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Estimated Time to Complete Activity: 0.25 hours

Participants should read the activity information, review the activity in its entirety, and complete the online post-test and evaluation. Upon completing this activity as designed and achieving a passing score on the post-test, you will be directed to a Web page that will allow you to receive your certificate of credit via e-mail or you may print it out at that time.

The online post-test and evaluation can be accessed at <https://tinyurl.com/ADInfoED>

Inquiries about continuing medical education (CME) accreditation may be directed to Postgraduate Institute for Medicine (PIM) at www.pimed.com or (303) 799-1930.

Joint Provider Statement

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Physician Continuing Medical Education

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Continuing Nursing Education: The maximum number of hours awarded for this Continuing Nursing Education activity is 0.2 contact hours.

Target Audience

This activity is designed for dermatologists, pediatricians, pediatric dermatologists, allergists, physician assistants, nurse practitioners, and nurses in dermatology and allergy actively treating patients with atopic dermatitis (AD).

Educational Needs

There is a wide variation in knowledge, competence, and practice among clinicians regarding the management of AD, especially in light of recent scientific advances and newly available therapies. To provide optimal care, specialists need foundational education regarding the etiology and pathophysiology of AD. To accurately assess their patients and decide on a course of treatment, they need awareness of available validated tools. They would further benefit from case-based AD education to facilitate application of the foundational learning into clinical practice.

Learning Objectives

At the conclusion of this program, participants should be better able to:

- Describe the current theories of the pathophysiology of AD
- Identify and apply the diagnostic criteria for AD
- Employ strategies to manage patients with mild-to-moderate and moderate-to-severe AD

This activity is jointly provided by:



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A Current Practice Snapshot of Atopic Dermatitis Management: Where We Are and Where We Need to Be

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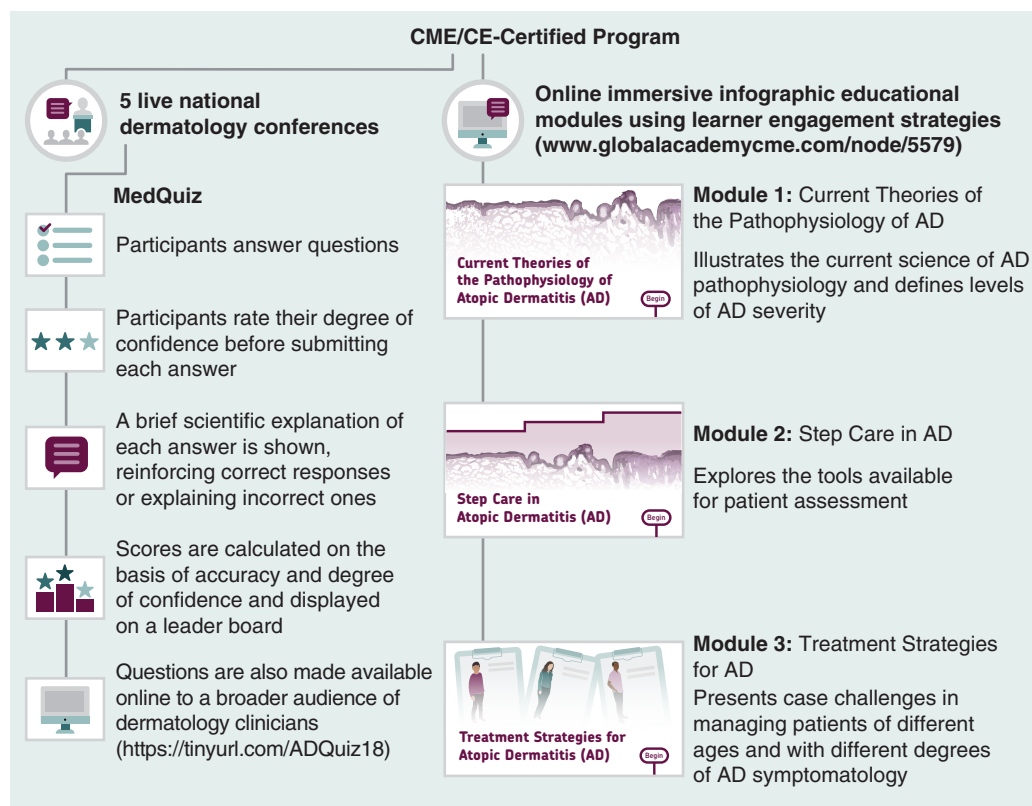
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Introduction

The evolving understanding of the pathophysiology of atopic dermatitis (AD) has led to the recent approval of new therapies with novel mechanisms of action, including crisaborole, a topical agent for mild-to-moderate AD, and dupilumab, a systemic agent for moderate-to-severe disease. Despite these advances, however, many clinicians may lack awareness of how to accurately assess disease severity and, subsequently, how to design an appropriate therapeutic strategy for the individual patient when the disease progresses. As an expert panel of the International Eczema Council (IEC) recently observed, “[G]uidelines for decision making about advancement to systemic therapy are lacking.”¹ To address these gaps in knowledge and practice—and to explore “where we are” with regard to contemporary AD management—a multicomponent CME-certified program was launched in the fall of 2018.



This supplement outlines the interim findings from pretest and post-test questions of the above educational activities and highlights the following:



Where we are in current practice for atopic dermatitis management

Data from key questions asked during the activities

Where we need to be for optimal atopic dermatitis management

Information on what clinicians should know and how they should apply that knowledge



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Faculty

Linda F. Stein Gold, MD, Speaker's Bureau: Allergan, Celgene, Galderma, LEO, Novartis, Pfizer, Valeant; **Consultant:** AbbVie, Allergan, Celgene, Galderma, LEO, Medimetrix, Novartis, Pfizer, Promius, Valeant; **Fees for Non-CME Services Received Directly from a Commercial Interest or Its Agent:** AbbVie, Allergan, Celgene, Dermira, Galderma, La Roche-Posay, LEO, Medimetrix, Novan, Novartis, Pfizer, Promius, Sebela, Valeant; **Contracted Research:** Allergan, Aqua, Dermira, Galderma, LEO, Pfizer, Valeant.

Luz Fonacier, MD, Consultant: Regeneron/Sanofi; **Fees for Non-CME Services Received Directly from a Commercial Interest or Its Agent:** Regeneron/Sanofi; **Contracted Research:** Pfizer, Regeneron/Sanofi, and Shire to NYU Winthrop Hospital. **Moise L. Levy, MD, Consultant:** Castle Creek Pharmaceuticals; **Contracted Research:** Amicus.

Staff and Planning Committee Members:

The PIM planners and managers have nothing to disclose. The Global Academy for Medical Education planners and managers have nothing to disclose.

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Exploring Mechanisms of Disease

Questions presented during the interactive modules and MedQuiz activity focused on learners' general understanding and knowledge of specific aspects of AD pathology.



Where we are



50% of respondents did not know that **irritants**, such as cigarette smoke, are an **undisputed trigger for AD**.



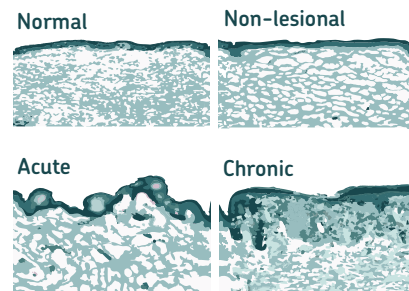
4 in 10 incorrectly believed that hygiene habits, such as regular bathing, were involved.

Where we need to be



The current model of AD posits a multifactorial pathogenesis involving both an "outside-in" and an "inside-out" process.² In this view, symptoms result from a combination of defects in the skin barrier that are exacerbated by dysregulated immune activity.³ Understanding this principle is important, since treatment for AD in the earlier stages involves topical management (including use of skin hygiene, emollients, and therapeutic lotions or ointments), while in the later stages a systemic approach may be needed to address the underlying inflammatory and allergic processes.⁴ Non-lesional AD has a greater response to irritants, including cigarette smoke.

AD Is a Progressive Disease Involving Defects in the Skin Barrier



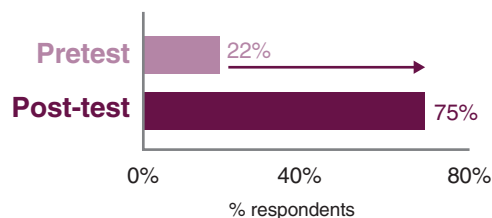
Where we are



Nearly 76% of learners reported that their understanding of the **pathophysiological mechanisms** contributing to the barrier defect in AD was only "fair" or "poor."



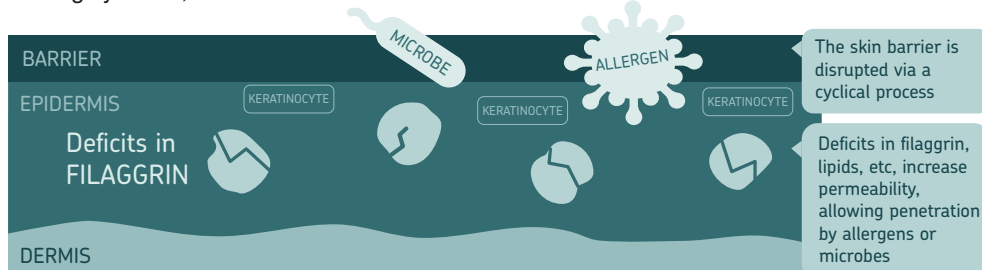
When asked about which factors contribute to the **skin barrier defect** seen in AD, the proportion of learners who identified the correct answer (**deficits in filaggrin**) rose from about 22% (pretest) to nearly 75% after exposure to the education.



Where we need to be

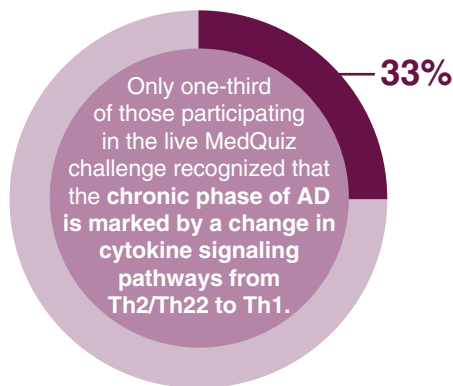


The above findings (and other similar outcomes results, not reported here) support the need for further education on AD mechanisms and the value of presenting such material in a highly visual, interactive format.





Where we are



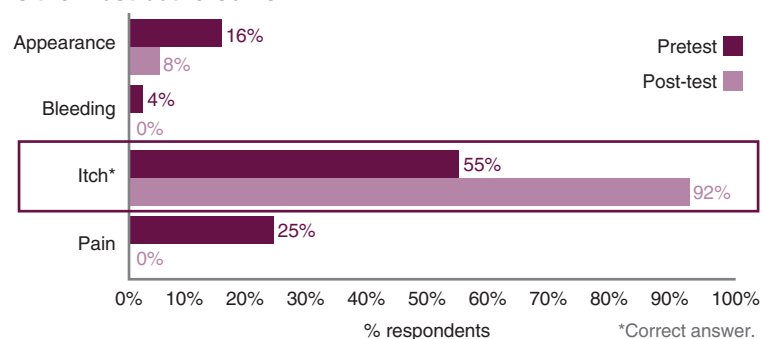
Current Concepts in Assessing AD

Learners' understanding of symptoms and comorbidities of AD was determined, along with their knowledge of tools that can be used to make such assessments.



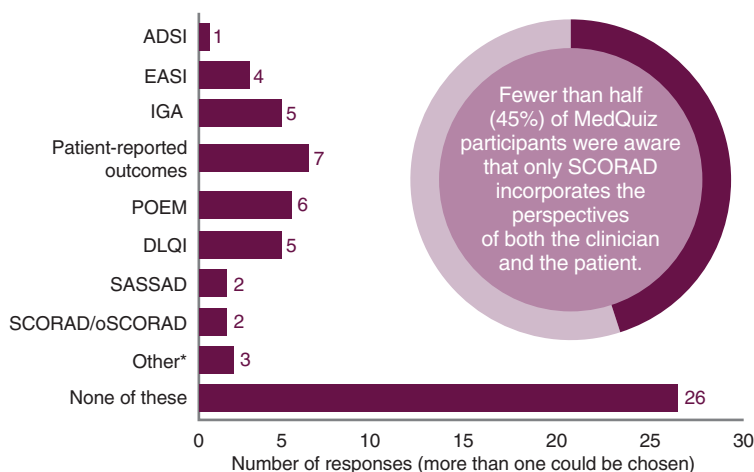
Where we are

Which of the following **symptoms** do patients with AD **typically report** is the **most bothersome**?



Where we are

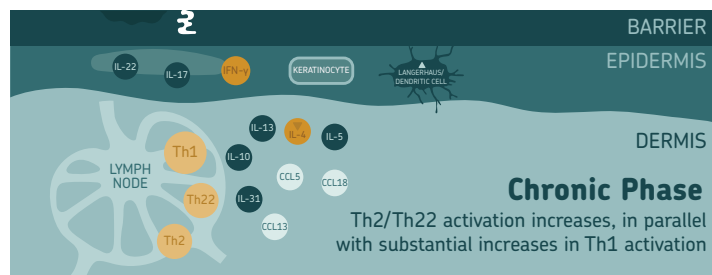
In the interactive modules, the majority of learners reported that they did not use any of the listed **assessment tools** when **clinically evaluating patients** with AD.



*Other = Body Surface Area (BSA); Clinician's Global Assessment of Severity (CGAS); Pittsburgh Sleep Quality Index (PSQI). **ADSI**, Atopic Dermatitis Severity Index; **EASI**, Eczema Area and Severity Index; **IGA**, Investigator Global Assessment; **POEM**, Patient-Oriented Eczema Measure; **SASSAD**, Six Area, Six Sign Atopic Dermatitis; **SCORAD/oSCORAD**, (Objective) Scoring Atopic Dermatitis.

Where we need to be

AD can progress through 3 phases of severity—non-lesional, acute, and chronic. The increase in severity reflects changes in the underlying immune mechanisms and dictates treatment choices.⁵



Where we need to be

The clinical presentation of AD involves the following:

Clinical Presentation:

- Pruritus
- Eczema
- Pain, bleeding, or oozing

Comorbidities:

- Sleep disturbance
- Depression
- Psychosocial challenges

Awareness of pruritus is important because treatment must be designed to address this aspect of disease to optimize outcomes and to improve patients' satisfaction with their therapy.

Where we need to be

Although most patients with AD can be managed effectively with topical agents, a significant number will require a more aggressive approach.¹ The decision to step up to systemic therapy should be made holistically, involving a mix of tools to assess both disease severity and the patient's overall quality of life. Many tools exist for evaluating these parameters, but most are intended for use in evaluating results of trials and may not be practical in the clinic. No "gold standard" tool has been identified, and only 3 have been adequately validated: EASI, SCORAD, and POEM.⁶⁻⁸ Experts recommend applying a mix of tools and strategies for assessment.

Tools

POEM

CGA

BSA

Itch Assessment

PSQI

DLQI/CDLQI

POEM is the only patient-reported outcome tool that addresses patients' concerns and that has sufficient validation for the assessment of AD

Pros

- Simple to administer
- Patient (or parent) fills it out in the waiting room before seeing the clinician
- Takes a few seconds to score

Cons

- Assesses only frequency of 7 characteristic signs and symptoms
- Does not assess extent or severity of lesions

Tool Assessment Items

Itch	Each item on the left is scored by the number of days that symptom has been present over the past week
Sleep disturbance	
Bleeding	
Weeping/oozing	
Skin cracked	
Flaking	
Dry/rough skin	

CDLQI, Children's Dermatology Life Quality Index; DLQI, Dermatology Life Quality Index; PSQI, Pittsburgh Sleep Quality Index.

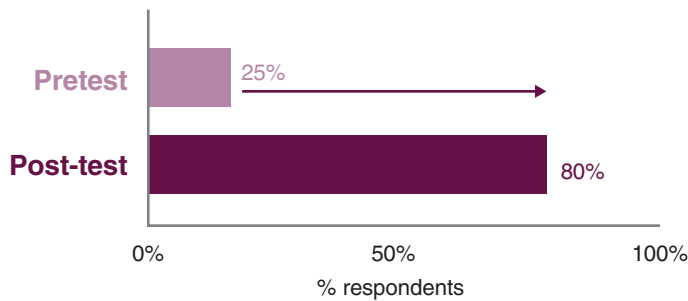
Current Concepts in Treating AD

Learners were assessed on their level of understanding of the mechanisms of action of current AD therapies as a way to emphasize the importance of selecting agents that target the pathophysiological disruptions that produce symptoms.



Where we are

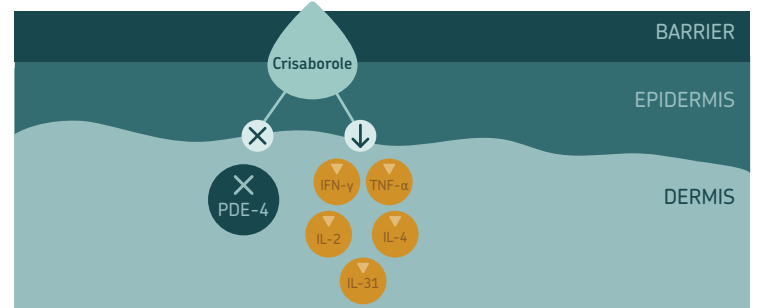
Asked which agent **targets phosphodiesterase 4 (PDE-4)** activity, only about 25% of respondents on the pretest correctly identified **crisaborole**; after the education, however, more than 80% answered correctly.



Where we need to be

Understanding the mechanism of action of available agents for managing AD will help clinicians identify appropriate treatment choices.

Crisaborole inhibits PDE-4 activity, resulting in a reduction in cytokine expression, including IFN- γ , TNF- α , IL-2, IL-4, and IL-31.⁹

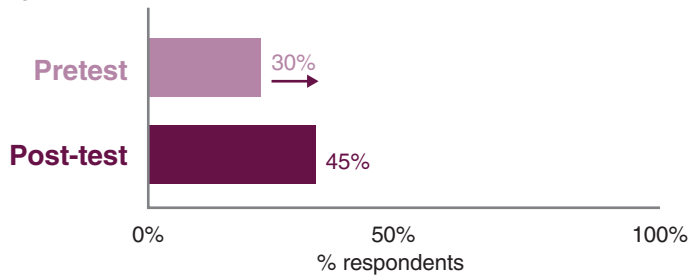


IFN, interferon; IL, interleukin; TNF, tumor necrosis factor.



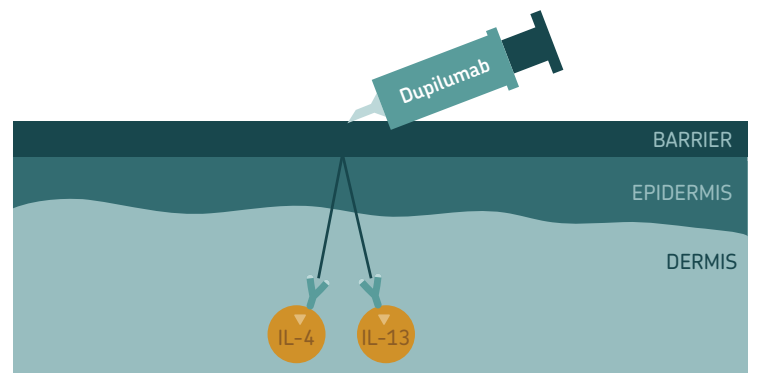
Where we are

The proportion of learners who knew that **dupilumab reduces inflammation in AD by inhibiting Th2 cytokines** also rose, from 30% on the pretest to 45% on the post-test, but such results, while encouraging, indicate there is still room to increase awareness about drug mechanisms.



Where we need to be

Dupilumab reduces inflammation in AD by inhibiting Th2 cytokines.



Summary

Atopic dermatitis is a complex disease that can arise at any age, including in very young patients, and that can persist and progress over the course of many years. Most cases are mild-to-moderate in severity and can be managed appropriately and effectively in the primary care setting. However, the underlying pathology of AD can change over time to involve different immune pathways, resulting in more severe symptoms and necessitating a reevaluation of the patient to determine whether referral for more aggressive treatment is needed. Specialists are in a position to offer stepped-up therapy that includes a systemic approach when appropriate. Education that explicates the disease process—and that challenges learners to select optimal treatment for an individual—can be effective, but the range of learner answers submitted by participants in a recent CME program series suggests that more effort is needed to close the educational gaps and improve therapeutic outcomes for patients with AD.

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