Skin Cleansers and Epidermal Barrier: A Narrative Review

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ABSTRACT

Introduction. Skin hygiene is essential, but the type of cleanser, its pH and the pattern of use determine the impact on the barrier function and the risk of irritant dermatitis. This review summarizes damage mechanisms and comparisons between alkaline soaps and syndet/moisturizing cleansers.
Methods. Narrative review under SANRA. Source: PubMed/MEDLINE (Humans; last 10 years). Reproducible strategy with MeSH terms/keywords related to cleansers, skin barrier and TEWL. Last search: Nov 1, 2025. Inclusion: human studies evaluating pH, TEWL, capacitance/hydration, NMF or clinical signs. Exclusion: animals, editorials without data, duplicates. n=53 articles were identified.
Results. Alkaline soaps raise pH, draw lipids from the stratum corneum, and increase TEWL; Frequent washing, wet work and occlusion with gloves increase the damage. In contrast, syndets (pH 4.5–5.5; nonionic/amphoteric surfactants) and wetting cleansers show less impact on pH/TEWL and better tolerance. In intensive hygiene scenarios, hydroalcoholic solutions with emollients may be less lipid-reducing than repeated washing with soap.
Conclusions. For daily and repeated use, it is recommended to prefer syndets with physiological pH or moisturizing cleansers, minimize friction and contact time, alternate with ABHR with emollients when hygiene is intensive, moisturize immediately after washing and limit occlusion. Standardized comparisons (pH/TEWL/capacitance) and longer studies are required in atopic dermatitis, older adults, and high-risk occupations.

Keywords: skin barrier, transepidermal water loss, irritant contact dermatitis, soaps.

INTRODUCTION

The skin constitutes the body's main protective barrier against the external environment, with the stratum corneum (SC) being the fundamental layer responsible for this function. This structure maintains a delicate homeostasis that prevents excessive water loss and blocks the penetration of harmful substances [1].

Skin hygiene is an essential practice for health and infection prevention, the importance of which has been accentuated in high-demand contexts such as the COVID-19 pandemic [2]. However, the frequent use of cleansing agents presents a dilemma: the need for cleansing directly conflicts with preserving the integrity of the skin barrier [3].

Traditional alkaline soaps contain anionic surfactants such as sodium lauryl sulfate (SLS), known to alter the lipid and protein architecture of the stratum corneum and, therefore, induce skin irritation [4,5]. This effect results from simultaneous mechanisms, including the elevation of pH towards neutral or alkaline values, which increases epidermal permeability and compromises the activity of acid mantle-dependent enzymes, essential for the maturation and repair of the skin barrier [6,7].

Furthermore, surfactants solubilize intercellular lipids and denature structural proteins such as filaggrin, which clinically translates into increased transepidermal water loss (TEWL) and decreased natural moisturizing factor (NMF) [1].

Repeated exposure to these irritants leads to a state of chronic microinflammation, characterized by the release of proinflammatory cytokines and sensitization of neural pathways that mediate pruritus [8].

This disruption of the skin barrier increases susceptibility to irritant contact dermatitis (ICD), especially in vulnerable populations or those with high occupational exposure [9]. In contrast, syndets (synthetic detergents) and moisturizing cleansers, formulated with milder surfactants and a physiological pH, have demonstrated a better ability to preserve the enzymatic activity and cohesion of the subcutaneous layer [6].

The aim of this narrative review is to analyze the pathophysiological mechanisms by which skin cleansing products compromise the barrier function, compare the irritation profile of alkaline soaps versus syndet cleansers, and offer a comprehensive perspective on the clinical implications of these alterations in the prevention and management of irritant dermatitis.

METHODS

For the development of this work, a narrative review was carried out following the guidelines of the SANRA scale (Scale for the Quality Assessment of Narrative Review Articles). The literature search was performed in the PubMed, Cochrane, Ovid, and Scopus databases, applying filters to limit the results to studies conducted in humans and published within the last 10 years.

The closing date for the final search was November 1, 2025.

The search strategy was structured by combining MeSH terms and specific keywords: ("soaps"[MeSH Terms] OR "cleansers" OR "detergents" OR "syndet" OR "skin cleansing agents" OR "skin cleanser" OR "soap") AND ("skin barrier"[MeSH Terms] OR "stratum corneum" OR "skin barrier function" OR "transepidermal water loss" OR "TEWL" OR "irritant contact dermatitis") AND (irritation OR "irritant effect" OR "skin irritation" OR "cutaneous irritation" OR "dermal irritation").

Regarding the selection criteria, human studies published in the last decade that evaluated objective parameters such as pH, transepidermal water loss (TEWL), skin capacitance and hydration, natural moisturizing factor (NMF), or clinical signs of irritation were included. Conversely, studies using animal models, editorial articles that did not contribute original data, and duplicate records were excluded. After this filtering process, 60 articles were identified for analysis.

The data extraction and quality assessment process was carried out by two independent reviewers, who performed the initial screening by title and abstract, followed by a detailed review of the full text; any discrepancies during this phase were resolved by consensus. Information was extracted from each study regarding the design, population characteristics, type of product evaluated (soap, syndet, moisturizing cleanser, or alcohol-based hand sanitizers [ABHR]), skin barrier metrics, and the main clinical message.

Finally, the analysis was conducted using a qualitative approach, the synthesis of which is presented through a thematic integration organized by mechanisms of action, comparisons between products, risk modifiers, and special clinical scenarios.

RESULTS

- **Function of the Skin Barrier**

The skin is the largest organ of the human body and is the body's first line of defense against physical, chemical, and microbiological aggressions. It is composed of three layers: epidermis, dermis, and hypodermis.

This structure provides an effective barrier between the body and the environment to ensure protection against transepidermal water loss (TEWL) due to evaporation (from the inside out), and a mechanism that prevents the penetration of irritants, allergens, and external microorganisms (from the outside in) [10].

There are three main pathways described for the penetration of substances through the skin: intercellular, transcellular, and transappendix. The intercellular pathway involves the passage of substances between the corneocytes of the stratum corneum; the transcellular pathway directly crosses the epidermal cells; the transappendicular pathway utilizes skin appendages, such as sweat glands, sebaceous glands, and hair follicles, with the latter being the most frequent route for water-soluble molecules [11].

The main function of this barrier is found in the stratum corneum of the epidermis, formed by anucleate corneocytes embedded in a lipid matrix composed of ceramides, cholesterol, and fatty acids. An intact barrier maintains water homeostasis, the skin's natural acidic pH (4.5–5.0), and immunological function mediated by cytokines, immune cells (Langerhans cells), and antibodies such as IgA. Its disruption leads to xerosis, irritant or atopic dermatitis, and increased sensitivity to surfactants [12]. The skin barrier function can be compromised by the repeated use of soaps and surfactants, as well as by conditions such as aging, certain topical medications, rosacea, or atopic dermatitis [13,14,15].

The most common causes of surfactant-induced irritant contact dermatitis include sodium lauryl sulfate and benzalkonium chloride [16].

- **Irritation Mechanisms**

Skin irritation from soaps and other cleansers is explained by interrelated phenomena (Figure 1):

1. *Alteration of Skin pH*

Elevating skin pH toward neutral or alkaline values interferes with enzymes dependent on the acid mantle (lipases, ceramidases) and with the proper degradation of filaggrin, causing disorganization of intercellular lipids, increased epidermal permeability, and an increase in TEWL. Considering that the isoelectric point of keratin is around pH 5, it has been shown that surfactants formulated predominantly with anionic compounds cause greater irritation and dryness compared to those with a more neutral pH, due to an alteration of electrostatic interactions [7,17,18].

Koudounas' study demonstrated that a sustained elevation of pH above 6.5 disrupts the lipid matrix of the stratum corneum and reproduces the irritant effect typical of alkaline soaps [6]. Repeated exposure to alkaline cleansers or frequent washing induces a persistent increase in surface pH and a progressive loss of skin homeostasis, compared to syndets with physiological pH, which even reduce pH after repeated use [17,19]. Patients with atopic and extremely dry skin present higher than normal pH values, around 5.5 or higher; it is unclear whether the change in pH is a cause or an effect of the skin condition [12].

Alkalization of the epidermal microenvironment also affects the commensal flora and amplifies the local inflammatory response, predisposing to irritant dermatitis and exacerbating pre-existing dermatoses, such as atopic dermatitis [14].

Taken together, these findings confirm that maintaining the skin's acid mantle is essential to preserving the integrity of the epidermal barrier and preventing barrier dysfunction induced by alkaline pH soaps.

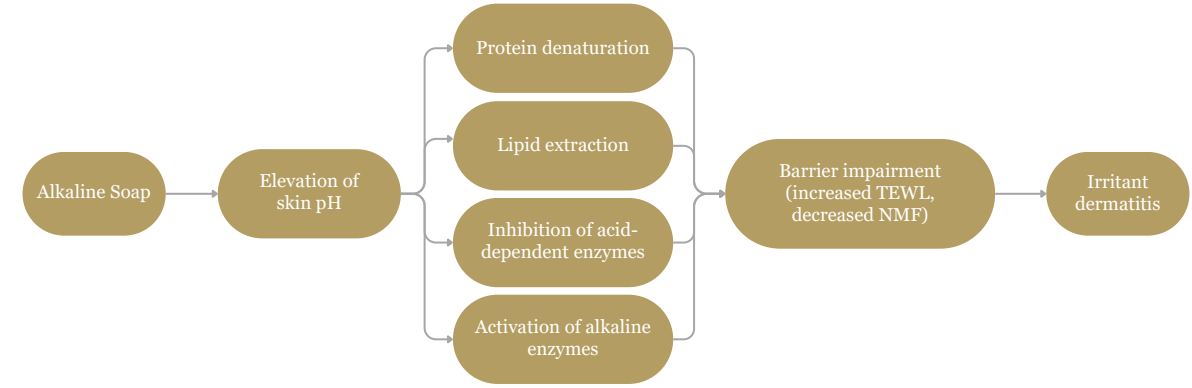


Figure 1. Mechanisms of Irritation by Alkaline Soaps: Pathways of Damage and Convergence to Barrier Impairment. *Original work by the author. TEWL: transepidermal water loss; NMF: natural moisturizing factor*. The diagram illustrates the cascade of pathophysiological events triggered by alkaline soap (pH 8-10). The cleansing agent initiates three main pathways of damage that converge in the impairment of the skin barrier function: 1) Elevation of skin pH, which alters enzymatic homeostasis by inducing the activation of alkaline enzymes and the inhibition of acid-dependent enzymes (essential for lipid synthesis); 2) Denaturation of structural proteins (such as filaggrin); and 3) Extraction of intercellular lipids. The sum of these effects leads to barrier impairment, clinically manifested by increased transepidermal water loss (TEWL) and decreased natural moisturizing factor (NMF), ultimately culminating in irritant dermatitis.

2. *Lipid Removal and Protein Damage*

Anionic surfactants, such as SLS, act as potent lipid-removing and protein-denaturing agents. Their detergent action solubilizes the main lipid components of the stratum corneum (ceramides, free fatty acids, and cholesterol) and alters structural proteins such as filaggrin and involucrin, resulting in a decrease in NMF and an increase in TEWL [16,20,21].

Repeated exposure to SLS has been shown to significantly reduce NMF and impair barrier function in a cumulative irritation model. In atopic skin, the same group showed a more pronounced effect, with a decrease in NMF and a higher TEWL than in healthy skin, demonstrating individual vulnerability to damage. It was confirmed that repeated application of SLS produces an increase in erythema and TEWL even in areas pretreated with protective creams, indicating that topical preventive measures do not always mitigate barrier damage induced by frequently used soaps [4].

The underlying mechanism is related to the extraction of lamellar lipids and the denaturation of corneocyte proteins, which disrupt the cohesion of the stratum corneum and facilitate the penetration of irritants. Anionic surfactants such as SLS and benzalkonium chloride are, therefore, the main culprits behind occupational irritant dermatitis [16].

Furthermore, structural damage is amplified by electrostatic interactions between negatively charged surfactants and positively charged skin proteins, leading to cellular edema, protein denaturation, and loss of barrier integrity [10,16,17].

3. *Keratinocyte Damage, Inflammation, and Microenvironment*

Surface-induced chemical and osmotic stress triggers pro-inflammatory cellular responses in the skin epithelium. Repeated exposure to detergents generates oxidative damage and keratinocyte apoptosis, with the release of cytokines such as IL-1α, IL-8, and TNF-α, which activate an inflammatory cascade responsible for amplifying irritation and barrier dysfunction [22]. The American Contact Dermatitis Society consensus statement noted that these mediators, along with the loss of the skin's natural moisture barrier (NMF) and elevated skin pH, lead to histopathological changes characteristic of irritant dermatitis: spongiosis, dyskeratosis, and persistent chronic microinflammation [3].

Furthermore, the hyperhydrated or alkaline microenvironment modulates the expression of filaggrin and type 2 cytokines (IL-4, IL-13), reproducing an immunological profile similar to that observed in atopic dermatitis. These alterations perpetuate epidermal inflammation and impaired barrier function [14].

4. *Risk factors for irritation*

The extent of skin irritation depends not only on the type of detergent but also on extrinsic (frequency, friction, humidity, occlusion) and intrinsic (atopy, age, previous barrier integrity) conditions (Table 1).

Occlusion and moisture. Prolonged glove use and working in wet conditions enhance the irritant effect of detergents by increasing skin temperature, humidity, and pH. Occlusion increases pH and the penetration of irritants in a dose-response relationship, amplifying barrier damage [23]. It has been observed that the combination of gloves and detergent exposure compromises epidermal integrity even with the use of protective creams, confirming an additive effect between occlusion and irritants [24]. The wet work phenomenon, which refers to repeated exposure to water, surfactants, and friction, causes maceration, lipid depletion, and increased permeability of the stratum corneum. Even in the absence of detergents, prolonged occlusion with gloves reduces barrier function [25].

Friction and repeated trauma. Constant friction or microtrauma promotes loss of corneocyte cohesion and facilitates the penetration of irritants, contributing to the chronicity of dermatitis. This mechanism has been documented in occupations involving mechanical handling or repeated contact with cleaning, cosmetic, or healthcare products [21,26].

Washing frequency and product type. The frequency of exposure is a determining factor; even "mild" soaps or syndets can produce cumulative alterations in TEWL and pH after repeated washings, indicating that irritation depends more on the number of exposures than on the type of product [22]. During the COVID-19 pandemic, excessive washing combined with alcohol-based disinfectants was shown to cause skin dysbiosis, local immune dysfunction, and persistent chronic eczema [2].

Individual predisposition. Genetic and underlying factors, such as atopy or mutations in the filaggrin gene, increase susceptibility to barrier damage and irritation from surfactants [14,21]. Likewise, occupational exposure, such as in health workers, personnel performing cosmetic procedures, and jobs exposed to radiation emitted by the sun, have been described as factors present in the appearance of damage from cleaners [9,21,26–28].

Table 1. Modifiable and non-modifiable factors that increase the risk of soap irritation.

Modifiable Factors	Non-modifiable Factors
• Washing Frequency • Friction • Use of Topical Medications • Contact Time • Water Temperature • Cleanser Formulation • Occlusion (gloves) • "Wet work" • Inadequate Drying • Lack of Post-Wash Emollient Use	• Genetics (atopy, filaggrin gene mutations) • Advanced Age • Previous Occupational Exposure

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- **Comparison of Soaps**

1. *Alkaline Soaps (Saponified, pH 8–10)*

Traditional alkaline soaps, made by saponifying fats with alkalis, have a pH between 8 and 10 and contain anionic surfactants, primarily SLS. These chemical characteristics alter the lipid architecture of the stratum corneum by solubilizing intercellular lipids (ceramides, cholesterol, and free fatty acids) and denaturing structural proteins such as filaggrin and involucrin.

Soaps containing SLS cause a sustained increase in skin pH (from 4.5–5.5 to >7) and an increase in TEWL, which reduces the activity of acid-dependent enzymes such as β-glucocerebrosidase and sphingomyelinase, responsible for lipid regeneration. This disruption promotes epidermal inflammation mediated by IL-1α, IL-8, and TNF-α, clinically manifesting as xerosis, pruritus, erythema, and microfissures, characteristic of irritant contact dermatitis [3].

2. *Syndets (physiological pH, non-ionic and amphoteric surfactants)*

Syndets (synthetic detergents) employ non-ionic or amphoteric surfactants, such as cocamidopropyl betaine or lauryl glucoside, formulated with a physiological pH (4.5–5.5). This range maintains the enzymatic activity of the acid mantle and corneocytic cohesion, reducing TEWL and preserving the skin microbiota [6]. An elevated skin pH has been shown to increase epidermal permeability, demonstrating that syndets, by maintaining physiological acidity, preserve the structural and functional integrity of the stratum corneum.

Clinically, their regular use is associated with better tolerance, less inflammation, and a lower risk of irritant dermatitis in sensitive or frequently washed skin [6].

While liquid syndets with a pH of 5.5 maintain epidermal hydration (TEWL <10 g/m²/h) and prevent irritation, alkaline soaps raised skin pH to 7.1 ± 0.3, reducing lipids and causing transient erythema. These results confirm that syndets are superior in skin compatibility, even in immature or sensitive skin [29].

3. *Moisturizing Cleansers and Combination Formulations*

Moisturizing cleansers incorporate active hydrating agents (urea, glycerin, ceramides) that modulate stratum corneum hydration and compensate for water loss induced by repeated washing. Alcohol-based disinfectants containing urea reduce dryness and redness by restoring the skin's natural moisture barrier (NMF) and improving stratum corneum water retention, without affecting antimicrobial efficacy [30]. Additionally, modified corneosurvep measurement (mCMS) was used to quantify the irritant potential of the cleansers.

Alkaline soaps showed the highest levels of corneocyte damage and protein denaturation, while syndets and moisturizing cleansers maintained cell cohesion and epidermal hydration, exhibiting minimal structural alteration [31].

These findings confirm that formulations with moisturizing agents offer superior protection of the skin barrier, reducing the erythema, scaling, and xerosis observed with prolonged use of conventional soaps.

4. *Antibacterial Soaps and Alternative Agents*

Analysis of antibacterial soaps containing triclosan revealed that their use does not offer greater antimicrobial efficacy than neutral soaps or syndets, but it does cause adverse dermatological and microbiological effects. Triclosan alters the commensal skin microbiota, decreases protective bacterial diversity, and promotes bacterial resistance, in addition to interfering with fatty acid synthesis in keratinocytes, reducing ceramides and increasing TEWL [32].

From a clinical perspective, prolonged use of soaps containing triclosan is associated with chronic irritant

dermatitis and skin dysbiosis, which is why the FDA (Food and Drug Administration) banned its use in general consumer products in 2016. In contrast, alternatives such as chlorhexidine, benzalkonium chloride, and lactic acid have demonstrated adequate antimicrobial efficacy with less irritant potential, depending on the concentration and formulation.

5. *Comparative Evidence and Clinical Applications*

Ex vivo and clinical evidence converges on the fact that alkaline soaps are the most irritating, causing an increase in TEWL, lipid loss, and pH alteration, while syndets with physiological pH and mild surfactants offer greater dermatological compatibility and better maintenance of the skin barrier function [3,6,29].

Moisturizing cleansers with urea or glycerin, on the other hand, provide additional restorative effects by replenishing the skin's natural moisture barrier (NMF) and promoting epidermal regeneration [30,31]. Finally, antibacterial soaps with triclosan pose dermatological and microbiological risks, with no proven clinical benefits compared to syndets [32].

- **Methods for Assessing the Skin Barrier**

Evaluating skin barrier function in response to soaps, detergents, and cleansers requires standardized and reproducible methods that allow for the quantification of structural, biophysical, and clinical alterations induced by these agents. The main parameters used are TEWL, skin pH, stratum corneum hydration by corneometry, erythema by colorimetry, and inflammatory biomarkers obtained by tape stripping (IL-1α, IL-1RA, NMF).

1. *In Vivo Biophysical Assessments*

TEWL is the most widely used quantitative method as an indicator of stratum corneum integrity, reflecting the passive diffusion of water through the epidermis. An increase in TEWL indicates damage or structural disruption of the barrier.

When measuring TEWL, pH, and erythema in in-use forearm models to compare soaps, it was demonstrated that TEWL correlates with the severity of irritant damage [17,19]. The sensitivity of TEWL and pH as measures for assessing the impact of wetwork and repeated friction has been confirmed [4,25]. An automated model (ACiD) was developed that standardizes pressure and washing time, allowing for the quantification of TEWL, hydration (corneometry), erythema (colorimetry), and pH, along with inflammatory markers and NMF (non-facial muscle fibers) using tape stripping [22].

2. *Evaluation of Hydration, pH, and Erythema*

Stratum corneum hydration, measured by corneometry, is a direct marker of surfactant-induced dryness. During the COVID-19 pandemic, the combination of corneometry, TEWL, and pH was established as the gold standard for determining dermal tolerance to cleansers and moisturizers.

Skin pH, measured with pH-metry, is another key indicator. Thus, maintaining a subacid pH (~5.5) preserves enzymatic homeostasis and prevents lipid degradation, while alkaline soaps raise the pH and increase irritation [7,14]. Erythema colorimetry complements these parameters, allowing for the quantification of the inflammatory vascular component associated with barrier damage.

3. *Irritation and Cytotoxicity Tests*

Experimental methods include repeated irritation models such as the Repeated Wash Test (RWT) or the Tandem Repeated Irritation Test (TRIT), which reproduce the cumulative damage from detergent exposure. Their application in the comparative study by Tasar (2021) between alcohols and soaps has confirmed their usefulness in measuring the severity of irritation [33].

In vitro studies have described predictive techniques such as modified corneosurvep measurement (mCMS), protein denaturation (zein or albumin), and reconstructed epidermis models (LSE) with IL-1α/IL-1RA quantification [12]. Cellular cytotoxicity has also been correlated with increases in TEWL and pH, demonstrating a correlation between biochemical damage and functional barrier disruption [18].

4. *Clinical and Occupational Assessments*

In occupational exposure settings, methods combine standardized clinical assessment, exposure interviews, and patch testing. Occupational irritant dermatitis has been documented through detailed occupational history and allergy exclusion via patch testing [21, 28].

Furthermore, glove occlusion has been observed to increase pH and TEWL, contributing to skin barrier impairment even without chemical exposure [23].

In healthcare workers, the combined monitoring of TEWL, pH, hydration, and clinical signs of eczema allows for the detection and quantification of barrier impairment resulting from intensive washing and the use of alcohol-based disinfectants [34,35].

5. *Methodological Integration and Standardization*

The literature agrees that no single parameter fully describes barrier function. The combination of TEWL, corneometry, pH, and erythema constitutes the minimum set of biophysical assessments, while biomarkers obtained by tape stripping (IL-1α, IL-1RA, NMF) and advanced techniques such as CRM (Confocal Raman Microscopy) allow for the detection of subclinical alterations and the evaluation of barrier recovery after the use of cleansers or soaps.

This underscores the need to standardize these methods in surfactant studies, combining instrumental measurements with clinical scales and patch tests to obtain a comprehensive view of skin damage induced by cleansing agents [16,20].

DISCUSSION

The synthesized findings confirm that the damage induced by cleansing products on the skin barrier depends on a multifactorial equation: the nature of the surfactant, the pH of the formulation, the frequency and method of use (contact time, friction), and exposure conditions such as humidity and occlusion. Within this framework, saponified alkaline soaps (predominantly anionic surfactants such as SLS) consistently show a greater increase in TEWL, a sustained elevation of pH, and erythema compared to syndets with physiological pH and non-ionic/amphoteric surfactants, which better preserve corneocyte cohesion and the commensal microbiota [7,12,17,19,20]. These differences are not only biophysical: they correlate with lipid disruption (ceramides/cholesterol/fatty acids), protein denaturation (e.g., filaggrin), and amplification of proinflammatory pathways (IL-1α, TNF-α), the histopathological basis of irritant contact dermatitis [11,16].

The frequency of washing and the "exposure dose," modulated by the context (healthcare, domestic, or occupational), act as risk multipliers. In vivo models and repeated irritation protocols demonstrate that even "mild cleansers" can induce cumulative damage if the number of cycles increases or if friction, high temperature, and aggressive drying coexist [22,24,25]. "Wet work" and occlusion with gloves (due to their impact on humidity, temperature, and pH) enhance the penetration of irritants and impair barrier recovery, even in the absence of added detergent [23,25]. This explains the high incidence of irritant contact dermatitis (ICD) in professions involving frequent handwashing or repeated contact with cleaning solutions [9,21,26,28].

During the COVID-19 pandemic, increased handwashing and the use of alcohol-based hand sanitizers raised the incidence of irritant contact dermatitis [2,32,35]. Although alcohols (n-propanol, isopropanol) have, on average, a lower irritant potential than alkaline soaps, the damage increases when other irritants (detergents) or occlusion by gloves are present [23,33]. Alternating handwashing with soap and alcohol-based hand sanitizers containing emollients, and applying immediate post-wash emollients, reduces the cumulative effect of chemical and mechanical irritation [30,35,36].

Certain populations require specific strategies. In atopic skin or skin with a pre-existing barrier compromise, the "irritation threshold" is lower; Choosing subacid pH syndets and moisturizing/lipid-replenishing formulations reduces relapses and better maintains skin elasticity [7,12,14]. In older adults, the decrease in natural moisture barrier (NMF) and ceramides makes it advisable to avoid alkaline soaps and favor cleansers with moisturizers/ceramides and systematic emollient routines [13,37]. In neonates, mild formulations at pH ~5.5 show better compatibility than traditional soaps [29].

Methodologically, the literature agrees that no single marker fully captures barrier function. The combination of TEWL, pH, corneometry, and erythema constitutes the "minimum common" for evaluation, and complementary techniques (e.g., mCMS/CIM, tape stripping with IL-1α/IL-1Ra, and, when available, confocal Raman spectroscopy) add granularity and sensitivity for lesions [7,10,22,31].

Finally, the review of "antibacterial" soaps qualifies their use: agents such as triclosan do not offer proven clinical superiority over syndets and carry dermatological and dysbiosis risks; their replacement with alternatives with a better safety profile is consistent with regulatory and dermatological recommendations [32].

LIMITATIONS

This review is narrative and, by design, does not incorporate meta-analysis or quantitative effect assessment. Heterogeneity between studies due to product type (soap, syndet, moisturizer, ABHR), anatomical areas, usage pattern, intervention duration, and measurement panel (TEWL, pH, corneometry, erythema, biomarkers) limits direct comparability and inferential strength.

Most trials include short-term exposures in healthy volunteers, which restricts extrapolation to patients with dermatoses (e.g., atopic dermatitis) or chronic occupational exposures. Several studies do not comprehensively control environmental variables (temperature, relative humidity) or standardize friction/contact time, with a risk of residual confounding. Although TEWL and pH are sensitive, they do not fully capture neurocutaneous phenomena (pruritus) or dysbiosis; advanced techniques (mCMS, Raman, tape stripping) are not yet universally used, hindering inter-study triangulation.

Furthermore, the time frame (last 10 years) and the filtering of studies in humans may have excluded valuable prior evidence or relevant mechanistic data. All of this suggests that the results should be interpreted with caution and reinforces the need for longer, standardized comparative trials in vulnerable populations.

DERMATOLOGICAL RECOMMENDATIONS

The accumulated evidence on the impact of cleansing agents on the skin barrier function allows us to establish a set of clinical guidelines focused on preventing irritant contact dermatitis (ICD) and preserving epidermal integrity.

1. **Cleansing Agent Selection**

The choice of cleanser should prioritize minimizing structural and biochemical damage to the stratum corneum (SC).

- ***Priority to physiological pH:*** The use of cleansers with a subacid or physiological pH (4.5–6.0), such as syndets, is recommended, as they prevent the transient alkalization of the skin, which has been shown to increase skin permeability [6].
- ***Avoid harsh surfactants:*** Alkaline soaps and those formulated with strong anionic surfactants should be avoided, especially on sensitive or

compromised skin. SLS is a known irritant that disrupts the skin barrier function by denaturing proteins and extracting epidermal lipids [1,15].

- ***Alternatives in intensive hygiene:*** In healthcare or intensive hygiene settings, hydroalcoholic solutions with emollients cause less disruption of the skin barrier than repeated washing with soap [38]. Furthermore, the use of ethanol-based disinfectants containing urea can reduce soap sensitivity [36].

2. **Modification of Hygiene Practices**

Cleansing technique and frequency should be optimized to reduce mechanical and chemical stress.

- ***Limiting Exposure:*** Repeated exposure to detergents and excessive washing, even with mild products, are correlated with barrier impairment and reduced skin barrier function [39]. Limiting the frequency and duration of contact with water and cleanser is recommended.
- ***Minimizing Friction:*** Mechanical manipulation during washing contributes to barrier damage. Cleaning procedures should be brief and gentle, avoiding unnecessary friction [24].
- ***Occlusion Management:*** In occupational settings, prolonged occlusion with gloves exacerbates detergent-induced damage [9]. Limiting the wear time of impermeable gloves and applying skin protection measures are recommended.

3. **Repair and Protection Strategies**

Active barrier repair is an essential component of prevention.

- ***Post-wash emollience:*** Immediate application of emollients is crucial to counteract the increase in TEWL and restore barrier integrity [37]. Moisturizers containing physiological lipids or NMF are preferred.
- ***Care in the elderly:*** In the geriatric population, it is essential to avoid alkaline soaps and use daily moisturizing cleansers and emollients to maintain epidermal integrity and reduce the risk of fissures and dermatitis [40].
- ***Susceptibility monitoring:*** Individual susceptibility to irritative damage can be assessed using structural biomarkers such as corneocytic nanotexture, allowing identification of individuals at higher risk of developing ICD [41].

CONCLUSIONS

Alkaline soaps with anionic surfactants (e.g., SLS) consistently disrupt the barrier function of the stratum corneum: they raise the pH, extract lipids, and increase TEWL, facilitating inflammation and irritant dermatitis. In contrast, syndets with a physiological pH and moisturizing cleansers show better dermatological compatibility and less impact on pH/TEWL, especially when friction and contact time are reduced. In intensive hygiene settings, alternating washing with ABHR soaps containing emollients, plus immediate emollience after each cleansing, effectively mitigates the cumulative effect of these irritations.

The regimen should be individualized according to skin type, age, skin comorbidities, and exposure (wet work/occlusion).

Looking ahead, pragmatic, long-term trials are needed to compare formulations under real-world conditions (frequency, friction, occlusion, temperature), with standardized outcome panels (TEWL, pH, corneometry, erythema, mCMS/CIM, and biomarkers by tape stripping), inclusion of vulnerable subgroups (atopic dermatitis, older adults, healthcare workers), and clinically significant outcomes (symptoms, flares, adherence, quality of life). In the meantime, the clinical recommendation is clear: favor syndets or moisturizing cleansers at pH 4.5–5.5, minimize friction and contact time, alternate with emollient ABHR cleansers when appropriate, and apply emollients after each cleansing to preserve skin barrier homeostasis.

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Conflict of interest

The authors declare no conflicts of interest.

Ethical considerations

This work did not involve the participation of human subjects or animals, nor the collection of primary data; therefore, it did not require approval from a research ethics committee. All data used came from previously published and publicly accessible secondary sources.

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