REVIEW

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Diaper Dermatitis: Etiology, Manifestations, Prevention, and Management

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Abstract: **Pediatricians and parents report diaper dermatitis (DD) to be one of the most common skin diseases that affects almost every child at some point during the early months and years of life. Diapered skin is exposed to friction and excessive hydration, has a higher pH than nondiapered skin, and is repeatedly soiled with feces that contains enzymes with high irritation potential for the skin. The combination of these factors frequently results in skin damage, leading to visible erythematous lesions that can be irritating and painful to the child. Behavioral changes such as increased crying and agitation and changes in eating and sleeping patterns indicate emotional distress. Appropriate skin care can help to prevent the occurrence of DD and to speed up the healing of affected skin. This includes frequent diaper changes and aeration, gentle cleansing, and the use of a barrier cream. Mild to moderate cases usually resolve after a few days of following this routine, but the use of harsh cleaning products can exacerbate DD.**

MANIFESTATIONS AND ETIOLOGY OF DIAPER DERMATITIS

The predominant form of diaper dermatitis (DD) is irritant contact dermatitis, an inflammation of the skin underneath the diaper manifested as erythematous dermatitis, which notably spares the inguinal fold. DD may worsen if left untreated and may recur until the infant stops wearing diapers (1). Areas affected are the buttocks, the perianal area, the genitals, the inner thighs, and the waistline. In its early stages, DD appears as mild erythema in localized areas with minimal scaling. This may progress to moderate erythema together with the appearance of papules affecting parts of the diapered area. This is frequently associated with visible discomfort or pain (1). Severe cases exhibit papules, pustules, and skin erosion with open wounds. The severity of DD can be clinically evaluated using a scale such as the one shown in Table 1 (2). Visual representation of DD severity is also shown with the range of images in Fig. 1A–D. A common complication of DD is candidiasis, which manifests with erythematous patches, plaques, and scales (3). For a precise diagnosis of DD, other types of dermatoses that can present with erythema and other lesions in the diaper area have

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TABLE 1. *Clinical Evaluation Scale for Characterization of the Severity of DD*

Score	Degree	Definition
0	None	Skin is clear (may have some very slight dryness and/or a single papule but no erythema)
0.5	Slight	Faint to definite pink in a very small area (<2%); may also have a single papule and/or slight dryness
1.0	Mild	Faint to definite pink in a small area (2%–10%) or definite redness in a very small area (<2%) and/or scattered papules and/or slight dryness/scaling
1.5	Mild/moderate	Faint to definite pink in a larger area (10%) or definite redness in a small area (2%–10%) or very intense redness in a very small area (<2%) and/or scattered papules (<10% area) and/or moderate dryness/scaling
2.0	Moderate	Definite redness in a larger area (10%–50%) or very intense redness in a very small area (<2%) and/or single to several areas of papules (10%–50%) with five or fewer pustules, may have slight desquamation or edema
2.5	Moderate/severe	Definite redness in a very large area (>50%) or very intense redness in a small area (2%–10%) without edema and/or larger areas (>50%) of multiple papules and/or pustules; may have moderate desquamation and/or edema
3.0	Severe	Very intense redness in a larger area (>10%) and/or severe desquamation, severe edema, erosion and ulceration; may have large areas of confluent papules or numerous pustules/vesicles

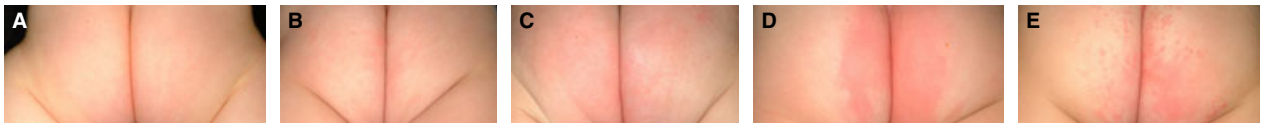

Figure 1. Visual digital images demonstrating the range of severity of DD: (**A**) slight, (**B**) mild, (**C**) moderate, (**D**) moderate to severe, (**E**) severe. Although this scale was used in the studies mentioned in the text, pediatricians have reported even more severe cases.

to be taken into consideration (e.g., intertrigo, seborrheic dermatitis, psoriasis) (3,4). Severe forms of DD require medical attention because they may indicate a serious underlying condition, such as nutritional deficiency, intestinal malabsorption syndrome, congenital abnormalities of the urinary or lower gastrointestinal tract, or toxic reactions.

Reports on the frequency of DD show that the majority of children of both sexes are affected at some point in infancy (5,6). In recent studies, the percentage of children with DD at the time of evaluation ranged from 16% to 70% (5,7). The age of maximum frequency reported in different studies varied, with peaks at approximately 9 to 12 months (8,9) and 12 to 24 months (5). Clinical signs of DD are absent at birth but may appear as early as the first month after birth (10). According to one study, by 4 weeks of age the incidence of DD was 25% (11). The incidence of DD in the general population may be underreported because not all cases are brought to the attention of a physician.

To understand the etiology of skin disorders, including DD, and to choose adequate preventive or treatment measures, it is important to keep in mind that infants have distinct skin physiology features that affect skin barrier function and water handling properties and that these continue to mature during the first years of life (12). These include histologic, biochemical, and functional differences, as well as differences in the microbial population on the skin (13–17). Histologically, infant epidermis is made of smaller keratinocytes than those in adults, microrelief structures are denser, the stratum corneum (SC) and the epidermis are thinner, cell proliferation is greater, and collagen fibers in the dermis are organized differently (17). The water handling properties of infant SC are also markedly different from those of adults. Newborn skin is initially drier but subsequently becomes more hydrated in older infants than the skin of adults. The concentration of the breakdown products of filaggrin proteolysis, collectively known as natural moisturization factor (NMF), is lower. A higher level of transepidermal water loss (TEWL) in some body areas of older infants, different water-holding capacity, and a more alkaline pH are indicative of a developing skin barrier function (15). The composition of the skin microbiome is different, with infant skin being colonized primarily by *Firmicutes*, whereas adult skin is colonized primarily by *Actinobacteria* (13).

Certain biophysical parameters of the diapered area of infants have site-specific differences from nondiapered regions, e.g., skin hydration is higher, as is the skin pH, and epidermal water handling properties are different from those of nondiapered regions (18). Moreover, diapered areas with DD have higher TEWL rates, greater SC hydration, and higher pH than healthy diapered skin (19). The microbial flora colonizing the skin surface in the buttocks area of infants is distinctly different from the microflora in

other skin areas (13). In particular, bacteria that are normally found in the gut are also present in the diaper area, which is explained by the skin coming in frequent contact with feces.

Figure 2 summarizes the factors involved in the pathophysiology of DD. DD occurs when prolonged exposure of the skin to factors that are characteristic of the diapered area, including excessive wetness, friction, high pH, and high enzymatic activity, compromise the epidermal barrier function. The epidermal barrier function resides in the outermost epidermal layer, the SC, which is the region of terminal skin differentiation and is composed of corneocytes (differentiated keratinocytes) and an extracellular lipid matrix. Specialized proteins, the corneodesmosomes, hold these cells, which contain intracellular keratin filaments with large water-holding capacity that are linked to highly cross-linked, insoluble proteins of the cornified envelope (CE) that surrounds the corneocytes (20,21). On its exterior, the CE is linked to the lipid matrix that fills up the intercellular space. The hydrophobic components of the lipid matrix provide the epidermal permeability barrier and protect against excessive water loss and irritant penetration, whereas the corneocytes and the CE provide mechanical strength to the SC. Water-soluble molecules of the NMF that are found inside the corneocytes and are able to absorb water influence the water-holding capacity of the SC (22).

Central in the etiology of DD is excessive hydration of the SC. Skin wetness and DD severity are correlated. Maceration of the SC increases susceptibility to friction between the skin and the diaper fabric, which can cause physical damage to the SC and compromise the epidermal barrier function (23). Furthermore, fecal ureases catalyze the breakdown of urea to ammonia (24), which increases the pH of the skin surface (18,23). This increase in pH contributes to the activity of fecal enzymes, proteases, ureases, and lipases (23), which are highly irritating to the skin (25),

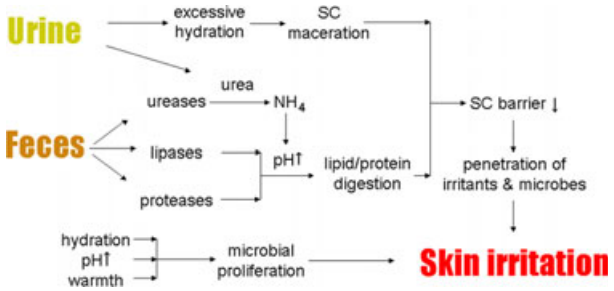

Figure 2. Physical and biochemical factors involved in the pathophysiology of DD.

as the development of severe skin erythema and epidermal barrier breakdown under occlusive exposure of skin to fecal digestive enzymes demonstrates (26). These enzymes increase the permeability to bile salts and other potential irritants (25). In turn, bile salts potentiate the irritant activity of fecal enzymes (25). The reportedly beneficial role of breastfeeding in the prevention of DD (8,9) may be linked to the lower irritation potential of the feces of exclusively breastfed babies, which has a significantly lower pH, lower protease and lipase activity, and lower urease content than that of formula-fed babies (23). The correlation between the number of bowel movements and the frequency of DD supports the damaging role of feces on skin (8). Diarrhea is also a risk factor for DD (5), which may be due to the higher concentration of residual digestive enzymes related to shorter transit times (27). Microorganisms present in the feces of the infant can gain access through the damaged SC, leading to more severe DD with secondary infection. The most frequently isolated microbial species from infected DD areas are *Candida albicans* (28) and *Staphylococcus aureus* (29).

Diaper dermatitis causes emotional stress for infants, as indicated in a study on infant behavior during and after an episode of DD (unpublished data). Parents reported an increase in the frequency of crying as the first symptom of pain together with agitation. Other behavioral indicators of distress, such as facial expressions (eyes squeezed shut, deepening of the nasolabial furrow), were more prevalent, normal eating habits and sleeping patterns were disrupted, and the frequency of urination and defecation diminished. Levels of salivary cortisol, a molecular indicator of stress (30), also increased in some infants during the period with DD.

PREVENTION AND MANAGEMENT

A number of good reviews have been published to address optimal management strategies and treatment algorithms for DD (4,31–33). In this section we will review common practices and present new data on the efficacy of routines relating to DD prevention and treatment.

The key to DD treatment lies in its prevention (33). The most effective routine to prevent DD involves frequent diaper changes to reduce exposure to urine and feces under occlusion. Exposure of the buttocks to air for as long as possible reduces the duration of direct contact of the skin with the wet fabric surface and reduces friction. A fresh diaper should be put on after each urination or defecation. For newborns with

a high wetting frequency, this means a diaper change ideally approximately every 2 hours during the day, whereas for older infants, every 3–4 hours should be sufficient (34).

To help reduce the risk of DD onset or further degradation, if the skin barrier is already compromised, aggressive approaches such as the use of harsh cleansers and scrubbing of the delicate skin should be avoided. Routine bathing of infants should be performed with warm water (37°C–40°C) and a small amount of a mild (nonirritating) cleanser with slightly acidic to neutral pH that respects the acid mantle of the skin (35,36). Traditional soaps with their alkaline pH are too harsh, increasing the skin pH and reducing the epidermal fat content (37,38). By comparison, synthetic cleansers affect the skin pH to a lesser degree and provoke less lipid depletion, less erythema, and a lower TEWL rate (38,39). Clinical studies on routine bathing in infants have shown that neither water alone nor the addition of a mild cleanser causes any physiologically aberrant changes in skin barrier function (TEWL, hydration, pH), skin condition (erythema, dryness, scaling), or microbial colonization (40–42), but water alone may not be sufficient for the removal of feces because of its fat content. Instead, water should be combined with a gentle cleanser (3). Cleansing with appropriately formulated diaper wipes is also an option (35). Evidence from one large assessor-blinded, randomized clinical trial (43) and another double-blind study (44) suggests that appropriately formulated wipes are safe for use on infant skin in the diaper area. Fragrance-free baby wipes impregnated with a mild surfactant system (Johnson's Baby; Johnson & Johnson Ltd., Maidenhead, UK) have been shown to have an effect on skin hydration equivalent to that of cotton wool and water, and they did not adversely affect infant skin (43). Moreover, it has been suggested that alcohol-free and fragrance-free wipes formulated with emollient cleansers and acidic pH protect the barrier function better than washing with a cloth and water alone (45). In cases in which the skin is severely damaged, gently patting dry rather than rubbing is advised.

Application of barrier creams or ointments at each diaper change is recommended as an additional protective or preventive measure and for the treatment of mild to moderate DD. They form a lipid film on the skin surface and protect it from contact with moisture and irritants. Most products contain zinc oxide (ZnO), petrolatum (petroleum jelly), or both as active ingredients. Underneath this protective film, injured skin is allowed to heal and is protected from contact with urine and feces and irritation. During diaper changes, complete removal of the barrier cream or ointment is not necessary to prevent additional injury to the skin; the skin in the diaper area should be gently patted, not rubbed. A beneficial effect may also be associated with reduced skin hydration after application of a barrier cream (46). Other ingredients in use are cod liver oil, aloe barbadensis, dimethicone, and dexpanthenol (47). In some European countries, application of an aqueous solution of 2% eosin after diaper changes is used for DD treatment (48). The lack of barrier cream use and a low frequency of diaper changes are among the factors associated with recurrent DD (5,8).

The efficacy of ZnO- or petrolatum-based creams, pastes, and ointments has been demonstrated in treating diaper rash (27,35,49). In an evaluator-blinded, randomized study, 112 diaper-wearing infants ages 2 to 36 months with mild to moderate DD (diaper rash severity score 1.5 or greater) were randomized to receive one of two ZnO-containing topical products in a petrolatum base (DES13 with 13% ZnO and DES40 with 40% ZnO; Desitin, Johnson & Johnson Consumer Products, Skillman, NJ) at each diaper change and after bathing (2). They were evaluated after 12 and 24 hours of treatment application by a trained evaluator. The data for the 111 evaluable infants showed clinically significant improvement of DD at both time points in all five assessed anatomic areas (buttocks, abdomen, sacrum, inner thighs, and anogenital area). The global mean DD score attributed to the two treatment groups decreased significantly from 1.7 at baseline (both treatment groups) to 1.1 and 1.0, respectively, at 12 hours and to 0.9 (both treatment groups) at 24 hours. In parallel, the parents of the evaluated infants confirmed alleviation of the DD symptoms and reported significant improvement in 79% and 96% of the cases, respectively, at 12 hours and in 84% and 87%, respectively, at 24 hours. ZnO-based topical products have also been shown to provide rapid improvement in the signs of diaper rash, such as erythema, which can be comforting to parents of babies with DD (observations by NKT). In a double-blinded clinical study, 60 diaper-wearing infants (ages 3–36 months) with mild to moderate DD (overall diaper rash severity score 0.5–2.0) were randomized to receive one of two ZnO-containing products (DES13 and DES40) and were asked to apply one application of the test product to the diaper area and to rediaper their infant. After 3 hours ($\pm$1 hour), the test product was gently removed and a trained evaluator and the parent reevaluated the diaper area. The data showed

clinically significant improvement in erythema after one application, evaluated on a visual scale (Fig. 1). The erythema score decreased by 20% ($p < 0.001$), from 1.61 (treatment 1) and 1.93 (treatment 2) at baseline to 1.29 and 1.55, respectively, at 3 hours. The parents of the evaluated infants confirmed the improvement in erythema and reported significant improvement in 64% (treatment 1) and 65% (treatment 2) of cases.

ZnO- or petrolatum-based diapering products exert their protective and preventive properties through the formation of a suitable protective film on the skin surface. In a study conducted on 36 adults, the overnight use of two ZnO-containing topical products (DES13 and DES40) prevented the penetration of a dye into the treated skin (unpublished data). The 36 participants received one test product on one site on one arm and the other test product on one site on the other arm through random assignment, whereas one site on each arm served as an untreated control. The treated areas were immediately covered with mini-diapers for 12 hours overnight. All four areas were then exposed to staining with crystal violet and the extent of staining was compared with that of an unstained area using a colorimetric technique. The results showed marked staining of the untreated areas, whereas the areas treated with either of the two test products completely protected the skin from the applied stain. Similarly, when skin barrier function was measured using in vivo Raman microspectroscopy, skin treatment using a ZnO-containing barrier cream (DES13) blocked externally applied caffeine penetration, unlike the untreated control (49). These results confirm the protective action of ZnO against the penetration of skin irritants. Petrolatum-based products also have the ability to prevent skin maceration and irritant penetration (50). The ability of petrolatum to penetrate deep into the SC (~30 μm), where it increases the lipid content and provides skin occlusion, as indicated by greater SC thickness in adults and infants, probably explains this (51).

Finally, diaper rash formulations have been shown to be well tolerated in the diaper area, with no adverse events reported with their use. This was demonstrated in the infant study mentioned above regarding treatment with two ZnO-containing products for 24 hours (2). Similar tolerability was observed in a double-blind study conducted on 72 diaper-wearing 3- to 24-month-old infants with healthy skin (group 1) or slight (diaper rash severity score of 0.5) to mild to moderate (diaper rash severity score of 1.5) DD (group 2) who were treated using a ZnO-containing product (DES13) under normal use conditions for 4 weeks, with 65 participants completing the study (unpublished data).

Diaper dermatitis can be successfully treated using the measures outlined above. If conventional measures fail for moderate to severe cases, topical corticosteroids and topical antifungal medications may be prescribed for treatment and applied under the barrier cream. Nystatin, clotrimazole, and ketoconazole are frequently prescribed antifungal drugs to control fungal growth when candidal infection is suspected, especially in DD that lasts for longer than a few days, and mild topical corticosteroids such as hydrocortisone are used to reduce inflammation (6). Creams containing mid- and high-potency corticosteroids in combination with antifungal treatment (nystatin–triamcinolone, clotrimazole–betamethasone dipropionate) are among those most frequently prescribed by pediatricians for the treatment of DD in the United States (6), but they are not considered appropriate for mild to moderate DD treatment and should be avoided to prevent possible side effects (52). If a secondary infection involving bacteria is expected, topical antibiotics may be considered (3).

CONCLUSION

In summary, DD is a common skin condition for newborn and young infants. The diaper occlusion creates an environment conducive to skin irritation beginning with damage to the skin barrier caused by components of urine and feces. Supporting the skin barrier with barrier creams coupled with frequent diaper changes is an effective preventive strategy.

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CONFLICT OF INTEREST

The authors are employees of Johnson & Johnson Family of Consumer Companies.

REFERENCES

1. Berg RW. Etiology and pathophysiology of diaper dermatitis. Adv Dermatol 1988;3:75–98.
2. Brown WM, Berg JE, Li Q et al. A clinical study to evaluate the efficacy of two marketed zinc oxide-based diaper rash ointments in children with diaper dermatitis. Clinical Dermatology Conference. Las Vegas, NV, 2006.

3. Shin HT. Diaper dermatitis that does not quit. Dermatol Ther 2005;18:124–135.
4. Boiko S. Treatment of diaper dermatitis. Dermatol Clin 1999;17:235–240.
5. Adalat S, Wall D, Goodyear H. Diaper dermatitis—frequency and contributory factors in hospital attending children. Pediatr Dermatol 2007;24:483–488.
6. Ward DB, Fleischer AB Jr, Feldman SR et al. Characterization of diaper dermatitis in the United States. Arch Pediatr Adolesc Med 2000;154:943–946.
7. Liu N, Wang X, Odio M. Frequency and severity of diaper dermatitis with use of traditional Chinese cloth diapers: observations in 3- to 9-month-old children. Pediatr Dermatol 2011;28:380–386.
8. Jordan WE, Lawson KD, Berg RW et al. Diaper dermatitis: frequency and severity among a general infant population. Pediatr Dermatol 1986;3:198–207.
9. Benjamin L. Clinical correlates with diaper dermatitis. Pediatrician 1987;14(Suppl 1):21–26.
10. Visscher MO, Chatterjee R, Munson KA et al. Development of diaper rash in the newborn. Pediatr Dermatol 2000;17:52–57.
11. Philipp R, Hughes A, Golding J. Getting to the bottom of nappy rash. ALSPAC Survey Team. Avon Longitudinal Study of Pregnancy and Childhood. Br J Gen Pract 1997;47:493–497.
12. Stamatas GN, Nikolovski J, Mack MC et al. Infant skin physiology and development during the first years of life: a review of recent findings based on in vivo studies. Int J Cosmet Sci 2011;33:17–24.
13. Capone KA, Dowd SE, Stamatas GN et al. Diversity of the human skin microbiome early in life. J Invest Dermatol 2011;131:2026–2032.
14. Giusti F, Martella A, Bertoni L et al. Skin barrier, hydration, and pH of the skin of infants under 2 years of age. Pediatr Dermatol 2001;18:93–96.
15. Nikolovski J, Stamatas GN, Kollias N et al. Barrier function and water-holding and transport properties of infant stratum corneum are different from adult and continue to develop through the first year of life. J Invest Dermatol 2008;128:1728–1736.
16. Saijo S, Tagami H. Dry skin of newborn infants: functional analysis of the stratum corneum. Pediatr Dermatol 1991;8:155–159.
17. Stamatas GN, Nikolovski J, Luedtke MA et al. Infant skin microstructure assessed in vivo differs from adult skin in organization and at the cellular level. Pediatr Dermatol 2010;27:125–131.
18. Visscher MO, Chatterjee R, Munson KA et al. Changes in diapered and nondiapered infant skin over the first month of life. Pediatr Dermatol 2000;17:45–51.
19. Stamatas GN, Zerweck C, Grove G et al. Documentation of impaired epidermal barrier in mild and moderate diaper dermatitis in vivo using noninvasive methods. Pediatr Dermatol 2011;28:99–107.
20. Elias PM, Menon GK. Structural and lipid biochemical correlates of the epidermal permeability barrier. Adv Lipid Res 1991;24:1–26.
21. Madison KC. Barrier function of the skin: "la raison d'etre" of the epidermis. J Invest Dermatol 2003; 121:231–241.
22. Rawlings AV, Harding CR. Moisturization and skin barrier function. Dermatol Ther 2004;17(Suppl 1):43–48.
23. Berg RW, Buckingham KW, Stewart RL. Etiologic factors in diaper dermatitis: the role of urine. Pediatr Dermatol 1986;3:102–106.
24. Cooke JV. The etiology and treatment of ammonia dermatitis of the gluteal region of infants. Am J Dis Child 1921;22:481–492.
25. Buckingham KW, Berg RW. Etiologic factors in diaper dermatitis: the role of feces. Pediatr Dermatol 1986;3:107–112.
26. Andersen PH, Bucher AP, Saeed I et al. Faecal enzymes: in vivo human skin irritation. Contact Dermatitis 1994;30:152–158.
27. Atherton DJ. The aetiology and management of irritant diaper dermatitis. J Eur Acad Dermatol Venereol 2001;15(Suppl 1):1–4.
28. Ferrazzini G, Kaiser RR, Hirsig Cheng SK et al. Microbiological aspects of diaper dermatitis. Dermatology 2003;206:136–141.
29. Brook I. Microbiology of secondarily infected diaper dermatitis. Int J Dermatol 1992;31:700–702.
30. Schmidt NA. Salivary cortisol testing in children. Issues Compr Pediatr Nurs 1998;20:183–190.
31. Boiko S. Diapers and diaper rashes. Dermatol Nurs 1997;9:33–39.
32. Friedlander SF, Eichenfield LF, Leyden J et al. Diaper dermatitis: appropriate evaluation and optimal management strategies. Contemp Pediatr 2009;April(Suppl).
33. Heimall LM, Storey B, Stellar JJ et al. Beginning at the bottom: evidence-based care of diaper dermatitis. MCN Am J Matern Child Nurs 2012;37:10–16.
34. Wolf R, Wolf D, Tuzun B et al. Diaper dermatitis. Clin Dermatol 2000;18:657–660.
35. Lund CH, Kuller J, Raines DA et al. Neonatal skin care—evidence-based clinical practice guideline, 2nd ed. Washington, DC: Association of Women's Health, Obstetric and Neonatal Nurses and the National Association of Neonatal Nurses, 2007.
36. Blume-Peytavi U, Cork MJ, Faergemann J et al. Bathing and cleansing in newborns from day 1 to first year of life: recommendations from a European round table meeting. J Eur Acad Dermatol Venereol 2009;23:751–759.
37. Gelmetti C. Skin cleansing in children. J Eur Acad Dermatol Venereol 2001;15(Suppl 1):12–15.
38. Gfatter R, Hackl P, Braun F. Effects of soap and detergents on skin surface pH, stratum corneum hydration and fat content in infants. Dermatology 1997;195:258–262.
39. Strube DD, Koontz SW, Murahata RI et al. The flex wash test: a method for evaluating the mildness of personal washing products. J Soc Cosmet Chem 1989;40:297–306.
40. Dizon MV, Galzote C, Estanislao R et al. Tolerance of baby cleansers in infants: a randomized controlled trial. Indian Pediatr 2010;47:959–963.
41. Garcia Bartels N, Scheufele R, Prosch F et al. Effect of standardized skin care regimens on neonatal skin barrier function in different body areas. Pediatr Dermatol 2010;27:1–8.
42. Lavender T, Bedwell C, O'Brien E et al. Infant skin-cleansing product versus water: a pilot randomized, assessor-blinded controlled trial. BMC Pediatr 2011; 11:35.

43. Lavender T, Furber C, Campbell M et al. Effect on skin hydration of using baby wipes to clean the napkin area of newborn babies: assessor-blinded randomised controlled equivalence trial. BMC Pediatr 2012;12:59.
44. Ehretsmann C, Schaefer P, Adam R. Cutaneous tolerance of baby wipes by infants with atopic dermatitis, and comparison of the mildness of baby wipe and water in infant skin. J Eur Acad Dermatol Venereol 2001;15 (Suppl 1):16–21.
45. Visscher M, Odio M, Taylor T et al. Skin care in the NICU patient: effects of wipes versus cloth and water on stratum corneum integrity. Neonatology 2009;96: 226–234.
46. Xhauflaire-Uhoda E, Henry F, Pierard-Franchimont C et al. Electrometric assessment of the effect of a zinc oxide paste in diaper dermatitis. Int J Cosmet Sci 2009;31:369–374.
47. Adam R. Skin care of the diaper area. Pediatr Dermatol 2008;25:427–433.
48. Arad A, Mimouni D, Ben-Amitai D et al. Efficacy of topical application of eosin compared with zinc oxide paste and corticosteroid cream for diaper dermatitis. Dermatology 1999;199:319–322.
49. Stamatas GN, Boireau-Adamezy E. Development of a non-invasive optical method for assessment of skin barrier to external penetration. In: Biomedical Optics and 3-D Imaging. OSA Technical Digest, paper JM3A.42. Miami, FL: Optical Society of America, 2012.
50. Hoggarth A, Waring M, Alexander J et al. A controlled, three-part trial to investigate the barrier function and skin hydration properties of six skin protectants. Ostomy Wound Manage 2005;51:30–42.
51. Stamatas GN, de Sterke J, Hauser M et al. Lipid uptake and skin occlusion following topical application of oils on adult and infant skin. J Dermatol Sci 2008;50: 135–142.
52. Railan D, Wilson JK, Feldman SR et al. Pediatricians who prescribe clotrimazole-betamethasone dipropionate (Lotrisone) often utilize it in inappropriate settings regardless of their knowledge of the drug's potency. Dermatol Online J 2002;8:3.