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Are Any Ancient Grains Gluten Free?

A Brief History of Wheat

When botanists discuss wheat breeding, they often point out that wheat is more complex than other cereal grains. And they aren't exaggerating. Every cell of every living creature contains deoxyribonuceic acid (DNA). And that DNA contains the instructions that define all the processes necessary for life and replication. DNA is structured as long ribbon-like strands known as chromosomes. Various species have evolved in various ways, so some have more chromosomes than others. And organisms tend to have differing numbers of copies of chromosomes. For example, a human cell has 23 pairs of chromosomes, for a total of 46 chromosomes. This is known as a diploid arrangement. Corn has a diploid number of 10 pairs of chromosomes and rice has 12 pairs. By contrast, wheat cells have a hexaploid arrangement — six copies of its seven chromosomes for a total of 42 chromosomes. Interestingly, wheat, corn, and rice are technically all grasses and they all originated from a common ancestor over 50 million years ago, but they have all evolved quite differently over the years.

Although wheat has fewer chromosomes than humans, it has many more genes. Wheat has somewhere between 164,000 and 334,000 genes, while humans have only 20,000 to 25,000 genes (Colorado wheat, 2013, Nov 15).[1]

The entire set of genetic material that contains the instructions for the reproduction and functioning of an organism is known as the genome of that organism. Among the commonly grown crops, wheat has the largest genome.

So how did wheat come to have such a much more complex arrangement?

Cross-breeding 2 different species of animals typically results in offspring that are sterile, unless they are very closely related. For example, mules which are the offspring of a horse and a donkey, are sterile.

However, cross-breeding in plants is much more likely to result in fertile

offspring because of chromosome duplication. Some examples besides wheat include cotton and peanuts. Wheat is very unusual because it took not just one, but two separate cross-breeding events to produce it.

The first lucky cross-breeding took place between 10,000 to 40,000 years ago.

Wild einkorn wheat is a diploid and has only 14 chromosomes. Somewhere between 10,000 and 40,000 years ago, wild einkorn (*Triticum monococcum*), crossed with a species of goat grass (*Aegilops speltoides*) and produced fertile offspring.

The result of that first fortuitous cross-breeding gave our ancestors wild emmer wheat, which is a tetraploid with 28 chromosomes (in comparison with the hexaploid arrangement of 42 chromosomes of modern wheat). As mentioned previously, this cross-breeding apparently occurred between two wild species, without human intervention.

If the hybridization had resulted in a diploid (two sets of chromosomes), the offspring would have been sterile. But the extra chromosomes in four sets of chromosomes allowed the cross to be fertile. The earliest clear evidence of the agricultural production of domesticated emmer wheat ranges from about 7,650 to 8,200 years BC near Damascus, Syria (Encyclopedia of life, n.d,).[2]

Emmer wheat may have been domesticated sooner than einkorn wheat. The earliest known production of einkorn wheat occurred in southeastern Turkey about 7,950 to 8,650 BC (Encyclopedia of life, n.d.).[3] But wild emmer wheat was gathered for thousands of years before this, so it's possible that it was grown as a crop elsewhere earlier than einkorn wheat.

The second lucky cross-breeding took place about 7,000 BC.

Wild emmer (known as *Triticum turgidum subspecies dicoccoides*) which is tetraploid, and another species of goat grass (*Aegilops tauschii*) which is diploid, crossed to yield *Triticum aestivum*, which is a hexaploid with 42 chromosomes (Crop Origins, n.d.).[4] It has become the basis for modern wheat and it has led to the development of over 25,000 (by some estimates) cultivars of bread wheat since then (Encyclopedia of life, n.d.).[5]

The seed pods of wild plants tend to shatter in order to scatter the seeds. As cereal grain plants are domesticated, they are selected for tighter husks which will help to hold the seeds tighter and longer to reduce loses before harvest. Over the years farmers select plants with more and larger grain, to further enhance yields. But the main benefit of the emmer/goat grass cross was the addition of 2 or 3 genes that upgraded the proteins that create elasticity in the dough when kneaded. This upgrade in the quality of the gluten in wheat was what makes wheat so uniquely suited for bread-making.

But note that this hybridization resulted in a plant that produced grain that had a new molecular structure in some of its proteins. Humans had not evolved with these proteins. Consequently, they could not completely digest some of the proteins in wheat. Normally, digestion results in breaking down the molecules in food into individual amino acids so that they can be used by the body as building blocks for new tissue. But digestion of wheat results in incomplete digestion. Molecules are composed of long strings of amino acids, and the human digestive system is not capable of completely cleaving parts of the gluten molecules in wheat. This results in certain short and medium-length chains of amino acids (known as peptides). These peptides provoke increased intestinal permeability (leaky gut) and they manage to escape the digestive system and get into the bloodstream of anyone who is sensitive to wheat gluten. They are the apparent cause of celiac disease.

Some wanna-be authorities claim that advancements in wheat breeding methods to increase gluten content in wheat that were made roughly half a century ago are the main cause of a recent perception that gluten sensitivity is increasing. That's simply not true.[6] The major changes in wheat genetics were made in that second prehistoric hybridization when bread wheat (*Triticum aestivum*) was created by the cross-breeding of emmer wheat and a species of goat grass (*Aegilops tauschii*). Every modification in the wheat genome that has been made since that second hybridization event can only be described as part of a series of comparatively minor refinements, whether the changes have occurred as a result of natural selection, or genetically modified organism (GMO) technology. The increase in celiac diagnoses that has occurred since then is due more to increased awareness of celiac disease, than to any changes in wheat seed breeding. As physicians become more aware of a medical condition, they are more likely to look for it in their patients, and this naturally boosts the diagnostic rate.

It's the gluten in wheat that causes celiac disease.

Gluten is primarily made up of two protein fractions, namely prolamins (storage proteins) and glutelins. All grains contain a form of gluten, but only the gluten in wheat, rye, and barley have been pinpointed as triggers for celiac disease.

The prolamins in wheat are called gliadins and the glutelins are known as glutenins. Both of these protein fractions have been shown to cause adverse reactions in people who are sensitive to them. And as mentioned above most people who are sensitive to the gluten components in wheat are also sensitive to the prolamins in rye (known as secalin) and barley (known as hordein). In addition, many of those same individuals are sensitive to the prolamin (known as avenin) in oats. Rice is the only other grain for which a glutelin is commonly mentioned (orycenin), but very rarely are people sensitive to it, and it is not associated with celiac disease.

Wheat digestion results in many different gliadin and glutenin peptides to which celiacs are known to react, but the digestion of rye, barley, or oats produces far fewer reactive peptides. Altogether, this group of grains has been shown to be associated with hundreds of different peptides that can provoke immune system responses (from people who are sensitive to them) (Tye-Din et al., 2010).[7]

A description of celiac disease can be found in the medical literature of approximately 2,000 years ago (Guandalini, 2007, summer. p. 1).[8] But until the 1920s, the medical community was clueless as to what caused celiac disease, so they had no idea how to treat it. Considering that celiac disease is a relatively simple disease caused by a sensitivity to gluten, with a straightforward treatment (avoid gluten), why has it taken the medical community almost 2,000 years to begin to address it? And after about another hundred years, why is their overall understanding of the disease still rather rudimentary? Some sources estimate that the diagnostic criteria are so primitive that as few as 5% of all celiacs are ever even diagnosed. How could this be?

The likely reason for this problem is due to inadequate training of physicians. Traditionally, physicians have received very little (if any) training in diet and nutrition. They're taught to diagnose health problems and treat them either by surgery or by prescribing a drug-based treatment. Consequently they are unable to even intelligently consider the issues caused by food sensitivities in their patients. Even though food sensitivities are at the root of many modern health problems, physicians are generally unable to even recognize the association, let alone understand how to properly treat food sensitivities.

Most doctors simply don't understand food sensitivities.
And they don't understand the differences between food allergies, food sensitivities, and other gastrointestinal issues well enough to be able to tell the difference. This was well illustrated by a study sponsored by several regulatory agencies of the U. S. government, as well as a number of industry organizations focused on food allergies (National Academies of Sciences et al., 2017). The study clearly showed that most medical professionals don't understand these issues and aren't equipped to treat them. They don't even have adequate testing methods available to them.

And part of the blame may be attributed to the excessive reliance of many physicians on their drug reps. Far too many physicians rely on their drug reps from the pharmaceutical companies to provide them with continued education in the form of up-to-date information on treatment methods. The drug reps can hardly be blamed for omitting information and training on any issue that does not require drugs as part of the treatment. It shouldn't be surprising that physicians don't understand gluten sensitivity very well, and that they often tend to overlook it, in view of the fact that it is so deemphasized in their continuing education.

So Which Ancient Grains Do Not Contain Any Gluten?

Wheat is a member of the grass family. In fact, all of the common grains that we use today were developed from ancient grasses. But not all of them share wheat's characteristics. The ancestors of modern grains such as corn, sorghum, and rice evolved independently from wheat, and normally they're safe for use by virtually all celiacs and MC patients. Likewise, there are a number of ancient grains still available that evolved independently from wheat, and they remain free of gluten. At least they contain no gluten in nature. As long as they are handled, processed, and stored properly, they should be safe options for most celiacs and MC patients. Some examples include millet, teff, amaranth, ragi, Job's tears, buckwheat, and quinoa (Kasarda, n.d).[9] Ragi and Job's tears (also called adlay) are special varieties of millet. Most of these have very tiny grain size, and they're available as whole grains and as flour. An online search will usually turn up a good selection of recipes.

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COVID-19 Update Especially for Microscopic Colitis (MC) Patients

Hyperglycemia is very common among Covid-19 patients.
Almost half of hospitalized Covid-19 patients had hyperglycemia when admitted, despite not having a prior diagnosis of diabetes, according to a study based on 605 patients in two hospitals in Wuhan, China (Tucker, 2020, July 13).[1] Researchers found that over 45 % of them had a fasting blood glucose level of 126 mg/dL (7.0 mmol/L) or higher.

What might we deduce from this? Hyperglycemia and type 2 diabetes are closely associated with a chronic magnesium deficiency. Therefore, this symptom appears to be due to COVID-19's ability to exploit poorly or inadequately-maintained immune systems.

Another study shows the value of higher vitamin D levels.
A study done in Israel showed that individuals who tested positive for COVID-19, and who had a prior serum test of their vitamin D level, were more than 50 % likely to have a 25(OH)D level below 30 ng/mL (McNamara, 2020, July 29).[2] Note that the U.S. National Institutes of Health (NIH) lists 20 ng/mL (50 nmol/L) or greater as “Generally considered adequate for bone and overall health in healthy individuals”(NIH, 2020, p. 1).[3] Obviously the Israeli study proves this recommended level to be woefully inadequate for maintaining resistance against COVID-19.

This research supports our position that we need to maintain a strong immune system in order to minimize our risk of becoming another COVID-19 victim. And that means at the very least, keeping our vitamin D and magnesium levels up well above NIH recommendations.

References

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