Tom O’Bryan, DC holds:

- Adjunct Faculty, The Institute for Functional Medicine,
- Adjunct Faculty, The National University of Life Sciences,
- Clinical Consultant on Functional Medicine - BioBotanical Research
- Clinical Consultant on Functional Medicine - NuMedica, Inc.
- Scientific Advisory Board - International and American Association of Clinical Nutritionists
- Medical Advisory Board National Association of Nutritional Professionals
MECHANISM OF ACTION OF LDN

LDN

- Increase in endogenous enkephalin and endorphin
- Inhibition of proinflammatory cytokines
- Interaction of the nuclear opioid growth factor receptor
- Blockade of opiate-R in GI tract
- Regulation of TReg and production of IL-10 and TGF-β

Enhancement of immune function
Improvement in inflammatory reaction
Healing of corneal ulcers
Improvement in Crohn’s disease activity
Down regulation of TH-17

Promotion of DNA synthesis
Effect on no. of liquid bowel movements
Healing & repair of mucosal tissue

Vojdani, LDN Conference 2008
7 Premises To Look At
Premise #1

Incompletely digested peptides of wheat (exorphins) modulate opioid receptor activity
Glutens, made up of two main fractions, gliadins and glutenins, are the main storage proteins of wheat and are comprised of about 100 different proteins in a given wheat cultivar (variety).
When the various wheat cultivars are considered, the number of different gluten proteins is even greater.
Increased urinary excretion of gluten peptides in patients with schizophrenia has been observed previously.
Referred to as gluten exorphins, these peptides have been shown to have potent opioid-like properties and to affect hormonal balance, behavior, and learning in animal models.
Peptides with activity similar to that of morphine and other opioids have been isolated from the brain and other sources such as the pituitary. These peptides, the endorphins and enkephalins, are synthesized in vivo and may function both as hormones and neurotransmitters.
The results presented here have shown that peptides with morphine-like activities, which we call exorphins, can be isolated from some food proteins (wheat and casein).
Dietary interventions like gluten-free and casein-free diets have been reported to improve intestinal, autoimmune and neurological symptoms in patients with a variety of conditions.
however, the underlying mechanism of benefit for such diets remains unclear
Epigenetic programming, including CpG methylation and histone modifications, occurring during early postnatal development can influence the risk of disease in later life, and such programming may be modulated by nutritional factors such as milk and wheat.
The hydrolytic digestion of casein (a major milk protein) and gliadin (a wheat-derived protein) releases peptides with opioid activity, and in the present study, we demonstrate that these food-derived proline-rich opioid peptides modulate cysteine uptake in human neuronal and gastrointestinal (GI) epithelial cells via activation of opioid receptors.
Decreases in cysteine uptake were associated with changes in the intracellular antioxidant glutathione and the methyl donor S-adenosylmethionine.
Restricted antioxidant capacity, caused by wheat- and milk-derived opioid peptides, may predispose susceptible individuals to inflammation and systemic oxidation, partly explaining the benefits of gluten-free or casein-free diets.
The inhibitory action of the exorphins in wheat has a specific opiate effect. This morphine-like psychoactive nature of the peptides results from the incomplete digestion of these dietary proteins binding to the opiate receptors in the brain, and offers a possible explanation for some of the reported psychiatric reactions to these gluten proteins, including the sense of ‘brain fog’ that often accompanies immune reactions to these foods and which may follow with panic attacks, depression, or other neurological complaints.

- Interchange. 28(2/3):183-189.
Premise #2

What is the Most Common Cause of Morbidity and Mortality in the Industrialized World?
While many individual autoimmune diseases are rare, collectively they are thought to affect approximately 8 percent of the United States population – 24 million persons.
To provide a context to evaluate the impact of autoimmune diseases, cancer affected approximately 9 million people and heart disease affected approximately 22 million people in the United States.
“Collectively Auto-immune Diseases have been identified in about 24 million people in the US, and only 1/3rd are diagnosed. That means about 72 million people have an AI Disease. It’s not looked for. Our system waits until the signs and symptoms are severe enough with organ failure and irreversible damage before we identify it.”
Autoimmune diseases are the third leading cause of morbidity and mortality in the industrialized world, surpassed only by cancer and heart disease.
In the United States, CVD prevalence in the general population is expected to reach 40%, with direct related costs set to reach 800 billion dollars per year in the next two decades.
In Europe, CVD causes 47% of all deaths accounting for 4 million fatalities each year, and costing 196 billion euros a year.
The first indicator of atherosclerosis for 30%-50% of patients was an acute, and in many cases fatal, myocardial infarction (MI).

How is it possible that our Health Care System could be so Blind? We’re looking in the wrong place. And we keep looking in the wrong place.
Perhaps if We Open to More Current Information.....
Immune-driven inflammation is key to the development of cardiovascular disease (CVD)
Atherosclerosis is increasingly considered an immune system–mediated process of the vascular system.
Autoimmunity plays a major role in the development of dyslipidemia and atherosclerotic plaque formation.
Atherogenesis has been proposed to be considered an autoimmune disease, raising the hope that CVD could be prevented by immunomodulation.
Fig. (5). Common changes in the lipid profile amongst the autoimmune rheumatic disease and their impact on atherosclerotic plaque formation. LDL: Low density lipoproteins, TG: Triglycerides, Lp(a): Lipoprotein (a), Anti-LPL: anti-Lipoprotein Lipase, HDL: high density lipoproteins, ApoA1: Apolipoprotein A1, Anti-APL: anti phospholipid.
Autoimmunity plays a major role in the development of dyslipidemia and atherosclerotic plaque formation. The mechanisms underlying these changes include the interplay of inflammation and auto-antibody formation.
Thus, If CVD has an Initiating Autoimmune Component, Arguably, What Becomes the #1 Mechanism in the Progression of Morbidity and Mortality?
Silently
Point to 2 People
Close By

How often do you see Autoimmune Disorders Currently in Your Practice and Given these Numbers, What Would the Impact Be IF You were Recognizing Autoimmune Disorders at this Frequency?

Detective Adrian Monk

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Prevention of Autoimmune Diseases:

- Define genetic make-up of susceptible individuals
- Identify environmental triggers
- Identify autoantibodies
- Develop preventive interventions
Autoimmunity plays a major role in the development of dyslipidemia and atherosclerotic plaque formation. The mechanisms underlying these changes include the interplay of inflammation and auto-antibody formation.

THERAPEUTIC TARGET
Identifying antibodies and Preventive interactions
The overexpression of inflammation genes, immune-response genes and genes associated with the lysosomal system  J Clin Immunol 29:397405, 2009
Premise #3

Genes Control Function
POTENTIAL TRIGGER

#1
Direct evidence for an association between elevated maternal levels of inflammatory mediators and the development of psychosis in offspring has been reported.
The association between maternal gliadin antibodies and the development of psychosis in offspring can potentially be explained by maternal diet.
IgG is actively transported across the placenta during the later stages of pregnancy to provide passive immunization of the fetus, and hence such antibodies reflect maternal exposures and immune responses to specific antigens.
A significantly elevated risk for nonaffective psychoses was associated with high levels (90th percentile) of IgG anti-gliadin antibodies (odds ratio=1.7), but not anti-casein antibodies (odds ratio=0.8).
The risk for future nonaffective psychosis increased further with levels of anti-gliadin antibodies at the 95th percentile (odds ratio=2.5)
FIGURE 1. Levels of IgG Directed at Gliadin and Casein and Odds of Developing Nonaffective Psychosis

Gliadin

Casein

Odds Ratio (95% CI)

75th 90th 95th Percentile

0 1 2 3 4 5

0 1 2 3 4 5
A mechanism potentially linking maternal antigliadin reactivity with the later development of psychosis in offspring involves maternal inflammation.

Note: These are Not Celiacs
So What is the Clinical Relevance of This?
How Do I Use This Information in My Practice?
EVERY PREGNANT WOMAN IS ACCURATELY TESTED FOR A GLUTEN RELATED DISORDER, NOT JUST CELIAC DISEASE
The seroprevalence of transglutaminase IgA was 6.70% in the group with recurrent abortion, 5.70% in the group with stillbirth, 5.65% in the group with infertility, 9.33% in the group with intrauterine growth restriction, and 1.30% in the control group.
Premise #4

Food Turns On and Turns OFF Our Genes

Detective Adrian Monk

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Our aim was to test whether carbohydrate dietary modifications improve insulin sensitivity and secretion and glucose tolerance in overweight or obese persons with the metabolic syndrome, even in the absence of weight loss.
Dietary carbohydrate modification induces alterations in gene expression in abdominal subcutaneous adipose tissue in persons with the metabolic syndrome: the FUNGENUT Study¹⁻⁴

Peteri Kallio, Marjukka Kolehmainen, David E Leaksoun, Jari Kekäläinen, Titu Salopuu, Katarina Sherins, Leena Pullkonen, Hannu M Mykkänen, Leo Niskanen, Matti Uusitupa, and Kaisa S Poutanen

ABSTRACT
Background: Diets rich in whole-grain cereals and foods with a low glycemic index may protect against type 2 diabetes, but the underlying molecular mechanisms are unknown.

Objectives: The main objective was to test whether 2 different car-
bohydrate sources (rye bread and pasta or oat and wheat bread and potato) were the main carbohydrate sources (34% and 37% of energy intake, respectively).

Introduction

The subjects were randomly assigned to 12-week diets in which either rye bread and pasta or oat and wheat bread and potato were the main carbohydrate sources (34% and 37% of energy intake, respectively).
Dietary carbohydrate modification induces alterations in gene expression in abdominal subcutaneous adipose tissue in persons with the metabolic syndrome: the FUNGENUT Study\(^1\-4\)

Petteri Kallio, Marjukka Kolehmainen, David E Leakounen, Jari Kokkola, Tiina Salopuro, Katarina Siivenius, Leena Pulkkinen, Haruna M Mykkänen, Leo Niskanen, Matti Uusitupa, and Kaisa S Poutanen

ABSTRACT

Background: Diets rich in whole-grain cereals and foods with a low glycemic index may protect against type 2 diabetes, but the underlying molecular mechanisms are unknown.

Objective: The main objective was to test whether 2 different carboxylic acid sections of the long-term (1-5). In line with this hypothesis, we found that high-fiber rye bread increased the acute insulin response, but insulin sensitivity remained unchanged (16). Furthermore, we recently showed that rye and pasta-based carbohydrate modification can enhance early insulin secretion in persons with the metabolic syndrome (17), although no changes in glucose tolerance or insulin resistance were observed. This effect was found to be independent of the fiber content of the diet.

Abdominal subcutaneous adipose tissue (SAT) produces a variety of secretory factors that have an important role in inflammation and insulin resistance via endocrine, paracrine, or autocrine signals (18, 19). Impaired insulin signaling occurs in

Results: We detected 71 down-regulated genes in the rye-pasta group, including genes linked to insulin signaling and apoptosis. In contrast, the 12-week oat-wheat-potato diet up-regulated 62 genes related to stress, cytokine-chemokine-mediated immunity, and the interleukin pathway. The insulinogenic index improved after the rye-wheat diet (P = 0.003) but not after the oat-wheat-potato diet. Body weight was unchanged in both groups.

Conclusions: Dietary carbohydrate modification with rye or oat or wheat, and potato differentially modulates the gene expression profile in abdominal subcutaneous adipose tissue, even in the absence of weight loss. Am J Clin Nutr 2007;85:1417-27.

KEY WORDS Gene-nutrient interactions, metabolic syndrome, insulin resistance, microarray, adipose tissue, diet intervention, insulinogenic response, rye, oat, wheat

INTRODUCTION

The pathogenesis of the metabolic syndrome is not well understood, but lifestyle, including diet, and genetic factors clearly interact in its development and progression. These interactions are likely to be reflected in gene expression. The metabolic syndrome, characterized by central obesity, abnormal insulin and glucose metabolism, dyslipidemia, and hypertension, predisposes to cardiovascular diseases and especially type 2 diabetes (T2DM) (1-5).

2 From the Department of Clinical Nutrition, Food and Health Research Centre (PK, MLK, and KSP), Department of Medicine (PK, MLK, and KSP), Department of Computer Science (JK), and Department of Clinical Nutrition (TS, KS, LP, BM, and MU), University of Kuopio, Kuopio, Finland, and YTT, Espoo, Finland (KSP).

3 PK and MLK contributed equally to this work.

4 Supported by Finnish Bakeries Ltd, Vassan & Vassan Oy, the Technology Development Center of Finland, the Academy of Finland (no. 208443), the Sigrid Juselius Foundation, and the ABS graduate school.

5 Address reprint requests to M. Kolehmainen, Department of Clinical Nutrition, Food and Health Research Centre, University of Kuopio, Finland. E-mail: marjukka.kolehmainen@uku.fi.

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Dietary carbohydrate modification induces alterations in gene expression in abdominal subcutaneous adipose tissue in persons with the metabolic syndrome: the FUNGENUT Study\(^1\)\(^{-4}\)

Peteri Kiili, Marjukka Kolehmainen, David E. Laakso, Jari Kekäläinen, Tiitu Selopuro, Katarina Sivenius, Leena Pulkkinen, Hanna M. Mykkänen, Leo Niskanen, Matti Uusitupa, and Raisa S. Poussa

ABSTRACT

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In contrast, the 12-week oat-wheat-potato diet upregulated 62 genes related to stress, cytokine–chemokine–mediated immunity, and the interleukin pathway.

Am J Clin Nutr. 2007;85:1417-1427

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Premise #5

Where Does the Persisting Inflammation Come From?
Proposed mechanisms of association (in AID development) include abnormal regulation of intestinal permeability and increased autoantibody production in the setting of chronic gut inflammation.
Fig. 1. Autoimmune and inflammatory diseases in relation to celiac disease. *Strongest associations.
Why Creating the Healthiest Intestinal Environment Possible Can Arrest Your Vulnerability to the #3 Cause of Getting Sick and Dying

Alessio Fasano, MD
Currently at Harvard’s Mass General Hospital for Children where he Chairs the Department of Pediatric Gastroenterology
Dr. Fasano, Could you tell us, what is the importance of pathogenic intestinal permeability?
It’s one of the key functions of the intestine that I probably think has been the most overlooked over human biology.
If we just pay attention to what nature has done in engineering this wonderland system that is the gut’s intestinal system, you start to wonder why the anatomy and the physiology is built in that way. And, you start to see, the amplified surface. That means we want to interface with the environment as much as we can.
Intestine: Interesting Facts

~20 ft long

~3,000 sf!!!
The **key function is to interface** with the environment and eventually exchange information, including molecules from the environment that comes in in a very tightly and coordinated and controlled manner.
And, the bottom line, the modern biology seems to suggest that the state of health or the state of disease is the combination between what we are—meaning what genetically makes us the way that we’re engineered—and the environment that’s around us.
And, the gut is the point of entry in which these two elements, they really meet. And, the way that, again, this exchange happens, it really is totally controlled by the permeability of the gut. They allow—if and when allowed—molecules to come through. And, on a specific genetic background, this brings us to the outcome of the overall picture of what, biologically, we are.
And, if everything goes fine and this traffic is tightly controlled, we stay in a state of health. But, if this tightly-controlled trafficking is, for whatever reason, jeopardized because of an infection, because of a change of the composition of bacteria in our gut--i.e. dysbiosis because we’re abusing antibiotics--because, again, we’re exposed to pollutants, chemicals, or genetically engineered foodstuffs, in other words, stuff that (will cause) dysfunction, we will pay a price.
“The state of health or the state of disease is the combination between what we are—meaning what genetically makes us the way that we’re engineered—and the environment that’s around us.”

Alessio Fasano, MD
So, with Intestinal Permeability, we have this uncontrolled trafficking of macromolecules. And, depending who we are, on what kind of genetic background we have, we can develop different problems.
For example, we can develop food allergies if we are skewed to develop allergies. We can develop autoimmune diseases. We can develop chronic inflammation that can lead to a stroke, Alzheimer’s, you name it, cancer. And, all this depends, again, on who we are genetically speaking, and what kind of environment is surrounding us.
So, I think that to make this in even more in simple terms, when we’re born, and, therefore, we have the entire genetic potentials, we are like a very precious single marble block. But, what is going to end up on this marble block in terms of what kind of sculpture, it depends on the environment. So, it can be an environment that you can become the painter Michelangelo’s David.
Or, you can be in a different environment and the outcome will not be so wonderful. And, that’s pretty much the story.
The result of the interface of our environment with our genes
Premise #6
What is the Impact of Intestinal Permeability?
Increased intestinal permeability is an early biological change that often precedes the onset of autoimmune diseases. Such increased permeability could be due to environmental factors (such as infection, toxic molecules, allergenic foods) that possibly initiate the (autoimmune) disease.
Mechanisms of Disease: the role of intestinal barrier function in the pathogenesis of gastrointestinal autoimmune diseases

Alessio Fasano* and Terez Shea-Donohue

The primary functions of the gastrointestinal tract have traditionally been perceived to be limited to the digestion and absorption of nutrients and electrolytes, and to water homeostasis. A more attentive analysis of the anatomic and functional arrangement of the gastrointestinal tract, however, suggests that another extremely important function of this organ is its ability to regulate the trafficking of macromolecules between the environment and the host through a barrier mechanism. Together with the gut-associated lymphoid tissue and the neuroendocrine network, the intestinal epithelial barrier with its intercellular tight junction, turn-like receptor

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A more attentive analysis of the anatomic and functional arrangement of the gastrointestinal tract, however, suggests that another extremely important function of this organ is its ability to regulate the trafficking of macromolecules between the environment and the host through a barrier mechanism.
The classical paradigm of autoimmune pathogenesis involving a specific genetic makeup and exposure to environmental triggers has been challenged by the addition of a third element: the loss of intestinal barrier function.
Together with the gut-associated lymphoid tissue and the neuroendocrine network, the intestinal epithelial barrier, with its intercellular tight junctions, controls the equilibrium between tolerance and immunity to non-self-antigens.
When the finely tuned trafficking of macromolecules is dysregulated in genetically susceptible individuals, both intestinal and extraintestinal autoimmune disorders can occur.
The autoimmune process can be arrested if the interplay between genes and environmental triggers is prevented by re-establishing intestinal barrier function.
Premise #7
How Might The Impact of Intestinal Permeability Present?
GUT INFLAMMATION

BRAIN INFLAMMATION

Uncontrolled inflammation drives neurodegeneration
Children with autism exhibited significantly elevated levels of IgG antibody to gliadin when compared with unrelated healthy controls or when compared with the combination of unaffected siblings and unrelated healthy controls (p<0.01).
Over the last decade there has been mounting evidence that supports a role for the GI tract and the enteric nervous system (ENS) in the pathogenesis of PD.
The GI system and the brain are directly linked anatomically with the dorsal motor nucleus of the vagus nerve, a brain region proposed to express Lewy pathology very early in the disease process.
Gut leakiness in patients with a genetic susceptibility to PD may be a pivotal early step promoting a pro-inflammatory/oxidative environment contributing to the initiation and/or progression of the PD process.
Indeed, it has been suggested that the GI tract might be a portal of entry for a putative PD pathogen, triggering pathological changes in the submucosal/myenteric neurons, which then spread through the vagus nerve to the medulla oblongata.
From there, pathological changes may move rostrally, ultimately resulting in the clinically-defining motor symptoms of PD when there is extensive involvement in the middle portion of the disease at the level of the midbrain substantia nigra.
An index of intestinal permeability, systemic exposure to intestinal bacterial products was determined by measuring plasma LPS binding protein (LBP). Lower levels of plasma LBP have been associated with increased exposure to gram negative bacteria.
Figure 4. Plasma LBP is significantly lower in PD patients. Plasma levels of LPS binding protein (LBP), an indirect measure of systemic endotoxin exposure, were determined for PD subjects and healthy controls as described in Materials and Methods. Values for plasma LBP in PD subjects were significantly lower than in healthy controls. Data are presented as means (ng/ml) ± SE. *p<0.05.
CASE STUDY #1

A Fatal Diagnosis
Addressing the autoimmune component of ALS


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A 44-year-old male was referred for a neurological opinion with a 6-month history of progressive right leg weakness, wasting and intermittent painful spasms of his right quadriceps. In the preceding month the patient had also noticed progressive weakness of his right arm and difficulty when writing. He had no sensory symptoms. The patient’s only past medical history of note was migraine with aura.
Examination revealed right-sided spastic hemiparesis with a pyramidal pattern of leg weakness associated with mild wasting of the right quadriceps. The patient had generalized bilateral hyperreflexia, sustained right ankle clonus and a right extensor plantar response. Results of cranial nerve, cerebellar and sensory examinations were normal.
Electromyography (EMG) of the masseter, biceps, first dorsal interosseous extensor digitorum communis and the vastus medialis muscles demonstrated widespread fasciculations, reduced recruitment of motor units, and frequent complex polyphasic waveforms. Fibrillation potentials were recorded in the right vastus lateralis.
The patient’s initial presentation:

- progressive motor syndrome with absence of sensory signs
- clinical evidence of upper and lower motor neuron degeneration
- electromyographic evidence of widespread acute denervation
- hyperintensity in the corticospinal tracts on MRI.

A dx of Amyotrophic Lateral Sclerosis (ALS).
His family hx revealed that a maternal aunt had CD, a sister had Crohn’s disease, and his maternal grandmother had MS.
MRI sequences revealed a region of hyperintensity along the course of the left corticospinal tract, arising from the subcortical white matter of the precentral gyrus and following the posterior limb of the internal capsule into the brainstem (Figure 1A).
Repeat neuroimaging 2 months later revealed more-extensive changes in the same pattern, with additional involvement of the opposite (right) subcortical region of the precentral gyrus (Figure 1B).
A is initial presentation, B is 2 months later
Routine blood tests revealed:

- a mild microcytic anemia
- ↓ levels of serum iron
- ↓ serum ferritin
- ↓ serum folate
Screening for celiac disease was prompted by the discovery of microcytic anemia with low serum iron and folate levels.
Blood tests revealed:
- elevated antiendomysial antibody
- duodenal biopsy demonstrated villous atrophy, crypt hyperplasia and increased intraepithelial lymphocytes (Marsh 3A), consistent with gluten-sensitive enteropathy (Celiac Disease)
The patient was started on a gluten-free diet approximately 7 months after the onset of his initial neurological symptoms. No drugs, including riluzole or other agents with neuroprotective potential were given.
9 months after initiation of treatment, the patient’s right arm function, had returned to normal. Improvement in the patient’s right leg function was noted, wasting was still present and there was some residual spasticity. He was now able to walk unaided, however, and his handwriting and ability to fasten buttons had returned to normal.
A case of celiac disease mimicking amyotrophic lateral sclerosis

Martin R Turner, Gurjit Chohan, Gerardine Quinn and Kevin Talbot*

SUMMARY

Background A 44 year-old male presented to a general neurology clinic with a 6-month history of progressive right-sided spastic hemiparesis without sensory symptoms or signs. The thigh muscle in the affected leg showed signs of wasting. The patient had a remote family history of celiac disease.

Investigations Neurological examination, neurophysiological studies, brain MRI scan, routine blood tests, duodenal biopsy, cerebrospinal fluid analysis including polymerase chain reaction test for JC virus DNA, serological testing for HIV and for the presence of serum antibodies to endomysium, gliadin and tissue transglutaminase.

Diagnosis Celiac disease with neurological involvement, mimicking Musculoskeletal and Neurological

(and) the hyperintensity of the left corticospinal tract is more confined, and the right motor cortical changes have resolved.

MR Turner is a Specialist Registrar in the Department of Neurology and G Quaghebeur is a Consultant Neuroradiologist in the Department of Neuroradiology at John Radcliffe Hospital, Oxford, UK. G Chohan is a Research Registrar at the National CJD Surveillance Unit, Edinburgh, UK. BCD Greenhill is a retired Consultant Neurollogist formerly of the Radcliffe Infirmary, Oxford, UK. M Hadjiyiannis is a Consultant Neuroradiologist at the Royal Hallamshire Hospital, Sheffield, UK. K Talbot is a Senior Clinical Lecturer and Consultant Neuroradiologist in the Department of Clinical Neurology at Oxford University, Oxford, UK.

Correspondence* University Department of Clinical Neurology, Level 8, West Wing, John Radcliffe Hospital, Oxford OX3 9DS, UK
bend.talbot@nhs.net

Received 5 April 2007; Accepted 2 July 2007

Electromyography (EMG) of the masseter, biops, first dorsal interosseous extensor digitorum
initial

2 months later

9 months after GFD
“Because ALS is a progressive and untreatable disease while CD is easily treatable, considering the latter as a cause of neurologic disorders in patients with ALS-like symptoms may be indicated”.

Am J Neuroradiol. 2010 May;31(5):880-1
Gluten sensitivity should be considered as a state of heightened immunologic responsiveness to ingested gluten proteins in genetically predisposed individuals. The brain seems to be particularly vulnerable.
Inside the cells of autoimmune targeted tissue, is a roaring fire
A Special Gift For You!

I’m going to send you 2 take-aways to help you implement this information!

Access to the 49 research articles used to create this presentation!

“Differentiating Gluten Related Disorders”

Both written by Dr. Thomas O’Bryan DC, CCN,
Get Out Your Phones
Text to:

58885
Text the message Gluten and your Email Address
Gluten

Your email address
Gluten
JaneDoe@gmail.com

Thank You
Dr. Tom O’Bryan
We will send you all 49 references I used to create the presentation you are hearing today.

- Use the references:
  - For your personal review to increase your knowledge about the connection between food sensitivities and autoimmunity
  - Share them with your patients, family, friends and Loved Ones
  - Share with your peers, your Study Groups, and begin the discussion with them as to how these research topics may relate to their Practices
Mechanisms by which a Gluten-Related Disorder may Impact on the Brain and Central Nervous System

7 Premises To Look At
Premise #1

Incompletely digested peptides of wheat (exorphins) modulate opioid receptor activity
Premise #2

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While many individual autoimmune diseases are rare, collectively they are thought to affect approximately 8 percent of the United States population – 24 million persons.
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Genes Control Function
Premise #4

Food Turns On and Turns OFF Our Genes
Premise #5

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Fig. 1. Autoimmune and inflammatory diseases in relation to celiac disease. *Strongest associations.
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GUT INFLAMMATION

BRAIN INFLAMMATION

Uncontrolled inflammation drives neurodegeneration
initial

2 months later

9 months after GFD
Take Care of Yourself
Make Sure to Tell those Important to You How Much You Love them
“Throughout your life the most profound influences on your health, vitality and function are not the Doctors you have visited or the drugs, surgery, or other therapies you have undertaken. The most profound influences are the cumulative effects of the decisions you make about your diet and lifestyle on the expression of your genes.”
“Thank You for Your Kind Attention”
Wishing you Sunrises of Beauty throughout your life