Article

The Critical Stage of Friction Ridge and Pattern Formation

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Abstract

This study provides an enhanced understanding of the biological structure and development of friction ridge skin for the latent print examiner who is called upon to explain the scientific principles of latent print identification as based on permanence and uniqueness. Cellular attachments ensure permanence, while variable stresses and cellular distributions account for individuality on all "three levels" of detail. Volar patterning is dependent upon the tension across the surface of the developing skin during a critical stage of approximately 10.5 to 16 weeks estimated gestational age. Fingerprint ridge counts are predominantly affected by two combined timing events: the onset of epidermal cellular proliferation and the timing of the regression of the volar pads. Fingerprint pattern types are predominantly affected by the symmetry of the volar pad.

Introduction

The accuracy and reliability of many of the forensic sciences are currently being challenged. Specific among these challenges is the admissibility and reliability of the science related to friction ridge skin identification. All latent print examiners should understand and be prepared to recite the philosophy of identification, define the methodology used in examinations, and explain the scientific basis of the permanence and uniqueness of friction ridge skin based upon empirical and scientific research data.

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In the United States, legal challenges relating to the admissibility of friction ridge skin identifications can occur on a case-by-case basis. Until 1993, the standard of admissibility was that the scientific technique or procedure be generally accepted and practiced by the relevant scientific community. *Daubert vs. Merrell Dow Pharmaceuticals* (1993) enhanced the authority of trial judges by giving them "gatekeeper" authority to alleviate possible "junk science" entering the courtroom [1]. Although Daubert issues have surfaced during the trial phase, traditionally, a "Daubert hearing" is conducted pre-trial and outside the presence of the jury. In addition to the relevancy of the subsequent case testimony, several factors may be considered by the trial judge in making a reliability determination regarding scientific principles or methodology including: (1) testing and validation, (2) publication with peer review, (3) error rate, (4) existence of standards, and (5) general acceptance.

Forensic sciences have inadvertently stepped into the limelight of prime time newscasts and television dramas, sparking public interest and debate. Latent print examiners are being questioned about their science not only in legal settings, but in social and public venues as well. As the international community grapples with updating policies and procedures to meet new court challenges, it must also address the concerns of the general public.

It is of the utmost importance that each examiner in the field of latent print examination be able to explain why friction ridge skin is unique and permanent. The traditional response, "because no two fingerprints have ever been found to be the same," is inadequate. Knowledge of the physical and natural scientific basis, rather than observational data alone, must be exhibited to explain why friction ridge skin is unique and why this uniqueness is permanent. This study explores the anatomical structure and growth process of human friction ridge (volar) skin presented in a manner to enhance latent print examiner understanding, and equip the latent print examiner with scientific information to accurately and concisely communicate the principles of the science of friction ridge skin identification.

The Structure of Friction Skin

Like all skin, volar skin is composed of two basic layers: the outer epidermis, and the inner dermis (Figure 1). The epidermis and dermis are separated by a basement membrane, which serves

as a boundary and a mechanical linkage between these two tissue layers. The epidermis is further divided into five cellular layers based on intrinsic changes in the cells as they progress from the bottom of the epidermis to the surface of the skin. The dermis is divided into two layers, reflecting differences in fiber composition, cell type and distribution, and vascular networks [2].

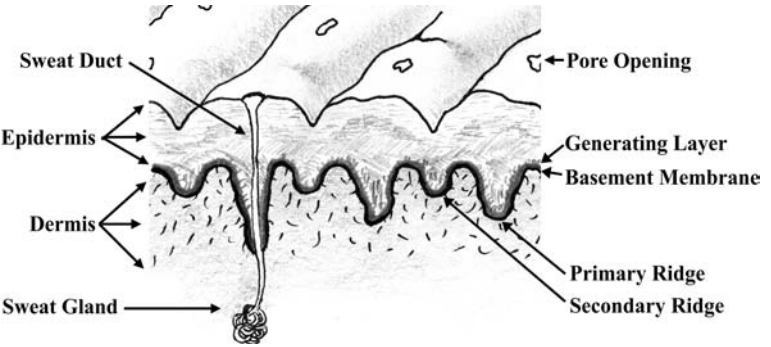

Figure 1

Three dimensional representation of the structure of mature volar skin.

The surface ridges and furrows of friction skin reflect, to a certain degree, the complex organization of the epidermis below the surface. The basal layer of the epidermis of friction skin has a series of folds protruding into the dermis, which correspond to the ridges and furrows on the outer surface of the epidermis. The ridges or folds of the basal layer containing ducts from the eccrine sweat glands of volar skin are termed primary ridges, and correspond to the surface ridges of friction skin. Secondary ridges, alternating between primary ridges, also protrude into the dermis, but correspond to the furrows on the surface of the skin.

Branches of epidermis, termed "anastomoses" bridge primary and secondary ridges and mold the papillae pegs of the dermis [3]. The series of primary and secondary ridges, accentuated by the anastomoses, increase the surface area of attachment between the epidermis and dermis. New epidermal cells are constantly generated in the basal layer and pushed toward the surface [2]. These new cells replenish the upper layers of epidermis and consistently reproduce the complex arrangements present at the epidermal-dermal junction.

The structure of volar skin is extremely complex, and is very difficult to represent in a two-dimensional drawing. Available literature contains representations of the structure of friction skin that, on first glance, appear to conflict with regard to primary, secondary, and surface ridge configuration. In some drawings of the structure of fetal skin, "double rows" of dermal papillae appear to correspond with surface furrows (Figure 2A), [4, 5, 6] whereas other drawings show the double rows as residing beneath the surface ridge itself (Figure 2B) [7, 8]. However, these apparent diagrammatical discrepancies can be resolved when the growth of friction ridge skin is viewed with the added consideration of the passage of time (Figures 2C and 2D). Further, Chacko noted skin of different structure in different individuals of the same age, as well as in different areas of friction ridge skin of the same person, designating them as types "DR0, DR1, DR2, and DR3" (Figure 2) [9].

The Principle of Permanence

The key to understanding why friction skin retains its features throughout natural growth and aging lies within the structure of skin. There are three principal structural elements of skin that allow for the permanence of friction ridge detail: (1) the adherence of the epidermal cells to each other, (2) the basal cell layer of the epidermis, and its attachment to the basement membrane, and (3) the attachment of the basement membrane to the dermis. A general discussion on the cellular structure of skin is included to demonstrate the specific attributes that enforce permanence.

Epidermis

Epidermis of volar skin is approximately 1.8mm in thickness [8], and is comprised mostly of cells called *keratinocytes.* The keratinocyte undergoes a maturation process, termed differentiation [2], as it moves from the basal layer of the epidermis to the surface, where it sloughs off (exfoliates) into the atmosphere. The term *differentiation* represents the progression of the keratinocyte from a newly generated cell, through synthesis and accumulation of the protein keratin, and, finally, to a dead, completely keratinized (cornified) cell [10].

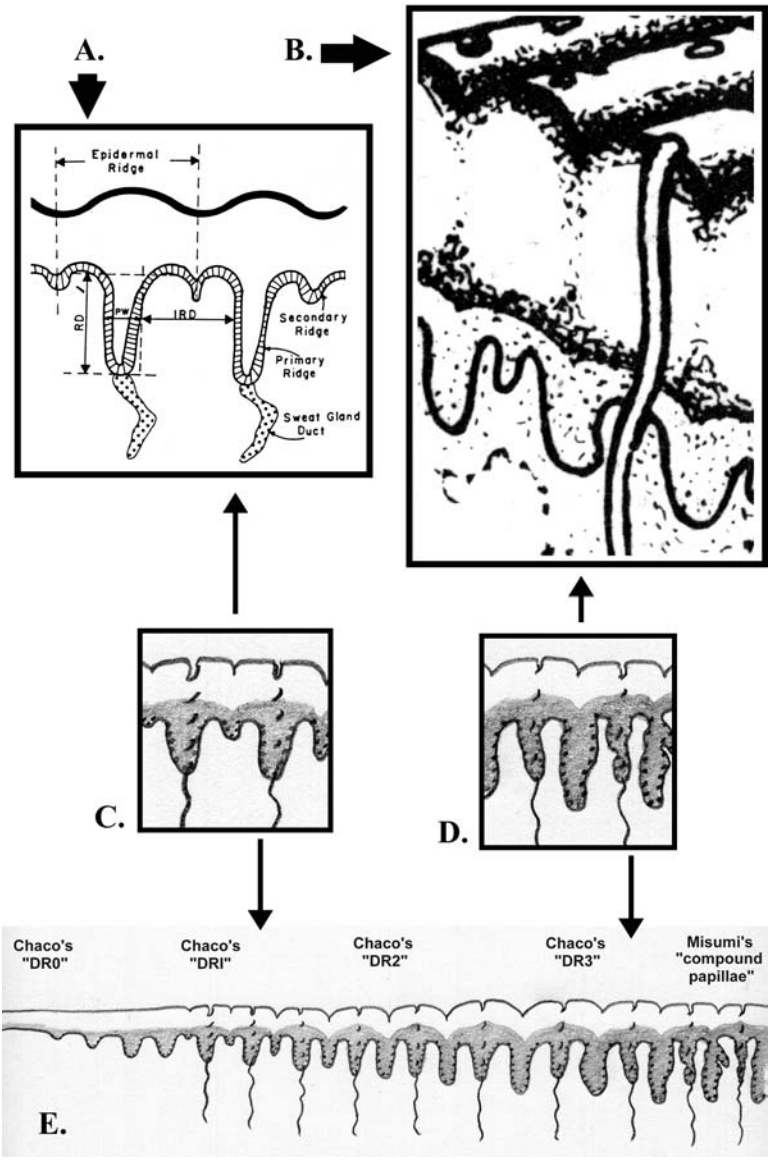

Figure 2

A: Drawing of fetal volar skin structure [4] B: Drawing of adult volar skin structure [7] C: A section of E which corresponds with A D: A section of E which corresponds with B E: An illustration of the progression of volar skin from DR0 (left) through DR3 (right) [9] and representing papillae of different sizes and shapes (right) [5]

The *stratum germinativum* (generating layer) is the basal layer of keratinocytes of the epidermis. It is a single layer of columnar shaped cells attached to the basement membrane. These cells undergo mitosis (cell division) and, because of the direction of division in these columnar cells, the new cells are pushed upward within the epidermis to replace exfoliated cells. Basal cells are joined to each other by complex cell junctions called *desmosomes* and are joined to the basement membrane by another type of junction called a *hemidesmosome* [2].

After dividing from the basal cells of the generating layer, keratinocytes are pushed upward and become part of the *stratum spinosum* (spinous layer), a zone 2 to 4 cell layers in thickness. Cells of the spinous layer exhibit the first stage of differentiation by organizing the necessary components of keratin synthesis. These cells are bound to one another by abundant desmosomes [2].

As cells progress toward the external surface of the epidermis, they become part of the *stratum granulosum* (granular layer). The cells of this layer are the last of the living cells of the epidermis, as they begin to display the first precursors of keratin. As keratinization occurs, all other cellular activities and components degrade, marking the beginning stages of cell death. Some researchers define the small layer of histologically clear cells at the top of the granular layer as the *stratum lucidum*, or Hyalin layer [11]. The cells remain linked by desmosomes as they progress into the *stratum corneum* (horny layer) [2].

By the time the cells have reached the horny layer of the upper epidermis, they have accumulated keratin, and cell death has occurred. The horny layer of adult volar skin can be up to 100 cells in thickness [2], making it 25 to 30 times thicker than most other areas of the body [12]. Cornified, dead cells are large and flat (Figure 3), overlap at the margins, and are joined to underlying and superficial layers through interlocking undulations and modified desmosomes [2]. The entire progression through the different stages of cell maturation, from cell birth to exfoliation, is approximately 30 days [12]. Individuals who live to be 60 years old will undergo complete turnovers of epidermis approximately 720 times in their lifetime. This fact reinforces the need for latent print examiners to understand why ridge detail is consistently represented on the surface of the skin, year after year.

The first structural element that enforces the permanence of friction ridges (and other skin features) is the complex and secure junction between cells of the epidermis. Cells generated from the basal layer are surrounded by and "cemented" to neighboring cells in the epidermis, and remain so until exfoliation occurs [11, 13]. The positional properties of the basement membrane zone are consistently transferred to the surface because individual cells move upward in concert with surrounding cells.

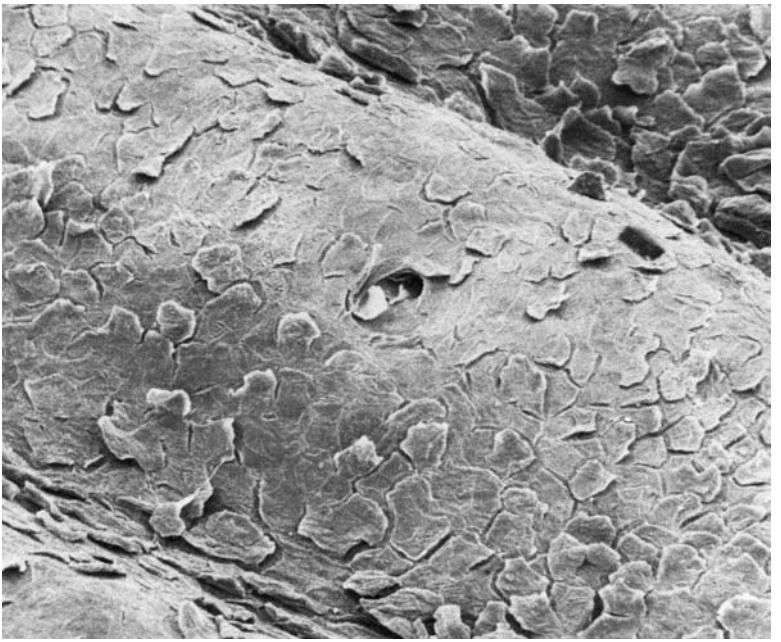

Figure 3

Scanning electron view of the surface of a friction ridge showing cornified epidermal cells undergoing exfoliation, and the orifice of a sweat gland. [11]

Basement Membrane

The second structural element of permanence is the attachment site between the basal cells of the epidermis and the basement membrane. Cell junctions and small fibers prevent the basal cells from sliding or migrating along the basement membrane. Further, the rate at which a basal cell produces new epidermal cells is reasonably consistent. For normal healthy tissue, a small isolated area of basal cells will not suddenly begin to produce more or less epidermal cells than it previously did, thereby causing a different formation on the surface ridge. This concept is supported by 100 years of observational data demonstrating that ridge detail is not subject to variations over time, barring growth, disease or injury.

Dermis

The dermis lies directly beneath the basement membrane and is anchored to it by penetrating fibrils and bundles of microfibrils. The dermis is divided into two regions, the upper papillary dermis and the lower reticular dermis. Both regions of the dermis are composed of fibrous and nonfibrous dermal matrix. The fibrous material (primarily collagen and elastic fibers) gives the skin bulk and tensile strength while allowing for flexibility. The nonfibrous material forms a ground substance which influences the passage of nutrients, allows for cellular migration, and provides a continuous medium for structural fibers [2].

The third structural element attributing to permanence is the attachment of the dermis to the basement membrane through small, anchoring fibers. The extensive network of fibrils and microfibrils prevents the dermis from sliding along the basement membrane.

Basement Membrane Zone Properties, Aging, and Third Level Detail

The epidermal-dermal junction is referred to as the basement membrane zone (BMZ). The BMZ is composed of constituents from the epidermis and the dermis and separates the two layers. In addition to its structural function, the BMZ acts as a filter between the epidermis and dermis. All nutrients, waste, and chemical

signals to and from the epidermis must pass through the basement membrane zone [2].

The epidermal-dermal junction becomes more complex with age. Dermal papillae increase in number and become arranged in a progressively more crowded state throughout adulthood [5, 9, 14]. These changes most likely reflect the continued addition of anastomoses between primary and secondary epidermal ridges, providing enhanced attachment sites for aging skin.

It is important for the latent print examiner to realize why changes in papilla configurations do not affect the detail represented on the surface of the skin. Certain concepts regarding the basement membrane zone must be presented and understood on a cellular level to address this issue.

It is easy to visualize a three-dimensional structure beneath the surface that exactly mirrors surface ridges, but this is not entirely accurate. Surface ridge configurations, down to third level detail (ridge and pore shape), are not solely related to a similar shape along the basement membrane. They are also rooted in the configuration and type of basal cells which feed new epidermal cells to the surface. These are very different concepts.

In an effort to differentiate the concepts, imagine a row of 10 devices and that each rolls a marble across a table at a constant interval. Further imagine in this analogy that each of these devices rolls marbles at different rates: one marble per second, two per second, three per second, etc. At the end of the table are 10 baskets that collect the marbles from each device. If the devices are started at the same time and stopped 5 minutes later, the number of marbles in each basket will be different. If the devices were misaligned to slightly different distances from the edge of the table, and the experiment were re-run, the number of marbles in each basket after 5 minutes would not be significantly different than the previous run; perhaps only by a marble or two. On the other hand, if the production rates of the devices were changed, large differences would be expected in the numbers of marbles in each respective basket. The rates of production of the devices have a much more drastic effect on the end result than simply changing their position on the table.

When visualizing the three-dimensional epidermal-dermal junction, whether an area is concave or convex does not appear to be as important as the basal cell types present and the mitosis rates of the different (heterogeneous) basal cells in determining the three-dimensional detail on the surface within that particular area. The devices in our analogy mimic basal cells, which do not supply new cells at the same rate [2, 13]. Stem cells directly underneath primary ridges (and not secondary ridges) in monkey palms have been shown to give rise to "transient amplifying cells" which, themselves, "undergo a few rounds of cell division" before differentiation [15]. It is important to understand that even though differences in the rate of cell proliferation may occur over time [13], those changes affect relatively large areas of epidermis, therefore, a particular detail in an isolated area would not change. In short, basal cells constantly produce new cell growth at rates proportional to surrounding basal cells [3].

Although latent print examiners may not be able to relate the scientifically-technical details of the mechanisms of human basal cell mitosis, they should be able to demonstrate in lay analogies the complex nature of the epidermal-dermal junction. This communication would explain why third level detail remains permanent during the aging process of skin, in spite of changes in the shape of the epidermal-dermal boundary.

The Principle of Individuality

The individuality of friction ridge skin, or all skin for that matter [13], falls under the larger umbrella of biological uniqueness. No two organisms are exactly alike. The intrinsic and extrinsic factors affecting the development of any individual are impossible to duplicate. The most obvious example is the fact that monozygotic twins are each unique, distinguishable individuals.

Just as homes built from the same blueprint are not the same house, individuals with identical DNA are not the same person. The same blueprint can be used to build six homes, but the final outcome of each home depends on available materials, manner of construction, and a host of environmental factors too numerous to even fathom. Although the six homes will be similar, they will not be exactly alike. The boards of the wood frame will not be in the same exact position, the thousands of nails and screws will not be

in the same exact locations, and the rooms will not have exactly the same dimensions.

DNA works in a very similar way. It provides a blueprint for assembling proteins. These proteins direct the cell's activities by facilitating biochemical processes within the cell. These processes not only depend on the protein derived from the gene, but also the many other components of the cell such as sugars, lipids, non-protein hormones, inorganic elements (e.g. oxygen), inorganic compounds (e.g. nitric oxide), and minerals. Additionally, the physical environment around and within cells, such as surface tension, electrical charge, and viscosity, contribute to the way the cell functions [16].

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 which define the species). However, within the species, for each aspect of an individual's appearance, there are many genes and external factors that 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 the many non-genetic components of the cell and react with each other in complex biochemical pathways during the growth and development of the fetus [16]. These biochemical pathways proceed under the omnipresent influence of external factors.

Although DNA is crucial for providing the blueprint for the development of a particular model, there are so many steps between the genesis of the DNA-encoded protein and the final product, that even the same DNA blueprint produces two completely unique models. With respect to fingerprint patterns, one milestone along the pathway from blueprint to model involves the development of the central nervous and cardiovascular systems.

Trigger Mechanism for the Onset of Friction Ridge Proliferation

At the time of embryonic friction ridge formation, the central nervous and cardiovascular systems are undergoing a critical period of development [17]. Many researchers have reported the appearance of nerve endings (innervation) at the sites of ridge formation

immediately preceding the appearance of ridges, and suggest this could be the trigger mechanism for the onset of cell division (proliferation) [18, 19, 20, 21]. Several researchers even postulated that the distribution of the capillary-nerve pairs at the junction of the epidermis and dermis *directly* influences the alignment of the primary ridges [19, 20, 21]. Earlier research on pattern distribution established "developmental fields," or groupings of fingers on which patterns had a greater tendency to be similar [22, 23, 24]. Later discoveries confirm the neurological relation of spinal cord sections C-6, C-7, and C-8 to innervation of the fingers [25]. This offers even more support of the link between innervation and volar patterning (dermatoglyphics).

The presence of nerves and capillaries in the dermis prior to friction ridge formation may be necessary for friction ridge proliferation. It would seem that all relevant areas of the developing fetus must be in communication with the central nervous system or the endocrine and exocrine (hormone) systems in order to orchestrate complex simultaneous productions such as friction ridge formation [26]. However, it is doubtful that the nerves and/or capillaries independently establish a map that directly determines subsequent ridge flow. It seems more likely that the alignment of the nerves and/or capillaries is directed by the same stresses and strains of the developing hand that establish ridge alignment [26, 27].

Another theory proposes that the same forces of compression on the deeper layers of the epidermis which condition ridge alignment also stimulate the proliferation of basal cells [28]. It is well recognized in cell biology that physical pressure on a cellular system can trigger electro-chemical changes within that system itself. Merkell cells occupy the epidermis just prior to innervation along those pathways [2], suggesting that even this early in fetal formation the stresses created by the different growth rates of the dermis and epidermis are causing cellular activity along invisible lines of stress which already delineates pattern characteristics. Regardless of the trigger mechanism controlling the onset of the first primary ridge proliferations, the structure of the skin during this critical stage of development has been well documented.

Primary Ridge Structure and Genesis

Prior to ridge development, the embryonic epidermis is three to four cell layers thick, and smooth on its outer surface (periderm) and its inner surface (dermal-epidermal junction). Keratinocytes are tightly bound to each other by desmosomes and the cells of the basal layer are attached to the basement membrane by hemidesmosomes [2]. At around 10 to 10.5 weeks Estimated Gestational Age (EGA), basal cells of the epidermis begin to rapidly divide [29]. As the cells proliferate, shallow "ledges" [3] can be seen on the bottom of the epidermis which already delineate the overall patterns that will become permanently established on the volar surfaces several weeks later [29, 30].

During embryonic development, primary ridges are the first visual evidence of interaction between the dermis and epidermis, and are first seen forming as continuous ridges (Figure 4). The prevailing theory of events prior to the visualization of primary ridge structure involves centers of active cell proliferation (Figure 5), which will be the center of sweat gland development [3]. Under this theory, the "units" of rapidly multiplying cells increase in diameter, somewhat randomly growing into one another (Figure 6) along the lines of stress and strain relief that run perpendicularly to the direction of tension. 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 [13].

In 1904, Inez Whipple presented research detailing a theory of evolutionary progression of the volar surface. Ashbaugh succinctly summarizes Whipple's proposition of the evolutionary genesis of friction ridges:

> "Early mammals were covered with a scale-like skin surface. Each scale had one hair protruding from it and an accompanying oil or sebaceous gland. On volar areas, which are the bottoms of the hands and feet, hairs slowly disappeared due to surface use. The pore that was related to the hair changed from a sebaceous gland to a sweat gland. Its purpose, to keep the surface skin damp which enhanced the grip of the volar surface.
>
> Starting in all likelihood as a mutation, scales started to line up in rows and fuse together. This further assisted the

grip of the skin surface by increasing friction. Through natural selection, this mutation became prevalent. Scales slowly evolved into wart-like units with pore openings near the centre. The fusing of these wart formations into rows is the predecessor to the friction ridge, the individual wart being the equivalent of a ridge dot." [31]

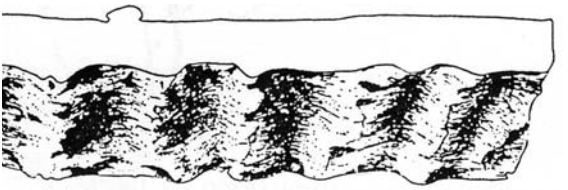

Figure 4

Reconstruction of the underside of fetal volar epidermis, displaying the first three-dimensional model of the undulations at the epidermal-dermal junction [3].

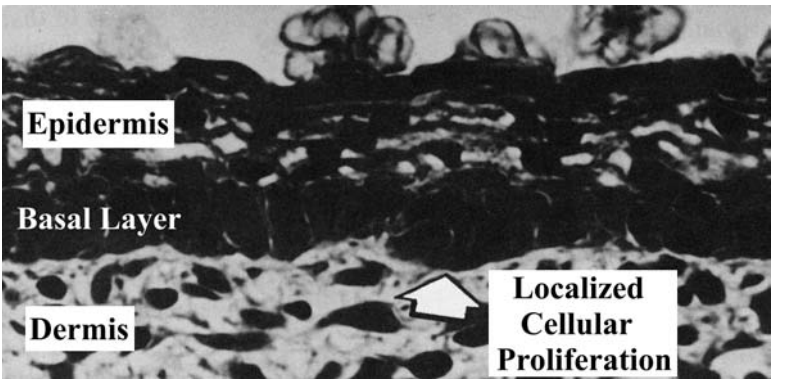

Figure 5

Histologic cross section of 10.5 week EGA fetal volar skin at the onset of cellular proliferation [29].

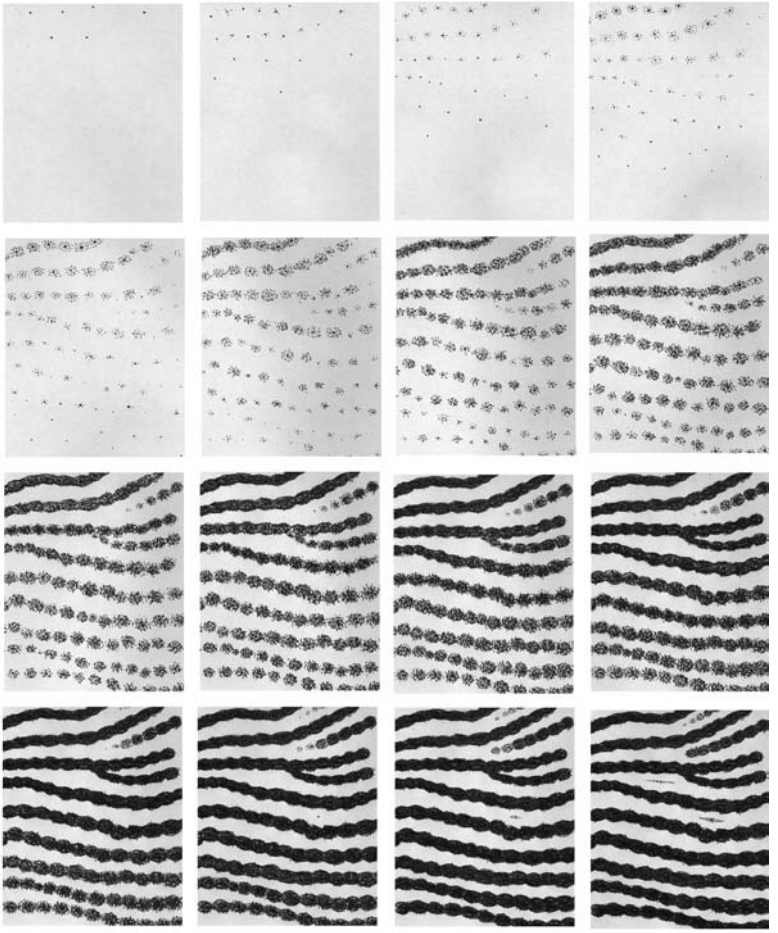

Figure 6

This series of drawings represent the currently accepted theory that localized cellular proliferations grow together into what will subsequently appear as ridges at the epidermal-dermal junction at around 10.5 weeks EGA.

Thirteen years after Whipple's *phylogenetic* (evolutionary history) theory was presented, researchers diverged from her theory and presented an *ontogenetic* (developmental or embryonic history of an individual) model, suggesting that fusion of warts into ridges occurs during the embryonic development process [32]. In 1926, Cummins refuted the ontogenetic scheme [33]. However, Hale later included the ontogenetic model in his conclusions [3]. Literature since that time has been mixed. The fact remains that, currently, the first visual evidence of interaction between the dermis and the epidermis clearly demonstrates ridges forming as ridges, not a series of units protruding into the dermis (Figure 4). Perhaps with advances in technology, the theory that localized cell proliferations grow together into linear ridges before the appearance of the structure as a ridge will be demonstrated. Until then, this will have to remain a possible model of development which could provide individuality prior to 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 [13], with the term "Localized Proliferation" being used to describe theoretical events of fetal formation [34].

Primary ridges mature and extend deeper into the dermis. Initial formation of primary ridges begins at about 10.5 weeks EGA and continues until about 16 weeks EGA, at which time secondary ridges begin to form. Although the exact mechanisms by which minutia (Galton's details, second level details) form is unclear, observational data by many researchers examining fetal tissue provides a detailed visual account of the structure of friction ridge skin in successive stages of the development process. The general consensus of the literature in detailing the formation of second level detail is represented in Figure 7. Many things are happening during this period of primary ridge growth. The finger is growing, new primary ridges are forming across the finger, and the existing primary ridges are beginning to separate due to growth of the digit. As existing ridges separate, a demand for new ridges is created because the surface has a tendency to be continually ridged at this point in development. New ridges pull away from existing primary ridges [3] to fill in these gaps, creating bifurcations by mechanical separation. Ending ridges form when a developing ridge becomes sandwiched between two established ridges. Under this theory, "fusion between adjacent ridges [which have already formed] seems improbable, although there is no evidence for or against this process." [3]

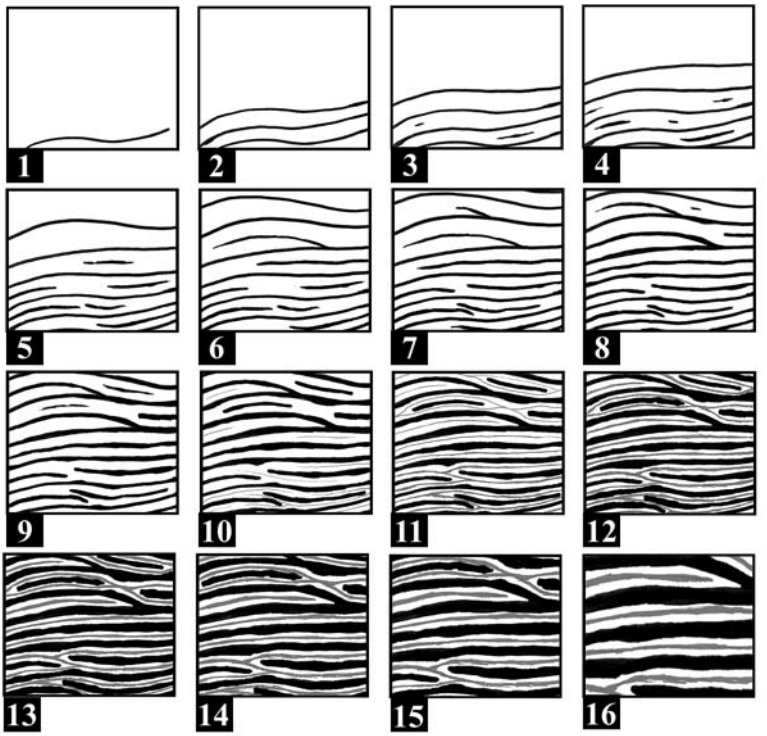

Figure 7

This series of drawings represents the consensus of the literature in demonstrating the theoretical formation of minutia arising from expansion of the volar surface and the tendency of volar skin during the critical stage (frames 1 - 10) to remain continuously ridged. Once secondary ridge formation begins at about 16 weeks EGA (frame 10), the minutia becomes set and the ridges will only increase in size during maturity.

An alternate theory is that all minutia form within the pattern from the onset of proliferation, remain transient during the critical stage, and become permanently set upon secondary ridge formation. The difference between the two theories is subtle: bifurcations would occur as a mechanical separation in the first theory versus a static formation in the second. Under this static theory, both bifurcations and ridge endings could form as a result of the random fusion of the localized cell proliferations, or by another mechanism such as chemical reaction-suppression models.

Regardless of which theory of minutia formation is considered (mechanical or static, fusion or chemical), the placement of any particular second level detail within the developing ridge field is governed by a random series of infinitely interdependent stresses, strains, and tensions across that particular area of skin at that critical moment. Slight differences in the mechanical stress, physiological environment and/or variation in the timing of development could affect any particular minutia placement in that area of skin.

Secondary Ridge Formation

Sweat glands begin to appear around 14 weeks EGA as the existing primary ridges increase in width and continue to penetrate the dermis [29]. By 15 weeks EGA, the primary ridges are experiencing development in two directions: the downward penetration of the sweat glands and the upward push of new cell growth. Between 15 and 17 weeks EGA, secondary ridges appear between the primary ridges (Figure 8) on the underside of the epidermis [29]. At this point in fetal development, the randomly located minutia within the fingerprint pattern become permanently set [3], marking the end of new primary ridge formation [35] and the end of the critical stage.

Secondary ridges are also cell proliferations resulting in downfolds of the basal epidermis. 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 the tension created by cell adhesion, cause infolding of the epidermal layers above the attachment site of the secondary ridges [3]. These infoldings are progressively mirrored on the surface as the furrows

of friction ridge skin, as secondary ridges continue to mature from 16 to 24 weeks EGA [36].

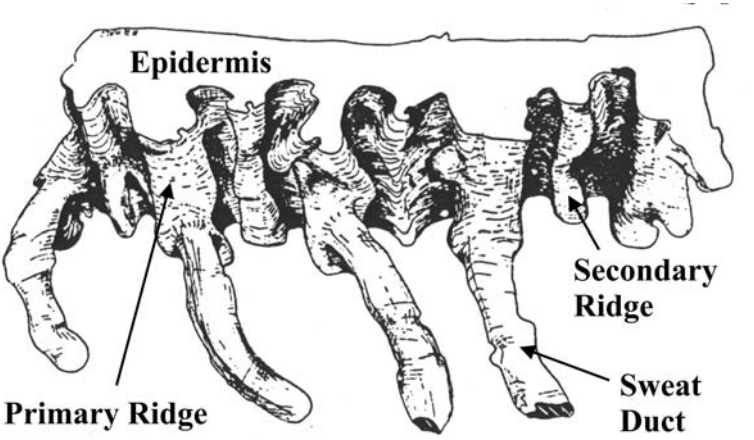

Figure 8

Reconstruction of the underside of fetal volar epidermis, displaying primary ridges with sweat duct formations and the beginning of secondary ridge formation [3].

Incipient Ridges

Little is known about the morphogenesis of incipient (nascent, interstitial, rudimentary) ridges, but several theories could account for their presence in volar skin. Because primary and secondary ridge formation are separate timing events, incipient ridges could be a result of an abnormal transition from primary ridge formation to secondary ridge formation at about 16 weeks EGA. One plausible mechanism of incipient ridge formation would involve a small period of time between primary and secondary ridge formation when no new primary ridges are forming. Such a window of time would allow the existing primary ridges to mature slightly before secondary ridges form, locking in the detail. Under these circumstances, incipient ridges could simply be the result of a malfunction in whatever mechanism signals the cessation of new primary ridge formation. Those primary ridges that are in the earliest stages of

development when secondary ridge formation begins would become incipient ridges. Another plausible mechanism of incipient ridge formation would involve a malfunction in the timing of secondary ridge formation, causing it to begin twice. The original secondary ridges would then have a secondary ridge on each side of them, causing their structure to become that of a small primary ridge. Either of these mechanisms could account for the presence of incipient ridges as an abnormal timing event during formation that would manifest itself across the entire volar area under development at that time. Further, both mechanisms are consistent with the observation that incipient ridges are inherited [37], as timing abnormalities could very well be based in genetics. Regardless of the mechanism of incipient ridge formation, they are based in the same structure as the surrounding friction ridges, and, therefore, inherit the principles of permanence and individuality that allow for their use in the identification process [38, 39]. Some examiners may be reluctant to rely on incipient ridges when comparing two prints due to absence of some detail in one of the exemplars. The two-dimensional representation of incipient ridges is more susceptible to being affected by deposition pressure due to their size difference and location between two larger structures.

Maturation Process

After maturation of the primary and secondary ridges at 24 weeks EGA, anastomoses begin to cross through the dermis [3], linking primary and secondary ridges and molding the upper portion of the dermis into papillae pegs (Figures 9 and 10).

As the skin progresses through this entire process of formation (Figure 11), a nearly infinite number of factors contribute to the end result: complete biological uniqueness, from ridge path down to the structure and shape of a single ridge and beyond. To say that duplication of the entire process of biological formation could occur in any given piece of skin and be indistinguishable from another piece of skin would be equivalent to an identical dump truck load of sticks being scattered twice along the same stretch of road, and saying each stick could land in the exact same position both times.

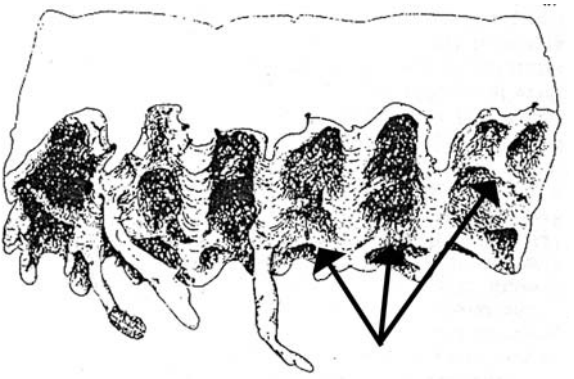

Figure 9

Reconstruction of the underside of the epidermis of fetal volar skin, the arrows demonstrating sections of epidermis which have bridged primary and secondary ridges, cordoning off sections of dermis commonly referred to as "papillae pegs" [3].

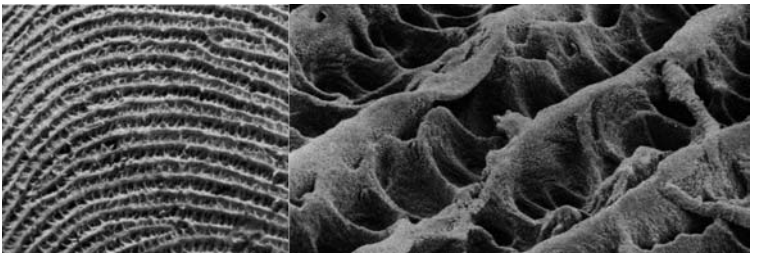

Figure 10

SEM image of the complex undersurface of the epidermis (approximate magnifications left: 8x and right: 80x) [11].

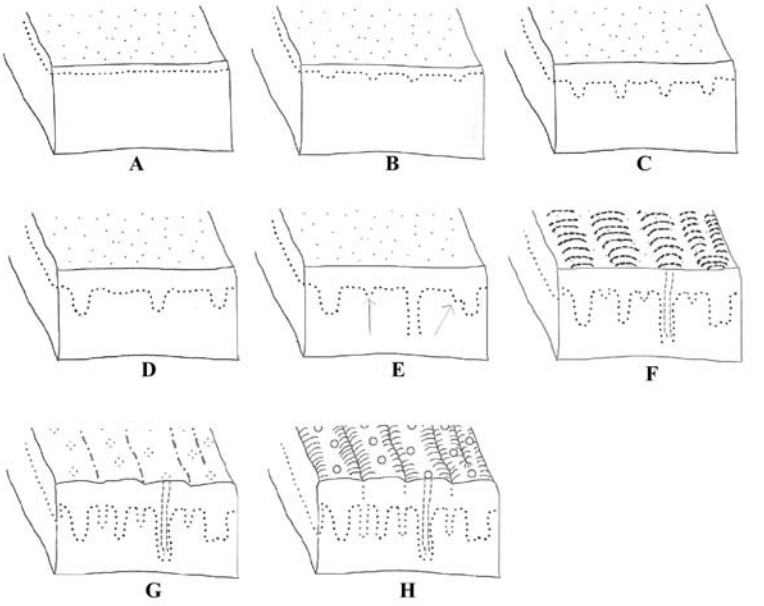

Figure 11

This series of three-dimensional drawings represents the epidermal-dermal junction and the surface of the skin before (A), during (B-E), and after (F-H) the critical stage of friction skin formation. A: The epidermis remains undifferentiated until about 10-11 weeks EGA. B: Primary ridge formation at the epidermal-dermal border, protruding into the dermis. C: Primary ridges continue to penetrate the dermis. D: The skin grows during the critical stage, separating existing primary ridges. E: New primary ridges are thought to form, pulling away from and between existing ridges. F: Secondary ridges begin forming between primary ridges at around 16 weeks EGA, and new primary ridge formation ceases. Surface ridges begin to take form as secondary ridges proliferate downward. Sweat gland ducts have begun to form by this time. G: Secondary ridges continue to mature and surface ridges continue to form. H: By about 24 weeks EGA, the secondary ridges are approaching the depth of the primary ridges, and the entire system begins the maturation process.

Pattern Formation

It is seen throughout the physical world that ridges tend to align perpendicularly to physical stress across a surface (Figure 12). Ridges also form transversely to the lines of growth stress in friction skin. The predominate growth of the hand is longitudinal (lengthwise), subsequently, ridges typically cover the volar surface transversely (side to side), as seen in the ridge flow in the joints of the fingers. However, localized eminencies on the volar surfaces of the hands and feet create stresses in directions other than longitudinal, and, therefore, redirect the flow of the ridges in a complex manner across these three-dimensional structures.

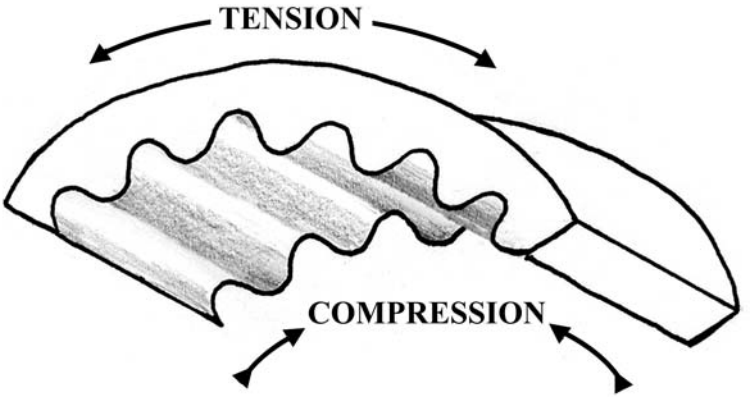

Figure 12

When a semi-flexible membrane is flexed, ridges form transversely to the forces of tension (on the top) and compression (on the bottom).

Development of the Hand and the Volar Pads

The hand changes topography greatly during the initial phases of formation (Figure 13). At approximately 5 to 6 weeks EGA, the hand is a flat, plate-like structure with thickenings of tissue that show the contours of what will become fingers. From 6 to 7 weeks EGA, these thickenings begin to form the bone models (cartilage at this stage) and muscular components of the hand. Also during this time, the fingers begin to separate and the first volar pads appear on the palm. Volar pads (Figure 14) are transient swellings of mesenchymal tissue under the epidermis (this tissue later forms the dermis) on the palmar surface of the hands and soles of the feet of the human fetus (Figure 15). The interdigital pads appear first, around 6 weeks EGA, followed closely in time by the thenar and hypothenar pads. At approximately 7 to 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. By 8 weeks EGA, the bone models begin to ossify and the joints begin to form between the bones of the hand. By 8.5 weeks EGA, the hand has an external morphology similar in proportion to the infant [29]. The pads remain well rounded during their rapid growth until about 9 to 10 weeks EGA, after which time they begin to demonstrate some individual variation in both shape and position. These timed events represent the general consensus of the researchers actually observing fetal tissue [4, 33, 34, 40].

As a result of their slowing growth, the pads become progressively less distinct contours on the more rapidly growing hand (Figure 16). This process is referred to as “regression,” but it is important to understand that the pad is not actually shrinking. The volar pads of the palm begin to regress first, followed 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 [29].

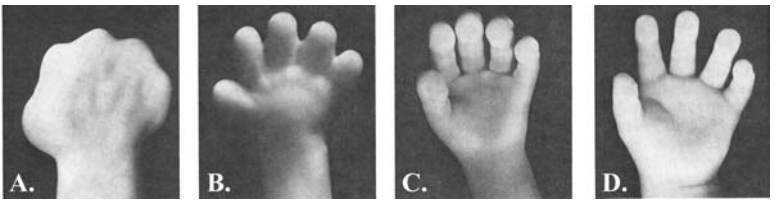

Figure 13

Growth of the hand begins from a paddle-like form (A), continues as the fingers take shape (B), the volar pads form (C), and continues to mature (D) [40].

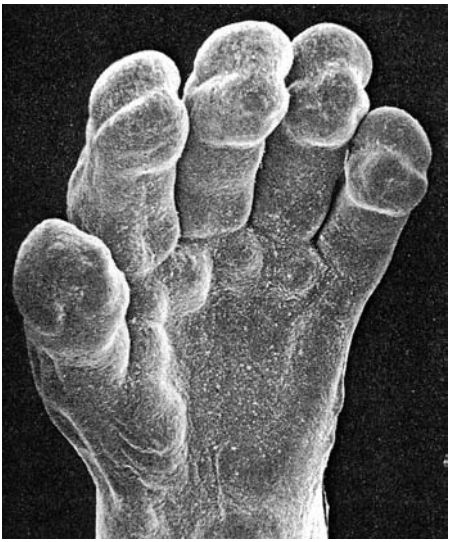

Figure 14

SEM view of the hand of a fetus, displaying prominent volar pads [65].

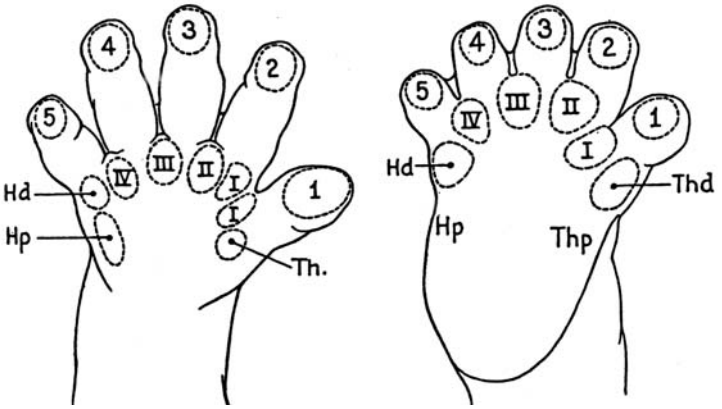

Figure 15

There are normally 11 volar pads on each limb (one on each digit and six on the palmar and plantar surface.) In Cummins' diagram above, he has noted that the hypothenar pad of the palm is divided into a distal (Hd) and proximal (Hp) portion, and that the first (I) interdigital volar pad (associated with the thumb) is also divided into two portions, making 13 distinct elevations on each palmar surface. On plantar surfaces, the proximal portions of the hypothenar pad (Hp) and the thenar pad (Thp) are absent, leaving 11 distinct plantar elevations [40].

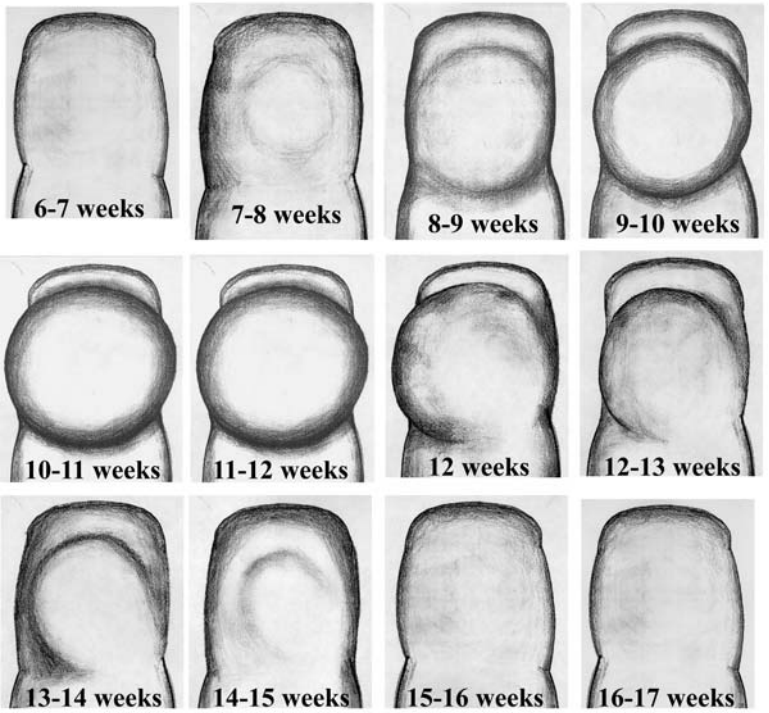

Figure 16

The life of a volar pad from formation (7-8 weeks EGA on the fingers) until complete regression (16 weeks EGA). It is understood that all EGA values related to volar pad size and shape are highly variable from fetus to fetus, and, therefore, are only included as approximations in this general developmental scheme.

Ridge Alignment: Growth Stresses on the Volar Surface

The growth and regression of the volar pads produce variable physical stresses across the volar surface that affect the alignment of the ridges as they first begin to form. Generally, volar pads are high and round when the cells along future primary ridges first begin to proliferate. Ridges form concentrically around the apex of a high round pad, conforming to the navigational pattern of the loxodrome [41] (Figure 17). Research in both the medical and mathematical fields support this same physical model applying across the entire volar surface of the hands and feet [28, 33, 40, 42]. Researchers have observed ridges forming on high, pronounced volar pads conforming to the surface as large-count whorl patterns. Conversely, ridges forming on a finger with a low or absent volar pad are low-count or arch type patterns [34]. The results of this physical model become extremely complex on asymmetrical variations of the spherical shape.

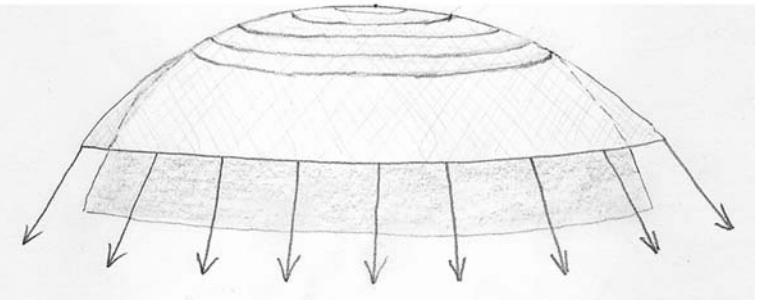

Figure 17

In navigation, this pattern is known as a loxodrome, and results from an elastic film being stretched over a hemisphere. Ridges form concentrically around the apex. The mathematical formula for this pattern can be found in tensor calculus, a field which offers much promise in predicting what ridge formations might form across different shaped surfaces.

The effect of the size and shape of the volar pad on ridge patterns has been studied intensely by several researchers over the years. Bonnevie first hypothesized that volar pad height affected fingerprint patterns in 1924 [18]. Three years later, Harold Cummins published an extensive analysis of malformed hands to elucidate the effect of the growth and topology of the hand on ridge direction [33]. Cummins concluded that ridge direction is established by the contours of the hands and feet at the time of ridge formation. Penrose examined fingerprint pattern formation from a mathematical perspective [28, 43].

The distinction between the size, height, and shape of the volar pad, and the effects of differences in each of these elements on a fingerprint pattern is a difficult topic to study, a fact that many recent researchers have acknowledged [44, 45, 46]. Naturally, such differences cannot be evaluated in one individual. Large bodies of data must be studied and correlated in order to deduce which factors may affect specific pattern elements. Fortunately, a literary review has allowed such study, and a unique picture of fingerprint pattern formation has emerged which combines portions of many theories, but goes beyond what has been previously published on this subject.

Ridge Count: Timed Events and Volar Pad Size

The size, particularly the height, of the volar pad during primary ridge formation affects the core to delta ridge count of normal fingerprint patterns [18, 22, 41]. Holt reported that the Total Finger Ridge Count (TFRC) of all 10 fingers is the most inheritable feature in dermatoglyphics [47]. This combined information points directly toward timing events related to volar pad and friction ridge formation affecting fingerprint patterns.

The ridge count of a fingerprint pattern is related to two different timed events: the timing of the onset of volar pad regression versus the timing of the onset of primary ridge formation. Differences in either timed event will affect the ridge count of that particular pattern if the events are timed independently. For example, early onset of volar pad regression would lead to a volar pad which was in a more regressed state by the time of the onset of primary ridge formation, and a lower ridge count pattern (or arch) would result. On the other hand, overall late onset of volar pad regression would mean that the pad was still relatively large when primary ridges

began forming, and, therefore, a high ridge count pattern would result (Figure 18A). This theory is supported by a study which found that "late maturers" had higher than average ridge counts, and "early maturers" had lower than average ridge counts [48]. If volar pad regression onset occurred at the normal time, then earlier than average onset of primary ridge formation would occur on a larger than average size volar pad. This circumstance would lead 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. This circumstance would lead to a lower than average ridge count (Figure 18A). When both early and late timing of both factors are taken into account, the results become even more complex (Figure 18B).

Ridge Count: Converging Ridge Fields

The onset of cellular proliferation which begins primary ridge formation occurs first in three distinct areas: (1) the apex of the volar pad (which corresponds with the core of the fingerprint pattern), (2) the distal periphery, or tip of the finger (near the nail bed), and (3) the distal interphalangeal flexion crease area, below the delta(s) 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 (Figure 19). This wave-like process of three converging fields allows for the visualization of how deltas most likely form (Figure 20).

The concept of "converging ridge fields" also offers a way to visualize the difference between the formation of large versus small ridge count patterns. If ridges begin formation 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 traveled by the field on the apex of the pad; and a large count pattern would be formed (Figure 21). However, if the ridges form first on the two outermost 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; a very small pattern is observed (Figure 22). The combined observations of different researchers examining fingerprints during the critical stage of development further support the validity of this model [19, 20, 27, 29].

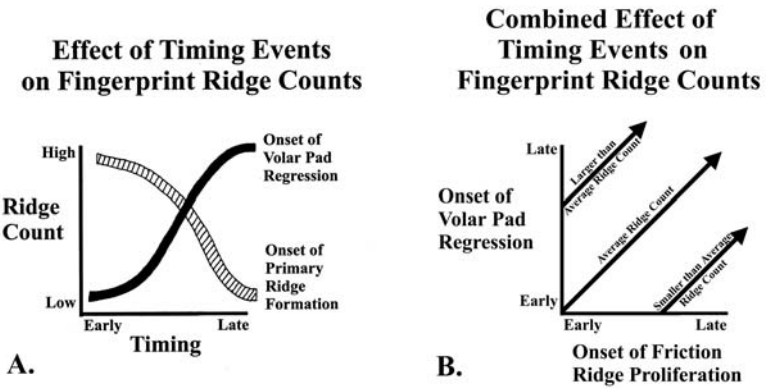

Figure 18

Chart A demonstrates the effects of two independent timing events on the resulting ridge count of a fingerprint pattern. Chart B demonstrates their combined effects on fingerprint pattern ridge count.

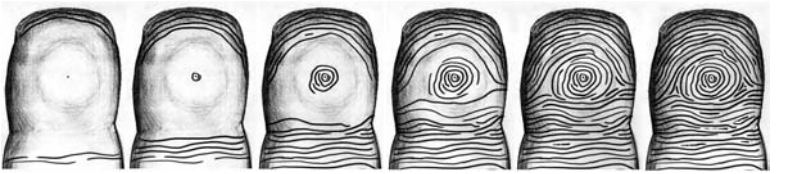

Figure 19

Ridges form in three distinct locations on the end joint of the finger, and converge.

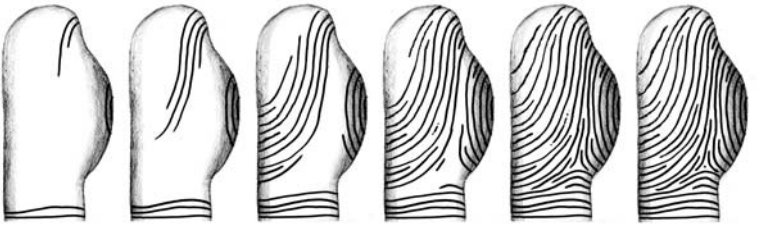

Figure 20

Deltas form where three ridge fields meet.

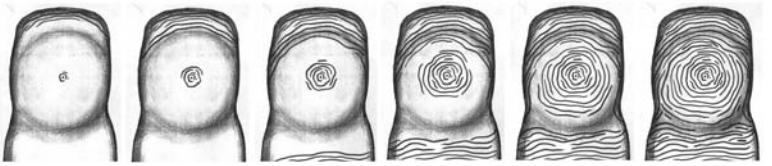

Figure 21

Large count pattern formation: ridges form in the center first and proceed outward before being met by the other ridge fields.

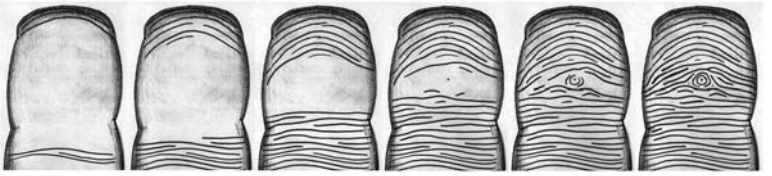

Figure 22

Small count pattern formation: ridges form on the outer perimeter of the pattern area and proceed inward until the last instant, when ridge formation begins at the apex of the pad.

Ridge Counts: External Factors

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 (10), hormone levels (44), radiation levels (49), and any other factor that could affect the growth rate of the fetus during the critical stage could all indirectly affect the ridge counts of the developing fingerprints. It is important to remember that anything that could affect the tension across the surface of the finger could affect the resulting ridge count. However, Holt's 1968 findings seem to indicate that timing events, rather than environmental factors, play the dominant role in determining TFRC. A significant point to remember is that ridge counts are affected primarily by the combined timing events of volar pad regression and primary ridge formation. Pattern type, on the other hand, is affected by a completely different set of factors.

Pattern Type: Volar Pad Shape and Symmetry

The overall shape and symmetry of the finger volar pad when ridges first begin to form determines pattern type. Cummins, Penrose, and others have long reported that high and round (and/or narrow) volar pads form a large whorl-type pattern, asymmetrical "leaning" pads form looping patterns, and low or absent volar pads form arch patterns [33, 43]. In 1987, Babler validated the correlation between pad symmetry and pattern type [34].

Whether ridge flow will conform to a whorl or a loop pattern depends entirely on the symmetry of the stress across the surface of the finger. If the volar pad and other elements of finger growth are perfectly symmetrical during the onset of primary ridge formation, then a symmetrical pattern (a whorl or an arch) will result. However, if the volar pad and/or 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 regression occurring either totally symmetrical or leaning all the way to one side (totally askew). In fact, there is a continuum involved from whorl patterns to loop patterns. Figure 23 illustrates several patterns from different individuals whose volar pads were roughly the same size at the critical stage (similar ridge counts), but differed in their degree of symmetry. 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.

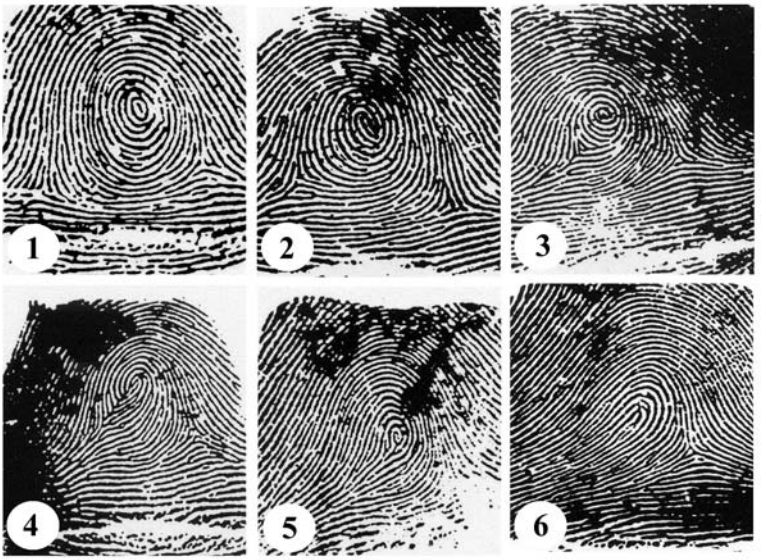

Figure 23

Patterns from different individuals representing the continuum of differing volar pad symmetry. (1) At the onset of friction ridge proliferation, the volar pad of this first pattern was nearly symmetrical. (2) The volar pad of the second pattern was only slightly displaced, the third slightly more, etc. (6) The volar pad of this pattern was completely displaced.

Timing (Size) and Symmetry of Volar Pads: Cumulative Effect

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 large ridge count. If timing is later in fetal life after the pad has almost completely been absorbed into the contours of the finger, then a low-count whorl pattern will result. Any further regression, and an arch pattern would form (Figure 24). 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 large count loop will result. Later timing leads to a low-count loop or arch-type pattern (Figure 25). Again, it is emphasized that volar pads are not simply symmetrical or asymmetrical, rather, a continuum of symmetry 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 asymmetrical as the volar pad regresses. This is supported by general fingerprint pattern statistics which show that over half of all fingerprint patterns are ulnar loops. More specifically, this scheme is supported by fetal research which has determined that early timing of primary ridge formation leads to a higher percentage (95%) of whorls [50]. Also, low and high ridge count patterns occur less frequently than average count patterns [51]. All the data studied 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 large inked print collections.

Figure 24

These patterns were formed on completely symmetrical volar pads. Top row illustrates fetal condition of volar pad, while bottom row illustrates resulting print. From left to right the images show the timing of friction ridge proliferation versus volar pad regression. Left: Ridge proliferation was early, and, therefore, occurred on a large volar pad. Right: Ridge proliferation was late, and, therefore, occurred on a small or non-existent volar pad.

Figure 25

These patterns were formed on completely asymmetrical volar pads.Top row illustrates fetal condition of volar pad, while bottom row illustrates resulting print. From left to right the images show the timing of friction ridge proliferation versus volar pad regression. Left: Ridge proliferation was early, and, therefore, occurred on a large volar pad. Right: Ridge proliferation was late, and, therefore, occurred on a small or non-existent volar pad.

Unusal Fingerprint Patterns

There are basically two fundamental factors that serve as a starting point during the critical stage of development in determining what pattern will result on a particular finger. The first is the timing of the onset of friction ridge formation with respect to the stage of volar pad regression. The second factor is the degree of volar pad symmetry (or asymmetry). If these two factors can be evaluated independently, then insight may be gained with respect to causal factors of unique patterning. Tension across the surface does not always conform to normal models, which is why we see double-loop whorls, accidental whorls, nutant loops, cuspal patterns, etc. These abnormal patterns could be caused by irregular volar pad growth or regression, unique growth of the bony distal phalanx, physical pressure on the digit, or any other factor affecting the symmetry of the volar pad. Perhaps a lack of tension across a portion of the pattern is the cause of dissociated ridges, or a complete lack of tension across the entire digit (stagnant growth rate) might explain dysplasia. Perhaps the fundamental reason that individuals with certain chromosomal diseases, such as Down's syndrome and Turner's syndrome, consistently display abnormal ridge counts [52, 53, 54] is that chromosomal abnormalities are closely linked with critical timing events in the development of the nervous system and other important fetal milestones during the 10.5 to 16 week EGA critical stage. The interrelated factors of timing and symmetry seem to be most significant in affecting tension across the surface, and account for the wide range of dermatoglyphic patterns seen by latent print examiners around the world.

Palm Prints and Vestige Patterns

Friction skin formation concepts apply to all volar skin, whether on the finger, palm, or sole. This paper primarily focuses on the fingers, because fingerprint patterns are the most widely discussed and studied in the literature. However, patterning on the palm and sole could be addressed using these same concepts. Volar pads and timing events still affect patterning, but terminology regarding ridge counts and pattern types would have to be redefined for application to each relevant area. The same concepts apply on the surface of the palm with respect to volar pad location and delta (triradii) formation: triradii in the palm also form along the shoulders of the volar pads, which have mostly regressed into the surface of the palm before the critical stage. Occasionally, pattern-

ing can be found in the thenar or hypothenar areas, but these volar pads are the first to regress and are usually completely absent during the onset of primary ridge formation. Palm prints open our eyes to the formation of friction skin over the entire volar surface. Congenital lymphedema (swelling of the limbs during fetal formation) is but one example which demonstrates this through both high ridge count whorl patterns on the fingers and complex palmar patterning. However, it can be generally said that the same formation concepts apply across all volar areas: tension during the critical stage conditions ridge alignment, stresses across small areas determine minutia placement, and heterogeneous basal cell distribution determines ridge shape and pore location.

Some flexion creases form concurrently with friction ridge skin [56]. Disruptions in ridge flow are sometimes found around creases in certain areas undergoing simultaneous development. The most common area is the distal transverse flexion crease (top crease) just under the little finger [57]. Ridges in this area occasionally appear to turn abruptly into the crease, suggesting the crease and ridges were both forming during the critical stage. The scope of this paper did not include an in-depth study of volar creases. Suffice it to say that flexion creases are part of the same volar skin structure just as ridges, and, therefore, share the same principles of permanence and individuality [56, 58].

The formation of vestige patterns (Figure 26) has not been addressed in depth in the literature, and is not widely discussed in the field. However, several researchers have independently studied and diagrammed the volar pads on the surface of the palm (Figure 27). When volar pads between the index finger and the thenar area in the palm are pronounced during the critical stage, it can be seen that the surface relief, as depicted by Kimura [56], would lead to the formation of a vestige pattern. (Figure 28)

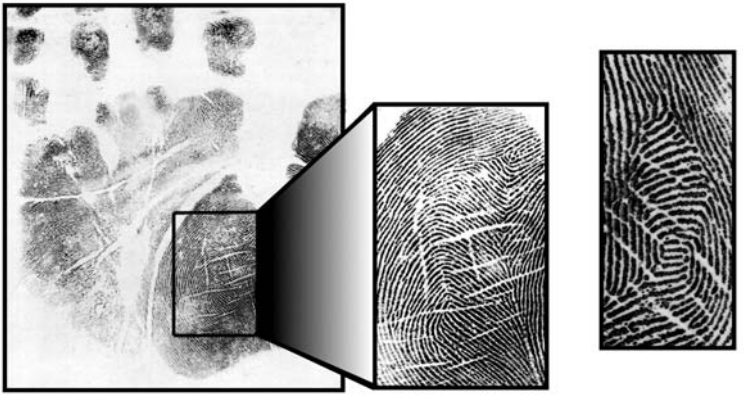

Figure 26

Vestige patterns can sometimes be found in the thenar area of palm prints.

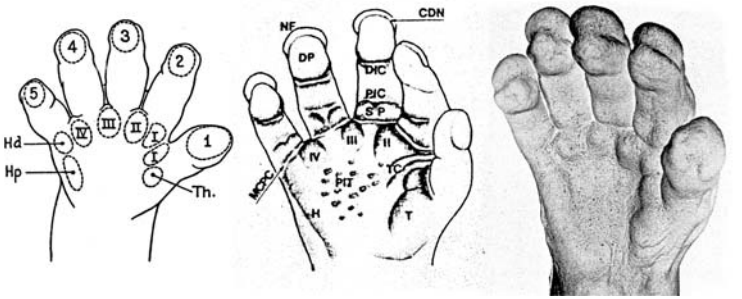

Figure 27

Researchers' observations are consistent throughout the literature in representing a portion of the first palmar volar pad being separate from the rest, on occasion being pushed up next to the thenar pad [40, 56, 66, respectively].

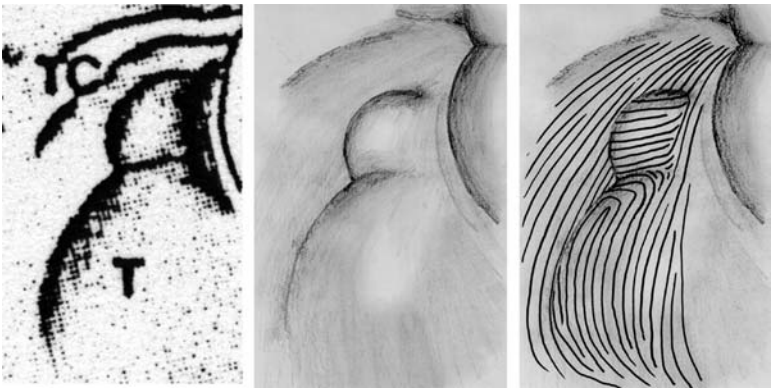

Figure 28

When the relief of the palmar surface, as drawn by Kimura, is present, it becomes easy to see how vestige patterns likely form.

Pattern Type: Bone Morphology

As demonstrated by fingerprint pattern frequency statistics, volar pads tend to regress in a manner conducive to the formation of ulnar loops. The cause of radial loops is simply a volar pad that is "leaning" or regressing in the opposite direction. In 1987, Babler found that radial loops and the distinctive ridge flow on the tips of the thumbs are associated with unique shapes in the bony distal phalanx [34]. Babler also reported a higher frequency of whorls associated with shorter distal phalanges and a higher frequency of whorls in instances where bones were less ossified [35]. Unique bone development in the end joint of the finger during the critical stage could affect tension across the skin, which, in turn, affects friction ridge patterning.

The Role of Genetics

Every aspect of the growth and development of a single cell to a fully formed human is directed by genetics. The capacity to form friction ridges is inherent. The patterns that these ridges form, however, are limited by nature and are defined as whorls, loops, arches, combinations and transitions of these basic patterns, or lack of a pattern [17]. Nature has established patterns, while genetics directs when and where ridges will form.

As with all biological traits, genetics does not independently control the resulting patterns on friction ridge skin. The ultimate example is monozygotic twins, who share identical genetic information and very similar 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 that patterning is affected by the environment [17, 59, 60, 61]. These genes most likely influence pattern formation indirectly through timing events, volar pad regression, growth rate of the fetus, and other factors which significantly influence pattern type and ridge count. Stresses across small areas of skin are not inherited, and represent but one of many environmental factors which influence pattern formation.

Until recently [45, 46], most researchers in the field of genetics and physical anthropology have traditionally viewed TFRC as evidence of direct genetic control of fingerprint pattern formation [18, 47]. The research of Holt, published in 1968, regarding the heritability of TFRC is a significant finding, and supports the two-tiered development scheme suggested by our study. Genetically controlled timed events would be less susceptible to environmental variations, and, therefore, TFRC would be more inheritable than pattern type. However, fingerprint pattern type and ridge count are *indirectly* inherited, and, therefore, are not affected by only one developmental factor. Ridge flow and ridge count are both affected by tension across the surface of growing fetal skin, as visualized through the model of differential timing (Figure 29).

Thousands of anthropological studies [62] have been conducted on distinct populations to identify trends in fingerprint pattern frequencies. Additionally, the medical community has been, and continues to be, very interested in dermatoglyphics as an indicator of abnormal fetal development during the critical stage [63,

64]. Using this new understanding of friction ridge and pattern formation, it may be possible to re-examine some of the massive amounts of previously published data, isolate certain aspects of the fingerprint patterns, and provide even further insight into the mechanism of friction ridge formation.

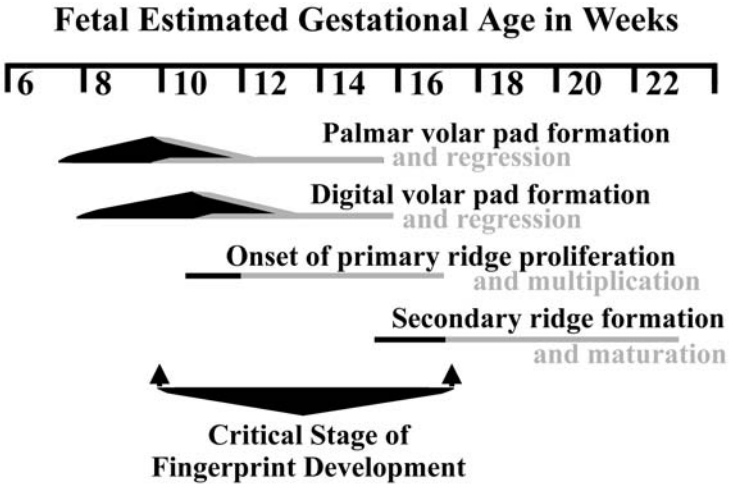

Figure 29

Summary of the timed events critical to the development of fingerprints, the exact timing of which varies among individuals. Black represents event onset, gray represents pad regression and ridge maturation.

Conclusion

Although different terminology was found throughout the literature in this study, different elements and general concepts contribute to form an over-all picture of the structure of skin (Figure 30).

The fundamental principle of permanence is based in the structure of friction ridge skin. The cells of the epidermis rise in concert from the constant but unique production of the basal layer, providing an outer layer that consistently represents the unique cell arrangements and production of the landscape from which it emerged. Within the skin, there are three primary structural

elements which enforce the permanence of the friction ridges: (1) the attachment of epidermal cells to each other, (2) the attachment of the basal epidermal cells to the basement membrane, and (3) the attachment of the dermis to the basement membrane. The properties of the basement membrane zone are consistently represented on the surface through the constant supply of new skin cells from that template. In addition to the typical structure of skin, the enhanced structure of friction ridge skin, with its alternating primary and secondary ridges, further anchors the surface ridges and furrows.

The uniqueness of friction skin is imparted to the permanent base structure from a sea of random forces, which, themselves, are affected by a seemingly infinite number of factors. The fetal volar pads play a major role in affecting the tensions that directly influence pattern formation (volar pad symmetry) and ridge count (volar pad size), but minutia formation occurs on a much smaller level. Localized stresses (tensions and compressions), resulting from growth of the tissue layers of the digit and interactions with existing ridge fields, create the foundations for second level uniqueness. Ridge morphology (third level detail) demonstrates a unique heterogeneous cellular community along the basement membrane, which constantly feeds the epidermis a three-dimensional portrait of its collective individuality. It is completely inconceivable that these physical stresses and cellular distributions could be exactly duplicated, on any level, in two different areas of developing fetal tissue. The fact is that each individual ridge is unique. Therefore, any ridge arrangement, regardless of quantity, is unique and could only come from one source. Wide variations in the amount of detail that transfers from the three-dimensional to the two-dimensional realm during any given contact may not permit individualization, but the arrangement itself is still unique.

More than ever, latent print examiners are expected to completely and accurately describe the principles upon which the science of friction skin individualization is founded: the concepts of *permanence* and *individuality* that permit the use of fingerprints as a means of identification. The scientific basis for stating that friction ridges and ridge formation are permanent and unique can be found in the biological sciences. It remains the responsibility of each expert, in every case worked, to be prepared to address these issues, defend the examination philosophy and methodology, and uphold a century of scientific excellence in the field of latent print examinations.

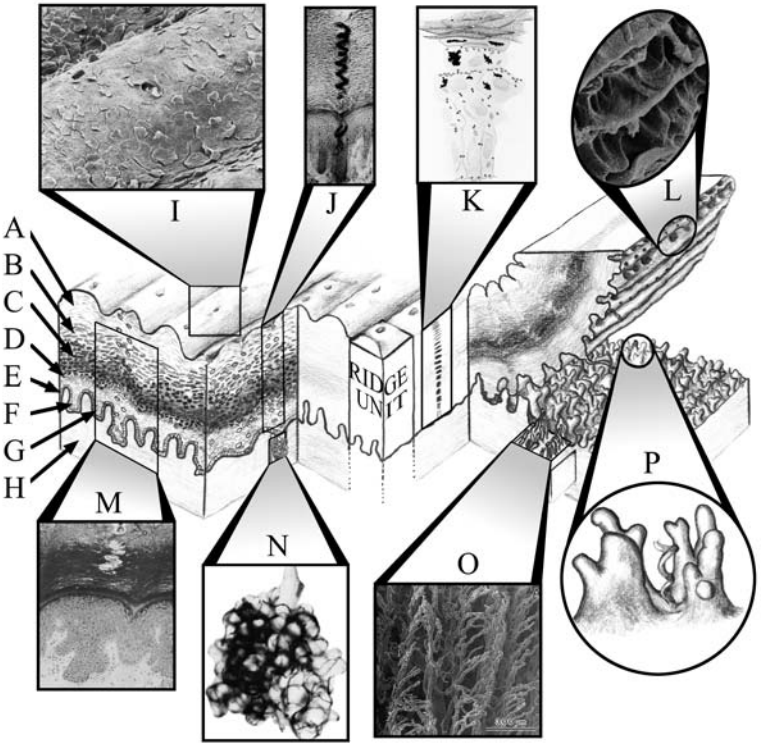

Figure 30

A: Stratum Corneum, or Horny layer
B: Stratum Lucidum, or Hyalin layer
C: Stratum Granulosum, or Granular layer
D: Stratum Spinosum, or Spinous layer
E: Stratum Basal, or Basal (Generating) layer
(A-E): Layers of the Epidermis
F: Primary Ridge (associated with surface ridge)
G: Secondary Ridge (associated with surface furrow)
H: Dermis
I: SEM view of a surface ridge [11]
J: Spiraling sweat duct [11]
K: Epidermal cell progression [11]
L: Under-surface of the epidermis [11]
M: Stained cross-section of volar skin [66]
N: Coiled sweat gland [11]
O: Capillaries underneath dermal papilla [66]
P: Close-up of dermal papilla [5]

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Animated illustrations for Figures 6, 7, 16, and 19 - 22 are available at www.clpex.com/animation.htm

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