Dairy and Wheat

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Richard Harding

Newcastle, NSW Australia

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Wheat and the Distorted Views of William Davis

William Davis, a cardiologist, author of *Wheat Belly*, claims that "modern wheat is a perfect chronic poison". He claims that modern wheat causes diabetes, inflammation, heart disease and high blood pressure and that eliminating wheat will cure these problems.

Davis recommends the avoidance of foods such as corn, rice, quinoa, millet, buckwheat, beans and potatoes – even though they do not contain wheat or gluten.

Davis's inconsistencies would be amusing if they did not have such serious health consequences.

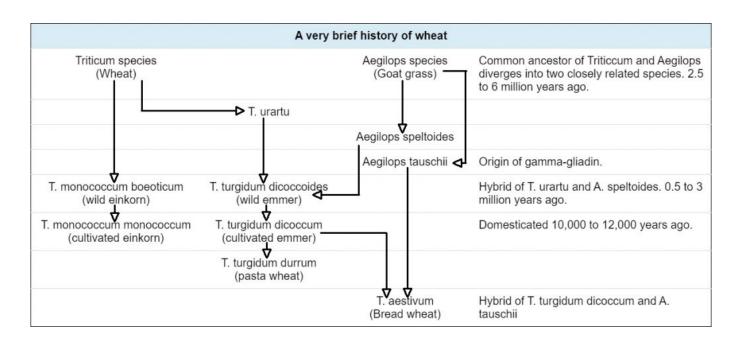
Wheat

Maize, wheat and rice are the most commonly grown cereal crops.

Common bread wheat, *Triticum aestivum*, has one of the most complex genomes known with 6 copies of each chromosome (that is, a hexaploid variety) and approximately 5 times the number of genes of humans.

The history of wheat evolution is complex.[1] [2]

Gamma-gliadin is found in goat grass from which the gliadins in modern wheat are derived.[3] Gliadin is present in all wheat, wild and cultivated.[4]



Davis's version of the origin of gliadin is several million years late.

Common or Bread Wheat

Triticum aestivum, a hexaploid species which is usually called "common" or "bread" wheat. It accounts for 95% of the wheat grown. Bread wheat is was probably formed 8,000-9,000 years ago in southeast Turkey from a hybrid of a variety of Durum wheat (Emmer), *Triticum turgidum*, and a closely related grass, Goat grass, *Aegilops tauschii*, that originally shared a common ancestor with wheat.

Pasta or Durum Wheat

Triticum turgidum var. durum, a tetraploid (four chromosomes) species is gown in the Mediterranean regions and adapted to hot, dry summer conditions. This accounts for most of the remaining 5%.

Einkorn

Cultivated Einkorn is a diploid (two sets of chromosomes, one from each parent) wheat variety, *Triticum monococcum var. monococcum*. The origins of wheat varieties are disputed.

South-east Turkey, the northern part of Tigris-Euphrates, the Balkans and Jordan near the Dead Sea is considered as a likely region. Wild Einkorn, *Triticum boeoticum*, is a different species.

Wild einkorn was found in modern Turkey, south Caucasian region, the Mediterranean region, southwestern Europe and the Balkans. It was one of the first cereals cultivated for food. Wild einkorn grain was harvested in the from 16,000-15,000 BC. Domestication of einkorn occurred around 7,500 BC (9,500 to 10,000 years ago). Einkorn continued to be grown widely until 4,000 BC before emmer wheat become more popular.

Einkorn cultivation continued in isolated regions to the current day where it is limited to small regions within France, India, Italy, Turkey and Yugoslavia.

Emmer

Modern Emmer wheat is also known as Farro, is a tetraploid (four set of chromosomes) variety, *Triticum turgidum var. dicoccum* – a variety of pasta wheat. Einkorn wheat and emmer wheat were the first crops domesticated in the grassland and woodland areas from Israel to Iran.

Spelt

Spelt (*Triticum aestivum var. spelta*) is considered to be a variety of common wheat. It appears to originate in the southern Caucasus Mountains region of Georgia, Armenia and Azerbaijan approximately 6,000 BC.[5]

Khorasan

Khorasan or Oriental wheat, *Triticum turgidum ssp. turanicum*, is similar to Emmer. Kamut is its registered name. It was first described in 1921 in the Persian province of Khorasan.[6]

It is generally considered an ancient relative of *Triticum turgidum var. durum.*[7]

Khorasan wheat has greater amount of gliadin than modern wheat.[8]

Gluten and Gliadin

Gluten is a group of proteins found in wheat, barley, rye and oats. Glutens are classified as either prolamins or glutelins. It is common not to include oats as a gluten source.

There is more of a similarity with wheat, barley and rye glutens than oats. This is why some people who react to wheat, barley and rye are able to eat oats. There is the added complication that oats may be contaminated with other cereals. The table classifying glutens is based on Schalk et al 2017.[9]

Gluten	Collective name for storage proteins found in cereals.			
Prolamins	Simple proteins - soluble in alcohol			
Gliadins	Wheat			
Secalins	Rye			
Hordeins	Barley			
Glutelins	Complex proteins - insoluble in water and alcohol			
Glutenins	Wheat			
Secalins	Rye			
Hordenins	Barley			
Non-Gluten Proteins				
Prolamins	Prolamins are plant storage proteins having a levels of the amino acids proline and glutamine amino acid content. They are found in cereal grains. Examples include corn (zein), sorghum (kafirin) and oats (avenin).			
Glutelins	Complex proteins - insoluble in water and alcohol. Includes oats (avenalins), corn (zeanins), rice (oryzenin), soy glutelins and millet glutelins.			

Gluten creates the elasticity of the kneaded dough which is important for making bread, pasta and other wheat-based products.

According to Davis:

It becomes clear that grains have always been a problematic calorie source, with problems amplified by changes introduced by genetics manipulations and agribusiness. Cutting-edge research, for example, has revealed that consumption of modern wheat is the first step in triggering autoimmune diseases such as type 1 diabetes, rheumatoid arthritis, and multiple sclerosis. As powerful as wheat elimination — or even better, all grains — can be, there is more to address as part of an effort to establish ideal health. [10]

Davis makes no support for his claims that wheat is associated with the diseases that he mentions other than anecdotal evidence. I have addressed the conditions that Davis raises in other posts.

- 1. Type 1 diabetes The problem with cow's milk
- 2. *Rheumatoid arthritis* Rheumatoid arthritis An autoimmune condition
- 3. Multiple sclerosis Multiple Sclerosis and Roy Swank

According to Davis,

Modern wheat is a perfect, chronic poison.

[Wheat today] is an 18-inch tall plant created by genetic research in the '60s and '70s. This thing has many new features nobody told you about, such as there's a new protein in this thing called gliadin. It's not gluten. I'm not addressing people with gluten sensitivities and celiac disease. I'm talking about everybody else because everybody else is susceptible to the gliadin protein that is an opiate. This thing binds into the opiate receptors in your brain and in most people stimulates appetite, such that we consume 440 more calories per day, 365 days per year. It's really a wheat issue.[11]

Davis makes the claim that "gliadin is not gluten". Incorrect. Gliadin is relatively simple protein that is a component of all wheat gluten.

Davis claims that gliadin is an opiate as it binds to opiate receptors. An opiate is a drug derived from opium. Binding to an opiate receptor does not make a substance an opiate. Davis relies on the paper[12] to support his claim that gliadin is an opiate. However, the paper states that "wheat gluten, alpha and beta caseinates and hemoglobin" bind with opiate receptors.

Alpha and beta caseinates are found in dairy and cheeses and hemoglobin is found in meat. Dairy, red and processed meat are acceptable foods, according to Davis.

A simpler explanation of the rising rate of obesity is that we are eating more and whilst the amount of fat as a proportion of food has dropped from an average of 40% to 33%, the total amount of fat has actually increased.

Even the so-called *Subway Diet* has been associated with significant weight loss. The core component of this diet was whole-grain Subway sandwiches without the cheese or mayonnaise.

Whole-grain bread is (or was in the 1960s) an important part of the diet in the Greece and the Mediterranean regions of Spain and France comprising of 30-40% of energy intake. Bread consumption was less in Italy because "they eat so much pasta".[13]

Approximately 5% of the population has some kind of issue consuming wheat or gluten products. For the remainder of the population, large-scale studies have shown that the consumption of grains provides a significant health benefit.

Glycemic Index and Whole-Grain Bread

Davis states that "Whole wheat bread has a GI of 72 which is higher than table sugar (GI = 59)". This is true but very misleading.

Glycemic index compares the blood glucose response over a two hour period obtained from 50 g of available carbohydrate (dietary fibre is not included) in the test food to the blood glucose response obtained from 50 g (12 teaspoons or 60 ml) of glucose. Sugar (sucrose) contains 50% of high GI glucose and 50% of low GI fructose.

It takes 5-6 slices of whole-grain bread to obtain 50 g of carbohydrate.

The measurements are taken with isolated food components. In real life, food is not eaten in isolation but in combination with other foods which changes the GI significantly and cannot be predicted from the GI of the individual foods. Fruits that are ripe have a higher GI. Finely ground foods (such as flour) to produce with smaller particle size results in increases to the GI.

Cooking and cooling changes the food's GI. Starch gelatinisation breaks the bonds of starch molecules when cooking with heat and water which makes the starch easier to digest. This process is reversed when the starch is cooled. Potatoes are a great example which is why hot cooked potatoes have a higher GI than potatoes cooled overnight.[14]

The ripeness of the food changes the GI. Removing fibre from a food and grinding food to produce smaller particle sizes makes the food more readily absorbed which increases the GI. Mixing foods together, which we do when we eat food, results in a glycemic response that cannot be predicted.

Comparing the glycemic index of 5-6 slices of whole-grain bread to 60 ml sucrose is not a valid comparison.

Ideal Diet - According to Davis

Davis claims that gliadin, which is found in wheat, is the culprit. However, he advocates eliminating not only wheat – both traditional and modern, but rye, barley and oats. If gliadin was the culprit, then oats, barley and rye would be safe to consume.

Davis fails to explain why the elimination of foods such as corn, rice, quinoa, millet, buckwheat, beans and potatoes is so important – after stating that gliadin is the monster that created our current health crisis.

According to Davis, dairy, eggs, ham, pepperoni and bacon wrapped chicken breasts are allowable foods – just stay away from "unhealthy" grains. Davis claims that, "pepperoni bread just looks unhealthy. After all, this 'bread' is really just made of ground nuts, coconut, cheese, eggs, and pepperoni".[15]

Examples of Wheat Belly "healthy" recipes include:

- Bacon Wrapped Chicken Breasts Stuffed with Spinach, Mushrooms and Roasted Red Peppers
- Italian Sausage Frittata with 8 eggs and hot chicken or turkey sausage meat ensure that the sausage casings are removed
- Pepperoni Bread made with pepperoni, eggs and cheese

Davis and The China Study

Davis claims that "Minger's analysis of the China study data showed that wheat ingestion is responsible for the heart disease". Denise Migner claims that Colin Campbell distorted data from China-Cornell-Oxford project ("The China Study") and that the data really showed that wheat is responsible for heart disease.[16]

Colin Campbell was a member of a team of researchers involved with The China Study. The epidemiologist and statistician was Sir Richard Peto from Oxford University. Junshi Chen of Beijing organised the survey teams that collected data including blood samples throughout China. Li Junyao was one of the authors of Chinese *Cancer Atlas Survey* – a comprehensive

survey of the causes of mortality of the Chinese that covered 96% of the population.

Denise Migner at the time of her revelations was a 23 year-old who majored in English. She is a Catholic school teacher with no training in statistics, mathematics or epidemiology. On reviewing the China Study data, she discovered serious flaws which escaped the attention of not only Campbell but also Peto, Li and Chen.

According to Mgner, [17], her intention 'is to highlight the weaknesses of "The China Study" and the potential errors in Campbell's interpretation of the original data'. It is not only Campbell's interpretation but Peto's, Li's and Chen's as well.

Migner, states that there exists a highly significant correlation between wheat flour consumption and two cardiovascular disease. She makes the observation that "none of these correlations appear to be tangled with any risk-heightening variables".

Campbell makes the following points.

- Higher wheat consumption was associated with a lower consumption of green vegetables as many of these people live in northern, arid and much colder regions
- Tuoli county data was omitted, which Migner called a "sin of omission". The data was omitted because the people of Tuoli county are nomadic which results in a high seasonal variation in meat consumption and fruit and vegetable consumption
- Consumption of wheat was associated with a lower serum levels of monounsaturated fats
- Consumption of wheat was associated with a greater body weight
- Consumption of wheat was associated with a higher serum levels of urea which is a marker of protein consumption

Migner's observation that there was no confounding factors is not valid.

Weight Reduction

Consumption of whole-grains has been shown to correlate to weight reduction, not weight gain.

An eight-year study of men showed, "an increase in whole-grain intake was inversely

associated with long-term weight gain".[18]

Similarly, a study of 74091 US female nurses aged 38–63 years in 1984 were assessed in 1984, 1986, 1990 and 1994. This showed that, "weight gain was inversely associated with the intake of high-fiber, whole-grain foods." That is, the more whole-grains consumed, lower the weight gain.[19]

That millions of non-sufferers banish gluten from their lives can be considered a public health farce. Dr Michael Greger

Related articles

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Footnotes

- 1. Brouns, F. J. P. H. et al. (2013) Does wheat make us fat and sick? *Journal of Cereal Science*. 58 (2), 209–215.
- Stallknecht, G. F. et al. (1996) 'Alternative Wheat Cereals as Food Grains: Einkorn, Emmer, Spelt, Kamut, and Triticale', in J Janick (ed.) *Progress in New Crops*. Alexandria VA: ASHS Press. pp. 156–170.
- 3. Goryunova, S. V. et al. (2012) Expansion of the gamma-gliadin gene family in Aegilops and Triticum. *BMC Evolutionary Biology*. 12 (1), 215
- 4. Rasheed, A. et al. (2018) The goat grass genome's role in wheat improvement. *Nature Plants*. 4 (2), 56–58.
- 5. Cubadda, R. & Marconi, E. (2002) 'Spelt wheat', in *Pseudocereals and Less Common Cereals*. Springer. pp. 153–175.
- 6. Grausgruber, H. et al. (2004) Khorasan wheat, Kamut and 'Pharaonenkorn': origin,

characteristics and potential.

- Colomba, M. S. & Gregorini, A. (2012) Are Ancient Durum Wheats Less Toxic to Celiac Patients? A Study of α-Gliadin from Graziella Ra and Kamut. *The Scientific World Journal*. 20121–8.
- Colomba, M. S. & Gregorini, A. (2012) Are Ancient Durum Wheats Less Toxic to Celiac Patients? A Study of α-Gliadin from Graziella Ra and Kamut. *The Scientific World Journal*. 20121–8.
- 9. Schalk, K. et al. (2017) Isolation and characterization of gluten protein types from wheat, rye, barley and oats for use as reference materials Karol Sestak (ed.). *PLOS ONE*. 12 (2), e0172819.
- Davis, W. (2017) Dr. William Davis | Cardiologist & Author of Wheat Belly Books [online]. Available from: https://www.wheatbellyblog.com/(Accessed 15 October 2017).
- Cochran, A. (2013) Modern wheat a 'perfect, chronic poison,' doctor says CBS News [online]. Available from: https://www.cbsnews.com/news/modern-wheat-a-perfect-chronic-poison-doctor-says/

(Accessed 26 June 2018).

- 12. Möller, N. P. et al. (2008) Bioactive peptides and proteins from foods: indication for health effects. *European Journal of Nutrition*. 47 (4), 171–182.
- 13. Keys, A. & Keys, M. (1975) *How to eat well and stay well the Mediterranean way*. Doubleday, Garden City, NY. p38
- 14. Fernandes, G. et al. (2005) Glycemic index of potatoes commonly consumed in North America. *Journal of the American Dietetic Association*. 105 (4), 557–562.
- 15. Davis, W. (2017) Dr. William Davis | Cardiologist & Author of Wheat Belly Books [online]. Available from: https://www.wheatbellyblog.com/ (Accessed 15 October 2017).
- 16. Davis, W. (2017) Dr. William Davis | Cardiologist & Author of Wheat Belly Books [online]. Available from: https://www.wheatbellyblog.com/ (Accessed 15 October 2017).
- 17. Migner, D. (2010) The China Study: Fact or Fallacy? | Denise Minger [online]. Available from: https://deniseminger.com/2010/07/07/the-china-study-fact-or-fallac/.
- Koh-Banerjee, P. et al. (2004) Changes in whole-grain, bran, and cereal fiber consumption in relation to 8-y weight gain among men. *The American Journal of Clinical Nutrition*. 80 (5), 1237–1245.
- 19. Liu, S. et al. (2003) Relation between changes in intakes of dietary fiber and grain products and changes in weight and development of obesity among middle-aged women. *The American Journal of Clinical Nutrition*. 78 (5), 920–927.

What is the Problem with Wheat and Gluten?

Gluten-free foods is a huge industry. It is estimated that approximately 2% of the US population, that has not been diagnosed with celiac disease, is consuming a gluten-free diet. [1]

The CSIRO reports that, " as many as 1 in 10 Australian adults, or approximately 1.8 million people, were currently avoiding or limiting their consumption of wheat-based products. Women were more likely to be avoiding wheat than men. The survey also revealed that over half (53%) of those who were avoiding wheat were also avoiding dairy-based foods."[2]

However, this survey was based on voluntary registration and is not a true reflection of the dietary habits of Australia.

There are 3 major issues that people may have from consuming wheat and gluten.

- **1.** Celiac disease: This is an autoimmune disease to gluten that affect less than 1% of the population.
- Wheat allergy: This is an adverse immunological reaction to wheat proteins that is indicated by high IgE antibodies. Prevalence is generally considered to be less than celiac disease.
- 3. Non-celiac gluten sensitivity (NCGS) is everyone else that is affect by wheat. There is no specific test and many have a wide range of other food intolerances. Common confounding foods are dairy, eggs and FODMAP foods. Some people are also intolerant to the nightshade family (tomatoes, capsicums, egg plants, potatoes).

FODMAP is an acronym for *Fermentable Oligo-, Di-, Mono-saccharides And Polyols*. FODMAP items includes:

- Short chain polymers of fructose (fructans) and galactose (galacto-oligosaccharides) raffinose, stachyose, inulin found in onion, garlic, beans, bananas, artichoke, wheat and rye.
- Lactose (milk sugar).
- Fructose fruit sugar found in many fruit with high concentrations in apples, pears, pineapples, high-fructose corn syrup, honey and molasses.
- Sugar alcohols (polyols) such as sorbitol, mannitol, xylitol and maltitol. Found naturally in fruits and berries and is added to commercial foods as a sweetener.

A 2017 study[3] reported that fructans was a more significant source of discomfort than gluten. Fructans and gluten are both found in wheat. Other sources of fructans are listed above. However, this involves removing healthy foods (many fruits, vegetables, beans, grains and mushrooms) whilst consuming detrimental items such as eggs, meat, poultry, fish and butter.

Eliminating FODMAP foods avoids the problem. It does not solve it.

There is a great deal of uncertainty in the prevalence of NCGS. The best estimates appear to be 2-3% of the American population.

Between 2004 and 2010, 5,896 patients were seen at the Center for Celiac Research, University of Maryland. The criteria for gluten sensitivity were noted for only 347 patients seen (6%).[4]

Since the centre specialises in celiac disease, then the prevalence in the general population is going to be much less.

An Italian study^[5] involved a group of 920 patients diagnosed with irritable bowel syndrome (IBS). This is characterised by abdominal pain or discomfort for 12 weeks over the past year. There is no diagnostic test for this condition.

This group is much more likely to have food sensitivities, allergies or intolerances. The

patients did have diagnostic test to show that they did not have CD or wheat allergy. They were subjected to a blinded food challenge. All of them went on a diet for four weeks that excluded wheat, cow's milk, tomato, eggs, chocolate, and any other foods that the patients had found to exacerbate their symptoms.

After four weeks they were given capsules containing either gluten or a non-gluten placebo. All the patients had 2 weeks of placebo and 2 weeks of gluten, but nobody knew what order they were getting them in. They recorded their symptoms. It was found that 276 of the original 920 IBS patients (30%) had improvement in their symptoms on the elimination diet and a return of symptoms (pain, bloating, change in stool consistency) when taking the gluten capsule.

That is, only 30% of the patients suffering from irritable bowel syndrome were diagnosed to be sensitive to gluten.

Researchers then tested these 276 wheat-sensitive patients to discover if they were also sensitive to other foods. 75% of these patients that were sensitive to gluten were also sensitive to cow's milk. These patients were also sensitive to eggs (120 cases – 43%) and tomatoes (112 cases – 41%). Fewer than 10% of those with IBS were sensitive to wheat only.

Popular commentators frequently misrepresent this finding to state that 30% of the general population are sensitive to wheat or gluten.

An Australian trial[6] [7] consisting of 34 patients was performed over a six week period.

The trial was a double-blind placebo controlled study—the patients did not know if they were receiving gluten-free or gluten in the diet. The gluten foods were free of FODMAP components.

The primary outcome was the proportion of patients answering "NO" on more than 50% of the occasions to the question, "Were your symptoms adequately controlled?"

32% of the patients reported an improvement, even when they were unknowingly consuming a gluten diet. 40% of the patients reported no improvement, even when they were

consuming a gluten-free diet.

William Davis is largely responsible for the low-wheat, low-gluten diets with the publication of his book *Wheat Belly*.[8] In this book he states that we live in a 'whole grain world'. This simply is not true.

The US guidelines for whole grains is the equivalent of four slices of whole-grain bread for males and three slices for females. 99% of the US population do not meet these guidelines. Only 10% of grains consumed are whole grains.[9]

Much of the processed grain consumed comes from yeast breads, grain-based desserts, pizza bases and Mexican mixed dishes.

When people give up gluten foods, they are also giving up the high-fat foods listed above, which is going to be of great benefit. When we hear stories people are feeling much better after eliminating gluten, they are also avoiding pizza (dairy, fat, and salt), highly-refined white bread which is spread with butter or margarine, cookies, cakes, desserts (fat, sugar, dairy) and pasta with its fat and cheese.

Many people who believe that they are eating a gluten-free diet are not. For celiac patients, even a very small amount of gluten (20 parts per million) can have serious consequences.

Davis claims that:

There are currently 22,000-25,000 varieties of wheat, all which are the result of human intervention, and these varieties are hundreds, perhaps thousands of genes apart from einkorn bred naturally. Prior to 1940, there had been little change in wheat flour for more than 200 years, but since then numerous changes in the wheat protein structure have cause severe problems for human immune responses.

Davis claims that gluten is the problem, not wheat which means that all gluten grains are problematic. If so, then the variety of wheat it is irrelevant – traditional and modern wheat, rye, barley and possibly oats will cause problems.

Davis greatly exaggerates the extant of changes to wheat genetics since 1940s.[10]

Whilst most discussion centres on gluten, wheat is relatively low on the list of foods likely to cause allergies. Below is a list of foods in order of prevalence of allergies.[11]

- Cow's milk
- Egg
- Shellfish
- Peanuts
- Fish
- Tree nuts
- Fruits
- Wheat

There is an increasing awareness of the importance of gut flora and its role in health. [12] [13] [14] [15]

People who embark on gluten-free diets frequently have significantly impaired health outcomes due to changes in gut bacteria.

Microbes in the intestines are essential for the breakdown of complex carbohydrates, the production of short chain fatty acids and synthesis of vitamins. More than 1000 different species have been identified. Despite the vast number of bacteria species and people, there are only two types of bacteriological ecosystems in the gut (enterotypes) – those dominated by *Prevotella* genus bacteria and those by *Bacteroides* genera. Both *Bacteroides* and *Prevotella* belong to Bacteroidetes phylum. Enterotypes were strongly associated with long-term diets, particularly protein and animal fat (*Bacteroides*) versus carbohydrates (*Prevotella*). Microbiome composition changed within 24 hours of initiating a high-fat/low-fiber

or low-fat/high-fiber diet. However, it takes a longer period of time to change the enterotype from one state to the other.[16]

A paper published in 2010, showed that healthy gut bacteria decreased and the numbers of unhealthy bacteria increased following the reductions in the intake of polysaccharides after following a gluten-free diet.[17]

This paper also reports that nutritional deficiencies and health complications frequently occur compared with non-gluten-free diet subjects and this could be explained by changes to the gut bacteria. Inflammation markers were also significantly affected, for the worse.

Similar results were also obtained from a 2009 study. In addition to the changes to the intestinal bacteria which is noted in the previous study, this study showed that people on a gluten-free diet consume a less dietary fibre and polysaccharides which affect intestinal bacteria. The average amount of polysaccharides consumed was halved (from 117 g/d to 63 g/d) and dietary fibre from 20 g/d to 18 g/d. The recommended amount of fibre is set at a very conservative 25 g/day.[18]

It is apparent that many people are consuming gluten-free diets that are not only unnecessarily restrictive but are associated with significant health risks.

If it is essential to consume a gluten-free, grain-free diet then it must be done with awareness to avoid the very real dangers associated with such a diet.

Changing one aspect of our diet frequently results in multiple changes and it is difficult to know what changes are associated with the benefits.

As a generalisation, the best diets are whole-food, plant-based diets, which are not only optimal for our health are also the best for the environment and for the animals we share the earth with.

Related articles

What is the Problem with Wheat? Wheat and Inflammation Impact of a Gluten-Free Diet Wheat and the Distorted Views of William Davis

Footnotes

- 1. Kim, H. et al. (2016) Time trends in the prevalence of celiac disease and gluten-free diet in the US population: results from the National Health and Nutrition Examination Surveys 2009-2014. *JAMA Internal Medicine*. 176 (11), 1716–1717.
- Golley, S. (2015) Understanding the choice to go gluten- or wheat-free CSIRO blog [online]. Available from: https://blog.csiro.au/understanding-the-choice-to-go-gluten-or-wheat-free/ (Accessed 30 November 2017).
- 3. Skodje, G. I. et al. (2017) Fructan, Rather Than Gluten, Induces Symptoms in Patients With Self-reported Non-celiac Gluten Sensitivity. *Gastroenterology*.
- 4. Sapone, A. et al. (2012) Spectrum of gluten-related disorders: consensus on new nomenclature and classification. *BMC Medicine*. 10 (1), 1.
- 5. Carroccio, A. et al. (2012) Non-Celiac Wheat Sensitivity Diagnosed by Double-Blind Placebo-Controlled Challenge: Exploring a New Clinical Entity. *American Journal of Gastroenterology*. 107 (12), 1898–1906.
- Biesiekierski, J. R. et al. (2011) Gluten Causes Gastrointestinal Symptoms in Subjects Without Celiac Disease: A Double-Blind Randomized Placebo-Controlled Trial. *The American Journal Of Gastroenterology*. 106508.
- 7. Ferch, C. C. & Chey, W. D. (2012) Irritable bowel syndrome and gluten sensitivity without celiac disease: separating the wheat from the chaff. *Gastroenterology*. 142 (3), 664–666.
- 8. Davis, W. (2011) Wheat Belly. Rodale Press, Inc.
- 9. Krebs-Smith, S. M. et al. (2010) Americans Do Not Meet Federal Dietary Recommendations. *Journal of Nutrition*. 140 (10), 1832–1838.
- Stallknecht, G. F. et al. (1996) 'Alternative Wheat Cereals as Food Grains: Einkorn, Emmer, Spelt, Kamut, and Triticale', in J Janick (ed.) *Progress in New Crops*. Alexandria VA: ASHS Press. pp. 156–170.

- 11. Campbell, Thomas. *The China Study Solution: The Simple Way to Lose Weight and Reverse Illness, Using a Whole-Food, Plant-Based Diet* (p. 84). Rodale Books.
- 12. Brown, K. et al. (2012) Diet-Induced Dysbiosis of the Intestinal Microbiota and the Effects on Immunity and Disease. Nutrients. 4 (12), 1095–1119.
- 13. Power, S. E. et al. (2014) Intestinal microbiota, diet and health. British Journal of Nutrition. 111 (03), 387–402.
- 14. Tang, W. H. W. & Hazen, S. L. (2014) The contributory role of gut microbiota in cardiovascular disease. Journal of Clinical Investigation. 124 (10), 4204–4211.
- 15. Wang, Z. et al. (2011) Gut flora metabolism of phosphatidylcholine promotes cardiovascular disease. Nature. 472 (7341), 57–63.
- 16. Wu, G. D. et al. (2011) Linking long-term dietary patterns with gut microbial enterotypes. *Science*. 334 (6052), 105–108.
- 17. Sanz, Y. (2010) Effects of a gluten-free diet on gut microbiota and immune function in healthy adult humans. *Gut Microbes*. 1 (3), 135–137.
- 18. De Palma, G. et al. (2009) Effects of a gluten-free diet on gut microbiota and immune function in healthy adult human subjects. *British Journal of Nutrition*. 102 (08), 1154.

Wheat and Inflammation

William Davis is largely responsible for the low-wheat, low-gluten diets with the publication of his book *Wheat Belly*.[1] In this book he states that we live in a 'whole grain world'. This simply is not true.

The US guidelines for whole grains is the equivalent of four slices of whole-grain bread for males and three slices for females. 99% of the US population do not meet these guidelines. Only 10% of grains consumed are whole grains.[2]

Much of the processed grain consumed comes from yeast breads, grain-based desserts, pizza bases and Mexican mixed dishes.

Popular commentators such as William Davis claims that wheat causes most of the diseases of modern civilisation and that it leads to inflammation. However, whole-grain wheat has consistently been shown to reduce inflammation in patients who are NOT suffering from CD.

Whole-grain consumption resulted in a lower PAI-1 (Plasminogen activator inhibitor-1) over a 8 week period. There was also a reduction of plasma tumor necrosis factor- α (TNF- α) after 8 weeks and increased interleukin (IL)-10, a marker of immune response, after 4 weeks.[3]

PAI-1 is a marker for cardiovascular disease, metabolic syndrome, a number of cancers, thrombosis (blood clots) and obesity. It is associated with an increase in fibrinogen.[4] [5] [6] [7]

A study examined the consumption of whole grains and dietary fiber among among 902 diabetic women in the Nurses' Health Study. Intakes of whole grains and bran were significantly associated with a decrease of the inflammation markers C-reactive protein (CRP) and tumor necrosis factor- α receptor 2 (TNF-R2). [8]

A Canadian study showed that whole grain consumption is related to lower levels of PAI-1 and C-reactive protein (CRP) inflammation markers.[9]

According to Esposito & Giugliano, "fiber content of a high-carbohydrate meal may influence plasma concentrations of adiponectin and interleukin 18 (IL-18): the greater the quantity of fiber in the load, the greater the inhibition of plasma IL-18 and the stimulation of adiponectin.

IL-18 is a potent pro-inflammatory cytokine that may be important in the process of plaque destabilization."

Adiponectin is an insulin-sensitizing adipocytokine with anti-inflammatory properties.[10]

A study published in 2006 showed a significant inverse trend between whole-grain intake and the metabolic syndrome and mortality from cardiovascular disease. This study used data from the Nurses Study.

Fasting glucose concentrations and body mass index decreased across increasing quartile categories of whole-grain intake independent of other factors. An intake of refined grain was associated with a higher fasting glucose concentrations and a higher prevalence of the metabolic syndrome.

The average whole-grain intake of the lowest quartile was a measly 0.3 servings a day with the average of the highest quartile being 2.9 servings a day.[11]

A research team lead by Alessio Fasano at the University of Maryland School of Medicine published an article in 2000 that announced the discovery of Zonulin, a protein which "induces tight junction disassembly and a subsequent increase in intestinal permeability". [12]

Increased permeability is "an early event in coeliac disease and not just a consequence of chronic intestinal inflammation."[13]

Zonulin is present in both coeliac disease and non-ceolic disease subjects but the "threshold of intestinal permeability upon gliadin exposure is not reached in non-coeliac disease".[14]

According to Dr, Michael Greger,

When I see books, websites, articles, and blogs parroting claims like "grains are inflammatory— even whole grains," I can't help but wonder what alternate dimension the authors call home.[15]

Dr. John McDougall writes,

Wheat Belly and Grain Brain take a backdoor approach to the Atkins lowcarbohydrate method. As the titles of these books suggest, wheat causes a big belly and grains damage the brain. Within their pages you learn that all starchy foods, including rice, corn, and potatoes—the traditional foods consumed by billions of people throughout human history—are now unhealthy and must be minimized or, better yet, avoided altogether.

If you believe these authors, then what is left to eat in order to meet your energy requirements? Meat, dairy, fish, and eggs. In order for the authors of these two books to pull off the monumental task of luring otherwise intelligent people into inherently dangerous diet plans, they have had to (1) ignore the bulk of the science, (2) exaggerate the truth, and (3) make false associations. [16]

Related articles

What is the Problem with Wheat? Wheat and Inflammation Impact of a Gluten-Free Diet Wheat and the Distorted Views of William Davis

Footnotes

- 1. Davis, W. (2011) Wheat Belly. Rodale Press, Inc.
- 2. Krebs-Smith, S. M. et al. (2010) Americans Do Not Meet Federal Dietary Recommendations. *Journal of Nutrition*. 140 (10), 1832–1838.
- 3. Vitaglione, P. et al. (2015) Whole-grain wheat consumption reduces inflammation in a randomized controlled trial on overweight and obese subjects with unhealthy dietary and lifestyle behaviors: role of polyphenols bound to cereal dietary fiber. *American Journal of Clinical Nutrition*. 101 (2), 251–261
- 4. Kohler, H. P. & Grant, P. J. (2000) Plasminogen-Activator Inhibitor Type 1 and Coronary Artery Disease Franklin H. Epstein (ed.). *New England Journal of Medicine*. 342 (24), 1792–1801.
- Juhan-Vague, I. et al. (1991) Increased plasma plasminogen activator inhibitor 1 levels. A possible link between insulin resistance and atherothrombosis. *Diabetologia*. 34 (7), 457–462.
- 6. Lijnen, H. R. (2005) Pleiotropic functions of plasminogen activator inhibitor-1. *Journal of Thrombosis and Haemostasis*. 3 (1), 35–45.
- 7. Huang, F. et al. (2012) Plasminogen activator inhibitor-1, fibrinogen, and lung function in adolescents with asthma and obesity. *Endocrine research*. 37 (3), 135–144.
- 8. Qi, L. et al. (2006) Whole-grain, bran, and cereal fiber intakes and markers of systemic inflammation in diabetic women. *Diabetes Care*. 29 (2), 207–211
- 9. Masters, R. C. et al. (2010) Whole and Refined Grain Intakes Are Related to Inflammatory Protein Concentrations in Human Plasma. *Journal of Nutrition*. 140 (3), 587–594.
- 10. Esposito, K. & Giugliano, D. (2006) Whole-grain intake cools down inflammation. *The American Journal of Clinical Nutrition*. 83 (6), 1440
- Sahyoun, N. R. et al. (2006) Whole-grain intake is inversely associated with the metabolic syndrome and mortality in older adults. *The American Journal of Clinical Nutrition*. 83 (1), 124–131.
- 12. Fasano, A. et al. (2000) Zonulin, a newly discovered modulator of intestinal permeability, and its expression in coeliac disease. *The Lancet*. 355 (9214), 1518–1519.
- Drago, S. et al. (2006) Gliadin, zonulin and gut permeability: Effects on celiac and nonceliac intestinal mucosa and intestinal cell lines. *Scandinavian Journal of Gastroenterology*. 41 (4), 408–419.
- 14. Drago, S. et al. (2006) Gliadin, zonulin and gut permeability: Effects on celiac and non-

celiac intestinal mucosa and intestinal cell lines. *Scandinavian Journal of Gastroenterology*. 41 (4), 408–419.

- 15. Greger, Dr Michael; Greger, Michael; Stone, Gene. *How Not To Die: Discover the foods scientifically proven to prevent and reverse disease*. Pan Macmillan UK. Kindle Edition.
- McDougall, J. (2014) The Smoke and Mirrors behind Wheat Belly and Grain Brain [online]. Available from: https://www.drmcdougall.com/misc/2014nl/jan/smoke.htm (Accessed 15 November 2016).

Impact of Gluten-free Diets

Prevalence and Motivations

An 2015 Australian study[1] examined the prevalence and motivations of those choosing a gluten-free diet in Australia.

Some highlights of the survey were:

- Approximately 4 times more women than men chose a gluten-free diet.
- 53% on a gluten-free diet also avoided dairy
- For 56%, the primary reason for a gluten free diet was based on symptoms, mainly gastrointestinal
- 16% chose a gluten-free diet based on medical diagnostic advice
- Those choosing a gluten-free diet had a greater receptiveness to complementary medicine than in the general population

For those who based their decision up symptoms, the top reasons with a incidence of greater than 10% are listed below.

Symptom	Incidence (%)
Bloating or wind	79
Stomach discomfort or cramps	55
Constipation	31
Diarrhoea	21
Heartburn of indigeation	16
Skin problems	14
Mucus buildup	13
Headaches	10

A list of the most common allergenic foods is listed below. Wheat does not rate highly on the list of allergenic foods.[2]

There can be variations in such lists due to how the data is collected and the regions that are involved. Apple and carrot can rate highly if fruit and vegetables are separated. This list is consistent with other allergenic list. Dairy, especially cow's milk, is particularly allergenic.

Food Item	Children	Adults	Total
Milk	2.23	1.89	1.97
Shellfish	0.55	1.91	1.60
Other	1.32	1.67	1.59
Fruits	1.14	1.61	1.50
Tree nut	1.73	1.07	1.22
Vegetables	0.45	1.29	1.10
Peanut	1.77	0.78	1.00
Egg	1.23	0.67	0.80
Wheat	0.45	0.86	0.77
Fish	0.18	0.60	0.51
Soy	0.32	0.16	0.20
Sesame	0.23	0.07	0.10
All foods	7.14	8.34	8.07

Comparison Standard Diet and Gluten-Fee Diet

There is a substantial difference between a standard western diet and a gluten-free diet. If a gluten-free diet is no warranted, a gluten-free diet may have unintended health consequences that are not beneficial as well as creating an additional inconvenience.

Consumption of complex carbohydrates (polysaccharides) and dietary fibre can be significantly less.

Impact of Low-Fibre Intake

The changes to the complex carbohydrate and dietary fibre has a significant impact on intestinal bacteria.[3]

Beneficial bacteria *Faecalibacterium prausnitzii*, *Bifidobacterium and Lactobacillus* was decreased as a result of the gluten-free diet.

Enterobacteriaceae which includes the dangerous Escherichia coli, Klebsiella, Salmonella, Shigella and Yersinia pestis was increased.

Satiety

With the removal of complex carbohydrates, satiety can be reduced. Foods that are filling are: high in starches, complex carbohydrates and dietary fibre; high volume foods; and have low energy density.

Also tasty foods flavored with herbs, spices and other condiments are more satisfying. A list of foods and their satiety rating according to *A satiety index of common foods* are listed.[4]

The comparison is made with an equal amount of energy (1000 kJ or 240 kCalories) of food compared with white bread.

- Potatoes, boiled 323%
- Porridge, Oatmeal 209%
- Apples 197%
- Brown pasta 188%
- Beef 176%
- Baked beans 168%
- Grain bread 154%
- Cheese 146%
- Brown rice 132%
- Eggs 150%
- Bananas 118%

Immune System Response

The reduction of beneficial intestinal bacteria compromises the response to the immune system inflammatory hormones TNF- α , interferon- γ , IL-10 and IL-8.[5]

Solutions

It is certainly possible to have a healthy, gluten free diet. Legumes are an important component of a gluten-free diet (and any other diet).[6]

Carbohydrates

The pseudo-cereals (amaranth, quinoa, buckwheat), beans (particularly lentils) and nuts provide a source of dietary fibre and complex carbohydrates. Particular attention should be given to increasing the amount of legumes (beans and peas).

Protein

There is a tendency to increase the amount of animal protein in gluten-free diets due to the consumption of meat, milk and dairy products, eggs and fish. This contributes to a reduction of beneficial bacteria.

Legumes, nuts, seeds, pseudo-cereals and gluten-free cereals including millet and teff are sources of protein.

Phytonutrients

Cereals are very high in phytonutrients which can more than adequately replaced by leafy green vegetables, millet, pseudo-grains (buckwheat and quinoa), berries (particularly plums and any other dark fruits), herbs and spices.

These foods are also high in vitamins and minerals.

Related articles

What is the Problem with Wheat? Wheat and Inflammation Impact of a Gluten-Free Diet Wheat and the Distorted Views of William Davis

Footnotes

- 1. Golley, S. et al. (2015) Motivations for avoiding wheat consumption in Australia: results from a population survey. *Public Health Nutrition*. 18 (3), 490–499.
- 2. Soller, L. et al. (2012) Overall prevalence of self-reported food allergy in Canada. *Journal of Allergy and Clinical Immunology*. 130 (4), 986–988.
- 3. De Palma, G. et al. (2009) Effects of a gluten-free diet on gut microbiota and immune function in healthy adult human subjects. *British Journal of Nutrition*. 102 (08), 1154.
- 4. Holt, S. H. et al. (1995) A satiety index of common foods. *European Journal of Clinical Nutrition*. 49 (9), 675–690.
- 5. De Palma, G. et al. (2009) Effects of a gluten-free diet on gut microbiota and immune function in healthy adult human subjects. *British Journal of Nutrition*. 102 (08), 1154.
- Saturni, L. et al. (2010) The Gluten-Free Diet: Safety and Nutritional Quality. *Nutrients*. 2 (1), 16–34.

The Gluten Lie: And Other Myths About What We Eat

Alan Levinovitz is an assistant professor of Religious Studies at James Madison University, Virginia.

His book, *The Gluten Lie: And Other Myths About What We Eat*[1], "takes on bestselling physicians and dietitians, exposing the myths behind how we come to believe which foods are good and which are bad—and pointing the way to a truly healthful life, free from the anxiety of what we eat."

Whilst the book was fascinating in describing how easily it is for society to be deluded about food issues, the book adds several misconceptions of its own.

Gluten and wheat is a serious health problem for a small percentage of people, but for the rest (approximately 92% – 95%) avoiding wheat and gluten has detrimental consequences.

Levinovitz quotes Dr Peter Gibson of Monash University (Melbourne, Australia) who claims FODMAP items are a much greater problem.[2]

FODMAP is an acronym for *Fermentable Oligo-, Di-, Mono-saccharides And Polyols*. FODMAP items includes foods such as onion, garlic, beans, bananas, artichoke, wheat, lactose (milk sugar), fruit sugar found in many fruit with high concentrations in apples, pears, pineapples, high-fructose corn syrup, honey and molasses as well as sugar alcohols (polyols) such as sorbitol, mannitol, xylitol and maltitol.

Items allowed on the FODMAP diet include potential problem foods such as hard cheeses (which apparently also includes brie and camembert), butter, meat, poultry, seafood, coconut milk and eggs. These foods have a detrimental impact on our intestinal microflora.

Beneficial foods such mushrooms, fruits, legumes (peas and beans) and cabbage family (broccoli, Brussel sprouts, cabbage, cauliflower) are avoided on a FODMAP diet. A highcarbohydrate, low-fat diet is most beneficial to our intestinal microflora and the FODMAP diet, rather than solving our intestinal microflora issues, intensifies it. Levinovitz contends that that medical profession's issues with fats and saturated fats fall into the same misguided category of undeservedly maligned foods. He quotes Aseem Malhotra as an authority. Malhotra is not a credible source of information.

I have written about Malhotra's shortcomings at The Pioppi Diet.

Levinovitz assertion is that "everything in moderation" is the dietary answer without offering any evidence why this is true and contends that "fake cheese is his one food taboo" – which is understandable.

As Oscar Wilde wrote,

Moderation is a fatal thing.[3]

The method of citing references is clumsy and does not easily allow for fact checking.

Related Articles

Moderation is a Fatal Thing

Seventh-day Adventists and Health – Moderation does not work Taiwanese Buddhist Study – Moderation does not work What is the problem with wheat and gluten? The nature of food allergies Eggs and the benefits of choline

Footnotes

- 1. Levinovitz, A. (2015) The Gluten Lie and other myths about what you eat. Nero.
- 2. Gibson, P. R. & Shepherd, S. J. (2010) Evidence-based dietary management of functional gastrointestinal symptoms: The FODMAP approach. *Journal of Gastroenterology and Hepatology*. 25 (2), 252–258.
- 3. Wilde, O. (1893) A Woman of No Importance.

The Problem With Cow's Milk

Skin and intestinal reactions to cow's milk was described by Hippocrates (460-370 B.C.) and Galen of Pergamum (130-210 AD). Both are ancient Greek physicians so there has been an awareness of problems with cow's milk for a considerable period of time.

Cow's milk is the most common form of allergic reactions, although the actual prevalence is disputed.

Digestion of Cow's Milk

In cattle, digestion of casein proteins is initiated by rennin, which produces a curd. In humans, since rennin is not present, curds are not formed. The precipitate formed from human milk is much finer and softer and easier to digest.

Casein from cows binds to bile acids which limits the ability to make fatty acids soluble. Calcium, iron, zinc magnesium and magnesium bind to casein which possibly limits their availability.

The net protein utilization of whey protein, irrespective of source, is superior to that of casein, 95% compared to 80%.

A significant amount of effort has been made in "humanising" cow's milk infant formula. The amino acid profile of pre-term newborn babies fed a whey-predominant formula more closely resemble those of breast-fed infants.[1]

Babies fed a casein-predominant formula have:

- higher levels of blood urea nitrogen
- higher levels of amino acids phenylalanine, methionine, tyrosine
- lower levels of taurine and cystine
- higher levels of ammonia
- lower serum pH blood is more acidic

Gastroenteritis

Since the 1950s, it has been known that "that breast-fed babies are relatively resistant to gastroenteritis". Breast-fed babies have greater amounts of *Lactobacillus* due to higher levels of lactose, low protein and low phosphate content.[2]

Flora of the breast-fed infant is dominated by *Bifidobacterium* and *Lactobacillus* which produce lactic acid and are beneficial. *Staphylococcus* bacteria is also higher in breast-fed babies which can be detrimental. *Lactobacillus* and *Bifidobacterium* bacteria inhibit the growth of many pathogenic bacteria such as *Staphylococcus*, *Salmonella*, *Yersinia*, *Clostridium*, *Listeria* species and *Escherichia coli*.

A decrease in *Bifidobacterium* bacteria in human intestine indicates an unhealthy state.

The flora of casein-based formula-fed infants contains more *Bacteroides*, *Clostridium* species as well as Enterobacteriaceae. Enterobacteriaceae is a large family of many mostly harmless bacteria but does include common pathogens such as *Salmonella* and *Escherichia coli* which are associated with gastroenteritis and other intestinal problems.[3] [4]

Bifidobacterium and *Lactobacillus* and other bacteria produce B vitamins and vitamin K. They ferment non-digestible carbohydrates (dietary fibre) into short-chained fatty acids (acetate, propionate and butyrate) which are essential to our well-being.

Other intestinal bacteria produce substances that are harmful to the host, such as putrefactive products, toxins and carcinogenic substances. When harmful bacteria dominate in the intestines, essential nutrients are not produced and the level of harmful substances rises.

Bovine casein promotes the growth of disease causing bacteria. Neonatal necrotizing enterocolitis, where a portion of the bowel dies) is much more evident in formula-fed infants.

Inflammation and Allergy

Casein has considerable inflammatory characteristics.[5]

The following table the prevalence of potential allergenic symptoms with cow's milk proteins.[6] These values are taken from a number of different studies and show a wide range of prevalence.

The homology represents the amount of similarity with the proteins in human milk compared with cow's milk.[7]

Component	Prevalence of Allergies %	Notes
Caesin		
• αS1-casein	65-100	Trace amounts only in human milk
• αS2-casein		
• β-casein	35-44	47% similarity (homology) with human milk
• к-casein	35-41	Low homology with human milk
Whey		
• α -lactoalbumin	0-67	72% homology with human milk
• β-lactoglobulin	13-62	Absent in humans
Immunoglobulins	12-36	
Serum albumin	4.9-5.1	80% homology with human milk
Lactoferrin	0-35	Much higher concentration in human milk
• Lysozyme		

Immune Response to Cow's Milk Proteins

Antibodies are produced when our bodies recognise a protein as foreign. Antibodies to cow's milk proteins can be detected at birth and levels generally increase until weaning. Antibodies to β -lactoglobulin and α -casein were measured in the blood of formula-fed infants of 31 to 41 weeks of gestation.[8]

Antibodies to bovine serum albumin (BSA), casein and to β -lactoglobulin found in the control group of "normal" children and those with type 1 diabetes in a Finnish study. The diabetic children had a significant increase in anti-BSA antibodies.[9] [10]

Serum antibodies to five major proteins of cow's milk: casein, bovine serum albumin (BSA), α -lactalbumin, β -lactoglobulin A, and β -lactoglobulin B, were compared in patients with Crohn's disease and 20 matched controls. IgG and IgM antibodies to cow's milk proteins were significantly elevated in patients with inflammatory bowel disease as compared to controls.[11]

β-Casomorphins

Casomorphins are formed from casein when mammal milk is digested. They bind to opiate receptors which results in calming the infant and most likely assists in bonding with the mother.

A 2009 paper[12] studying the effects of breast feeding on motor development, showed that elevated levels of antibodies to bovine β -casomorphins-7 was associated with a "a risk factor for delay in psychomotor development and other diseases such as autism". The study concluded that "breast feeding has an advantage over artificial feeding for infants' development during the first year of life".

Bovine β -casomorphins has also been associated with apnea (the suspension of breathing)

and sudden infant death syndrome (SIDS).[13]

Recently, significantly higher levels of bovine β -casomorphins have been detected in the urine of children that have impaired early child development. Their hypothesis is that casomorphins interact with opioid and serotonin receptors and thereby "setting the stage for autistic disorders".[14]

Lactose Intolerance

Milk is toxic to approximately 75% of the world's population.[15] Adults do not produce the enzyme lactase which is required to break down lactose (milk sugar). Children have this ability but the ability is lost by 7 or 8 years.[16]

The production of cheese and yogurt around 9,000 BCE in the Middle East, allowed adults to consume dairy products without the ill-effects of bloating and diarrhoea.

By approximately 5,500 BCE, herders reached central Europe, a genetic mutation allowed lactase to be produced into adulthood, allowing milk to be consumed without discomfort.

As well as northern Europe, western Africa (Algeria, Mauritania, Senegal, Guinea), Arabia, Pakistan & Gujarati have independently developed populations that are lactose tolerant as adults.

Lactose comprises of two simple sugars: glucose and galactose.

Research[17] funded by The National Dairy Promotion and Research Board, and the US Department of Agriculture, tested a treatment for lactose intolerance by feeding patients with *Lactobacillus acidophilus*, which is found in yogurt. The study failed to show that *Lactobacillus* had any benefit.

Since galactose is routinely used by researchers to promote aging in animal experiments, it is

apparent that evolution (or nature, if you prefer) has a good reason to ensure that our consumption of galactose is limited.

Type 1 Diabetes

In 1990s, Finland had the highest incidence of diabetes and cow's milk consumption in the world.

In Finland, researchers compared levels of incompletely digested cow's milk protein (Bovine Serum Albumin – BSA) in 142 diabetic children. Levels of IgG anti-BSA antibodies were higher than 3.55 RFUs (relative fluorescence units) for the 142 diabetic children whilst each nondiabetic child in the control group of 79 children had levels of less than 3.55.[18]

There was no overlap of the levels between the two groups of children. All children with diabetes had a higher level of the antibodies (which can only occur from consuming cow's milk) than the group without diabetes.

Significant increases in BSA antibodies in diabetic children have been found in other studies in Finland[19] and France.[20]

For Type I diabetes, there is a specific sequence of 17 amino acids that is found in proteins in cow's milk. The immune system recognizes this sequence as a foreign intruder so antibodies are produced to eliminate the unwanted invaders. Unfortunately, the same 17 amino acid sequence is found on the cells of the pancreas that produce insulin. Consequently, the immune system is unable to distinguish the cow's milk protein fragments from the pancreatic cells. It therefore destroys both which leads to the inability of the pancreas to produce insulin and leads to a life time dependency of insulin injections and their consequences.[21]

Summary

Mammals have evolved over millions of years to provide nutrition for their infants in the first stage of life. There are significant difference between species depending upon factors such as rates of growth.

A bull reaches maturity at 9-10 months, so the rate of growth is markedly different to humans. Consequently, the composition of bovine milk is very different to that of humans. The consequences of cow's milk consumption are potentially harmful.

Related articles

The A2 Milk Story Comparison of Dairy Milks with Human Milk

Footnotes

- Miller, J. et al. (1990) Casein : A Milk Protein with Diverse Biologic Consequences. *Casein*. (43129), 143–159.
- 2. Bullen, C. L. & Willis, A. T. (1971) Resistance of the breast-fed infant to gastroenteritis. *British Medical Journal*. 3 (5770), 338–343.
- 3. Bullen, C. L. & Willis, A. T. (1971) Resistance of the breast-fed infant to gastroenteritis. *British Medical Journal* 3 (5770), 338–343.
- 4. Lara-Villoslada, F. et al. (2007) Beneficial effects of probiotic bacteria isolated from breast milk. *British Journal of Nutrition*. 98 (S1)
- 5. Miller, J. et al. (1990) Casein : A Milk Protein with Diverse Biologic Consequences. *Casein*. (43129), 143–159.
- 6. Hochwallner, H. et al. (2014) Cow's milk allergy: From allergens to new forms of diagnosis, therapy and prevention. *Methods*. 66 (1), 22–33.
- Miller, J. et al. (1990) Casein : A Milk Protein with Diverse Biologic Consequences. *Casein*. (43129), 143–159.
- 8. Müller, G. et al. (1986) Cow milk protein antigens and antibodies in serum of premature infants during the first 10 days of life. *The Journal of Pediatrics*. 109 (5), 869–873.
- 9. Karjalainen, J. et al. (1992) A Bovine Albumin Peptide as a possible trigger of insulin-

dependent Diabetes Mellitus. New England Journal of Medicine. 327 (5), 302-307.

- Dahlquist, G. et al. (1992) An increased level of antibodies to β-lactoglobulin is a risk determinant for early-onset Type 1 (insulin-dependent) diabetes mellitus independent of islet cell antibodies and early introduction of cow's milk. *Diabetologia*. 35 (10), 980–984.
- 11. Knoflach, P. et al. (1987) Serum antibodies to cow's milk proteins in ulcerative colitis and Crohn's disease. *Gastroenterology*. 92 (2), 479–485.
- 12. Kost, N. V. et al. (2009) β -Casomorphins-7 in infants on different type of feeding and different levels of psychomotor development. *Peptides*. 30 (10), 1854–1860.
- 13. Sun, Z. et al. (2003) Relation of β -casomorphin to apnea in sudden infant death syndrome. *Peptides*. 24 (6), 937–943.
- 14. Sokolov, O. et al. (2014) Autistic children display elevated urine levels of bovine casomorphin-7 immunoreactivity. *Peptides*. (56), 68–71.
- 15. Saltzman, J. R. et al. (1999) A randomized trial of Lactobacillus acidophilus BG2FO4 to treat lactose intolerance. *The American Journal of Clinical Nutrition*. 69 (1), 140–146.
- 16. Curry, A. (2013) The Milk Revolution. Nature. 500.
- 17. Saltzman, J. R. et al. (1999) A randomized trial of Lactobacillus acidophilus BG2FO4 to treat lactose intolerance. *The American Journal of Clinical Nutrition*. 69 (1), 140–146.
- 18. Karjalainen, J. et al. (1992) A Bovine Albumin Peptide as a possible trigger of insulindependent Diabetes Mellitus. *New England Journal of Medicine*. 327 (5), 302–307.
- Saukkonen, T. et al. (1994) Children With Newly Diagnosed IDDM Have Increased Levels of Antibodies to Bovine Serum Albumin But Not to Ovalbumin. *Diabetes Care*. 17 (9), 970–976.
- 20. Levy-Marchal, C. et al. (1995) Antibodies against bovine albumin and other diabetes markers in French children. *Diabetes Care*. 18 (8), 1089–1094.
- 21. Karjalainen, J. et al. (1992) A Bovine Albumin Peptide as a possible trigger of insulindependent Diabetes Mellitus. *New England Journal of Medicine*. 327 (5), 302–307.

Comparison of Dairy Milks with Human Milk

Milks are complex lipid emulsions in water containing protein, fat, lactose, vitamins and minerals, as well as enzymes, hormones and immunoglobulins which provide initial immunity functions,

There is approximately 5,500 species of mammals which initially supply their young with milk. There are vast differences in milk composition among the mammal species.

Mammals provide milk for their growing infants that provide a unique collection of proteins, carbohydrates and fats.

Human milk is markedly different to other mammals, in particular to its protein content.

Proteins in human milk provide sufficient of protein to sustain infants for the first six months without any additional food, as well as supplying the means of establishing suitable environment for the growth of healthy intestinal bacteria and providing the proteins involved in the immune system.

The name *Casein* is derived from the Latin word for cheese. Casein is the curds portion of milk (as in curds and whey) which is insoluble portion that is formed in acid conditions or by the action of rennin.

The main nutritional function of casein is the transport of calcium and phosphorus. Fast growing species (rodents and cattle) have a much higher casein content than humans and elephants which are slower growing.[1]

Casein is only found in milk whilst whey proteins are also found in the blood.

Digestion of casein curds is much slower than that of whey proteins.

Since casein is insoluble in water, it forms globules (micelles) which vary in size and composition according to species.

There are three main divisions of casein: alpha (α), beta (β) and kappa (κ), with α -casein separated into S1 and S2 subdivisions. β -casein has two variants which are discussed separately. β -casein contains 209 amino acids. The A1 and A2 variants differ only at amino acid position 67, which is histidine in A1 or proline in A2 milk.

A comparison of the milk of dairy animals and humans is shown below.[2] This paper suggests that mare's milk is a more appropriate substitute for human milk than other dairy species.

Component	Unit	Mare	Cow	Sheep	Goat	Human
Gross energy	kcal/g	480	674	1090	670	677
Fat	% of energy	23	48	62	55	48
Carbohydrate	% of energy	60	32	18	25	44
Protein	% of energy	18	20	20	20	8
Caesin	% of protein	56	87	88	80	29
• α-casein	% of casein	47	55	50	26	12
• β-casein	% of casein	46	30	40	54	65
• к-casein	% of casein	8	15	10	16	24
Whey	% of protein	44	20	22	25	61
• α-lactoalbumin	% of whey	30	21	12	30	35
 β-lactoglobulin 	% of whey	30	58	68	55	0
Immunoglobulins	% of whey	17	12		13	22
Serum albumin	% of whey	4	7	4	0	8
• Lactoferrin	% of whey	9	2		3	30
• Lysozyme	% of whey	9				5
Micelles size	nm	255	182	210	260	64

• The values listed are averages from a number of sources. These values vary according to the study and the age of the infant. Tables such as these can indicate a precision that does not exist.

- Some authors state that α -casein is absent from human milk.[3] [4] The published figure of 0.06 g / 100 g of milk is significantly less than the dairy species.
- With its smaller micelle size, human milk precipitate is finer and more easily digestible than the firm curds of cow's milk. They contain less calcium and phosphorus than that of cow's milk.
- The need for protein is the greatest in the first year of life. Human milk contains only 6-8% of protein (by energy). This is the lowest of any mammal that has been studied. Other dairy species contain approximately 20% protein.
- The amount of casein as a proportion of protein is lower in humans than other dairy species. This varies throughout lactation. Whey to casein ratios vary from 80:20 in early lactation to 50:50 in late lactation.[5]
- The amount of lactoferrin is higher in humans than other species. In humans, 30% of whey proteins is lactoferrin compared with 2% in cows. As well as providing iron, lactoferrin has antimicrobial activity and is important in growth of beneficial bacteria.[6] Many other factors in human milk are also involved in immune response.

It is fascinating that breast milk contains a significant concentration of amylase. Amylase is involved in the digestion of starches which are not present in milk. This suggests that breast milk aids in the digestion of complex carbohydrates when solids are introduced. This is consistent with the view that starches are important in human nutrition.[7]

Related articles

The A2 Milk Story

Comparison of Dairy Milks with Human Milk

Footnotes

- De Kruif, C. G. & Holt, C. (2003) 'Casein Micelle Structure, Functions and Interactions', in P. F. Fox & P. L. H. McSweeney (eds.) *Advanced Dairy Chemistry—1 Proteins*. [Online]. Boston, MA: Springer US. pp. 233–276.
- 2. Potocnik, K. et al. (2011) Mare's milk: composition and protein fraction in comparison with different milk species. *Mljekarstvo*. 61 (2), 107–113.
- 3. Jensen, R. G. (1995) Handbook of Milk Composition. Academic Press.

- Miller, J. et al. (1990) Casein : A Milk Protein with Diverse Biologic Consequences. *Casein*. (43129), 143–159.
- 5. Lönnerdal, B. (2003) Nutritional and physiologic significance of human milk proteins. *American Journal of Clinical Nutrition*. 77 (6), 1537S–1543S.
- 6. Lönnerdal, B. (2003) Nutritional and physiologic significance of human milk proteins. *American Journal of Clinical Nutrition*. 77 (6), 1537S–1543S.
- 7. Lönnerdal, B. (2003) Nutritional and physiologic significance of human milk proteins. *American Journal of Clinical Nutrition*. 77 (6), 1537S–1543S.

The A2 Milk Story

Casein is the group of insoluble proteins found in milk. Whey is the group of soluble proteins. Casein comes in three types: α -casein, β -casein and κ -casein with β -casein occurring in three variants: A1, A2 and B. The B variant is minor.

Most cow's milk contains a mixture of A1 and A2 β -casein. A2 milk refers to milk that only contains the A2 variant of β -casein.

Milk from Guernsey, Jersey, Asian cattle, human milk, and other dairy animals such as sheep, goat, donkeys, yaks, camel and buffalo contain mostly A2 β -casein.

Holstein Friesians cows are the black and white dairy cattle that originated in the Netherlands. More recently, Holstein refers to stock that has been further developed in America whereas Friesians refer to the traditional European stock. These breeds contain significantly more A1 β -casein.[1]

 β -casein from cows contains 209 amino acids. The only difference between A1 and A2 variants is one amino acid at position 67. A1 milk contains histidine at position 67 whilst A2 contains proline. A peptide containing seven amino acids, β -casomorphin-7 (BCM-7), is formed when A1 milk is digested but not when A2 milk digested.[2]

 β -casomorphin is a casomorphin which is a biological active opioid.

Robert Elliott observed a much lower rates of type 1 diabetes amongst Polynesian children that were raised on the Polynesian islands compared with those raised in Auckland. He attributed this to the differences in the β -casein profile. [3]

Elliott was the lead author of a conference paper[4] that examined the effects of feeding casein to non-obese diabetic (NOD) mice. The conclusion was that "the induction of diabetes by casein in the NOD mouse appears to be restricted to casein containing the A1 variant of beta-casein".

An experiment was performed independently at three centres (Ottawa, London and Auckland) in an attempt to duplicate these results. The diets for the mice were coded so that the researchers were not aware of nature of the diets fed to the mice.[5]

The conclusion of this paper was:

These findings show that it is not likely that diabetes could be prevented solely by removing or altering the cows' milk component of the diet [...].

The above conclusion did not prevent Elliott and Hill from applying for US and New Zealand patents. Patents have been issued, listing Robert B Elliott and Jeremy P Hill as the inventors. The US patent has been assigned to New Zealand Dairy Board and the A2 Corporation Limited. The patent claims that:

The invention is based on the discovery that certain variants of β -casein may induce Type-1 diabetes in susceptible individuals while other variants do not. The invention consists of the selection of non-diabetogenic milk producing cows and recovering and processing their milk and milk products. Another aspect of the invention is selectively breeding cows which produce the non-diabetogenic milk.[6]

The a2 Milk Corporation was established to "pioneer the scientific understanding of the A2 protein type so more people can enjoy the nutritional goodness that only comes from real and natural milk".[7]

Note that it does not assist people who are lactose intolerant.

Elliott was a co-author of a paper[8] that surveyed more than "75 foods and over 100 nutritive food supply variables" and compared those to heart disease rates (in 18 countries) and type 1 diabetes rates (in 19 countries)[9].

Cow milk proteins	Correlation	Note
Milk & cream protein g/day	0.68	
Cheese protein g/day	0.23	
A1 protein in milk & cream g/ay	0.92	
A2 protein milk & cream g/day	0.47	
Ratio A1 protein to total beta casein in milk and cream	0.47	
Ratio A2 protein to total beta casein in milk and cream	0.47	
Milk consumption (L/capita)	0.73	[10]

Cow milk proteins and correlation with type 1 diabetes

Below are some observations relating to the published data.

- Elliott's initial hypothesis was that the lower rates of type 1 diabetes in Polynesian children raised in the Pacific Islands was attributed to the differences in the β -casein profile that is, differences in A1 and A2 proteins.
- Dozens, if not hundreds, of studies have shown that the duration of breast feeding protects infants from type 1 diabetes. The importance of feeding infants breast milk has not been considered in this paper.[10] [11] [12] (There is always the rather unlikely possibility that the benefits of breast milk results from its high in A2 casein content.)
- The food consumption figures are derived from national consumption estimates. This does not necessarily correlate to the milk consumption of children that was observed in the initial 1992 study.
- Infant formula derived from cow's milk frequently contains increased whey and reduced casein components in an effort to "humanise" cow's milk. This will not be reflected in the milk protein consumption figures.
- Milk and cream consumption has a greater impact on incidence of type 1 diabetes than cheese consumption.
- A1 and A2 values were estimated by breed from dairy science literature.
- The correlation between A1 protein and diabetes was 0.91 which is a strong correlation. The correlation between A2 protein and diabetes is 0.47, a moderate correlation. Both

correlations are positive – that is, the more A1 protein and the more A2 protein, the incidence of type1 diabetes increases. Neither are protective.

- The correlation with the ratio of A1 protein to total β -casein (in milk and cream) to the incidence of diabetes is, at best, only moderate, at 0.47.
- This is exactly the same as the correlation with the ratio of A2 protein to total β -casein to the incidence of diabetes (in milk and cream), which is supposed to be protective. A higher A2 β -casein ratio results in a higher incidence of type 1 diabetes.
- Consumption of milk, without any regarding for the protein content, shows a strong correlation of 0.73.
- As a generalization, 90% indicates a very strong correlation, 70-90% a strong correlation and 50-70% a moderate correlation.

Professor Boyd Swinburn prepared a 43-page report[13] for the New Zealand Food Safety Authority. His conclusion:

The evidence does not support such dietary changes [replacing A1 β -casein with A2 β -casein] as a recommended clinical approach with a known likelihood of benefit.

Professor Stewart Truswell conclusions is:

The A1/A2 milk hypothesis was ingenious. If the scientific evidence had worked out it would have required huge adjustments in the world's dairy industries. This review concludes, however, that there is no convincing or even probable evidence that the A1 β -casein of cow milk has any adverse effect in humans.[14]

Footnotes

1. Pasin, G. (2017) A2 Milk Facts - California Dairy Research Foundation [online]. Available

from: http://cdrf.org/2017/02/09/a2-milk-facts/ (Accessed 18 December 2017).

- 2. Truswell, A. S. (2005) The A2 milk case: a critical review. *European Journal of Clinical Nutrition*. 59 (5), 623–631.
- 3. Elliott, R. (1992) Epidemiology of diabetes in Polynesia and New Zealand: child health research unit. *Pediatric and adolescent endocrinology*. 2166–71.
- 4. Elliot, R. et al. (1997) 'The role of beta-casein variants in the induction of insulin-dependent diabetes in the non-obese diabetic mouse and humans', in 1997 International Dairy Federation.
- Beales, P. et al. (2002) A multi-centre, blinded international trial of the effect of A1 and A2 β-casein variants on diabetes incidence in two rodent models of spontaneous Type I diabetes. *Diabetologia*. 45 (9), 1240–1246.
- 6. Elliott, R. B. & Hill, J. P. (2002) *Method of selecting non-diabetogenic milk or milk products and milk or milk products so selected*. United States Patent 6451368
- The a2 Milk Company (2017) About us The a2 Milk Company [online]. Available from: https://thea2milkcompany.com/about-us/ (Accessed 27 December 2017).
- 8. Laugesen, M. & Elliott, R. B. (2003) Ischaemic heart disease, Type 1 diabetes, and cow milk A1 β-casein. *The New Zealand Medical Journal*. 116 (1168), 1–19.
- 9. AT=Austria; AU=Australia; CA=Canada; CH=Switzerland; DE=Germany; DK=Denmark; FI=Finland; FR=France; GB=United Kingdom; HU=Hungary; IL=Israel; IS=Iceland; IT=Italy; JP=Japan; NO=Norway; NZ=New Zealand; SE=Sweden; US=United States; VE=Venezuela
- 10. Blom, L. et al. (1989) The Swedish childhood diabetes study social and perinatal determinants for diabetes in childhood. *Diabetologia*. 32 (1), 7–13.
- 11. Verge, C. F. et al. (1994) Environmental factors in childhood IDDM: a population-based, case-control study. *Diabetes Care*. 17 (12), 1381–1389.
- 12. Dahl-Jorgensen, K. et al. (1991) Relationship Between Cows' Milk Consumption and Incidence of IDDM in Childhood. *Diabetes Care*. 14 (11), 1081–1083.
- 13. Swinburn, B. (2004) Beta casein A1 and A2 in milk and human health. *Report to New Zealand Food Safety Authority*.
- 14. Truswell, A. S. (2005) The A2 milk case: a critical review. *European Journal of Clinical Nutrition*. 59 (5), 623–631.

Dairy, Gluten and Autism

Autism is defined by a set of behaviours. Key symptoms are: [1]

- an extreme introversion
- social indifference
- stereotyped repetitive behaviours
- language problems
- problems with hygiene
- temper tantrums
- in some cases hyperactivity
- irrational fears
- decreased but fluctuating insensitivity to pain.

The Centers for Disease Control and Prevention (CDC) monitors the prevalence of autism in 14 states of the US for children with autism at 8 years, as this is the age of peak prevalence. It is not a representative sample of the US. The reference area represents 9% of the US population of the same age.[2]

Median age at diagnosis in the reference area is 53 months which is *less* than the median age in the total US population.

At 2010, the rate of autism at age 8 was 14.7 per 1,000 which is 1 in 68. Boys are 4.5 times more likely to be affected than girls—rate for boys is 1 in 42 compared with 1 in 189 for girls.

Rates of autism have been rising dramatically. The 2010 rate is:

- 29% higher than the preceding estimate of 1 in 88 children in 2008.
- 64% higher than the 2006 estimate of 1 in 110 children.
- 123% higher than the 2002 estimate of 1 in 150 children.

Black children are affected at a rate 14% higher than Hispanic children and white children affected 45% more than Hispanic children.

The association of autism with severe gastrointestinal problems has been documented since the 1990s.[3] [4] [5] [6] [7] [8]

A group of 10 children were assigned to a gluten-free, casein-free diet for a year along with a control group of 10 children. The development for the group of children on the diet was significantly better than for the controls.[9]

Below is a table showing the changes in the number of autistic traits in the diet group over the year.

Code	Number of Autistic Traits		
	Before	After	
А	15	5	
В	10	8	
С	16	9	
D	10	2	
E	10	6	
F	12	5	
G	12	3	
Н	12	5	
I	13	4	
J	13	9	

Similar results were obtained from an 8 week trial. As well as eliminating wheat and dairy, egg white, rice and soy was also eliminated in susceptible children.[10]

As previously noted, food allergies may have many potential confounding allergies. Simply removing dairy and gluten may obscure other potential issues. Eggs and tomatoes have been shown to be problematic.[11]

Also, people on a gluten-free diet consume a less dietary fibre and polysaccharides which affect intestinal bacteria.[12]

Despite the vast number of bacteria species and people, there are only two types of bacteriological ecosystems in the gut (enterotypes). Enterotypes were strongly associated with long-term diets, particularly protein and animal fat versus carbohydrates diets. Microbiome composition changed within 24 hours of initiating a high-fat/low-fiber or lowfat/high-fiber diet.[13]

There is sufficient evidence to suggest a low-fat, high-fibre, whole-food, plant-based diet would be more effective than a dairy-free, gluten-free diet.

In 2000, Hannah Poling was a 19 month old normal girl when she received a series of vaccinations.

Within 48 hours after immunizations to diphtheria, tetanus, and pertussis; Haemophilus influenzae B; measles, mumps, and rubella; polio; and varicella (Varivax), the patient developed a fever to 38.9°C, inconsolable crying, irritability, and lethargy and refused to walk. Four days later, the patient was waking up multiple times in the night, having episodes of opistho-tonus, and could no longer normally climb stairs. Instead, she crawled up and down the stairs. Low-grade intermittent fever was noted for the next 12 days.

Ten days following immunization, the patient developed a generalized erythematous macular rash beginning in the abdomen. The patient's pediatrician diagnosed this as due to varicella vaccination. For 3 months, the patient was irritable and increasingly less responsive verbally, after which the patient's family noted clear autistic behaviors, such as spinning, gaze avoidance, disrupted sleep/wake cycle, and perseveration on specific television programs. All expressive language was lost by 22 months. The patient continued to have chronic yellow watery diarrhea intermittently for 6 months, which was evaluated with negative testing for Clostridium difficile, ova/parasites, and culture.

Four months later, an evaluation with the Infant and Toddlers Early Intervention program for possible autism was initiated. Along with the regression, her appetite remained poor for 6 months and her body weight did not increase. This resulted in a decline on a standard growth chart for weight from the 97th to the 75th percentile Evaluation at 23 months showed atopic dermatitis, slow hair growth, generalized mild hypotonia, toe walking, and normal tendon reflexes. *The Childhood Autism Rating Scale* (CARS) score was 33 (mild autism range), and she also met *Diagnostic and Statistical Manual for Mental Disorders-IV* criteria for autism. [14]

Her father, Dr Jon Poling, PhD, was a neurologist at John Hopkins Hospital.

A three-member panel of Federal Claims Court ruled in November 2007 that, "the vaccinations received on July 19, 2000, significantly aggravated an underlying mitochondrial disorder, which predisposed her to deficits in cellular energy metabolism, and manifested as a regressive encephalopathy with features of ASD [Autism Spectrum Disorder]." Note that the court ruled that Hannah had "features of ASD".

On the 29th March 2008, Dr Julie Gerberding, who was the Director Centers for Disease Control and Prevention at the time, was interviewed by CNN.

Dr Gerberding stated:

Well, you know, I don't have all the facts because I still haven't been able to review the case files myself. But my understanding is that the child has a what we think is a rare mitochondrial disorder. And children that have this disease, anything that stresses them creates a situation where their cells just can't make enough energy to keep their brains functioning normally. Now, we all know that vaccines can occasionally cause fevers in kids. So if a child was immunized, got a fever, had other complications from the vaccines. And if you're predisposed with the mitochondrial disorder, it can certainly set off some damage. Some of the symptoms can be symptoms that have characteristics of autism.[15]

- The court ruled that Hannah had "features of ASD" despite the fact that she was diagnosed as having autism. By using the acronym, the court avoiding using the word "autism".
- Gerberding states that she has not been able to review the case files. The court handed down the decision 4 months previously.
- In the interview, Gerberding does not answer the question, "Does Hannah have autism?" She states that Hannah has "symptoms characteristic of autism". Hannah does have autism.[16]
- Gerberding states that this can be a result of a rare mitochondrial dysfunction. An estimate for mitochondrial dysfunction in autistic children is 5%. However, several biomarkers for mitochondrial dysfunction are much higher: low total carnitine (90.0%); elevated AST (45.6%); elevated creatine kinase (46.8%); elevated ammonia (35.0%); elevated lactate (31.1%).[17]
- Vaccination-associated adverse events occur in ~1 of every 6 toddlers receiving measlesmumps-rubella vaccine dose 1, with high fever (greater than 39.5°C) occurring in 1 of 20.[18]

Footnotes

- 1. Reichelt, K. L. et al. (2012) Peptides role in autism with emphasis on exorphins.pdf. *Microbial Ecology in Health & Disease*. 23 (18958),
- Centers for Disease Control and Prevention (2014) Prevalence of autism spectrum disorder among children aged 8 years-autism and developmental disabilities monitoring network, 11 sites, United States, 2010. *Morbidity and mortality weekly report. Surveillance summaries* (Washington, DC: 2002). 63 (2), 1-24.
- 3. Galiatsatos, P. et al. (2009) Autistic Entercolitis Fact ot Fiction. *Canadian Journal of Gasteroentrology*. 23 (2), 95–98.

- 4. Horvath, K. et al. (1999) Gastrointestinal abnormalities in children with autistic disorder. *The Journal of Pediatrics*. 135 (5), 559–563.
- 5. Furlano, R. I. et al. (2001) Colonic CD8 and $\gamma\delta$ T-cell infiltration with epithelial damage in children with autism. *The Journal of Pediatrics*. 138 (3), 366–372.
- Krigsman, A. et al. (2010) Clinical presentation and histologic findings at ileocolonoscopy in children with autistic spectrum disorder and chronic gastrointestinal symptoms. *Autism Insights.* 2 (1), 1–11.
- 7. de Magistris, L. et al. (2010) Alterations of the Intestinal Barrier in Patients With Autism Spectrum Disorders and in Their First-degree Relatives: *Journal of Pediatric Gastroenterology and Nutrition*. 51 (4), 418–424.
- 8. de Magistris, L. et al. (2013) Antibodies against Food Antigens in Patients with Autistic Spectrum Disorders. *BioMed Research International*. 20131–11.
- 9. Knivsberg, A. M. et al. (2002) A Randomised, Controlled Study of Dietary Intervention in Autistic Syndromes. *Nutritional Neuroscience*. 5 (4), 251–261.
- 10. Lucarelli, S. et al. (1995) Food allergy and infantile autism. *Panminerva*. 37 (3), 137–141.
- Carroccio, A. et al. (2012) Non-Celiac Wheat Sensitivity Diagnosed by Double-Blind Placebo-Controlled Challenge: Exploring a New Clinical Entity. *American Journal of Gastroenterology*. 107 (12), 1898–1906.
- 12. De Palma, G. et al. (2009) Effects of a gluten-free diet on gut microbiota and immune function in healthy adult human subjects. *British Journal of Nutrition*. 102 (08), 1154.
- 13. Wu, G. D. et al. (2011) Linking long-term dietary patterns with gut microbial enterotypes. *Science*. 334 (6052), 105–108.
- 14. Poling, J. S. et al. (2006) Developmental regression and mitochondrial dysfunction in a child with autism. Journal of child neurology. 21 (2), 170–172.
- 15. Gupta, S. (2008) Unraveling the Mystery of Autism. *Health Call*. 29 March. [online]. Available from: http://transcripts.cnn.com/TRANSCRIPTS/0803/29/hcsg.01.html.
- 16. Poling, J. S. et al. (2006) Developmental regression and mitochondrial dysfunction in a child with autism. *Journal of child neurology*. 21 (2), 170–172.
- 17. Rossignol, D. A. & Frye, R. E. (2012) Mitochondrial dysfunction in autism spectrum disorders: a systematic review and meta-analysis. *Molecular psychiatry*. 17 (3), 290–314.
- LeBaron, C. W. et al. (2006) Evaluation of potentially common adverse events associated with the first and second doses of measles-mumps-rubella vaccine. *Pediatrics*. 118 (4), 1422–1430.