ALLETESS
MEDICAL LABORATORY
The first step... Alletess

CLIA #22D0080258
14 Howard Street, Rockland, MA 02370

PHONE: 800.225.5404 or 781.871.4426
FAX: 781.871.4182 | www.foodallergy.com

PATIENT INFORMATION
Sample Patient
DOB: 00/00/0000
REQUISITION ID: 000000

PROVIDER INFORMATION
Sample Provider
Street Address
City, State 00000

Common Food Allergen Panel (IgE)

SPECIMEN TYPE: Serum
COLLECTION DATE: 00/00/0000
REPORT DATE: 00/00/0000

ALLERGEN (IgE)	SCORE	CLASS
Almond	0.13	0/1
Apple	0.20	0/1
Beef	<0.10	0
Cashew	<0.10	0
Corn	<0.10	0
Egg	0.12	0/1
Milk (Cow's)	0.16	0/1
Orange	<0.10	0
Peanut	**0.53**	**1**
Salmon	<0.10	0
Sesame Seed	0.13	0/1
Shrimp	**0.47**	**1**
Soybean	**0.77**	**2**
Strawberry	<0.10	0
Wheat	0.11	0/1

IgE Ranges kUA/l
< 0.10 = 0
0.10 – 0.34 = 0/1
0.35 – 0.69 = 1
0.70 – 3.49 = 2
3.50 – 17.49 = 3
17.50 – 49.99 = 4
50.00 – 99.99 = 5
> 100 = 6

Ranges are reflective of increasing concentrations of allergen specific IgE.

- In the interpretation of some food allergen test results, cross reactivity with other homologous food and/or environmental allergens can occur. The test findings should be interpreted in the context of the clinical findings and the individual's health history.

- Diagnostic features of an IgE-mediated allergy include sensitization to a specific allergen and an individual's clinical history of allergic symptoms on exposure to that allergen. A negative immunoassay laboratory test result especially in an individual with a strongly suggestive clinical and symptomatic history does not rule out allergy and further evaluation should be considered.

ALLETESS
MEDICAL LABORATORY
The first step... Alletess

You have tested positive to *Soy*, ask your provider about ordering a *Soy Component Panel*

Soy Allergen Component testing can help determine which proteins you are sensitized to.

Knowing the proteins, or components, within each allergen that are triggering your symptoms can help guide your management plan.

Soybean consists of different types of proteins that all have different characteristics that may be associated with varying risk of causing severe allergic reactions. Some people with soybean allergy may be able to eat soybean if it is cooked, as high temperatures break down the causative proteins. For another patient, soybean should be avoided completely, as it could potentially cause a severe event, also called anaphylaxis.

Your specific risk profile depends on which proteins you are allergic to.[1]:

rGly m 4
- Usually associated with mild symptoms when ingesting mildly processed soy products, e.g., oral allergy syndrome (OAS), but also sometimes severe reactions, due to birch pollen allergy (cross reactivity).
- Sensitive to heat and digestion, cooked foods are often tolerated.

nGly m 5, nGly m 6
- Symptoms are likely caused specifically by soy.
- Usually associated with severe reactions.
- Stable to heat and digestion, both cooked and raw foods may cause symptoms.

MUXF3 (CCD)
- Positive specific IgE for soy in combination with MUXF3 CCD (Cross-reactive Carbohydrate Determinant), being the only positive component test indicates that the cause of symptoms may be something other than soy.

Please note that the test results should be interpreted by your healthcare provider, in the context of your clinical history. Final diagnosis and decision on further management is made by your healthcare provider.

Some people with soybean allergy may also experience symptoms when eating other seemingly unrelated foods.

This is called cross reactivity and occurs when your body's immune system identifies the proteins, or components, in different substances as being structurally similar or biologically related, thus triggering a response.

The most common cross reactivities with soybeans are fruits (i.e., apple, cherry), vegetables (i.e., carrot), legumes (i.e., peanut), seeds, and tree nuts.

1. EAACI, et al. Molecular allergology user's guide. Pediatr Allergy Immunol. 2016 May;27 Suppl 23:1-250. doi: 10.1111/pai.12563. PMID: 27288833. (225-234 p.) Available from: http://www.eaaci.org/documents/Molecular_Allergology-web.pdf.

ThermoFisher SCIENTIFIC

ALLETESS
MEDICAL LABORATORY
The first step... Alletess

You have tested positive to *Peanut,* ask your provider about ordering a *Peanut Component Panel*

Specific IgE testing for allergen components helps to identify the specific proteins that may cause reactions. So instead of just knowing that you're allergic to peanuts, an allergy blood test can pinpoint exactly which protein within the peanut may trigger your reaction.

Knowing the specific protein to which you're allergic can help your healthcare provider determine if you may be at risk for a more severe, systemic reaction or a mild, localized reaction

77.6% **The percentage of patients sensitized to peanut who may not be at risk for a systemic reaction.[1]**

Characteristics of individual proteins

Peanut f 13	• High levels of peanut IgE can predict the likelihood of peanut sensitivity, but may not be solely predictive of reactions or allergic response[1]
CCD MUXF3	• **LOWEST RISK** of systemic reaction[2] • Highly cross-reactive with pollen, plant food and venoms[2]
Profilin Bet v2	• **LOWER RISK** of systemic reaction[2,3] • Cross-reactive with pollens[2]
Ara h 8 f 352	• **LOWER RISK** of systemic reaction[4,5] • Risk of mild, localized symptoms, such as itching/tingling of the lips, mouth, and oropharynx[6] • Cross-reactive with pollens (e.g., birch)[6]
Ara h 9 f 427	• **VARIABLE RISK** of systemic reaction including anaphylaxis[7,8] • Often accompanied by sensitization to other peanut proteins[8] • Cross-reactive with fruits with pits (e.g., peaches)[7]
Ara h 1, 2, 3, 6 f 422, f 423, f 424, f 447	• **HIGHER RISK** of systemic reaction including anaphylaxis[7, 10] • Sensitization to Ara h 2 is nearly always associated with clinical peanut allergy[11]

CCD, Profilin, Ara h 8 MUXF3, Bet v2, f 352	Ara h 9 f 427	Ara h 1, 2, 3, 6 f 422, f 423, f 424, f 447	Management Considerations[1,4,12-18]
+	-	-	Oral food challenge (OFC) with a specialist may be recommended. High likelihood that patient may pass OFC. If patient passes an OFC: • Foods prepared with or around peanuts may be consumed • Patient not restricted to peanut-free zones
+/-	+	-	• If there is no clinical history of symptoms, please see considerations above • If there is a clinical history of symptoms, please see considerations below
+/-	+/-	+	• Choose peanut-free zones for patient's safety • Consider prescribing epinephrine auto-injector • Family, colleagues, and teachers should be made aware of allergy and have a plan

As in all diagnostic testing, any diagnosis or treatment plan must be made by the clinician based on test results, individual patient history, the clinician's knowledge of the patient, as well as their clinical judgment.

1. Nicolaou N, Poorafshar M, Murray C, et al. Allergy or tolerance in children sensitized to peanut: prevalence and differentiation using component-resolved diagnostics. J Allergy Clin Immunol. 2010;125(1):191-197. **2.** Bradshaw N, A Clinical Reference Guide to Molecular Allergy. Go Molecular! Molecular Allergy –The Basics, 2014. **3.** Katelaris CH: Food allergy and oral allergy or pollen-food syndrome. Curr Opin Allergy Clin Immunol 2010, 10:246–251. **4.** Asarnoj A, Nilsson C, Lidholm J, et al. Peanut component Ara h 8 sensitization and tolerance to peanut. J Allergy Clin Immunol. 2012;130(2):468-472. **5.** Nucera E, et al. Hypersensitivity to major panallergens in a population of 120 patients. Postepy Dermatol Alergol. 2015 Aug; 32(4): 255–261. **6.** Mittag D, Akkerdaas J, Ballmer-Weber BK, et al. Ara h 8, a Bet v 1-homologous allergen from peanut, is a major allergen in patients with combined birch pollen and peanut allergy. J Allergy Clin Immunol. 2004;114(6):1410-1417. **7.** Lauer I, Dueringer N, Pokoj S, et al. The nonspecific lipid transfer protein, Ara h 9, is an important allergen in peanut. Clin Exp Allergy. 2009;39(9):1427-1437. **8.** Sastre J: Molecular diagnosis in allergy. Clin Exp Allergy 2010, 40:1442–1460. **9.** Movérare R, Ahlstedt S, Bengtsson U, et al. Evaluation of IgE antibodies to recombinant peanut allergens in patients with reported reactions to peanut. Int Arch Allergy Immunol. 2011;156(3):282-290. **10.** Peeters KA, Koppelman SJ, van Hoffen E, et al. Does skin prick test reactivity to purified allergens correlate with clinical severity of peanut allergy? Clin Exp Allergy. 2007;37(1):108-115. **11.** Asarnoj A, Movérare R, Östblom E, et al. IgE to peanut allergen components: relation to peanut symptoms and pollen sensitization in 8-year-olds. Allergy. 2010;65(9):1189-1195. **12.** Dang, Thanh, et al. Increasing the accuracy of peanut allergy diagnosis by using Ara H 2. J Allergy Clin Immunol. 2012;129(4):1056-1063. **13.** Nicolaou, N, et al. Quantification of specific IgE to whole peanut extract and peanut components in predication of peanut allergy. J Allergy Clin Immunol. 2011:1-2. **14.** Vereda, A, et al. Peanut allergy: Clinical and immunologic differences among patients from 3 different geographic regions. J Allergy Clin Immunol. 2010;3(2):1-5. **15.** Lange L, Beyer K, Kleine-Tebbe J. Benefits and limitations of molecular diagnostics in peanut allergy. Allergo J Int. 2014;23:158–63. **16.** Pedrosa, M, et al. Utility of specific IgE to Ara h 6 in peanut allergy diagnosis. Ann Allergy Asthma Immunol. 2015;115:108-112. **17.** van der Valk, JP, et al. Mono-sensitisation to peanut component Ara h 6: a case series of five children and literature review. Eur J Pediatr. 2016;175:1227–1234. **18.** Sicherer SH, Sampson HA. Food allergy: Epidemiology, pathogenesis, diagnosis, and treatment. J Allergy Clin Immunol. 2014;133(2):291-307. **19.** Lemon-Mulé H, Sampson HA, Sicherer SH, Shreffler WG, Noone S, Nowak-Wegrzyn A. Immunologic changes in children with egg allergy ingesting extensively heated egg. J Allergy Clin Immunol. 2008;122(5):977-983. **20.** Benhamou AH, Caubet JC, Eigenmann PA, et al. State of the art and new horizons in the diagnosis and management of egg allergy. Allergy. 2010;65(3): 283-289. **21.** Ando H, Movérare R, Kondo Y, et al. Utility of ovomucoid-specific IgE concentrations in predicting symptomatic egg allergy. J Allergy Clin Immunol. 2008;122(3):583-588. **22.** Shin M, Han Y, Ahn K. The influence of the time and temperature of heat treatment on the allergenicity of egg white proteins. Allergy Asthma Immunol Res. 2013;5(2):96-101. **23.** Tomicic S, Norrman G, Fälth-Magnusson K, Jenmalm MC, Devenney I, Böttcher MF. High levels of IgG4 antibodies to foods during infancy are associated with tolerance to corresponding foods later in life. Pediatr Allergy Immunol. 2009;20(1):35-41. **24.** Urisu A, Yamada K, Tokuda R, et al. Clinical significance of IgE-binding activity to enzymatic digests of ovomucoid in the diagnosis and the prediction of the outgrowing of egg white hypersensitivity. Int Arch Allergy Immunol. 1999;120(3):192-198. **25.** Bernhisel-Broadbent J, Dintzis HM, Dintzis RZ, Sampson HA. Allergenicity and antigenicity of chicken egg ovomucoid (Gal d III) compared with ovalbumin (Gal d I) in children with egg allergy and in mice. J Allergy Clin Immunol. 1994;93(6):1047-1059. **26.** Montesinos E, Martorell A, Félix R, Cerdá JC. Egg white specific IgE levels in serum as clinical reactivity predictors in the course of egg allergy follow up. Pediatr Allergy Immunol. 2010;21(4 pt 1):634-639. **27.** Järvinen KM, Beyer K, Vila L, Bardina L, Mishoe M, Sampson HA. Specificity of IgE antibodies to sequential epitopes of hen's egg ovomucoid as a marker for persistence of egg allergy. Allergy. 2007;62(7):758-765. **28.** Nowak-Wegrzyn A, Bloom KA, Sicherer SH, et al. Tolerance to extensively heated milk in children with cow's milk allergy. J Allergy Clin Immunol. 2008; 122(2): 342-347. **29.** Shek LP, Bardina L, Castro R, Sampson HA, Beyer K. Humoral and cellular responses to cow milk proteins in patients with milk-induced IgE-mediated and non-IgE-mediated disorders. Allergy. 2005;60(7):912-919. **30.** Wal JM. Bovine milk allergenicity. Ann Allergy Asthma Immunol. 2004;93(5 Suppl 3):S2-S11. **31.** Sicherer SH, Sampson HA. Cow's milk protein-specific IgE concentrations in two age groups of milk-allergic children and in children achieving clinical tolerance. Clin Exp Allergy. 1999;29(4):507-512. **32.** Boyano-Martinez T, Garcia-Are C, Pedrosa M, Diaz-Pena JM, Quirce S. Accidental allergic reactions in children allergic to cow's milk proteins. Allergy Clin Immunol. 2009;123(4):883-888. **33.** Caubet J, et al. Utility of casein-specific IgE levels in predicting reactivity to baked milk. J Allergy Clin Immunol. 2013;131(1):222-224. **34.** CM Allergy Review. Diagnosis of cow's milk allergy in children: determining the gold standard? Expert Rev. Clin. Immunol. 2014;10(2):257-267. **35.** Kim J, et al. Dietary baked milk accelerates the resolution of cow's milk allergy in children. J Allergy Clin Immunol. 2011;128(1):125-131. **36.** Ito K, et al. The usefulness of casein-specific IgE and IgG4 antibodies in cow's milk allergic children. Clinical and Molecular Allergy. 2012;10:1:1-7.

Thermo Fisher Scientific 4169 Commercial Avenue, Portage, MI 49002, 800.346.4364 www.thermofisher.com

ThermoFisher SCIENTIFIC