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# Aetiology of Disruption of Normal Skin Barrier in Urban Communities

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## ABSTRACT

The anatomy of the skin is a well-organized network of three layers, the epidermis is the outermost layer and is composed of keratinocytes or skin cells. Beneath the epidermis is the dermis, composed mostly of collagen but also adjunctive structures like hair follicles and sweat glands. Sebaceous glands are found next to hair follicles and produce sebum, a combination of natural lipids that coat the skin's surface and provide a protective nourishing role. Sweat glands function to help regulate temperature through evaporation and cooling. Their ducts pass through the dermis and epidermis to empty directly onto the skin's surface. The dermis also contains vital blood vessels and nerves which traverse the collagen network there. The subcutaneous layer which holds fat and larger blood vessel lies underneath the dermis. It acts as a heat insulator and provides protection from mechanical trauma. Pathophysiological processes affect all layers of the skin which damage to the skin barrier is the usually the first step.

**Keywords:** Skin barrier; Trimethoprim-Sulfamethoxazole; Particulate matter; Desquamation

## INTRODUCTION

This article provides a thorough review of the multiple normal homeostatic functions of the stratum corneum, comprehensive analysis of endogenous and exogenous social, pharmacological and physical factors causing significant damage to normal skin barrier in urban environments. The outermost layer of the epidermis is the stratum corneum. The stratum corneum provides permeability barrier to the skin which is crucial for regulating trans-epidermal water loss thereby preventing excessive water evaporation from the body and maintaining proper hydration levels. It also acts as a barrier against the penetration of exogenous substances like allergens, irritants,

chemicals and pathogens. This barrier is formed by corneocytes embedded in a lipid matrix, often described using the "bricks and mortar" analogy, where corneocytes are the bricks and the lipid layers are the mortar. The stratum corneum provides mechanical strength and resilience, protecting against physical damage and shear forces. The dense packing and keratinization of corneocytes, along with the lipid matrix, contribute to the skin's structural integrity.Another important function of the stratum corneum is maintenance of the skin's slightly acidic pH, known as the acid mantle, to discourage the growth of harmful bacteria and support beneficial microorganisms. The stratum corneum also contains lipids with antimicrobial properties and produces antimicrobial peptides like defensins and cathelicidins that combat pathogens [1]. Antioxidant barrier of the stratum corneum contains a network of enzymatic and non-enzymatic antioxidants like alpha-tocopherol (Vitamin E), ascorbic acid (Vitamin C) and glutathione that help neutralize harmful reactive oxygen species from environmental sources like Ultraviolet UV radiation and environmental pollution. This protects skin lipids and proteins from oxidative damage. Photo-protective barrier of the SC contributes to photo-protection through the presence of melanin, which absorbs and scatters UV radiation. Desquamation and cohesion; the stratum corneum maintains its thickness by shedding old corneocytes through a process called desquamation. This process is regulated by enzymes that break down corneodesmosomes, the structures that hold corneocytes together and adequate hydration is essential for this process.

The stratum corneum plays a vital role in initiating immune responses against invaders through the involvement of dendritic cells and toll-like receptors.1The stratum corneum has a surface pH of approximately 4 to 5.5 and this acidic pH, the “acid mantle” of the SC may play a role in protecting against colonization of the skin surface by harmful bacterial [2]. Korting, et al [2] also reported increases in skin pH following the use of natural soaps and a concomitant increase in skin colonization by coagulase-negative staphylococci. Below the SC is the stratum granulosum, made of keratinocytes that have granules containing proteins such as filaggrin. Keratinocytes also produce lipids such as triglycerides and cholesterols functioning as part of the chemical level. Tight junction proteins connect adjacent keratinocytes within the stratum granulosum to form a barrier against water and solutes [3].

Trimethoprim-sulfamethoxazole has been identified as a key pharmacological agent that plays a role in the damage of skin barrier proteins specifically desquamation. The body's immune cells may recognize the drug or its metabolites as foreign, leading to an inflammatory response that damages the skin cells (keratinocytes) and causes the epidermis to detach or peel off (Figure 1).The established literature hypothesizes that drug-specific CD8+ cytotoxic T cells utilizing perforin/granzyme B trigger keratinocyte apoptosis [4]. Environmental pollution in urban areas (Figure 2) contribute significantly to damage of skin barrier and expose individuals to a mixture of airborne pollutants, including particulate matter (PM), gases (ozone, nitrogen dioxide) and volatile organic compounds (VOCs). These pollutants can damage the skin barrier through mechanisms like oxidative stress and inflammation; IL-1α, IL-1β, IL-6, TNF-α are the major proinflammatory cytokines that are responsible for early response. Exposure to fine particulate matter (PM2.5) has been linked to increased trans epidermal water loss (TEWL) and decreased levels of filaggrin, a protein essential for skin barrier function.

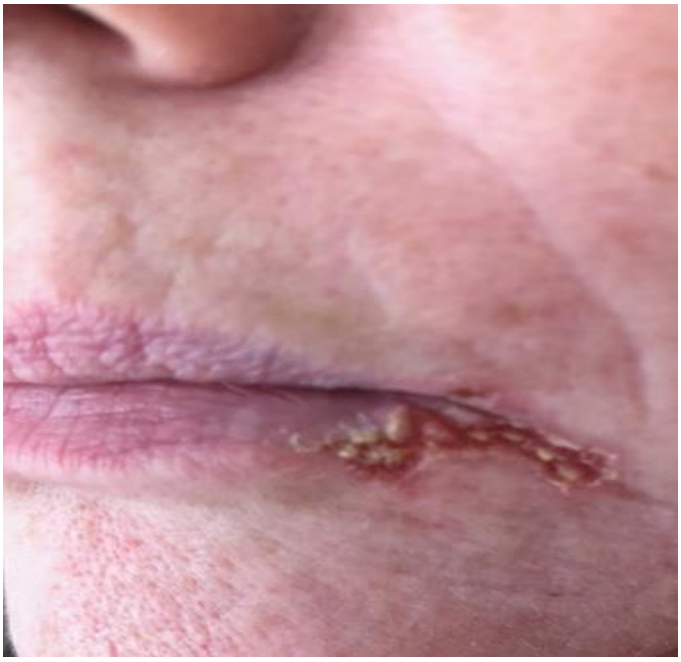

**Figure 1:** Shows abnormal desquamation triggered by ingestion of Trimethoprim-sulfamethoxazole

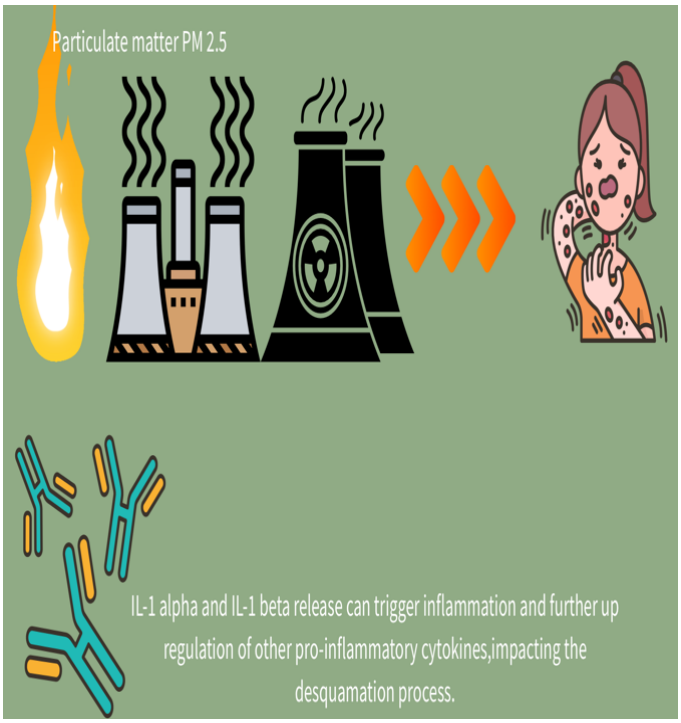


**Figure 2:** Effects of Environmental Pollution on skin barrier.

## CONCLUSION

The different mechanisms involved in disrupting normal and healthy skin barrier are a chain reaction of exposure to airborne pollutants; gases, particulate matter from open wild fires, boilers, diesel engines (Figure 2), volatile organic compounds, damage by oxidative stress and important pro-inflammatory cytokines. Consequently, Atopic dermatitis has been associated with dysfunctional skin barrier. Impairment of the skin barrier increases the likelihood of external antigens, irritants, and pathogens passing into the skin and driving inflammation, potentially leading to skin infections or allergies. The challenges identified can be approached through prevention strategies; oat-based creams and emollients.

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