A PRIMER ON THE SKIN BARRIER

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Note from the Authors

We hope you will enjoy our Primer on the Skin Barrier and find it helpful to your understanding of the skin and how it functions to keep us well. We have written it to be understandable to all readers who are curious to learn more about the skin and the skin’s barrier.

A medical background is not required to understand the material we present; we will define all technical terms and offer informative illustrations to help our readers visualize the concepts we discuss.

The barrier, in many respects, is at the center of the skin’s universe.

Although this material will be accessible to all, we are particularly eager to reach the many varieties of skin professionals, from the cosmetologists working to enhance the beauty of their client’s skin, to the cosmetic scientists working to develop new skin care products, to the broad community of medical professionals who treat skin and its disorders.

We believe that the material that we present here, though basic to skin function, may nonetheless be new to skin professionals across this wide range of interests. It is our hope that even dermatologists may find that some of our ideas offer them a new way to understand skin function that is useful to them in their clinical practice.”
A PRIMER ON THE SKIN BARRIER

Introduction

The primary duties of skin are to provide a protective shield against assaults from the outside and at the same time to erect a barrier capable of preserving the body’s internal milieu. The primary task of this latter function, the ’permeability barrier’, is to preserve our water molecules, because we are mostly water (about 80% of our body is water), but we live in a dry (‘terrestrial’) environment.

A skin barrier of necessity evolved as a means to prevent the outward movement of water when plants and animals left the wet seas to inhabit the land. Our skin’s barrier minimizes the evaporation of our body’s water into the surrounding, drier atmosphere. Thanks to it, we do not shrivel up; we are grapes and not raisins, and plums, not prunes.

Of all the skin’s many functions, this, its permeability barrier to the loss of body water, is its most important task.

Of all of skin’s many functions, this, its permeability barrier to the loss of body water, is its most important task, because without a competent skin barrier, life on earth would not be possible for more than just a few hours. Many of the skin’s other important protective functions – for example, its ability to withstand frictional injury and its ability to prevent invasion by microbes (bacteria, viruses and fungi), as well as to prevent the uptake of foreign chemicals – are closely tied to the
competence of its permeability barrier function. When the permeability barrier fails, these other protective functions also decline; and conversely, a defect in one of these functions may also impair the permeability barrier. The barrier, in many respects, is at the center of the skin’s universe.

Our skin generates its permeability barrier in the outermost layers of epidermis, the stratum corneum. To understand how it accomplishes this task, let us briefly examine the structure of the skin.

**The Structure of Skin**

![Illustration of skin structure](image)

Illustrated by *Jessica Kraft*

Skin has two major components, the outer layer or ‘epidermis’, and the inner layer or ‘dermis’. Below the dermis is a layer of subcutaneous fat, while still further below reside muscles, fascia, and finally, cartilage, tendons and bone. The epidermis is the principal player in the barrier story, relegating the dermis to play a critical,
Although the epidermis is much smaller in volume than the dermis, it is the ‘business end’ of the skin, because it must produce the permeability barrier and keep it operational.

pressure. Finally, extending deep into the dermis, but originating from, and connected to the overlying epidermis, are many sweat glands, oil or ‘sebaceous’ glands and hair follicles.

Although the epidermis is much smaller in volume than the dermis, it is the ‘business end’ of the skin, because it must produce the permeability barrier and keep it operational. The epidermis itself is composed of multiple layers of cells, called ‘keratinocytes’, that are bound to each other by special protein bridges called ‘desmosomes’. Scattered amongst the keratinocytes are other specialized cells of the immune system (e.g. Langerhans cells) and of the sensory nervous system (e.g. Merkel cells); and still other cells (melanocytes) that generate skin pigmentation.
The Epidermis

The innermost layer of the epidermis, which rests upon the dermis, is composed of progenitor or mother cells, called ‘basal cells’. These mother cells divide periodically, sending their progeny outward toward the skin surface. As these daughter keratinocytes push outwards to make way for new siblings, they undergo a series of carefully orchestrated changes. Like children, they progressively mature – or in medical parlance, they ‘differentiate’. They increase in size (‘hypertrophy) as more and more proteins accumulate.

One group of these accumulating proteins comprise a family of fibrous molecules, collectively termed ‘keratins’. While keratins are found in many types of cells,
Certain keratins are unique to epidermis, as are several other proteins produced by these differentiating keratinocytes.

Finally, the keratinocytes undergo ‘terminal differentiation,’ as they digest away their nuclei and other recognizable internal organelles, including energy-generating mitochondria. These outermost epidermal cells that lack nuclei are called ‘corneocytes.’ Layer-upon-layer of flattened corneocytes arrange themselves into vertical, interwoven stacks, forming the outermost part of the epidermis, the ‘stratum corneum.’

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Corneocytes, too, are bound to their neighbors by their own type of specialized protein bridges, called ‘corneodesmosomes.’

While there are approximately 20 layers of corneocytes over much of our skin surface, on certain body surfaces that are specially adapted to resist mechanical and frictional insults, for example, the palms and soles, 40 or more cell layers may be present. With the continual addition of cells from below, eventually each corneocyte reaches the skin surface, where it detaches from its neighbors, and is shed.
This process, desquamation, is usually invisible to the naked eye, but in some skin conditions where desquamation is faulty, instead of loss of individual cells, clusters of corneocytes visibly flake off.

Conditions with abnormal desquamation can be as common as dry skin or dandruff and as rare as some members of a family of genetic skin disorders called ‘ichthyosis’. Although these inherited disorders of desquamation are uncommon, they have provided important insights into the normal process of skin shedding. The mechanics of desquamation are closely linked to both the cohesion of the corneocytes one to another and to the competence of the permeability barrier. (For more information, see our articles Scaly Skin and Ichthyosis (Nothing to do with fish skin) and “Scaly Skin and the Barrier”)

Structure of the Barrier: The Bricks and Mortar

Paradoxically perhaps, the stratum corneum, which is made up of ‘dead’ cells that lack nuclei, is the business end of the epidermis, for it is here that the permeability barrier resides. This fact can be demonstrated readily by using adhesive tape to strip away the stratum corneum layer by layer: only when all its layers are stripped away, does water begin to leak out of the skin.

Because the cells that make up the stratum corneum are filled with keratin fibers, it has been often assumed that keratins are the magic molecule behind the skin’s barrier. But, if instead of peeling off the stratum corneum with adhesive tape, one merely wipes the skin surface repeatedly with a solvent or a detergent, the same
result is observed: water losses accelerate. Yet, little or no keratin was removed or denatured by these solutions. Instead of dissolving or inactivating keratins, solvents and detergents abrogate the barrier by extracting fats or ‘lipids’. In the stratum corneum these lipids comprise a very unique mixture of highly water-repellant species that fill the extracellular matrix, and surround and envelop each corneocyte.

Think of the stratum corneum as analogous to a brick wall in which the corneocytes are the bricks, and the ‘bricks’ are embedded in a lipid-enriched ‘mortar’. In a simplistic sense, the cells are packages of fibrous proteins or keratins and each protein package is surrounded by a thick slice of fat. Then, translating structure into function, it is the accumulation of layers of tough, fibrous protein-enriched cells, surrounded by sheets of water-repellant lipids, that produces the permeability barrier, allowing us to live on land without desiccating and to bathe without drowning.

How does our remarkable external tissue, the epidermis, and its outermost layer, the stratum corneum, accomplish this potentially daunting task? To see how the bricks and the mortar came to be where they are, and how they work together to generate
These smaller, breakdown products of filaggrin have been collectively referred to as ‘natural moisturizing factor’ or NMF, because they attract and bind water within the corneocyte, keeping the outer skin layers hydrated.

A Closer Look at the Bricks

We will first turn our magnifying glass on the corneocytes. These ‘dead’ cells are filled with bundles of keratin proteins, like cables of coiled wire, forming its backbone and contributing greatly to the mechanical integrity of skin. Also residing inside the corneocyte, and surrounding the keratins, is another protein, ‘filaggrin’, which, like the keratins, persists after the nucleus and other organelles of the terminally differentiating keratinocyte have been digested and their components recycled to the living cells below.

Yet, filaggrin, too, eventually is digested by ‘proteases’ (enzymes that break down proteins) in the corneocyte, generating a bevy of smaller molecules. These smaller,
breakdown products of filaggrin have been collectively referred to as ‘natural moisturizing factor’ or NMF, because they attract and bind water within the corneocyte, keeping the outer skin layers hydrated.

Still, other proteins form a sheath around the corneocyte, called the ‘cornified envelope’. This structure incorporates over twenty different proteins, many of which are unique to epidermis, which are progressively bound together (or ‘cross-linked’) by chemical connections. The cornified envelope forms a rigid, mechanically resistant, but porous edifice that surrounds each corneocyte, much like a hedgerow of roses around a small plot of land. Along with the keratin fibers, the cornified envelope is an important component of our skin, providing strength and resilience against the frictional assaults of every day life.

The permeability barrier lies at the heart of all of these defensive functions - it is the kingpin of the skin’s protective apparatus.

A Closer Look At the Mortar

Let us now turn our magnifying glass to the mortar. It is composed of multiple sheets of lipids or lamellar membranes. The lipids that make up these membranes are for the most part manufactured by epidermal cells and packaged within small ovoid structures inside the keratinocytes, called ‘lamellar bodies’.

As the first or innermost layer of stratum corneum forms, the contents of each lamellar body package are extruded (‘secreted’) from the keratinocyte, and then reorganized into the lamellar membranes that fill the extracellular matrix surrounding the corneocytes.
**Electron micrograph of the lamellar body secretory system:** The epidermal lamellar body is a small organelle, filled with lipids forming pleated sheets (Lower left). At the outermost nucleated cell layer of the epidermis (the granular cell layer), the lamellar bodies release (‘secrete’) their contents into the spaces between the cells of the innermost stratum corneum layer. (Lower right). The secreted contents are then modified (‘metabolized’) by ‘lipid-processing’ enzymes to form broad sheets or lamellae (Top), that fill the ‘extracellular’ regions between the corneocytes.
This organization of lipids into lamellar membrane structures is a critical feature of a competent permeability barrier. In other words, it is not sufficient merely to have fat, even if highly water-repellent, between the corneocytes - these lipids must also be present in the correct form, as multiple layers of lamellar membranes, to ensure optimal barrier function. And this reveals another function of the cornified envelope beyond its mechanical strength. Because, despite the fact the cornified envelope itself is a leaky structure, it is needed to provide a scaffold for the lipids to organize themselves into the membrane structures that will form the barrier to water movement.

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The lipid mixture forming the stratum corneum lamellar membranes is quite unique in nature. In contrast to all other membranes made by cells throughout the rest of the body, these membranes lack a class of lipids called 'phospholipids'. These relatively 'polar' lipids are ordinarily required for the organization of other, less polar lipids, such as cholesterol, into membrane structures. Indeed, all other membrane structures in the body are in the form of phospholipid-based membranes. Yet, the lipids of the stratum corneum, though lacking the membrane-forming phospholipids, are also assembled into highly organized membrane structures.

The lipids that make up the stratum corneum membranes are strikingly non-polar, i.e., they are unusually hydrophobic (or ‘water-avoiding’). Three major types of lipids make up these membranes: sterols (and specifically, cholesterol), ceramides and free fatty acids. The carbon chain length of the fatty acids – both as free fatty acids.

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1 Lipids are categorized as polar vs. nonpolar depending on their relative affinity for water.
acids and as esters in the ceramides is ‘long’ (16 to 18 carbons) to ‘very-long’ (20 to 34 carbons); these long hydrocarbon tails are highly hydrophobic. Each class is essential to form the membrane structures. Moreover, the essential fatty acid, linoleic acid is also required for formation of the lamellar membranes.

If any of these 3 lipid classes is deficient, the result is disorganized or non-lamellar regions that provide an avenue for the loss of water from the body and for the entry of other molecules from the outside world. In addition, each of the three lipid classes must be present in the proper proportions; i.e., a 1:1:1 molecular ratio, termed the ‘critical mole ratio’, in order to form these lamellar membranes. Finally, one must produce and secrete enough of the lipids to generate multiple stacks of lamellar membranes (see Top panel in the electromicrograph, above). Normally, the lipids of the stratum corneum comprise about 10% of the mass (weight) of the tissue.
How The Barrier Works

Think of the body as an oil and water system: its cells with their many membranes are like the oil droplets, and the ‘interstitial’ fluid between the cells, which is in continuity with the blood stream, is a water compartment around these oil droplets.

Reversal of aqueous and lipid compartments: The cells of the living epidermis, ‘keratinocytes’, like other cells in the body, contain many membranes that are water repellant or ‘hydrophobic’. They are surrounded by ‘interstitial’ fluid that is in equilibrium with the fluid in the blood stream, forming a water-friendly or ‘hydrophilic’ compartment. In contrast, the cells of the stratum corneum, ‘corneocytes’ are devoid of lipids and hence form a water-friendly compartment, whereas the lamellar membrane lipids of the external ‘mortar’ are hydrophobic. This role reversal – the water-loving external milieu of the living epidermal cells layers overlaid by the water-repellant external milieu of the stratum corneum – forms the skin’s permeability barrier.

By reversing the two oil-and-water systems - cells that previously were ‘oil’ are now ‘water’, and the intercellular water has now been replaced by ‘oil’ - in this way, the stratum corneum seals in the body’s water.
Now visualize the stratum corneum, where the situation is reversed: the corneocytes, now devoid of a plasma membrane or organelles, but still filled with small water-loving or ‘hydrophilic’ molecules, have become the water compartment; while the lamellar membranes, made up of water-repellant or ‘hydrophobic’ lipids that surround the corneocytes, are now the oil (figure from page 16).

By reversing the two oil-and-water systems – cells that previously were ‘oil’ are now ‘water’, and the intercellular water has now been replaced by oil - in this way the stratum corneum seals in the body’s water.

Illustrated by Jessica Kraft

The stratum corneum, with its multiple layers of cells each coated by multiple layers of water-repellant membranes, interposes a highly redundant barrier to the loss of water from the aqueous system of the interior. Both the outward and inward movement of water effectively stop; and simultaneously, the movement of water-soluble molecules is halted.

**The Stratum Corneum as a Dynamic Interface**

The stratum corneum contains abundant, ongoing metabolic activity, even though its cells are no longer capable of synthesizing new proteins, because they lack a nucleus and the other cellular machinery of molecular synthesis. Although the
corneocytes cannot make new molecules from scratch, the stratum corneum can modify existing molecules for its purposes.

Many enzymes are non-energy-requiring and remain active in the stratum corneum. Stratum corneum deploys a mixture of proteolytic enzymes and their inhibitors to exquisitely orchestrate the invisible shedding of corneocytes from its surface. It also continues to refortify the corneocytes, by cross-linking additional peptides into the cornified envelope, rendering the cells even more rigid as they migrate outwards. It also modifies the lipids that were secreted from the lamellar bodies into even more hydrophobic species that will form the lamellar membranes.

This latter sequence is orchestrated by yet another set of enzymes, collectively termed ‘lipid processing’ enzymes. In addition, the extracellular matrix of the stratum corneum is studded with proteins that can interdict invading microorganisms, called ‘antimicrobial peptides’.

Although officially ‘dead’, the stratum corneum can ‘talk back’. Whenever the skin’s critical permeability barrier function is threatened, for example as the skin becomes leaky during a hot, soapy shower, or when the layers of
cells are mechanically stripped away, when it is burned by exposure to ultraviolet light or, indeed, disturbed by any other type of insult - chemical signals are released from the stratum corneum that direct the underlying nucleated cells to respond. These signals order the manufacture of the constituents necessary to restore normal barrier function.

Using biochemical language, they order the epidermis to quickly ‘Spit out the lipids that it already has made!’; then, to ‘Make more lipid, as quickly as possible!’; and finally, it orders the epidermis to ‘Make more cells!’ Sequentially, when the barrier is abrogated: lamellar bodies are secreted within a few moments and granular cells transform themselves into corneocytes; then, within minutes-to-hours new lipids begin to be generated in the differentiating cell layers of the epidermis; and then several hours later, the basal layer accelerates its production of new cells. In a somewhat paradoxical fashion, then, this ‘dead’ tissue, the stratum corneum, which is composed of cells without nuclei, can be analogized to the queen bee, with the cells of the underlying nucleated epidermal layers, her willing nymphs and workers. It is the counter-intuitive nature of this relationship - dead cells commanding the activities of the living - that have continued to impede a full appreciation of stratum corneum function.

The stratum corneum uses a variety of molecular signals for these purposes. Two of these signaling molecules are the ‘cytokines’, interleukin-1alpha and interlekin-1beta. These cytokines lie in wait within the corneocytes. When the barrier is threatened by one of these insults, these two molecules are released (‘activated’), initiating a cascade of other cytokines and signaling molecules (e.g., TNFα, nerve growth factor, amphiregulin, etc.) that will direct the repair process.
This sequence of injury-signaling-repair is completed within a few hours of an acute insult to the skin barrier, but if the injury persists or is repeated, cytokines continue to be released, and eventually they recruit an inflammatory response of increased blood flow to the region, followed by influx of lymphocytes or other leukocytes. Inflammation, with the recruitment of white blood cells to the skin, is not all bad – these cells are the warriors in the battle against infectious microbes that dare to enter the skin. But persistent inflammation is not always good either, because the resultant redness, itching, and irritation will eventually produce a visible and symptomatic rash.

This sequence is operative in some skin conditions like atopic dermatitis, and perhaps even in psoriasis, where function of the barrier is compromised due to inherited defects in its structure. In these disorders, the inherently defective skin
barrier initiates a process of inflammation that cannot turn itself off. Indeed, the recent awareness that outbreaks of eczema may be initiated by a barrier defect is changing the current treatment paradigms - away from merely treating the consequence (inflammation) and towards treating the cause (impaired barrier).

The Super-Barrier

Though at first glance, the barrier to water loss lies in the skin, as we look closer, we now see that the barrier is in the epidermis, and then upon still-closer examination, it’s in the stratum corneum. Finally, we see that the barrier localizes to the lipids that form the lamellar bilayers between the cells of the stratum corneum. The lamellar membranes of the stratum corneum not only restrict the movement of water into and out of the body, but they also pose a formidable barrier to the ingress of foreign molecules, such as allergens (antigens), free radicals and toxins, as well as against disease-causing microorganisms.

Although it is convenient to consider each of these functions (permeability barrier to water loss, prevention of toxin/allergen penetration, antioxidant defense, blockade of microbial entry, and of UV light penetration) as discrete processes, there is evidence that each of these functions is interdependent and intertwined with the other. In other words, you can’t have one without the other. And if you repair one function, as for example by applying the proper mixture of lipids to repair a damaged permeability barrier, you will simultaneously correct a whole bevy of other functions.

This ‘super-barrier’ then protects not only against bugs, toxins and allergens, but also against free radicals, UV-irradiation, mechanical/frictional insults, and changes in environmental humidity or temperature (see Table below). The permeability barrier lies at the heart of all of these defensive functions – it is the kingpin of the skin’s protective apparatus. Therefore, understanding how the barrier operates in health, how it changes over our lifetime and in response to environmental challenges and how a damaged barrier contributes to skin diseases is central to our appreciation of this miracle, our protective skin.
Table: Protective Functions of Skin and Their Relationship to the Permeability Barrier

1. Stratum Corneum Hydration
   - The permeability barrier affects corneocyte hydration:
     - An incompetent barrier allows water within the corneocytes to escape.
   - Hydration affects the barrier:
     - Breakdown products derived from filaggrin hydrate corneocytes and contribute to the acidification of the stratum corneum.
     - Filaggrin deficiency results in impaired corneocyte hydration.
     - ‘Dry’ corneocytes steepen the water gradient between the interior and external environments, putting additional ‘stress’ on the barrier.
     - Filaggrin deficiency also results in loss of the skin’s ‘acid mantle’.
     - A less acidic stratum corneum produces a defective permeability barrier.

2. Antimicrobial Defense
   - The permeability barrier affects antimicrobial defense:
     - Free fatty acids in the lamellar membranes are potent antimicrobial agents, especially active against S. aureus and yeasts.
     - Barrier competence regulates expression of at least 2 key antimicrobial peptides, CRAMP (a cathelicidin) and human beta-defensin 2.
     - When the barrier is impaired, CRAMP content also declines.
     - When barrier competence is restored or enhanced, CRAMP content increases in parallel.
• Antimicrobial competence affects the permeability barrier:
  ◆ Reduced free fatty acid content results both in poor barrier function and in impaired antimicrobial defense.
  ◆ Reduced CRAMP (a mouse cathelecidin) content results in poorly formed lamellar membranes and an impaired permeability barrier, as well as in impaired antimicrobial defense.
  ◆ Cathelecidins may also function as apoproteins for lamellar membrane organization.

3. Mechanical Strength

• Corneocyte envelope integrity affects the barrier:
  ◆ An intact corneocyte envelope is required to provide a scaffold for the deposition of the lamellar membrane structures.
  ◆ A poorly formed corneocyte envelope results in poorly formed membranes and a defective permeability barrier.

• Keratin filament integrity affects the barrier:
  ◆ Abnormally formed keratin fibers reduce the mechanical strength of skin.
  ◆ An intact keratin filament cytoskeleton is required for normal secretion of lamellar body contents into the stratum corneum.
  ◆ Abnormally formed keratin fibers impede the secretion of lamellar bodies and result in a reduced quantity of lamellar membranes and an impaired permeability barrier.

4. Ultraviolet Light Defense

• The barrier affects UV defense:
  ◆ Filaggrin breakdown products are important both for barrier competence (see above) and for UV defenses, because ~50% of incident UV light is absorbed by these molecules.
• UV light exposure affects barrier competence:
  ◆ Moderate UV exposure increases epidermal production of barrier-related lipids and proteins that improve barrier competence.
  ◆ High dose UV light exposures (e.g. sun-burns) damage the barrier though toxic effects on the epidermis.

5. Antioxidant Defenses
• The barrier affects antioxidant defenses:
  ◆ A competent permeability barrier blocks the penetration of free radicals and other oxidants in the environment.
• Antioxidants improve barrier competence:
  ◆ Polyphenolic antioxidants, such as apigenim and hesperidin, increase epidermal production of barrier-related lipids and proteins that improve barrier competence

6. Neurosensory Interface
• Changes in the physical environment alter barrier competence
  ◆ Physical changes, such as temperature, acidity, mechanical pressure and osmolarity acting through TRPV receptors increase epidermal production of barrier-related lipids and proteins that improve barrier competence.
• Neurotransmitters in epidermis affect barrier competence:
  ◆ Histamines decrease barrier competence by decreasing production of barrier-related lipids and proteins by the epidermis.
  ◆ Antihistamines increase barrier competence by increasing epidermal production of barrier-related lipids and proteins.
Suggested Reading


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Peter M. Elias, M.D. is a Professor in the Department of Dermatology at the University of California San Francisco, and a Staff Physician at the Veterans Affairs Medical Center, San Francisco. He received his medical degree from the University of California San Francisco and trained in dermatology at Harvard University. Dr. Elias has authored or co-authored over 550 peer-reviewed scientific articles, innumerable review articles, and he has edited or coauthored three books on the subject of the skin barrier in health and disease. He is an invited speaker at national and international scientific meetings, is a consultant to the pharmaceutical and cosmetic industries, and has received numerous awards for his scientific discoveries including the William Montagna and the Kligman-Frost Awards from the Society of Investigative Dermatology.

Mary L. Williams, M.D.

Mary L. Williams, M.D. is a Clinical Professor of Dermatology and Pediatrics at the University of California San Francisco. She received her medical degree from the University of Chicago, trained at the University of California San Francisco, and is board certified in both pediatrics and dermatology. Her laboratory research has focused on the pathogenesis of inherited disorders of cornification (the ‘ichthyosis’) and on the prenatal development of the epidermal permeability barrier. She is the author of over 150 peer-reviewed scientific publications, and an invited speaker at regional, national and international scientific meetings. She is the recipient of a Distinguished Service Award from the American Academy of Pediatrics. Currently Dr. Elias and Dr. Williams are writing a book on the skin permeability barrier.

To contact Elias and Williams, please click here.