The Basis for the Uniqueness and Persistence of Scars in the Friction Ridge Skin

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ABSTRACT
At the site of an injury, the skin must accomplish two things: 1) repair itself and 2) return to a state of normal maintenance. In the process of repair, the skin undergoes guided, but random, cell growth. The random nature of this process means that the exact appearance of the scar is not foretold. Once repaired, the skin is brandished with a unique reminder of the injury and must return to the normal maintenance program. The skin of a scar follows the same maintenance program as uninjured skin and consistently reproduces the features on the surface of the scar.

INTRODUCTION
Scars! To use or not to use? That is the question. At least for the purposes of this article, that is the question. There appears to be a wide range of opinions regarding the use of scars when comparing impressions of the friction ridge skin. Some examiners consider their absence or presence, some compare the details of the scars, and others largely ignore them. It seems as though the same fundamental questions we ask about friction ridges must also be asked of scars: Are they unique? Do they persist?

The answers to these questions lie in the anatomy (structure) and physiology (function) of the skin responding to a wound. Before embarking on the journey to explain the anatomical and physiological changes that take place when the skin responds to an injury, it is necessary to review the normal anatomy and physiology of friction ridge skin.

Normal Skin: Outer Morphology
The outer surface of friction ridge skin is composed of alternating ridges and furrows with sweat pores occurring in regular intervals along the tops of the ridges. Figure 1 is an image of friction ridge skin. This outer surface comes into contact with objects and leaves the impressions that keep many readers of this article gainfully employed.

Normal Skin: Anatomy
Skin is composed of three anatomical layers: the outer epidermis, the inner dermis, and the lower hypodermis. The hypodermis contains, for better or worse, insulating fat that serves as an energy reserve and gives the contours of the body. The dermis is a network of cells, fibers, blood vessels, and gelatinous material that provides structural support and nourishment for the epidermis. The epidermis is composed predominantly of keratinocytes and is the outer protective barrier that provides temperature regulation and sensory input.
The ridges and furrows on the surface of the friction ridge skin are firmly rooted in the dermis by primary ridges (under the surface ridges) and secondary ridges (under the valleys). The primary and secondary ridges are part of the epidermis and feed cells to the surface ridges and furrows to replace cells lost at the surface. The primary and secondary ridges are interlocked with the dermis to provide support and strength to the friction ridge skin. Additionally, sweat glands extend from the primary ridges, through the dermis, and are anchored in the hypodermis.

**Normal Skin: Layers of the Epidermis**

The epidermis is described as a “stratified, continually renewing epidermis.” In other words, it is a layered tissue that must constantly replace the cells leaving the surface. Figure 2 demonstrates the five layers of the friction ridge skin epidermis: Stratum Basale, Stratum Spinosum, Stratum Granulosum, Stratum Lucidum, and Stratum Corneum. Notice the supra-basal layer between the Stratum Basale and the Stratum Spinosum. This is not a formal layer, but is an important transitional zone in the primary ridges.

![Figure 2. Cross-sectional view of the friction ridge skin](image)

**Normal Skin: Cells of the Epidermis**

The predominant cell (90-95%) of the epidermis is the keratinocyte. In Figure 2, the blue, purple, pink, red, orange, and yellow cells are ALL keratinocytes. They are presented in different colors to demonstrate the changes that occur as the keratinocytes reproduce in the Stratum Basale, transition into the Stratum Spinosum, and continue to be displaced toward the surface through the remaining layers. The epidermal cell layers are named based on the microscopic appearance of the cells in slide preparations of the epidermis. The cells change in appearance as they are pushed toward the surface because the chemical activity inside the cells changes as they move outward.

The brown, grey, and green cells in Figure 2 represent the few non-keratinocyte cells present in the epidermis: the melanocytes, Langerhans, and Merkel cells respectively. Melanocytes produce the pigments that are deposited into the keratinocytes. This pigment, melanin, protects the DNA of the keratinocytes from ultra-violet damage. The Langerhans cells are an extension of the body's immune system. Upon exposure to invading bacteria, Langerhans cells initiate an alert that causes the body to recruit more aggressive immune cells (T cells) to attack the invaders. The Merkel cells are an extension of the nervous system and transmit the sensation of touch: “shape, size, and texture of objects and two-point discrimination”.

**Normal Skin: Attachments**

The unique features of the friction ridge skin persist due to attachment sites and cell regulation. Cell regulation will be considered under the physiology section. In regards to attachment sites, there are structural features of the overall skin and of the skin cells that maintain the structure of the epidermis (even though skin cells are always sloughing at the surface). There are three levels of attachment in the friction ridge skin: the primary/secondary ridge attachment with anastomoses, the basement membrane zone, and cell-to-cell attachments.

The first level of attachment is the topography at the junction of the epidermis and dermis. The alternating system of primary and secondary ridges on the bottom of the epidermis provides general structural support for the surface ridges and furrows. Additional reinforcement of this system is provided by dermal papillae and epidermal anastomoses. As shown in Figure 3, dermal papillae are the peg-like projections of dermis between the primary and secondary ridges of the epidermis. Dermal papillae remodel with age in response to

![Figure 3. Cross-section of friction ridge skin with detail of the epidermis separated from the dermis to display the dermal papillae and complementary epidermal anastomoses.](image)
shearing stress on the surface of the skin. This remodeling causes the dermal papillae to become more complex, like a head of cauliflower. As the dermal papillae branch, the epidermis responds by creating complementary sheets of tissue to fill in and around the branches of the dermal papillae. These complementary sheets of epidermis are called anastomoses. The formation of dermal papillae and epidermal anastomoses increase the surface area of attachment between the epidermis and dermis, thereby increasing the bond between the epidermis and dermis. Figure 3 depicts a simplified schematic of the anastomoses and dermal papillae between primary and secondary ridges. Additionally, Figure 3 has a detail showing the complementary epidermal anastomoses and dermal papillae upon separation of the epidermis from the dermis.

The second level of attachment is the basement membrane. The basement membrane is a fibrous sheet that attaches the basal cells (keratinocytes of the Stratum Basale) of the epidermis to the underlying dermis. The basement membrane is generated by the basal cells of the epidermis and the dermis. As shown in Figure 4, the basal cells of the epidermis have specialized attachment plaques, termed hemidesmosomes, which project fibers down toward the dermis. The dermis projects anchoring fibers back up toward the epidermis. These fibers, generated by the basal cells and the dermis, are interwoven to create the fibrous sheet locking the epidermis to the dermis. The hemidesmosomes and interlocking fibers prevent the basal cells from migrating. The basal keratinocytes are locked down to their position in the epidermis.

The third level of attachment is the cell-to-cell attachments of the keratinocytes throughout the layers of the epidermis. Desmosomes and focal tight junctions, Figure 5, attach the keratinocytes to one another. Desmosomes are round plaques on the cell surface with fibers extending into the cells. Focal tight junctions are small “spot welds” of the cells’ surfaces. Desmosomes are reinforced as the cells move from the basal layer to the surface. Upon reaching the outer portion of the Stratum Corneum, the desmosomes and focal tight junctions are broken down to release the cells from the surface.

The three levels of attachment of the friction ridge skin contribute to the maintenance of the skin by preventing the epidermis from sliding across the dermis and by preventing the keratinocytes from moving laterally in the epidermis.

Normal Skin Physiology

The second component of friction ridge skin persistence is the physiology, or function, of the skin. The epidermis exists in a dynamic, steady state. Cells lost at the surface must be replaced (dynamic) in order for the skin to maintain (steady) its protective barrier (state). The concept of keeping things the same despite fluctuations is referred to as homeostasis. Homeostasis is defined as “the condition in which the body's internal environment remains relatively constant, within physiological limits.” Homeostasis is critical to the functioning of all organisms. Homeostasis of the skin is achieved through the physical attachments and the careful regulation of cell production in the Stratum Basale.

All keratinocytes of the epidermis are produced by the keratinocytes anchored in the Stratum Basale. The Stratum Basale is also referred to as the Generating Layer because it is the wellspring of all the upper layers. When a basal keratinocyte divides, it undergoes the process of mitosis. Mitosis is the mechanism by which a cell replicates its DNA, the two copies of which remain in the basal layer, cell A (Figure 6) and the newly generated cell sits on top of it (cell B in Figure 6). When the basal keratinocytes divide again, the first generated cell (B) is displaced into the Stratum Spinosum by the newly generated cell (cell C in Figure 6).

As the basal keratinocytes continue to divide (mitosis), the newly generated cells continually push previously generated cells toward the surface of the epidermis. As the cells move away from the Stratum Basale, they begin to differentiate. Differentiation refers to the process in which the keratinocytes lose their ability to divide, increase in size and flatten.
change their internal structure and chemistry, change their external surface structure, and dehydrate. The process of differentiation is necessary to create and maintain the protective outer barrier of the skin. As the cells move toward the surface, they undergo programmed cell death and fill with the protein keratin. Keratin is the durable protein that allows the skin to act as a protective barrier.

The cells anchored in the Stratum Basale continuously divide. The newly generated cells become part of the Stratum Spinosum and are committed to differentiating. In Figure 2, the basal keratinocytes of the primary ridges are diagrammed differently than the basal cells of secondary ridges. Additionally, the primary ridges have a supra-basal layer between the Stratum Basale and Stratum Spinosum. The keratinocytes in the basal layer of the secondary ridges have long finger-like projections on their bottoms for additional anchorage of the furrows to the dermis. The basal keratinocytes of the secondary ridges continuously divide as described above — each basal cell dividing to push one cell at a time into the Stratum Spinosum.

The basal cells of the primary ridges behave a little differently. The basal keratinocyte of the primary ridge divides to create a new cell. This new cell does not immediately enter the Stratum Spinosum and commit to differentiation. The newly generated cell, termed a transient-amplifying cell, undergoes a double rounds of division while it sits in the supra-basal layer. “Transient-amplifying cell” means that the cell has a temporary ability to replicate itself prior to moving into the Stratum Spinosum.

Even though the cells in the Stratum Basale are all keratinocytes, they have a slightly different anatomy and physiology depending on their position in the Stratum Basale. The basal cells of the primary ridges produce transient amplifying cells; consequently more cells are generated in the primary ridges than the secondary ridges. The cells of the primary ridges maintain the surface ridges. More abrasion occurs on the surface ridges than the furrows. The primary ridges need to create more cells to keep up with the greater rate of loss on the surface ridge.

Skin must maintain the protective barrier while existing in a dynamic steady state — cells leaving the surface must be replaced. The rate at which basal cells divide in the basal layer must coincide with the rate at which cells are leaving at the surface. There must be a mechanism in place to control the rate of cell division of the basal keratinocytes and to monitor the thickness of the skin. This mechanism is cell communication. The keratinocytes are in constant communication with one another and with the melanocytes, Langerhans, and Merkel cells. The keratinocytes are also in communication with the rest of the body via the dermis. Cell communication is achieved through direct cell-to-cell communication via gap junctions and through the use of cell surface receptors on the periphery of the cells. The gap junctions are direct links between the cells that can pass electrical signals. Cell surface receptors are proteins embedded in the cell membrane (boundary of the cell) that can bind chemical signals that induce or inhibit the cell division.

Physical attachments prevent the skin (and the keratinocytes) from moving laterally. As the basal keratinocytes divide, the cell-to-cell attachments ensure that the new cells will move upward toward the surface in concert. Control of the rate at which the basal keratinocytes divide ensures that the appropriate skin thickness is maintained (homeostasis). The three-dimensional ridge morphology on the surface persists . . . until there is an accident.

Wound Healing — Phase I

When an injury occurs, the protective barrier of the skin is breached. Figures 7 through 11 are diagrams of a skin model that will be used to demonstrate the cellular response of the keratinocytes to a wound. Figure 7 demonstrates the skin intact and after injury. Upon assault, keratinocytes have been removed and damaged and the dermis has been injured. The dermis and the epidermis undergo a remarkable change in anatomy and physiology in order to repair the damage. The process of wound healing is broken down into three phases, although there is considerable overlap: inflammation, proliferation and tissue formation, and tissue remodeling.

![Figure 7. Intact skin (A) and wounded skin (B).](image)

Phase I, inflammation, begins immediately after the injury. The disruption of the blood vessels in the dermis causes blood to spillover into the surrounding tissue. The platelets from the blood direct the clotting of the blood and send out signals to recruit cells from the immune system and the cells of the dermis. The immune cells kill bacteria and scavenge damaged cells. The dermal cells (fibroblasts) are concentrated in the wound area to repair the dermis. Additionally, endothelial cells (cells from the blood vessels) begin repair of the damaged blood vessels. It should be noted that the repair of the dermis and epidermis occur underneath the formed blood clot, although the blood clot is not shown in the following diagrams.

Wound Healing — Phase II

As the fibroblasts and endothelial cells continue to repair the dermis, the basal keratinocytes on the edge of the wound take over control of the healing process to start Phase II. As a result of the injury, the basal keratinocytes are suddenly exposed to the dermis by disruption of the basement membrane. Contact with the dermis causes the basal keratinocytes to undergo dramatic changes: the desmosomes and hemidesmosomes dissolve, actin filaments form inside the periphery of the cell, and pseudopodia (foot-like projections) are extended from the cell. The dissolution of the desmosomes and hemidesmosomes releases the basal keratinocytes from their firm attachments. The actin filaments, which act like miniature cell muscles, and the pseudopodia allow the skin cells to crawl across the wound. As the basal keratinocytes at the edge of the wound crawl, the basal keratinocytes behind them divide to create additional cells to help cover the wound. As the opposing sheets of basal keratinocytes march toward one another, the upper layers of cells are pinched together in friction ridge skin, this pinching creates the classic puckering of the ridges at the scar site. Figure 8C demonstrates the repair of the dermis and the beginning of the basal keratinocyte migration. Figure 8D demonstrates the continued migration, causing the puckering of the skin surface, and the proliferation of the basal keratinocytes behind the migrating cells.
Figure 8. Repair of the dermis and start of the migration of basal keratinocytes (C). Continued migration of the basal keratinocytes and production of new keratinocytes (D).

When the leading cells of migrating basal keratinocytes contact one another, they form gap junctions. These gap junctions reestablish communication. The keratinocytes stop their migration and begin to reconstitute the basement membrane (including hemidesmosomes) and the desmosomes and tight junctions between the keratinocytes. Once the basal layer is reestablished, the basal keratinocytes begin dividing upward until the appropriate skin thickness is attained. See Figures 9, 10, and 11.

Figure 9. Migrating basal keratinocytes meet in the middle of the wound and reconstitute the basement membrane (E). The new basal layer begins dividing to reconstitute the upper layers (F).

Figure 10. Keratinocytes begin to differentiate as they are pushed upward in the new epidermis (G, H).

Figure 11. New epidermis is completely formed.

Once the appropriate barrier has been formed, the scab formed by the blood clot during Phase I is released and the skin returns to its normal physiological state. The friction ridges are not reconstituted. The new basal layer of keratinocytes covering the wound forms the new template for the epidermis at that site. There are no primary or secondary ridges formed, consequently the epidermis does not regenerate the surface ridges and furrows. Additionally, sweat glands are not reformed. When the sweat glands are damaged as a result of the injury, the cells of the gland also migrate to cover the wound and the gland is lost.

Wound Healing – Phase III

Once the epidermis has resurfaced, Phase III begins in the dermis. The dermis continues to remodel and reinforce the scar tissue for weeks or months after the injury.

Wound Healing Model for Friction Ridge Skin

The following series of figures are diagrams created to demonstrate wound healing in friction ridge skin. The skin is undergoing the same series of events described above, but this model will focus on the layers, rather than the cells, as the skin heals. The layers of the epidermis are color-coded the same as Figure 2 and the friction ridge skin is viewed from both a three dimensional perspective and an aerial perspective.

Figure 12. Intact friction ridge skin.
Figure 13. Injured friction ridge skin.

Figure 14. Repair of the dermis.

Figure 15. Initial migration of basal keratinocytes.

Figure 16. Continued migration of basal keratinocytes and pinching of upper layers of skin.

Figure 17. Final migration of basal keratinocytes and reconstitution of basement membrane.

Figure 18. Basal keratinocytes begin dividing upward to form the Stratum Spinosum.
Considerations of Scars on the Impressions of Friction Ridge Skin

Formation of the scars explains what is seen on the skin, and subsequently on the impressions left by the skin. Scars may appear as a void, or may contain partial voids, in an impression because all or part of the newly formed epidermis sits below the level of the surface ridges. Like friction ridges, scars are a three-dimensional structure with surface contours and edges. Also like the friction ridges, the features of the scars will have some variability in appearance depending on deposition pressure and movement. Figure 22 is an image of a finger bearing a mature scar and an inked impression of the same finger.

Figure 22. Mature scar on the friction ridge skin and the resulting impression taken with ink, position reversed. (Printed with permission of the source, Richard Hansell of the Las Vegas Metropolitan Police Department Forensic Laboratory.)

Figure 23. Alteration of crease due to keratinocyte migration to cover the injury. (Printed with permission from the injured party, Michael Grimm of the Virginia Division of Forensic Science.)

It is readily apparent that the ends of the friction ridges approaching the scar are altered as the result of the basal keratinocyte migration during the healing process. In a relatively minor injury,
only the very ends of the friction ridges obtain a new configuration. With a more severe injury, the healing process can alter the skin away from the injury site. Figure 23 is the before and after images of a finger subjected to a drill with a screwdriver attachment. Notice that the flow of the ridges under the delta has stretched upward and the distal crease has shifted position.

Uniqueness of Scars
Scars are unique for the very same reason the friction skin is unique – developmental noise. Developmental noise is chance events that occur during development. As Richard Lewontin, research professor at Harvard University, eloquently states in his book, Human Diversity, “Wherever cell growth and division are involved, we can expect such noise to contribute its effects. The exact placement of hair follicles on our heads, the distribution of sweat glands on our bodies, a hundred such small details of our morphology are largely under the influence of such random events in development.” When the friction ridges are forming on the fetus and when the basal keratinocytes are activated by an injury, they are under the influence of developmental noise. The cells are rapidly proliferating and are tasked with forming the fetal skin or reconstituting injured skin. These cells are guided, but not given specific instructions on their position in the epidermis. In the case of an injury, the cells rapidly proliferate and migrate. The reconstitution of the Stratum Basale (the new template for the surface) and the effects on the surrounding epidermis (pinching) are the result of this guided, yet random process. Two injuries cannot duplicate the same scar.

Persistence of Scars
Scars persist for the same reason that the friction ridges persist: attachment sites and regulation of keratinocyte mitosis. The basal keratinocytes regenerate the basement membrane, reestablishing the attachment of the epidermis to the dermis. The keratinocytes also reestablish the cell-to-cell attachments: desmosomes and tight junctions. The keratinocytes resume communication with each other, with the melanocytes, Langerhans, and Merkel cells, and with the dermis. Communication allows for regulation of cell division in the basal layer, ensuring that the epidermis retains its appropriate thickness (homeostasis). As the cells divide, they move outward in concert and maintain the surface features of the scar. The impressions in Figure 24 were taken over fourteen years apart and clearly demonstrate the persistent nature of scars.

Figure 24. Known prints of a subject taken in July 1990 and over 14 years later in September 2004.

Conclusion
The use of scars in the comparison of friction ridge impressions has the same basis, and follows the same application, as the use of friction ridges. Once formed, scars are unique and persistent. When an impression of the skin is made, the features of the scar will be reproduced at variable levels of clarity. The clarity of the detail in the impression may reveal the overall flow of the scar, the path of the scar, and the edge shapes of the scar.

Scars! To use, or not to use? Based on the anatomy and physiology of wound healing in the epidermis, the comparison of scars in friction ridge impressions is perfectly valid. The details of a scar, when present in two impressions, have the same individualizing power as friction ridge paths and friction ridge morphology. In the examination process, the significance of a scar should be weighted on its clarity in the impressions. Scars certainly should not be ignored. If a scar is present in one impression, but not in the comparable area of the second impression (i.e. prior to injury), the examiner should be well equipped to explain the aberrations in ridge flow and crease path of the scarred impression based on the knowledge of how the skin heals.

When analyzing the details of friction ridge impressions, always remember the source of those details – the skin! An examiner is better equipped to evaluate the significance of the details of ridges, creases, and scars if the examiner understands why those features are unique and how they persist.

Bibliography

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