http://www.ncbi.nlm.nih.gov/pubmed/15497770

Nutr Rev. 2004 Sep;62(9):360-3.

Comment in:
Nutr Rev. 2004 Dec;62(12):490; author reply 491.

A milligram of gluten a day keeps the mucosal recovery away: a case report.

Biagi F, **Campanella J**, **Martucci S**, **Pezzimenti D**, **Ciclitira PJ**, **Ellis HJ**, **Corazza GR**.

IRCCS Policlinico S Matteo, University of Pavia, Italy.

In recent years it has been suggested that patients with celiac disease can be adequately followed up on the basis of merely clinical and serological response to a gluten-free diet. Thus, a duodenal biopsy some months after commencement of a gluten-free diet would no longer be necessary. We report here the case of a celiac patient in whom the ingestion of a milligram of gluten every day for 2 years prevented histological recovery in spite of satisfactory clinical and serological response. The literature regarding the minimal amount of gluten that could be harmless to celiac patients is reviewed.

http://www.ncbi.nlm.nih.gov/pubmed/15730422

Clin Endocrinol (Oxf). 2005 Mar;62(3):372-5.

Growth hormone deficiency and coeliac disease: an unusual association?

Bozzola M, **Giovenale D**, **Bozzola E**, **Meazza C**, **Martinetti M**, **Tinelli C**, **Corazza GR**.

Pediatric Department, University of Pavia, Pavia, Italy. m.bozzola@smatteo.pv.iy

OBJECTIVE: To assess the occurrence of growth hormone deficiency (GHD) in patients with coeliac disease (CD).

STUDY DESIGN: A total of 1066 children diagnosed elsewhere with short stature were referred to our centre for second-line evaluation in a 6-year period. All patients were screened for CD by antiendomysial antibodies (EMA) and those with positive sera underwent intestinal biopsy.

RESULTS: Among the 1066 short children, 210 (19.7%) had GHD and 12 (1.12%; chronological age from 3.6 to 12.3 years, bone age from 1.5 to 10.5 years, SDS height from -3.05 to -0.48), having positive EMA, showed histologically confirmed CD. Nine of these latter 12 CD children had a beneficial effect on growth rate after the first year of gluten-free diet, while the remaining three showed no catch-up growth. A careful endocrinological investigation in these three CD boys showed an isolated GHD in two cases and a multiple GHD in one case. The congenital origin of GHD is supported by the congenital abnormalities documented by magnetic resonance imaging. GH therapy associated with gluten-free diet led to an increased growth rate.

CONCLUSION: GH secretion should be evaluated in coeliac patients showing no catch-up growth after a period on a gluten-free diet in spite of reversion to seronegativity for EMA. In the case of GHD and CD, replacement GH therapy should be started during a gluten-free diet.

http://www.ncbi.nlm.nih.gov/pubmed/16126954

Ann N Y Acad Sci. 2005 Jun;1051:148-55.

Neurological complications of celiac disease and autoimmune mechanisms: preliminary data of a prospective study in adult patients.

Briani C, **Zara G**, **Toffanin E**, **Ruggero S**, **Ferrarini A**, **De Lazzari F**, **Luca M**, **Faggian D**, **Grassivaro F**, **Ermani M**, **Pezzani R**, **Giometto B**, **D'Odorico A**.

Department of Neurosciences, University of Padua, Via Giustiniani 5, 35128 Padua, Italy.
chiara.briani@unipd.it

Antibodies to gangliosides and Purkinje cells have been reported in patients with celiac disease (CD) with neuropathy and ataxia, respectively. Whether these antibodies are pathogenic is not clear. The response of neurological symptoms and antibody titers to a gluten-free diet is still controversial. The objective of our study was to assess whether neurological manifestations in CD patients correlate with antibody titers and a gluten-free diet.Thirty-five CD patients (9 males, 26 females, mean age 37.1 +/- 12.6 yrs) were followed prospectively. At initial evaluation, 23 were on a gluten-free diet, 12 were not. At recruitment and during follow-up, patients underwent neurological and electrophysiological evaluation. IgG, IgM, and IgA anti-ganglioside antibodies were assayed by ELISA; anti-neuronal antibodies were assessed by immunohistochemistry and Western blot. Four patients, all males, had electrophysiological evidence of neuropathy; three had been on a gluten-free diet for several months, and one was newly diagnosed. One had reduced tendon reflexes; another complained of distal paresthesias. With regard to anti-ganglioside antibodies, three patients had a moderate increase in antibodies without symptoms or signs of neuropathy. No patients had ataxia or cerebellar dysfunction, although in four patients reactivity to neuronal antigens was found. In 17 patients, an electrophysiological follow-up (mean duration of follow-up, 9 months) showed no changes. In conclusion, the preliminary results of this prospective study indicate that neuropathy, usually subclinical, may accompany CD. Antibody titers do not seem to correlate with neurological symptoms/signs or diet. Ongoing follow-up will help confirm these data and clarify the role, if any, of antibodies in neurological involvement in CD.

http://www.ncbi.nlm.nih.gov/pubmed/18343508

J Neuroimmunol. 2008 Mar;195(1-2):171-5. Epub 2008 Mar 17.

Neurological complications of celiac disease and autoimmune mechanisms: a prospective study.

Briani C, **Zara G**, **Alaedini A**, **Grassivaro F**, **Ruggero S**, **Toffanin E**, **Albergoni MP**, **Luca M**, **Giometto B**, **Ermani M**, **De Lazzari F**, **D'Odorico A**, **Battistin L**.

Department of Neurosciences, University of Padova, Padova, Italy. chiara.briani@unipd.it

Humoral immune mechanisms may have a role in the neurological complications of celiac disease (CD). We assessed 71 CD patients for neurologic manifestations and presence of serum antibodies to neural antigens. Sixteen patients (22.5%) were found to have neurological deficits including headache, depression, entrapment syndromes, peripheral neuropathy, and epilepsy. Antibody reactivity to neural antigens was detected in 30/71 (42.2%) patients. There was no clear correlation between anti-neural reactivity and neurologic dysfunction. Follow-up of 62 patients did not reveal change in electrophysiology or antibodies, regardless of diet. However, in 2 patients with neuropathy, symptoms improved or worsened depending on the diet.

http://www.ncbi.nlm.nih.gov/pubmed/17122729

Neurologist. 2006 Nov;12(6):318-21.

Epilepsy and celiac disease: favorable outcome with a gluten-free diet in a patient refractory to antiepileptic drugs.

Canales P, **Mery VP**, **Larrondo FJ**, **Bravo FL**, **Godoy J**.

Department of Neurology, Hospital Regional de Talca, Talca, Chile. kikimery@gmail.com

BACKGROUND: There is a well-documented relationship between epilepsy and celiac disease, including a syndrome characterized by epilepsy, occipital calcifications, and celiac disease.

REVIEW SUMMARY: We report the case of a 23-year-old woman with an 11-year history of refractory epileptic seizures and newly diagnosed biopsy-proven celiac disease with increased antiendomysium immunoglobulin A antibodies. The patient showed a dramatic improvement after starting a gluten-free diet.

CONCLUSION: This case emphasizes the need to include celiac disease in the differential diagnosis when investigating the etiology of epilepsy in refractory patients.

http://www.ncbi.nlm.nih.gov/pubmed/11984490

Rev Neurol (Paris). 2002 Apr;158(4):467-9.

Comment in:
Rev Neurol (Paris). 2004 Jan;160(1):96; author reply 96.

[Chronic meningitis before diagnosis of celiac disease]

[Article in French]

Cartalat-Carel S, **Pradat PF**, **Carpentier A**, **Delattre JY**.

Fédération de Neurologie Mazarin, Groupe Hospitalier Pitié-Salpétrière, Paris, France.

Neurological complications, mainly cerebellar syndrome, have been associated with coeliac disease. We report the first observation of a 56-year-old woman with a chronic meningitis revealing coeliac disease. Neuralgia of the trijeminal nerve and cerebellar syndrome only appeared seven years after the onset of meningitis. MRI examination showed a cerebellar atrophy and a leucoencephalopathy. This case report emphasizes the utility of detection of anti-gliadin antibodies for the diagnosis of a chronic meningitis.

http://www.ncbi.nlm.nih.gov/pubmed/14716525

Neurol Sci. 2003 Dec;24(5):311-7.

Clinical and neurological abnormalities in adult celiac disease.

Cicarelli G, **Della Rocca G**, **Amboni M**, **Ciacci C**, **Mazzacca G**, **Filla A**, **Barone P**.

Department of Neurological Sciences, Federico II University of Naples, Via S. Pansini 5, I-80131 Naples, Italy.

We assessed the occurrence of neurological signs and symptoms in adult patients with celiac disease and evaluated the correlation between neurological features and diet. A total of 176 patients and 52 age-matched controls underwent a semistructural interview and a neurologic examination. The effect of gluten-free diet was evaluated by comparing the prevalence of signs and symptoms among patients adhering to a gluten-free diet and patients on an unrestricted diet. The occurrence of headache, dysthymia and signs of peripheral neuropathy was significantly higher in patients with celiac disease than in control subjects. Adherence to a strict gluten-free diet was associated with a significant reduction of headache, dysthymia, cramps and weakness, but did not modify the occurrence of paresthesia or hyporeflexia. Neurological signs and symptoms are associated with celiac disease and can be ameliorated by a gluten-free diet.

http://www.ncbi.nlm.nih.gov/pubmed/8689146

Gastroenterologist. 1996 Mar;4(1):54-64.

Primary gastrointestinal lymphomas.

Cooper DL, **Doria R**, **Salloum E**.

Department of Internal Medicine, Yale University School of Medicine, New Haven, CT 06856, USA.

Recent evidence suggests that a significant proportion of primary gastrointestinal lymphomas are driven by exogenous agents/antigens. In the stomach, Helicobacter pylori appears to be responsible for most cases of low-grade lymphomas (MALToma), whereas an infectious etiology is suspected in immunoproliferative small intestine disease (IPSID). Similarly, enteropathy-associated T-cell lymphomas appear to result from a disordered response to gluten, although this profile remains controversial. Accordingly, although traditional antineoplastic treatments, such as surgery and radiation, are still important for the treatment of primary GI lymphomas, antibiotics may be the first line of therapy for low-grade gastric MALToma, and they are often used alone or in combination with chemotherapy for IPSID. In patients with celiac sprue, a gluten-free diet appears to markedly reduce the risk for lymphoma. An important caveat for the treatment of gastric lymphomas is that only low-grade gastric MALTomas have consistently responded to antibiotics. Treatment of high-grade gastric lymphoma is evolving. Although surgery was once considered central to diagnosis, staging, and treatment of gastric lymphoma, most patients can now have a diagnosis established by endoscopic biopsy and are candidates for chemotherapy and adjuvant radiation. The risks of fatal hemorrhage and perforation have probably been vastly overestimated and appear to be equal or less than the mortality associated with surgery. In addition, the long-term effects of gastric resection on quality of life have been almost completely ignored. Systemic lymphomas involve the GI tract far more often than is clinically apparent. In most cases, treatment should not be affected.

http://www.ncbi.nlm.nih.gov/pubmed/17353316

AJNR Am J Neuroradiol. 2007 Mar;28(3):479-85.

Whole-brain histogram and voxel-based analyses of apparent diffusion coefficient and magnetization transfer ratio in celiac disease, epilepsy, and cerebral calcifications syndrome.

Della Nave R, **Magaudda A**, **Michelucci R**, **Capizzi G**, **Calabrò A**, **Guerrini L**, **Gavazzi C**, **Diciotti S**, **Riguzzi P**, **Daniele O**, **Villari N**, **Tassinari CA**, **Mascalchi M**.

Section of Radiodiagnostics, University of Florence, Florence, Italy.

BACKGROUND AND PURPOSE: Diffusion and magnetization transfer (MT) techniques have been applied to the investigation with MR of epilepsy and have revealed changes in patients with or without abnormalities on MR imaging. We hypothesized that also in the coeliac disease (CD), epilepsy and cerebral calcifications (CEC) syndrome diffusion and MT techniques could reveal brain abnormalities undetected by MR imaging and tentatively correlated to epilepsy.

MATERIALS AND METHODS: Diffusion and MT weighted images were obtained in 10 patients with CEC, 8 patients with CD without epilepsy and 17 healthy volunteers. The whole brain apparent diffusion coefficient (ADC) and MT ratio (MTR) maps were analyzed with histograms and the Statistical Parametric Mapping 2 (SPM2) software. We employed the non-parametric Mann-Whitney U test to assess differences for ADC and MTR histogram metrics. Voxel by voxel comparison of the ADC and MTR maps was performed with 2 tails t-test corrected for multiple comparison.

RESULTS: A significantly higher whole brain ADC value as compared to healthy controls was observed in CEC ($P = 0.006$) and CD ($P = 0.01$) patients. SPM2 showed bilateral areas of significantly decreased MTR in the parietal and temporal subcortical white matter (WM) in the CEC patients.

CONCLUSION: Our study indicates that diffusion and MT techniques are also capable of revealing abnormalities undetected by MR imaging. In particular patients with CEC syndrome show an increase of the whole brain ADC histogram which is more pronounced than in patients with gluten intolerance. IN CEC patients, voxel-based analysis demonstrates a localized decrease of the MTR in the parieto-temporal subcortical WM.

Full free study available

http://www.ncbi.nlm.nih.gov/pubmed/18379737

Neurol Sci. 2008 Feb;29(1):29-31. Epub 2008 Apr 1.

A case of multiple sclerosis with atypical onset associated with autoimmune hepatitis and silent coeliac disease.

Ferrò MT, **Franciotta D**, **Riccardi T**, **D'Adda E**, **Mainardi E**, **Montanelli A**.

Neurological Department Ospedale Maggiore, Multiple Sclerosis Center, L.go Dossena 2, 26013 Crema, Italy. maraferro@libero.it

Multiple sclerosis (MS) and coeliac disease (CD) are an uncommon association. Recently "MS-like illness and CNS white-matter abnormalities" have been demonstrated in patients with CD. We report the case of a 19-year-old female with MS, who presented an episode of headache at onset of disease and developed acute hepatitis (AH) 14 months later. After the diagnosis of AH, an occult CD, confirmed by jejunal biopsy, was disclosed. Constipation was the only gastrointestinal symptom. A serum sample collected before onset of MS was positive for CD. Anti-central nervous system antibodies were negative in both retrospective and current serum samples.

Conclusions The concomitant presence of MS with atypical onset, AH and CD likely represents an unusual chance association in our patient but inflammatory immune-mediated damage of the central nervous system triggered by gluten could not be excluded.

http://www.ncbi.nlm.nih.gov/pubmed/8338215

Brain Dev. 1993 Jan-Feb;15(1):79-82.

Progressive cerebral calcifications, epilepsy, and celiac disease.

Fois A, **Balestri P**, **Vascotto M**, **Farnetani MA**, **Di Bartolo RM**, **Di Marco V**, **Vindigni C**.

Department of Pediatrics, University of Siena, Italy.

A case with progressive cerebral calcifications, white matter involvement, and drug-resistant epilepsy in a 9-year-old boy is described. The final diagnosis was celiac disease (CD). The relationship of CD with epileptogenic lesions is considered, and the possible significance of this association is discussed.

http://www.ncbi.nlm.nih.gov/pubmed/18056506

J Bone Joint Surg Am. 2007 Dec;89(12):2732-6.

Crowned Dens syndrome.

Goto S, **Umehara J**, **Aizawa T**, **Kokubun S**.

Department of Orthopaedic Surgery, Senboku Kumiai General Hospital, 1-30 Omagari-torimati, Daisen, Akita 014-0027, Japan. cfq40980@par.odn.ne.jp

BACKGROUND: Patients with crowned dens syndrome typically present with severe neck pain and have calcium deposits around the odontoid process of the axis on radiographs. To our knowledge, the cases of only thirty-five patients have been reported in the English-language literature and the clinical features remain unclear. The purposes of this study were to examine the clinical features of crowned dens syndrome, determine treatment outcomes, and propose diagnostic criteria.

METHODS: Forty patients with severe neck pain had calcium deposition around the odontoid process on computed tomography scans, and they were thus diagnosed as having crowned dens syndrome. Data were collected in relation to these patients, including the date of onset of neck pain, the presence of inflammatory indicators (increased body temperature, C-reactive protein levels, and white blood-cell count), and treatment outcomes.

RESULTS: The male-to-female ratio was 0.6, and two-thirds of the patients were more than seventy years of age. All patients had markedly restricted neck motion, particularly in rotation, and all had one or more positive inflammatory indicators. Calcium deposition was detected in all areas around the odontoid process, but chiefly behind the process. Pain was typically relieved by nonsteroidal anti-inflammatory drugs, prednisolone, or both. A combination of both appeared to be the most effective.

CONCLUSIONS: We believe that crowned dens syndrome is more common than previously recognized, especially in elderly patients. It is diagnosed on the basis of acute and severe neck pain; marked restriction of neck motion, particularly in rotation; the presence of inflammatory indicators, such as an elevated C-reactive protein level; calcium deposition around the odontoid process detected by computed tomography; no history of trauma; and the exclusion of other inflammatory diseases and tumors. Prednisolone and nonsteroidal anti-inflammatory drugs in combination are the recommended treatment for symptom relief.

http://www.ncbi.nlm.nih.gov/pubmed/11794474

Neurol Sci. 2001 Nov;22 Suppl 2:S117-22.

Neurological manifestations of gastrointestinal disorders, with particular reference to the differential diagnosis of multiple sclerosis.

Ghezzi A, **Zaffaroni M**.

Centro Studi Sclerosi Multipla, Ospedale di Gallarate, Università di Milano, Gallarate, Varese, Italy.

Neurological manifestations of gastrointestinal disorders are described, with particular reference to those resembling multiple sclerosis (MS) on clinical or MRI grounds. Patients with celiac disease can present cerebellar ataxia, progressive myoclonic ataxia, myelopathy, or cerebral, brainstem and peripheral nerve involvement. Antigliadin antibodies can be found in subjects with neurological dysfunction of unknown cause, particularly in sporadic cerebellar ataxia ("gluten ataxia"). Patients with Whipple's disease can develop mental and psychiatric changes, supranuclear gaze palsy, upper motoneuron signs, hypothalamic dysfunction, cranial nerve abnormalities, seizures, ataxia, myorhythmia and sensory deficits. Neurological manifestations can complicate inflammatory bowel disease (e.g. ulcerative colitis and Crohn's disease) due to vascular or vasculitic mechanisms. Cases with both Crohn's disease and MS or cerebral vasculitis are described. Epilepsy, chronic inflammatory polyneuropathy, muscle involvement and myasthenia gravis are also reported. The central nervous system can be affected in patients with hepatitis C virus (HCV) infection because of vasculitis associated with HCV-related cryoglobulinemia. Mitochondrial neurogastrointestinal encephalopathy (MNGIE) is a disease caused by multiple deletions of mitochondrial DNA. It is characterized by peripheral neuropathy, ophthalmoplegia, deafness, leukoencephalopathy, and gastrointestinal symptoms due to visceral neuropathy. Neurological manifestations can be the consequence of vitamin B1, nicotinamide, vitamin B12, vitamin D, or vitamin E deficiency and from nutritional deficiency states following gastric surgery.

http://www.ncbi.nlm.nih.gov/pubmed/12566288

Brain. 2003 Mar;126(Pt 3):685-91.

Comment in:
Brain. 2003 Sep;126(Pt 9):E4; author reply E5.

Gluten ataxia in perspective: epidemiology, genetic susceptibility and clinical characteristics.

Hadjivassiliou M, **Grünewald R**, **Sharrack B**, **Sanders D**, **Lobo A**, **Williamson C**, **Woodroofe N**, **Wood N**, **Davies-Jones A**.

Department of Neurology, The Royal Hallamshire Hospital, Sheffield, UK.
m.hadjivassiliou@sheffield.ac.uk

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. Gluten ataxia is therefore the single most common cause of sporadic idiopathic ataxia. Antigliadin antibody testing is essential at first presentation of patients with sporadic ataxia.

Full free study available

http://www.ncbi.nlm.nih.gov/pubmed/7854913

Nord Med. 1995;110(2):56-9.

[Anders Jahre Prize for young researchers 1994. Chronic intestinal inflammation studies with multi-colored immunohistochemistry]

[Article in Norwegian]

Halstensen TS.

Laboratorium for Immunhistokjemi og Immunpatologi (LIIPAT), Rikshospitalet, Oslo.

Multi-colour immunofluorescence can be a potent tool in investigating the cause of chronic inflammation of unknown etiology. The immune system uses two main mechanisms for target destruction; antibody-induced, complement-mediated or cell-mediated lysis. Immunohistochemical tracing of complement activation and immunoglobulin deposition in intestinal lesions from patients with ulcerative colitis and Crohn's disease show that ulcerative colitis may be an autoimmune disease characterized by an autoantibody of the IgG1 subclass to an apically located colonic autoantigen, which induce in situ complement activation and cellular lysis. The apical immune complex deposits co-localized with a previous identified putative autoantigen of 40kD, with peptide sequence homology with tropomyosin. Coeliac disease is a mucosal hypersensitivity to the wheat protein gluten. The intestinal lesion shows a peculiar disease specific increased percentage of T cell receptor (TCR)gamma/delta cells in the epithelium; differential use of CD45-isoforms and proliferative activation of intraepithelial T cells. T-helper(CD4) cells in the lamina propria show a non proliferative activation (CD25) which can be induced after gluten challenge in vitro. Immunomagnetic isolation of such in vitro activated T cells renders it possible to establish gluten reactive T cell clones that respond to gluten presented in the context of the disease associated human leucocyte antigen HLA-DQ2.

http://www.ncbi.nlm.nih.gov/pubmed/7427205

Br Med J. 1980 Jul 12;281(6233):115.

Reversal of premature hair greying in adult coeliac disease.

Hill LS.

Full free study available : VERY INTERSTING !

Ph. Hug remark :

When I'm under EMF exposition, I loose my hair and they become more grey.

Some weeks after exposure, I don't loose my hair, but it takes long, long time for them to recover the original color !

http://www.ncbi.nlm.nih.gov/pubmed/15965221

J Neurol Neurosurg Psychiatry. 2005 Jul;76(7):1028-30.

Gluten sensitivity and neuromyelitis optica: two case reports.

Jacob S, **Zarei M**, **Kenton A**, **Allroggen H**.

Department of Neurology, University Hospitals of Leicester NHS Trust, Royal Infirmary, UK.

Neuromyelitis optica is a clinical syndrome characterised by acute transverse myelitis plus an acute or subacute optic neuritis with or without recovery. Although once believed to be a variant of multiple sclerosis, diagnostic criteria have recently been proposed for neuromyelitis optica, making it a clinically distinct syndrome. The term gluten sensitivity refers to a state of heightened immunological responsiveness to ingested gluten in genetically susceptible individuals, as indicated by circulating antibodies to gliadin. Several neurological complications have been described associated with gluten sensitivity ranging from peripheral neuropathy and cerebellar ataxia to an increased risk of epilepsy. Although myelopathy has been described in some case reports of coeliac disease, neuromyelitis optica has never been described in association with gluten sensitivity. We describe two cases of gluten sensitivity presenting as neuromyelitis optica with no previous history of significant gastrointestinal symptoms. Gluten sensitivity was confirmed by immunological and histological studies.

Full free study available

http://www.ncbi.nlm.nih.gov/pubmed/12080401

Eur J Clin Nutr. 2002 Jul;56(7):622-8.

Depression of the glycemic index by high levels of beta-glucan fiber in two functional foods tested in type 2 diabetes.

Jenkins AL, **Jenkins DJ**, **Zdravkovic U**, **Würsch P**, **Vuksan V**.

Clinical Nutrition and Risk Factor Modification Centre, St Michael's Hospital, Toronto, ON, Canada.

OBJECTIVES: To determine the extent to which beta-glucan reduces the glycemic index (GI) of oat products and whether high levels of beta-glucan impair palatability.

DESIGN: The study design was an open-label, randomized cross-over study with six treatment segments.

SETTING: Free-living outpatients.

SUBJECTS: Sixteen volunteers with type 2 diabetes (10 men, six women, 61+/-2 y, body mass index 29+/-2 kg/m(2), HbA1c 7.4+/-0.4%) were recruited from the St Michael's Hospital diabetes clinic.

INTERVENTIONS: Volunteers were given, in random order, 50 g available carbohydrate portions of: white bread; a commercial oat bran breakfast cereal (4.4 g% beta-glucan); and a prototype beta-glucan-enriched breakfast cereal and bar, both high in beta-glucan (8.1 and 6.5 g% beta-glucan, respectively) and sweetened with fructose. Capillary blood samples were taken fasting and then 30, 60, 90, 120, 150 and 180 min after the start of the meal. Palatability was recorded using two different methods.

RESULTS: The glycemic indices of the prototype beta-glucan cereal (mean+/-s.e.m.; 52+/-5) and beta-glucan bar (43+/-4.1) were significantly lower than the commercial oat bran breakfast cereal (86+/-6) and white bread (100; $P<0.05$). All foods were highly palatable and not significantly different. It was found that the GI of the test foods used in this study decreased by 4.0+/-0.2 units per gram of beta-glucan compared to our estimate of 3.8+/-0.6 for studies reported in the literature.

CONCLUSION: Addition of beta-glucan predictably reduces the GI while maintaining palatability. In a 50 g carbohydrate portion each gram of beta-glucan reduces the GI by 4 units, making it a useful functional food component for reducing postprandial glycemia.

SPONSORSHIP: Nestec, Switzerland.

Free full study available

http://www.ncbi.nlm.nih.gov/pubmed/12483040

Int Arch Allergy Immunol. 2002 Dec;129(4):348-50.

Enhancement of allergic skin wheal responses by microwave radiation from mobile phones in patients with atopic eczema/dermatitis syndrome.

Kimata H.

Department of Allergy, Unitika Central Hospital, Uji, Uji City, Kyoto, Japan.
unitikah@m12.alpha-net.ne.jp

Microwave radiation from mobile phones enhanced skin wheal responses induced by house dust mite and Japanese cedar pollen while it had no effect on wheal responses induced by histamine in patients with atopic eczema/dermatitis syndrome (AEDS). Microwave radiation also increased plasma levels of substance P (SP) and vasoactive intestinal peptide (VIP) in patients with AEDS. These results indicate that microwave radiation from mobile phones may enhance allergen-induced wheal responses in association with the release of SP and VIP. This finding may be useful in elucidating the pathophysiology and treatment of AEDS.

http://www.ncbi.nlm.nih.gov/pubmed/15369195

Behav Med. 2004 Winter;29(4):149-52.

Laughter counteracts enhancement of plasma neurotrophin levels and allergic skin wheal responses by mobile phone-mediated stress.

Kimata H.

Department of Allergy, Ujitakeda Hospital, Japan. unitikah@m12.alpha-net.ne.jp

Laughter caused by viewing a comic video (Rowan Atkinson's The Best Bits of Mr. Bean) reduced the plasma nerve growth factor, neurotrophin-3 levels, and allergic skin wheal responses in patients with atopic dermatitis, whereas viewing a nonhumorous video (weather information) failed to do so. In contrast, stress induced by writing mail on a mobile phone enhanced the plasma nerve growth factor, neurotrophin-3 levels, and allergic skin wheal responses. However, previewing the comic video counteracted mobile phone-mediated enhancement of plasma neurotrophins or allergic skin wheal responses, whereas previewing the weather information failed to do so. Taken together, these results suggest that, in patients with atopic dermatitis, writing mail on a mobile phone causes stress and enhances allergic responses with a concomitant increase in plasma neurotrophins that are counteracted by laughter. These results may be useful in the study of pathophysiology and treatment of atopic dermatitis.

http://www.ncbi.nlm.nih.gov/pubmed/10975212

Diabet Med. 2000 Jun;17(6):441-4.

Autoantibodies to tissue transglutaminase are sensitive serological parameters for detecting silent coeliac disease in patients with Type 1 diabetes mellitus.

Kordonouri O, **Dieterich W**, **Schuppan D**, **Webert G**, **Müller C**, **Sarioglu N**, **Becker M**, **Danne T**.

Department of Paediatrics, Charité, Humboldt University, Berlin, Germany.
olga.kordonouri@charite.de

AIMS: To investigate the clinical significance of the determination of IgA antibodies to tissue transglutaminase (tTG) for the detection of silent coeliac disease in patients with Type 1 diabetes mellitus.

METHODS: A total of 520 patients with diabetes (median age 14.2 years, range 1-27) were tested for IgA antibodies to tTG (IgA anti-tTG, ELISA), endomysium (EmA, indirect immunofluoresence) and gliadin (IgA-AGA, enzyme immunometric assay) after ruling out IgA deficiency.

RESULTS: The prevalence of IgA anti-tTG among patients with diabetes was 4.4% (23 of 520), and that of EmA and IgA-AGA 3.5% (18 of 520, respectively). The coefficient of agreement between IgA anti-tTG and EmA was high (Cohen's kappa = 0.87, $P < 0.001$). Thirteen of the 23 IgA anti-tTG-positive patients underwent duodenal biopsy. Coeliac disease was confirmed in nine of 13 patients. One of them was negative for EmA and AGA, but positive for IgA anti-tTG. Retrospective annual determinations up to 8 years in six IgA anti-tTG-positive patients showed both permanent and transient elevations of the serological markers.

CONCLUSIONS: These data show that a positive IgA antibody test to tTG is a more sensitive parameter than EmA for silent coeliac disease in patients with diabetes. Confirmatory small bowel biopsy, however, remains necessary for diagnosis as some patients with positive antibodies may be without histological changes.

http://www.ncbi.nlm.nih.gov/pubmed/10525659

Diabetologia. 1999 Oct;42(10):1195-8.

Comment in:
Diabetologia. 2000 Jun;43(6):815-6.

Antibodies to tissue transglutaminase C in type I diabetes.

Lampasona V, **Bonfanti R**, **Bazzigaluppi E**, **Venerando A**, **Chiumello G**, **Bosi E**, **Bonifacio E**.

Department of Laboratory Medicine, San Raffaele Scientific Institute, Milan, Italy.

AIMS/HYPOTHESIS: Silent coeliac disease is a gluten driven autoimmune disease which is relatively frequent in patients with Type I (insulin-dependent) diabetes mellitus. To determine the extent of gluten associated autoimmunity in Type I diabetes, autoantibodies to tissue transglutaminase C, a major autoantigen in coeliac disease, were measured in patients with new-onset Type I diabetes.

METHODS: We measured IgG and IgA tissue transglutaminase C autoantibodies using human recombinant antigen and radio-binding assays in a cohort of 287 patients with new-onset Type I diabetes, 119 with Type II (non-insulin-dependent) diabetes mellitus and in 213 control subjects.

RESULTS: We found IgA and IgG tissue transglutaminase C antibodies in 24 (8 %) patients with Type I diabetes; 97 (33 %) patients had IgG antibodies only and 1 IgA antibodies only. Antibody concentrations were highest in those with both IgA and IgG antibodies. Only 2 (2 %) patients with Type II diabetes and 2 (1 %) control subjects had either IgG or IgA tissue transglutaminase C antibodies. Patients with HLA DRB1(*)04 alleles had the highest prevalence of IgG tissue transglutaminase C antibodies.

CONCLUSION/INTERPRETATION: These data show that almost 10 % of patients have autoimmunity typical of coeliac disease and that another 30 % have low level tissue transglutaminase C antibody binding. This high prevalence suggests either involvement of the gut in the pathogenesis of Type I diabetes or that transglutaminase is a secondary autoantigen resulting from beta-cell destruction.

[Diabetologia (1999) 42: 1195-1198]

http://www.ncbi.nlm.nih.gov/pubmed/12556782

Gastrointest Endosc. 2003 Feb;57(2):187-91.

Comment in:
Gastrointest Endosc. 2004 Jan;59(1):158-9; author reply 159-60.
Gastrointest Endosc. 2004 Jan;59(1):160-1; author reply 161.

Duodenal histology in patients with celiac disease after treatment with a gluten-free diet.

Lee SK, **Lo W**, **Memeo L**, **Rotterdam H**, **Green PH**.

Department of Surgical Pathology and Medicine, Columbia University College of Physicians and Surgeons, New York, New York, USA.

BACKGROUND: The diagnosis of celiac disease requires characteristic histopathologic changes in an intestinal biopsy with clinical improvement in response to a gluten-free diet. Endoscopy with procurement of biopsy specimens is often performed to document response to the diet, but there are little data on the appearance of treated celiac disease. This study examined the endoscopic and histopathologic appearance of the duodenum of patients with celiac disease whose diet was gluten-free.

METHODS: A cohort of 39 adult patients (mean age 52 years, range 20-74 years) with biopsy-proven celiac disease was retrospectively reviewed. All had responded clinically to a gluten-free diet that they had maintained for a mean of 8.5 years (range 1-45 years). The endoscopic and histopathologic appearances of the duodenal mucosa were reviewed. Blinded review of the diagnostic (initial) and post-treatment biopsy specimens was also performed to assess response of individual patients to the diet.

RESULTS: The endoscopic appearance was normal in 23%, reduced duodenal folds were present in 46%, scalloping of folds in 33%, mucosal fissures in 44%, and nodularity in 33%. There was more than 1 abnormality present in 46%. Histology was normal in only 21%. The remainder had villous atrophy (69% partial, 10% total). Paired (diagnostic and follow-up) biopsy specimens were reviewed blindly for 12 patients. The mean (SD) intraepithelial lymphocyte count fell from 61 (22) to 38 (17) (normal <30 per 100 epithelial cells) and the crypt-to-villous ratio improved although it did not normalize.

CONCLUSIONS: Despite a good clinical response, abnormal endoscopic and histopathologic appearances persist in the majority of patients with celiac disease treated with a gluten-free diet.

http://www.ncbi.nlm.nih.gov/pubmed/12949716

Gastroenterology. 2003 Sep;125(3):696-707.

Alterations of the intestinal transport and processing of gliadin peptides in celiac disease.

Matysiak-Budnik T, **Candalh C**, **Dugave C**, **Namane A**, **Cellier C**, **Cerf-Bensussan N**, **Heyman M**.

INSERM EMI 0212, Faculté Necker-Enfants Malades, Paris.

BACKGROUND & AIMS:The hypothesis of a defect in the intestinal transport and processing of toxic (31-49) or immunostimulant (57-68 and the 33-mer 56-89) gliadin peptides was tested in patients with active celiac disease (ACD), patients with treated celiac disease (TCD), and controls.

METHODS: Using duodenal biopsy specimens mounted in Ussing chambers, we measured electrical resistance, mucosal-to-serosal radiolabeled-peptide fluxes, and peptide processing during transport using radio-reverse-phase high-performance liquid chromatography.

RESULTS: Peptide 31-49 fluxes (24.7 microg x 3 h(-1). cm(-2)) were increased in patients with ACD compared with controls and patients with TCD (12.7 and 12.3 microg x 3 h(-1). cm(-2); $P < 0.01$). In contrast, no increase was observed for peptide 57-68 or 56-89 (33-mer). Electrical resistance was decreased in patients with ACD versus controls (15.3 vs. 23.9 ohms. cm(2); $P < 0.001$). Peptide 57-68 was partially degraded by brush-border peptidases in controls but not in patients with celiac disease. However, it was totally degraded after intestinal transport both in controls and patients with celiac disease. Peptides 31-49 and 56-89 were resistant to brush-border peptidases in all groups of patients but were totally degraded during intestinal transport in controls and patients with TCD. In patients with ACD, however, 50% of peptide 31-49 was delivered intact into the serosal compartment and only partial degradation of the 33-mer was observed. These abnormalities were not related to a nonspecific paracellular leakage.

CONCLUSIONS: Our data indicate that gliadin peptides, although poorly or not digested by intraluminal enzymes, can be fully digested by enterocytes in controls and patients with TCD. In patients with ACD, incomplete degradation of the 33-mer and protected transport of the peptide 31-49 might favor their respective immunostimulatory and toxic effects.

Ph. Hug remark : what are the EMF effects on peptide's electrical resistance ? I bet the resistance will decrease...

http://www.ncbi.nlm.nih.gov/pubmed/16194732

Pediatr Neurol. 2005 Oct;33(4):292-5.

Successful treatment of epilepsy and celiac disease with a gluten-free diet.

Mavroudi A, **Karatza E**, **Papastavrou T**, **Panteliadis C**, **Spiroglou K**.

Department of Pediatrics, 3rd Pediatric Clinic, Division of Digestive Diseases, Aristotle University of Thessaloniki, Hippokration Hospital, Thessaloniki, Greece.

Celiac disease is a gluten-sensitive enteropathy, which recently has been described in association with epilepsy or other neurologic disturbances. This study describes a case of a 7-year-old female with intractable-to-treatment epilepsy and late-onset celiac disease, who was treated successfully with a gluten-free diet plus antiepileptic therapy. It is important for children with intractable cases of epilepsy and weight loss to undergo screening for celiac disease.

http://www.ncbi.nlm.nih.gov/pubmed/8948386

J Am Diet Assoc. 1996 Dec;96(12):1254-61.

Oat bran concentrate bread products improve long-term control of diabetes: a pilot study.

Pick ME, **Hawrysh ZJ**, **Gee MI**, **Toth E**, **Garg ML**, **Hardin RT**.

Department of Agricultural, Food and Nutritional Science, University of Alberta, Edmonton, Canada.

OBJECTIVE: To evaluate the long-term effects oat bran concentrate bread products in the diet of free-living subjects with non-insulin-dependent diabetes (NIDDM) via dietary, clinical, and biochemical methods.

DESIGN: A 24-week crossover study consisting of two 12-week periods.

SUBJECTS/SETTING: Eight men with NIDDM (mean age = 45 years) who lived in the community. Glucose and insulin profiles were conducted in a clinical investigation unit.

INTERVENTION: Palatable, high-fiber, oat bran concentrate (soluble fiber [beta-glucan] content = 22.8%) bread products were developed. Four randomly chosen subjects ate oat bran concentrate breads first; the other subjects ate control white bread first.

MAIN OUTCOME MEASURES: Dietary intake (four 48-hour dietary recalls per period) was assessed. Blood glucose and insulin (8-hour profiles) and lipid parameters after fasting were measured (at 0, 12, and 24 weeks).

STATISTICAL ANALYSES PERFORMED: Analysis of variance and repeated-measures analysis of variance.

RESULTS: Total energy and macronutrient intakes were similar in both periods. Mean total dietary fiber intake was 19 g/day in the white bread period and 34 g/day (9 g soluble fiber per day from oat bran concentrate) in the oat bran concentrate period. Body weight remained stable. Mean glycemic and insulin response areas (area under the curve) were lower ($P < or = .05$ and not significant, respectively) for the oat bran concentrate period than the white bread period. After breakfast, area under the curve for the oat bran concentrate period was lower for glucose ($P < or = .01$) and insulin ($P < or = .05$); insulin peak was reached earlier ($P < or = .05$) than in the white bread period. Dietary fiber intake was correlated negatively with insulin area under the curve ($P < or = .05$). Mean total plasma cholesterol and low-density lipoprotein cholesterol levels were lower ($P < or = .01$) in the oat bran concentrate period than in the white bread period. In the oat bran concentrate period, the mean ratio of low-density lipoprotein cholesterol to high-density lipoprotein cholesterol was reduced by 24% ($P < or = .05$).

CONCLUSIONS: The well-accepted oat bran concentrate bread products improved glycemic, insulinemic, and lipidemic responses.

http://www.ncbi.nlm.nih.gov/pubmed/18217518

Am Fam Physician. 2007 Dec 15;76(12):1795-802.

Summary for patients in:
Am Fam Physician. 2007 Dec 15;76(12):1809-10.

Celiac disease.

Presutti RJ, **Cangemi JR**, **Cassidy HD**, **Hill DA**.

Department of Family Medicine, Mayo Clinic, Jacksonville, Florida 32224, USA.

As many as one in every 100 to 200 persons in the United States has celiac disease, a condition resulting from an inappropriate immune response to the dietary protein gluten. The manifestations of celiac disease range from no symptoms to overt malabsorption with involvement of multiple organ systems and an increased risk of some malignancies. When celiac disease is suspected, initial testing for serum immunoglobulin A (IgA) tissue transglutaminase (tTG) antibodies is useful because it offers adequate sensitivity and specificity at a reasonable cost. A positive IgA tTG result should prompt small bowel biopsy with at least four tissue samples to confirm the diagnosis. However, 3 percent of patients with celiac disease have IgA deficiency. Therefore, if the serum IgA tTG result is negative but clinical suspicion for the disease is high, a serum total IgA level may be considered. Screening of asymptomatic patients is not recommended. The basis of treatment for celiac disease is adherence to a gluten-free diet, which may eliminate symptoms within a few months. Patients should also be evaluated for osteoporosis, thyroid dysfunction, and deficiencies in folic acid, vitamin B12, fat-soluble vitamins, and iron, and treated appropriately. Serum IgA tTG levels typically decrease as patients maintain a gluten-free diet.

http://www.ncbi.nlm.nih.gov/pubmed/17226827

Muscle Nerve. 2007 May;35(5):675-7.

Celiac disease presenting with motor neuropathy: effect of gluten free-diet.

Rigamonti A, **Magi S**, **Venturini E**, **Morandi L**, **Ciano C**, **Lauria G**.

Neuromuscular Diseases Unit, National Neurological Institute Carlo Besta, Via Celoria 11, 20133 Milan, Italy.

Celiac disease is a rare cause of neuropathy that most commonly presents with symmetric distal sensory disturbances. We describe two patients with celiac disease in whom neuropathy presented unusually with progressive weakness of the limbs. In both patients a gluten-free diet induced a significant improvement of muscle strength and neurophysiological abnormalities, suggesting a direct pathogenetic role of sensitivity to gluten. Celiac disease should be considered in patients with idiopathic neuropathy even when gastrointestinal symptoms are absent.

http://www.ncbi.nlm.nih.gov/pubmed/16416238

Dig Dis Sci. 2006 Jan;51(1):213-4.

Liver dysfunction after a gluten-free diet in a patient with celiac disease: a new link?

Selcuk H, **Kanbay M**, **Murat K**, **Yilmaz U**.

No abstract available on PubMed
First page abstract free on the editor paper

http://www.ncbi.nlm.nih.gov/pubmed/11909917

J Neurol Neurosurg Psychiatry. 2002 Apr;72(4):527-9.

Ataxia in the setting of complicated enteropathy: double jeopardy.

Shams PN, **Waldman A**, **Dogan A**, **MacKenzie JM**, **Plant GT**.

National Hospital for Neurology and Neurosurgery, Queen Square, London, UK.

The differential diagnosis of subacute onset ataxia in the setting of enteropathy is wide. A 54 year old patient with a pancerebellar syndrome and known ulcerative jejunoileitis is described. Small bowel biopsy showed evidence of enteropathy associated T cell lymphoma and subsequent neuropathological analysis and immunophenotyping confirmed metastasis of this tumour to the cerebellum. The presence of anti-gliadin antibodies and MRI evidence of a more longstanding process suggested additional immunologically mediated cerebellar dysfunction. Lymphomatous involvement of the CNS is rare in patients with complicated enteropathies, and has not been previously reported to involve the cerebellar parenchyma. This diagnostic possibility should be borne in mind before attributing cerebellar dysfunction in patients with the coeliac related enteropathies to nutritional compromise or immunological dysfunction (gluten ataxia) alone.

Full free study available

http://www.ncbi.nlm.nih.gov/pubmed/15586305

Clin Rheumatol. 2005 Jun;24(3):301-4. Epub 2004 Dec 7.

Midaortic syndrome and celiac disease: a case of local vasculitis.

Stadlmaier E, **Spary A**, **Tillich M**, **Pilger E**.

Rheumatic Diseases Division, Department of Internal Medicine, Medical University Graz, Auenbruggerplatz 15, 8036, Graz, Austria. elke.stadlmaier@klinikum-graz.at

Midaortic syndrome is a variety of aortic coarctation, located in the distal thoracic aorta, the abdominal aorta or both, involving the intestinal and renal vessels, usually presenting with renovascular arterial hypertension. Underlying conditions are thought to be Takayasu's arteritis, von Recklinghausen's disease, and connate hypoplasia. Celiac disease is an inflammation in the small intestine, triggered by an allergic reaction to gluten. It is known to be associated with a variety of other autoimmune disorders, e.g., dermatitis herpetiformis (Duhring's disease), insulin-dependent diabetes mellitus, and IgA nephropathy. We describe the case of a young woman who presented with claudication of the lower limbs, therapy-refractory arterial hypertension, and untreated celiac disease. We found a midaortic syndrome, characterized by severe stenosis of the infrarenal aorta, of both renal arteries (more pronounced on the right side) and of the inferior mesenteric artery. We assume that-after having excluded other possible pathogeneses-the underlying condition is a local vasculitis in the abdominal aorta and the renal and mesenteric arteries due to the chronic inflammation of untreated celiac disease. We performed a percutaneous transluminal angioplasty together with implantation of two stents into the infrarenal aorta and the right renal artery and started treating the celiac disease by dietary intervention. The patient is now under regular medical control and observation.

http://www.ncbi.nlm.nih.gov/pubmed/16766754

Gut. 2006 Jul;55(7):1037-46.

Comment in:
Gut. 2006 Nov;55(11):1672.
Gut. 2007 Feb;56(2):310; author reply 310-1.

Recent advances in coeliac disease.

van Heel DA, **West J**.

Institute of Cell and Molecular Science, Barts and The London, Queen Mary's School of Medicine and Dentistry, Turner St, London E1 2AD, UK. d.vanheel@qmul.ac.uk

No abstract available
Full free study available

http://www.ncbi.nlm.nih.gov/pubmed/12219789

Am J Clin Pathol. 2002 Sep;118(3):459-63.

Histologic follow-up of people with celiac disease on a gluten-free diet: slow and incomplete recovery.

Wahab PJ, **Meijer JW**, **Mulder CJ**.

Department of Gastroenterology and Hepatology, Rijnstate Hospital Arnhem, The Netherlands.

To assess histologic recovery in response to gluten withdrawal in celiac disease, 158 patients seen in our hospital during a 15-year period underwent follow-up small intestine biopsies (SIBs) within 2 years after starting a gluten-free diet; further SIBs were done if villous atrophy was present. A modified Marsh classification was used (IIIA, partial villous atrophy; IIIB, subtotal villous atrophy; IIIC, total villous atrophy). Of patients with Marsh IIIA, IIIB, or IIIC lesions, histologic remission was seen in 65.0% within 2 years, 85.3% within 5 years, and 89.9% in long-term follow-up. Eleven patients (7.0%) with persisting (partial) villous atrophy had symptoms and signs of malabsorption and were considered to have refractory celiac disease; 5 of them developed an enteropathy-associated T-cell lymphoma. Children recovered up to 95% within 2 years and 100% in the long-term. Histologic recovery in celiac disease after starting a gluten-free diet takes time and is incomplete or absent in a substantial subgroup of patients (10.1% villous atrophy after 5 years). Systematic follow-up of patients with celiac disease and the malabsorption syndrome and secondary complications is needed.

Full free study available

http://www.ncbi.nlm.nih.gov/pubmed/9353622

Diabetes Care. 1997 Nov;20(11):1774-80.

The role of viscous soluble fiber in the metabolic control of diabetes. A review with special emphasis on cereals rich in beta-glucan.

Würsch P, **Pi-Sunyer FX**.

Nestlé Research Centre, Lausanne, Switzerland.

Recent recommendations for the dietary management of diabetes mellitus state that diet needs to be individualized so that there is improved glucose and lipid control in the patient. In a majority of individuals with diabetes, this is best done with a diet that is low in fat and high in carbohydrate, particularly that of cereal origin. However, symptoms of hyper- and hypoglycemia must be averted. Most cereal products, however, tend to have a high glycemic index Cereals such as Prowashonupana barley or fractions of oat bran are particularly high in the soluble fiber beta-glucan, which when taken with a meal increases the viscosity of the meal bolus once it has reached the small intestine, where the absorption of nutrients occurs. This high viscosity delays absorption. A 50% reduction in glycemic peak can be achieved with a concentration of 10% beta-glucan in a cereal food. A significant lowering of plasma LDL cholesterol concentrations can also be anticipated with the daily consumption of > or = 3 g of beta-glucan. Diabetic individuals can benefit from diets that are high in beta-glucan, which, as a component of oats and barley, can be incorporated into breakfast cereals and other products.

http://bastyrcenter.org/content/view/313/

Allergic Reactions Enhanced by Cell Phone Use

Cellular phone use for one continuous hour exacerbates allergic responses to dust and pollen in adults with eczema, according to a new study in *International Archives of Allergy and Immunology* (**2002**;129:348–50). The findings of the study, while only preliminary, suggest that the microwave radiation emitted from cell phones may increase sensitivity to specific allergens, potentially making allergy symptoms worse.

In the new study, 52 men and women between the ages of 21 and 52 with eczema and known allergies to dust and Japanese cedar pollen were divided into two groups. One group was exposed to 60 minutes of continuous cell phone calling while the other group was not exposed to cell phone calls. Skin scratch testing for dust, Japanese cedar pollen, histamine, and a control solution was performed before and after the cell phone exposure. The diameter of the skin response was measured after 15 minutes, two hours, and four hours following the scratch to determine the degree of sensitivity. (A greater diameter of reaction on the skin suggests a stronger allergic response.) Blood levels of substance P and vasoactive intestinal peptide (VIP), substances associated with causing allergy symptoms, were also taken at the same intervals.

Those exposed to microwave radiation from cell phones had a significant increase in the allergic response to dust and Japanese cedar pollen, as well as increased blood levels of substance P and VIP. In contrast, no significant changes in any measurement were seen in the group that had not received the phone calls. The enhanced allergic responses to dust and Japanese cedar pollen was maintained after two hours, but not after four hours in the group receiving cell phone calls.

In the current study, microwave radiation from cell phones stimulated the release of substance P and VIP, both of which are associated with triggering allergic reactions and are elevated in the secretions of people with nasal allergies. While stronger skin scratch responses were observed in cell phone users, it is not clear how microwave radiation affects symptoms of allergies, such as runny nose, itchy eyes, scratchy throat, and hives. It is also unknown whether shorter or longer periods of microwave radiation exposure would produce similar results. Future studies examining the effect of cell phones on allergy symptoms are needed to clarify these issues.

The effect of cell phones on human health is not clear thus far. Some studies suggest the microwave radiation emitted from cell phones may lead to brain cancer, but not all studies agree. Other studies suggest cell phones may cause other neurological problems, such as altered motor coordination and sleep difficulties. Since cell phones have only been in commercial use for just over a decade, it may be too soon to determine their long-term effects on health and disease.

Darin Ingels, ND, MT (ASCP), received his bachelor's degree from Purdue University and his Doctorate of Naturopathic Medicine from Bastyr University in Kenmore, WA. Dr. Ingels is the author of *The Natural Pharmacist: Lowering Cholesterol* (Prima, 1999) and *Natural Treatments for High Cholesterol* (Prima, 2000). He currently is in private practice at New England Family Health Associates located in Southport, CT, where he specializes in environmental medicine and allergies. Dr. Ingels is a regular contributor to Healthnotes and *Healthnotes Newswire*.

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