Brockwell Bake

Humans eating cereals, benefits, problems and solutions

Review of research into and incidence of problems in human cereal grain consumption with special reference to "heritage" wheats and celiac disease

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Definitions used:
Celiac disease: Also known as coeliac disease (outside US) and celiac sprue, often abbreviated to CD.
Heritage wheat: used here to refer to any wheats not including dwarfing genes introduced in the "Green Revolution" by Norman Borlaug working at CIMMYT from 1961 onwards. "Heritage wheat" means essentially the same as commonly understood as "heirloom wheat" in US, "ble paysannes" in French, "kultursorter" in Swedish and "Kultursorten" in German. "Modern wheat" is conversely used to refer to post-Green revolution "dwarfed" wheats.

"Heritage wheats" can be further divided into farmer selected and genetically diverse "landrace" wheats adapted to local agronomy and usage and pre-modern wheat "early cultivar" lines created by selection or hybridization by wheat breeders, branded and sold by seedsmen. Conversely "modern cultivars" refers to post Green Revolution commercial wheat lines.

Contents

Introduction

Cereals in the human diet and culture

Cereal cultivation is the cornerstone of farming and by extension human culture and civilization as we currently know it. Debates continue about the transition from hunter gatherer society to farming, was there a clear division or was the transition gradual and so forth. It now seems clear from the archaeological record that the immediate consequence for those who first adopted a sedentary farming life was a reduced quality of overall diet with less protein and less variety of foodstuffs. The main initial benefit was some security of food supply through winter and other lean periods with the ability to store food and this was mainly due to storing grain. However as the range of food from domesticated crops and then livestock expanded the diet balance tipped from hunter gatherers to farmers and it became more obvious farming could support many more people per hectare. As farming expanded hunting and gathering territories which may have already become over-crowded by humans were further eroded.

Grain storage and grain surpluses enabled the creation of specialists roles within the neolithic community including priest-astrologers to administer the farming calendar, utensil and tool producers and professional soldiers (in the first place to guard the grain store). Trade in grain provided the basis for expansion of trade and the creation of trading and administrative centres, the beginnings of towns and then cities.

The adoption of cereals and in particular wheat to take the central position in providing protein and the largest part of numerous other vital trace elements in the human diet worldwide has not been without its downsides, one commentator remarked even that “humans did not domesticated wheat, wheat domesticated humans”. Certainly those freed by grain surpluses to take up specialists roles soon exploited their position to rule over others.

FERMENTED CEREALS. A GLOBAL PERSPECTIVE…

2013/14 world production
estimate in millions of tons - International Grain Council

maize	991
wheat	713
rice	476
barley	145

2012 world production
in millions of tons - FAO stats

maize	872
rice	734
wheat	671

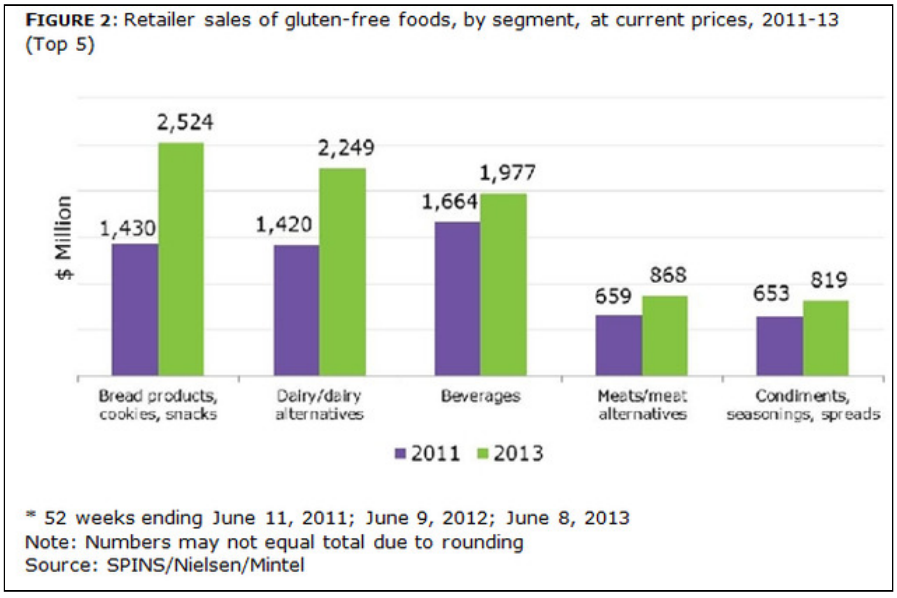
Current world wheat production is second or third in tonnage for cereals however most maize is used as animal feed and the protein content of rice is considerably less than wheat. Wheat can therefore be described as the most important crop in the current human diet contributing some 20% of calorific value.

Whilst it still remains key constituent in most humans' diets our modern relationship to wheat and cereals in general is ambivalent and detached with a very small percentage of the population now involved in wheat's cultivation and considerably less than in previous eras in its processing into food. There is an almost overwhelming pressure to apply capital and research into making wheat and other cereals an ever more abundant yet cheaper and cheaper to produce and process food whilst extracting maximum profit from capital expenditure. Because so few people are directly involved in the wheat crop to table process nowadays the possibilities of informed scrutiny of modern production techniques is diminished. The threat in the driving down of cereal cultivation and processing costs is that we may have overstepped the nutritionally beneficial bounds of cereals defined by their original prime quality as a foodstuff, their stability in storage.

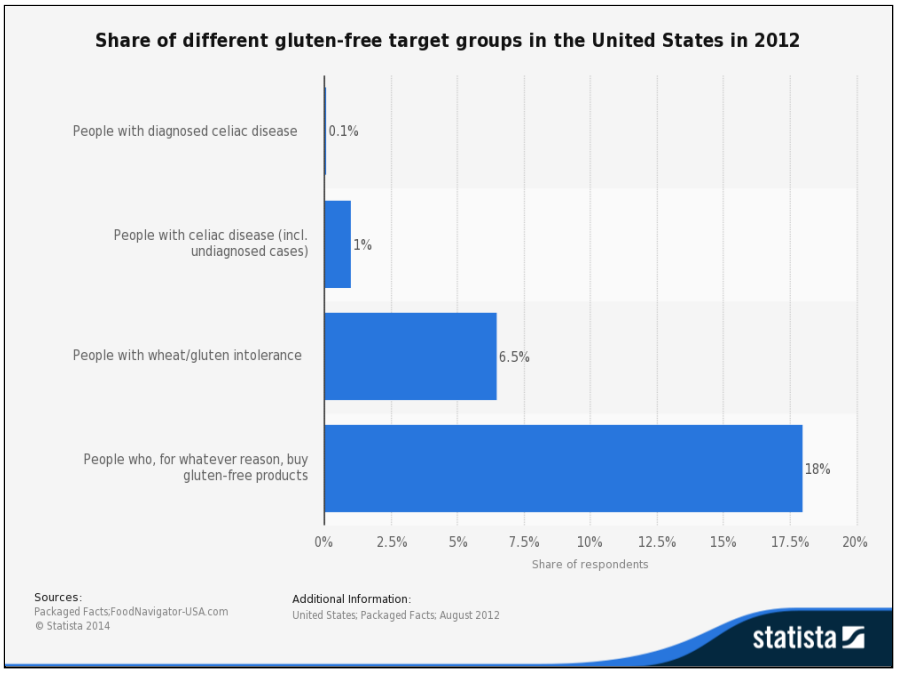
The "Gluten-Free" phenomenon

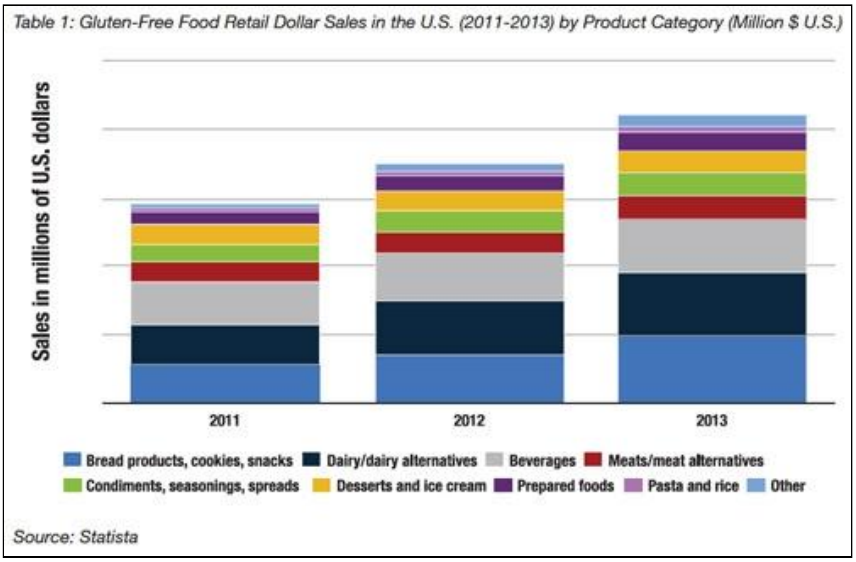
The gluten-free food product market has passed from the preserve of specialist wholefood shops and chemists to occupying complete aisles in major supermarket chains in many developed countries. Consumption is spreading from those who have diagnosed gluten digestion problems and those who think they have undiagnosed 'medical' gluten digestion problems to a wider consumer pool which simply perceives a gluten-free diet as more healthy.

A return to a pre-neolithic hunter gatherer style of diet is advocated by some but it is evident that on the one hand we lack the culture to re-create this diet and on the other hand that the resources for this style of diet are severely limited and can be the preserve of only a small fraction of the current world population. Indeed its evident from human genetic changes that have occurred that we are for the most part adapted to a cereal based diet and this cannot be reversed short of some human cataclysm.



US gluten-free market 2011 v 2013





This trend for ‘gluten-free’ is a public health concern - are the health claims verifiable? Are there costs and risks associated with gluten-free diets? “Gluten-free” is also obviously also a concern for those producing products containing gluten principally arable farmers, millers and bakers

The current document seeks to review these health claims with particular attention to epidemiology of wheat intolerances and possible connections to heritage v “modern” wheats.

The context - cereal grain biology

That some humans should have problems digesting cereals and in particular wheat should not be a surprise. Cereals formed the core of the earliest farming precisely because of their suitability for storage compared to other food sources.

Biologically cereal grains are in volume per grain mostly a food source for the germ to feed on when conditions are favourable, which in some cases can be a wait of more than one year in wild relatives. Until germination this food bank is effectively locked and protected against predators either animal or biological. The bran layer with its cellulose content is almost an armour plating, the endosperm starch is relatively unattractive compared to the sugars that will be produced from it by amylase activity at germination meantime enzyme-inhibitors protect the energy store, storage proteins such as glutenin and gliadin are insoluble in water, phytic acid is the insoluble store of phosphorus to be unlocked at germination by the enzyme phytase, until when it binds to itself trace elements making these unavailable to any predator's digestion and acts as a proteolytic enzymes inhibitor, effectively becoming an anti-nutrient. Perhaps most obviously to a degree that it gets overlooked, ripe grain is very hard and very dry.

The earliest farmers were able to utilise grain as a food source because they learnt to unlock the grain's food bank by first developing tools to smash/mill the grain (or alternatively sprout grain), developing pre-cooking processing of the resulting meal from mill or quern usually by fermentation and finally cooking and/or baking. Other elements of early farming that were also dependant on the grain store were chosen because of their own inherent methods of unlocking the grain's energy, the ruminants, sheep, goats and cows with their own supply of phytase and cellulases produced by specific microorganisms to digest cellulose. Still another element of farming, the cat, became domesticated in order to defend against those predators who could attack the granaries contents.

There is plenty of scientific evidence that humans did undergo some genetic changes and/or selection with the introduction of farming in order to start eating large quantities of grain products and the unevenness of these changes in the current human population indubitably explains in particular part of the picture for celiac disease. Nonetheless if we part from the mechanics of traditional processing of grain to food established by previous generations we can expect adverse effects for digestibility of foods containing grain. Similarly substantial changes in those aspect of the grain which defend it from predators pre-germination, the enhancement of quantity or 'quality' of gluten content for example, might be expected to possibility adversely affect human digestibility even with application of traditional processing.

We have to question and re-evaluate those developments of wheat as it is typically grown currently for which the primary driving force has been extractable profit rather than long term nutritional value and agronomic sustainability criteria that largely applied in previous epochs.

Gluten intolerances

Human health problems associated with encountering gluten can be divided into 3 categories, **celiac disease**, **wheat intolerance** or 'sensitivity' sometimes referred to as non-celiac gluten sensitivity (NCGS) and **wheat allergies**.

Wheat allergies

Wheat allergies, like hay fever and other allergies, develop when the body's immune system becomes sensitized and overreacts to something in the environment that typically causes no problem to most people. Symptoms can be induced by digestion of or external contact with wheat and include hives or skin rash, nausea, stomach cramps, indigestion, vomiting or diarrhea, stuffy or runny nose, sneezing, headaches, asthma, Anaphylaxis (less common), a potentially life-threatening reaction that can impair breathing and send the body into shock. Wheat allergy is most common in children, and is usually outgrown before reaching adulthood, often by age three. Individual sufferers will be allergic to different elements of wheat, individual gliadins (omega-5 gliadin most commonly) and glutenins in gluten but also in some cases to the pollen and the plant. Food service workers especially bakers and millers can develop respiratory focused allergy to wheat and other cereals flour which has been also associated with the common flour additive, fungally grown amylase.

Prevalence of wheat allergy is hard to clearly report and it is certain more people think they are allergic to wheat (4.5% in 2009 UK study) than actually are.

Table 2. Prevalence of self-reported and confirmed allergy to any food and to wheat.

	Food allergy or intolerance		Wheat allergy or intolerance	
	Self-reported	Confirmed	Self-reported	Confirmed*
1 yrs	At 1yr: 7.2% (11)	≤1yr: 2.5-2.8% (11)	0.4% (11)	0.3% (11)
3 yrs	8.3% (12)	5-6% (12)	0.3% (12)	0.2% (12)
6 yrs	11.8% (13)	1.6-2.5% (13)	1.3% (13)	0.4% (13)
11 yrs	11.6% (14)	1.4-2.3% (14)	1.3% (14)	0.0% (14)
15 yrs	12.4% (14)	2.1-2.3% (14)	1.2% (14)	0.1% (14)
>18 yrs (1994)	20.4% (8)	1.4-1.8% (8)	0.9% (8)	-
>18 yrs (2009)	21.3% (9)	-	4.5% (9)	-

*Confirmed on the basis of a positive or negative skin prick test and performing food challenges over a one week period provided 4-6 portions of wheat in a blinded fashion i.e. both food allergy and intolerances were investigated in this trial.

source "NABIM Wheat allergy and intolerance report" 2010

It has been clearly shown that celiac disease sufferers must have a specific genetic pre-disposition (see below) but this has not been proven for wheat allergies.

Wheat intolerance

“Wheat intolerance” also referred to as “wheat sensitivity” or more precisely as non-celiac gluten sensitivity (NCGS) is the most difficult to pinpoint of wheat/human contact problems, perhaps most usefully defined for the time being as a problem with consumption of gluten which is not diagnosable as celiac disease or as a wheat allergy. At the moment there is no diagnostic test for it but symptoms are reported as feeling bloated and gassy; and experiencing abdominal pain, diarrhea, and abdominal cramping. These symptoms are also typical for celiac disease therefore any sufferers should get tested for this in the first instance as CD can have severe general health consequences including fatal. “Wheat intolerance” blends into the condition Irritable Bowl Syndrome for which also there is no diagnostic test. By some estimates over a fifth of people will experience some period of “Irritable Bowl Syndrome” during their lives.

A possible subgroup of those suffering non-celiac gluten sensitivity i.e. of those who have symptoms akin to classic celiac disease but do not test for celiac disease it has been suggested by recent research are reacting to wheat amylase trypsin inhibitors (ATIs) rather than the typical gliadin epitopes of celiac disease. ATIs are in the wheat grain to defend against pest attack and it may be significant that a focus of modern wheat breeding has been to increase the ATI content of HYV wheats.

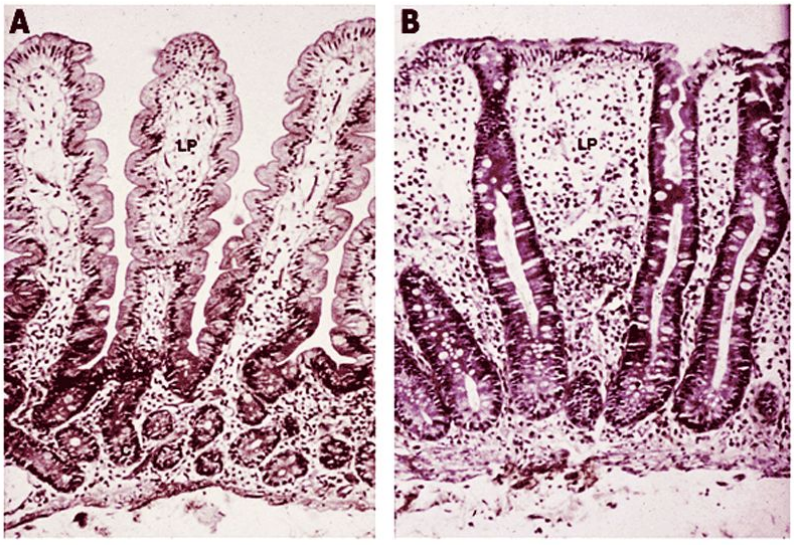
http://en.wikipedia.org/wiki/FODMAP

Celiac disease

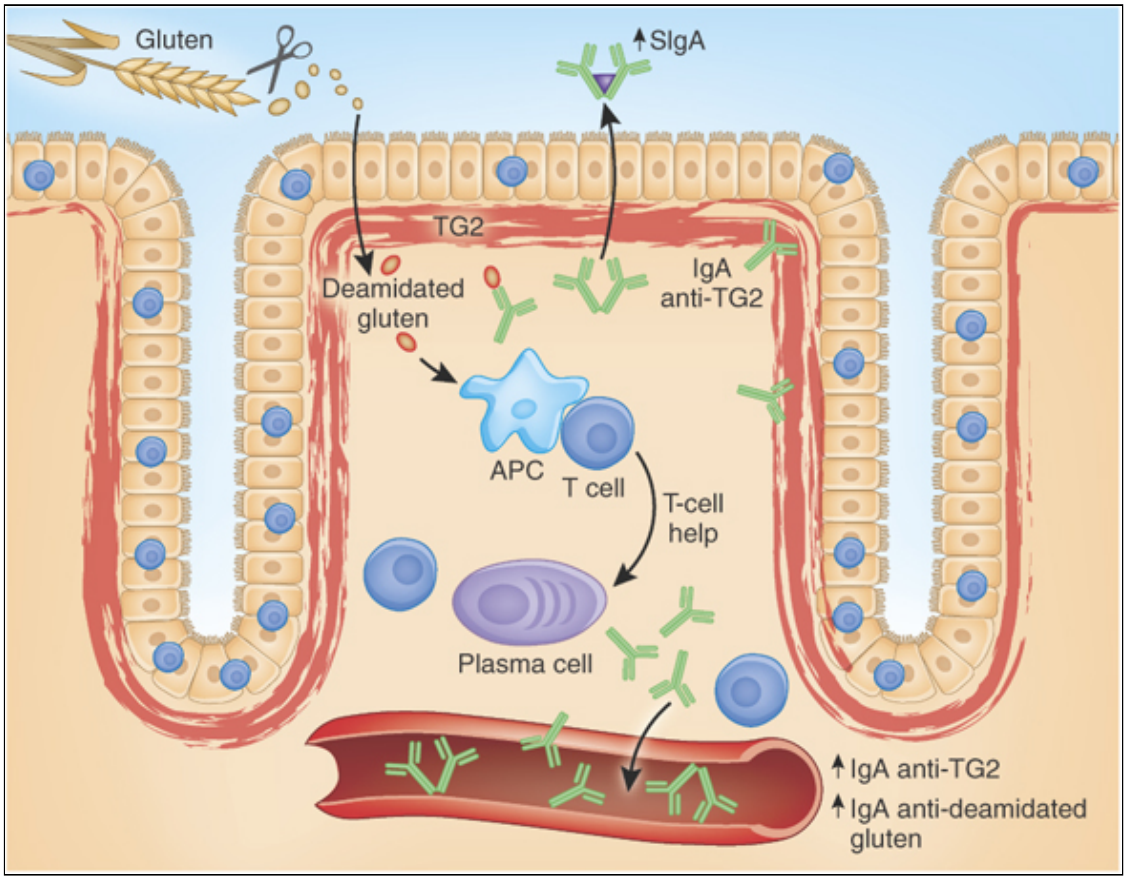
The disease is is an autoimmune disorder of the small intestine that occurs in genetically predisposed people of all ages from middle infancy onward. These symptoms may include iron deficiency that results in a low number of red blood cells (anemia), vitamin deficiencies, low bone mineral density (osteoporosis), itchy skin rashes (dermatitis herpetiformis), defects in the enamel of the teeth, chronic fatigue, joint pain, poor growth, delayed puberty, infertility, or repeated miscarriages and ‘failure to thrive’ as a child. Neurological problems have also been associated with celiac disease; these include migraine headaches, depression, attention deficit hyperactivity disorder (ADHD), and recurrent seizures (epilepsy). Celiac disease untreated is potentially life threatening.

Celiac disease is caused by a reaction to gliadin, a prolamin (gluten protein) found in wheat, and similar proteins found in other domesticated cereals from tribe Triticeae including rye, barley and oats though most celiac sufferers have less reaction to oats. Upon exposure to gliadin, and specifically to three peptides (which are therefore epitopes for CD) found in the prolamins, the enzyme tissue transglutaminase modifies the protein, and the immune system cross-reacts with the small-bowel tissue, causing an inflammatory reaction. That leads to a truncating of the villi lining the small intestine (called villous atrophy). The effect has been compared to small intestine lining that should look like a deep shag pile carpet instead resembling a parquet floor. This interferes with the absorption of nutrients because the intestinal villi are responsible for absorption. The only known effective treatment is a lifelong gluten-free diet. Repair of the villi once on a gluten-free diet can take between one and three years. Some cases known as “refractory CD” fail to respond to the gluten-free diet treatment and the consequence inevitably is premature death.

Incidence of celiac disease has been described as being like an iceberg in that the vast majority of sufferers go undiagnosed, in Europe in range of 1:5 to 1:13. It is the most common diagnosable problem associated with wheat consumption. If answers and solutions for celiac sufferers can be found other than wheat (gluten)-free diet it would seem quite conceivable that other more ill defined problems might also be solved and therefore the present document tries to pay particular attention the issues around CD.



healthy villi left and CD damaged villi right



The epitopes that CD sufferers produce antibodies that have now been identified are numerous. Gluten peptides resulting from partial digestion of all gluten protein groups (α/β-, γ-, ω-gliadins, low molecular weight glutenins, and high molecular weight glutenins) may contain T-cell stimulatory epitopes (Koning 2008; Stepniak et al. 2008), but the epitopes from the α-gliadins are considered to have by far the highest clinical relevance with regard to both the adaptive immune response and the innate immune response that lead to the development of CD (Sjöström et al. 1998; Arentz-Hansen et al. 2000a, b, 2002; Anderson et al. 2000; Janatuinen et al. 2002; Vader et al. 2002; Maiuri et al. 2003; Molberg et al. 2003; Schuppan et al. 2003; Qiao et al. 2005; Marti et al. 2005; Camarca et al. 2009). The Glia-α9 epitope is especially known as a major immunodominant epitope that can be recognized by the majority of CD patients (Vader et al. 2002; Camarca et al. 2009). The Glia-α9 epitope sequence (αI) is part of the proteolytic-resistant 33-mer in α-gliadins that has a strong T-cell stimulatory effect (Shan et al. 2002; Shan et al. 2005).

Genetics of celiac disease incidence

The risk of developing celiac disease is increased by certain variants of the HLA-DQA1 and HLA-DQB1 genes. These genes provide instructions for making proteins that play a critical role in the immune system. The HLA-DQA1 and HLA-DQB1 genes belong to a family of genes called the human leukocyte antigen (HLA) complex. The HLA complex helps the immune system distinguish the body's own proteins from proteins made by foreign invaders such as viruses and bacteria.

The proteins produced from the HLA-DQA1 and HLA-DQB1 genes attach (bind) to each other to form a functional protein complex called an antigen-binding DQαβ heterodimer. This complex, which is present on the surface of certain immune system cells, attaches to protein fragments (peptides) outside the cell. If the immune system recognizes the peptides as foreign (such as viral or bacterial peptides), it triggers a response to attack the invading viruses or bacteria.

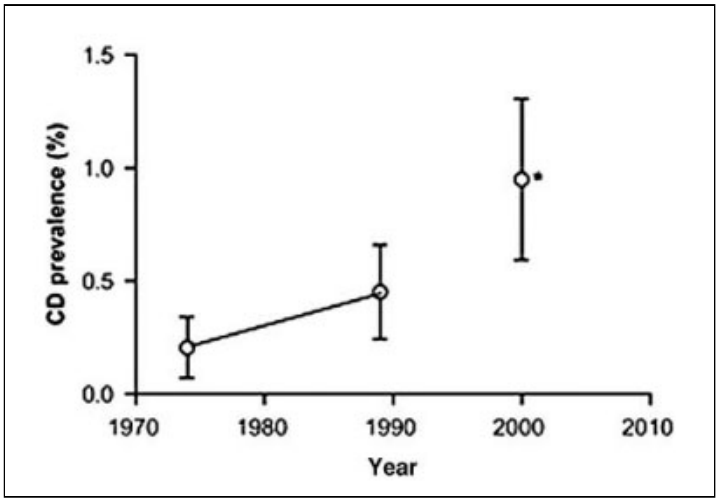
Almost all people with celiac disease test positive for HLA-DQ2 and HLA-DQ8 genes. These variants of HLA-DQA1 and HLA-DQB1 genes in turn are part of the HLA A1-B8-DR3-DQ2 haplotype which is a multigene haplotype that covers a majority of the human major histocompatibility complex on chromosome 6 also known by the nickname “Super B8” from the HLA-B8 portion and referred to as the ancestral European haplotype because of its common occurrence in Europe. However whereas the HLA A1-B8-DR3-DQ2 haplotype is more prevalent across all Europe than on other continents it is still more prevalent within Europe’s further North and West reaches, Ireland, the Basque country and Scandinavian countries. This suggests a negative selection with later arriving Europe colonizers and/or a farming culture, assumed to be cereal cultivation, but an earlier positive selection amongst earlier European colonizers for this halotype, possibly relating to a high shellfish diet. Prevalence of the HLA-B8 serotype in various countries is listed here.

Epidemiology of celiac disease

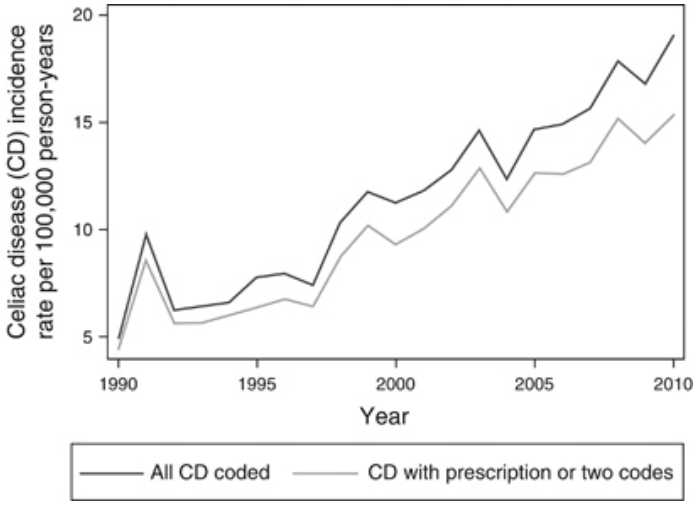
As a whole between 30% and 40% of Caucasians have the HLA A1-B8-DR3-DQ2 haplotype apparently making them candidates for celiac disease yet currently only <1% do test for celiac disease antibodies - diagnosed or undiagnosed, symptomatic or nonsymptomatic, however this incidence appears to be rising.

Diagnosis of celiac disease until relatively recently relied on patients presenting themselves with relevant symptoms followed by biopsies of the small-bowel to identify damage to the villi. However this damage can be patchy so may be missed by biopsy - currently at least tests on upto 6 biopsy site are recommended. About 20 years ago a much more sensitive and easier test was developed to find antibodies from the immune system in blood including those relating to celiac disease. Testing for Immunoglobulin A (IgA) tissue transglutaminase antibody (tTG) is now the preferred methodology in CD diagnosis though biopsies are still performed to confirm and establish progression of CD. From what had been considered a relatively rare problem, celiac disease was revealed as one of the most common chronic diseases in some populations. A 1996 study in Italy of 17,201 students revealed 0.54% incidence confirmed by biopsy, the ratio of previously undiagnosed to previously diagnosed was 1:7. A consequent 2010 European wide study of 29,212 people gave an overall 1% incidence with large variations between countries, 2.0% in Finland, 1.2% in Italy, 0.9% in Northern Ireland and 0.3% in Germany.

The most commonly quoted figure for celiac disease incidence in the US is now 1:133. However a 2010 study using stored serum showed an increase in celiac disease incidence in the US with 1:501 in 1974, 1:219 in 1989 and 1:105 in 2001. In another 2009 US study preserved blood samples from 1950's US airmen were compared with 90 year old retired airmen and current airmen with same age as participants from the 1950's sample. The 9,133 1950s sample showed 0.2% CD incidence, the retired airmen showed 0.9% and amongst current airmen 0.8%. A 2104 UK study showed a fourfold increase in celiac disease diagnosis between 1990 and 2011 but with strong regional variations. One implication of these results is that what had been considered a lifelong problem from childhood was revealed to be also a problem for older people who had previously tested negative for celiac disease antibodies. Another implication is that from being a disease that afflicts currently perhaps 1% of a genetically predisposed general population (+3% of those specifically genetically disposed within such a population) when certain gliadin peptides were present in the diet, other factors are needed to be found to explain the fivefold increase over 40 to 50 years in these studies. Without finding these factors it is difficult to predict future incidence of celiac disease or to put this increase into reverse though some studies point to a current plateauing of the increase.



Prevalence of celiac disease by antibody screening in the US 1974 to 2001



Prevalence of celiac disease diagnosis in the UK 1990-2011 (possibly 1:7 of actual sufferers)

Potential causes of CD and other gluten intolerances

Whilst two pre-conditions for celiac disease are known, consumption of cereals and genetic disposition, obviously most Western hemisphere people fulfill the first condition and between 30% to 40% of Caucasians fulfill the second yet at most 1:100 of this pool of potential sufferers get celiac disease, diagnosed or undiagnosed. Therefore a further condition must be supposed and since the evidence is that celiac disease is increasing, fulfillment of this condition or conditions must be on the increase also.

A number of “life events” have been reported as triggers for onset of celiac disease including severe gastrointestinal infection, pregnancy, severely emotionally testing episodes, however these are generally “life events” that have always existed so are unlikely to explain increasing incidence of celiac disease. A number of factors that may have may have had an increasing impact from mid 20th C into the 21st C have been suggested, including **changes in gut flora and immune system development**, especially relating to breast feeding and post-weaning diet, changes in diet related to **quantitative and qualitative changes in content of wheat** and **changes in processing to food of wheat**.

Factor: Gut flora and immune system development

A 2008 study compared results of celiac disease antibody tests of 3654 Finnish children with those of 1988 children from Russian Karelia, positive antibody tests were followed by duodenal biopsies showing 5 times higher celiac disease prevalence in Finland. Given that Russian Karelia is next to Finland, the ethnic and cultural background of the populations is the same and that if anything the Russian Keralians eat more wheat compared to rye than the other side of the border neither diet nor genetic makeup explain this difference. The study concluded that the much lower living standard and lower childhood hygiene standards in Russian Karelia compared Finland were responsible. Other autoimmune syndromes such as Type 1 autoimmune diabetes and a range of allergies were also up to 6 times less likely in Russian Karelia. At the time of this research, roughly a decade ago, Russia's per-capita income was one-fifteenth of Finland's. Analysis of house dust and potable water suggests that the Russian Karelians encountered a greater variety and quantity of microbes, including many that were absent in Finland. Not surprisingly, they also suffered from more fecal-oral infections. For example, three of four Russian Karelian children harbored Helicobacter pylori, a corkscrew-shaped bacterium, while just one in 20 Finnish children did.

The theory to explain this finding is that modern developed countries childhood environment and diet has become so sanitised that the developing immune system does not get challenged by the pathogenic bacteria, viruses and parasitic intestinal worms it is designed to encounter and learn to defend against and therefore finds other false and auto targets. This is sometimes known as the "hygiene hypothesis" first articulated by 2001 Lancet study that showed children from large families and/or farm based families had less incidence of hay fever. Positive selection for the HLA A1-B8-DR3-DQ2 haplotype responsible for most autoimmune diseases in Europe may be explained by the increasing scale and 'cramptness' of human colony populations in late Paleolithic and very early Neolithic eras, the genetic selection for an immune system in early childhood champing at the bit to tackle a multitude of challenges due to poor hygiene which in a more sanitised environment behaves like a young dog without exercise and appropriate stimulation.

Alongside these findings an epidemic of serious celiac disease in under 2 year olds occurred in Sweden starting between 1985 and 1987 rising to 200-240 cases per 100000 followed in 1995 by a sharp decline back to the previous 50-60 cases per 100000 persons. Research revealed that the epidemic ironically occurred when the Swedish government, in an effort to reduce celiac disease, recommended wheat should not be introduced to babies diets till six months old, coincidentally this was the typical age at which Swedish mums weaned their children and at the same time as this government directive occurred manufacturers happened to increase the wheat content of baby foods.

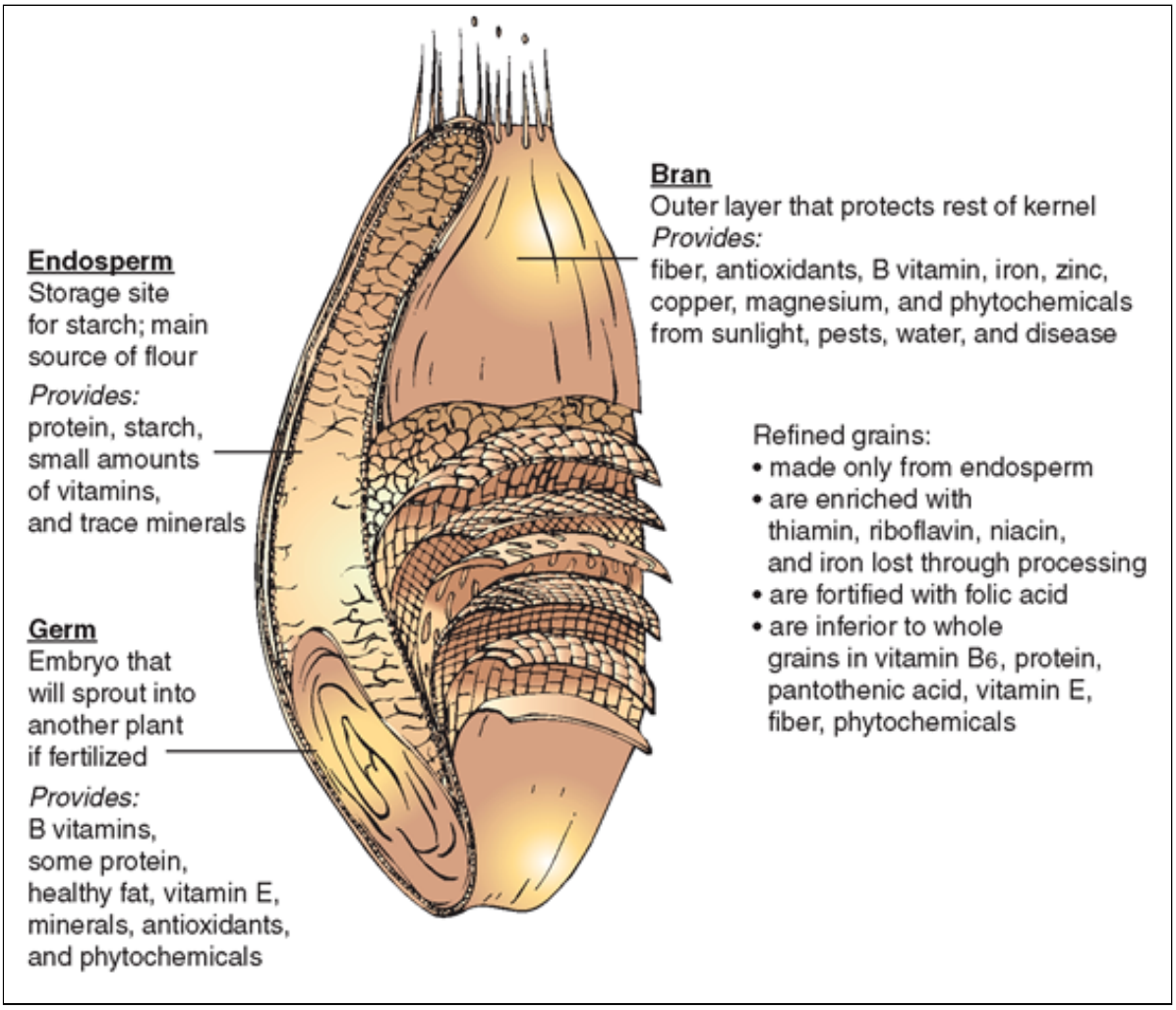
Changing the recommendation to overlap introduction of wheat with breast feeding combined with a reduction in wheat in the baby foods appears to have caused the subsequent decline in celiac disease back to previous level. The causal mechanism appears to centre around levels of beneficial bifidobacteria in the infant gut with studies revealing reduced levels in celiac sufferers which can be rectified by breast feeding which provides protective antibodies and immune-signaling proteins and prebiotic sugars targeted to feed bifidobacteria and other beneficial microbial gut flora. Use of antibiotics is also implicated in reducing beneficial gut flora. The coincidence of reduced breast-feeding and antibiotics in developed countries post-WWII may give one set of factors that match the dating of the reported modern increase of celiac disease.

Much of the above from NYT article here

Factor: changes in wheat and its processing to food

A four to fivefold increase in the incidence of celiac disease since the 1960's has been reported by various studies in developed countries. Factors that could explain this include changes in the processing of cereals into human food. Most cereals are consumed after first **milling** then some further **preparation** followed by a **cooking or baking** process. Essential to this processing of cereal grain into human food is understanding the locked down and guarded nature of a cereal grain's food/energy reserve and the biochemical process of unlocking this food supply for germination or for processing to food.

The biochemistry of the cereal grain



enzyme activity in grain and flour

In the wild progenitors of wheat the rachi connecting seed to stem shatters on ripening and seed inside its glumes (hull) with awn (beard) attached is carried away from the parent plant by wind and water. In the dry lands of their origin the objective was to find some crack in a dried up wadi into which to insert oneself, directed by the awn and drilled down by small hairs on the glume which rise and fall with morning dew, then wait for rain. With a good fall of rain in its new location comes the time to unlock and unpack the carefully guarded energy pack which is over 80% of grain with the objective of getting established below and above ground as quickly as possible. A one night soaking is the time taken to germinate wheat, the resulting explosion of energy released is remarkable, the heat given off by a pile of germinating grain is considerable. The keys to this unlocking process are enzymes.

Enzymes are highly selective biological catalysts creating chemical reactions to convert starting molecules (substrates) into different molecules (products). Enzyme inhibitors can stop or slow down enzyme activity, activators get them going. Different enzymes may have different requirements for activity such as pH level, temperature, availability of specific trace elements for their activity and so on.

Wheat grain contains a host of enzymes to aid germination each specific to a particular substrate **amylases** (starches to sugars), **glucanases** (glucan to glucoses), **proteases** (proteins to amino acids by hydrolysis of the peptide bonds), **phytase** (hydrolysis of insoluble phytic acid to release inorganic phosphorus and chelated trace elements), **lipases** (hydrolysis of fats and oils), **lipoxygenase** (hydrolysis of fats and oils) and **catalases** (hydrogen peroxide to oxygen and water).

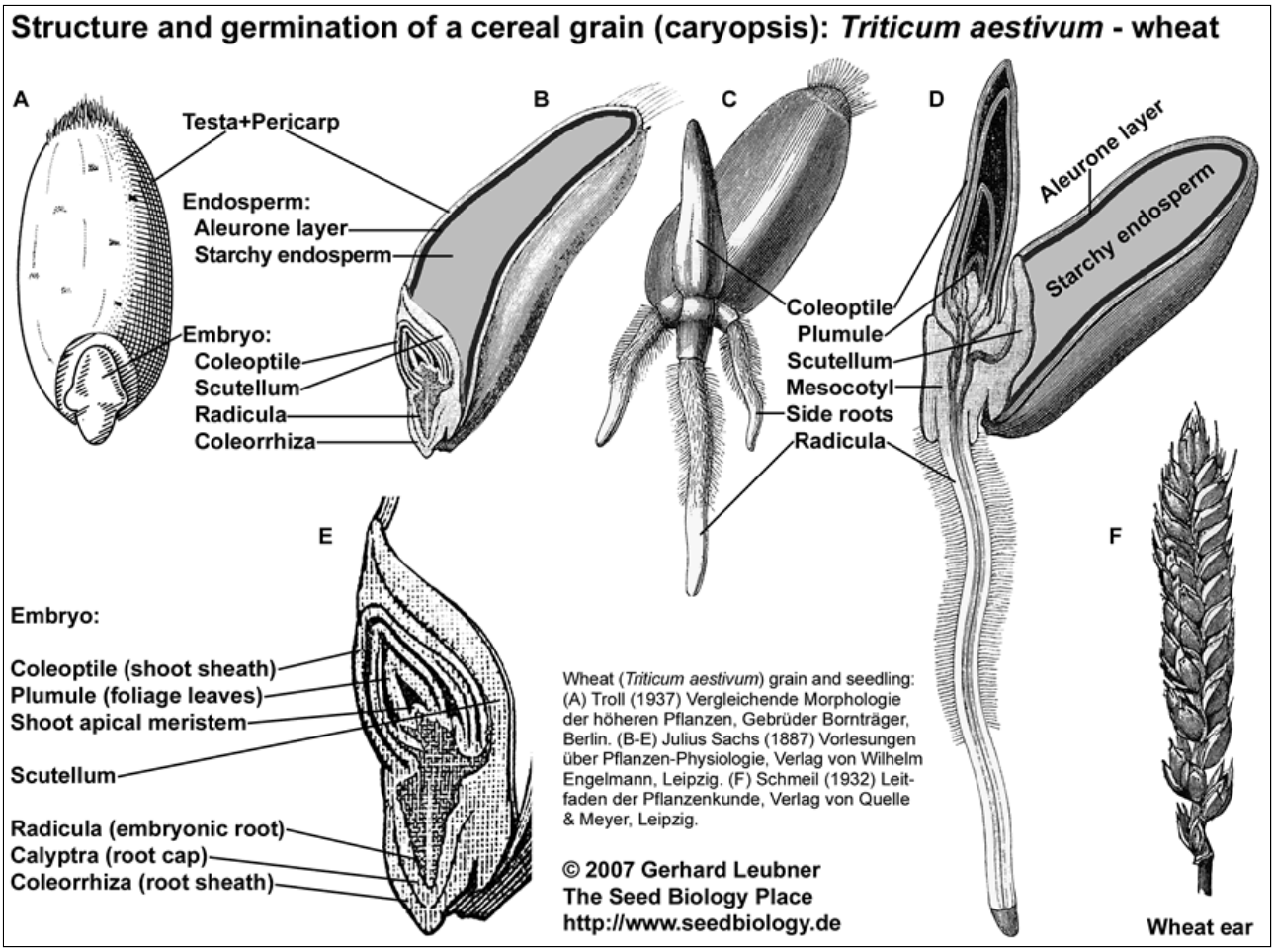
amylases

Bakers are probably most aware of the **amylases** which serve to convert the starch of the endosperm portion of the grain into sugars at germination to power early phases of plant growth. Endogenous (from inside) grain amylases are divided into **α-Amylases** (alpha) and **β-Amylases** (beta).

α-Amylases produce maltose and other sugars by cutting at random along the starch chain and therefore work quicker than β-Amylases which can only chop sugars off an end of starch chains. α-Amylases require the presence of calcium. α-Amylases are present in the grain prior to grain ripening but then die away till germination. Between ripening and germination the embryo and the aleurone layer immediately underneath the bran layers are the parts of the grain that remain alive, at germination signals from the plant hormones gibberellin (GA) and abscisic acid (ABA) emanating from the scutellum between embryo and endosperm initiate the renewed synthesis of α-Amylases in the aleurone layer. After generation of α-Amylases at germination the aleurone layer cells are programmed by further GA and ABA signals to die.

β-Amylases work to produce two units of maltose at a time off the non-reducing end of starches. They are stored ready to be activated by hydrolysis in the endosperm proportion of the grain between ripening and germination.

Amylase inhibitors have been found in wheat that do not act on its own amylases but target amylases found in mammalian saliva (including our own) and those of the yellow mealworm and mites. These can act as antigens in human allergic reactions including baker's asthma.



phytic acid, minerals and phytase action in grain

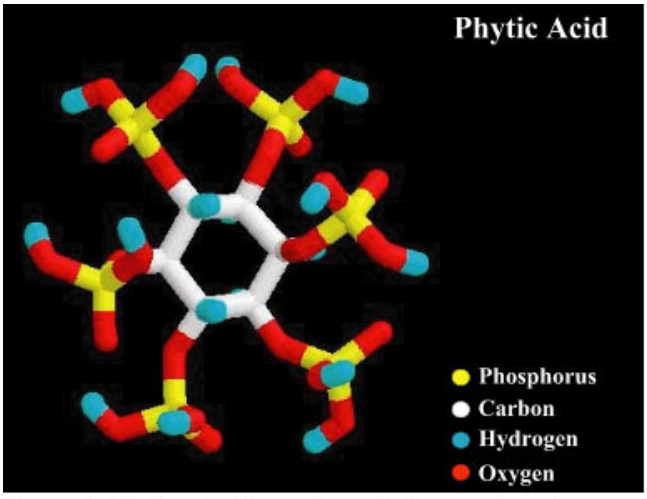


Figure 3. Phytic acid, the predominate storage form of phosphorus in mature seeds (figure courtesy of W. Schmidt – USDA/ARS).

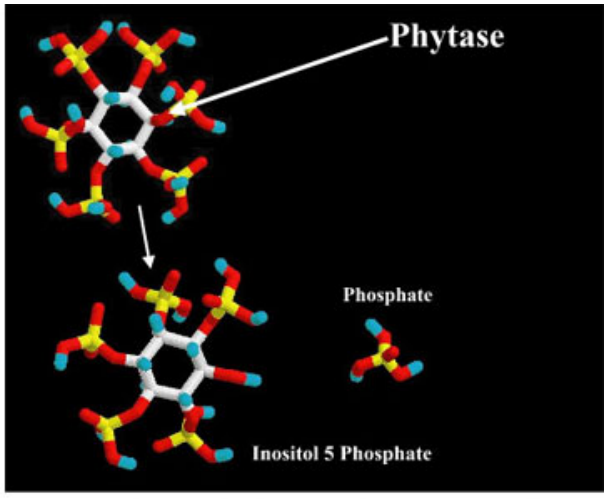
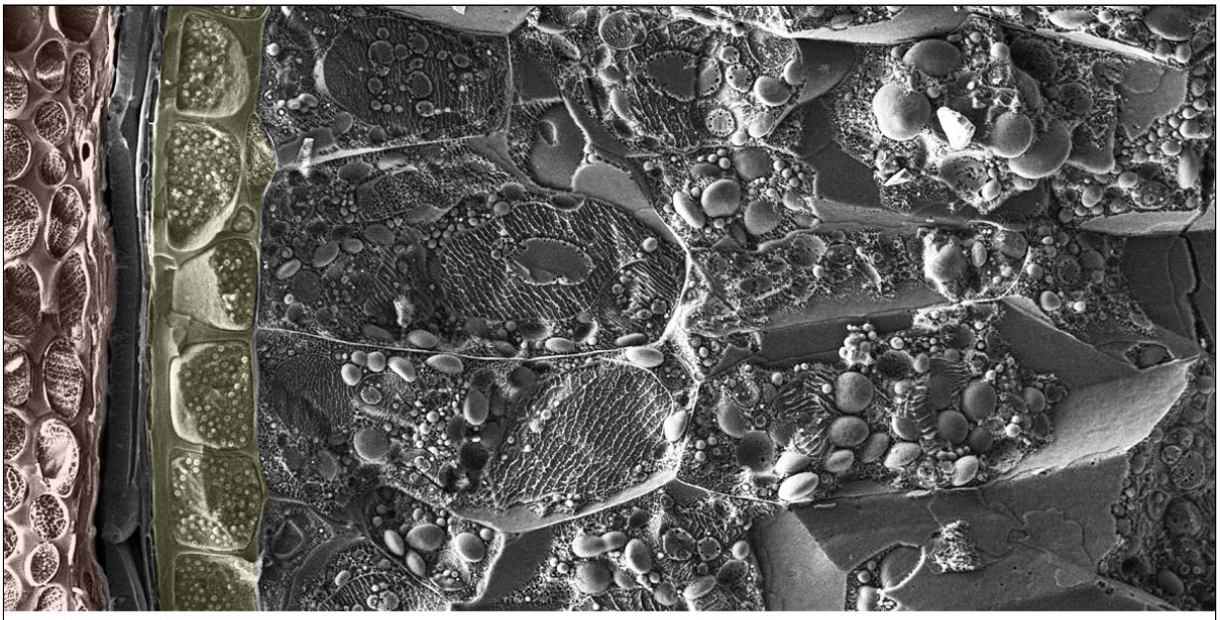


Figure 4. Diagram of release of phosphate from phytin by the enzyme, phytase. (figure courtesy of W. Schmidt – USDA/ARS).

Phytate is the salt form of phytic acid (also known as inositol hexakisphosphate (IP6)). **Phytate** is used by plants and in particular grains and nuts to store phosphorous, inositol and a variety of minerals and it accounts for 75–80% of the total phosphorus in seeds. Phosphorus is necessary component for all life on Earth present in DNA, RNA and ATP, the second most common mineral in the human body after calcium being essential to bone building together with calcium and critical to plant life and is the main component of fertilizers.

Phytate is insoluble and also binds to it (chelates) biologically essential trace elements including zinc, iron, copper, magnesium, manganese, potassium and niacin (vitamin B_3) making it an anti-nutrient for would be animate thieves. At germination phytate is cleaved by the enzyme phytase into inorganic phosphorus and any trace elements that were bound to the phytate will also become bio available.

Phytate is concentrated in the grain in the aleurone layer immediately under the bran layers (pericarp and testa) and this part remains alive along with the germ embryo between maturity and germination.



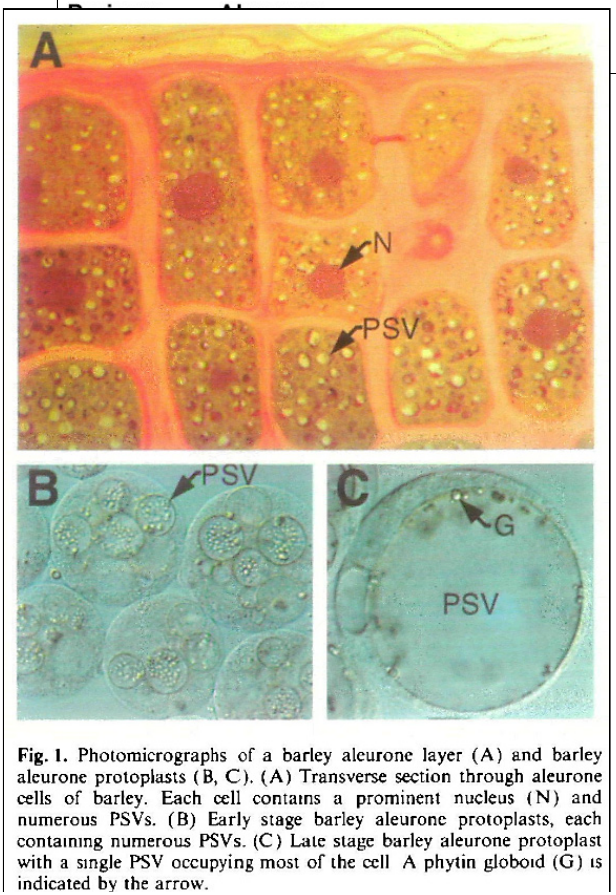


Fig. 1. Photomicrographs of a barley aleurone layer (A) and barley aleurone protoplasts (B, C). (A) Transverse section through aleurone cells of barley. Each cell contains a prominent nucleus (N) and numerous PSVs. (B) Early stage barley aleurone protoplasts, each containing numerous PSVs. (C) Late stage barley aleurone protoplast with a single PSV occupying most of the cell A phytin globoid (G) is indicated by the arrow.

Phytate salts as phytin ‘globoid’ crystals are stored between maturity and germination within numerous protein storage vacuoles (PSVs also known as aleurone grains) within the cells of the aleurone layer.

At germination using the plant hormones, gibberellic acid (GA) and abscisic acid (ABA) signals are emitted from the embryo across the scutellum layer separating embryo and endosperm and picked up by cells in the aleurone which respond by beginning the synthesis and activation of enzymes necessary for unlocking of the food store of the grain to feed the embryo on germination. In wheat GA signals go out into a neutral pH environment (mature barley endosperm before germination is already somewhat acidic) and then acidification of the aleurone and endosperm is initiated from the aleurone layer to create optimum conditions for a number of enzymatic hydrolysis processes. In wheat acidification proceeds from the embryo towards the distal end of the grain from pH 6.8 (neutral) to within pH 5.2-6 within 6 days of germination and then plateaus. Calcium is released by this acidification making α-amylase activity possible, phytase stored in the aleurone layer and also synthesised there de novo operates best at 55°C and at pH 4.5 to 5.0. The sugars, peptides, and amino acids produced by this enzymatic hydrolysis also require an acidic environment for their transport across the scutellum to the embryo.

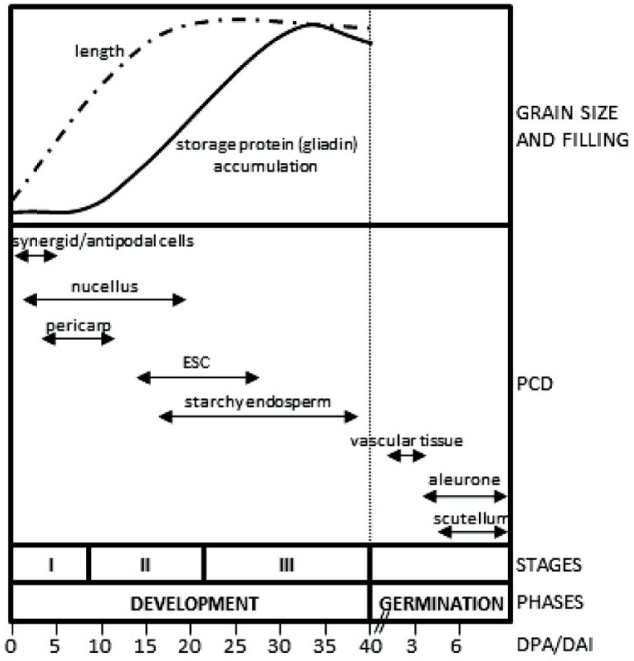
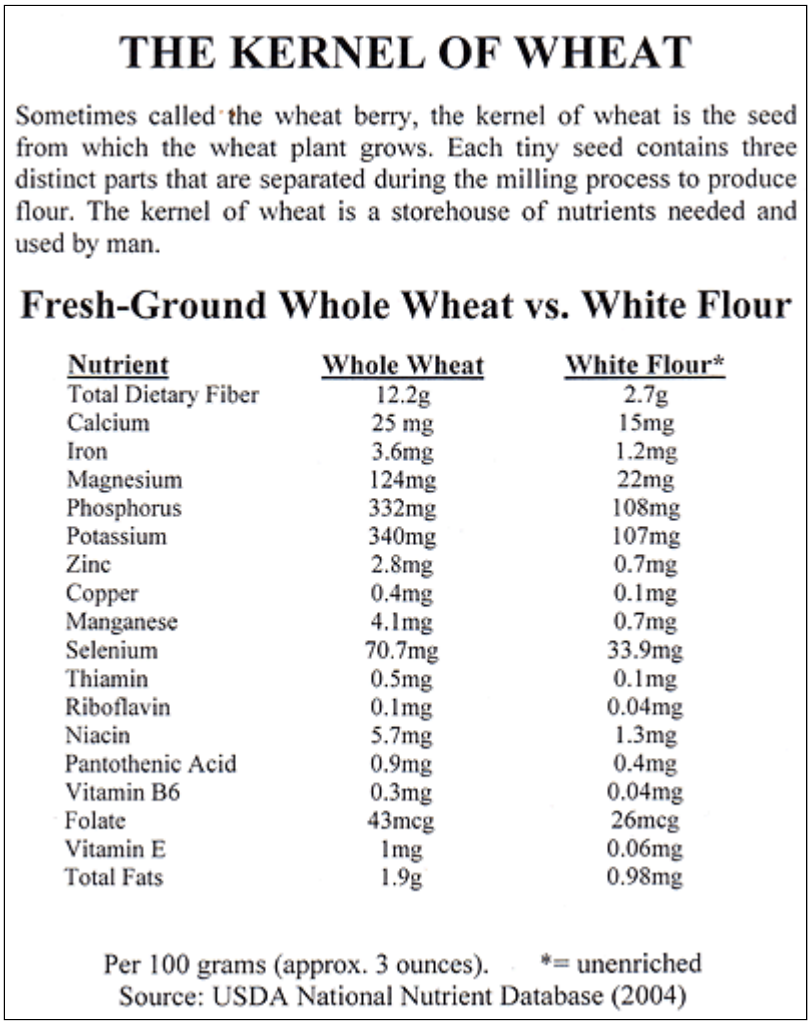


Diagram of Programmed Cell Death in cereal grain with days post anthesis (DPA) scale and days after imbibition (DAI). ESC = embryo-surrounding cells

Cells in the developing grain and in the grain post-germination that no longer have a function alive respond by dieing and their content is then used for to build other needed cells. Programmed cell death (PCD) occurs at in various areas of the grain at various stages in its life cycle. As the grain matures first the bran layer (pericarp) becomes dead and nutrients from this are transferred into the endosperm. When mature the endosperm portion of the seed dies and just the embryo and aleurone layer remain alive. After germination when the cells of the aleurone layer cells have completed the synthesis and secretion of hydrolytic enzymes they die. The cells of the scutellum are the last to die as they perform transfer of nutrients to the embryo so are needed till this is complete, proceeding from day 4 after germination till around day 7.

The degree to which sifting out bran and therefore much of the aleurone layer, which tends to adhere to bran particles when milled, reduces the quantity of trace elements in final flour is quite stark as the table below shows.



THE KERNEL OF WHEAT

Sometimes called the wheat berry, the kernel of wheat is the seed from which the wheat plant grows. Each tiny seed contains three distinct parts that are separated during the milling process to produce flour. The kernel of wheat is a storehouse of nutrients needed and used by man.

Fresh-Ground Whole Wheat vs. White Flour

Nutrient	Whole Wheat	White Flour*
Total Dietary Fiber	12.2g	2.7g
Calcium	25 mg	15mg
Iron	3.6mg	1.2mg
Magnesium	124mg	22mg
Phosphorus	332mg	108mg
Potassium	340mg	107mg
Zinc	2.8mg	0.7mg
Copper	0.4mg	0.1mg
Manganese	4.1mg	0.7mg
Selenium	70.7mg	33.9mg
Thiamin	0.5mg	0.1mg
Riboflavin	0.1mg	0.04mg
Niacin	5.7mg	1.3mg
Pantothenic Acid	0.9mg	0.4mg
Vitamin B6	0.3mg	0.04mg
Folate	43mcg	26mcg
Vitamin E	1mg	0.06mg
Total Fats	1.9g	0.98mg

Per 100 grams (approx. 3 ounces). *= unenriched
Source: USDA National Nutrient Database (2004)

The lack of various of these elements in white flour is the reason many countries legally oblige fortification of flour, in the UK four are required calcium carbonate (chalk), iron, thiamin (vitamin B1) and niacin. But to repeat again most of this long list of elements vital to good human health are not bioavailable to humans unless phytase has had a chance to separate them from phytate.

The “gluten-free” industry pumps out a lot of PR about the evils of bread wheat and phytate in particular, however much of their evidence turns out to be self-referencing or anecdotal and not to peer reviewed scientific standards. However there is a parallel stream of scientific and industrial study relating to livestock feed especially for non-ruminants i.e. those farm livestock without their own internal phytase supply produced by internal bacteria, pigs and hens. Pig and poultry are not of course always fed a high wheat diet and not all alternatives contain as much phytase potential as each other. Analysis in 1993 gives the following figures for phytase content rye (5130 units kg^{-1}), triticale (1688 units kg^{-1}), wheat (1193 units kg^{-1}) and barley (582 units kg^{-1}) with other feed stock has much less than these figures.

Phytate phosphorus content and phytase activity of some common feed ingredients.

Ingredient	Phytate P[a] (%)	Phytate P[a] (% of total P)	Phytase activity[b] (units kg^{-1})
Cereals and by-products			
Maize	0.24	72	15
Wheat	0.27	69	1193
Sorghum	0.24	66	24
Barley	0.27	64	582
Oats	0.29	67	40
Wheat bran	0.92	71	2957
Oilseed meals			
Soybean meal	0.39	60	8
Canola meal	0.70	59	16
Sunflower meal	0.89	77	60
Groundnut meal	0.48	80	3
Cottonseed meal	0.84	70	NA

[a]Data adapted from Ravindran (1996) and Ravindran *et al.* (1994, 1995b).
[b]Data from Eeckhout and De Paepe (1994). One unit is defined as that amount of phytase which liberates inorganic phosphorus from a 5.1 mM Na-phytate solution at a rate of 1 µmol min^{-1} at pH 5.5 and 37°C (98.6°F).

In addition unless animal feedstuff contains byproducts of human food preparation where grain has undergone a process that allows time for phytase activity such as malting for brewing or agricultural processes such as ensiling the phytase potential activity may not be realised prior to use of the animal feedstuff. As a result since 1991 commercial phytase supplements for pig and poultry have been available and widely used in conventional farming across the world to improve bioavailability of phosphorus and trace elements to the livestock as well as reducing pollution from undigested phosphorus in manure. Currently ~90% of poultry - and ~70% of pig - diets worldwide include a phytase supplement. Phosphorus digestion can increase with phytase supplement by 26% in pigs and 13.9% in chickens as a result. In addition to uncleaved phytate reducing the availability of Zn, K, Ca, Cu, Co, Mn, Fe and Mg - calcium and zinc being the most actively chelated by phytate, some studies implicate phytate in reduction of protein and amino acid digestion inhibiting proteolytic enzymes.

Animal feed phytase supplements are derived not from endogenous plant phytase but are exogenous from fungally grown or bacterially created phytase. The activity of these phytases differs from plant endogenous phytase acting on the 3 position on phytate rather than the 6 position. Feeding as mash giving time and means for phytase activity prior to feeding rather than as pelleted feed may be more effective but this is not favoured in conventional industrial scale pig and poultry farming. Addition of rye with its much higher phytase and amylase levels even than wheat would also seem a good option.

When we apply this animal feed research to human situation we can see dramatic potential in properly prepared cereal based diet especially in those developing countries with lower income levels and therefore less consumption of protein rich food such as meat and fish. In Pakistan for instance 47% children and 30% adult women have anemia whilst there appears to be zinc deficiency in children 54.2% though Pakistan has one of the highest wheat consumption levels at .per capita of around 124kg/year (348g per day). This level of wheat consumption with modern wheats with best preparation should be able to contribute 70% of iron daily requirements and 65% of zinc requirements. With older “heritage” wheat lines the contribution might be still higher - see table X below.

changes in wheat cultivated

The Green Revolution

In 1961 the release of radical new varieties of wheat began from the International Maize and Wheat Improvement Center (CIMMYT) under the direction of Nobel laureate Norman Borlaug. These new 'High Yield Variety' (HYV) wheats are characterised by their dwarf stature in comparison with typical earlier wheats as a result of breeding in genes from the Japanese line Norin 10. This reduced stature enabled application of chemical fertilizer to the crop that would otherwise result in the crop falling over ('lodging') at maturity. As long as used with fertilizer this change in the wheat plant enabled a 4 to 5 fold increase in yield of grain worldwide. The CIMMYT programme also bred for resistance to a variety of wheat plant diseases. A similar dwarfing of rice was carried out and released in 1964. In conjunction these developments are known as the Green Revolution.

The obvious down side of the new dwarfed wheat lines is that they no longer have the same capacity to shade out weeds in the same manner as earlier taller 'heritage' wheats. This in conventional farming is typically countered by application of weed killers. The resulting field with a single wheat plant genetic line in it and often the same plant in the next field and across a region multiplies the risk of attack by disease and pests which once having succeeded on one plant will find the next door plant and across whole field and maybe region equally susceptible. As a result most conventionally grown wheat crops are sprayed with fungicides and often pesticides preventatively. CIMMYT and other modern wheat breeders engage in an unending 'arms race' to keep ahead of new disease variants with most new modern lines only lasting 8 to 9 years before needing to be replaced.

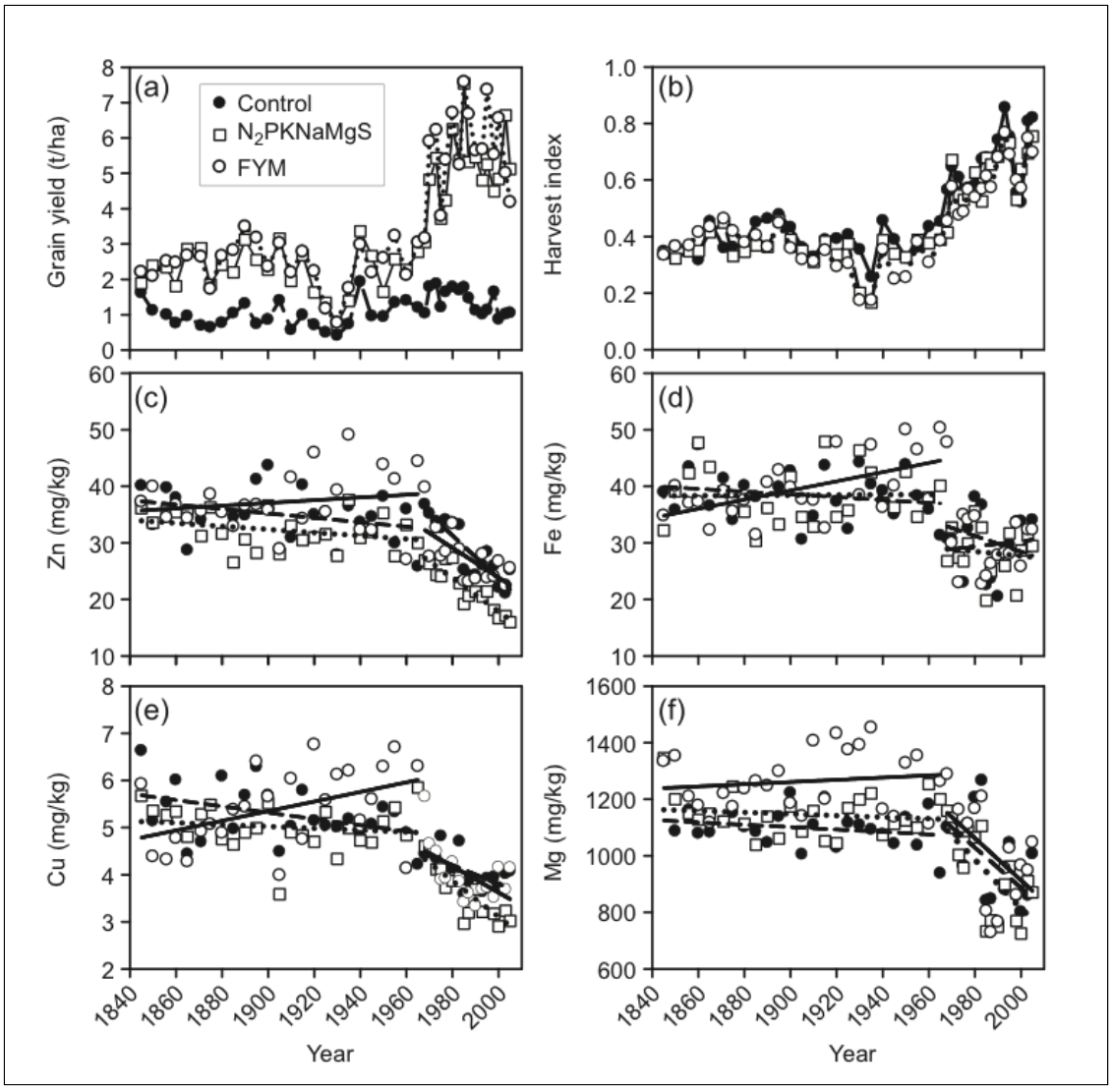
Belatedly the genetic bottleneck the Green Revolution had created was recognised and the saving of traditional landrace and cultivar wheats and other cereals to genebanks was stepped up in case germplasm in these included characteristics need to counter future threats to modern wheats and other cereals.

Green Revolution cereals with their symbiotic relationship to the use of chemicals in agriculture are not as much use for organic and biodynamic farmers. In addition as chemical inputs have tended to rise in price the profit margin on growing the HYV lines has fallen and the capital requirements of farming risen.

Another down side to the Green Revolution wheat lines is that breeding has concentrated on yield, disease and pest resistance and technical baking qualities possibly at expense of other qualities such as nutritional and flavour content.

Reduction in mineral content

In one of the historically longest running agronomy tests the Broadbalk wheat experiment (image left - 1880 harvest on Broadbalk) at Rothamstead Research station in the UK since 1843 has grown for analysis the popular wheat lines of the day on the same plot of land. In 2008 research showed the decreasing mineral content per kilo of grain recorded by the 160 years of the Broadbalk wheat experiment amounting to an average 20-30 per cent less zinc, iron, copper and magnesium.



The explanation for this decrease in mineral content per grain are not entirely obvious but increase in yield per square metre where a finite quantity of minerals may be available in the that area could be one factor but another is that modern wheats have a root biomass of around two thirds of pre-Green revolution wheats and 2007 research showed that in the most recent popularly grown HYV wheats this trend is continuing. This phenomenon seems to have occurred simply because modern wheat breeding programmes have not taken notice of it.

Further research from 2010 based in Sweden on wheat mineral content has shown that when grown organically even higher variations in some genotypes, particularly for 'primitive wheats' (non Triticum Aestivum) and landrace and early cultivar wheats, are possible and that by selection of particular genotypes as much as 100% of recommended daily dietary intake of many minerals is possible from consumption of 200g flour per day.

The reduction of magnesium in modern wheats is given as a possible explanation of increased cardiovascular disease mortality worldwide in research from 2012.

Comparison of mineral concentration (mg/kg) in the present study with previous studies, recommended intake of minerals and percentage of recommended intake of minerals from flour consumption 200 g/person/day. This study was performed in organic system while rest of the studies, except Ryan *et al.* [14] were carried out under inorganic condition.

	Present study	Spiegel *et al.*, 2009 [40]	Zhao *et al.*, 2009 [12]	Kirchmann *et al.*, 2009 [42]	Fan *et al.*, 2008 [31]	Ryan *et al.*, 2004 [14]	Graham *et al.*, 1999 [11]	Recommended intake (mg/day) according to DGE 2001 [25]	Percentage of recommended intake from flour consumption 200 g/person/day
B	1.96	0.69	n.a	n.a	n.a		2.3	1	39
Cu	5.26	3.9	n.a	3.51	4	3.3	n.a	1.5	70
Se	0.11	n.a	0.09	0.03	n.a	n.a	n.a	0.03–0.07	31–73
Fe	37.9	31	38.2	30.3	30.4	18	37.2	10	76
Mg	1,261	1,208	n.a	n.a	1,015	630	1,130	300–350	72–84
Zn	38.9	23.9	21.4	27.3	27.4	25	35.0	10	78
Ca	378	284	n.a	n.a	n.a	420	416	1,000	8
Mn	22.5	36.9	n.a	33.3	n.a	41	44.7	5	90
Mo	1.71	0.81	n.a	1.19	n.a	n.a	n.a	0.05–0.1	>100
P	4,124	3,293	n.a	n.a	n.a	2,800	3,380	700	>100
S	1,298	n.a	n.a	n.a	n.a	1,400	1,670	850–1,500	17–30
K	4,075	3,289	n.a	n.a	n.a	4,000	3,600	2,000	41

n.a indicates not analyzed.

Table 2. Mean concentration of minerals (mg/kg) among genotype groups, locations and wheat type for a total number of 493 wheat samples.

		B	Cu	Se	Fe	Mg	Zn	Ca	Mn	Mo	P	S	K
Selections	n = 32	1.59 c	5.27 b	0.18 a	35.8 abc	1,330 a	41.6 b	358 b	23.7 a	2.58 a	4,670 a	1,310 ab	4,050 b
Old cultivars	n = 191	1.90 bc	5.10 b	0.10 b	39.4 a	1,220 c	38.1 bc	390 a	24.2 a	1.53 b	3,890 d	1,260 bc	3,980 b
Primitive	n = 32	2.41 a	5.75 a	0.11 b	32.2 c	1,300 ab	45.6 a	383 ab	17.6 c	1.36 b	4,540 a	1,350 a	4,670 a
Spelt	n = 103	1.95 bc	5.50 ab	0.10 b	38.0 ab	1,280 abc	39.2 bc	327 c	20.0 bc	1.75 b	4,280 b	1,360 a	4,150 b
Landraces	n = 107	2.10 ab	5.33 b	0.09 b	38.5 ab	1,290 ab	38.1 bc	408 a	22.7 ab	1.66 b	4,130 bc	1,300 abc	4,000 b
Cultivars	n = 28	1.59 c	4.49 c	0.11 b	33.3 bc	1,240 bc	36.2 c	388 a	23.3 ab	2.23 a	4,020 dc	1,230 c	4,070 b
Alnarp	n = 278	1.84 b	5.29 b	0.15 a	38.4 b	1,300 a	39.9 b	382 b	24.2 b	2.19 a	4,470 a	1,320 b	4,180 a
Bohuslän	n = 29	1.45 c	3.79 c	0.03 c	38.2 b	1,190 b	35.8 c	330 c	41.2 a	1.33 b	3,300 d	988 c	3,390 c
Gotland	n = 141	2.33 a	5.34 ab	0.04 c	33.1 c	1,220 b	36.2 c	369 b	17.0 c	1.13 b	3,800 b	1,280 b	4,070 a
Uppsala	n = 45	1.79 b	5.66 a	0.08 b	49.6 a	1,210 b	43.4 a	411 a	17.0 c	0.71 c	3,500 c	1,420 a	3,890 b
Spring wheat [a]	n = 176	2.11 a	5.62 a	0.10 a	47.5 a	1280 a	41.2 a	420 a	22.6 a	1.32 b	3870 b	1430 a	4160 a
Winter wheat [a]	n = 317	1.86 b	5.05 b	0.10 a	32.5 b	1250 a	37.7 b	355 b	22.5 a	1.92 a	4260 a	1220 b	3920 b

Mean values followed by the same letter do not differ significantly from each other. [a] Cultivation period for spring and winter wheat are April–August and September–August, respectively.

Whilst it's important to take these findings in concert with making the mineral content of any wheat product actually bioavailable through processing to food, clearly effective processing to food maybe it cannot make available as nutrition what is not present in the grain. It should also be noted that trace mineral content is important to nutritional value once digested but may also be necessary to enzyme activity both in processing to digestibility e.g. Ca and Cu availability necessary for amylase activity and for proteolytic activity in the gut once consumed.

Changes in grain gluten content - possible link to celiac disease incidence

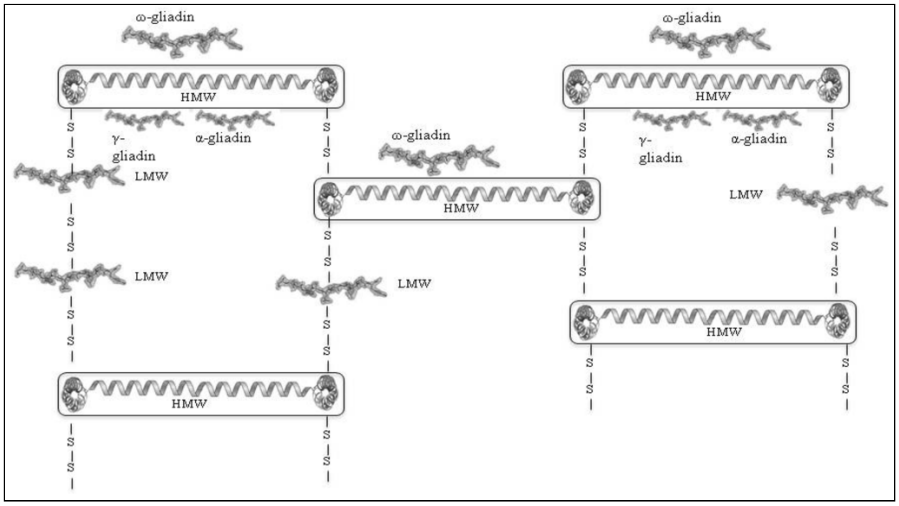
Gluten is the complex of insoluble prolamin storage proteins that comes together in dough made from a flour of wheat and some other cereals. By storage protein is meant proteins that store biological reserves of metal ions and amino acids for use by the plant embryo at germination, in the case of the prolamins these have a high proline amino acid content and are primary stores of nitrogen and sulphur. The constituents of gluten are deposited during maturation of the grain as a matrix like structure around starch granules as these dry in the endosperm area of the grain providing both a structure for the starches' storage and protection of them from predation pre-germination. After milling because of gluten's insolubility dough made from wheat and other flours can be washed and what is left will be the 'gluten' fraction.

In wheat the gluten content is between 80 and 47% of the total protein content of the grain which forms 10% to 15% of the total grain, the remainder being mostly the storage proteins globulin and albumin. Both of these are more easily digestible than the constituents of gluten being soluble in the case of the globulins slightly salty water and pure water in the case of the albumins. Oats are particularly high in globulins relative to gluten constituents which explains their high rating as animal fodder and possibly also toleration of oat products by many CD sufferers.

Gluten is responsible for the ability of dough made from the flour of cereals containing gluten, in particular from wheat, to trap carbon monoxide given off by fermentation creating the very attractive aerated finished product that we are familiar with, bread. Its insoluble and elastic properties also means it is used in many other food and other products as a binding and emulsifying agent.

The elasticity of gluten in dough is an attribute of the combination of the constituent classes of proteins in gluten, the gliadins and the glutenins. The essential mechanism by which the gluten structure with its functionality in dough is formed from its constituent elements of the gliadins and glutenins is pretty well understood but many details are still unclear.

The basics are as follows. The glutenins are are polymer protein aggregates of high molecular weight (HMW or sometimes HMM) and low molecular weight (LMW) subunits with molar masses from c. 200,000 to a few million which are only soluble by dilute acids. The gliadins are monomeric proteins which are divided based on their amino acid sequences into α, γ, and ω types and are slightly soluble in ethanol. Glutenins appear in dough formation as very large, loosely coiled protein polymers while gliadins appear in dough as much smaller and tightly coiled spheres. The two combine into an elastic sheet or sandwich like formation linked together by disulfide bonds. The formation of this sandwich like structure is encouraged by kneading dough.



The HMW units of glutenin can be described as the heavy lifters or 'backbone' of gluten functionality contributing the strength of elasticity in a particular gluten formation, its ability to resist and spring back from stretching whereas the LMW glutenin and gliadin components contribute stretchability, the ability of the gluten to be stretched without ripping. An increase in HMW glutenin subunits in the wheat and increase in ratio of glutenins to gliadins has been one focus of modern wheat for milling breeding and related voluminous research after the initial increases in yield with attempts to identify genes which control HMW to LMW balance and then target these for breeding although results have been unpredictable via this gene marker (MAS) led breeding strategy. More traditional wheat breeding selection based on dough mixing performance tests such as on a farinograph or a Chopin alveograph may however have had the same effect where it has been demonstrated that alleles Glu-A1b (Ax2∗) and Glu-D1d (Dx5 + Dy10) are normally associated with superior end-use quality, especially dough strength. In some cases (France, Spain) modern wheats may have an overall decrease in protein level but increase in dough strength associated with an increase in specific HMW glutenins.

There is not scientific literature positively identifying HMW glutenins in particular or changes in these for modern wheat as causal factors in increases in incidence of human wheat intolerances. However various gliadins are the most prominent epitopes for celiac disease and some literature relates this to changes in the overall gluten content in modern wheat.

The gliadin, Glia-α9 epitope is especially known as a major immunodominant epitope that can be recognized by the majority of CD patients (Vader et al. 2002; Camarca et al. 2009). The Glia-α9 epitope sequence (αI) is part of the proteolytic-resistant 33-mer in α-gliadins that has a strong T-cell stimulatory effect (Shan et al. 2002; Shan et al. 2005). Some studies also indicate that some fragments of Glia-α9 at positions 31-55, 31-43/49 have the ability to trigger an innate immune response in genetically susceptible individuals, that is an immune response not triggered by T-cell mechanism and it is conceivable that this could be a first stepping stone in the onset of CD.

A 2010 study by Hetty C. van den Broeck et al in the Netherlands tested a basket of 50 landrace heritage wheats and a basket of 36 modern wheats for Glia-α9 and Glia-α20 (as reference). The results of this study showed that although both tested groups included wheats with exceptionally high intensities of Glia-α9 and wheats with particularly low intensities of Glia-α9 the mean relative intensity for the landrace was considerably lower. Other researchers having highlighted that the degree of exposure to gliadin epitopes is important in the onset of and progress of CD the researchers here concluded that it should be worth further studying the possibility of working with heritage wheats to create diets that would less likely to provoke the onset of CD in a susceptible individual and even possibly a diet including wheat that at least some CD sufferers can tolerate.

What the researchers of this study do not hazard is an explanation of why pre-modern wheats might have a lower intensity of Glia-α9 than modern wheats, whether it could have occurred purely coincidentally with some common ancestor across modern wheats or has effectively being bred for either because its presence confers some desirable characteristics or it is part of some genotype some other aspect of which confers benefits. Certainly for those working with heritage wheats it would seem sound to have the particular lines they are working with tested for Glia-α9 intensity and if possible work with CD researchers to ascertain whether CD sufferers can better tolerate these wheats.

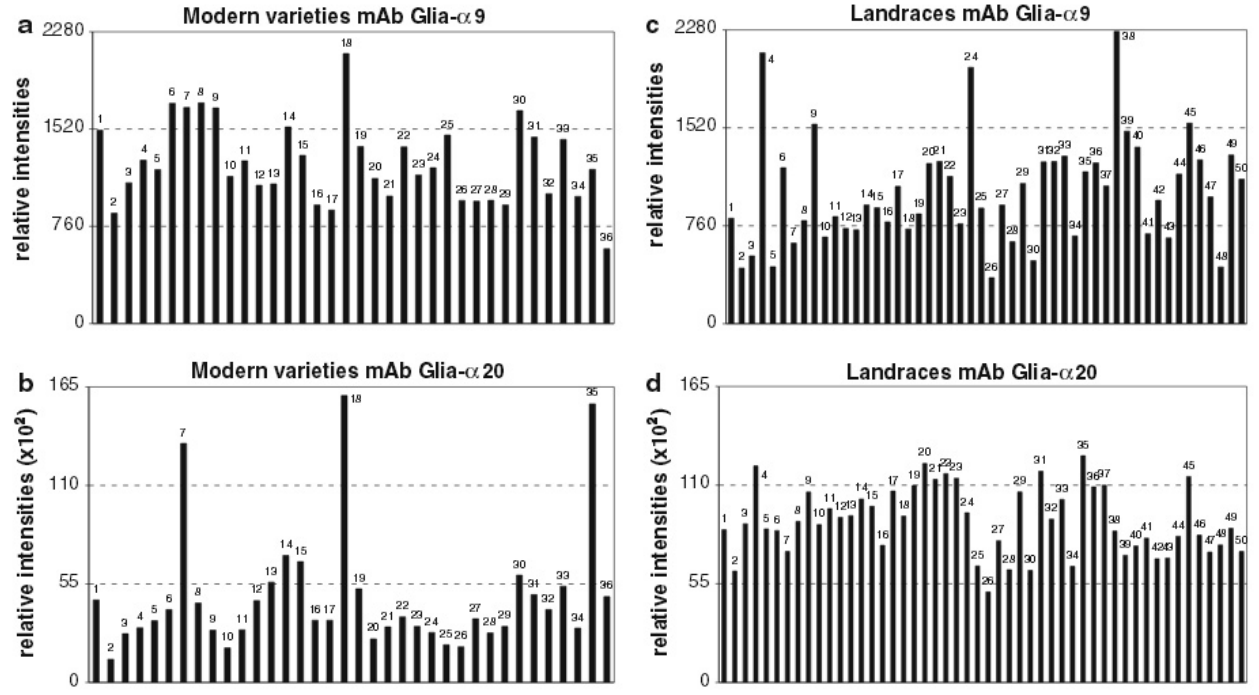


Fig. 4 Immunoblots from Fig. 1 were scanned and the relative intensities are shown for mAbs Glia-α9 and Glia-α20. **a** Glia-α9 in modern wheat varieties, **b** Glia-α20 in modern wheat varieties, **c** Glia-α9 in wheat landraces, and **d** Glia-α20 in wheat landraces

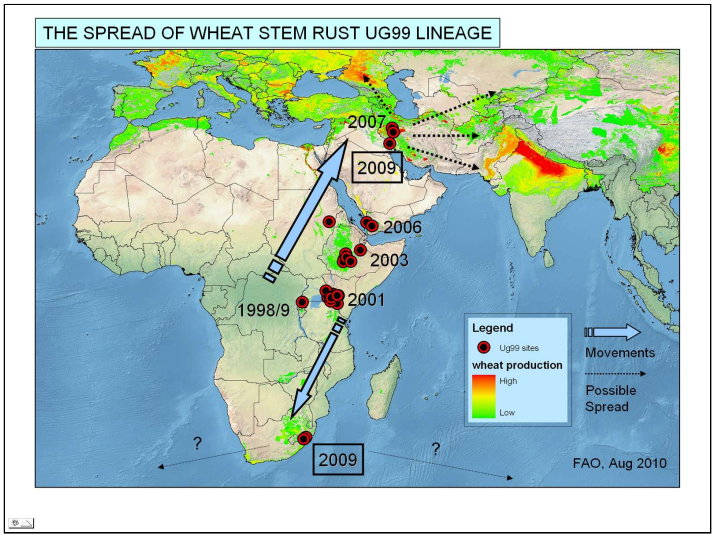
changes in cultivation and processing of cereals to food

changes in cultivation - agrochemical usage

The key change in the cultivation of wheat in the 20th C has been the introduction of agrochemicals. First and foremost was the increasing availability of synthetic fertilizer post WWII. Existing wheat lines of the period were not suitable for cultivation with addition of chemical fertilizer as they would become so tall as a result that lodging (falling over towards maturity) would become inevitable. In response a new range of wheat lines crossed with the Japanese Noren 10 dwarf wheat and having a much reduced height was created by Norman Borlaug and his team at CIMMYT and released across the world in the early 1960s. These new wheats of the “Green Revolution” had a higher grain to plant matter ratio, known as High Yield Varieties - HYVs and did not lodge nearly so readily when grown with fertilizer additions as older lines. However their decreased height meant that they faced increased competition from weeds the response to which in conventional farming has been the increased use of herbicides both broad-spectrum and selective. Once weed killers have been successfully used on a cultivation and given that modern wheat lines comprise genetically identical individual plants there is a strong potential that any successful disease or pest attack on a single plant will quickly spread across the crop. This risk is reinforced by the tendency of farmers across a region to all be growing lines from the same limited recommended list and a common recent genetic inheritance across modern wheat worldwide. In reaction conventional farmers apply fungicides and pesticides often in a preventative manner.

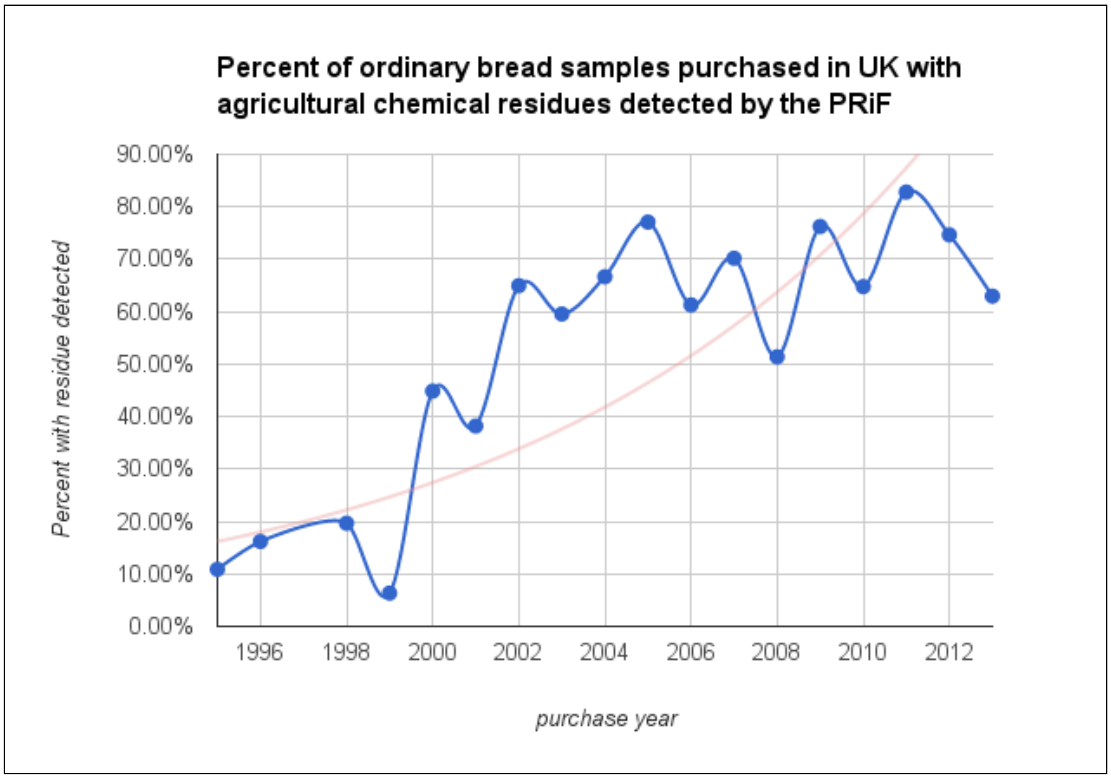
Overall the emphasis of modern conventional farming is to manipulate the environment through the use of high inputs, both mechanical and chemical to enable the cultivation of high yielding but comparatively demanding and fragile crops. This thrust of conventional farming requires high levels of capital investment, beyond the reach of many small scale farmers both in developed and developing countries and leaves agriculture vulnerable to rises in input costs. New threats can emerge that play on the limited genetic diversity of crops or exploit the uniformity of cultivation techniques globally.

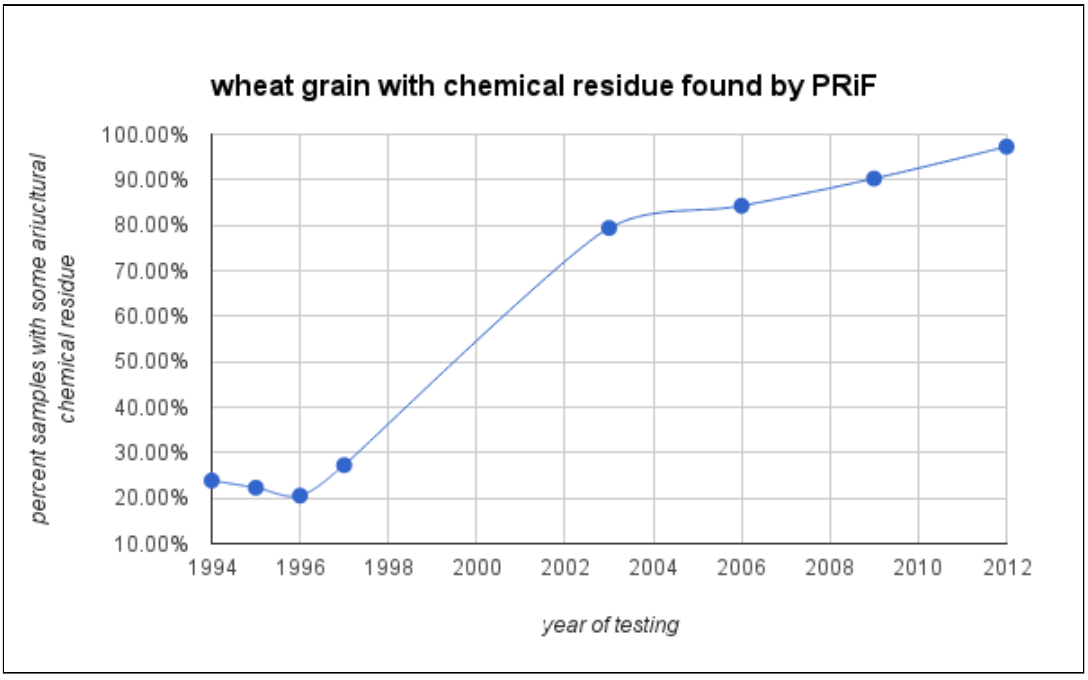
A recent disease threat is the emergence of a wheat stem rust variant in Uganda in 1999 called Ug99. This circumvented what had been seen by wheat breeders as a “silver bullet” against stem rust by the incorporation into modern wheats of the stem rust resistance gene SR31 from rye. It has spread from Uganda through to Kenya, Yemen and Iran and down to South Africa as well as further mutating to overcome first efforts to breed new resistance to it into wheat lines. Spraying has become a main defence for farmers without which they risk losing up to 80% of their crop.



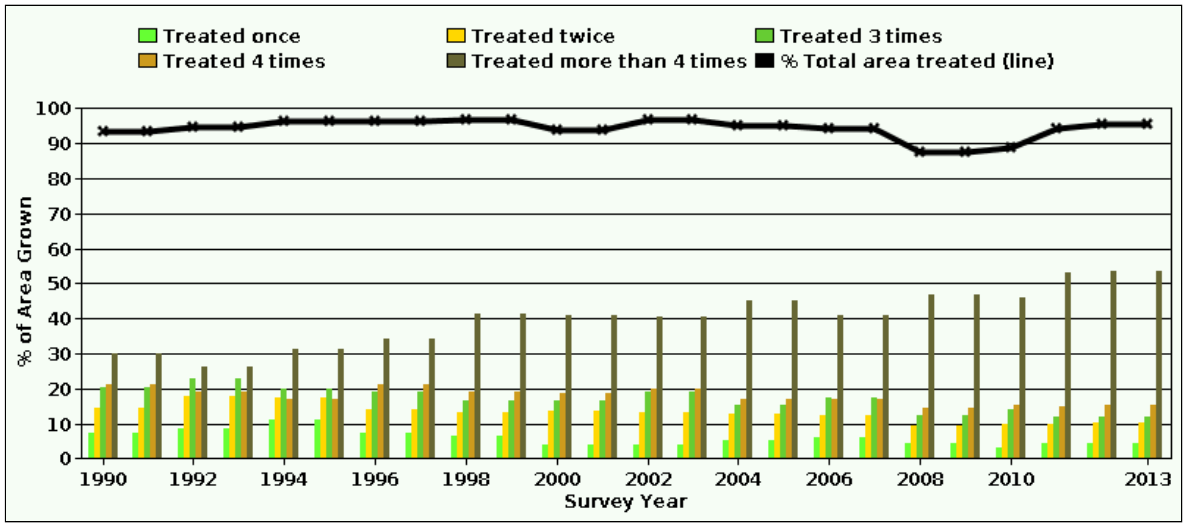
An example of an emerging weed problem associated with conventional growing of wheat out of rotation is the spread of weed killer resistant "black grass" in the UK and across Europe. In the US "super-weeds" resistant to the glyphosate herbicide (marketed as "Roundup" by the Monsanto company) now infest 5% of US agricultural land thanks to the reliance on creating and cultivating Roundup resistant GMO crops.

In the UK we are seeing an increasing incidence of agrochemical residues found by the Health and Safety Executive's Expert Committee on Pesticide Residues in Food (PRiF) in bread products made from conventionally grown crops and in UK grain before milling.



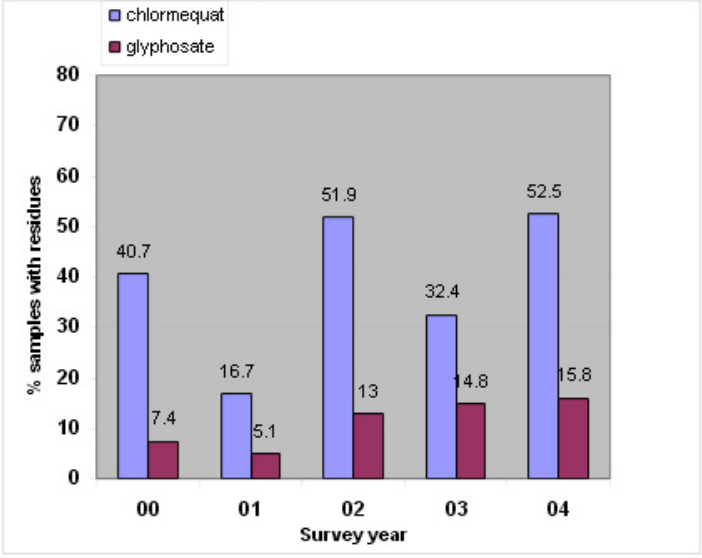


Since the turn of the 20th century the quantity of agro chemicals applied to the UK wheat crop by weight may have fallen very slightly however the number and variety of applications has risen sharply and the occurrence of residues in “ordinary bread” PRiF has risen from around 20% to over 70%. On average 2012 UK wheat crops received 3 fungicides, 3 herbicides, 2 growth regulators and 1 insecticide application, 0.6% of the total harvest remained untreated - presumably the organic crop.



The three agrochemical residues found most commonly by the PRiF in their tests are the Plant Growth Regulator (PRG), **chlormequat**, the broad spectrum herbicide, **glyphosate** commonly known as Roundup and the insecticide used on stored grain, **pirimiphos-methyl**.

Fig. 3: Ordinary and speciality bread samples containing residues of chlormequat and glyphosate 2000-2004 (%) (Source: PRC survey data)



Ordinary bread

Pesticide residue	1994	1995	1996	1998	1999	2000	2001	2002	2003	2004
Total samples	255	239	241	239	142	214	144	137	136	144
No. samples with no residues detected	214	213	202	192	133	118	89	48	55	48
% samples with no residues detected	83.9	89.1	83.8	80.3	93.7	55.1	61.8	35	40.4	33.3
Chlormequat (PGR)	-	-	-	-	-	88 (0.05-0.2)	32 (0.05-0.2)	80 (0.05-0.2)	63 (0.05-0.2)	88 (0.05-0.3)
Chlorpyrifos-methyl (I)	Nil	14 (0.1)	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Etrimfos (I)	3 (0.06-0.07)	1 (0.1)	4 (0.06-0.3)	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Glyphosate (H/D)	-	-	-	-	-	16 (0.1-0.3)	9 (0.1-0.2)	26 (0.1-0.4)	30 (0.1-0.5)	27 (0.1-0.6)
Gamma HCH (I)	-	-	1 (0.01)	Nil	Nil	Nil	Nil	Nil	-	-
Malathion (I)	12 (0.05-0.1)	5 (0.08-0.3)	Nil	Nil	1 (0.05)	1 (0.06)	Nil	Nil	4 (0.05-0.1)	4 (0.05-0.1)
Pirimiphos-methyl (I)	34 (0.05-0.4)	6 (0.1-0.2)	37 (0.05-0.2)	47 (0.05-0.2)	9 (0.05-0.1)	7 (0.07-0.2)	15 (0.05-0.1)	6 (0.06-0.2)	2 (0.08-0.2)	7 (0.05-0.2)
MRL exceedances	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil

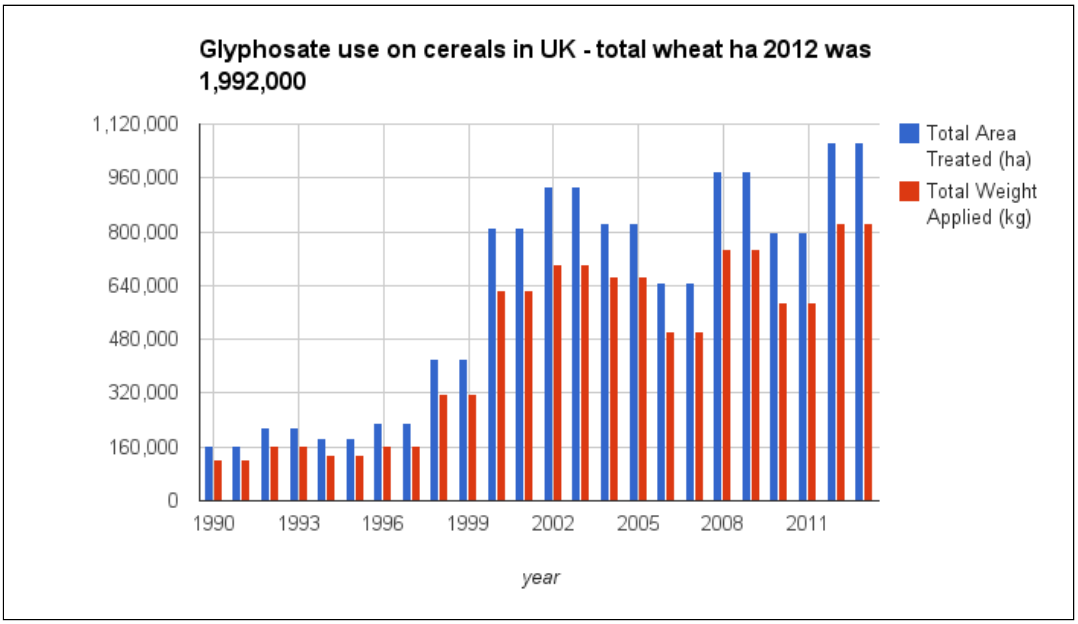
Chlormequat is the most commonly found agricultural chemical residue in the UK wheat crop which is now found in the majority of UK grain samples and in over half bread tested annually. It is a gibberellin biosynthesis inhibitor acting to stop cell elongation and therefore is used as a PGR and is applied in order to shorten plant height and minimise the risk of lodging (crop falling over) enabling maximisation of nitrogen application. It is not approved for use on food crops in the United States but PGRs are applied in the spring to 89% of the winter UK wheat crop area and 79% of these applications include chlormequat. The EU legislated Maximum Residue Level (MRL) for wheat is 2 mg/K though the more typical MRL for most foodstuffs is 0.05 mg/K. No MRL is set for bread but the UK PRiF calculates a "default" MRL for bread of 1.

A 2006 review reported Danish studies in the 1980's showed pigs display impaired reproduction, mainly impaired oestrus, when fed grain from crop treated with chlormequat at a level calculated to 0.0023 mg/kg bw/day as a result of which the Danish advisory board recommended limiting the use of grain (maximum 30% of diet energy) from crop treated with chlormequat given to breeding stock due to the risk of reproduction problems. Further experiments showed that epididymal spermatozoa from mice on feed or water containing chlormequat at a rate of 0.024 mg/kg bw/day had compromised fertilizing competence in vitro, while reproduction in female mice was not compromised.

Glyphosate is a broad spectrum herbicide. Unusually for herbicides, it has the ability to move down the plant with the photo-assimilates in the phloem to 'sinks' caused by the growth of perennating organs (Caseley & Coupland, 1985). Its slow mode of action assists this process by ensuring that damage to plant tissue that might otherwise prevent its movement does not occur until after there has been sufficient transport to the perennating organs.

Originally designed by Monsanto as a water softener glyphosate is a powerful chelator of trace minerals such as Cu, Fe, Mg, Mn, Ni, and Zn and it is this removal of micronutrient availability to plants that interrupts the activity of EPSP synthase and other enzymes that participate in the shikimic acid pathway that plants and other organisms use to produce the aromatic amino acids including phenylalanine, tryptophan, histidine, tyrosine all of which are considered essential amino acids in the human diet. The micronutrient chelating effect of glyphosate extends into the growing environment by the killing of microflora in soil which enable micronutrient uptake by plant roots while the breakdown of glyphosate in soil including that from decay of plants killed by the herbicide is very variable, from 2 to 147 days.

Glyphosate's herbicide potential was discovered in 1971 by a Monsanto chemist. It was introduced in the UK in 1974 for the control of perennial weeds, notably common couch. It replaced other herbicides with higher doses of active ingredient which were often used in conjunction with repeated cultivation to control common couch. In 1980 its use on cereal crops pre-harvest was introduced and by 2002 12% of UK conventionally grown wheat was treated with glyphosate rising to over half the crop by 2011. The area of all cereal species treated with glyphosate increased significantly by 660% from 161,213 ha in 1990 to 1,064,529 ha in 2013.



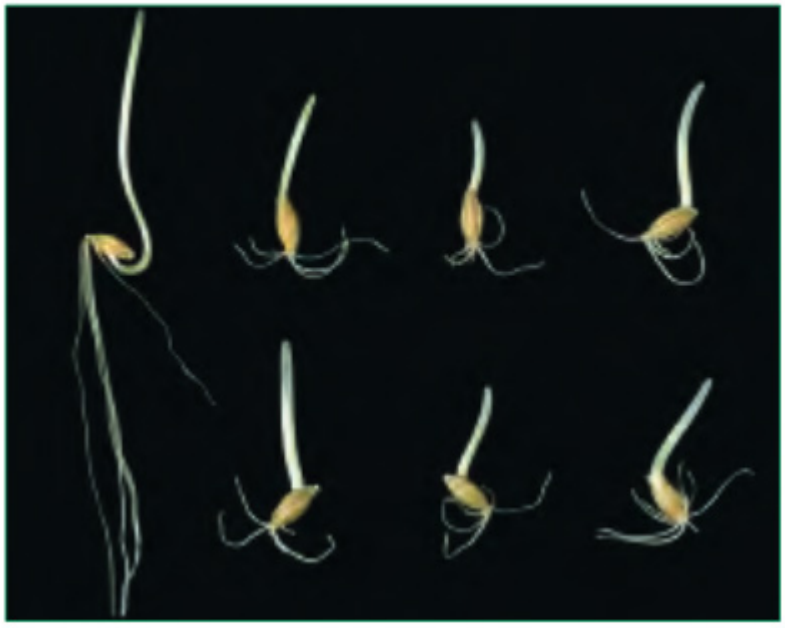
Source https://secure.fera.defra.gov.uk/pusstats/

Much of this extreme increase in the use of glyphosate (commonly available as Roundup) on cereals and its increased detection as a residue in bread is explained by the increased pre-harvest use on wheat as a **desiccant**. The commercial reasoning for pre-harvest spraying was initially to gain time in field operations when there would normally have been a glyphosate application in between one cereal crop and the sowing of a second cereal in order to control particularly perennial weeds and which may be more vulnerable to glyphosate with pre-harvest application. However other commercial advantages to pre-harvest application known as **desiccation** emerged. The effect on the wheat plant is similar to other threats to a plant's viability in that the plant will initially respond by concentrating energy on seed/fruit filling. The result is a somewhat speeded up and evened out grain maturation, the glyphosate being applied once a sample of grain from the crop has reached 30% moisture, between a fortnight to seven days from normal harvest.

In years where secondary tillering may have occurred when rain has followed a prolonged period of drought in early summer these unwanted tillers will be killed along with the plant as a whole and perennial weeds in the crop will of course also be killed and tend to dry out both of which will tend to make combining easier and cheaper.

The assertion is made both in HGCA review and repeated on glysophate promotion website http://www.glyphosate.eu that pre-harvest application improves the Hagberg Falling Numbers (HFN) of a wheat crop by killing green grain from late tillers. This relates to the high pre-maturity alpha-amylase activity in the pericarp (RPAA) and aleurone layer of the grain which if included in harvested grain is one of four roots to low HFN make the flour less suitable for milling and baking with. Pre-harvest glyphosate application appears to lower potential germination of resulting wheat grain by around 16% and those seed that do germinate show less vigour as well as abnormalities of growth. Pre-harvest glyphosate application is not allowed for barley destined for malting and the brewing industry as a result.

Figure 2.3 Seedlings showing glyphosate-induced abnormalities in paper germination tests (normal seedling for comparison is on the left of the plate)

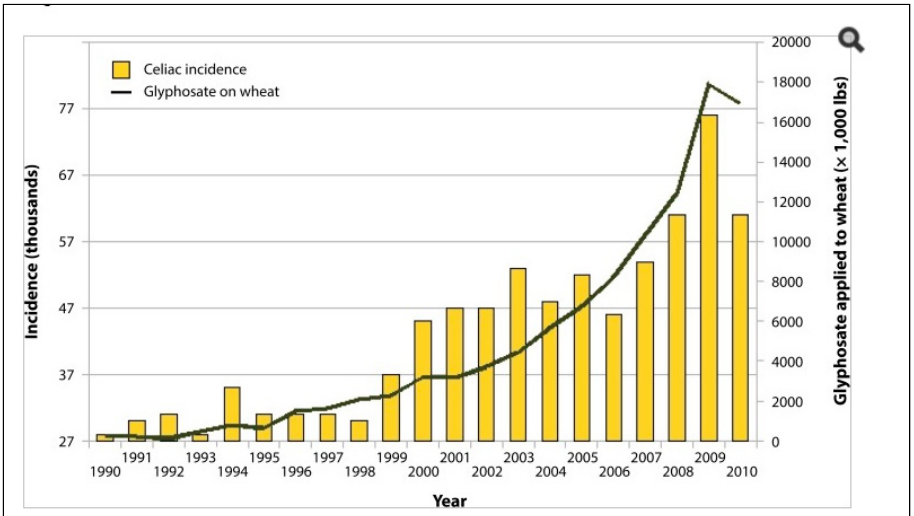


The concern is that wheat in the supply chain to millers and bakers in the UK that has been treated with glyphosate as a desiccant will not perform in a normal manner, in particular enzyme potential activity may have been reduced or altered which could be a particular concern especially in the case of phytase.

Aside from potential damage to seed wheat grain functionality there is the further issue of increasing glyphosate residues in bread and other foods with wheat as an ingredient.

In March 2015 the World Health Organization's relevant panel of experts issued a report concluding glysophate was "probably a carcinogen" on the basis of review of existing research. This carcinogenic substances or professions category is second "2a" from that of 1, "definitely carcinogenic".

Researchers publishing in 2013 have identified glyphosate residues in the human diet as a major health threat and blamed these residues for causing the increase in CD and several other major diseases. This they partly conclude from the coincidence of rising diagnosis of CD and other diseases with increasing use of glyphosate on wheat.



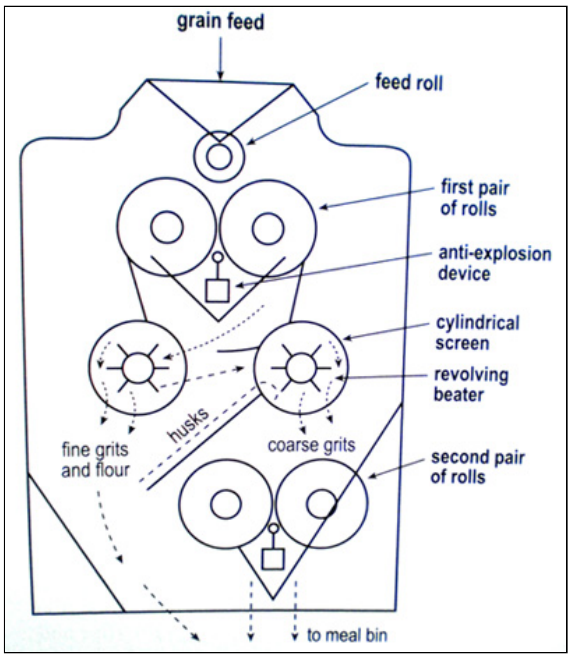
Hospital discharge diagnosis (any) of celiac disease ICD-9 579 and glyphosate applications to wheat (R=0.9759, $p \leq 1.862e\text{-}06$).
Sources: USDA:NASS; CDC. (Figure courtesy of Nancy Swanson).

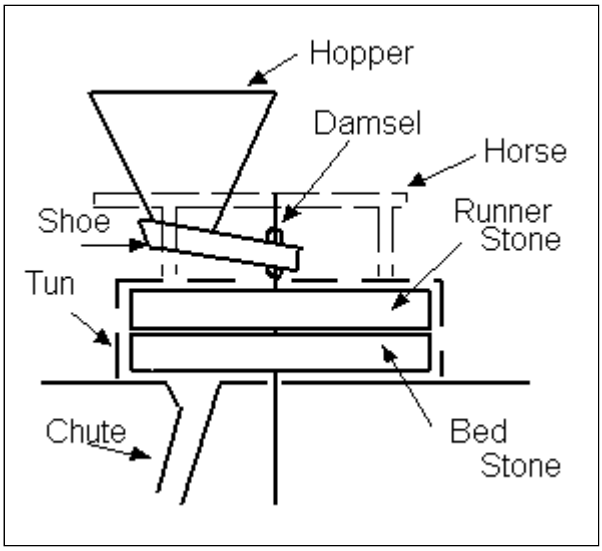
The primary routes through which these researchers propose use of glyphosate on wheat particular as a desiccant are disruption of cytochrome P450 class enzymes activity in human digestion, chelation of micronutrients in human digestion and lack of tryptophan, tyrosine, methionine and selenomethionine essential amino acids in human diet as a result of consuming GMO Roundup Ready crops. The researchers fail to provide experimental evidence for many of these assertions or suggest experiments which might confirm or disprove instead reinforcing their ideas with graphs showing coincidence of glyphosate use on US wheat and various diseases. In the case of the above for CD its ridiculous to show that a fall in glyphosate use on US wheat in 2010 should instantly produce a decline in CD testing where the resulting wheat crop would take up to a year to reach the food chain and CD is a disease that predominantly takes years from onset to testing for diagnosis to happen. Nonetheless in the absence of definitive explanations for the increase in prevalence of CD amongst various human populations during the 20th C coincidental increase in glyphosate use pre-harvest on wheat and increased residue detection in bread and human urine the issue should not be dismissed from examination.

Another change in typical cultivation and harvest is that drying of grain for storage is normally done mechanically and at high speed and relatively high temperatures compared to previous practice of stooking of sheaves in the field followed by storage in ricks. This may block the typical maturation process to millable grain that takes several months from harvest, grain not being suitable for milling according to millers' lore either till November from August/September or according to some till after Christmas. This may relate to resistance to pre-harvest sprouting that some wheat lines exhibit, seed dormancy and the subsequent natural process of lifting of dormancy. This would explain how it is that industrial millers justify adding fungal amylase to flour to make up for lack of endogenous plant amylase in grain milled soon after harvest.

changes in milling

In the 1870's a radical change in milling technology got under way, the previous stone milling method was replaced by roller milling.





In stone milling the whole grain is fed into the centre of a top "running" (turning) stone with a stationary "bed" stone underneath it, the grain is gradually milled finer and finer as it moves from the centre between the stones to their outer edges. If a flour that is less than the wholemeal is to be produced the flour from the mill is then sifte. In stone milling even when sifted small fragments of bran will remain and the germ with its vitamin bearing fats and oils will have been crushed and mixed with the endosperm.

Roller milling was partly introduced to deal with the harder wheat coming out of North America in the late 19th C but also brought much greater separation between the main elements of the grain. Roller milling works with pairs of rollers with one roller running at a slightly different speed to the other with typically over 20 pairs of rollers operating in a chain. The grain is gradually stripped down into streams which are essentially pre-sifted so that for instance the stream from centre of endosperm contains none of the germ or bran and aleurone layers. Higher temperatures during milling also serve to kill enzyme content. The only way to make a wholemeal with roller milling is in fact to add the streams back together again.

Roller milling was able to produce a much whiter flour than stone milling which aside from lack of colour gave the resulting product, which lacked the oils of the germ and the enzymatic content of the aleurone layer, a much longer shelf life - being essentially dead in biological terms. Roller mill suppliers sales pitch included offering less pest problems than stone milling as the pests couldn't find much worth eating, at the same time the more nutritious germ and bran streams found a ready market as animal feed. Along with easier and longer storage and shelf life, roller mills are much more productive but more capital intensive than typical stone mills producing a rapid concentration of the milling industry during the 20th C across the world, destroying everywhere local and independent milling businesses.

As described above modern roller milling to produce white flour removes much of the micronutrient value of the grain. Wholemeal and darker flours restore all or some of this nutritional value but whereas the fibre content supplied by including pericarp fractions will be of recognised benefit, trace element content to a large extent is bound to phytic acid as phytins and will require cleaving by phytase during fermentation in order to become bioavailable. The effectiveness of this cleaving occurring will depend on the amount of phytase available and the time and conditions created and allowed for this activity to take place.

One specific issue with possible implications for enzyme activity is whether flour should be used freshly milled or used ‘aged’ as suggested by some milling and baking practice. The issues are as follows.

First, the cut off point for flour use in the case of wholemeal flour is it going rancid which happens between 2 and 3 months depending on temperature of storage. The element that goes off is wheat germ oil which is a main reason why one main objective of roller milling is to remove the germ. But this is actually one of the most nutritious elements of the grain, the germ in general and its oil in particular which contains essential fatty acids omega 5 and 3 and is highest natural source of vitamin E.

In stone milling the wheat germ oil will get crushed into the rest of the grist. Most stone millers (in UK, the Traditional Corn Millers Guild members) will list 3 months best before date for their wholemeal but vary between 6 and 9 months for their sifted flour. More logically any stone milled flour will be best within 3 months. Ideally all stone millers would provide their milling date as well as “Best Before” date on packaging.

At the other end of the scale roller milled white flour usually has best before date of between one or two years being an essentially dead product.

Things are more complicated from the opposite time approach - how fresh to use flour? Can flour be too fresh (green)?

Much advice is given that flour needs 'ageing' by natural means or oxidising flour 'improvers' because a nice product is more easily made - flour becomes stronger and whiter.

Oxidising agents used as flour “improvers” to artificially “age” include benzoyl peroxide, Calcium peroxide, Nitrogen dioxide, Chlorine, Chlorine dioxide, Azodicarbonamide, potassium bromate (E924, the component which gives bromated flour its name, used mainly in the U.S. East and Midwest, acts as a bleaching agent, banned in the EU).

Its quite easy to see that a cream or yellow flour will become whiter after 4 or 5 weeks. The yellow disappearing is carotenoids oxidising but these are what carry bread aroma and sweetness as well acting as antioxidants with valuable health benefits when eaten - so does the baker actually want to get them deactivated by allowing or encouraging them to oxidise?

'Ageing' or oxidation of flour acts to strengthen dough by robbing sulfhydryl, sulphur bonded to hydrogen of its hydrogen freeing sulphur content to create more disulphide bonds in your dough's gluten structure - it makes your dough more elastic but less extensible.

Ageing also certainly increases Hagberg Falling Number, that is reduces amylase enzyme activity which chops starches into sugars. Some amylase activity to feed your yeasts but not too much otherwise your dough will turn to slop. UK farmers who try to grow wheat for milling have it rejected if HFN are too low (too much amylase activity), however the level set is in reality not ideal as big millers prefer to then top up with fungally grown amylase to a consistent level year on year - this is one of the most common additives in conventional flour and is implicated in causing Baker's Asthma.

The biggest potential problem with using “aged” flour is the question of whether other enzymes than the amylases de-activate during storage - especially phytase and in relation to that especially for wholemeal and darker flours.

If it is the case that phytase becomes deactivated progressively post-milling it would easily explain the observed 'liveliness' of fresh 'green' flour - one French 'boulanger paysanne' described it being like a young child in the bakery in a video. This baker liked it to feed his starter but preferred six month old flour from year old grain for his main mix - but when one saw his dough one understand why. a +100% hydration dough that could only be chopped wrapped in copious flour rather than shaped. Conversely most 'boulanger paysannes', often with their own mills, prefer the opposite, totally fresh to 3 to 4 day old flour. Some bakers who like fresh flour in principle may however only use totally fresh in starter and for main flour use after a week when they feel it becomes more predictable and uniform - but still relatively fresh.

A main reason given by one roller milling spokesperson for chemical ageing of flour is that modern millers often need to mill grain that is fresh from harvest. Its perfectly true that new season grain can't be successfully milled/baked without a two to three months wait from harvest but whether chemical ‘ageing’ rather than simply waiting is an acceptable process that produces an equivalent product in terms of enzyme functionality is questionable. The exact mechanism by which freshly harvested grain is not suitable for milling is not understood.

flour fortification

- calcium carbonate (chalk),
- iron,
- thiamin (vitamin B1) and
- nicotinic acid or nicotinamide

changes in baking process

In 1961 Chorleywood research laboratory of the British Baking Industries Research Association developed the baking process named after it, the Chorleywood bread process (CBP) and this quickly came to dominate industrial plant baking in the UK, Australia, New Zealand and India and many other countries. The main objectives of the process are more water to flour, using lower protein UK grown flour and reducing time. Today over 80% of UK bread is produced using CBP. Originally sold to bakers as the "no time method" the original mixing machines to fulfill the essential requirement for an extremely high energy mix lasting between 2 and 5 minutes under a partial vacuum were adapted from paint mixing machines. This violent mixing or whipping is aided by including in the dough slurry, solid fat–hydrogenated or fractionated plant oils, ascorbic acid (Vitamin C) and other "improvers" including various possible enzyme additions plus a 2 to 3 times the usual quantity of commercial yeast. The mixing creates so much heat that the next stages require computer controlled cooling, the dough is divided and given an 8 minute rest before re-shaping and proofing in tins for around an hour. Baking then takes 20 minutes followed by a couple of hours cooling before slicing and packaging.

CBP is not the dominant process in the US as typical US hard red spring wheat is too high in protein so that more mixing force is needed and with higher prevailing ambient temperatures the cooling costs prior to baking are excessive. Instead a batch processing using a sponge mix of around 60% of total dough is proofed for between 2 and 4 hours before final mix and baking.

Into the ingredients for various speeded up industrial bread production processes go an array of additives to compensate for nutritional and technical baking inadequacies in flour used and to substitute for the activity of enzymes endogenous to grain that do not have sufficient time to function under these industrial schedules.

Other than for wholemeal flour, many countries require “fortification” of all flour by millers. In the UK under the The Bread and Flour Regulations 1998 calcium carbonate (chalk, once considered an adulterant), iron, thiamin (vitamin B1) and niacin (also known as vitamin B_3 or nicotinic acid) must be added to compensate for the large proportion of these lost in sifted roller milled flour and to compensate for the proportion of these chelated by phytic acid in a bread not allowed sufficient proofing time for endogenous phytase to cleave the phytic acid content. At the time of the introduction of the initial UK legislation in 1953 when the rate of extraction legally permitted was reduced from 80% to 70% these fortificants were aptly referred to in the Commons as ‘token’ as they by no means encompass all the trace elements and vitamins lost through modern roller milling to white flour and in the case of iron especially it is not clear the form added is actually nutritionally an adequate bio-available substitute.

A bewildering list of legally permitted additives are termed “bread conditioners” to help in various aspects of the industrial production of bread as well as to increase shelf life, some permitted chemicals are listed here http://www.sustainweb.org/realbread/food_additives/#permitted and some enzymes in use are shown in a list from a manfacturer here http://specialtyenzymes.com/products/term/baking. In the case of enzymes their use in baking without any ingredient labelling providing in industry speak “clean labelling” was totally deregulated in the UK in 1996 on the grounds that all would be destroyed in actual baking although this is scientifically inaccurate as some enzymes are heat stable and this also begs the question of the desirability of some these enzymes’ activity for nutrition and digestibility. Added enzyme ‘solutions’ typically stand in for enzyme activity related to the production process by enzymes endogenous to grain but requiring extra time either prior to milling or prior to baking compared to these additions. These enzyme ‘solutions’ for technical baking qualities may mask the lack of time in industrial bread making processes for other enzymatic processes to occur that less obviously impact the technical appearance of the baked loaf but do impact nutritional and digestibility qualities.

Additives for shelf life of non-sourdough bread are prevalently fats designed to barrier water held in the gluten structure of the bread from evaporation. Fats are also added in the Chorleywood Bread Process to help give structure to the dough in mixing. The nature of these fats which are nowadays typically palm oil raise questions both around polyunsaturated fats in relation to health, especially cardiovascular and around social and environmental issues of forest clearance and monoculture in palm oil cultivation in Malaysia and elsewhere.

The currently most common additives in baking processes are ascorbic acid and fungal grown alpha amylase.

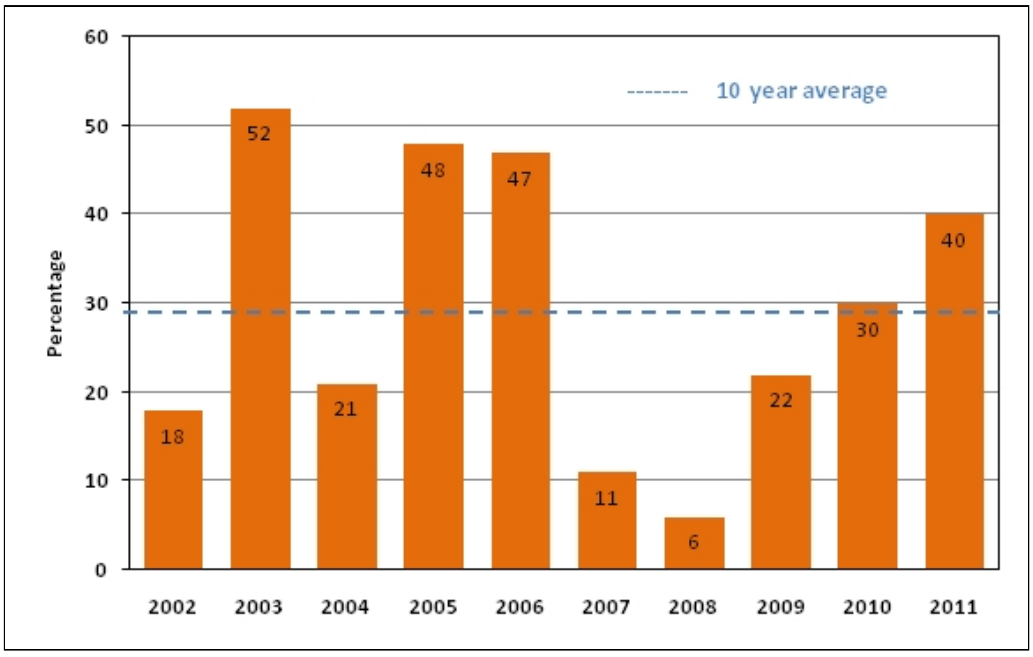
Ascorbic acid in many EU jurisdictions is the only permitted “flour improver”. It may be benefits from also being termed Vitamin C making it sound like a possibly nutritionally beneficial addition. In fact the form used is a very purified form of that found in citrus fruit and other high vitamin C natural sources and is denatured in the baking process so that it delivers no vitamin supplement. Ascorbic acid is a reducing agent but in dough mixing in the presence of oxygen and ascorbate oxidase enzyme endogenous to the grain ascorbic acid becomes dehydroascorbic acid and in this form it serves to strengthen disulphide bonds and therefore gluten structure in dough. The net result is greater oven spring, a finer crumb structure and greater bounce back from compression of the finished loaf.

There has been no evidence that the addition of ascorbic acid in the quantities used in baking is a human health threat. Its use is fairly ubiquitous in some baking traditions in particular in the creation of French baguettes and has become common in Australian ‘artisan’ baking but in both these cases there are within the baking industry ardent advocates against its’ use such as the Australian sourdough pioneer John Downes and so is a subject that can provoke heated debate. Generally it can be seen as a baker’s crutch substituting for higher quality flour and greater attention to detail in dough mixing and baking.

The addition of fungal amylase has been discussed earlier in this document. Fungal amylase is implicated as a cause of baker's asthma but has not been identified as a problem in consumption of bread products made with this additive. However the role of fungal amylase, to make up for insufficient endogenous plant amylases in wheat is a cause for concern.

In the UK the Agriculture and Horticulture Development Board, Cereals and Oilseeds (AHDB) (previously known as the Home Grown Cereals Authority) each season issues a list of recommended varieties of wheat and other cereals for cultivation which is a selection from with the current Permitted for Cultivation list as a result of trials for disease and pest resistance, yield and quality when farmed conventionally. Wheat lines recommended are divided into nabim (National Association of British and Irish Flour Millers) groups according to their likely suitability for different usages, group 1 being those most likely to be good for making bread. Since most UK farmers both conventional and organic follow these recommendations this institution in itself contributes to the genetic homogeneity of the UK wheat crop with for instance only two varieties listed as nabim group 1 Spring sown Spring wheats in 2015 and this therefore increases overall disease pressure.

When a farmer sows a nabim group 1 wheat he is hoping to make a premium by selling the crop for milling and bread making. However the resulting crop must meet a variety of quality standards at the mill for the farmer to get this premium the most important of which are a standard for size of grain (bushel weight), a standard for protein level and a Hagberg Falling Number standard which indicates amylase levels. In an average UK season only 28% of wheat sown to try and reach these milling standards fully reaches them and the grower threfore receiving full premium price.



Proportion of Group 1 Wheat Samples Achieving Full Milling Specification

A typical cause of a nabim group 1 UK wheat variety crop not meeting milling standard is Hagberg Falling Numbers too low, i.e. too much amylase activity caused by a wet harvest season and therefore some signs of pre-harvest germination/sprouting. However the standard set is in fact for lower amylase levels than is actually needed hence the practice of adding fungal amylase. In effect the large scale millers are demanding less than optimal amylase levels of farmers because in order to produce a year on year consistent product the millers take the root of topping up with fungal amylase. The bi-product of this solution may well be a lack of other endogenous enzymes activity particular those that may relate to digestibility as opposed to functional aspects of bread making in particular phytase and proteolytic enzymes. Avoiding millers taking this route in flour preparation would be encouraged by greater knowledge, skill and willingness amongst bakers to adapt their methods and recipes to season on season variations in flour - for which millers in turn need to provide greater information to their bakery customers as to flour batch characteristics as exampled by the "Cook Naturally" mill in the USA.

benefits of sourdough bread making process

In recent decades there has been a resurgence in traditional baking practice broadly termed "artisan baking" across many developed countries. Central to this renaissance of traditional baking processes has been use of the sourdough method. In sourdough baking the "straight dough" method use of "baker's yeast" as the leavening agent is replaced by a sourdough culture or starter. The sourdough method typically takes much more time than common straight dough method. Straight dough methods can take between and hour and 4 hours compared to between 12 and anything up to 3 days of fermentation steps for sourdough methods. A sourdough culture is made up of yeast(s) and lactobacilli type bacteria co-existing in a symbiotic relationships and these tend to keep out any intruders and in some cases provide inputs to each other and to specific aspects of the dough fermentation process such as additions to enzyme activity or optimization of conditions for enzyme activity.

The motivating force behind this sourdough and artisan baking renaissance in the first case has been the re-discovery of the enhanced organoleptic qualities that long sourdough fermentations bring with increase of flavours and aromas from the ingredients developed principally through increased enzymatic activity. Alongside these re-discovered gustatory benefits of sourdough methods from the early days of this renaissance advocates and practitioners have extolled the nutritional and digestive advantages of the process. Most sourdough bread bakers will have experienced reaction from recipients of their bread who report that they can't eat typical modern industrially processed bread without symptoms in the "wheat-intolerance" range such as eczema, hay fever and bloatedness but can eat the producer's sourdough bread without such problems. Various relatively recent scientific papers have elucidated why this can be the case. Aside from the extra time in fermentation in sourdough processes - analogous to timing of a soak to induce germination of grain - a principal consideration is the pH of the fermenting dough and whether this optimises phytase activity for the cleaving of phytic acid, research such as that recorded in Reduction of phytic acid during breadmaking of whole-meal breads demonstrates that a pH of 4.5 is optimal for hydrolysis of phytic acid. This pH level is clearly consistent with most sourdough methods and not achievable in common straight dough production methods.

Typically a sourdough bakery keeps one or more sourdough cultures from one bake to another feeding a quantity remaining from previous bake to create enough freshly fed starter for the subsequent bake. Bakeries have often been using the same culture(s) for their entire existence in some cases over multiple human generations. Sourdough cultures have been increasingly analysed to identify their biological content and the list of possibilities is now extensive with some noted regional variations. However the approach to creation or acquisition and maintenance of sourdough cultures by bakers is quite variable with some authors suggesting simply to capture into a flour and water solution local airborne spores or spores from sources such as wine grape skins, whilst others recommending procuring from existing bakeries or from collectors such as Sourdough International. Many bakeries maintain their sourdough culture by feeding simply with cereal flour, wheat and/or rye but others feed their “mother” culture with additions of other substrates such as apple juice or baked potatoes.

In almost no cases, other than the very rare industrial applications of sourdough method, do bakers relate scientific analysis of the biological contents of a culture to its appropriateness for their particular breads either at conception or in studying how their maintenance regime may accentuate the biological constituents and functionality of a culture. This seems in stark contrast to the modern approach to beer brewing where hundreds of races of different yeasts for different styles of beers can be purchased online and a good new strain is immediately sent to be frozen in liquid nitrogen or preserved by other methods in case of accidental loss or change in production. Although there

Taxonomic Structure and Stability of the Bacterial Community in Belgian Sourdough Ecosystems as Assessed by Culture and Population Fingerprinting

http://www.sourdoughlibrary.org/traditional-breads-italy/

Whole grain foods are valuable sources of minerals. A high content of phytate in these products has been considered a factor for limited bioavailability of these nutrients. Degradation of phytate may, however, result in an increased bioavailability of the minerals (29). This could be done during food processing like soaking, germination, malting, and fermentation. At optimal conditions for the enzyme phytase (55°C, pH 4.5–5.0) the phytate could be effectively reduced after 12–16 h of soaking. The acidity of the dough during breadmaking is of great importance for phytate degradation during scalding and sourdough fermentation. After 8 h of fermentation at 37°C, a reduction of 65% of the phytate content may be obtained in regular dough, compared to 97% in sourdough.
Whole grain foods and health - a Scandinavian perspective

Plant phytase has an optimum pH of 4.0-7.5 and optimum temperature of 40-60°C (Wodzinski and Ullah, 1996).

enzyme additives in baking
http://specialtyenzymes.com/products/term/baking

Further reading

Celiac Disease

Increasing prevalence of coeliac disease over time

Celiac Disease and Gluten: Multidisciplinary Challenges and Opportunities

Wheat deficient in gliadins: promising tool for treatment of coeliac disease

New Clues in Celiac Disease Epidemiology, Pathogenesis, Clinical Manifestations, and Treatment

Prevalence of Celiac Disease among Children in Finland

The Prevalence of Celiac Disease in the United States

http://www.webmd.com/digestive-disorders/celiac-disease/news/20100927/celiac-disease-can-develop-at-any-age

http://www.researchgate.net/profile/Javier_Gil-Humanes

Mapping of Gluten T-Cell Epitopes in the Bread Wheat Ancestors: Implications for Celiac Disease

Can an Increase in Celiac Disease Be Attributed to an Increase in the Gluten Content of Wheat as a Consequence of Wheat Breeding? including vital gluten consumption

Dessication with glysophate

http://www.agriville.com/cgi-bin/forums/viewThread.cgi?1376346944

Glyphosate, pathways to modern diseases II: Celiac sprue and gluten intolerance

Human cell toxicity of pesticides associated to wide scale agricultural GMOs

THE EFFECT OF GLYPHOSATE TREATMENT ON THE GERMINATION POTENTIAL OF RESULTANT CROPS

Physiological control of Hagberg falling number and sprouting in winter wheat and development of a prediction scheme

Sustainability of UK - grown wheat for breadmaking

Stabilising the Hagberg falling number in wheat - HGCA.com

The prevalence of celiac disease in the United States

A critical review of glyphosate findings in human urine samples and comparison with the exposure of operators and consumers

Pre-harvest glyphosate for weed control and as a harvest aid in cereals Review 065
no mention of possible bad effect of dead grain in flour

Pre Harvest Glyphosate and Desiccation

Human cell toxicity of pesticides associated to wide scale agricultural GMOs

Pre-harvest glyphosate for weed control and as a harvest aid in cereals

Expert Committee on Pesticide Residues in Food (PRiF)

Farmers Cope With Roundup-Resistant Weeds

WHO issues report categorizig glysophate as “probably carcinogenic”

Grain chemistry

Wheat gluten proteins and processing properties

Grain yield, nitrogen-use efficiency and baking quality of old and modern Italian bread-wheat cultivars grown at different nitrogen levels

Effects of HMW- & LMW-glutenins and grain hardness on size of wheat storage proteins polymers

The Chemistry of Cereal Proteins, Second Edition Radomir Lasztity

Phytate Degradation during Breadmaking: The Influence of Flour Type and Breadmaking Procedures

Highly efficient gluten degradation by lactobacilli and fungal proteases during food processing: new perspectives for celiac disease.

Historical shifts in the seed mineral micronutrient concentration of US hard red winter wheat germplasm

Degradation of Phytate by the 6-Phytase from Hafnia alvei: A Combined Structural and Solution Study

Increased understanding of the cereal phytase complement for better mineral bio-availability and resource management

Evidence of decreasing mineral density in wheat grain over the last 160 years Ming-Sheng Fan, Fang-Jie Zhao, Susan J. Fairweather-Tait, Paul R. Poulton, Sarah J. Dunham, Steve P. McGrath

Sourdough process for phytic acid and gluten degradation

Bread chemistry

Sourdough bread made from wheat and nontoxic flours and started with selected lactobacilli is tolerated in celiac sprue patients

Strains of Lactic Acid Bacteria Isolated from Sourdoughs Degrade Phytic Acid and Improve Calcium and Magnesium Solubility from Whole Wheat Flour

Highly Efficient Gluten Degradation by Lactobacilli and Fungal Proteases during Food Processing: New Perspectives for Celiac Disease

Phytate Degradation during Breadmaking: The Influence of Flour Type and Breadmaking Procedures

The Importance of Lactic Acid Bacteria for Phytate Degradation during Cereal Dough Fermentation

Moderate Decrease of pH by Sourdough Fermentation Is Sufficient To Reduce Phytate Content of Whole Wheat Flour through Endogenous Phytase Activity

Phytase active yeasts isolated from bakery sourdoughs

Reduction of phytic acid during breadmaking of whole-meal breads

Purple and Blue Grain

Cloning and characterization of purple acid phosphatase phytases from wheat, barley, maize, and rice

Use of wheat gene resources with different grain colour in breeding

Development of the New Winter Wheat Variety Skorpion (purple)

Miscellaneous

the HMW glutenin subunit allele database

Allelic variation in HMW glutenins in Spanish wheat landraces and their relationship with bread quality

http://www.slate.com/articles/health_and_science/human_evolution/2012/10/evolution_of_lactose_tolerance_why_do_humans_keep_drinking_milk.html

Interaction between protein, phytate, and microbial phytase. In vitro studies.

Eeckhout and De Paepe (1994) reported that rye (5130 units/kg), triticale (1688 units/kg), wheat (1193 units/kg) and barley (582 units/kg) were rich in phytase

Against the grain - NY Times

Making bread with sourdough improves mineral bioavailability from reconstituted whole wheat flour in rats

'Good bacteria' key to stopping asthma

Tummy-friendly breads

The good news is that you might not need to cut out bread completely.

Some people with wheat sensitivity have no problems when they eat toast (cooked wheat tends to be easier to digest), sourdough bread, bread cooked with flour made from French wheat, or any bread from a specialist bakery rather than a supermarket.

"Bakeries in supermarkets use the Chorleywood bread-making process, which cuts out the second rising to speed up the baking. People seem to have more problems digesting supermarket breads, so I'd always recommend avoiding store-bought loaves," says Dr Skypala.

Chemical composition of cereal grains (average values)

	Wheat	Rye	Corn	Barley	Oats	Rice	Millet
	(g/100 g)						
Moisture	12.6	13.6	11.3	12.1	13.1	13.0	12.0
Protein (N×6.25)	11.3	9.4	8.8	11.1	10.8	7.7	10.5
Lipids	1.8	1.7	3.8	2.1	7.2	2.2	3.9
Available carbohydrates	59.4	60.3	65.0	62.7	56.2	73.7	68.2
Fiber	13.2	13.1	9.8	9.7	9.8	2.2	3.8
Minerals	1.7	1.9	1.3	2.3	2.9	1.2	1.6
	(mg/kg)						
Vitamin B_1 (thiamine)	4.6	3.7	3.6	4.3	6.7	4.1	4.3
Vitamin B_2 (riboflavin)	0.9	1.7	2.0	1.8	1.7	0.9	1.1
Nicotinamide	51.0	18.0	15.0	48.0	24.0	52.0	18.0
Panthothenic acid	12.0	15.0	6.5	6.8	7.1	17.0	14.0
Vitamin B_6	2.7	2.3	4.0	5.6	9.6	2.8	5.2
Folic acid	0.9	1.4	0.3	0.7	0.3	0.2	0.4
Total tocopherols	41.0	40.0	66.0	22.0	18.0	19.0	40.0