The Gluten Sensitivity Spectrum

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September, 2011
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You must first arm yourself with knowledge!

The most important thing to remember is this:

Celiac disease and gluten intolerance are not the same thing.
Gluten Sensitivity ≠ Celiac Disease

Science Finally Confirms Gluten Sensitivity

A new double blind, randomized, placebo controlled study published in the *American Journal of Gastroenterology* confirms the presence of gluten sensitivity in the absence of celiac disease. This is the first study of its kind confirming the existence of gluten intolerance in the absence of celiac disease.

**Source:**

• Gluten Sensitivity has traditionally been used synonymously with Celiac disease because that has been the focus of research.

• These terms have been created in the medical literature to separate Celiac Disease from Gluten Sensitivity
  • Non-Celiac Gluten Sensitivity – Dr. Marsh
  • Gluten Syndrome – Rodney Ford, M.D.
Unpublished data from Dr. Kenneth Fine, laboratory director at Enterolab, speculates that one in three have some degree of gluten intolerance!
Clinical symptoms of Celiac disease taught in graduate school are extreme weight loss, diarrhea, stomach pain, bloating, and vomiting.

In actuality symptoms can be and usually are systemic and we now know that different people respond in different ways.
On Celiac vs. Gluten Sensitivity

“Recent studies are showing the gluten sensitivity may be much more common than previously thought. It may, in fact, be a separate disease entity that involves different organs and different mechanisms than celiac disease. While there is no doubt that the condition exists, the lack of definite criteria for a diagnosis has resulted in a skeptical attitude on the part of many doctors.” He goes on to say: “The acceptance of gluten sensitivity as a valid condition has evolved.”

Dr. Peter Green - Director of The Celiac Disease Center at Columbia University
On Celiac vs. Gluten Sensitivity

60-70% of those who think they have celiac disease and seek help from his research center are actually gluten sensitive – they do not have celiac disease.

Communication from Dr. Alessio Fasano – University of Maryland Celiac Research Center

Courtesy of Dr. Vikki Petersen (Author of the Gluten Effect)
Why is the focus primarily on celiac disease?

Historical landmarks relating to digestive illnesses (references in process to be posted at www.glutensensitivity.net history page)

BC
The book of Exodus refers to related general and digestive symptoms in reference to bread and grains.

100 AD
Comment describing celiac symptoms in ancient medical literature

300 AD
Comment and description by Aretaeus, refers to “Coeliac disease”, meaning “abdominal”.

1855
Dr. Gull Guy’s Hospital Reports, symptoms described symptoms of gluten intolerance.

1887
Dr. Samuel Gee, “We must never forget that what the patient takes beyond his power to digest does harm.”

1850’s+
Bechamp-Pasteur debate re: microbiology/vaccines. Continues for many years. Bechamp’s predictions fulfilled.

1850’s+
Processed flour, sugar and commercially canned vegetables and milks become more widely available with industrialization.

1900
Coronary Heart disease is no more than 10% of annual cause of death from all causes. Butter consumption 18 lbs/person

1908
Drs. Emmett Holt and Christian Horsley publish “On Infantile Diseases from Chronic Intestinal Infecions”

1910
Myocardial infarction is responsible for no more than 6000 deaths in 1930.

1911
Crisco, a synthetic substitute for animal tallow, lard and beef fat. Has a long shelf life.

1914
Dr. Paul D. White of Harvard U.

1920-51
Holt reads “Prudent Diet” and studies the diet of 350 Highlanders for better nutrition.

1922
Dr. Robert G. Heberlein, “Science of Medical Nutrition”

1930
Myocardial infarction is responsible for no more than 8000 deaths in 1930.

1932
Dr. B.B. Crohn speaks of “new intestinal disorder” he calls “regional ileitis”, now called Crohn’s Disease.

1939
Dr. Weston A. Price publishes his 10 year travel research “Nutrition and Physical Degeneration”, detailing his comparisons of health and diets of isolated cultures with “modern” societies, and related butter and soil fertility studies.

1940-50
Dr. Willem Dicke notices certain patients’ digestion improves during grain shortages in Holland during World War 2. Their illnesses relapse when grain is again available. This turns attention to gluten grains.

1949
Drs. Sidney and Merrill Haas successfully treat 600 cases of “celiac disease” with the Specific Carbohydrate Diet.

1950
Butter consumption has dropped in the US from 18 lbs/person in 1900 to 10 lbs/person per year, 1950’s. Hydrogenated vegetable oils replace butter. Coronary heart disease is now the leading cause of death, 30%.

1951
Drs. Haas publish “The management of Celiac Disease” in 1951, focusing on certain complex dietary sugars and starches.

1952
University of Birmingham tests ten children and concludes that gluten, not starch is the culprit. The new focus on gluten widens the number of foods allowed, but few patients meet the diagnostic criteria and recognized symptoms are narrow, mainly wasting diarrhea and failure to thrive in children. They are diagnosed with “gluten induced celiac disease” via early blood and later newly developed capsule biopsy testing. USA doctors are taught that “celiac disease” is rare and they will likely never see a case. Most practitioners don’t think of it, so most gluten induced celiac patients and those who might have responded to a carbohydrate based approach alike fall by the wayside.

1950-60s
Numerous researchers perform studies finding benefit in saturated animal fats and tropical oils vs. disturbing results from inexpensive hydrogenated vegetable oils.

1960
The American Heart Association launches the Prudent Diet (replace red meat, eggs on TV networks based on the “lipid hypothesis” amid an era of protests from lipid researchers and heart specialist Dr. Paul Dudley White. “I began my practice as a cardiologist in 1921 and I never saw an MI patent until 1928. Back in the MI free days before 1920, the fats were butter and lard and I think that we would all benefit from the kind of diet that we had at a time when no one had ever heard the word corn oil.”

1960
Myocardial infarction claims 500,000 lives in 1960.

1960
Margot Shiner and Cyrus Rubin separately invent a small bowel biopsy capsule facilitating dx of small bowel diseases.
One of the diseases it can trigger is...
Rheumatoid Arthritis

Bone Loss Osteoporosis

Celiac Disease

Cancer (Lymphoma)

Psychological Disorders

Fibromyalgia & CFS

Asthma

Thyroid Disease

Gluten Sensitivity/Intolerance is not a disease, but it causes disease.
“Gluten sensitivity is a systemic autoimmune disease with diverse manifestations. This disorder is characterised by abnormal immunological responsiveness to ingested gluten in genetically susceptible individuals. Coeliac disease, or gluten-sensitive enteropathy, is only one aspect of a range of possible manifestations of gluten sensitivity. Although neurological manifestations in patients with established coeliac disease have been reported since 1966, it was not until 30 years later that, in some individuals, gluten sensitivity was shown to manifest solely with neurological dysfunction.”
All Patients with Autoimmune Disease should be screened for Gluten Sensitivity...
Table 1. Manifestations of silent celiac disease (predominantly extra-intestinal).

<table>
<thead>
<tr>
<th>Manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dermatitis herpetiformis</td>
</tr>
<tr>
<td>Anemia</td>
</tr>
<tr>
<td><strong>Autoimmune disorders</strong></td>
</tr>
<tr>
<td>Osteoporosis</td>
</tr>
<tr>
<td>Neurological disorders</td>
</tr>
<tr>
<td>Epilepsy with cerebral calcification</td>
</tr>
<tr>
<td>Neuropathy</td>
</tr>
<tr>
<td>Cerebellar ataxia</td>
</tr>
<tr>
<td>Chorea</td>
</tr>
<tr>
<td>Infertility/subfertility</td>
</tr>
<tr>
<td>Non-alcoholic fatty liver disease</td>
</tr>
<tr>
<td>Unexplained chronic hypertransaminasemia</td>
</tr>
</tbody>
</table>

Symptoms and signs in individuals with serology positive for celiac disease but normal mucosa

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Published: 22 July 2009
Received: 1 April 2009
Accepted: 22 July 2009

BMC Gastroenterology 2009, 9:57
This article is available from http://www.biomedcentral.com/1471-230X/9/57

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Abstract

Background: Antibody serology is an important tool in the investigation of celiac disease (CD), but does not always correlate with mucosal appearance in the small intestine. Patients with positive CD serology but normal mucosa (Marsh 0) are at increased risk of future CD. In this study we describe a model for identifying and characterizing individuals with normal mucosa but positive CD serology. Such individuals are sometimes referred to as having latent CD.

Methods: The records of ten Swedish pathology departments were used to identify individuals with biopsies indicating normal duodenaljejunal mucosa. Using the national personal identification number, these data were linked with CD serology data (anti-gluten, anti-endomysial and tissue transglutaminase antibodies), and we thereby identified 3,736 individuals with normal mucosa but positive CD serology. Two independent reviewers then manually reviewed their biopsy reports to estimate comorbidity. We also randomly selected 112 individuals for validation through patient chart review.

Results: The majority of the 3,736 individuals were females (63%). Children (0–15 years) made up 21.4%. The median number of biopsy specimen was 3. Our review of biopsy reports found that other gastrointestinal comorbidity was rare (inflammatory bowel disease: 0.4%, Helicobacter pylori infection: 0.2%). Some 22% individuals selected for patient chart review had a relative with CD. The most common symptoms among these individuals were diarrhoea (46%) and abdominal pain (45%), while 26% had aches. Although 27% of the individuals selected for validation had been informed about gluten-free diet, only 13% were adhering to a gluten-free diet at the end of follow-up.

Conclusion: Individuals with positive CD serology but normal mucosa often have CD-like symptoms and a family history of CD.
Results: 1 to 20 of 262

   Clin Gastroenterol Hepatol. 2010 Jul. [Epub ahead of print]
   PMID: 20601132 [PubMed - as supplied by publisher]
   Related citations

2. Severe iron deficiency anaemia as a manifestation of silent coeliac disease: case report and literature review.
   Paul SP, Taylor TM, Barnard P.
   PMID: 20518373 [PubMed - indexed for MEDLINE]
   Related citations

3. Silent celiac disease presenting with polyarthritis.
   Efe C, Urün Y, Purnak T, Ozaslan E, Ozbalan Z, Savaş B.
   PMID: 20511962 [PubMed - in process]
   Related citations

   Majorana A, Bardellini E, Ravelli A, Plebani A, Polimeni A, Campus G.
   PMID: 20384326 [PubMed - indexed for MEDLINE]
   Related citations

5. Celiac disease in Middle Eastern and North African countries: a new burden?
   Barada K, Bitar A, Mokadem MA, Hashash JG, Green P.
We have, however, shown that neurological dysfunction can not only precede coeliac disease but can also be its only manifestation. Of even more interest is the demonstration of a high prevalence of circulating antigliadin antibodies (IgG, IgA, or both) in patients with neurological dysfunction of obscure aetiology (57% ± 9% in neurological controls and 12% in normal controls). Only 35% of these patients had histological evidence of coeliac disease. The remaining 65% have gluten sensitivity where the target organ is the cerebellum or the peripheral nerves, a situation analogous to that of the skin in dermatitis herpetiformis.

In the light of these findings, the specificity of the concept of gluten sensitivity is not merely a question of degree, nor is it to be described merely as a histologically normal small bowel while on a normal diet who at some stage of their lives have had or will have an abnormal small bowel that responds to a gluten free diet. A number of case reports and case series have described patients with neurological disorders associated with gluten sensitivity. Further case reports have since been published, but most are based on patients with coeliac disease who later develop neurological dysfunction, implying that gut disease is a prerequisite. Unlike antinuclear antibodies or antireticulin antibodies, antigliadin antibodies are antibodies against the extrinsic causal factor for gluten sensitivity. Antigliadin antibodies may be more specific for coeliac disease, but no large scale data are available as yet on their specificity or sensitivity in patients with gluten sensitivity where the immunological target organ may be other than the gut.
Gluten-Sensitive Enteropathy (Celiac Disease): More Common Than You Think

TABLE 2
Symptoms of Celiac Disease and Possible Causes

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Possible causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatigue, malaise</td>
<td>Anemia, general immune system activation</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Nutrient malabsorption</td>
</tr>
<tr>
<td>Diarrhea, abdominal pain</td>
<td>Accelerated gastrointestinal tract transit time, steatorrhea, malabsorption</td>
</tr>
<tr>
<td>Anemia</td>
<td>Most commonly, iron deficiency; less commonly, vitamin B₁₂ and/or folate deficiency</td>
</tr>
<tr>
<td>Bone pain</td>
<td>Osteoporosis</td>
</tr>
<tr>
<td>Aphthous oral ulcers, glossitis, stomatitis</td>
<td>Vitamin deficiency, “oral” celiac disease</td>
</tr>
<tr>
<td>Infertility</td>
<td>Postulated cause: iron, folate, and/or zinc deficiency</td>
</tr>
<tr>
<td>Male impotence, decreased libido</td>
<td>Peripheral insensitivity to circulating testosterone</td>
</tr>
<tr>
<td>Alopecia areata</td>
<td>Immunologic attack on hair follicles</td>
</tr>
<tr>
<td>Dental enamel defects</td>
<td>Demineralization during tooth bud development in children</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>Delayed absorption of glucose</td>
</tr>
<tr>
<td>Gas, flatus, borborygmus</td>
<td>Secondary digestion of sugars by intestinal flora</td>
</tr>
<tr>
<td>Seizures, gluten ataxia, central nervous system symptoms</td>
<td>Increased affinity of celiac antibodies for brain vasculature</td>
</tr>
</tbody>
</table>

Celiac disease is commonly called celiac disease, is an intolerance of the small intestine that is precipitated by the ingestion of gluten in genetically susceptible persons. The mucosa, resolution of the malabsorptive effects of celiac disease. Recent studies of celiac disease in approximately one-milliliter common manifestations as “silent” serologic tests for antibodies against the gluten peptides of patients with celiac disease. Patients who are at increased genetic risk for celiac disease or personal history of chronic diarrhea, unexplained anemia, early diagnosis and management are in the 20th century when celiac disease was described late in the century, treatment responsive until the mid-20th century when all-bowel biopsy technique was established. Features of villous flattening, crypt hyperplasia, and increased intraepithelial lymphocytes (Figure 1) were shown to normalize after the institution of a gluten-free diet.

In the mid-1960s, an enteropathy strikingly similar to celiac disease was identified in patients with dermatitis herpetiformis. Subsequently, this skin disorder was shown to be a manifestation of gluten-sensitive enteropathy. In the mid-1960s, adult celiac disease was also noted to be associated with numerous neurologic abnormalities. In 1974, Russell and colleagues published an excellent review of the complications of celiac disease. Since that time, many other complications have been described, including the development of cancer and autoimmune diseases. The inclusion of IgA antibodies directed against tissue transglutaminase in the diagnostic workup of celiac disease is of great importance because it allows for the diagnosis of celiac disease in patients who do not have a gluten-sensitive enteropathy or have other conditions that may lead to a false-positive serologic test for celiac disease. In general, the treatment of celiac disease is the same as that for other enteropathies, with a gluten-free diet being the cornerstone of treatment. The diagnosis of celiac disease can be challenging, and a variety of clinical and serologic tests are available to help make the diagnosis. The most commonly used serologic tests are the anti-tissue transglutaminase antibody test and the anti-endomysial antibody test. The anti-tissue transglutaminase antibody test is more sensitive than the anti-endomysial antibody test, and it has been shown to be more specific for celiac disease. The anti-endomysial antibody test is more specific than the anti-tissue transglutaminase antibody test, and it has been shown to be more sensitive for celiac disease. The anti-tissue transglutaminase antibody test and the anti-endomysial antibody test are both useful for the diagnosis of celiac disease, and the choice of test should be based on individual patient factors.
“The study provided evidence that children who are EmA positive have a celiac-type disorder and benefit from early treatment despite normal mucosal structure, indicating that the diagnostic criteria for celiac disease should be re-evaluated.” (J Pediatr. 2010 Apr 16).
A new report in the *New England Journal of Medicine* identifies antibodies against osteoprotegerin (a protein that prevents bone breakdown) in several patients with celiac disease. This protein is responsible for helping maintain bone density. When it is attacked by the body’s immune system, bone loss becomes accelerated contributing to osteoporosis. (N Engl J Med 2009; 361: 1459 – 65).
"a wide spectrum of liver injuries in children and adults may be related to CD and in particular: (1) a mild parenchymal damage characterised by absence of any clinical sign or symptom suggesting a chronic liver disease and by non-specific histological changes reversible on a gluten-free diet; (2) a chronic inflammatory liver injury of autoimmune mechanism, including autoimmune hepatitis, primary sclerosing cholangitis and primary biliary cirrhosis, that may lead to fibrosis and cirrhosis, generally unaffected by gluten withdrawal and necessitating an immunosuppressive treatment; (3) a severe liver failure potentially treatable by a gluten-free diet. Such different types of liver injuries may represent a spectrum of a same disorder where individual factors, such as genetic predisposition, precocity and duration of exposure to gluten may influence the reversibility of liver damage."
What?
Keep in mind...

Gluten intolerance/sensitivity is not the sole cause of the following diseases. In cases where a person does not have a known cause for their diagnosis, gluten sensitivity should be ruled in or out. Therefore; those with the following conditions should be genetically screened...
• Angina Pectoris (chest pain/pressure)
• Anorexia
• Immunoglobulinopathies
• Antiphospholipid syndrome
• Anxiety
• Apathy
• Aphthous ulcers and canker sores
• Aortic Vasculitis
• Arthritis
  • Juvenile rheumatoid
  • Enteropathic
  • Psoriatic
  • rheumatoid
- Abdominal pain and distention
- Spontaneous abortion
- Addison’s Disease
- ADHD
- Alopecia (hair loss)
- Anemia
  - Iron deficiency
  - Folate deficiency
  - B-12 deficiency
  - B-6 deficiency
  - Vitamin C deficiency
  - Vitamin E deficiency
  - Copper deficiency
• Ataxia
• Atherosclerosis
• Autism and other learning disorders
• Cholangitis (gall bladder)
• Dermatitis Herpetiformis
• Autoimmune hepatitis
• Polyglandular syndrome
• Thyroiditis (hypothyroidism)
• Bitot’s spots
• Blepharitis
• Abnormal blurry vision
- Bone pain
- Bone fractures
- Cachexia
- Bronchiectasis
- Barrett’s Esophagus
- Bronchoalveolitis
- Adenocarcinoma of the intestine
- Small cell esophageal cancer
- Melanoma
- Asthma
- Cardiomegaly
- Cardiomyopathy
- Cataracts
- Cerebral perfusion abnormalities
- Cheilosis
- Chorea
- Coagulation abnormalities
- Crohn’s disease
- Ulcerative colitis
• Chronic constipation
• Coronary artery disease
• Diarrhea
• Lymphoma
• Cutaneous vasculitis
• Cystic fibrosis
• Delayed puberty
• Failure to thrive
• Dementia
• Depression
• Dermatomyositis
• Diabetes Mellitus type I
• Down syndrome
• Dysmenorrhea
• Dysgeusia
• Duodenal erosions
- Edema
- Eczema
- Dysphagia
- Epilepsy
- Spontaneous nose bleeds
- Erythema nodosum
- CFS
- Growth retardation
- Mental retardation
- Secondary food allergy response
- Blood in the stool
- Gastric bloating
- Grave’s disease
• Bleeding gums
• Hair loss
• Heartburn
• H. pylori infection
• Hives
• NAFL
• Malnutrition and nutritional deficiencies
• Infertility
• Hypogonadism
• Hypoglycemia
• Hypospleenism
• Thrombocytopenia
• Impotence
• Osteoporosis
• Insomnia
• IBS
• Keratomalacia
- Lactose intolerance
- Loss of smell
- Non Hodgkin lymphoma
- Early menopause
- Migraine headache
- Multiple sclerosis
- Muscle wasting
- Myopathy
- Obesity
- Osteomalacia
- Osteopenia
- Parathyroid carcinoma
- Pancreatic insufficiency
- Polymyositis
- PMS
- Biliary cirrhosis
- Psoriasis
- Dermatitis
- Sjogren’s syndrome
- Short stature
- Scleroderma
- Steatorrhea
- Spina bifida
- SLE
- Tremors
- Parkinson’s disease
- Glossitis
- Vitiligo
- Vomiting
- Vaginitis
- UTI
Aside from physical stress, gluten has been shown to contribute to all of these mechanisms...
Diagram 1 – Possible Mechanisms and Consequences of Magnesium Deficiency

Diet (i.e. ↑ refined CHO)  
Stress  
Diuretics (alcohol, caffeine, anti HTN meds)

↓ Mg ++ 
(serum and cellular)

↑ blood lipids  
↑ Platelet aggregation

Depression  
Vaso-constriction  
↓ Bone Mineralization  
Muscle spasm & Pain

?SSRI’s?  
Hypertension

?HRT?  
NSAIDS?  
?Aspirin?  
?Diuretics?

CoQ10

SSRI – Selected Serotonin Reuptake Inhibitors  
HRT – Hormone Replacement Therapy  
NSAIDS – Non steroidal Anti-inflammatory Drugs
The Gluten Sensitivity HYDRA

Joint pain
Acne
Bloating
Hormone Imbalance
Fatigue
Nausea
Weight Gain

Treating these symptoms with medicine does not resolve the origin of a patient’s problem...
What is Gluten?

• Gluten is a mixture of proteins found in all grains. It is composed of two primary subfractions:
  • Prolamines
  • Glutelins

• The prolamine gliadin is the most studied piece of gluten in the medical literature as it relates to celiac disease.
# The Prolamine Fraction of Proteins in Grains

<table>
<thead>
<tr>
<th>Grain</th>
<th>Prolamine</th>
<th>% Total Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wheat</td>
<td>Gliadin</td>
<td>69</td>
</tr>
<tr>
<td><strong>Rye</strong></td>
<td>Secalinin</td>
<td><strong>30-50</strong></td>
</tr>
<tr>
<td>Oats</td>
<td>Avenin</td>
<td>16</td>
</tr>
<tr>
<td><strong>Barley</strong></td>
<td>Hordein</td>
<td><strong>46-52</strong></td>
</tr>
<tr>
<td>Millet</td>
<td>Panicin</td>
<td>40</td>
</tr>
<tr>
<td><strong>Corn</strong></td>
<td>Zien</td>
<td><strong>55</strong></td>
</tr>
<tr>
<td>Rice</td>
<td>Orzenin</td>
<td>5</td>
</tr>
<tr>
<td><strong>Sorghum</strong></td>
<td>Kafirin</td>
<td><strong>52</strong></td>
</tr>
<tr>
<td>Teff</td>
<td>Penniseiten</td>
<td>11</td>
</tr>
</tbody>
</table>
“Unexpectedly, a sequence from ω-gliadin (wheat) and C-hordein (barley) but not α-gliadin was immunodominant regardless of the grain consumed.”

The antigenic peptides recognized by the T cells that cause this disease are incompletely defined. Our understanding of the epitopes of pathogenic CD4+ T cells is based primarily on responses shown by intestinal T cells in vitro to hydrolysates or polypeptides of gluten, the causative antigen. A protease-resistant 33-amino acid peptide from wheat α-gliadin is the immunodominant antigen, but little is known about the spectrum of T cell epitopes in rye and barley or the hierarchy of immunodominance and consistency of recognition of T-cell epitopes in vivo. We induced polyclonal gluten-specific T cells in the peripheral blood of celiac patients by feeding them cereal and performed a comprehensive, unbiased analysis of responses to all celiac toxic prolamins, a class of plant storage protein. The peptides that stimulated T cells were the same among patients who ate the same cereal, but were different after wheat, barley and rye ingestion. Unexpectedly, a sequence from ω-gliadin (wheat) and C-hordein (barley) but not α-gliadin was immunodominant regardless of the grain consumed. Furthermore, T cells specific for just three peptides accounted for the majority of gluten-specific T cells, and their recognition of gluten peptides was highly redundant. Our findings show that pathogenic T cells in celiac disease show limited diversity, and therefore suggest that peptide-based therapeutics for this disease and potentially other strongly HLA-restricted immune diseases should be possible.
Prolamine Definition

• Any of a class of simple proteins soluble in alcohol and usually having a high proline and glutamine content, found in the grains of cereal crops such as wheat, rye, barley, corn, and rice.

• Prolamines are further subclassified into:
  • alpha, beta, gamma and omega fractions
  • Alpha and beta gliadins are the most well studied in relation to celiac disease.
Grains are the seeds of grass. The seed has a bran casing, a starchy endosperm which contains 90% of the protein (including gluten), and a small germ nucleus which is the plant embryo, waiting to grow. Any flour made from the starchy endosperm contains prolaminides and is potentially toxic to the grain sensitive/intolerant person.

Excerpt from: "Nutrition Therapy" by Stephen J. Gislason, MD
What is Gluten Sensitivity?

• The current yet antiquated definition is as follows:
  • Gluten sensitivity is an immune reaction to the protein gluten found in wheat, barley, and rye. The definition sometimes includes oats & sometimes does not. This definition is often times incorrectly used synonymously with celiac disease.
  • Why is it inconsistent?
  • What about those with non celiac symptoms?
  • What about other gluten containing grains?
Definitional Differences

- **Gluten Allergy** is typically considered to be an allergy (immune mediated response).

- **Gluten intolerance** is considered to be an inability to tolerate gluten (immune and non immune mediated).

- **Gluten Sensitivity** is a mesh of the above two terms.

- **Celiac Disease** is an autoimmune disease of the small intestine caused by gluten induced damage.
Allergy = Immune Reaction

Acute

IgE = Antibody

Chemical Inflammation

This is what your allergy doctor measures with a skin prick test

Delayed Hypersensitivity

T-Cell Reaction

IgG, IgA, IgM, (IgD?) Antibodies

Immune Complexes

Chemical Inflammation

Tissue Damage

Disease

This is what your GI doctor measures in your blood when testing for celiac disease.

What about these?
True Allergy Reaction to Gluten (IgE)

Schematic presentation of the pathophysiology of the immediate hypersensitivity reactions (Type 1 allergy) of the intestine.
**Common Acute Food Allergy Reactions**

- Hives - itching, burning and swelling of the skin
- Eczema – redness and small blistering of skin
- Bronchitis
- Asthma
- Coughing
- Sneezing
- Diarrhea
- Colic
- Vomiting or excessive spitting up
Gluten Intolerance

- Inability to Digest Gluten
- Gut Dysbiosis
- Leaky Gut/Intestinal Permeability
- Acquired Allergy
- Tissue Damage

Production of immune system production of antibodies and inflammatory chemicals
Rheumatoid Arthritis
Bone Loss Osteoporosis
Cancer (Lymphoma)
Gluten Sensitivity/Intolerance
Psychological Disorders
Fibromyalgia & CFS
Thyroid Disease
Asthma

Gliac Disease
Diagnosing Gluten Sensitivity

- **Blood tests**
  - Non specific
  - High tendency towards false negative

- **Biopsy**
  - Only diagnostic for celiac disease
  - Not an accurate representation of the entire intestine or of extra intestinal damage

- **Genetics**
  - Very accurate for identifying potential to react to gluten

- **Stool tests**
  - More accurate than blood but still limited to gliadin

- **Predictive antibody testing**
  - in development
  - Used to monitor more than diagnose
Old School vs. New School  

1. Celiac disease is the only manifestation of gluten sensitivity  
2. Intestinal biopsy is the gold standard for diagnosis of celiac disease  
3. Antibody blood tests are used for gliadin  
4. Extraintestinal manifestations of celiac disease are rare  

1. Celiac is a rare manifestation of gluten sensitivity  
2. HLA-DQ testing with clinical symptoms is the gold standard for gluten sensitivity recognition  
3. Extraintestinal manifestations of gluten intolerance are a major cause of missed diagnosis in developed nations worldwide.
Genetic Influence on the Gut Response

Depiction of the intestinal mucosa with emphasis on the factors involved in genetic influence on gut response.
Gluten specific, HLA-DQ restricted T cells from coeliac mucosa produce cytokines with Th1 or Th0 profile dominated by interferon γ

E M Nielsen, K E A Lundin, P Kragi, H Scott, I M Solid, P Brandtzæg

Abstract
Coeliac disease is precipitated in susceptible subjects by ingestion of wheat gluten or gluten related prolamins from some other cereals. The disease is strongly associated with HLA-DQ2. Coeliac disease or gluten sensitive enteropathy is a proximal small intestinal disorder characterised by various degrees of crypt cell hyperplasia and villous atrophy. The result is malabsorption and often diarrhoea. The cause is not known.

All TCC were found to secrete interferon (IFN) γ, often at high concentrations (>2000 U/ml); some secreted in addition interleukin (IL) 4, IL 5, IL 6, IL 10, tumour necrosis factor (TNF), and transforming growth factor (TGF) β. The last TCC thus displayed a ThO-like cytokine pattern. However, other TCC produced IFN γ and TNF but no IL 4, or IL 5, compatible with a Th1-like pattern.
The Gluten Positive Genes

• HLA-DQα1 Gene
  • 0505 (DQ2)*
  • 0501 (DQ2)*
  • 0301 (DQ8)*

• HLA-DQβ1 Gene
  • 0201 (DQ2)*
  • 0202 (DQ2)*
  • 0302 (DQ8)*
  • 03xx (DQ3)
  • 01xx (DQ1)
  • 05xx (DQ1)
  • 06xx (DQ1)
Gluten sensitivity related to HLA alleles other than HLA-DQ2 or DQ8


High prevalence of celiac sprue-like HLA-DQ genes and enteropathy in patients with the microscopic colitis syndrome.


OBJECTIVE: Celiac sprue is associated with specific HLA-DQ genes (mainly DQ2). Because there are epidemiological and histopathological similarities between celiac sprue and microscopic colitis, we hypothesized that these syndrome may share an HLA genetic predisposition and pathogenesis. METHODS: The HLA-DQ genes of 25 patients with celiac sprue, 53 patients with the microscopic colitis syndrome, and 429 normal controls were typed and compared. Serum was analyzed for antigliadin and antiendomysial antibodies. Small intestinal biopsies were analyzed for signs of histopathology. RESULTS: HLA-DQ2 or DQ1,3 (the latter as DQ1,7,DQ1,8, or DQ1,9) were seen more frequently in both patient groups relative to controls. In patients with the microscopic colitis syndrome, serological tests for celiac sprue were weakly positive in 17%; mild inflammation of the small intestine without villous atrophy was present in 43%, and inflammation plus partial or subtotal villous atrophy was present in 27%. CONCLUSIONS: A shared set of predisposing HLA-DQ genes account for the epidemiological overlap of celiac sprue and microscopic colitis. Mild to moderate mononuclear cell inflammation of the small intestine, often accompanied by partial or subtotal villous atrophy, is frequent in patients with the microscopic colitis syndrome. Although further studies will be necessary to determine if this enteropathy is induced by dietary gluten, we speculate that the small intestinal but not colonic histopathology in patients with microscopic colitis is caused by immunological gluten sensitivity.
Gluten ataxia in perspective: epidemiology, genetic susceptibility and clinical characteristics.

Brain. 2003 Sep;126(Pt 9):E4; 685-691

Department of Neurology, The Royal Hallamshire Hospital, Sheffield, UK.

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We previously have described a group of patients with gluten sensitivity presenting with ataxia (gluten ataxia) and suggested that this disease entity may account for a large number of patients with sporadic idiopathic ataxia. We have therefore investigated the prevalence of gluten sensitivity amongst a large cohort of patients with sporadic and familial ataxia and looked at possible genetic predisposition to gluten sensitivity amongst these groups. Two hundred and twenty-four patients with various causes of ataxia from North Trent (59 familial and/or positive testing for spinocerebellar ataxias 1, 2, 3, 6 and 7, and Friedreich's ataxia, 132 sporadic idiopathic and 33 clinically probable cerebellar variant of multiple system atrophy MSA-C) and 44 patients with sporadic idiopathic ataxia from The Institute of Neurology, London, were screened for the presence of antigliadin antibodies. A total of 1200 volunteers were screened as normal controls. The prevalence of antigliadin antibodies in the familial group was eight out of 59 (14%), 54 out of 132 (41%) in the sporadic idiopathic group, five out of 33 (15%) in the MSA-C group and 149 out of 1200 (12%) in the normal controls. The prevalence in the sporadic idiopathic group from London was 14 out of 44 (32%). The difference in prevalence between the idiopathic sporadic groups and the other groups was highly significant (P < 0.0001 and P < 0.003, respectively). The clinical characteristics of 68 patients with gluten ataxia were as follows: the mean age at onset of the ataxia was 48 years (range 14-81 years) with a mean duration of the ataxia of 9.7 years (range 1-40 years). Ocular signs were observed in 84% and dysarthria in 66%. Upper limb ataxia was evident in 75%, lower limb ataxia in 90% and gait ataxia in 100% of patients. Gastrointestinal symptoms were present in only 13%. MRI revealed atrophy of the cerebellum in 79% and white matter hyperintensities in 19%. Forty-five percent of patients had neurophysiological evidence of a sensorimotor axonal neuropathy. Gluten-sensitive enteropathy was found in 24%. HLA DQ2 was present in 72% of patients. DQ1 accounts for 20% of the gluten ataxia patients. Gluten ataxia is therefore the single most common cause of sporadic idiopathic ataxia.

Gastrointestinal symptoms were present in only 13%. MRI revealed atrophy of the cerebellum in 79% and white matter hyperintensities in 19%. Forty-five percent of patients had neurophysiological evidence of a sensorimotor axonal neuropathy. Gluten-sensitive enteropathy was found in 24%. HLA DQ2 was present in 72% of patients. DQ1 accounts for 20% of the gluten ataxia patients. Gluten ataxia is therefore the single most common cause of sporadic idiopathic ataxia.
Physicians at Mayo Clinic are now recommending HLA-DQ Gene testing for patients with irritable bowel syndrome (IBS) symptoms.

References:
2. J Gastrointestin Liv Dis 2006. 15;3:221-25
Fortification of Grain?

In the United States, manufacturers of cereals, rices, breads and other grains are federally required to fortify their products with the mineral iron and several B vitamins. In 1943 the government mandated that grain products be fortified with niacin, riboflavin, thiamine and iron, while 1998 saw folate added to this list of nutrients. The addition of these nutrients into everyday products was undertaken to reduce the incidence of beriberi, pellagra, birth defects and other issues.
Traditional Gluten Free Diets Fail

Researchers give the traditional gluten free diet an F...

In this study only 8% of the patients recovered from intestinal damage while following a traditional gluten free diet.

“”

After a median 16 months GFD, 38 (8%) patients had histological ‘normalization’, 300 (65%) had ‘remission’ with persistent intraepithelial lymphocytosis, 121(26%) had ‘no change’ and 6 (1%) had ‘deterioration’.

Source:

Why are Gluten Free Diets Failing to Heal So Many Patients?

A recent study published in the *American Journal of Gastroenterology* finds that more than 30% of patients with celiac disease following a gluten free diet fail to exhibit recovery of intestinal damage after 5 years on a gluten free diet.

“Mucosal recovery was absent in a substantial portion of adults with CD after treatment with a GFD. There was a borderline significant association between confirmed mucosal recovery (vs. persistent damage)

Source:

*The American Journal of Gastroenterology*, (9 February 2010)
Let's take a closer look:

1. The Cardinal Rule – One cannot achieve or maintain health eating unhealthy foods.
2. Processed and packaged food is not healthy regardless of whether or not the label claims to be gluten free.
3. Eating unhealthy foods leads to poor health (I know, this should be a no brainer).
4. Many over the counter packaged foods contain cross contamination of gluten.
5. Many “gluten free” products contain other types of grain based gluten that have not been adequately studied to be safe for those with gluten sensitivity (see video tutorial #1 for more on this).
6. Most processed “gluten free” products contain genetically modified grains, high amounts of sugar, and are devoid of any significant nutrient density.
Gluten Free Whiplash

Going gluten free can be a saving grace for many. However, a common clinical manifestation called *Gluten Whiplash* occurs for many who do not go TRUE gluten free.

*The Gluten Whiplash Effect* typically occurs 3-6 months after starting a gluten free diet. Let me explain. When one initially goes gluten free, a state of dietary distress and confusion sets in. Many limit their diets to an extreme because they are not quite sure what to eat. The typical gluten free diet learning curve takes 8-12 weeks. This is because one must spend enough time educating themselves about acceptable products, restaurants, etc. During this time, the body starts to heal and most people do very well noticing dramatic improvements in their health.

Once the learning curve is conquered, people tend to gravitate toward the processed, packaged “gluten free” food items. People tend to get lazy and make the choice of convenience over health. BIG MISTAKE! This is where *Gluten Whiplash* tends to set in.
Modern Wheat Breeding Increases Celiac Disease Occurrence?

New research claims that the toxicity of wheat gluten potentially worsened by cross breeding different strains...

suggests that modern wheat breeding practices may have led to an increased exposure to CD epitopes

Source:

1. Genetic manipulation of grains – no long term research has been done on safety, yet we assume these foods are OK contrary to common sense. Many studies show these foods to be dangerous.

2. The pervasive use of grains in the food supply. Almost all packaged foods contain grain either as a main ingredient or an agent to alter food texture, viscosity, etc. More grain exposure = more people reacting to grain.

3. The use of herbicides, pesticides, fungicides, etc. Much like genetically modified foods, these chemicals are used under the assumption that they are safe.

4. Over use of antibiotics. Although life saving if one has a bacterial infection, the over utilization of these drugs contributes to a change in the normal healthy gut flora thus weakening the immune system. Additionally, we feed them to chickens, pigs, cows, and fish that are being raised for human food consumption.

5. Anti-acid medications. Nexium, Tums, Prilosec, Rolaids, and more, these drugs suppress acid in the stomach. Acid suppression weakens the immune system and leads to wide spread malabsorption of nutrients.

6. Non steroidal anti-inflammatory medications (NSAIDS). These medications contribute to the destruction of the gastric and intestinal lining thus weakening immunity and predisposing one to intestinal permeability (leaky gut syndrome).

7. Medications in general. Many OTC and prescription medications contain grain based adhesives. Sick from gluten? Take this pill (with gluten in it) and you will get better?!?

8. Grain is cheap food. The government subsidizes grain making it much less expensive to use as a staple food.

9. Commercialization. Everywhere you look, there is a billboard, TV commercial, nutritionist, Food Guide Pyramid, etc telling us how healthy whole grains are.

10. Degradation of the eduction system. Public schools focus on teaching students how to pass standardized tests. Nutrition and physical education are given minimal time in the classroom. Many of those teaching nutrition do not lead by example thus devaluing the lesson. The nutrition basics taught focus on a Food Guide Pyramid based in grain.
What about corn?

“Maize prolamines had low but definite activity even though maize is reported to be harmless”

*(Gut, 1983, 24, 825-830)*
Antibodies to maize in patients with Crohn's disease, ulcerative colitis and coeliac disease

<table>
<thead>
<tr>
<th>Group</th>
<th>Number</th>
<th>Maize antibody positive</th>
<th>Significance*</th>
<th>Wheat antibody positive</th>
<th>Significance*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crohn's disease</td>
<td>33</td>
<td>11 (33%)</td>
<td>$P = 0.037$</td>
<td>19 (58%)</td>
<td>$P = 0.00035$</td>
</tr>
<tr>
<td>Ulcerative colitis</td>
<td>18</td>
<td>9 (50%)</td>
<td>$P = 0.0054$</td>
<td>9 (50%)</td>
<td>$P = 0.0027$</td>
</tr>
<tr>
<td>Total inflammatory bowel disease</td>
<td>51</td>
<td>20 (39%)</td>
<td>$P = 0.0061$</td>
<td>28 (55%)</td>
<td>$P = 0.00014$</td>
</tr>
<tr>
<td>Coeliac disease</td>
<td>36</td>
<td>16 (44%)</td>
<td>$P = 0.0032$</td>
<td>21 (58%)</td>
<td>$P = 0.00017$</td>
</tr>
<tr>
<td>Coeliac disease off GFD†</td>
<td>22</td>
<td>10 (45%)</td>
<td>$P = 0.0079$</td>
<td>15 (68%)</td>
<td>$P = 0.000094$</td>
</tr>
<tr>
<td>Coeliac disease on GFD†</td>
<td>14</td>
<td>6 (43%)</td>
<td>$P = 0.0307$</td>
<td>6 (43%)</td>
<td>$P = 0.018$</td>
</tr>
<tr>
<td>Controls</td>
<td>41</td>
<td>6 (14%)</td>
<td>—</td>
<td>5 (12%)</td>
<td>—</td>
</tr>
</tbody>
</table>

* Patients vs controls (probabilities estimated either by the Chi-square test with Yate's correction or Fisher's exact test, as appropriate).

† GFD = Gluten-free diet.

"It is of interest that patients with coeliac disease on a gluten-free diet had a lower incidence of wheat, but not of maize, antibodies when compared with those patients not on a diet."
mTG treatment increased reactivity to wheat and maize prolamins in patients with celiac disease...

Celiac disease (CD) is an enteropathy triggered by dietary proteins of wheat gluten and related cereals, which has increased to an estimated worldwide prevalence of 1–2% (1). Among the causes for the increase in the incidence of CD could be the use of infant formulas, instead of breastfeeding and the early introduction of cereals in the diet, which have been related to the earlier onset of CD (2). Additionally, in recent decades, cereal food technology has changed to fast processes by which proteins are not degraded during manufacture, which could initiate or exacerbate CD in predisposed individuals (3). A change related to CD (4, 5) is the increasing industrial use of microbial transglutaminase (mTG) for improving functional properties of dairy and bakery products (6).

CD is characterized by the presence of antibodies against gluten peptides, especially after deamination by the tissue transglutaminase (tTG), which is also the autoantigen (7). Therefore, it was not sure that immunoreactivity of IgA from CD patients to gluten proteins increased after mTG treatment (4, 5). In addition, some other dietary proteins, such as milk caseins and maize tein, induced in a contrast, a specific inflammatory reaction in the CD mucosa of 59% of the patients (8) and were recognized by IgA antibodies from other gastrointestinal conditions, but with a different affinity to gliadins to cross the intestinal barrier in CD-predisposed individuals, and it might initiate the cascade of autoimmune reactions (10).

Although CD onset can appear at any age, there are some differences in the immune responses among infants and older children or adults. In young children, the cellular immune response is against amino acid sequences, which are not substrates for tTG, whereas in older children and adults, deamination of the sequences by mTG increases the reactivity (11). In another study, we found that the reactivity of serum IgA from a 16-year-old celiac patient to gliadins increased after treatment with mTG, whereas the IgA reactivity of a 2.9-year-old patient was the same against gliadins, whether it was mTG-treated or not.

There are also age-related differences in CD manifestations. In children under 2 years of age, CD is characterized by diarrhea and abdominal distension, whereas abdominal pain is more common in children older than 2 years old (12). Atypical features (e.g., affecting other organ systems) occur in patients with later onset of the disease (13). Additionally, D'Amico et al. (14) found that the onset of CD symptoms was mainly in the first to second year for nonbreastfed children, whereas it was in the second to third year for exclusively breastfed children. Therefore, we hypothesized that reactivity to serum IgA from CD patients, which is a manifestation of the immune
Bovine milk intolerance in celiac disease is related to IgA reactivity to α- and β-caseins

Francisco Cabrera-Chávez, M.Sc., and Ana María Calderón de la Barca, Ph.D.*
Centro de Investigación en Alimentación y Desarrollo, A. C. Carrera a La Victoria, Hermosillo, Sonora, Mexico
Manuscript received October 16, 2001; accepted January 9, 2002.

Abstract
Celiac disease is an autoimmune disease triggered mainly by ingestion of wheat gluten proteins.
However, some other dietary proteins, such as those of cow’s milk, induce celiac disease-like symptoms in some patients with celiac disease. Different approaches have been done to detect the component responsible for this problem, including the possibility of gluten peptides present in cow’s milk.

Keywords: Bovine casein; immunoglobulin A reactivity; Celiac disease

In a recent issue of Nutrition [1], intolerance to bovine milk of some patients with celiac disease (CD) was reported to not be due to the presence of epitopes from wheat gluten.

In the excellent work by Dolkings et al. [1], the investigators did not detect gluten proteins or peptides in bovine milk from cows fed their containing large amounts of wheat.

Thus, it was demonstrated that the symptoms seen in patients with CD after cow’s milk consumption are not related to gluten proteins in bovine milk, confirming that whole-phase-conjugated goat anti-rabbit antibodies. Alkaline phosphatase activity was developed.

Figure 1 shows the gliadins (Fig. 1A, lane 2) and bovine caseins (Fig. 1B, lane 2) electrophoretic patterns and their respective immunodetection with serum IgA from patients with CD (lane 2 for gliadins in Fig. 1A and lane 2 for caseins in Fig. 1D). As expected, there was a clearly different electrophoretic mobility for the two protein types. In Figure 1A, lane 1, gliadins had a molecular weight from 15 to 45 kDa.

“the serum IgA response of patients with CD to bovine milk could be related to gliadins and caseins sharing epitopes recognized by antigliadin IgA antibodies, as previously proposed.”

The minority fraction of caseins, αs-casein, has the higher antigenicity for milk-intolerant individuals [7]. Therefore, the IgA immunoactivity found against α- and β-caseins is not attributable to antigenicity.

Previous studies [2,3] have demonstrated a reaction to caseins, although these were mixtures of αs, βs, and κ-caseins and probably other milk proteins; however, a distinctive identification had not been done. It has been published that there is a high homology of some peptides in bovine β-casein to the gluten peptide, mainly with the amino acid...
“The observation that corn gluten challenge induced an abnormal NO reaction in some of our patients with CD is intriguing as maize is considered safe and is recommended as the substitute cereal in a gluten free diet.”

Gut 2005;54;769-774
“The allergens in rice, corn, millet and buckwheat should be better studied before they can be recommended as alternatives for cereal allergic children.”

“High titres were also found when coeliac sera were tested against wheat glutenins, albumins, and globulins, as well as against barley, oats, and maize prolamines”

In this case, corn flakes triggered her symptoms!

Recent Studies on Rice...


“Causative foods for the 35 children were rice ($n = 14$), soy ($n = 12$), cow’s milk ($n = 7$), vegetables and fruits ($n = 3$), meats ($n = 2$), oats ($n = 2$), and fish ($n = 1$). In the 66 episodes, vomiting was the most common clinical feature (100%), followed by lethargy (85%), pallor (67%), and diarrhea (24%). A temperature of $<36°C$ at presentation was recorded for 24% of episodes.”

Gluten Aside. Isn’t Grain Supposed to Be Healthy?

The food guide pyramid recommends up to 11 servings per day with 50% coming from whole grain sources.
Isn’t Grain Supposed to Be Healthy

- The seeds are sprayed with fungicides and insecticides.
  - Xenoestrogens which effect hormone balance and contribute to many diseases (breast cancer, endometriosis, fibrocystic breasts)
Isn’t Grain Supposed to Be Healthy?

• The seeds are doused with hormones to aid in growth
• The grains are stored in bins sprayed with additional pesticides
• Drying of the grain causes damage to it’s proteins
• Processing adds…
  • Dough conditioners
  • Preservatives
  • Soy flour
  • Extrusion creates acrylamide
  • Hydrogenated oils
Nutrient Properties of Grains

• Poor source of protein leads to inadequate growth (archeological fossil records show reduction in stature and osteoporosis with the introduction cereal grain based diets)

• Low in EPA and DHA

• Contain Anti-nutrients

• Contain Autoimmune inducing peptides for genetically susceptible individuals
Hormonal influences linked to obesity

- Much like sugar, Grains cause insulin excess...
  - Tells the body to store fat
  - Prevents muscle building
  - Reducing vitamin C uptake into white blood cells
  - Causes magnesium loss
    - Leads to cyclical hypertension (muscle constriction)
  - Sodium retention and excess
    - Contributes to congestive heart failure
What about infant cereals?
So What Do I Eat?

**Meat** — any variety is ok. You must consider the source of the animal. In the case of animal based foods you are not what you eat, you’re what you eat eats!

- **Beef** — should come from grass fed animals.
- **Fish** — Should be wild caught not farm raised.
- **Poultry and eggs** — should be free range organic
• **Dairy**
  • Only from grass fed (grazing animals). Raw dairy from a reputable farm is recommended.

• **Fruits and Vegetables**
  • Any organic variety that you are not allergic to.

• **Nuts, non grain seeds, and beans**
  • Any organic variety that you are not allergic to.

• **Processed food**
  • Including processed food labeled “gluten free” are better left avoided.
Gluten contamination of grains, seeds, and flours in the United States: a pilot study.

“Twenty-two inherently gluten-free grains, seeds, and flours not labeled gluten-free were purchased in June 2009 and sent unopened to a company who specializes in gluten analysis. All samples were homogenized and tested in duplicate using the Ridascreen Gliadin sandwich R5 enzyme-linked immunosorbent assay with cocktail extraction… Nine of 22 (41%) samples contained more than the limit of quantification, with mean gluten levels ranging from 8.5 to 2,925.0 ppm. Seven of 22 samples (32%) contained mean gluten levels >/= 20 ppm and would not be considered gluten-free under the proposed FDA rule for gluten-free labeling. Gluten contamination of inherently gluten-free grains, seeds, and flours not labeled gluten-free is a legitimate concern.” (J Am Diet Assoc. 2010 Jun; 110 (6): 937 – 940).
If the following terms are found on the food label or ingredient list the food should be avoided:

- Malt
- Wheat
- Gluten
- Barley
- Rye
- Oats
- Teff*
- Sorghum*
- Buckwheat***
- Amaranth***
- Quinoa***
- Spelt*
- Rice*
- Corn or maize*
- Millet*
- Triticale (wheat hybrid)*
Processed foods are not recommended!

- Textured vegetable protein **
- Hydrolyzed plant protein **
- Extenders and binders **
- Hydrolyzed vegetable protein **
- Modified Food Starch**
- MSG**
- Natural Flavors

*These grains are classically considered gluten free, but are not recommended on a TRUE gluten free diet.

** These items are only found in processed food items.

*** These items are technically not grains, but are at high risk for cross contamination and not recommended on a TRUE gluten free diet unless verification can be obtained. These pseudo cereals are also very high in glutamic acid and should be discouraged as substitutes for patients with neurological symptoms.
Additional Recommendations

Because gluten sensitivity has been shown to cause malabsorption of vitamins, minerals, and other nutrients, it is recommended that you see your doctor to be tested for nutritional deficiencies. Spectracell labs has the most comprehensive and scientifically advanced test available. You can visit their website at www.spectracell.com to find physicians in your area capable of performing the testing for you.
LABORATORY REPORT

Account Number: 191473
Name: 
Gender: Female
DOB: 09/30/1970
Accession Number: K49200
Requisition Number: 382097
Date of Collection: 06/07/2011
Date Received: 06/08/2011
Date Reported: 06/17/2011

Summary of Deficient Test Results

Micronutrient analysis (WBC) determined the following deficiencies:

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<th></th>
<th>Folate</th>
<th>Asparagine</th>
<th>Oleic Acid</th>
<th>Calcium</th>
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</thead>
</table>

Gluten sensitivity, migraine headaches, and chronic fatigue

John F. Crawford, Ph.D.
Laboratory Director

CLIA#: 45D0710715

All tests performed by Spectrachell laboratories, Inc. 10401 Town Park Drive Houston, TX 77024
Summary of Deficient Test Results

Micronutrient analysis (WBC) determined the following deficiencies:

- Calcium
- Zinc
- Spectrox

Irritable Bowel Syndrome, Gluten Sensitivity

John F. Crawford, Ph.D.
Laboratory Director

CLIA# 45D0710715
Summary of Deficient Test Results

Micronutrient analysis (WBC) determined the following deficiencies:

- Pantothenate
- Biotin

Antibiotic induced waisting, muscle pain, IBS (diarrhea)

John F. Crawford, Ph.D.
Laboratory Director

CLIA# 45D0710715

All tests performed by SpectraCell Laboratories Inc. * 10401 Town Park Drive Houston, TX 77072
Summary of Deficient Test Results

Micronutrient analysis (WBC) determined the following deficiencies:

- Zinc
- Magnesium
- Selenium
- Vitamin E

This patient was diagnosed with the following:
- Gluten Sensitivity
- Hypothyroidism
- Type II Diabetes
Summary of Deficient Test Results

Micronutrient analysis (WBC) determined the following deficiencies:

<table>
<thead>
<tr>
<th>Vitamin B1</th>
<th>Vitamin B2</th>
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</thead>
<tbody>
<tr>
<td>Folate</td>
<td>Glutamine</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

This gluten sensitive patient was diagnosed with PCOS, obesity, and chronic muscle pain.

John F. Crawford, Ph.D.
Laboratory Director
LABORATORY REPORT

Account Number: 191473
Name: 
Gender: Female
DOB: 09/16/1993
Accession Number: K47855
Requisition Number: 382050
Date of Collection: 05/01/2011
Date Received: 06/01/2011
Date Reported: 06/10/2011

Summary of Deficient Test Results

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<td>B12</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>SpectroX</td>
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</tbody>
</table>

Gluten Sensitivity - Chronic anemia, IBS, muscle pain, and intermittent fainting

John F. Crawford, Ph.D.
Laboratory Director

CLIA# 45D0710715

All tests performed by Spectracell Laboratories, Inc., 14034 Town Park Drive, Houston, TX 77089
Before seeing me, this patient was diagnosed with idiopathic peripheral neuropathy, depression, hypothyroid, and migratory joint pain.
Additional Recommendations

Gluten can cause leaky gut syndrome. Because of this, many people develop additional food allergies. Measuring for food allergies is an important next step to help to determine what other dietary exposures are contributing to disease.

ELISA/ACT Biotechnologies LLC
Have Family Members Genetically Tested!
For more information and for physician affiliate inquiries:
Contact Dr. Osborne or visit
www.GlutenFreeSociety.org
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Commonly asked questions

1. Will I receive a copy of the presentation slides?

   YES

2. Is the presentation being recorded?

   YES

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