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Wheat: The Unhealthy Whole Grain Book Excerpt: Wheat Belly

By William Davis, MD

Flip through your parents' or grandparents' family albums and you're likely to be struck by how *thin* everyone looks. The women probably wore size-four dresses and the men sported 32-inch waists. Overweight was something measured only by a few pounds; obesity rare. Overweight children? Almost never. Any 42-inch waists? Not here. Two-hundred-pound teenagers? Certainly not.

The women of that world didn't exercise much at all. How many times did you see your mom put on her jogging shoes to go out for a three-mile run? Nowadays I go outdoors on any nice day and see dozens of women jogging, riding their bicycles, power walking—things we'd virtually never see 40 or 50 years ago. And yet, we're getting fatter and fatter every year.

I am going to argue that the problem with the diet and health of most Americans is wheat—or what we are being sold that is called “wheat.”

Documented peculiar effects of wheat on humans include appetite stimulation, exposure to brain-active *exorphins* (the counterpart of internally derived endorphins), exaggerated blood sugar surges that trigger cycles of satiety alternating with heightened appetite, the process of *glycation* that underlies disease and aging, inflammatory and pH effects that erode cartilage and damage bone, and activation of disordered immune responses. A complex range of diseases results from consumption of wheat, from celiac disease—the devastating intestinal disease that develops from exposure to wheat gluten—to an assortment of neurological disorders, diabetes, heart disease, arthritis, curious rashes, and the paralyzing delusions of schizophrenia.

The sad truth is that the proliferation of wheat products in the American diet parallels the expansion of our waists. Advice to cut fat and cholesterol intake and replace the calories with whole grains that was issued by the National Heart, Lung, and Blood Institute through its National Cholesterol Education Program in 1985 coincides precisely with the start of a sharp upward climb in body weight for men and women. Ironically, 1985 also marks the year when the Centers for Disease Control and Prevention (CDC) began tracking body weight statistics, documenting the explosion in obesity and diabetes that began that very year.

So why has this seemingly benign plant that sustained generations of humans suddenly turned on us? For one thing, it is not the same grain our forebears ground into their daily bread. Wheat has changed dramatically in the past fifty years under the influence of agricultural scientists. Wheat strains have been hybridized, crossbred, and introgressed to make the wheat plant resistant to environmental conditions, such as drought, or pathogens, such as fungi. But most of all, genetic changes have been induced to increase yield *per acre*. Such enormous strides in yield have required drastic changes in genetic code. Such fundamental genetic changes have come at a price.

Wheat starches are the complex carbohydrates that are the darlings of dietitians. “Complex” means that the carbohydrates in wheat are composed of polymers (repeating chains) of the simple sugar, glucose. Conventional wisdom, such as that from your dietitian or the USDA, says we should all reduce our consumption of simple carbohydrates in the form of candy and soft drinks, and increase our consumption of complex carbohydrates.

Of the complex carbohydrate in wheat, 75 percent is the chain of branching glucose units, *amylopectin*, and 25 percent is the linear chain of glucose units, *amylose*. In the human gastrointestinal tract, both amylopectin and amylose are digested by the salivary and stomach enzyme amylase. Amylopectin is efficiently digested by amylase to glucose, while amylose is much less efficiently digested, some of it making its way to the colon undigested. Thus, the complex carbohydrate amylopectin is rapidly converted to glucose and absorbed into the bloodstream and, because it is most efficiently digested, is mainly responsible for wheat's blood-sugar-increasing effect.

Wheat: Super Carbohydrate

People are usually shocked when I tell them that whole wheat bread increases blood sugar to a higher level than sucrose.¹ Aside from some extra fiber, eating two slices of whole wheat bread is really little different, and often worse, than drinking a can of sugar-sweetened soda or eating a sugary candy bar.

This information is not new. A 1981 University of Toronto study launched the concept of the glycemic index, i.e., the comparative blood sugar effects of carbohydrates: the higher the blood sugar after consuming a specific food compared to glucose, the higher the glycemic index (GI). The original study showed that the GI of white bread was 69, while the GI of whole grain bread was 72 and Shredded Wheat cereal was 67, while that of sucrose (table sugar) was 59.2 Yes, the GI of whole grain bread is higher than that of sucrose. Incidentally, the GI of a Mars Bar nougat,

chocolate, sugar, caramel, and all—is 68. That's *better* than whole grain bread. The GI of a Snickers bar is 41—far better than whole grain bread.

This has important implications for body weight, since glucose is unavoidably accompanied by insulin, the hormone that allows entry of glucose into the cells of the body, converting the glucose to fat. The higher the blood glucose after consumption of food, the greater the insulin level, the more fat is deposited. This is why, say, eating a three-egg omelet that triggers no increase in glucose does not add to body fat, while two slices of whole wheat bread increases blood glucose to high levels, triggering insulin and growth of fat, particularly abdominal or deep visceral fat.

Trigger high blood sugars repeatedly and/or over sustained periods, and more fat accumulation results. The consequences of glucose-insulin-fat deposition are especially visible in the abdomen—resulting in, yes, wheat belly. The bigger your wheat belly, the poorer your response to insulin, since the deep visceral fat of the wheat belly is associated with poor responsiveness, or “resistance,” to insulin, demanding higher and higher insulin levels, a situation that cultivates diabetes. Moreover, the bigger the wheat belly in males, the more estrogen is produced by fat tissue. The bigger your wheat belly, the more inflammatory responses that are triggered: heart disease and cancer.

The extremes of blood sugar and insulin are responsible for growth of fat specifically in the visceral organs. Experienced over and over again, visceral fat accumulates, creating a fat liver, two fat kidneys, a fat pancreas, fat large and small intestines, as well as its familiar surface manifestation, a wheat belly. (Even your heart gets fat, but you can't see this through the semi-rigid ribs.)

Visceral fat is different. It is uniquely capable of triggering a universe of inflammatory phenomena. Visceral fat filling and encircling the abdomen of the wheat belly sort is a unique, twenty-four-hour-a-day, seven-day-a-week metabolic factory. And what it produces is inflammatory signals and abnormal cytokines, or cell-to-cell hormone signal molecules, such as leptin, resistin, and tumor necrosis factor.^{3,4} The more visceral fat present, the greater the quantities of abnormal signals released into the bloodstream.

All body fat is capable of producing another cytokine, adiponectin, a protective molecule that reduces risk for heart disease, diabetes, and hypertension. However, as visceral fat increases, its capacity to produce protective adiponectin diminishes.⁵ The combination of lack of adiponectin along with increased leptin, tumor necrosis factor, and other inflammatory products underlies abnormal insulin responses, diabetes, hypertension, and heart disease.⁶ The list of other health conditions triggered by visceral fat is growing and now includes dementia, rheumatoid arthritis, and colon cancer.⁷ This is why waist circumference is proving to be a powerful predictor of all these conditions, as well as of mortality.⁸

High blood insulin provokes visceral fat accumulation, the body's means of storing excess energy. When visceral fat accumulates, the flood of inflammatory signals it produces causes tissues such as muscle and liver to respond less to insulin. This so-called insulin resistance means that the pancreas must produce greater and greater quantities of insulin to metabolize the sugars. Eventually, a vicious circle of increased insulin resistance, increased insulin production, increased deposition of visceral fat, increased insulin resistance, etc., etc., ensues.

But you could remove wheat and an entire domino effect of changes develop: less triggering of blood sugar rises, no exorphins to drive the impulse to consume more, no initiation of the glucose-insulin cycle of appetite. And if there's no glucose-insulin cycle, there's little to drive appetite except genuine physiologic need for sustenance, not overindulgence. If appetite shrinks, calorie intake is reduced, visceral fat disappears, insulin resistance improves, blood sugars fall. Diabetics can become nondiabetics, prediabetics can become nonprediabetics. All the phenomena associated with poor glucose metabolism recede, including high blood pressure, inflammatory phenomena, glycation, small LDL particles, triglycerides.

If you also count the people who don't yet meet full criteria for prediabetes but just show high after-meal blood sugars, high triglycerides, small LDL particles, and poor responsiveness to insulin (insulin resistance)—phenomena that can still lead to heart disease, cataracts, kidney disease, and eventually diabetes—you would find few people in the modern age who are not in this group, children included.

This disease is not just about being fat and having to take medications; it leads to serious complications, such as kidney failure (40 percent of all kidney failure is caused by diabetes) and limb amputation (more limb amputations are performed for diabetes than any other non-traumatic disease). We're talking *real* serious.

Pancreatic Assault and Battery

The cost of Americans becoming obese dwarfs the sum spent on cancer. More money will be spent on health consequences of obesity than education.

The early phase of growing visceral fat and diabetes is accompanied by a 50 percent *increase* in pancreatic beta cells responsible for producing insulin, a physiologic adaptation to meet the enormous demands of a body that is resistant to insulin. But beta cell adaptation has limits.

High blood sugars, such as those occurring after a nice cranberry muffin provoke the phenomenon of “glucotoxicity,” actual damage to pancreatic insulin-producing beta cells that results from high blood sugars.⁹

The higher the blood sugar, the more damage to beta cells. The effect is progressive and starts at a glucose level of 100 mg/dL, a value many doctors call normal. After two slices of whole wheat bread with low-fat turkey breast, a typical blood glucose would be 140 to 180 mg/dL in a nondiabetic adult, more than sufficient to do away with a few

precious beta cells—which are never replaced.

Your poor, vulnerable pancreatic beta cells are also damaged by the process of lipotoxicity, loss of beta cells due to increased triglycerides and fatty acids, such as those developing from repeated carbohydrate ingestion. Recall that a diet weighted toward carbohydrates results in increased VLDL particles and triglycerides that persist in both the after-meal and between-meal periods, conditions that further exacerbate lipotoxic attrition of pancreatic beta cells.

Pancreatic injury is further worsened by inflammatory phenomena, such as oxidative injury, leptin, various interleukins, and tumor necrosis factor, all resulting from the visceral fat hotbed of inflammation, all characteristic of prediabetic and diabetic states.¹⁰

Over time and repeated sucker punches from glucotoxicity, lipotoxicity, and inflammatory destruction, beta cells wither and die, gradually reducing the number of beta cells to less than 50 percent of the normal starting number.¹¹ That's when diabetes is irreversibly established.

Part of the prevailing standard of care to prevent and treat diabetes, a disease caused in large part by carbohydrate consumption . . . is to advise increased consumption of carbohydrates.

Fighting Carbohydrates with Carbohydrates

Years ago, I used the ADA diet in diabetic patients. Following the carbohydrate intake advice of the ADA, I watched patients gain weight, experience deteriorating blood glucose control and increased need for medication, and develop diabetic complications such as kidney disease and neuropathy. *Ignoring* ADA diet advice and cutting carbohydrate intake leads to improved blood sugar control, reduced HbA1c, dramatic weight loss, and improvement in all the metabolic messiness of diabetes such as high blood pressure and triglycerides.

The ADA advises diabetics to cut fat, reduce saturated fat, and include 45 to 60 grams of carbohydrate—preferably “healthy whole grains”—in each meal, or 135 to 180 grams of carbohydrates per day, not including snacks. It is, in essence, a fat-phobic, carbohydrate-centered diet, with 55 to 65 percent of calories from carbohydrates. If I were to sum up the views of the ADA toward diet, it would be: Go ahead and eat foods that increase blood sugar, just be sure to adjust your medication to compensate.

Reduction of carbohydrates improves blood sugar behavior, reducing the diabetic tendency. If taken to extremes, it is possible to *eliminate* diabetes medications in as little as six months. In some instances, I believe it is safe to call that a cure, provided excess carbohydrates don't make their way back into the diet. Let me say that again: If sufficient pancreatic beta cells remain and have not yet been utterly decimated by long-standing glucotoxicity, lipotoxicity, and inflammation, it is entirely possible for some, if not most, prediabetics and diabetics to be cured of their condition, something that virtually never happens with conventional low-fat diets such as that advocated by the American Diabetes Association.

We might gain better understanding of the aging process if we were able to observe the effects of *accelerated* aging. We need not look to any mouse experimental model to observe such rapid aging; we need only look at humans with diabetes. Diabetes yields a virtual proving ground for accelerated aging, with all the phenomena of aging approaching faster and occurring earlier in life—heart disease, stroke, high blood pressure, kidney disease, osteoporosis, arthritis, cancer. Specifically, diabetes research has linked high blood glucose of the sort that occurs after carbohydrate consumption with hastening your move to the wheelchair at the assisted living facility.

Advanced glycation end products, appropriately acronymed AGE, is the name given to the stuff that stiffens arteries (atherosclerosis), clouds the lenses of the eyes (cataracts), and mucks up the neuronal connections of the brain (dementia), all found in abundance in older people.¹² The older we get, the more AGEs can be recovered in kidneys, eyes, liver, skin, and other organs. Although we can see evidence of some AGE effects—saggy skin and wrinkles, the milky opacity of cataracts, the gnarled hands of arthritis—none are truly quantitative. AGEs nonetheless, at least in a qualitative way, identified via biopsy as well as some aspects apparent with a simple glance, yield an index of biological decay.

AGEs are useless debris that result in tissue decay as they accumulate. They provide no useful function: AGEs cannot be burned for energy, they provide no lubricating or communicating functions, they provide no assistance to nearby enzymes or hormones. Beyond effects you can see, accumulated AGEs also mean loss of the kidneys' ability to filter blood to remove waste and retain protein, stiffening and atherosclerotic plaque accumulation in arteries, stiffness and deterioration of cartilage in joints such as the knee and hip, and loss of functional brain cells with clumps of AGE debris taking their place.

While some AGEs enter the body directly because they are found in various foods, they are also a by-product of high blood sugar (glucose), the phenomenon that defines diabetes.

The sequence of events leading to formation of AGEs goes like this: Ingest foods that increase blood glucose. The greater availability of glucose to the body's tissues permits the glucose molecule to react with any protein, creating a combined glucose-protein molecule. Once AGEs form, they are irreversible and cannot be undone. They also collect in chains of molecules, forming AGE polymers that are especially disruptive.¹³ AGEs are notorious for accumulating right where they sit, forming clumps of useless debris resistant to any of the body's digestive or cleansing processes.

Thus, AGEs result from a domino effect set in motion anytime blood glucose increases. Anywhere that glucose goes (which is virtually everywhere in the body), AGEs will follow. The higher the blood glucose, the more AGEs will

accumulate and the faster the decay of aging will proceed.

Diabetes is the real-world example that shows us what happens when blood glucose remains high, since diabetics typically have glucose values that range from 100 to 300 mg/dL all through the day as they chase their sugars with insulin or oral medications. If such repetitive high blood sugars lead to health problems, we should see such problems expressed in an exaggerated way in diabetics . . . and indeed we do. Diabetics, for instance, are two to five times more likely to have coronary artery disease and heart attacks, 44 percent will develop atherosclerosis of the carotid arteries or other arteries outside of the heart, and 20 to 25 percent will develop impaired kidney function or kidney failure an average of eleven years following diagnosis.¹⁴ In fact, high blood sugars sustained over several years virtually *guarantee* development of complications.

With repetitive high blood glucose levels in diabetes, you'd also expect higher blood levels of AGEs, and indeed, that is the case. Diabetics have 60 percent greater blood levels of AGEs compared to nondiabetics.¹⁵

AGEs that result from high blood sugars are responsible for most of the complications of diabetes, from neuropathy (damaged nerves leading to loss of sensation in the feet) to retinopathy (vision defects and blindness) to nephropathy (kidney disease and kidney failure). The higher the blood sugar and the longer blood sugars stay high, the more AGE products will accumulate and the more organ damage results.

AGEs form even when blood sugar is normal, though at a much lower rate compared to when blood sugar is high. AGE formation therefore characterizes normal aging of the sort that makes a sixty-year-old person look sixty years old. But the AGEs accumulated by the diabetic whose blood sugar is poorly controlled cause *accelerated* aging. Diabetes has therefore served as a living model for age researchers to observe the age-accelerating effects of high blood glucose. Thus, the complications of diabetes, such as atherosclerosis, kidney disease, and neuropathy, are also the diseases of aging, common in people in their sixth, seventh, and eighth decades, uncommon in younger people in their second and third decades. Diabetes therefore teaches us what happens to people when glycation occurs at a faster clip and AGEs are permitted to accumulate.

AGE formation is therefore a continuum. But while AGEs form at even normal blood glucose levels (fasting glucose 90 mg/dL or less), they form faster at higher blood sugar levels. The higher the blood glucose, the more AGEs form. There really is no level of blood glucose at which AGE formation can be expected to cease entirely.

Being nondiabetic does *not* mean that you will be spared such fates. AGEs accumulate in nondiabetics and wreak their age-advancing effects. All it takes is a little extra blood sugar, just a few milligrams above normal, and—voilà—you've got AGEs doing their dirty work and gumming up your organs. Over time, you too can develop all the conditions seen in diabetes if you have sufficient AGE accumulation.

Thus, wheat products such as your poppy seed muffin or roasted vegetable focaccia are triggers of extravagant AGE production. Wheat, because of its unique blood glucose-increasing effect, makes you age faster. Via its blood sugar/AGE-increasing effects, wheat accelerates the rate at which you develop signs of skin aging, kidney dysfunction, dementia, atherosclerosis, and arthritis.

The Great Glycation Race

There is a widely available test that, while not capable of providing an index of biological age, provides a measure of the *rate* of biological aging due to glycation. Knowing how fast or slow you are glycating the proteins of your body helps you know whether biological aging is proceeding faster or slower than chronological age. Thankfully, a simple blood test can be used to gauge the ongoing rate of AGE formation: hemoglobin A1c, or HbA1c. HbA1c is a common blood test that, while usually used for the purpose of diabetes control, can also serve as a simple index of glycation.

Hemoglobin is the complex protein residing within red blood cells that is responsible for their ability to carry oxygen. Like all other proteins of the body, hemoglobin is subject to glycation, i.e., modification of the hemoglobin molecule by glucose. The reaction occurs readily and, like other AGE reactions, is irreversible. The higher the blood glucose, the greater the percentage of hemoglobin that becomes glycated.

Red blood cells have an expected life span of sixty to ninety days. Measuring the percentage of hemoglobin molecules in the blood that are glycated provides an index of how high blood glucose has ventured over the preceding sixty to ninety days, a useful tool for assessing the adequacy of blood sugar control in diabetics, or to diagnose diabetes.

A slender person with a normal insulin response who consumes a limited amount of carbohydrates will have approximately 4.0 to 4.8 percent of all hemoglobin glycated (i.e., an HbA1c of 4.0 to 4.8 percent), reflecting the unavoidable low-grade, normal rate of glycation. Diabetics commonly have 8, 9, even 12 percent or more glycated hemoglobin—twice or more the normal rate. The majority of nondiabetic Americans are somewhere in between, most living in the range of 5.0 to 6.4 percent, above the perfect range but still below the “official” diabetes threshold of 6.5 percent.^{16,17} In fact, an incredible 70 percent of American adults have an HbA1c between 5.0 percent and 6.9 percent.¹⁸ HbA1c does not have to be 6.5 percent to generate adverse health consequences. HbA1c in the “normal” range is associated with increased risk for heart attacks, cancer, and 28 percent increased mortality for every 1 percent increase in HbA1c.^{19,20}

That trip to the all-you-can-eat pasta bar, accompanied by a couple of slices of Italian bread and finished off with a little bread pudding, sends your blood glucose up toward 150 to 250 mg/dL for three or four hours; high glucose for a sustained period glycates hemoglobin, reflected in higher HbA1c.

HbA1c—i.e., glycated hemoglobin—therefore provides a running index of glucose control. It also reflects to what degree you are glyating body proteins beyond hemoglobin. The higher your HbA1c, the more you are also glyating the proteins in the lenses of your eyes, in kidney tissue, arteries, skin, etc.²¹ In effect, HbA1c provides an ongoing index of aging rate: The higher your HbA1c, the faster you are aging.

So HbA1c is much more than just a feedback tool for blood glucose control in diabetics. It also reflects the rate at which you are glyating other proteins of the body, the rate at which you are aging. Stay at 5 percent or less, and you are aging at the normal rate; over 5 percent, and time for you is moving faster than it should, taking you closer to the great nursing home in the sky.

Dr. William Davis is medical director of the online heart disease prevention and reversal program, Track Your Plaque (www.trackyourplaque.com). Join his conversations on Facebook and on his blogs, wheatbellyblog.com and trackyourplaque.com/blog.

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References

1. Foster-Powell, Holt SHA, Brand-Miller JC. International table of glycemic index and glycemic load values: 2002. *Am J Clin Nutr.* 2002;76(1):5–56.
2. Jenkins DJH, Wolever TM, Taylor RH, et al. Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr.* 1981 Mar;34(3):362–6.
3. Klötting N, Fasshauer M, Dietrich A, et al. Insulin sensitive obesity. *Am J Physiol Endocrinol Metab.* 2010 Jun 22.
4. DeMarco VG, Johnson MS, Whaley-Connell AT, Sowers JR. Cytokine abnormalities in the etiology of the cardiometabolic syndrome. *Curr Hypertens Rep.* 2010 Apr;12(2):93–8.
5. Matsuzawa Y. Establishment of a concept of visceral fat syndrome and discovery of adiponectin. *Proc Jpn Acad Ser B Phys Biol Sci.* 2010;86(2):131–41.
6. *Ibid.*
7. Funahashi T, Matsuzawa Y. Hypoadiponectinemia: a common basis for diseases associated with overnutrition. *Curr Atheroscler Rep.* 2006 Sep;8(5):433–8.
8. Deprés J, Lemieux I, Bergeron J, et al. Abdominal obesity and the metabolic syndrome: contributions to global cardiometabolic risk. *Arterioscl Thromb Vasc Biol.* 2008;28:1039–49.
9. Marchetti P, Lupi R, Del Guerra S, et al. The beta-cell in human type 2 diabetes. *Adv Exp Med Biol.* 2010;654:501–14.
10. *Ibid.*
11. Wajchenberg BL. Beta-cell failure in diabetes and preservation by clinical treatment. *Endocr Rev.* 2007 Apr;28(2):187–218.
12. Bengmark S. Advanced glycation and lipoxidation end products—amplifiers of inflammation: The role of food. *J Parent Enter Nutr.* 2007 Sept-Oct;31(5):430–40.
13. Uribarri J, Cai W, Peppas M, et al. Circulating glycotoxins and dietary advanced glycation endproducts: Two links to inflammatory response, oxidative stress, and aging. *J Gerontol.* 2007 Apr;62A:427–33.
14. Epidemiology of Diabetes Interventions and Complications (EDIC). Design, implementation, and preliminary results of a long-term follow-up of the Diabetes Control and Complications Trial cohort. *Diabetes Care.* 1999 Jan;22(1):99–111.
15. Kilhovd BK, Giardino I, Torjesen PA, et al. Increased serum levels of the specific AGE-compound methylglyoxal-derived hydroimidazolone in patients with type 2 diabetes. *Metabolism.* 2003;52:163–7.
16. Sarwar N, Aspelund T, Eiriksdottir G, et al. Markers of dysglycaemia and risk of coronary heart disease in people without diabetes: Reykjavik prospective study and systematic review. *PLoS Med.* 2010 May 25;7(5):e1000278.
17. International Expert Committee. International Expert Committee report on the role of the HbA1c assay in the diagnosis of diabetes. *Diabetes Care.* 2009;32:1327–44.
18. Khaw KT, Wareham N, Luben R, et al. Glycated haemoglobin, diabetes, and mortality in men in Norfolk cohort of

European Prospective Investigation of Cancer and Nutrition (EPIC-Norfolk). *Brit Med J.* 2001 Jan 6;322(7277):15–8.

19. Gerstein HC, Swedberg K, Carlsson J, et al. The hemoglobin A1c level as a progressive risk factor for cardiovascular death, hospitalization for heart failure, or death in patients with chronic heart failure: an analysis of the Candesartan in Heart failure: Assessment of Reduction in Mortality and Morbidity (CHARM) program. *Arch Intern Med.* 2008 Aug 11;168(15):1699–704.

20. Khaw KT, Wareham N, Luben R, et al. Glycated haemoglobin, diabetes, and mortality in men in Norfolk cohort of european prospective investigation of cancer and nutrition (EPIC-Norfolk). *BMJ.* 2001 Jan 6;322(7277):15-8.

21. Swami-Mruthinti S, Shaw SM, Zhao HR, et al. Evidence of a glycemic threshold for the development of cataracts in diabetic rats. *Curr Eye Res.* 1999 Jun;18(6):423–9.

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“The Wheat Belly Diet is not particularly easy to follow... Banishing food groups is not recommended unless there is scientific evidence to back that decision up. And there is no proof that gluten is the bad guy in the obesity epidemic”. *Wheat Belly Diet Quality of Ingredients.* Many unhealthy and high calorie foods are banned on this diet. It limits foods which are known to be nutritionally weak as well. While this can lead to weight loss, it's difficult to uphold due to the severe restrictions. “we conclude that whole-wheat consumption cannot be linked to increased prevalence of obesity in the general population”. It's clear from all the science available that there is no need to follow a diet like this. Click the link cited here for a comprehensive listing of the top ranked weight loss diets. As the titles of these books suggest, wheat causes a big belly and grains damage the brain. Within. foods consumed by billions of people throughout human history—are now unhealthy and must be. minimized or, better yet, avoided altogether. Page 2. Examples of whole grains included whole wheat, dark bread, oats, brown rice, rye, barley, and bulgur. Even those few people intolerant of gluten (wheat, barley, and rye) can healthfully consume non- gluten rice, corn, oats, and other grains. Steering people away from the few healthy components of our diet (grains and other starchy vegetables) and toward the unhealthy foods (meat, dairy, fish, and eggs) makes matters worse. People are desperate for a solution to their weight and health problems, and many of them are easily deceived.

So Gliadin joins the party " Gliadin proteins; wheat germ agglutinin joins the party and they get access to your bloodstream and other foreign substances gain access to your bloodstream, all because of this effect that wheat has. So if you read the science that comes from all of this " that is this Gliadin effect of opening the intestinal barriers, you'll see that the scientists say you know what, this seems an awful lot like cholera toxin. The most typical joints inflamed from wheat are the fingers and wrists, elbows followed by shoulders and then the big joints, less common but still can be a pain. You get inflammation of the airway, asthma. You get inflammation of the brain. You get inflammation of thyroid. Book Excerpt: *Wheat Belly*. October 2011. By William Davis, MD. Documented peculiar effects of wheat on humans include appetite stimulation, exposure to brain-active exorphins (the counterpart of internally derived endorphins), exaggerated blood sugar surges that trigger cycles of satiety alternating with heightened appetite, the process of glycation that underlies disease and aging, inflammatory and pH effects that erode cartilage and damage bone, and activation of disordered immune responses.