# The Relationship of Proper Skin Cleansing to Pathophysiology, Clinical Benefits, and the Concomitant Use of Prescription Topical Therapies in Patients with Acne Vulgaris

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**KEYWORDS**

• Acne vulgaris • OTC products • Surfactants • Skin irritation • Skin barrier function • Sensitive skin • Retinoids • Benzoyl peroxide

**KEY POINTS**

- Patients often perceive the cause of their acne to be related to poor hygiene and a lack of proper cleansing, therefore many patients with acne attempt to treat their acne either alone or with prescription therapy by frequent aggressive skin cleansing with harsh cleansing agents.
- Altered epidermal barrier function, inflammation, and *Propionibacterium acnes* are related components to acne vulgaris (AV) pathophysiology; proper cleansing can favorably modulate the development of AV.
- Benzoyl peroxide (BP) and topical retinoid therapy (ie, tretinoin) can adversely alter skin barrier function and cause cutaneous irritation, thus affecting patient tolerability and compliance with AV. Improvements in vehicle technology may mitigate the barrier impairment that may be associated with these therapeutic agents.
- Harsh cleansers, such as true soap and cleansers with high alkaline pH, adversely affect the skin by increasing skin pH, impairing the stratum corneum (SC) permeability barrier function, altering skin bacterial flora, desiccating the SC, increasing erythema, inducing symptoms of subjective irritation, and promoting follicular plugging.
- Combars with an added antibacterial agent do not decrease the amount of *P acnes* on skin and may promote gram-negative folliculitis if there is preferential reduction in commensal gram-positive bacteria. Therefore, true soap and combars are not ideal products to use in most skin diseases, including AV.
- Syndet bars and lipid-free cleansers have the potential to gently cleanse the skin without markedly diminishing epidermal barrier function. This process optimally prepares the SC for the application and absorption of topical therapies while minimizing skin irritation, reducing skin dehydration from prescription therapies, and maintaining the physiologic acid mantle pH of the skin.
- The limited clinical studies available support the benefit of gentle cleansing in AV by showing the ability to contribute to improving AV lesion counts and severity and minimizing the irritation seen with topical AV therapies such as retinoids and BP.

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## INTRODUCTION

Worldwide, acne vulgaris (AV) is one of the skin disorders for which patients most frequently consult a dermatologist.[1] The economic and psychosocial burden of AV is high, and it constitutes the most common reason for dermatologist consultation.[2,3] The direct cost of AV in the United States is estimated to exceed $1 billion per year, with $100 million spent on over-the-counter (OTC) AV products.[3,4]

AV is a polymorphic skin disorder that produces a series of lesions: comedones, cysts, pustules, papules, or nodules. The primary goals of acne therapy are to achieve initial control, maintain therapy to prevent flares, and prevent persistent or permanent sequelae such as scarring. An important aspect of AV management that is often forgotten by physicians is to dispel any myths and misperceptions the patient may have about the cause of their AV, and to develop an appropriate management plan that includes adjunctive OTC products that serve to support their prescription regimen.[4]

Many patients with AV mistakenly think that aggressively cleansing their skin with soap and water several times a day is therapeutic for AV. A survey of patient perceptions of AV showed that 29% of patients thought AV was caused by poor skin hygiene, and 18% thought it was caused by infection, with 61% of patients thinking dirt was an aggravating factor.[5,6] Even among medical students, 25% thought poor facial hygiene was an exacerbating factor.[6] For generations, even physicians thought that successful treatment of AV depended on the degreasing of the skin to an extent that produces desquamation with noticeable peeling.[7] Because of these perceptions, patients tend to cleanse diligently and harshly with the belief that the more cleansing the better.

In the 1980s things started to change and the suggestion was made that inducing visible inflammation and desquamation of the skin was not necessary for acne control.[5,6] Also, it was discovered that the lipid in the follicular reservoir that plays a role in AV pathogenesis cannot likely be reached by harsh soaps and detergents or by frenetic washing; aggressive cleansing with harsh soaps can aggravate AV and, under certain circumstances, cause a detergent-induced acneiform eruption.[8] In addition, overzealous cleansing can lead to disruption of the epidermal barrier,[9–11] increased transepidermal water loss (TEWL),[9–11] roughened and irritated skin,[10,12] increased bacterial colonization,[12] increased comedonal formation,[8] secondary irritant contact dermatitis,[10] and burning and stinging.[13–15] These negative effects caused by harsh soaps and aggressive cleansing make many prescription topical AV medications less tolerable.[4,16] It is for these reasons that many dermatologists are now recommending gentle cleansers rather than the harsh soaps and cleansers used in the past with the hope of improved patient outcomes and increased compliance.[9]

Although there is a plethora of data on the tolerability and benefits of mild cleansers in other skin disease, such as atopic dermatitis, data are more limited concerning their benefit in AV.[11,17–21]

This article presents and summarizes the available scientific evidence concerning the use of gentle cleansers in AV.

## ACNE PATHOGENESIS

The pathogenesis of acne is multifactorial, involving follicular hyperkeratinization leading to (1) comedo formation; (2) hormonal (androgenic) stimulation of the sebaceous glands leading to increased sebaceous gland size and sebum secretion; (3) proliferation of *Propionibacterium acnes*; and (4) induction of a variety of inflammatory cascades, some triggered in response to *P acnes* proliferation. These factors are summarized in **Box 1**.[22]

### *Follicular Wall Barrier Dysfunction*

One of the primary events in the pathophysiology of acne is subclinical aberrant follicular wall hyperkeratinization,[23,24] leading to a plugged follicular orifice (microcomedo).[23] There several hypotheses regarding the pathophysiologic mechanism of follicular wall hyperkeratinization seen in acne; however, decreased stratum corneum (SC) barrier function (BF) has been suggested as a cause of reactive follicular wall hyperkeratinization, abnormal desquamation, and follicular plugging or comedo formation.[25,26] Because pilosebaceous units have long canals through which sebum flows, hyperkeratinization of the follicular epithelium easily leads to sebum sequestration, which forms a microenvironment conducive to *P acnes* proliferation.[26]

**Box 1**
**Summary of the 3 key pathogenic factors in acne**

1. Follicular hyperkeratinization
2. Hormonal stimulation of the sebaceous glands
3. Inflammation in response to *P acnes*

*Data from* Webster GF. Acne vulgaris and rosacea: evaluation and management. Clin Cornerstone 2001;4(1):15–22.