Current guidelines for the evaluation and management of atopic dermatitis: A comparison of the Joint Task Force Practice Parameter and American Academy of Dermatology guidelines

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Atopic dermatitis (AD) is a chronic pruritic inflammatory disease that commonly presents in the pediatric population. Although definitions and diagnosis of AD have largely been agreed upon, allergists and dermatologists have similar and divergent approaches to the management of AD. This review facilitated integration of the American Academy of Allergy, Asthma & Immunology/American College of Allergy, Asthma & Immunology Joint Task Force 2012 AD Practice Parameter and the 2014 American Academy of Dermatology guidelines to highlight the basic principles of AD management and discuss therapies and management of AD from the distinct perspectives of the allergist and dermatologist. (J Allergy Clin Immunol 2017;139:S49-57.)

Key words: *Atopic dermatitis, eczema, guidelines, Joint Task Force, American Academy of Dermatology, topical and systemic treatments*

Atopic dermatitis (AD) is a chronic, remitting-relapsing inflammatory dermatitis often managed by a multidisciplinary group of providers, including allergists, dermatologists, and primary care practitioners. Because the pathogenesis of AD is complex and multifactorial, there are numerous approaches to therapeutic management. Current AD management guidelines cover a broad range of interventions, from treating acute flares to environmental modifications. Allergists and dermatologists have both common and distinct approaches to AD management highlighted in their respective guidelines, providing multiple approaches to disease management. We compare and contrast the recent American Academy of Allergy, Asthma & Immunology (AAAAI)/American College of Allergy, Asthma & Immunology (ACAAI) Joint Task Force (JTF) 2012 AD Practice Parameter and the American Academy of Dermatology (AAD) 2014 guidelines, highlighting differing approaches to disease management.

Abbreviations used

AAAAI:	American Academy of Allergy, Asthma & Immunology
AAD:	Academy of Dermatology
ACAAI:	American College of Allergy, Asthma & Immunology
AD:	Atopic dermatitis
JTF:	Joint Task Force
MMF:	Mycophenolate mofetil
TCI:	Topical calcineurin inhibitors
TCS:	Topical corticosteroids

METHODS

We reviewed and compared the following published guidelines:

1. the 2014 AAD guidelines: "Guidelines of care for the management of atopic dermatitis" sections 1 to 4[1-4] and
2. the 2012 JTF Practice Parameter Update, representing the AAAAI, the ACAAI, and the Joint Council of Allergy, Asthma & Immunology: "Atopic dermatitis: a practice parameter update 2012."[5]

The AAD guidelines and JTF Practice Parameter used similar review processes to develop their guidelines and recommendations by performing a systematic search of PubMed and the Cochrane Library for relevant articles using subject headings, such as "atopic dermatitis," "eczema," "topical corticosteroid," and "calcineurin inhibitor," among others.[1,5] However, the AAD additionally used the Global Resources for Eczema Trials database for all newly identified clinical questions, whereas the JTF excluded this source.[1]

The practice parameter was developed by the JTF, which has contributed 33 practice parameters to the field of allergy and immunology, including the original parameter on AD.[5] These guidelines were created primarily by subspecialists in allergy and immunology but also included dermatologists from the United States and Europe, as well as a psychologist. In contrast, the AAD work group generally consisted of academic dermatologists. Both sets of work group participants are experts in the field of AD.

The JTF Practice Parameter was an update of the 2004 parameter on AD, whereas the previous AAD guidelines were also published in 2004. The JTF Practice Parameter is a single document with an executive summary, followed by evidence-based summary statements and an annotated flowchart of the diagnosis and management of AD. For simplicity, the JTF Practice Parameter will be hereafter referred to as the JTF guidelines. The AAD guidelines are organized into 4 separate publications, with data highlighted in tabular form. The AAD work group ranked the strength of their recommendations in descending order from "A" to "C," and the JTF used a similar strength of recommendation scale from "A" to "D," both of which were based on the grade of evidence available for these clinical practices (values are shown in parentheses in the text, as described in Tables I and II).[1,5]

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Disclosure of potential conflict of interest: L. F. Eichenfield serves as a consultant for Allergan, Anacore/Pfizer, Galderman, Lilly, Otsuka/Medimetriks, TopMD, and Valeant; serves on the board for Valeant; and receives speakers' fees from Valeant. M. Boguniewicz receives personal fees from Omnia Education. The rest of the authors declare that they have no relevant conflicts of interest.

Received for publication December 6, 2016; revised January 16, 2017; accepted for publication January 20, 2017.

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0091-6749/$36.00

http://dx.doi.org/10.1016/j.jaci.2017.01.009

TABLE I. Strength of recommendation and grade of evidence from the AAD

Level A	Recommendation based on consistent and good-quality patient-oriented evidence	Level I	Good-quality patient-oriented evidence
Level B	Recommendation based on inconsistent or limited-quality patient-oriented evidence	Level II	Limited-quality patient-oriented evidence
Level C	Recommendation based on consensus, opinion, case studies, or disease-oriented evidence	Level III	Other evidence, including consensus guidelines, opinion, case studies, or disease-oriented evidence

Adapted from Eichenfield et al.[1]

TABLE II. Strength of recommendation and category of evidence from the JTF

Level A	Directly based on category I evidence	Level Ia	Evidence from meta-analysis of RCT
Level B	Directly based on category II evidence	Level Ib	Evidence from at least 1 RCT
Level C	Directly based on category III evidence or extrapolated recommendation from category I or II evidence	Level IIa	Evidence from at least 1 controlled study without randomization
Level D	Directly based on category IV evidence or extrapolated recommendation from category I, II, or III evidence	Level IIb	Evidence from at least 1 other type of quasiexperimental study
		Level III	Evidence from nonexperimental descriptive studies, such as comparative studies
		Level IV	Evidence from expert committee reports, opinions, or clinical experience of respected authorities or both

Adapted from Schneider et al.[5] *RCT*, Randomized controlled trial.

DEFINITIONS AND DIAGNOSIS

Both guidelines define AD as a chronic pruritic inflammatory disease that commonly presents in the pediatric population but can also affect adults. Although both guidelines agree that the disease can be familial, only the AAD guidelines associate AD with an additional history of type I allergies, allergic rhinitis, and asthma.[1,5]

Both guidelines concur that AD is diagnosed clinically based on the patient's history, characteristic clinical findings, and exclusion of other dermatoses.[1,5] Although the AAD guidelines distinguish atopy as an important but not required feature for the diagnosis of AD, the JTF guidelines assert the necessity of an atopic history.[1,5] The JTF guidelines outline the typical appearance according to the chronicity of AD lesions as pruritic, erythematous papulovesicular lesions associated with excoriation and serous exudate in the acute setting, whereas findings of lichenification, papules, and excoriations can be seen in patients with chronic AD (JTF: D). In contrast, AAD guidelines delve further into standardized criteria based on revised Hanifin and Rajka diagnostic schemes (Table III).[1,5]

Additionally, although the AAD guidelines mandate the exclusion of other common cutaneous disorders before diagnosis, such as contact dermatitis and cutaneous lymphomas, the JTF guidelines suggest this thorough re-evaluation, particularly in patients recalcitrant to optimal therapeutic management.[1,5] Other considerations discussed by the AAD work group include the lack of specific biomarkers required for diagnosis or severity assessment (AAD: BII) and the recommendation against obtaining routine IgE levels (AAD: AI).[1,5]

NONPHARMACOLOGIC INTERVENTIONS

Bathing practices

Recommendations for bathing practices largely stem from expert consensus, with few objective measures documented in the literature. The JTF and AAD guidelines both recommend bathing with warm water, followed by application of moisturizers (JTF: D; AAD: CIII).[2,5]

Bathing boosts skin hydration while eliminating residual bacteria, crusting, and irritants. However, transepidermal water loss can occur through evaporative losses after bathing. Application of moisturizers after bathing is critical to maintain adequate cutaneous hydration, as discussed below. Although not explicitly defined in the AAD guidelines, the JTF group delineates an appropriate bathing duration of at least 10 minutes (JTF: D).[5] The JTF guidelines also support the addition of bathing additives (ie, oatmeal or baking soda) for symptomatic relief of pruritus and skin irritation while acknowledging that they do not decrease transepidermal water loss (JTF: D).[5] In contrast, the AAD guidelines recommend against use of bath additives and acidic spring water, with the exception of bleach (AAD: CIII).[2]

Both guidelines encourage the avoidance of damaging, drying, and irritating soaps with alkaline pH. The JTF and AAD guidelines similarly recommend the limited use of neutral pH, fragrance-free hypoallergenic soaps or nonsoap cleaners (JTF: B; AAD: CIII).[2,5]

Moisturizers

Topical therapies are the cornerstone of AD treatment and, when used in conjunction with other interventions, can target different components of AD pathogenesis. Both the JTF and AAD guidelines concur that moisturizers improve skin barrier function and reduce transepidermal water loss, thereby increasing skin hydration (JTF: D).[2,5] Moisturizers have been indicated as primary therapy (JTF: D; AAD: AI) for mild AD especially and as adjunctive therapy for moderate-to-severe AD.[2,5] Clinically, moisturizers lessen the signs and symptoms of AD, including erythema, fissuring, and pruritus, thus impeding the itch-scratch

TABLE III. Features to be considered in the diagnosis of patients with AD

Essential features
Required:
- Pruritus and eczema (acute, subacute, or chronic)
- Typical morphology and age-specific patterns
- Chronic or relapsing history

Important features
Observed in a majority of cases and adds support to diagnosis:
- Early age of onset, atopy, personal and/or family history, IgE reactivity, xerosis

Associated features
Help suggest the diagnosis but are nonspecific:
- Atypical vascular responses
- Keratosis pilaris/pityriasis alba/hyperlinear palms/ichthyosis ocular/periorbital changes
- Other regional findings (eg, perioral changes/periauricular lesions)
- Perifollicular accentuation/lichenification/prurigo lesions

Adapted from Eichenfield et al.[1]

cycle. Notably, their use also decreases the amount of prescription topical agents needed for adequate control of AD.[2,5]

Although the JTF and AAD guidelines acknowledge that most hydrophilic ointments carry the advantage of not containing preservatives, solubilizers, or fragrances, no specific recommendations regarding vehicle systems and types of moisturizers (eg, petrolatum vs more expensive "barrier creams") have been detailed.[2,5] Moreover, these guidelines discuss the paucity of systematic studies to define the optimal amount and frequency of moisturizer application; however, generous and frequent application on hydrated skin is suggested.[2,5] Guidelines recommend the application of a moisturizer soon after bathing to improve skin hydration in patients with AD (JTF: D; AAD: BII).[2,5]

Prescription emollient devices are a class of topical nonsteroidal agents that have been proposed to target specific skin barrier defects in patients with AD. However, AAD guidelines do not recommend their use because they have not shown superiority over other moisturizing products.[2]

In conclusion, both guidelines recommend moisturizing agents for the treatment of active disease, maintenance, and prevention of flares. No single moisturizer has been proved to be superior, and this choice can be made based on patient and provider preference.[2,5]

TOPICAL PHARMACOTHERAPIES

Corticosteroids

Topical corticosteroids (TCS) are endorsed as effective therapies for AD.[2,5] TCS are indicated when nonpharmacologic interventions have failed (JTF: A; AAD: AI).[2,5] It has been hypothesized that TCS therapy can impede the mechanisms of antigen processing, thereby inhibiting the release of proinflammatory cytokines.[2] TCS are effective for both active inflammation and disease prophylaxis; however, there are no data to support a specific agent among the 7 TCS classes, and there is limited evidence to recommend an optimal dosing or frequency regimen.[2,5] According to both guidelines, low-potency TCS are generally

TABLE IV. TCI formulations

Formulations for tacrolimus ointment for moderate-to-severe AD:
- 0.03% formulation for patients aged 2-15 y
- 0.1% formulation for patients aged ≥16 y

Pimecrolimus 1% cream is indicated for patients with mild-to-moderate AD aged ≥2 y.

Source: Eichenfield et al.[2]

suggested for maintenance therapy, whereas intermediate and high-potency TCS are recommended for the acute control of AD (JTF: A; AAD: BII).[2,5] There is limited evidence to recommend an optimal dosing or frequency of TCS. Twice-daily application of TCS is commonly recommended for the treatment of acute AD based on the registration studies of most TCS, which were approved with twice-daily indication. However, the guidelines reference studies that discuss the utility of daily TCS use as being effective as twice-daily application.[2] For "proactive" maintenance therapy, the AAD suggests once- to twice-weekly application of TCS in commonly flaring areas to prevent relapses, whereas the JTF discusses long-term control with twice-weekly TCS, and this therapeutic option has been added to the updated flow chart as "Box 7."[2,5] Although no optimal quantity of TCS has been definitively recommended, AAD guidelines highlight the "fingertip unit" method applied over an area equivalent to 2 palms in addition to the use of charts that propose quantities that are age and area based.[2]

Both guidelines caution use of TCS on areas of thin skin, such as the face, neck, and skin folds, because adverse effects are directly related to the surface area of affected skin, skin thickness, use of occlusive dressing, and potency and duration of TCS administered (JTF: D; AAD: AI & BIII).[2,5] Several local adverse effects from TCS include the development of acneiform or rosacea-like eruptions, focal hypertrichosis, purpura, skin atrophy, striae, and telangiectasia. Systemic sequelae have been rare but include risk of the hypothalamic-pituitary-adrenal axis and growth suppression. Despite this concern, the JTF and AAD guidelines differ, with the latter strongly recommending against undertreatment in the context of steroid phobia, and emphasize the importance of patient education and counseling to improve adherence.[2,5]

Calcineurin inhibitors

Topical calcineurin inhibitors (TCI), including tacrolimus and pimecrolimus (Table IV),[2] are a distinct class of steroid-sparing, anti-inflammatory agents that have been shown to be efficacious in acute flares and maintenance therapy of AD in both adults and children older than 2 years (JTF: A; AAD: AI).[2,5] TCI exert their anti-inflammatory properties by inhibiting calcineurin-dependent T-cell activation, thereby impeding production of proinflammatory cytokines and mediators. The guidelines agree that use of TCI at sites of sensitive or thin skin offers an advantage over use of TCS (JTF: A; AAD: AI).[2,5] TCI are usually offered as a second-line therapy for acute and chronic treatment of AD in patients who have not responded adequately to other topical treatments or when those treatments are not recommended.[2]

Twice-daily application of either tacrolimus ointment or pimecrolimus cream is efficacious in treating inflamed AD lesions

and resolving pruritus. In contrast, proactive or maintenance therapy for AD that includes intermittent application of TCI twice daily or twice or thrice weekly to recurrent sites of involvement has been shown to reduce relapse (JTF: A; AAD: AI).[2,5]

The most common side effects of TCI are localized site reactions, including burning, stinging, and pruritus, which commonly occur during the first week of treatment. Both guidelines emphasize the importance of counseling patients on these potential side effects to prevent premature discontinuation of treatment (JTF: A; AAD: BII).[2,5] Although not discussed in the JTF Practice Parameter, the AAD guidelines recommend the preceding use of TCI with TCS, where appropriate, to lessen the severity of local skin reactions (AAD: BII). In addition to informing patients about immediate site reactions, both guidelines advocate for proactive guidance regarding the boxed warning on TCI, although a causal relationship has not been established (AAD: CIII). JTF and AAD guidelines review large prospective studies that suggest a correlation between an increased risk of lymphoma with AD disease severity, without an association with TCI use.[2,5] Moreover, TCI can increase the prevalence of local viral infections while decreasing the population of *Staphylococcus aureus*; however, both guidelines cite the evidence supporting these theoretical risks as inconsistent.[2]

Both guidelines suggest similar efficacy and safety profiles at either the 0.03% or 0.1% strengths of tacrolimus in children, with 0.1% being superior in adults; however, this is based on inconsistent evidence.[2,5] Although not discussed in the JTF guidelines, AAD guidelines recommend against routine blood monitoring of TCI levels in patients with AD given the low to negligible systemic absorption after topical application.[2]

Distinct from the JTF guidelines, AAD guidelines suggest concurrent therapy with TCI and TCS (AAD: BII).[2] These guidelines recommend that TCS can be initially used to control a flare, whereas TCI can be applied as maintenance therapy to prevent relapse, although the evidence for this regimen has been inconsistent.[2]

Other topical therapies

Both JTF and AAD guidelines note that topical coal tar preparations (one of the oldest therapies) in the treatment of AD have fallen out of favor because of the lack of well-controlled randomized studies demonstrating their efficacy.[2,5] Despite inconsistent evidence, the JTF advocates the use of topical coal tar when AD involves the scalp.[5]

Both guidelines similarly recommend consideration of proactive antimicrobial bleach baths in patients with recurrent skin infections (JTF: A; AAD: BII). The AAD guidelines endorse intranasal mupirocin in conjunction with bleach baths in this population of patients (AAD: AI).[2] Moreover, although the JTF guidelines highlight the potential antimicrobial benefit of topical antiseptics in conjunction with topical anti-inflammatory medications, the AAD guidelines advise against these topical antiseptic and antimicrobial preparations, citing concerns of widespread antimicrobial resistance (AAD: AI).[2]

Use of topical antihistamines in the treatment of pruritus in patients with AD was not discussed in the JTF guidelines. These agents are not recommended by the AAD guidelines because of the risk of absorption and development of photoallergic contact dermatitis.[2]

Wet wrap therapy

Both guidelines recommend wet wrap therapy in conjunction with TCS for the management of recalcitrant AD (JTF: A; AAD: BII).[2,5] The JTF guidelines caution against overuse of wet wrap therapy, highlighting the detrimental consequences of prolonged therapy, including folliculitis, skin maceration, and secondary infections. This group discourages the use of TCI in conjunction with wet wrap therapy.[5]

SYSTEMIC THERAPY

Phototherapy

The AAD and JTF guidelines both recommend phototherapy as a treatment for AD refractory to topical treatments (JTF: A; AAD: BII).[3,5] Consideration of availability, cost, patient skin type, skin cancer history, and patient use of photosensitizing medications might help with the selection of phototherapy.[3] Although AAD guidelines outline the multiple forms of light therapy without a definitive recommendation of a particular therapy, the JTF guidelines consider narrow-band UVB as the most effective phototherapy option, given its low-risk profile, relative efficacy, availability, and provider comfort level.[3,5] JTF guidelines additionally suggest using UVA1 for acute exacerbations, UVB modalities for chronic AD, and photochemotherapy with psoralen and UVA only for patients with severe widespread AD.[5] AAD guidelines state that phototherapy can be used as maintenance therapy in patients with chronic disease.[3]

Dosing and frequency of phototherapy are dependent on minimal erythema dose, Fitzpatrick skin type, or both.[3] Both guidelines review several cutaneous adverse reactions, including actinic damage, local erythema and tenderness, and altered pigmentation, in addition to less common systemic effects, such as increased risk for cutaneous malignancies and cataract formation, among others.[3,5]

Systemic immunosuppressants

Both guidelines recommend immunomodulatory agents in a subset of patients with severe AD refractory to topical regimens and phototherapy or when quality of life is severely affected (JTF: A; AAD: see below).[3,5] Although both guidelines agree that there is a paucity of data indicating the relative efficacy of each systemic agent,[3,5] the AAD guidelines suggest that cyclosporine (AAD: BI-II), methotrexate (AAD: BII), mycophenolate mofetil (MMF; AAD: CIII), and azathioprine (AAD: BII) are widely used and more efficacious in treating AD when compared with IFN-γ (AAD: BII) and oral calcineurin inhibitors.[3] Insufficient data exist to definitively recommend optimal dosing, duration of therapy, and monitoring protocols for these drugs.[3,5] Although the AAD tabulates dosages, monitoring, adverse effects, interactions, and contraindications of systemic immunomodulants, the JTF emphasizes their potential serious adverse effects.[3,5]

Cyclosporine is an immunosuppressant of T cells and IL-2 through impedance of cytokine gene expression.[3,5] JTF guidelines suggest that short-term treatment with 5 mg/kg/d cyclosporine in patients with severe refractory AD might reduce disease severity and improve quality of life.[5] In contrast, AAD guidelines mention great variability in dosages and duration but otherwise suggest higher initial doses to achieve disease control.[3] Both guidelines agree on monitoring for side effects of hypertension, renal impairment, and liver function. Although JTF

guidelines additionally emphasize gastrointestinal issues associated with cyclosporine, the AAD guidelines recommend further monitoring for dyslipidemia, gingival hyperplasia, increased risk for malignancies, and immunosuppression (tuberculosis and HIV testing, if indicated).[3,5]

Azathioprine is a purine analog that inhibits DNA production and thus affects rapidly dividing cells, such as B and T cells in inflammatory settings.[3] As with other systemic agents, there is no consistent dosage recommended for azathioprine.[3] AAD guidelines strongly recommend obtaining a patient's thiopurine methyltransferase activity level to determine dosages of azathioprine, whereas the JTF recommends obtaining this level to determine the risk for myelosuppression while taking azathioprine.[3,5] AAD guidelines discuss further adverse effects in addition to leukopenia, including gastrointestinal disturbances, headache, hypersensitivity reactions, liver abnormalities, and increased risk for malignancy.[3]

Methotrexate functions as a folic acid antagonist, which interferes with purine and pyrimidine synthesis and thus impairs the production of nucleotide synthesis.[3] Although the JTF suggests that methotrexate is effective, the AAD guidelines discuss the lack of consistency among studies regarding methods, dosing, and duration of therapy to comment on its true efficacy. Gastrointestinal toxicity has been the predominant side effect, although bone marrow suppression, pulmonary fibrosis, and malignancies have also been associated with methotrexate.[3,5] The AAD recommends folate supplementation for all patients with AD taking methotrexate to reduce the incidence of several aforementioned effects.[3]

MMF impairs purine synthesis by inhibiting inosine monophosphate dehydrogenase, selectively affecting B and T cells because they lack a purine scavenger pathway.[3,5] Distinct from the JTF guidelines that suggest comparable efficacy with other agents, the AAD considers MMF as an alternative and variably effective therapy option for patients with refractory AD.[3,5] The most common side effects include gastrointestinal symptoms, which might improve on administration of the enteric-coated formulations.[3] Other side effects include hematologic disturbances and genitourinary symptoms in addition to increased risk for malignancies.

IFN-γ is a cytokine that enhances natural killer cell proliferation and increases macrophage oxidation, affecting both the innate and adaptive immune systems.[3] Similar to MMF, AAD guidelines discuss the efficacy of IFN-γ as inconsistent and thus recommend it as an alternative therapy in patients with AD refractory to phototherapy or other systemics.[3,5] Although the JTF work group considers IFN-γ an effective agent with a strength of recommendation level of A, references citing the efficacy of IFN-γ have varying levels of evidence.[5] Both guidelines detail several constitutional side effects associated with its use.[3,5]

Systemic corticosteroids

Corticosteroids are made naturally by the adrenal gland to regulate the human stress response and immune system.[3] The guidelines differ in their recommendation of systemic corticosteroids (JTF: A; AAD: BII). Although the JTF guidelines support their use as a short course in patients with acute disease, they strongly recommend against long-term use and use in children. In contrast, the AAD guidelines recommend avoidance of systemic steroids, if possible, for the treatment of AD, with exclusive reservation for acute severe exacerbations and as a bridge therapy to another systemic, steroid-sparing treatment.[3]

Other systemic treatments

Limited data exist to determine the utility of rituximab, omalizumab, intravenous immunoglobulin, and oral calcineurin inhibitors in the management of AD.[3,5] Neither guideline includes discussion of newer biologics or small-molecule agents (eg, dupilumab and Janus kinase inhibitors).

Systemic antihistamines

The AAD advises against general use of systemic sedating (AAD: CIII) and nonsedating (AAD: AII) antihistamines.[3] However, both JTF and AAD guidelines suggest sedating antihistamines for short-term sporadic use in patients with disturbed sleep caused by pruritus (JTF: C).[3,5]

Vitamin D therapy

Although not discussed in AAD guidelines, JTF guidelines suggest vitamin D supplementation for patients with AD, especially if they have either a documented low level or poor vitamin D intake (JTF: B).[5]

Systemic antimicrobials

As previously discussed, it is widely acknowledged that patients with AD have a high rate of infectious complications, directly resulting from cutaneous bacterial colonization with *S aureus* and other pathogens.[3] The use of systemic antimicrobial agents in AD management is not routinely recommended in the absence of clinical findings consistent with cutaneous bacterial superinfection (AAD: BII).[3] However, systemic antibiotics can be recommended for use in patients with evidence of bacterial infection alongside other therapies (AAD: A). Both guidelines advocate for the use of systemic antivirals in the treatment of eczema herpeticum (JTF: B; AAD CII). The JTF guidelines support the consideration of fungal infections as a possible complication of AD and suggest diagnostic testing, including KOH prep, skin culture, or specific IgE *Malassezia* species testing (JTF: C).[5]

Hospitalization

The JTF Practice Parameter provides a rationale for hospitalization of patients with AD not responsive to therapy (JTF: D).[5]

IRRITANTS, ALLERGENS, AND ENVIRONMENTAL MODIFICATIONS

Irritants

The JTF and AAD recognize the potential role of irritants, aeroallergens, and foods in AD symptomatology. The avoidance of allergens and irritants specific to the patient might decrease disease severity and provide symptomatic relief.

In light of the lower threshold for cutaneous irritation in patients with AD, both groups recommend avoidance of common triggering irritants (ie, acids, bleaches, fragrances, solvents, and wool; JTF: B; AAD: CIII). The JTF guidelines endorse additional environmental modifications, including temperature and

humidity control; avoidance of activities involving contact, heavy uniforms, and/or extreme perspiration; and preferential use of nonirritating sunscreens (JTF: D).

The AAD and JTF guidelines fundamentally differ on their respective views of clothing modifications in AD management. The JTF group acknowledges the potential benefit of removing residual irritating chemicals and detergents from new clothing before cutaneous contact. They specifically endorse liquid instead of powder detergent and suggest an additional rinse cycle to remove remaining detergents and other irritating chemicals (JTF: B). They also discourage occlusive clothing in favor of loose-fitting open-weave cotton or cotton blend attire (JTF: B). In contrast, the AAD guidelines recommend against special laundering techniques or specific products (AAD: CIII). Additionally, the AAD guidelines favor smooth clothing without irritating fabrics and fibers to minimize cutaneous irritation while citing insufficient evidence to recommend specialized clothing fabrics (AAD: BII).

Environmental and food allergies

Although the relationship between allergic sensitization and AD is widely recognized, the role of food and environmental allergies in the disease remains debatable. Both groups acknowledge the increased frequency of food allergies in patients with AD, especially patients less than 5 years of age with moderate-to-severe disease. The AAD and JTF guidelines similarly encourage active assessment for clinical signs of food allergy during AD office visits.[4,5]

Both groups oppose allergy testing independent of clinical assessment. The JTF encourages initial diagnostic use of food-specific IgE antibody testing in patients with AD with clinical presentation suspicious for food allergy. In this subset of patients, oral food challenge is subsequently recommended if IgE test results are negative.[5] The AAD emphasizes the utility of placebo-controlled oral food challenge for definitive diagnosis and states the unlikely improvement in disease severity with elimination diets (AAD: BII).[4]

Both groups strongly recommend against food elimination based on allergy tests alone, citing the low specificity of such testing and the potential for nutritional deficiencies (JTF: B; AAD: BII). The AAD also recommends against elimination diets based on the presence of AD or a suspicious history alone. However, in the event of a consistent correlation of symptoms and intake of a specific food, a 4- to 6-week dietary elimination trial can be initiated.[4] Citing recommendations from the National Institute of Allergy and Infectious Diseases Food and Allergy Expert Panel, both guidelines suggest the consideration of limited food allergy testing in patients less than 5 years of age with refractory AD despite optimal therapeutic management and/or a clinical history of an allergic reaction immediately after specific food exposure (JTF: D; AAD: BII). Moreover, both AAD and JTF guidelines strongly support avoidance of proved IgE-mediated food allergens to prevent serious adverse reactions and potentially promote improvement in AD severity (AAD: AI).

The role of aeroallergens in AD disease severity remains highly controversial. Both groups acknowledge the increased prevalence of aeroallergens in patients with AD, especially older children and adolescents. The JTF guidelines strongly recommend minimizing exposure to aeroallergens (ie, animal dander, house dust mites, and pollens), especially house dust mites, in patients with AD (JTF: A). They additionally suggest the use of house dust mite mattress and pillow covers based on multiple studies highlighting their successful reduction in house dust mite sensitization levels (JTF: A). Although the AAD acknowledges this reduction in house dust mite sensitization, they emphasize the limited and controversial evidence of mattress and pillow covers in reducing AD severity.[4] In fact, the AAD advises against routine use of house dust mite covers in patients with AD without proved allergen sensitization (AAD: BII). This group acknowledges the potential benefit of mattress and pillow covers in patients with proved dust mite sensitivity refractory to optimal AD management, citing greater clinical improvement in this subset of patients in one clinical investigation (AAD: BII).

Patch testing for allergic contact dermatitis

Although minimally addressed by the JTF group, the AAD endorses consideration of patch testing in patients with clinical assessments suspicious for allergic contact dermatitis (AAD: BII). Dermatitis characterized by atypical distribution or lesion morphology (ie, facial or periorbital, flexural neck, and dorsal hands) or exacerbated by moisturizers or topical medications might warrant referral for patch testing. The AAD guidelines highlight additional indications for patch testing, including recalcitrant AD, negative family history of atopy, and/or an unexplained increase in disease severity. A diagnosis of allergic contact dermatitis is further contingent on improvement in AD severity with allergen elimination.

Allergy immunotherapy

The JTF guidelines assess allergen-specific immunotherapy, suggesting that a clinician might consider immunotherapy in selected patients with aeroallergen sensitivity (JTF: B).[5] They base this recommendation on its usefulness in patients with AD associated with aeroallergen. In contrast, the AAD guidelines discuss the literature for both sublingual and injection immunotherapy but conclude that the data do not support recommendation for use at this time.[4]

EMERGING FOOD ALLERGY GUIDELINES

Recently, a subset of infants and children has been distinguished as potential beneficiaries of the early introduction of specific foods, potentially thwarting the subsequent development of food allergy. The Learning Early about Peanut Allergy trial, which was published in 2015, represents the first large-scale clinical investigation of allergen prevention by proactive early allergen introduction. The results of this groundbreaking investigation reveal an inverse correlation between early peanut introduction and subsequent development of peanut allergy (Learning Early about Peanut Allergy study). The promising results of this study provided the basis for the novel 2015 interim guidelines for the prevention of peanut allergy in the United States. To proactively reduce the risk of peanut allergy development, this addendum endorses the early introduction of age-appropriate peanut-containing foods in infants with severe eczema, egg allergy, or both as early as 4 to 6 months of age and delineates suggested methods for assessment of peanut sensitization before challenge.[6,7]

TABLE V. Comparing and contrasting JTF and AAD guidelines on select treatment methods for AD

	JTF	Similarities	AAD	
Moisturizers		Moisturizers are front-line therapy for both acute and proactive treatment (JTF: D; AAD: AI) Recommend application of moisturizers after bathing (JTF: D; AAD: BII)	Discusses PEDs that have not shown superiority over other moisturizers	Moisturizers
TCS		First-line therapy when nonpharmacologic interventions have failed (JTF: A; AAD: AI) Advocate for caution regarding TCS use in thinned-skin areas (JTF: D; AAD: AI & BIII)	Emphasis placed on avoiding undertreatment and recognizing "steroid phobia"	TCS
TCI		TCI are effective, steroid-sparing agents used in acute and maintenance therapy of AD (JTF: A; AAD: AI) Discuss need for consideration of cutaneous side effects with use (JTF: A; AAD: BII) Recommend counseling on black-box warning, although a causal relationship has not been identified (AAD: CIII)	Recommend preceding use of TCS to lessen severity of cutaneous reactions (BII) Recommend against routine monitoring of TCI blood levels	TCI
Topical coal tar	Consider use when AD involves scalp	Generally not recommended for treatment of AD		Topical coal tar
Topical antimicrobials/ antiseptics	Suggest topical antiseptic preparations in conjunction with topical anti-inflammatory therapy in infection-prone patients	Consideration of antimicrobial bleach baths (0.005% sodium hypochlorite) twice weekly in patients prone to skin infections (JTF: A; AAD: BII)	Generally not recommended with the exception of bleach baths with intranasal mupirocin (AI)	Topical antimicrobials/ antiseptics
Topical antihistamines			Generally not recommended	Topical antihistamines
Phototherapy		Recommend for recalcitrant AD or after failure of first-line treatment with topical agents (JTF: A; AAD: BII) Indicate preference toward narrow-band UVB	Outlines specifics of therapy options and dosing Emphasis placed on use with standard topical therapy	Phototherapy
Systemic immunosuppressants	Comparable efficacy among all immunomodulators, although inconsistent evidence supporting IFN-γ exists	Recommend these agents in a subset of patients with severe AD refractory to topical treatments and phototherapy (JTF: A; AAD: BI, BII, CIII)	Cyclosporine, azathioprine, and methotrexate should be considered before MMF and IFN-γ	Systemic immunosuppressants
Systemic corticosteroids		Short courses can lead to atopic flares after discontinuation	Generally should be avoided given the risk/benefit ratio unless in cases with acute severe exacerbations and as a bridge therapy to other systemic treatments (BII)	Systemic corticosteroids
Systemic antimicrobials	Use of systemic antibiotics in noninfected patients not discussed Consider workup for fungal infection (*Malassezia* species) (C)	Recommend use of systemic antibiotics in patients with evidence of infection *(S aureus)* (AAD: A) Recommend systemic antivirals for patients with signs and symptoms consistent with eczema herpeticum (JTF: B; AAD CII)	Recommends against use of systemic antibiotics in noninfected patients (BII)	Systemic antimicrobials

(Continued)

TABLE V. *(Continued)*

	JTF	Similarities	AAD	
Systemic antihistamines		Endorses short-term antihistamine use in patients with sleep disturbance secondary to pruritus	Recommends against use of nonsedating antihistamines in the absence of urticaria or rhinoconjunctivitis (AII)	Systemic antihistamines
Vitamin D therapy	Consider vitamin D therapy in the treatment of AD (B)			Vitamin D
Cleansers, detergents, and clothing modifications	Endorses liquid over powder detergent and suggests a second rinse cycle to remove residual detergents from new clothing (B)		Cites insufficient evidence for specific recommendations	Cleansers and detergents
Irritant avoidance	Endorse temperature and humidity control, avoidance of irritating sunscreens, and avoidance of specific athletics (D)	Recommend avoidance of common triggers (ie, acids, bleaches, fragrances, and wool) (JTF: B; AAD: CIII)		Irritant avoidance
Bathing practices	Supports bathing additives for symptomatic relief (D)	Encourage bathing in warm water and subsequently applying emollients (JTF: D; AAD: CIII) Recommend limited use of nonsoap or mild soap cleansers	Cites lack of clinical evidence to support the use of bathing additives (CIII)	Bathing practices
Wet wrap therapy	Cautions against overuse of wet wrap therapy	Suggest wet wrap therapy in conjunction with TCS in recalcitrant patients (JTF: A; AAD: BII)		Wet wrap therapy
Food allergy testing	Recommend IgE antibody testing as initial diagnostic test	Advise against allergy testing independent of supportive clinical history (JTF: B; AAD: BII) Suggest consideration of limited food allergy testing in patients <5 y old with refractory AD and/or suggestive clinical history (JTF: D; AAD: BII) Caution against broad food-elimination diets based solely on positive IgE test result (low specificity)	Supports 4- to 6-wk dietary elimination of suspected food allergen before formal allergy testing	Food allergy testing
Aeroallergens	Recommend minimizing exposure to aeroallergens, particularly house dust mites, and recommend house dust mite mattress and pillow covers (A)		Advises against routine use of house dust mite covers in subject without true dust mite allergy and recalcitrant disease (BII)	Aeroallergens
Patch testing	Minimally discussed by the JTF		Recommend consideration of testing in clinical presentations suspicious for ACD (BII)	Patch testing

PEDs, Prescription emollient devices.

Quality of life and disease effect

AD has a profound effect on the quality of life of patients and their families. Both guidelines recommend the clinician assess for severity of pruritus, sleep difficulty, and effect on daily activity of affected patients and their caregivers. The JTF also emphasizes the effect on family quality of life and suggests psychoeducation regarding strategies to minimize patients' pruritus and increase adherence to skin care regimens.[5] AAD guidelines discuss various quality-of-life measurement scales that have been implicated in clinical trials but not generally used in clinical practice.[1]

Educational interventions

Both guidelines detail evidence supporting the importance of education to patients and their caregivers regarding disease mechanisms and course and skin care techniques (JTF: D; AAD: AI). Both guidelines advocate for multidisciplinary training programs in addition to video interventions as a useful adjunct. Written information, including detailed action plans and methods of environmental control, has been encouraged by both the AAD and JTF.[4,5]

Consultations and referrals

A multidisciplinary team, including the AD specialist (pediatrician, allergist, or dermatologist or a combination of the aforementioned), psychotherapist or psychologist, and nurses, are often used in the management of AD. The JTF guidelines recommend referral to an AD specialist (1) when the patient has severe disease with multiple comorbidities as a result, (2) for identification of allergic triggers, (3) when the diagnosis of AD is questioned, and (4) for recommendations for alternative therapies.[5]

Limitations of current guidelines

As mentioned above, the previous JTF and AAD guidelines on AD were both published in 2004. Thus the current guidelines attempt to synthesize advances in both science and therapeutics spanning more than 10 years. Ideally, guidelines need to be able to incorporate key advances in our understanding of disease processes or therapeutic breakthroughs that affect patient care in a timely manner. In addition, guidelines should help clinicians by identifying appropriate patients for specific therapies and providing critical details, such as how much medication to use and for how long and the next steps. Although specialists in allergy and dermatology might be able to read through the extensive guidelines and supporting references, primary care clinicians, who take care of the majority of patients with mild-to-moderate AD, need streamlined guidelines that can be incorporated into their busy practices.[8]

CONCLUSIONS

AD is a common skin condition frequently encountered by primary care physicians, dermatologists, and allergists. This review facilitated integration of the AAAAI/ACAAI JTF 2012 AD Practice Parameter and 2014 AAD guidelines to highlight the basic principles of AD management and discuss therapies and management of AD from distinct perspectives of the allergist and dermatologist. Several key similarities and differences have been annotated in Table V. Although both guidelines provided comparable recommendations in discussions of first-line and topical treatments, their recommendations diverged on systemic treatments, adjunctive therapies, and preventative measures. This incongruence in recommendations is largely because of (1) inconsistent evidence from different sources in the literature and (2) bias reflective of each AD expert's training focus. Although these guidelines have several limitations noted above, they serve as a platform to enhance clinician awareness and knowledge of the respective clinical practice parameters to tailor a patient's treatment and management accordingly.

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