JOURNAL OF
ANTHROPOLOGICAL RESEARCH

(Formerly Southwestern Journal of Anthropology)

VOLUME 66 • NUMBER 2 • SUMMER • 2010

THE OMNIVORE'S DILEMMA
The Evolution of the Brain and the Determinants of Food Choice

George J. Armelagos

Goodrich C. White Professor of Anthropology, Emory University,
Atlanta, GA 30320 USA. Email: antga@emory.edu

KEY WORDS: Evolution of the brain, Food choice, Industrial food system, Paleolithic diet

More than 72 million Americans, over a third of the population, are obese. In the past three decades, the rates of obesity in adults have doubled, and rates in children have tripled. Obesity rates have markedly increased among all segments of society, including those defined by age, sex, race, ethnicity, socioeconomic status, education level, and geographic region. Michael Pollan (2006) argues that the obesity crisis is due to the abundance of foods now available to satisfy the omnivore's dilemma, the desire for dietary variety required to meet energy requirements paired with the often fearful and perilous search for new foods. The abundance of food is an important factor in the obesity problem, but the solution to this perplexing riddle is more complex and is buried in our evolutionary history. A biocultural perspective, which highlights coevolutionary processes, is most useful for understanding humans' dietary and nutritional adaptation to changing social and physical environments. In our early evolution, the evolving body—with an expanding brain, lengthening small intestine, and shrinking large intestine—required nutritionally dense foods. Our current pattern of eating reflects the way in which Homo sapiens *evolved and resolved the omnivore's dilemma. The resolution of the omnivore's dilemma lies in the development of cuisine to mediate this biological conflict. Cuisine defines which items found in nature are edible, how these substances are processed into food, how the foods are flavored, how and with whom we eat, and the rules of eating—the code of etiquette.*

With the transition to primary food production during the Neolithic, the variety of foods dramatically decreased. The need for variety was met by creatively experimenting with food preparation, despite the availability of limited ingredients. The shift toward large-scale agriculture in the past century led to an

overall decline in human nutrition by reducing dietary breadth. More recently, industrialization of the food system has made an overwhelming abundance of inexpensive, high-density foods (sugar and fats) available to populations in some areas of the world. The disjunction between the small amount of physical energy they expend to obtain significant numbers of calories has created the modern obesity epidemic.

> "When you wake up in the morning, Pooh," said Piglet at last, "what's the first thing you say to yourself?"
> "What's for breakfast?" said Pooh. "What do you say, Piglet?"
> "I say, I wonder what's going to happen exciting today?" said Piglet.
> Pooh nodded thoughtfully.
> "It's the same thing," he said.
>
> —A. A. Milne, *Winnie-the-Pooh*

OMNIVORE'S DILEMMA REVISITED

Michael Pollan (2006; Chevat and Pollan 2009) reintroduced, reinterpreted, and popularized the concept of the omnivore's dilemma for a new audience of children and adults. Pollan suggests that "a national eating disorder" has been perpetuated by the omnivore's dilemma—the conflict generated by humans' desire for the dietary variety needed to meet energy requirements combined with the often fearful and perilous search for new foods. In Pollan's version of the omnivore's dilemma, an abundance of food exacerbates the generally poor food choices we make. Pollan illustrates the dilemma by focusing on the decisions one makes on a daily or weekly basis at the supermarket. Choices there involve deciding between an organic apple, for example, or a traditional apple. To understand the dilemma, Pollan decides to "go to the beginning," to follow the food chain—a journey that highlights the role of the modern industrial food system in creating the perplexing abundance found at the supermarket. To this end, he follows three food chains: the industrial, the organic, and that of the hunter-gatherer. The last of his nutritional trips takes him to his own "hunting ground" in California, armed with a GPS to aid his search for wild mushrooms and meat for the table. During the journey, he uses a high-powered rifle to down a wild boar, which he prepares along with other foods he had foraged.

In a sense, just as Pollan trivializes the antiquity of hunting and gathering with his high-technology tools, he trivializes the omnivore's dilemma, which is generally a choice of what is and what is not edible when the selection of one item over another can have deadly consequences. In contrast, for Pollan, everything in the supermarket is edible, though it may carry attendant risks of increased mortality later in life. For the most part, the modern food shopper is little concerned with the immediate mortality risks of eating a fat-marbled steak or cholesterol-laden fried shrimp; neither threatens impending death, only an increased waistline.

The omnivore's dilemma in its original context represents a serious predicament for omnivores. It references the conflict in which organisms have a need to increase the variety of foods to meet their nutritional requirements (neophilia) in the context of fear that the novel substance they are about to eat may be deadly (neophobia). Although Pollan is correct in focusing on the abundance of food as a key element in the national eating disorder, he does not consider the roots of the problem. In this essay, I provide a discussion of the origin of the omnivore's dilemma, the role that it plays in the development of human food choices, and how it impacts food choice in an environment of abundance. To elucidate these complex issues, I employ a biocultural approach, which considers the contribution of both biological and cultural processes. One of the keys to dealing with the contemporary food crisis is an understanding of how the abundance of food has become a component of the environment for many human populations.

BIOCULTURAL ANTHROPOLOGY

The biocultural approach in anthropology (Durham 1990, 1991; Goodman and Leatherman 1998, eds. 1998), which originated more than a half century ago, has been an integrative force in the discipline (Armelagos 2008). At its core, the biocultural perspective focuses on the coevolution of cultural and biological features. Such studies have had an impact on bioarcheology (Armelagos 2003; Zuckerman and Armelagos 2010), medical anthropology (Armelagos et al. 1992; McElroy and Townsend 2009), and work on the evolution of diet (Armelagos 1987; Harris 1987; Schutkowski 2008; Turner et al. 2008), the origins of obesity (Brown 1991; Brown and Krick 2001), the emergence of disease (Barrett et al. 1998), and the impacts of social inequality on the health of past populations (Armelagos, Brown, and Turner 2005; Ortner and Schutkowski 2008).

Although challenges to the use of the approach in anthropology have arisen (e.g., Segal and Yanagisako, eds. 2005), biocultural anthropology has become embedded in the discipline and persists as a powerful synthesizing force. Segal and Yanagisako (2005) claim that the biocultural approach is reductionistic and privileges a biological examination at the expense of cultural interpretations, inhibiting its development as a perspective for understanding the social system. But Segal and Yanagisako have conflated the biocultural perspective by claiming that it offers only a narrow sociobiological interpretation (Armelagos 2008), which is a misrepresentation of the approach. The evolution of human diet, the topic of this essay, is particularly amenable to biocultural interpretations (Turner et al. 2008).

The coevolution of biological and cultural processes (Aoki 2001; Durham 1991) has shaped the evolution of diet and nutrition of human populations. Cultural factors have influenced the foods that we eat, and their consumption has biological consequences. For example, lactose intolerance, or the inability to digest lactose, a type of sugar found in milk and other dairy products (Beja-Pereira et al. 2003; Ingram et al. 2009; Tishkoff et al. 2007), is the result of a suite of coevolutionary adaptive responses. Individuals who are lactose intolerant lack lactase, the enzyme that is essential for hydrolyzing disaccharide lactose into galactose and glucose. This genetic condition has been considered a disease because those who lack the enzyme and consume milk experience digestive upset, such as gas, bloating, and diarrhea. The gas in the large intestine is produced when

E. coli digest the lactose (Weiss 2004). Nearly 70% of the world's population is lactose-intolerant. The distribution of this trait reveals that absence of the lactase enzyme and the resulting lactose intolerance are responses triggered in humans at the time of weaning from breast-feeding in societies where adults do not drink milk. Selection for the persistence of lactase only occurs in populations where milk from domesticated animals is consumed after weaning.

Geographers (e.g., Simons 1969, 1970) and anthropologists (Durham 1991; McCracken 1971) have long speculated about the coevolutionary relationship between milk-producing domestic animals and the persistence of lactase in modern human populations. Recently, definitive evidence has emerged. Beja-Pereira and colleagues (2003) have demonstrated a geographic concordance between the six most important milk proteins found in 70 native European cattle breeds and lactose tolerance in contemporary Europeans. Furthermore, the distribution of Neolithic archaeological sites with evidence of European cattle domestication, dating to around 5,000 years ago (Zvelebil 2000), corresponds to the area of the highest frequencies of lactase persistence.

Weiss (2004) and Swallow (2003) note that the distribution of lactose intolerance is highly variable. In a study of 270 indigenous populations from Europe and Africa, Bloom and Sherman (2005) found that lactose intolerance was associated with the extreme climates characteristic of both high and low latitudes. In the lactase-persistent northern latitudes, milk provides a source of calcium in an environment where the level of ultraviolet light limits the production of vitamin D, which is essential for bone growth. Interestingly, in the same areas where it has proven impossible or dangerous to herd cattle because of the presence of deadly infectious cattle diseases, lactose intolerance predominates (Bloom and Sherman 2005).

The lactase gene (LCT), which allows the persistence of the lactase enzyme, is a recent mutation relative to the scope of human evolutionary history. The gene appears to have undergone rapid selection only after the domestication of cattle. Lactose tolerance apparently developed in European and East African populations during the domestication event for cattle (Myles et al. 2005) and arose independently in Arab populations (Enattah et al. 2008). Swallow (2003:201) notes that some horse herders from Inner Mongolia, who have rates of 87.9% lactose intolerance, process mare's milk by fermentation, thus reducing the lactose levels so it can be consumed without any alimentary distress (Yongfa et al. 1984).

THE OMNIVORE'S DILEMMA AND THE ORIGIN OF CUISINE

The invention of cuisine, which mediates the omnivore's dilemma, was a defining event in human evolution. Cuisine is a cultural system that defines the items in nature that are edible; how these items can be extracted, eaten, or processed into food; the flavors used to enhance the taste of the food; and the rules about consuming it (Rozin 1982). Cuisine arose as the solution to the omnivore's dilemma, and it remains an essential feature of human dietary adaptation.

For Pollan, the danger in foods is the long-term health consequences from the overconsumption of sugars, fats, and salt. Although the foods available in

American supermarkets are thought by most to be safe, there are hidden dangers in the industrial food system—and in the supermarket aisles—that Pollan does not discuss in detail. These dangers are not hidden in the sense that they are well-documented and might kill you in 20 years, but rather in the sense that they are invisible but might kill you or seriously sicken you immediately. With conglomerates such as American Food Services grinding 365 million pounds of meat a year (more than 165,000 metric tons), and with the trimmings that are used to make the product coming from many sources from within the United States and internationally, the potential for food-borne diseases is great (Moss 2009). Paul Mead and colleagues (1999) estimate that food-borne diseases cause approximately 76 million illnesses, 325,000 hospitalizations, and 5,000 deaths in the United States each year. Of these illnesses, known pathogens account for an estimated 14 million illnesses, 60,000 hospitalizations, and 1,800 deaths. A study of more than 4,000 ground beef samples found a 4.2% prevalence of *Salmonella* contamination (Bosilevac et al. 2009). The globalization of the food network has increased the immediate threat of contamination by toxins (Ingelfinger 2008), viruses (Verhoef et al. 2009), and bacteria (Moran, Scates, and Madden 2009). Even the international market in produce has become a serious to health issue, as vegetables have become contaminated with *E. coli* O157:H7 and *Salmonella enterica* (Franz and van Bruggen 2008).

Most contemporary shoppers (63%) go to a supermarket without any concern for food safety (NPD Group 2009). The nonhuman primate, on the other hand, faces an uncertain food choice. The omnivore's dilemma for our primate cousins is informative; the rain forest environment may seem on the surface to provide an unlimited source of food for a primate that has the ability to digest secondary compounds as well as large amounts of fiber. In fact, in the rain forest, food search is restricted by the toxins that plants have evolved for their own protection. Janzen (1978:73) observes how the omnivore's dilemma affects primate food choice when he comments that "the plant world is not colored green; it is colored morphine, caffeine, tannin, phenol-terpene, cavanine, latex, phytohaemagglutin, oxalic acid, saponin." The jungle demands taste discrimination, and as Jolly (1985:57) notes, primates "must choose carefully the items worth harvesting, or safe to harvest, in a limited world."

The term *omnivory* is derived from the Latin *omni-*, "everything," and *vorous*, "feeding on." The trait appeared early in evolutionary history, among the insects (Eubanks, Styrsky, and Denno 2003), but its origin in Eocene primates (Sussman 1991) is more relevant to examining human adaptation. The composition of the early hominin diet is still a matter of debate (Hladik and Pasquet 2002). Katherine Milton (1999, 2003) argues that meat was an essential feature of the high-density foods that characterized early hominin diet, but others have argued that plants, fruits, and oil-rich seeds and tubers were also essential features of the early hominin diet (Wrangham and Conklin-Brittain 2003; Wrangham et al. 1999). This debate reflects the flexibility of the human diet and the ambiguity of the evidence (Ungar 2007) used to reconstruct Paleolithic diets.

Paul Rozin (1976) and Elisabeth Rozin (1982) suggest that the solution to the omnivore's dilemma was the development of cuisine, which mediated this biological conflict in several key ways. First, a given cuisine (Rozin 1982) includes a very limited number of foods selected from what the environment offers. The selection is usually based on availability and the efficiency with which nutrients can be extracted. Cuisine calls, second, for preparation techniques, such as cooking (Lévi-Strauss 1979), and third, for traditional principles of flavoring food staples. The fourth aspect of cuisine consists of rules: the number of meals eaten each day, whether they are eaten alone or with others, the ceremonial use of foods, the observation of taboos—all of which comprise the code of etiquette (Elias 1978).

The adaptive significance of flavors used to enhance food has generated some controversy (Krebs 2009). Although the notion that spices were used to mask the smell of rotting foods has been dispelled (Keay 2006), there is evidence that they have antioxidant (Yanishlieva, Marinova, and Pokorný 2006) and antibacterial (Shan et al. 2009; Sherman and Billing 1999; Sherman and Hash 2001) properties. These biological benefits of spices are a regionally variable adaptive by-product of their primary use as a solution to neophobia and to enhance dietary variety. For example, the use of certain flavors to identify foods of a particular group may provide a reassuring familiarity that blunts the fear of new foods. Introducing a familiar flavor in an unfamiliar food is also instrumental in facilitating children's acceptance of new foods and resolving their neophobia (Pliner and Stallberg-White 2002; Wardle and Cooke 2008). Interestingly, these same flavors can also be manipulated to provide variety; for example, Indian curries can be combined in a number of ways to create, in the Rozins' terminology, themes and variations that help to solve the omnivore's dilemma (Armelagos 1987; Rozin and Rozin 1981).

Rolls and colleagues (1982:120) see this search for variety as an aid to ensuring a nutritionally balanced diet. They also suggest that "built-in" mechanisms may ensure that humans pursue some variety in their food search. When people eat a particular food over an extended period, they develop sensory-specific satiety or palate fatigue and lose their taste for it (Rolls 1986; Rolls et al. 1986). The desire to eat other foods is not affected; we therefore maintain palatability at a high level by eating a variety of foods (Rolls 2007).

The !Kung of the Kalahari Desert in Botswana well illustrate how food diversity can play an integral role in cuisine. The !Kung recognize 105 species of plants and 260 species of animals as edible. These 365 food items have been selected from the more than 500 plants and animals that have been identified as edible by contemporary scientists in the !Kung's environment (Lee 1984:36). The mogongo nut is the !Kung's primary food source, but thirteen other widely available plants are also considered major food sources. Another nineteen species of locally available plants are held as minor foods. Even with this variety, the !Kung select fourteen items that are primary and major food categories, and that make up 75% of the overall diet (Lee 1979:159). The remainder of the plants and animals they consume function as sources of culinary variety and as famine foods. Plant foods constitute 67% of the Kung diet (Lee 1968). This pattern of food exploitation may not be representative, however, since the food list reported by Lee was limited to one month of data collection and transportation was provided for the women who were collecting mogongo nuts (Kaplan et al. 2000).

BRAIN SIZE, GUT SIZE, AND FOOD CHOICE

The relationship of dietary choice to the expansion of the brain during human evolution remains a relevant topic of research, with researchers progressively revealing how the selection for increasingly larger brains in humans and their recent ancestors is related to a number of factors. Dunbar (1998) suggests that the expansion of the brain's neocortex, which is involved in social cognition, is associated with social interaction and memory. Although Dunbar minimizes the impact of the food search, a high-quality diet requires knowledge as well as temporal and spatial memory to extract resources that are patchily distributed (Snodgrass, Leonard, and Robertson 2009). The cognitive ability to deal with the complexity of the search for plants and prey and the vagaries of an unpredictable climate was facilitated by increases in the social networks of early hominins (Dunbar 2003; Dunbar and Shultz 2007; Sol 2009). The need to maintain intricate knowledge of the environment necessitates a larger brain. Larger brains, however, come with attendant metabolic demands; our brain, which represents only 2% of our overall body mass, consumes 20% of our energy budget (Magistretti 2009). In its resting state, the human brain requires almost 25% more energy than do the brains of other primates at rest. As such, brain expansion created selective pressures on behavioral strategies to meet increasing energy demands. Notwithstanding the specific selective pressures that led to the expansion of the brain, its larger size would have provided the cognitive ability needed in the food search.

Humans, relative to other primates, have a small total gut size given their body size (Milton 1999). The human small intestine makes up more than 56% of the total gut, whereas the colon constitutes only 17% to 23% of its length. In comparison, an ape's colon makes up more than 45% of the total gut, and the small intestine makes up between 14% and 29% (Milton 1999, 2003). These figures reflect an evolutionary trend in the relationship between gut morphology and brain size. Folivores (leaf-eaters) have smaller brains and longer large intestines than frugivores (fruit-eaters) (Milton 1993). As large-brained omnivores, humans are the outliers when the size and morphology of their gut are compared with those of other primates.

Allometric scaling of the relationship between the human brain and gut shows that observed brain size is 300% of expected brain size, and observed gut size is 73% of what would be expected, on average, if humans followed the proportional trajectories characteristic of other primates. Two theories have been generated to explain the energy constraints related to the increase in fuel needed to meet the brain's demands. The first, the expensive tissue hypothesis (Aiello and Wheeler 1995; Aiello, Bates, and Joffe 2001), argues that our big brains are fueled by the energy saved by our having a smaller gastrointestinal tract than other, smaller-brained primates. High-density and volumetrically concentrated foods are more easily digestible and provide added energy. In contrast, the second—the maternal investment hypothesis—claims that mothers provide the extra energy needed for brain development through the placenta during pregnancy and via breast milk between birth and age four, when the human brain reaches 85% of its adult size.

Although the origin and causes of this pattern of meeting the increased energy demands of big brains are matters of controversy, there is no question that the changes in the alimentary canal would necessitate diets of high-quality, high-density foods, such as fruit, oil-rich nuts, tubers, and animal protein, regardless of the selective forces that were driving brain expansion. The composition of the early hominin diet is still a matter of debate (Hladik and Pasquet 2002). Milton (1999, 2003), for one, has argued that meat was an essential feature of the high-density foods that characterized the early hominin diet. In particular, Milton finds it "highly unlikely" that early hominins could have achieved their large and complex brains while on the evolutionary trajectory of large and highly social primates without increasing their intake of animal protein (2003:3886S). In this case, the inclusion of meat in the diet would have allowed hominins to evade the nutritional constraints placed on body size (Milton 2003). Children especially need "volumetrically concentrated, high quality food" to feed their rapidly growing brain given their relatively small guts (Milton 2003). In turn, Hamilton (1987) has argued that consumption of animal protein by primates and early humans created an evolutionary inertia that ultimately explains the contemporary overconsumption of meat in humans. In contrast, others have argued that plants, fruits, oil-rich seeds, and tubers were also essential features of early hominin diet (Wrangham and Conklin-Brittain 2003; Wrangham et al. 1999), especially because plant material makes up 87% to 99% of the primate diet (Milton 2003). Wrangham (2009) has made the case that high-density foods such as seeds and tubers would have been an alternative to meat eating.

Tubers and other starch-rich foods are improved by cooking. The advent of cooking was a major development in hominin evolution and enabled the processing of starches into high-density foods (Gibbons 2007; Keller 2009). In essence, it is a technological means of "predigesting" foods (Milton 2000). The control of fire, an essential component in cooking, is said to have "made us human" (Wrangham 2009) and "ignited human evolution" (Burton 2009). According to Wrangham and colleagues, cooking defined and shaped human evolution (Wrangham and Conklin-Brittain 2003; Wrangham et al. 1999). Cooking makes inedible plants edible, and cooked foods are preferred by humans and even captive apes (Wobber, Hare, and Wrangham 2008). Cooking breaks down the skin, softens cellulose, denatures toxins, and reduces a complex protein which enhances sweetness and would have increased the caloric intake of early hominins (Wrangham et al. 1999). Apes can finely discriminate levels of sugar (Hladik and Simmen 1996), and cooking increases the susceptibility of starch to amylase degradation (Svihus, Uhlen, and Harstad 2005; Tester, Qi, and Karkalas 2006).

Wrangham and Conklin-Brittain (2003) have suggested that cooked foods had evolutionary consequences, such as the evolution of smaller teeth and the previously mentioned changes in the intestinal tract, which allowed for faster rates of passage through the gut and better digestion of various foods. These changes would eventually have reduced hominins' abilities to digest uncooked fibers and to detoxify secondary compounds, limiting many potential dietary items and creating a dietary dependence on cooking.

Wrangham and colleagues' hypothesis is complicated, however, by issues of timing. Evidence of the control of fire appears much later in the evolutionary record than the origin of features such as smaller teeth that they assert were the result of cooking. There are possible indications of the use of fire around 1.9 million years ago, with evidence of controlled fires in the form of the hearths between 800,000 (Goren-Inbar et al. 2004) and 250,000 (Gibbons 2007; Preece et al. 2006) years ago, though evidence of cooking is difficult to distinguish in the archaeological record. Nonetheless, while fire may have been a more recent event in human evolution, it certainly was a factor in increasing access to a variety of high-density foods.

THE BIOLOGY OF TASTE

Sugar-rich foods represent classic examples of high-density food for Americans. Johnson and colleagues (2009) report the average American consumes 22.2 teaspoons of added sugar (sugar added as an ingredient during food preparation/processing) each day, representing 355 additional calories. Put into a broader perspective, this represents the equivalent of more than 5,892 pounds (2,672 kg) of added sugar over the course of an individual's lifetime. In caloric terms, this represents 10 million calories which would translate to 2,946 pounds (1,336 kg) of added weight if the energy were not expended. To dissipate these calories, a 155-pound (70 kg) man would have to run a mile in 11.5 minutes for 17,761 hours.

There is a biological basis for the consumption of sucrose and other sweet substances such as simple carbohydrates, and even sweet-tasting amino acids such as alanine. The 10,000 or so taste buds on the tongue (Pfaffmann 1959) have between 50 and 100 taste receptors that can detect sweetness. Moreover, even receptors in the gut can detect sweetness. When these receptors are stimulated, they cascade pleasurable impulses to the hypothalamic region of the brain. Even infants prefer substances that are sweeter than lactose, the sugar found in human breast milk (Desor, Maller, and Turner 1972; Schiffman 1983). This feedback system is an important evolutionary adaptation that ensures a search for rich energy sources.

From an evolutionary perceptive, access to an abundant source of refined sugar that is available to the masses is a recent blip in evolutionary time. Only with the globalization of trade in the 1500s and ultimately the industrialization of the food system did refined sugar become an accessible commodity. By the fifteenth century, Venetian ships were bringing 100,000 pounds (>45 metric tons) of sugar to England every year. During this period, sugar transitioned from being a luxury item to one that was widely—and inexpensively—accessible to the lower classes (Mintz 1985). By 2009, the world production of sugar had reached 3.1 trillion pounds (Tian 2009), of which 61 billion pounds were produced in the United States. However, even 61 billion pounds does not meet our society's demands for sweetness; additional sugar (limited by the federal government to 15% of domestic production) is imported into the United States each year.

Beyond sugar, there is evidence of a "fat tooth" that has an even greater biological propensity than the well-known "sweet tooth." According to

Drewnowski and colleagues (1995), when binge eaters who feast on sugary, high-fat treats are given an opiate inhibitor (naloxone), their cravings for the high-fat treats like chocolate decrease but their consumption of other foods is not diminished (Drewnowski et al. 1995). More recent studies suggest that it is not the ingestion of preferred foods but the stimulation of the feeding and reward centers of the brain that leads to permanent neurochemical changes in the brain (Levine, Kotz, and Gosnell 2003). Ultimately, though, even if these biological changes create a propensity for foods that affect the brain reward centers, it is the economic factors that explain their use (Drewnowski 2003; Drewnowski and Levine 2003).

At the other end of the taste scale is the ability to taste bitter substances, such as PTC (phenylthiocarbamide) and PROP (or 6-n-propylthiouracil), which are similar to plant alkaloids that are anti-thyroxin agents (Johnston, Hertzog, and Malina 1966). Our ability to taste these substances would have prevented foragers from consuming the anti-thyroxin substances or forced them to process the plant products to remove the bitterness (Johns 1990, 1999). The intensity of this tasting ability is related to the number of fungiform papillae on the tongue (Arvidson and Friberg 1980; Essick et al. 2003). Recent work has revealed taste receptors, similar to those on the tongue, on respiratory cilia, the hairlike projections on the primary bronchi that remove microbes and debris (Shah et al. 2009). When bitter substances are sensed, they increase motility to remove the substances from the airways (Kinnamon and Reynolds 2009). In contemporary populations, taste sensitivity to PTC and PROP is sometimes related to dislikes of certain foods, such as eggplant, cabbage, and brussels sprouts (Yackinous and Guinardf 2003). Coffee and chocolate are also bitter, repellant substances, but they have been made palatable by the addition of sugar (Mintz 1985).

Issues of sweetness and bitterness show how cultural factors can interact with the biological underpinnings of taste and distaste on the tongue and in the brain. The coevolution of genes and taste perceptions illustrates the ways in which cultural factors and adaptive trade-offs impact the omnivore's diet. This cultural layering suggests that the nutritional failings of Americans cannot be solely tied to biological factors, opening an examination of another evolutionary trend that will provide a basis for our nutritional dilemma. Two major events in evolutionary history set the stage for our contemporary dietary problems: the transition to primary food production (Aldenderfer 2009; Cohen 2009) and the industrialization of the food system (Grey 2000). Both have systematically reduced the number of foods available for consumption by human populations.

THE AGRICULTURAL TRANSFORMATION

The importance of agriculture in the development of modern civilization has made most of us agricultural chauvinists who have never evaluated the biological costs of the transformation to primary food production. However, some researchers have critically evaluated the effects of Neolithic development. Manning (2004) describes agriculture as a maladaptation that constitutes the basis of imperialism, slavery, diseases, and other scourges of humanity; others have called it the metaphorical "childhood's end" for humanity (Bar-Yosef 2004:S1). Diamond (1987) echoes Manning's criticism in calling it "the worst mistake in the history of the human race." For Diamond, agriculture is the fundamental cause of malnutrition, starvation, infectious disease, and social inequalities.

Even the Bible (Genesis 3:17–19) despairingly describes agriculture as the penalty for original sin. Humans were driven from the Garden of Eden, wherein all of their needs were being met, because of their first moral transgression. As punishment, God decreed that they were ordained to toil the weed-filled land forever:

> I have placed a curse on the ground. All your life you will struggle to scratch a living from it. It will grow thorns and thistles for you, though you will eat of its grains. All your life you will sweat to produce food, until your dying day. Then you will return to the ground from which you came. For you were made from dust, and to the dust you will return.

Although some of the negative impacts of agriculture, such as increases in loads of intestinal parasites from prolonged stays in one living location, are intuitive (Wolfe, Dunavan, and Diamond 2007), the emergence of nutritional diseases (Cohen and Armelagos 1984) is not. Nonetheless, the rise of social inequality (Armelagos, Brown, and Turner 2005), famine, seasonal hunger, blights, and trade (which often leads to exchange of high-quality food for items of symbolic value, creating nutritional problems) attendant with the development and intensification of agriculture increased the threat of reductions in dietary resources. Skeletal samples from populations that underwent the shift to agriculture and intensified primary food production show an array of pathologies that suggest a corresponding decline in health (Cohen and Armelagos 1984; Steckel and Rose 2002). A rise in the prevalence of infectious diseases resulted from increases in the size and density of sedentary populations (Barrett et al. 1998). The domestication of animals introduced zoonotic infections—diseases carried by animal hosts that can infect humans as well, such as tuberculosis (Wolfe, Dunavan, and Diamond 2007). For example, of the 1,407 species recognized as human pathogens, 58% are of zoonotic origin (Woolhouse and Gowtage-Sequeria 2005). In turn, 177 species are the sources of emerging and reemerging pathogens that continue to affect modern populations. Agricultural populations also had further difficulty with the accumulation of human wastes that could pollute their sources of water (Brothwell 1972), resulting in increased loads of intestinal parasites.

Empirically, agricultural populations experienced significant instances of nutritional deficiencies even though they were able to produce surpluses that drove cultural development. Reduction in overall dietary breadth was a particular product of the Neolithic revolution, as the increased reliance on a small number of domesticated plants limited the variety of foods consumed. Agricultural subsistence invariably reduces the variety of available foods (Armelagos 1987), and farming societies frequently specialize in a single cereal cultigen such as millet, rice, wheat, or maize, which further reduces the types of foods available. Plant blights and droughts also interrupted the flow of food. Skeletal samples from populations that underwent the shift to agriculture and intensified primary food production show evidence of retarded and interrupted growth during childhood, which ultimately had an impact on life expectancy (Cohen and Armelagos 1984).

Surprisingly, the dietary deficiencies produced by primary food production may have also exacerbated the diseases afflicting early agriculturalists. The reduction of the dietary niche (Katz 1987) results in nutritional deficiencies that can increase the impact of infectious disease (Hulsewé et al. 1999; Ulijaszek 2000). Adding to the disease profile, food storage, possible because of food surpluses generated by agriculture, also increased the potential for food poisoning (Brothwell 1972; Brothwell and Brothwell 1998). The combination of a complex society, increasing divisions of class, epidemic disease, and dietary insufficiencies had a major effect on the stress levels in agricultural populations (Goodman et al. 1984).

Agriculture leads to a reduction in the dietary niche that creates a nutritional bottleneck. According to the Human Resources Institute (Reid and Miller 1989), humans have used about 5,000 species of plants as food. Of these, only 150 have become major products of world commerce, with less than 20 providing most of the world's food. Of these 20 species, wheat, rice and corn (maize) account for 60% of the calories and 56% of the protein that humans consume directly from plants. In this context, the diversity that humans require comes from variation in the manner of preparation and from the use of spices to provide perceived, if somewhat artificial, variety.

INDUSTRIALIZATION OF THE FOOD SYSTEM

Industrialization of the food system and the rise of factory farms have further reduced humanity's dietary breadth. The genetic diversity of crops has declined with industrial agriculture. According to United Nations estimates, 75% of the genetic diversity of crop plants was lost in the twentieth century (Food and Agricultural Organization 1997). Rural Advancement Foundation International used a U.S. Department of Agriculture list of seeds that were available in 1903 to illustrate the extent of genetic erosion (Fowler and Mooney 1990:62–67). The survey revealed that by 1983, 97% of the 8,172 seed varieties on the list had been lost. The loss includes 96% of the 688 varieties of green beans (*Phaseolus*), 95% of the 544 varieties of cabbage, and 93% of the 789 varieties of corn.

Filipino farmers once grew thousands of varieties of rice; today, only two varieties account for 98% of the area sown. Mexico has lost an estimated 80% of its varieties of maize. Of 8,000 traditional rice varieties being grown in China in 1949, only 50 remained in 1970. Modern varieties have supplanted traditional varieties for 70% of the world's corn, 75% of Asian rice, and half of the wheat in Africa, Latin America, and Asia. In 1950, India had 30,000 wild varieties of rice, but by 2015, only 50 are expected to remain (Fowler and Mooney 1990).

In Peru, 100 varieties of potatoes can be grown in a single valley, and some households plant as many as a dozen varieties in their small plots. In comparison, only about 100 varieties of potatoes are grown in the entire United States (Brush 1992; Brush et al. 1995). But, 70% of the U.S. crop is of the russet variety, and half of this production is processed into french fries, chips, and dehydrated potato products.

Despite the loss of many varieties of corn, the number of products made from this crop has increased. Of 45,000 food products found in the modern American supermarket, more than 11,000 are made from corn (Pollan 2006:19). In 2007, 800 million tons of corn were grown worldwide (FAO 2007). Corn has a relatively telltale chemical signature (Schoeninger 2009) that persists as it travels through the complex system that turns it into feed, which is consumed and processed by cattle to grow tissue. It continues after the animals are slaughtered and the meat is cooked. Studies of these telltale chemical signatures show that 30% of beef is derived from cows fed exclusively on corn (Chesson, Ehleringer, and Cerling 2009; Jahren and Kraft 2008, 2009). This pattern is repeated and enlarged upon in industrially produced fast foods; not only the cheeseburger (which is 52% corn), but the remainder of the meal is mostly corn as well. Sodas turn out to be 100% corn, with similar contributions in milk shakes (78%), salad dressing (65%), and chicken nuggets (56%). Even french fries have a 23% corn signature because of the oil in which they are fried (Pollan 2006:117).

Just as the corn signature is apparent in fast-food meals, those who eat fast foods or other highly processed, industrially produced foods also reflect this signature. For example, when CNN Medical Correspondent Dr. Sanjay Gupta had a strand of his hair isotopically analyzed, the results demonstrated that 69% of the carbon in his body came from corn (Gupta 2007).

In 2007, the average American spent 9.8% of their disposable personal income on food (5.7% spent at home and 4.1% away from home; Clauson 2008). The lower the income, the greater the proportion spent on food. Americans who earned $10,000 to $14,999 a year, before taxes, spent a quarter of their income on food, while those earning $15,000 to $19,999 a year spent 19% of their income on food. As food prices increase, lower-income families will spend an increasing share of their income on food. On the other hand, those making $70,000 spend 11% of their income on food (Capehart and Richardson 2008). This $7,700 spent on food is more than the income of a family of two at the poverty level (U.S. Department of Health and Human Services 2009).

One justification for the industrial food system is its ability to produce enormous quantities of energy using minimal human energy. The techno-environmental efficiency ratio measures the amount of human energy invested in the food search or food production compared with the amount of energy extracted from that activity (Harris 1975:203–17). Hunter-gatherers, for example, extract 9.6 calories for every calorie put into the system. Irrigation agriculturalists extract nearly 54 calories for each calorie inputted, while industrial farming yields a whopping 210 calories for every human calorie expended. However, factory farming systems involve not only the human energy expended, but also the subsidy of energy from fossil fuels input into the system. For every 100 calories of energy put into factory farming, 13 calories are produced. Translated into what we eat, this means that it takes 23,200 calories to produce a 6-ounce (170 g) ribeye steak (Pimentel and Pimentel 2003)! Even a 2.8-ounce (80 g) serving of peas (117 calories) requires 3,637 calories of energy in its production (Rao 1977). We are, in a sense, "eating oil" (Green 1978): Americans use 120 billion gallons (U.S.) of oil to produce a year's supply of food (Pimentel et al. 2008).

The industrialization of the food system creates opportunities for incredibly dense fast foods that can contain 65% more energy than is found in a typical meal, but with minimal concern for issues of health. Energy density refers to the amount of calories an item of food contains in relation to its weight. A prime example of such a high-density food has been introduced by Kentucky Fried Chicken (KFC). For $4.99, KFC offers the breadless "Double Down," which is made with two Original Recipe® fried chicken filets, two strips of bacon, a slice of pepper jack cheese, a slice of Swiss cheese, and dollop of "the Colonel's secret sauce," but with no bread—the chicken filets serve as the bun. The *Vancouver Sun* (Parry 2009) estimates that it has 1,228 calories and supplies more than 124% of the daily recommended allowance of fat, 117% of the saturated fat, 105% of the cholesterol, 125% of the sodium, 194% of the protein, and 61% of the daily recommended calorie intake.

Although Americans may have difficulty with health-related issues associated with eating a "Double Down," they certainly seem to be comfortable eating meat. There is evidence that eating red meat generates a modest increase in mortality risk from cancer and cardiovascular disease (Sinha et al. 2009), but Americans are not deterred. In 2001, Americans consumed an amount of meat (195.2 pounds or 87 kg; U.S. Department of Agriculture 2003:15) equivalent to the average body weight of the American male (Ogden et al. 2004). In 2009, 35 million cows, 150 million pigs, and 9 billion birds, mainly chickens and turkeys, were killed to provide this meat (Kolbert 2009). Americans show little meaningful concern about the effects of meat eating on their health. However, meat eating certainly has an impact on the health of the planet. McMichael and coworkers (2007) suggest that 22% of greenhouse emissions is derived from agricultural activity relating to livestock production. They argue for agricultural policies that will consider health risks from consumption and from climate change. The average consumption of meat worldwide is about 100 g per person per day, with about a tenfold range between high-volume and low-volume meat consumers. They suggest a target of 90 g of meat per person per day that is more evenly distributed, and that less than 50 g of meat should come from methane-producing ruminants such as cattle, goats, and sheep.

Drewnowski and Darmon (2005) state that the relationship they see between obesity and socioeconomic factors is related to dietary energy density and energy cost. Energy-dense foods, such as refined grains, sugars, and fats, are inexpensive largely because of major government subsidies on production of primary ingredients, such as seed oil and sugar; they taste good and are also convenient. The nutrient-dense lean meats, fish, fresh vegetables, and fruits generally cost more. An inverse relationship between the energy density of foods (kilojoules per gram) and their energy cost (dollars per megajoule) means that energy-dense diets are associated with lower daily food costs and may be an effective way for individuals to save money. According to Drewnowski and Darmon, these decisions to purchase and consume energy-dense food may have economically burdensome biological consequences. They cite laboratory studies showing that energy-dense foods and diets have a lower satiating power and may result in passive overeating. Raynor et al. (2004) offer an interesting slant on the consumption of energy-dense foods, suggesting that increased variety in the food supply may contribute to the development and maintenance of obesity. Studies in animals and humans show an increase in consumption when there is more variety in a meal or a diet. However, the variety discussed here comes not from diversity in the types of foods or in their preparation, but primarily from variations in the amounts of added fat, sugar, and salt.

Lifestyle factors also have an impact on food choices made by individuals and families. Carol Devine and coworkers (2009) found that half of the mothers and fathers they surveyed depended on mealtime coping strategies. Fathers tended to skip family meals, eat at work, or feed their families take-out meals; mothers were likely to skip breakfast and buy restaurant or prepared entrees instead of cooking. Devine et al. claim that "food prepared outside the home has been shown to be lower in nutritional quality than food prepared at home. Less healthful diets have been positively associated with work conditions such as low job status, poor job conditions, high workloads, high work demands, and low control at work" (2009:365).

We live in a land of conspicuous abundance and hidden poverty. American supermarkets stock more than 50,000 items, renewing the offerings with 11,000 new items every year (Nestle 2002). More than 7,000 of these new items are condiments, candy, snacks, baked goods, soft drinks, and ice cream novelties. This abundance exists in a nation in which 36.2 million Americans (12.2%) live in food-insecure households and 11.9 million live with hunger (Cook 2009:146–47). The food insecurity persists even with sizable amounts of federal food assistance (Chilton and Rose 2009).

Food security is defined by the United Nations as occurring "when all people at all times have access to sufficient, safe, nutritious food to maintain a healthy and active life" (FAO 1996). It describes not only the availability of food, but the ability to purchase food. Food security means having a reliable source of food and sufficient resources to purchase it. A family is considered food-secure when its members do not live in hunger or fear of starvation (U.S. Department of State 2009). Hadley and Maes (2009) suggest that the primary issues for most families at risk are the anxiety and uncertainty that come with food insecurity.

Half of all child deaths in the developing world are related to undernutrition. Chronic hunger and undernutrition primarily result from poverty—people who are poor often simply cannot afford to buy food. This year, for the first time, the number of hungry people in the world will exceed one billion, with up to two billion facing intermittent food insecurity. This trend exists at a time when the flow of food aid is at a 20-year low according to the United Nations World Food Programme (Moszynski 2009). Problems with food security in the next 20 years will become even more challenging as global demand for food is projected to increase by 50% (U.S. Department of State 2009). Ironically, this food insecurity coexists with rampant overnutrition, with half of adult Americans being overweight or obese (Mokdad et al. 2001).

The international agency Save the Children (2006) estimates that each year four million babies are stillborn, dying between week 22 of pregnancy and birth, and another four million die before they reach the age of one month. Almost all of these newborn deaths occur at home, in the absence of skilled health care. Enormous disparities exist between rich and poor nations. The mortality rate in Africa for newborns during their first months of life is 30 times greater than in Western Europe or North America (Save the Children 2006).

Even if infants and children survive stress during these critical periods, the impact of nutritional deficiency has long-term consequences (Armelagos et al. 2009). Stresses in utero (Kuzawa 2009), infancy, and childhood (Law 2005) have adverse health consequences in adulthood (Paneth 1995) and form the basis of a variety of adult diseases (Fall et al. 1995), including hypertension (Barker et al. 1990), respiratory disease (Barker et al. 1991), type 2 diabetes (Hales et al. 1991), insulin resistance and metabolic syndrome (Barker et al. 1993), osteoporosis (Cooper et al. 1997), and sacropenia (loss of muscle mass; Sayer et al. 2006) later in life (Syddall et al. 2005). This pattern of early life stress and its impact on adult disease has resulted in the "developmental origins of health and disease" hypothesis (Gillman 2005), which remains controversial (Solomons 2009) but has been gaining adherents (Gluckman and Hanson 2004; Gluckman, Hanson, and Beedle 2007).

An unexpected source of nutritional stress has been added to the problem of undernutrition with an increase in obesity. The term "nutrition transition" was coined by Popkin in 1994 to describe the coexistence of undernutrition and overnutrition in different segments of the same population. The segment that is overnourished experienced a rise in prevalence of chronic diseases (Popkin 1994). These epidemiological changes were associated with dietary changes related to an increase in the consumption of sugars and fats (Popkin 1994). This epidemiological pattern was thought to have emerged in developed nations that had controlled infectious diseases (Barrett et al. 1998; Omran 1971) but saw the rise of obesity, cardiovascular disease, and type 2 diabetes. We currently see in developing countries a growth in fast food outlets and global trade contributing to an increase in heart disease and other consequences of overnutrition (Astrup et al. 2008).

The consequences of the omnivore's dilemma have come full circle. The solution to the dilemma in the early hominins in Africa was the development of cuisine that could mediate the problem of neophobia. Cuisine resolved the need for defining what is edible and expanded the range of high-density foods needed to satisfy the inherent requirement for dietary variety. Two central events led to a reduction in the variety of plants and animals that formed the diet. The first occurred in the Fertile Crescent, Asia (the Yangtze and Yellow river basins), and the Americas (the eastern United States, Central Mexico, and Amazonia) and New Guinea Highlands, which were centers of development of complex processes of domestication (Armelagos and Harper 2005a, 2005b). The invention of new methods for preparing and processing food created variety. The second event was the industrialization of the food system led by the United Kingdom and then the United States, which reduced the number of foods that were used to feed their populations. Both of these nations have increased the variety of available foods through myriad processing methods that produce a large number of high-density foods featuring the addition of sugar and fats. The exportation of these high-density foods to the developing nations of Africa and Asia has created an unexpected scenario in which under- and overnutrition coexist. The omnivore's dilemma solved the perplexing issue of neophobia, but the inherent need for variety has created problems in an industrial age that paradoxically produces an overabundance of homogenous, high-density foods that deliver salt, sugar, fats, and little else, to the masses.

CONCLUSION

On October 30, 1991, off the coast of New England, a rare atmospheric phenomenon occurred with the convergence of a dry cold front, moisture from Hurricane Grace, and an extratropical low, creating 65-knot winds and 39-foot (nearly 12 m) waves. The event has been chronicled in Sebastian Junger's 1997 book, *The Perfect Storm.* The present nutritional dilemma is similarly the result of a "perfect storm" of an evolutionary journey that began with the original omnivore's dilemma. The cultural development of a cuisine that mediated the biological problem of what to eat inadvertently provided the background for the perfect storm. The elements of the perfect storm began with the need for high-density food in a food system that demands variety in diet, which combines with an industrial food system that produces inexpensive, high-density food in an economic system that creates dual-income families who face issues of time constraints. Add to this a vast advertising industry that capitalizes on these time demands. This storm spread globally with the expansion of high-density foods to poorer populations. The perfect storm of 1991 dissipated after a few days. The perfect storm that has produced this global nutritional dilemma has and will endure. It is a dilemma that has clearly perceptible origins in our evolutionary history, but one that we will have difficulty resolving.

ACKNOWLEDGMENTS

I would like to thank Peter J. Brown, Jennifer Sweeney, Molly K. Zuckerman, Kenneth Maes, and Bethany Turner for thoughtful reading and comments on this paper. An earlier version of this paper was given as the XXIX Journal of Anthropological Research Distinguished Lecture at the University of New Mexico in November 2009. Many thanks to Lawrence G. Straus for the invitation and to him and Ann Braswell for making the visit to UNM a thoroughly pleasurable experience.

REFERENCES CITED

Aiello, L. C., and P. Wheeler. 1995. The expensive-tissue hypothesis: The brain and the digestive system in human and primate evolution. *Current Anthropology* 36:199–221.

Aiello, L. C., N. Bates, and T. Joffe. 2001. "In defense of the expensive tissue hypothesis," in *Evolutionary anatomy of the primate cerebral cortex*. Edited by D. Falk and K. R. Gibson, pp. 57–78. Cambridge: Cambridge University Press.

Aldenderfer, M. 2009. Editorial: The continuing conversation about the origins of agriculture. *Current Anthropology* 50:585.

Aoki, K. 2001. Theoretical and empirical aspects of gene-culture coevolution. *Theoretical*

Population Biology 59:253–61.

Armelagos, G. J. 1987. “Biocultural aspects of food choice,” in *Food and evolution*. Edited by M. Harris and E. Ross, pp. 579–94. Philadelphia: Temple University Press.

———. 2003. “Bioarcheology as anthropology,” in *Archaeology is anthropology*, vol. 13. Edited by S. D. Gillespie and D. Nichols, pp. 27−40. Arlington, VA: Archaeological Papers of the American Anthropological Association.

———. 2008. “Biocultural anthropology at its origins: Transformation of the new physical anthropology in the 1950s,” in *The Tao of anthropology*. Edited by A. J. Kelso, pp. 269–82. Gainesville: University of Florida Press.

Armelagos, G. J., P. J. Brown, and B. Turner. 2005. Evolutionary, historical and political economic perspectives on health and disease. *Social Science & Medicine* 61:755–65.

Armelagos, G. J., A. H. Goodman, K. N. Harper, and M. L. Blakey. 2009. Enamel hypoplasia and earlier mortality: Bioarchaeological support for the Barker hypothesis. *Evolutionary Anthropology* 18:261−71.

Armelagos, G. J., and K. N. Harper. 2005a. Genomics at the origins of agriculture, part 1. *Evolutionary Anthropology* 14:68–77.

———. 2005b. Genomics at the origins of agriculture, part 2. *Evolutionary Anthropology* 14:109–21.

Armelagos, G. J., T. Leatherman, M. Ryan, and L. Sibley. 1992. Biocultural synthesis in medical anthropology. *Medical Anthropology* 14:35–52.

Arvidson, K., and U. Friberg. 1980. Human taste: Response and taste bud number in fungiform papillae. *Science* 209:807–8.

Astrup, A., J. Dyerberg, M. Selleck, and S. Stender. 2008. Nutrition transition and its relationship to the development of obesity and related chronic diseases. *Obesity Reviews* 9 (Suppl 1):48–52.

Bar-Yosef, O. 2004. Editorial: East to West—Agricultural origins and dispersal into Europe. *Current Anthropology* 45:S1–S3.

Barker, D. J., A. R. Bull, C. Osmond, and S. J. Simmonds. 1990. Fetal and placental size and risk of hypertension in adult life. *British Medical Journal* 301:259–62.

Barker, D. J., K. M. Godfrey, C. Fall, C. Osmond, P. D. Winter, and S. O. Shaheen. 1991. Relation of birth weight and childhood respiratory infection to adult lung function and death from chronic obstructive airways disease. *British Medical Journal* 303:671–75.

Barker, D. J. P., C. N. Hales, C. H. D. Fall, C. Osmond, K. Phipps, and P. M. S. Clark. 1993. Type 2 (non-insulin-dependent) diabetes mellitus, hypertension and hyperlipidaemia (syndrome X): Relation to reduced fetal growth. *Diabetologia* 36:62–7.

Barrett, R., C. Kuzawa, T. McDade, and G. J. Armelagos. 1998. Emerging and re-emerging infectious diseases: The third epidemiologic transition. *Annual Review of Anthropology* 27:247–71.

Beja-Pereira, A., G. Luikart, P. R. England, D. G. Bradley, O. C. Jann, G. Bertorelle, A. T. Chamberlain, T. P. Nunes, S. Metodiev, N. Ferrand, and G. Erhardt. 2003. Gene-culture coevolution between cattle milk protein genes and human lactase genes. *Nature Genetics* 35:311–13.

Bloom, G., and P. W. Sherman. 2005. Dairying barriers affect the distribution of lactose malabsorption. *Evolution and Human Behavior* 26:301–12.

Bosilevac, J. M., M. N. Guerini, N. Kalchayanand, and M. Koohmaraie. 2009. Prevalence and characterization of Salmonellae in commercial ground beef in the United States. *Applied and Environmental Microbiology* 75:1892–1900.

Brothwell, D. 1972. “The question of pollution in earlier and less developed societies,” in *Population and pollution*. Edited by P. R. Cox and J. Peel, pp. 15–27. London: Academic Press.

Brothwell, D. R., and P. Brothwell. 1998. *Food in antiquity: A survey of the diet of early peoples*, expanded edition. Baltimore: Johns Hopkins University Press.

Brown, P. 1991. Culture and the evolution of obesity. *Human Nature* 2:31–57.

Brown, P. J., and S. V. Krick. 2001. "The etiology of obesity: Diet, television and the illusion of personal choice," in *Obesity, growth and development*. Edited by F. E. Johnston and G. D. Foster, pp. 111–27. London: Smith-Gorden.

Brush, S. 1992. Reconsidering the green revolution: Diversity and stability in cradle areas of crop domestication. *Human Ecology* 20:145–67.

Brush, S., R. Kesseli, R. Ortega, P. Cisneros, K. Zimmerer, and C. Quiros. 1995. Potato diversity in the Andean center of crop domestication. *Conservation Biology* 9:1189–98.

Burton, F. D. 2009. *Fire—the spark that ignited human evolution*. University of New Mexico Press, Albuquerque.

Capehart, T., and J. Richardson. 2008. Food price inflation: Causes and impacts. *CRS Report to Congress*. (Available online at http://assets.opencrs.com/rpts/RS22859_20080410.pdf)

Chesson, L., J. Ehleringer, and T. Cerling. 2009. American fast food isn't all corn-based. *Proceedings of the National Academy of Sciences (USA)* 106:E8.

Chevat, R., and M. Pollan. 2009. *The omnivore's dilemma: The secrets behind what you eat*. Young Readers' Edition. New York: Dial Books.

Chilton, M., and D. Rose. 2009. A rights-based approach to food insecurity in the United States. *American Journal of Public Health* 99:1203–11.

Clauson, A. 2008. Despite higher food prices, percent of U.S. income spent on food remains constant. *Amber Waves (*September). (Available online at http://www.ers.usda.gov/AmberWaves/September08/Findings/PercentofIncome.htm)

Cohen, Mark N. 2009. Introduction: Rethinking the origins of agriculture. *Current Anthropology* 50:591–95.

Cohen, M. N., and G. J. Armelagos, editors. 1984. *Paleopathology at the origins of agriculture*. Orlando: Academic Press.

Cook, J. T. 2009. "Food security and insecurity in the United States and their consequences for child health," in *Critical food issues: Problems and state-of-the art solutions: Environment* Edited by L. E. Phoenix, pp. 143–56. Santa Barbara: ABC-CLIO.

Cooper, C., C. Fall, P. Egger, R. Hobbs, R. Eastell, and D. Barker. 1997. Growth in infancy and bone mass in later life. *Annals of the Rheumatic Diseases* 56:17–21.

Desor, J. A., O. Maller, and R. E. Turner. 1972. Taste acceptance of sugars by human infants. *Journal of Comparative and Physiological Psychology* 84:496–501.

Devine, C. M., T. J. Farrell, C. E. Blake, M. Jastran, E. Wethington, and C. A. Bisogni. 2009. Work conditions and the food choice coping strategies of employed parents. *Journal of Nutrition Education and Behavior* 41:365–70.

Diamond, J. 1987. The worst mistake in the history of the human race. *Discover* (May): 64–66.

Drewnowski, A. 2003. The role of energy density. *Lipids* 38:109−15.

Drewnowski, A., and N. Darmon. 2005. The economics of obesity: Dietary energy density and energy cost. *American Journal of Clinical Nutrition* 82:265S–273S.

Drewnowski, A., and A. S. Levine. 2003. Sugar and fat—from genes to culture. *Journal of Nutrition* 133:829S–30S.

Drewnowski, A., D. Krahn, M. Demitrack, K. Nairn, and B. Gosnell. 1995. Naloxone, an opiate blocker, reduces the consumption of sweet high-fat foods in obese and lean female binge eaters. *American Journal of Clinical Nutrition* 61:1206–12.

Dunbar, R. I. M. 1998. The social brain hypothesis. *Evolutionary Anthropology: Issues,* *News, and Reviews* 6:178–90.

———. 2003. The social brain: Mind, language, and society in evolutionary perspective. *Annual Review of Anthropology* 32:163–81.

Dunbar, R. I. M., and S. Shultz. 2007. Evolution in the social brain. *Science* 317:1344–47.

Durham, W. H. 1990. Advances in evolutionary culture theory. *Annual Review of Anthropology* 19:187–210.

———. 1991. *Coevolution: Genes, culture, and human diversity*. Palo Alto: Stanford University Press.

Elias, N. 1978. *The civilizing process: The history of manners*. New York: Urizen Books.

Enattah, N. S., T. G. K. Jensen, M. Nielsen, et al. 2008. Independent introduction of two lactase-persistence alleles into human populations reflects different history of adaptation to milk culture. *American Journal of Human Genetics* 82:57–72.

Essick, G. K., A. Chopra, S. Guest, and F. McGlone. 2003. Lingual tactile acuity, taste perception, and the density and diameter of fungiform papillae in female subjects. *Physiology & Behavior* 80:289–302.

Eubanks, M. D., J. D. Styrsky, and R. F. Denno. 2003. The evolution of omnivory in heteropteran insects. *Ecology* 84:2549–56.

Fall, C. H., C. Osmond, D. Barker, P. M. Clark, C. N. Hales, Y. Stirling, and T. W. Meade. 1995. Fetal and infant growth and cardiovascular risk factors in women. *British Medical Journal* 310:428–32.

Food and Agricultural Organization (FAO). 1996. Report of the World Food Summit, 13–17 November 1996. Rome. (Available online at http://www.fao.org/docrep/003/w3548e/w3548e00.htm)

———. 1997. *The state of the world's plant genetic resources for food and agriculture*. Rome: Food and Agricultural Organization of the United Nations.

———. 2007. *Corn*. Rome: Food and Agricultural Organization of United Nations, Economic and Social Department, Statistical Division.

Fowler, C., and P. R. Mooney. 1990. *Shattering: food, politics, and the loss of genetic diversity*. Tucson: University of Arizona Press.

Franz, E., and A. H. C. van Bruggen. 2008. Ecology of *E. coli* O157:H7 and *Salmonella enterica* in the primary vegetable production chain. *Critical Reviews in Microbiology* 34:143–61.

Gibbons, A. 2007. Food for thought. *Science* 316:1558–60.

Gillman, M. W. 2005. Developmental origins of health and disease. *New England Journal of Medicine* 353:1848–50.

Gluckman, P. D., and M. A. Hanson. 2004. Living with the past: Evolution, development, and patterns of disease. *Science* 305:1733–36.

Gluckman, P. D., M. A. Hanson, and A. S. Beedle. 2007. Early life events and their consequences for later disease: a life history and evolutionary perspective. *American Journal of Human Biology* 19:1–19.

Goodman, A. H., and T. L. Leatherman. 1998. "Traversing the chasm between biology and culture: An introduction," in *Building a new biocultural synthesis: Political-economic perspectives on human biology*. Edited by A. H. Goodman and T. L. Leatherman, pp. 3–41. Ann Arbor: University of Michigan Press.

Goodman, A. H., and T. L. Leatherman, eds. 1998. *Building a new biocultural synthesis: Political-economic perspectives on human biology*. Ann Arbor: University of Michigan Press.

Goodman, A. H., D. Martin, G. J. Armelagos, and G. Clark. 1984. "Indications of stress from bones and teeth," in *Paleopathology at the origins of agriculture*. Edited by M. N. Cohen and G. J. Armelagos, pp. 13–49. Orlando, FL: Academic Press.

Goren-Inbar, N., N. Alperson, M. E. Kislev, O. Simchoni, Y. Melamed, A. Ben-Nun, and E. Werker. 2004. Evidence of hominin control of fire at Gesher Benot Ya`aqov, Israel. *Science* 304:725–27.

Green, M. B. 1978. *Eating oil: Energy use in food production*. Boulder, CO: Westview Press.

Grey, M. A. 2000. The industrial food stream and its alternatives in the United States: An introduction. *Human Organization* 59:143–50.

Gupta, S. 2007. " If we are what we eat, Americans are corn and soy." CNN, September 27. (Available online at http://www.cnn.com/2007/HEALTH/diet.fitness/09/22/kd.gupta.column/index.html)

Hadley, C., and K. Maes. 2009. A new global monitoring system for food insecurity? *Lancet* 374:1223–24.

Hales, C. N., D. J. Barker, P. M. Clark, L. J. Cox, C. Fall, C. Osmond, and P. D. Winter. 1991. Fetal and infant growth and impaired glucose tolerance at age 64. *British Medical Journal* 303:1019–22.

Hamilton, W. J. 1987. "Omnivorous primate diet and the overconsumption of meat," in *Food and evolution*. Edited by M. Harris and E. B. Ross, pp. 117–32. Philadelphia: Temple University Press.

Harris, M. 1975. *Culture, people, nature: An introduction to general anthropology*, second ed. New York: Crowell.

———. 1987. "Foodways: Historical overview and theoretical prologomen," in *Food and evolution*. Edited by M. Harris and E. B. Ross, pp. 57–98. Philadelphia: Temple University Press.

Hladik, C. M., and P. Pasquet. 2002. The human adaptations to meat eating: A reappraisal. *Human Evolution* 17:199–206.

Hladik, C. M., and B. Simmen. 1996. Taste perception and feeding behavior in nonhuman primates and human populations. *Evolutionary Anthropology* 5:58–71.

Hulsewé, K. W. E., B. A. C. van Acker, M. F. von Meyenfeldt, and P. B. Soeters. 1999. Nutritional depletion and dietary manipulation: Effects on the immune response. *World Journal of Surgery* 23:536–44.

Ingelfinger, J. R. 2008. Melamine and the global implications of food contamination. *New England Journal of Medicine* 359:2745–48.

Ingram, C. J., C. A. Mulcare, Y. Itan, M. G. Thomas, and D. M. Swallow. 2009. Lactose digestion and the evolutionary genetics of lactase persistence. *Human Genetics* 124:579–91.

Jahren, A. H., and R. A. Kraft. 2008. Carbon and nitrogen stable isotopes in fast food: Signatures of corn and confinement. *Proceedings of the National Academy of Sciences (USA)* 105:17855–60.

———. 2009. Reply to Chesson et al.: Carbon stable isotopes in beef differ distinctly between corporations. *Proceedings of the National Academy of Sciences (USA)* 106:E9.

Janzen, D. H. 1978. "Complications in interpreting the chemical defenses of trees against tropical arboreal plant-eating vertebrates," in *Ecology of the arboreal folivores*. Edited by G. G. Montgomery, pp. 73–78. Cambridge: Cambridge University Press.

Johns, T. 1990. *"With bitter herbs they shall eat it": Chemical ecology and the origins of human diet and medicine.* Tucson: University of Arizona Press.

———. 1999. The chemical ecology of human ingestive behaviors. *Annual Review of Anthropology* 28:27–50.

Johnson, R. K., L. J. Appel, M. Brands, et al., on behalf of the American Heart Association. 2009. Dietary sugars intake and cardiovascular health: A scientific statement from the American Heart Association. *Circulation* 120:1011–20.

Johnston, F. E., K. P. Hertzog, and R. M. Malina. 1966. Phenylthiocarbamide taste sensitivity and its relationship to growth variation. *American Journal of Physical Anthropology* 24:253–55.

Jolly, A. 1985. *The evolution of primate behavior*. New York: Macmillan.

Junger, S. 1997. *The perfect storm: A true story of men against the sea*. Thorndike, ME: Thorndike Press.

Kaplan, H., K. Hill, J. Lancaster, and A. M. Hurtado. 2000. A theory of human life history evolution: Diet, intelligence, and longevity. *Evolutionary Anthropology* 9:156–85.

Katz, S. H. 1987. “Food and biocultural evolution: A model for the investigation of modern nutritional problems,” in *Nutritional anthropology*. Edited by F. E. Johnston, pp. 41–63. New York: Alan R. Liss.

Keay, J. 2006. *The spice route: A history*. Berkeley: University of California Press.

Keller, A. 2009. The cooking ape. *Science* 325:394–95.

Kinnamon, S. C., and S. D. Reynolds. 2009. Using taste to clear the air(ways). *Science* 325:1081–82.

Kolbert, E. 2009. “Flesh of our flesh.” *The New Yorker* (November 9):74–78.

Krebs, J. R. 2009. The gourmet ape: Evolution and human food preferences. *American Journal of Clinical Nutrition* 90:707S–711S.

Kuzawa, C. W, and E. A. Quinn. 2009. Developmental origins of adult function and health: Evolutionary hypotheses. *Annual Review of Anthropology* 38:131−47.

Law, C. 2005. Early growth and chronic disease: A public health overview. *Maternal & Child Nutrition* 1:169–76.

Lee, R. B. 1968. “What hunters do for a living, or How to make out on scarce resources,” in *Man the hunter*. Edited by R. Lee and I. DeVore, pp. 30–48. Chicago: Aldine.

———. 1979. *The !Kung San: Men, women, and work in a foraging society*. Cambridge: Cambridge University Press.

———. 1984. *The Dobe !Kung: Case studies in cultural anthropology*. New York: Holt, Rinehart and Winston.

Lévi-Strauss, C. 1979. *The raw and the cooked*. New York: Octagon Books.

Levine, A. S., C. M. Kotz, and B. A. Gosnell. 2003. Sugars and fats: The neurobiology of preference. *Journal of Nutrition* 133:831S–834S.

Magistretti, P. J. 2009. Low-cost travel in neurons. *Science* 325:1349–51.

Manning, R. 2004. *Against the grain: How agriculture has hijacked civilization*. New York: North Point Press.

McCracken, R. D. 1971. Lactase deficiency: An example of dietary evolution. *Current Anthropology* 12:479–517.

McElroy, A., and P. K. Townsend. 2009. *Medical anthropology in ecological perspective*, fifth ed. Boulder, CO: Westview Press.

McMichael, A. J., J. W. Powles, C. D. Butler, and R. Uauy. 2007. Food, livestock production, energy, climate change, and health. *Lancet* 370:1253–63.

Mead, P. S., L. Slutsker, V. Dietz, L. F. McCaig, J. S. Bresee, C. Shapiro, P. M. Griffin, and R. V. Tauxe. 1999. Food-related illness and death in the United States. *Emerging Infectious Disease* 5:607–25.

Milton, K. 1993. Diet and primate evolution. *Scientific American* 269:86–93.

———. 1999. A hypothesis to explain the role of meat-eating in human evolution. *Evolutionary Anthropology* 8:11–21.

———. 2000. Hunter-gatherer diets: A different perspective. *American Journal of Clinical Nutrition* 71:665–67.

———. 2003. The critical role played by animal source foods in human (*Homo*) evolution. *Journal of Nutrition* 133:3886S–3892S.

Mintz, S. 1985. *Sweetness and power: The place of sugar in modern history*. New York: Penguin Books.

Mokdad, A. H., B. A. Bowman, E. S. Ford, F. Vinicor, J. S. Marks, and J. P. Koplan. 2001. The continuing epidemics of obesity and diabetes in the United States. *Journal of the American Medical Association* 286:1195–2000.

Moran, L., P. Scates, and R. H. Madden. 2009. Prevalence of *Campylobacter* spp. in raw retail poultry on sale in Northern Ireland. *Journal of Food Protection* 72:1830–35.

Moss, M. 2009. *E. coli* path shows flaws in ground beef inspection. *New York Times* (October 4):1. (Available online at http://www.nytimes.com/2009/10/04/health/04meat.html?_r=1)

Moszynski, P. 2009. Hunger poses the "world's greatest health risk" as aid deficit bites, says UN. *British Medical Journal* 339:3849.

Myles, S., N. Bouzekri, E. Haverfield, M. Cherkaoui, J. M. Dugoujon, and R. Ward. 2005. Genetic evidence in support of a shared Eurasian-North African dairying origin. *Human Genetics* 117:34–42.

Nestle, M. 2002. The soft sell: How the food industry shapes our diets. *Nutrition Action Healthletter*. (Available online at http://findarticles.com/p/articles/mi_m0813/is_7_29/ai_90980246/)

NPD Group, Inc. 2009. NPD finds most Americans agree foods from supermarkets are safe but percentage is slipping. (Available online at http://www.npd.com/press/releases/press_090330.html)

Ogden, C. L., C. D. Fryar, M. D. Carroll, and K. M. Flegal. 2004. Mean body weight, height, and body mass index, United States 1960–2002. *Advanced Data from Vital and Health Statistics* 347:1–20.

Omran, A. R. 1971. The epidemiologic transition: A theory of the epidemiology of population change. *Millbank Memorial Fund Quarterly* 49:509–38.

Ortner, D. J., and H. Schutkowski. 2008. "Ecology, culture and disease in past populations," in *Between biology and culture*. Edited by H. Schutkowski, pp. 105–29. Cambridge: Cambridge University Press.

Paneth, N. 1995. Early origin of coronary heart disease (the "Barker hypothesis"). *British Medical Journal* 310:411−12.

Parry, C. 2009. KFC tests out new bunless burger to rave reviews in America's heartland. *Vancouver Sun* (August 25). (Available online at http://www.vancouversun.com/health/tests+artery+clogging+burger+rave+reviews+America+heartland/1917736/story.html)

Pfaffmann, C. 1959. "The sense of taste," in *Handbook of Physiology,* Section 1, Vol. 1: *Neurophysiology*. Edited by J. Field, H. W. Magoun, and V. E. Hall, pp. 507–33. Washington DC: American Physiological Society.

Pimentel, D., and M. Pimentel. 2003. Sustainability of meat-based and plant-based diets and the environment. *American Journal of Clinical Nutrition* 78:660S–663S.

Pimentel, D., S. Williamson, C. E. Alexander, O. Gonzelez-Pagan, C. Kontak, and S. E. Mulkey. 2008. Reducing energy inputs in the US food system. *Human Ecology* 36:459–71.

Pliner, P., and C. Stallberg-White. 2002. "Pass the ketchup, please": Familiar flavors increase children's willingness to taste novel foods. *Appetite* 34:95–103.

Pollan, M. 2006. *The omnivore's dilemma: A natural history of four meals*. New York: Penguin Press.

Popkin, B. M. 1994. The nutrition transition in low-income countries: An emerging crisis. *Nutrition Reviews* 52:285–98.

Preece, R. C., J. A. J. Gowlett, S. A. Parfitt, D. R. Bridgland, and S. G. Lewis. 2006.

Humans in the Hoxnian: Habitat, context and fire use at Beeches Pit, West Stow, Suffolk, UK. *Journal of Quaternary Science* 21:485–96.

Rao, M. A. 1977. Energy consumption for refrigerated, canned, and frozen peas. *Journal of Food Process Engineering* 1:149–65.

Raynor, H. A., R. W. Jeffery, D. F. Tate, and R. R. Wing. 2004. Relationship between changes in food group variety, dietary intake, and weight during obesity treatment. *International Journal of Obesity Related Metabolic Disorders* 28:81–820.

Reid, W. V., and K. R. Miller. 1989. "Food crops and biodiversity," in *Keeping options alive: The scientific basis for the conservation of biodiversity*. World Resources Institute. (Available online at http://www.wri.org/node/8584/)

Rolls, B. J. 1986. Sensory-specific satiety. *Nutrition Reviews* 44:93–101.

Rolls, B. J., E. T. Rolls, and E. A. Rowe. 1982. "The influence of variety on human food selection and intake," in *The psychobiology of human food selection*. Edited by L. M. Barker, pp. 101–22. Westport, CT: AVI.

Rolls, E. T. 2007. Sensory processing in the brain related to the control of food intake. *Proceedings of the Nutrition Society* 66:96–112.

Rolls, E. T., E. Murzi, S. Yaxley, S. J. Thorpe, and S. J. Simpson. 1986. Sensory-specific satiety: Food-specific reduction in responsiveness of ventral forebrain neurons after feeding in the monkey. *Brain Research* 368:79–86.

Rozin, E. 1982. "The structure of cuisine," in *The psychobiology of human food selection*. Edited by L. M. Barker, pp. 189–203. Westport, CT: AVI.

Rozin, E., and P. Rozin. 1981. Culinary themes and variation. *Natural History* 90:6–14.

Rozin, P. 1976. "The selection of food by rats, humans, and other animals," in *Advances in the Study of Behavior,* Vol. 6. Edited by J. Rosenblatt, R. A. Hinde, C. Beer, and E. Shaw, pp. 21–76. New York: Academic Press.

Save the Children. 2006. *State of the World's Newborns*. Washington, DC. (Available online at http://www.savethechildren.org/publications/technical-resources/child-survival/newborns_report.pdf)

Sayer, A. A., H. E. Syddall, H. J. Martin, E. M. Dennison, F. H. Anderson, and C. Cooper. 2006. Falls, sarcopenia, and growth in early life: Findings from the Hertfordshire Cohort Study. *American Journal of Epidemiology* 164:665–71.

Schiffman, S. S. 1983. Taste and smell in disease, part 2. *New England Journal of Medicine* 308:1337–43.

Schoeninger, Margaret J. 2009. Stable Isotope Evidence for the Adoption of Maize Agriculture. *Current Anthropology* 50:633–40.

Schutkowski, H. 2008. "Thoughts for food: Evidence and meaning of past dietary habits," in *Between biology and culture*. Edited by H. Schutkowski, pp. 141–65. Cambridge: Cambridge University Press.

Segal, D. A., and S. J. Yanagisako. 2005. "Introduction," in *Unwrapping the sacred bundle: Reflections on the disciplining of anthropology*. Edited by D. A. Segal and S. J. Yanagisako, pp. 1–23. Durham, NC: Duke University Press.

———, eds. 2005. *Unwrapping the sacred bundle: Reflections on the disciplining of anthropology*. Durham, NC: Duke University Press.

Shah, A. S., Y. Ben-Shahar, T. O. Moninger, J. N. Kline, and M. J. Welsh. 2009. Motile cilia of human airway epithelia are chemosensory. *Science* 325:1131–34.

Shan, B., Y.-Z. Cai, J. D. Brooks, and H. Corke. 2009. Antibacterial and antioxidant effects of five spice and herb extracts as natural preservatives of raw pork. *Journal of the Science of Food and Agriculture* 89:1879–85.

Sherman, P. W., and J. Billing. 1999. Darwinian gastronomy: Why we use spices. *BioScience* 49:453–63.

Sherman, P. W., and G. A. Hash. 2001. Why vegetable recipes are not very spicy. *Evolution & Human Behavior* 22:147–63.

Simons, F. 1969. Primary adult lactose intolerance and the milking habit: A problem in biological and cultural interrelations, I: Review of the medical research. *American Journal of Digestive Diseases* 14:819–36.

———. 1970. Primary adult lactose intolerance and the milking habit: A problem in biological and cultural interrelations, II: A culture historical hypothesis. *American Journal of Digestive Diseases* 15:695–710.

Sinha, R., A. J. Cross, B. I. Graubard, M. F. Leitzmann, and A. Schatzkin. 2009. Meat intake and mortality: A prospective study of over half a million people. *Archives of Internal Medicine* 169:562–571.

Snodgrass, J. J., W. R. Leonard, and M. L. Robertson. 2009. "The energetics of encephalization in early hominids," in *The evolution of hominin diets: Integrating approaches to the study of palaeolithic subsistence*. Edited by J.-J. Hublin and M. P. Richards, pp. 15–29. New York: Springer.

Sol, D. 2009. Revisiting the cognitive buffer hypothesis for the evolution of large brains. *Biology Letters* 5:130–33.

Solomons, N. W. 2009. Developmental origins of health and disease: Concepts, caveats, and consequences for public health nutrition. *Nutrition Reviews* 67 (Suppl. 1):2–6.

Steckel, R. H., and J. C. Rose. 2002. *The backbone of history: Health and nutrition in the Western Hemisphere*. Cambridge: Cambridge University Press.

Sussman, R. W. 1991. Primate origins and the evolution of angiosperms. *American Journal of Primatology* 23:209–23.

Svihus, B., A. K. Uhlen, and O. M. Harstad. 2005. Effect of starch granule structure, associated components, and processing on nutritive value of cereal starch: A review. *Animal Feed Science and Technology* 122:303–20.

Swallow, D. M. 2003. Genetics of lactase persistence and latose intolerance. *Annual Review of Genetics* 37:197–219.

Syddall, H. E., A. A. Sayer, S. J. Simmonds, C. Osmond, V. Cox, E. M. Dennison, D. J. Barker, and C. Cooper. 2005. Birth weight, infant weight gain, and cause-specific mortality: The Hertfordshire Cohort Study. *American Journal of Epidemiology* 161:1074–80.

Tester, R. F., X. Qi, and J. Karkalas. 2006. Hydrolysis of native starches with amylases. *Animal Feed Science and Technology* 130:39–54.

Tian, Y. 2009. Global sugar-production deficit to widen, ISO says (Update1). Bloomberg.com. (Available online at http://www.bloomberg.com/apps/news?pid=20601087&sid=antxF2zirii4)

Tishkoff, S. A., F. A. Reed, A. Ranciaro, et al. 2007. Convergent adaptation of human lactase persistence in Africa and Europe. *Nature Genetics* 39:31–40.

Turner, B. L., K. Maes, J. L. Sweeney, and G. J. Armelagos. 2008. "Human evolution, diet and nutrition: Where the body meets the buffet," in *New perspectives in evolutionary medicine*. Edited by W. R. Trevathan, E. O. Smith, and J. J. McKenna, pp. 55–71. Oxford: Oxford University Press.

Ulijaszek, S. J. 2000. Nutrition, infection and child growth in Papua New Guinea. *Collegium Antropologicum* 24:423–29. Available online.

Ungar, P. S. 2007. *Evolution of the human diet: The known, the unknown, and the unknowable*. Oxford: Oxford University Press.

U.S. Department of Agriculture, Office of Communications. 2003. *Agriculture fact book, 2001–2002*. Washington, DC.

U.S. Department of Health and Human Services, Assistant Secretary for Planning and Education. 2009. The 2009 HHS poverty guidelines. Washington, DC. (Available online at http://aspe.hhs.gov/poverty/09poverty.shtml)

U.S. Department of State, Bureau of Public Affairs. 2009. Global Hunger and Food Security Initiative: Consultation document. Washington, DC. (Available online at http://www.state.gov/s/globalfoodsecurity/129952.htm)

Verhoef, L. P. B., A. Kroneman, Y. v. Duynhoven, H. Boshuizen, W. v. Pelt, and M. Koopmans. 2009. Selection tool for foodborne norovirus outbreaks. *Emerging Infectious Disease* 15:31–38.

Wardle, J., and L. Cooke. 2008. Genetic and environmental determinants of children's food preferences. *British Journal of Nutrition* 99:S15–S21.

Weiss, K. M. 2004. The unkindest cup. *Lancet* 363:1489–90.

Wobber, V., B. Hare, and R. Wrangham. 2008. Great apes prefer cooked food. *Journal of Human Evolution* 55:340–48.

Wolfe, N. D., C. P. Dunavan, and J. Diamond. 2007. Origins of major human infectious diseases. *Nature* 447:279–83.

Woolhouse, M. E. J., and S. Gowtage-Sequeria. 2005. Host range and emerging and reemerging pathogens. *Emerging Infectious Disease* 11:1842–47.

Wrangham, R. W. 2009. *Catching fire: How cooking made us human*. New York: Basic Books.

Wrangham, R. W., and N. L. Conklin-Brittain. 2003. Cooking as a biological trait. *Comparative Biochemistry and Physiology,* Part A: *Molecular & Integrative Physiology* 136:35–46.

Wrangham, Richard W., James H. Jones, G. Laden, D. Pilbeam, and N. Conklin-Brittain. 1999. The raw and the stolen: Cooking and the ecology of human origins. *Current Anthropology* 40:567–94.

Yackinous, C. A., and J.-X. Guinardf. 2003. Relation between PROP (6-n-propylthiouracil) taster status, taste anatomy and dietary intake measures for young men and women. *Appetite* 38:201–9.

Yanishlieva, N. V., E. Marinova, and J. Pokorný. 2006. Natural antioxidants from herbs and spices. *European Journal of Lipid Science and Technology* 108:776–93.

Yongfa, W., Y. Yongshan, X. Jiujin, D. Ruofu, S. D. Flatz, W. Kühnau, and G. Flatz. 1984. Prevalence of primary adult lactose malabsorption in three populations of northern China. *Human Genetics* 67:103–6.

Zuckerman, M. K., and G. J. Armelagos. 2010. "The origins of the biosocial dimensions in bioarcheology," in *Handbook of Social Bioarchaeology*. Edited by S. C. Agarwal and B. Glencross. Hoboken, NJ: Wiley. (in press)

Zvelebil, M. 2000. "The social context of the agricultural transition in Europe," in *Archaeogenetics: DNA and the population prehistory of Europe*. Edited by C. Renfrew and K. Boyle, pp. 57–79. Cambridge: McDonald Institute for Archaeological Research.