CEREAL GRAINS: HUMANITY’S DOUBLE-EDGED SWORD

Long before our early ancestors had a written language or even a fulsome vocal one, Oonga somehow figured out how to let her kids know which plants, animals, insects, etc. were okay to eat and which would give them a bellyache, or worse. Oonga had picked this up from her mother and grandmother, as they in turn had learned from theirs. Nothing less than survival depended on these handed-down lessons.

Our own times are characterized by so much language—written, spoken, even sung—that survival lessons are hard to distinguish from commercials. But I’m zeroing in on clues to safe and sane nutrition: how close is a food to ones that nourished us as a species?

Most of the answers come from researchers who examine foods and/or diet records of isolated preindustrial hunter-gatherer societies, only some of which still exist. In the 1930s Weston Price and his wife produced irreplaceable documentation on indigenous diets that supported superb dental and overall health. Modern investigators like Michael Crawford, Melvin Konner, S. Boyd Eaton, Loren Cordain, Andrew Sinclair, Janette Brand-Miller, Katherine Milton and others carry on the search. Technology today enables them to analyze nutrient composition of foods believed to be identical, or similar, to those eaten by our ancestors during the 2-million-year stretch of our evolution (the Paleolithic period, or old Stone Age) that ended in different parts of the world between 40,000 and 10,000 years ago.

Mongongo Nuts, Anyone?

In the March 2000 Am J Clinical Nutrition (vol 71, no. 3), Loren Cordain et al. suggest that for the majority of 20th century hunter-gatherers, animal food (including “fished animal food”) made up a much greater percentage of diet compared to plant foods than formerly estimated, even though plant foods were basic to all diets. They say this added up to a higher energy intake from protein, and a much lower energy intake from carbohydrates, than in the U.S. diet today.

Katherine Milton in her editorial in the same issue argues that these societies were too varied for such a broad conclusion; our origins as primarily a plant-eating species incline us towards including plenty of plant foods as well. She points to the !Kung hunter-gatherers of Botswana, who may live “in conditions close to the ‘ideal’ hunting and gathering environment. What do the !Kung eat? Animal foods are estimated to contribute 33% and plant foods 67% of their daily energy intakes. Fifty percent (by wt) of their plant-based diet comes from the mongongo nut, which is available throughout the year in massive quantities.”

Both articles point to the lack of “diseases of civilization” in hunter-gatherer societies, regardless of whether more or less animal or plant food is eaten. Cordain et al. write: “Although cereal grains, dairy products, beverages, oils and dressings, and sugar and candy comprise [more than] 60% of the total daily energy consumed by all people in the United States, these types of foods would have contributed virtually none of the energy in the typical hunter-gatherer diet.”

Farewell to Paleolithic Life!

This issue’s heading is the title of a magnificent, long-needed review by Prof. Cordain of Colorado State University. About 10,000 years ago, human populations increased while large grazing herbivores became either extinct or severely depleted. Planting and harvesting, i.e., agriculture, gradually “became the dominant way of life, and cereal grains became the dominant caloric and protein source in many, but not all prehistoric cultures.”

As the title implies, we paid a price for this switch. Bones got weaker. Teeth became subject to decay. People got shorter. Yes, pre-agricultural Cro-Magnons who lived about 30,000 years ago were taller and stronger than we are today!

But I didn’t know until I read a 1998 paper by S. Boyd Eaton et al. that our brains had gotten smaller:

“Brain size has declined since the Paleolithic; the current average, 1,350 cm³, is about 11% less than estimates for early anatomically modern humans living before the development of agriculture. This decrease has occurred very rapidly compared with the several million year period during which the human brain enlarged.”

Dr. Eaton et al. conjecture that a factor in this decline might be “decreases in the availability of brain structural substrate,” specifically brain-building fatty acids. “...current DHA and AA consumption are both much below Paleolithic levels.”

That’s one effect of reliance on cereal grains, Cordain says: they have no omega-3 (w-3) DHA and very little alpha-linolenic from which DHA might be made. Grains don’t have AA either, but they supply w-6 linoleic acid, some of which can be converted in the body to AA (arachidonic acid). But gray matter needs DHA and AA about equally.

The Heart, Too

The w-3s play such intrinsic roles in cardiovascular health, that any strategy that reduces them becomes potentially dangerous. For instance, the U.S. Dept. of Agriculture’s “food pyramid” urges 6 - 11 servings of grain products a day (bread, pasta, cereals, etc.) at the expense of fruits, vegetables, nuts, seafood, and lean meat. Cordain says this would further overburden the excessively high w-6/w-3 ratio “from vegetable oils, margarine, and shortening” and may contribute indirectly to an essential fatty acid profile that promotes coronary heart disease.

Although cereal grains played little or no part in our evolution as a species, small amounts that don’t displace basic plant and animal foods may not harm us. But now they provide at least 50% of calories for the world: the poorer the country, the higher the percentage of calories from grains. The dilemma is, of course, that we can no longer survive without them.

Calcium

Cordain’s scholarly work clarifies how this dependence affects us, nutrient by nutrient. For instance, while cereal grains provide minerals, they have a quite low ratio of


calcium to phosphorus, as well as a calcium/magnesium ratio that also favors calcium excretion. The net effect of these unfavorable ratios and “low bioavailability of calcium via a high phytate content” is more bone mineral pathologies such as osteomalacia, rickets, and osteoporosis in populations “where cereal grains provide the major source of calories.”

Vitamin D

(T his one took me by surprise.) “Consumption of high levels of whole grain cereal products impairs bone metabolism not only by limiting calcium intake, but by indirectly altering vitamin D metabolism. In animal studies it has been long recognized that excessive consumption of cereal grains can induce vitamin D deficiencies in a wide variety of animals, including primates....” [My emphasis. CF]

Zinc

This essential trace mineral is needed in enzymes that conduct most of the body’s business. Deficiency is known to cause dwarfism and hypogonadism in men. Consumption of “whole grain cereals (wheat, rye, barley, oats and triticale) impairs zinc absorption...Yeast leavening of whole grain breads can reduce their phytate content and improve the bioavailability of zinc...” The bioavailability of zinc from meat “is four times greater than that from cereals.”

Growth

Human fossil records show a characteristic reduction in stature with adoption of cereal-based agriculture. Present-day populations “depending upon cereal grains for the bulk of their energy and protein also tend to be of short stature. There are a variety of reasons why cereal-based diets may impair linear growth. These include deficiencies in energy, protein, zinc, iron, copper, calcium, vitamin D, vitamin B₁₂ and vitamin A.”

Antinutrients

Cereal grains are seeds of grasses (monocotyledons) containing a variety of substances “which can be either toxic, antinutritional, benign or somewhere in between,” depending on the creature consuming them. While many birds, rodents, insects, and herbivores do well on grains, the majority of primates are not adapted to them in the same way they are to dicotyledons that form their traditional plant foods. Consequently, humans, like all other primates have little evolutionary experience developing resistance to secondary and antinutritional compounds which normally occur in cereal grains.” Here are a few and their actions:

Alpha-Amylase Inhibitors inhibit our starch-digesting enzymes, and also are allergens. Inhalation of cereal flours is the cause of baker’s asthma, an occupational allergy triggered by alpha-amylase inhibitor proteins.

Lectins are recognized “as the major antinutrient of food,” because these glycoproteins [complex sugars attached to protein], can bind to virtually every cell and extracellular substance in the body. Lectins can cause red blood cells to clump (agglutinate).

Of the eight commonly consumed cereal grains, lectin activity has been demonstrated in wheat, rye, barley, oats, corn, and rice, but not in sorghum or millet.” Lectins, “by their ability to increase the permeability of the intestine,” may facilitate passage into the blood of dietary antigens, i.e., stuff the immune system spots as foreign. Lectins themselves may be transported across the intestinal wall into systemic circulation, where they have “the potential to interfere with the body’s normal hormonal, metabolism, and health.”

A Role in Autoimmune Illness

Autoimmune diseases occur when the body loses the ability to discriminate self proteins from nonself proteins. This loss of tolerance ultimately results in destruction of self tissues by the immune system.”

Cereal grains are the “known environmental causative agent for at least two autoimmune diseases: celiac disease and dermatitis herpetiformis.” Gluten proteins in wheat, rye, and barley are the culprits, principally gliadin. (Other autoimmune ailments where circulating antibodies to gliadin are seen include rheumatoid arthritis, the kidney ailment IgA nephropathy, Sjögren’s Syndrome, and recurrent mouth canker sores.)

Dermatitis herpetiformis (DH) is a nasty chronic skin eruption causing severe itching and burning. The only “medical” cure is a gluten-free diet. In celiac disease (CD) the tiny villi that line the small intestine are destroyed and along with them much ability to absorb nutrients. Fortunately, rice, corn, millet, sorghum, quinoa, buckwheat, and amaranth don’t trigger these illnesses.

Psychological & Neurological Illnesses in Celiac Disease

“Neurological complications have long been recognized in celiac patients and can include epilepsy, cerebellar ataxias, dementia, degenerative central nervous system disease, peripheral neuropathies (of axonal or demyelinating type), and myopathies.”

Incidence of schizophrenia is about 30 times higher in celiac patients than in the general population...schizophrenics have elevated circulating IgA antibodies to gliadin.” Multiple clinical studies have shown that “schizophrenic symptoms improved on gluten-free diets and worsened upon reintroduction.” In populations eating little or no wheat, rye and barley, the prevalence of schizophrenia is quite low.

The Good and the Bad

“From an evolutionary perspective, humanity’s adoption of agriculture, and hence cereal grain consumption, is a relatively recent phenomenon,” Cordain writes. “Cereal grains obviously can be included in moderate amounts in the diets of most people without any noticeable, deleterious health effects, and herein lies their strength.” The energy efficiency in their cultivation “allowed for the dramatic expansion of worldwide human populations which, in turn, ultimately led to humanity’s enormous cultural and technological accomplishments.”

But we didn’t know, until nutrition science’s great strides in the early part of the century, that people who subsisted largely on grain products could suffer grim vitamin and mineral deficiencies.

Nor did we know until the early 1950s that wheat gluten caused celiac disease; and in the past 10 years “has come the evidence (admittedly incomplete) that certain cereal peptides may interact with the immune system to elicit a variety of autoimmune-related diseases...the human immune, digestive and endocrine systems have not yet fully adapted to a food group which provides 56% of humanity’s food energy and 50% of its protein.”

He concludes: “Cereal grains are truly humanity’s double-edged sword. For without them, our species would likely have never evolved the complex cultural and technological innovations which allowed our departure from the hunter-gatherer niche. However, because of the dissonance between human evolutionary nutritional requirements and the nutrient content of these domesticated grains, many of the world’s people suffer disease and dysfunction directly attributable to the consumption of these foods.”

A Felix Aside on Gluten

In Italy, where much good research on gluten intolerance is being done, the national health service provides gluten-free staple foods to people diagnosed with DH and/or CD. The U.S. medical community has a lot of catching up to do, both in awareness and research, but a new multisector study that screened over 7000 individuals for CD found the prevalence to be 1 in 257 (that’s about 1 million persons – huge potential consumer market for gluten-free foods), “indicating that CD is as common in the U.S. as in Europe.” From Celiac Disease Foundation Newsletter, Summer 2000. Foundation tel. 818-995-2354. 13751 Ventura Blvd, Suite 1, Studio City CA 91604-1838. email: cdff@celiac.org
MY DRUG OF CHOICE

The connection of smoking to heart disease may appear to be less direct than its link to lung cancer, but a new study proves cigarette smoking "strikingly impairs coronary microcirculation, and interferes with regulation of myocardial blood flow....However, these derangements are reversed by acute administration of vitamin C," according to Lancet of Sept 16.

Philipp Kaufmann (Zurich, Switzerland) and coworkers found considerably reduced blood flow in heart muscle in 11 healthy smokers compared to 8 non-smokers. After infusing 3 grams (3000 milligrams) of vitamin C into all subjects, there was no change in the non-smokers, but smokers responded with normalized myocardial blood flow.

Researcher Paolo Camici said smokers have too-low plasma and tissue concentrations of vitamin C, so smoking leads to both "decreased natural antioxidants and an increased oxidative burden." A large-scale trial is warranted, he said, to test whether daily oral vitamin C supplementation can prevent coronary artery disease in smokers.

Although he encourages smokers to eat more fruits and vegetables, he stresses it's "premature to extrapolate from our study to encourage people who smoke to use vitamin C as a drug."

RESCUING THE HEART

A mysterious molecule is showing up in the news, and according to Dr. John Danesh of Oxford University, the news is not good. His group gathered data from 27 studies tracking over 3,000 people who had heart disease or survived a heart attack. During a decade of follow-up there were 70% more heart attacks in individuals who had the highest serum levels of this curious molecule: lipoprotein(a), or Lp(a) for short.

The Oxford report in Circulation (Sept. 4), a journal of the American Heart Assoc., struck a despairing note. Although Lp(a) is a fat and cholesterol-transporter in blood, like LDL (low-density lipoprotein), little can be done to modify it, either by diet or drugs. Danesh says, because most of Lp(a) blood levels are determined by one's genes.

New Light on Heart Disease

Hold on there, Danesh et al.! There's a lot more to the story and it's by no means gloomy. In a series of articles in 1991, Matthias Rath and Linus Pauling described Lp(a) and the roles it plays in human health. Rath, an MD from Hamburg University, having proven with colleagues that atherosclerotic lesions in human arteries are largely composed of Lp(a) rather than LDL molecules, sought a reason for this phenomenon. He found it in the discovery that Lp(a) exists primarily in species which can't synthesize their own vitamin C. "In contrast, animals able to produce optimum amounts of vitamin C do not need lipoprotein(a) in any significant amount."

Actually, Lp(a) is LDL plus a super-adhesive protein--apolipoprotein(a)--surrounding it. The adhesive apo-protein(a) makes Lp(a) "one of the stickiest particles in the body," Rath writes.2

The Price for Low Ascorbate

Vitamin C's depletion in scurvy causes breakdown of the body's connective tissues, including blood vessel walls. As blood oozes out, Lp(a) goes into emergency mode, binding to the blood-clotting factor, fibrinogen, to form a tangled fibrin network to stop hemorrhage.

Fatal scurvy may be uncommon in the U.S., but chronic vitamin C (ascorbate) deficiency affects millions. The vitamin is needed to synthesize the 'cement' (e.g., collagen and elastin) needed for sturdy, resilient artery walls. Rath and Pauling proposed that human cardiovascular disease is "primarily a degenerative disease caused by chronic ascorbate deficiency." [My emphasis. CF]

"The extracellular deposition of Lp(a) and fibrinogen is a defense mechanism to limit the damage done by this deficiency. Under chronic conditions the defense may, however, turn into a pathologic process leading to the continued accumulation of Lp(a) and fibrinogen in the vascular wall."3

In his 1997 book, Dr. Rath explains how this deficiency leads to "millions of small lesions and cracks in the artery wall, particularly in the coronary arteries. The coronary arteries are mechanically the most stressed arteries because they are squeezed flat from the pumping action of the heart more than 100,000 times per day...."

Cholesterol and other repair factors "are produced at an increased rate in the liver and transported in the bloodstream to the artery walls...in order to mend and repair the damage there." But with chronic ascorbate deficiency over many years, "the repair process overshoots. Atherosclerotic plaques form predominantly at those locations in the cardiovascular system with the most intensive repair: the coronary arteries."

There goes the hi-cholesterol theory of heart disease! Cholesterol carriers like LDL and Lp(a) become villains only after they've overdone their job as emergency repair crews, when the 'emergency' turns into a chronic state.

The Solution

Since sticky Lp(a) accumulates the most, yet its blood levels are mainly genetically determined, how do we get around the problem? The answer, of course, lies in nutrition. (That's why I love my work!)

Keep your blood vessels strong. Avoid any need for repair jobs (except in case of real wounds). Ascorbate is the major tool, but not in silly RDA amounts. Think more in terms of the intake of Dr. Katherine Milton's wild monkeys. Dr. Rath espouses a full-bore high-dosage, total vitamin-mineral program. (Both vitamin C and niacin have been shown to lower Lp(a) levels, he says.)


In addition, he says the amino acids lysine and proline provide a teflon-like surface for artery walls that not only discourages Lp(a) and LDL molecules from latching on, but nudges those already stuck to quit the artery wall! As Lp(a) and LDL molecule by molecule are released from atherosclerotic deposits, the process leads "to a natural reversal of cardiovascular disease."

He offers an example from nature. Hibernating bears don't eat, become depleted of many vitamins, make very little vitamin C, and build up big fatty deposits in their arteries. Come spring, as the bears' food/vitamin intake and ascorbate production soar, the fatty deposits in their artery walls "gradually reverse, and artery walls retain their natural stability and function."

Rath's book is easy to read, lays out his supplement program, and is well-referenced. He also offers the most passionate accusations I've ever read against the international pharmaceutical cartels: "There is an entire industry with an innate economic interest to obstruct, suppress, and discredit any information about the eradication of diseases...This mission of this industry is to make money from ongoing diseases. The cure or eradication of a disease leads to the collapse of a multi-billion dollar market of pharmaceuticals." Get the book--there's much more.

NEW AHA GUIDELINES

Researcher and friend Paul Stitt sent me the new American Heart Association Dietary Guidelines, and it's full of the usual admonitions to limit intake of saturated fats and cholesterol. (It also has a nice surprise, as you'll see.) Major monies still flow towards research propping up the AHA's shaky premise that high serum cholesterol is the cause of coronary heart disease. Because of the false alarm that cholesterol-rich foods raise havoc with our arteries, invaluable traditional foods like shell fish and especially organ meats are feared in the U.S.

Also, the food industry, in response to medical outcry about its use of saturated animal and tropical vegetable fats, now substitutes partially or lightly hydrogenated vegetable oils in its products. These are the sources of trans-fatty acids, consumed in epic amounts in chips, cookies, cakes, breads, puddings -- even in baby biscuits. These phony get into cell membranes, where they bollix up normal cell functions and increase requirements for the real fatty acids.

The new Guidelines recommend limiting intake of trans-fatty acids.

Saturated Fats Are Not Villains

On the other hand, natural saturated fats are old friends of humankind. Even if we don't eat them, our bodies are very handy at making them--not just to round out our tushies, but to support every one of our cell membranes. Breast milk is full of saturated fats, with lots of 'medium-chain' ones that are easy for babies to digest. So I don't go along with the advice to keep saturated fats and cholesterol intake very low. Cordain, Eaton, and others say our late Paleolithic ancestors went after fish and game that provided both, but also enough w-3s to keep things in balance.

Thankfully, the Guidelines do not recommend very-low-fat diets. Not only can these lead to essential fatty acid deficiencies, they say, but they're "often associated with the use of processed low-fat foods that are calorie dense." Many of us believe the alarming epidemic of fatties has much of its roots in the medical low-fat campaigns that lead the public to misinterpret as 'medically recommended' the most worthless junk carobs, so long as these sport shiny LO-FAT! or NO-FAT! labels.

Antioxidants Yes, Supplements No

While possible protection from heart disease by antioxidants is noted, the AHA insists these come solely from fruits, vegetables, etc. (Use of supplemental vitamins C and E, to be sure, is discouraged.)

The Guidelines acknowledge the newer, sounder theory that excess plasma homocysteine levels are associated with cardiovascular disease, and that folic acid, vitamins B6, B12, and riboflavin are needed to metabolize homocysteine properly. They commend folate fortification of food grain, but say nothing about supplements. (I, on the other hand, approve of supplements of folic acid, B6, B12, and trimethyl glycine to accomplish what diet alone may not.)

A Big Step Forward

Now for the AHA's surprise. Increase intake of w-3-rich foods, they say, because foods rich in EPA and DHA confer such benefits as:
• reduction in sudden death
• decreased risk of arrhythmia
• lower plasma triglyceride levels
• reduced blood-clotting tendency

Moreover, "There is some evidence from epidemiological studies that another w-3 fatty acid, a-linolenic acid, reduces risk of myocardial infarction and fatal ischemic heart disease in women. Several randomized controlled trials recently have demonstrated beneficial effects of both a-linolenic acid and marine w-3 fatty acids on both coronary morbidity and mortality in patients with coronary disease. Because of the beneficial effects of w-3 fatty acids on risk of coronary artery disease as well as other diseases such as inflammatory and autoimmune diseases, the current intake, which is generally low, should be increased. Foods source of w-3 fatty acids include fish, especially fatty fish such as salmon, as well as plant sources such as flaxseed and flaxseed oil (emphasis mine. CF), canola oil, soybean oil, and nuts. At least 2 servings of fish per week are recommended to confer cardioprotective effects."

No, I'm not dreaming this. There's hope, even for the AHA.

ERRATUM REPAIR

Having met and spoken a number of times with Artemis Simopoulos, who happens to be a great research toer and a very good Greek dancer, I no longer refer to her, as I once did in a newsletter, as a he. She kindly informed me, however, that she was born in Greece, not, as I wrote in Fls 110/111, on the Greek island of Crete. (I fell in love with both Ancient Olympia and Crete in June, during and after participating in the great conference she co-chaired.)

Illustrations are by the late Clay Geerdes and other artists as noted.

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