Environmental and behavioral mitigation strategies for patients with atopic dermatitis

Nadia Shobnam, MD, Grace Ratley, MSPH, Jordan Zeldin, BS, Manoj Yadav, PhD, and Ian A. Myles, MD, MPH

Objective: Herein, we aimed to summarize the evidence-base for these interventions with a focus on the role of specific chemicals in driving AD.

Methods: A narrative review of nonprescription mitigation strategies in AD was conducted.

Results: We identified avoidance strategies for the various routes of exposure such as air pollution, water contamination, or inclusion in home goods, skin care products, and cleansers. Evidence for and against dietary modification and emollient use as primary prevention were also elucidated. To remember these interventions we propose a mnemonic, HELPSS AD: Home decor, Emollients, Laundering, Probiotics, Soaks, Social support, Air quality, and Diet.

Limitations: Each of these categories presents nuanced molecular differences that must be considered. For example, probiotic responses vary by the specific species while home products and pollution must be analyzed by the specific toxins.

Conclusion: Although the interventions discussed lack the level of evidence required for inclusion into formal guidelines, awareness of these approaches may offer aid to, and build trust with, patients and caregivers. (JAAD Int 2024;17:181-91.)

Key words: atopic dermatitis; behavior change; eczema; environment; mitigation strategies; patient care.

INTRODUCTION

Atopic dermatitis (AD) is a chronic, inflammatory skin disease with significant psychological and immunologic burdens.[1,2] AD is characterized by barrier dysfunction and immune mediated inflammation.[2] Although numerous medications have been recently approved,[3,4] there are many aspects of disease management that are not well understood. For example, there is uncertainty surrounding the optimal skin care regimen for soaps and moisturizers.[5] Similarly, patients need to consider their disease when planning their diet, choice of clothing, and cleansers used in the home.[6] Although current treatment guidelines detail both prescription and nonprescription medications,[7,8] these guidelines less often comment on patient and parents' preference for nonpharmacologic means for primary or secondary prevention of flares.[9] The emerging understanding of AD as having a microbiome-mediated, lipid-centric mechanism may help clarify the murky field of nonprescription mitigation strategies.[10-17] This review aims to summarize the current evidence for nonprescription and environmental mitigations that may allow patients, caregivers, and providers to improve outcomes in AD.

From the Epithelial Therapeutics Unit, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Maryland.

Authors Ratley and Zeldin contributed equally to this article.

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Correspondence to: Ian A. Myles, MD, MPH, Epithelial Therapeutics Unit, National Institute of Allergy and Infectious Diseases, National Institutes of Health, 9000 Rockville Pike, Building 10, Room 11N228, Bethesda, MD 20895. E-mail: mylesi@niaid.nih.gov, X: @lcdriammdmph.

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Bathing practices

Impaired skin barrier and subsequent water loss across the skin surface contributes to the pathophysiology of AD.[18] Thus, a mainstay of therapy has been the "soak and seal" method that incorporates daily soaks of the entire body for 10 to 15 minutes followed by immediate application of an emollient. There is some evidence to suggest daily bathing is more beneficial than bathing several times a week[19]; however, the literature lacks a robust body of evidence to suggest the optimal temperature, timing (morning or evening), or duration of bathing.[20] Whatever bathing regimen is used, water softeners do not provide benefit in AD.[21,22] Although for most patients bleach baths are no more efficacious than water baths,[23] there may be a small yet debated, benefit in moderate to severe AD when used as an adjunct therapy.[7]

CAPSULE SUMMARY

- Environmental exposures have long been known to be triggers of atopic dermatitis flares but are increasingly recognized as causal in atopic dermatitis etiology.
- Practicing dermatologists can inform their patients about the evidence-based methods for environmental mitigations which can improve outcomes in atopic dermatitis.

Considerations when selecting soaps and cleaners

Navigating the numerous options for topical products is a source of frustration for patients and caregivers.[24] A majority of patients report avoidance of topical products containing fragrances, while many avoid parabens, benzyl alcohol, and formaldehyde.[6] In *in vitro* assays, parabens, benzyl alcohol, formaldehyde, as well as lanolin demonstrated greater inhibition of ceramide-producing commensal bacterial growth than isolates of *Staphylococcus aureus*.[5] Thus, products containing these ingredients would be expected to exacerbate, if not induce, the type of skin dysbiosis associated with AD.[25] Additional risk may come from topical products which contain food allergens, such as tree nuts, soy, or milk.[26] However, topical products are rarely 1 ingredient and therefore complexity arises in recommending optimal regimens. For example, fragrances differed in their propensity to induced dysbiosis[5] which is broadly defined as a microbial "imbalance", and in AD often reflects an reduced presence of Gram-negative bacteria and coagulase negative *Staphylococcus* with an overabundance of *S. aureus*.[25]

Cleansers containing sodium lauryl sulfate (SLS) may present a risk for barrier damage. SLS is found in numerous products including hand soaps, shampoos, laundry detergents, dishwashing detergents, home cleaners, and even toothpaste.[14,27] Similar barrier-eroding properties are also found with the common rinse-aid ingredient alcohol ethoxylate.[28] Unfortunately, what little data is available suggests that while "green" labeled products release a lower amount of total volatile chemicals compared to conventional products, such labeling does not provide assurance that any given product will be free of AD-associated chemicals.[29] Furthermore, all soaps possess some degree of lipid disrupting properties[30,31] and will definitionally present a risk to normal skin physiology. In addition to barrier disruptors, soaps and cleansers may contain contact allergens.

Considerations when selecting emollients

Recent American Academy of Allergy, Asthma, and Immunology practice parameters recommend the avoidance of prescription moisturizer in favor of a "bland fragrance-free moisturizer".[7] Thicker emollients (gels and ointments) may be more efficacious while lighter emollients (creams and lotions) tend to have better compliance and skin absorption.[32] While some recent studies have reported ceramide-dominant emollients as superior to other formulations,[33,34] it remains controversial to recommend one emollient over another.[32] For example, while our lab has identified specific topical products which do not induce AD-associated dysbiosis (Table I), only some of these products contain ceramides, and some contain contact allergens that risk inducing flares.

As with soaps and cleansers, emollients containing parabens, formaldehyde, lanolin, or SLS would present a predicted risk of encouraging dysbiosis and/or barrier dysfunction. Therefore, while the current evidence is insufficient to recommend gravitation towards any specific emollient, avoidance of select ingredients may be advisable even if untested. However, providers should acknowledge the increased costs that emollients present to patients and counsel patients to trial an emollient that is affordable to determine tolerability.

While several studies are still ongoing, published reports on emollients as primary prevention in AD are mixed.[36,37] Overall, the data do not support recommending preventative use of emollients to all patients, but there may be benefit for high-risk children, those with parental atopy.[38] In addition to a lack of overall efficacy, a recent Cochrane review identified a

Abbreviations used:

AD: atopic dermatitis
SLS: sodium lauryl sulfate

potential increased risk for subsequent food allergy and skin infections with certain emollient exposures depending on the ingredients.[39] However, there are substantial differences in the molecular contents of emollients and, even for the same formulation, the risk of infectious contamination is higher when the product is stored in tubs versus pump bottles[40] (unless a sterile device is used to scoop the emollient from the tubs). Therefore, a meta-analysis which ignores these nuances may lack validity.

Air quality as a trigger for AD

Rates of AD in the US and UK have increased 3-6-fold since approximately 1970,[41-44] and as much as 30-fold compared to developing nations.[45,46] Additional evidence linking air pollution to AD includes the significant impact of migration to or from highly industrial countries and further influence of urbanicity within a given country.[45-48] Until recently, the epidemiologic links between AD and the environment felt disconnected. For example, living near a highway or major road is associated with an increased risk for AD[49] especially for the children of mothers who live near the roadway while pregnant,[50] reflecting a connection with the recently reviewed traffic-related air pollution.[51,52] Yet risks also include wildfires,[53,54] indoor air pollution,[55] humidity and temperature fluctuations,[43,56] as well as active and passive exposure to cigarettes[57,58] or e-cigarettes.[59] Additional risk for children under 2 years of age may come from home remodeling practices such as wall papering, painting, using floor sealant, or purchasing new furniture.[15]

Recent discoveries may have offered a throughline for the links between AD and pollution sources that range from natural disasters to industrial waste to home goods. Contrasting databases on AD visits with industrial release data from the US Environmental Protection Agency identified isocyanate-containing compounds (such as toluene diisocyanate) and xylene as having the strongest association with AD risk for adults and children.[15,60] These chemicals are all within the benzene, toluene, ethylbenzene, and xylene (BTEX) family of toxins and have been implicated in various ADs.[61] Mechanistic studies revealed that these toxics harm health-associated, ceramide-producing commensal bacteria,[15,60] activate host itch- and thermo-receptors,[62-65] and directly induces AD-like dermatitis in mice.[17,66]

These chemicals became part of car exhaust circa 1970 as an unintended consequence of catalytic converters and as a replacement for lead as the primary antiknock agent in gas.[67] Cigarette smoke also contains both isocyanate and xylene.[68] Meanwhile, the most common natural source of isocyanate or xylene is wildfire smoke.[69,70] Other in-home exposures also began to rise with modern manufacturing of toluene diisocyanate and xylene for use in paints, wallpaper glue, polyurethane based sealants, and the types of nonlatex foam used in new furniture.[61,71] Thus, isocyanate and xylene are found in the primary exposures linked to AD; erode the production of protective ceramides by commensal microbes; directly activate itch and rash through temperature-sensitive host receptors; and became increasingly common exposures just prior to the modern rise in AD rates.

Patients should thus be advised against home remodeling if children under 2 are in the house and those with active AD may need to avoid exposure to these products. In addition to inquiring about changes in detergents or topical skin care regimens, providers evaluating patients with recent worsening of AD control should ask about a history of moving to new residence, any home remodeling work, and if they purchased any new foam-containing furniture. However, mitigating nearby highways, factories, or wildfires may be impossible. As is currently the practice for those with severe asthma, patients with severe AD may consider staying indoors during low-quality air days. Yet, given that outdoor pollutants may concentrate and linger indoors,[72-75] ventilation and cleaning contaminated surfaces is advised.[75]

One retrospective study suggested a possible protective effect of air purifiers against AD.[56] The only interventional air purification study found an 18.5% improvement in itch, rash, and sleep disturbance, as well as an 18.7% improvement in transepidermal water loss after 24 weeks of use.[56] Of note, the off-the-shelf filter assessed was not specifically assessed against any pollutant linked to AD. Thus, future work into optimizing the filtration type may improve clinical response. In the interim, we suggest patients consider a filter rated against volatile organic compounds that does not produce ozone during its operation. These air purifiers are often expensive and thus any such suggestion must consider the patients' financial situation and possible insurance coverage.[76] While of untested benefit in AD, studies indicate that specific house plants can metabolize AD-relevant toxins like the BTEX compounds and formaldehyde[77-80] (Table I).

Table I. Summary of recommendations for environmental mitigations for atopic dermatitis

Recommendation(s)
Home décor (In-home products that may contribute to AD)
• Avoidance of in-home products with isocyanates, xylene, or other BTEX compounds.
• Consider pushing for requiring products containing AD relevant chemicals be marked with a warning label for consumers.
• After remodeling of the home, wallpaper changes, refurnishing, and resurfacing, ventilate the home and dust/clean surfaces to reduce VOCs.
• Preference for cotton, silk, or natural-fiber linen and clothing, but avoidance of wool, nylon, spandex, and polyester. Consider individual experimentation.
Emollients (and other topical skin care products)
• Consider emollients with lipid base, preferencing ceramides.
• No formal recommendation for emollients as primary prevention for all patients currently.
• High risk infants (parental atopy) may benefit from twice daily emollients for the first 8 wk of life.
• Preference for topical products with no in vitro evidence of dysbiosis.
• Consider phone apps, such as Yuka, EWG's Healthy Living, SkinSafe, or the CAMP website from the American Contact Dermatitis Society to screen products for concerning ingredients.
• Advocate for warning labels for products containing ingredients of concern for AD, akin to established labels for food allergy.
• Preference for topical products with no in vitro evidence of dysbiosis, such as[5,15]: Altopalm MLE Cream, Aveeno Colloidal oatmeal, CeraVe Daily Moisturizing lotion, Cetaphil Moisturizing cream, Eczema Honey lotion, Eucerin Original Healing lotion, Rareglo Skin & Hair lotion, Simple Sugar Coconut Body lotion, Vanicream lotion, CeraVe Healing ointment, Vaseline Advanced ointment, Dr Bronner's coconut oil, Kiyamel Eczema Relief Baby Oil, Nutiva Liquid coconut oil, Spectrum Culinary sunflower oil, Neutrogena SheerZinc sunblock, Sun Bum sunblock spray.
• Screen all topical products for relevant contact allergens.
• When using topical products stored in tubs, use a fresh tongue blade to scoop contents rather than scooping out by hand.
• Avoid topical skin care products containing parabens, formaldehyde, lanolin, and/or SLS. SLS may also be indicated on the label as: sodium monolauryl sulfate, sodium dodecyl sulfate, sodium monolauryl sulfate, sodium dodecane sulfate, lauryl alcohol, or hydrogen sulfate.
• Consider avoiding products found to have dysbiotic potential including[5,15]: Aveeno Eczema Therapy, Curel Hydrotherapy, Eucerin Eczema Relief, Lubriderm Moisturizing lotion, Aquaphor Healing ointment, Spectrum Culinary canola oil, Spectrum Culinary grapeseed oil, Spectrum Culinary olive oil, Banana Boat Ultra Sport sunblock spray, Neutrogena Beach Defense sunblock spray, Coppertone Sport sunblock spray.
Laundering (cleaning products for home, linens, and body)
• Avoid cleaning products containing parabens, formaldehyde, lanolin, alcohol ethoxylate, and/or SLS.
• Advocate for warning labels for products containing ingredients of concern for AD, akin to established labels for food allergy.
Probiotics
• Only take probiotics labeled with genus, species, and strain identification.
• For treatment, preference probiotics containing L. casei (CECT9104), B. longum ES1, B. lactis BPL1, and L. rhamnosus CNCM I-4036.
• For prevention, preference probiotics containing B. longum BL999 combined with L. paracasei ST11.
• Topical probiotic consideration with Roseomonas mucosa RSM2015; under study in clinical trial NCT06096857.
• Advise patients that probiotic therapy takes at least 8-12 wk to become evident.
Soaks (bathing practices)
• Soaking for 10-15 min prior to emollient application may be optimal.
• No support for use of water softeners.
Social support
• Seek out counseling and support networks for patients and caregivers.
• Providers and patients should familiarize themselves with local support group resources.
• NEA offers toll free counseling (1-800-818-7546); limited to USA business hours.
• Discuss enrollment in Support For Eczema caregivers (www.gper.org/caregiver).

Continued

Table I. Cont'd

Recommendation(s)
Air quality
• Consider staying indoors during poor air quality days and/or avoid prolonged outdoor exposures near wildfire smoke or highways.
• Ensure proper ventilation indoors: open windows on opposing sides of the building when possible or set up inlet and outflow fans.
• Evaluate the specific chemicals released from factories in your zip code using the EPA Risk Assessment resource (https://www.epa.gov/trinationalanalysis/where-you-live).[35]
• Ventilate the home and dust/clean surfaces. If feasible, avoid living near busy roads or regions with a high burden of wildfires.
• Consider the use of a VOC rated air purifier that does not produce ozone.
• Inquire with insurance providers if such purifier may be covered under insurance.
• Consider use of house plants shown to reduce BTEX compounds and formaldehyde: Areca palm, Spider plant, Golden pothos, Gerbera daisy, Boston fern, Weeping fig, Peace lily, Chrysanthemum, Snake plant, and/or Aloe vera.
Diet
• No specific dietary plan or avoidance recommended.
• Do not restrict maternal dietary exposures during pregnancy or lactation.
• Consider supplementation of vitamin D at 1600 IU/d.
• Consider increasing consumption of fruit, vegetable, and naturally fermented foods (such as kefir, yogurt, fermented rice flour, fermented milk, kimchi).
• Advocate for interventional studies for dietary modification as AD treatment.
• Consider evaluation for systemic contact dermatitis (including nickel, trace metals, and balsam of Peru).

AD, Atopic dermatitis; *BTEX*, benzene, toluene, ethylbenzene, and xylene; *CAMP*, Contact Allergen Management Program; *EPA*, Environmental Protection Agency; *SLS*, sodium lauryl sulfate; *VOC*, volatile organic compound.

Considerations when selecting linens and clothing fabric

Current AD treatment guidelines advise patients to avoid coarse wool fiber clothing.[8] However, exacerbations of AD are also linked to nylon, spandex, and polyester.[81,82] Fabric tightness and thickness may induce sweating and thereby impact AD and the skin microbiome.[83,84] However, interventions aimed at minimizing sweat did not demonstrate clinical benefit.[85] The recent insights into air pollutants[15,60] may offer mechanistic insights into these reactions to synthetic fabrics given that spandex is made from diisocyanates, polyester derived from xylene, and nylon production requires benzene.[86] Consistent with direct modulation of the skin microbiome by the chemicals in these fabrics, a recent report demonstrated that the synthetic fabrics facilitate the growth of *S. aureus* while corrupting the metabolism of health-associated commensals.[60]

While *S. aureus* cannot survive for more than a few days on cotton, it can persist on both nylon and a spandex/polyester blend.[60] Meanwhile, commensal microbes could persist on all fabric tested, but synthetic fabrics disrupted the phospholipid metabolism needed for ceramide and sphingolipid production.[60] The balance of organism growth on bamboo was potentially beneficial, but there are fewer regulations as to the processing or purity of bamboo; bamboo may also be naturally contaminated with polycyclic aromatic hydrocarbons and heavy metals.[87,88] Fabrics infused with antimicrobial agents may have therapeutic utility, but they carry a high-cost burden and risks for contact dermatitis and antimicrobial resistance.[89-91]

Dietary considerations

Numerous meta-analyses failed to find any consistent association between AD risk and material avoidance of antigens during either pregnancy or lactation nor does the data inform formula selection.[92] However, a consistent protective benefit against AD development is evident for the consumption of fermented foods during pregnancy, such as yogurt or kimchi.[93-97] Similarly, higher consumption of fruits, vegetables, fish, and vitamin D rich foods was associated with a decreased risk of all allergic diseases.[92,95] Although the data on supplementation in AD is less conclusive[98] than for dietary intake, the data remain positive after meta-analysis only for vitamin D at doses over 1600 IU/day.[99,100] In contrast, consumption of processed foods, emulsifiers, refined oils, and nuts increase AD risk.[92,101] Additional harm may come from food contaminated with inducers of contact dermatitis such as nickel, other trace metals, or balsam of Peru.[102,103] Screening patients for systemic contact dermatitis and/or a trial on a low nickel diet may be beneficial.[103,104]

The prior recommendations of food avoidance until 6 months have been rescinded and the current consensus is that dietary elimination confers minimal benefit while risking the induction of immunoglobulin E-mediated anaphylactic reactions.[92,105]

While the literature on the potential for dietary prevention of AD is growing, relatively less is known about the potential for dietary modification as treatment. While food avoidance is officially discouraged, one recent report found that 95.1% of patients reported clinical improvement associated with dietary changes.[106] Diet and natural products were also among the most frequently discussed topics in online support forums for AD.[24] While most interventional dietary studies are small and without control groups, trials of naturally fermented foods (kefir, yogurt, rice flour, milk, and kimchi) have been reported to improve AD symptoms.[107] Overall, providers should be mindful of their patients' interest in dietary modifications and be careful not to conflate the evidence against food avoidance with the conclusion that diet does not play a role in AD.

Considering probiotics

Similar to emollients, probiotics are often erroneously grouped together despite wide variations in the ingredients and mechanisms of actions. This categorical error complicates the interpretation of meta-analyses which collate studies with interventions that vary in their biochemistry.[108] While species from the *Lactobacillus* and *Bifidobacteria* genre are most often linked to improvement in AD, these bacteria are only related at the kingdom level of taxonomy; conflating them would therefore be analogous to meta-analyzing all vertebrate animals. Thus, attention should be paid to how species- or stain-level differences might impact efficacy or safety.[109] For example, combination of *L. casei* (CECT9104), *B. longum* ES1, *B. lactis* BPL1, and *L. rhamnosus* CNCM I-4036 may be the most effective probiotic regimen for AD treatment while the more popular *L. rhamnosus* GG fails to offer clinical benefit.[110] Meanwhile, *B. longum* BL999 combined with *L. paracasei* ST11 appears to be the most efficacious in AD prevention.[110] A provocative, albeit less practical, approach of stool transplant therapy for AD was recently reported.[111] Topical probiotics, such as *Roseomonas mucosa RSM2015*,[15,112-117] are now available over the counter[118] while being further studied in clinical trials.

Providers should be aware of how probiotic strains are indicated and advise their patients to research any considered product by searching for the stain-level identifier(s). Patients should also be advised that clinical improvements are often not noticeable until at least 8-12 weeks of use. Furthermore, most probiotic studies have been performed only in adults and thus lack guidance on pediatric use. Another caveat is that what little data is available suggests that use of oral probiotics without consumption of a healthier diet may not yield sustained improvements in health.[119,120]

Social and psychological support

Managing AD requires a significant amount of time and money.[7] In addition to atopic consequences (asthma, rhinitis, and food allergy), patients with AD must also handle an increased risk of attention deficit hyperactivity disorder, anxiety, depression, social isolation, and no less than a 1.5-fold increased risk for suicide.[121-123] The financial, social, and physical burdens of the disease lead to reduced quality of life for patients and caregivers.[7,123,124] Some of the psychological burdens are secondary to the disrupting influence of itch and sleep loss; however, the inflammatory cytokines and lipid mediators associated with AD each have direct effects on mood and behavior.[125,126] In addition, evidence of bidirectionality of the stress-skin axis indicates that mental wellbeing can directly impact AD symptoms.[127] Therefore, providers should not assume that resolution of AD-related skin symptoms indicate a resolution of AD related psychological taxation. Various groups offer supportive communities and networks for patients and caregivers, including more formal counseling for patients (Table I). Overall, patients and providers should familiarize themselves with local resources and routine screening for behavioral health is recommended for all AD patients and their families.[7,127]

DISCUSSION

Overall, there are numerous nonpharmacological strategies for improving AD outcomes for patients (Table I; Supplementary Fig 1, available via Mendeley at https://data.mendeley.com/datasets/738367jk4s/1). While this advice has some evidence-base, many of these mitigations still lack the level of evidence one would expect for formal treatment guidelines (Table II). For example, substantial knowledge gaps persist for whether specific topical products (representing a mixture of several chemicals) have similar barrier-eroding or dysbiotic properties as individual ingredients. For now, smartphone apps are available, such as Yuka,[128] EWG's Healthy Living,[129] or SkinSafe[130] which scan barcodes and report to US and Canadian consumers whether the product contains potentially harmful chemical ingredients. Notably however, these apps use internal scoring criteria which may not

Table II. Knowledge gaps in the environmental mitigation strategies

Knowledge gaps
Home décor (In-home products that may contribute to AD)
• No current RCT for avoidance of these chemicals.
• Is there a safe exposure level for these chemicals?
• Do all products containing these ingredients have the same harmful properties as the chemicals in isolation?
• No RCT to establish impact of fabric choice on AD.
Emollients (and other topical skin care products)
• Can excipients counter the benefits of tri-lipid based products?
• Does the efficacy of primary prevention differ by emollient formulation (base and excipients)?
• Are there subgroups for which primary prevention is efficacious?
• The number of topical products tested for dysbiotic potential is only a fraction of the total number of commercially available formulations.
• Is there a safe concentration for the chemical additives?
• Do all products containing these ingredients have the same harmful properties as the chemicals in isolation?
Laundering (cleaning products for home, linens, and body)
• Is there a safe concentration for the chemical additives?
• Do all products containing these ingredients have the same harmful properties as the chemicals in isolation?
Probiotics
• Are there manufacturing and/or batch effects that impact efficacy of a given strain?
• What is the optimal dosing strategy for children?
• Can probiotics provide primary prevention against AD?
• Can strain-specific findings be repeated in larger-scale studies?
• Repeat clinical trials are ongoing to confirm efficacy.
• Can adjunct treatment with TRPA1 (itch receptor) blockade enhance clinical benefit?
• Can efficacy be enhanced by coadministering oral and topical probiotics?
Soaks (bathing practices)
• Is there an optimal water temperature?
• Is there an optimal frequency of bathing and duration?
Social support
• Can improvement in mental health directly influence AD symptoms?
• Does AD pathology include primary impacts on anxiety or depression that are independent from secondary reactions to disease?
Air quality
• What is the threshold air pollutant concentration that influences AD risk?
• Which pollutants initiate the atopic cascade vs aggravate existing disease?
• No current RCT for air pollution avoidance.
• Can efficacy be reproduced in additional trials?
• Can efficacy be enhanced by optimizing filter selection for specific chemicals?
Diet
• Are there subgroups for which avoidance is recommended?
• Is a proinflammatory diet required for cutaneous pathogenesis in AD?
• Are there specific therapeutic windows for dosing and/or timing of administration?
• Can food serve as a therapeutic or only as prevention?
• Does diet impact the efficacy of medications?

AD, Atopic dermatitis; *RCT*, randomized controlled trials; *TRPA1*, transient receptor potential ankyrin 1.

appropriately flag products with relevant contact allergens; the Contact Allergen Management Program run by the American Contact Dermatitis Society may serve as an additional resource.[131] Additional details regarding the increased risk of contact dermatitis in patients with AD has been recently well reviewed.[132]

Pollution detection systems capable of tracking trace levels of toxins will be required to establish the levels of ambient pollutants that pose a risk for exacerbating or inducing AD and to identify if any air purifiers are capable of mitigating AD-associated toxins. For now, providers and patients can educate themselves about which chemicals are released by factories in their zip code using the Environmental Protection Agency Risk Assessment resource (https://www.epa.gov/trinationalanalysis/where-you-live).[35] Although a case-control or cohort study evaluating the utility of changing from synthetic to natural fabric could be developed, such research will be limited by

the fact that a placebo-controlled study for fabric is likely impossible. Furthermore, interventional studies on which dietary practices may aid AD control and/or maximize benefit to medication or other mitigation strategies are needed.

It will be imperative for providers and researchers to cease broad categorizations such as probiotics, emollients, cosmetics, or cleansers in favor of more refined approach that specifies their molecular contents. Similar sophistication is needed for assessments of pollution or diet modification; evaluations of these interventions must respect the molecular complexity of each exposure. In summary, although much remains unanswered about the mitigatable environmental factors that contribute to AD, greater awareness of the current state of data as well as openness to nonpharmacologic interventions may improve outcomes and build trust between patients and providers.

The authors no longer hold the patent to *Roseomonas mucosa* and thus do not have any financial conflicts for its sales.

Conflicts of interest

The National Institute of Allergy and Infectious Diseases receive royalties from the sales of *Roseomonas mucosa RSM2015*.

REFERENCES

1. Chong AC, Schwartz A, Lang J, et al. Patients' and caregivers' preferences for mental health care and support in atopic dermatitis. *Dermatitis*. 2024;35:S70-S76.
2. Paller A, Jaworski JC, Simpson EL, et al. Major comorbidities of atopic dermatitis: beyond allergic disorders. *Am J Clin Dermatol*. 2018;19:821-838.
3. Schuler CF IV, Billi AC, Maverakis E, Tsoi LC, Gudjonsson JE. Novel insights into atopic dermatitis. *J Allergy Clin Immunol*. 2023;151:1145-1154.
4. Adam DN, Gooderham MJ, Beecker JR, et al. Expert consensus on the systemic treatment of atopic dermatitis in special populations. *J Eur Acad Dermatol Venereol*. 2023;37:1135-1148.
5. Castillo CR, Alishahedani ME, Gough P, et al. Assessing the effects of common topical exposures on skin bacteria associated with atopic dermatitis. *Skin Health Dis*. 2021;1:e41.
6. Ratley G, Sun AA, Capozza K, Barta K, Myles IA. Survey of topical exposure concerns for patients and caregivers dealing with atopic dermatitis. *Front Allergy*. 2023;4:1210973.
7. AAAAI/ACAAI JTF Atopic Dermatitis Guideline Panel, Chu DK, Schneider L, Asiniwasis RN, et al. Atopic dermatitis (eczema) guidelines: 2023 American Academy of Allergy, Asthma and Immunology/American College of Allergy, Asthma and Immunology Joint Task Force on Practice Parameters GRADE- and Institute of Medicine-based recommendations. *Ann Allergy Asthma Immunol*. 2024;132:274-312.
8. Sidbury R, Alikhan A, Bercovitch L, et al. Guidelines of care for the management of atopic dermatitis in adults with topical therapies. *J Am Acad Dermatol*. 2023;89:e1-e20.
9. Maleki-Yazdi KA, Heen AF, Zhao IX, et al. Values and preferences of patients and caregivers regarding treatment of atopic dermatitis (eczema): a systematic review. *JAMA Dermatol*. 2023;159:320-330.
10. Chaudhary PP, Myles IA, Zeldin J, et al. Shotgun metagenomic sequencing on skin microbiome indicates dysbiosis exists prior to the onset of atopic dermatitis. *Allergy*. 2023;78:2724-2731.
11. Berdyshev E, Kim J, Kim BE, et al. Stratum corneum lipid and cytokine biomarkers at age 2 months predict the future onset of atopic dermatitis. *J Allergy Clin Immunol*. 2023;151:1307-1316.
12. Yamamoto-Hanada K, Saito-Abe M, Shima K, et al. mRNAs in skin surface lipids unveiled atopic dermatitis at 1 month. *J Eur Acad Dermatol Venereol*. 2023;37:1385-1395.
13. Rinnov MR, Halling AS, Gerner T, et al. Skin biomarkers predict development of atopic dermatitis in infancy. *Allergy*. 2023;78:791-802.
14. Akdis CA. Does the epithelial barrier hypothesis explain the increase in allergy, autoimmunity and other chronic conditions? *Nat Rev Immunol*. 2021;21:739-751.
15. Zeldin J, Chaudhary PP, Spathies J, et al. Exposure to isocyanates predicts atopic dermatitis prevalence and disrupts therapeutic pathways in commensal bacteria. *Sci Adv*. 2023;9:eade8898.
16. Zeldin J, Tran TT, Yadav M, et al. Antimony compounds associate with atopic dermatitis and influence models of itch and dysbiosis. *Environ Sci Technol Lett*. 2023;10:452-457.
17. Yadav M, Chaudhary PP, D'Souza BN, et al. Diisocyanates influence models of atopic dermatitis through direct activation of TRPA1. *PLoS One*. 2023;18:e0282569.
18. Montero-Vilchez T, Segura-Fernandez-Nogueras MV, Perez-Rodriguez I, et al. Skin barrier function in psoriasis and atopic dermatitis: transepidermal water loss and temperature as useful tools to assess disease severity. *J Clin Med*. 2021;10:359.
19. Cardona ID, Kempe EE, Lary C, Ginder JH, Jain N. Frequent versus infrequent bathing in pediatric atopic dermatitis: a randomized clinical trial. *J Allergy Clin Immunol Pract*. 2020;8:1014-1021.
20. Allan GM, Craig R, Korownyk CS. Atopic dermatitis and bathing. *Can Fam Physician*. 2021;67:758.
21. Jabbar-Lopez ZK, Ung CY, Alexander H, et al. The effect of water hardness on atopic eczema, skin barrier function: a systematic review, meta-analysis. *Clin Exp Allergy*. 2021;51:430-451.
22. Jabbar-Lopez ZK, Ezzamouri B, Briley A, et al. Randomized controlled pilot trial with ion-exchange water softeners to prevent eczema (SOFTER trial). *Clin Exp Allergy*. 2022;52:405-415.
23. Chopra R, Vakharia PP, Sacotte R, Silverberg JI. Efficacy of bleach baths in reducing severity of atopic dermatitis: a systematic review and meta-analysis. *Ann Allergy Asthma Immunol*. 2017;119:435-440.
24. Yin Y, Capozza K, Shao Y, et al. What are patients saying about their experience with atopic dermatitis? Insights from a machine learning analysis of online comments. *Skin Health Dis*. 2022;2:e100.
25. Gough P, Khalid MB, Hartono S, Myles IA. Microbial manipulation in atopic dermatitis. *Clin Transl Med*. 2022;12:e828.
26. Ryczaj K, Chojnowska-Wojtowicz M, Dumycz K, Feleszko W, Kulus M. Prevalence of major food allergens in skincare products for atopic dermatitis. *Postepy Dermatol Alergol*. 2023;40:762-765.

27. Rinaldi AO, Li M, Barletta E, et al. Household laundry detergents disrupt barrier integrity and induce inflammation in mouse and human skin. *Allergy*. 2024;79:128-141.
28. Ogulur I, Pat Y, Aydin T, et al. Gut epithelial barrier damage caused by dishwasher detergents and rinse aids. *J Allergy Clin Immunol*. 2023;151:469-484.
29. Calderon L, Maddalena R, Russell M, et al. Air concentrations of volatile organic compounds associated with conventional and "green" cleaning products in real-world and laboratory settings. *Indoor Air*. 2022;32:e13162.
30. Henriksen JR, Andresen TL, Feldborg LN, Duelund L, Ipsen JH. Understanding detergent effects on lipid membranes: a model study of lysolipids. *Biophys J*. 2010;98:2199-2205.
31. Mijaljica D, Spada F, Harrison IP. Skin cleansing without or with compromise: soaps and syndets. *Molecules*. 2022;27:2010.
32. Ridd MJ, Roberts A, Grindlay D, Williams HC. Which emollients are effective and acceptable for eczema in children? *BMJ*. 2019;367:l5882.
33. Sindher S, Alkotob SS, Shojinaga MN, et al. Pilot study measuring transepidermal water loss (TEWL) in children suggests trilipid cream is more effective than a paraffin-based emollient. *Allergy*. 2020;75:2662-2664.
34. Elias PM. Optimizing emollient therapy for skin barrier repair in atopic dermatitis. *Ann Allergy Asthma Immunol*. 2022;128:505-511.
35. Epa.gov. Where You Live: Risk Assessment. EPA United States Enviromental Protenction Agency. Accessed September 11, 2024. epa.gov/trinationalanalysis/where-you-live
36. Chalmers JR, Haines RH, Bradshaw LE, et al. Daily emollient during infancy for prevention of eczema: the BEEP randomised controlled trial. *Lancet*. 2020;395:962-972.
37. Xu D, Stengel R, Sun P. Effectiveness of emollients in the prevention of atopic dermatitis in infants: a meta-analysis. *Dermatology*. 2022;238:711-716.
38. Ni Chaoimh C, Lad D, Nico C, et al. Early initiation of short-term emollient use for the prevention of atopic dermatitis in high-risk infants-The STOP-AD randomised controlled trial. *Allergy*. 2023;78:984-994.
39. Kelleher MM, Phillips R, Brown SJ, et al. Skin care interventions in infants for preventing eczema and food allergy. *Cochrane Database Syst Rev*. 2022;11:CD013534.
40. Lee JM, Ryu SI, Kim JI, et al. Contribution of container types on cosmetics contamination. *Ann Dermatol*. 2019;31:588-590.
41. Hou A, Silverberg JI. Temporal trends of birthweight are not driving trends of atopic dermatitis prevalence in United States children. *Arch Dermatol Res*. 2022;314:609-611.
42. Hou A, Silverberg JI. Secular trends of atopic dermatitis and its comorbidities in United States children between 1997 and 2018. *Arch Dermatol Res*. 2022;314:267-274.
43. Silverberg JI, Hanifin J, Simpson EL. Climatic factors are associated with childhood eczema prevalence in the United States. *J Invest Dermatol*. 2013;133:1752-1759.
44. Latvala J, von Hertzen L, Lindholm H, Haahtela T. Trends in prevalence of asthma and allergy in Finnish young men: nationwide study, 1966-2003. *BMJ*. 2005;330:1186-1187.
45. Hadi HA, Tarmizi AI, Khalid KA, Gajdacs M, Aslam A, Jamshed S. The epidemiology and global burden of atopic dermatitis: a narrative review. *Life (Basel)*. 2021;11:936.
46. Odhiambo JA, Williams HC, Clayton TO, Robertson CF, Asher MI, Group IPTS. Global variations in prevalence of eczema symptoms in children from ISAAC Phase Three. *J Allergy Clin Immunol*. 2009;124:1251-1258.e23.
47. Wang Y, Allen KJ, Suaini NHA, Peters RL, Ponsonby AL, Koplin JJ. Asian children living in Australia have a different profile of allergy and anaphylaxis than Australian-born children: a State-wide survey. *Clin Exp Allergy*. 2018;48:1317-1324.
48. Tham EH, Loo EXL, Zhu Y, Shek LP. Effects of migration on allergic diseases. *Int Arch Allergy Immunol*. 2019;178:128-140.
49. Nevid MZ, Rabinovitch N, Crooks J, Goleva E, Leung D, Hui J. The association of residential distance from highly trafficked roads with atopic dermatitis risk. *J Allergy Clin Immunol Pract*. 2023;11:1554-1555.
50. Lee JY, Lamichhane DK, Lee M, et al. Preventive effect of residential green space on infantile atopic dermatitis associated with prenatal air pollution exposure. *Int J Environ Res Public Health*. 2018;15:102.
51. Matz CJ, Egyed M, Hocking R, Seenundun S, Charman N, Edmonds N. Human health effects of traffic-related air pollution (TRAP): a scoping review protocol. *Syst Rev*. 2019;8:223.
52. Fadadu RP, Abuabara K, Balmes JR, Hanifin JM, Wei ML. Air pollution and atopic dermatitis, from molecular mechanisms to population-level evidence: a review. *Int J Environ Res Public Health*. 2023;20:2526.
53. Fadadu RP, Green M, Jewell NP, Grimes B, Vargo J, Wei ML. Association of exposure to wildfire air pollution with exacerbations of atopic dermatitis and itch among older adults. *JAMA Netw Open*. 2022;5:e2238594.
54. Fadadu RP, Grimes B, Jewell NP, et al. Association of wildfire air pollution and health care use for atopic dermatitis and itch. *JAMA Dermatol*. 2021;157:658-666.
55. Choi DW, Moon KW, Byeon SH, et al. Indoor volatile organic compounds in atopy patients' houses in South Korea. *Indoor Build Environ*. 2009;18:144-154.
56. Kim YM, Kim J, Ha SC, Ahn K. Effects of exposure to indoor fine particulate matter on atopic dermatitis in children. *Int J Environ Res Public Health*. 2021;18:11509.
57. Kantor R, Kim A, Thyssen JP, Silverberg JI. Association of atopic dermatitis with smoking: a systematic review and meta-analysis. *J Am Acad Dermatol*. 2016;75:1119-1125.e1.
58. Kim SY, Sim S, Choi HG. Atopic dermatitis is associated with active and passive cigarette smoking in adolescents. *PLoS One*. 2017;12:e0187453.
59. Smith B, Engel P, Collier MR, et al. Association between electronic-cigarette use and atopic dermatitis among United States adults. *J Am Acad Dermatol*. 2023;89:163-165.
60. Ratley G, Zeldin J, Sun AA, Yadav M, Chaudhary PP, Myles IA. Spatial modeling connecting childhood atopic dermatitis prevalence with household exposure to pollutants. *Commun Med (Lond)*. 2024;4:74.
61. Bolden AL, Kwiatkowski CF, Colborn T. New look at BTEX: are ambient levels a problem? *Environ Sci Technol*. 2015;49:5261-5276.
62. Moparthi L, Sinica V, Moparthi VK, et al. The human TRPA1 intrinsic cold and heat sensitivity involves separate channel structures beyond the N-ARD domain. *Nat Commun*. 2022;13:6113.
63. Montell C. TRP channels: it's not the heat, it's the humidity. *Curr Biol*. 2008;18:R123-R126.
64. Mahmoud O, Soares GB, Yosipovitch G. Transient receptor potential channels and itch. *Int J Mol Sci*. 2022;24.
65. Yao L, Chen S, Tang H, et al. Transient receptor potential ion channels mediate adherens junctions dysfunction in a toluene diisocyanate-induced murine asthma model. *Toxicol Sci*. 2019;168:160-170.
66. Noroes MM, Santos LG, Gavioli EC, et al. Role of TRPA1 receptors in skin inflammation induced by volatile chemical irritants in mice. *Eur J Pharmacol*. 2019;858:172460.

67. Epa.gov. Final rule for control of hazardous air pollutants from mobile sources. Epa.gov: EPA United States Enviromental Protenction Agency. 40 CFR Parts 59, 80, 85, nad 86.
68. Polzin GM, Kosa-Maines RE, Ashley DL, Watson CH. Analysis of volatile organic compounds in mainstream cigarette smoke. *Environ Sci Technol*. 2007;41:1297-1302.
69. Roberts JM, Veres PR, Cochran AK, et al. Isocyanic acid in the atmosphere and its possible link to smoke-related health effects. *Proc Natl Acad Sci U S A*. 2011;108:8966-8971.
70. Liu X, Huey LG, Yokelson RJ, et al. Airborne measurements of western U.S. wildfire emissions: comparison with prescribed burning and air quality implications. *J Geophys Res Atmos*. 2017;122:6108-6129.
71. Agency for Toxic Substances and Disease Registry. Medical Management Guidelines for Toluene Diisocyanate. Accessed July 4, 2024. Cdc.gov
72. Azimi P, Stephens B. A framework for estimating the US mortality burden of fine particulate matter exposure attributable to indoor and outdoor microenvironments. *J Expo Sci Environ Epidemiol*. 2020;30:271-284.
73. Messier KP, Tidwell LG, Ghetu CC, et al. Indoor versus outdoor air quality during wildfires. *Environ Sci Technol Lett*. 2019;6:696-701.
74. Walker ES, Stewart T, Jones D. Fine particulate matter infiltration at Western Montana residences during wildfire season. *Sci Total Environ*. 2023;896:165238.
75. Li J, Link MF, Pandit S, et al. The persistence of smoke VOCs indoors: partitioning, surface cleaning, and air cleaning in a smoke-contaminated house. *Sci Adv*. 2023;9:eadh8263.
76. CMS.gov. Durable Medical Equipment (DME) Reference List. Accessed September 11, 2024. CMS.gov/medicare-coverage-database/view/ncd.aspx?NCDId=190
77. Sriprapat W, Boraphech P, Thiravetyan P. Factors affecting xylene-contaminated air removal by the ornamental plant Zamioculcas zamiifolia. *Environ Sci Pollut Res Int*. 2014;21:2603-2610.
78. Kim KJ, Kim HJ, Khalekuzzaman M, Yoo EH, Jung HH, Jang HS. Removal ratio of gaseous toluene and xylene transported from air to root zone via the stem by indoor plants. *Environ Sci Pollut Res Int*. 2016;23:6149-6158.
79. Matheson S, Fleck R, Irga PJ, Torpy FR. Phytoremediation for the indoor environment: a state-of-the-art review. *Rev Environ Sci Biotechnol*. 2023;22:249-280.
80. Kumar R, Verma V, Thakur M, Singh G, Bhargava B. A systematic review on mitigation of common indoor air pollutants using plant-based methods: a phytoremediation approach. *Air Qual Atmos Health*. 2023;16:1-27.
81. Langan SM, Silcocks P, Williams HC. What causes flares of eczema in children? *Br J Dermatol*. 2009;161:640-646.
82. Mason R. Fabrics for atopic dermatitis. *J Fam Health Care*. 2008;18:63-65.
83. Swaney MH, Nelsen A, Sandstrom S, Kalan LR. Sweat and sebum preferences of the human skin microbiota. *Microbiol Spectr*. 2023;11:e0418022.
84. Williams JR, Burr ML, Williams HC. Factors influencing atopic dermatitis-a questionnaire survey of schoolchildren's perceptions. *Br J Dermatol*. 2004;150:1154-1161.
85. Kaneko S, Murota H, Murata S, Katayama I, Morita E. Usefulness of sweat management for patients with adult atopic dermatitis, regardless of sweat allergy: a pilot study. *BioMed Res Int*. 2017;2017:8746745.
86. *Energy and Environmental Profile of the U.S. Chemical Industry*. U.S. Department of Energy Office of Industrial Technologies; 2000:2-6.
87. Shen D, Yuan X, Han Y, et al. Evaluation of polycyclic aromatic hydrocarbons (PAHs) in bamboo shoots from soil. *Bull Environ Contam Toxicol*. 2021;106:589-593.
88. Bian F, Zhong Z, Zhang X, Yang C, Gai X. Bamboo - an untapped plant resource for the phytoremediation of heavy metal contaminated soils. *Chemosphere*. 2020;246:125750.
89. Haug S, Roll A, Schmid-Grendelmeier P, et al. Coated textiles in the treatment of atopic dermatitis. *Curr Probl Dermatol*. 2006;33:144-151.
90. Gauger A, Fischer S, Mempel M, et al. Efficacy and functionality of silver-coated textiles in patients with atopic eczema. *J Eur Acad Dermatol Venereol*. 2006;20:534-541.
91. Koller DY, Halmerbauer G, Bock A, Engstler G. Action of a silk fabric treated with AEGIS in children with atopic dermatitis: a 3-month trial. *Pediatr Allergy Immunol*. 2007;18:335-338.
92. Khan A, Adalsteinsson J, Whitaker-Worth DL. Atopic dermatitis and nutrition. *Clin Dermatol*. 2022;40:135-144.
93. Tan T, Xiao D, Li Q, et al. Maternal yogurt consumption during pregnancy and infantile eczema: a prospective cohort study. *Food Funct*. 2023;14:1929-1936.
94. Kim HJ, Ju SY, Park YK. Kimchi intake and atopic dermatitis in Korean aged 19-49 years: the Korea National Health and Nutrition Examination survey 2010-2012. *Asia Pac J Clin Nutr*. 2017;26:914-922.
95. Venter C, Palumbo MP, Glueck DH, et al. The maternal diet index in pregnancy is associated with offspring allergic diseases: the Healthy Start study. *Allergy*. 2022;77:162-172.
96. Park S, Bae JH. Fermented food intake is associated with a reduced likelihood of atopic dermatitis in an adult population (Korean National Health and Nutrition Examination Survey 2012-2013). *Nutr Res*. 2016;36:125-133.
97. Ercelik HC, Kaya V. The effects of fermented food consumption in pregnancy on neonatal and infant health: an integrative review. *J Pediatr Nurs*. 2023;75:173-179.
98. Venter C, Agostoni C, Arshad SH, et al. Dietary factors during pregnancy and atopic outcomes in childhood: a systematic review from the European Academy of Allergy and Clinical Immunology. *Pediatr Allergy Immunol*. 2020;31:889-912.
99. Hattangdi-Haridas SR, Lanham-New SA, Wong WHS, Ho MHK, Darling AL. Vitamin D deficiency and effects of vitamin D supplementation on disease severity in patients with atopic dermatitis: a systematic review and meta-analysis in adults and children. *Nutrients*. 2019;11:1854.
100. Javanbakht MH, Keshavarz SA, Djalali M, et al. Randomized controlled trial using vitamins E and D supplementation in atopic dermatitis. *J Dermatolog Treat*. 2011;22:144-150.
101. Venter C. Immunonutrition: diet diversity, gut microbiome and prevention of allergic diseases. *Allergy Asthma Immunol Res*. 2023;15:545-561.
102. Nijhawan RI, Molenda M, Zirwas MJ, Jacob SE. Systemic contact dermatitis. *Dermatol Clin*. 2009;27:355-364. vii..
103. Ahuja K, Issa CJ, Nedorost ST, Lio PA. Is food-triggered atopic dermatitis a form of systemic contact dermatitis? *Clin Rev Allergy Immunol*. 2024;66:1-13.
104. Mislankar M, Zirwas MJ. Low-nickel diet scoring system for systemic nickel allergy. *Dermatitis*. 2013;24:190-195.
105. Oykhman P, Dookie J, Al-Rammahy H, et al. Dietary elimination for the treatment of atopic dermatitis: a systematic review and meta-analysis. *J Allergy Clin Immunol Pract*. 2022;10:2657-2666.e8.
106. Rotter G, Teut M, Binting S, Michalsen A, Ortiz M, Brinkhaus B. Lifestyle behaviors in patients with atopic dermatitis: results of a cross-sectional study following a randomized controlled trial. *Complement Med Res*. 2022;29:419-428.

107. Lund E. Comparison of additive and multiplicative models for reproductive risk factors and post-menopausal breast cancer. *Stat Med*. 1995;14:267-274.
108. Xue X, Yang X, Shi X, Deng Z. Efficacy of probiotics in pediatric atopic dermatitis: a systematic review and meta-analysis. *Clin Transl Allergy*. 2023;13:e12283.
109. Makrgeorgou A, Leonardi-Bee J, Bath-Hextall FJ, et al. Probiotics for treating eczema. *Cochrane Database Syst Rev*. 2018;11:CD006135.
110. Tan-Lim CSC, Esteban-Ipac NAR, Recto MST, Castor MAR, Casis-Hao RJ, Nano ALM. Comparative effectiveness of probiotic strains on the prevention of pediatric atopic dermatitis: a systematic review and network meta-analysis. *Pediatr Allergy Immunol*. 2021;32:1255-1270.
111. Deng WY, Chen WJ, Zhong HJ, Wu LH, He XX. Washed microbiota transplantation: a case report of clinical success with skin and gut microbiota improvement in an adolescent boy with atopic dermatitis. *Front Immunol*. 2023;14:1275427.
112. Myles IA, Moore IN, Castillo CR, Datta SK. Differing virulence of Healthy skin commensals in mouse models of infection. *Front Cell Infect Microbiol*. 2018;8:451.
113. Myles IA, Williams KW, Reckhow JD, et al. Transplantation of human skin microbiota in models of atopic dermatitis. *JCI Insight*. 2016;1:e86955.
114. Myles IA, Reckhow JD, Williams KW, Sastalla I, Frank KM, Datta SK. A method for culturing Gram-negative skin microbiota. *BMC Microbiol*. 2016;16:60.
115. Jacobson ME, Myles IA, Paller AS, Eichenfield LF, Simpson EL. A randomized, double-blind, placebo-controlled, multicenter, 16-week trial to evaluate the efficacy and safety of FB-401 in children, adolescents, and adult subjects (ages 2 years and older) with mild-to-moderate atopic dermatitis. *Dermatology*. 2024;240:85-94.
116. Myles IA, Earland NJ, Anderson ED, et al. First-in-human topical microbiome transplantation with Roseomonas mucosa for atopic dermatitis. *JCI Insight*. 2018;3:e120608.
117. Myles IA, Castillo CR, Barbian KD, et al. Therapeutic responses to Roseomonas mucosa in atopic dermatitis may involve lipid-mediated TNF-related epithelial repair. *Sci Transl Med*. 2020;12.
118. Defensin. Roseomonas mucosa in Eczema. Accessed June 10, 2024. Skinesa.com
119. Song Z, Song R, Liu Y, Wu Z, Zhang X. Effects of ultra-processed foods on the microbiota-gut-brain axis: the bread-and-butter issue. *Food Res Int*. 2023;167:112730.
120. Ridaura VK, Faith JJ, Rey FE, et al. Gut microbiota from twins discordant for obesity modulate metabolism in mice. *Science*. 2013;341:1241214.
121. Cai XC, Wang SH, Wang CX, et al. Epidemiology of mental health comorbidity in patients with atopic dermatitis: an analysis of global trends from 1998 to 2022. *J Eur Acad Dermatol Venereol*. 2024;38:496-512.
122. Sandhu JK, Wu KK, Bui TL, Armstrong AW. Association between atopic dermatitis and suicidality: a systematic review and meta-analysis. *JAMA Dermatol*. 2019;155:178-187.
123. Kelly KA, Balogh EA, Kaplan SG, Feldman SR. Skin disease in children: effects on quality of life, stigmatization, bullying, and suicide risk in pediatric acne, atopic dermatitis, and psoriasis patients. *Children (Basel)*. 2021;8:1057.
124. Kim RW, Barta K, Begolka WS, et al. Qualitative analysis of the impact of atopic dermatitis on caregivers. *Br J Dermatol*. 2022;187:1038-1041.
125. Schneider M, Levant B, Reichel M, Gulbins E, Kornhuber J, Muller CP. Lipids in psychiatric disorders and preventive medicine. *Neurosci Biobehav Rev*. 2017;76:336-362.
126. Konstantinou GN, Konstantinou GN, Koulias C, Petalas K, Makris M. Further understanding of neuro-immune interactions in allergy: implications in pathophysiology and role in disease progression. *J Asthma Allergy*. 2022;15:1273-1291.
127. Radtke S, Grossberg AL, Wan J. Mental health comorbidity in youth with atopic dermatitis: a narrative review of possible mechanisms. *Pediatr Dermatol*. 2023;40:977-982.
128. Yuka. Make the right choices for your health. Accessed July 1, 2024. Yuka.io/en/
129. EWG. EWG's Healthy Living App. Accessed August 1, 2024. https://www.ewg.org
130. SkinSafe. 2024. Accessed September 1, 2024. www.skinsafeproducts.com
131. American Contact Dermatitis Society. CAMP Access 2024. Accessed September 10, 2024. contactderm.org/resources/acds-camp
132. Johnson H, Aquino MR, Snyder A, et al. Prevalence of allergic contact dermatitis in children with and without atopic dermatitis: a multicenter retrospective case-control study. *J Am Acad Dermatol*. 2023;89:1007-1014.