Remove Watermark Now

Michel Foucault. 1994. *The Birth of the Clinic*. New York: Vintage.

Margaret Lock. 2002. *Twice Dead: Organ Transplants and the Reinvention of Death*. Berkeley: University of California Press.

Daniel Moerman. 2002. *Meaning, Medicine, and the "Placebo Effect."* Cambridge, UK: Cambridge University Press.

Lynn Payer. 1996. *Medicine and Culture*. New York: Holt.

Lesley Sharp. 2006. *Strange Harvest: Organ Transplants, Denatured Bodies, and the Transformed Self*. Berkeley: University of California Press.

Loudell F. Snow. 1993. *Walkin over Medicine*. Boulder, CO: Westview Press.



Chapter 4

Diet and Nutrition in Health and Disease

CHAPTER GOALS

- To understand the basics of human nutrition
- To understand how an evolutionary perspective on diet and nutrition helps clarify some contemporary diet-related diseases
- To see how diet changed with the transition from hunting-gathering to agriculture and the effects on human health
- To demonstrate how evolutionary and developmental dietary conditions are related to the risk of chronic diseases such as diabetes
- To understand the role of biological variation in response to milk and wheat and its relationship with current dietary policies and patterns

The Paleo diet. Gluten free. Vegan. Low fat. Low carb. MyPlate. These are all contemporary types of dietary advice. They are also all very different, but each purports to improve your health. Amid this confusing and contradictory array of dietary guidance, what should you eat? This is a decision individuals make every day. The effect of diet on the body is the topic that interests scientists in nutrition and also public health because a population's dietary patterns contribute to their disease patterns, whether they are rates of cardiovascular disease or child stunting from protein-energy malnutrition. What constitutes the "ideal" or "healthiest" diet for humans is the subject of a great deal of ongoing research. Many countries develop their own dietary guidelines, which are supposed to be authoritative statements about the best diet to enhance health, given food availability and local culinary traditions, but these guidelines are also controversial because they may be shaped by the interests of powerful food industries (Nestle 2013). Understanding the links between diet and health is an especially pertinent issue given both the variability in human diets across the world and the globalization of food, which is generating greater homogeneity in dietary practices.

Diet plays a major role in health and disease. Specific nutrient deficiencies or surpluses can result in discrete syndromes, whereas more generalized shortages or

Remove Watermark Now

excess can result in dysfunction across many physiological systems. All humans require roughly the same nutrients, although the absolute amount varies depending on age, size, activity patterns, and other individual factors. However, the range of possible diets that provides these nutrients is wide and there is tremendous variation in the types of diets humans consume, although traditional diets are eroding as "Western" diets have become increasingly global. Consider, for example, traditional Inuit (Arctic peoples) diets, which were composed mainly of fatty meat because plant foods were available in the Arctic only during the brief summer. Contrast those with the heavily plant-based diets of populations living in tropical forests, where large game animals are scarce and plant life is highly diversified. Then there are typical American diets, in which refined grains are staple foods, as are beef, chicken, and dairy products.

In this chapter we present a brief overview of basic nutrition and its relationship to human diets and biology. We then explore the links between nutrition and health and examine how anthropology provides a unique perspective on these relationships. Human biological characteristics evolved in the context of specific dietary environments, yet most contemporary humans consume foods different from those of our ancestors in the distant past. This dietary "mismatch" is likely to result in health problems of dietary origin because our bodies evolved to process a diet that little resembles those that are common today (Cordain et al. 2005; Eaton, Eaton, and Konner 1999). Many of the foods that are relatively cheap and ubiquitous in markets around the world are dense in calories, fat, or salt; such foods would have been rare in human diets of the past. It is thus crucial to understand when, how, and why these particular foods came to predominate in global diets because their relationship to disease is becoming clear. As human populations experienced different types of diets in their unique pasts, genetic adaptations emerged in some human groups that were specific to their dietary environments. These emerged as populations transitioned from foraging to food production around 10,000 years ago and adopted different types of new staple foods. In this new dietary and subsistence context, new modes of food processing also enabled human populations to extract nutrients from what became a much less diverse diet.

Fundamentals of Nutrition

Nutrition is a relatively young science, with its origins in the 1910s and 1920s. Much remains to be learned about how specific components of the diet affect human health and well-being, but most of the basic knowledge about human nutritional requirements was worked out by the mid-twentieth century. Importantly, nutrient deficiencies as a cause of disease were initially not considered likely because medicine was dominated by enthusiasm over Koch and Pasteur's germ theory of disease at the turn of the century (see Chapter 8; Carter 1977). For example, beriberi was known as a widespread disease in Southeast Asia in the late nineteenth century, but efforts to demonstrate its infectious cause were fruitless. Christiaan Eijkman and Frederick Gowland Hodgkins were able to show that it was caused by a deficiency of thiamine (now also known as vitamin B_1), a discovery consistent with growing evidence that other syndromes such as scurvy or pellagra were also caused by vitamin deficiencies (vitamin C and niacin [vitamin B_3]), respectively).

It is interesting to note that training in nutrition remains somewhat marginal to medical education; until the mid- to late-twentieth century nutrition was often folded into departments of home economics rather than in medical or public health schools. A recent survey of the medical schools in the United States (Adams, Butsch, and Kohlmeier 2015) found that although the National Research Council recommends 25–30 hours of nutrition education, the average number of hours reported in the survey was 19. More than one-third of schools required less than 12 hours and 9 percent required none. Furthermore, less than 20 percent provided a dedicated nutrition course, with most integrating nutrition into other preclinical classes and few providing nutrition training in the context of clinical practice. What is even more discouraging is that there appears to be a downward trend in requirements for hours of nutrition education in medical schools and fewer schools providing a dedicated nutrition course (Adams, Butsch, and Kohlmeier 2015).

Organisms eat primarily to get energy to fuel their metabolic and activity requirements. A certain number of calories is required each day just to stay alive, a number that varies by age, sex, and body size and composition.[1] For humans, up to 20 percent of that goes to fuel our large brain! Energy is derived from two main sources: carbohydrates in plant foods (which provide glucose) and fat in animal and some plant foods (especially nuts and seeds). Although energy comprises the largest component of the diet, other nutrients are needed in smaller quantities. Protein is required as a source of amino acids. Of the 20 known amino acids, 9 are considered essential for humans, and the remaining 11 can be made from the essential amino acids. These are used to make the body's own proteins, which serve a variety of purposes, from the structure of muscle and bone to carrier proteins that transport oxygen to tissues or molecules across the membranes of cells. Amino acids also serve as the sole source of metabolizable nitrogen, which is necessary for the synthesis of the genetic material deoxyribonucleic acid (DNA) and related molecules. Although animals can synthesize fat from excess calories, one fatty acid (linoleic acid), which is found in seeds and dark-green leafy vegetables, is considered essential because humans cannot make it themselves. This "essential" fat is used for making other fat-based biochemicals, such as steroid hormones.

Carbohydrates, protein, and fat are collectively referred to as "macronutrients" because they are needed in relatively large quantities, supply energy (calories), and are used to make fundamental body structures. In addition, there are many "micronutrients" required for various functions. The vitamins and minerals and estimates of daily required amounts are listed in Table 4.1. These substances all play some vital role (hence the term "vitamin") in diverse physiological functions. The vitamins are divided into those that are water soluble (the B vitamins and vitamin C) and those that are fat soluble and can be stored in the body (A, D, E, and K). Many of the B vitamins are involved in energy metabolism, a complex set of cycles requiring numerous enzymes and cofactors that include these vitamins. Vitamin B_{12} is of particular

[1] A calorie is actually more accurately referred to as a kilocalorie, which is equivalent to the amount of energy needed to raise one liter of water 1 degree centigrade (a calorie itself is the amount of energy needed to raise 1 gram of water 1 degree centigrade, but this "small" calorie designation is rarely used). The standard international unit for energy is the kilojoule. One kilocalorie is equivalent to 4.18 kilojoules.

Do not distribute

Remove Watermark Now

Table 4.1 Vitamins and Minerals in Human Nutrition

Element	Rich Source	DRI for Adults	Function in the Body
Calcium	Milk Cheese Some green vegetables	RDA 1.0 g daily, 1.3 g in pregnancy and lactation	Bone and tooth formation; muscle contraction, including heartbeat; activates enzymes
Sodium	Common salt Animal products	AI 1.5 g	Regulates extracellular electrolyte and water balance, muscle and nerve function
Potassium	Meats Cereals Vegetables Legumes Fruits	AI 4.7 g	Regulates intracellular electrolyte and water balance and cell metabolism
Iron	Liver Meat Legumes Whole grains Potatoes Egg yolk Green vegetables Dried fruits	RDA: Women, 18 mg Men, 8 mg	Constituent of hemoglobin, and myoglobin; oxygen transport
Iodine	Seafood Iodized salt	RDA: 150 mcg	Required for synthesis of thyroxine, a thyroid hormone
Zinc	Seafood Meat Liver Eggs Milk	RDA: Women, 8 mg Men, 11 mg	Constituent of insulin and metalloenzymes; wound healing; taste acuity

Fat-Soluble Vitamins	A	D	E	K
Important food sources	Liver Egg yolk Butter Green and yellow vegetables Apricots Cantaloupe	Butter Egg yolk Liver Salmon Sardines Tuna fish	Wheat germ Leafy vegetables Vegetable oils Egg yolk Legumes Peanuts	Cabbage Cauliflower Spinach Other leafy vegetables Soybean and other vegetable oils
Function	Epithelial cell function, in mucous membranes, skin, and bone; in visual pigments	Calcium and phosphorous absorption and utilization in bone growth	Protects cell structures	Blood clotting
Deficiency: signs and symptoms	Night blindness; rough, dry skin; dry mucous membranes	Rickets; soft bones; bowed legs; poor teeth; skeletal deformities	Increased hemolysis of red blood cells	Slow blood clotting
DRI	RDA Women, 700 mcg Men, 900 mcg	RDA 15 mcg	RDA 15 mg	AI Women, 90 mcg Men, 120 mcg

Water-Soluble Vitamins	Vitamin C Ascorbic Acid	Fractions of the Vitamin B Complex Thiamine	Riboflavin	Niacin
Important food source	Citrus fruits Strawberries Cantaloupe Tomatoes Peppers Cabbage Potatoes Kale Turnip greens	Pork Organ meats Whole grains Nuts Legumes Potatoes	Liver, milk Meat, eggs Green leafy vegetables	Liver, poultry Meat, fish Whole grains Legumes Mushrooms
Function: Essential in	Collagen formation, cellular oxidation, and reduction	Carbohydrate metabolism	Carbohydrate, fat, and protein metabolism	Carbohydrate, fat, and protein metabolism
Deficiency manifests as	Scurvy Sore mouth Bleeding gums Weak-walled capillaries	Beriberi Poor appetite Fatigue Constipation	Eye sensitivity	Pellagra Dermatitis Depression Diarrhea
RDI	RDA Women, 75 mg Men, 90 mg	RDA Women, 1.1 mg Men, 1.2 mg	RDA Women, 1.1 mg Men, 1.3 mg	RDA Women, 14 mg Men, 16 mg

	Vitamin B6	Folacin	Vitamin B12	Pantothenic Acid	Biotin
Important food sources	Pork Organ meats Legumes, seeds Grains Potatoes Bananas	Green leafy vegetables Liver and organ meats Milk Eggs	Organ meats Milk Eggs	Organ meats Eggs Peanuts Legumes Mushrooms Salmon Whole grains	Organ meats Peanuts Mushrooms

[continued]

Pearson Education

Remove Watermark Now

Table 4.1 *(continued)*

	Vitamin B$_{12}$	Folacin	Vitamin B$_6$	Pantothenic acid	Biotin
Function: Essential in	Metabolism of amino acids	Blood formation; synthesis of DNA, RNA; amino acid metabolism	Growth, blood formation, choline synthesis, amino acid metabolism	Carbohydrate, fat, and protein metabolism	Fatty acid synthesis carboxytation reactions
Deficiency manifest as	Convulsions Anemia	Megaloblastic anemia Glossitis Diarrhea	Macrocytic anemias and pernicious anemia	Deficiency very rare in humans	Lassitude Anorexia Depression Anemia
DRI	RDA 1.3 mg	RDA 400 mcg	RDA 2.4 mcg	AI 5 mg	AI 30 mcg

AI, adequate intake; DRI, dietary reference intake; RDA, recommended dietary allowance.
Source: http://www.iom.edu/Activities/Nutrition/SummaryDRIs/DRI-Tables.aspx.

interest because it is found only in animal foods and requires a special mechanism, the intrinsic factor, for absorption in the stomach. Vitamin C is an antioxidant but is also involved in the synthesis of collagen, the protein in muscle and connective tissue. Many of the water-soluble vitamins act as antioxidants, protecting cells from sustaining damage from oxygen-**free radicals**, which are common highly reactive molecules that are known to cause cellular damage. Among the fat-soluble vitamins, vitamin A is found exclusively in animal foods, but can be synthesized from plant-based β-carotenes. Vitamin E is broadly distributed in plants, and vitamin K, which is involved in blood clotting, can be synthesized by specialized bacteria in the colon.

Vitamin D is found primarily in fish oils and is involved in calcium absorption and deposition of calcium in bone. It can also be synthesized; specialized skin cells convert a vitamin D precursor into active vitamin D in the presence of ultraviolet light. At high latitudes it is not possible to synthesize sufficient vitamin D during the winter months because the sun's rays are not sufficiently powerful to induce the conversion, but because vitamin D is stored to a limited degree, this is not necessarily problematic. However, covering up most of the skin or having darker skin also reduces vitamin D synthesis. In some countries such as the United States, milk is fortified with vitamin D because of its connections to calcium utilization.

The mineral humans need in the greatest quantity is calcium. Calcium forms the inorganic matrix of bone and also regulates neuron activity, muscle contraction, and blood clotting. Sodium is required for maintaining fluid balance in the body and, in conjunction with potassium, for the conduction of nerve impulses. Among the trace minerals (those needed in minute quantities), iron is a component

of hemoglobin, the protein in red blood cells that carries oxygen throughout the body, and it also plays other roles in energy generation and the synthesis of collagen and various neurotransmitters. Zinc is an important cofactor for more than 300 enzymes and hence is involved in numerous functions throughout the body. Last, iodine is a key component of thyroid hormones, which regulate metabolic rate and promote growth and development.

What Is Hunger?

Everyone knows the feeling of being hungry, although it is hard to describe in words. Hunger pangs result from contractions in the stomach, possibly stimulated by the hormone ghrelin, which is associated with appetite for food. Prolonged hunger can stem from crop failures and famine, economic inability to procure food, or purposeful dieting, and can result in starvation, characterized by fatigue, weakness, or irritability, and ultimately the catabolism of the body's own energy and protein resources and death. Yet hunger is hard to quantify, and in 2006 in the United States, the problem of hunger was recast as varying levels of food security, with four categories:

Food Security:

- High food security (*old label = Food security*): no reported indications of food-access problems or limitations.
- Marginal food security (*old label = Food security*): one or two reported indications—typically of anxiety over food sufficiency or shortage of food in the house. Little or no indication of changes in diets or food intake.

Food Insecurity:

- Low food security (*old label = Food insecurity without hunger*): reports of reduced quality, variety, or desirability of diet. Little or no indication of reduced food intake.
- Very low food security (*old label = Food insecurity with hunger*): Reports of multiple indications of disrupted eating patterns and reduced food intake.

It did not escape notice that this new label removed the emotional impact of the word "hunger," replacing it with the more neutral term "food insecurity." The panel recommending this change distinguished hunger from food insecurity, noting that hunger is an individual-level physiological condition that may result from food insecurity and would require collection of more detailed and extensive information on physiological experiences of individual household members than could be reasonably accomplished in national surveys. Food insecurity is a household-level economic and social condition of limited or uncertain access to adequate food that can be assessed

(continued)

Remove Watermark Now

(continued)

through national household surveys. (http://www.ers.usda.gov/topics/food-nutrition-assistance/food-security-in-the-us.aspx).

In the United States, about 14 percent of all households currently fall into the "food insecurity" categories (48 million people), but 19 percent of all households with children and more than one-third of households below the poverty line are classified as "food insecure." The prevalence of food insecure households has increased 10 percent Since the turn of the twenty-first century, remaining more or less consistent for the past three years. For comparative purposes, about 8 percent of all households in Canada are food insecure, with much higher rates among households living in the northern and northwestern territories (http://www.statcan.gc.ca/pub/82-624-x/2015001/article/14138-eng.htm). At the global level, the Global Hunger Index is calculated for countries based on the number of individuals with insufficient access to calories, along with rates of stunting (low height), wasting (low weight for height) and mortality among children under five years (http://www.ifpri.org/topic/global-hunger-index). About 800 million people are thought to be undernourished, with about 90 million children under five years stunted and/or wasted. At present, countries in sub-Saharan Africa and South Asia have some of the highest rates of hunger. The International Food Policy Research Institute website contains interactive mapping tools to demonstrate variation in hunger across countries and time.

Ending hunger has been a part of international food and health agencies for the past few decades. One of the UN's Millennium Development Goals was to halve the number of people suffering from hunger by 2015, which was close to being reached (prevalence was 23 percent in 1990; it is now 13 percent). Among the UN Sustainable Development Goals for 2030, adopted in September 2015, is Sustainable Development Goal 2, which is bolder than the Millennium Development Goal: End hunger, achieve food security and improved nutrition, and promote sustainable agriculture. There is widespread agreement that hunger or food insecurity is not simply attributable to a lack of food production, but rather to problems in distribution and economic access, but there is also a great deal of current concern about climate change's impacts on food production and sustainability (R. C. Patel 2012, Downing 2013, see also box on Ellen Messer).

Undernutrition is well known to impair survival and contribute to numerous health problems (from chronic disease to infection) (Martins et al. 2011; R. E. Black et al. 2013), and the medical anthropologists Leslie Jo Weaver and Craig Hadley have considered the ways in which food insecurity also has negative effects on mental health: "Whether expressed as acute feelings of anguish and despair, as anxiety, resignation, hopelessness, and shame, or as embodied symptoms, qualitative analysis of this group of studies suggests that food insecurity compromises mental health" (Weaver and Hadley 2009, 275). Furthermore, food insecurity may be overshadowed by more visible health concerns such as HIV/AIDS, yet sufficient food is essential to effective treatment (Kalofonos 2010).

How Are Dietary Reference Intakes Constructed?

Dietary Reference Intakes (DRIs) for macro- and micronutrients are a diverse set of standards that are not always straightforward to interpret. They are developed by the Food and Nutrition Board of the Institute of Medicine, which is part of the National Academies of Science in the United States. They are the basis for the Dietary Guidelines for Americans (DGAs), which translate nutrient requirements into dietary recommendations. These in turn are the standards for a variety of food and nutrition programs.

The DRIs include the following:

- Estimated Average Requirements (EARs): This amount should satisfy the needs of 50 percent of the population.
- Recommended Dietary Allowances (RDAs): This amount should meet the requirements of nearly all healthy individuals in particular age/sex groups.
- Adequate Intake (AI): When no RDA is established, this is the amount that is believed to be adequate for a demographic group. It is used when firm scientific evidence does not exist for a given nutrient.
- Tolerable Upper Intake Levels (UL): For nutrients whose overconsumption can be dangerous (such as sodium or vitamin A), the UL is the maximum intake that does not appear to be associated with deleterious effects.

For macronutrients, there is a new reference, the Acceptable Macronutrient Distribution Range (AMDR), which provides a range of intake associated with protection from chronic disease while maintaining essential nutrient intake.

Adding to the confusion are Daily Values (DVs), which are used in food labeling. DVs are reported as percentages of the Recommended Daily Intakes (RDIs), based on the highest levels of the 1968 RDAs for adults and older children, standardized to a 2,000-kilocalorie intake. However, given that some are ULs and some are RDAs, the meaning of the percentage varies by nutrient.

DRIs have fluctuated over time as a function of new scientific evidence but also as a result of changing health concerns. For example, when the RDAs were established in 1941, they were used to assess the sufficiency of the American diet during wartime. However, current concerns about chronic diseases have resulted in changes in recommendations as linkages between nutrient intakes and cardiovascular disease, stroke, or cancer are demonstrated. Importantly, DRIs are based on the assessment of overall scientific evidence, but in fact most of the data come from the United States or Europe, so it is not known with any certainty whether there are meaningful population differences in nutrient requirements beyond those reported for different age or sex groups. See Nestle (2013) for a critical history of the development of nutrient recommendations and the DGAs.

When people receive all of the nutrients they need in the proper amounts, they are said to be in nutrient balance, or to have good nutritional status. If they receive too few nutrients they are undernourished (or suffer from **undernutrition**). If they receive too many it is called **overnutrition**. A word that can cover both conditions is **malnutrition**—which simply means "bad nutrition." In practice, malnutrition is most often used as a synonym for undernutrition. **Protein-energy malnutrition**

Not to be reproduced

Remove Watermark Now

occurs when there are shortages of these macronutrients. Deficiencies in the macronutrients usually lead to weight loss or, in children, deficits in growth in weight or height, as we will see in Chapter 5.

Digestive Physiology

Humans have a generalized digestive system, which seems well adapted for a high-quality omnivorous diet. This means a diet that includes both plant and animal foods and that is rich in nutrients relative to undigestible fiber. Other organisms, such as cows and colobine (leaf-eating) monkeys, have specialized stomachs that house colonies of bacteria to ferment the large quantities of fiber they consume from grass or leaves. An enlarged cecum, the pouch between the small and large intestine, fills the same role in other plant-eating organisms. Humans lack such obvious gut specializations, as shown in Figure 4.1.

The basic physiology of human digestion is as follows. Digestion starts in the mouth, where the teeth break up large food particles and the salivary enzyme amylase begins to break down starches (complex carbohydrates). After swallowing, food goes through the esophagus into the stomach, where it is subjected to strong acids that kill potential pathogens and enzymes that begin the process of digesting proteins. After two to three hours there, the resulting material (chyme) is sent into the small intestine. Most of the work of digestion and absorption is done within the small intestine. Here digestive enzymes (many coming from the pancreas) interact with food complexes to break down starches, complex sugars, fats, and proteins into their component parts, which can then be absorbed by the cells that line the small intestine. The remaining undigestible material continues on to the large intestine (colon), where water is absorbed along with the products of bacterial fermentation (such as volatile fatty acids) and other remaining biochemicals. The colon has large colonies of bacteria, most of which are helpful in that they can ferment some fibrous material that humans cannot break down with their own digestive enzymes. Treatment with broad-spectrum antibiotics often kills these bacteria, resulting in symptoms of diarrhea or gastrointestinal discomfort. The role that gut microbiota play in human health and disease has become a major new area of research because some bacteria are associated with greater risk of obesity or metabolic disorders, whereas others may protect against allergies or influence mood (Voreades, Kozil, and Weir 2014; Neuman et al. 2015). Gut microbiota vary across human populations (Obregon-Tito et al. 2015) and undoubtedly diet plays a role in shaping this variation (Xu and Knight 2015).

Nutrients that have been absorbed are transported via the lymph system to the bloodstream or they are sent via the portal vein to the liver for further processing. Ultimately, nutrients go to tissues that need to utilize them immediately. If they are not needed, the surplus is either stored or excreted. Excess fats are stored, and excess sugar and amino acid skeletons can be converted to adipose tissue (fat) as well. In contrast, the body has a limited capacity to store protein, minerals, or water-soluble vitamins, so surpluses of these are excreted in urine.

It is often difficult to diagnose nutrient deficiencies except in their extreme forms. Symptoms often take time to manifest, and a few days of nutrient deficiencies are not likely to have any serious or long-lasting consequences. However, chronic

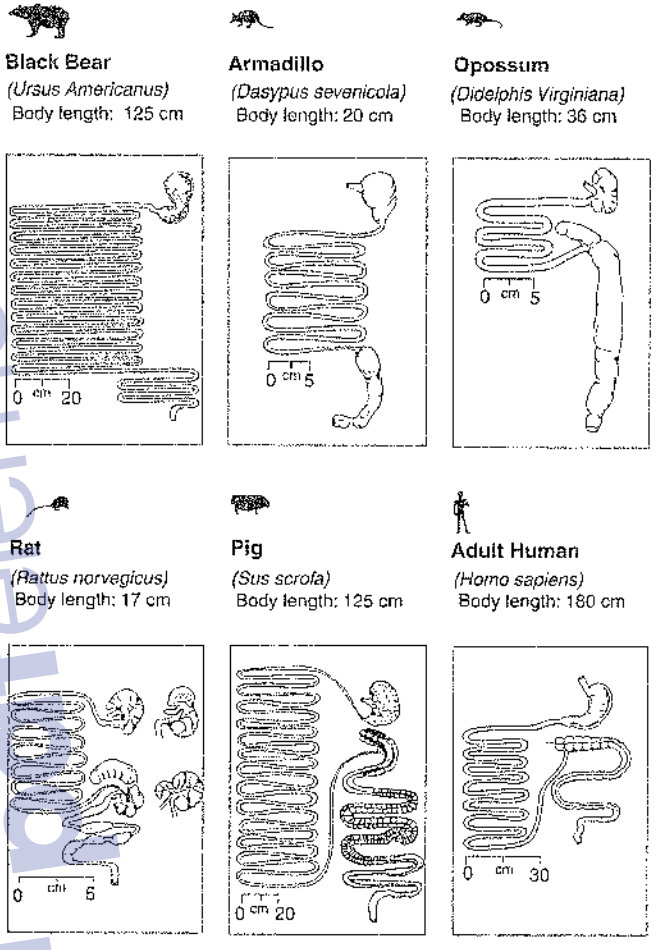


Figure 4.1
The diversity of gastrointestinal systems across mammals. Compared with other mammals, humans have a simple digestive tract, with no elaborated specialized digestive organs. This likely reflects an adaptation to a high-quality omnivorous diet.
Source: Stevens, C. Edward, and Ian D. Hume 1998. "Contributions of microbes in vertebrate gastrointestinal tract to production and conservation of nutrients." *Physiological Reviews* 78 (2):393–427. Reprinted with permission from The American Physiological Society.

deficiencies can. In response to low intake of nutrients, there are several adaptive physiological responses: more efficient absorption, conservation, mobilization of stores, or breakdown of active body tissues (e.g., muscle for protein) and prioritization of nutrient flow to more critical organs such as the brain. In the case of energy deficiency (as in food shortages or deliberate dieting), fat stores are mobilized first

Intellectual Property

Remove Watermark Now

because they can be used to fuel muscle activity. However, other organs such as the brain cannot use fat, but they can use ketones, which are a product of fat breakdown in the liver. When these energy resources are exhausted, muscle protein, as well as that from the heart, liver, kidneys, and other organs, can be used to synthesize glucose, which is the preferred source of energy for your cells. Fatigue is common to low-energy diets and probably acts to reduce energy expenditure. Deficiencies of protein and energy are associated with reduced fat deposits and muscle wasting, as well as impaired growth among children (see Chapter 5).

Just as there are nutrient deficiency syndromes, there are also potential negative health effects of nutrient surpluses, although most vitamin and mineral excesses are likely to come from nutrient supplements rather than from food. The most common nutrients that are overconsumed are fat, carbohydrates, protein, and sodium. It is possible to overconsume vitamin A if the liver of carnivorous animals such as polar bears is eaten because that is where vitamin A is concentrated. Indeed, hypervitaminAosis has been suggested as a cause of *pibloktoq* (see Chapter 11), a syndrome associated with temporary hysteria among Inuit populations of the Arctic (Landy 1985). Excesses of sodium may contribute to **hypertension**. Overconsumption of the macronutrients can contribute to weight gain, which in turn is associated with increased risk of various chronic diseases such as heart disease, diabetes, and various cancers.

An Evolutionary Approach to Nutrition

As discussed in Chapter 2, the biology of contemporary organisms has been shaped by their evolutionary history. The traits that organisms now possess reflect the advantages that accrued to individuals with those traits in some past environment, what is referred to as the **environment of evolutionary adaptation** (EEA). What constitutes the relevant EEA depends on what trait is being considered because traits evolved at different times in the past. For example, having flexible fingers, including an opposable thumb, first evolved among early primates some 50 million to 60 million years ago. Bipedalism, however, evolved sometime around 6 million to 8 million years ago, and the large brain of humans began to emerge around 2 million years ago.

With respect to general dietary and activity trends, the most relevant EEA for humans is considered the Upper Paleolithic, when humans were biologically "modern" (meaning indistinguishable from contemporary humans in their anatomy) and lived as hunter-gatherers. This period is roughly between 200,000 and 10,000 years ago, with the latter date signaling a transition away from foraging to food production (i.e., agriculture). There was a great deal of geographic variation among these Upper Paleolithic hunter-gatherers that resulted in differences in diet, with some relying more heavily on animal foods (e.g., high-latitude populations), whereas others had access to more plant foods. Populations foraged for wild foods and ate a broad diversity of plants and animals. Although historically this was considered a "hand-to-mouth" existence, where populations were constantly faced with the threat of starvation, ethnographic research among contemporary hunter-gatherers suggests that although they move around to sites with foods available, their diet is rich in nutrients and they need not spend all, or even the majority, of their time searching for food (Howell 2010; R. B. Lee 1968). Across hunter-gatherer groups, energy expenditure has been described as "moderate" rather than "high" (Pontzer et al. 2012; Gurven et al. 2013).

Scurvy in Evolutionary Perspective

A good example of a nutritional disease that is illuminated by the evolutionary perspective is scurvy. Scurvy is caused by a deficiency of vitamin C. Its symptoms manifest as breakdown of connective tissues—bleeding gums, pinpoint hemorrhages, and opening up of old wounds, among others. It was recognized initially in the sixteenth century primarily as a disease of sailors, who were often at sea for long periods of time without fresh fruits or vegetables. In Europe there were more than 100 scurvy epidemics between 1550 and 1857, and ships often lost up to half of their crews to this deficiency. With the discovery in the eighteenth century that citrus fruits such as oranges, lemons, and limes prevented scurvy, cases among sailors plummeted (this is also how British sailors came to be called "limeys") (Magiorkinis, Beloukas, and Diamantis 2011).

Interestingly, most mammals are not vulnerable to scurvy, with the exception of fruit-eating bats, guinea pigs, monkeys, apes, and humans. What do these organisms have in common that makes them differ from other mammals? All have lost the ability to synthesize vitamin C. Vitamin C is similar in structure to the sugar glucose, and most other animals can synthesize vitamin C from glucose or other sugars. All vitamin C–requiring animals lack the enzyme L-gulano-γ-lactone oxidase, the final step in the synthesis of ascorbic acid from glucose.

To understand why humans lack this enzyme, it is necessary to trace its loss in the anthropoid primate lineage, some 30 million to 40 million years ago. The most compelling hypothesis for why this would have occurred is that anthropoid primate diets tend to be high in fruit and other plant materials, which contribute large amounts of vitamin C to the diet. There would have been no need to produce vitamin C under these conditions, and the glucose used for its synthesis could be used for other important functions. In other words, organisms that did not produce vitamin C would have had access to more energy than those that continued to synthesize it.

With the transition to agriculture among populations throughout the world around 10,000 years ago, this way of life was dramatically altered. First, the types and diversity of plant foods that lent themselves to domestication and large-scale production were different from the foods consumed by hunter-gatherers. Hunter-gatherers consumed a diverse array of fruits, leafy vegetables, tubers, and nuts. With food production, it is not efficient to try to replicate this diet because each plant food has unique ecological requirements, so larger quantities of a smaller number of plants

Instructor Content

Remove Watermark Now

were grown. Grains such as wheat, barley, rice, or corn were more easily manipulated by early farmers, although the wild progenitors of these crops were tiny compared with their modern highly domesticated forms. Likewise, tubers such as potatoes or cassava (also known as manioc or yucca) are highly productive sources of carbohydrates, although they are not rich in micronutrients or protein. At the same time, plots of a single plant crop were vulnerable to random droughts or other weather problems and also to predation and plant pathogens, which could spread rapidly among a densely planted single-species crop. The net effect was a decline in dietary diversity, increased vulnerability to food shortages, and potential nutrient deficits (M. N. Cohen 1989). Furthermore, energy expenditure was likely increased, not decreased, by this transition and would have been more intensive during particular seasons (planting, weeding, harvesting; see also Dufour and Piperata 2008).

Jared Diamond once argued that agriculture was the "worst mistake in the history of the human race" (Diamond 1987). Although he can be critiqued for indulging in hyperbole, several studies of the fossil and archaeological record have shown that health generally declined in the post–food production era compared with earlier hunter-gatherers (M. N. Cohen 2009; Mummert et al. 2011). Before considering these studies, however, it is important to note that the reasons that most hunter-gatherer populations took up food production are not entirely clear. The most likely scenario is that Paleolithic hunter-gatherers were successful at acquiring food, which led to population growth. Over time, sufficient food became more difficult to access, resulting in intensification of resource usage. This may have included greater protection of favored foods (plants and animals), enhancing environmental conditions that supported the growth of foods, or consumption of less preferred foods, among others (M. N. Cohen 1989). Ultimately, this could have created settlements around sites of food production and greater "domestication" of plant and animal foods. Once these were in place, returning to hunting and gathering became increasingly untenable, especially because this transition appears to have stimulated further population growth. However, this new "lifestyle" came with multiple new challenges to health, as well as population growth (Mummert et al. 2011; Lambert 2009).

Ascertaining Diet and Nutritional Status from Ancient Bones

Ancient skeletons can provide a great deal of information on the health and nutrition of past populations. The study of health and disease using ancient skeletal materials is called paleopathology. Bioarchaeology is another related field that focuses on the biological remains to provide insight into past lifeways (such as diet and activity patterns). Several conditions can be ascertained from these materials, as shown in Figures 4.2a–d.

(continued)

(continued)

Harris Lines

Harris lines are horizontal bands visible on the long bones. They indicate periods of growth stoppage and an approximation of the age at which growth was disrupted. Multiple bands on a bone are evidence of multiple periods during which growth was interrupted during childhood. Growth may have been disrupted because of undernutrition, infection, or another form of stress.

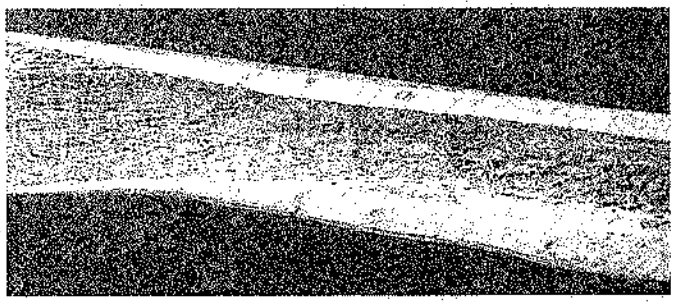


Figure 4.2a
Harris Lines, visible as darkened bands on the outer (cortical) bone.
Photo courtesy of Della Cook and Jenny Raff, Bioanthropology Collections, Indiana University.

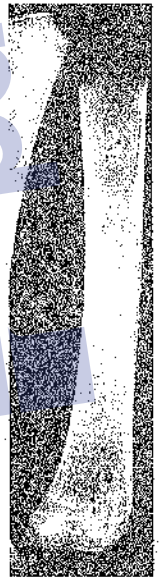


Figure 4.2b
Harris Lines, Late Woodland period, Ledders Mound Group, Illinois, child.
Photo courtesy of Della Cook, Bioanthropology Collections, Indiana University.

(continued)

Preview Department

Remove Watermark Now

(*continued*)

Enamel Hypoplasia

Enamel hypoplasia is also an indicator of growth disruption. Bands of thin enamel in teeth indicate periods of growth disruption. The width of these bands can be used to ascertain the relative duration of the growth stoppage, with wider bands indicating a longer duration of growth disruption. The placement of these bands also provides insight into the age at which the growth disruption occurred. Growth may have been disrupted because of undernutrition, infection, or another form of stress.



Figure 4.2c
Enamel hypoplasia, visible as bands in the tooth enamel. Adult male with linear enamel hypoplasia. From the Mississippian period, Schild Cemetery, Illinois.
Photo courtesy of Della Cook, Bioanthropology Collections, Indiana University.

Porotic Hyperostosis

Porotic hyperostosis is an indicator of anemia (reduced oxygen-carrying capacity of blood). Iron deficiency is a common cause of anemia, although there are many other causes. In response to anemia, red blood cell production increases, resulting in expansion of the bone marrow relative to the outer layer of bone. Porotic hyperostosis is visible when the outer bone is so thin or nonexistent that the porous inner bone is exposed.

Cavities

Cavities most often occur in the context of a diet made up of sugary or high-carbohydrate foods. Corn, for example, is high in sugar and is sticky, leading to the buildup of bacteria in the teeth, which ultimately can result in tooth decay.

(*continued*)

(*continued*)

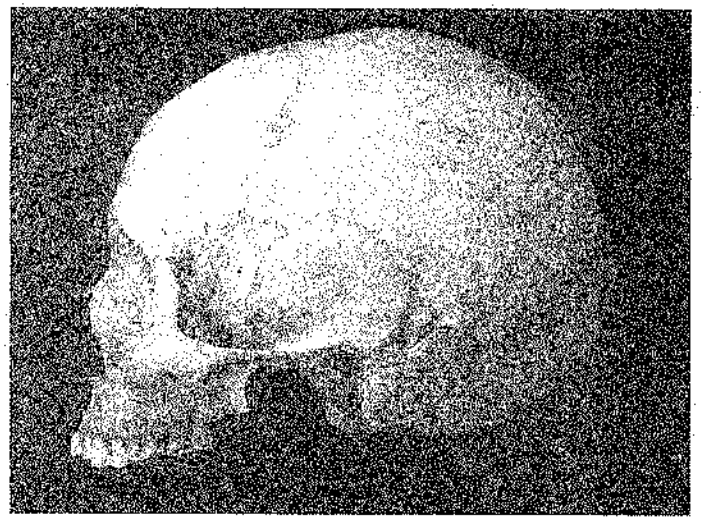


Figure 4.2d
Porotic hyperostosis, visible as the porous texture of the back of the cranium. Early nineteenth-century Euro-American young adult male, Petersburg, Illinois.
Photo courtesy of Della Cook and Jenny Raff, Bioanthropology Collections, Indiana University.

Osteoarthritis

Osteoarthritis most often results from repetitive use of joints, which results in visible modifications of these joints. The joints that show these bony distortions were likely those used routinely in hard work.

C_3 versus C_4 in Bones

Plants have different carbon "signatures" that, when eaten by humans, leave a distinct chemical trace in ancient bones. Corn is a common C_4 plant, and fruits, wheat, and nuts tend to be C_3 plants. This method is thus useful to get a sense of the components and quality of the diet of past populations, although it is impossible to distinguish whether the C_3 or C_4 plants were consumed directly or indirectly through consumption of herbivores eating them.

Nitrogen Isotopes

Different isotopic forms of nitrogen are found in marine and terrestrial plants and the animals that consumed these plants. Thus, the relative balance of these nitrogen forms can provide insight into the relative importance of marine versus terrestrial sources of food.

Not to be reproduced

Remove Watermark Now

The health consequences of this transition are evident from a variety of studies (see review in Mummert et al. 2011, with a focus on height as a measure of nutritional status). Claire Cassidy's classic work on the skeletons from two sites in Kentucky—one from a prehistoric hunting-and-gathering Native American group and one from an agricultural Native American group during the early colonial period (although they do not seem to have had contact with any Europeans prior to the site being abandoned)—provides an example (Cassidy 1980). The two sites were ecologically similar, but subsistence patterns and diet were different. The hunter-gatherers at the site called Indian Knoll appear to have eaten shellfish, fish, deer, other small mammals, and turkey, as well as a variety of wild plant foods. Members of the agricultural village (Hardin Village) ate corn, beans, and squash. Wild plants and some animals were supplementary to these staple plants. Table 4.2 shows some important differences in growth and nutrition between the two groups. Growth was disrupted in a more irregular fashion among the agriculturalists than it was among the hunter-gatherers and was generally of longer duration. Rates of anemia were also higher, and there were more cavities (most likely from the corn-based diet).

There is an important caveat to this interpretation, however. In an influential article from 1992, James Wood and colleagues (Wood et al. 1992) pointed out that increases in the number of individuals with nutritional pathologies in agricultural populations do not necessarily mean that health was getting worse. It may instead indicate underlying population growth. If populations are larger in the agricultural sites than in the hunter-gatherer sites, which is likely, the increased number of individuals with anemia, for example, could just be a reflection of there being more people, and the prevalence of disease could have actually decreased (this is what is known as the "osteological paradox"). Without knowledge of population size, increases in skeletal pathologies cannot necessarily be attributed to worsening health overall. Furthermore, because skeletal samples are by definition from dead individuals, it may be that they overestimate the true extent of disease in the population. That is, if a skeleton is estimated at 20 years of age at death, it is probably more likely to show evidence of pathology than a representative sample of living 20-year-olds in the population. The sample of skeletons is likely biased toward higher rates of disease. One last problem is

Table 4.2 Differences in Health between Agricultural and Hunter-Gatherer Groups in Kentucky

	Indian Knoll (Hunter-Gatherers) 2350 BC to 2015 BC Skeletons (*n* = 265)	Hardin Village (Agriculturalists) 1500 AD to 1675 AD Skeletons (*n* = 205)
Porotic hyperostosis (iron deficiency anemia)	0.00%	8.20%
Harris lines (growth disruption)	11.30% (regular; mild)	4.10% (random; severe)
Tooth decay (number of cavities)	0.73, men 0.91, women	6.74, men 8.51, women

Source: Table based on data from Cassidy (1980).

that those with evidence of pathology could in fact be healthier than others because the bony response develops when there is chronic exposure or as the problem heals. Thus, individuals with bony lesions may be the ones who survived because those who died of the disease may not have had time to develop the osteological traces. Thus, estimates of population health from skeletal pathologies must take these considerations into account (see updated discussion of this issue in Vercellotti et al. 2014).

Other archaeological evidence suggests that changes in social organization also occurred and contributed to disparities in health within agricultural populations. For example, some sites show that women became more likely to have evidence of nutritional deficiencies relative to men. With agriculture, surpluses of food are produced and must feed larger populations throughout the year, including seasons with little production. Those who could control land or food surpluses gained in economic and political power and were able to maintain better health than those with fewer resources (Goodman and Armelagos 1985; Vercellotti et al. 2014).

These patterns are common to many postagricultural populations and remain persistent issues in peasant agriculture. During the colonial period (roughly 1650 through the early twentieth century) with the rise of large plantations producing large amounts of crops for export to the colonial powers, the possibility of even further global differentiation in nutritional status emerged, with some receiving the benefits of these relatively cheap surpluses, even as others toiled to produce them for export. For example, Clark Spencer Larsen has shown how colonization in North America caused a worsening of diets of Native Americans in the Southeast and increased workloads (Larsen 2005). Corn came to predominate in the diet, resulting in cavities, hypoplasias, and anemia; Native Americans were used as laborers on plantations, and their bones show evidence of osteoarthritis and heavy upper-body workloads.

This trend has continued in the postcolonial period with the rise of multinational corporations that control vast areas of farmland and produce single crops such as wheat, corn, rice, sugar, fruits (such as bananas or pineapples), cows and pigs, or nonnutritive crops (such as tobacco, coffee, or tea). The net result is a surfeit of inexpensive carbohydrate, fat, and protein sources marketed primarily in wealthy countries, overconsumption of these foods, and reduced energy expenditure among individuals. Among those who produce them, decreased nutrient intake and increased caloric expenditure render them vulnerable to undernutrition, in part as a function of their increased reliance on market foods as subsistence land is used for export crops (Pelto and Pelto 1983).

So how does this historical overview relate to an evolutionary approach to nutrition and health? Contemporary dietary patterns are at odds with those that typified most of our evolution as a species. In other words, we are biologically adapted to a diet and lifestyle that no longer prevails, and our bodies are now being asked to function in different ways with different resources. Remember that genetic evolution is slow, with changes in gene frequencies accumulating slowly across generations. In contrast, cultural changes (e.g., in technologies and dietary patterns) can be rapid, with radical changes occurring within just one or two generations. It is no surprise that many chronic diseases such as cardiovascular disease, diabetes, obesity, stroke, and cancers that are prevalent in wealthy countries (and becoming more common in nations with emergent middle classes) are the outcomes of these

Taylor & Francis
Not for distribution

Remove Watermark Now

diet and activity changes. It is not only that access to cheap sources of energy, fat, and protein has increased (and these products dominate grocery stores and food advertisements), but also that other micronutrients and dietary components that were commonly consumed by our hunter-gatherer ancestors are missing from the diet. As a result, despite advances in prevention and treatment of disease, overconsumption combined with less physical activity contributes to high rates of chronic disease and early death.

Anthropologist in Action: Ellen Messer

Ellen Messer holds a Ph.D. in anthropology from the University of Michigan and is currently a visiting scholar at Brandeis University's Heller School's Institute for Global Health and Development. Formerly she was director of the Alan Shawn Feinstein World Hunger Program at Brown University, where she worked with interdisciplinary teams that sought to end the connections between hunger and conflict. In that capacity she was co-editor of the *World Hunger Report 1995* (with Peter Uvin). She is currently a member of the American Anthropological Association's Food Crisis Task Force and has long been involved in bringing anthropological sensitivities to understanding both the roots of hunger and the policies designed to alleviate it.

Dr. Messer's areas of expertise include food security, hunger, and human rights. She has spent the past decades investigating how hunger and inadequate access to safe, sufficient, and nutritious food (i.e. food insecurity) are involved in conflicts throughout the developing world. Messer writes and consults frequently on policy issues related to food security. An "activist-scholar," she is engaged in debates over access to food as a human right, genetically modified foods as solutions to global hunger issues, and the role of food in local and national conflict. To that end, she and her colleagues coined the phrase "food wars" in the 1980s to describe how conflicts may stem from lack of food, how conflicts may result in widespread hunger or lack of access to food, and how food is used as a tool in wars and other smaller-scale conflicts.

With regard to the contemporary global situation regarding food security, she notes, "We live in a world that has more than enough food to feed everyone on the planet a nutritionally adequate, basic diet, and that has been the case since the 1970s. The problem is who controls food and the global food system. In 2007 and 2008, there was a food crisis caused by spot food shortages and food-price spikes in various countries around the world. In some cases, food was diverted for use as biofuel or as livestock feed; in other cases, it was hoarded. As a result, according to estimates by the U.N. Food and Agriculture Organization, an additional 100 million people experienced extreme food poverty: They could not afford to access a nutritionally adequate diet" (adapted from http://www.brandeis.edu/magazine/2011/summer/featured-stories/foodwars.html).

Nutrition and Contemporary Chronic Diseases

The most common causes of death in the United States, most European countries, and some Asian countries such as Japan are cardiovascular disease, cancer, and stroke. These predominantly affect older individuals, although there is evidence that some of the risk factors for these diseases are becoming apparent at younger ages, including among children (Van Cleave, Gortmaker, and Perrin 2010; Juonala et al. 2011). **Cardiovascular disease** (CVD) can include any dysfunction in the heart or circulatory system, but the most common is **atherosclerosis**, or hardening of the arteries because of plaque buildup or inflammation. **Stroke** involves an interruption in the blood supply to all or part of the brain, usually because of a ruptured blood vessel or blood clot. This can result in the deaths of nerve cells (neurons) and, depending on where the stroke occurs in the brain, paralysis or loss of motor and other functions. **Cancer** is the uncontrolled proliferation of malignant cells that, if they achieve broad distribution in the body, fatally disrupt physiological function. There are overlaps in the behavioral risk factors for these three diseases, especially CVD and stroke, including obesity, diabetes, high-fat and high-salt diets, lack of physical activity, and smoking, among others. Diet plays an important role—both directly and indirectly by influencing risk factors such as diabetes and obesity.

In 1985, S. Boyd Eaton and Melvin Konner wrote a groundbreaking article in the *New England Journal of Medicine* outlining the ways that diets of wealthy countries such as the United States deviate substantially from the diets of contemporary hunter-gatherers. Similarly, populations of wealthy countries tend to have a high prevalence of chronic diseases, whereas hunter-gatherers have notably lower risks of these diseases (Eaton and Konner 1985). These ideas and data have been updated and refined since (Konner and Eaton 2010; Cordain et al. 2005; Lindeberg 2012), and there is a difference of opinion about the foods and their relative contributions to the Paleolithic diet (also dubbed the "Paleo diet"), but the general principles remain the same. Most of the data on the Paleolithic diet come from studies of modern hunter-gatherers because it is difficult to accurately assess diet from the fossil record. This is because plant materials degrade rapidly, whereas animal bones are larger and contain inorganic material, making them more durable and evident in the fossil record. It is also important to remain cognizant of the fact that contemporary hunter-gatherers are neither "premodern" nor our ancestors; they are using a subsistence strategy that was, however, common among preagricultural populations.

People in subsistence-level societies engage in higher levels of physical activity compared with those in wealthy countries where people often do not use physical labor to meet their subsistence needs (Dufour and Piperata 2008), although differences between hunter-gatherer and Western population energy expenditure are not as great as anticipated (Gurven et al. 2013; Pontzer et al. 2012). Many forms of contemporary work offer little opportunity for physical exertion and more or less confine people to their desks for hours a day, and prolonged sitting itself may increase the risk of many chronic diseases and mortality (Biswas et al. 2015). Leisure activities such as television watching or video games are also often sedentary, and there may be few safe and accessible opportunities for physical activity. In addition, hunter-gatherers consume almost no grains, have no dairy foods, and have limited

Remove Watermark Now

access to saturated fat and salt. Their diets are high in fiber, micronutrients, potassium (compared with sodium), and protein, as shown in Table 4.3. With the exception of high-latitude groups, most of the diet derives from a diverse array of plants but also includes meat as a source of animal protein, although not saturated fat because wild animals rarely have the opportunity to accumulate rich fat deposits. Tobacco and alcohol historically were not available, although contemporary hunter-gatherers may use such products.

In contrast, diets in wealthy industrialized societies are based on carbohydrate-rich cereals, most often processed to remove the bran (which contains micronutrients and fiber); dairy products, including those high in saturated fat (cheese); pure sugars, sodium, and hydrogenated oils in highly processed snack foods; and meat with a high percentage of saturated fat because of the calorie-rich diets fed to livestock and their lack of activity. It should also be noted that the DGAs reflect the fact that nutritional science and policies have developed in the context of the American agricultural economy, where it is expected that carbohydrates will be coming from grains and that calcium will be coming from milk, two food types that would not have been found in hunter-gatherer diets.

Hunter-gatherer diets contain a broader diversity of plant foods than typify agricultural diets, and importantly, this provides a wide array of **phytochemicals**, which are nonnutritive but biologically active compounds present in plants.

Table 4.3 Widely Agreed-on Qualitative Differences between Average Ancestral (Hunter-Gatherer) Diets and Contemporary Western Diets

	Ancestral/ Hunter-Gatherer	Contemporary Western
Total energy intake	More	Less
Caloric density	Very low	High
Total carbohydrate intake	Less	More
Added sugars/refined carbohydrates	Very little	Much more
Fruits and vegetables	Twice as high	Half as much
Fiber	More	Less
Protein intake	More	Less
Total fat intake	Equal	Equal
Polyunsaturated fat	More	Less
ω-6:ω-3	Roughly equal	Far more ω-6
Long-chain essential fatty acids	More	Less
Micronutrient intake	More	Less
Sodium:potassium ratio	<1	>1
Milk products	Mothers milk only	High, lifelong
Cereal grains	Minimal	Substantial

Redrawn from data in Konner, Melvin, and S. Boyd Eaton 2010. "Paleolithic nutrition twenty-five years later." *Nutrition in Clinical Practice* 25 (6):594–602.

Although these are not like vitamins in that they are not considered "essential" to life, more attention is being paid to them as important modulators of physiological function. They are common in most hunter-gatherer diets, but with plant domestication these often-bitter substances are frequently bred out in favor of more palatable plant strains. Phytochemicals are often missing in contemporary diets dominated by refined carbohydrates, saturated fats, and animal protein. Important classes of these include alkaloids, saponins, cardiac and cyanogenic glycosides, terpenoids, nonprotein amino acids, and proteinaceous phytohemagglutinins and proteinase inhibitors (Johns 2007). Although some of these are potentially toxic (and are produced by plants as defenses against being eaten or parasitized), their chemical properties may render them beneficial to health in both specific and generalized ways (Frison et al. 2006; Traka and Mithen 2011). For example, many plants contain antibiotics that ward off bacteria, helminths, or other disease-causing microorganisms. Fava beans, which are widely consumed in the Mediterranean region, contain a substance called vicine, which inhibits malaria infection.

Aside from these antimicrobial properties, many other phytochemicals have more general effects that are important in chronic disease (Howes and Simmonds 2014; Traka and Mithen 2011). These are most broadly distributed in fruits and vegetables, and most dietary recommendations for avoiding chronic disease such as CVD and cancer include consumption of more and different kinds of fruits and vegetables. Many such phytochemicals have benefits via their actions as antioxidants, which reduce cell damage from oxygen-free radicals. Others have anti-inflammatory properties, important for cardiovascular health and potentially of significance in hampering tumor growth. Isoflavones, common in soy products, act as weak estrogenic compounds and appear to influence endogenous sex hormone activity, cell proliferation, and differentiation. Many spices used in traditional cuisines such as turmeric or cumin also have neuroprotective, antioxidant, anti-inflammatory, and potentially anticancer properties, among others (Perrone et al. 2015; Opara and Chohan 2014). Although any given phytochemical may not be especially important when it comes to chronic disease, diets containing an abundance and diversity of plant foods have a mix of phytochemicals that act synergistically in the reduction of chronic disease risk. Most hunter-gatherer diets fall into this category.

Because *Homo sapiens* evolved primarily under hunter-gatherer conditions, a diet characterized by a great diversity of wild plant and animal foods and limited in sugars, fats, and salt is likely to be the one to which our bodies are well adapted. Given that the latter nutrients are important to physiological function and energy storage and yet they are rare in nature, it is not surprising that consumption of foods with these nutrients is experienced as pleasurable and that many mammals perceive these as "tasty" and seek them out preferentially (Bachmanov et al. 2011). However, in an environment characterized by cheap and widely available foods high in these nutrients, they are more likely to be consumed in large quantities. The dietary and physical activity patterns common in many countries (and the wealth on which it is predicated) thus represent an *evolutionarily novel* set of environmental conditions, and they contribute to an increased risk of chronic diseases.

Importantly, because these diseases generally cause morbidity and mortality relatively late in life, individuals suffering from them are not being "selected

Taylor & Francis Not for distribution

Remove Watermark Now

against." That is, whatever genes contribute to chronic disease vulnerability will not be removed from the gene pool, because these diseases most commonly occur after an individual has reproduced (i.e., their fitness is established). One often-cited explanation for this is that life expectancy in wealthy societies is so much longer than that of hunter-gatherers, on the order of some 35 years, that only now do humans have the opportunity to suffer from these chronic diseases. The conclusion is that if hunter-gatherers had only lived that long, they too would experience these diseases, or, we live so long now that these are "inevitable" consequences of the extension of the lifespan. This is incorrect. The major factor that influences life expectancy is infant and child mortality. If large numbers of very young individuals die, the average life span will appear to be quite short. If deaths of the very young are exempted, life expectancies are less dramatically different across populations (Blurton Jones, Hawkes, and O'Connell 2002). In contrast, as mortality risk goes up with increasing age independent of chronic disease, reducing death from chronic disease is likely to have only a modest effect on life expectancy, with some estimates of four to five years if all of the major known risk factors for the leading chronic diseases were eliminated (Danaei et al. 2010).

The take-home message from the evolutionary approach to nutrition and chronic disease is that eating more like our hunter-gatherer ancestors and engaging in a high level of physical activity on an ongoing basis should result in a reduced risk of diseases such as CVD, diabetes, and stroke, and there is some evidence that individuals on a Paleolithic diet have better outcomes (in terms of chronic disease risk factors) than those on diets based on traditional dietary guidance (Manheimer et al. 2015). However, given that most contemporary humans cannot return to a hunter-gatherer lifestyle and that most humans live in agricultural or industrial societies, how is this supposed to occur? Most contemporary dietary guidelines, including the official DGAs from 2015, include recommendations that, in general terms, provide a template, although with marked differences from a Paleolithic diet (see Table 4.3). The DGAs advocate eating a variety of plant-based foods, including fruits, vegetables, and whole grains (which were not a large part of hunter-gatherer diets) and limiting fatty meats, foods with added sugars or those that are highly processed, and alcohol. The DGAs also recommend increased consumption of low-fat dairy products, which would not have been part of the ancestral diet (although hunter-gatherers appear to get more than sufficient calcium from their diet; Eaton, Eaton, and Konner 1999). Both recommend a physically active lifestyle and a balance between caloric intake and physical activity such that body weight does not increase. These are all reasonable goals, but they are more easily recommended than enacted within the social and economic contexts of contemporary societies and our biology.

It is worth noting the proliferation of evolution-based diet books that advocate a hunter-gatherer-type diet, such as *The Paleolithic Prescription*, *The Paleo Diet*, *The New Evolution Diet*, and *The Primal Blueprint*. All of these are predicated on certain assumptions about human ancestral diets, often that they were rich in meat and light in seeds/grains or legumes. There are even groups of people who attempt to recreate this lifeway by eating large quantities of meat, fruits, and vegetables, but no bread or dairy products, fasting for lengthy periods, and engaging in physical activities such as sprinting and jumping, and some eschew foods native to the New World or donate blood regularly to simulate blood loss from accidents (Goldstein 2010). Whether these types of diets actually benefit long-term health or lengthen life remains to be seen; suffice it to say that there probably is some benefit to consuming a diet more similar to that of our ancestors, but it is wise to keep in mind that we have no firm evidence for exactly what that diet entailed in its entirety.

Obesity

As of 2014, the World Health Organization (WHO) estimates that 13 percent of the adult population of the world is obese, although this percentage ranges from 5 percent in Southeast Asia to 27 percent in the Americas. The obesity epidemic is an issue of global proportions, as Figure 4.3 illustrates (see also Brewis 2011). Obesity prevalence is rising in many developing countries, where it often coexists with undernutrition, the so-called "double burden" of malnutrition (Abdullah 2015). **Obesity** has been defined in different ways over the years, and currently the most common measure used is the **body mass index** (BMI). BMI is calculated as weight (kg)/height (m)^2 or, to calculate it in pounds, weight (lbs)/height (in)^2 × 703. Currently a BMI between 18.5 and 24.9 is considered healthy; 25 to 29.9 is considered **overweight**; and 30 or greater is defined as obese. A BMI less than 18.5 is considered underweight. Because of their different body proportions, BMIs are interpreted somewhat differently for children. BMIs are supposed to be used as a screening tool to identify possible health problems, but a high or low BMI is not, in and of itself, a diagnosis of ill health.

Mammals store excess energy as fat and appear to have virtually unlimited ability to do so. The function of this storage mechanism is to have a source of energy available for times when food is rare or difficult to find or when an animal hibernates. However, most likely for the first time in evolutionary history, a large number of humans are able to store a large quantity of fat that becomes a more or less permanent part of their anatomy. A question is whether obesity is a problem in and of itself or whether its health significance is via increased risk for impairments in physiological function or individuals' ability to carry out daily activities, or whether its social valuation (e.g., stigma) contributes to its associated health outcomes (Brewis et al. 2011; Saguy 2012). It used to be thought that fat was metabolically inert, but recent research indicates that fat, especially that stored in the abdomen, is metabolically active and has the potential to influence the amount of fat in circulation or inflammation, both risk factors for metabolic and cardiovascular disease (P. Patel and Abate 2013). Thus, fat patterning is emerging as a more important risk factor for metabolic disease, but overall, having more fat tends to correlate with a greater risk of metabolic, cardiovascular, and cancer morbidity, even with a BMI in the normal range (Romero-Corral et al. 2010). Some obese individuals are metabolically healthy, but may nonetheless have some subclinical health risk factors (Roberson et al. 2014), and there is a growing "fat acceptance" movement (Saguy 2012).

Researchers are not only concerned with obesity and its health effects among adults, but also have become increasingly worried about its rising prevalence among children. In the United States, about 17 percent of all children and

Routledge Taylor & Francis

Remove Watermark Now

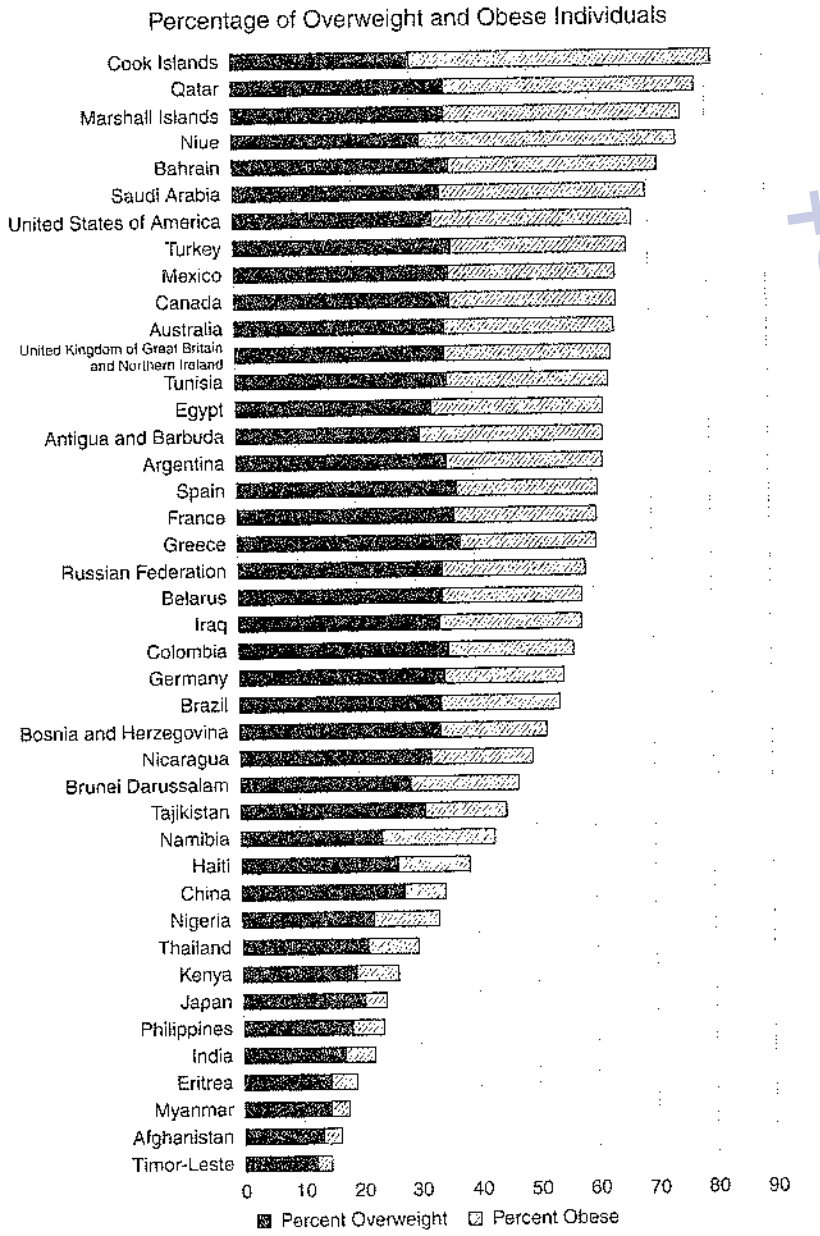


Figure 4.3
Current age-standardized prevalence of overweight and obesity from selected countries across the globe.
Based on data from the World Health Organization, http://apps.who.int/gho/data/view.main.2450A?lang=en

adolescents are considered overweight or obese, almost three times higher than the prevalence of obesity in 1980 (https://www.cdc.gov/obesity/data/childhood.html). Obesity in childhood appears to be difficult to reverse and is associated with a large increase in the risk of diabetes and markers for CVD at an earlier age. Indeed, because of the "obesity epidemic," epidemiologists worry that the next generation of people in wealthy countries may have a lower life expectancy than their parents, which would be a striking reversal of the increases in life expectancy that have been sustained over the twentieth century (Stewart, Cutler, and Rosen 2009).

At the same time, it is worth questioning some of the causes of this epidemic. Cheryl Ritenbaugh has described how standards for "ideal weights" (presumably those associated with the lowest mortality) have declined over the latter part of the twentieth century, especially for females, as shown in Figure 4.4. This alone could account for a sudden "rise" in rates of overweight and obesity evident in the 1970s and 1980s. Ritenbaugh (1982) suggested that changing ideals for weight and

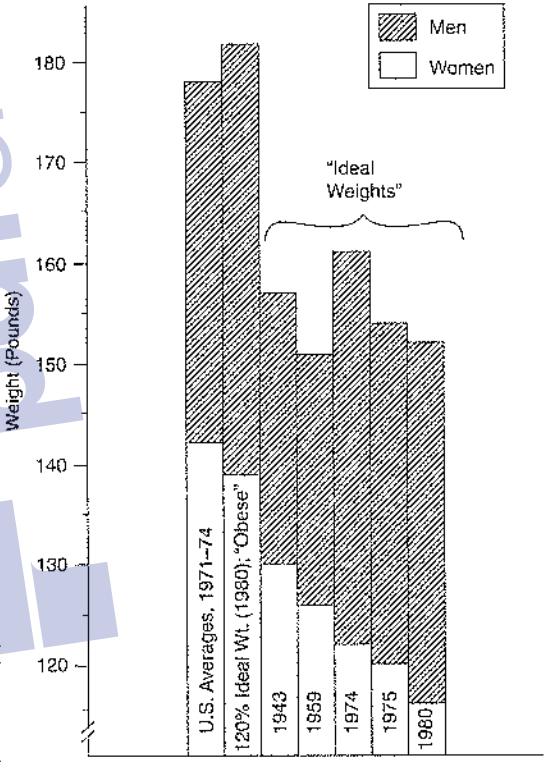


Figure 4.4
Changes in "ideal" weights over time.
From Cheryl Ritenbaugh. 1982. "Obesity as a culture bound syndrome." *Culture, Medicine, and Psychiatry* 6 (4):355. Reprinted with kind permission from Springer Science and Business Media.

Taylor & Francis

Remove Watermark Now

overweight are more reflective of shifting cultural ideas of the ideal body than based on strong scientific evidence that lower weights are associated with reduced morbidity and mortality. Starting in the 1980s, BMI replaced weight as a standard, so it is difficult to see whether this trend has continued and is evident in current recommendations. During the 1980s and 1990s, BMI standards for overweight tended to hover around 27 or 28 and allowed for increases with age and differences between males and females (males had slightly higher cutoffs for overweight). In 1997, the WHO established a standard of 25.0 for overweight for all adults, and the subsequent year the U.S. standard was aligned with it (Kuczmarski and Flegal 2000). This effectively redefined 35 million people previously considered of normal weight as overweight!

What is the relationship between BMI and mortality risk? Studies have shown a U-shape relationship between body weight and mortality (see Figure 4.5). That is, both high and low BMIs are associated with higher mortality than BMIs in between. The high mortality at the low end can occur because individuals who are very thin may be those who are already sick or who substitute smoking for eating, but thinness remains a risk for mortality even when these other factors are controlled for statistically. Furthermore, the mortality risk associated with a given BMI changes with age; higher BMIs early in life (age 30 years) are associated with higher mortality than they are at age 70 years, and it differs by gender. Thus, the message should not be that thinness is, by definition, healthier than fatness, but rather that the behaviors that contribute to a given weight (such as eating patterns, physical activity, or smoking) are associated with different health consequences.

Moreover, it appears that the relationship between body size and health has itself changed over time and that it may be variable across populations. Barry Popkin has argued that Asians, Hispanics, and Africans experience more negative health outcomes at lower BMIs than do whites, suggesting that there is population variation in the meaning of BMI (Popkin 2002). Moreover, the association between BMI and fatness may also differ; in India, higher levels of fatness are seen at lower BMIs, resulting in what has been termed the "thin-fat" phenotype (Yajnik 2004). As a result, different countries and regional bodies set the cutoffs for normal, overweight, and obesity at different points. In Asians, for example, the cutoff for overweight and obesity is lower than the WHO standards (WHO Expert Consultation 2004). Max Henderson's historical work on BMI in the United States also shows that although BMIs have increased since the nineteenth century (and obesity rates have increased 10-fold), the ideal range of BMI for men (in terms of lower mortality) has shifted upward, from 20–26 to 22–28. There are a variety of reasons for this, including changing height, patterns of disease, dietary components, and infectious disease exposure, all of which affect the height-for-weight measure (Henderson 2005). In sum, standards for body size change over time as a function of cultural trends and scientific understandings, and the associations between a given body size and health are neither fixed nor universal.

What else has contributed to this rise in obesity, especially in children? Generally, it is assumed that increased consumption of more energy-dense, nutrient-poor foods with high levels of sugar and saturated fats combined with reduced physical activity are responsible, but the evidence supporting these claims is less strong than

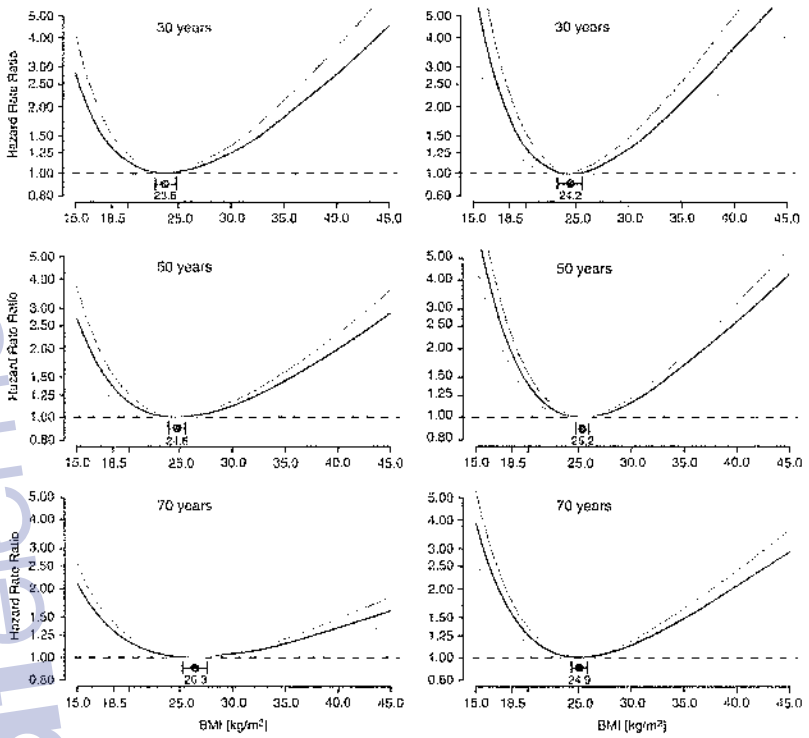


Figure 4.5
The relationship between body mass index (BMI) at different adult ages and mortality for women (left) and men (right) at different ages. Lines represent the risk relative to the optimum BMI and 95% confidence intervals. The black dot represents the BMI associated with minimum mortality. Mortality rises at both low and high BMIs, although the difference in risk associated with higher or lower BMI varies by gender and age.
Reprinted by permission from Macmillan Publishers Ltd: Peter, R. S., B. Mayer, H. Concin, and G. Nagel. 2015. "The effect of age on the shape of the BMI–mortality relation and BMI associated with minimum all-cause mortality in a large Austrian cohort." *International Journal of Obesity* (2015)39, 530–534; doi:10.1038/ijo.2014.168.

one would think, and there are myriad other factors that may also play a role, such as reduced sleep duration, some infections, gut bacteria, and positive assortative mating (Dhurandhar and Keith 2014). Increasing numbers of studies are pointing to the influence of advertising, especially that of high-calorie foods marketed to children in television programming, as one of the nonproximate causes (Powell, Schermbeck, and Chaloupka 2013); "pouring" contracts in schools whereby soft drink companies have been allowed to put vending machines in schools in exchange for financial support or educational materials; reduction in physical activity programs in school or safe places for children to engage in such activities outside of school; the decline in home-prepared foods and "family dinners" concurrent

Interdependent

Remove Watermark Now

with the spread of fast-food restaurants in which calorie-dense foods predominate on the menu; large serving sizes of home meals and those served at restaurants; and the disproportionate amount of space in grocery stores devoted to foods that dietary guidelines suggest should be consumed "sparingly" (Brownell 2004; Cheyne et al. 2014). In other words, there are strong political, economic, and social interests at work that conspire against a healthy diet (Brewis 2011; Brownell 2004; Critser 2003; Pollan 2006; Nestle 2013, 2015). These are especially relevant for individuals living in working-class urban areas where caretakers work long hours and where there is limited access to fresh foods and large grocery stores and a high density of fast-food restaurants.

In addition, a widely cited study in the *New England Journal of Medicine* found that obesity seems to "spread" through social networks (Christakis and Fowler 2007). The researchers found that if one person in a social network was obese, there was an increased probability that others in the network would also become obese (while controlling for numerous other environmental factors), even if members of the network were not geographically close. The likelihood was higher still among close friends and siblings, especially those of the same sex. The authors of the study suggested that these trends could be a result of changing perceptions of the social norms regarding the acceptability of obesity. They noted,

> To the extent that obesity is a product of voluntary choices or behaviors, the fact that people are embedded in social networks and are influenced by the evident appearance and behaviors of those around them suggests that weight gain in one person might influence weight gain in others. Having obese social contacts might change a person's tolerance for being obese or might influence his or her adoption of specific behaviors (e.g., smoking, eating, and exercising). In addition to such social mechanisms, it is plausible that physiological imitation might occur; areas of the brain that correspond to actions such as eating food may be stimulated if these actions are observed in others. (Christakis and Fowler 2007, 370)

Thus, obesity has social roots that must be addressed as part of any sort of "treatment" program.

In the past, and among contemporary hunter-gatherers or subsistence agriculturalists, only individuals with ample resources would have been able to achieve overweight or obesity. In a study in Samoa, where obesity rates are very high, it was found that weight was positively associated with social status. Large-bodied men were considered to look more regal and commanded more respect, and high-status men and women could become fat because they could order others to work for them (Brewis et al. 1998). Anthropologists have noted that as traditional societies transition to urban, industrial societies with greater exposure and access to Western goods and ideals, perceptions of weight tend to reverse, and fatness is no longer associated with wealth or health, especially among women, and becomes highly stigmatized (Brewis et al. 2011; Becker 2004). It is notable that obesity in the United States is more common among groups of lower socioeconomic status and minorities (the two categories often go together in the United States) such as African Americans, Native Americans, Hispanics, and Mexican Americans (Ogden et al. 2014).

As noted earlier, access to fresh produce and grocery stores is more limited in lower-income areas, fast-food restaurants have a much higher density in such areas, and access to safe parks or sports facilities is much lower than in middle-class neighborhoods (Critser 2003; Lake and Townshend 2006; Engler-Stringer et al. 2014).

The reversal of a positive association of socioeconomic status and weight reveals an important evolutionary change. Historically and in the prehistoric period, weight would have been a symbol of resource accumulation and hence wealth: it reflects storage of energy that could be mobilized in seasons of hunger or food scarcity. The current negative relationship between socioeconomic status and weight in industrialized countries is therefore an evolutionary and historical anomaly. It seems likely that only in the context of unprecedented food security could the lack of fat be socially valued. This trend is spreading, however, with the global presence of Western celebrities and cultural values that emphasize slimness, and there is also evidence of a global spread of fat stigma, especially among middle-income and developing countries (Brewis et al. 2011).

Thus, the conundrum of rising rates of overweight and obesity across the globe seems unrelenting and irreversible, and interventions of various kinds designed to promote weight loss have been notoriously ineffective and unsustainable. As Brewis notes, "Obesity is a complex, multifaceted, deep-rooted part of the contemporary human condition that resists simple, singular, quick, or easy fixes. Because it has stemmed from fundamental social, economic, and ecological changes our species has faced over the past few decades, only sustained and fundamental examination of those conditions is likely to do much about it (Brewis 2011, 128–129). How fatness ultimately impacts human health on a local and global scale will be a critical issue for the twenty-first century.

Diabetes

Diabetes is a disease often found along with obesity, in which levels of sugar (glucose) in the blood are too high. Blood glucose levels normally range between 70 and 100 milligrams per 100 milliliters in the fasting state. If they exceed the upper limit, glucose begins to be excreted in urine, and the person feels hunger and thirst. **Insulin**, a hormone produced in the pancreas, is released as soon as glucose enters the bloodstream after a meal, and it stimulates the uptake of glucose by muscle, fat, and other cells and the conversion of glucose into glycogen (the only form in which glucose is stored) in the liver. With diabetes, blood glucose levels are continually high because of problems with either insulin production or cellular responses to insulin.

There are two major types of diabetes. In diabetes mellitus type 1 (also called insulin-dependent diabetes, IDDM), the problem derives from reduced insulin production and release by the pancreas. This type of diabetes typically starts during childhood and requires insulin therapy. Much more common is type 2 diabetes mellitus, or non–insulin dependent diabetes mellitus (NIDDM). NIDDM results when the cells that are the target of insulin (such as fat or muscle cells) become resistant to it and more and more insulin is required to stimulate those cells to take up glucose. This can happen as a consequence of repeated insulin stimulation, as in a high-glucose diet, or as a consequence of reduced cellular need, as in a lack of

Preview Only

Remove Watermark Now

activity. This type of diabetes is generally manageable or reversible. Reduced intake of sugars or other foods that increase blood sugar or insulin levels, combined with increases in physical activity, can reverse insulin resistance. Currently, more than 12 percent of the U.S. population age 20 or older has diabetes, and rates vary by minority grouping, with non-Hispanic whites having the lowest rates (7.6 percent), whereas 9 percent of Asian Americans, 13 percent of both blacks and Hispanics, and 16 percent of Native Americans have diabetes (http://www.cdc.gov/diabetes/pubs/statsreport14/national-diabetes-report-web.pdf).

In 1962, the geneticist James Neel proposed an evolutionary hypothesis in an attempt to explain the relatively high frequency of diabetes (he initially did not make a distinction between NIDDM and IDDM) in many populations. In what became known as the "thrifty genotype" hypothesis, James Neel (1962) posited that our hunter-gatherer ancestors would have experienced alternating periods of food abundance and food scarcity. Those with a "quick insulin trigger," who quickly released a large quantity of insulin in the presence of glucose in the bloodstream, would have had a fitness advantage. This is because they would be able to more efficiently store energy as fat by rapidly converting glucose into storable fat. During times of relative scarcity, such individuals would have had more fat (i.e., energy reserves) to use than those with a less-efficient energy storage mechanism. This would have served our ancestors well. However, in the context of continual access to abundant quantities of food, especially glucose-rich carbohydrates, this quick trigger is now highly detrimental because individuals continually produce high levels of insulin, leading to insulin resistance among target cells.

Subsequent to Neel's proposal, it became evident that not everyone eating a calorie-rich diet was equally likely to develop NIDDM. Instead, certain populations seemed to be more susceptible than others, even when eating similar kinds of diets. In particular, Native North Americans have high rates of both NIDDM and obesity. The Pima, a Native American group living in the southwestern United States (also known as the Akimel O'odham), have been singled out as having the highest rates of any population, with 38 percent of U.S. Pima adults having NIDDM (Schulz et al. 2006). Among Pima living in Mexico, however, the rate was much lower, at 7 percent, which was not significantly different from the rate found among nearby non-Pima Mexicans. For those 55 years of age and older, the rate for U.S. Pima is 77 percent, compared with 9 percent among the Mexican Pima. Similarly, obesity rates were 3 to 10 times higher in the U.S. Pima group than the Mexican Pima (Schulz et al. 2006). Much research has focused on genetic characteristics of the Pima, and Native Americans in general, as predisposing them to higher rates of NIDDM and obesity, but it remains an open question as to whether and, if so, why there is a genetic predisposition among Native American groups that puts them at higher risk of NIDDM.

Variation among the Pima stems, in part, from differences in activity patterns. The Mexican Pima have much higher activity levels because they have been able to maintain their farming tradition, whereas the U.S. Pima rely on wage labor and have become more sedentary. Their traditional farming system was undermined in the nineteenth century by the usurping of their water supply by growth in the Anglo population in Arizona and increased competition from mechanized farming in the region (Smith-Morris 2006). In the wake of these trends, the Pima became

more reliant on government commodities and other processed foods, including eggs, bacon, potatoes, lard, cheese, beans, canned meat, dry cereals, and dried or canned milk, along with some fruits and vegetables (Smith-Morris 2006). More recently, frybread has become an important food, with religious and social significance; it, along with other fried or sweet foods, is central to culturally valued forms of generosity and hospitality.

Carolyn Smith-Morris, who conducted extensive ethnographic work on diabetes among the Pima, argues that effective prevention and treatment of diabetes among the Pima must incorporate political–economic, cultural, and genetic and biological factors; they must "fit" the current conditions of Pima life but also address the structural problems that underlie these conditions and give rise to high rates of diabetes and its negative health consequences (Smith-Morris 2004). Although genetic factors may indeed contribute to greater vulnerability to diabetes among the Pima, it must be emphasized that the major proximate contributors to NIDDM risk derive from their diet and activity patterns. As Schulz and colleagues concluded,

> The low prevalence of type 2 diabetes and obesity in the Pima Indians in Mexico in a more traditional rural environment contrasts sharply with that in the U.S. Pima population living in a Westernized environment. The difference in diabetes prevalence in these populations is mirrored by the differences in physical activity and obesity. The findings indicate that, even in a genetically highly susceptible population, type 2 diabetes is not inevitable and is preventable in environments that promote low levels of obesity and high levels of physical activity. (Schulz et al. 2006, 1870)

In contrast, Europeans appear to have lower rates than other populations. John Allen and Susan Cheer have argued that the abundance of milk in the diets of northern Europeans made "thrifty genes" deleterious (Allen and Cheer 1996). This is because milk stimulates a larger than expected rise in blood insulin levels after it is consumed. Thus, individuals who had a thrifty genotype and who drank a lot of milk would have been likely to develop diabetes early in life and likely suffered higher rates of mortality at an earlier age as well. As a consequence, thrifty genes may have been eliminated from the population. Allen and Cheer also pointed out that Neel's assumption that hunter-gatherers were markedly more prone to food scarcity than agriculturalists was difficult to support, requiring a different explanation for the evolution of thrifty genes.

More recently, a new hypothesis has been put forth to explain the high frequency of diabetes among populations transitioning from more traditional diets to a more "Western-style" diet. The Pima and other Native American groups fall into this category, and rates of diabetes are now rising most rapidly among populations in transitional economies such as India (Yajnik 2004). The "thrifty phenotype" or "developmental origins of health and disease" hypothesis posits that exposure to nutrient scarcity during the fetal and infant stages of development results in a kind of metabolic programming for thriftiness (Barker and Thornburg 2013; Yajnik 2014; Gluckman and Hanson 2006; Benyshek 2013; see Figure 4.6). Development gets channeled in different directions depending on the environment in which it

Preliminary

Remove Watermark Now

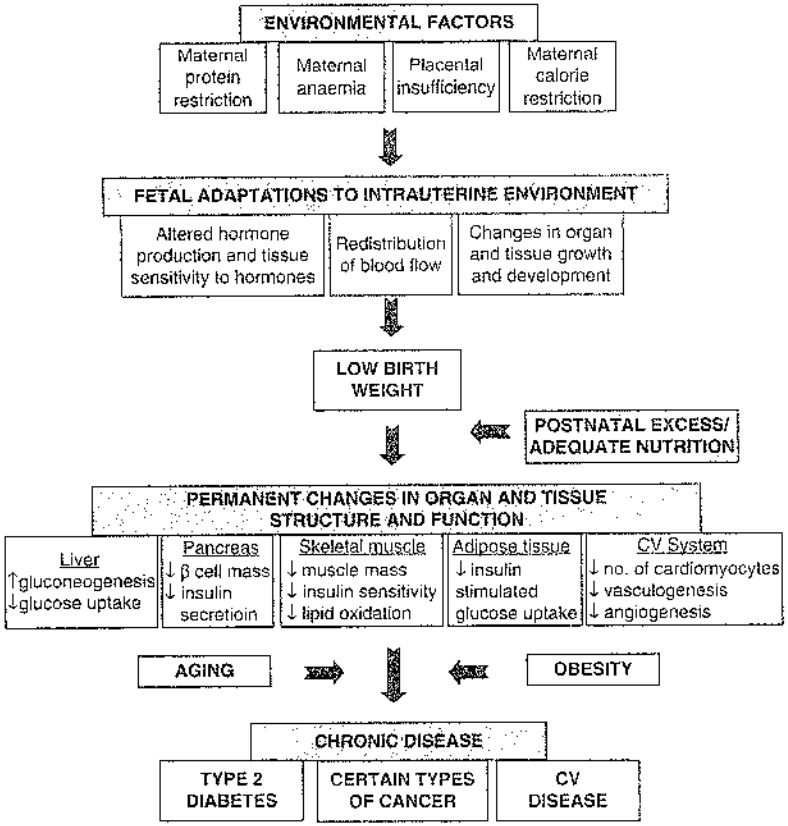


Figure 4.6
A schematic representation of the relationship between prenatal environments and later-life chronic disease.
From M. S. Martin-Gronert and S. E. Ozanne. 2010. Mechanisms linking suboptimal early nutrition and increased risk of type 2 diabetes and obesity. *J. Nutr.* 140: 662–666. Reprinted with permission from American Society for Nutrition.

occurs (as a form of developmental adaptation). If the environment is characterized by nutrient scarcity, the developing body makes a "prediction" that future conditions will be similar and alters its trajectory accordingly by developing more thrifty mechanisms for nutrient uptake, retention, and metabolism. However, if during childhood or adulthood resources become abundant for a sustained period of time, those efficient metabolic adaptations serve to increase the risk of diabetes. Numerous studies show that low birth weight, as a measure of poor fetal growth, is a strong predictor of later life (including later childhood) risk of developing CVD, obesity, and diabetes in populations where children and adults have ample access to calories (Barker and Thornburg 2013; Yajnik 2014). Both the thrifty genotype

and the thrifty phenotype models make use of the same concept of mismatch or discordance between the environment the body was "expecting" (i.e., one characterized by some degree of resource scarcity) and that in which the individual lives (i.e., one of resource abundance) (Gluckman and Hanson 2006).

Although there appears to be population variation in susceptibility to NIDDM that may be genetically or developmentally based (although no gene associated with vulnerability to type 2 diabetes has been located and found to be variable across populations in ways that correlate with diabetes prevalence), the disease itself will not manifest except in the context of exposure to a hyperabundance of energy-rich foods and dramatic reductions in energy expenditure. The Pima study and the dramatic rise of diabetes in India are clear demonstrations of this. Thus, any attempt to reduce the risk of diabetes must first concentrate on the environmental conditions that trigger it and the obesity that is often correlated with it. This is a difficult situation because currently a biological predisposition for sweet, fatty foods is aligned with ubiquitous, heavily advertised, and relatively cheap sweet fatty foods supported by political and economic interests (lobbying groups, industry involvement in setting dietary guidelines, reduced federal and state funding for educational programs that force schools into alliances with food corporations, etc.) and the rise of a sedentary lifestyle with few safe, accessible opportunities for routine vigorous activity. These contribute to the high rates of chronic disease in general and NIDDM in particular. These conditions are not evenly distributed across societies, with poor and minority groups disproportionately vulnerable.

Sugar and High-Fructose Corn Syrup in Biocultural Perspective

Sugar would have been rare in hunter-gatherer diets (see Table 4.3) because it is only found in high quantities in fruits, grains, and tubers such as potatoes. Because of its relative scarcity and its importance as the primary source of energy for nerve cells, humans, along with many other mammals, show a decided preference for sweetness. That would have served hunter-gatherers well as a motivation to seek out sweet-tasting, sugar-rich foods. This ancestral preference can now be satisfied easily by populations throughout the world with access to commercial foods by any number of cheap, accessible sugar-rich foods, from sugar-laden cereals ("kid's cereals"), cookies, and candy to any carbohydrate-rich food such as pasta or bread. Sidney Mintz (1985) elegantly traced the political–economic history of sugar, which became the first truly global and commercially produced foodstuff. Sugar cane (the main source of pure sugar: sucrose) was originally domesticated in the Middle East and was a rare and expensive commodity. However, during the colonial period, European powers such as the British

(*continued*)

Preferred Proof

Remove Watermark Now

(*continued*)

found that sugar was amenable to large-scale production on the plantations of the tropical New World, where it could be produced cheaply by slaves in large quantities. It was then shipped to Europe, where it served as an affordable energy source (along with nonnutritive stimulants such as coffee, tea, and tobacco) for the new urban factory workers of the industrial revolution. Sugar became a ubiquitous part of European diets, added to tea or coffee, converted to alcoholic beverages such as rum, added to fruit to make preserves, and various other preparations. Sugar was among the first commodities to be widely advertised as a way of encouraging consumption of this previously rare and expensive product.

Many of the foods (especially cookies, crackers, cereals, prepared foods, soda, fruit drinks, sports drinks, and jams, among others) found in grocery stores today are made not with sugar, but with high-fructose corn syrup. This is a derivative of corn and is relatively inexpensive to produce, transport, and utilize in processed foods (especially drinks) compared with sugar cane. This is especially true in the United States, where corn is cheap and produced in vast amounts (Pollan 2006). High-fructose corn syrup intake has increased more than 1,000-fold since 1970 and now makes up more than 40 percent of added sweeteners in foods (Bray, Nielsen, and Popkin 2004). Fructose does not generate an insulin spike like glucose does, but promotes insulin resistance and abdominal fat deposition (Stanhope and Havel 2009; Dornas et al. 2015). It also does not stimulate the production of the hormone leptin, which, like insulin, promotes feelings of satiety (Dornas et al. 2015). Furthermore, calories from drinks do not promote the same feeling of satiety as do those from solid foods (Bray, Nielsen, and Popkin 2004). There is currently vigorous discussion of the specific contributions of soda consumption to the obesity epidemic, a topic taken up by Marion Nestle in her new book *Soda Politics* (Nestle 2015), but there is a clear positive correlation across countries between soda consumption and prevalence of obesity across countries (Basu et al. 2013).

Lactose Intolerance

Another useful example that illustrates variation in historical exposure to a food is lactose intolerance. **Lactose intolerance** occurs in some individuals when they drink milk and is characterized by nausea, cramping, gas, and diarrhea. This is most common among individuals of sub-Saharan African or Southeast and East Asian origin, Native Americans, South Pacific Islanders, and Native Australian populations. It is relatively rare among Europeans, some populations of Central or South Asia, and some herding populations of sub-Saharan Africa such as the Fulani, Hausa, Turkana, and Maasai, among others (Itan et al. 2010).

Lactose intolerance results from incomplete digestion of the milk sugar lactose. Lactose is a disaccharide (a double sugar) made up of glucose and galactose. The bond between these sugars must be cleaved before these simple sugars can be absorbed in the small intestine. The enzyme that does this is called lactase. Lactase is produced by most juvenile mammals, and it breaks down the sugar in their food source: mother's milk. However, baby mammals eventually get weaned and then they never consume milk again. The gene that manufactures lactase is shut off, and the adult mammal no longer produces this enzyme. The regulation of lactase activity appears to be genetic; an area of DNA just upstream from the lactase gene itself appears to be the "switch" that turns on or shuts off lactase production. Importantly, the "shutting off" of lactase production occurs independent of the presence of milk in the diet; that is, it cannot be kept on simply by continuing to consume milk (Sahi 1994). In general, there tends to be a close temporal correspondence between the decline in lactase production and age at weaning across mammalian species.

A small number of human populations are exceptions to the basic mammalian pattern in that they continue to produce lactase in adulthood (i.e., they are **lactase persistent**) and hence relatively few individuals in these populations suffer from lactose intolerance when they consume milk. Why do populations vary in this way? One attribute of all populations with high rates of lactase persistence is a history of keeping domestic animals such as cows, water buffalo, goats, or sheep for their milk. This would suggest that they have adapted to exploit this new dietary resource by keeping the lactase gene on throughout life. Individuals that could make use of milk without suffering ill effects would have access to a rich new source of nutrients. Thus, individuals with a mutation that kept lactase on would have had a nutritional advantage and the mutation would have spread. However, this is not the whole story because it appears that some populations that keep dairy animals do *not* have high frequencies of lactase persistence. It turns out that these groups tend to use processed dairy products exclusively and do not consume fresh milk. Importantly, lactose is found in the highest quantities in fresh milk; the conversion of milk into yogurt or cheese removes much if not all of the lactose, depending on the preparation method. If only these dairy products are consumed, individuals are not being exposed to lactose, yet they can access the other nutrients in milk.

According to Bill Durham (1991), there are two clusters of populations with high rates of lactase persistence: those living at high latitudes (such as northern Europeans) and those living in arid or semiarid environments of the more tropical latitudes where they graze their animals. For northern Europeans, fresh milk may have provided an additional advantage over dairy products: The lactose in milk can substitute for vitamin D in its role in facilitating calcium absorption in the small intestine. Recall that vitamin D can be produced by exposure of the skin to ultraviolet light, but this exposure is dramatically limited among high-latitude populations, where it is also cold (and so people cover up their skin to stay warm) and often cloudy. Drinking fresh milk in this context would have been advantageous as a way of avoiding vitamin D deficiency and its manifestation as rickets—weight-bearing bones that are soft and breakable because of the lack of calcium being deposited in them by vitamin D. Among other low-latitude herding populations, fresh milk would have been useful as a source of water in arid environments and a way to include sugar (lactose) in a diet that is high in animal products but low in plant foods. Thus, only in the presence of these additional environmental conditions was lactase persistence advantageous.

Preview Not for Retail Sale

Remove Watermark Now

Although most humans are lactase impersistent and are at high risk of lactose intolerance if they consume milk, milk is included as a component of dietary guidelines in countries such as the United States, those in Europe, Australia, and New Zealand, and increasingly others throughout the world (Wiley 2016). In the United States, two to three servings of milk or other dairy products are considered necessary parts of a healthy diet. But what about the 30 million to 50 million Americans who are lactase impersistent? If they follow these guidelines, they may suffer from lactose intolerance. This begs the question as to the rationale for including dairy products in the dietary guidelines (Wiley 2004b, 2016). Most frequently, the calcium in milk is invoked as the primary reason for this, and Americans have been found to consume well less than the recommended daily allowance. Given the importance of milk production to the American agricultural economy, it comes as no surprise that its consumption should be encouraged (Wiley 2016). Some researchers have gone as far as to suggest that the higher rates of many chronic diseases found among minority populations are attributable to their low rates of milk intake (Jarvis and Miller 2002; Bailey et al. 2013). Their high rates of "perceived" lactose intolerance contribute to low milk consumption and are thus considered an important "barrier" to good health. Suffice it to say that health disparities cannot be explained away as a consequence of low rates of milk consumption.

Most important, this example illustrates the dangers inherent to ideas about what constitutes medical or biological "normalcy." Until the 1960s, most European and American scientists assumed that lactase persistence was the norm for *H. sapiens* and that those who developed gastrointestinal symptoms when they drank milk had some sort of pathology. After all, the populations that these scientists came from were largely lactase persistent, and milk and dairy products were important parts of the diet. Although it is now widely recognized that lactase impersistence is the norm among humans, it is still labeled "abnormal" (Wiley 2016). For example, the American Academy of Pediatrics uses the language "*lactose malabsorption*," but in the discussion of prevalence, it refers to *lactase deficiency*:

> Approximately 70% of the world's population has primary lactase *deficiency*. The percentage varies according to ethnicity and is related to the use of dairy products in the diet, resulting in genetic selection of individuals with the ability to digest lactose. In populations with a predominance of dairy foods in the diet, particularly northern European people, as few as 2% of the population has primary lactase deficiency. In contrast, the prevalence of primary lactase deficiency is 50% to 80% in Hispanic people, 60% to 80% in black and Ashkenazi Jewish people, and almost 100% in Asian and American Indian people. (Heyman and the Committee on Nutrition 2006)

Likewise, lactose intolerance is described by the American Gastroenterological Association as "a *shortage* of the enzyme lactase, which is *normally* produced by the cells that line the small intestine. . . . For most people, lactase *deficiency* is a condition that develops naturally, over time" (American Gastroenterology Association 2013, emphasis added). Writing in the *American Academy of Family Physicians* journal, Swagerty, Walling, and Klein state that "lactose *malabsorption* is a normal physiologic pattern" and that there is "speculation that *lactase deficiency* is the 'normal' or 'natural' state, and the persistence of significant lactase activity into adult life in northern European populations is an 'abnormal' mutation" (Swagerty, Walling, and Klein 2002, 1845–1846, emphasis added). Thus, although it is generally acknowledged that lactase impersistence is common, it is still seen as a medical condition characterized by a "lack" of lactase production. The assumption that European physiology is the healthy norm for the species remains widespread. This also plays out in donations of milk to populations experiencing food scarcity. The anthropologist Marvin Harris described his experience in Brazil in the early 1960s when the United States sent some 88 million pounds of powdered milk to Brazil as part of a Food for Peace program. Many Brazilians complained that the milk gave them upset stomachs and diarrhea, much to the disbelief of American officials, who suggested that they must have been preparing it improperly. Within the United States, surplus powdered milk was distributed to impoverished citizens as part of poverty-relief programs. Native Americans and African Americans were primary recipients, and frequent gastrointestinal complaints were noted to local clinicians (Harris 1985).

Celiac Disease

One last example of human biological variation that is related to diet and often manifests clinically is **celiac disease** (CD), also called celiac sprue, or gluten-sensitive enteropathy. CD is an immunologically mediated sensitivity to gluten, a protein found in wheat, rye, and barley. When individuals with CD consume these foods, their immune system responds by damaging or destroying the villi, the cells that line the small intestine and that are responsible for the absorption of nutrients. CD is considered an autoimmune disorder, meaning that gluten stimulates the immune system to attack the body's own cells, in this case the villi of the small intestine. The resulting symptoms include those associated with malnutrition, as the villi become inefficient at absorbing nutrients, but also gastrointestinal pain and discomfort, dermatitis, and irritability. In addition to CD, individuals may have an allergic reaction to gluten or other wheat proteins, and there is another category of "gluten sensitivity" that has no clear biomarkers, but that is used when individuals suffer a variety of symptoms that resolve when gluten or cereals containing gluten are removed from the diet (Fasano et al. 2015).

The prevalence of CD varies among the world's populations, with the highest prevalence in North Africa and northern Europe and an overall prevalence of 0.9 percent (Lionetti et al. 2015). In the United States, the prevalence has doubled every 15 years since 1974 for reasons that are not understood (Catassi et al. 2010). Global estimates are not available, but CD is—not surprisingly—more common in populations that consume wheat (those in Europe, North and South America, and the Middle East; Lionetti et al. 2015). What is interesting to note is that CD, like lactose intolerance and milk, will not manifest unless gluten is in the diet—that is, if the diet does not contain wheat, rye, or barley. Thus, it was unlikely to exist in populations relying on traditional staple foods such as rice (such as in Southeast or East Asia) or corn (among Native North and South American populations). It was also unlikely to have been a problem for hunter-gatherer *H. sapiens*. However, in what has been dubbed the "evolutionary paradox of CD," CD

Remove Watermark Now

prevalence and the underlying genetic correlates of the disease are more common among populations with longstanding histories of wheat production and consumption. It is unclear why the alleles associated with CD have not been eliminated from populations with long term usage of wheat. Furthermore, the consumption wheat in its various preparations such as bread, pasta, and other baked goods has increased markedly throughout the world, and rates of CD are escalating (Malekzadeh, Sachdev, and Fahid Ali 2005).

Shirley Lindenbaum has described how wheat became an important part of the diet in Bangladesh, where rice had traditionally been the staple food. Indeed, the question "Have you had a meal?" translates as "Have you eaten rice?" (Lindenbaum 1987). Wheat production escalated in the 1970s and as foreign countries provided wheat as part of their foreign aid to Bangladesh. The United States in particular became a source of wheat, which became a part of a complex "gastropolitics," with the United States cutting off aid during the 1974 famine until Bangladesh agreed to stop exporting jute to Cuba. The net effect was that Bangladeshis were importing, producing, and consuming more wheat. However, wheat grows best there in the winter and has replaced the more proteinaceous crop of lentils, thus increasing rates of malnutrition. It also requires more work and during the hottest months of the year (unlike rice, which is harvested during the cooler months). Wheat is working its way into the Bangladeshi diet, particularly among the urban middle classes, who see it as a more "progressive" or "Western" food. Although it is not clear whether rates of CD have risen in Bangladesh as a result, the disease is now widely recognized in South Asia, and in all likelihood rates will rise in populations now including more wheat or processed foods containing gluten in their diets.

Because the symptoms of CD or gluten sensitivity are similar to those of other gastrointestinal tract disorders such as irritable bowel syndrome or may present with more generalized symptoms, individuals who suffer from CD may find the path to official diagnosis long and difficult (Copelton and Valle 2009) and the disease is considered widely underdiagnosed (Lionetti et al. 2015). Thus, they often turn to self-diagnosis by either eliminating gluten from their diets and/or paying for stool testing via direct-access testing (when patients send samples for a laboratory to analyze, without a clinician's orders). As Copelton and Valle note,

> Celiac's permissive symptomology, its non-medical remedy, and the difficulty patients experience when trying to gain a medical diagnosis, support self-diagnosis. Self-diagnosed celiacs adopt their own subjective diagnostic standard in place of the biopsy, the objective medical gold standard. . . . Given the ubiquity of gluten in the average American diet, the social nature of meals, and the close scrutiny of ingredients and preparation techniques necessary to maintain a gluten-free diet, some self-diagnosed persons continue to seek medical legitimation for their diagnosis to garner social support from friends, family, and coworkers. (Copelton and Valle 2009, 629)

The difficulty of adhering to a gluten-free diet in the United States or Europe should not be underestimated given how widespread wheat is in typical foods and the high cost of gluten-free products, and individuals following a gluten-free diet are often met with skepticism that such extreme measures are necessary. A similar

situation exists for food allergies, which also appear to be on the rise. A global survey of food allergies found rates varying from 1 to 2 percent in Thailand to 10 percent in Australia (Ashley et al. 2015). In 2007 a national survey indicated that about 4 percent of all U.S. children under the age of 18 reported food allergies, an 18 percent increase from 10 years prior (Branum and Lukacs 2009), although a more recent survey of about 40,000 children found a rate of 8 percent (Gupta et al. 2011). An upward trend has been found in other industrialized countries. Most common were peanut, milk, and shellfish allergies, many of which were described as severe. Rates are higher among young children, who often outgrow allergies as they age. Children born in the winter in northern U.S. cities have higher allergy rates than those born in the summer, and it has been suggested that vitamin D (recall that vitamin D is synthesized in the presence of ultraviolet light, which is insufficient during winter months in such areas) may play a role in this finding (Vassallo and Camargo 2010). Food allergies act through a different immunological mechanism than autoimmune disorders like CD, and symptoms range from mild tingling of the mouth and tongue or hives (rash) to anaphylaxis, which can be fatal.

In a systematic review of the literature, Chafen and colleagues (2010) found that there was no uniform definition, diagnostic standard, therapeutic course, or strategy for prevention of food allergies. They argued that food allergies are likely overreported and individuals unnecessarily restrict their diets as a result, while at the same time, the ambiguity surrounding their diagnosis "obscures the substantial morbidity caused in patients truly affected by immune-mediated food allergy and serves to perpetuate some public misperceptions that food allergy is a trivial medical condition" (Chafen et al. 2010, 1854). Individuals with allergies or their caretakers suffer from a variety of psychosocial stresses that negatively impact their quality of life, especially when the condition is trivialized or challenged by peers (Cummings et al. 2010).

A common theme emerges in the discussion of food intolerances, allergies, or autoimmune disorders such as CD: clinicians—and often peers—are skeptical of individuals' needs for dietary restriction. Why should this group of syndromes be singled out? In some ways they are not—other illnesses such as chronic fatigue syndrome, irritable bowel syndrome, or fibromyalgia are often treated with similar disdain, and individuals' suffering is not legitimated (that is, they are not allowed to take on the sick role). All have diverse symptoms including chronic malaise that overlap with multiple other disease states. There is a lack of clear diagnostic procedures and/or treatments, and these syndromes are often attributed to psychological stress by clinicians (Nelson and Ogden 2008). What makes food-related syndromes even more complicated is the social nature of eating. To reject food is to reject conviviality and engagement with others in routine social activity, and ostracism or outright challenges with the contested foods can result (Lieberman et al. 2010).

But food-related disorders also offer an option not available to those suffering from other kinds of diseases: the ability to self-treat by modifying one's own diet. Such "treatment" does not require medical authorization. That said, to be eligible for insurance, special government-funded meal plans, or exemptions from work, a medical diagnosis is usually required, and general practitioners find themselves having to negotiate their skepticism with patients' perceptions of food intolerance.

Do not distribute

Remove Watermark Now

As Mia Nelson and Jane Ogden report from their interviews with general practitioners, the negotiation and arrival at common ground did not involve the GPs' acceptance of the whole of the patients' belief systems or of food intolerance itself, but rather acceptance of the reality of the patients' distress and desire for relief, and acceptance that food intolerance is an uncertain diagnosis rather that an illegitimate one. This "accepting approach" was reported as being reinforced by the GPs impression that, for whatever reason—placebo effect, secondary gain or direct biophysical mechanism—many patients felt better for acting on their belief in food intolerance and for excluding foods from their diet. (Nelson and Ogden 2008, 1044)

Conclusion

This chapter has focused on basic nutritional requirements, as well as the links between nutrition and health. It has also outlined the ways in which there exists population variation in the risk of these diseases and how an evolutionary perspective can inform the understanding of why these differences occur. Although a wide range of diets can be used to meet our nutritional needs, some are clearly healthier than others. An abundance of calories combined with reduced activity are evolutionarily anomalous, but with increasing globalization of Western diets, occupations, and lifestyles, this package is becoming more common. Along with it comes a rise in chronic disease, especially CVD, stroke, and diabetes. It is notable that CVD and stroke are now the most common causes of death in the world, accounting for more than 25 percent of all deaths. Diabetes is now the seventh most common cause of death, although it also contributes to CVD deaths (http://www.who.int/mediacentre/factsheets/fs310/en/). All of these diseases have increased as a percentage of all deaths since 2000.

It is also important to recognize that the health consequences of a particular diet occur over the long term; especially when considering chronic disease, it is the cumulative effects of years of consumption and activity patterns, in combination with genetic inheritance and the conditions in utero, that ultimately impact disease risk. Because chronic diseases do not emerge overnight, the links to diet are necessarily indirect and complicated by numerous other factors. Making healthy "choices" about what to eat is not simple. Our evolved biology, with its preference for energy- and salt-rich foods, in combination with a dietary environment characterized by the ubiquitous presence and inexpensiveness of these foods in supermarkets, restaurants, and government surplus commodity programs, along with advertisements on television among myriad other places, makes it virtually impossible to do so. It is this dietary environment that has "gone global" and the negative health consequences of it have also achieved a global distribution. Whether this ultimately reverses a recent history of increases in life expectancy remains to be seen, but it is also the case that these chronic diseases are on the rise in the same places where undernutrition and infectious disease are also still common. The latter two conditions are particularly potent sources of child ill health and poor growth, which will be considered in greater detail in the next chapter.

Preview Not Available

Study Questions

1. Why do we say that there is a mismatch between our bodies and our contemporary environments, at least in terms of diet and nutrition?
2. What are the basic macronutrient and micronutrient requirements of the human diet?
3. How does the human digestive system reflect the fact that humans are omnivorous?
4. In general terms, how do the diets of agriculturalists differ from those of hunter-gatherers?
5. What are some of the problems associated with the assessment of the nutritional health of past populations based on bioarchaeological data?
6. Why do people living on hunter-gatherer-type diets have less risk of developing chronic disease than those who have a more "modern" diet?
7. What are phytochemicals and how might they be important in the relationship between diet and health?
8. What are some of the underlying biocultural causes of the worldwide "obesity epidemic"? In what ways does the epidemic vary among the world's populations? How does obesity affect mortality?
9. What are the two primary types of diabetes? What are some of the hypotheses offered to explain the population distribution of NIDDM?
10. What is lactase persistence? How does the biocultural distribution of lactase persistence inform biomedical conceptions of biological normalcy?
11. Why are food intolerances often greeted with skepticism? What factors might be responsible for these?

Critical Thinking Questions

1. Consider a cheeseburger and its component parts. Evaluate them in a biocultural and evolutionary context regarding their real and potential impact on human health and disease.
2. All studies indicate that obesity is a public health problem that will only increase in severity in the coming decades. What should we, as a society, do about this? Do you agree that it really is a problem that must be fixed?
3. What are the most important factors in your own food choices? Do you feel that you could make healthier food choices? Does the biocultural perspective help you understand the food choices you make?

Suggested Ethnographies to Read with This Chapter

Alexandra A. Brewis. 2011. *Obesity: Cultural and Biocultural Perspectives.* New Brunswick, N.J: Rutgers University Press.

Frederick Errington, Deborah Gewertz, and Tatsuro Fujikura. 2013. *The Noodle Narratives: The Global Rise of an Industrial Food into the Twenty-first Century.* Berkeley: University of California Press.