Functional Medicine University's Functional Diagnostic Medicine Training Program

Module 3 * FDMT 527A

Celiac Disease / Gluten Sensitivity/Gluten Intolerance

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http://www.FunctionalMedicineUniversity.com

Contents

Recommended Reading (Articles located on FMU website library):

Celiac Disease News; A Changing Environment and the Increasing Prevalence of Celiac Disease*; NIH*

The Immunology of Gluten Sensitivity Beyond the Intestinal Tract; *A.Vojdani, T.O'Bryan, G.H. Kellerman*

The Immunology of Immediate and Delayed Hypersensitivity Reaction to Gluten*; A. Vojdani, T.O'Bryan, G.H. Kellermann*

Natural History of Antibodies to Deamidated Gliadin Peptides and Transglutaminase in Early Childhood Celiac Disease*; Edwin Liu, Marcella Li, Lisa emery, Iman Taki, Kathy Barriga, Claudio Tibeti, George S. Eisenbarth, Marian J. Rewers, and Edward J. Hoffenberg*

Sprue is a general term for diseases that cause a decrease in absorption by the intestinal mucosa. *Nontropical sprue* is also called celiac disease, gluten-sensitive enteropathy, or idiopathic sprue. Nontropical Sprue results from the toxic effects of gluten. Gluten causes the destruction of the intestinal enterocytes and may destroy the microvilli and villi. *Tropical Sprue* is believed to be caused by a bacterial infection that causes inflammation of the intestinal mucosa. In both types of sprue, fat absorption is usually affected first, causing steatorrhea. As the disease progresses, absorption of protein and carbohydrates become impaired.

Nutrient deficiencies and associated pathology

- Malabsorption of fats, proteins, carbohydrates, calcium, vitamin K, folic acid and B12
- Muscle wasting
- Osteomalacia
- Decreased coagulation time
- Anemia

CELIAC DISEASE

Celiac disease is an inherited autoimmune disorder of unknown etiology. It is also a disease of malabsorption. Celiac disease damages the small intestine via an immunological reaction to gluten. The inflammation of the small intestinal lining then causes malabsorption. Once activated, the immune response may progress from damaging the small intestine to damaging other organ systems of the body. Environmental, immunologic and genetic factors seem to contribute to the disease. The disease may become active for the first time after surgery, pregnancy, childbirth, viral infection, or severe emotional stress.

Celiac disease originates as a result of a combination of both adaptive and innate immunity. There is a strong genetic predisposition to celiac disease, with the major risk attributed to the specific genetic markers known as HLA-DQ2 and HLA-DQ8 (HLA= Human Leukocyte Antigen). The genetic markers can induce an adaptive immune response to gluten. The innate immune response is orchestrated by the MHC type 1 (Major Histocompatibility Complex) that is on the surface of the enterocytes caused by stress and inflammation. The presence of these specific types of MHC type 1 receptors leads to activation of natural killer cells.

Celiac disease is the only autoimmune disease with a known trigger, that being *gluten*. Some of the proteins that make up gluten are called gliadin. It is the gliadin portion of the gluten that causes the immunological reaction in the small intestine. Wheat is the main source of gliadin. Barley and rye contain gliadin-like proteins, hordeins and secalins respectively, and can cause the same toxic immunological reaction. Oats contain gliadin-like proteins, called *avenins*; however, they usually cause a weak immunological reaction. Gliadin is made up of a long chain of amino acids, mainly glutamine. Gliadin is not completely digested by the brush border intestinal enzymes and long chains of amino acid remain intact. These larger chains enter the enterocytes, probably by increased intestinal permeability (leaky gut), and attach to an enzyme in the cell called *tissue transglutaminase*. The complex of tissue transglutaminase and long chains of glutamine initiates an immune reaction that damages the intestinal lining. This inflammatory reaction destroys the microvillus and villi causing a "flattening "of the lining. The flattening causes a significant decrease in surface area, which leads to malabsorption.

Celiac disease affects people in all parts of the world. Originally thought to be a rare childhood syndrome, celiac disease is now known to be a common genetic disorder. The range of the onset of symptoms is from the first year of life through the eighth decade. More than 2 million people in the United States have the disease, *or about 1 in 133 people*. Among people who have a first-degree relative (parent, sibling or child) diagnosed with celiac disease, as many as 1 in 22 people have the disease and people with second-degree relatives diagnosed with celiac disease (aunt, uncle or cousin), as many as 1 in 39 people have the disease. Up to 60% of children and 41% of adults with celiac disease may be asymptomatic.

There is an existing classification of patients with supposed subphenotypes. Four possible presentations of celiac disease are presently recognized:

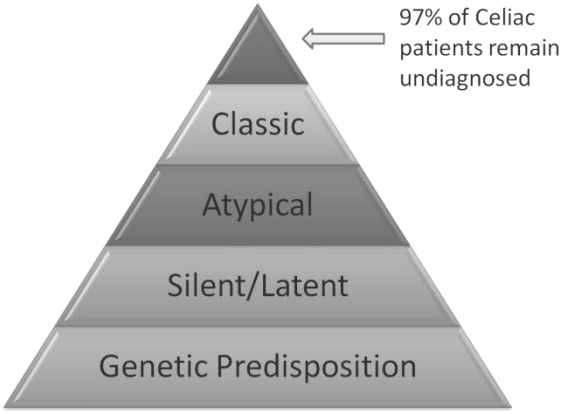
- **Classic celiac disease** (typical celiac disease) – Classic celiac disease is dominated by symptoms and sequelae of gastrointestinal malabsorption. The diagnosis is established by serological testing, biopsy evidence of villous atrophy and improvement of symptoms on a gluten-free diet.

- **Atypical celiac disease** (extraintestinal) – Atypical celiac disease is characterized by few or no gastrointestinal symptoms with extraintestinal manifestations predominate (thyroid, skin, etc). Atypical is the *most common presentation* of celiac disease. As with classic celiac disease, the diagnosis is established by serological testing, biopsy evidence of villous atrophy and improvement of symptoms on a gluten-free diet.

- **Silent celiac disease** (asymptomatic celiac disease) – Silent celiac disease refers to individuals who are asymptomatic but have a positive serological test and villous atrophy on biopsy. These individuals usually are detected via screening of high risk individuals, or villous atrophy detected by endoscopy and biopsy taken for a different reason. Silent celiac disease may develop to classic or atypical later in life.

- **Latent celiac disease** – Individuals with latent celiac disease possess genetic markers and may have a positive serological test, however, the intestinal mucosa is morphologically normal. These individuals are usually asymptomatic, but they may develop symptoms and/or histological changes later in life.

***NOTE**: These classifications do not take into account the individuals with who suffer from gluten intolerance or gluten sensitivity. Discussion of these conditions will follow.*



Celiac disease generally presents with a variety of seemingly unrelated symptoms making it difficult to diagnose. Symptoms also vary depending on a person's age and the degree of damage to the small intestine. Many adults have the disease for a decade or more before they are diagnosed. The average length of time to diagnose celiac in the United States is about four years. The longer a person remains undiagnosed, and therefore untreated, the greater the chance of developing autoimmune disorders, neurological disorders and cancer. As functional medicine practitioners, we have the opportunity to decrease the length of time establishing a diagnosis by utilizing a **detailed patient history (esp. family history),** nutritional assessment, comprehensive examination and basic and advanced laboratory testing.

Signs and Symptoms of Celiac Disease

There are more than two hundred signs and symptoms associated with Celiac Disease. Keep in mind that the disease may have no symptoms at all. The symptoms may or may not cause digestive dysfunction. The disease can present as iron deficiency anemia or infertility, to name a few. There are two categories of signs and symptoms: those due to *malabsorption*, and those due to *malnutrition* (deficiencies of fats, proteins, carbohydrates, vitamins and minerals).

1. Recurring abdominal cramps, gas and bloating
2. Chronic diarrhea
3. Vomiting
4. Liver and biliary dysfunction (fatty liver/ sclerosing cholangitis)
5. Fatigue
6. Weight loss
7. Greasy, gray or tan, foul-smelling stools that are high in fat content (steatorrhea)
8. Anemia (iron-deficiency that does not respond to treatment and B12 deficiency)
9. Skin rash (dermatitis herpetiformis)
10. Stunted growth in children (delayed puberty)
11. Osteopenia or osteoporosis (lack of vitamin D and calcium)
12. Infertility (recurrent miscarriage)
13. Amenorrhea
14. Sores in the mouth
15. Peripheral neuropathy [(tingling and numbness in the legs and arms) B-complex deficiency]
16. Anxiety/depression
17. Joint pain
18. Fluid retention (low serum total protein)
19. Bruising easily (lack of vitamin K)

Possible Presenting Symptoms of Celiac Disease Based on Age

- **Infants** – diarrhea, steatorrhea, abdominal cramps, abdominal distension, irritability, muscle wasting, failure to thrive and failure to grow. The usual onset of symptoms occurs shortly after the introduction of gluten containing cereals.

- **Children** – diarrhea/constipation, steatorrhea, flatulence, short stature, behavioral difficulties, delayed puberty, abdominal pain and learning difficulties.

- **Adult** – May have many of the signs and symptoms listed above or only a few such as iron deficiency anemia, abnormal bleeding and/or osteoporosis. A common misdiagnosis is irritable bowel syndrome.

The Celiac Disease Center at Columbia University Medical Center in New York obtained data on 1138 **adults** with biopsy proven celiac disease. The following is a synopsis of their research:

- Majority of individual were diagnosed in the four to sixth decades
- Female predominance 2.9 to 1 (less marked in the elderly)
- Diarrhea presented in 85%
- Symptoms were present a mean of 11 years prior to diagnosis
- Iron deficiency anemia 8 to 15 %
- Peripheral neuropathy 8%
- Less common presentations were as follows: elevated serum amylase, hypoalbuminemia, hypocalcemia, vitamin deficiency states, hypospenism, dental enamel defects and infertility.

Diseases Associated With Celiac Disease (This List Is Not All Inclusive)

NOTE: *Patient's with the following symptoms and/or diseases warrant screening for celiac disease. Additionally, anyone suffering unexplained, non-resolving illness may benefit from a screening. Screening is especially important for first degree relatives of patients with celiac disease (parent, child or sibling). Second degree relatives should also be screened.*

- *Gastrointestinal dysfunctions*

  - Recurring abdominal pain
  - Chronic diarrhea (not resolving in three weeks)
  - Steatorrhea
  - Flatulence
  - Constipation
  - Chronic bloating
  - Ulcerative jejunoileitis (rare)
  - Ulcerative colitis

- *Systemic dysfunction*
  - Anemia (remains unresolved with treatment/unexplained)
  - Chronic fatigue
  - Infertility
  - Osteopenia/osteoporosis
  - Unexplained weight loss
  - Persistent muscle cramps
  - Tooth enamel decay

- *Autoimmune Disease*
  - Dermatitis herpetiformis
  - Aphthous stomatitis (recurrent mouth ulcers)
  - Peripheral neuropathy, ataxia and epilepsy
  - Rheumatoid Arthritis
  - Autoimmune thyroid disease
  - Systemic lupus
  - Sjogren's syndrome
  - Insulin-dependent diabetes (Type 1 diabetes)
  - Chronic active hepatitis, primary biliary sclerosing, cholangitis
  - Addison's disease

- *Malignant Disease*
  - Small intestinal Lymphoma [Non-Hodgkin's] (anemia, blood loss, abdominal pain, weight loss, fever, small intestinal obstruction)
  - Small intestinal adenocarcinoma
  - Esophageal carcinoma
  - Papillary thyroid cancer

- The prevalence of celiac disease is increased in: Down syndrome, Turner syndrome and Williams Syndrome.

- *Dermatitis Herpetiformis*: Skin manifestation affecting 15 to 25% of people with celiac disease

Dermatitis herpetiformis is characterized by small, clustered papules and vesicles that erupt symmetrically on the elbows, knees, buttocks, back and scalp. Men may also have oral or genital lesions. A burning sensation may precede lesion formation. DH is caused by the deposit of immunoglobulin A (IgA) in the skin, which triggers further immunologic reactions resulting in lesion formation. DH is an external manifestation of an abnormal response to gluten, in which IgA antibodies form against the skin antigen epidermal transglutaminase. A skin biopsy is the first step in diagnosing DH. Direct immunofluorescence of clinically normal skin adjacent to a lesion shows granular IgA deposits in the upper dermis. Histology of lesional skin may show microabscesses containing neutrophils and eosinophils but may only reveal excoriation due to the intense itching patient's experience. Skin biopsies performed on the affected skin are always positive for IgA deposition. Blood test for antiendomysial or anti-transglutaminase antibodies may also suggest celiac disease.

HEALTHY GUT ILLUSTRATION (see next page)

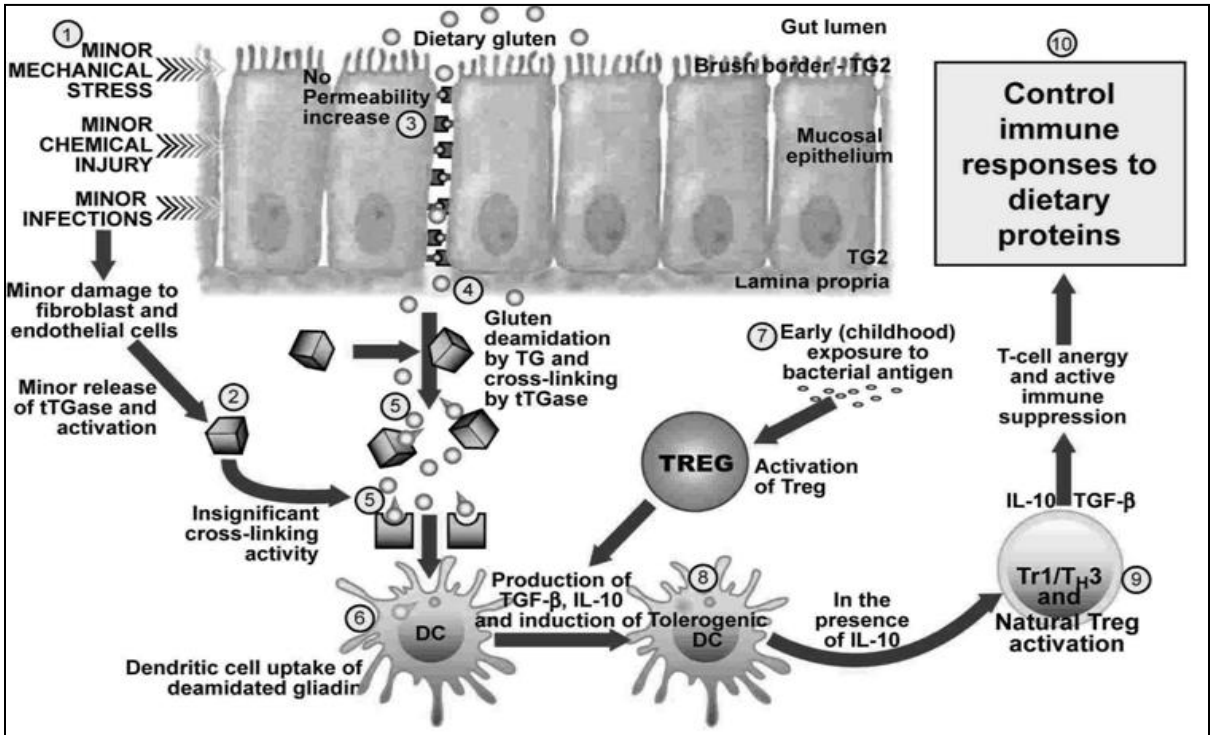
Healthy Gut response to gluten exposure (and similar substances) in the presence of minor stress, minor chemical injury (toxins), and minor infections.

A. Stress, chemical injury (toxins) and infection are minor

B. Very few gluten molecules pass the "healthy" gut wall even in the presence of minor stress, toxins and infection

Early childhood exposure to bacteria produces a protective mechanism that suppresses immune response (allergies and intolerances). This healthy process takes place with most foods that enter the normal gut. Simply put, children need some normal common sense exposure to dirt and bacteria to build their immunities.

HEALTHY GUT ILLUSTRATION



Cellular and molecular induction of immune tolerance to dietary proteins (gliadin).

Although HLA-DQ2 or HLA-DQ8 is found in roughly 30% of the western population, celiac sprue is encountered in 1 out of 50 carriers. Most carriers of these genes, like the rest of the population, harbor some form of immune protection, as shown in this figure.

In the absence of major mechanical and chemical stress or infection (1), no damage is done to fibroblasts and endothelial cells, and only small quantities of tissue transglutaminase are released into the environment (2).

Since under these conditions the tight junctions are in perfect shape (3), only a few gliadin molecules may survive digestion and be transported across the mucosal epithelium (4).

If these molecules of gliadin are deamidated by transglutaminase (5), the key regulator of the immune system called dendritic cells or antigen-presenting cells (6) prime T cells for anergy or tolerance.

Early exposure to dietary proteins and bacterial antigens such as LPs (7) can activate regulatory T cells to produce TGF-b and IL-10, inducing activation of tolerogenic DCs (8) to control immune response to dietary proteins (gliadin). Further activation of TR_1, TH_3 and natural Treg (9) by IL-10 results in induction of central or peripheral tolerance (10).

The regulatory T cells are divided into two major groups:

a - Natural Tregs, which act in a contact-dependent fashion, and express CD25 and transcription factor $FOXP^3$;

b - Adoptive Treg Type1 cells (TR_1), which function in a contact-independent manner and may or may not express CD25 and $FOXP^3$. The TR_1 and TH_3 cells preferentially synthesize immunosuppressive cytokines IL-10 and TGF-b respectively in order to maintain homeostasis of responses to foreign antigens, including gliadin.

GLUTEN SENSITIVITY ALLERGY ILLUSTRATION (see next page)

Gluten Sensitivity (IgE type 1 immediate hypersensitivity reaction) to toxins or strenuous exercise

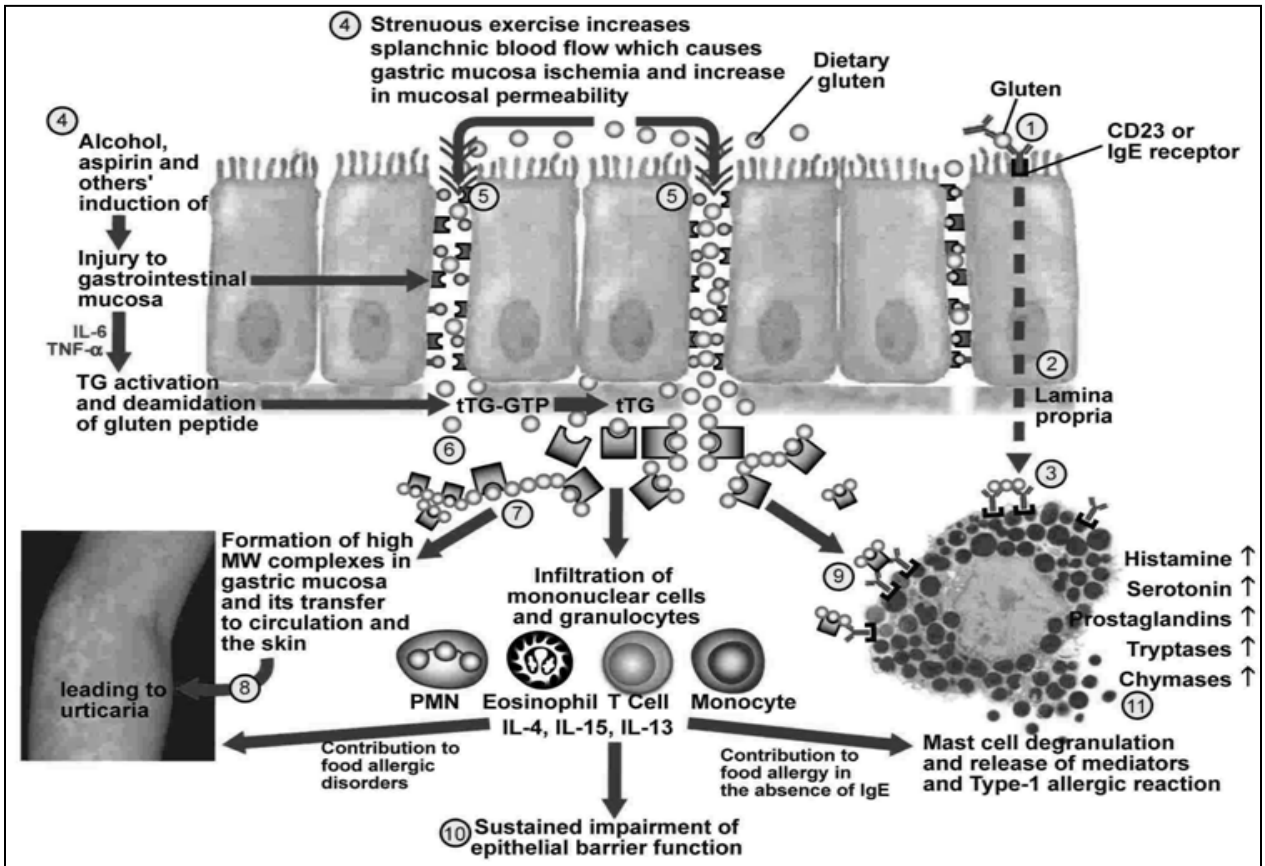
A. This is an allergic reaction

B. This reaction pathway can be triggered by alcohol, chemical toxins found in food along with gluten, several classes of medications including aspirin and also strenuous exercise.

C. The gut is significantly leaky

D. Continued exposure to gluten causes the mast cells to release histamine and other primary and secondary mediators. This may trigger urticaria, respiratory distress and other symptoms related to Type 1 hypersensitivity reactions

GLUTEN SENSITIVITY ALLERGY ILLUSTRATION



Schematic presentation of the pathophysiology of the immediate hypersensitivity reactions (Type 1 allergy) of the intestine.

This hypersensitivity reaction may occur by the binding of dietary peptides (gluten) to low affinity IgE receptor CD23, which is expressed on the epithelium of the small intestine (1), facilitating uptake of antigen in an IgE-independent manner (2).

Gluten cross-links to IgE on the surface of MAST cells to induce degranulation (3). This MAST cell degranulation could be induced by strenuous exercise, alcohol and medication [aspirin] (4), causing injury to gastrointestinal mucosa and an increase in mucosal permeability (5).

Under these conditions, parts of gluten that are resistant to processing by luminal and brush-border enzymes will survive digestion and be transported across the mucosal epithelium as polypeptides.

Upon activation of transglutaminase in the subepithelial region (6), many gliadin peptides form high molecular weight complexes with transglutaminase (7) which can be transferred into the circulation and the skin, leading to urticaria (8).

These complexes can also bind to IgE receptors on MAST cells and induce further degranulation (9). Finally, infiltration of granulocytes, mononuclear cells and their cytokines can contribute to late phase responses, which results in the impairment of epithelial barrier function (10).

Also, products released from MAST cells, including histamine, serotonin, prostaglandins, tryptases and chymases (11) have been shown to have direct and indirect effects (via activation of the enteric nerve) on epithelial ion secretion, barrier function, and intestinal motility. Gluten Sensitivity

GLUTEN INTOLERANCE ILLUSTRATION (see next page)

Gluten Intolerance Reaction to stress, chemical injury (toxins), and infections *without* gene involvement. This can lead to widespread autoimmune vulnerability.

A. The infections listed on the illustration are simply the agents observed in research studies. They are not necessarily the only infections that may contribute to this type of reaction.

B. Stress, chemical injury (toxins) and/or infections contribute significantly to gut wall damage.

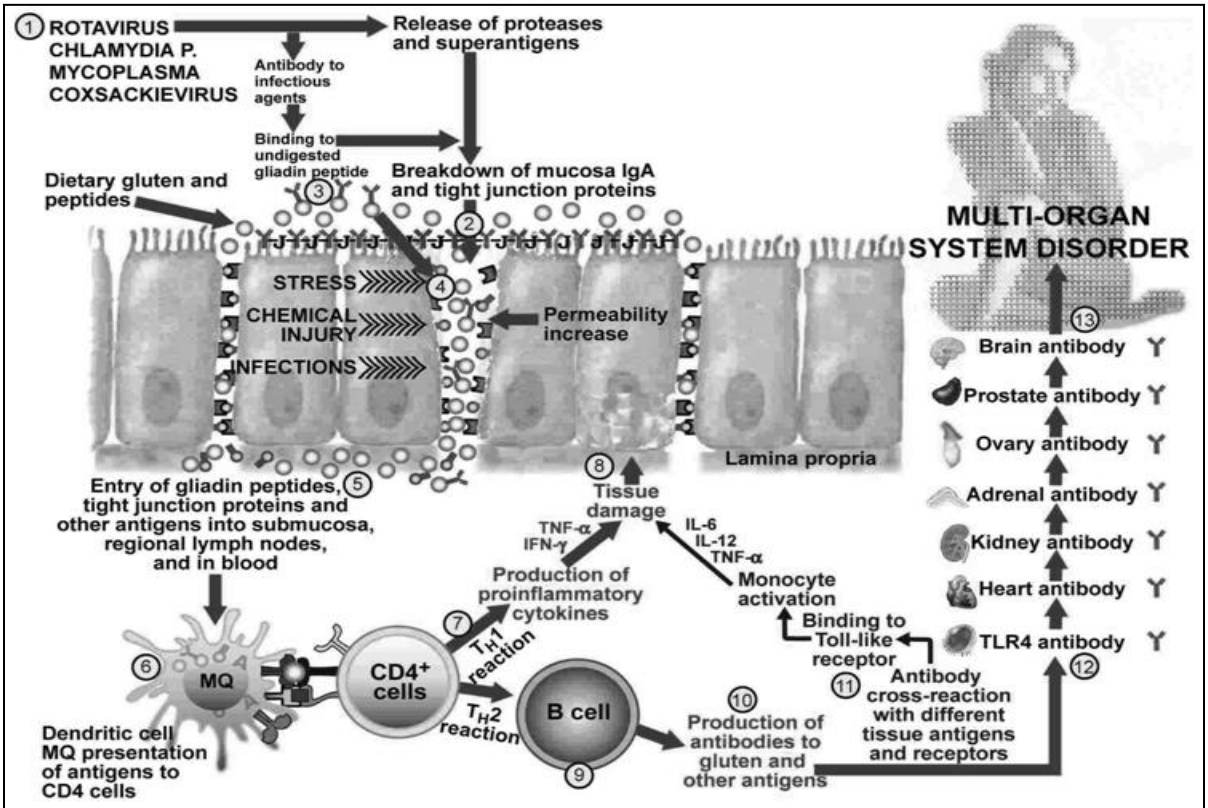
C. The gut wall is badly damaged and leaky. This allows many large food peptides to cross the gut wall before sufficient digestion can appropriately break them into very tiny pieces. These pieces enter the cell and are rejected and pushed back to the cell surface to be recognized and destroyed by killer cells.

D. *This reaction can happen to anyone.* This gluten or other food intolerance pathway does *not* require a predisposing gene.

E. This pathway produces antibodies which "stick" to many body or organ tissues. This marks that tissue for autoimmune destruction by various killer cells. (The list of effected body parts is not complete on the illustration)

F. Since this pathway involves cross reactions with many body/organ tissues and eventually with multiple foods, testing is recommended for a number of gluten antibodies and milk and other problematic food proteins, plus several susceptible organs and other related markers.

GLUTEN INTOLERANCE ILLUSTRATION



Depiction of immunological mechanisms underlying gluten intolerance and its immunopathological consequences.

Precipitation of gluten intolerance appears to be preceded by acute gastroenteritis symptoms induced by infections such as rotavirus and others(1).

Rotavirus and its super-antigens can break down mucosal IgA directly (2) or indirectly by the local production of anti-rotavirus antibody. Due to partial linear homology or cross-reactivity between rotavirus protein and a-gliadin, the anti-rotavirus antibody binds to gliadin and forms complexes with it (3).

The combination of infection antibody cross-reactivity with gliadin and additional stressors can severely impair mucosal integrity (4) and the entry of gliadin peptides, tight junction proteins and other antigens into the submucosa, regional lymph nodes, and the blood (5).

Gliadin peptides, rotavirus antigens, rotavirus antibody bound to gliadin, and tight junction proteins are presented by dendritic cells with or without HLA-DQ2/DQ8 to $CD4^+$ cells (6).

This antigenic presentation results in driving the cell $CD4^+$ response either towards TH_1 reaction (7), production of proinflammatory cytokines, mucosal cell destruction and autoimmunity (8); or towards TH_2 response B-cell activation (9) and antibody production against gluten, rotavirus, and tight junction proteins (10).

Cross-reaction of these antibodies with cell receptors such as toll-like receptor (11) and tissue antigens such as heart kidney, adrenal gland, ovary, prostate, brain and others (12) results in further tissue damage and multi-organ system disorders (13).

CELIAC DISEASE ILLUSTRATION (see next page)

Gene mediated response to stress, chemical injury (toxins), and infections. This leads to widespread autoimmune, malabsorptive, inflammatory and other vulnerabilities.

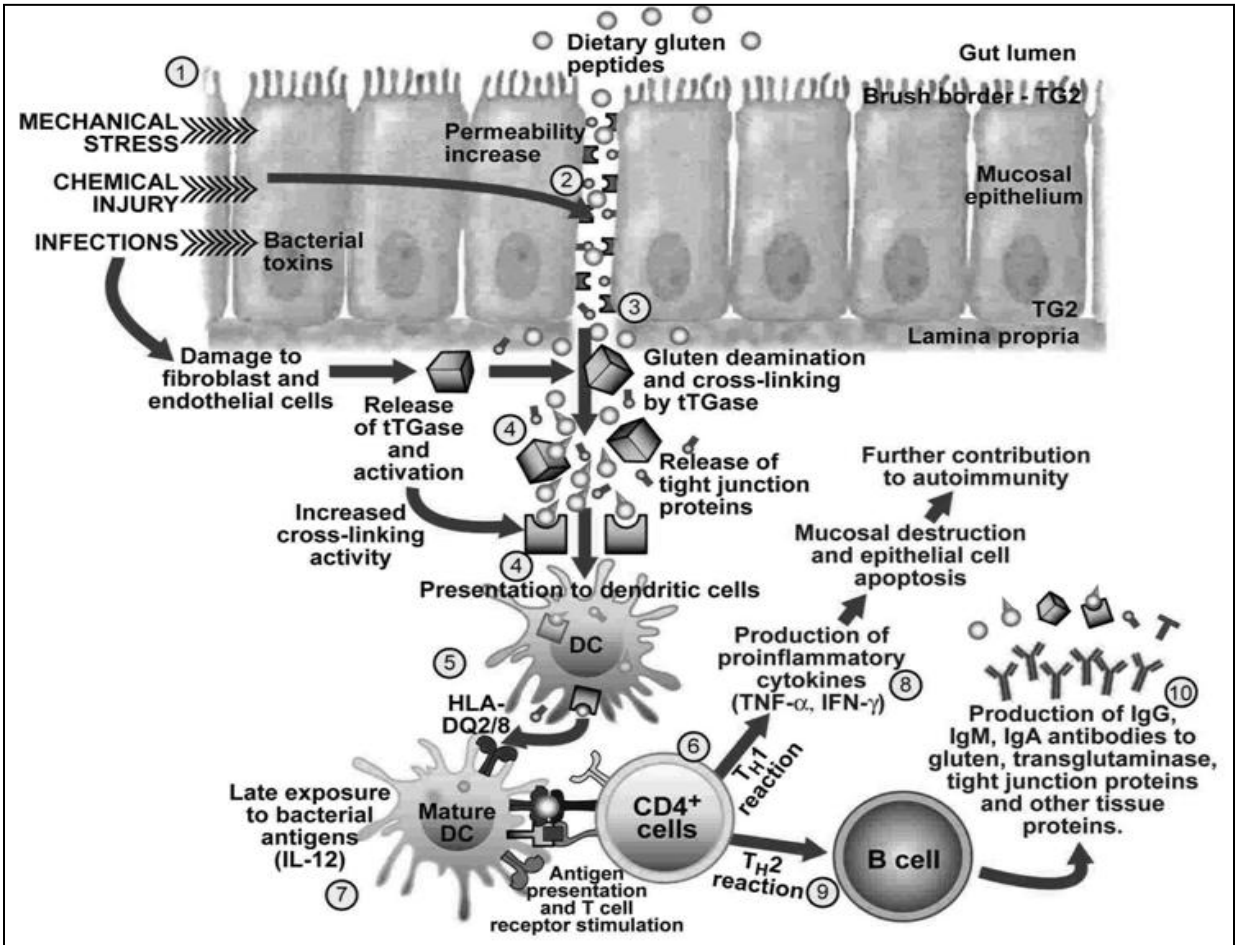
A. Stress, chemical injury (toxins) and/or infections are significant.

B. Gut damage is significant and more gluten molecules pass through the gut - "leaky gut". This activates antibodies to form in the blood stream against one or more gluten/gliadin/wheat related molecules.

C. Specific genes contribute to this pathway, making it easier for antibodies to be formed. The antibodies bind to many substances including body tissues that "look similar" to gluten (molecular mimicry). The antibodies then attract killer cells to the substances with which they bind including body tissues causing an autoimmune reaction.

D. Formation of antibodies to various products of this process cross reacts with many tissues to produce autoimmunity.

CELIAC DISEASE ILLUSTRATION



Depiction of the intestinal mucosa with emphasis on the factors involved in the development of celiac disease in individuals with HLA DQ2/DQ8-positive.

Infection, mechanical and chemical stress (1) can impair mucosal integrity (2).

The parts of gluten that are resistant to brush-border enzymes will survive digestion and can be transported across the epithelial barrier as polypeptides (3).

Tissue transglutaminase in the intestinal mucosa (lamina propria) become activated and deamidate gluten peptides. Some of the deamidated gliadins may cross-link to transglutaminase and form complexes of gliadin with TG (4).

Deamidated gliadin peptide by itself, deamidated gliadin peptide cross-linked to TG, and released tight junction proteins are presented by dendritic cells or antigen-presenting cells as well as B cells (5) which carry HLA-DQ2 or DQ8 molecules to the CD4+ T cells in the lamina propria (6).

It is believed that this antigenic presentation is enhanced in an individual with later-in-life exposure to bacterial antigens whose mature dendritic cells produce significant amounts of interleukin-12 [IL-12] (7).

This antigenic presentation results in driving the CD4+ cell response either towards TH1 reaction, production of inflammatory cytokines (8), mucosal cell destruction and autoimmunity, or, toward TH2 response B-cell activation (9), and antibody production against deamidated gluten, transglutaminase gliadin cross-linked to transglutaminase and different tissue antigens T (10).

Testing For Celiac Disease

- **Biopsy** - Biopsy of the proximal small intestine remains the gold standard in the diagnosis of celiac disease. The pathology report should indicate the extent of crypt hyperplasia, villous atrophy and intraepithelial lymphocyte infiltration. *See article on Marsh classifications on FMU website library*.

- **Genetic Testing** – Genetic (DNA) testing is used to determine whether or not an individual is carrying the HLA-DQ2 and HLA-DQ8 genes. Between 35 and 40% of the United States population carry the alleles for celiac disease. These genes are responsible for the development of celiac disease. A person who has a positive genetic test means that they have a predisposition for celiac disease - it does not mean they have the disease. Follow-up serological and/or biopsy are used to confirm the diagnosis. If the genetic test is negative, another cause for the patient's symptoms must be investigated.

- **Serological Testing**- Serologic tests look for antibodies produced in response to celiac disease. The most sensitive serological tests for celiac disease are based on the immunoglobulin A Isotypes (IgA).

  - Tissue Transglutaminase Antibodies (tTG IgA and tTG IgG) – tTG IgA is considered an excellent first-line marker. It has a high sensitivity and specificity in untreated individuals.

  - Endomysial Antibodies (EMA) – Endomysial IgA has a specificity of close to 100% and a sensitivity of about 90%. There is a close correlation between the titer of endomysial antibodies and mucosal damage. This test must be interpreted by an experience laboratory.

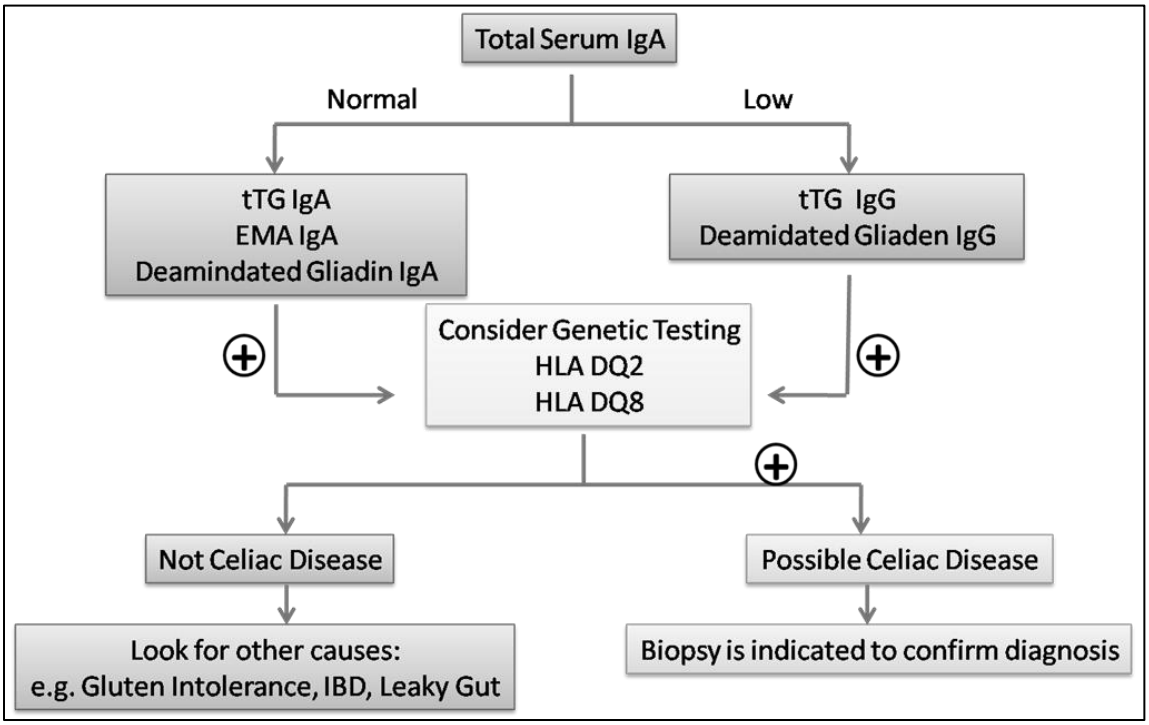
  - Deamidated Gliadin Antibodies (IgA/IgG) – these test measures antibodies to a fragment of gliadin that has been acted upon by tissue transglutaminase. These tests are relative new and may be more sensitive than tTG antibodies. *Please read the article: "Natural History of Antibodies to Deamidated Gliadin Peptides and Transglutaminase in Early Childhood Celiac Disease".*

  - Gliadin Antibodies (IgA and IgG) have a lower sensitivity and specificity for celiac disease and are not as reliable. Interfering factors include other gastrointestinal diseases such as Crohn's disease, colitis and severe lactose intolerance. Anti-Reticulin Antibodies (ARA) is also not as specific or sensitive as the other antibodies mentioned.

  - Selective IgA deficiency is a genetic disorder that causes immunodeficiency of IgA. IgA deficiency is more common in patients with celiac disease. Total serum IgA is measured to identify patient that have selective IgA deficiency. tTG IgA and emdomysial IgA will most likely be negative with low levels of total serum IgA; however, tTG IgG may be positive. You also need to order gliadin IgG antibodies and deamidated gliadin IgG antibodies, if total serum IgA is low.

Note: Levels of anti-tTG and EMA will wax and wane according to the amount of gluten ingested or lack of. You may want your patient to ingest some gluten containing foods prior to testing if they can tolerate it. These antibodies can also be used for patient compliance.

CELIAC DISEASE DIAGNOSTIC ALGORITHM



Recommended Functional Medicine Testing

- CBC with iron panel – evaluate for iron deficiency anemia and pernicious anemia
- CMP – evaluate for blood chemistry changes in electrolytes, protein and calcium levels. (total protein may be low, globulin may be high, low calcium)
- CRP – evaluate for inflammation
- ESR – evaluate for inflammation
- Vitamins D, B12, homocysteine, methylmalonic acid
- Thyroid Panel
- Functional Medicine Stool Analysis – evaluate for steatorrhea, pancreatic enzyme insufficiency, bacterial overgrowth and intestinal pathogens
- Adrenal Stress Index (This test will also evaluate for salivary sIgA levels)
- Dexa Test – evaluate bone density
- Food Allergy Testing – IgE, IgG, IgG4 (IgG4 antibody is a more clinically relevant marker of chronic food-immune reactions and possible intestinal hyperpermeability.)
- Lactulose-Mannitol Test (Intestinal Hyperpermeability Test)
- BMI

Treatment Recommendations

The only treatment for celiac disease is lifelong gluten-free diet. A gluten-free diet means not ingesting any food that contains wheat (including spelt, triticale, and kamut) rye and barley. Pancreatic enzyme supplementation can enhance the clinical benefit of a gluten-free diet during the first few months after confirming the diagnosis. Continued pancreatic supplementation after that time period has not proven to be beneficial.

Note: *Gluten is also used in some medications and cosmetics*

The following are the six key elements in the management of individuals affected by celiac disease as outline by the *NIH Consensus Development Conference on Celiac Disease 2004*:

Consultation with a skilled dietitian
Education about the disease (label reading is a must)
Lifelong adherence to a gluten-free diet
Identification and treatment of nutritional deficiencies
Access to an advocacy group
Continuous long-term follow-up by a multidisciplinary team

As seen in the previous illustrations, it is paramount that all gastrointestinal dysfunctions be treated whether the patient has celiac disease, gluten intolerance or gluten allergy. This requires using the protocols outlined in the GI module that focuses on healing and repairing the mucosal lining. *Treat all nutritional deficiencies*.

Follow-up laboratory testing is paramount. Follow-up testing can disclose:

1. Patient's compliance to gluten-free diet
2. Effectiveness of treating nutritional deficiencies
3. Effectiveness of treating gastrointestinal dysfunctions
4. Allows for the possible of a concomitant disease (e.g. iron deficiency from occult blood loss)

I recommend retesting all abnormal finding in six to eight weeks.

Celiac Disease and Gluten Sensitivity/Intolerance/Allergy Resources

The Gluten-Free Diet: Some Examples

In 2006, the American Dietetic Association updated its recommendations for a gluten-free diet. The following chart is based on the 2006 recommendations. This list is ***not*** complete, so people with celiac disease should discuss gluten-free food choices with a dietitian or physician who specializes in celiac disease. People with celiac disease should always read food ingredient lists carefully to make sure that the food does not contain gluten.

Adapted from the following resource: Thompson T. *Celiac Disease Nutrition Guide.* 2nd ed. Chicago: American Dietetic Association; 2006. Used with permission. For a complete copy of the *Celiac Disease Nutrition Guide,* please visit *www.eatright.org*.

Allowed Foods

Amaranth
Arrowroot
Buckwheat
Cassava
Corn
Flax
Indian rice grass
Job's tears
Legumes
Millet
Nuts
Potatoes
Quinoa
Rice
Sago
Seeds
Soy
Sorghum
Tapioca
Wild Rice
Yucca

Foods To Avoid

Wheat
- Including einkorn, emmer, spelt, kamut
- Wheat starch, wheat bran, wheat germ, cracked wheat, hydrolyzed wheat protein

Barley
Rye
Triticale (a cross between wheat and rye)

Other Wheat Products

Bromated flour
Durum flour
Enriched flour
Farina
Graham flour
Phosphated flour
Plain flour
Self-rising flour
Semolina
White flour

Processed Foods that May Contain Wheat, Barley, or Rye*

Bouillon cubes
Brown rice syrup
Chips/potato chips
Candy
Cold cuts, hot dogs, salami, sausage
Communion wafer
French fries
Gravy
Imitation fish
Matzo
Rice mixes
Sauces
Seasoned tortilla chips
Self-basting turkey
Soups
Soy sauce
Vegetables in sauce

* Most of these foods can be found gluten-free. When in doubt, check with the food manufacturer.
www.nih.gov

Hidden Gluten

Gluten is often hidden through the processing of ingredients in foods. The following terms could indicate that the food contains gluten, unless it states 'gluten free':

Soy Sauce
Malt or Malt Flavoring (unless it is derived from corn)
Cereals (unless made with rice, corn, potato, or soy flours)
Hydrolyzed Vegetable Protein (unless it is made from soy or corn)
Vegetable Protein (unless from soy or corn)
Modified Starch or Modified Food Starch (unless from corn, tapioca, arrowroot, or maize)
Stabilizer Starch Flavoring Emulsifier Hydrolyzed Plant Protein

Gluten Free Food Resources

Many recipes containing gluten ingredients can be converted with the use of gluten-free products. Although there are many resources available, a few are listed below:

Gluten-Free Pantry (www.glutenfreepantry.com): Specializes in gluten-free all purpose flour and ready- made mixes for bread, pizza and cake mixes.

Bob's Red Mill (www.bobsredmill.com): Specializes in gluten-free and organic flours and mixes

Ener-G Foods (www.ener-g.com): Specializes in a number of gluten-free products

Glutino (www.glutino.com) : Specializes in a variety of gluten free products including breads, pastas, cookies, crackers and other snacks.

(www.allergygrocer.com), (www.consorzio.com) : Offers a variety of gluten-free condiments

(www.applegatefarms.com): Many of their cold-cuts and hot-dogs are gluten-free

Celiac Disease Organizations

American Celiac Disease Alliance
2504 Duxbury Place
Alexandria, VA 22308
Phone: 703-622-3331
Fax:
Email: info@americanceliac.org
Web: www.americanceliac.org

American Dietetic Association
120 South Riverside Plaza, Suite 2000
Chicago, IL 60606
Phone: 1-800-877-1600
Fax: 312-899-4739
Email:
Web: www.eatright.org

Celiac Disease Foundation
13251 Ventura Boulevard, #1
Studio City, CA 91604-1838
Phone: 818-990-2354
Fax: 818-990-2379
Email: cdf@celiac.org
Web: www.celiac.org

Celiac Sprue Association/USA Inc.
P.O. Box 31700
Omaha, NE 68131-0700
Phone: 877-272-4272
Fax: 402-643-4108
Email: celiacs@csaceliacs.org
Web: www.csaceliacs.org

Children's Digestive Health and Nutrition Foundation
P.O. Box 6
Flourtown, PA 19031
Phone: 215-233-0808
Fax: 215-233-3918
Email: mstallings@naspghan.org
Web: www.cdhnf.org or www.celiachealth.org

Gluten Intolerance Group of North America®
31214 – 124th Ave SE
Auburn, WA 98002
Phone: 206-246-6652
Fax: 206-246-6531 Email: info@gluten.net
Web: www.gluten.net

National Foundation for Celiac Awareness
P.O. Box 544
Ambler, PA 19002–0544
Phone: 215-325-1306
Fax: 215-283–0859
Email: info@CeliacCentral.org
Web: www.CeliacCentral.org

North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition
P.O. Box 6
Flourtown, PA 19031
Phone: 215-233-0808
Fax: 215-233-3918
Email: naspghan@naspghan.org
Web: www.naspghan.org

Helpful Links

www.livingwithout.com

www.celiac.com

www.glutenfreemall.com

www.glutenfreerestaurants.com

www.glutenintolerance.com

References

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2. University of Chicago Celiac Disease Center; 5839 S. Maryland Ave., Chicago, IL 60637; Overview of Celiac Disease article; www.CeliacDisease.net
3. The Journal of Family Practice; *What Blood Tests Help Diagnose Celiac Disease*; article December 2006 (Vol 55, No. 12); www.jfponline.com
4. U.S. Department of Health and Human Services; National Institutes of Health; Celiac Disease; National Digestive Diseases Information Clearinghouse; *What is celiac disease?*
5. NIH Consensus Development Conference on Celiac Disease; June 28-30, 2004; http://consensus.nih.gov/2004/2004CeliacDisease118html.htm
6. Celiac Disease, A Hidden Epidemic; Peter H.R. Green, M.D.
7. Mosby's Manual of Diagnostic and Laboratory Tests; 3rd ed.; Pagana & Pagana
8. Laboratory Evaluations for Integrative and Functional Medicine; 2nd ed.; Richard S. Lord & J. Alexander Bralley
9. Encyclopedia of Natural Medicine; Revised 2nd ed.; Michael Murray, N.D., and Joseph Pizzorno