Plants, Food, and Human Health: You Are What You Eat?

We need food to survive, and plants are an important part of any human diet, whether eaten directly, indirectly (by consuming plant-eating animals or animal products), or as modified by industrial processing. Changes to our diet, brought about by the development of agriculture and industrial food processing, have had both positive and negative effects on human health. Humans evolved consuming only foods that grew and grazed naturally, with big seasonal variations and periods of food scarcity. The development of agriculture ~10,000 years ago changed patterns of food consumption, leading to an increased reliance on grains and the potential for a more predictable food supply. Also as a consequence of the agricultural revolution, our capacity to produce food has increased enormously, as has our life expectancy. More recently, postindustrial changes in food production and changes in lifestyles have led to many people habitually consuming diets whose compositions lie outside any that were met during our evolutionary history. The rate of change of our eating habits has vastly exceeded any opportunities for evolutionary change, so often there is an imbalance in what the body needs and what the body gets. While many people suffer from chronic hunger and malnutrition, currently more humans in the world suffer the ill effects of eating too much overall, and with a poor nutritional balance (i.e., too much of the wrong kinds of foods and not enough of the right kinds). Increased incidences of most chronic diseases can be traced back to the misalignment of modern diets with the physiological needs of our bodies. About 80% of all incidents of type 2 diabetes and 30% of heart disease and cancers can be attributed to obesity, poor diet quality, and physical inactivity, along with the increased longevity that provides more time for these chronic diseases to develop. This lecture describes the chemical composition of food, how different foods affect us physiologically and can contribute to the prevention or development of chronic diseases, and how nutrition research, agricultural practices, and plant breeding programs can contribute to more healthful diets.

WHY WE EAT WHAT WE EAT

Twenty years ago, the Five a Day for Better Health program was initiated, with the objective of encouraging Americans to increase their dietary intake of fruits and vegetables to five servings a day (similar programs were launched in other countries). Although most Americans and Europeans are familiar with this dietary guideline, on average no more than 30% follow it. This raises one of the challenges of public health; even when a clear cause-and-effect relationship is established, people have the liberty to ignore it. In the 1950s, the correlation between smoking and lung cancer was established, but more than a billion people currently smoke (among countries, the percentage of the adult male population that smokes varies from <10 to >70). The powerfully addictive effect of nicotine is a culprit, but the choice to smoke is more complicated; the decision to start smoking has other nonphysiological origins. Food choices are similarly complex. We eat because it is necessary to do so. Multiple neuro-endocrine regulator systems (including those involved in addictive behaviors) ensure that some time after eating, the feeling of satiety wanes and appetite develops. If eating is delayed, we go on to develop hunger and would fight for food if necessary. These are basic evolutionary survival mechanisms. There are subtle physiological controls over taste sensation that direct us to a balance of foods and nutrients. However, the most potent drivers over our choices of what and how much we eat, which can overcome our physiology, have roots in our cultural, social, and emotional status, economic pressures, and the pervasive effects of marketing and media. Confusion about which foods or balances of foods are and are not beneficial is rampant, and as scientists, we can help others to understand how food affects health and understand how to interpret research and often misleading news stories.

YOU ARE WHAT YOU EAT? FOOD AND NUTRITIONAL STUDIES

The old adage “you are what you eat” is overly simplistic and fails to capture the complexity of nutritional sciences. Our diet, what we eat, is one of the three interacting components that together define nutritional status, which in turn influences our growth, our development, our physical, mental, and social function, and our health. Nutritional status comprises (1) what we eat, diet quantity and nutritional quality; (2) what we are, body tissue and organ sizes and compositions; and (3) what we can do, our functional capacities, at molecular up to social levels, importantly including physical activity and enzymatic function. Seen in this way, it is clear that what we eat can only influence what we are if what we are has equipped the gut with adequate digestive functional capacity and the liver with synthetic enzyme capacity, and so on.

WHAT WE GET FROM FOOD

Unlike plants, humans cannot synthesize our own food, and so instead we must eat other organisms; we are heterotrophs (other
eaters). We eat plants and animals (or their milk or egg products) that themselves eat plants, so plants directly or indirectly are the foundation of our food supply. In the context of food, plants and animals are chemical assemblages that we can define as macronutrients (carbohydrates, proteins, and lipids) and micronutrients (vitamins, minerals, and phytonutrients, some of which are bioactive). Our bodies require macronutrients as fuel and as building blocks for growth, and micronutrients for their protective properties, their structural properties, and their roles as coenzymes. We will briefly describe these nutrients, how we assimilate them, and their functions in our bodies.

**Macronutrients**

Macronutrients supply energy (calories) and building blocks of cellular structions. They comprise carbohydrates (3.75 kcal/g), proteins (4 kcal/g), and lipids (9 kcal/g). Dietary fiber is a subtype of carbohydrates that provides ~2 to 3 kcal/g.

**Carbohydrates**

Carbohydrates consist of carbon, hydrogen, and oxygen, the name deriving from the proportion of elements [C-(H₂O)], and include sugars, starches, and celluloses. The basic unit of a carbohydrate is a simple sugar, a monosaccharide. Glucose (Glc) and fructose (Fru) are common monosaccharides. Sucrose (Suc) is a disaccharide made from two simple sugars, Fru and Glc. More complex carbohydrates are polymers of simple sugars. Complex carbohydrates include starches and celluloses, both of which are made from Glc but differ in how the Glc monomers are joined. Starch is readily digested to sugars because we produce an enzyme, α-amylase, that breaks apart the α-bonds that join the Glc units in starch. By contrast, we do not produce an enzyme capable of breaking apart the β-bonds that join the Glcs in cellulose, so to us this carbohydrate is indigestible. As will be discussed later, even indigestible carbohydrates affect our health in a variety of ways.

Simple sugars, particularly Glc, are key players in cellular energy production. In an animal, Glc is distributed to cells throughout the body through the bloodstream. Within the cell, Glc is metabolized to produce the real currency of cellular energy, ATP; the by-products of Glc metabolism are carbon dioxide and water. An animal that ingests more carbohydrate than it needs can store the excess as another complex carbohydrate, glycogen, or convert it to the more compact storage form of fat. Each [C-(H₂O)] unit of digested carbohydrates can be oxidized by two oxygen atoms to CO₂ + H₂O.

Indigestible carbohydrates are referred to as dietary fiber, of which there are two forms, soluble and insoluble. Soluble fiber includes pectins, gums, and mucilages and can have a gel-like consistency that is familiar to those who eat soluble fiber-rich foods like oatmeal, okra, and eggplant. Soluble fibers are beneficial in that they can slow the rate of assimilation of fats and sugars from the intestine. Most undigested carbohydrates (i.e., dietary fiber and starch that has been denatured [retrograded] by cooking) are used as fuel by gut bacteria. Some are referred to as prebiotics, meaning that they preferentially enhance the growth of beneficial bacteria in the intestine. These beneficial bacteria compete with pathogens and generate metabolic products, such as short-chain fatty acids, which contribute to intestinal and hepatic nutrition and so indirectly enhance digestion. Insoluble fibers include plant cell wall materials, such as celluloses, hemicelluloses, and lignins, and are abundant in skins of fruits and seed coats (sometimes called the bran). Some of these compounds are metabolized by gut bacteria, but some are inert and not metabolized, although they may contribute to a feeling of satiation.

**Proteins**

Proteins are polymers assembled from amino acids that are used for protein assembly in all organisms. Amino acids contain nitrogen as well as carbon, hydrogen, and oxygen, and two protein-forming amino acids also contain sulfur. Within the digestive system under the actions of enzymes secreted into the gut, dietary proteins are broken down into the component amino acids, which are assimilated and distributed in the bloodstream to cells throughout the body. Some amino acids can be converted into others, but a few are not made by our bodies, so these essential amino acids must be acquired through our diets. Because humans (and other animals) are heterotrophs and ingest complex foods, there has been low to no selective pressure to retain the amino acid biosynthetic pathways present in our autotrophic ancestors; thus, many have been lost, presumably through genetic drift.

Most ingested amino acids are used as a source of energy, entering metabolic pathways similar to those for carbohydrates, and providing a similar amount of energy (4 kcal/g). However, some amino acids are used to contribute to growth and physiological turnover of proteins, entering the amino acid pools to be taken up by cells and reassembled into proteins as directed by the cell’s genetic material. Meat, milk, and eggs are concentrated sources of protein from animals, but vegetarians can get ample dietary proteins through the consumption of protein-rich plant foods, such as beans and grains. Too little dietary protein can retard growth and cause immunological deficiencies, whereas when proteins are ingested in excess of the body’s needs, the excess amino acids can be further degraded and used for energy production. High-protein diets (e.g., >25% of energy) suppress appetite and can result in weight loss, but at a price. Diets excessively high in dietary proteins, including some trendy very-low-carbohydrate diets, have been associated with damage to the kidneys, which are involved in the excretion of the degraded amino acid by-products.

**Lipids: Fats and Oils**

Lipids are hydrocarbons, made primarily from carbon and hydrogen, largely as chains of 16 to 20 carbons in the proportion C-H₂, hence, their name. Dietary lipids are mostly assimilated as fatty acids, plus a variety of other compounds, such as sterols (including cholesterol from animal sources). Although once thought to be a dietary hazard, it is now recognized that dietary cholesterol has relatively little bearing on blood cholesterol levels because most blood cholesterol is synthesized in the body. Fatty acids are composed of a carboxylic acid and a long
hydrocarbon tail and occur in foods mainly as triglycerides, with three fatty acids linked to a glycerol molecule. To be absorbed, dietary triglyceride must first be kept in solution by mixing with bile, to form micelles, and then triglyceride molecules are cleaved into free fatty acids and mono- or diglycerides by the action of enzymes in the gut lumen. They are absorbed and reassembled into triglycerides in the mucosal cells. The medium-chain triglycerides enter the portal vein directly, whereas other fats are transported via lymph and the thoracic duct. Within cells, fatty acids are broken down through \( \beta \)-oxidation to produce acetyl-CoA, which then enters the citric acid cycle to release energy and carbon dioxide. Sterols and lipids are also used as building blocks, for example, for the formation of cellular membranes and as precursors for the synthesis of steroid hormones.

The physical and patho-physiological properties of lipids are dictated by the length of the hydrocarbon tail and the extent to which it is saturated with hydrogen. A fatty acid that contains the maximum number of possible hydrogens is said to be saturated. In unsaturated fatty acids, fewer hydrogens are present, so adjacent carbon atoms form a double bond with each other. A fatty acid with a single double bond is said to be monounsaturated, and one with multiple double bonds is polysaturated.

Because of the way the carbon atoms line up, a saturated fatty acid is a very straight molecule, and saturated fatty acids pack together very tightly into a compact, rigid substance (like a well-built brick wall). Saturated fatty acids are abundant in animal tissues (e.g., lard and butterfat) and are solids at room temperature. By contrast, double bonds cause the fatty acids to bend, so they fit together in a much looser, more flexible arrangement (like a fence built from twigs). Unsaturated fatty acids are usually liquids at room temperatures and are abundant in oils extracted from plant seeds or some fruits (e.g., olives).

Trans-fatty acids are present in some animal foods, particularly in milk and dairy products like cheese, but contribute <2% of dietary energy from these sources. Human biology largely lacks the capacity to metabolize trans-fatty acids, and they have similar adverse effects to saturated fatty acids of increasing low-density lipoprotein (LDL) cholesterol (see below). About 100 years ago, a method was developed to convert less expensive plant oils into butter-like solids (i.e., margarine), which is easier to store and transport and useful for cheap baking. This process is called hydrogenation and involves passing hydrogen gas through unsaturated fatty acids, converting them into more highly saturated, solid form. The problem is that the resulting hydrogenated fatty acids form in an unnatural orientation. Naturally occurring fatty acids are bent around double bonds in such a way that the two carbons fall on the same, cis, side of the bond. The hydrogenated fatty acids form bonds in which the carbons fall on opposite, trans, sides of the bond. This orientation makes the molecule straighter, more closely resembling a saturated fatty acid and causing the molecules to pack more tightly into room temperature solids. Over time, trans fats became widely used ingredients in many processed foods, but more recently we’ve learned that they contribute to chronic health problems. Because of these proven health effects, many countries require foods containing trans fats to be labeled, and efforts are underway to ban or limit their use in prepared foods and restaurants. Denmark was the first country to ban sales of foods with over 2% trans fats, and many have now followed. Unfortunately, the food industry has reacted by replacing it with saturated fatty acids. Denmark has now put a large tax on foods high in saturated fat. It is likely that this will finally lead to a less atherogenic food supply and less diabetes, heart disease, and strokes.

Omega-3 (or n-3) and omega-6 (n-6) long-chain polyunsaturated fatty acids (PUFAs) have special importance in our diet. The omega in the name of these fatty acids refers to the last carbon group and the number to the first double bond encountered from the end. The n-3 fatty acids have a double bond three carbons in, and n-6 fatty acids have a double bond six carbons in (as well as at other positions in the fatty acid). Our bodies cannot synthesize the n-6 PUFA linoleic acid (LA) and the n-3 PUFA \( \alpha \)-linolenic acid (ALA), so these must be assimilated through our diet. Both are abundant in seed oils; LA is abundant in common vegetable oils, including safflower, sunflower, maize, and soybean oils, and ALA in flaxseed (\textit{Linum usitatissimum}). These PUFAS have structural roles in cell membranes and act as precursors for signaling molecules, the eicosanoids. Eicosanoids can have either pro- or anti-inflammatory functions. The eicosanoids derived from n-3 fatty acids are typically anti-inflammatory and health enhancing.

The catalytic reactions downstream of LA and ALA are very inefficient in humans, so our health can be enhanced by dietary uptake of some of these downstream products. Eicosapentaenoic acid and docosahexaenoic acid are n-3 fatty acids that are abundant in fish oils. The fish accumulate them from the marine algae they consume, and some vegetarians supplement their diet with algae-derived eicosapentaenoic acid or docosahexaenoic acid. Most health organizations have concluded that a diet enriched for n-3 fatty acids helps protect against chronic disease. Although n-6 fatty acids are also essential, a high dietary ratio between n-3 and n-6 fatty acids is particularly beneficial, in part through increased production of anti-inflammatory eicosanoids. Since most people consume sufficient n-6 fatty acids without much effort because they are abundant in cereals and in meat from animals that have been reared on cereal feed, boosting n-3 consumption through ingestion of fatty fish, flaxseed oil, or supplements is generally considered to be a good idea. There is no need to consume n-3 fats daily because they are stored in adipose tissue. Americans are encouraged to eat more seafood, and in the UK, there is a national recommendation to eat two portions of fish per week (including one portion of oil-rich fish) or three portions for people at high risk of heart disease. However, world fish stocks could not supply this rate of consumption, so other sources must be considered. Additionally, due to the high levels of mercury found in predatory marine fish (e.g., albacore, tuna, halibut, and shark), freshwater and small sea fish (e.g., salmon and pollock) are preferred sources of omega-3 fatty acids.

\section*{Micronutrients: Vitamins, Minerals, and Phytonutrients}

The macronutrients described above are the major sources of energy and building blocks needed by our bodies. For optimal health, our diets must also include several small bioactive molecules, including vitamins, minerals, and phytonutrients.
Vitamins

The term vitamin was coined early in the 20th century, a time during which many small molecules containing amine groups were recognized as being vital for life (vital + amine); subsequently, it was found that not all vitamins have amine groups, but the name remains. Vitamins’ discoveries came from studies of people or lab animals with very restricted diets. Healthy people can meet their needs for most vitamins through diet alone, without supplements. However, many vitamins are only present in significant amounts in fruits and vegetables, and as indicated earlier, most people do not eat the recommended five servings a day. This problem is aggravated by poverty and poor education, both in developing and developed countries.

The number of recognized vitamins currently stands at 13, but the classification and decalification of substances as vitamins as an ongoing process. The 13 vitamins are A, C, D, E, K, and eight B vitamins; furthermore vitamins D and E are found in multiple forms. Vitamins have several functions. Vitamin A, which is synthesized in the body from provitamin A (β-carotene) in the diet, is converted to retinol, the photo-pigment in the eye. Dietary provitamin A and other closely related carotenoids are important for eye health and vision; vitamin A deficiency is widespread and a leading cause of blindness, which can be prevented by increasing provitamin A in the diet, as described further below. Vitamin A has many other functions, perhaps the most important being in our defenses against infections. It is fat soluble and extensively stored in the liver, being transported to other tissues by a dedicated retinol binding protein. In overdosage it is toxic and can even be fatal. However, hypervitaminosis A has only been associated with diets rich in vitamin A and not with plant-derived β-carotene (provitamin A).

The eight B vitamins share the feature of being water soluble and mainly acting as enzyme cofactors but otherwise vary in their structure and function. As an example, vitamin B9 (folic acid) is found in leafy green vegetables and legumes, but because there is a strong correlation between maternal folic acid deficiency and birth defects, in many countries, folic acid is added to fortified flours and bread. In the US, vitamins B1 (thiamine), B2 (riboflavin), and B3 (niacin) are also added to fortified flour and bread. Vitamin B12 (cobalamin), which is found mainly in meat, eggs, milk, and fermented foods, may be required as a supplement for vegans.

Vitamins C and E are often referred to as antioxidants, although this is not their only or most critical role; there are many other antioxidant systems in the body. The health benefits of antioxidants have long been thought to be through their protective function against cellular oxidants (e.g., reactive oxygen species, environmental toxins, and by-products of metabolism), but their roles are now thought to be more complex. Vitamin C (ascorbic acid) is water soluble and found in most fruits and vegetables but is particularly abundant in citrus fruits. Vitamin E comprises eight closely related fat-soluble compounds (tocopherols and tocotrienols) found in some oils (e.g., wheat germ oil and nut oils) and other foods. Among these different naturally occurring compounds, α-tocopherol has the greatest vitamin E activity.

Deficiencies in vitamins can cause serious health problems, but health problems are also associated with excessive ingestion of vitamins. Some vitamins may have pharmaceutical actions when taken in very large doses (e.g., vitamin A derivatives are used to treat some skin diseases). Some may become toxic, particularly the fat-soluble vitamins (e.g., A and D), which are prone to accumulate in the body. Water-soluble vitamins also can be unhealthy in high doses; for example, excessive vitamin C can cause uric acid crystals to deposit in joints, and there is some concern that high doses of folic acid may exacerbate tumor formation. The evidence for antioxidant vitamins and carotenoids is fairly consistent that in supplemental high doses they increase heart disease and cancer deaths. Thus, in general, vitamin supplements should only be taken to correct deficiencies or proven malabsorption conditions, but unfortunately they are still widely marketed to a vulnerable public.

Minerals

Vitamins and the phytonutrients described below are complex carbon-containing organic molecules that are synthesized in plants or other organisms. By contrast, minerals are elements. Elements are not biologically interconverted to other elements, so an organism’s needs must be met by assimilation. Many dietary minerals are necessary for human health. Iron deficiency is the most widespread nutritional disorder and affects over two billion people. Iron is the oxygen binding element of hemoglobin and myoglobin that is found in red blood cells and muscle. Menstruating women who regularly lose iron in blood and young growing children are most susceptible to iron deficiency. Zinc deficiency is nearly as widespread. Calcium, selenium, iodine, and potassium can also be limiting for optimal health.

Mineral nutrition is affected by the mineral’s bioavailability, which is determined by whether it is conjugated to or chelated by other molecules, and the presence of other nutrients in the gut. Mineral assimilation from plant material can be less efficient than from animal-derived food, meaning those who have largely vegetarian diets are most at risk of mineral deficiencies. For example, iron can be readily assimilated from red meat because it is in conjunction with heme. Plants are also sources of iron, but the non-heme iron in plants is less readily assimilated than heme iron. Zinc is also readily assimilated from meat but less so from plant materials. Furthermore, there is a physiological competition for the mechanisms to absorb some minerals, which can become a problem for people who take mineral supplements; iron supplements can lead to zinc deficiency and zinc supplements to copper deficiency.

Iodine is an essential mineral nutrient that is incorporated into thyroid hormones. Dietary iodine comes from seafood as well as plants and milk, but in these terrestrial sources the amount varies with soil levels. The World Health Organization estimates that 2 billion people around the world are iodine deficient. In many mountainous regions, such as the Alps and the Himalayas, soil iodine levels are low, so grass and then milk are deficient in iodine and human deficiencies are not uncommon. Subclinical iodine deficiency recently has been recognized as a problem in European countries, possibly as a consequence of changing diets. Adequate iodine is most critical during pregnancy, when deficiency can impair neurodevelopment. The World Health Organization (2007) describes iodine deficiency as “the world’s
greatest single cause of brain damage.” Although iodized table salt continues to be an important instrument to address iodine deficiency, salt consumption is being discouraged, so alternative sources like seaweed are being encouraged.

Several compounds in plants, including positively charged compounds, such as phytate (myoinositol hexaphosphate) and oxalate, and polyphenols, interfere with mineral assimilation by binding strongly to the minerals, whereas other compounds, including vitamin C can enhance mineral uptake. Similarly, the uptake of calcium and phosphorous is highly dependent upon adequate levels of the active form of vitamin D; a deficiency of vitamin D is manifested as a calcium deficiency and leads to rickets. Finally, the amount of a mineral found in a plant is correlated with the availability of the mineral in the soil. Some soils are very deficient in selenium or zinc, and people whose food is grown in deficient soils can themselves become deficient.

One of the challenges to addressing human vitamin and mineral nutrition is the uncertainty as to what are the minimum levels necessary to support good health. It is worth reflecting that the Dietary Reference Values, used to characterize the diets of groups as adequate or not, were derived from many isolated and sometimes inconsistent results from research, and most tend to err on the generous side, so many individuals with apparently low dietary intakes are perfectly healthy. There is very active debate, for example, over the most desirable level of calcium consumption. Recommendations vary widely, and most evidence shows rather little or no hazard with substantially lower intakes than recommended. Similarly, research is showing no adverse effects for most people with levels of iodine consumption well below the lower cutoff considered adequate. More accurate and reliable data are still needed to determine when and where to focus intervention efforts.

**Phytonutrients and Functional Foods**

Plants produce a huge number of chemicals, many of which have roles in their protection against pathogens or herbivores or as attractants for pollinators or seed dispersers. Many of these chemicals are relatively recent evolutionary innovations and found only in a small number of plants or plant families. Many of these compounds, commonly called phytonutrients, can have beneficial effects on human health. Although phytonutrients have not been shown to be essential for human health in the same way that vitamins and minerals are, they may be important for long-term human health.

Polyphenols are a large class of chemicals that includes many well-described phytonutrients. Because the beneficial roles of these compounds have been recognized only recently, confirmation of their properties is ongoing. Nevertheless, there is compelling evidence for the health benefits conferred by several polyphenols. Among these are several anthocyanins (i.e., red, blue, and purple pigmented compounds found in many fruits), flavonoids (e.g., quercetin in many fruits and vegetables and epigallocatechin gallate in green tea), isoflavones (e.g., genistein in soybeans), and curcumin in the spice turmeric. Several of these compounds have antioxidant or anti-inflammatory properties, but their beneficial effects extend beyond these properties (for example, see Martin et al., 2011).

Carotenoids, including β-carotene (provitamin A) are a family of lipid-soluble phytonutrients, many of which are yellow, orange, or red in color. In plants, carotenoids are photosynthetic pigments that contribute to light harvesting and photoprotection. In humans, carotenoids have photoprotecting and antioxidant functions. Zeaxanthin and lutein accumulate to high levels in the macula of the human retina (to such an extent that it appears orange in color) and are thought to protect the retina from phototoxic damage and may help prevent the onset of age-related macular degeneration. Lycopene (a red pigment found in tomato fruit) has been implicated as an anticarcinogen.

Organosulfur compounds, such as diallylsulfides, are found in onions and garlic and other members of the Allium genus and are widely reported to have beneficial health effects through antioxidant, antibacterial, anti-inflammatory, and anticarcinogenic properties. Isothiocyanates are sulfur-containing metabolites of glucosinolates, which are produced in cruciferous plants, including broccoli, cauliflower, and mustard. There are over 150 known glucosinolates that accumulate differently in different species and that have different bioactive properties. There is evidence that some of the derived isothiocyanates are involved in the detoxification of carcinogenic compounds as well as having effects on inflammation and cell proliferation.

Foods whose health benefits go beyond traditional nutrients and vitamins are sometimes called functional foods. Although there is no universally accepted definition of functional foods, a labeling scheme in Japan that has been in place since the 1980s recognizes more than 700 Foods for Specified Health Use; these include foods that benefit the gastrointestinal tract or blood Glc levels (e.g., by containing phytochemicals or prebiotic carbohydrates that support the growth of beneficial intestinal microbes (see Yamada et al., 2008 for more on the Foods for Specified Health Use labeling program). The European Union is currently attempting to review, and then to regulate, the vast numbers of functional health claims being made in food marketing, based on a systematic review of the totality of the evidence for each. The publicly available European Union register of health claims that have been approved or rejected includes the approval of claims that oat β-glucan (a soluble fiber) and plant sterols help to reduce blood cholesterol and the rejection of many other claims, usually because “a cause and effect relationship between the consumption of the food for which the claim is made and the claimed effect has not been established” (http://ec.europa.eu/nutri/claims/). It is likely that as our understanding of phytonutrients and functional foods grows, our ability to identify and define those with legitimate health benefits versus those whose benefits are negligible will grow as well.

**Nutrient Assimilation and the Intestinal Microbiota**

The movement of nutrients from food into the bloodstream and cells of the body of the consumer is a complex and variable process. How food is stored and processed affects its nutritional content and nutrient availability. Finely ground wheat is a very different food than coarsely ground wheat, uncooked carrots are different from cooked carrots, and fresh-squeezed orange juice...
is different from reconstituted orange juice. Food digestion begins in the mouth, and people who chew their food thoroughly will take in nutrients differently from those that swallow it in chunks. The combination of foods eaten together affects the assimilation of each; for example, a recent study shows that eating foods high in calcium alongside red meat can mitigate some of the harmful effects of the red meat but also interfere with the absorption of non-heme iron. The presence or absence of fiber also affects how nutrients are assimilated across the intestinal epithelium; for example, the cholester醇 in a buttered baguette will be absorbed differently from that in a buttered fiber-rich oatcake.

One of the hottest topics in nutrition research is the role of intestinal microbes, also known as the gut microbiota, in food assimilation and human health. There are literally trillions of bacteria (comprising hundreds of species) living in your intestine; the number of microbes in your gut outnumbers the number of your cells in your body by 10-fold. Rodent studies initially revealed how dramatically these bacteria affect nutrient assimilation. A mouse reared in a germ-free environment that has no intestinal bacteria is leaner and eats more than a conventional mouse because intestinal bacteria are necessary to extract maximal energy from food. (The germ-free mouse excretes more calories in its feces showing that matter is indeed conserved.) A fecal transplant from the conventional mouse to the germ-free mouse restores its ability to extract nutrients maximally. Another study showed that the population structure of the gut microbiota from genetically obese mice differs from that in a buttered fiber-rich oatcake.

The importance of the gut microbiota on human health has been known for some time, but new advances in large-scale DNA sequencing mean that it is now possible to identify the entire population of bacteria and bacterial genes (the microbiome) in the gut. This method is particularly informative when surveys are compared across time, individuals, and regions. From such comparisons, there is considerable evidence that certain types of bacteria and certain genes are correlated with an increased incidence of obesity. Furthermore, the variable effects of some phytoneutrinents on different people have been correlated with differences in their gut microbiome. For example, only some individuals assimilate the beneficial phytoneutrinent equol from soybeans. The reason for this is that soybeans produce daizein, not equol. Certain types of intestinal bacteria can convert daizein to equol, and these bacteria are more prevalent in people who regularly eat soybeans. Another study showed that the ability to break down polysaccharides from nori, the kelp used to wrap sushi, is determined by microbes that are abundant in Japanese microbiota, but rare in others. Perhaps you are what your gut microbiota eat?

Although modifying diet composition with probiotics (i.e., food enriched with specific types of bacteria) can lead to modest changes in gut microbial populations, there is as yet very little evidence that this is either helpful or hazardous to health, and questions remain about potential very-long-term influences on gut health and human health.

HOW NUTRITION RESEARCH IS CONDUCTED

The modern, scientific approach to understanding nutrition and health has its origins in the early studies of the effects of vitamin deficiencies. Each discovery is fascinating, and it has resulted in Nobel Prizes in 1929, 1937, and 1943.

The first controlled dietary experiment that was correctly interpreted and acted upon appropriately is described in the Old Testament book of Daniel (see Daniel 1, 12-15). It concluded that young men fared better on diets rich in vegetables than those lacking them.

Later, in the mid-18th century, James Lind tried to find a solution to scurvy through a controlled study. Advances in navigation from the 15th century paved the way to long sea voyages and intercontinental travel. These voyages were frequently lethal to the sailors, whose diet was severely restricted for months on end. Following up on reports that acidic solutions prevented scurvy’s “putrid gums, the spots and lassitude, with weakness of the knees,” Lind augmented the diet of six pairs of sailors with either cider, vinegar, seawater, elixir of vitriole (dilute sulphuric acid), or two oranges and a lemon. Those receiving fruit recovered quickly, and they even helped to nurse their inflicted colleagues. Nevertheless, more than 50 years and countless deaths ensued before fresh citrus fruit became regularly incorporated into the sailors’ diet.

Nutritional scientists use several types of studies to examine the effects of diet on health (for more, see Traka and Mithen, 2011), including studies involving humans. Epidemiological studies look at the distributions of diet and disease across populations to identify correlations; these kinds of studies helped to demonstrate a connection between saturated fat and cardiovascular disease (CVD). Retrospective case-control studies ask individuals with a disease to record their past history, which can be compared with histories of individuals without the disease. These kinds of studies helped to identify the correlation between smoking and lung cancer; individuals with lung cancer were far more likely to have been smokers than individuals without it. Cross-sectional studies of these kinds are very prone to produce erroneous or erroneously strong apparent associations between food exposures and diseases. More reliable evidence comes from longitudinal studies, where baseline characteristics are recorded, rather than remembered or gathered from old records. Longitudinal cohort studies collect data about dietary and other habits from large numbers of individuals for many years. At some point, some of these individuals develop diseases, at which time their dietary histories can be compared with those of their cohort. The advantage of a cohort study versus a retrospective study is that people are more likely to record their dietary habits more accurately on an ongoing basis rather than by trying to remember their past behaviors.

The most rigorous method by which to establish causality is a randomized, controlled experimental study, in which individuals are randomly assigned to groups that are provided with specific dietary advice (or diets plus supplements) or to a control group. Both groups are otherwise treated exactly the same and health outcomes, or proxy measures, are monitored over time. Experimental studies are conducted either in very controlled metabolic ward settings, to assess physiological and
biochemical mechanistic outcomes, or in free-living subjects, whose actual behaviors may not match the advice given, so effect sizes are often very small.

Laboratory studies supplement human studies to explore mechanisms after an effect has been seen or to generate new questions for human research. Cell cultures are used to assess how foods or isolated compounds affect cell viability, whether they are capable of suppressing the unrestrained proliferation of cancer cells, or how compounds are metabolized within cells. Cell cultures derived from the intestinal lining are used to determine whether a compound is assimilated into these cells, which gives an indication of whether it is likely to be assimilated into the body. Animal models can reveal the uptake and metabolism of compounds in vivo and can also be models by which to examine the effects of diet on long-term health. Of course, mice are not men, and there are some very real differences between the rodent digestive system and our own, so these models are necessarily limited. Human clinical trials are the most convincing but also the most complex, expensive, and difficult to conduct.

Ultimately, if research points to net health benefit from particular nutrient or nutrient balance and dietary advice is shown to be effective in the complexity of the real world, the final step needed for translational research is to embed the desired dietary constituents into a sustainable food supply for the population, without the need for eternal health promotion expenditure, in other words, to change habits. This aspirational goal has not been achieved; in fact, modern dietary habits are leading people in the wrong direction and contributing to serious long-term health problems.

**HOW DIET AFFECTS HUMAN HEALTH**

**Dietary Deficiencies Lead to Disability and Death**

Throughout almost the entirety of our evolutionary history, our ancestors regularly faced the threat of malnourishment and starvation. Only in the past few years has this situation turned around, and since 2005, more people in the world are suffering from overweight rather than underweight conditions. Nevertheless, more than a billion people are chronically hungry, more than two billion are chronically malnourished, and the rapid pace of population growth particularly in the most impoverished regions means that these numbers will continue to grow. War and corruption contribute to these problems, and children suffer disproportionately.

Malnourishment stems from insufficient food but also from an overreliance on a limited number of food sources and insufficient dietary diversity. One of the most pervasive types of malnourishment is vitamin A deficiency, which affects more than 100 million children and is a leading cause of infections and of blindness. Staple crops in the most affected regions include rice and cassava, both of which contribute little vitamin A to the diet. Efforts to ameliorate it include vitamin A supplements, genetic improvements in staple foods, and increased affordability and distribution of foods rich in the provitamin-A carotenoids (e.g., β-carotene).

Other dietary deficiencies occur, often but not always in association with poverty. Although sometimes assumed to be a problem of developing nations, most Americans do not get the full daily recommended amounts of zinc, calcium, and vitamin A, and a quarter of U.S. households with children reported experiencing food hardship in 2012. In the developed world, food fortification programs initiated in the 20th century have helped to reduce the incidence of vitamin and mineral deficiencies. Efforts to extend these programs are ongoing through efforts to directly enrich plants with vitamins and minerals, a process called biofortification. These range from breeding varieties with improved mineral uptake capacities or increased rates of vitamin biosynthesis, altering mineral bioavailability through altering the abundance of phytate or oxylate, which can bind minerals and prevent their absorption, and agronomic practices to enrich the soils in which plants are grown.

**Diet Is a Factor in Chronic Diseases**

Chronic diseases, including type 2 diabetes, CVD, and cancer, have supplanted communicable diseases as leading causes of death; chronic diseases account for over 60% of all deaths, 80% of which occur in low- and middle-income countries. Besides causing premature death, chronic diseases take away sufferer’s health and energy and often lead to poverty through increased expenditures and loss of wage-earning capabilities. Diet-related chronic diseases are increasing in incidence and are to some extent preventable. It has been estimated that removing risk factors, such as poor diet and physical inactivity, could prevent around three-quarters of cases of CVDs and type 2 diabetes and 40% of cancers. Obesity, which is a consequence of unhealthy eating and physical inactivity, directly contributes to the development of chronic diseases, particularly type 2 diabetes and CVD. One of the strongest indicators of the development of chronic diseases is an inadequate consumption of fruits and vegetables.

**Metabolic Syndrome**

Metabolic syndrome defines a set of related health conditions that predispose people to further health problems, including type 2 diabetes and CVD. Although not a disease per se, it is a syndrome strongly correlated with the onset of future health problems and a useful way to describe a constellation of related causal, metabolic risk factors, which are all reversible by a single measure—weight loss. The components that define metabolic syndrome include a high waist circumference, high fasting Glc, high serum triglyceride, high blood pressure, and low high-density lipoprotein (HDL) cholesterol. The cutoffs are set at values well below those that individually would constitute an independent diagnosis or would be treated; it is the combination that gives the elevated risk. Most, but not all, of these metabolic risk factors are linked to insulin resistance.

About 30% of all Americans or Europeans develop metabolic syndrome by age 60, and a higher proportion of Asians living in obesogenic environments (such as those associated with sedentary lifestyles and high-calorie or Western diets). Many of those affected have larger waists than might be expected from other dimensions because there is a deposition of fat inside the abdomen, specifically in the liver and pancreas. Unlike
subcutaneous fat, abdominal fat is a significant source of inflammatory cytokines and affects the release and uptake of Glc by the liver and muscles, contributing to chronic health problems. The tendency toward abdominal fat deposition has a large genetic component, which is reflected in the greater susceptibilities of some racial and ethnic groups. Thus, there are two processes going on: total fat accumulation, which is mainly environmental and a little bit genetic, and the distribution of fat deposition, which is mainly genetic with a smaller environmental component.

Metabolic syndrome and the increased risks of chronic diseases associated with it can be avoided by maintaining a healthy body weight (as defined by a low waist circumference), physical activity (e.g., over 10,000 steps daily or 30 min of moderate to vigorous activity five times a week), and by not smoking.

**Type 2 Diabetes**

Diabetes is two main diseases, with very different causes and features. Both have extensive vascular consequences that affect eye, kidney, nerve, heart, and brain functions over time. Type 1 diabetes (~10% of cases) is an autoimmune disease with a large genetic contribution, in which the body destroys the pancreatic insulin-producing cells, causing a loss of insulin production. Type 1 diabetes requires insulin replacement as treatment and is the most frequent type diagnosed in childhood, although it can occur at any age. The incidence of type 1 diabetes is increasing, but only slightly.

By contrast, the incidence of type 2 diabetes (90% of cases) is increasing very rapidly. Type 2 diabetes is largely the result, in genetically susceptible individuals, of body fat accumulation, physical inactivity, and the associated development of insulin resistance. High intakes of saturated fats also contribute. Obesity is the single most reliable predictor of type 2 diabetes. Although the risk goes up with more severe obesity, there is a demonstrated increased risk even in people who are slightly overweight (i.e., those with a body mass index above 23 kg/m² or a waist circumference above 94 cm in men or 80 cm in women); waist circumference is a better indicator because it is more directly correlated with health risks. Essentially, type 2 diabetes is a preventable chronic disease. It is also reversible in most cases, with both prevention and reversal largely feasible through a change in dietary habits and physical activity. Although type 2 diabetes is usually diagnosed in adulthood, it has begun to be seen around puberty in very obese children.

Those with diabetes (type 1 or type 2) must carefully manage their intake of sugars to avoid the damaging effects of high blood Glc levels, but to what extent do dietary sugars contribute to the onset of type 2 diabetes? Systematic reviews have concluded that sugar itself has little or no effect on the incidence of diabetes, heart disease, cancers, or any other disease. There are potential mechanisms that could point to such an effect (e.g., the role of the Fru half of the Suc molecule in elevating serum triglycerides), but no unambiguous causal effect has been seen. Rather, diets high in sugar are correlated with other factors that themselves may be of greater importance (e.g., being overweight and sedentary). As an example, there is consistent evidence that higher consumption of sugary drinks is associated with greater weight gain in children and young people and with the obesity epidemic. It is easy to conclude that this must be causal, but frequency of sugary drink consumption is also a marker for sedentariness and for other diet and lifestyle patterns, which may be the real cause of obesity. High consumption of low-calorie carbonated drinks (with artificial sweeteners) is also associated with being overweight. Furthermore, interventions to reduce sugary drinks have shown small effects on the weight gain patterns of children (although it is hard to be sure that they did not change other food choices). The rising incidence of childhood obesity and its accompanying increased risk for the development of type 2 diabetes is a significant public health concern.

**CVDs**

CVDs, including stroke, heart attack, atherosclerosis, and high blood pressure, are the leading causes of death globally and account for 30% of all deaths. There are many risk factors for heart disease, some of which are considered part of the causal pathway by leading to arterial plaque formation or to thrombosis (blood clots within blood vessels). Many risk factors, like age or male sex, are not reversible. Obesity, inactivity, and metabolic syndrome are reversible contributors to CVD.

Blood lipid levels and forms are important indicators for the risk of CVD and metabolic syndrome, which is contributed to by high levels of serum triglycerides and low levels of HDL cholesterol. High serum triglycerides are correlated with high intakes of dietary fats and sugars and physical inactivity. Dietary fats are assimilated primarily as fatty acids and then reformed into triglycerides. Triglycerides are packaged along with cholesterol and proteins into lipoprotein particles. There are several different kinds of lipoproteins, but the two major forms are LDLs and HDLs. LDLs are particularly damaging, but HDLs are actually beneficial because in these particles lipids are carried to the liver for removal from the body. Therefore, the total serum lipid levels and the relative levels of LDLs versus HDLs are all important indicators of health.

LDLs are prone to oxidation, and when oxidized adhere to arterial walls and initiate inflammation and the formation of a buildup called atheroma plaque. Plaque narrows arteries and makes them less flexible, contributing to high blood pressure, but plaque can also break off to form clots that cause strokes and heart attacks. High levels of dietary fats, particularly trans fats and saturated fats from animal sources, contribute to LDL accumulation, oxidation, adhesion, arterial inflammation, and plaque formation.

Twenty-five years ago, epidemiological studies revealed the interesting observation that although the French ate a diet high in saturated fats, their incidence of CVD was unexpectedly low; this effect was named the French Paradox. A number of potential explanations have been put forward, with no definitive answer. Leading possibilities are that the French also have higher intakes of monounsaturated fatty acids, such as oleic acid from olive oil, that they drink modest regular amounts of wine, which elevates HDL cholesterol, and that they consume many more fruits and vegetables.

There is a good correlation that, despite similar dietary saturated fat consumption, regions where more fruits and vegetables are consumed show lower rates of heart disease. Vegetarians similarly
have less heart disease than meat consumers. Several mechanisms appear to contribute: greater intake of potassium, less sodium, more dietary fiber, more antioxidants, and more folate. Largely based on epidemiological associations, a number of plant products, including polyphenols and n-3 fatty acids, and antioxidants, including vitamins C and E, may contribute to preventing plaque buildup. Some of these compounds have in vitro properties that suggest plausible mechanisms for CVD protection, and there are some encouraging animal studies, for example, with anthocyanins. As yet, experimental evidence in humans is lacking, and in vitro studies are confounded by uncertainties about how these compounds are metabolized in the gut and to what extent they are absorbed from the gut. Although the mechanisms remain uncertain, the evidence that plant-rich diets are desirable is compelling.

Cancer

Cancer is a leading cause of death and was responsible for nearly eight million deaths in 2008. Cancer is caused by an accumulation of genetic changes in cells that leads to a loss of their ability to control cell division and overproliferation. Cancer cells become resistant to inhibition by neighboring cells and metastasize by breaking away from their site of origin to initiate tumors at new sites. Because cancers typically develop asymptptomatically over many years, it is more difficult to examine the role of diet in cancer incidence and progression than in other chronic diseases. Nevertheless, evidence is accumulating that some phytonutrients may interfere with various stages of the progression of the disease. These include polyphenols and carotenoids from diverse plants. There is also evidence from animal and other studies for a protective function for isothiocyanates, which are derived from glucosinolates that are present in plants of the Brassicaceae family (e.g., broccoli, cabbage, cauliflower, and their relatives) and which may detoxify carcinogenic compounds, suppress inflammation, and suppress cellular overproliferation.

Colorectal cancer is unique among the chronic diseases in that dietary compounds do not necessarily need to be digested or absorbed to be beneficial. Volatile fatty acids produced by bacterial fermentation within the gut lumen may have differential effects on tumor development. Several nonabsorbable phytonutrients could have the potential to detoxify carcinogenic agents within the lumen of the intestine or prevent their assimilation. As an example of the latter, heme compounds from red meat (or nitrosyl-heme compounds from processed meats) are carcinogenic, and burned (e.g., barbecued) meat is associated with colorectal cancer. There is evidence to indicate that the presence of calcium or chlorophyll in the intestine at the same time as heme can prevent its assimilation. Most traditional diets contained very little meat, and the increase in colorectal tumors at new sites. Because cancers typically develop asymptptomatically over many years, it is more difficult to examine the role of diet in cancer incidence and progression than in other chronic diseases. Nevertheless, evidence is accumulating that some phytonutrients may interfere with various stages of the progression of the disease. These include polyphenols and carotenoids from diverse plants. There is also evidence from animal and other studies for a protective function for isothiocyanates, which are derived from glucosinolates that are present in plants of the Brassicaceae family (e.g., broccoli, cabbage, cauliflower, and their relatives) and which may detoxify carcinogenic compounds, suppress inflammation, and suppress cellular overproliferation.

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More than 30 years ago, epidemiological studies indicated that a high-fiber diet may protect against colorectal cancer. One model was that the bulking effect of insoluble fiber diluted potential carcinogens and accelerated their movement through the intestine. More recent studies have indicated that the benefits of a high-fiber diet are not solely attributable to the fiber, but also to the many micronutrients and phytochemicals ingested along with it. Thus, although our understanding of their protective action has changed, the recommendation to eat fiber-rich foods, including whole grains, fruits, and vegetables, remains.

Obesity is another major cause of colorectal cancer, as it is for breast, uterus, and prostate cancers. It is likely that there are multiple mechanisms contributing to these effects. Some well-supported hypotheses include the increased production of hormones (e.g., estrogen or leptin) from fat cells or the increased levels of insulin and low-level inflammation that accompany obesity.

MEDIA AND MARKETING MUDDY IMPRESSIONS OF HEALTHY EATING

Many consumers get their nutritional information from newspapers, magazines, and the Internet, and three-quarters of those surveyed indicated that their healthy eating goals were confounded by “too much conflicting information about which foods are healthy and which are not.” Popular news outlets can present scientific findings accurately and rationally, but many do not. In their desire to sell papers (or clicks), the media often employs hyperbole in its headlines, only sometimes reporting the conditionals and caveats that accompanied the original report. Several organizations (for example, see Harvard School for Public Health and Sense about Science) can help consumers sort through the conflicting information by guiding them to look for evidence of peer review and to ask questions about how large and what type of study is being reported.

Because selling the promise of health and weight management is a multi-billion dollar business, the Internet is replete with self-professed health experts selling empty promises and quick health fixes. The types of health claims that these products can make are regulated, but even restricted claims can sound convincing to someone seeking a packaged health solution. Sensible health information is widely, freely available from health-oriented charities and public health and nutrition organizations. The advice from these organizations is surprisingly simple and includes recommendations to eat whole grains and plenty of varied vegetables. Or as summarized eloquently by Michael Pollan, “Eat food, not too much, mostly plants.”

One simple measure that would help consumers toward nutritionally balanced meals would be to start to standardize what is sold as a meal. The introduction of nutritional labeling on packaged foods is a good start, but no such guidelines are readily supplied for prepared or takeout meals. Furthermore, the number of takeout meals and the size of the portions of those meals have been increasing with time. It would be a simple matter to establish a sensible range for the nutrients that should be provided in a meal, say a third of daily requirements, plus or...
minus 10%, and to identify very clearly meals that do this and those that are unbalanced. In some places, school meals follow such guidelines; it could easily be put into place for all meals. Furthermore, guidelines that address marketing of low-nutrient foods, particularly to children, can be enforced or established to help parents as they strive to promote healthier eating habits in their children.

WHAT CAN SCIENTISTS DO TO PROMOTE HEALTH
AND NUTRITION?

The long-term consequences of nutritional ignorance and overnourishment are as much a threat to human productivity as are the consequences of undernourishment, and both are unnecessary. One of our roles as scientists is to add our voices in support of nutritional education. Chronic diseases are easier to prevent than to reverse, yet consumers continue to blame poor eating habits in part on confusion about what foods are healthy or not. Furthermore, promising biofortification efforts are hindered by public uncertainty of genetic modification (GM) methods. Educated consumers will be able to dismiss a great deal of the unsubstantiated hype about food and health that uneducated consumers futilely follow.

Scientists can also contribute to discussions about the role of government in regulating how food is distributed and marketed. In some countries, the number of people smoking has decreased as a consequence of increased taxes levied upon tobacco products and smoking restrictions. Do the rising incidences of chronic diseases that result from poor food choices warrant intervention? For example, the clear connection between trans fats and chronic health problems has led to their being restricted in some countries, so should others follow suit? The introduction of food fortification programs in the early part of the 20th century is an example of a government-led program that has had far-reaching health benefits; are there similar opportunities to pursue now that we have a better idea of what constitutes a healthy diet? School lunch guidelines may improve children’s nutrition in the short term, but should cooking classes be reintroduced to the curriculum to support their long-term nutritional needs? Should governments regulate or subsidize food prices to help to reduce the economic and geographical barriers that interfere with the consumption of healthy foods? These are complex questions that need input from all members of society and in which scientists should certainly engage.

Plant scientists also have a more direct role in improving human health through their research efforts. Fruits and vegetables are expensive compared with some other foods, for many reasons including their relatively lower yields and shorter shelf lives compared with grains. Research into improving yields, reducing losses to stresses and pests, and improving shelf life can help make these foods more affordable, widely grown, and accessible. Advances in plant breeding tools and better understanding of phytochemistry and plant metabolomics will help to identify candidate genes and germplasms for breeding strategies. Biofortification of grains and other staples is another promising approach. For recent and comprehensive surveys of biofortification efforts, see Hirshi (2009) and Newell-McGloughlin (2008); we will highlight only a few efforts here. Golden rice may be the most familiar biofortified food. Through expression of β-carotene biosynthesis genes in the rice endosperm, Golden rice has been modified into a significant source of provitamin A. Because Golden rice is a genetically modified food, it has been subject to an arsenal of regulatory hurdles, but it may soon begin to be distributed. Provitamin A levels in other staple foods, including maize (Zea mays) and cassava (Manihot esculenta), have been or are being increased through GM and conventional breeding methods. The strategy of increasing vitamin content by elevating the biosynthetic pathway has been used to enhance levels of folic acid, vitamin C, and vitamin E; superbiofortified foods can be produced that are enhanced for multiple nutrients. Similarly, foods enriched for phytonutrients can be produced by metabolic engineering approaches, both by introducing biosynthetic genes or, as has been successful for flavonoid production, by expression of transcription factors that coordinately regulate a biosynthetic pathway. Metabolic engineering has also been employed to increase levels of fiber and to alter fatty acid compositions of foods. These foods also can be useful for controlled human or animal feeding studies, in which the biofortified food can be compared with its near-isogenic progenitor, eliminating many of the commonly confounding variables.

Nutritional content can also be enhanced through non-GM methods. For example, the Beneforte broccoli variety that has increased levels of glucoraphanin was developed by crossing conventionally grown varieties with a high glucoraphanin–containing wild relative, followed by several years of marker-assisted selection. High provitamin A cassava and sweet potatoes are being developed through conventional breeding methods, and genetic approaches are being used to identify high-iron beans and zinc-enriched wheat and rice varieties. Mineral nutritional quality can be further enhanced by increasing mineral content of the soil, by intercropping with other species, by increasing the plant’s ability to take up or sequester mineral nutrients, or efforts to increase their bioavailability. For example, mineral content may be enhanced by the expression of high affinity transporters in the roots or mineral binding proteins in the seed. Phytate, which chelates mineral nutrients and interferes with their assimilation, can be eliminated by expression of the enzyme phytase.

CONCLUSIONS AND FUTURE DIRECTIONS

The agricultural revolution, and the industrialization of food production in the 19th and 20th centuries, radically changed the way we eat. Food supplies have become more abundant (in some regions) and more centralized, and food processing practices have led to more stable foods, sometimes at the expense of nutritional quality. Supermarket and fast food selling practices alongside the industrial scale of food production have led to the bizarre situation in which processed foods are often cheaper than unprocessed foods. We are beginning to realize the health consequences, particularly the epidemics of chronic diseases, brought about by all of this convenience. The adoption of some of these eating habits by the developing world has made these truly global health problems.

Science has important roles in responding to the chronic disease crisis. One of the main roles is to improve our understanding
of the links between food and health and to ensure that consumers and governments get and respond to sound advice. Some of the ongoing questions include understanding the role of human genetic diversity in nutrient assimilation (also known as nutrigenomics) and the role of the gut microbiome in nutrition. Plant scientists are developing nutrient-enriched foods and working with nutritionists to identify phytochemicals with beneficial roles in human health. An important challenge is to understand how food components interact with each other in the gut to affect bioavailability and uptake as well as activity within the body.

Finally, scientists from all disciplines need to work cooperatively toward defining and then achieving changes in national and global diets that will significantly improve diet quality and health. There is evidence that dietary advice through health promotion has, at best, a limited impact on food choices and eating behavior, which is largely restricted to the most educated and most affluent groups. Health inequalities continue to widen. Scientists need to educate politicians and the consuming public so that policies and funding support sound public health strategies that are in line with evidence-based science, and one of the strongest correlations we have indicates that a diet rich and varied in plant matter is optimal. After many years of efforts to ensure that policies and funding support sound public health strategies and most affluent groups. Health inequalities continue to widen.

RECOMMENDED READING


Centers for Disease Control and Prevention (2011). National estimates and general information on diabetes and prediabetes in the United


