CHAPTER 3

Diabesity and Darwinian Medicine

The Evolution of an Epidemic

Leslie Sue Lieberman

Lieberman's eclectic and comprehensive review of the metabolism of eating and the ecology of factors surrounding eating, including why we eat as we eat, what we eat, and why so much at each setting, is remarkable for its depth and range. Integrating proximate and ultimate explanations, she traverses across the many different disciplines, illuminating an evolutionary point of view on diabesity and the neuroendocrinology and physiology of eating, carbohydrate metabolism, and fat storage. Yet she also addresses the range of emotional factors and learned processes that explain why it is apparently so difficult for our species to get the proper proportions of food down to more optimal levels, especially in industrialized nations. She talks of the role of novelty, our need for companionship, the way friendship affects eating, as well as mood, and discusses the meaning of food during rituals and ceremonies, all of which "seduces" omnivores to eat often, even if we are less hungry than our actual behavior might suggest. She discusses the texture and colors of food and shows why, from a psychological point of view, the old expression "Your eyes are bigger than your stomach" is really true, as humans are not capable (see also Chapter 2) of judging more optimal portions using internal hunger cues. Finally, she points out the many ways in which cooking has augmented our intellectual capacities by incorporating tools needed to harvest food and how cooking expands our dietary breadth by killing potential pathogens such as parasites. Her chapter is a bountiful feast, full of all consuming tidbits of knowledge!

INTRODUCTION

Comparative and evolutionary perspectives provide lenses through which to view the growing pandemic of the co-occurring conditions of obesity and type 2 diabetes. The conditions are linked because type 2 diabetes results from metabolic processes associated

with obesity. "Diabesity," first proposed in 1980, has been used by a number of researchers (Astrup & Finer, 2000; Daly, 1994; James, 2005; Zimmet, Alberti, & Shaw, 2001).

The underlying assumption is that the diabesity pandemic is the result of a mismatch of Stone Age genotypes adapted to environments with fluctuating food availability and high energy demands and contemporary Space Age lifestyles with abundant food and low energy costs (Broadhurst, 1997; Chakravarthy & Booth, 2004; Eaton, Strassman, Neese, Elwald, Williams, et al., 2002; Neel, 1962) (see also Chapters 2, 4, 8, and 18). These ancient cognitive, behavioral, and physiological genotypes and phenotypes adapted to feast and famine and exercise and rest cycles persist today in evolutionary novel obesogenic environments (Boon, Stroebe, Schut, & Jensen, 1998; Booth, Pinkston, & Poston 2005; Brownell & Horgan, 2003; Cordain, Gotshall, Eaton, & Eaton, 1998; Hill & Peters, 1998; Liebman, 1999; Lieberman, 2006; Nestle, 2003; Speakman, 2004; Wansink, 2004a, b, c). Obesogenic landscapes coupled with our metabolic efficiency have created a perverse normalcy of excessive weight gain (Bray, 1987; Brownell and Hogan, 2003; Kuzawa, 1998; Nestle, 2003; Popkin, 2001; Rolls, 2003; Speakman, 2004; Spurlock, 2005).

This chapter examines the nexus of human biology and behavior and characteristics of contemporary environments that are conducive to obesity and type 2 diabetes. A Darwinian medical perspective is used to suggest approaches that differ from current cognitive–behavioral interventions to stem the growing pandemic of diabesity and its adverse health outcomes.

Definitions and Diagnostic Criteria

Diabetes is a group of metabolic diseases characterized by high blood sugar or hyperglycemia. Type 1 diabetes, previously called insulin-dependent diabetes or juvenile diabetes, is an autoimmune disease with usual onset in childhood. It affects about 5% of all individuals with diabetes. Type 2 diabetes, previously called non–insulin-dependent or adult-onset diabetes, usually occurs in adults who are obese but has been increasing among overweight children and adolescents. It affects approximately 90% of all individuals with diabetes. The remaining 5% of individuals with diabetes include women with gestational diabetes caused by metabolic alterations in pregnancy and people with diabetes caused by genetic abnormalities, infectious diseases, and trauma (American Diabetes Association, 2004a; Centers for Disease Control, 2005b). The focus of this chapter is type 2 diabetes.

Diagnostic Criteria for Diabetes

Chronic hyperglycemia or a high blood sugar level is the defining hallmark of diabetes. It can result from defects in pancreatic insulin secretion and/or insufficient insulin action in muscle or adipose (fat) tissue. The American Diabetes Association (2004a, b) defines type 2 diabetes as a random glucose $\geq$ 200 mg/dl (11.1 mmol/l) or a fasting plasma glucose $\geq$ 100 mg/dl (5.6 mmol/l) or a 2-hour oral glucose tolerance test with a plasma glucose $\geq$ 200 mg/dl (11.1 mmol/l). Individuals with diabetes may have the following symptoms: frequent urination, hunger, thirst, weight loss, blurred vision, and skin itchiness, among others (American Diabetes Association, 2004a). Insulin levels may be high, low, or normal. Neurological and vascular damage can occur throughout the body, leading

to kidney failure, blindness, and lower limb amputations (Zimmet, Alberti, & Shaw, 2001). Diabetes currently affects more than 20 million American adults (American Diabetes Association, 2004a, b; 2006). More than 85% of individuals with type 2 diabetes are significantly overweight, obese, or have a recent history of obesity (American Diabetes Association, 2004; Centers for Disease Control, 2002).

Diagnostic Criteria for Obesity

Obesity is the primary risk factor for type 2 diabetes and related conditions such as metabolic syndrome, syndrome X, insulin resistance metabolic syndrome, and impaired glucose tolerance. All these conditions are characterized by elevated insulin and glucose levels. Medically, obesity is defined as an excess amount of body fat (National Institute of Diabetes, Digestive and Kidney Diseases, 2001), but body fat is rarely measured by clinicians or researchers. The diagnosis of obesity is based on standard values for waist circumference, hip/waist circumference ratio, body weight for age, sex, and height, and body mass index [(BMI) = wt (kg)/ ht (m^2)]. BMI is widely used in epidemiological and clinical research. BMI has the following classification system: underweight (less than 18.5), normal weight (18.5–24.9), overweight (25–29.9), obese class 1 (30–34.9), obese class 2 (35–39.9), and morbid obesity (40 and greater). The BMI classification system applies to both males and females (National Institute of Diabetes, Digestive and Kidney Diseases, 2001).

In addition to total body fat, the distribution of fat in the body is involved in the regulation of energy use and risk for chronic diseases (Bouchard & Johnson, 1988; Bray, Bouchard, & James, 1998). A centripetal distribution of fat (i.e., apple shape) is indicated by a large waist circumference and large stores of fat in the abdominal viscera. Visceral fat confers a greater risk for cardiovascular disease and type 2 diabetes than large deposits of fat on the hips and thighs (i.e., pear shape) (Bouchard & Johnson, 1988; Bray, Bouchard, & James, 1998).

There are a number of ways of measuring body fat. Skinfold (fatfold) measurements are taken at standardize sites on the body (e.g., mid-upper arm over the triceps muscle, medial mid-thigh) and used in population-, age-, and sex-specific equations to estimate total body fat. Other techniques that measure body fatness, such as dual-energy X-ray absorptiometry (DEXA), underwater or hydrostatic weighing, bioelectrical impedance, near infrared interactance, computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound, require expensive equipment, are time consuming, and are rarely used in public health contexts (Heymsfield, Lohman, Wang, & Going, 2005; University of Georgia, 1998).

In summary, type 2 diabetes and related conditions, (for example, metabolic syndrome) are defined by high levels of blood glucose (i.e., hyperglycemia). In contrast to the direct measures of blood glucose, obesity or an excess amount of body fat is inferred from physical measurements of body size and mass, such as height and weight. Because body composition is not measured, BMI classification may not accurately reflect fat stores for women with low bone mass or males with high muscle mass. However, BMIs of 30 or greater are used worldwide to define obesity and report its prevalence (Figure 3-1).

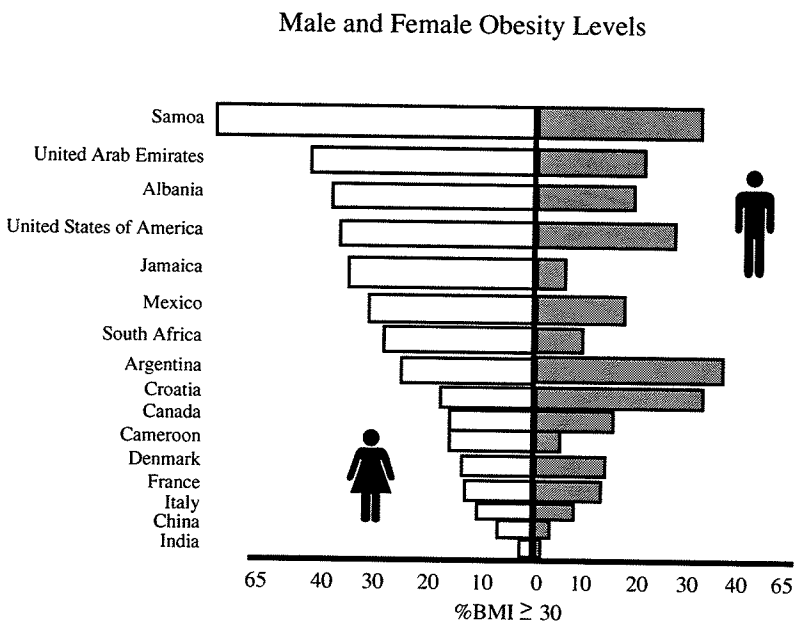

FIGURE 3-1 Prevalence of obesity in selected countries. (From International Obesity Task Force 2006 www.itof.org. Figures from late 1990s to 2002.)

METABOLIC MECHANISMS REGULATING OBESITY AND DIABETES

Humans have a complex physiological system used to maintain energy balance. Hormonal and neurological mechanisms control appetite (i.e., hunger and satiety), food consumption, and energy and nutrient storage in organs and tissues (Wynne, Stanley, McGowan, & Bloom, 2005). There are multiple pathways with significant redundancy that favor both a drive to eat and a low sensitivity to satiety (Melanson, 2004; Wynne, Stanley, McGowan, & Bloom, et al. 2005). Additionally, humans have an extraordinary ability to store fat, a good ability to store proteins, and, with moderate physical activity, a daily turnover of carbohydrate stores (Bray, 1987). Natural selection has favored fat storage and metabolic thriftiness in infants, children, and pregnant and lactating women, especially when energy intakes are low and/or energy demands are high due to illness burden, growth and development, pregnancy, and lactation (Kuzawa, 1998; Lieberman, 2003; Neel, 1962, 1999a, b; see also Chapter 18).

Energy balance is closely regulated despite huge variations in daily food intake and energy expenditure (Bray, 1987; Bray, et al. 1989). Bray calculated that the "average" nonobese, adult American male consumes approximately 1 million kcal a year. With only a 10% alteration in either energy intake or expenditure, the result can be a 30-lb.

(13.6-kg) weight gain in a single year. Even a tiny surfeit of 5 kcal a day can result in a 24-lb. (11-kg) weight gain over a 40-year time span (Bray, 1987). During most of human history changes in caloric intake or activity levels compensated for these small surfeits (Jenike, 2001; Leonard, 2001). However, contemporary urban and suburban environments provide few incentives or opportunities to maintain a "normal" body weight so that populations around the world are now experiencing unprecedented increases in obesity (Brownell & Horgan, 2003; International Obesity Task Force, 2004).

Contemporary environments are providing the means—high food availability and low-energy-expenditure lifestyle—and evolution has provided the mechanisms of efficient energy storage and metabolic thriftiness that are driving the diabesity pandemic (Eaton, Eaton, & Konner, 1999, Lieberman, 2006).

The Regulation of Appetite

The regulation of food intake involves physical factors (e.g., gastric distension and emptying), gut enzymes and other peptides (e.g., amylase, cholecystokinin, ghrelin), neuropeptides (e.g., dopamine, neuropeptide Y (NPY), pancreatic peptide (PP) and hormones (e.g., insulin, glucagon, leptin, ghrelin). Table 3-1 presents the key peptides and hormones involved in the internal or endogenous control of appetite. Neuropeptides are chemicals that are found in the brain that aid in communication among neurons (Wikipedia, 2006b). Hormones are chemical substances that are produced by organs and tissues and secreted into the blood. They influence the activities, including metabolism, of other organs and tissues (Wikipedia, 2006a).

TABLE 3-1 Internal (Endogenous) Appetite Control

Hormone or neuropeptide	Source	Effect on appetite
Enhances appetite		
Neuropetide Y (NPY)	Hypothalmus	↑
Agouti-related peptide (AG RP)	Hypothalmus	↑
Orexin	Hypothalmus, stomach, intestine, pancreas	↑
Dopamine	Hypothalmus, central nervous system	↑
Resistin	Adipocytes	↑
Ghrelin	Stomach, intestine	↑
Depresses appetite		
Insulin	Pancreas	↓
Pro-opimelanocortin (POMC)	Hypothalmus	↓
Cocaine & amphetamine regulated Transcriptase (CART)	Hypothalmus	↓
Endocanabinoids	Hypothalmus	↓
Leptin	Adipocytes, stomach, placenta	↓
Adiponectin	Adipocytes	↓
PP-fold peptides PYY, PP, NPY	Intestine, pancreas (PP)	↓
Cholecystokinin (CCK)	Intestine	↓
Proglucagon, GLP-1, OXM	Pancreas, intestine, central nervous system	↓

Source: Wynne et al., 2005.

Insulin is a hormone essential in regulating carbohydrate and fat metabolism by facilitating energy storage in cells (Porte, Baskin, & Schwartz, 2002). It is produced by the pancreas and secreted in response to eating and the concentration of glucose in the blood. Insulin levels reflect the sensitivity of muscle and adipose tissue to the insulin-mediated transport of glucose into cells. This sensitivity is determined to a large extent by the total amount of body fat, and particularly, the amount of visceral or abdominal fat. This fat is more metabolically active than fat located in other parts of the body (Wynne et al., 2005). When adipose tissue cells (i.e., adipocytes) fill with triglycerides and fatty acids, they become resistant to the transport of glucose, causing a rise in blood levels of glucose (i.e., hyperglycemia) that trigger a rise in insulin secretion. If the high insulin levels are unable to reduce blood glucose concentrations to normal levels, then individuals are considered to have insulin resistance or "prediabetes" (ADA, 2004b). Although insulin primarily regulates carbohydrate metabolism, it also has roles in increasing fat storage and decreasing the breakdown of muscle protein to make glucose (Wynne et al., 2005). If glucose or its stored form, glycogen, are not used for fuel, then fat is oxidized or "burned" for fuel and ketones are produced. Using ketones to meet energy demands prevents the muscle protein from being used to meet energy needs and preserves muscles that are needed for foraging and other activities. Ketones also decrease hunger and elevate mood (Cahill and Veech, 2003).

Insulin receptors are widely distributed in the brain. Insulin has a direct effect on the hypothalamus and acts to decrease food intake and body weight. However, experiments with administration of insulin causes a drop in blood glucose levels, which stimulates eating (Nicolaidis & Rowland, 1976). When this occurs, the counterregulatory hormone glucagon is produced by the pancreas and facilitates the release of energy stored as glycogen in the liver. This results in a rise in blood sugar to re-establish glucose homeostasis (Wynne et al., 2005). Insulin responses linking peripheral sensitivity to the central nervous system are mediated by interactions with other hormones and neurotransmitters (e.g., leptin, NPY) (Wynne et al., 2005).

The hypothalamus has both hunger and satiety centers that influence feeding behaviors. Specific hypothalamic nuclei are the targets of neuropetides and hormones (Wynne et al., 2005). These peptides are involved in energy homeostasis but also have pleiotropic or other effects. For example, the hypothalamic peptide pro-opiomelanocortin (POMC) is decreased by fasting and increased in the presence of leptin, a hormone secreted by adipocytes (fat cells). Mutations in the POMC gene or abnormalities in processing POMC result in early onset obesity, adrenal insufficiency, and red hair in humans (Wynne et al., 2005).

Eating activates many central nervous system neuropeptides that create pleasant sensations and act as endogenous rewards. These peptides include dopamine, endocannabinoids, and serotonin (Wynne, et al., 2005). Other hormones and neuropeptides produced by the stomach, intestines, adipocytes, pancreas, and other organs respond to eating and influence these reward pathways (Wynne et al., 2005). For example, leptin has a direct influence on the hypothalamic reward pathways (Fulton, Woodside, & Shizgal, 2000).

The links between obesity and diabetes involve the balance of several adipocyte signaling proteins or neuropeptides, including leptin, adiponectin, and resistin. After a meal, adipocytes release leptin, which acts on the appetite-control center activating the

pro-opiomelanocortin (POMC) and cocaine and amphetamine regulated transcript (CART) neurons in the hypothalamus and creating a state of satiety or "fullness." People (may) stop eating. Mice lacking the gene for leptin production or lacking leptin receptors overeat and become obese. When leptin-deficient mice are given leptin, they eat less and lose weight (Halaas et al., 1995). However, mice and rats are more sensitive to leptin deficiency that promotes weight gain than they are to high levels of leptin that decrease appetite and promote weight loss (Levin & Dunn-Meynell, 2002). This imbalance in leptin sensitivity may reflect selection for high sensitivity in dietary environments of unstable or inadequate food availability and a diminished regulatory role for leptin in environments of food abundance (Wynne et al., 2005). Although leptin was the basis for the development of antiobesity drugs, the administration of leptin has not been an effective approach to appetite control or weight loss in humans (Wynne et al., 2005).

Adiponectin is produced by the adipocytes in response to food restriction and weight loss in laboratory rodents and humans (Hu, Liange, & Spiegelman, 1996). It decreases insulin resistance and increases muscle cell metabolism (Wynne et al., 2005). If adipocytes fail to produce adiponectin, a decreasing adipocyte sensitivity to feeding and energy intake could lead to overconsumption of food and obesity. Because some overweight diabetic patients are deficient in adiponectin, it has become a new target for development of obesity drugs to boost adiponectin levels (Wynne et al., 2005).

Resistin is a recently identified adipocyte peptide that decreases insulin resistance and promotes fat storage. Levels of resistin, unlike adiponectin, increase with obesity and decrease with weight loss (Ahima, Qi, & Singhal, 2006). Drugs that decrease resistin also increase adiponectin. Researchers are actively seeking other adipocyte peptides.

The gastrointestinal (GI) tract also produces many neuropeptides that respond to energy homeostasis by regulating hunger and satiety. (Table 3-1) Ghrelin is a protein that acts in the hypothalamus to stimulate appetite. It is produced primarily by the stomach in response to fasting, and levels decrease after eating (Cummings et al., 2001). In humans, ghrelin levels, like leptin, are high in the morning and low at night, which is appropriate for diurnally active humans (Cummings et al., 2001). Some obese individuals have low ghrelin levels or demonstrate a slow response in the decline of ghrelin levels with eating. Therefore, they continue to eat increasing their risk of excessive calorie intake and obesity (Wynne et al., 2005).

Other GI track neuropeptides include the PP-fold peptides (PYY, PP, NPY) secreted by cells in the intestine and pancreas. These peptides, like ghrelin, decrease rapidly after eating and produce satiety by acting in the hypothalamus. Some obese humans have low levels of PYY and a slow response to food intake (Arosio et al., 2003; Challis et al., 2003). Rodents and humans given PYY and PP reduce their food intake, lose weight, and improve their control of glucose levels (Challis et al., 2003). However, acute stress has been shown to override the satiety effects of PYY (Wynne et al., 2005).

Cholecystokinin (CCK) and the proglucogon products (GLP-1, OXM) are hormones produced primarily in the intestine and released in response to eating. They increase satiety and decrease food intake (Wynne et al., 2005). CCK stimulates the release of enzymes from the pancreas and gallbladder, increases intestinal motility, slows gastric emptying, and decreases food intake (Wynne et al., 2005). CCK is also found in the brain, where it acts as a neurotransmitter involved in the regulation of reward behavior, memory, anxiety, and satiety (Crawley & Corwin, 1994). CCK and GLP-1 normalized blood

glucose levels when administered to people with diabetes (Nauck et al., 1993). Administration of OXM reduces ghrelin levels (Cohen et al., 2003)

In summary, food intake and metabolism are regulated by complex interactions of many peptides produced by and acting in the central nervous system, GI tract, and other organs. Because the prime regulatory mechanisms are sensitive to hunger and relatively insensitivity to satiety, eating is favored over restricted food intake. This makes evolutionary sense in environments with fluctuating and uncertain food supplies. The regulatory system promotes eating when you can, eating as much as you can, and storage of energy for times when food is not available. The identification of adipocyte hormones and neuropeptides and the elucidation of their mechanisms of action on appetite provide helpful insights into the development of new therapeutic products and molecular and cellular targets that have an evolutionary basis rooted in dietary constraints present throughout most of hominin evolution (Lee & DeVore, 1968; Ungar & Teaford, 2002).

Other Biological Factors Implicated in the Etiology of Diabesity

In addition to the endocrine and GI systems involved in diabesity, recent research has found that obese individuals suffer chronic, systemic low-grade inflammation. Adipocytes secrete a number of different cytokines (e.g., interleukin-6, tumor necrosis factor-α), which are a class of proteins that promote inflammation (Lee and Pratley, 2005). Moreover, obesity increases the number of macrophages (i.e., immune cells associated with inflammation that engulf and digest cellular debris) in adipose tissue. Both cytokines and macrophages increase with psychosocial stress (Black, 2006). Cellular inflammation and chronic activation of the immune system subsequently lead to obesity, insulin resistance, and type 2 diabetes (Lee and Pratley, 2005). Reducing fat stores decreases the inflammatory response and reduces insulin resistance (Black, 2006).

Cutting-edge research points to intestinal microorganisms or microflora, which are implicated in obesity because of their heightened ability to extract calories from food, particularly polysaccharides (i.e., sugars found in plant foods). Some bacteria also enhance fat deposition (Backhed, Ding, Wang, Hooper, Koh, et al., 2004; Ley, Blackhed, Turnbaugh, Lozupone, Knight, et al., 2005). Experiments with mice demonstrate that the presence of Firmicute bacteria in the gut is correlated with obesity and insulin resistance (Backhed et al., 2004; Henig, 2006; Ley et al., 2005). If research on humans confirms this association, then gut microorganisms will present a new target for therapeutic interventions (Backhed et al., 2004; Henig, 2006).

COGNITIVE AND BEHAVIORAL REGULATION OF APPETITE AND EATING

Appetite and food intake are regulated not only by biological factors but also by psychological states (e.g., depression, anxiety) (Boon et al., 1998; Polivy & Herman, 2006), social contacts (Wansink, 2004b), beliefs (Rozin, 2000, 2005) cultural practices and habits (Kittler & Sucher, 2004), hedonics (e.g., taste) (Drewnowski, 1997; Johns & Keen, 1985), and atmospherics (e.g., lighting, music) (Wansink, 2004a, b). Vision, olfaction, taste of edible stimuli, and even thinking about food initiate the cephalic phase of digestion with

the secretion of saliva, gastric acid, gastrointestinal hormones, and pancreatic enzymes (Katschinski, 2000).

Although individuals voluntarily diet to lose weight for health and esthetic reasons, both experimental and naturalistic observations document that humans are very stressed by voluntary food restriction when food is available (Polivy & Herman, 2006; Rogers, 1999). When individuals do restrict food, they rebound with overconsumption and "yo-yo" between weight loss and regain (de Lauzon-Guillain et al., 2006; Hill, 2004; Polivy & Herman, 2006). Cognitive control of eating is a demanding task, especially in environments with persistent cues to eat. These stimuli (for example, the sight and smell of food) lead to the disinhibition of eating behaviors in which people lose control, "go off their diets," and sometimes develop disordered eating patterns (e.g., binging and purging) (Hill, 2004; Rogers, 1999; Polivy & Herman, 2006). Polivy and Herman (2006) argue that restrained eating and chronic dieting are behaviors that defy our evolutionary heritage and that restrained eating or "calorie conservation" makes evolutionary sense only when food is in limited supply. Self-control when the food supply is limited may be helped by the tendency to decrease intake when there is limited variety (i.e., sensory-specific satiety) (Raynor & Epstein, 2001). Our evolutionary history reflects food deprivation by necessity and not by choice so that when food is available humans are biologically and psychologically prepared to eat. Polivy and Herman conclude that dieting in obesogenic environments is a "recipe for overeating and distress" (2006, p. 34).

Psychosocial distress increases cortisol, a hormone that raises blood sugar levels and increases the storage of abdominal fat (Melanson, 2004). Researchers have begun to examine the role of stress in populations with high rates of obesity and diabetes. For example, diabesity has been linked to stress-inducing historical, social, and economic circumstances reflecting discrimination and health disparities among indigenous peoples, including Native Americans (Ferrier and Lang, 2006). Many good examples of historical trauma, genocidal and ethnic "cleansing" campaigns, and blaming-the-victim policies and practices are documented in *Indigenous Peoples and Diabetes. Contemporary Empowerment and Wellness*, edited by Ferrier and Lang (2006).

Stress is related to a rise in the number of people with sleep deprivation and sleeping disorders (Keith et al., 2006). Short habitual sleep duration (e.g., 5 hours vs. 8 hours per night) is associated with reduced leptin and elevated ghrelin levels (Taheri, Lin, Austin, Young, & Mignot, 2004). People with shorter sleep duration have higher BMIs due to increased appetite and energy intake (Keith et al., 2006). They also suffer a higher prevalence of sleep apnea (i.e., temporary cessation of breathing), wake frequently during the night, and are fatigued with low activity levels during the day (Keith et al., 2006).

Other Behavioral and Lifestyle Factors Contributing to Diabesity

Other behavioral factors may be contributing to the diabesity pandemic. Keith and colleagues (2006) have generated a long list of potential contributors. These include the declining number of people who are smoking tobacco. Smoking suppresses appetite, reduces food intake, and increases metabolism, thereby reducing the risk of obesity. Temperature regulation of microenvironments (e.g., air-conditioned cars) reduces energy expenditures needed to maintain comfortable body temperatures. Pharmaceutical use has increased, and some drugs are linked to weight gain (e.g., injected insulin, antipsychotic drugs). Endocrine disruptors (e.g., insecticides, herbicides, detergents, resins, plasticizers) that may alter fat metabolism are found in food and water.

In addition, there are a number of recent demographic trends that may be contributing to the pandemic. For example, there is an increase in weight with age, and people are living longer. With declining birth rates in many countries, the middle and older age segments of populations are increasing relative to the younger component (Popkin, 1994, 2004). An increasing percentage of the U.S. population is comprised of obesity-tolerant ethnic groups who value larger body sizes (Brown & Konner, 1987; Kittler & Sucher, 2004). People choose mates who look like themselves (i.e., positive assortative mating) so that large-bodied people marry other large-bodied people, providing a potential genetic and social basis for increases in the proportion of the population who are large-bodied and/or obese (Keith et al., 2006). The etiology of the diabesity pandemic is clearly multifactorial.

EVOLUTIONARY MECHANISMS, THRIFTY GENOTYPES, AND PHENOTYPES IN OBESOGENIC ENVIRONMENTS

Although this chapter focuses on the contemporary environment, it is important to review the theoretical underpinnings of the current ideas about the deleterious nature of genotypic thriftiness. Geneticist J. V. Neel proposed a thrifty genotype for glucose utilization among Native Americans as an evolutionary explanation for the high prevalence of type 2 diabetes seen in those populations (see Chapters 4, 17, 18, and 19; Neel, 1962, 1999a, b). He hypothesized that a feast-and-famine existence conferred a selective advantage and increased reproductive fitness for those individuals who had the ability to release insulin quickly, to thriftily store energy during times of food abundance, and to efficiently utilize energy deposits during dietary deprivation. Shifts to modern lifestyles rendered a once adaptive genotype detrimental, leading to obesity and type 2 diabetes (Neel, 1962, 1999a, b).

Many authors have expanded on the thrifty gene hypothesis for other populations with additional selective pressures: cold stress from water and long oceangoing voyages for Pacific Islanders, extreme cold stress for Eskimo and Aleut populations, and high seasonal energy demands under conditions of slavery for African Americans (Lieberman, 2003). In conclusion, the thrifty genotype hypothesis does not fit well for all cases, but it raises the possibility of a well-supported genetic explanation for the proneness to obesity and the high prevalence of type 2 diabetes for many populations. In addition, other microevolutionary processes (e.g., founder effects for island populations) and recent dietary and lifestyle changes have helped to increase the prevalence of "diabesity."

Thrifty Phenotypes and Prenatal Programming

Alternatively, David Barker has proposed thrifty phenotypes that develop through processes of prenatal "programming" in response to conditions such as high circulating glucose levels and nutrient deficiencies in the uterine environment (Barker, 1998a, b, 1999, 2004, 2006). During pregnancy nutrient supply and demand vary depending on

a number of factors: maternal body composition, maternal dietary intake, placental blood flow, and the fetal genotype (Barker, 1998a, 2004; de Moura & Passos, 2005; Kuzawa, 2005, see also Chapter 18). Many researchers have substantiated that low birth weight and/or small body size or thinness in full-term infants are associated with an increased risk for obesity, type 2 diabetes, insulin resistance or metabolic syndrome, increased blood pressure, coronary heart disease, hypertension, and stroke when coupled with moderate to high weight gains in infancy, childhood, and later in life (Barker, 1998b, 2004, 2006; de Moura & Passos, 2005, Hales & Barker, 2001, Kuzawa, 2004, 2005; Plagemann, 2006). These associations have been established in populations around the world including British men (Hales et al., 1991; Baker 1998a, b), Asian Indians (Yajnik, 2004; Yajnik et al., 1995), Filipino children (Kuzawa, 2004), Chinese infants (Jie et al., 2000), Native Americans (Hanson, Imperatore, Bennett, & Knowler, 2002), and Mexican Americans (Valdez et al., 1994).

Barker postulates that low-birth-weight babies have metabolically thrifty mechanisms for fat storage and reduced rates of glucose oxidation in insulin-sensitive target tissues (Barker 1999, 2005; Barker et al., 1993; Yajnik, 2004; Yajnik et al., 1995). Adverse uterine environments alter many hormones involved in energy homeostasis, including leptin and cortisol. In addition, catabolic steroids rise with undernutrition, limiting lean body mass. In contrast, high-birth-weight infants or exposure in utero to excess glucose levels in diabetic mothers may preferentially decrease (i.e., downregulate) their sensitivity to glucose, predisposing them to insulin resistance, metabolic syndrome, obesity, and type 2 diabetes (Kuzawa, 1998). Barker calls for preconception, prenatal, and early life interventions to limit later life chronic diseases (Barker, 1998a, 2005). These preventive interventions would affect intergenerational health outcomes.

Thrifty Energy Cycles and Physical Activity

Chakravarthy and colleagues (Booth et al., 2002; Chakravartthy and Booth, 2004) argue that the combination of continuous food abundance and physical inactivity disrupts the evolutionarily programmed biochemical cycles that were adaptive for the feast/famine and physical activity/rest cycles of past lifeways. They view sedentary lifestyles and the reduction in energy expenditure as key conditions leading to a disruption of normal cycling of carbohydrate and fat stores, thus altering the insulin sensitivity of muscle and adipose tissues and production and use of metabolic regulatory peptides. Obesity and diabetes are referred to as "inactivity-mediated diseases," where neither glycogen stores in muscle (about 900 kcal) nor tryglycerides stored in adipose tissue (about 120,000 kcal) are routinely expended (Chakravarthy and Booth, 2004). Sedentary lifestyles result in an "unabated storage of fuel without the stimulus for its utilization" (Chakravarthy and Booth, 2004, p. 10).

Data from many sources support an argument that more attention should be paid to the overall reduction in energy expenditure and the frequency and intensity of activity in the etiology of diabesity (Campbell & Cajigal, 2001; Cordain et al., 1998; Jenike, 2001; Leonard, 2001). For example, endurance-trained athletes used as a proxy for hunting and foraging populations exhibit very efficient use of triglycerides or fatty acids for energy and use muscle glycogen sparingly, improving the potential for survival (Booth et al., 2002; Chakravarthy and Booth, 2004). Conversely, bed rest is

associated with increases in plasma glucose, insulin resistance, and poor use of energy stores.

Exercise increases skeletal muscle mitochondria, which enhance cellular metabolism and insulin sensitivity. There is a low prevalence of type 2 diabetes in contemporary populations with economies demanding high levels of physical activity such as hunting and foraging, horticulture, and pastoralism (Diamond, 2003). Even the extremely high caloric intake of U.S. lumberjacks (4500–8000 kcal/day) balanced by energy expenditures results in an average BMI of only 25.5 (Lehtonen & Viikari, 1978). In summary, some researchers make a strong case for physical activity as a "core catalyst" to regulate the physiology of energy balance and call for an increased focus on physical-activity interventions to control the pandemic of diabesity (Chakravarthy and Booth, 2004; Eaton & Eaton, 2003; Eaton et al., 2002).

THE SCOPE OF THE PROBLEM: GLOBAL OBESITY PREVALENCE

The epidemic of overweight and obesity has now reached more than 1.6 billion persons, and in the United States two thirds of adults are overweight or obese, with some segments of the adult population reaching a nearly 80% prevalence (Kuczmarski, Flegal, Campbell, & Johnson, 1994; World Health Organization, 2006). The prevalence of overweight and obesity among adults and children has grown threefold or more since 1980 in many developed and developing countries (International Obesity Task Force, 2004; Kuczmarski et al., 1994; Lobstein, Baur, Uauy, & International Association for the Study of Obesity [IASO], 2004; Wild, Roglic, Green, Sicree, & King, 2004). In some small island populations, for example, the Samoans of the South Pacific, 75% are obese. In other countries such as China and India, where obesity rates are low (1–5%), the number of obese individuals is high because the populations are very large (International Obesity Task Force, 2004; WHO, 2000a, b). These countries have a growing public health burden from obesity and continue to cope with segments of the population that are underweight and undernourished (Popkin, 1994, 2004) (see Figure 3-1).

The U.S. National Health and Nutrition Examination Surveys (NHANES) showed a 40% increase in the number of overweight adults and 100% increase in the number of obese adults in the 25-year period between the 1976–80 and the 1999–2002 surveys (Flegal, Carroll, Ogden, & Johnson, 2002). Recent estimates are that more than two thirds of children 10 years of age and older who are obese will remain obese as adults (Magarey, Daniels, Boulton, & Cockington, 2003).

The obese also are getting fatter. The fastest growing group in the United States are the morbidly obese, whose BMI is $\geq$50. There are 9 million U.S. adults who are at least 100 lb. (45 kg) overweight (Hedley et al., 2004; Anonymous, 2005).

In 2000 the World Health Organization estimated that there were 22 million overweight children and adolescents worldwide, representing a remarkable increase in obesity within the last decade. A more recent estimate classifies 20 million children age 5 years and younger as overweight (World Health Organization, 2006). A number of studies indicate that 15–30% of children in developed countries are overweight (de Onis & Blossner, 2000; Lobstein et al., 2004) and more boys than girls are overweight (Hedley

et al., 2004). Kim and colleagues documented a 73% increased in overweight babies in the United States in the period 1980–2001 (Kim et al., 2006). The prevalence of overweight and obesity is higher among Hispanic/Latino and African American/black compared to non-Hispanic white youth (Hedley et al., 2004; Kim et al., 2006). There are no geographic regions untouched by the pandemic of overweight and obesity (Bellenger & Bray, 2005; Ford, Giles, & Deitz, 2002; Popkin, 1994, 2001, 2004; Popkin & Gordon-Larson, 2004, Martorell, Khan, Hughes, & Gummer-Strawn, 2000).

THE SCOPE OF THE PROBLEM: DIABETES GLOBAL PREVALENCE

In 2003 the worldwide estimate for the number of adults with type 2 diabetes was 194 million compared to 135 million in 1995 and 30 million in 1985 (Figure 3-2). The projected global estimate for 2025 is 333 million (International Diabetes Federation, 2003) and for 2030 it is 366 million adults with diabetes (Wild et al., 2005). Wild and colleagues estimate that from 2000 to 2030 the worldwide population of adults will increase 37% but the percentage change in the number of adults with diabetes will be 114% (Figure 3-2). The *Diabetes Atlas 2003* contains estimates of diabetes prevalence for 212 countries within the seven regions of the International Diabetes Federation (International Diabetes Federation, 2003). The lowest regional prevalence (1.2%) is in sub-Saharan Africa, and the highest regional prevalence estimates are in the eastern Mediterranean, Middle East, and North America (7.8%). Many small island populations have the highest prevalence

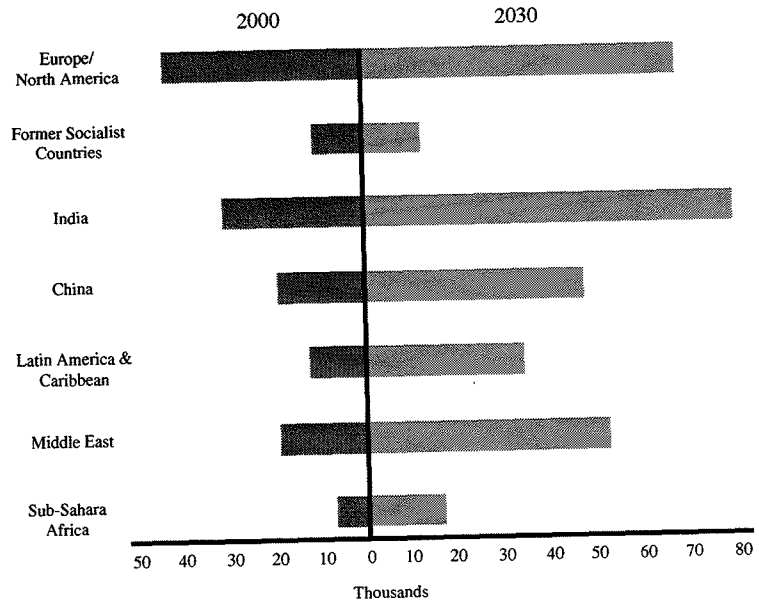

FIGURE 3-2 Worldwide prevalence of type 2 diabetes. (From Wild et al., 2004.)

estimates of type 2 diabetes, for example: Mauritius (15%), Barbados (13.2%), and Tonga (11.5%) (International Diabetes Federation, 2001, 2003). Currently India, China, and the United States account for nearly half of the world's population with type 2 diabetes (International Diabetes Federation, 2003).

Countries and regions with the greatest increases in diabetes prevalence are undergoing rapid demographic, lifestyle, and nutritional transitions characterized by increases in the number of people over 65 years of age, the proportion of the population living in urban areas, and increased access to prepared foods and public transportation (Drewnowski & Popkin 1997; IASO International Obesity Task Force, 2004; Popkin 2001; 2004; Popkin & Larson, 2004; Wild et al., 2005).

The United States in 2005 had an estimated 20.8 million individuals with diabetes (Centers for Disease Control, 2005b). There is wide variation in the estimated prevalence among ethnic groups within the United States: 15.1% American Indians/Alaskan natives, 13.3% non-Hispanic blacks, 10.2% Hispanic/Latino Americans, and 8.7% non-Hispanic whites (Centers for Disease Control, 2005b; International Diabetes Federation, 2003; Mokdad et al., 2000, 2001). Type 2 diabetes is increasing rapidly among overweight and obese children, especially among children from populations with a high prevalence of adults with diabetes. Six million (25%) U.S. children are overweight or obese, and one quarter of this group are estimated to have impaired glucose tolerance or "prediabetes" (Miller, Rosenbloom, & Silverstein, 2004). Eighty-five percent of children with type 2 diabetes were overweight or obese at the time of diagnosis (Young & Rosenbloom, 1998). These diabesity trends are expected to continue because contemporary environments are conducive to a surfeit of calories and fat stores. These aspects of our obesogenic environment are explored in the remainder of this chapter.

CHARACTERISTICS OF THE DIETARY OBESOGENIC ENVIRONMENT

There are a number of aspects of contemporary environments and lifestyles that predispose humans to excessive fat storage and type 2 diabetes. Some of the physiological/metabolic and cognitive/behavioral factors have been described previously in this chapter. The remainder of this chapter will focus on the dietary environment and food-related behaviors. The hallmarks of global dietary modernization are increased energy intake with processed energy-dense foods, increased quantity, and increased ingestion frequency. These dietary changes are coupled with behaviors that reduce energy expenditure. For example, television viewing is a low-energy-expenditure activity (i.e., couch potato), and it is frequently associated with increases in energy intake (e.g., potato chips) (Wansink, 2004a).

CONTEMPORARY DIETS: MACRO- AND MICRONUTRIENTS

S. Boyd Eaton and colleagues have extensively researched and written about Paleolithic diets and their relationship to contemporary diets and health outcomes (Eaton et al., 2002, Eaton & Eaton, 1999; Eaton, Shostak, & Konner, 1988). They note that there is

very little in common between Paleolithic diets composed primarily of wild plant and animal foods and contemporary diets. Although geographic variability was high, Paleolithic diets were generally lower in fat and considerably higher in fiber than diets today. The fats in Paleolithic diets were primarily long-chain polyunsaturated fatty acids. Carbohydrates were chiefly from fruits (e.g., berries, figs) and vegetables (e.g., squashes, tubers) and consumed with little processing. In contrast, for most contemporary populations, macronutrients and calories are from processed grains, fatty domesticated animals, high-fat dairy products, and refined sugars. Contemporary, globalized diets are "fat rich and fiber poor" (Drewnowski & Popkin, 1997; Lieberman, 2003; Popkin, 2001).

Carbohydrate foods in contemporary diets are processed and refined. They often have high glycemic indexes. The glycemic index is a measure of the metabolic impact of a food or meal and the resulting increase in insulin and plasma glucose concentrations (Foster-Powell, Holt, & Brand-Miller, 2002). The enhanced insulin response promotes the storage of energy and increased adiposity. Globalized fast food meals often have a high glycemic load. They are a combination of low bulk, fiber-poor starches, fatty or oily foods (e.g., French fries), and highly sugared drinks. High fructose corn syrup used to sweeten both foods and beverages does not produce a quick insulin release so people do not experience satiation and may continue to eat for a longer period of time, increasing their intake of calories (Melanson, 2004).

Food-processing technologies are used to produce foods that have desirable characteristics, including sweet taste and smooth or crunchy textures. Fat produces a smooth texture at room temperature (e.g., butter) and when it is cold (e.g., ice cream). When fat

TABLE 3-2 Food Qualities and Atmospherics

Food Qualities and Serving Characteristics
Variety (color, taste, shape)
Attractiveness (shape, color, arrangement)
Amount (large serving sizes, stockpiles)
Presentation (jumbled rather than in discrete piles)
Environmental (Exogenous) Appetite Control Increasing Food Intake
Containers & Serving Dishes (large containers, dishes and bowls; short; wide glasses, visible food)
Atmospherics
Eating effort (low effort-fingers, forks, bit sizes, shelled nuts, easy-open packaging)
Music (slow prolongs eating, fast increases rate of eating)
Aromas (initially pleasant increases eating)
Comfortable seating (increases eating time)
Low lighting (disinhibits and increases eating time)
Number of people (the number of people eating at a table is positively correlated with the amount of food consumed per person)
Eating norms (people conform to expectations and observations of others)
Distractions (TV, games, conversation, other distractions)

Source: Wansink, 2004.

is subjected to high temperatures, it produces a crunchy texture (e.g., French fries, fried chicken) (Drewnowski, 1997; Spurlock, 2005). In addition to taste and texture, food technology has produced foods with high energy and nutrient density and low bulk, facilitating increased consumption. In addition, there are many other characteristics of food that compel people to overeat (e.g., variety, portion size) and characteristics of the obesogenic environment that promote eating (e.g., food advertisements in print and electronic media, low lighting in restaurants, bulk purchases, and stockpiling) (Wansink, 2004a, b) (see Table 3-2).

GLOBALIZATION OF FOODS: ENORMOUS VARIETY

The globalization of diets has increased local dietary diversity, which is an important factor increasing consumption (Rolls et al., 1981; Wansink, 2004b, c). As big-brained omnivores we enjoy and seek dietary variety to balance our nutrient requirements. For example, people eat more at buffets that often have an enormous variety of foods (Schacter & Rodin, 1974; Wansink, 2004b).

Humans seek variety in their food even when taste is not varied. In a series of experiments with jelly beans, people given a bowl with jelly beans of six different colors ate about 60% more than those given a bowl with jelly beans of only four colors, even though both bowls had the same number of jelly beans and all the colors tasted the same. In another experiment people were presented with jelly beans either in piles separated by color or as a mixed assortment. People ate an average of 13 jelly beans if they were separated by color but 22 jelly beans (69% more) if colors were mixed (Wansink, 2004c). Further experiments with M&M's® candies corroborated these findings. People given the bowl with 10 colors ate 91 versus 64 (43% more) than those given 7 colors (Wansink, 2004a, b).

Successful fast food chains have capitalized on these findings. Offering small alterations in the basic hamburger, for example, increases consumption in both experimental and naturalistic settings (Schlosser, 2001; Wansink, 2004a). Fast food giants such as McDonald's® have responded to the obesity epidemic and the popularity of low-carbohydrate diets by adding salads and other healthful options to their menus, thereby increasing choices and profits (Brownell & Horgan, 2003; Buckley, 2004; Nestle, 2003; Schlosser, 2001). McDonald's also has menu items that are specific to national tastes. For example, there is the Prosperity Burger in Malaysia, a Kampong Burger in Singapore, and Pizza McPuffs in India. The strategy is to produce a cuisine with novel fusions of food items and spice appealing to local tastes.

In addition, unique and appealing names and descriptions of foods are positively related to increased consumption. How many ways can you say "chocolate cake"? For example, people more frequently selected and ate "Belgian Black Forest Double Chocolate Cake" than "Chocolate Cake," although the items were identical (Wansink, 2004c). Hundreds of new food items are added to the market place each year. In 2004, 948 new reduced-sugar products were introduced into the U.S. marketplace (Spurlock, 2005). In addition to product variety, the globalized food market has variety in product sizes and a penchant to supersize.

GLOBALIZATION OF FOOD: SUPERSIZING

For fast foods, supersized portions are more profitable, consumers expect them, and they get more food for their money (Spurlock, 2005). Food costs associated with supersizing are low relative to the costs of advertising, packaging, and labor (Brownell & Horgan, 2003; Nestle, 2003; Spurlock, 2005). Research has demonstrated that portion size and perceived portion size are powerful determinants of how much a person eats (Diliberti, Bordi, Conklin, Roe, & Rolls, 2004; Rolls, 2003; Wansink, 2004a, b; Young and Nestle, 1995).

Often packaging connotes a unit size that is expected to be one serving. A single packaged large muffin appears to be a single serving, but the label informs the consumer that the 660 kcal muffin is really three servings (Nestle, 2003). The USDA portion sizes are often only one eighth to one fourth of the sizes served in fast food and other restaurants. For example, a USDA standard soft drink is 8 ounces (100 kcal), but many restaurants routinely serve 32 ounces (400 kcal), and it is often part of a supersized "meal deal" (Anonymous, 2005). Another example is cooked pasta, with official serving sizes ranging from 1/2 cup (USDA) to 1 cup (U.S. Food and Drug Administration), whereas restaurant servings averages 2.6–3.3 cups (Young and Nestle, 2003). Hamburgers are supersized and loaded with energy-dense fatty extras like cheese and bacon and "special sauces." One recent U.S. trend has been an increase in average size and caloric content of appetizers. Examples include fried mozzarella sticks (830 kcal), spicy chicken wings often called "buffalo wings" (1000 kcal), and fried onion rings with dipping sauce (2130 kcal) (Jacobson & Hurley, 2002).

A large body of research exists on portion size that demonstrates that individuals miscalculate portion sizes for all types of foods (Godwin, Chambers, & Cleveland, 2004; Rolls, 2003; Wansink 2004c; Wansink & van Ittersum, 2003; Young & Nestle, 2003). People routinely select sizes that they consider normative that are larger than the USDA "standard" sizes. Even with the aid of measuring devices, the ability of young adults to estimate correct portion sizes is poor. Using photographs of foods to teach portion sizes did not significantly improve the ability to estimate correct sizes (Furbisher and Maxwell, 2003). Furthermore, experience does not insure accurate assessment of portion sizes. Bartenders misjudged and poured 26% more alcohol into short, wide glasses compared to tall, narrow glasses (Wansink, 2004b; Wansink & van Ittersum, 2003). Teaching portion sizes using common objects or the human body as a guide may be helpful; for example, a deck of cards as the equivalent of 3.5 oz of meat, a tennis ball the size of one-half cup, and the tip of the thumb the equivalent of one teaspoon are common examples. Cross-cultural and ethnic differences on culturally salient "common" measures have not been well researched or developed.

When individuals choose larger portion sizes, they consume more (Rolls, 2003; Young and Nestle, 2003). In a movie theater people given large buckets of popcorn ate 50% more than those given smaller buckets. When both groups were asked to estimate the amount or the calories they had eaten, there were no significant differences in their reported estimates. In an M&M candy experiment, people who were given 1-lb. bags of M&M's ate 120 M&M's, compared to people given a 1/2 lb. bag, who ate 63 M&M's while they watched a videotape. The researchers suggest that the size of the package or portion gives people a perceptual consumption cue as to what is "normal" or acceptable regardless of what they actually consume (Grier and Rozin, 2006; Wansink, 1996; Wansink & van Ittersum, 2003). In another experiment nutrition experts using smaller spoons and bowls consumed less than half the amount of ice cream as those using the larger utensils and bowls. The two groups did not report a significant difference in estimated intake (Wansink, van Ittersum, & Painter, 2006).

Additionally, people who buy food in large quantities or bulk also eat more of those foods than if they buy them in smaller units (Wansink, 2004b). Experiments that alter the caloric density without changing portion sizes (Rolls, 2003) indicate that humans miscalculate food portion sizes and appear to be insensitive to the amount of food consumed and its caloric density (Melanson, 2004; Wansink, 2004c).

From an evolutionary perspective the lack of sensitivity to internal cues of satiation, defined as the inhibition of hunger and appetite within meals, may have been advantageous so that eating continued when food was available in feast and famine conditions. Furthermore, there may have been little selective advantage to be able to accurately and precisely judge portion sizes because the natural environment already constrained the size and amount of available food (Jenike, 2001; Ulijaszek & Strickland, 1993; Ungar and Teaford, 2002). The paradox of relying on visual cues, but being a poor judge of portion size and how much one can eat is exemplified by common saying and aphorisms. In English this is "*Your eyes are bigger than your stomach*"; in Spanish "*Come con las ojos*" (You eat with your eyes); and in German "*Die Augen sind grösser als der Magen*" (Your eyes are bigger than your stomach).

Furthermore, judging portion sizes and consumption volume is confounded by consuming food that you do not see or only partially see. For example, people who acquire food from a fast food drive-in window will eat in their cars and do not see the full contents of their purchase as they consume the food from the bag or container. Bags, boxes, and canisters of fast foods and snacks are packaged in ways that preclude viewing the contents (Wansink, 2004a), and some foods are heated in microwave ovens and eaten directly from their containers.

Additionally, people are distracted by a wide range of stimuli and will continue to eat until food is consumed or until an environmental cue, such as the end of a television program, signals that it is time to stop eating (Wansink, 2004b). Obesogenic environments provide both the cues and the resources that apparently decouple physiological factors such as hunger and satiety from the amount and timing of food ingestion (Melanson, 2004). In sum, people do not see what they are eating, misjudge portion sizes, are susceptible to labeling tricks, are insensitive to the amount of food they ingest, and are distracted or multitask during consumption. All of these factors foster overconsumption and diabesity (Wansink, 2004b).

GLOBALIZATION OF FOOD: UBIQUITOUS VISUAL FOOD STIMULI

Although much has been written about the roles of gustation (i.e., taste) and olfaction (i.e., smell) in food selection and eating behaviors (Drewnowski 1997; Johns & Keen, 1985), relatively little has been written about the evolution and role of primate vision as the primary sense used to promote food and negotiate the current obesogenic environment. Two features of primate vision are particularly important: binocular vision allowing

depth perception and color vision (Cartmill, 1974; Conroy, 1990; Jolly, 1985). This primate legacy of binocular or stereoscopic vision aided hominins relying on depth perception to locate geographical landmarks, track, trap, and hunt game (Lee and DeVore, 1968; Winterhalder and Smith, 1981). R. H. Post (1962) postulated that color vision conferred a selective advantage to human hunters and foragers because they could see prey more readily.

Modern environments are filled with an array of visual stimuli of actual food items, their representations in media, and universally recognized iconic images and logos (e.g., McDonald's Golden Arches). These images transcend cultural and linguistic boundaries and easily identify sources of food. The visual appeal is purposeful and effective because vision is the primary primate mode of perception (Jones, Martin, & Pilbeam, 1992). Visual stimuli and just thinking about food initiate the cephalic phase of digestion (Katschinski, 2000).

These stimuli signal learned messages about the nutritional and physiological effects of particular foods. For primates, initial signals are often visual rather than olfactory, with color as a primary salient property (Dominey & Lucas 2001, 2004; Post, 1965). Color changes in ripening vegetables and fruits signal sensory changes, for example, increased aroma, increased sweetness, softer texture, reduction in toxins (e.g., lectins, phenols), and presence of other biologically active substances (e.g., plant estrogens, vitamins) (Dominy & Lucas, 2004; Vorobyev, 2004; Wolf, 2002). Leaf maturity is also signaled by color change from reddish to green and used by folivorous primate species to select edible leaves. Young leaves often contain more protein and micronutrients, lower levels of toxins, and less fiber than older leaves (Dominy & Lucas, 2001, 2004). Color changes may also signal potential physiological consequences such as the production and/ or release of insulin and increased thermogenesis (LeBlanc, 2000). In turn, these metabolic responses to eating may promote further eating behaviors, initiate activity, and facilitate growth and development, all of which have evolutionary implications for survival (Chakravarthy & Booth, 2004; Cordain et al., 1998; Jenike, 2001; Ulijaszek & Strickland, 1993).

Humans also use color changes to signal when food is cooked. For example, vegetables, if sautéed, will retain their color but with continued cooking become dull, and red meats are "browned" to retain moisture and flavor. Some foods (e.g., lobsters) change color on cooking. Applying heat and observing color changes (e.g., rare vs. well-cooked meat) can indicate that pathogenic bacteria (e.g., *Escherichia coli)*, other microorganisms, and parasites (e.g., *Trichinella spiralis)* have been killed. Food that is spoiling often changes color (e.g., browning of lettuce leaves). Food technology is used to enhance and retain natural colors in fruits, vegetables, fish, and meat (Fellows, 2000). Color is often used to enhance the serving of food, and cooks from around the world are encouraged to prepare attractive food service that incorporates consideration of color (Rombauer and Becker, 1997). Cookbooks often have beautiful color illustrations of food dishes and serving suggestions along with recipes.

Humans are attracted to colorful foods. For example, Mars "color-refreshed" M&M's by adding blue and holiday colors (e.g., pastels for Easter), potato and vegetable chips are available in many colors, and children like brightly colored soft drinks such as Kool-Aid®. Supermarkets display fruits and vegetables in colorful arrangements often appealing to the eye rather than the nose because they are wrapped or in refrigerated displays. Americans talk about the importance of taste and aroma as variables used in the selection

of fruits and vegetables, but they often select fresh produce based on color, size, shape, and blemish-free appearance (Lieberman, unpublished data; Nestle, 2003).

Food images are ubiquitous in obesogenic environments and stimulate purchases and eating (Brownell & Horgan, 2003; Spurlock, 2005; Wansink, 2004a, b). Restaurants display plastic food models, and menus with colorful pictures are common throughout the world. Restaurant take-out windows place colorful food pictures on marquees near ordering stations. Full-color food ads in magazines and newspapers, billboards, and hand fliers are also common.

Colorful television and computer Internet ads for foods are abundant, especially advertisements aimed at children. In 2004 $13 billion of an approximately $144 billion fast food advertising budget was spent on food advertising aimed at children in the United States (Spurlock, 2005, p. 307). Seventy percent of U.S. children age 6–8 years in one poll thought that fast food was healthier than home-prepared food (Story & French, 2004), and 73% of Pakistani children thought that soft drinks were a healthy choice for frequent consumption (Rachagan, 2004).

In the United States children watched an average of 20,000–40,000 TV commercials per year and 11–19 food commercials per hour on Saturday mornings (Story & French, 2004). Based on a six-nation study in Asia, children in the Philippines and Malaysia watched 20 minutes of advertising per hour. Forty to 75% of the commercials on television programming for Asian children were for food and beverages often high in sugar, fat, and salt (Rachagan, 2004). Children develop brand loyalty at an early age and frequently influence family purchases via "pester power" to the extent that they request particular products and brands (Rachagan, 2004). Multinational food companies have begun to exploit other technologies that accommodate visual advertising including, cell phones, iPods, MP3 players, digital video discs, and corporate-sponsored web sites (Kaiser Family Foundation, 2006).

GLOBALIZATION OF FOOD: FORAGING, EATING, AND REDUCED ENERGY EXPENDITURE

The abundant visual cues in contemporary environments facilitate successful foraging and efficient exploitation. These environments present evolutionarily novel opportunities to forage for highly reliable and abundant energy-dense foods with low energy expenditures (Lieberman, 2006). The application of foraging theory provides insights into why humans have become so "successful" in optimizing their energy intakes and producing a diabesity pandemic. Foraging theory, a component of behavioral ecology, uses cost/benefit models to predict food-related behaviors, prey (including plants)–predator interactions, and characteristics of the environment (Mann, 2004; Winterhalder & Smith, 1981). Most cost/benefit models use energy as the currency. Ultimately the goal is to link the costs and benefits to reproductive success (Illius, Tolkamp, & Yearsley, 2002; Drewnowski & Popkin, 1997; Popkin, 2004; Popkin & Gordon-Larsen, 2004; Winterhalder & Smith, 1981).

The costs of obtaining foods have all drastically diminished with dietary globalization. Urban environments have abundant food patches: in homes (pantries, refrigerators, freezers), school cafeterias, movie theaters, kiosks, street vendors, convenience stores,

shopping malls, supermarkets, gas stations, and restaurants. McDonald's, has more than 31,000 stores worldwide (McDonalds, 2006; Spurlock, 2005), Subway® 22,000 (Subway, 2006), Pizza Hut® 20,000 (Pizza Hut, 2006), and Kentucky Fried Chicken® (KFC) 12,000 (Kentucky Fried Chicken, 2006). Many of these fast food franchises are found in more than 100 countries.

A number of studies have demonstrated a relationship between geographical density of fast food restaurants and obesity rates (Drewnowski & Spector, 2004). Within low-income neighborhoods in the United States there are more fast food restaurants, fewer supermarkets, and fewer opportunities for safe exercising compared to higher income neighborhoods (Booth et al., 2005; Maddock, 2004). In Chicago the median distance between any school and the nearest fast food restaurant was 520 m and 78% of schools had a fast food restaurant within 800 m. Children can walk to these "food patches" in about 5–10 minutes (Austin et al., 2006). Accessible and well-advertised food patches reduce search and travel times and, hence, time and human energy expended in these activities. Urban and suburban areas provide the convenience of having food delivered to your home or worksite, thereby reducing search and travel to zero.

An array of food patches (e.g., school cafeterias, fast food restaurants, gas station convenience stores, supermarkets, etc.) provide high-calorie, nutrient-dense, and inexpensive food. There is appealing variety, and the food requires little preparation prior to consumption. There are no issues of diminishing returns in exploiting these patches because the supply of food is limitless. Only purchasing power and satiety are limitations. Drewnowski and Spector (2004) demonstrated that individuals with limited incomes purchase energy-dense fast foods that provide more calories per dollar than expensive less energy-dense fruits and vegetables. The marginal value theorem predicts this purchasing behavior because people are expected to maximize their rate of return in calories given their investment in time, energy, and money (Sinervo, 1997). Although eating fast food optimizes energy intake per unit cost, it can lead to poor nutritional balance (Drewnowski, 2004).

Americans increasingly value convenience and low time and energy investments in food preparation (Rozin, 2005). In a recent survey, 34% of Americans indicated that they preferred to "dine out" or have a meal catered in their home even for holiday meals at Thanksgiving and Christmas (Hales, 2005). Americans consume a large proportion of their meals away from home. On an average day in 2004, 40% of American adults were eating in restaurants. Food consumed in restaurants requires little time or energy spent on acquisition, preparation, cooking, service, or cleaning up after the meal. Restaurants are attractive and create atmospheres that are conducive to eating with lighting, aromas, comfortable seating, distractions, and high food salience (Wansink, 2004) (see Table 3-2).

In addition to low-energy-cost access to food, industrialized nations are modern marvels of human energy-saving lifestyles created by technology, the built environment, daily habits, and cultural values (Lieberman, 2006). Appealing to the human inclination for energy conservation, contemporary environments reduce human energy expenditure with motorized mobility (e.g., cars, buses, elevators, moving sidewalks) and wireless and remote control devices (e.g., cell or mobile phones, garage and TV remote controls) (Cordain et al.,1998; Rozin, 2005). For example, the number of hours of sedentary

activities such as watching TV and DVDs and playing videogames are positively correlated with weight gain for both children (Dennison et al., 2002; Rachagan, 2004) and adults (Leonard, 2001; Stroebele & de Castro, 2004; Wyatt, Peters, Reed, Barry, & Hill, 2005). The Kaiser Family Foundation has been documenting children's television and computer viewing behavior and program content for a number of years (1999, 2003, 2005, 2006). Children 2–18 years of age watched or used media nearly 6.5 hours per day, with 81% reporting daily television watching (Rideout, Roberts, & Ulla, 2005). Television watching both reduces energy expenditure and increases energy intake, contributing in both ways to the net energy surfeit and diabesity (Rachagan, 2004). For every hour of TV watched the obesity percentage rose by 6% in children (America's Obesity Crisis, 2004). Because television remote controls and cordless and cellular phones are widely available, people no longer get up from chairs to change channels or answer the telephone. We continue to reduce the small amounts of energy spent as part of our daily routines, and these cumulative changes in lifestyle contribute to the global diabesity pandemic (Rozin, 2005; Wyatt et al., 2005).

DIABESITY AND DARWINIAN MEDICINE

The current pandemic of obesity and type 2 diabetes is the result of very rapid changes in lifestyles that have capitalized on millennia of evolutionary selective pressures favoring physiological patterns, nutritional requirements, and cognitive and behavioral repertoires adapted to environments with fluctuations in food availability and periodic high energy expenditures (Lieberman, 2003, 2006). Humans have evolved a complex system of adipose, gut, and pancreatic hormones, enzymes, and neurotransmitters that respond to food intake and signal the hypothalamus to regulate appetite and influence various organs and tissues to metabolize or store energy (Chakravarthy & Booth, 2004; Neel, 1962). These regulatory mechanisms are more sensitive to hunger than satiety, and humans respond to food characteristics (i.e., variety, large portion sizes) and characteristics of the dietary environment (i.e., abundant food patches, convenience, pleasant eating environments) with increases in food consumption (Wansink, 2004b).

Cognitive/behavioral approaches to slow the growing diabesity pandemic have not been successful in managing it. For example, billions of dollars have been and continue to be spent on weight-reduction diets, special foods, books, over-the-counter remedies, exercise and health club memberships, and nutritional counseling (Jeffcoat & Bakker, 2005). We are a nation of dieters. In the United States dieting to reduce body weight is the most common approach, but numerous studies document that restricted eating is not an evolutionary adaptive strategy (Polivy & Herman, 2006). Annually, 44% of woman and 29% of men in the United States are dieting (Friedman, 2003). If people lose weight, 95% regain the weight, often exceeding their baseline weight when they initiated dieting (Ikeda et al., 2005). We are losing the "war" on obesity and diabetes because the strategies are ineffective in obesogenic environments (Friedman, 2003; Spiegel & Nabel, 2006).

Eaton and colleagues (2002) have called for "evolutionary health promotion" that takes into account issues of both health and disease, based on adopting a conceptual framework that interprets contemporary human conditions in light of our evolutionary

past (Eaton et al., 2002; Speakman, 2004). The approaches that Eaton and colleagues (2002) suggest in *Preventive Medicine* alert physicians to the evolutionary context in which once adaptive genotypes and phenotypes become mismatched with contemporary lifestyles—Stone Age genes in Space Age environments (Eaton et al., 2002). Based on the data presented in this chapter, their strategies do not appear to go far enough in suggesting a closer look at physiological/metabolic and cognitive/behavioral regulatory mechanisms. They suggest conventional approaches that require dietary restraint and motivation to increase activity. Current approaches focusing on behavioral interventions are contrary to what we know about human cognition, behavior, and physiology that drive eating behavior and fat storage in obesogenic environments. Interventions to reduce psychosocial stress and increase sleep duration may be more amenable to change than behaviors associated with eating (Keith et al., 2006).

As noted previously, one approach to preventing, controlling, and reversing the undesirable sequelea of diabesity is to develop new pharmaceuticals that operate at the molecular level opposing or enhancing the effects of gut and adipose tissue peptides, hormones, and neurotransmitters on the appetite-control nuclei in the hypothalamus. Some rodent experiments have been promising, and new drugs are being developed (Wynne et al., 2005). Thus far, antiobesity drugs have either not proved effective (e.g., leptin) or they have had serious side effects (e.g., PhenFen and pulmonary hypertension) (Bray et al., 1998; Wynne et al., 2005). A novel approach to decreasing diabesity is to develop drugs that interfere with the inflammatory response and restore insulin sensitivity (Lee and Pratley, 2005). Primary prevention and pharmacological therapies targeting molecular changes are most likely the approaches that will have some impact on the diabesity pandemic.

The only long-term successful approach to sustained weight loss has been surgery (i.e., gastric bypass, stomach stapling), which carries a number of health risks (e.g., rupture of the stomach) (Pear, 2006). An estimated 200,000 surgeries were performed in 2006 (Centers for Disease Control, 2005b; Pear, 2006).

An intergenerational approach would focus on maternal health and the intrauterine environment in the prevention of low-birth-weight babies, who are at increased risk for the development of diabesity (Barker, 1998a, b, 2004, 2005). Ideally the interventions would occur preconception and continue throughout the pregnancy. This primary prevention approach has the potential for a large impact on the diabesity pandemic because it would alter fetal metabolic programming.

Finally, there are alterations in the food supply, such as smaller portion sizes served in restaurants, foods packaged in smaller units, and the elimination of soda and candy from schools. These changes would be more acceptable if portion sizes were made to appear larger, for example, by serving them on smaller plates (Wansink, 2004b, c). Alterations in the eating environment to reduce the salience of food cues (e.g., fewer food commercials on television) and increasing attention to eating by eliminating distractions and multitasking have the potential to curb eating and reduce the risk for diabesity (Wansink, 2004a, b). If the decline in tobacco use can be used as an example, then broad policy and legal interventions might prove effective in changing behaviors associated with eating and physical activity. For example, in December 2006 the Board of Health of New York City passed a ban on trans fat use in restaurants that would go into effect in 2008 (MSNBC News Service, 2006).

CONCLUSION

Evolution has honed human proclivities for eating large portions of food and physiological capacities for energy storage that, when coupled with modern lifestyles that include energy-dense food abundance and energy-saving conveniences, promotes weight gain, obesity, and type 2 diabetes. Predictions are that the worldwide pandemic of diabesity will continue to escalate over the next few decades, with the majority of new cases in India and China. An evolutionary medical approach would de-emphasize alterations in food intake or dieting and refocus efforts on early life prevention and pharmaceutical intervention.