



ARE YOU A GLUTTON FOR GLUTEN?

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It is almost impossible to discuss health and nutrition these days without discussing the topic of gluten with our patients. Are food companies merely taking advantage of vulnerable consumers looking for the latest fad or is there merit to “going gluten-free”? With all the questions that we get surrounding gluten, we think that it is necessary to set the record straight. By now, many of you have certainly heard of celebrities following a gluten free diet, or have heard of someone's health turning around as a result of gluten elimination. This report will give you a deeper understanding of what gluten is, where it is found, and the implications to your health.



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The Gluten Free Industry

In the last few years, sales of gluten-free food has grown 27% and is estimated at being a \$4.2 billion market in 2012. To say this is an exploding market is an understatement. Many foods have always been gluten free, but are now being labelled as such. Gluten free foods include nuts, seeds, dairy, meats, eggs, vegetables, fruits and several grains. To be considered Gluten-free typically means that a food must be processed in a separate plant that is certified Gluten-free. Technically, gluten-free means that a food has less than 30 parts per million (ppm) of gluten. Those with Celiac disease and severe reactivities to gluten can react at 3 ppm. Often the certification process is what accounts for the increased cost of gluten free foods. Also keep in mind that wheat is heavily subsidized unlike other gluten-free grains such as amaranth.



As consumer demand grows there will be more and more gluten-free products coming to the market. Our position on these foods is to consume them in extreme moderation. Replacing one grain with another will still have a negative impact on blood sugar, insulin and weight. Gluten free items should be used as a last solution, not a staple in your diet.

What is gluten?

Gluten is a protein molecule found in wheat, barley, rye, spelt, kamut, and 98% of oats (due to cross-contamination). Gluten literally means “glue”. Gluten is what gives bread its elasticity and texture. Modern wheat has been cultivated to contain higher amounts of gluten. This is great for manufacturers, but at the price of your health. Currently there is no genetically modified (GM) wheat on the market, but big business is trying to change that. Some animals that have consumed GM wheat have been reported to develop liver failure.

Hidden sources of gluten

Some hidden or unknown sources of gluten are soy sauce, food starches, food emulsifiers, food stabilizers, artificial food coloring, malt extract, beer, some red wines, seasonings and sauces. When in doubt, call the manufacturer or look for the gluten free label. Products that are manufactured in a plant that processes gluten may also be contaminated due to shared equipment.

Gluten And Chronic Illness

It is estimated that there are at least 18 million Americans that have a gluten sensitivity. We feel that number is grossly underestimated, but it is difficult to estimate the breadth of the problem since many people are misdiagnosed and never receive the right treatment.



Celiac's disease gets the lions share of attention when talking about gluten, we will discuss the many spin-off conditions that arise from gluten exposure later. To date, research indicates that over 200 diseases are associated with gluten sensitivity. These can range from seizures to skin conditions. The list would surprise you.

What is gluten sensitivity?

Gluten sensitivity can often be challenging for an untrained practitioner to identify. A sensitivity can cause subtle to severe symptoms depending on the patient. Another unfortunate fact regarding sensitivities is that they often occur outside of the digestive system, such as joints, skin, and nervous system. These reactions may also be delayed (30 minutes - 4 days), making it challenging for one to recognize gluten as the offending agent.

Once ingested the gluten molecule is particularly difficult for your body to digest. When ingested, anti-bodies to gluten are produced and gluten is "tagged" by the immune system as a foreign invader. This type of subtle and delayed reaction is known as an IGG response, much different than a IGE response often associated with foods like peanuts.

My parents ate wheat and they seem fine

We have been eating wheat for generations, but not the commercial wheat that is grown today. By modifying wheat to contain more gluten than it originally did, the agriculture business has vastly increased our exposure. Also, the wheat a person eats today goes through a process called de-amidation, which your immune system happens to not like and it causes more of an immune reaction. The Standard American Diet (SAD) has very little nutritional value and our society is much more stressful, which contributes to the breakdown of our immune tolerance for substances like gluten. Lastly, without proper nutrition, our bodies have a tendency to make less of the enzymes required to digest and break down

gluten particles. Chronic exposure combined with a compromised nutritional status and immune health seem to be the perfect storm for chronic illness related to gluten.

My doctor told me I do not have Celiac disease

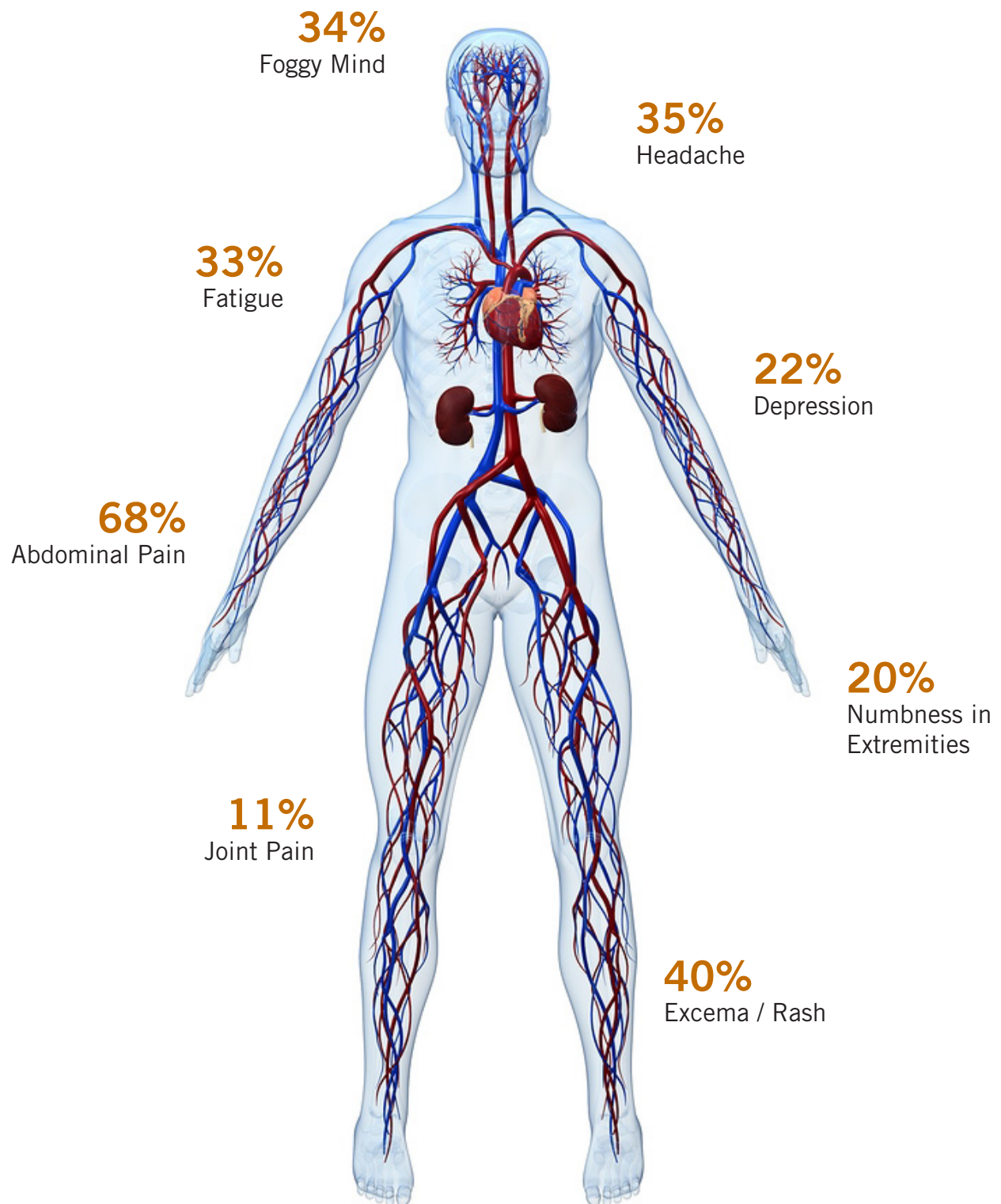
As mentioned earlier, when a person hears the word gluten it is synonymous with Celiac's Disease. However, the research clearly states that gluten sensitivity being regarded principally as a disease of the small bowel is a historical misconception. The research also demonstrates that for every gluten sensitive patient with symptoms of an enteropathy (Classic Celiac Disease), there are 8 people with no GI symptoms!

Thinking Beyond The Gut

Other organs and areas that have been demonstrated to be involved in gluten sensitivity are the joints (Rheumatoid Arthritis and Ankylosing Spondylitis), the heart, thyroid, bone, and most notably the brain, cerebellum, and neuronal synapses (think Multiple Sclerosis and Cerebellar Ataxia). There is a connection with gluten sensitivity and type 1 diabetes, osteoporosis, Addison's disease, and ADHD. Gluten sensitivity has also been demonstrated to play a critical role in infertility and stillbirths. Lastly, people with a gluten sensitivity are 10 times more likely to develop an autoimmune disease. As you can see, the notion that gluten only affects the small bowel is archaic to say the least, it is time for a radical paradigm shift.



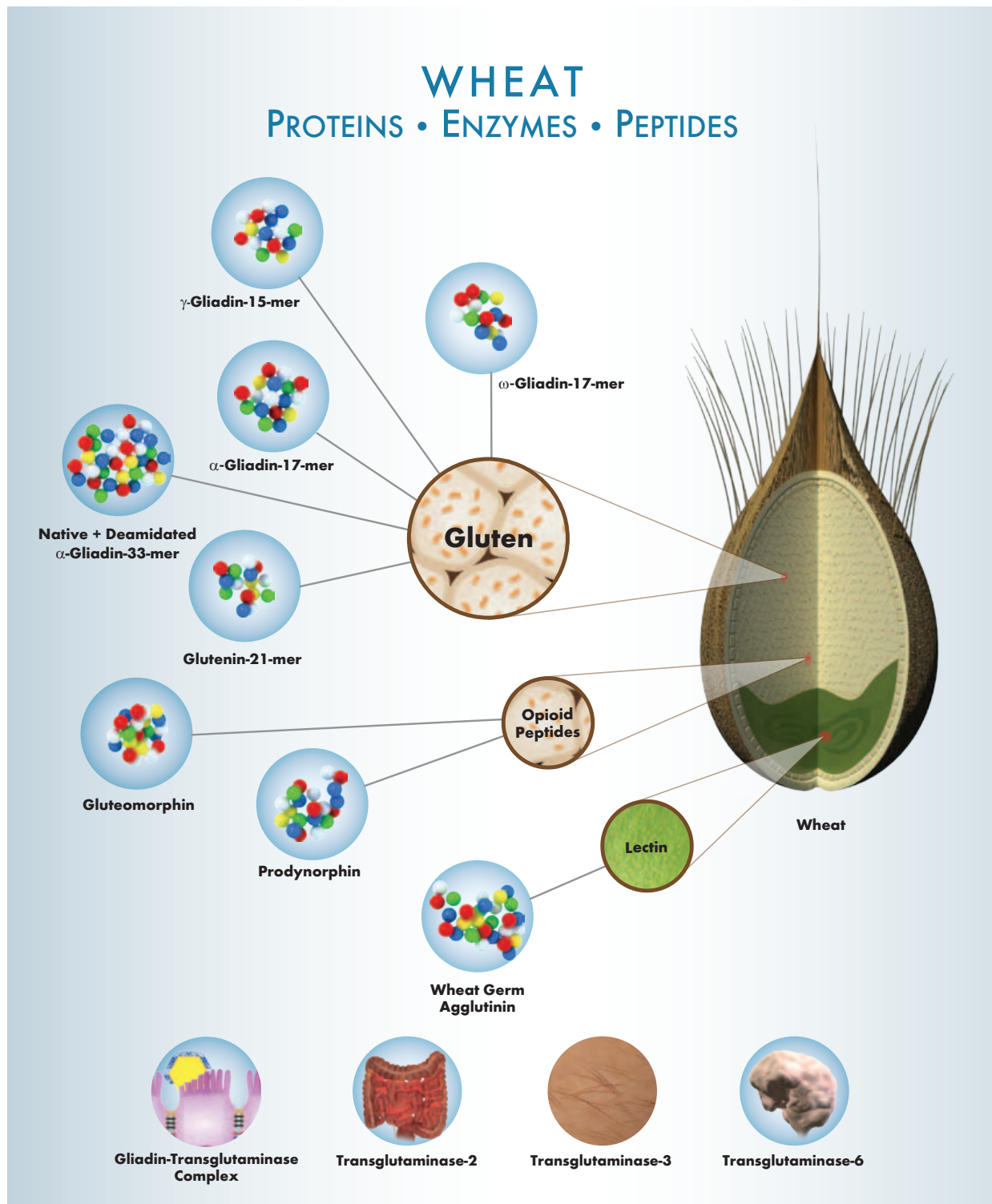
The Spectrum of Gluten Disorders



What type of testing is best?

The blood test that we recommend looks at 12 different peptides of gluten. Luckily, there is testing out there that is not only more accurate than the traditional blood test, but also easier to administer because it is done through saliva. This can be an excellent screen for people suspecting gluten sensitivity.

Despite advanced testing, it should be noted that the gold standard for any type of allergy or sensitivity is complete elimination for up to 6 months.



When going gluten-free goes wrong

In rare instances patient may report that their symptoms worsen when going on a gluten free diet. There are three possible explanation for this. Firstly, gluten binds to opioid receptors in the brain and literally acts like a drug. Similar to coming off of any addictive substance there will be a withdrawal period. This may last a few days.

Secondly, around 36% of people that go gluten free are still getting accidental exposure. This exposure may be from cross contamination, poor technique at restaurants, lack of due diligence and the occasional ingestion such as at birthdays and celebrations.

Lastly, when people go gluten-free, they usually start ingesting other grains in place of gluten. In some cases, this may initiate an immune response very similar to eating gluten. This is a notion called cross-reactivity. Cross-reactivity is the ability of an antibody to bind with similar-looking parts on different proteins called epitopes. This phenomenon is also known as molecular mimicry. Up to 82% of gluten sensitive patients have antibodies to other foods including rice, milk, beef, sheep, and eggs. Other studies have identified cross-reactivity with chocolate, sesame, hemp, rye, kamut, buckwheat, sorghum, millet, spelt, amaranth, quinoa, yeast, tapioca, oats, coffee, corn, and potatoes. As you can see, a lot of the foods that people use to replace gluten in their diet are on this list. Luckily, there is a test that we recommend that looks at these cross-reactivities as well as other common food sensitivities in those that are unresponsive to gluten elimination.

Gluten and Autism

80% of children diagnosed with autism not only produce antibodies to gluten, they also produce antibodies to their brain. This is a mind blowing number of children that are being mismanaged, misdiagnosed, and mistreated. How can a solution so simple, so effective and so cheap be ignored? At PrimeLife, going gluten free is our first line of therapy for our autistic patients.

Why has my doctor never mentioned this to me and what can I do?

The answer is easy, most clinicians are not trained to look at a gluten sensitivity when areas other than the GI tract are affected. Also, when most clinicians run an antibody test they are only looking for the alpha-gliadin marker. The problem with that is that only around 50% of celiac patients have antibodies to the alpha-gliadin portion of wheat, the other half are sensitive to a different portion.





The PrimeLife Approach

In the face of so much research and empirical data, it is difficult to ignore the elephant in the room. Going gluten free can be an extremely easy dietary change with the right guidance and by taking small efforts to read food labels more closely. When in doubt, stick to a whole food diet that excludes processed and packaged foods.

References

Vojdani A, O'Bryan T, Green J, McCandless J, Woeller K, Vojdani E, Nourian E, Cooper E, Immune Response to Dietary Proteins, Gliadin and Cerebellar Peptides in Children with Autism, Nutritional Neuroscience, Volume 7 Number 3 (June 2004), pp. 151–161.

Marks J, Shuster S, Watson AJ. Small bowel changes in dermatitis herpetiformis. *Lancet* 1966; 2:1280- 1282.

Cooke WT, Thomas-Smith W. Neurological disorders associated with adult celiac disease. *Brain* 1966; 89:683-722.

3. Hvatut M, Kanerud L, Hällgren R, Brandtzaeg P. The gut-joint axis: cross-reactive food antibodies in rheumatoid arthritis. *Gut* 2006; 55:1240-1247.

4. Kjeldsen-Kragh J, Hvatut M, Haugen M, Forre O, Scott H. Antibodies against dietary antigens in rheumatoid arthritis patients treated with fasting and a one-year vegetarian diet. *Clin Exp Rheumatol* 1995; 13:167-172.

5. Koot VC, Van Straaten M, Hekkens WT, Collee G, Dijkmans BA. Elevated level of IgA gliadin antibodies in patients with rheumatoid arthritis. *Clin Exp Rheumatol* 1989; 7:623-626.

O'Farely C, Marten D, Melcher D, McDougall B, Price R, Goldstein AJ, Sherwood R, Fernandes L.A. VOJDANI ET AL. Association between villous atrophy in rheumatoid arthritis and a rheumatoid factor and gliadin-specific IgG. *Lancet* 1988; 2:819-822.

Quan CP, Berneman A, Pires R, Avrameas S, Bouvet JP. Natural polyreactive secretory immunoglobulin A autoantibodies as a possible barrier to infection in humans. *Infect Immun* 1997; 65:3997-4004.

Frustaci A, Cuoco L, Chimenti C, Pieroni M, Fioravanti G, Gentiloni N, Maseri A, Gasbarrini G. Celiac disease associated with autoimmune myocarditis. *Circulation* 2002; 105:2611-1618.

Chimenti C, Pieroni M, Frustaci IA. Celiac disease in idiopathic dilated cardiomyopathy. *Ital Heart J* 2001; 2:658-659.

Counsell CE, Taha A, Ruddell WS. Coeliac disease and autoimmune thyroid disease. *Gut* 1994; 35:844- 846.

Natter S, Granditsch G, Reichel GL, Baghestanian M, Valent P, Elfman L, Gronlund H, Kraft D, Valenta R. IgA cross-reactivity between a nuclear autoantigen and wheat proteins suggests molecular mimicry as a possible pathomechanism in celiac disease. *Eur J Immunol* 2001; 31:918-928.

Pedeira S, Vazquez H, Sugai E, Niveloni S, Smecuol E, Mazure R, Flores D, Maurino E, Bai JC. Clinical significance of anti-smooth muscle antibody (SMA) fluorescence in patients with celiac disease. *Gastroenterol* 2000; 118:A363.

Sugai E, Cherniavsky A, Pedeira S, Smecuol E, Vazquez H, Niveloni S, Mazure R, Mauriño E, Rabinovich GA, Bai JC. Bone-specific antibodies in sera from patients with celiac disease: characterization and implications in osteoporosis. *J Clin Immunol* 2002; 22:353-362.

Vazquez H, Mazure R, Gonzalez D, Flores D, Pedeira S, Niveloni S, Smecuol E, Mauriño E, Bai JC. Risk of fractures in celiac disease patients: A cross-sectional, case-control study. *Am J Gastroenterol* 2000; 95:183-189.

Pratesi R, Gandolfi L, Friedman H, Farage L, De Castro CAM, Catassi C. Serum IgA antibodies from patients with coeliac disease react strongly with human brain blood-vessel structures. *Scand J Gastroenterol* 1998; 33:817- 821.

Volta U, De Giorgio R, Petrolini N, Stangbellini V, Barbara G, Granito A, De Ponti F, Corinaldesi R, Bianchi FB. Clinical findings and anti-neuronal antibodies in celiac disease with neurological disorders. *Scand J Gastroenterol* 2002; 137: 1276- 1281.

Hadjivassiliou M, Grünewald R, Sharrack B, Sanders D, Lobo A, Williamson C, Woodroffe N, Wood N, Davies-Jones A. Gluten ataxia in perspective: epidemiology, genetic susceptibility and clinical characteristics. *Brain* 2003; 126:685-691.

Abele M, Schöls L, Schwartz S, Klockgether T. Prevalence of antigliadin antibodies in ataxia patients. *Neurology* 2003; 60:1674-1675.

Hadjivassiliou M, Boscolo S, Davies-Jones GA, Grünewald RA, Not T, Sanders DS, Simpson JE, Tongiorgi E, Williamson CA, Woodroffe NM. The humoral response in the pathogenesis of gluten ataxia. *Neurology* 2002; 58:1221-1226.

Hadjivassiliou M, Davies-Jones GAB, Sanders DS, Grünewald RA. Dietary treatment of gluten ataxia. *J Neurol Neurosurg Psychiatry* 2003; 74:1221-1224.

Vojdani A, O'Bryan T, Green JA, McCandless J, Woeller KN, Vojdani E, Nourian AA, Cooper EL. Immune response to dietary proteins, gliadin and cerebellar peptides in children with autism. *Nutritional Neuroscience* 2004; 7(3):151-161.

Alaedini A, Okamoto H, Briani C, Wollenberg K, Shill HA, Bushara KO, Sander HW, Green PH, Hallett M, Latov N. Immune cross-reactivity in celiac disease: anti-gliadin antibodies bind to neuronal synapsin I. *J Immunol* 2007; 178:6590- 6595.

Green PH, Alaedini A, Sander HW, Brannagan TH III, Latov N, Chin RL. Mechanisms underlying celiac disease and its neurologic manifestations. *Cell Mol Life Sci* 2005; 62:191-199.

Hilfiker S, Benfenati F, Doussau F, Nairn AC, Czernik AJ, Augustine GJ, Greengard P. Structural domains involved in the regulation of transmitter release by synapsins. *J Neurosci* 2005; 25:2658- 2669.

Sommer C, Weishaupt A, Brinkhoff J, Biko L, Wessig C, Gold R, Toyka KV. Paraneoplastic stiff- person syndrome: passive transfer to rats by means of IgG antibodies to amphiphysin. *Lancet* 2005; 365: 1406-1411.

Takenoshita HM, Shizuka-Ikeda M, Mitoma H, SongS-Y, Harigaya Y, Igeta Y, Yaguchi M, Ishida K, Shoji M, Tanaka M, Mizusawa H, Okamoto K. Presynaptic inhibition of cerebellar GABAergic transmission by glutamate decarboxylase autoantibodies in progressive cerebellar ataxia. *J Neural Neurosurg Psychiatry* 2001; 70:386-389.

Zelnick N, Pacht A, Obeid R, Lerner A. Range of neurologic disorders in patients with celiac disease. *Pediatr* 2004; 113:1672-1676.

Hadjivassiliou M, Williamson CA, Woodroffe N. The immunology of gluten sensitivity beyond the gut. *Trends Immunol* 2004; 25:578-582.

Kumar V, Rajadhyaksha M, Wortsman J. Celiac disease associated autoimmune endocrinopathies. *Clin Diag Lab Immunol* 2001; 8:678-685.

Funda DP, Kaas A, Bock T, Tlaskalová-Hogenová H, Buschard K. Gluten-free diet prevents diabetes in NOD mice. *Diabetes Metab Res Rev* 1999; 15:323-327.

Westman E, Ambler GR, Royle M, Peat J, Chan A. Children with coeliac disease and insulin-dependent diabetes mellitus-growth, diabetes control dietary intake. *J Pediatr Endocrinol Metab* 1999; 12:433-37.

Cronin CC, Shanahan F. Insulin-dependent diabetes mellitus and coeliac disease. *Lancet* 1997; 349:1096- 442.

Lorini R, Scotta MS, Cortona L, Avanzini MA, Vitali L, De Giacomo C, Scaramuzza A, Severi F. Celiac disease and type I (insulin-dependent) diabetes mellitus in childhood: follow-up study. *J Diabetes Complications* 1996; 10:154-159.

Velluzzi F, Caradonna A, Boy MF, Mossa P, Corda G, Pinna MA, Cabula R, Lai MA, Piras E, Atzeni F, Loviselli A, Usai P, Mariotti S. Thyroid and celiac disease: clinical, serologic and echographic study. *Am J Gastroenterol* 1998; 93:976-979.

Sategna-Guidetti C, Bruno M, Mazza E, Carlino A, Predebon S, Tagliabue M, Brossa C. Auto-immune thyroid disease and coeliac disease. *Eur J Gastroenterol Hepatol* 1998; 10:927-931.

Venture A, Neri E, Ughi C, Leopaldi A, Citta A, Not T. Gluten-dependent diabetes-related and thyroid- related antibodies in patients with celiac disease. *J Pediatr* 2000; 137:263-265.

Valentino R, Savastano S, Tommaselli AP, Dorato M, Scarpitta MT, Gigante M, Lombardi G, Troncone R. Unusual association of thyroiditis, Addison's disease, ovarian failure and celiac disease in a young woman. *J Endocrinol Investig* 1999; 22:390-394. Selby P. Bone loss in celiac disease is related to secondary hyperparathyroidism. *J Bone Mineral Res* 1999; 14:652-657.

Iafusco D, Rea F, Chiarelli F, Mohn A, Prisco F. Effect of gluten-free diet on the metabolic control of type I diabetes in patients with diabetes and celiac disease. *Diabetes Care* 2000; 23:712-713.

Hadjivassiliou M., Gluten Sensitivity as a Neurological illness, *J Neurol Neurosurg Psychiatry* 2002;72:560–563.

van Heel D., West J, Recent Advances in Coeliac Disease, *Gut* 2006;55:1037–1046.

Fasano A, Catassi C., Current Approaches to Diagnosis and Treatment of Celiac Disease: An Evolving Spectrum *Gastroenterology* 2001;120:636-651.

Goddard CJ., Gillett H R., Complications of coeliac disease: are all patients at risk? *Postgrad. Med. J.* 2006;82:705-712.

Green PHR, Stavropoulos SN, Panagi SG, et al. Characteristics of adult celiac disease in the USA: results of a national survey. *Am J Gastroenterol* 2001;96:126–31.

Olesen M, Eriksson S, Bohr J, Jarnerot G, Tysk C. Microscopic colitis: a common diarrhoeal disease. An epidemiological study in Orebro, Sweden, 1993-1998. *Gut*, 2004; 53:346-350.

Gillet HR, Freeman HJ. Prevalence of celiac disease in collagenous and lymphocytic colitis. *Can J Gastroenterol*, 2000; 14: 919-921.

Koskela RM, Niemelä SE, Karttunen TJ, Lehtola JK. Clinical characteristics of collagenous and lymphocytic colitis. *Scand J Gastroenterol*, 2004;39: 837-845.

Corrao G, Corrazza GR, Bagnardi V, et al. Mortality in patients with coeliac disease and their relatives: a cohort study. *Lancet* 2001;358:356–61.

Hill I Salem W, Dirks M, Liptak G, Colletti R , Fasano A, Guandalini S, Hoffenberg E, Horvath K, Murray J, Pivor M, Salem W, Seidman E, Guideline for the Diagnosis and Treatment of Celiac Disease in Children: Recommendations of the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition, *J Pediatr Gastroenterol Nutr*, Vol. 40, No. 1, January 2005.

Hadjavassiliou M, Gluten Sensitivity: from Gut to Brain. *Lancet Neurol* 2010; 9: 318–30.

Fasano, A, Celiac Disease-How to handle a Clinical Chameleon, NEJM 348;25 June 19,2003.

Arnson Y, Amital H, and Shoenfeld Y, Vitamin D and autoimmunity: new aetiological and therapeutic considerations, J of Immunology, 2005, 175: 4119–4126.

Alaiedini A, Okamoto H, Briani, C, Wollenberg K, Shill H, Bushara K, Sander H, Green P, Hallett M, Latov N, Immune Cross-Reactivity in Celiac Disease: Anti-Gliadin Antibodies Bind to Neuronal Synapsin I, The Journal of Immunology, 2007, 178: 6590– 6595.

Bland J., Understanding the Origins and Applying Advanced Nutritional Strategies for Auto-immune Disease, Metagenics Seminar Series, 2006.

Green P, Alaiedini A, Sander HW, Brannagan III TH, Latov N, Chin R, Mechanisms underlying celiac disease and its Neurologic Manifestations Cell. Mol. Life Sci. 62 (2005) 791–799.

Marietta E, Black K, Camilleri M, Krause P, Rogers RS 3rd, David C, Pittelkow MR, Murray JA., A new model for dermatitis herpetiformis that uses HLA-DQ8 transgenic NOD mice, J Clin Invest. 2004 Oct;114(8):1090-7.

Lindqvist U, Rudsander A, Boström A, Nilsson B, Michaëlsson G., IgA antibodies to gliadin and coeliac disease in psoriatic arthritis, Rheumatology (Oxford). 2002 Jan;41(1):31-7.

Humbert P, Pelletier F, Dreno B, Puzenat E, Aubin F, Gluten intolerance and skin diseases, Eur J Dermatol 2006; 16 (1): 4-11.

Selva-O'Callaghan A, Casellas F, de Torres I, Palou E, Grau-Junyent JM, Vilardell- Tarrés M., CELIAC DISEASE AND ANTIBODIES ASSOCIATED WITH CELIAC DISEASE IN PATIENTS WITH INFLAMMATORY MYOPATHY, Muscle Nerve. 2007 Jan;35(1):49-54.

Hadjivassiliou M, Grünewald R, Sharrack B, Sanders D, Lobo A, Williamson C, Woodroffe N, Wood N, Davies-Jones A., Gluten ataxia in perspective: epidemiology, genetic susceptibility and clinical characteristics, Brain. 2003 Mar;126(Pt 3):685-91.

Hadjivassiliou M, Aeschlimann D, Grünewald RA, Sanders DS, Sharrack B, Woodroffe N, GAD antibody-associated neurological illness and its relationship to gluten sensitivity, Acta Neurol Scand. 2010 Apr 15.

Eaton W, Mortensen PB, Agerbo E, Byrne M, Mors O, Ewald H., Coeliac disease and schizophrenia: population based case control study with linkage of Danish national registers, BMJ. 2004 Feb 21;328(7437):438-9.

Hadjivassiliou M, Grünewald RA, Chattopadhyay AK, Davies-Jones GAB, Gibson A, Jarratt JA, et al. Clinical, radiological, neurophysiological and neuropathological characteristics of gluten ataxia. Lancet 1998;352:1582-5.

J Neurol Neurosurg Psychiatry. 2006 Nov;77(11):1262-6., Hadjivassiliou M, Grünewald RA, Kandler RH, Chattopadhyay AK, Jarratt JA, Sanders DS, Sharrack B, Wharton SB, Davies-Jones GA, Neuropathy associated with gluten sensitivity. Gluten sensitivity: from gut to brain., Hadjivassiliou M, Sanders DS, Grünewald RA, Woodroffe N, Boscolo S, Aeschlimann D, Lancet Neurol. 2010 Mar;9(3):318-30.

Hopper A., et.al., Pre-endoscopy serological testing for coeliac disease:evaluation of a clinical decision tool, BMJ. 2007 Apr 7;334(7596):729.

Hill ID., What are the sensitivity and specificity of serologic tests for celiac disease? Do sensitivity and specificity vary in different populations? Gastroenterology. 2005 Apr;128(4 Suppl 1):S25-32.

Memeo L, Jhang J, Hibshoosh H, Green PH, Rotterdam H, Bhagat G., Duodenal intraepithelial lymphocytosis with normal villous architecture: common occurrence in H. pylori gastritis, Mod Pathol. 2005 Aug;18(8):1134-44.

Abrams JA, Diamond B, Rotterdam H, Green PH. Seronegative celiac disease: increased prevalence with lesser degrees of villous atrophy, Dig Dis Sci. 2004 Apr;49(4):546-50.

Tursi A., Seronegative Coeliac Disease: a Clinical Challenge. BMJ 26 April, 2005. Rostami, K., Unforgiving Master of Non-Specificity and Disguise, BMJ 27, April 2005.

Lebwold, Green P, Screening for Celiac Disease. N Engl J Med Oct.23 2003,1673-4.

Freeman HJ., Pearls and pitfalls in the diagnosis of adult celiac disease. Can J Gastroenterol 2008;22(3):273-280.

Bonamico M., Serologic and Genetic Markers of Celiac Disease: A Sequential Study in the Screening of First Degree Relatives, Journal of Pediatric Gastroenterology and Nutrition 42:150–154.

Fasano A., Catassi C., Current Approaches to Diagnosis and Treatment of Celiac Disease: An Evolving Spectrum, GASTROENTEROLOGY 2001;120:636–651. Hadjavassiliou M., Grunewald R., The Neurology of Gluten Sensitivity:Science vs. Conviction Practical Neurology, 2004, 4, 124–126.

Camarca, A., et.al., Intestinal T Cell Responses to Gluten Peptides Are Largely Heterogeneous: Implications for a Peptide-Based Therapy in Celiac Disease, J. Immunol. 2009;182:4158-4166.

Meresse B., , Ripoche J., Heyman M., Cerf-Bensussan N., Celiac disease: from oral tolerance to intestinal inflammation, autoimmunity and lymphomagenesis, Nature Vol 2 No 1, JANUARY 2009.

Bethune M.,Parallels Between Pathogens and Gluten Peptides in Celiac Sprue, Plos Pathogens Feb 2008 Vol 4: 2:e34.

Ehrhardt G., et.al. Discriminating gene expression profiles of memory B cell subpopulations JEM VOL. 205, August 4, 2008.

Martucci S., Corazza G., Spreading and Focusing of Gluten Epitopes in Celiac Disease GAS-TROENTEROLOGY Vol. 122, No. 7, 2002.

Pastore L., et.al., Orally Based Diagnosis of Celiac Disease: Current Perspectives, J Dent Res 87(12):1100-1107, 2008.

Vader W., et.al., The Gluten Response in Children With Celiac Disease Is Directed Toward Multiple Gliadin and Glutenin Peptides, GASTROENTEROLOGY 2002;122 :1729–1737.

Green P, Cellier C, Celiac Disease NEJM 357;17 Oct 25, 2007. Leffler DA, Dennis M, Hyett B, Kelly E, Schuppan D, Kelly CP, Etiologies and predictors of diagnosis in nonresponsive celiac disease, Clin Gastroenterol Hepatol. 2007 Apr;5(4):445-50.

Sicherer SH., Clinical implications of cross-reactive food allergens, J Allergy Clin Immunol. 2001 Dec;108(6):881-90.

Kristjánsson G, Venge P, Hällgren R., Mucosal reactivity to cow's milk protein in coeliac disease, Clin Exp Immunol. 2007 Mar;147(3):449-55.

Bürgin-Wolff A, Hernandez R, Just M, Signer E., Immunofluorescent antibodies against gliadin: a screening test for coeliac disease, Helv Paediatr Acta. 1976 Dec;31(4-5):375-80.

Ferguson A, Carswell F, Precipitins to dietary proteins in serum and upper intestinal secretions of coeliac children, Br Med J. 1972 Jan 8;1(5792):75-7.

Becker CG, Van Hamont N, Wagner M. Tobacco, cocoa, coffee, and ragweed: cross-reacting allergens that activate factor-XII-dependent pathways. Blood, 1981; 58(5):861-867.

Gangur V, Kelly C, Navuluri L. Sesame allergy: a growing food allergy of global proportions? Ann Allergy Asthma Immunol, 2005; 95:4-11.

Kagi Mk, Wuthrich B. Falafel burger anaphylaxis due to sesame seed allergy. Ann Allergy, 1993; 71(2):127-129.

Keskinen H, Ostman P, Vaheria E, et al. A case of occupational asthma, rhinitis and urticaria due to sesame seed. Clin Exp Allergy, 1991; 21:623-624.

Pecquet C, Leynadier F, Saïag P. Immediate hypersensitivity to sesame in foods and cosmetics. Contact Dermatitis, 1998; 39:313.

Perkins MS. Raising awareness of sesame allergy. Pharma J, 2001; 267:757-758. Popa V, Gavrilescu N, Preda N, et al. an investigation of allergy in byssinosis: sensitization to cotton, hemp, flax and jute antigens. Brit J Industr Med, 1969; 26:101-108.

Ciclitiera PJ and Ellis HJ. Relation of antigenic structure of cereal proteins to their toxicity in coeliac patients. Brit J Nutr, 1985; 53:39-45.

Kasarda DD. Grains in relation to celiac disease. Cereal Foods World, 2001; 46:209-210.

Simonato B, Pasini G, Giannattasio M, Curioni A. Allergenic potential of Kamut® wheat. Allergy, 2002; 57:653-654.

Göhle C-J, Wislander G, Ancker K, Forsbeck M. bucksheat allergy: health food, an inhalation health risk. Allergy, 2007; 38(3):155-159.

Hekkens WT. The determination of prolamins in gluten-free food. Introductory remarks. Panminerva Med, 1991; 33(2):61-64.

Kim J-L, Wieslander G, Norbäck D. Allergy/Intolerance to buckwheat and other food products among Swedish subjects with celiac disease. Proc. 9th Int'l Symp Buckwheat, Prague, 2004:705-709.

Lee SY, Lee KS, Hong CH, Lee KY. Three cases of childhood nocturnal asthma due to buckwheat allergy. Allergy, 2001; 56:763-766.

Pomeranz Y, Marshall HG, Robbins Gs, Gilbertson JT. Protein content and amino acid composition of maturing buckwheat (Fagopyrum esculentum moench). Cereal Chem, 1975; 52:479-484.

De Maat-Bleeker F, Stapel SO. Cross-reactivity between buckwheat and latex. Allergy, 1998; 53:538-539.

Sdepanian VL, Scaletsky ICA, Fagundes-Neto U, de Moraes MB. Assessment of gliadin in supposedly gluten-free foods prepared and purchased by celiac patients. J Ped Gastroenterol Nutr, 2001; 32:65-70.

Skerrett JH, Devery JM, Hill AS. Chemistry, coeliac-toxicity and detection of gluten and related prolamins in foods. Panminerva Med, 1991; 33(2):65-74.

Wieslander G, Norbäck D. Buckwheat allergy. Allergy, 2001; 56:703-704.

Bietz JA. Cereal prolamin evolution and homology revealed by sequence analysis. Biochem Genetics, 1982; 20(11/12):1039-1053.

Cicek M and Esen A. Structure and expression of a dhurrinase (-glucosidase) from sorghum. *Plant Physiol*, 1998; 116:1469-1478.

Mazhar H, Chandrashekar A, Shetty HS. Isolation and immunochemical characterization of the alcohol-extractable proteins (kafirins) of *Sorghum bicolor* (L.) Moench. *J Cereal Sci*, 1993; 17(1):83-93.

Gaitan E, Cooksey RC, Legan J, Lindsay RH. Antithyroid effects *in vivo* and *in vitro* of vitexin: a C-glucosylflavone in millet. *J Clin Endocrinol Metab*, 1995; 80(4):114-1147.

Monteiro PV, Virupaksha TK, Rao DR. Proteins of Italian millet: amino acid composition, solubility fractionation and electrophoresis of protein fractions. *J Sci Food Agric*, 1982; 33(11):1072-1079.

Monteiro PV, Sudharshna L, Ramachandra G. Japanese barnyard millet (*Echinochloa frumentacea*): protein content, quality and SDS-PAGE of protein fractions. *J Sci Food Agric*, 1988; 43(1):17-25.

Parameswaran KP and Thayumanavan B. Homologies between prolamins of different minor millets. *Plant Foods Human Nutr*, 1995; 48:119-126.

Parameswaran KP and Thayumanavan B. Isolation and characterization of a 20 kD prolamin from kodo millet (*Paspalum scrobiculatum*) (L.): homology with other millets and cereals. *Plant Foods Human Nutr*, 1997; 50:359-373.

Grela ER. Nutrient composition and content of antinutritional factors in spelt (*Triticum spelta* L.) cultivars. *J Sci Food Agric*, 1996; 71(3):399-404.

Jones SM, Megnoli CG, Cooke SK, Sampson HA. Allergens, IgE, mediators, inflammatory mechanisms: immunologic cross-reactivity among cereal grains and grasses in children with food hypersensitivity. *J Allergy Clin Immunol*, 1995; 96:341-351.

Pastorello EA, Farioli L, Robino A, et al. A lipid transfer protein involved in occupational sensitization to spelt. *J Allergy Clin Immunol*, 2001; 108(1):145-146.

Skrabagnja V, Kovac B, Golob T, et al. Effect of spelt wheat flour and kernel on bread composition and nutritional characteristics. *J Agric Food Chem*, 2001; 49:497-500.

Aphalo P, Castellani OF, Martinez EN, Anón MC. Surface physicochemical properties of globulin-P amaranth protein. *J Agric Food Chem*, 2004; 52:616-622.

Gorinstein S, Delgado-Licon E, Pawelzik E, et al. Characterization of soluble amaranth and soybean proteins based on fluorescence, hydrophobicity, electrophoresis, amino acid analysis, circular dichroism, and differential scanning calorimetry measurements. *J Agric Food Chem*, 2001; 49:5595-5601.

Vasco-Méndez NL and Paredes-López O. antigenic homology between amaranth glutelins and other storage proteins. *J Food Biochem*, 1995; 18(4):227-238.

Aluko RE and Monu E. Functional and bioactive properties of quinoa seed protein hydrolysates. *J Food Sci*, 2003; 68(4):1254-1258.

Lee AR, Ng DL, Dave E, et al. The effect of substituting alternative grains in the diet on the nutritional profile of the gluten-free diet. *J Hum Nutr Diet*, 2009; 22:359-363.

Wright KH, Huber KC, Fairbanks DJ, Huber CS. Isolation and characterization of Atriplex hortensis and sweet Chenopodium quinoa starches. *Cereal Chem*, 2002; 79(5):715-719.

Heelan Bt, Allan S, Barnes RMR. Identification of a 200-kDa glycoprotein antigen of *Saccharomyces cerevisiae*. *Immunol Lett*, 1991; 28:181-186.

Oshitani N, Hato F, Kenishi S, et al. Cross-reactivity of yeast antigens in human colon and peripheral leukocytes. *J Pathol*, 2003; 199:361-367.

Sendid B, Quinton JF, Charrier G, et al. Anti-Saccharomyces cerevisiae mannan antibodies in familial Crohn's disease. *Am J Gastroenterol*, 2001; 93(8):1306-1310.

Vojdani A, Rahimian P, Kalhor H, Mordechai E. Immunological cross reactivity between candida albicans and human tissue. *J Clin Lab Immunol*, 1996; 48:1-15.

Young Ca, Sonnenberg A, Berns EA. Lymphocyte proliferation response to baker's yeast in Crohn's disease. *Digestion*; 1994;55(1):40-43.

Beezhold DH, Sussman GL, Liss GM, Chang NS. Latex allergy can induce clinical reactions to specific foods. *Clin Exp Allergy*, 1996; 26(4):416-422.

Brehler R, Theissen U, Hohr C, Luger T. "Latex-fruit syndrome": frequency of cross-reacting IgE antibodies. *Allergy*, 1997; 52:404-410.

Ibero M, Castillo MJ, Pineda F. Allergy to cassava: a new allergenic food with cross-reactivity to latex. *J Investig Allergol Clin Immunol*, 2007; 17(6):409-412.

Mikkola JH, Alenius H, Kalkkinen N, et al. Hevein-like protein domains as a possible cause for allergen cross-reactivity between latex and banana. *J Allergy Clin Immunol*, 1998; 102:1005-1012.

Arentz-Hansen H, Fleckenstein B, Molberg Ø, et al. The molecular basis for oat intolerance in patients with celiac disease. *PLoS Med*, 2004 1(1):084-092.

Janatuinen EK, Pekka HP, Kemppainen TA, et al. A comparison of diets with and without oats in adults with celiac disease. *N Engl J Med*, 1995; 333:1033-1037.
Reunala T, Collin P, Holm K, et al. Tolerance to oats in dermatitis herpetiformis. *Gut*, 1998; 43:490-493.

Silano M, Dessì M, De Vincenzi M, Cornell H. *In Vitro* tests indicate that certain varieties of oats may be harmful to patients with coeliac disease. *J Gastroenterol Hematol*, 2007; 22:528-531.

Srinivasan U, Jones E, Carolan J, Feighery C. Immunohistochemical analysis of coeliac mucosa following ingestion of oats. *Clin Exp Immunol*, 2006; 144:197-203.
Thompson T. Gluten contamination of commercial oat products in the United States. *N Engl J Med*. 2004; 351(19):2021-2022.

Axelsson IG. Allergy to the coffee plant. *Allergy*, 1994; 49(10):885-887.
Caballero Tm, Garcia-Ara C, Pascual C, et al. Urticaria induced by caffeine. *J Investig Allergol Clin Immunol*, 1993; 3(3):160-162.

Moneret-Vautrin DA, Kanny G, Faller JP, et al. [Severe anaphylactic shock with heart arrest caused by coffee and gum Arabic, potentiated by beta-blocking eyedrops]. *Rev Med Interne*, 1993; 14(2):107-111.

Osterman K, Johansson SG, Zetterström O. Diagnostic tests in allergy to green coffee. *Allergy*, 1995; 40(5):336-343.

Treudler R, Tebbe B, Orfanos CE. Coexistence of type I and type IV sensitization in occupational coffee allergy. *Contact Dermatitis*, 1997; 36:109.

Davidson IW, Lloyd RS, Whorwell PJ, Wright R. Antibodies to maize in patients with Crohn's disease, ulcerative colitis and coeliac disease. *Clin Exp Immunol*, 1979, 35:147-148 lxxvi

Lehrer SB, Reese G, Malo J-L, et al. Corn Allergens: IgE antibody reactivity and cross-reactivity with rice, soy, and peanut. *Int Arch Allergy Immunol*, 1999; 118:298-299.

Paulis JW and Bietz JA. Separation of alcohol-soluble maize proteins by reversed-phase high performance liquid chromatography. *J Cereal Sci*, 1986; 4:205-216.

Asero R, Amato S, Alfieri B, et al. Rice: another potential cause of food allergy in patients sensitized to lipid transfer protein. *Int Arch Allergy Immunol*, 2007; 143:69-74.

Horikoshi M, Kobayashi H, Yamazoe Y, et al. Purification and complete amino acid sequence of a major prolamin of rice endosperm. *J Cereal Sci*, 1991; 14(1):1-14.

Urisu A, Yamada K, Masuda S, et al. 16-kilodalton rice protein is one of the major allergens in rice grain extract and responsible for cross-allergenicity between cereal grains in the poaceae family. *Int Arch Allergy Immunol*, 1991; 96(3):244-252.

Wen T-N and Luthe DS. Biochemical characterization of rice glutelin. *Plant Physiol*, 1985; 78:172-177.

Yamada K, Urisu A, Komada H, et al. [Involvement of rice protein 16KD in cross-allergenicity between antigens in rice, wheat, corn, Japanese millet, Italian millet]. *Arerugi*, 1991; 40(12):1485-1495.

Racusen D and Foote M. A major soluble glycoprotein of potato tubers. *J Food Biochem*, 1980; 4(1):43-52.

Vos-Scheperkeuter GH, De Boer W, Visser RGF, et al. Identification of granule-bound starch synthase in potato tubers. *Plant Physiol*, 1986; 82:411-416.

Vos-Scheperkeuter GH, de Wit JG, Ponstein AS, et al. Immunological comparison of the starch branching enzymes from potato tubers and maize kernels. *Plant Physiol*, 1989; 90:75-84.

Hvatum M, Scott H, Brandtzaeg P, Serum IgG subclass antibodies to a variety of food antigens in patients with coeliac disease, *Gut*. 1992 May;33(5):632-8.

Husby S, Foged N, Oxelius VA, Svehag SE, Serum IgG subclass antibodies to gliadin and other dietary antigens in children with coeliac disease, *Clin Exp Immunol*. 1986 Jun;64(3):526-35.

Breiteneder H, Ebner C., Molecular and biochemical classification of plant-derived food allergens, *J Allergy Clin Immunol*. 2000 Jul;106(1 Pt 1):27-36. Review

Scott H, Fausa O, Ek J, Brandtzaeg P, Immune response patterns in coeliac disease. Serum antibodies to dietary antigens measured by an enzyme linked immunosorbent assay (ELISA), *Clin Exp Immunol*. 1984 Jul;57(1):25-32.

Farthing M, Rees L, Edwards C, Dawson A. Male gonadal function in coeliac disease: 2. Sex hormones. *Gut*, 1983; 24, 127-135. 26. Betterle C, Zanchetta R. Update on autoimmune polyendocrine syndromes (APS). *Acta Bio Medica*, 2003; 74; 9-33.

F, Facciuto E, Migliaccio S, Bardella MT, Dubini A, Borghi MO, Saraifogher S, Teti A, Bianchi ML. Imbalance of osteoclastogenesis-regulating factors in patients with celiac disease. *J Bone Miner Res*, 2004; 19: 1112-1121.

da Silva K, Kotze L, Nishihara R, daRosa U, Piovezan G, Kotze L. Thyroid disorders in Brazilian patients with celiac disease. *J Clin Gastroenterol*, 2006; 40(1):33-36.

Stone S, Khamashta MA, Poston L. Placental, antiphospholipid syndrome and pregnancy

outcome. *Lupus*, 2001;10:67-74.

Shamir R, Shoenfeld Y, Blank M, Eliakim R, Lahat N, Sobel E, Shinar E, Lerner A. The prevalence of coeliac disease antibodies in patients with the antiphospholipid syndrome. *Lupus*, 2003;12:394.

La Villa G, Pantaleo P, Tarquini R, Cirami L, Perfetto F, Mancuso F, Laffi G. Multiple immune disorders in unrecognized celiac disease: a case report. *World J Gastroenterol*, 2003;9(6): 1377-1380.

Sategna-Guidetti C, Volta U, Ciacci C, Usai P, Carlino A, De Franceschi L, Camera A, Pelli A, Brossa C. Prevalence of Thyroid Disorders in Untreated Adult Celiac Disease Patients and Effect of Gluten Withdrawal: An Italian Multicenter Study. *AJG*, 2001;Vol. 96, No. 3: 751-757.

Levine JS, Branch DW, Rauch J. The antiphospholipid syndrome. *NEJM*, 2002; 346: 752-763.

Djuric Z, Zivic S, Katic V. Coeliac disease with diffuse cutaneous Vitamin K deficiency bleeding. *Adv Ther*, 2007;24(6):1286- 1289.

Rujner J. Age at menarche in girls with celiac disease. *Ginek Pol*, 1999;70:359-362.

Sher KS, Mayberry JF. Female fertility, obstetric and gynaecological history in coeliac disease: a case control study. *Acta Paediatr Suppl*, 1996;412:76-77.

Martinelli P, Troncone R, Paparo F, Torre P, Trapanese E, Fasano C, et al. Coeliac disease and unfavourable outcome of pregnancy. *Gut*, 2000;46(3):332-335.

<http://www.greenmedinfo.com/blog/200-clinically-confirmed-reasons-not-eat-wheat>

http://www.naturalnews.com/037170_gm_wheat_liver_failure_gmo.htm