hands and soles of the feet. Within the body cavity, the major organs such as the liver, pancreas, and gall bladder become visible. By the end of week 8, the embryo has grown to about 25 millimeters in length and weighs about 1 gram.

**Fetal Growth: 9–12 Weeks EGA (Embryonic growth activity)**

During the third month, the embryo’s nervous system and sense organs develop, and the arms and legs begin to move. Primitive reflexes such as sucking are noticed and early facial expressions can be visualized. Friction ridges begin to form at about 10.5 weeks EGA and continue to mature in depth as the embryo passes into the second trimester. From this point on, the development of the embryo is essentially complete, and further maturation is referred to as fetal growth rather than embryonic development.

**Second Trimester**

The second trimester is marked by significant growth to 175 millimeters and about 225 grams. Bone growth is very active and the body becomes covered with fine hair called lanugo, which will be lost later in development. As the placenta reaches full development, it secretes numerous hormones essential to support fetal bone growth and energy. Volar pads regress and friction ridges grow until about 16 weeks EGA, when the minutiae become set.
Sweat glands mature, and the epidermal–dermal ridge system continues to mature and grow in size. By the end of the second trimester, sweat ducts and pores appear along epidermal ridges, and the fetus begins to undergo even more rapid growth.

**Third Trimester**

In the third trimester, the fetus doubles in weight several times. Fueled by the mother's bloodstream, new brain cells and nerve tracts actively form. Neurological growth continues long after birth, but most of the essential development has already taken place in the first and second trimesters. The third trimester is mainly a period for protected growth.

**Limb and Hand Development**

During the initial phases of formation, the hand undergoes significant changes in topography. Until approximately 5–6 weeks EGA, the hand appears as a flat, paddlelike structure with small protrusions of tissue that will become fingers. From 6 to 7 weeks EGA, these finger protrusions in the hand plate begin to form muscle and cartilage that will become bone at later stages of hand growth.

From 7 to 8 weeks EGA, the fingers begin to separate and the bone begins to “ossify” or harden. By 8 weeks EGA, the joints begin to form between the bones of the hand, and the external hand morphology appears similar in proportion to that of an infant.

**Volar Pad Development**

Volar pads are transient swellings of tissue called mesenchyme under the epidermis on the palmar surface of the hands and soles of the feet of the human fetus. The interdigital pads appear first, around 6 weeks
EGA, followed closely in time by the thenar and hypothenar pads. At approximately 7–8 weeks EGA, the volar pads begin to develop on the fingertips, starting with the thumb and progressing toward the little finger in the same radio-ulnar gradient that ridge formation will follow. Also at about 8 weeks EGA, the thenar crease begins to form in the palm, followed by the flexion creases in the fingers at around 9 weeks EGA (Kimura 1991).

**Volar Pad “Regression”**

The pads remain well rounded during their rapid growth around 9–10 weeks EGA, after which they begin to demonstrate some individual variation in both shape and position (Cummins 1926, 1929, Burdi et al 1979, Babler 1987). During the period from 8 to 10 weeks EGA, thumb rotation is achieved (Lacroix et al, 1984). Also at about 10 weeks EGA, the flexion creases of the toes begin formation, followed at about 11 weeks EGA by the distal transverse flexion crease in the palm, and at about 13 weeks EGA by the proximal transverse flexion crease in the palm (Kimura 1991).

As a result of the volar pads slowing growth, their contour becomes progressively less distinct on the more rapidly growing surface. This process has been defined as “regression” (Lacroix et al 1984) but it is important to understand that the pad is not actually shrinking; rather, the volar pads are overtaken by the faster growth of the larger surrounding surface. The volar pads of the palm begin to regress as early as 11 weeks EGA, followed closely by the volar pads of the fingers. By 16 weeks EGA, volar pads have completely merged with the contours of the fingers, palms, and soles of the feet (Cummins 1929).
Differentiation of the Friction Ridge Skin

Development of the Epidermis

The primitive epidermis is formed at approximately 1 week EGA, when ectoderm and endoderm are separately defined. A second layer of epidermis is formed at about 4–5 weeks EGA. The outermost layer is the periderm. The middle layer, which is the actual epidermis, is composed of basal keratinocytes (named because of the keratins these cells manufacture). At about 8 weeks EGA, the basal cells between the epidermis and the dermis begin to divide consistently and give rise to daughter cells that move vertically to form the first of the intermediate cell layers (Holbrook 1991b). At this point, the embryonic epidermis is three to four cell layers thick, but it is still smooth on its outer and inner surfaces. Keratinocytes are tightly bound to each other by desmosomes, and the cells of the basal layer are attached to the basement membrane by hemidesmosomes (Holbrook 1991a).

Development of the Dermis

Fibroblasts are the first dermal components to originate from the mesoderm. These cells with irregular branching secrete proteins into the matrix between cells. Fibroblasts synthesize the structural (collagen and elastic) components that form the connective tissue matrix of the dermis. During the period 4–8 weeks EGA, many of the dermal structures begin formation. Elastic fibers first appear around 5 weeks EGA at the ultrastructural level in small bundles of 20 or fewer fibrils (Holbrook, 1991b). Nerve development occurs in different stages from 6 weeks EGA onwards. Neurovascular bundles and axons with growth cones are seen in the
developing dermis as early as 6 weeks EGA (Moore and Munger 1989). In fact, axons can be traced to the superficial levels of the dermis, and in some cases they almost about the basal lamina of the epidermis. By 9 weeks EGA, innervation (the appearance of nerve endings) of the epidermis has begun to occur, although there are some Merkel cells in the epidermis that are not yet associated with axons. In embryos older than 10 weeks EGA, Merkel cells are predominant in the developing epidermis, and their related axons and neurofilaments are present in the dermis (Smith and Holbrook 1986, Moore and Munger 1989).

The dermis becomes distinguishable from deeper subcutaneous tissue due largely to a horizontal network of developing blood vessels. From 8 to 12 weeks EGA, vessels organize from dermal mesenchyme and bring much-needed oxygen and hormones to the underside of the developing epidermis. Unlike other epidermal structures, blood vessels continue to alter with aging, as some capillary loops are lost and new ones arise from the interpapillary network. This continues into late adulthood (Smith and Holbrook 1986).

A second vascular network forms deep in the reticular dermis by about 12 weeks EGA. Unlike the developing primary ridges, the vascular network is not a permanent structure. There is significant reorganization of capillary beds during the period 8–20 weeks EGA to keep pace with skin growth; even after birth, microcirculation continues to form and re-model (Smith and Holbrook 1986, Holbrook 1991).
Primary Ridge Formation

Initiation of Primary Ridge Formation

At around 10–10.5 weeks EGA, basal cells of the epidermis divide rapidly (Holbrook and Odland 1975, Babler 1991). Shallow “ledges” (Hale 1952) can be seen on the bottom of the epidermis as the volar epidermal cells divide. These ledges delineate the overall patterns that will become permanently established on the volar surfaces several weeks later (Evatt 1906, Babler 1991). Primary ridges are the first visual evidence of interaction between the dermis and epidermis and are first seen forming as continuous ridges.

The prevailing theory of events before the visualization of primary ridge structure involves centers of active cell proliferation, which will become the centers of sweat gland development (Babler1991).

As per this theory, the “units” of rapidly multiplying cells increase in diameter, somewhat randomly, growing into one another along lines of relief perpendicular to the direction of compression.

As the series of localized proliferations “fuse” together, the resulting linear ridges of rapidly dividing epidermal cells fold into the dermis, creating the first visible ridge structure at the epidermal–dermal junction (Ashbaugh1999). Another plausible theory is that developing nerves may interact with epidermal cells to stimulate clustered interactions that blend together in the early stages of ridge development.

At the time of embryonic friction ridge formation, the central nervous and cardiovascular systems are undergoing a critical period of development (Hirsch 1964). Researchers have reported innervation at the
sites of ridge formation immediately preceding the appearance of friction ridges and suggest that innervation could be the trigger mechanism for the onset of proliferation (Bonnevie 1924, Dell and Munger 1986, Moore and Munger 1989). Several researchers even postulate that the patterning of the capillary nerve pairs at the junction of the epidermis and the dermis is the direct cause of primary ridge alignment (Hirsch and Schweichel 1973, Dell and Munger 1986, Moore and Munger 1989, Morohunfola et al 1992).

Early research on pattern distribution established “developmental fields” or groupings of fingers on which patterns had a greater tendency to be similar. (Siervogel et al 1978, Meier 1981, Roberts 1982). Later discoveries confirmed the neurological relation of spinal cord sections C–6, C–7, and C–8 to innervation of the fingers (Heimer 1995). Specifically, Kahn and colleagues (2001) reported that a large ridge-count difference between C–8 controlled fingers 4 and 5 may predict a larger waist-to-thigh ratio and, therefore, an increased risk of some major chronic diseases such as heart disease, cancer, and diabetes. Other interesting hypotheses have been published regarding the connection between innervation and friction ridge patterning, but the main consideration for the purposes of friction ridge formation is that specific parts of the nervous system are undergoing development at the same time that ridges begin to appear on the surface of the hands.

The presence of nerves and capillaries in the dermis before friction ridge formation may be necessary for friction ridge proliferation. It would seem that complex simultaneous productions such as friction ridge formation would benefit from being in communication with the central
nervous system or the endocrine and exocrine (hormone) systems (Smith and Holbrook 1986). However, it is doubtful that nerves or capillaries independently establish a map that directly determines the flow of the developing friction ridges. It seems more likely that the alignment of the nerves and capillaries is directed by the same stresses and strains on the developing hand that establish ridge alignment (Smith and Holbrook 1986, Babler 1999). It is well recognized in cell biology that physical pressure on a cellular system can trigger electrochemical changes within that system. Merkel cells occupy the epidermis just prior to innervation along those pathways (Holbrook 1991), suggesting that even before ridge formation, the stresses created by the different growth rates of the dermis and epidermis are causing differential cell growth along invisible lines that already delineate pattern characteristics (Loesch 1973). Regardless of the trigger mechanism controlling the onset of the first primary ridge proliferations, the propagation of primary ridges rapidly continues.

**Propagation of Primary Ridge Formation**

Primary ridges mature and extend deeper into the dermis for a period of approximately 5.5 weeks, from their inception at 10.5 weeks EGA until about 16 weeks EGA. The cell growth during this phase of development is along the primary ridge, in what has been labeled the “proliferative compartment”. The proliferative compartment encompasses basal and some suprabasal cells, ultimately governed by stem cells, and is responsible for new skin cell production of the basal layer of skin (Lavker and Sun 1983).
Minutiae Formation

Although the exact mechanisms for formation of minutiae are unclear, the separate accounts of many researchers who have examined fetal tissue allow for a fairly accurate reconstruction of the morphogenesis of friction ridges in successive stages of the development process.

Many events happen during this rapid period of primary ridge growth. The finger rapidly expands, new primary ridges form across the finger, and the existing primary ridges begin to separate because of growth of the digit. As existing ridges separate, the tendency of the surface to be continually ridged creates a demand for new ridges. Hale reports that new ridges pull away from existing primary ridges to fill in these gaps, creating bifurcations by mechanical separation. Ending ridges form when a developing ridge becomes sandwiched between two established ridges. According to this theory, “fusion between adjacent ridges (which have already formed) seems improbable, although there is no evidence for or against this process” (Hale 1952).

Other models explain ridge detail in nature as a chemical reaction-suppression scheme in which morphogens react and diffuse through cells, causing spatial patterns (Murray 1988). According to these models, hormones circulate first through newly formed capillaries just before ridge formation in the epidermis, offering another potential factor in the genesis of ridge formation (Smith and Holbrook 1986). Secondary ridges continue to mature from 16 to 24 weeks EGA, this structure is progressively mirrored on the surface of friction ridge skin as the furrows (Burdi et al 1979).
Formation of Dermal Papillae

Dermal papillae are the remnants of dermis left projecting upward into the epidermis when anastomoses bridge primary and secondary ridges. They begin to form at approximately 23 weeks EGA (Okajima, 1975) and continue to become more complex throughout fetal formation and even into adulthood (Chacko and Vaidya 1968, Misumi and Akiyoshi 1984).

Pattern Formation

Shape of the Volar Pad

It is observed throughout the physical world that ridges tend to align perpendicularly to physical compression across a surface. Ridges also form transversely to the lines of growth stress in friction skin. The predominant growth of the hand is longitudinal (lengthwise) and ridges typically cover the volar surface transversely (side to side). This phenomenon is seen in the ridge flow across the phalanges.

Bonnevie first hypothesized in 1924 that volar pad height affects friction ridge patterns (Bonnevie 1924). Disruptions in the shape of the volar surfaces of the hands and feet create stresses in directions other than longitudinal. The ridges flow in a complex manner across these three-dimensional structures.

The distinction between the size, height, and shape of the volar pad, and the effects of differences in each of these elements on a friction ridge pattern, is a difficult topic to study (Jamison 1990, Chakraborty 1991, Mavalwala et al 1991). However, almost all research points to the conclusion that the shape of the volar pad influences the stress across the skin that directs ridge alignment. One contrary viewpoint to this conclusion
exists. In 1980, Andre Wilde proposed a theory that pattern formation is directed much earlier in fetal life, before volar pads form, while the hand is still in a paddlelike shape (De Wilde 1980). Degree of asymmetry will be reflected in the ridge flow of the resulting pattern. This biological process cannot be thought of as limited to the extremes of volar pad regression, occurring either completely symmetrically or asymmetrically (leaning all the way to one side). In fact, there is a continuum involved from whorl patterns to loop patterns.

Subtle variations in the symmetry of a volar pad could affect the formation of a whorl pattern versus a central pocket loop whorl pattern, or a central pocket loop whorl pattern versus a loop pattern. Any one of the numerous genetic or environmental factors present during the critical stage could cause a slight deviation in the normal developmental symmetry of the volar pad and, therefore, affect the resulting pattern type.

**Size of the Volar Pad Pattern Size.**

The size, particularly the height, of the volar pad during primary ridge formation affects the ridge count from the core to the delta of normal friction ridge patterns (Bonnevie 1924, Mulvihill and Smith 1969, Siervogel et al 1978). Researchers have observed that ridges that form on high, pronounced volar pads conform to the surface as high-count whorl patterns. Conversely, ridges that form on a finger with a low or absent volar pad create low-count or arch-type patterns (Babler 1987). Holt (1968) reported that the total finger ridge count (TFRC) of all 10 fingers, taken by adding the ridge counts from the core to the delta in loops, or the core toward the radial delta in whorls, is the most inheritable feature in dermato-
glyphics. This combined information points directly to the conclusion that timing events related to volar pad and friction ridge formation affect friction ridge patterns.

**Timing Events:** The ridge count of a friction ridge pattern is related to two different events: the timing of the onset of volar pad regression and the timing of the onset of primary ridge formation. Differences in the timing of either event will affect the ridge count of that particular pattern. For example, early onset of volar pad regression would lead to a volar pad that was in a more regressed state at the time of the onset of primary ridge formation, and a relatively low-ridge-count pattern (or arch) would likely result. Conversely, overall late onset of volar pad regression would mean that the pad was still relatively large and hypothesized that ridges direct the size and shape of the volar pads. However, no other theoretical or empirical support for this theory could be found. All other research indicates that friction ridges align according to volar pad shape and symmetry at approximately 10.5 weeks EGA.

**Symmetrical Volar Pad:** The growth and regression of the volar pads produce variable physical stresses across the volar surface that affects the alignment of the ridges as the ridges first begin to form. Whether ridge flow will conform to a whorl or a loop pattern appears highly correlated with the symmetry of the stress across the surface of the finger. If the volar pad and other elements of finger growth are symmetrical during the onset of primary ridge formation, then a symmetrical pattern (a whorl or an arch) will result. Ridges will form concentrically around the apex of a volar pad that is high and round when the generating layer of friction ridge skin first begins to
rapidly produce skin cells. The ridge flow from a symmetrical volar pad conforms to the navigational pattern of the loxodrome (Mulvihill and Smith 1969, Elie 1987). Research in both the medical and mathematical fields suggests that this same physical model applies across the entire volar surface of the hands and feet (Cummins 1926, 1929, Loesch 1973, Penrose and O’Hara 1973).

**Asymmetrical Volar Pad:** The degree of asymmetry of the finger volar pad when ridges first begin to form determines the asymmetry of the pattern type. Many researchers have reported that asymmetrical “leaning” pads form looping patterns and that low or absent volar pads form arch patterns (Cummins 1926). Babler perhaps conducted the most scientific validation of the correlation between pad symmetry and pattern type through extensive examination of fetal abortuses (Babler 1978).

Cummins published an extensive analysis of malformed hands to demonstrate the effect of the growth and topology of the hand on ridge direction (Cummins 1926). Cummins also concluded that ridge direction is established by the contours of the hands and feet at the time of ridge formation. Penrose examined friction ridge pattern formation from a mathematical perspective, arriving at the same conclusion (Penrose and Plomley 1969, Loesch 1973). More recently, Kücken and Newell (2005) modeled stress fields across bounded three-dimensional, spherical virtual surfaces, creating relatively accurate-appearing ridge patterns.

If the volar pad and other growth factors of the finger are asymmetrical during the critical stage, then that same a recent model of the process of friction ridge morphogenesis has been likened to mechanical
instability (Kücken and Newell 2005). Building on the folding hypothesis of Kollmann (1883) and Bonnevie (1924), Kucken and Newell (2005) consider the basal layer as “an overdamped elastic sheet trapped between the neighboring tissues of the intermediate epidermis layer and the dermis”, which they mathematically model as “beds of weakly nonlinear springs”.

Their computer program models the results of forcing enough compressive stress to cause a buckling instability on a virtual three-dimensional elastic sheet constrained by fixed boundaries on two sides. The resulting ridge patterns are similar to all three major fingerprint pattern types oriented by the upper fixed boundary of the nailbed and the lower fixed boundary of the distal interphalangeal flexion crease.

Regardless of the exact mechanism of minutiae formation (mechanical or static; fusion or chemical), the exact location of any particular bifurcation or ridge ending within the developing ridge field is governed by a random series of infinitely interdependent forces acting across that particular area of skin at that critical moment. Slight differences in the mechanical stress, physiological environment, or variation in the timing of development could significantly affect the location of minutiae in that area of skin.

Secondary Ridge Formation Initiation of Secondary Ridge Formation

By 15 weeks EGA, the primary ridges are experiencing growth in two directions: the downward penetration of the sweat glands and the upward push of new cell growth. Generally, the entire volar surface is ridged by 15 weeks EGA. Okajima (1982) shows a fully ridged palm of a 14-week-old fetus.
Between 15 and 17 weeks EGA, secondary ridges appear between the primary ridges on the underside of the epidermis (Babler 1991). Secondary ridges are also cell proliferations resulting in downfolds of the basal epidermis. At this time in fetal development, the randomly located minutiae within the friction ridge pattern become permanently set (Hale 1952), marking the end of new primary ridge formation (Babler 1990).

**Propagation of Secondary Ridge Formation**

As the secondary ridges form downward and increase the surface area of attachment to the dermis, the primary ridges are pushing cells toward the surface to keep pace with the growing hand. These two forces, in addition to cell adhesion, cause in folding of the epidermal layers above the attachment site of the secondary ridges (Hale 1952). As secondary ridges continue to mature from 16 to 24 weeks EGA, this structure is progressively mirrored on the surface of friction ridge skin as the furrows (Burdi et al 1979)

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**Symmetrical Volar Pad:** The growth and regression of the volar pads produce variable physical stresses across the volar surface that affect the alignment of the ridges as the ridges first begin to form. Whether ridge flow will conform to a whorl or a loop pattern appears highly correlated with the symmetry of the stress across the surface of the finger. If the volar pad and other elements of finger growth are symmetrical during the onset of primary ridge formation, then a symmetrical pattern (a whorl or an arch) will result. Ridges will form concentrically around the apex of a volar pad that is high and round when the generating layer of friction ridge skin first begins to rapidly produce skin cells. The ridge flow from a symmetrical volar pad conforms to the navigational pattern of the loxodrome (Mulvihill and Smith 1969, Elie 1987). Research in both the medical and mathematical fields suggests that this same physical model applies across the entire volar surface of the hands and feet (Cummins 1926, 1929, Loesch 1973, Penrose and O’Hara 1973).

**Asymmetrical Volar Pad:** The degree of asymmetry of the finger volar pad when ridges first begin to form determines the asymmetry of the pattern type. Many researchers have reported that asymmetrical “leaning” pads form looping patterns and that low or absent volar pads form arch patterns (Cummins, 1926). Babler perhaps conducted the most scientific validation of the correlation between pad symmetry and pattern type through extensive examination of fetal abortuses (Babler 1978).
Cummins published an extensive analysis of malformed hands to demonstrate the effect of the growth and topology of the hand on ridge direction (Cummins 1926). Cummins also concluded that ridge direction is established by the contours of the hands and feet at the time of ridge formation. Penrose examined friction ridge pattern formation from a mathematical perspective, arriving at the same conclusion (Penrose and Plomley 1969, Loesch 1973). More recently, Kücken and Newell (2005) modeled stress fields across bounded three-dimensional, spherical virtual surfaces, creating relatively accurate-appearing ridge patterns.

If the volar pad and other growth factors of the finger are asymmetrical during the critical stage, then that same degree of asymmetry will be reflected in the ridge flow of the resulting pattern. This biological process cannot be thought of as limited to the extremes of volar pad regression, occurring either completely symmetrically or asymmetrically (leaning all the way to one side). In fact, there is a continuum involved from whorl patterns to loop patterns.

Subtle variations in the symmetry of a volar pad could affect the formation of a whorl pattern versus a central pocket loop whorl pattern, or a central pocket loop whorl pattern versus a loop pattern. Any one of the numerous genetic or environmental factors present during the critical stage could cause a slight deviation in the normal developmental symmetry of the volar pad and, therefore, affect the resulting pattern type.

**Size of the Volar Pad Pattern Size:** The size, particularly the height, of the volar pad during primary ridge formation affects the ridge count from the core to the delta of normal friction ridge patterns (Bonnevie 1924, Mulvihill
Researchers have observed that ridges that form on high, pronounced volar pads conform to the surface as high-count whorl patterns. Conversely, ridges that form on a finger with a low or absent volar pad create low-count or arch-type patterns (Babler, 1987). Holt (1968) reported that the total finger ridge count (TFRC) of all 10 fingers, taken by adding the ridge counts from the core to the delta in loops, or the core toward the radial delta in whorls, is the most inheritable feature in dermatoglyphics. This combined information points directly to the conclusion that timing events related to volar pad and friction ridge formation affect friction ridge patterns.

**Timing Events:** The ridge count of a friction ridge pattern is related to two different events: the timing of the onset of volar pad regression and the timing of the onset of primary ridge formation. Differences in the timing of either event will affect the ridge count of that particular pattern. For example, early onset of volar pad regression would lead to a volar pad that was in a more regressed state at the time of the onset of primary ridge formation, and a relatively low-ridge-count pattern (or arch) would likely result. Conversely, overall late onset of volar pad regression would mean that the pad was still relatively large when primary ridges began forming, and a high-ridge-count pattern would more likely result. This theory is supported by a study that found that “late maturers” had higher-than-average ridge counts, and “early maturers” had lower-than-average ridge counts (Meier et al 1987).

If the onset of volar pad regression occurred at the normal time, then earlier-than-average onset of primary ridge formation would occur on a
larger-than-average volar pad, leading to a higher-than-average ridge count. Likewise, later-than-average onset of primary ridge formation would occur on a smaller-than-average volar pad, leading to a lower-than-average ridge count. When both early and late timing of both factors are taken into account, the results become even more complex.

To make matters even more complex, the size of the volar pad with respect to the finger is also affected by many factors. Diet and chemical intake of the mother (Holbrook 1991b), hormone levels (Jamison 1990), radiation levels (Bhasin 1980), and any other factors that affect the growth rate of the fetus during the critical stage could all indirectly affect the ridge counts of the developing friction ridges on the finger. It is important to remember that anything that affects the tension across the surface of the finger could affect the resulting ridge alignment and pattern type. However, Holt’s findings seem to indicate that timing events, rather than environmental factors, play the dominant role in determining TFRC (Holt 1968).

**Delta Placement:** The onset of cellular proliferation, which begins primary ridge formation, occurs first in three distinct areas: (i) the apex of the volar pad (which corresponds to the core of the fingerprint pattern) (ii) the distal periphery, or tip of the finger (near the nailbed) (iii) the distal interphalangeal flexion crease area below the delta in a fingerprint.

As ridge formation continues, new proliferation occurs on the edges of the existing ridge fields in areas that do not yet display primary ridge formation. These three “fields” of ridges converge as they form, meeting in
the delta area of the finger. This wavelike process of three converging fields allows for the visualization of how deltas most likely form.

The concept of “converging ridge fields” also offers a way to visualize the difference between the formation of high-versus low-ridge-count patterns. If ridges begin forming on the apex (center) of the pad first and proceed outward before formation begins on the tip and joint areas, then by the time the fields meet, a relatively large distance will have been traversed by the field on the apex of the pad; in that instance, a high-count pattern will be formed. However, if the ridges form first on the two outer-most portions and proceed inward, and formation begins at the last instant on the apex of the pad, then only a few ridges may be formed by the time the fields meet; in that instance, a very low-count pattern is observed. The combined observations of different researchers examining friction ridges on the finger during the critical stage of development further support the validity of this model (Hirsch and Schweichel 1973, Dell and Munger 1986, Babler 1991, 1999).

3.7.3 Combined Effect of Timing and Symmetry on Ridge Formation

When it is understood that timing and symmetry control two very different elements of ridge flow, it becomes easy to see how both small and large loop and whorl patterns form. A finger pad that regresses symmetrically will form a whorl pattern, regardless of early or late timing of friction ridge formation with respect to volar pad regression. If the timing of the onset of primary ridge formation in this situation is early in fetal life, then the volar pad will still be high on the finger, and the whorl pattern will have a high ridge count. If timing is later in fetal life, after the volar pad has
almost completely been absorbed into the contours of the finger, then a low-count whorl pattern will result. With further regression, an arch pattern will form.

Likewise, asymmetrical finger pads will form loop patterns and will also be affected by timing. If ridges begin forming early with respect to volar pad regression on an asymmetrical pad, then the pad will be large, and a high-count loop will result. Later timing leads to a low-count loop or arch-type pattern. Again, volar pad placement is not simply symmetrical or asymmetrical; a continuum of volar pad symmetry occurs and accounts for the variety of pattern types observed.

A regression scheme seems to exist whereby the volar pad is symmetrical at the onset and becomes progressively more asymmetrical as it regresses. This is supported by general fingerprint pattern statistics that show that more than one-half of all fingerprint patterns are ulnar loops. More specifically, this scheme is supported by fetal research that has determined that early timing of primary ridge formation leads to a higher percentage (95 percent) of whorls (Babler 1978). Also, low and high ridge count patterns occur less frequently than average count patterns (Cowger 1983). All research tends to indicate that volar pads regress from an early symmetrical position to an asymmetrical position later in fetal life. Although this is the norm, it is certainly not without exception, because whorl patterns with extremely low ridge counts and loop patterns with extremely high ridge counts can both be found with relative ease in even small collections of recorded fingerprints.
Introduction to Genetic Diversity and Friction Ridge Skin: In 1904, Inez Whipple presented research that provided a detailed theory of evolutionary progression of the volar surface (Whipple 1904). Ashbaugh succinctly summarizes Whipple’s proposition of the evolutionary genesis of friction ridges. Fourteen years after Whipple’s phylogenetic (evolutionary history) theory was presented, researchers diverged from her theory and presented an ontogenetic (individual developmental or embryonic history) model, suggesting that fusion of warts into ridges occurs during embryonic development (Wilder and Wentworth 1918). In 1926, Cummins refuted the ontogenetic scheme (Cummins 1926). However, Hale later included the ontogenetic model in his conclusions (Hale 1952). Literature since that time has been mixed. Multiple researchers have demonstrated that the first visual evidence of interaction between the dermis and the epidermis is ridges, not a series of units, protruding into the dermis. Perhaps with advances in technology, the theory that localized cell proliferations grow together into linear ridges before the appearance of the ridge as a structure will be demonstrated. Until then, fusion of units into ridges remains a possible model of development that could provide individuality before the appearance of the first ridge structures. The term “ridge unit” might be limited to a description of an adult sweat pore and surrounding ridge (Ashbaugh 1999), with the term “localized proliferation” being used to describe theoretical events of fetal formation (Babler 1987)

2.3 The Role of Genetics

Every aspect of the growth and development of a single cell into a fully formed human is initiated by a genetic blueprint. The capacity to form
friction ridges is inherent within the developing embryo. The patterns that these ridges form, however, are limited by nature and are defined by the fingerprint community as whorls, loops, arches, combinations and transitions of these basic patterns, or lack of a pattern (Hirsch 1964). Although genetics may direct when and where ridges will form by providing the blueprint for proteins, nature provides the boundaries for patterning through physical mechanisms (Ball 1999).

Proteins direct cellular activity by facilitating biochemical processes within the cell. These processes depend not only on the protein derived from the gene but also on the many other non-protein components of the cell such as sugars, lipids, hormones, inorganic elements (e.g: oxygen), inorganic compounds (e.g: nitric oxide), and minerals. Additionally, the physical environment around and within cells, including surface tension, electrical charge, and viscosity, contributes to the way the cell functions (Ball 1999).

Genetic information directs cellular function, serves as a link between generations, and influences an individual's appearance. Some aspects of appearance are similar for each individual of that species (i.e., those characteristics that define the species). However, within the species, for each aspect of an individual's appearance, many genes and external factors affect the final outcome of physical appearance. The genes involved with a specific attribute (e.g: skin color) produce the appropriate proteins, which in turn react with each other and with the many non-genetic components of the cell in complex biochemical pathways during the growth and development of the fetus (Ball 1999). These biochemical pathways proceed
under the omnipresent influence of external factors. The ultimate example of the role of the environment in friction ridge formation is monozygotic twins, who share identical genetic information and very similar intrauterine environments, but on many occasions have very different patterns. The role of genetics is currently understood by the indication that several main genes, in conjunction with a number of modifying genes, may be responsible for volar patterning, but it is well established that friction ridge patterning is also affected by the environment (Hirsch 1964, Slatis et al 1976, Weninger et al 1976, Loesch 1982, 1983, Chakraborty 1991).

Like many traits, genetics influences pattern formation indirectly by contributing to the timing of the onset of friction ridge skin, the timing of the onset of volar pad regression, the growth rate of the fetus, and other factors. Stresses across small areas of skin are not inherited, but rather they represent one of many environmental factors that influence pattern formation.

Until recently (Chakraborty 1991, Mavalwala et al 1991) most researchers in the field of genetics and physical anthropology have traditionally viewed TFRC as evidence of direct genetic control of fingerprint pattern formation (Bonnevie 1924, Holt 1968). The research of Sara Holt (1968) regarding the inheritability of TFRC is a significant finding that supports the two-tiered development scheme suggested by this and other literary reviews of fingerprint pattern formation. Logic also supports this scheme. Genetically controlled timed events would be less susceptible to environmental variations, and therefore, TFRC would be more inheritable than pattern type. Additionally, the wide range of patterns found on the
palms (Malhotra 1982) demonstrates the complex nature of factors that affect ridge alignment. Patterning and ridge counts are indirectly inherited and are not affected by only one developmental factor. However, ridge flow and ridge count are both affected by tension across the surface of growing fetal skin.

**LR Patterning**

The geometrical invariance known as symmetry is a prominent aspect of developmental morphology during embryogenesis (Levin 2005). Animal body-plans occur in a wide variety of symmetries: spherical (e.g. volvox), radial (e.g. sea anemone), chiral (e.g. snails, ciliates), bilateral (e.g. planaria) and pseudo-bilateral (e.g. man). Vertebrates have a generally bilaterally symmetrical body-plan, but this symmetry is broken by the consistently asymmetric placement of various internal organs such as the heart, liver, spleen, and gut, or the asymmetric development of paired organs (such as brain hemispheres and lungs) (Levin 2005). Symmetries are repeatedly broken during development. For example, the radial symmetry of the early chick blastoderm is broken into a bilateral symmetry by the appearance of Kohler's sickle and then the primitive streak (Levin 2005). This is further broken into definitive pseudo-symmetry by the right-sided looping of the heart tube. A fascinating atlas of morphological asymmetries throughout the animal kingdom is given in Neville (1976).

Developmental noise often results in pseudo-random characteristics and minor stochastic deviations known as fluctuating asymmetry; however, the most interesting phenomenon is invariant (i.e. consistently biased among all normal individuals of a given type) differences between the left and right
sides. For reasons of space as well as because these are likely to be secondary to embryonic asymmetries, this review largely neglects behavioral/sensory asymmetries (such as lobster claw morphology which is determined by neurological activity). A huge literature on brain lateralization phenomena in human beings exists as well (Harnad 1977), but many of these asymmetries are secondary and arise as a result of cultural environmental biasing factors. The LR axis itself follows automatically from the definition of the AP and DV axes, as it is perpendicular to both; however, consistently imposed asymmetry across it is fundamentally different from patterning along the other two axes (Levin 2005). Firstly, while the AP and DV axes can be set by exogenous cues such as gravity, or sperm entry point, there is no independent way to pick out the left (or right) direction, since no known macroscopic aspect of nature differentiates left from right (Levin 2005). One possible way to use a fundamental force to orient the LR axis relative to the other two axes was suggested by Huxley and deBeer (1963). They proposed that LR asymmetry was oriented during embryonic development by an electric current running down the length of the notochord, which would generate a magnetic field vector pointing R or L, if measured at the dorsal or ventral sides. Although a correlation between the earth’s geomagnetic field reversals and shell chirality has been observed (Harrison and Funnel, 1964), the nature of a causal relationship (if any) is unknown, and there is no evidence to date of a magnetic field being utilized during LR patterning in any species. In the final phase, individual organs utilize cell migration, differential proliferation, cytoskeletal organization, and other mechanisms to achieve asymmetries
in their location or morphogenesis (Stalsberg 1969a, b, Manasek 1981, Horne-Badovinac et al 2003). Consistent with their downstream position, and counter to earlier proposals (Waddington 1937), a number of recent studies have shown that the individual organs literalities are set, and can be experimentally randomized, independently (Levin et al 1997b, Chin et al 2000a). Biophysical mechanisms used to shape organogenesis include the extracellular matrix (Tsuda et al 1996, Yue et al 2004) and actin bundles (Itasaki et al 1989, 1991) in the chick heart tube, and differential rates of elongation in the frog gut tube (Muller et al 2003). Genetic control of these pathways is mediated proximately (if not directly) by genes such as flectin, the bHLH family members EH AN D and DH AND, and the transcription factor Tbx5 (Srivastava 1995, Tsuda et al 1996, Sparrow et al 1998, Bruneau et al 1999, Angelo et al 2000, Fernandez-Teran et al 2000, Hatcher et al 2000, Takeuchi et al 2003). The mechanisms underlying embryonic turning remain poorly understood (Constam and Robertson 2000).

The topological deformations undergone by asymmetric tissues are more complex than usually assumed (Manner 2004) and complete understanding is likely to require mathematical or physical models in addition to molecular biology. Upstream of these processes lies a pathway of asymmetric genes which are expressed in cell fields only on one side of the embryo’s midline. By inducing or repressing transcription of downstream asymmetric targets, they propagate signals among sub-populations of cells (such as node and lateral plate mesoderm), which eventually dictate sideness for the organs undergoing asymmetric
morphogenesis. These cascades of asymmetric gene expression form the middle phase of LR patterning. However, for whichever asymmetric gene is at the top of the pathway, it is necessary to ask what determined its asymmetry. Thus, in the first phase of LR patterning, an as-yet unknown mechanism must orient the LR axis with respect to the other two axes. While theoretical candidate mechanisms have been proposed (Brown and Wolpert 1990) no mechanism has been conclusively shown to initiate asymmetry. The developmental timing of each phase differs among species, though asymmetric gene expression almost always begins at or shortly after gastrulation. The LR axis is probably specified after the AP and DV axes, and is determined with respect to them (McCain and McClay, 1994; Danos and Yost, 1995a). The timing of the initiation of LR asymmetry in the various species is particularly controversial, but the mechanisms underlying different aspects of LR patterning in various species are beginning to be uncovered in significant detail. The earliest conserved asymmetric gene known is Nodal, which is left-sided at somite stages in all vertebrates in which it has been examined. Downstream Lefty and Pitx-2 genes appear to be well conserved also. However, neither Shh nor any of the other early genes known to be asymmetric during chick gastrulation (cAct-RIIa, cHNF3-b, Follistatin, cWnt-8C, etc.) have been reported to asymmetric in other species despite in situ hybridization searches by a number of labs (Ekker et al 1995, Stolow and Shi 1995), although Shh is left-sided in ducks and quails (Levin, 1996). Interestingly, misexpression of Hedgehog proteins in frog embryos is known to randomize asymmetry (Sampath et al 1997), raising the possibility that the asymmetric Hedgehog
signal exists in amphibia but perhaps utilizes an as yet uncharacterized family member. The situation with respect to the early asymmetric genes is the same in mouse, where genetic deletions have suggested roles for some of the same molecules (Oh and Li 1997, Tsukui et al, 1999b). It is possible that the asymmetry in Hedgehog signaling exists at a level other than mRNA (protein processing, translation, etc.) or is anatomically so subtle as to have been missed. While no asymmetric expression upstream of Nodal has been reported in mice, two mouse pathways (the first conserved to chicks, the second to Xenopus play a role upstream of Nodal: the Notch pathway (Krebs et al 2003, Raya et al 2003b) and Vg-1 (Rankin et al 2000). Two more areas which are of relevance to questions of evolutionary conservation are retinoic acid signaling and induction of Nodal genes by Hedgehog signals in amphibian.

Dental Caries:

Dental caries may probably be considered as a disease of modern civilization, since pre-historic man rarely suffered from this form of tooth destruction. There is no evidence of dental caries in the relatively few teeth found in skull fragments of our earliest known direct ancestors the pithencanthropen.

Thousands of years ago the Sumerians and the Chinese offered worm as the cause of the disease. Fossils of early ancestors of man have shown the incidence of caries in them. Egyptians were more concerned about the treatment and started compounding prescriptions and placing fillings to treat the ravages of caries and increase in caries in Egyptian population which was thought to coincide with the replacement of wheat for
other grains in their diet. Centuries later there was an increase in caries during roman occupation of Europe, probably owing to increased use of cooked food.

Anthropologic studies of Van Len Hossek revealed that the dolicocephalic skulls of men from pre Neolithic periods (12,000 B.C) did not exhibit dental caries. But skulls from brachycephalic men of the Neolithic period (12,000-3000 B.C) had carious teeth. Palentological evidence shows that it has affected humans at least from the time that agriculture replaced hunting as the principal source of food. Examination of skulls in Britain suggested that caries prevalence changed little from Anglo-Saxon period (5th-7th centuries) to the end of the middle ages, approximately the year 1500. The modern pattern of caries was not evident in Britain until the 16th century.

Dietary changes during the 17th century principally increased refinement and greater use of sucrose, as sugars became more available, are considered to be chiefly responsible for the development of the modern pattern of caries. Import duties on sugar in Britain began to be removed in 1845 and were completely eliminated in 1875, a period during which the severity of caries greatly increased. By the end of the 19th century, dental caries was well established as an epidemic disease of massive proportions in most developed countries. However, evidence now indicates that this trend peaked and began to decline in many countries in the late 1970s and early 1980s, and the decline was most notable in certain segments of the population of the United States, western Europe, New Zealand, and Australia.
The exact cause of the decline is unknown but is attributed to the addition of trace amounts of fluoride ion to public drinking water. Trace amounts of fluoride were discovered to have a marked limiting effect on the progression of caries lesions originating on the adjacent contacting, or proximal, surfaces of teeth. However the increase was nothing as compared to the dramatic rise in dental caries from the middle age until the 1950s. Infact by the 1950s dental caries had reached epidemic proportions affecting 90% to 95% of the population in the developed world.

History of dental caries and management throughout the second millennium can be divided in two distinct periods.

- First which lasted more than 900 years and may still be going on today is the “observational” era.
- The second which has developed and revolutionized our understanding of the cause and treatment of all diseases is scientific era.

During observational era there were several theories on why dental caries develops. However one theory that is based on limited “observational and experimental data” was the Chemico-parastic theory. Dietary or “constitutional” or nutritional factors also were associated with dental caries.

During the late 19th century American dentists began reporting on the epidemic of dental caries. The rise in dental caries was most noticeable among affluent, urban & white Americans. This observation led to several theories.
ETIOLOGY

2.4 ANCENT THEORIES OF CARIES ETIOLOGY

WORM THEORY

According to ancient Sumerian text, toothache was caused by a worm that "drunk the blood of teeth and fed on roots of jaw." this legend of worms is estimated to date back to 5000 B.C as evidenced by the discovery of clay tablets. The ancient Chinese of 1000 B.C also believed worms to be invaders of the mouth. This idea was once universally accepted and the treatment was advocated by Chinese and Egyptians. For these mouth worms, fumigation with seeds of onion and leech was recommended.

HUMOR THEORY

The ancient Greeks considered that a person’s physical and mental constitution was determined by the relative proportions of the four elemental fluids of the body - blood, phlegm, black bile, and yellow bile. These four elemental fluids of our body i.e. Blood, phlegm, black bile and yellow bile corresponds to the four humors - sanguine, phlegmatic, melancholic and choleric. All diseases, including caries could be explained by an imbalance of those humors.

VITAL THEORY

The vital theory regarded dental caries as originating within the tooth itself, analogous to bone gangrene. This theory proposed at the end of
eighteenth century, remained dominant until the middle of the nineteenth century. A clinically well known type of caries is characterized by extensive penetration into the dentin, and even into the pulp, but with a barely detectable catch or a fissure.

**CHEMICAL THEORY**

Parmly (1819) rebelled against the vital theory and proposed that an unidentified “chemical” agent was responsible for caries. He stated that caries began on the enamel surface in locations where the food putrefied and acquired sufficient dissolving power to produce the disease chemically. Support for the chemical theory came from Robertson (1835) and Regnart (1938) who actually carried out experiments with different dilutions of organic acids (such as sulphuric and nitric) and found that they corroded the enamel and dentin.

**PARASITIC THEORY**

In 1843, Erdl described filamentous parasites to be responsible for caries. Ficinus called these micro-organisms as "denticolae". All these theories, although at one time universally accepted, are now a days obsolete.

**2.5 EARLY THEORIES OF CARIES ETIOLOGY**

**CHEMO-PARASITIC- THEORY**

Pasteur discovered that micro-organisms transform sugar to lactic acid due to fermentation. Emil Magitot (1867) demonstrated that fermentation of sugars causes dissolution of tooth mineral in vitro. Leber Rottenstein (1867) presented additional evidence implicating acid and bacteria as the causative agents of dental caries. They believed leptothrix
buccalis to be responsible for dental caries. William D. Miller gave the chemo-parasitic theory which had a profound effect in understanding of caries etiology. Carbohydrate food material lodged between and on surfaces of teeth is the source of the acid. The enamel is destroyed by the acid produced by fermentation of sugars and the disintegrated enamel is subsequently mechanically removed by forces of mastication.

The significance of Miller's observations is that he assigned an essential role to three factors in the caries process: the oral microorganisms in acid production and in proteolysis; the carbohydrate substrate which microorganisms fermented, and the acid which causes dissolution of tooth minerals. Miller used a mixed microbial flora of saliva and carbohydrates in order to demonstrate the destruction of teeth in vitro. He concluded that caries was caused not by a single species of microorganisms but was related to multiple microbial activity involving acid production and protein degradation. He believed that no single species of microorganisms are capable of producing acid and digesting proteins. This lead to the CHEMO-PARASITIC THEORY which includes:

1. Decalcification or softening of tissues and
2. Dissolution of the softened residue

Modern research has shown beyond doubt that acids are involved in caries as evidenced by decrease in pH following a rinse with a suitable substrate for bacterial fermentation.

**Critique of Miller's chemo-parasitic theory**

It was unable to explain the predilection of specific sites on a tooth to dental caries. The initiation of caries on smooth surfaces was not accounted
for by this theory. The concept of the dental plaque adhering to teeth and serving to localize bacterial enzymatic activity was not proposed until 1897 by Williams and in 1898 by Miller, while a disciple of Koch who was an avid advocate of specific bacterial etiology of infectious disease, nevertheless worked with mixed cultures from saliva and with techniques that did not attempt to ascertain types of organisms present. Miller's theory does not explain why some populations are caries-free. The phenomenon of arrested caries is not explained by the chemico-parasitic theory. Miller believed that in some systemic conditions the inorganic salts within a tooth could be withdrawn and that the organic-inorganic bonds would be weakened. He did not produce any experimental evidence that the adult tooth is subject to such systemic influences.

**PROTEOLYTIC THEORY**

The human tooth contains only 1.5%-2.0% of organic material of this 0.3 to 0.4% account for protein. According to the proteolytic theory, the organic component is most vulnerable and is attacked by hydrolytic enzymes of microorganisms. This precedes the loss of inorganic phase. Gottlieb (1944) believed that the initial action was due to proteolytic enzymes of S.mutans attacking the lamellae, rod sheaths, tufts and walls of dentinal tubules. Pincus (1949) believed that the proteolytic organisms first attacked the protein elements and then destroyed the prism sheaths. The loosened prisms would then fallout mechanically.

**PROTEOLYSIS-CHELATION THEORY (SCHWATZ et al 1955)**

The word 'chelate' is derived from the Greek word 'chele' meaning claw, and refers to compounds that are able to bind metallic ions such as
calcium, iron, copper, zinc and other metals, by the secondary valence bonds. The resulting chelates are nonionic and usually soluble. Biological substances such as amino acids and other chelators may be used to remove calcium and other metal ions from a solution.

According to this theory chelation has been proposed as an explanation for tooth decay whereby the inorganic components of enamel can be removed at neutral or alkali pH. This theory considers dental caries to be a bacterial destruction of teeth where the initial attack is on organic components of enamel. Breakdown products of this organic matter have chelating properties and hence dissolve the enamel. Thus, both the organic and inorganic constituents of enamel are simultaneously demolished.

This theory suggests that the microorganisms produces an initial carious lesion and then releases a variety of complexing agents, such as amino acids, polyphosphates and organic acids. The complexing agents then dissolve the crystalline apatite. Less than 1% of mature enamel is organic in nature and the suggestion that this material upon degradation can give rise to a significant concentration of chelator sufficient to dissolve up to 96% mineral matter has no experimental support. Also, there is no substantial experimental evidence that the initial caries lesion stems from a breakdown of organic matter, i.e. due to proteolytic action. While proteolysis-chelation is an important biological phenomenon, its primary role in the etiology of dental caries has not been corroborated.
2.6 OTHER THEORIES OF CARIES ETIOLOGY SUCROSE-CHELATION THEORY (COMPLEXING AND PHOSPHORYLATING THEORY)

Eggers-Lura (1967) proposed that sucrose itself and not the acid derived from it, can cause dissolution of enamel by forming unionized calcium saccharates. It can be readily demonstrated that an uptake of phosphate by plaque bacteria occurs during aerobic and anaerobic glycolysis and the synthesis of polyphosphates. The theory is that calcium saccharides and calcium complexing intermediaries require inorganic phosphate, which is subsequently removed from the enamel by phosphorylating enzymes. Soluble calcium complexing compounds produced by bacteria cause further tooth disintegration.

Saliva is an abundant source of inorganic phosphate for bacterial utilization. Hence, it is highly improbable that depletion of phosphate in plaque by oral microbial metabolism results in phosphate withdrawal from enamel. Kreitzman et al (1969) stated that that alkaline phosphatase causes a release of enamel phosphate from hypothetical organically bound phosphate is without experimental proof. How alkaline phosphatase which acts on organic phosphates could degrade a solid enamel substrate, in which virtually none of the phosphate is organically bound, remains to be elaborated. Release of phosphate from teeth may be nonspecifically achieved by ammonium sulfate and, since this salt was used in the preparation of the commercial alkaline phosphatase, it may explain this anomalous finding.
AUTO IMMUNE THEORY

Burch & Jackson (1966) analyzed caries epidemiologic data and suggested that genes, partly inherited and partly mutational, determine whether a site on a tooth is at risk. In discussing this hypothesis, Jenkins points out that most of the data on which the theory is based are epidemiologic. It is doubtful whether these data, collected during routine clinical examinations are sufficiently accurate for mathematical analysis.

SULFATASE THEORY

Pincus (1950) advanced the sulfatase theory, whereby bacterial sulfatase hydrolyzes the 'mucoitin sulfate' of enamel and the chondroitin sulfate of dentin producing sulfuric acid that in turn causes decalcification of the dental tissues. The concentration of sulfated polysaccharides in enamel is very small and not readily accessible as a substrate for enzymatic degradation. This is a highly unlikely hypothesis for the degradation of tooth enamel.

Caries as Nutritional Deficiency

Some researches consider caries as nutritional deficiency caused either by sufficient phosphate intake or improper dietary calcium - phosphate ratio. None of these theories have adequate statistical or experimental support and hence these theories remain primarily conjectural.

2.7 CURRENT CONCEPTS OF CARIES ETIOLOGY

Keyes (1960) proposed a triad of factors for caries formation. According to him caries could not occur even if one of the triad was missing. He proposed it in the form of a venn diagram as
The current concepts of caries etiology believe dental caries to be a multifactorial disease in which there is interplay of four perpetual factors (1978 Newbrun): Host, Micro flora, Substrate (diet) and Time.

Circle for caries etiology
Burt (1986) modified the Keyes circle by redefining the contents of the Venndiagram as

**Modified Keyes Circle**

Factors involved in caries development - Fejerskov and Manji
Nutrition is the balance between the supply and physiological expenditure of energy and nutrients of all the cells in the body. Nutrition is concerned with the systemic “metabolic” effects of eating habits and nutrients in food.

2.8 Nutritional influences on dental tissues and Caries

Enamel, Dentin and Cementum are highly dynamic tissues which are exposed to a constant supply of ions of both external (oral) and internal (pulpal) origin re-precipitation of minerals after pH-induced mineral lost causing formation of an organic protein matrix, followed by mineralization and maturation, which may disturb the tooth structure as well as the form and position of the teeth and delay eruption into the mouth.

Nutritional deficiency such as deficiencies of calcium, phosphate, vitamin A, D and C protein and energy affect tooth tissue formation. Impairment of enamel and dentin quality increases the progression of dental caries.

Vitamin D

Vitamin D along with parathyroid hormone and calcitonin play primary role in regulating the concentration of calcium and inorganic phosphate in plasma ECF in regulating the movements of these ion into and out of the cells and in controlling the mineralization of bone and teeth.

Enamel hypoplasia

Mellariby (1936) reported that 74% of the hypoplastic teeth were affected by caries and 80% of normal teeth were non carious. This has also been supported by Bibby (1943), Carr (1953). Infante and Gillespie (1977) did a study on children with linear enamel hypoplasia, where in the
incidence of caries was significantly higher in non hypoplastic molars of children with LEH on anteriors, when compared to children who did not have this condition.

**Vitamin A**

Vitamin A deficiency leads to atrophic changes of the ameloblasts, reduced number of salivary acini in the major and minor salivary glands. This may lead to hypoplastic defects and xerophthalmia.

**Other Vitamins**

**Pyridoxine (B6)** has been stated to reduce caries in rats (Cole 1980). Human studies involving administration of large doses of pyridoxine to pregnant women and children have been shown to reduce caries. The vitamin acts by modifying the oral flora.

**Lipids**

Fat consumed posteruptively has been co-related with reduction in caries. The anticariogenic action of the fats has been co-related to 2 reasons: The formation of a fat film which reduces the demineralization action. And the contact between the carbohydrate foods and bacteria is reduced in the presence of fat. Certain fats have an antimicrobial action, but whether this occurs in the mouth has not been adequately studied.

**Proteins**

Many studies on animals have shown a strong correlation between the caries formation and protein deficiency. Observational studies done by many (Johansson, Rajan BP 1992) have shown similar results in children of India. The exact mechanism of action is still not clear. The following factors may contribute: - Reduced salivary flow and therefore reduced total
buffering capacity, a reduced rematerializing activity, altered morphology of dentition and decrease in the immune response.

**Carbohydrates**

It has been firmly established that dietary carbohydrates are caries conducive and they exert their cariogenic effect locally on the tooth surface.

**Carbohydrate Intolerance and Dental Caries**

Hereditary fructose intolerance provides evidence of a direct link between sugar ingestion and dental caries. In 1959 Froesch described an inborn error of fructose metabolism transmitted by an autosomal recessive gene. This condition results in episodes of pallor, nausea, vomiting, coma and convulsion following ingestion of fruit containing fructose or cane sugar. Treatment consists of complete dietary exclusion of sucrose; although other carbohydrates, such as glucose, lactose, and galactose, may be included. Persons with HFI show a strikingly reduced dental caries experience when compared to a control population of the same age (Marthaler 1967).

**2.9 Plaque and its Role in Caries**

One of the prime culprits of dental decay and periodontal diseases is plaque – a soft adherent collection of salivary products and bacterial colonies on the teeth. It accumulates on the surface of the teeth continuously throughout the life span of most people in varying degrees. The patients only hope in eliminating this disease producing material is to continuously remove it by tooth brushing and dental flossing. Unrestricted plaque growth produces local environmental conditions that may selectively promote the accumulation of pathogenic bacterial species. High frequency
sucrose exposure is the single most important factor in producing a cariogenic plaque. Frequent sucrose ingestion begins a series of changes in the local tooth environment that promotes the growth of highly acidogenic bacteria and eventually leads to caries.

Multiple factors determine the characteristics of plaque. The factors that control the presence of individual species in plaque are termed ecological determinants: Plaque growth begins approximately six hours after the thorough cleaning of the teeth. The first phase of plaque development is the deposition of adherent products from the saliva. These products are primarily composed of mucin, which forms the thin adherent layer on the teeth called the pellicle. Once the pellicle has been formed on the clean tooth surface, bacteria that inhabit the oral cavity attach themselves to the pellicle. After attachment the bacteria multiplies to form large masses of bacterial colonies. These begin to occur approximately eighteen hours after thorough cleaning of the teeth and continue until the plaque is fully mature at the end of three weeks.

Mature plaque consists primarily of bacteria of various types. Each type of organism functions in a different way. Some bacteria produce harmful chemical substances and others produce substances that are needed by neighboring bacteria to survive. Still other organisms produce adherent substances that are interspersed with the bacteria and hold the plaque intact on the surface. Mature plaque is in reality a microscopic community of different bacteria and other substances that function to produce dental disease. The disease producing potential of dental plaque is that of the supra gingival plaque, because of its acidic nature, is
responsible for the production of dental caries. Subgingival plaque because of its capacity to produce substances that are toxic to soft tissues, is responsible for periodontal diseases.

Streptococcus mutans is one of the first organisms to attach to the pellicle and multiply. The streptococci are capable of producing both polysaccharides and acids from carbohydrates that are consumed by the patient. This is important because the polysaccharides help attach the streptococci to the pellicle. The acid they produce is capable of demineralizing the enamel layer of the tooth. This demineralization is the first stage of dental caries. Other organisms in dental plaque produce various substances that help the bacteria mass attach to the pellicle. The acid producing bacteria are attached to the tooth surface contributes to a greater effectiveness of acid demineralization of tooth enamel. The plaque because of its thickness and density prevents acid produced within it from being diluted by saliva or neutralized by chemicals contained in the saliva. Therefore the acid rather concentrated adjacent to the tooth surface and can break down the enamel more quickly.

Once the caries process is initiated, another organism, lactobacillus, can become retained in the decayed area. Since the lesion is acidic, these organisms thrive and like those of streptococcus mutans, they convert sugar to acid, which in turn attacks tooth structure. It is also believed that lactobacillus organisms can become lodged in the retentive pits and fissures in the tooth surface, where they multiply and the acid they produce attack tooth structure.
2.10 GENETICS IN DENTAL CARIES

Dental caries has an important role in the manifestation of tooth pain and loss, and has been associated with problems in school and absenteeism in the workplace, leading to a decrease in quality of life. Moreover, oral health presents a close association with the individual’s general health, and may be a risk factor for several diseases.

Since the 1920s, the question about the genetic influence in dental decay has been discussed. From the 1970s through the 1990s numerous studies concerning genetic aspects of caries searched for gene variants in cariogenic bacteria. The involvement of S. mutans and its different genotypes in susceptibility to dental decay, and many S. mutans strains have already been identified as having influence on the disease.

The natural history of caries development and the relative contributions of genetic and environmental factors to caries of the primary dentition have been poorly understood. Hence, certain crucial features of caries onset and progression have been difficult to model. (William, 1978) Understanding the relative roles of heredity and environmental factors (“nature vs. nurture”) in the pathogenesis of dental caries, has occupied clinical and basic researchers for decades. Critical is the realization that genes and environment do not act independently of each other; the appearance or magnitude of heritability may differ with various environments. (Werneck, 2010) Dental caries incidence is affected by host factors that may be related to the structure of dental enamel, immunologic response to cariogenic bacteria, or the composition of saliva. Genetic variation of the host factors may contribute to increased risks for dental
Numerous reports have described a potential genetic contribution to the risk for dental caries. The pathogenesis of the caries process is rather well understood today, and although it is quite more complex than was believed in the early days of dental research, for the sake of simplification we can presuppose that the caries attack rate in humans is a consequence of at least five distinctly separate traits or attributes (Osborne 1963)

1. The density or structural integrity of the enamel
2. Topical and/or communal water fluoridation
3. The composition of the secretions of the salivary glands
4. Nutrition and day-to-day dietary habits
5. Personal and professional oral hygiene.

The latter could be considered to include the spectrum of oral bacterial flora. Numbers (1) and (3) are obviously the most likely candidates for direct genetic control; (4) and (5) less so, at least directly; and (2) must be viewed as “purely environmental”. Studies on twins have provided strong evidence for the role of inheritance. Establishing a basis for a genetic contribution to dental caries will provide a foundation for future studies utilizing the human genome sequence to improve understanding of the disease process. The evidence of a genetic contribution to caries was based on following factors.

1. Examining inheritance that altered the dental hard tissues- This was based on the role of the dental hard tissues, the target for acid dissolution by cariogenic bacteria, and the genetic contribution of altered enamel biomineralization.
2. The immune response-based on alterations in the immune response reducing the clearance of the bacteria
3. The dietary consumption of sugar
4. The saliva and its composition.

Genetically regulated processes identified as contributing to caries incidence included tooth eruption and development, salivary flow and saliva components, and tooth morphology. The most convincing data on the role of genetics in the pathogenesis of dental caries has been developed by analyzing the caries incidence in monozygotic and dizygotic twins. Inheritance and the incidence of dental caries were achieved by analyzing twins reared apart who were enrolled in the Minnesota Boraas Study of Twins Reared Apart. These studies had a major advantage in that the patients did not share similar environments from shortly after birth until the time of analysis. The analysis demonstrated a highly significant (p<0.001) relationship between the numbers of teeth present and the percentage of teeth with dental caries when comparing monozygotic and dizygotic twins reared apart. Boraas et al (1988) performed matched pair study design between 64 MZ and 33 DZ twins. Using 2-way ANOVA test as the data analysis method, they concluded that there was a marked genetic contribution to dental caries. He concluded that the study provided “New evidence for a marked genetic component to dentate status and dental caries experience.”

He also speculated on the particular inherited traits that could contribute to the results by stating “Several genetically variable factors which may be involved in the development of dental caries and could
contribute to the greater MZ (monozygotic) similarity in dental caries experience (are)

1. Salivary factors and oral flora,
2. Tooth eruption time and sequence,
3. Tooth morphology,
4. Arch shape,
5. Dental spacing,
6. Propensity for diet.

The analysis of twins raised apart provides the strongest evidence of agenetic contribution to the incidence of dental caries. The analysis of dental caries incidence in monozygotic and dizygotic twins indicates that a large number of different genes contribute to the observed outcomes.

Individuals with either an inherited or acquired immune deficiency are subject to increased risks for and incidence of dental caries. Evidence for genetic contribution to the susceptibility to dental caries was done by matched pair twin analysis studies. The genetic analysis was performed using zygosity testing method between monozygotic twins (MZ) and dizygotic twins (DZ).

These include studies by Bachrach and Young (1927) studied 300 twin populations. It included 130 MZ twins as a study group and 170 DZ twins as a control group. The correlation existed between MZ twins and caries; thus they concluded heredity as a subsidiary part in caries incidence. Goldberg (1930) performed comparative study on 42 pairs of MZ and DZ twins population. He found that identical twins showed decay in corresponding teeth and concluded “Heredity affects dental decay only in as much as it
controls the shape of a tooth and pits and fissures, its position in the dental arch.” (Charles 2001) Horowitz et al (1958) performed match pair study between MZ (n=30) and DZ (n=9) twins. They concluded that MZ twins had a greater caries concordance than DZ twins.

Goodman et al (1959) performed match pair study between MZ (n=19) and DZ (n=19) twins. They concluded that intrapair caries variance of DZ twins was greater than in MZ twins. Mansbridge (1959) performed match pair study between MZ (n=96) and DZ (n=128) twins. Using chi-square data analysis he found genetic contribution to dental caries was lesser than environmental factors. Finn and Caldwell (1963) performed matched pair study design between MZ (n=35) and DZ (n=31) twins. Using F test as the data analysis method, they concluded that smooth surface caries had a genetic susceptibility. Bordoni et al (1973) performed matched pair study design between MZ (n=17) and DZ (n=17) twins. Using F test as the data analysis method, they concluded that there is a “strong genetic component in primary teeth which affects the incidence of caries.” (Charles 2001).

Fairpo (1979) performed matched pair study design between MZ (n=100) and DZ (n=120) twins. Using F test as the data analysis method, they concluded that his study “indicates that there is some genetic influence on the susceptibility to caries of both deciduous and permanent teeth.” (Charles 2001) Conry et al (1993) performed matched pair study design between MZ (n=46) and DZ (n=22) twins. Using 2-way ANOVA test as the data analysis method, they concluded that there was a marked genetic contribution to dental caries.
Several MHC analysis studies were performed to evaluate the genetic susceptibility to dental caries. The earliest study was conducted by Lehner et al. (1981), who analyzed the distribution of HLA DR antigens in a group of twenty-four individuals with either high or low Decayed Missing Filled Surfaces (DMFS) indices. It was shown that HLA DRw6-1,2, 3 had a significant relationship to the DMFS index and to low dose response to Streptococci mutans antigens. Mariani et al. (1994) performed matched pair analysis study on 271 individuals. Using serological test method they concluded HLA DR3 increased risk of dental caries and HLA-DR 5, 7 associated with a reduced frequency of dental caries.

Two different lines of investigation have provided evidence that the genes in the HLA complex are associated with altered enamel development and increased susceptibility to dental caries. The role of these genes in the immune response to cariogenic bacteria represent a mechanism that is based on inherited genetic complements and thus provides the opportunity in the future to study specific allelic variants of these genes as a potential marker for increased dentalcaries risk.


Senpuku et al. (1998) performed serological test analysis method on 9 individuals and concluded that Streptococcus mutans antigens bound strongly to HLA DR8, DR5 and DR6.
Acton et al (1999) performed comparative study on 186 individuals. Using HLA typing genetic analysis method they concluded DRB1-3 and BRB1-4 linked to high levels of Streptococcus mutants number and thus concluded a increased risk of dental caries. Applying the family/population method of analysis, Klein (1946) examined 5,400 individuals who were members of 1,150 different families, and demonstrated that the amount of dental disease (viz., caries, "DMF") that appeared in the offspring was quantitatively related to that which had been experienced by their parents. He concluded that it is difficult to exclude the view that caries susceptibility in children involves strong familial vectors which very likely have a genetic basis, perhaps sex-linked. Book and Grahnen (1953) in the first sentence of their landmark paper on "Clinical and Genetical Studies of Dental Caries", concluded that "genetic factors play an appreciable part in determining individual resistance against dental caries".

Realizing that dental caries is a pathologic entity that results from the interaction of endogenous and exogenous traits, Goodman et al (1959) studied 38 like sexed MZ and DZ twin pairs in Michigan in an attempt to relate tooth decay to other factors that might be under genetic control. They reported significant heritability for the presence of several oral microorganisms, including Streptococci, and also for salivary flow rate, salivary pH, and salivary amylase reactivity. Aside from hereditary factors relating directly to enamel constitution, Goodman and co-workers thus established other genetically influenced factors as operative in caries etiology. Fairpo (1979), expanded on previous studies of caries susceptibility by greatly increasing the "n" in the twin study population, and by evaluating total caries
experience in a racially discreet, age-matched sample. The results authenticated previous conclusions (Finn and Caldwell, 1963; Akhmedov, 1973) that both in permanent and deciduous dentitions, a strong genetic factor has been implicated in caries susceptibility.

As noted by Boraas and co-workers (1988), several genetically controlled factors have previously been identified or implicated to be involved in the susceptibility to dental caries-for example, salivary factors and the oral microflora (Mandel 1974), tooth eruption patterns (Gedda and Brenci 1966), tooth morphology (Konig 1963, Wood and Green 1969), dental arch shape (Kolmakow and Puranen 1985), and interdental space dimension (Corruccini and Potter, 1980), as well as nutritional factors (Forrai and Bankovi 1984). (Werneck R 2010) It is rather well-accepted today that tooth morphology (e.g., occlusal fissure shape and depth) can play a significant role in caries susceptibility as tooth shape and size are genetically determined is well established.

Introducing the term Caries Experience Rate (CER) into the dental literature, Horowitz et al demonstrated a genetic component of variability in caries incidence in adults who were otherwise in good systemic health, and claimed, therefore, to have found a definitive hereditary factor in susceptibility to caries.

Quantitative trait locus (QTL) analysis was performed on genetic crosses of C3H/HeJ (caries-resistant) and C57BL/6J (caries-susceptible) mice inoculated with Streptococcus mutans serotype C. In a genome wide scan, three suggestive QTLs were detected on chromosomes 1, 2 and 7; one significant QTL was found on chromosome 2, and one highly significant
QTL was detected on chromosome 8. The likelihood ratiostatistic (LRS) was raised around the marker D1Mit21 in the middle region of chromosome 1, between D2Mit255 and D2Mit311 in the distal region of chromosome 2, and the region distal to D7Mit31 on chromosome 7. A significant QTL was located between the markers D2Mit237 and D2Mit101 on chromosome 2. The LRS was highly significantly raised between markers D8Mit208 and D8Mit280 on chromosome 8, and exceeded a highly significant level between markers D8Mit211 and D8Mit280. These results suggest that major gene(s) responsible for dental caries susceptibility or resistance are located in one or more of these regions. The longitudinal twins studies show that a significant proportion of the caries variance in young children is heritable, indicating a genetic contribution (Bretz et al 2005). Current findings also indicate that in longitudinal assessments of site specific incidence rates in lesion progression, heredity may play a significant role. Heritability estimates were greatest at the age ranges 1.5 - < 4 yrs and > 6 yrs. These findings suggest that risk for caries incidence and progression may be greatest when the dentitions are emerging into the oral cavity.

Newly born caries-resistant rats were fostered by caries-susceptible mothers and vice versa. The caries-resistant young remained caries-resistant; the caries susceptible young remained susceptible and the foster mothers did not affect the caries experience of the young they nursed. It may be concluded that, in these animals, genotype played a role in their resistance or susceptibility to dental caries. Identifying the genetic factors contributing to caries risk and resistance will provide clinicians with new tools for targeting individuals and/or populations for more efficient and
effective preventive therapies. For example, individuals with certain types of understanding the human hereditary traits contributing to dental caries coupled with genetic knowledge of the virulence and pathogenicity associated with cariogenic bacteria will allow new diagnostic and novel therapeutic approaches to be applied in the management of this disease. Thus multifactorial nature of dental caries has limited the opportunity to link patterns of inheritance with susceptibility to dental caries.

Bacteria genetic studies by Wen and Burne 2002, Cheryl et al 2005 identified genetic changes able to encode the proteins involved in biofilm development. The relationship among human leukocyte antigen (HLA) class II and TNFα alleles, levels of oral bacteria was identified to play a role in the etiology of dental caries by Afro-American women (Acton et al 1999).

Genetic studies comprehend the genetic component associated with the individual susceptibility to dental decay development. Steggerda and Hill 1936, hypothesized that differences in susceptibility to caries could be due to hereditary factors. Hunt–Hoppertstudies strongly suggest the influence of genetic differences in controlling caries progression. (Hunt et al 1944, 1955) Matsumoto et al suggested that the E2f1 gene, which mutation cause a decreased volume of saliva production and protein production rate, affected susceptibility for oral biofilm formation by streptococci. Maeda et al 1995 suggested that the salivary immune response plays an important factor in regulating dental decay development.

Several studies of familial aggregation in caries have been reported since the 1930. A solid body of evidence was created indicating familial
clustering of caries experience and allowing for speculation whether or not there are genetic factors controlling the disease.

High concordance rates between twins for several dental phenotypes such as dental decay, tooth size, dental arch dimensions, intercuspal distances and occlusal traits have been described (Townsend et al 2003, 2008). Horowitz et al (1958), suggested that there was genetic influence in the susceptibility to dental decay development in both deciduous and permanent teeth. Bretz et al 2005b demonstrated that there was a genetic factor influencing dental decay development. Bordoni et al (1973) concluded that a genetic component is more important in tooth morphology and eruption timing than caries.

Linkage and association analysis helps in identification of the polymorphisms associated with susceptibility and / or resistance and several candidate genomic regions and genes have been identified. Genetic linkage between dental decay and loci of chromosomes two, eight and 17 where mice major histocompatibility complex (MHC) is localized, was identified. Maeda et al, 1995 suggested that the immune response may be an important factor in regulating dental decay development. Lehner et al, 1981 demonstrated that, in caries resistant subjects, a lower dose of Streptococcus was necessary to stimulate T-helper activity and the low-dose feature was associated with the specificity HLA-DRw while the high-dose was associated with HLA-DR4.

A polygenic nature for the genetic control of caries disease has been discussed since the 1970s. Muhlemann (1972) suggested that a set of genes could influence the enamel resistance while a different set could
influence the saliva composition and host response to infection. Nevertheless, only recently linkage and association studies have begun to be conducted in an attempt to identify genomic regions and polymorphisms related to dental caries. The first linkage analysis for dental decay was carried out in 2008 and the authors highlighted the presence of genes related to saliva flow control and diet preferences in regions 13q31.1, 14q24.3 and 14q11.2.

Slayton et al 2005 demonstrated that the effect of the TUFT1 gene combined with the effect of high level of S. mutans increases the susceptibility to dental decay. Strong evidence for association was found for one AMELX marker with higher DMFT and increased age adjusted caries experience by Deeley et al 2008. They concluded that increased DMFS is composed of a combined over representation of specific alleles of a marker of TUFT1 and a marker of AMELX.

Decreased blood levels of mannose-binding lectin (MBL) may cause predisposition to infections and autoimmune diseases. Bagherian et al 2008 found significant association between HLADRB1*04 and ECC. Yu et al 1986 found an association between DMFS increase and saliva levels of a specific proline-rich protein (PRPs), a saliva component that influences the attachment of bacteria. De Soet et al 2008 observed that CD14 genotype was significantly associated with the presence of 4 or more carious lesions.

Identifying the genes that play a role in controlling caries susceptibility is essential for a full understanding of the molecular basis of the disease pathogenesis, and would have potential impact on the
development of new preventive and therapeutic strategies – such as molecular vaccines and even gene therapy. Clinicians would be able to screen and identify susceptible patients, adopting individual, tailor-made intervention with a potential high impact over maintenance and preservation of individual oral health. Finally, the identification of genetic risk factors for caries would help reduce costs associated with treatment and prevention of one of the most frequent oral diseases.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study Population</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Klein and Palmer, 1938</td>
<td>Siblings (4416)</td>
<td>Similarities in caries rate between Siblings</td>
</tr>
<tr>
<td>Klein, 1946</td>
<td>Parents and Children 5400</td>
<td>Offspring dental disease quantitatively related to parents experience</td>
</tr>
<tr>
<td>Klein, 1947</td>
<td>Parents and Children (-)</td>
<td>Similarities in caries rate between</td>
</tr>
<tr>
<td>Book and Grahnen, 1953</td>
<td>Parents and Siblings (317)</td>
<td>Correlation between siblings and parents of caries free individuals.</td>
</tr>
<tr>
<td>Garn et al 1976b</td>
<td>Parents and Children (6580)</td>
<td>Mother-child similarities in the DMFT scores are symmetrically higher than father-child.</td>
</tr>
<tr>
<td>Garn et al 1977</td>
<td>Spouse pairs (1800)</td>
<td>Positive spouse DMFT correlation</td>
</tr>
<tr>
<td>Bedos et al 2005</td>
<td>Mothers and Children (-)</td>
<td>Positive correlation between edentulous mother and their children</td>
</tr>
<tr>
<td>Bachrach</td>
<td>MZ (130) and DZ</td>
<td>No difference between MZ and DZ</td>
</tr>
<tr>
<td>Study</td>
<td>MZ (N) and DZ (N)</td>
<td>Conclusion</td>
</tr>
<tr>
<td>------------------------------</td>
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<td>-----------------------------------------------------------------------------</td>
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<tr>
<td>and Young 1927</td>
<td>(171)</td>
<td>twin pairs.</td>
</tr>
<tr>
<td>Horowitz et al, 1958</td>
<td>MZ (30) and DZ (19)</td>
<td>MZ more alike caries experience than DZ twin pairs.</td>
</tr>
<tr>
<td>Mansbridge, 1959</td>
<td>MZ (96) and DZ (128)</td>
<td>MZ twins with greater similarity in caries experience.</td>
</tr>
<tr>
<td>Goodman et al, 1959</td>
<td>MZ (19) and DZ (19)</td>
<td>Intapair variance of DZ greater than MZ</td>
</tr>
<tr>
<td>Finn and Caldwell, 1963</td>
<td>MZ (35) and DZ (31)</td>
<td>MZ and DZ differences greater for smooth surface caries in anterior teeth</td>
</tr>
<tr>
<td>Bordoni et al 1973</td>
<td>MZ (17) and untreated controls</td>
<td>Greater similarity in morphology and eruption timing in primary teeth between MZ than unrelated controls.</td>
</tr>
<tr>
<td>Gao, 1999</td>
<td>MZ and DZ (280)</td>
<td>Higher correlation in MZ twins but not statistically significant</td>
</tr>
<tr>
<td>Conry et al 1993</td>
<td>MZ (46) and DZ (22) reared apart</td>
<td>MZ with greater within pair similarity than DZ pairs for teeth present, teeth present excluding third molars, teeth restored, teeth restored index, surfaces restored, surfaces restored index and surfaces restored or carious in reared apart twin pairs.</td>
</tr>
<tr>
<td>Boraas et al 1988</td>
<td>MZ and DZ (44) reared apart</td>
<td>Resemblance within MZ for number of teeth present, percentage of teeth and surfaces restored, or carious, tooth size and malalignment.</td>
</tr>
<tr>
<td>Liu et al 1998</td>
<td>MZ and DZ (82)</td>
<td>Strong evidence of genetic influence to third molar presence, tooth size, arch size and upper lateral incisor malformation.</td>
</tr>
<tr>
<td>Bretz et al 2005a</td>
<td>MZ (142) and DZ (246)</td>
<td>For surface based caries prevalence rates, the heritability was strong-76.3 ; for lesion severity the heritability was also strong -70.6</td>
</tr>
<tr>
<td>Bretz et al</td>
<td>MZ (114) and DZ</td>
<td>For surface based caries prevalence</td>
</tr>
</tbody>
</table>

84
rates the heritability was moderate (H=30.0) and greatest for the oldest groups (H=46.3); for lesion severity, the heritability was also moderate (H=36.1) and greatest for the youngest group (H=51.2)

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study population/ Type of study</th>
<th>Candidates region(s)/ gene(s)</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slayton et al 2005</td>
<td>Children dmfs &gt;4 (92) and dmfs=0 (343)/ case control</td>
<td>AMELX, AMBN, TUFT1, ENAM, TFIP11, KLK4</td>
<td>Tuftelin gene and high levels of S.mutans associated with susceptibility to dental caries.</td>
</tr>
<tr>
<td>Pehlivan et al 2005</td>
<td>Children caries free (40) and with carious teeth (42)/ case-control</td>
<td>MBL</td>
<td>No significant difference between two groups and genotypes distribution.</td>
</tr>
<tr>
<td>Zakhary et al 2007</td>
<td>Adult Caucasians (60); Children of Caucasian parentage (89), African American Parentage (96) and Mixed Parentage (23)/ case-control</td>
<td>PRH1 locus (Db)</td>
<td>Db negative Caucasians had significantly more caries.</td>
</tr>
<tr>
<td>Bagherian et al (2008)</td>
<td>ECC children (44) and caries free children (35)/ case contro;</td>
<td>HLA- DRB1, HLA-DQB1</td>
<td>HLA-DRB1*04 was associated with ECC susceptibility.</td>
</tr>
<tr>
<td>Deeley et al (2008)</td>
<td>DMFT≤ 2(44) and DMFT ≥3 (66)/ Case- Control</td>
<td>AMELX, AMBN, TUFT1, ENAM, TFIP11, KLK4</td>
<td>Strong association of AMELX with DMFT≥ 20 and increased age adjusted</td>
</tr>
<tr>
<td>Authors</td>
<td>Study Design</td>
<td>Genes/phenotype</td>
<td>Findings</td>
</tr>
<tr>
<td>-------------------------</td>
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</tr>
<tr>
<td>Patir et al (2008)</td>
<td>dmfs≥4 (91) and dmfs =0 (82) case-control</td>
<td>AMELX, AMBN, TUFT1, ENAM, TFIP11, KLK4</td>
<td>TUFT1 overexpression of T allele and AMELX over representation of the c Allele.</td>
</tr>
<tr>
<td>De Soet et al (2008)</td>
<td>5 groups: Caries free (53); full dental treatment (75); extraction only (66); ART filling only (77); and no treatment (77)/ case-control Study</td>
<td>CD 14-260</td>
<td>Protection effect of the CD 14-260 TT genotype for AFF in children with dmft+ DMFT&gt;4 at baseline</td>
</tr>
<tr>
<td>Peres et al (2009)</td>
<td>Children (245) caries free and with caries (Case-Control)</td>
<td>CA 6</td>
<td>Positive association between buffer capacity and the rs2274327(C/T) Polymorphism.</td>
</tr>
<tr>
<td>Vieira et al (2008)</td>
<td>46 families/ genome wide linkage analysis</td>
<td></td>
<td>Five suggestive loci were identified: -3 for low caries susceptibility,(5q13.3, 14q11.2 and Xq27)-2 for high caries susceptibility (13q31.1 and 14q24.3).</td>
</tr>
</tbody>
</table>

**Dermatoglyphics in Diseases:**

Down's Syndrome (Trisomy 21): Cummins (1939) demonstrated characteristic differences in frequency of dermal configurations between affected and normal children, long before chromosomal basis of diagnosis of Down's syndrome was established. Walker (1957) derived an estimate of the probability, that a child has Down's syndrome by a probability index. It
was derived by multiplying the probabilities for each pattern or after conversion to logarithms, by adding them.

Lu (1968) listed all possible combinations of finger patterns and their frequencies in a group of patients with Down's syndrome and controls and discriminated (89%) of those from without Down's syndrome. Reed et al (1970), in his study, constructed a nomogram using only four dermatoglyphic traits, chosen for their high discriminant values (Borgaonkar et al 1971) developed another method, using predictive discrimination by which 88% of patients with Down's syndrome and 92% of controls were discriminated. Marylin Preus et al in 1972 in his review article concluded that the hands and feet of a patient with trisomy 21 were generally short and broad. There was a high frequency of simian creases, in curved fifth digit (Clinodactyly) with or without a short or missing middle phalynx and a wide space between the first and second toe, with a deep plantar crease. Dermatoglyphic pattern showed an increase in the bilateral "t" 10 ulnar loops on the fingers, radial loops in 4 or 5 fingers. Rajangam et al (1995) studied dermatoglyphic patterns of 235 cytogenetically confirmed patients of Down's syndrome. The data were correlated and compared with 230 controls. Patients total finger ridge counts and 'atd' angles differed significantly from that of the controls. Mostly ulnar loop pattern was observed in the patients. Abnormal dermatoglyphic features such as simian crease, Sydney line and patterns in the hypothenar and interdigital areas have occurred more frequently in the patients. Katznelson et al (1999) carried out study in order to evaluate the dermatoglyphic traits (DT) in males and females with Down's syndrome (DS). The aim of the study was
to explore the possibility of using DT of the parents of DS patients to predict the likelihood of the disease appearing in the offspring. The samples were of DS patients (198 males and 140 females) and their parents (84 fathers and 153 mothers), all Israeli Jews. The prints were collected and interpreted. This included identification of patterns, ridge counts and the measurements of distances and angles in the palm of the hands; 79 DT for every individual: 28 continuous traits, 9 discrete traits, 11 indices of intra-individual diversity (Div), 15 indices of directional asymmetry were estimated. The results were compared between parents and control groups of women and men. The present study found proof of the existence of an additive genetic component in DT, while an increased ridge counts and arch patterns (ulnar and radial loops) was observed in parents of DS patients in comparison to control groups. The DT which is typical to DS patients was confirmed also in parents. Thomas Fogle (2002) performed comparative Dermatoglyphic study between 180 Downs syndrome patient and 180 normal individuals. He concluded that ulnar loops and radial loops were mostly seen on index fingers and ring fingers respectively in Down syndrome compared to normal individuals. Sardool Singh (2005) studied Dermatoglyphics of schizophrenics; patients with Down's syndrome and mentally retarded males were compared with those of normal Australian Europeans. It was noticed that the patients with Down's syndrome separated significantly from the rest of the groups. They showed a significant increase in the ulnar loop patterns compared to normal group.

Matsuyama, Ito (2006) studied each fingerprint type (arch, ulnar loop, radial loop, and whorl) of the parents of children with Trisomy 21
(Fathers:71; Mothers: 128) born between 1965 and 1970 obtained from the Tokyo Medical and Dental University Hospital. Japanese controls were taken from dermatoglyphics data in Japan. Results from a statistical analysis based on the above data showed significant differences, more arches and fewer whorls in mothers of children with Trisomy 21. Among fathers of Trisomy 21 children, a significant difference was found in there being fewer whorls and ulnar loops. Considering the mothers' fingerprints, they suspected that females with a higher frequency of arches and a lower frequency of whorls had a stronger possibility of bearing Trisomy 21 babies. (b) 45, XO Turner's syndrome: Marylin Preus et al (1972) in his review article concluded that individuals with XO Turner's syndrome often had a short fifth finger, dystrophic or hyperconvex nails, shortening of the third to fifth metacarpals and lymphoedema of the hands and feet in infancy. The A-line exists in the thenar area more frequently than in normal individuals with an increase in the atd angle greater than 120 and with an increase in the bilateral hypothenar area.

Kobyliansky et al (1997) studied dermatoglyphic patterns among 57 Turner female patients and compared it with healthy individuals. 79 dermatoglyphic variables for every patient: 28 continuous traits, 9 discrete traits, 10 indices of arch patterns, 16 indices of directional asymmetry and 16 indices of fluctuating asymmetry were estimated. They found that there was significant increase in the ulnar loop patterns among these patients compared to control group.

c) Klinefelter's syndrome (47 XXY) Marylin Preus et al (1972) in their review article concluded that there is a slight increase in height of the axial
triradius in hypothenar patterns, and a decrease in thenar patterns were noted.

d) Pseudo hypoparathyroidism: Forbes (1967) reported an increase in patients with high axial triradius. An increase in arch patterns was noted, but the data was not presented in a form that can be evaluated for clinical use. Marylin Preus et al (1972) in his review article concluded that these patients had short, broad hands and feet, with short metacarpals and metatarsals especially the fourth and fifth.

e) Rubinstein–Taybi syndrome (R-T Syndrome) Marylin Preus et al (1972) in his review article concluded that one of the major diagnostic criteria for individuals with this syndrome were broad thumbs and great toes, a deep plantar crease, overlapping toes, and fifth finger clinodactyly or polydactyly. Dermatoglyphic features include an increase in the radial loop pattern, thenar pattern and bilateral I3 pattern.

Marylin Preus et al (1972) in his review article concluded that Breast cancer is hereditary and earlier studies have revealed two genes BRCA and BRCA2 on the q arm of chromosome 17 at the 36th position. Engler et al (1982), analyzed dermatoglyphic patterns in breast cancer patients, and concluded that the presence of six or more whorls on the finger tips of a person provides a high risk of obtaining breast cancer. Huang (1997) studied Fingerprints of 570 breast cancer cases and the same number of matched controls from the population-based finger print file in Hawaii for studying the association between breast cancer and digital dermal patterns and ridge counts. The results showed that breast cancer patients had a significant excess of radial loops on the left hand. It was also found that the
frequency of ulnar loops on the left hand was significantly elevated for premenopausal women with breast cancer, whereas an excess of radial loops on the left hand was observed for the postmenopausal women with breast cancer. Bierman et al (2003) studied fingerprints of 200 women with histologically proven breast cancer (case group) and compared to fingerprints from 138 women with no history of any malignant disease (control group). Of the patterns analyzed, four were significantly associated with breast cancer: accidentals, transitionals, angled ulnar loops, and horizontal ulnar loops. Of 200 patients in the case group, 27 had one or more accidental prints, 58 had one or more transitionals, 34 had one or more horizontal ulnar loops, and 93 had one or more angled ulnar loop patterns. In 138 control subjects there were 2 with accidental patterns, 21 with one or more transitionals, 6 with horizontal ulnar loops, and 16 with one or more angled ulnar loops. Thus the prints described will represent a non invasive anatomical marker of breast cancer risk. Seltzer (2005) studied fingerprints and palm prints in 78 breast cancer patients, 391 patients at increased risk for developing breast cancer, and 64 control patients for the purpose of finding a pattern that would identify those women with breast cancer or those who are predisposed to its development. A pattern of 6 or more digital whorls was identified more frequently in women with breast cancer than in those without the disease. He concluded that digital dermatoglyphics may have a future role in identifying women either with or at increased risk for breast cancer such that either risk reduction measures or earlier therapy.
Chintamani et al (2007) conducted a study on 60 histo-pathologically confirmed breast cancer patients and their digital dermatoglyphic patterns were studied to assess their association with the type and onset of breast cancer. Simultaneously 60 age-matched controls were also selected that had no self or familial history of a diagnosed breast cancer and the observations were recorded. The differences of qualitative (dermatoglyphic patterns) data were tested for their significance using the chi-square test, and for quantitative (ridge counts and pattern intensity index) data using the t-test. It was observed that six or more whorls in the finger print pattern were statistically significant among the cancer patients as compared to controls. It was also seen that whorls in the right ring finger and right little finger were found increased among the cases as compared to controls. The differences between mean pattern intensity index of cases and controls were found to be statistically significant. Thus they concluded that the dermatoglyphic patterns may be utilized effectively to study the genetic basis of breast cancer and may also serve as a screening tool in the high-risk population.

A recent study (Sridevi et al 2010) on a small group of breast cancer patients in Karnataka revealed that those with breast cancer disposition show an ulnar loop on the little finger of both hands and a ridge count of between 6 and 8.

Reed & Rose (1994), published the replication of a study relating to the combined use of dermatoglyphics and the MMPI tests. The tests indicated that, identical twin subjects with asymmetric (dissimilar) patterns on their left and right hands were likely to suffer from environmental
distresses, than identical twins who had symmetric patterns. Twins with asymmetric palmar patterns were considered to have poorer genetic buffering against environmental factors than those with symmetrical corresponding palmar patterns. These, with the asymmetrical patterns exhibited heightened developmental sensitivity to extraneous environmental stress". Their findings suggested such persons had poorer genetic buffering" and environmental sensitivity differences could be manifested in clinical correlative behaviors of anxiety or depression.

Balgir et al (2001) studied 50 patients with a predisposition to obsessive-compulsive disorder and compared with 50 healthy individuals. They concluded that study group showed a significant increase in the ulnar loop with aridge count of 2-3 on the forefinger or any other finger, and a proximal crease on the palm with a line running right cross compared to control group.

Bets et al (1994) studied dermatoglyphic patterns among group of Russian children with clinically diagnosed diabetes mellitus. Pattern asymmetry was observed in children of both sexes compared to control group. The examined population was characterized by reduced incidence of loop patterns.

Ravindranath et al (1995) studied total finger ridge count, absolute finger ridge count and finger print pattern in 150 maturity onset diabetes mellitus patients and compared to 120 controls. Significant findings were: in males, with both hands combined and separately (i) an increase in radial and ulnar loops and arches (ii) A decrease in whorls. (iii) In females, an
increase in ulnar loops and a decrease in whorls in the left hand were observed.

Vera et al (1995) studied hand and palm dermatoglyphics in 158 insulin-dependent diabetic children. The findings in this group were compared with those in 400 control subjects, with a similar racial distribution. The main dermatoglyphics alterations found in diabetic patients were increase in the number of t'-axial triradii and ulnar loops.

Bali et al (2005) studied dermatoglyphic patterns of 108 male and 65 female patients diagnosed as diabetes mellitus. The control population consisted of 536 males and 234 females from the same population. Their results an increase in the ulnar loop patterns among diabetes mellitus patients compared to control population.

Barta et al (2006) studied Dermatoglyphic features of 180 adults with diabetes mellitus. They found that the loop patterns and arch pattern was mostly seen on the thumb in diabetes mellitus patients compared to healthy individuals.

VI) Dermatoglyphics in cleft lip and cleft palate.

Egle Zarakauskaite et al (2004), in their case control study, suggested that there are some significant dermatoglyphic peculiarities in persons with cleft lipand/or cleft palate (CLP) in comparison with control group. The patterns on thenar eminence in hands of those with CLP were six times rarer than in controls (p<0.05). There was a significant difference (p<0.05) between the control group and persons with CLP by count of all triradii (controls-98%, CLP-87.3%). The main line A ended more often in fields 5' and 5" in persons with CLP in comparison with their parents. There
were significantly more arches, double loops and ulnar loops in with CLP than in control dermatograms. (Matsunga E 1977) Scott NM et al (2005) studied dermatoglyphic prints from individuals with non-syndromic CL/P (n = 460) and their unaffected relatives (n = 254) from the Philippines and China. For both samples three raters designated the patterns as arch, ulnar loop, radial loop, and whorl. Chi-square analysis and standard ANOVA were used to investigate heterogeneity between subjects. The significant associations between particular pattern types and CL/P were not the same in both populations. An increased radial and ulnar loop was observed in Cleft lip and palate patients.

Mathew et al (2005) studied dermatoglyphic patterns of 100 children between age of 5-15 years with no difference between sexes of which 50 consisted of the study group (nonsyndromic children with oral clefts) and remaining 50 consisted of control group (healthy children without any anomalies). Bilateral finger prints were collected and analysed. It was observed that oral cleft individuals had an increased frequency of ulnar loops as the ridge configuration as compared to control group.

Balgir (2006) studied dermatoglyphic characteristics of sixty nine cases of cleft lip with or without cleft palate and twenty eight isolated cleft palate cases. They were evaluated for finger patterns, digital patterns, interdigital patterns, types of C- and D-line. It showed variations in patients and controls. Wider 'atd' angle (more than 30 degrees) and dermatoglyphic asymmetry were noted in the patient groups. There was also a significant increase in the ulnar loop, arch patterns among the cleft palate patients.
Jalali et al (2002) studied the relation between the dermatoglyphic pattern of myocardial infarction patients. A multi-centre cross-sectional study was conducted among 900 patients diagnosed with myocardial infarction. The control group consisted of 900 subjects in patients group, the distribution of dermatoglyphic pattern was 7.2 % arch type, 46.8 % loop type, and 46% whorl type of fingerprints. In contrast, in the control group, there were 3.7%, 50.7% and 45.5% respectively. This result showed a statistical significant increase in the rate of arch type fingerprints in patients with MI roughly two times. Also, in subgroup analysis, the percentage of arch type was significantly increased in left thumb, left forefinger and left ring finger among cases (P < 0. 0001).Their finding indicated that there is a significant relation between the arch types of fingerprint and the risk of MI.

Kuklin (2001) studied the ratios between dermatoglyphic patterns of different types in males and females with and without hereditary diseases of the skin. It was found that ridge patterns of fingers were determined by special polygenes. Patients with monogenic dermatoses (X-linked ichthyosis and autosomal recessive ichthyosiform erythroderma) exhibited a suppressed formation of the loop pattern compared to control subjects. Blackwell (2002) studied palmar and fingerprints of 70 patients with Darier’s disease and 409 normal controls. The dermatoglyphic characteristics of each group were determined and comparisons were made between them. Dermatoglyphic abnormalities were found. There was significant increase in the ulnar loop pattern among the Darier’s disease patients. Cusumano (1983) studied fingertip dermatoglyphic patterns of forty-five patients with atopic dermatitis and compared to those of sixty healthy
individuals. The average number of whorl pattern were detected was significantly higher in the atopic group than in the control group. However, atopic patients with hand dermatitis had, on the average, a greater number of whorl pattern than did control patients. Rodewald (1982) studied finger, palmar, and plantar prints of 8 males with X-linked hypohidrotic ectodermal dysplasia (HED), 8 carrier mothers, 7 sisters, and 1 carrier grandmother and compared with data from 552 controls. The patients with HED and the carrier females had higher incidence of arches on the fingertips, triradii, of hypothenar patterns (especially ulnar loops), and of transversal direction of the main lines on the palms than the control individuals. The affected males were also characterized by severe hypoplasia and/or dysplasia of the dermal ridges ("ridge flattening") the carrier females also showed ridge flattening and hypoplasia.

Kargül et al (2001) studied dermatoglyphic patterns in 3 hypohidrotic ectodermal dysplasia (HED) patients and compared with 45 controls. This clinical evaluation (intraoral and radiological), genetic findings, dermatoglyphic patterns were analysed. The HED patients had a higher incidence of ulnar loop patterns compared to controls.

**Dermatoglyphics in Dental caries:**

Metin Atasu (1998) studied dermatoglyphic configurations in caries-free students and the students with extensive caries and found there was significant difference in dermatoglyphic patterns in these two groups. In other words caries-free students had more ulnar loops on the fingertips and the students with extensive caries had more whorls on the finger tips. The role of heredity in dental caries has been shown in this study.
Somani studied to determine if there is any significant correlation between salivary bacteria interactions, dermatoglyphics and dental caries. They found highly significant difference in loops between the subject (caries) and control groups, since the observed value ($Z_{cal} = 7.9762, 4.0248$) was more than the standard value ($Z_{tab} = 3.79$) at $P < 0.001$ and also observed significant difference between subject and control groups for microbial growth since the observed value ($Z_{cal} = 2.43, 2.09, 2.29, 2.61$) was more than the standard value ($Z_{tab} = 1.96$) at $P < 0.05$. The results of the study inferred that there existed a statistically significant difference between subject and control groups for dermatoglyphics, and *Smutans* levels. The above study had linked two important parameters of caries causation- dermatoglyphics and saliva in children. Sharma et al (2013) in their study on 90 subjects correlated dermatoglyphics and salivary pH. These authors concluded that dermatoglyphics may serve as a noninvasive reliable genetic marker for dental caries.

Abhilash et al (2012) had studied dermatoglyphic patterns in dental caries among 1250 individuals between the age group of 5-12 years. Of the 1250 individuals 625 were in the study group, 625 were in the control group. The authors described that the dental caries susceptibility of an individual increased with whorl pattern (83% correlation). The authors concluded that the dermatoglyphic patterns are efficient and can predict in assessing the risk of susceptibility of dental caries.

To summarise the review, dermatoglyphics is considered as a window of congenital abnormalities and is a sensitive indicator of intrauterine anomalies. The dermatoglyphic patterns have been significantly
correlated with dental caries thereby making it an effective non-invasive tool to study the role of heredity in dental caries. The children and their parents are observed to show similar pattern of occurrence of dental caries. This can be attributed to the genetic inheritance of salivary pH, enzymes, salivary flow and tooth morphology. Literature search has revealed only sparse studies with small sample size predominantly in children with no direct evidence to prove the association between dental caries and finger dermatoglyphics.
METHODOLOGY

ORGANIZATION AND ADMINISTRATION WORKOUT

1) APPROVAL FROM AUTHORITIES

Permission to implement the project was obtained from the concerned authorities, college principals, and HR managers in private sector companies, village panchayats, municipal corporation authorities, school head masters, school teachers and parents of school children.

2) REQUIRED INFORMATION ABOUT STUDY AREAS

All required and relevant information regarding the areas including map was obtained from the census office.

3) SCHEDULE OF THE PROJECT

The project was systematically scheduled to spread over a period of two years. (Months of January, February, June, July and August in 2009 to 2011) A detailed weekly and monthly schedule was prepared well in advance by informing and obtaining consent from authorities of respective rural areas. For phase I an average of 100 subjects, Phase II an average, of 500 subjects and Phase III an average of 400 subjects were interviewed, examined and treated on any given day during the survey period excluding the week ends. Even though a detailed schedule plan was prepared well in advance, few adjustments and changes had to be made while working it out practically.

4) INFORMED CONSENT

Voluntary informed consent was obtained from the parents of selected school children and the school teachers and relevant volunteers before the commencement of the survey.
Ethical committee approval was obtained prior to the start of the study and an informed consent was obtained from all the study subjects. Each step in the study was verbally explained to the participant and adequate opportunities were given for discussion of questions with the interviewer. A written description of the study was shown to the participant for obtaining his/her consent. The consent form was translated into the regional language (Tamil) for the convenience of individuals who did not know English. It was also back translated to check the accuracy of the translation. The consent form was explained to each participant and consent was obtained only after the interviewer was sure that the participant understood and accepted the contents.

5) METHOD OF OBTAINING DATA

The required data, for conducting this study, was collected and recorded using printed proforma. A structured questionnaire proforma was used which included questions regarding personal data, socio-demographic profile and all the probable common risk factors associated with dental caries. This questionnaire in English script was translated into tamil script (local language) by a recognized translator so that it could be used conveniently during field work.

6) DIAGNOSTIC CRITERIA FOR DENTAL CARIES

Dental caries was recorded according to the criteria of dentition status and treatment need index as described by International Caries Detection and Assessment System (ICDAS II)
7) CALIBRATION AND TRAINING

Before the implementation of the project, the principal investigator carried out training of the whole team regarding the criteria for diagnosing the dental caries. A group of subjects were selected and examined for dental caries. Subjects were reexamined on successive days using same diagnostic criteria. The kappa statistics for inter-examiner variability was 0.7 and for intra-examiner variability was 0.8.

8) INFECTION CONTROL

The examiner used disposable mouth masks and gloves during examination. The sterilization of the instruments was done using both chemical and physical methods. Gluteraldehyde – 7.0 gms; 1-6 dihydroxy 2.5 dioxyhexane – 8.2 gms and polymethyl urea derivative – 11.6 gms was diluted by adding 1 part to 9 parts of potable water and the instruments were disinfected using this disinfectant and later sterilization was carried out by placing dental instruments in the pressure cooker. At the end of the day’s clinical examination and treatment, the instruments were sterilized in autoclave.

Sample Size Calculation

Based on the published studies on selected populations in India, the proportion of exposure given dental caries is not present is 7%, and the anticipated odds ratio is 3, and the proportion of exposure when dental caries is present is 18%. For a power of 80% at 5% significance level, the sample size was estimated to be 382. The upper limit of 382 was taken as our target. However, considering dropout rate of 5% due to factors of non-
compliance of patients as we were dealing with children we recruited 400
individuals for the study. Adjusting for 5% drop out the projected sample
size is 402-400. Hence, the Phase I study consisted of 400 children in
study group and 400 children in the control group.

The preparatory phase of the study included training of a team to
conduct the study, obtaining approval from the authorities, obtaining
informed consent and conducting the study. Approval was obtained from
school head masters, school teachers and parents of school children. The
required data, for conducting this study, was collected and recorded using
printed proforma. A structured questionnaire proforma was used which
included questions regarding personal data, socio-demographic profile and
all the probable common risk factors associated with dental caries. This
questionnaire in English script was translated into Tamil script (local
language) by a recognized translator so that it could be used conveniently
during field work.

Training programme

Training was provided on a one to one basis to all the team
members by the principal investigator. Dental caries was recorded
according to the criteria of dentition status and treatment need index as
described by International caries assessment and detection system. The
whole team was trained regarding the criteria for diagnosing the dental
caries. A group of subjects were selected and examined for dental caries.
Subjects were reexamined on successive days using same diagnostic
criteria. Each trainee was evaluated individually. The inter and intra
observer variability was evaluated. The kappa statistics for inter-examiner
variability was 0.7 and for intra-examiner variability was 0.8. The trainees were considered fit to conduct the study only if they achieved good inter observer and intra observer reliability.

**Infection Control**

The examiner used disposable mouth masks and gloves during examination. The sterilization of the instruments was done using both chemical and physical methods. Gluteraldehyde – 7.0g; 1-6 dihydroxy 2.5 dioxyhexane – 8.2g and polymethyl urea derivative – 11.6g was diluted by adding 1 part to 9 parts of potable water and the instruments were disinfected using this disinfectant and later sterilization was carried out by placing dental instruments in the pressure cooker. At the end of the day’s clinical examination and treatment, the instruments were sterilized in autoclave.

**Source of data**

**Phase I**

**Time of Study:** October 2008

**Inclusion Criteria**

Subjects in the age group between 5 – 12 years with no caries, carious teeth / Filled teeth / Extracted due to caries.

**Exclusion Criteria**

- Children and adults with genetic, congenital and developmental anomalies
  - Adults with systemic diseases
  - Patients with hereditary and environmental structural defects in teeth.
Phase I was a case-control study and comprised a total number of 800 cases selected following the inclusion criteria. They were recruited from urban and suburban schools of Chennai. Data was collected from these 800 children between the ages of 5-12 years with no difference between the sexes. Out of 800 subjects, 400 subjects were grouped into study group and 400 subjects were the control group.

The study group included children with dental caries in 5 or more teeth based on the DMFT index (Children with 1-4 caries teeth were not included as per WHO criteria for dental caries survey also local factors may initiate the caries process and the role of heredity may not be very contributory. Local factors like trauma, anachoressis, environmental hypoplasia, orthodontic appliances, Poor Oral hygiene in physically and mentally challenged individuals, disorders and medications leading to xerostomia, radiation etc are other suggestive factors which could initiate caries irrespective of the hereditary factor as recognised by the WHO health information systems.) performed and control group consisted of normal, healthy children without any dental caries. The study was conducted for one week and the data was analyzed within one month period.

**Phase II**


**Type of Study: Descriptive Study**

Urban and suburban population across all socio-economic status in and around Chennai, Hyderabad, Bangalore and Trivandrum.
Since dental caries is the most common disease in dentistry we decided to conduct a survey across the population. The study was conducted in the form of camps across schools, colleges, community camps in residential areas across ages 5-35 years and gender so we can assess the trend in order to test our hypothesis. The survey had been conducted in stages adhering strictly to ethical considerations. The first phase was that permission was sought from the concerned authorities and then subsequently information pamphlets were displayed and distributed among the population. After procuring the informed consent, volunteers were then screened and data was collected.

**Inclusion Criteria**

Subjects in the age group between 5 – 35 years with no caries, carious teeth / Filled teeth / Extracted due to caries.

**Exclusion Criteria**

- Children and adults with genetic, congenital and developmental anomalies.
  - Adults with systemic diseases.
  - Patients with hereditary and environmental structural defects in teeth.

Since dermatoglyphic pattern is reported to be altered in many genetic conditions and the susceptibility of teeth with heredity and environmental defects to dental caries is different from normal condition, such subjects were excluded from the study. Literature reports of possible association of certain systemic diseases like diabetes mellitus,
malignancies apart from the physical state of the subjects made us exclude adults with systemic disease.

The study was planned in 4 major cities which will represent the population in South India. Hence, in order to achieve equal distribution of sample size, the weighted average was calculated based on the total population of the individual cities namely Chennai, Trivandrum, Bangalore and Hyderabad, which was subsequently used to determine the sample size.

<table>
<thead>
<tr>
<th>City</th>
<th>Weighted average of population</th>
<th>Sample Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chennai</td>
<td>4,216,268</td>
<td>1935</td>
</tr>
<tr>
<td>Trivandrum</td>
<td>1,687,408</td>
<td>774</td>
</tr>
<tr>
<td>Bangalore</td>
<td>9,621,551</td>
<td>4415</td>
</tr>
<tr>
<td>Hyderabad</td>
<td>6,809,970</td>
<td>3125</td>
</tr>
</tbody>
</table>

Sample size: 10,250 subjects

**Phase III**

**Time of Study:** March- June 2010.

**Study Design: Cluster Sampling**

**Inclusion Criteria**

Subjects in the age group between 5 – 35 years with no caries, carious teeth / Filled teeth / Extracted due to caries.
Exclusion Criteria

- Children and adults with genetic, congenital and developmental anomalies
- Adults with systemic diseases
- Patients with hereditary and environmental structural defects in teeth.

The states were divided into a few homogeneous regions, comprising of a number of districts, on the basis of agro-climatic factors used by the Planning Commission and the physio-geographic factors used by the Office of the census Commissioner and the Registrar General of India. The total sample of households from a state thus depended upon the number of such homogeneous regions.

A three-stage sampling design was adopted to select 210 rural households from each homogeneous region. The first stage was the random selection of a district from a region. The second was selection of 15 villages with probability proportional to size of the village and, finally selection of 14 households randomly from each selected village.

In the case of the urban sample of 105 households from a homogeneous region, eight blocks/wards were randomly selected from the selected district. From these eight blocks, 15 wards or census enumeration blocks (CEBs) were randomly selected (each CEB has almost equal population). In the next stage, 7 households were selected from each CEB. Again, 105 subjects from each group (5-7y, 12-14y, 15-17y, 25-28y, and 35-45y) were to be examined, with males making up half the number and females the other half.
Armamentarium

The armamentarium used included the clinical kit used for general dental examination along with the materials required to take the fingerprints, magnifying glass.

Method of Collection of Data

Considering the ethical issue and confidentiality of fingerprints of patients, the procedure was explained to the parents of the subjects and permission was obtained through written consent forms before recording the fingerprints. Brief case history with clinical examination and DMFT index was recorded.

Dermatoglyphic patterns of all 10 palmar digits were recorded using Cummins and Midlo (1943) method. The hands were cleaned with soap and water and then scrubbed thoroughly with an antiseptic lotion (Savlon) and allowed to dry. This was done to enhance the quality of the dermatoglyphic prints, by removing sweat, oil or dirt from the skin surface. The student's right palm was pressed in the ink pad followed by pressing it firmly against the bond paper. If the recording was not clear, a second or third recording was made whichever was satisfactory and readable. The same procedure was repeated for the left hand.

Evaluation of Patterns

The various patterns of finger prints were analysed according to the standard guidelines for classification of patterns. The study was designed as a double blinded assessment to increase the validity of results and was done by forensic fingerprint experts, retired from CB-CID, Tamilnadu Police. Each data interpretation form containing the finger outline of both
left and right hand was given to the experts who had the same code as the case sheet with no other clinical information.

The data was recorded and Statistical analysis was performed using SPSS software 16.0. Chi Square test, Pearson Coefficient test and t-test to compare the dermatoglyphic pattern changes between the study group and the control group for statistical significance.