PTO Form 1930 (Rev 9/2007)
OMB No. 0651-0050 (Exp. 4/30/2009)

Request for Reconsideration after Final Action

The table below presents the data as entered.

Input Field	Entered
SERIAL NUMBER	85184182
LAW OFFICE ASSIGNED	LAW OFFICE 102
MARK SECTION (no change)	
ARGUMENT(S)	

1. <u>Procedure History</u>

This is in response to the September 14, 2011 Final Office Action for Application Serial No. 85/184,182 for the mark PEACE, LOVE AND GLUTEN FREE owned by Rock-N-Roll Gourmet, LLC ("Applicant"). The Examiner issued a final refusal on the grounds that U.S. Registration Nos. 3,542,995 and 3,546,281 ("Cited Registrations") for crackers are likely to be confused with Applicant's mark when used in connection with potato chips. Previously, the Examiner issued a Non-final Office Action on the same grounds, to which Applicant responded on August 31, 2011. All arguments made in Applicant's August 31, 2011 response are incorporated in this response.

2. <u>Summary</u>

The Examiner argued that PEACE, LOVE AND GLUTEN FREE is likely to be confused with PEACE, LOVE AND CRUNCH because the dominant portion of the marks is PEACE, LOVE. Furthermore, the Examiner argued that gluten-free potato chips, Applicant's applied-for products, are typically marketed alongside regular snacks, and that gluten-free potato chips and crackers are highly related. Finally, the Examiner argued that the goods of both the Applicant and the owner of the Cited Registrations are marketed as gluten-free, and thus the goods are in the same narrow market of "gluten free salty snacks."

Applicant respectfully disagrees with the Examiner's conclusions. PEACE, LOVE AND GLUTEN FREE is not likely to be confused with PEACE, LOVE AND CRUNCH because the *overall* marks are distinct in appearance, sound, meaning and commercial impression, despite the Examiner's argument that PEACE, LOVE is the dominant and distinctive portion of the marks. In addition, even if Applicant's goods and the owner of the Cited Registrations' goods are marketed together in outlets such as grocery stores, it does not follow that consumers are likely to be confused because grocery stores sell thousands of items, and reasonable consumers would not assume that all of these items emanate from the same source. Finally, the fact that the goods of the parties are both gluten-free supports Applicant's argument that consumers who are on a gluten-free diet are discerning and sophisticated, and would unlikely to be confused.

3. <u>The Presence of Identical Dominant Terms Does Not Necessarily Make Two Marks Confusingly</u>

Similar

The Examiner argued that PEACE, LOVE is the dominant portion of the registrant's and Applicant's mark, and that GLUTEN FREE and CRUNCH, being the descriptive portions, are not the dominant features of the trademarks. While it is true that "dominant features will, of course, weigh heavier in the overall impression of a mark, . . . no element of a mark is ignored simply because it is less dominant, or would not have trademark significance if used alone." *In re Electrolyte Laboratories Inc.*, 16 U.S.P.Q.2d 1239, 1240 (Fed. Cir. 1990). Thus, even assuming, without Applicant's admitting, that the dominant portion of its mark is PEACE, LOVE, it does not automatically follow that PEACE, LOVE AND GLUTEN FREE is confusingly similar to PEACE, LOVE & CRUNCH. *Gen. Mills, Inc. v. Kellogg Co.*, 824 F.2d 622, 627 (8th Cir. 1987) ("The use of identical, even dominant, words in common does not automatically mean that two marks are similar."). The proper comparison is between the overall commercial impressions of the marks. PEACE, LOVE AND GLUTEN FREE and PEACE, LOVE & CRUNCH have different ***overall*** appearances, sounds, meanings and commercial impressions, and thus consumer confusion is unlikely.

4. The Parties' Goods Are Different

The Examiner argued that potato chips and crackers are the type of snacks that emanate from the same source. In particular, the Examiner provided the web pages created by an individual advocate of a gluten-free diet as proof that Frito-Lay markets its gluten-free chips alongside regular snacks (Exhibit A). There is no evidence that these web pages reflect market reality, as this is merely an informational website created by an individual about gluten-free snacks.

Even if gluten-free chips and snacks are marketed together in grocery stores or websites featuring groceries or snacks, "there exists no 'per se' rule that all food products are to be deemed related goods by nature or by virtue of their capability of being sold in the same food markets." *In re August Storck KG*, 218 U.S.P.Q. 823, 824 (T.T.A.B. 1983); *Riviana Foods, Inc. v. Societe Des Products Nestle S.A.*, 33 U.S.P.Q.2d 1669, 1670 (S.D. Tx. 1994) ("Both products are sold primarily in grocery stores and supermarkets. This is of little significance, however, because approximately 15,000-20,000 products are sold in today's supermarkets.").

Therefore, even if snacks and gluten-free potato chips are sold by the same retail outlets, this fact is of little significance because grocery stores sell a wide variety of products and consumers do not assume that all goods sold at these stores emanate from the same source.

5. Individuals on a Gluten-Free Diet are Sophisticated and Discerning

As Applicant previously argued in the response to the Non-final Office Action, a gluten-free diet is used to treat celiac disease, and consumers with the disease are likely to exercise more care when purchasing gluten-free products. Individuals allergic to gluten may suffer from schizophrenia, headaches, pain or diarrhea (Exhibit B), and it is highly likely that these individuals would be discerning and educated about the products. If these individuals do not exercise care when purchasing gluten-free products, it could cause them great physical harm.

Therefore, the fact that Mary's Gone Crackers, Inc. and Applicant both target consumers on a gluten-free diet, as the Examiner pointed out, weighs against likelihood of confusion because this class of consumers are sophisticated and discerning.

6. Conclusion

Applicant's registration of its PEACE, LOVE AND GLUTEN FREE mark is not likely to lead to confusion with the Cited Registrations because the *overall* marks are different in appearance, sound, meaning and commercial impression, are for different products that are sold in grocery stores that also sell thousands of other items that consumers do not reasonably assume that they all emanate from the same source, and consumers for gluten-free products are sophisticated and discerning. As such, Applicant respectfully requests approval of the PEACE, LOVE AND GLUTEN FREE application for publication.

EVIDENCE SECTION	
EVIDENCE FILE NAME(S)	
ORIGINAL PDF FILE	evi_156471510-153154016_._peace__love_and_gluten_free_exhibits.pdf
CONVERTED PDF FILE(S) (22 pages)	\\TICRS\EXPORT11\IMAGEOUT11\851\841\85184182\xml8\RFR0002.JPG
	\\TICRS\EXPORT11\IMAGEOUT11\851\841\85184182\xml8\RFR0003.JPG
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DESCRIPTION OF EVIDENCE FILE	third party website printouts
GOODS AND/OR SERVICES SECTION (current)	
INTERNATIONAL CLASS	029
DESCRIPTION	Gluten-free potato chips
FILING BASIS	Section 1(a)
FIRST USE ANYWHERE DATE	At least as early as 07/01/2007
FIRST USE IN COMMERCE DATE	At least as early as 07/01/2007
GOODS AND/OR SERVICES SECTION (proposed)	
INTERNATIONAL CLASS	029
DESCRIPTION	Gluten-free potato chips
FILING BASIS	Section 1(a)
FIRST USE ANYWHERE DATE	At least as early as 11/13/2007
FIRST USE IN COMMERCE DATE	At least as early as 11/13/2007
SIGNATURE SECTION	
DECLARATION SIGNATURE	/pouibonnielee/
SIGNATORY'S NAME	Pou-I "Bonnie" Lee
SIGNATORY'S POSITION	Attorney of record, New York bar member
DATE SIGNED	03/14/2012
RESPONSE SIGNATURE	/pouibonnielee/
SIGNATORY'S NAME	Pou-I "Bonnie" Lee
SIGNATORY'S POSITION	Attorney of record, New York bar member
DATE SIGNED	03/14/2012
AUTHORIZED SIGNATORY	YES
CONCURRENT APPEAL NOTICE FILED	YES

FILING INFORMATION SECTION	
SUBMIT DATE	Wed Mar 14 15:42:49 EDT 2012
TEAS STAMP	USPTO/RFR-156.47.15.10-20 120314154249075075-851841 82-490460ce38ac4f39efe36f 1b97f13512e9-N/A-N/A-2012 0314153154016795

PTO Form 1930 (Rev 9/2007)
OMB No. 0651-0050 (Exp. 4/30/2009)

Request for Reconsideration after Final Action

To the Commissioner for Trademarks:

Application serial no. **85184182** has been amended as follows:

ARGUMENT(S)
In response to the substantive refusal(s), please note the following:

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EVIDENCE
Evidence in the nature of third party website printouts has been attached.
Original PDF file:
evi_156471510-153154016_._peace__love_and_gluten_free_exhibits.pdf
Converted PDF file(s) (22 pages)
Evidence-1
Evidence-2
Evidence-3
Evidence-4
Evidence-5
Evidence-6
Evidence-7
Evidence-8
Evidence-9
Evidence-10
Evidence-11
Evidence-12
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Evidence-19
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Evidence-21
Evidence-22

CLASSIFICATION AND LISTING OF GOODS/SERVICES
Applicant proposes to amend the following class of goods/services in the application:

Current: Class 029 for Gluten-free potato chips
Original Filing Basis:
Filing Basis: Section 1(a), Use in Commerce: The applicant is using the mark in commerce, or the applicant's related company or licensee is using the mark in commerce, on or in connection with the identified goods and/or services. 15 U.S.C. Section 1051(a), as amended. The mark was first used at least as early as 07/01/2007 and first used in commerce at least as early as 07/01/2007, and is now in use in such commerce.

Proposed: Class 029 for Gluten-free potato chips
Filing Basis: Section 1(a), Use in Commerce: The applicant is using the mark in commerce, or the applicant's related company or licensee is using the mark in commerce, on or in connection with the identified goods and/or services. 15 U.S.C. Section 1051(a), as amended. The mark was first used at least as early as 11/13/2007 and first used in commerce at least as early as 11/13/2007, and is now in use in such commerce.
SIGNATURE(S)
Declaration Signature
If the applicant is seeking registration under Section 1(b) and/or Section 44 of the Trademark Act, the applicant has had a bona fide intention to use or use through the applicant's related company or licensee the mark in commerce on or in connection with the identified goods and/or services as of the filing date of the application. 37 C.F.R. Secs. 2.34(a)(2)(i); 2.34 (a)(3)(i); and 2.34(a)(4)(ii); and/or the applicant has had a bona fide intention to exercise legitimate control over the use of the mark in commerce by its members. 37 C.F. R. Sec. 2.44. If the applicant is seeking registration under Section 1(a) of the Trademark Act, the mark was in use in commerce on or in connection with the goods and/or services listed in the application as of the application filing date or as of the date of any submitted allegation of use. 37 C.F.R. Secs. 2.34(a)(1)(i); and/or the applicant has exercised legitimate control over the use of the mark in commerce by its members. 37 C.F.R. Sec. 2.44. The undersigned, being hereby warned that willful false statements and the like so made are punishable by fine or imprisonment, or both, under 18 U.S.C. Section 1001, and that such willful false statements may jeopardize the validity of the application or any resulting registration, declares that he/she is properly authorized to execute this application on behalf of the applicant; he/she believes the applicant to be the owner of the trademark/service mark sought to be registered, or, if the application is being filed under 15 U.S.C. Section 1051(b), he/she believes applicant to be entitled to use such mark in commerce; to the best of his/her knowledge and belief no other person, firm, corporation, or association has the right to use the mark in commerce, either in the identical form thereof or in such near resemblance thereto as to be likely, when used on or in connection with the goods/services of such other person, to cause confusion, or to cause mistake, or to deceive; that if the original application was submitted unsigned, that all statements in the original application and this submission made of the declaration signer's knowledge are true; and all statements in the original application and this submission made on information and belief are believed to be true.

Signature: /pouibonnielee/ Date: 03/14/2012
Signatory's Name: Pou-I "Bonnie" Lee
Signatory's Position: Attorney of record, New York bar member

Request for Reconsideration Signature
Signature: /pouibonnielee/ Date: 03/14/2012
Signatory's Name: Pou-I "Bonnie" Lee
Signatory's Position: Attorney of record, New York bar member

The signatory has confirmed that he/she is an attorney who is a member in good standing of the bar of the highest court of a U.S. state, which includes the District of Columbia, Puerto Rico, and other federal

territories and possessions; and he/she is currently the applicant's attorney or an associate thereof; and to the best of his/her knowledge, if prior to his/her appointment another U.S. attorney or a Canadian attorney/agent not currently associated with his/her company/firm previously represented the applicant in this matter: (1) the applicant has filed or is concurrently filing a signed revocation of or substitute power of attorney with the USPTO; (2) the USPTO has granted the request of the prior representative to withdraw; (3) the applicant has filed a power of attorney appointing him/her in this matter; or (4) the applicant's appointed U.S. attorney or Canadian attorney/agent has filed a power of attorney appointing him/her as an associate attorney in this matter.

The applicant is filing a Notice of Appeal in conjunction with this Request for Reconsideration.

Serial Number: 85184182
Internet Transmission Date: Wed Mar 14 15:42:49 EDT 2012
TEAS Stamp: USPTO/RFR-156.47.15.10-20120314154249075
075-85184182-490460ce38ac4f39efe36f1b97f
13512e9-N/A-N/A-20120314153154016795

EXHIBIT A

Home
Most Recent Updates
Gluten Sensitivity
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Quick Start Guide
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Kitchen Prep
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Disclaimer
Advertising Policy

[?] Subscribe To This Site
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Bloglines

About Us

Welcome to Gluten-Free-Diet-Help.com!

Why I Created This Site

My husband, Paul, was diagnosed with Osteoporosis at the age of 45. I had Chronic Fatigue and Fibromyalgia. Although our symptoms were very different, it turned out both of our symptoms were related to gluten.

Since going gluten and casein free in November of 2006, we have seen many changes.

My energy is better, the Fibromyalgia is gone, skin rashes are gone, and no more brain fog!

Paul has not had a migraine in 2 years! His hyperactivity disappeared after 10 months on the diet. His bone mass is slowly improving.

At our age it takes at least two years to heal. We are looking forward to even more improvement.

I was fortunate in having the time to read and read and learn about the gluten free diet.

I want to pass along what I've learned to others. To give the struggling mom who works full time especially, a resource. A place to get the information to make shopping easier. Recipes and cooking techniques to prepare gluten free meals. A head start, with everything in one place.

That's why I created this website. I hope it will be of great help to you!

Note: We do not give medical advice, as we are not medical professionals. Just some guidance on the practical aspects. If you have medical questions, please contact your doctor.

from About Us back to Gluten Free Diet Help Home Page

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Frs|ujkw› 533;05345#Dozqhq Uuhh0Glhw Khos1frp

EXHIBIT B

Gluten sensitivity

From Wikipedia, the free encyclopedia

Gluten sensitivity (also known as "gluten intolerance") (GS) belongs to a spectrum of disorders in which gluten has an adverse effect on the body. It can be defined as a non-allergic and non-autoimmune condition in which the consumption of gluten can lead to symptoms similar to those observed in celiac disease or wheat allergy (other conditions which fall under the gluten-related disorders spectrum).

Gluten sensitivity is thought to affect approximately 6% of the general population.[1] Symptoms of gluten sensitivity include bloating, abdominal discomfort, pain or diarrhea; or it may present with a variety of extraintestinal symptoms including headaches and migraines, lethargy and tiredness, attention -deficit disorder and hyperactivity, schizophrenia, muscular disturbances as well as bone and joint pain.[2][3][4][5]

Until recently, the terms gluten sensitivity and celiac disease were used interchangeably in literature. However, emerging research is beginning to identify the differences that exist between celiac disease and gluten sensitivity. If the medical history of a patient, along with clinical tests, rule out celiac disease and wheat allergy, a diagnosis of gluten sensitivity can be considered. However, certain criteria need to be met before a diagnosis of gluten sensitivity can be confirmed (see diagnosis section). Treatment for all three conditions is a gluten-free diet; the difference being that with wheat allergy the interruption is temporary and drugs may be administered; in the case of celiac disease the diet is lifelong and even ingesting very small amounts of gluten-containing food could damage their health and, in the case of gluten sensitivity the withdrawal of gluten from the diet may only be temporary.

Gluten is a protein composite found in foods processed from wheat and related species, including barley and rye. It gives elasticity to dough helping it to rise and to keep its shape. It is found in many staple foods in the Western diet. Gluten is composed of a gliadin fraction (alcohol soluble) and a glutenin fraction (only soluble in dilute acids or alkali).

Contents

Symptoms

Symptoms of gluten sensitivity may include bloating, abdominal discomfort, pain, or diarrhea; or it may present with a variety of extraintestinal symptoms including headaches and migraines, lethargy and tiredness, attention-deficit disorder and hyperactivity, muscular disturbances as well as bone and joint pain.[4][5][6]

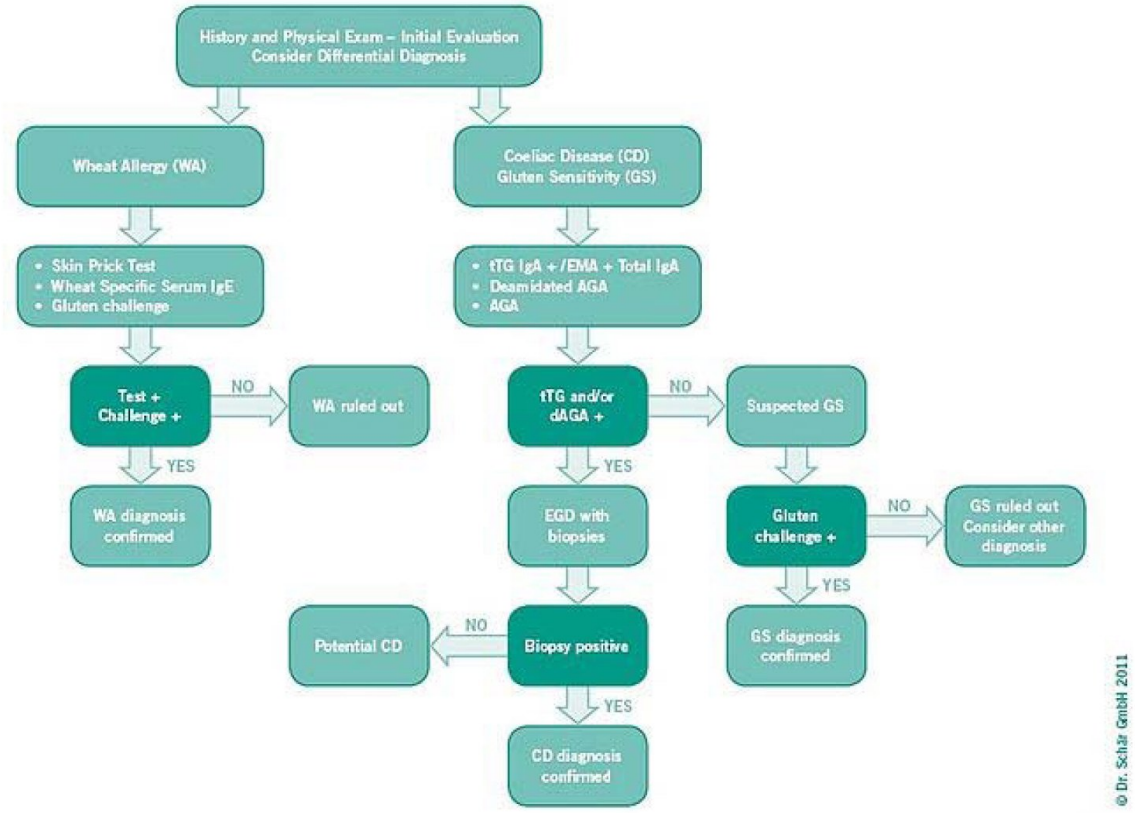
Diagnosis of Gluten Sensitivity

If the medical history of a patient, along with clinical tests, rule out celiac disease and wheat allergy, a diagnosis of gluten sensitivity can be considered. However, before a diagnosis of gluten sensitivity can be confirmed the following criteria need to be met:

- Wheat allergy excluded (anti IgE antibody negative)
- Celiac disease excluded (negative serological results of tTG/EMA/dAGA and IgA deficiency)
- Not HLA restricted- the absence of HLA DQ2/8 heterodimers makes it possible to practically rule out celiac disease, however, it is also important to note that the presence of HLA DQ 2 or DQ8 does not always mean celiac disease
- Intestinal biopsy shows no villous atrophy (Marsh III classification) but there may be minimal changes to the lining of the gut (Marsh 0-I classification)
- Possible presence of serum anti-gliadin antibodies (AGA) IgA and/or IgG investigated
- If the patient reports alleviation of symptoms on a gluten-free diet

If the above criteria are met then the person can be classed as ‘gluten sensitive’ and it can be treated with a gluten-free diet for a period of time. This should lead to symptom resolution.

A diagnostic algorithm for gluten sensitivity has been proposed:



Difference between gluten sensitivity and celiac disease

Whilst the research surrounding gluten sensitivity is still very much emerging, celiac disease is a well-defined condition. It is a lifelong autoimmune condition characterised by the chronic inflammation of the intestine. In genetically predisposed children and adults the intake of foods containing gluten leads to an immune response in the small intestine. This results in the flattening of the intestinal villi and in reduced absorption of nutrients from food which can lead to nutritional deficiencies and associated long-term complications such as osteoporosis. It is believed celiac disease affects 1% of the general population in the Western world.

In comparison, in a recent clinical paper, gluten sensitivity was defined as 'one or more of a variety of immunological, morphological or symptomatic manifestations that may also be shared by celiac disease and irritable bowel syndrome (IBS).[7] In cases where there is reactivity to gluten, yet celiac disease and wheat allergy are eliminated as possibilities, gluten sensitivity may be considered. Whilst the general clinical picture for gluten sensitivity is similar to celiac disease in particular, it is usually less severe and neither anti-tissue transglutaminase antibodies nor autoimmune comorbidities are found.

It is believed that approximately 40-50% of gluten sensitivity patients may have IgG or IgA anti-gliadin antibodies (AGA)[8][9] There is also a study identifying approximately 50% of gluten sensitivity patients, few more than the general population, carry either HLA DQ 2 or 8 [10]

On closer inspection, it has also been found that gluten-sensitive subjects do not develop full histological lesions; their lesions, if any, are limited to types 0-1 of the Marsh classification. In addition, it has been found that they have normal intestinal permeability and an increased expression of Toll Like receptors 2 (TLR2) but no change in the cytokines involved in adaptive immune responses Th1 and Th17 such as IL -6, IL-17 A, IL 21, which are increased only in patients with celiac disease. The knowledge of the response in gluten sensitivity to date suggests that only the innate immune system is involved, whereas celiac disease is an adaptive immune response (autoimmunity).

Gluten sensitivity should have a defined cause, although not apparent always with first examination, affected individuals should eventually fall into GSE or wheat allergy. Only rarely should gluten sensitivity be idiopathic. Idiopathic gluten sensitivity (IGS) arises *spontaneously or from an obscure or unknown cause* and may involve neuropathy, myopathy, dermal, or intestinal abnormalities. Anti-gliadin antibodies are the primary link between gluten and idiopathic sensitivity in instances "in which enteropathy or allergy are not clearly involved".[11] This form of gluten sensitivity is controversial at the moment but there is a growing body of research to support the concept of gluten sensitivity that is different from celiac disease and wheat allergy.

Etiology

Gluten sensitivity can develop at any point in life, and symptomatic disease may appear years after disease develops. When enteropathy develops in early childhood symptomatic disease is more rapidly evident. A survey of geriatrics with celiac disease in Finland[12] revealed that the incidence of disease was much higher than the general population. Allergic disease may rise or fall with age; however, certain evidence points to the increased or daily use of non-steroidal anti-inflammatory factors (aspirin, ibuprofen) as an increased risk factor for urticaria or anaphylaxis, and the sensitizing dose may include low-dose aspirin therapy used in the treatment of heart disease. Idiopathic disease appears largely late onset.

Gluten-sensitive enteropathy develops as a consequence of genetic and environmental factors. Other than the involvement of certain HLA-DQ isoforms (antigen presenting proteins in humans) and certain wheat proteins, there is no clarity in the involvement of other genes or other environmental factors (see risk modifiers). Strong genetic factors such as seen in GSE have not been seen in gluten allergy, and with idiopathic gluten-sensitivity the HLA-DQ associations are weak.

Researchers reported extreme fatigue and pain in patients without celiac disease, with gliadin antibodies. They called this a “non-celiac gluten intolerance” for which there is no explanation as to the mechanisms involved.[13]

Causes of gluten sensitivity

Triticeae and the potential role of selective evolution in gluten sensitivities

The fruiting bodies of plants contain genes as well as reserves of nutrients that allow seedlings to grow. The enrichment of nutrients is an attractant to herbivores and omnivores. For annual grasses that release

Underlying conditions

The normal intestine

Wheat proteins interact with the immune system by means of DQ2-mediated programmed cell death (apoptosis) of the gut in sensitive individuals. New research is finding that the celiac gut may be predisposed to sensitivity in the absence of HLA genetic factors.

seeds during a brief period each year there is a need to protect seeds during maturation from insects or animals, which might stock seeds for year round usage. For wheat, alpha-gliadins are seed-storage proteins, but also act as inhibitors of alpha -amylase activity in some animals, particularly in insects.[14] It is also known that wheat gliadins create intestinal disease when fed to very young rodents.[15]

One recent publication even raises the question 'is wheat safe for anyone to eat?'.[16] Critically, pathology in insects or artificially fed rodents does not reflect what causes disease in humans, but it is interesting that toxicological effects of wheat are being uncovered that do have the potential to cause pathology in humans. One interesting consequence of these studies is that there may be a general gluten sensitivity that underlies various pathological manifestations, such as celiac disease, urticaria and idiopathic sensitivity.

The rise of gluten sensitivity (particularly in adults) may reflect the convergence of many phenomena. An aging population, genetic risks associated with westernization, excesses in the diet, sensitizing chemicals (e.g.NSAIDs),[*citation needed*] and allergy-enhancing chemical treatment of foods (e.g. enzymatic deamidation of gluten) may act together with natural defensive agents in foods to cross the threshold between normality and pathology.[*citation needed*]

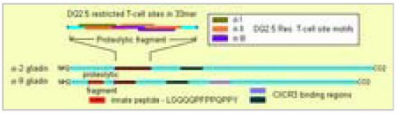


Illustration of 2 alpha gliadins showing 2 proteolytically resistant sites, Top shows 6 T-cells sites in 33mer, and bottom shows innate immune peptide and two CXCR3 binding sites

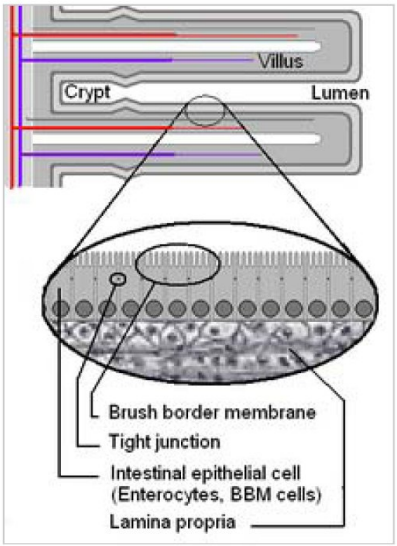
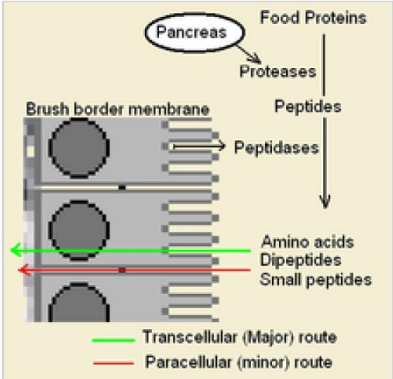


Illustration of the brush border membrane of small intestinal villi

How diet proteins reach the blood



The fate of digestible protein in the small intestine

In the normal gut, proteins are digested to peptides by pepsin (stomach), trypsin and chymotrypsin (derived from the pancreas and activated in the gut). Peptides are further digested when they enter the villi, where brush border peptidase break proteins into amino acids. Over much of the small intestine only small solutes, like water, can cross the tight junctions, however some regions of the intestine peptides as large as 500 daltons (4 amino-acids residues in length can cross).

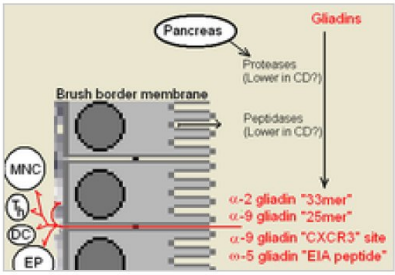
The gluten sensitive gut

Gluten toxicity

Further information: Innate immunity of gluten

An increasing number of studies on gliadin indicate gluten has a direct and modifying effect on the cells of the small intestine. Two different lines of research show that different gliadins can increase permeability of the epithelial cells (outermost cells of the villus) allowing food proteins to enter. One study examined the effect of ω-5 gliadin, the primary cause of Wheat Dependent exercise/aspirin induced anaphylaxis, and found increased permeability of intestinal cells caused by this gliadin and another wheat albumin.[17]

Another line of research shows gliadin binds a chemoattractant receptor and causes increases of a factor that destroys tight junctions.[18] These junctions prevent leakage around the cells that line the small intestine, resulting in the leaking of food proteins into the body.[19] These toxicities of gluten that are not part of the adaptive immune response may be the link between wheat and gluten sensitivity, and possibly type 1 diabetes.



The fate of gluten in celiac disease or EIA

There is a growing body of evidence that the gluten-sensitive intestine differs from the normal gut. Several gluten peptides can infiltrate the region behind the cells lining the small intestine. The "33mer" of α-2 gliadin is a magnitude larger than the maximum size allowable by the barrier around the cell, the tight junctions. Omega-5 gliadin peptides have been found in the blood stream of people with exercise-induced anaphylaxis, aided by salicylates. And the innate "25mer" is capable of reaching mononuclear cells in celiac gut, but in normal gut is broken down by brush border peptidases. It may be a lower peptidase activity that explains the presence of these peptides behind the brush border membrane. Recently, it was found that an α-9 gliadin peptide was capable of binding the "CXCR3" receptor, increasing zonulin production and weakening tight junctions, this may explain how, generally, larger peptides can enter the gluten-sensitive gut.

Immunochemistry of glutens

Main article: Gluten immunochemistry

Triticeae glutens are important factors in several inflammatory diseases. The immunochemistry can be subdivided into innate responses (direct stimulation of immune system), class II mediated presentation (HLA DQ), class I mediated stimulation of killer cells, and antibody recognition. The responses to gluten proteins and polypeptide regions differs according to the type of gluten sensitivity. The response is also dependent on the genetic makeup of the human leukocyte antigen genes. In enteropathy, there are at least 3 types of recognition, innate immunity (a form of cellular immunity priming), HLA-DQ and antibody recognition of gliadin and transglutaminase.[20]

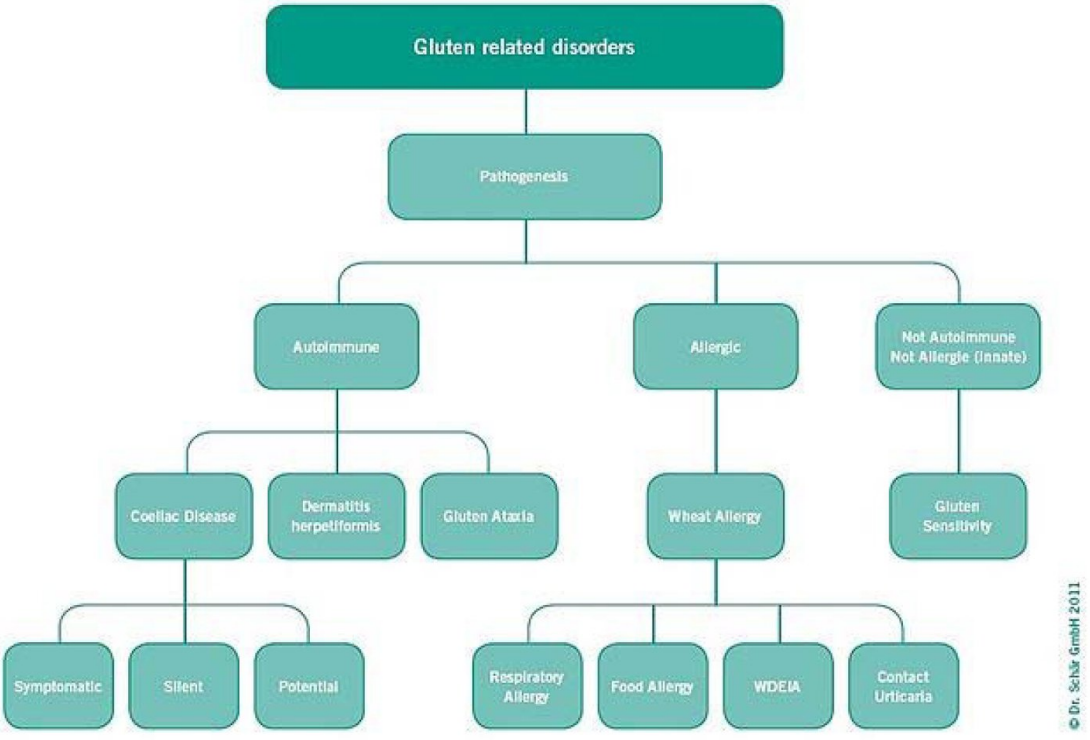
The three dominant sequences responsible for the antibody reaction have been identified.[21][22] With idiopathic disease only antibody recognition to gliadin has been resolved. In wheat allergy, there appears to be an innate components and the response pathways are mediated through IgE against gliadin and other wheat proteins.[23][24][25]

Separating forms of gluten sensitivity

Only rarely should gluten sensitivity be without cause. Generally the sensitivity can be split between celiac disease, gluten sensitivity and wheat allergy. Since individuals with celiac disease can also have wheat allergy, a finding of wheat allergy does not eliminate the possibility of enteropathy. Individuals highly suspect of celiac disease may be tested for anti-transglutaminase antibodies followed by duodenal biopsy, this will confirm or refute active celiac disease.[26] The study that recommends this, however, has a number of ATA positive/biopsy-negative individuals, this could result from patchy villous atrophy or subclinical pathology.[27][28]

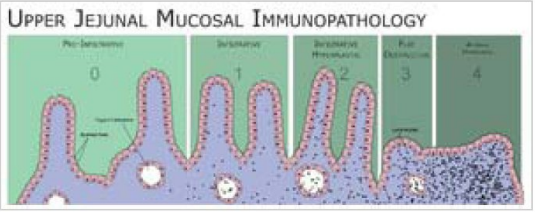
One current study recommended at biopsy samples running distally from the duodenum to avoid the risk of false negatives. Eliminating the possibility of celiac disease can generally be done by adding HLA-DQ typing, in which DQ2 and DQ8 are found in enteropathy 98% of the time in caucasians, DQ7.5 the remaining 1.6% and 0.4% not found with either of these 3. Without ATA or HLA-DQ2/8 positivity, celiac disease is not likely the cause of the sensitivity. In either case, other avenues of diagnostics, such as allergy testing are available.[29]

Rarely gluten sensitivity may be idiopathic, a potential that wheat proteins play a role in other disease, in these instances DQ1 may be associated with sensitivity. There is research showing that in certain patients with gluten ataxia early diagnosis and treatment with a GFD can improve ataxia and prevent its progression.[30] Recently, a classification of gluten-related disorders has been proposed:



Gluten-sensitive enteropathy (GSE)

Main article: Coeliac disease (CD)



Schematic of the Marsh classification of upper jejunal pathology in celiac disease.

Celiac disease as the classically defined gluten-sensitivity and dermatitis herpetiformis was appended to a broadening definition of gluten sensitivity. The diagnostic "gold standard" of celiac disease is the villus atrophy detected in duodenal biopsies. However, it is now recognized that inflammation of the epithelial tissue of the small intestine precedes atrophy. Early in the disease, gluten elicits T-lymphocyte recognition of gluten hydrolysates (polypeptides of gluten) and gluten peptides bind to mammalian tissue transglutaminase (tTG).

This second interaction results in the production of "self" antibodies to tTG. This increases lymphocytes within the epithelia of the small intestine (Marsh grade 1 and 2) and antibody-tTG complexes seen as deposits. This usually progresses to celiac disease (Marsh grade 3 and 4). The dietary cause of GSE is not limited to wheat gluten; 'glutens' from all known edible cultivars of *Triticeae* can induce GSE in susceptible individuals (see: Gluten immunochemistry).

Main article: Associated Conditions

There are a large number of medical conditions that result from GSE that can occur prior to the development of celiac disease and might be gluten responsive. While the level of villus atrophy in some cases of GSE may not reach clinical celiac disease recognition, the elevation of cellular immunity is capable of producing disorders more frequently found in celiac disease. Conditions secondary to GSE are important diagnostic criteria for gluten sensitivity when there may be no obvious intestinal abnormality.

Presentation of GSE is often the result of initial recognition of the secondary condition which in followup testing (ATA test, AGA test, HLA-DQ typing, and/or biopsy) recognizes the primary condition. The secondary conditions associated with GSE tend to make late onset celiac disease a systemic phenomena.

Frequencies of phenotypes in Celiac disease, Normal Americans, Odds ratios

Idiopathic gluten sensitivity

Idiopathic diseases are proposed as an expansion of the gluten-sensitivity. By the definition of idiopathic disease, the cause is not well defined. One hundred years ago, before gluten was discovered as the cause of celiac disease, celiac disease in adults was called adult idiopathic steatorrhoea, non-tropical sprue, sprue nostras, and many other names. The debate over this subset stems from the fact that identification of all grades of GSE and allergies is not uniformly approached. Most cases of early GSE go undetected, particularly before 2005.

There appears to be a small fraction of non-GSE gluten-sensitive individuals that show neither gluten-allergies but do have elevated anti-gliadin IgA or IgG. Common symptoms are peripheral neuropathies and cerebellar ataxia. Within the GSE set these may be explained by calcification of brain channels and avitaminosis. Within the remaining 'DQ2 and DQ8'less cohort. Given that this cohort of GS is idiopathic, the role of allergies, other sensitivities (e.g. aspirin), or other factors in IGS is also unresolved.

Further information: Anti-gliadin antibodies

Silent Disease. Depending on testing somewhere between 3 and 15% of the normal population have anti-gliadin antibodies (AGA). Studies using anti-gliadin antibodies (AGA) reveal that in undiagnosed or untreated individuals with AGA, with increasing risk for lymphoid cancers and decreased risk for other associated with affluence.[31] Though it is unknown in these studies the percentage that are early stage GSE.

	DQ haplotypes -Celiac Disease				
DQ hap	**2.5**	**2.2**	**7.5**	**8.0**	**Other**
2.5	34	22	4.0	2.0	22
2.2		1.1	4.0	1.1	2.9
7.5			0.3	0.0	1.3
8.1				2.9	2.0
other					0.4
	DQ haplotypes -Normal Population				
DQ hap	**2.5**	**2.2**	**7.5**	**8.1**	**Other**
2.5	1.7	2.9	2.9	1.8	15.1
2.2		1.2	2.4	1.6	12.8
7.5			1.2	1.5	1.3
8.1				0.5	8.0
other					33.4
	Odds ratios				
DQ hap	**2.5**	**2.2**	**7.5**	**8.1**	**Other**
2.5	20:1[1]	8:1[2]	1.4:1[6]	1.1:1	1.5:1[5]
2.2		1:1.1	1.6:1[4]	1:1.3	1:5
7.5			1:4	0	1:10
8.1				6:1[3]	1:4
other					1:100
	DQ Types by allele numbers (e.g. 0501) can be found here				

Neuropathies

Main article: Gluten-sensitive idiopathic neuropathies

Other conditions

Antibodies to α-gliadin have been significantly increased in non-celiacs individuals with oral ulceration.[32] Anti-α-gliadin antibodies are frequently found in celiac disease(CD), to a lesser degree subclinical CD, but are also found in a subset who do not have the disease. The 1991 reference comes from a period when testing for subclinical CD was undeveloped. Of people with pseudo-exfoliation syndrome, 25% showed increased levels of anti-gliadin IgA.[33] One fourth of people with Sjögren's syndrome had responses to gluten, of 5 that had positive response to gluten, only one could be confirmed as CD and another was potentially GSE, the remaining 3 appear to be gluten-sensitive. All were HLA-DQ2 and/or DQ8-positive.[34]

Treatment to produce remission of Crohns disease(CrD) symptoms on elimination diet indicated the most important foods provoking symptoms were wheat and dairy.[35] A later paper showed little IgE mediated response except to the dairy,[36] while another paper showed no significant anti-food IgE association.[37] Crohn's disease (CrD) may have a link to wheat that is independent of gluten. CrD appears to be associated with high anti-yeast antibodies (ASCA - yeast antigens that are found in bread and other cereal derived products) and affected individuals lack lectin binding proteins such that the mannins in yeast, the antibodies that bind them and aggravate inflammatory colitis. One concern of the above studies is the high prevalence of markers for gluten-sensitive enteropathy, one has to question how idiopathic these conditions are if close examination for GSE has not been undertaken.

Gluten-allergy related sensitivities

Main article: Wheat allergy

Why treat gluten allergies as sensitivities? Over the last 10 years it has become apparent that allergies to certain substances do not behave in predictable ways. One clear example of this is exercise induced anaphylaxis and asthma, WDEIA (Wheat Dependent Exercise Induced Anaphylaxis) is now believed to be induced by ingested gluten that finds a way into the blood stream. This pathway is now believed responsible for some forms of eczema.[*citation needed*] Recent studies on two wheat allergens show that they possess the capability of bypassing the gut/blood barrier. The most active of these is ω-5 gliadin, a gluten component that is a strong allergen and causes WDEIA. Allergy tests may not reveal allergies to gluten because the unfractionated allergens are 'hidden' from these tests, and most currently available tests cannot detect these new allergens. Finally, allergies typically involve IgE, but some studies indicate there are several classes of responses, for example IgG1,IgG2, IgG4 that are associated with IgE.[38] Gluten allergy may be a cause of some idiopathic gluten sensitivity and gluten allergy can be a secondary consequence of gluten-sensitive enteropathy.

Comparative pathophysiology

Comparison of different forms of gluten sensitivity

	Gluten-sensitive enteropathy	Wheat allergy	Gluten-sensitive idiopathic neuropathy
Typical symptoms	steatorrhoea, malnutrition, diarrhea, lactose intolerance, food allergies	eczema, asthma	ataxia, peripheral neuropathies
Primary tissue targets	epithelia of small intestine	(epi) dermis, bronchi, intestines	CNS, Peripheral nerves
Atypical pathologies	other autoimmune diseases, chronic constipation, neuropathies, cancer (lymphoid)	arthritis, migraines, anaphylaxis (exercise or aspirin induced)	unknown
Secondary targets (common)	blood (chemistry), bowel, nervous system, autoantigens	connective tissue, CNS, vascular	
Immunoglobin isotype	IgA, IgG	IgE, IgG, IgA	IgG, IgA
Antibody recognition	α/β,γ-gliadin (AGA), transglutaminase (ATA)	albumins, globulins, prolamins (ω-gliadin)(AGA), glutelins (LMW)(AGA)	α/β-gliadin
HLA associations	DQ2.5, DQ8, DQ2.2/DQ7.5	unknown	DQ2, DQ8?, DQ1?
Cellular immunity	T-cells, Eosinophils, Monocytes	Mast cells, Eosinophils	unknown
Innate responses	(α-gliadin) immune, increased permeability	(ω-5 gliadin)- increased permeability	unknown
Background & references	Celiac disease, GSEA conditions	Wheat allergy	IGS Neuropathies

Notes on table. Features of idiopathic neuropathy assume that all GSE cohort has been removed, assuming there is a gluten-sensitive, but not GSE contingent. Anti-gliadin antibodies covers all immunoglobulin isotypes and all gliadin isoforms. T-cell, Killer cell, and other gluten recognitions are covered in Gluten immunochemistry.

Gluten sources

From the perspective of gluten sensitivity there is no single definition of gluten that concisely defines all potentially pathogenic glutens. With wheat allergies, there can be a wide spectrum of species that may trigger allergies with similar proteins, the omega-gliadin proteins have similar proteins found in oats at high frequency, but omega-gliadin allergy is not a predictor of oat allergy or intolerance.[45] A person can have an allergy to wheat, but not rye.[46]

Glutelins have not been characterized over broad taxa. With idiopathic gluten sensitivity, the antibodies that correlate with disease are anti-gliadin antibodies. Whether these antibodies are pathogenic or are simply indicators of circulating gliadin is unknown. For gluten-sensitive enteropathy, gliadin and homologous proteins from rye and barley cause disease. T-cell epitopes implicated in disease have been found in glutinous protein genes in all species sequenced within the tribe *Triticeae*.[47]

Also, since barley is distantly related to wheat, but carries pathogenic epitopes it can be assumed that all members of *Triticeae* should carry T-cell sites capable of sustaining disease (see also Genetics of Triticeae). While often not explicitly stated in some standards, pathogenic glutens found in wheat are also found in Spelt and Kamut(both types of wheat), Triticale (a trans-species Triticeae hybrid).

The oat controversy

Oats are a species within the grass tribe *Aveneae*, which is in the *Pooideae* subfamily along with *Triticeae* (contains wheat, rye, barley and many other genera). Oats are the most closely related cereal species to *Triticeae* cereals. Some, but not all, cultivars of oat contain the pathogenic

Politics of Gluten-Free and Oats

Current guidelines

As a consequence, the current international standard for the "Gluten-free" designation, drafted in 1981 and agreed on in 1983[39] within the Codex Alimentarius (CA), states:

> *For the purpose of this standard, gluten is defined as those proteins, commonly found in wheat, triticale, rye, barley or oats to which some persons are intolerant.*[40]

The American Dietetic Association's *Nutrition Care Manual* position on the use of oats in a medically necessitated gluten-free diet is:

> *However, commercially available oats in the United States may be contaminated with small amounts of wheat, barley, or rye. For this reason, if you are newly diagnosed with celiac disease, you should not eat oats. Once your intestine heals, you may want to discuss the use of oats with your dietitian and physician.*[41]

indicating the need for a separate standard of purity for people with gluten sensitivity.

New standards in development

Codex Alimentarius is undergoing revision and a revised standard will be presented at the meeting of the Codex Alimentarius Commission at the end of June 2008.[42] The proposed standard limits the amount of contaminant in product that would qualify that product as gluten-free:

> *Gluten-free foods are dietary foods a) consisting of or made only from one or more ingredients which do not contain wheat (i.e., all Triticum species, such as durum wheat, spelt, and kamut), rye, barley, oats[1] or their crossbred varieties, and the gluten level does not exceed 20 mg/kg in total, based on the food as sold or distributed to the consumer, and/or b) consisting of one or more ingredients*


Oat grains in their husks

proteins that provoke a response in gluten sensitive individuals and those with celiac disease.[48] Alternatively, oat seeds appear similar to seeds of wheat, barley and rye; cross-contamination between these grains is difficult to resolve.

> *Further information: Oat-sensitive celiac disease*

Origin of controversy

After World War II, wheat was suspected as the cause of celiac disease, and the gluten from wheat was identified as a cause soon after. At the time, duodenal biopsy—the current "gold standard" of diagnosis—had not yet been developed;[49] indirect measures of disease were used. In two studies, three children were fed 75 to 150 grams of oats per day and developed symptoms. In three concurrent studies, 10 children and two adults were allowed to eat 28 to 60 grams of oats and developed no symptoms.[50] Since wheat, barley and sometimes rye are common contaminants in oats,[41][51] until this was investigated, oats were considered to be toxic to celiacs.

> *Further information: History of coeliac disease*

Current findings

While the problem of contamination has been known for several years, scientists' understanding of how oats and gluten are related continues to evolve. A study published in February 2011 uncovered differing levels of toxicity amongst different varieties of oat, indicating that cross-contamination is not the only reason why some oats provoke reactions in some people with a gluten intolerance.[48] A study published in June 2008 found that of 109 sources of oats screened, 85 had unacceptable levels of gluten from wheat, barley or rye.[52] *Triticeae* contaminated oats in the study came from many countries indicating that most sources of oats are unacceptable for GS based on contamination.

> *from wheat (i.e., all Triticum species, such as durum wheat, spelt, and kamut), rye, barley, oats[1] or their crossbred varieties, which have been specially processed to remove gluten, and the gluten level does not exceed 20 mg/kg in total, based on the food as sold or distributed to the consumer.*[43]
>
> [1] The Committee agreed to specify that the allowance of oats that are not contaminated with wheat, rye or barley in foods covered by the standard may be determined at national level."[43]

In realizing the benefit of whole oats in a gluten free diet, the Canadian Celiac Association sought to assure oats and oat products fulfill the gluten-free standards set by the Canadian Food Inspection Agency and Health Canada:

> *in consultation with Health Canada, Agriculture & Agri-Food Canada and the Canadian Food Inspection Agency, has established requirements for growing, processing, and purity testing and labelling of pure oats.*[44]

Tolerable levels of gluten

In summary of recent developments, oats can be tolerated in a gluten-free diet, but oat products should be limited in contamination from *Triticeae* derived gluten to 20 PPM (20 mg per kg). US states are free to deny the GF-label standard for oat products, if warranted (see Politics of Gluten-Free and oats).[53]

Gluten-free testing

As of February 2011, G12, the newest monoclonal antibody (moAb) available, is the only one proven to detect both cross-contamination in oats and also the inherent gluten / avenin that is only found in some varieties of oat.[48] Alternative methods of detection, while currently accepted by many gluten-free certification organizations, are not in fact able to detect this second form of gluten in oats. This may partially explain why some celiacs react to oats and others do not.

A barley-sensitive ELISA called the R5 sandwich assay does not detect gluten in any of 25 pure oat varieties, but it does detect barley, wheat and rye.[52] Disease-sensitive farming practices, antibody testing and species specific genetic testing are capable of producing pure oats.[52] In the United States, 3 domestic GF-brands are available and one brand imported from Ireland 'reckons' to be 99.95% pure oats.[54][55] Two brands in the United States use the R5 antibody test and claim to be below 20 PPM in defined gluten.[54][56] However, the R5 antibody test has not been proven to be as sensitive as the G12 test.

Further information: Antibody testing for gluten-free foods

Diets

Main article: Gluten-free diet

Gluten-free oats in a gluten-free diet. Gluten-free oats can provide a valuable source of fiber, vitamin B, iron, zinc and complex carbohydrates.[57] Recent studies show that gluten-sensitive individuals on a gluten-free diet often get too much simple starch, too little fibre and vitamin B. Currently most guidelines do not include oats in a gluten-free diet. While this is likely to change, oats are not recommended within a year of diagnosis because of the oat-sensitive enteropathy (ASE) risk, the desire to establish a clinical baseline and complexity of the contamination issue.[58]

Consuming oats when anti-gliadin antibodies or gliadin are present increases anti-avenin antibodies, and may promote ASE. Duodenal biopsy may be recommended after oat consumption is initiated. The DQ phenotype of all 3 ASE individuals studied so far indicated DQ2 homozygotes are at risk for ASE. Preferably, newly diagnosed celiacs seek the help of a dietician. However, guidelines are also available for the introduction of pure, uncontaminated oats into the gluten-free diet.[58]

Further information: Oat sensitivity

See also

- List of allergies

References

1. ^ Fasano, A; Data from his clinic in Maryland
2. ^ Sapone A et al. Differential mucosal IL-17 expression in two gliadin-induced disorders: gluten sensitivity and the autoimmune enteropathy celiac disease. International Archives of Allergy and Immunology. 2010;152:75-80
3. ^ Ford RP. The gluten syndrome. A neurological disease. Medical Hypotheses. 2009; 73 (3):438-440
4. ^ [a] [b] Dickerson F et al. Markers of gluten sensitivity and celiac disease in recent onset psychosis and multi-episode schizophrenia. Biological Psychiatry. 2010;68(1):100-104
5. ^ [a] [b] Hadjivassiliou M et al. Myopathy associated with gluten sensitivity. Muscle Nerve. 2007;35:443-450
6. ^ Sapone A et al. Differential mucosal IL-17 expression in two gliadin-induced disorders: gluten sensitivity and the autoimmune enteropathy celiac disease. International Archives of Allergy and Immunology. 2010;152:75-80; Ford RP. The gluten syndrome. A neurological disease. Medical Hypotheses. 2009; 73 (3):438-440
7. ^ Verdu EF, Armstrong D, Murray JA. Between celiac disease and irritable bowel syndrome: The 'No Man's Land' of Gluten Sensitivity. The American Journal of Gastroenterology. 2009;104(6):1587-1594
8. ^ Sapone A et al. (2010). Differential mucosal IL-17 expression in two gliadin-induced disorders: gluten sensitivity and the autoimmune enteropathy celiac disease. International Archives of Allergy & Immunology; 152: 75-80
9. ^ Bizzaro N et al. (2010) Cutting edge issues in celiac disease and in gluten intolerance. Clinical Reviews in Allergy & immunology.
10. ^ Sapone A et al. (2010). Differential mucosal IL-17 expression in two gliadin-induced disorders: gluten sensitivity and the autoimmune enteropathy celiac disease. International Archives of Allergy & Immunology; 152: 75-80.
11. ^ Hadjivassiliou M, Grünewald R, Sharrack B, et al. (2003). "Gluten ataxia in perspective: epidemiology, genetic susceptibility and clinical characteristics". Brain 126 (Pt 3): 685–91. doi:10.1093/brain/awg050. PMID 12566288.
12. ^ A. Vilpulla, P. Collin, M. Maki et al (2008) "Undetected coeliac disease in the elderly. A biopsy-proven population-based study." Digestive and Liver Disease 40 (2008) 809-813
13. ^ Biesiekierski, Jessica R et al (March 2011). "Gluten Causes Gastrointestinal Symptoms in Subjects Without Celiac Disease: A Double-Blind Randomized Placebo-Controlled Trial". *Am J Gastroenterol* **106** (3): 508–514. doi:10.1038/ajg.2010.487 (http://dx.doi.org/10.1038%2Fajg.2010.487) . PMID 21224837 (http://www.ncbi.nlm.nih.gov/pubmed/21224837) .
14. ^ Bandani AR (2005). "Effect of plant a-amylase inhibitors on sunn pest, Eurygaster integriceps Puton (Hemiptera: Scutelleridae), alpha-amylase activity". *Commun. Agric. Appl. Biol. Sci.* **70** (4): 869–73. PMID 16628930 (http://www.ncbi.nlm.nih.gov/pubmed/16628930) .
15. ^ Stepánková R, Kofronová O, Tucková L, Kozáková H, Cebra JJ, Tlaskalová- Hogenová H (January 2003). "Experimentally induced gluten enteropathy and protective effect of epidermal growth factor in artificially fed neonatal rats" (http://meta.wkhealth.com/pt/pt-core/template-journal/lwwgateway/media/landingpage.htm?issn=0277-2116&volume=36&issue=1&spage=96) . *J. Pediatr. Gastroenterol. Nutr.* **36** (1): 96–104. doi:10.1097/00005176-200301000-00018 (http://dx.doi.org/10.1097%2F00005176-200301000-00018) . PMID 12500003 (http://www.ncbi.nlm.nih.gov/pubmed/12500003) . http://meta.wkhealth.com/pt/pt-core/template-journal/lwwgateway/media/landingpage.htm?issn=0277-2116&volume=36&issue=1&spage=96.
16. ^ Bernardo D, Garrote JA, Fernández-Salazar L, Riestra S, Arranz E (2007). "Is gliadin really safe for non-coeliac individuals? Production of interleukin 15 in biopsy culture from non-coeliac individuals challenged with gliadin peptides" (http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=1954879) . *Gut* **56** (6): 889–90. doi:10.1136/gut.2006.118265 (http://dx.doi.org/10.1136%2Fgut.2006.118265) . PMC 1954879 (http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=1954879) . PMID 17519496 (http://www.ncbi.nlm.nih.gov/pubmed/17519496) . http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=1954879.
17. ^ Bodinier M, Legoux MA, Pineau F, *et al.* (May 2007). "Intestinal translocation capabilities of wheat allergens using the Caco-2 cell line". *J. Agric. Food Chem.* **55** (11): 4576–83. doi:10.1021/jf070187e (http://dx.doi.org/10.1021%2Fjf070187e) . PMID 17477542 (http://www.ncbi.nlm.nih.gov/pubmed/17477542) .

18. ^ Thomas KE, Sapone A, Fasano A, Vogel SN (February 2006). "Gliadin stimulation of murine macrophage inflammatory gene expression and intestinal permeability are MyD88-dependent: role of the innate immune response in Celiac disease" (http://www.jimmunol.org/cgi/pmidlookup?view=long&pmid=16456012) . *J. Immunol.* **176** (4): 2512–21. PMID 16456012 (http://www.ncbi.nlm.nih.gov/pubmed/16456012) . http://www.jimmunol.org/cgi/pmidlookup?view=long&pmid=16456012.
19. ^ Lammers KM, Lu R, Brownley J, *et al.* (March 2008). "Gliadin Induces an Increase in Intestinal Permeability and Zonulin Release by Binding to the Chemokine Receptor CXCR3" (http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=2653457) . *Gastroenterology* **135** (1): 194–204.e3. doi:10.1053/j.gastro.2008.03.023 (http://dx.doi.org/10.1053%2Fj.gastro.2008.03.023) . PMC 2653457 (http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=2653457) . PMID 18485912 (http://www.ncbi.nlm.nih.gov/pubmed/18485912) . http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=2653457.
20. ^ van Heel DA, West J (July 2006). "Recent advances in coeliac disease" (http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=1856316) . *Gut* **55** (7): 1037–46. doi:10.1136/gut.2005.075119 (http://dx.doi.org/10.1136%2Fgut.2005.075119) . PMC 1856316 (http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=1856316) . PMID 16766754 (http://www.ncbi.nlm.nih.gov/pubmed/16766754) . http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=1856316.
21. ^ http://www.livescience.com/health/celiac-disease-gluten-peptides-100721.html
22. ^ Tye-Din JA, Stewart JA, Dromey JA, Beissbarth T, van Heel DA, Tatham A, Henderson K, Mannering SI, Gianfrani C, Jewell DP, Hill AV, McCluskey J, Rossjohn J, Anderson RP (2010). "Comprehensive, quantitative mapping of T cell epitopes in gluten in celiac disease". *Sci Transl Med* **2** (41): 41ra51. doi:10.1126/scitranslmed.3001012 (http://dx.doi.org/10.1126%2Fscitranslmed.3001012) . PMID 20650871 (http://www.ncbi.nlm.nih.gov/pubmed/20650871) .
23. ^ Bittner C, Grassau B, Frenzel K, Baur X (March 2008). "Identification of wheat gliadins as an allergen family related to baker's asthma". *J. Allergy Clin. Immunol.* **121** (3): 744–9. doi:10.1016/j.jaci.2007.09.051 (http://dx.doi.org/10.1016%2Fj.jaci.2007.09.051) . PMID 18036646 (http://www.ncbi.nlm.nih.gov/pubmed/18036646) .
24. ^ Matsuo H, Dahlström J, Tanaka A, *et al.* (February 2008). "Sensitivity and specificity of recombinant omega-5 gliadin-specific IgE measurement for the diagnosis of wheat-dependent exercise-induced anaphylaxis". *Allergy* **63** (2): 233–6. doi:10.1111/j.1398-9995.2007.01504.x (http://dx.doi.org/10.1111%2Fj.1398-9995.2007.01504.x) . PMID 18186814 (http://www.ncbi.nlm.nih.gov/pubmed/18186814) .
25. ^ Akagawa M, Handoyo T, Ishii T, Kumazawa S, Morita N, Suyama K (August 2007). "Proteomic analysis of wheat flour allergens". *J. Agric. Food Chem.* **55** (17): 6863–70. doi:10.1021/jf070843a (http://dx.doi.org/10.1021%2Fjf070843a) . PMID 17655322 (http://www.ncbi.nlm.nih.gov/pubmed/17655322) .
26. ^ Hopper AD, Cross SS, Hurlstone DP, *et al.* (April 2007). "Pre-endoscopy serological testing for celiac disease: evaluation of a clinical decision tool" (http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=1847864) . *BMJ* **334** (7596): 729. doi:10.1136/bmj.39133.668681.BE (http://dx.doi.org/10.1136%2Fbmj.39133.668681.BE) . PMC 1847864 (http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=1847864) . PMID 17383983 (http://www.ncbi.nlm.nih.gov/pubmed/17383983) . http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=1847864.
27. ^ Hopper AD, Cross SS, Sanders DS (March 2008). "Patchy villous atrophy in adult patients with suspected gluten-sensitive enteropathy: is a multiple duodenal biopsy strategy appropriate?". *Endoscopy* **40** (3): 219–24. doi:10.1055/s-2007-995361 (http://dx.doi.org/10.1055%2Fs-2007-995361) . PMID 18058655 (http://www.ncbi.nlm.nih.gov/pubmed/18058655) .
28. ^ Kaukinen K, Peräaho M, Collin P, *et al.* (May 2005). "Small-bowel mucosal transglutaminase 2-specific IgA deposits in celiac disease without villous atrophy: a prospective and randomized clinical study". *Scand. J. Gastroenterol.* **40** (5): 564–72. doi:10.1080/00365520510023422 (http://dx.doi.org/10.1080%2F00365520510023422) . PMID 16036509 (http://www.ncbi.nlm.nih.gov/pubmed/16036509) .
29. ^ Kaukinen K, Turjanmaa K, Mäki M, *et al.* (September 2000). "Intolerance to cereals is not specific for coeliac disease". *Scand. J. Gastroenterol.* **35** (9): 942–6. doi:10.1080/003655200750022995 (http://dx.doi.org/10.1080%2F003655200750022995) . PMID 11063153 (http://www.ncbi.nlm.nih.gov/pubmed/11063153) .
30. ^ Hadjivassiliou M, Sanders DS, Woodroofe N et al. (2008). Gluten ataxia. The Cerebellum; 494-498

31. ^ Anderson LA, McMillan SA, Watson RG, *et al.* (2007). "Malignancy and mortality in a population-based cohort of patients with coeliac disease or "gluten sensitivity"". *World J. Gastroenterol.* **13** (1): 146–51. PMID 17206762 (http://www.ncbi.nlm.nih.gov/pubmed/17206762) .
32. ^ O'Farrelly C, O'Mahony C, Graeme-Cook F, Feighery C, McCartan BE, Weir DG (1991). "Gliadin antibodies identify gluten-sensitive oral ulceration in the absence of villous atrophy". *J. Oral Pathol. Med.* **20** (10): 476–8. doi:10.1111/j.1600-0714.1991.tb00407.x (http://dx.doi.org/10.1111%2Fj.1600-0714.1991.tb00407.x) . PMID 1753350 (http://www.ncbi.nlm.nih.gov/pubmed/1753350) .
33. ^ Ringvold A, Overgaard RG (1995). "Increased IgA antibodies to gluten and gliadin in serum of persons with ocular pseudo-exfoliation". *Acta ophthalmologica Scandinavica* **73** (2): 171–2. doi:10.1111/j.1600-0420.1995.tb00662.x (http://dx.doi.org/10.1111%2Fj.1600-0420.1995.tb00662.x) . PMID 7656149 (http://www.ncbi.nlm.nih.gov/pubmed/7656149) .
34. ^ Lidén M, Kristjánsson G, Valtýsdóttir S, Hällgren R (2007). "Gluten sensitivity in patients with primary Sjögren's syndrome". *Scand. J. Gastroenterol.* **42** (8): 962–7. doi:10.1080/00365520701195345 (http://dx.doi.org/10.1080%2F00365520701195345) . PMID 17613926 (http://www.ncbi.nlm.nih.gov/pubmed/17613926) .
35. ^ Workman EM, Alun Jones V, Wilson AJ, Hunter JO (1984). "Diet in the management of Crohn's disease". *Human nutrition. Applied nutrition* **38** (6): 469–73. PMID 6526690 (http://www.ncbi.nlm.nih.gov/pubmed/6526690) .
36. ^ Frieri M, Claus M, Boris M, Zitt M, Scalise D, Harris N (1990). "Preliminary investigation on humoral and cellular immune responses to selected food proteins in patients with Crohn's disease". *Annals of allergy* **64** (4): 345–51. PMID 2321808 (http://www.ncbi.nlm.nih.gov/pubmed/2321808) .
37. ^ Huber A, Genser D, Spitzauer S, Scheiner O, Jensen-Jarolim E (1998). "IgE/anti-IgE immune complexes in sera from patients with Crohn's disease do not contain food-specific IgE". *Int. Arch. Allergy Immunol.* **115** (1): 67–72. doi:10.1159/000023832 (http://dx.doi.org/10.1159%2F000023832) . PMID 9430498 (http://www.ncbi.nlm.nih.gov/pubmed/9430498) .
38. ^ Pfeil T, Schwabl U, Ulmer WT, König W (1990). "Western blot analysis of water-soluble wheat flour (Triticum vulgaris) allergens". *Int. Arch. Allergy Appl. Immunol.* **91** (3): 224–31. doi:10.1159/000235121 (http://dx.doi.org/10.1159%2F000235121) . PMID 1693911 (http://www.ncbi.nlm.nih.gov/pubmed/1693911) .
39. ^ p.47, JOINT FAO/WHO FOOD STANDARDS PROGRAMME CODEX ALIMENTARIUS COMMISSION 15th Session Rome, 4–15 July 1983 REPORT OF THE 13TH SESSION OF THE CODEX COMMITTEE ON FOODS FOR SPECIAL DIETARY USES (http://www.codexalimentarius.net/download/report/241/al83_26e.pdf) Bonn-Bad Godesberg, 20–24 September 1982
40. ^ "*Codex standard for "GLUTEN-FREE FOODS", CODEX STAN 118-19811 (http://www.codexalimentarius.net/download/standards/291/CXS_118e.pdf)* . Codex alimentaris*, Food and Agricultural Organization of the United Nations*
41. ^ [a] [b] American Dietetic Association. Nutrition Care Manual: Celiac Disease. Available at: http://www.nutritioncaremanual.org. Accessed December 15, 2004.
42. ^ "CODEX ALIMENTARIUS COMMISSION - Thirty first Session - Geneva, Switzerland, 30 June - 5 July 2008". From Title page. Committee on Nutrition and Foods for Special Dietary Uses. JOINT FAO/WHO FOOD STANDARDS PROGRAMME CODEX ALIMENTARIUS COMMISSION. Thirty-first Session Geneva, Switzerland, 30 June – 4 July 2008, Codex Alimentarius Commission REPORT OF THE 29th SESSION OF THE CODEX COMMITTEE ON NUTRITION AND FOODS FOR SPECIAL DIETARY USES (http://www.codexalimentarius.net/download/report/687/al08_26e.pdf)
43. ^ [a] [b] *"Draft Revised Standard for Foods for Special Dietary Use for Persons intolerant to Gluten (at Step 8)".* page 50-51. Committee on Nutrition and Foods for Special Dietary Uses. JOINT FAO/WHO FOOD STANDARDS PROGRAMME CODEX ALIMENTARIUS COMMISSION. Thirty-first Session Geneva, Switzerland, 30 June – 4 July 2008, Codex Alimentarius Commission REPORT OF THE 29th SESSION OF THE CODEX COMMITTEE ON NUTRITION AND FOODS FOR SPECIAL DIETARY USES (http://www.codexalimentarius.net/download/report/687/al08_26e.pdf)
44. ^ Rashid M, Butzner D, Burrows V, *et al.* (October 2007). "Consumption of pure oats by individuals with celiac disease: A position statement by the Canadian Celiac Association" (http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=2658132) . *Can. J. Gastroenterol.* **21** (10): 649–51. PMC 2658132 (http://www.pubmedcentral.gov/articlerender.fcgi?

tool=pmcentrez&artid=2658132) . PMID 17948135 (http://www.ncbi.nlm.nih.gov/pubmed/17948135) . http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=2658132.

45. ^ Baldo BA, Krilis S, Wrigley CW (1980). "Hypersensitivity to inhaled flour allergens. Comparison between cereals". *Allergy* **35** (1): 45–56. doi:10.1111/j.1398-9995.1980.tb01716.x (http://dx.doi.org/10.1111%2Fj.1398-9995.1980.tb01716.x) . PMID 6154431 (http://www.ncbi.nlm.nih.gov/pubmed/6154431) .
46. ^ Karatay S, Erdem T, Kiziltunc A, *et al.* (2006). "General or personal diet: the individualized model for diet challenges in patients with rheumatoid arthritis". *Rheumatol. Int.* **26** (6): 556–60. doi:10.1007/s00296-005-0018-y (http://dx.doi.org/10.1007%2Fs00296-005-0018-y) . PMID 16025333 (http://www.ncbi.nlm.nih.gov/pubmed/16025333) .
47. ^ Kupper C (2005). "Dietary guidelines and implementation for celiac disease". *Gastroenterology* **128** (4 Suppl 1): S121–7. doi:10.1053/j.gastro.2005.02.024 (http://dx.doi.org/10.1053%2Fj.gastro.2005.02.024) . PMID 15825119 (http://www.ncbi.nlm.nih.gov/pubmed/15825119) .
48. ^ [a] [b] [c] Comino, Isabel; Ana Real, Laura de Lorenzo, Hugh Cornell, Miguel Ángel López-Casado, Francisco Barro, Pedro Lorite, Ma Isabel Torres, Ángel Cebolla, Carolina Sousa (12). "Diversity in oat potential immunogenicity: basis for the selection of oat varieties with no toxicity in coeliac disease" (http://gut.bmj.com/content/early/2011/02/11/gut.2010.225268.abstract) . *Gut* **60** (First Online): 915–22. doi:10.1136/gut.2010.225268 (http://dx.doi.org/10.1136%2Fgut.2010.225268) . PMC 3112367 (http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=3112367) . PMID 21317420 (http://www.ncbi.nlm.nih.gov/pubmed/21317420) . http://gut.bmj.com/content/early/2011/02/11/gut.2010.225268.abstract. Retrieved 12 March 2011.
49. ^ SMITH RB, SPRINZ H, CROSBY WH, SULLIVAN BH (September 1958). "Peroral small bowel mucosal biopsy" (http://linkinghub.elsevier.com/retrieve/pii/0002-9343(58)90077-9) . *Am. J. Med.* **25** (3): 391–4. doi:10.1016/0002-9343(58)90077-9 (http://dx.doi.org/10.1016%2F0002-9343%2858%2990077-9) . PMID 13571252 (http://www.ncbi.nlm.nih.gov/pubmed/13571252) . http://linkinghub.elsevier.com/retrieve/pii/0002-9343(58)90077-9.
50. ^ Garsed K, Scott BB (February 2007). "Can oats be taken in a gluten-free diet? A systematic review". *Scand. J. Gastroenterol.* **42** (2): 171–8. doi:10.1080/00365520600863944 (http://dx.doi.org/10.1080%2F00365520600863944) . PMID 17327936 (http://www.ncbi.nlm.nih.gov/pubmed/17327936) .
51. ^ Thompson T (November 2004). "Gluten contamination of commercial oat products in the United States". *N. Engl. J. Med.* **351** (19): 2021–2. doi:10.1056/NEJM200411043511924 (http://dx.doi.org/10.1056%2FNEJM200411043511924) . PMID 15525734 (http://www.ncbi.nlm.nih.gov/pubmed/15525734) .
52. ^ [a] [b] [c] Hernando A, Mujico JR, Mena MC, Lombardía M, Méndez E (June 2008). "Measurement of wheat gluten and barley hordeins in contaminated oats from Europe, the United States and Canada by Sandwich R5 ELISA". *Eur J Gastroenterol Hepatol* **20** (6): 545–54. doi:10.1097/MEG.0b013e3282f46597 (http://dx.doi.org/10.1097%2FMEG.0b013e3282f46597) . PMID 18467914 (http://www.ncbi.nlm.nih.gov/pubmed/18467914) .
53. ^ Catassi C et al (2007) "A prospective, double-blind, placebo-controlled trial to establish a safe gluten threshold for patients with coeliac disease." American Journal of Clinical Nutrition, Vol 85, No 1, 160-166
54. ^ [a] [b] *Cleaning/Inspection Process* Gluten Free Oats (http://www.glutenfreeoats.com/cleaning.aspx)
55. ^ "*Are McCann's Oat products gluten free?*". URL:FAQ - Frequently asked questions (http://www.mccanns.ie/faq.html)
56. ^ *GLUTEN FREE ROLLED OATS* Bob's Red Mill Natural Foods (http://www.bobsredmill.com/catalog/index.php?action=showdetails&product_ID=680)
57. ^ Størsrud S, Hulthén LR, Lenner RA (July 2003). "Beneficial effects of oats in the gluten-free diet of adults with special reference to nutrient status, symptoms and subjective experiences". *Br. J. Nutr.* **90** (1): 101–7. doi:10.1079/BJN2003872 (http://dx.doi.org/10.1079%2FBJN2003872) . PMID 12844381 (http://www.ncbi.nlm.nih.gov/pubmed/12844381) .
58. ^ [a] [b] Rashid, Mohsin (2007-06-08). "Guidelines for Consumption of Pure and Uncontaminated Oats by Individuals with Celiac Disease" (http://www.celiac.ca/Articles/PABoatsguidelines2007June.html) . Professional Advisory Board of Canadian Celiac Association. http://www.celiac.ca/Articles/PABoatsguidelines2007June.html. Retrieved 2008-08-14.

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