

Consensus Conference on Pediatric Atopic Dermatitis*

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C current information identifying risk factors for cutaneous diseases such as atopic dermatitis in childhood is limited. Cutaneous disease prevention programs and health promotion strategies in children and adolescents have not been addressed in a concerted way by government agencies or relevant medical societies. Disease-based interventions often emphasize adult disease, but many diseases may be prevented or minimized by intervention during childhood years. Research may be more effectively coordinated and carried out with the establishment of consensus on how best to diagnose and treat diseases such as atopic dermatitis (AD) in children and identify the most critical issues related to this condition.

A consensus conference was convened by the American Academy of Dermatology on January 19-21, 2001 to (1) define the core dermatologic issues related to AD in children and adolescents that can influence their dermatologic health over a lifetime and (2) prioritize future research efforts on epidemiology, pathogenesis, prevention, and management of AD. The following focus questions were addressed and responses/recommendations were developed:

SECTION I: EPIDEMIOLOGY OF AD

- I. Are there adequate, reliable, pathogenetic, and other defining features of atopic dermatitis (AD)?

AD is best viewed as a syndrome. Clinical findings that define this syndrome and clinical criteria of AD include the following:

- A. Essential features (must be present)
 1. Pruritus
 2. Eczema (acute, subacute, chronic)
 - a. Typical morphology and age-specific patterns*
 - b. Chronic or relapsing history
- B. Important features (seen in most cases, adding support to the diagnosis)
 1. Early age at onset
 2. Atopy
 - a. Personal and/or family history
 - b. IgE reactivity
 3. Xerosis
- C. Associated features (these clinical associations help to suggest the diagnosis of AD but are too nonspecific to be used for defining or detecting AD for research or epidemiologic studies)
 1. Atypical vascular responses (eg, facial pallor, white dermographism, delayed blanch response)
 2. Keratosis pilaris/hyperlinear palms/ichthyosis
 3. Ocular/periorbital changes
 4. Other regional findings (eg, perioral changes/periauricular lesions)
 5. Perifollicular accentuation/lichenification/prurigo lesions

Exclusionary conditions: It should be noted that a diagnosis of AD depends on excluding conditions such as scabies, seborrheic dermatitis, allergic contact dermatitis, ichthyoses, cutaneous lymphoma, psoriasis, and immune deficiency diseases.

- II. Are the incidence and prevalence of atopic dermatitis in children increasing in North America? We have minimal data that directly measure the prevalence and incidence in the United States, except for the published work by the Portland group (Laughter D, Istvan JA, Tofte SJ, Hanifin JM. The

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*Patterns include (1) facial, neck, and extensor involvement in infants and children, (2) current or prior flexural lesions in any age group, (3) sparing of groin and axillary regions.

prevalence of atopic dermatitis in Oregon school-children. *J Am Acad Dermatol* 2000;43:649-55).

There is great interest in the natural history/remission rates to help develop an ecologic approach to understanding of the etiology of AD. Prevalence, remission rate/cure/ age at onset, population differences, and ethnic/environmental/regional differences all need to be addressed. Ideally, a birth cohort conducted longitudinally at several centers would provide the needed information. The data collected should be focused on assisting practitioners in making rational decisions about allocation of limited resources in their practices.

There is limited interest in more studies to precisely demonstrate the increasing incidence/prevalence of AD in the United States. Trends are probably sufficient to direct future policy decisions and to help direct research dollar allocations.

School-based studies to measure point or lifetime prevalence are another option to collected needed information.

Pharmaceutical companies might be able to fund such studies. The Health Care Financing Administration, National Health and Nutrition Examination Survey, or other federal agencies that administer Current Procedural Terminology codes (691.8) may be in the best position to assess the population receiving reimbursable services.

III. Are there definable population differences?

Data are scanty about definable population differences, at least in North America. Birth cohort data and other ethnographic information are needed. Studies are available that show racial predilections, but they are not North American-based and thus of limited value. It does appear that the highly industrialized nations are being disproportionately affected by AD because of the rising prevalence of atopy in these geographic areas. A published textbook on AD provides adequate references concerning this question (Williams HC. *Atopic dermatitis*. London (UK): Cambridge University Press; 2000).

IV. What is the impact of AD on individuals, families, and society?

One study on economic impact has been published by Lapidus et al (Lapidus CS, Schwarz DF, Honig PJ. *Atopic dermatitis in children: who cares? Who pays?* *J Am Acad Dermatol* 1993;38:699-703). It primarily addressed the costs of treatment, rather than costs to the families. More recently, the cost of third-party payors (public and private) has been examined; this study included data on comorbidities as well as dermatitis itself (Ellis CN, Drake LA, Prendergast MM, Abramovits W, Boguniewicz M, Daniel CR, et

al. Third party payor cost of atopic dermatitis and eczema in the United States. *J Am Acad Dermatol* 2002;46:361-70.) Future studies should examine the costs of atopic dermatitis at the family level more broadly, including the loss of work income. Age- and disease-specific quality-of-life (QOL) studies are badly needed.

The emotional impact of AD needs better measurement, especially regarding intimacy and interpersonal relationships. Anecdotally, the "no-sex-syndrome" appears to be a real phenomenon among families with young children with severe AD who frequently deprive the parents of the needed privacy for intimacy needed to preserve the essential structure of the family. However, we know of no studies that directly measure this. School performance and other indicators of well-being among children are also lacking. Such studies will help guide the practitioner's treatment plans and will improve the likelihood that compliance with prescribed therapies will be achieved.

Improved self-assessment instruments or QOL tools to help practitioners gauge the impact of AD on children and adolescent patients are also needed to improve treatment outcomes.

V. What is the rate of remission/cure among children and adolescents?

Remission should be defined in both objective, physiologic terms as well as with the use of subjective parameters. Objective parameters that may be useful include severity scores, transepidermal water loss, tape stripping, and other objective tools. Clinical criteria such as extent of excoriation and resolution of acute, subacute, and chronic AD should also be developed. Associated extracutaneous signs and psychosocial outcomes should also be included in measures of remission/cure.

Most studies looking at age at onset and remission are poorly designed or have other methodological problems. There are no well-controlled trials designed to measure remission. All remission rates we can now measure will be essentially measuring past treatments. Remission cannot be measured accurately until there is general agreement about defining features of AD and the clinical tools to be used to define AD (see Section I. I. above).

VI. What are the currently used rating or classifying systems for measuring the severity of clinical symptoms? How useful are they? Should a uniform system be recommended for general use? The Eczema Area and Severity Index (EASI) and Scoring Atopic Dermatitis (SCORAD) systems are the two systems that appear to be the most reliable and

user-friendly and share the advantage that they are most commonly used in contemporary clinical trials. Both systems are useful because they generate quantifiable data that are amenable to analysis. The EASI generates a composite score, whereas the SCORAD system picks a specific area of the body to be assessed. Both systems have merit, are reasonably reproducible, and should be viewed as complementary methods to gauge efficacy. Any scoring system that is chosen should address QOL issues as well as purely clinical criteria (see IV. above). Innovative new approaches such as quantitative digital photography and physical instrumentation are also worthy of further examination.

The differences between surface to body ratios among infants and adults need to be accounted for, and there is also a need to modify evaluation criteria to readily permit corrections for body surface area age-dependent differences.

Persons contemplating adoption of a rating system are advised that a useful Web site can be found at <http://www.scorad.sante.univ-nantes.fr>. This site also includes a useful training module.

SECTION II. PATHOGENESIS OF ATOPIC DERMATITIS

I. What are the immunologic, biochemical, and other pathogenic mechanisms of AD?

The pathogenesis of AD is a complex interplay of numerous elements including immune, genetic, metabolic, infectious, and neuroendocrine factors, and their interaction with the environment. Even an introductory discussion of this topic is beyond the scope of this article. Factors that are considered the most important or newer concepts that have the potential to be important contributors to the understanding of pathogenesis of AD are outlined below.

A. Immunologic abnormalities

That immune alterations are involved in the pathogenesis of AD is virtually universally accepted. There remains considerable controversy as to cause and effect, among immunologic abnormalities, as well as between immune abnormalities and other findings seen in patients with AD. Immunologic abnormalities associated with AD appear to involve the following:

1. IgE (total, specific)
2. Cytokine balance
3. Fcε receptors
4. Hyperstimulatory antigen-presenting cells (Langerhans cells, B cells, DC-2)
5. Eosinophilia
6. Mast cell factors
7. Basophil hyper-releasability

8. Decreased cell-mediated immunity
9. Anti-IgE antibodies
10. Autoimmunity
11. Superantigens
12. Cutaneous lymphocyte antigen-bearing T cells and antigen responsiveness
13. Decreased apoptosis of T cells/eosinophils in the skin
14. Chemokines

B. Phosphodiesterase abnormalities and cyclic nucleotide dysregulation

Elevated phosphodiesterase (PDE) activity in AD monocytes and the resulting disordered cyclic nucleotide regulation is an early change in AD. High-activity PDE isoforms in atopic immune and inflammatory cells reduce cyclic adenosine monophosphate functional modulation and can account for the increased inflammatory reactivity in AD and for many of the immune defects.

C. Neural mediators

Neural influences on AD may affect pathogenesis, including not only the pruritus sensation of affected individuals but also through interactions with immune and other elements as well. Mediators may include the following:

1. Activation of proteinase-activated receptor-2
2. α-Melanocyte-stimulating hormone
3. Substance P
4. Acetylcholine
5. Calcitonin gene-related peptide
6. Neurokinin A

D. Effector cells

Effector cells important in pathogenesis include the following:

1. Eosinophils
2. Mast cells
3. Basophils
4. T cells

E. Barrier function

The skin in AD has abnormalities in barrier function. The exact relationship between barrier function, immunologic and neural mediators of disease remains to be clarified. Barrier function abnormalities associated with AD include the following:

1. Increased transepidermal water loss
2. Decreased stratum corneum moisture content
3. Increased permeability to hydrophilic substances
4. Decreased lipids/ceramides
5. Decreased barrier to infectious agents
6. Decreased endogenous humectants
7. Likely increased epicutaneous antigen absorption

F. Proposed research goals for immunologic and pathogenetic mechanisms

Some areas of research are proposed to be of high interest in advancing our understanding of pathologic mechanisms of AD.

1. Identify genes predisposing to AD. Identify genes, if any, that contribute to AD specifically versus atopy in general.
2. Define relationships among identified abnormalities (genetics/immune mediators/barrier function and dysfunction/effector cells).
3. Identify necessary (but not necessarily sufficient) abnormalities—phenomena versus epiphenomena).
4. The mechanisms of disease amelioration in most patients warrant study: What is the influence of therapy on disease course? What are the rates and factors involved in spontaneous remission or cure? What factors may prevent active dermatitis in affected patients or prevent the initiation of AD?
5. The balance of immune regulation should be studied. An analogy to guide research is posed as a question: Is AD a disease of “too much gas” or “faulty brakes”? (ie, a disease of abnormal initiation or abnormal down-regulation?).
6. Better in vitro and in vivo model systems should be developed, including specific models that best characterize pediatric AD.

II. What are the genetic influences on the development of AD?

Population-based studies of genetic determination of traits may be very difficult to perform, but are potentially very important. An approach that studies informative kindreds with early-onset AD may be useful; 90% of patients will develop AD by 5 years of age; 95% by 15 years of age. Multigenerational families may be needed.

A. There are many mediators, enzymes, or potential mediators of AD that serve as potential candidates for genetic influences in AD, including the following:

1. Interleukin 4 (IL-4)
2. IL-4R
3. FcεRI
4. IL-13R
5. PDE4
6. ?CD14 (asthma)
7. ?Lipopolysaccharide receptor
8. ?Stat6 (pro)

9. ?Bcl6 (down-regulate)

B. The causes of several genetic syndromes may relate to the pathogenesis of AD and provide useful model systems to compare and contrast with AD, including the following:

1. WASP (associated with Wiskott-Aldrich syndrome)
2. SPINK5 (associated with Netherton syndrome)
3. Hyper-IgE syndrome

C. A set of factors may mediate the provocation of AD (in susceptible individuals). It is unknown whether susceptibility to these influences is genetically determined. This set may include the following:

1. Microbial colonization and infection, including viruses and bacteria
2. Stress
3. Barrier disruption
4. Environmental exposure
5. Allergens: contactant, inhalant, ingestant
6. Chemical irritants (eg, soap)
7. Sweating
8. Pollutants
9. Sensory irritants (eg, wool)
10. Breastfeeding/gastrointestinal exposures
11. Maternal ingestants (eg, foods/drugs)
12. Maternal infection

SECTION III: ATOPY, ALLERGEN EXPOSURE, AND ATOPIC DERMATITIS DEVELOPMENT

I. What is the influence of maternal food intake and early infant feeding patterns on AD development?

The influence of maternal food intake and infant feeding patterns on AD development is a complex issue. Food allergies are more common in children with established AD than in nonatopic children, and IgE-mediated sensitization is demonstrable in the majority of, but not all, AD patients by a variety of methods including skin prick tests, specific IgE antibodies and allergen patch tests. Prospective studies have displayed a concordance of sensitization with food and aeroallergens with the development of AD, but no specific cause-and-effect relationship has been convincingly displayed. There is uncertainty as to the ways in which exposure, or the timing of exposure of allergens (in utero and beyond), may influence the development of AD, and there is uncertainty about the utility of allergen avoidance in unselected populations of children with AD.

Study data are supportive of the benefits of maternal avoidance of major food allergens in families

at high risk for atopy. However, avoidance of major food allergens is difficult; the hardship on a family of pursuing avoidance should be a significant factor when considering this intervention. A highly motivated mother might pursue this avenue.

Data are inconsistent regarding the impact of infant feeding patterns on the development of AD, and there is a lack of consensus about feeding recommendations.

The influence of maternal factors on the ultimate development of AD should be investigated. The risks and benefits of maternal dietary avoidance should be studied. In families at high risk for atopy, investigation should be pursued for means to avoid development of disease in offspring.

II. Do exposures to infections in early childhood mediate AD development?

The lack or scarcity of intense systemic infections in early life has been theorized to increase the susceptibility of being sensitized to otherwise harmless antigens in later life. Prospective data in childhood are limited. Insufficient information exists as to whether immunizations have a positive or negative effect on the development of AD.

Studies should be performed to assess the relationship of AD development to the occurrence of early infections. Studies looking at the influence of gut colonization, skin colonization, and maternal infections on the development of AD are also indicated.

III. What is the influence of allergen sensitivity on the acquisition and duration of AD in children, and how is this best studied?

The influence of allergen exposure on the acquisition of AD is uncertain and controversial. Allergen sensitivity is recognized as a trigger to AD in a subset of children with AD. Many patients with AD have food allergies. Food allergies in patients with AD may induce eczematous dermatitis in some patients, whereas in others urticarial reactions, contact urticaria, or other noncutaneous symptom complexes may be elicited.

In high-risk families, further study of the effect of environmental and allergic influences on AD is warranted. The influence of allergen sensitivity on the acquisition and duration of AD in children should be further investigated.

Recommendations:

- A prospective study correlating the longitudinal history of AD with allergen sensitization and the respective laboratory data (eg, CAP-radioallergosorbent test) is recommended.

- The influence of specific allergen avoidance should also be studied.
- Investigation of factors that identify patients who would benefit from study of allergic triggers should be pursued.
- The influence of early initiation and control of AD on disease control and the course of atopy should be studied.

SECTION IV: TREATMENT

- I. What is the evidence basis for standard treatments of AD, including topical corticosteroids, systemic corticosteroids, systemic antihistamines, allergen avoidance, and bathing regimens? How safe are topical corticosteroids when used over a long period?

The evidence supporting the efficacy and safety of standard treatments of AD varies greatly. A useful summary has been compiled by the Cochraine group (Hoare C, LiWan Po A, Williams H. *Health Technol Assess* 2000;4:1-191; available at <http://www.ncchata.org/htapubs.htm#437>).

A discussion of therapies and the safety and utility of topical steroids follows. The goal of treatment is control of symptoms, not cure of the disease.

A. Role of bathing

Regular bathing can hydrate the skin and debride crust. There is no evidence that bathing, when performed as outlined below, adversely affects disease.

1. Bathe once daily for several minutes in warm (not hot) water.
2. Use a moisturizing cleanser.
3. Avoid antibacterial cleansers because of the risk of inducing bacterial resistance. Products designed for infants may have no demonstrable advantage over other products.
4. After bathing, pat dry and immediately apply topical medication followed by an emollient, or emollient alone.

Recommendations: Additional study is required to determine the efficacy of various cleansers, the role of bathing as a steroid-sparing modality, and the optimal duration of bathing.

B. Role of emollients

There is evidence that emollients are effective as first-line agents in the management of AD and may be steroid sparing. Ointments and creams provide greater barrier function than lotions. Emollients should be applied frequently since their maximum duration of action is 6 hours.

Recommendations: Further study is needed to determine the mechanism of action of emollients, their optimal formulation, and the frequency and amount of application.

C. Role of general measures

There is consensus that certain general measures may be beneficial as adjuncts in the management of AD. These include the following:

1. Avoid the use of clothing that may act as an irritant (eg, wool or synthetics). Cotton clothing is preferable.
2. To the extent possible, avoid overheating and external irritants.
3. Keeping the skin covered by clothing may reduce exposure to irritants and trauma from scratching.

D. Use of topical corticosteroids

Multiple comparative trials have documented the efficacy of low- and mid-potency topical corticosteroids in the management of AD in pediatric patients. These agents are the first-line therapy for the management of disease exacerbations. In general, these agents should be applied twice daily to induce control of inflammation (some preparations may be used once daily); there is no evidence that more frequent application enhances efficacy. Once disease activity is controlled, topical corticosteroids should be used intermittently, typically twice weekly to maintain control if long-term therapy is needed to control intermittent flares. The least potent preparation that adequately controls the disease process should be selected.

The vehicle base of a corticosteroid influences potency; ointments are generally more potent than creams. Creams may contain preservatives that may induce an irritant contact dermatitis.

Once control is achieved, the topical corticosteroid should be tapered to a less potent preparation or withdrawn. When selected and used appropriately, these agents are not associated with significant adverse effects (eg, suppression of the hypothalamic-pituitary-adrenal axis or growth).

There is empiric evidence that in the management of children with severe disease, the application of a topical corticosteroid under wet wrap occlusion may be beneficial. In the presence of such occlusion, less potent corticosteroid preparations or a corticosteroid compounded in an emollient are effective and reduce the risk of local and systemic adverse reactions.

Recommendations: Additional investigation is needed to determine possible synergism between topical corticosteroids and other modalities, the ef-

ficacy and safety of alternate dosing schedules (eg, pulsed application and brief duration, high-potency therapy), and the safety of long-term corticosteroid application.

II. What are the utility and safety of new nonsteroidal immunosuppressive therapies (systemic and topical) for AD?

A. Use of topical immunomodulatory agents

Immunomodulatory agents hold promise in the management of AD. Both topical tacrolimus and topical pimecrolimus have been approved for use in the treatment of AD in children. On the basis of studies involving several thousand children, there is evidence that these agents are effective in the treatment of dermatitis.

There is consensus, however, that concern remains about potential long-term adverse effects. For this reason, tacrolimus and pimecrolimus should be used as follows:

1. Tacrolimus should be used as second-line therapy for the management of moderate or severe AD.
2. The 0.03% ointment is the approved therapy for children.
3. Tacrolimus is not approved for children younger than 2 years.
4. Tacrolimus should be employed for episodic, not prolonged continuous use until further studies on long-term safety are performed.
5. Because of concerns regarding the potential for the development of cutaneous malignancy, patients being treated with tacrolimus and their families should be counseled about the use of appropriate sun protection, including the application of sunscreen.
6. Pimecrolimus 1% cream should be used as a second-line therapy for the management of mild to moderate AD.
7. Pimecrolimus is not approved for use in children younger than 2 years.
8. Pimecrolimus should be employed for episodic, not prolonged continuous use until further studies on long-term safety are performed.
9. Because of concerns regarding the potential for the development of cutaneous malignancy, patients being treated with pimecrolimus and their families should be counseled about the use of appropriate sun protection, including the application of sunscreen.

Recommendations: Further studies are required to determine systemic absorption of immunomodulatory agents particularly in infants and young children, their long-term safety, and the efficacy and safety of combined topical immunomodulatory corticosteroid therapy.

III. What is the role of other systemic therapy, psychosocial interventions, and adjuvant therapies for AD?

A. Use of systemic steroids

The use of systemic corticosteroids may be associated with significant adverse effects (eg, suppression of the hypothalamic-pituitary-adrenal axis or growth) and is not routinely recommended.

B. Use of antihistamines

Data regarding the efficacy of oral antihistamines in the management of AD are conflicting. However, these agents may provide relief from pruritus (through mechanisms unknown) and facilitate sleep. To avoid daytime sedation, it is prudent to administer sedating agents at bedtime.

Recommendations: Well-designed trials of sedating and nonsedating antihistamines as adjuncts to the management of pediatric AD may be useful. Issues that merit investigation are the comparative efficacy, safety, and optimal dosing schedule of various agents.

C. Use of antibiotics

The use of an oral or topical antibiotic active against *Staphylococcus aureus* and *Streptococcus pyogenes* is indicated for patients with evidence of secondary bacterial infection (eg, erythema, crusting, pustule formation). Long-term use of oral or topical antibiotics for other indications (eg, management of disease recalcitrant to standard therapy) is not recommended.

Recommendations: Well-designed trials of topical and systemic antibiotics as adjuncts to the management of AD are required. Issues that merit investigation are the comparative efficacy of various topical agents, the comparative efficacy of topical and systemic agents, and the efficacy and safety of these agents for other indications (eg, the management of disease recalcitrant to standard therapy).

D. Use of environmental modification

There is evidence that a small proportion of children and adolescents with severe, recalcitrant AD experience clinical improvement after the institution of measures to reduce exposure to house dust mites

(eg, enclosure of mattresses, frequent vacuuming). The role of testing for house dust mite sensitivity has not yet been defined.

There is evidence that effective management of other environmental allergies, including environmental modification and avoidance of contact allergens, may ameliorate the symptoms of AD in selected patients.

Recommendations: Further studies are needed to clarify the role of environmental allergens in the causation or exacerbation of AD, the role of testing for house dust mite sensitivity, the adjunctive efficacy of house dust mite or other allergen avoidance, and the value of patch testing.

E. The psychosocial impact of AD

There is evidence that AD may have a significant negative psychosocial impact on the patient and family. There is evidence that children with active or inactive disease have disturbed sleep that adversely affects other family members and may disrupt activities of daily living (eg, effective learning). At present, the identification and management of these psychosocial consequences are inadequate.

Recommendations: Dermatologists should become familiar with and disseminate to patients and families information about local and national resources to assist in coping with the disease. Further study of the prevalence of psychosocial disturbances and strategies for effective management are required.

F. Phototherapy

There is evidence supporting the efficacy of ultraviolet light therapy in adults with AD. However, data regarding safety and efficacy of this modality in the pediatric population are minimal. Further study is indicated.

G. Tar/coal tar solutions

In selected patients, tar preparations, administered topically or in the bathtub, may be effective as described by experts, but this is not evidence based.

H. Systemic immunomodulators

Oral immunomodulators have been used predominantly for severe AD, including cyclosporine, azathioprine, and mycophenolate, among others. The literature concerning their utility and safety in pediatrics is limited, and these were not discussed by the Consensus Committee. Further study may be indicated.

I. Use of alternative or complementary therapies

There is evidence that patients or their families use alternative or complementary therapies. In many cases, the safety and efficacy of these measures are not known. There are no data supporting the efficacy of Chinese herbal medicine, and concerns have been raised about hepatotoxicity associated with its use. Alternative and complementary therapies for AD should be subject to controlled trials.

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