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Friction Ridge Dysplasia and Its Preponderance in the Afghanistan Population

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FRICTION RIDGE DYSPLASIA AND ITS PREPONDERANCE IN THE AFGHANISTAN POPULATION

by

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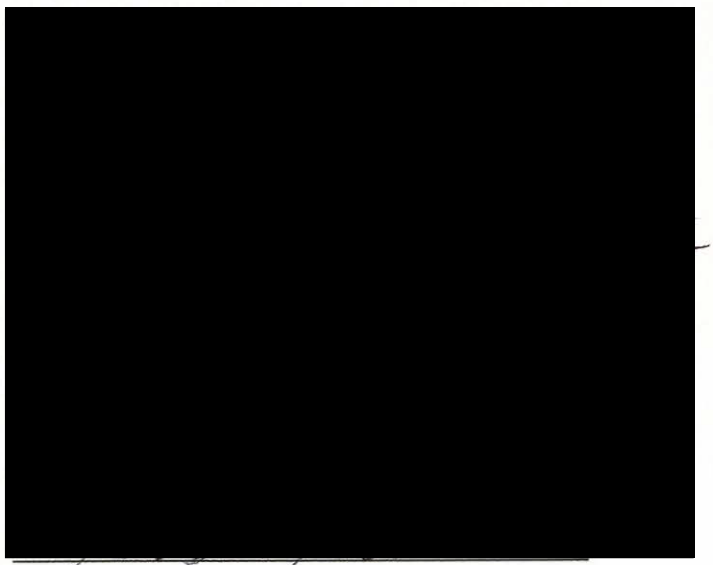

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ABSTRACT

FRICTION RIDGE DYSPLASIA AND ITS PREPONDERANCE IN THE AFGHANISTAN POPULATION

by Chelsea Elyse Woullard

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Friction Ridge Dysplasia is a rare genetic disorder in which the friction skin ridge units do not fuse together to form continuously flowing friction ridges. The skin affected by Friction Ridge Dysplasia is generally localized to one area and gives a similar appearance of the pebbled state of skin on the snout of a dog. Many authors have briefly discussed Friction Ridge Dysplasia in their publications; however, in-depth reflection as to the cause and rate of occurrence of Friction Ridge Dysplasia has not been documented. The objective of this study is to determine whether or not Friction Ridge Dysplasia is caused by certain factors such as, but not limited to, interfamilial reproduction and genetic inheritance in Middle Eastern populations. The latent print examiners deployed to Afghanistan noticed a more frequent occurrence of Friction Ridge Dysplasia within record fingerprints R. Schenck (personal communication, April 25, 2013). Due to the typically infrequent occurrence of Friction Ridge Dysplasia in general casework, this type of data has never been collated and examined for the rate of occurrence in any population. The study sought to determine whether the preponderance of Friction Ridge Dysplasia is consistent between the Afghanistan and United States populations. The study indicated that Friction Ridge Dysplasia is dependent on country; however, the disease is independent of handedness.

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CHAPTER I

INTRODUCTION

Friction ridges are the raised ridges that appear on the palm side of the hand and form the fingerprint patterns. Although these patterns grow in relation to the individual from intrauterine life to adulthood, the pattern does not change after final configuration unless disease causes deterioration, injury occurs to the basal layer, or death initiates decomposition (Ashbaugh, 1999). In some instances fingerprint patterns are interrupted by pebble-like dots that closely resemble the snout of a dog. This abnormal fingerprint characteristic is a genetic disease called Friction Ridge Dysplasia.

Friction Ridge Dysplasia is a rare genetic disease in which the individual friction ridge units do not fuse together to form continuously flowing friction ridges. This abnormality has stirred the curiosity of many Latent Print Examiners in the field of Forensic Science as well as many medical doctors. Although the interest was sparked, no definitive cause of Friction Ridge Dysplasia has been reported within either community. Many published authors such as Ashbaugh (1999), Babler (1978), Hale (1952), Maceo (2011), and Wertheim (2011) have briefly discussed Friction Ridge Dysplasia in their research and publications; however, in depth reflection as to the cause of the disease has not been documented.

The scarcity of research conducted on Friction Ridge Dysplasia affects the knowledge, or lack thereof, of the basis that forms the Latent Print discipline. While well researched Latent Print Examiners may understand when and how friction skin forms continuously flowing ridges, few, if any, understand what causes the lack of ridge fusion. Understanding the cause of Friction Ridge Dysplasia will ensure Latent Print Examiners

understand the biological and physiologic structures that form the basis of their discipline, which is friction ridge skin. Friction Ridge Dysplasia is one of hundreds of disorders classified under the wide umbrella of Ectodermal Dysplasias, or EDs, which affect the hair, skin, and nails (Ectodermal Dysplasia Society, 2012). No studies have been undertaken to explain how often the specific malfunction occurs; however according to the Nation Foundation for Ectodermal Dysplasia (2010),

> The latest estimate, published in the 1990 edition of The Birth Defects Encyclopedia, is that as many as 7 out of every 10,000 babies are born affected by the specific Ectodermal Dysplasia disorder. No one is really sure how many people are affected. (1)

This study seeks to determine if the preponderance of Friction Ridge Dysplasia in the Afghanistan population differs from the rate of occurrence in the United States population, as well as possible causes of Friction Ridge Dysplasia.

Objectives of the Study

The main focus of the current study is to determine the Friction Ridge Dysplasia rate of occurrence throughout the Afghanistan population and compare the data to the United States population. The cause of Friction Ridge Dysplasia will also be investigated in this exploratory study. The main focus of this study was maintained by completing the following:

1. Determine the Friction Ridge Dysplasia rate of occurrence for the Afghanistan population.
2. Determine the Friction Ridge Dysplasia rate of occurrence for the United States population.

3. Compare the Friction Ridge Dysplasia rate of occurrence of the Afghanistan population to the United States Friction Ridge Dysplasia rate of occurrence to determine if the differences are statistically different or similar.
4. Explore plausible causes of Friction Ridge Dysplasia.

CHAPTER II

REVIEW OF RELATED LITERATURE

Anatomy of Friction Ridge Skin

Smooth skin is found on all areas of the body excluding the palms of the hands and soles of the feet and contains hair, sweat glands, and sebaceous glands (Ashbaugh, 1999). The skin found on the palms of the hands and soles of the feet, called volar skin, is the focal point in this study. It possesses sweat glands and lacks sebaceous glands as well as pigmentation causing the surrounding smooth skin to usually be darker and more easily visualized on darker races (Ashbaugh, 1999) (Figure 1). Volar skin bears a striking difference from smooth skin in that the volar skin possesses furrows that increase tactile function. The surface of volar skin is crowded with continuously flowing ridges that serve to increase resistance between the skin and opposing surfaces, hence the name friction ridges (Ashbaugh, 1999).

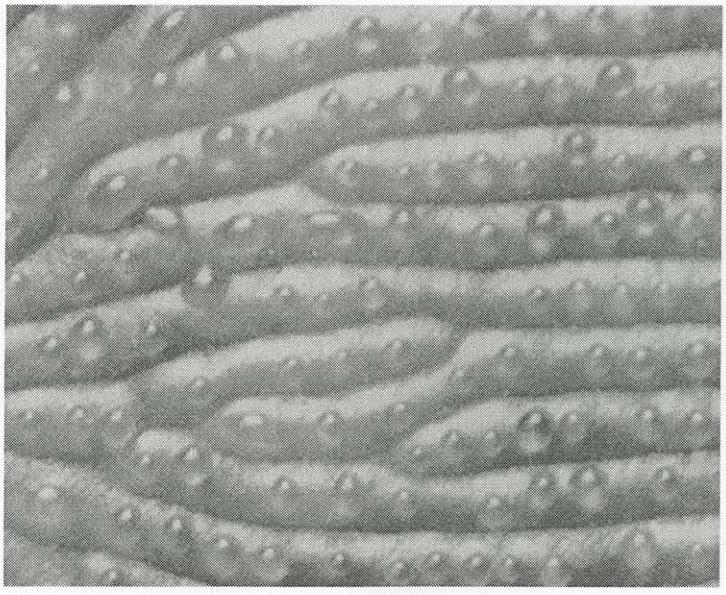

Figure 1. Sweat emitting from the pores on the friction ridge skin (Montagna and Parakkal, 1974).

The two main layers of skin are the inner dermis and the outer epidermis; however, there is also a sub layer called the hypodermis. Though the dermis and epidermis are two separate layers, they function together to provide essential bodily functions such as regulating body temperature, excreting wastes, providing a protective barrier, producing vitamin D and enabling sense of touch (Maceo, 2011). Excessive water loss is prevented by the outer layer of skin called the epidermis. The epidermis, primarily composed of keratinocytes, also provides protection even though it is constantly shedding keratinocytes when they reach the surface (Freinkel and Woodley, 2001). Freinkel and Woodley report that melanocytes, which are pigment-producing cells, protect the DNA of the keratinocytes from harmful sun exposure (2001). Keratinocytes mature, or differentiate, as they travel from the generating layer to the surface. The epidermis is described as a "stratified, continually renewing epithelium that exhibits progressive differentiation (keratinization, cornification) in a basal to superficial direction" (Freinkel and Woodley, 2001, 25). In simplest terms, this means that the epidermis is constantly generating new cells and pushing old cells upwards resulting in multiple layers of cells. Cells generated in the basal layer undergo changes in their chemical composition as they are displaced towards the surface.

The epidermis is comprised of five layers of cells, the innermost being the *stratum germinativum* or Basal layer. The Basal layer, often referred to as the generating layer of cells, is a single layer of columnar shaped keratinocytes that continuously divide, displacing old cells upwards. The basement membrane is where the basal cells, held together by desmosomes and focal tight junctions, are locked into their position by hemidesmosomes. The basement membrane is also referred to as the epidermal-dermal

junction and affords many different functions. Nutrients, wastes, and chemical signals must pass through basement membrane, which separates the dermis from the epidermis (Freinkel and Woodley, 2001).

When the keratinocytes divide from the basal cells, they are forced upwards to form the *stratum spinosum*, or Spinous layer. In this layer the desmosomes are reinforced and grow in size while keratin production is increased (Freinkel and Woodley, 2001). Differentiation occurs in this layer as the cells are no longer columnar shaped, but polyhedral (Maceo, 2011). Inactive lamellar granules, or pockets of lipids, begin to appear in the cells as desmosomes become more plentiful in the Spinous layer (Maceo, 2011). In the *stratum granulosum* (granular layer) the lamellar granules activate and begin secreting their lipid content onto the surrounding cells, in turn creating a hydrophobic barrier (Freinkel and Woodley, 2001). Chemical and structural modifications continue as keratinization causes cellular activities to disintegrate (Freinkel and Woodley, 2001). The *stratum lucidum*, or Hyalin layer, marks the death of the now keratinized cells although the chemical activity continues (Wertheim, 2011). As the cells continue to be displaced upwards, they finally reach the *stratum corneum* or Horny layer and have become cornified. Wertheim (2011) describes the arrangement of keratinocytes in this layer is described as a "brick-and-mortar model" meaning the keratin-filled cells (bricks) are surrounded by the lipids (mortar) secreted while the cells were in the stratum granulosum (34). As the cells approach the surface, the desmosomes begin degrading so the cells can be exfoliated once they reach the surface (Ashbaugh, 1999) (Figure 2).

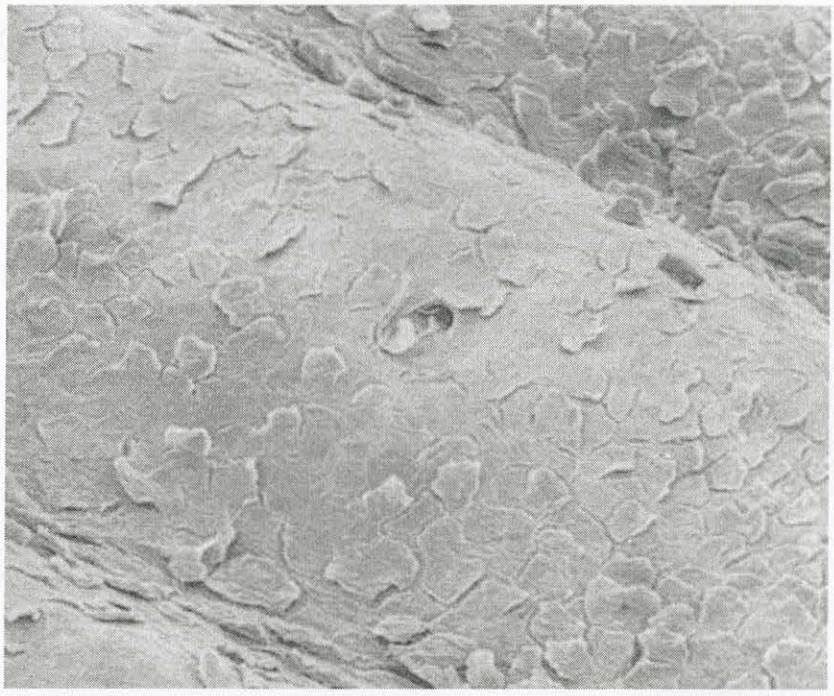

Figure 2. Surface of the friction ridges showing the cells shedding from the surface (Montagna and Parakkal, 1974).

The dermis is a protective barrier for the inner body and is often referred to as "true skin" (Ashbaugh, 1999, 61). This inner layer of skin, composed of connective tissue and fibrous proteins, is much thicker than the epidermis and provides nutrients as well as support to the dermis (Ashbaugh, 1999). With the support of the dermis, the skin is strengthened, water is stored, temperature is regulated and nutrient-saturated blood is supplied to the epidermis (Freinkel and Woodley, 2011). The dermis is divided into two layers, the papillary dermis and the reticular dermis. The papillary dermis, or papillary layer, is composed of anchoring fibrils which anchors the dermis to the epidermis (Maceo, 2011). Malleable peg-like projections between primary and secondary ridges called dermal papillae are formed in the papillary dermis (Misumi and Akiyoshi, 1984). Dermal papillae increase surface area, in turn binding the epidermis to the dermis more securely. The reticular dermis, composed of compact connective tissues, collagen, and

elastic fibers that boost pliability, is connected to the hypodermis (Freinkel and Woodley, 2001). The hypodermis, directly below the dermis, is composed of loose connective tissue, large blood vessels, and lobules of fat (Freinkel and Woodley, 2001).

The thermo regulating eccrine sweat gland is the only appendage of friction ridge skin although they are all over the body where other appendages are located as well (Maceo, 2011). Friction skin houses the larger and more active sweat glands. The simple tubular glands have ducts that penetrate through the dermis and hypodermis and open at the surface of the skin (Maceo, 2011). Thousands of fibrous connective tissue tufts called dermal papillae jacket the underside of the epidermis (Ashbaugh, 1999) (Figure 3). Directly below the surface ridges are mirrored primary ridges (Figure 3). Later in fetal development, the secondary ridges fit like a puzzle into the primary ridges to continue to cover the underside of the epidermis (Ashbaugh, 1999) (Figure 3). Unlike primary ridges, secondary ridges lack pore structures yet penetrate deeper into the dermis than the primary ridges (Ashbaugh, 1999). Between the primary and secondary epidermal ridges are small voids. These small voids are filled with dermal papillae that contain capillary loops to feed oxygen and food supplies to the basal layer (Ashbaugh, 1999). The dermal papillae also play a role in removing waste from the inner body (Ashbaugh, 1999).

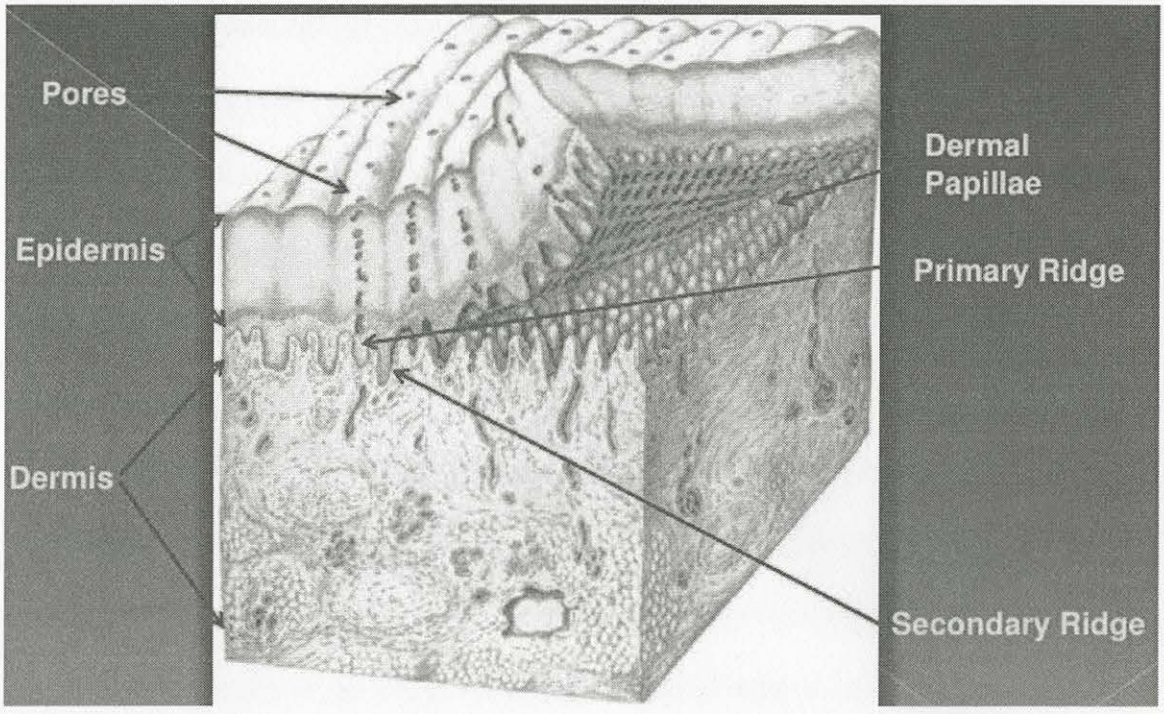

Figure 3. Three-dimensional representation of ridged skin with epidermis lifted to expose the dermal papillae (Rocamora, 2011)

Growth and Development of Friction Ridge Skin

The scientific study of fingerprints, or Dactylography, is a useful tool for forensic scientists during the process of identifying an individual or frequency of a ridge event. The growth and development of friction ridges are unique to one individual. Due to random and independent growth of friction ridges, they may stop, turn, or diverge at any point along its path (McRoberts 2005). Although the patterns may appear to be identical to the untrained eye, they are not. The various influences that occur in the womb due to inheritance, genetic malfunction, and fetal movement make it impossible for any two fingerprint patterns from different sources to be identical. The current study examines the frequency of a rare condition called Friction Ridge Dysplasia.

Fetuses possess different developmental times, according to Babler (1991). Hale (1952) reports that Crown to Rump, or C.R. length is frequently used. The first week's

estimated gestational age, or EGA, marks the formation of the primitive layer of the epidermis. At this point, the epidermis is approximately one cell thick and recognizable as an overall fetal covering at a very early stage (Ashbaugh, 1999). Patten and Corliss (1976) states that in early development, the epidermal layer is so thin that the color of the underlying dermal layer of vascular connective tissue shows through. The epidermis continues to form multiple layers of protection as the embryo grows and the basal cells divide (Ashbaugh, 1999). For the first few months basal cells multiply adding layers to the epidermis. As the basal cells divide the new cells displace old cells by forcing them upwards towards the surface as previously mentioned. On the word of Ashbaugh (1999), on volar areas the trip to the surface takes approximately one month, but there are numerous variables that can alter that rate. At approximately four weeks EGA, formation of shape, or morphogenesis, occurs when the arms, legs, knees, elbows, fingers, and toes begin to rapidly develop (Wertheim, 2011). The second layer of epidermis forms between the fourth and fifth week. At approximately six weeks, the first noticeable friction skin on volar surfaces appears (Ashbaugh, 1999). Patten and Corliss (1976) state that limb buds make their appearance at about the transition between the fourth and fifth week. Until approximately the fifth to sixth weeks EGA, the hand is expanded and flattened like a paddle and scalloped around the paddle-like boarder (Wertheim, 2011) (Figure 4).

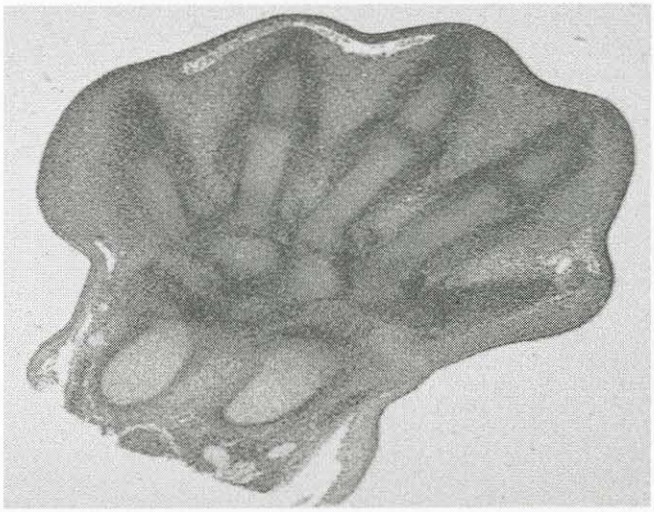

Figure 4. At six weeks the hand is paddle-like hand and the fingers are scalloped around its edge (Ashbaugh, 1999).

As Figure 5 shows, the second, third, and fourth interdigital pads are the first to appear at an approximate six weeks EGA (Ashbaugh, 1999). Followed closely are the thenar and hypothenar volar pads, which are trailed by the five distal pads (Ashbaugh, 1999). The swellings of tissue that appear as localized bulges on the palms of the hands and tips of the fingers are called volar pads. Volar pads begin to form on the tips of the thumbs as the basal cells rapidly divide, adding depth to the skin and progress towards the little finger at approximately the seventh and eighth weeks (Ashbaugh, 1999).

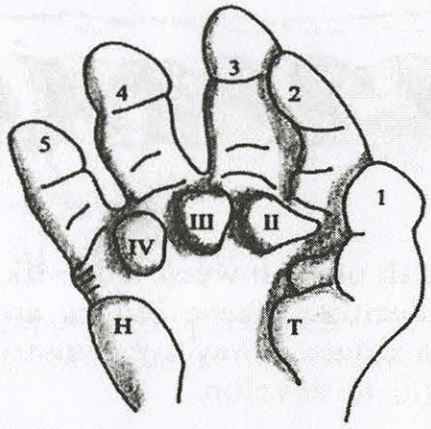

Figure 5. Volar pads conforming in their placement to the morphologic plan (Ashbaugh, 1999). Interdigital pads (II, III, IV). Thenar and Hypothenar pads (T and H). Distal volar pads (1, 2, 3, 4, 5).

Between approximately the seventh and eighth weeks EGA, the finger protrusion in the hand plate separate and elongate, causing the muscle and cartilage that was previously present to ossify into the bones of the fingers as depicted in Figure 6 (Wertheim, 2011). As ossification occurs, the joints of the hand begin to form as well (Ashbaugh, 1999). The thenar crease begins to form in the palm marking the rotation of the thumb as early as eight weeks EGA (Wertheim, 2011). At this time the fetus is only 2.5 cm C.R.; however, the hand resembles that of an infant (Cummins, 1929). Interdigital and palmer pads become very prominent during the eighth to tenth weeks, according to Ashbaugh (1999).

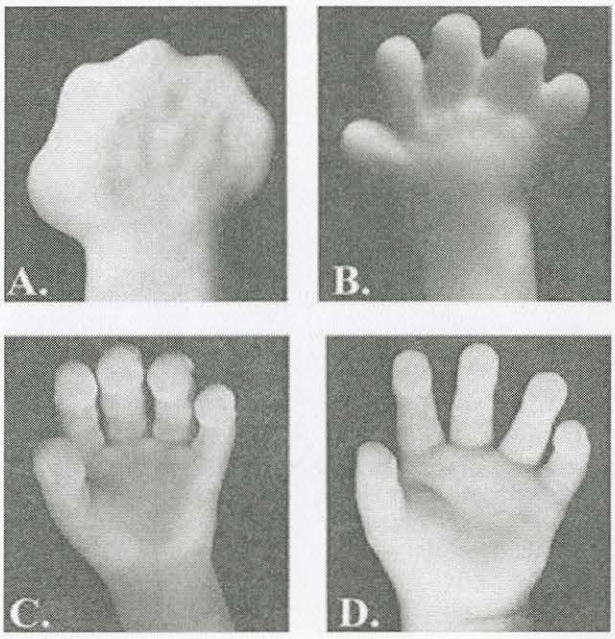

Figure 6. Growth of the hand progresses from (A) a paddle-like form, (B) continues as fingers separate, (C) the volar pads becomes prominent, and (D) achieves infantlike appearance by 8 weeks EGA (Cummins, 1929).

During the third month, the arms and legs begin to move (Wertheim, 2011). The prominent interdigital and palmer volar pads begin regressing between the tenth and eleventh weeks as the cells surrounding the pads continue rapid regeneration and reach the surface much quicker. The distal pads follow the pattern of volar pad regression. At this time the interdigital and palmer pads have regressed more than the digital pads. Over the next few weeks, the volar pads continue to reduce in size until their boundaries are indefinable from the surrounding skin (Ashbaugh, 1999). Embryonic development ends as fetal growth begins at the twelfth week (Wertheim, 2011). The primary ridges begin their formation in the basal layer on the underside of the epidermis when the fetus is approximately ten centimeters C.R. around the twelfth week (Wertheim, 2011).

Ashbaugh states that during the time of volar pad regression, bands of thickening tissue appear on the bottom of the epidermis, which is the first formations of primary ridges (1999). The epidermis has a fundamental biological factor encoded mandating it to

produce ridge units and have those ridge units fuse into rows to form friction ridges (Ashbaugh, 1999) (Figure 7).

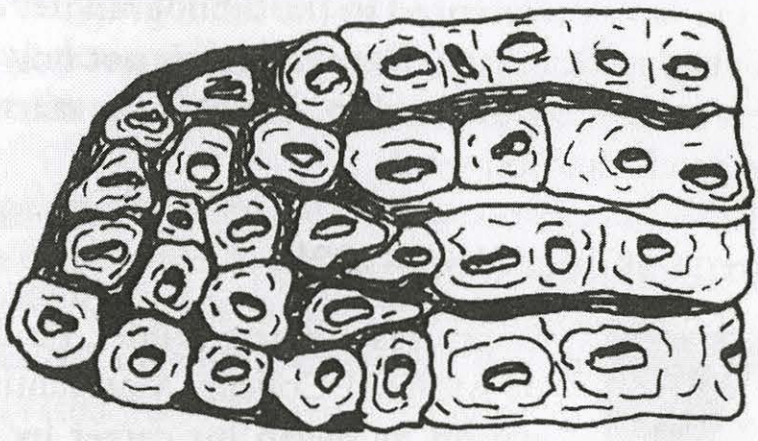

Figure 7. Ridge units fusing together to construct friction ridges (Ashbaugh, 1999).

Each ridge unit will develop a sweat gland deep in the dermis with a pore opening on the ridge unit's surface (Ashbaugh, 1999). Within three to four weeks, the cells developed inside the primary ridges have completely covered the bottom of the epidermis (Ashbaugh, 1999). When the digits have reached their largest state, around thirteen weeks, friction ridges begin forming on the digital pads as strong concentric formations begin to appear. By sixteen weeks EGA, volar pads have completely merged with the contours of the fingers, palms, and soles of the feet (Cummins, 1929). As the fetus reaches approximately fourteen centimeters C.R., the secondary ridges begin forming between the primary ridges on the underside of the epidermis (Wertheim, 2011). By approximately fifteen to sixteen weeks, when the secondary ridges begin forming, the primary ridges discontinue their formation in their final position (Ashbaugh, 1999). Although the primary ridges are fixed in their final location, they do grow in size as fetal growth continues (Wertheim, 2011). Around the fourth to fifth month of fetal

development, when the surface friction ridges are visible, the final configurations of the friction ridges have been established and are immutable. The surface stresses experienced during volar pad development as well as the timing and location of the developments influence the overall pattern and flow of ridges. The studies of topology, according to Cummins (1929), suggest that lines of curvature tend to follow the greatest convexity of a surface under stress. As the ridge patterns begin to develop from the center of the volar pad outward, a delta, or meeting of three ridge units in formation, is produced. Intrauterine volar pad regression and the start of friction ridge development have a direct impact on the type of fingerprint pattern developed as shown in Figure 8.

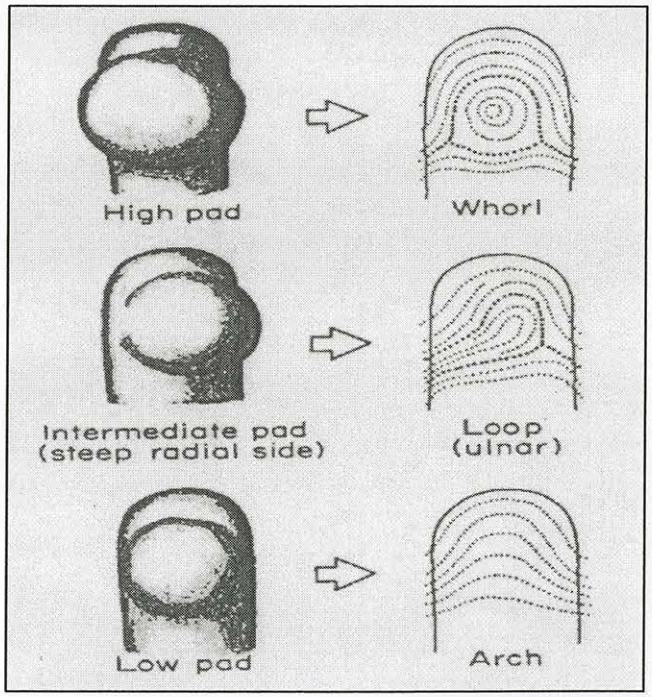

Figure 8. The impact of volar pad placement on fingerprint patterns (Ashbaugh, 1999).

The formation of second level detail during ridge growth is contingent on a multitude of physical and genetic variances, making friction ridges unique to that area of friction skin (Babler, 1978).

Friction Ridge Dysplasia

According to Ball (1999), though genetics may direct when and where ridges will form by providing the blueprint for proteins, nature provides the boundaries for patterning through physical mechanisms. Ashbaugh (1999) states that there are occasions when ridge units are present but do not fuse together to form continuously flowing friction ridges. This disease produces a rare condition called Friction Ridge Dysplasia.

According to the Ectodermal Dysplasia Society, Friction Ridge Dysplasia is one of hundreds of Ectodermal Dysplasias that plague the ectodermal tissues such as the hair, skin and nails (2012). Aquino et al. (2012) states that clinical features of Hypohidrotic Ectodermal Dysplasias include spare hair, abnormal or missing teeth, and an inability to sweat as shown in Figure 9. There are many different forms of hypohidrotic ectodermal dysplasias that may or may not affect the formation of friction ridges. The study conducted by Aquino et al. (2012) measured the severity of hypohidrosis, the presence or absence of specific teeth, ear position, and forehead prominence along with many other characteristics as depicted in Figure 9.

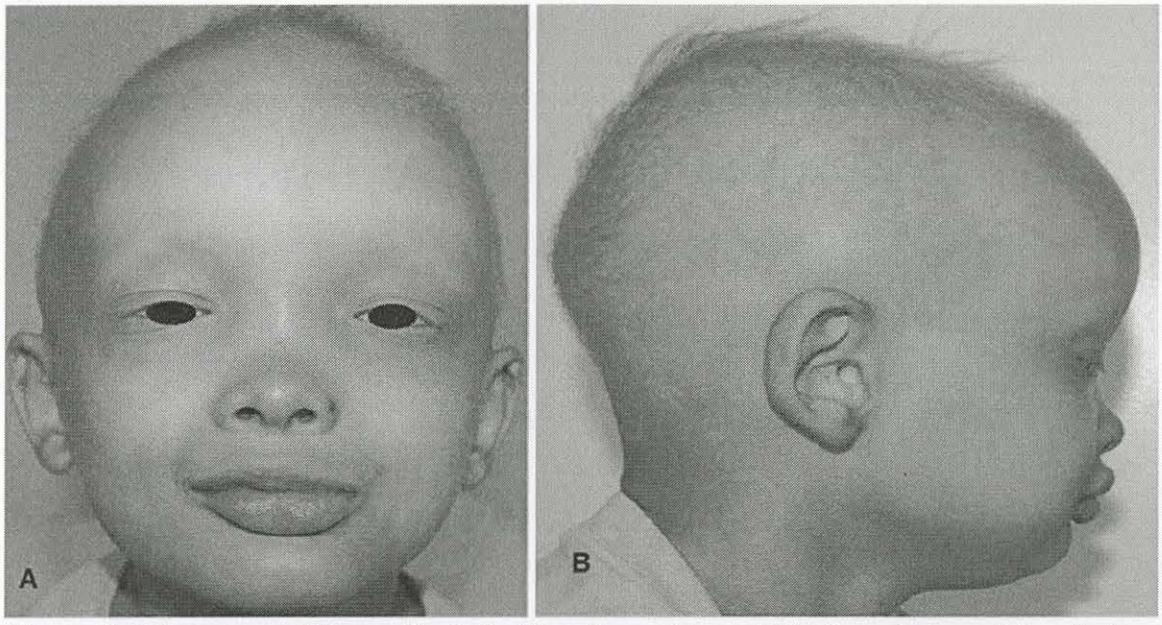

Figure 9. A four year old boy affected with a form of hypohidrotic ectodermal dysplasia (Aquino et al. 2012).

Friction Ridge Dysplasia is a hypohidrotic disease that is usually inherited X-linked recessively but can also be inherited autosomal recessively. Hypohidrotic diseases cause a lack of or complete obliteration of the sweat glands. Hypohidrotic disorders cause a disruption in continuity of ridge flow on volar areas when sweat pores are absent. According to Genetics Home Reference, most cases are inherited X-linked recessively and are caused by mutations in the EDA gene ("Hypohidrotic ectodermal dysplasia," n.d., related genes section, para. 1). The Genetic Home Reference goes on to explain the less frequently inherited patterns of the EDAR and EDARADD genes ("Hypohidrotic ectodermal dysplasia," n.d., related genes section, para. 1). The EDAR gene mutations can be inherited in an autosomal dominant or autosomal recessive pattern ("Hypohidrotic ectodermal dysplasia," n.d., related genes section, para. 1). The EDARADD gene mutations are inherited in an autosomal recessive pattern ("Hypohidrotic ectodermal dysplasia," n.d., related genes section, para. 1). The disruption in ridge flow is caused because each ridge unit contains one sweat pore. If the ridge unit does not contain a sweat

pore, it will not form or fuse to surrounding ridge units properly causing small or large non-fused pebble shaped ridge units, completely disrupting the pattern. These disruptions resemble the skin on the snout of a dog. Friction Ridge Dysplasia can be miniscule by affecting a small area or severe by completely obliterating the entire pattern (Figure 10).

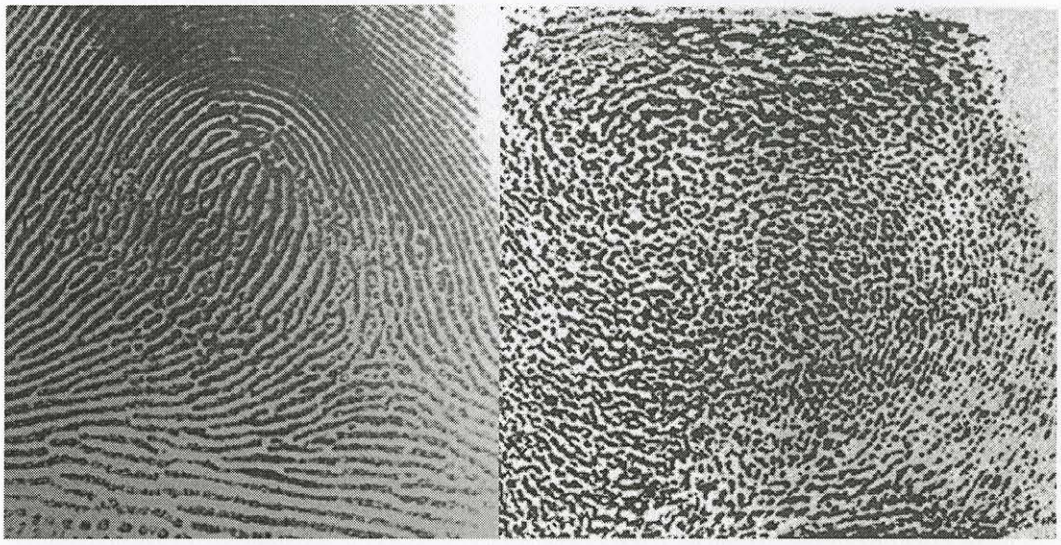

Figure 10. Impressions of epidermis displaying mild (left) and severe (right) dysplasia (Ashbaugh, 1999).

A study called "The Immigration Delay Disease: Adermatoglyphia-Inherited Absence of Epidermal Ridges" examined the complete absence of epidermal ridges on the palmer side of the hands. The condition was inherited over four generations of a family in an autosomal fashion. Burger et al. (2011) stated that the complete absence of epidermal ridges is extremely rare, resulting in only four kindred with additional features. T. Reed and R.L. Schreiner (1983) observed the complete absence of friction ridges passed through five generations of an Irish-American family. Like Friction Ridge Dysplasia, the individuals in this study lacked sweat pores. The study titled "Absence of Dermal Ridge Patterns: Genetic Heterogeneity" concurs with the belief of the previous studies that heredity plays a major role in the formation of friction ridges, or lack thereof.

Due to the rare occurrences of friction ridge dysplasia, the condition is not heavily researched. Hundreds of genetic EDs can occur when certain genes mutate causing the body to fail to transmit the signals needed to form friction ridges. Many theories have been explored in hopes of determining the trigger mechanism that causes friction ridge units to form and fuse together. These same theories could explain why friction ridge units do not fuse together. According to K. Wertheim, the capacity to form friction ridges is inherent within the developing embryo. The proteins that direct cellular activity by facilitating biochemical processes within the cell depends on many factors, one being the protein derived from the gene (Wertheim, 2011). In some individuals, the genes that derive the proteins are abnormal due to genetic mutation or inheritance from the parent.

Consanguinity

Afghanistan is located in Southern Asia, Northwest of Pakistan, and possesses numerous ethnic groups. According to the research found in the "Afghanistan's Ethnic Groups Share a Y-Chromosomal Heritage Structured by Historic Events" study by Haber et al. (2012), the current Afghan population shares a common heritage consequent to the unstructured ancestral population that likely surfaced during the Neolithic revolution. The study also stated that migration to and invasion of the region is a direct cause of the genetic differences found in the Afghans' population of Central Asia. The Hazara, Pashtun, Tajik, and Uzbek are the four major ethic groups of Afghanistan (Haber et al., 2012).

Many of these tribal communities still practice consanguineous marriage and reproduction. Consanguinity is derived from two Latin words "con" meaning of the same or common and "sanguineous" meaning blood. Linguistically consanguinity refers to two

people in a relationship who share a common ancestor. Referring back to Haber et al.'s (2012) previously-mentioned article, the four major ethnic groups in present-day Afghanistan share a common ancestor. The likelihood that mutations and diseases will be passed from parent to offspring is increased when consanguinity is practiced. According to Tadmouri et al. (2009), the main impact of consanguinity is an increase in the rate of homozygotes for autosomal recessive genetic disorders. The article also goes on to say that

> In mathematical terms, consanguinity does not alter the allele frequencies of common disorders, but increases the probability of a mating between two individual heterozygotes for the same recessive mutant allele. In this regard, the risk for birth defects in the offspring of first-cousin marriage is expected to increase sharply compared to non-consanguineous marriages particularly for rare autosomal recessive disease genes, because for common recessive conditions, there is a high chance that the abnormal gene may be carried by unrelated spouses and may be expressed in their progeny. (Tadmouri et al., 2009, 5)

CHAPTER III

MATERIALS AND METHODS

The purpose of this research was to determine if there were any statistical differences between the rate of occurrence of Friction Ridge Dysplasia in the Afghanistan population and the rate of occurrence of Friction Ridge Dysplasia in the United States population. The scope of the study, in terms of Friction Ridge Dysplasia, was focused on the differences in rates of occurrence in the Afghanistan and United States populations. The statistical analysis used to determine if the independent variable had any significant effect on the dependent variable is discussed.

Tenprint Record Collection

The individual fingerprints data concerning Friction Ridge Dysplasia in the sample of the Afghanistan population were collected from previously recorded electronic and hard copy tenprint records supplied by the Afghanistan Automated Biometric Index System (AABIS). The tenprint records originating from the United States population were obtained from the dermatologic research records base located in Arthell Kelly Hall of the University of Southern Mississippi. The United States tenprint records were donated to the University of Southern Mississippi for research purposes only. Information regarding race, age, gender and all other identifiable characteristics were removed by the donating source prior to being donated to the University of Southern Mississippi's dermatoglyphic research records base. Similar to the United States population, all identifiable characteristics were removed by the AABIS coordinators prior to being released to the researcher. The exclusion of all identifiable characteristics was important to the research to ensure anonymity of the subjects as well as protect the

personal identifying information of each subject. This method was selected to avoid direct interaction with live human subjects.

Population Information

The United States population dermatoglyphic record included 918 hard copy tenprint records, which totaled 9,180 individual recorded prints. Of those 9,180 individual recorded prints, 642 individual recorded prints were excluded from the study due to the lack of overall sufficiency and clarity of the individual recorded print. After the exclusion of 642 nonsufficient individual recorded prints, 8,538 individual recorded prints were left that were suitable for analysis. A Sirchie Model M2 fingerprint magnifying glass was used to analyze the hard copy tenprint records for the presence of Friction Ridge Dysplasia. The Afghanistan population included 102 electronic tenprint records, which totaled 1,020 individual recorded prints. Of those 1,020 individual recorded prints, 278 individual recorded prints were excluded from the study due to the lack of overall sufficiency and clarity of the individual recorded print. After the exclusion of 278 nonsufficient individual recorded prints, 742 individual recorded prints were left that were suitable for analysis. Universal Latent Workstation (ULW) was utilized to determine the presence of Friction Ridge Dysplasia on electronic tenprint records. Microsoft Excel Spreadsheet was used to monitor each individual recorded print. The occurrence of Friction Ridge Dysplasia for the Afghanistan and United States populations were monitored and recorded separately. The individual recorded prints were logged in order from left to right in a Microsoft Excel Spreadsheet as right thumb, right index, right middle, right ring, right little, left thumb, left index, left middle, left ring, and left little. The spreadsheet containing the Friction Ridge Dysplasia occurrences for the Afghanistan

population was named Afghan Population Statistical Values. The spreadsheet containing the Friction Ridge Dysplasia occurrences for the United States population was named US Population Statistical Values. Each instance of Friction Ridge Dysplasia was recorded under the column that described the affected finger. The presence of Friction Ridge Dysplasia was noted with a one (1) in Microsoft Excel Spreadsheet. Fingers that did not have any Dysplasia were noted in Microsoft Excel Spreadsheet as a two (2) under the column that described the non-affected finger. The area of analysis was limited to the tips of each finger to the first knuckle of each finger. All other areas below the first knuckle and on the palms were disregarded and excluded from the study. The frequency of Friction Ridge Dysplasia was also individually measured in both the Afghanistan and United States populations for the left and right hands separately, as well as the left and right hands collectively. The frequency of arches, loops, and whorls were not evaluated to avoid any replication of past studies.

Limiting Factors

The lack of information regarding gender was a limiting factor of this research. Additional statistical analysis could not be completed to determine if the rate of occurrence of Friction Ridge Dysplasia differed between male and female subjects. Further biometric information was also excluded from the study for privacy protection purposes causing a second limiting factor. Access to the Afghanistan population tenprint records served as a third limiting factor. The Afghanistan Automated Biometric Index System coordinators had access to a limited number of tenprint records where contributing individuals waived their right to privacy. The lack of privacy waivers within the Afghanistan population lead to the fourth limiting factor. This factor caused a

relatively small population sample available for study from the Afghanistan sample. A fifth limiting factor was the significantly larger United States population sample size. Ideally, the research would have been conducted on populations with similar sample sizes; however, due to unforeseen circumstances, only limited data was available from the Afghan population for statistical analysis. Finally, the sixth limiting factor was the clarity of tenprint records. Numerous Afghanistan and United States tenprint records had to be excluded from the study due to poor clarity of the recorded print. Prints that displayed incomplete recordings, smudges, warts, cracks and other barriers that decreased the overall quality were disregarded and excluded from each data set. To ensure the accuracy of the analysis, only pristine recorded prints were included in the data sets. The prints that displayed sufficient recordings were included in the data sets and were referenced as records that were suitable for analysis.

Statistical Analysis

To determine if a relationship between the independent variable (country of residency) and the dependent variable (increased rate of Friction Ridge Dysplasia), existed, a statistical analysis was utilized. As previously mentioned in the Population Information section, the occurrence of Friction Ridge Dysplasia was noted as a one (1) on the Microsoft Excel Spreadsheet and the absence of Friction Ridge Dysplasia was noted as a two (2). The data that was recorded in the Microsoft Excel Spreadsheet was merged into SPSS to obtain statistical results that would allow an accurate interpretation. In SPSS, the independent variable (country of residency) was noted as a one (1) for Afghanistan and a two (2) for the United States. SPSS performed a chi-square test of independence to determine if the independent variable influenced the dependent variable.

The chi-square test of independence also measured the relative amount of influence on the dependent variable if present.

CHAPTER IV

RESULTS

As noted in the review of literature, consanguinity increases the likelihood that diseases may occur. Tadmouri et al. (2009), stated that the main impact of consanguinity is an increase in the rate of homozygotes for autosomal recessive genetic disorders. For the United States sample, 918 tenprint records, totaling 9,180 individual recorded prints, were analyzed for the presence of Friction Ridge Dysplasia. Of those 9,180 individual recorded prints, 8,538 individual recorded prints were suitable for analysis. For the Afghanistan sample, 102 tenprint records, totaling 1,020 individual recorded prints, were analyzed for the presence of Friction Ridge Dysplasia. Of those 1,020 individual recorded prints, 742 individual recorded prints were suitable for analysis. Table 1 shows the presence and absence of Friction Ridge Dysplasia in both the Afghanistan and United States population samples. In the Afghanistan sample, a total of 742 individual recorded prints were analyzed. Of these individual prints, 121 of them had Friction Ridge Dysplasia and 621 did not have any type of dysplasia. This data translates to 16.3% (121/742) with dysplasia and 83.7% (621/742) without any dysplasia. Of the 8,538 individual prints in the United States population, 47 individual prints had Friction Ridge Dysplasia and 8,491 did not have any type of dysplasia. This translates to 0.6% (47/8,491) with dysplasia and 99.4% (8491/8538) without any dysplasia. A total of 9,280 individual prints from Afghanistan and the United States population samples were collectively analyzed for the presence of Friction Ridge Dysplasia. Of the collective population samples, 168 individual prints had dysplasia and 9,112 prints did not have any dysplasia. Table 2 shows the chi-square test of independence for Friction Ridge

Dysplasia and Country. A chi-square test of independence was calculated to determine if the occurrence of Friction Ridge Dysplasia was distributed differently across Afghanistan and the United States. A significant interaction was found ($\chi^2(1) = 953.50$, $p < 0.05$). Afghanistan was likely to have more cases of Friction Ridge Dysplasia (16.3%) than the United States (0.6%).

Table 1

The Expected and Observed Friction Ridge Dysplasia Compared to Afghanistan and the United States

Country * FRD Crosstabulation					
					Total
			FRD Present	FRD Absent	
Country	Afghanistan	Count	121	621	742
		Expected Count	13.4	728.6	742.0
		% within Country	16.3%	83.7%	100.0%
	United States	Count	47	8491	8538
		Expected Count	154.6	838.4	8538.0
		% within Country	0.6%	99.4%	100.0%
Total		Count	168	9112	9280.0
		Expected Count	168.0	9112.0	9280.0

Table 2

Chi-Square Test of Independence for Friction Ridge Dysplasia and Country

	Value	Df	Asymp.Sig (2-sided)
Pearson Chi-Square	953.501[a]	1	0.000
N of Valid Cases	9280		

Table 3 depicts the presence and absence of Friction Ridge Dysplasia in the left and right hands of both the Afghanistan and United States population samples. On the left

hand, a total of 4,676 individual recorded prints were analyzed. Of these individual prints, 81 of them had Friction Ridge Dysplasia and 4,595 did not have any type of dysplasia. This data translates to 1.7% (81/4676) with dysplasia and 98.3% (4595/4676) without any dysplasia. Of the 4,605 individual prints on the right hand, 87 individual prints had Friction Ridge Dysplasia and 4,518 did not have any type of dysplasia. This translates to 1.9% (87/4605) with dysplasia and 98.1% (4521.6/4605) without any dysplasia. A total of 9,281 individual prints from the left and right hands were collectively analyzed for the presence of Friction Ridge Dysplasia. Of the collective sample, 168 individual prints had dysplasia and 9,113 prints did not have any dysplasia. Table 4 shows a chi-square test of independence of Handedness and Friction Ridge Dysplasia. A chi-square test of independence was calculated comparing the distribution of Friction Ridge Dysplasia on handedness. No statistical relationship was found ($\chi^2(1) = 0.322$, $p > 0.05$). Friction Ridge Dysplasia appears to be independent of handedness.

Table 3

Expected and Observed Friction Ridge Dysplasia Compared to Handedness

Handedness * FRD Crosstabulation					
					Total
			FRD Present	FRD Absent	
Handed-ness	Left Hand	Count	81	4595	4676
		Expected Count	84.6	4591.4	4676.0
		% within Country	1.7%	98.3%	100.0%
	Right Hand	Count	87	4518	4605
		Expected Count	83.4	4521.6	4605.0
		% within Country	1.9%	98.1%	100.0%
Total		Count	168	9113	9281
		Expected Count	168.0	9113.0	9281.0

Table 4

Chi-Square Test of Independence for Friction Ridge Dysplasia and Handedness

	Value	Df	Asymp.Sig (2-sided)
Pearson Chi-Square	0.322[a]	1	0.571
N of Valid Cases	9281		

Table 5 shows the presence and absence of Friction Ridge Dysplasia on the left and right hands of the Afghanistan population sample. On the left hand, a total of 376 individual recorded prints were analyzed. Of these individual prints, 59 of them had Friction Ridge Dysplasia and 317 did not have any type of dysplasia. This data translates to 15.7% (59/376) with dysplasia and 84.3% (317/376) without any dysplasia. Of the 366 individual prints on the right hand, 62 individual prints had Friction Ridge Dysplasia and 304 did not have any type of dysplasia. This translates to 16.9% (62/366) with dysplasia and 83.1% (304/366) without any dysplasia. A total of 742 individual prints from the left and right hands of the Afghanistan population sample were collectively analyzed for the presence of Friction Ridge Dysplasia. Of the collective sample, 121 individual prints had dysplasia and 621 prints did not have any dysplasia. Table 6 shows a chi-square test of independence for Friction Ridge Dysplasia and handedness in the Afghanistan sample. A chi-square test of independence was calculated comparing the rate of Friction Ridge Dysplasia in handedness of the Afghanistan sample. No significant relationship was found ($\chi^2(1) = 0.212$, $p > 0.05$) Friction Ridge Dysplasia appears to be independent of handedness in the Afghanistan sample.

Table 5

Expected and Observed Frequencies of Friction Ridge Dysplasia on the Left and Right Hands of Afghanistan Population

Afghanistan Handedness * FRD Crosstabulation					
					Total
			FRD Present	FRD Absent	
Handedness	Left Hand	Count	59	317	376
		Expected Count	61.3	314.7	376.0
		% within Handedness	15.7%	84.3%	100.0%
	Right Hand	Count	62	304	366
		Expected Count	59.7	306.3	366.0
		% within Handedness	16.9%	83.1%	100.0%
Total		Count	121	621	742
		Expected Count	121.0	9113.0	742.0

Table 6

Chi-Square Test of Independence for Friction Ridge Dysplasia and Handedness in the Afghanistan Sample

	Value	df	Asymp.Sig (2-sided)
Pearson Chi-Square	0.212[a]	1	0.645
N of Valid Cases	742		

Table 7 depicts the presence and absence of Friction Ridge Dysplasia on the left and right hands of the United States population sample. On the left hand, a total of 4,300 individual recorded prints were analyzed. Of these individual prints, 22 of them had Friction Ridge Dysplasia and 4,278 did not have any type of dysplasia. This data translates to 0.5% (22/4300) with dysplasia and 99.5% (4278/4300) without any

dysplasia. Of the 4,239 individual prints on the right hand, 25 individual prints had Friction Ridge Dysplasia and 4,214 did not have any type of dysplasia. This translates to 0.6% (25/4239) with dysplasia and 99.4% (4214/4239) without any dysplasia. A total of 8,538 individual prints from the left and right hands of the United States population sample were collectively analyzed for the presence of Friction Ridge Dysplasia. Of the collective sample, 47 individual prints had dysplasia and 8492 prints did not have any dysplasia. Table 8 shows a chi-square test of independence for Friction Ridge Dysplasia and handedness in the United States sample. A chi-square test of independence was calculated comparing the rate of Friction Ridge Dysplasia in handedness of the United States sample. No significant relationship was found ($\chi^2(1) = 0.238$, $p > 0.05$). The rate of Friction Ridge Dysplasia appears to be independent of handedness in the United States sample.

Table 7

Expected and Observed Frequencies of Friction Ridge Dysplasia on the Left and Right hands of the United States Population

United States Handedness * FRD Crosstabulation					
					Total
			FRD Present	FRD Absent	
Handedness	Left Hand	Count	22	4278	4300
		Expected Count	23.7	4276.3	4300.0
		% within Handedness	0.5%	99.5%	100.0%
	Right Hand	Count	25	4214	4239
		Expected Count	23.3	4215.7	4239.0
		% within Handedness	0.6%	99.4%	100.0%
Total		Count	47	8492	8539
		Expected Count	47.0	8492.0	8539.0

Table 8

Chi-Square Test of Independence of Friction Ridge Dysplasia and Handedness in the United States Sample

	Value	df	Asymp.Sig (2-sided)
Pearson Chi-Square	0.238[a]	1	0.626
N of Valid Cases	8539		

CHAPTER V

DISCUSSION

In this research, the Friction Ridge Dysplasia of the Afghanistan population sample was studied to determine the relative frequency of the ectodermal dysplasia, especially of the friction ridge. Of the 742 individual fingerprints studied, 121 prints had visible ectodermal dysplasia (16.3%), while the remaining 621 prints had no visible dysplasia (83.7%) (Table 1). These figures are considerably high in the Afghanistan population sample compared to the United States population sample. The United States population sample data shows only 0.6% of the population studied has dysplasia while the remaining 99.4% were normal (Table 1). This indicates that the rates of Friction Ridge Dysplasia are dependent on the country (Table 2). Thus, the null hypothesis, that the Afghanistan population sample is likely to have more cases of Friction Ridge Dysplasia than prints from the United States, is retained. Analysis 2, depicted in Table 3, compares the rate of Friction Ridge Dysplasia to handedness in both the Afghanistan and United States population samples. The results of this analysis showed that the rate of Friction Ridge Dysplasia is independent of handedness (Table 4). In layman's terms, this simply means that rates of Friction Ridge Dysplasia are not likely to differ from the left hand to the right. Although findings support that, collectively, Friction Ridge Dysplasia is independent of handedness, both the Afghanistan and United States population samples were compared separately to handedness. The purpose of this was to determine if the rates within the populations differed. Determining if the rate of Friction Ridge Dysplasia on the right hand in the United States population is higher than the left and vice versa for Afghanistan would provide further insight into the inheritance patterns of the two

population samples. If Friction Ridge Dysplasia indicated dependence of handedness in either population, future studies could use the information obtained about handedness to make links to other diseases and disorders. The results of rates of Friction Ridge Dysplasia compared to handedness separately in each population maintained the same outcome as when both populations were compared collectively. In both populations, Friction Ridge Dysplasia was independent of handedness (Table 6, Table 8).

During the critical stages of development, normal proliferation may be disrupted by genetic mutation, inherited disease, or improper nerve development, all of which are more likely to occur when two individuals, related by blood (i.e., first cousins) reproduce, according to Tadmouri et al. (2009). Unfortunately, the exact recessive trait linked to Friction Ridge Dysplasia is unknown due to the lack of DNA research conducted on affected persons. No genetic data is available for this research to give definitive confirmation to the links between increased rates of Friction Ridge Dysplasia and consanguineous marriages in the Afghanistan population, however many practical possibilities are provided.

The reproductive practices of Afghanistan differ substantially from the practices of the United States. It is practical to predict that the preponderance of Friction Ridge Dysplasia in the Afghanistan population is attributed to two very significant factors. First, the current Afghanistan population shares a common ancestor. Second, the tribal community participates in consanguineous reproduction. In combination with one another, these two factors can cause an increased rate of Friction Ridge Dysplasia due to inbreeding. Autosomal recessive genes, such as hypohidrotic ectodermal dysplasias, can stay hidden for generations within a family, but when a child is born to consanguineous

parents, it is more likely that the recessive gene will surface. Aquino et al. (2012) stated hypohidrotic ectodermal dysplasia is inherited X-linked recessively and impinge on the hair skin, teeth and nails of affected individuals. When populations with a common ancestor reproduce with members of their family, the likelihood that mutations and diseases will be passed from parent to offspring is increased. Thus, the occurrence of Friction Ridge Dysplasia is higher in the Afghanistan population than in non-consanguineous populations, such as the United States. Biometric information, such as iris and facial photographs, were not included in this research. The lack of aforementioned data proves to be a limitation of the study. In future studies, this limiting factor should be explored to determine if certain facial characteristics similar to the ones described in the review of literature are more prevalent or absent in individuals with Friction Ridge Dysplasia.

In this research, the chi-square test of independence was used to determine if the occurrence of Friction Ridge Dysplasia was distributed differently across Afghanistan and the United States. The chi-square test of independence was chosen over the chi-square goodness of fit test due to the type of results needed to test the null hypotheses. The goodness of fit test is used to determine how the observed value is significantly different from the expected value. This means that the test compares the observed sample distribution with the expected probability distribution. The chi-square test of independence determines the differences between the pattern of observed frequencies and the pattern of expected frequencies. Due to the need to determine whether the two variables were independent of each other, the chi-square test of independence was utilized.

The lack of genetic analysis is a limiting factor of the research. Statistical analysis of consanguinity and the rate of Friction Ridge Dysplasia could not be tested due to this limitation. Further research is needed in this area. The severity of Friction Ridge Dysplasia was not included in this study. Future research should be completed on this limiting factor to determine if the severity of Friction Ridge Dysplasia differs between the Afghanistan and United States populations. If a difference in severity is significant, the difference would provide a likely starting point for collecting intelligence based information for the population that was most likely to have deposited the fingerprint. The statistical analysis proves that the Afghanistan population is likely to have more cases of Friction Ridge Dysplasia than the United States population. This fact provides insight into the inheritance pattern, which is quite possibly affected by being a product of consanguineous reproduction.

APPENDIX A

FINGERPRINT AUTHORIZATION FORM

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The palm cards that were provided to Chelsea M. Woullard were donated to the University of Southern Mississippi for research purpose only. All personal identifying information was removed prior to being donated to the university (age, gender, ethnicity, etc.).

Thank You,

Levi Buck
Laboratory Technician

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