

Atopic Dermatitis Testing

Test Code #403068 Complete Atopic Dermatitis Panel

Tests Included in the Complete AD Panel (CPT Code)

403071 Atopic Dermatitis Food IgE Panel	(86003 x 6)
• Allergen-specific IgE to 6 common food triggers (Panel includes milk, egg, wheat, soy, peanut, fish)	
403069 Atopic Dermatitis Microbial Panel	(86003 x 4)
• <i>Malassezia Mix</i> —specific IgE	
• <i>Candida albicans</i> —specific IgE	
• <i>S. aureus</i> Enterotoxin A-specific IgE	
• <i>S. aureus</i> Enterotoxin B—specific IgE	
403070 Atopic Dermatitis Autoimmunity	
• Anti-IgE (83516)	
• Manganese Superoxide Dismutase-Specific IgE (86003)	
00001 Total Serum IgE	(82785)

Other immunology tests available for evaluating AD patients:

42	Eosinophil cationic protein (ECP)
2102	Soluble E-Selectin
402472	Nut Food Panel (Includes IgE to 5 nut allergens)
403100	Filaggrin ProGenotyper [®] Test

For regional aeroallergen-specific IgE tests that might be a trigger, consult the IBT Laboratories Service Directory

Clinical Utility:

Although the diagnosis of AD is primarily based on clinical criteria, the immunologic findings are useful in assessing causative agents and monitoring treatment and can direct specific therapeutic options for the patients with this disease.

Specimen Requirement:

- 5.0 mL serum for Complete AD Panel and 2.0 mL for each of the individual tests/panels.
- Ship at ambient temperature.
- Special collection protocol is required for ECP (call IBT).

Background:

Atopic Dermatitis (AD) is a chronic relapsing severely pruritic inflammatory skin disease. The pathology is driven by cellular and antibody-mediated processes. More than 80% of patients with AD have elevated total serum IgE and many show specific IgE responses to a variety of unusual allergens. The importance of IgE in AD is also supported by the observation that patients have IgG autoantibodies to IgE, IgE autoantibodies to human epithelial proteins and a high prevalence of IgE to microbial antigens.

Food Allergy & AD. In children, an IgE mediated food sensitivity is more commonly associated with AD than with adults. Approximately 40% of infants and young children with moderate to severe AD have food allergy.

Inflammation & Microbial Antigens. Fungal and bacterial infections can exacerbate AD. *Staphylococcus aureus* infection has been shown to maintain skin inflammation by secreting toxins known to act as “superantigens”. *S. aureus* is found in > 90% of AD skin lesions and most patients with AD make specific IgE to these toxins (Enterotoxin A&B). Aggressive anti-Staphylococcal antibiotics are effective in these patients. The inflammatory response to fungi, especially *Malassezia* and *Candida* species are important triggers in AD. Treatment with anti-fungal drugs provides benefit in patients with elevated IgE to these fungi.

IgE and Autoimmunity. The total serum IgE is frequently very high in AD patients, sometimes exceeding 10,000 IU/mL. A subpopulation of these patients produce autoantibodies to their own IgE and this correlates with disease severity. The activation of IgE bearing Langerhans cells and macrophages by autoantibodies to IgE contribute to skin inflammation. The majority of sera from AD patients contain IgE specific for human skin proteins (e.g. MnSOD) and this autoantibody appears to exacerbate the inflammatory process once initiated.

Other peripheral blood findings in AD

- Expansion of cells secreting Th2 cytokines
- Eosinophilia and increased ECP
- Increased spontaneous basophil histamine release
- Increased soluble E-selectin levels

References:

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2. Bunkowski R, et al. Prevalence and role of serum IgE antibodies to the *S. aureus*-derived superantigens SEA and SEB in children with atopic dermatitis. *J Allergy Clin Immunol* 1999; 103: 119-124.
3. Nissen D et al. IgE-binding components of staphylococcal enterotoxins in patients with atopic dermatitis. *Ann Allergy Asthma Immunol* 1997; 79: 403-408.
4. Paller AS. Atopic Dermatitis. In *Current therapy in allergy, immunology and rheumatology*. 1996. Fifth Edition. Edited by Lichtenstein LM and Fauci AS. Pages 83 – 86. Mosby Pubs.
5. Sicherer S and Sampson H, Food hypersensitivity and atopic dermatitis: pathophysiology, epidemiology, diagnosis and management. *J Allergy Clin Immunol* 1999; 104:S114-122.
6. McGirt LY and Beck LA. Innate immune defects in atopic dermatitis. *J Allergy Clin Immunol* 2006; 118: 202-208.
7. Adinoff A and Clark R. Atopic Dermatitis. In *Allergy, asthma, and immunology from infancy to adulthood*. 1996, Edited by Berman CW, Pearlman, DS, Shapiro, GG, and Busse, WW. Third Edition, W.B. Saunders, Philadelphia
8. Werfel T and Kapp A. Environmental and other major provocation factors in atopic dermatitis. *Allergy*1998; 53: 731-739.
9. Valenta R Natter, S, Seiberler et al. Molecular characterization of an autoantigen, Hom s 1, identified by serum IgE from atopic dermatitis patients. *J Invest Dermatol* 1998; 111: 1178-1183.

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