Obesity from a Biocultural Perspective

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Abstract

Obesity is new in human evolutionary history, having become possible at the population level with increased food security. Across the past 60 years, social, economic, and technological changes have altered patterns of life almost everywhere on Earth. In tandem, changes in diet and physical activity patterns have been central to the emergence of obesity among many of the world's populations, including the developing world. Increasing global rates of obesity are broadly attributed to environments that are obesogenic, against an evolutionary heritage that is maladaptive in these new contexts. Obesity has been studied using genetic, physiological, psychological, behavioral, cultural, environmental, and economic frameworks. Although most obesity research is firmly embedded within disciplinary boundaries, some convergence between genetics, physiology, and eating behavior has taken place recently. This chapter reviews changing patterns and understandings of obesity from these diverse perspectives.

Human genetics likely have undergone selection for traits that promote energy intake and storage and minimize energy expenditure. Thus it is no surprise that there are a great many obesity-related genotypes. Models linking genetic susceptibilities with physiology and feeding behavior are emerging, and although they only explain a small proportion of global obesity rates, might underlie the more-common and genetically complex forms of human obesity. Human physiology can only exert weak control on the reduction of food intake and the increase of energy expenditure when energy stores are replete and food security is high, and obesity is almost an inevitable human biological outcome in the environments that have been constructed in industrialized nations over the past 60 years and that have emerged with modernization elsewhere.

INTRODUCTION

Obesity is the condition in which excess body fat has accumulated to a degree that health and function are negatively affected. It is new in human evolutionary history, having been essentially nonexistent until approximately 10,000 years ago (Brown 1991, Brown & Krick 2001). Across history, individuals and groups of privilege have been able to display embodied wealth by above-average body size, including weight and fatness (Brown & Konner 1987, Brown 1991, de Garine & Pollock 1995). Obesity was known in ancient Greece (Bevegni & Adami 2003) and was a common condition among the English upper classes in the late eighteenth century (Trowell 1975). It emerged more generally among North American men in the nineteenth century (Kahn & Williamson 1994), increasing in successive surveys in both the United States and Britain across the twentieth century (Garrow 1978). Across the past 60 years, social, economic, and technological changes have altered patterns of life worldwide. During post–World War II reconstruction, economic development grew on a more-global scale, as colonial models of economic management fell from favor. In tandem, changes in diet and activity patterns have been central to the emergence of obesity among many of the world's populations, including poorer ones (Popkin & Doak 1998). In the vast majority of nations for which comparative data are available, rates of obesity are increasing (de Onis 2005, Nishida & Mucavele 2005). It is estimated that over 300 million adults are currently obese, as defined by having a body mass index (BMI) above 30 kg/m^2. A further 700 million people are considered overweight, with BMIs between 25 kg/m^2 and 30 kg/m^2. The prevalence of obesity among children is also rising.

Increasing rates of obesity across the world are broadly attributed to environments that are obesogenic (French et al. 2001, Brownell 2002, Hill et al. 2003), against an evolutionary heritage that is maladaptive in these new contexts (Neel 1962, Eaton et al. 1998, Neel et al. 1998, Lev-Ran 2001). The term obesogenic environment was coined by Swinburn et al. (1999), who argued that the physical, economic, social, and cultural environments of the majority of industrialized nations encourage positive energy balance in their populations. A dominant explanatory framework for the emergence of obesogenic environments is that of nutrition transition (Popkin 2004), which relates globalization, urbanization, and westernization to changing food environments across the populations of the world (Drewnowski & Popkin 1997, Griffiths & Bentley 2001, Contaldo & Pasanisi 2004). Central to this transition are shifts in diet toward increased consumption of energy-dense foods (Drewnowski & Popkin 1997) and declines in physical activity (Erlichman et al. 2002). In this formulation, global food supply becomes increasingly abundant, less expensive, and more-aggressively marketed; coupled with declines in physical activity, this has led to higher prevalences of obesity (Nielsen et al. 2002, Drewnowski & Darmon 2005). In addition, economic inequalities within and between nations have ensured food security

for significant sectors of society and for some nations as a whole, while denying food security for many others (Dyson 1996). Underpinning such global phenomena are interrelated physiological, genetic, and behavioral factors (Shuldiner & Munir 2003, Clegg & Woods 2004, Flier 2004), as well as cultural norms that make humans susceptible to obesity (Brown 1991, de Garine & Pollock 1995). How food use is structured socially and culturally has been slow to adjust to changing patterns of food security, as have perceptions of appropriate body size for health and beauty, which has contributed to the emergence of obesity in various societies (de Garine & Pollock 1995).

Obesity has been studied using genetic, physiological, psychological, behavioral, cultural, environmental, and economic frameworks. Although most obesity research is firmly embedded within disciplinary boundaries, some convergence between the study of genetics, physiology, and eating behavior has taken place recently (Barsh & Schwartz 2002, Flier 2004, de Castro 2006). Anthropologists use biocultural perspectives to understand the interacting factors that may have made fatness-related traits advantageous across evolutionary time and in relation to cultural change (Brown 1991). Much has happened in obesity research since 1991, making a review from biocultural perspectives timely. In this chapter, we place new observations in the genetics and physiology of obesity (and the social, cultural, and behavioral forces that interact with them) in an updated biocultural description of this phenomenon. We rely heavily on research carried out in the United States, largely because this is where most of the best work has been carried out. Our focus is predominantly on obesity among adults.

We begin by defining measures of obesity and continue by considering its health implications and population trends. We use a definition of obesity as a BMI greater than 30 kg/m^2, and its age equivalent for children, which is recommended for international use (Shetty & James 1994, Cole et al. 2000), although this measure is not independent of stature, lean body mass, or body proportion (Norgan 1994a,b). This index is widely used largely because of the need for a simple comparative measure of obesity in the world's populations.

MEASUREMENT OF OBESITY AND FATNESS

Measures used to assess body fatness and obesity include (*a*) visual appearance; (*b*) anthropometry; (*c*) body density by underwater weighing, isotopic dilution, dual X-ray absorptiometry, or bioelectrical impedance; and (*d*) body imaging by ultrasound, computed tomography, or magnetic resonance imaging (Poskitt 1995). The most-frequently used measures for population-based work are anthropometric; the most common of these is the BMI, which is derived by dividing body weight in kilograms by the square of height in meters. It has been adopted for epidemiological and public health usage because it reflects body energy stores and shows strong associations with morbidity and mortality from a number of chronic diseases and disorders.

Relationships between BMI and both morbidity and mortality are usually either J- or U-shaped, with risk increasing outside of a normative range. At the lower end of the BMI distribution, increased morbidity risk owing to infectious disease has been demonstrated for populations in India (Campbell & Ulijaszek 1994), Pakistan and Kenya (Garcia & Kennedy 1994), but not in the Philippines and Ghana (Garcia & Kennedy 1994). At the upper end of the distribution, the BMI has been shown to be associated with chronic disease risk and mortality (Shetty & James 1994). These include coronary heart disease; high blood pressure; stroke; noninsulin-dependent diabetes (NIDDM) (World Health Organization 2000, Li et al. 2002); endometrial, ovarian, cervical, and postmenopausal breast cancer in women; and prostate cancer in men (Bianchini et al. 2002). The normative

BMI range of 18.5–25 kg/m^2 is likely to be maintained in many populations by way of balancing selection. The BMI cut-offs are used to define overweight and obesity in adults are 25 kg/m^2 and 30 kg/m^2, respectively (Shetty & James 1994). However, the relationship between BMI and fatness varies across populations, as do relationships between morbidity and BMI. In some Chineset (Li et al. 2002) and South Asian populations (Gill 2001, Sullivan 2001, Wahlqvist 2001), increased chronic disease risk occurs at lower BMIs than among European populations (Li et al. 2002). The BMI does not give a measure of intra-abdominal (visceral) or lower-body fatness, however. High levels of intra-abdominal fatness are independently associated with risk markers of cardiovascular disease, NIDDM, and various cancers (World Health Organization 2000). High lower-body fatness relative to waist size is associated with lower risk of the same disorders, and among females it is important for buffering the energetic stresses of pregnancy and lactation (Garaulet et al. 2000).

Classification of childhood obesity using BMI is more problematic than for adults because of the variability in the growth rates of children both within and between populations. The BMI changes with age, and Cole et al. (2000) have proposed for international use of age-specific cut-offs for childhood overweight and obesity that pass through BMIs of 25 kg/m^2 and 30 kg/m^2, respectively, at the age of 18, using a normative distribution that varies by age and sex. However, unlike in adults where it is possible to establish increased health risks associated with an increased BMI, most health effects of childhood obesity are manifested in adult life and not childhood, with the possible exception of risk markers for NIDDM.

POPULATION TRENDS IN OBESITY

Obesity at the population level was largely unknown in the 1950s. However, by the 1990s, 33 nations had obesity rates exceeding 10% of their adult populations (**Figure 1**, see color insert) (Nishida & Mucavele 2005). Currently, obesity is most prevalent in some Pacific Island nations; the United States; most European, many Middle Eastern, and some Latin American nations; and South Africa. Four Pacific Island nations (Nauru, Tonga, the Cook Islands, and French Polynesia) have the highest rates of obesity in the world, in all cases exceeding 40% of their adult populations. Obesity rates for Bahrain and Kuwait lie close to that of the United States, at a little below 30% of the adult population. The rate for Canada is approximately half that of the United States. For the adult South African population, the rate is 22%, similar to Egypt, Turkey, Hungary, and Germany, the latter two having the highest rates in Europe. The lowest rate in Europe, 5%, is among adults in Norway and Switzerland. Of Latin American populations, Mexico, Uruguay, and Peru have rates that exceed 15%. Rates among wealthier Asian nations vary from 6% in Singapore to 3% of adult populations in Japan and South Korea. In the vast majority of nations, obesity rates among females are higher than for males by an average of 5%. Obesity rates of females exceed those of males by more than 2% in 32 of the 66 nations for which data exist for both sexes, whereas obesity rates of males exceed those of females in only 4 nations.

Rising obesity rates have varied in different nations. In Pacific Island nations, rates of obesity were already high by the 1960s and continued to increase dramatically into the 1990s (Ulijaszek 2005). The United States and Canada had similar rates of obesity 40 years ago, at approximately 10% of the adult population; subsequently, rates became higher in the United States than in Canada (Nishida & Mucavele 2005). In the past 20 years, obesity rates have risen in the majority of nations with available data. Of 28 nations for which data are available, increased obesity rates have been observed for adult males in 20 and for adult females in 19 (Nishida & Mucavele 2005). The

fastest increases have taken place in Hungary, Russia, Ireland, Turkey, and Nauru. Of the nations with no increase or a decrease in obesity, three of them (France, Italy, and Japan) continued to have the low levels of obesity they experienced approximately 20 years ago and are economically advanced nations with excellent food security. The extensive emergence and rise of obesity among most of the world's populations indicate that the ability to become obese is universal (Lev-Ran 2001). Furthermore, the great variation in obesity rates between nations in the same regions with different economic standing supports the view that increasing food security is but one component of the recent emergence of obesity.

Although previously a condition predominantly of the wealthy, the relationship between social class and obesity has become inverted in wealthier and economically emerging nations (Sobal & Stunkard 1989, Kirchengast et al. 2004, Rennie & Jebb 2005, Stamatakis et al. 2005). Similar inversions are also found in urban areas of less-developed countries (Monteiro et al. 2000, Peña & Bacallao 2002), as increased food security and sedentization of life have increasingly permeated poorer sectors of society, as well as wealthier ones. Within more-affluent nations, minority populations and rural communities show the highest rates of obesity (Swinburn et al. 2004). For example, obesity rates among adult Pacific Islanders living in New Zealand in the early 1990s were above 65% (Swinburn et al. 2004), compared with nationwide values of 15%. In the United States, Native Americans, African Americans, Puerto Ricans, and Mexican Americans have higher body mass than European Americans (Denney et al. 2004). Nearly 40% of black non-Hispanic adults have a BMI above 30 kg/m^2, much higher than obesity rates for Mexican American and white non-Hispanic adults, who have rates of 35% and 29%, respectively (Flegal et al. 2002).

FATNESS AND HUMAN EVOLUTION

Larger body mass and increased ability to accumulate fat relative to other nonhuman primates in seasonal environments are two key adaptive features of human life history (Aiello & Wells 2002). The rapid brain evolution observed with the emergence of *Homo erectus* at approximately 1.6–1.8 million years ago is likely associated with increased body fatness as well as diet quality (Leonard et al. 2003), i.e., the greater availability of animal fat and cholesterol that would have come with increased diet quality, possibly facilitating encephalization (Horrobin 1999). Whereas Cunnane & Crawford (2003) proposed that the modern human brain was an outcome of earlier natural selection for greater fatness in neonates and infants, Kuzawa (1998) has argued that the two phenomena coevolved in feedback with each other. Regardless of whether fatness preceded encephalization, greater levels of body fatness and reduced levels of muscle mass relative to other primate species allow human infants to accommodate brain growth by having adequate stored energy for brain metabolism when intake is limited and by reducing the total energy costs of the rest of the body (Aiello & Wells 2002). Compared with apes, humans have a similar proportion of maternal daily nonmaintenance energy budget invested in fetal tissue, but humans have a much higher diet quality. This allows both larger brain size and higher body fatness at birth (Ulijaszek 2002a). Energy stored in adipose tissue buffers against mortality risk soon after birth and at weaning, when nutrition is often disrupted (Kuzawa 1998).

Fatness in human females is linked to fertility (Brown & Konner 1987, Norgan 1997), e.g., female ovarian function being particularly sensitive to energy balance and energy flux (Ellison 2003). At any BMI, females have a greater proportion of body weight as fat than males. Furthermore, they have a greater proportion of lower-body fat than

males. This has importance in reproductive function: Lower-body fat is less-readily available for everyday energetic needs than upper-body or abdominal fat, but lower-body fat is mobilized during pregnancy and lactation (Garaulet et al. 2000). Reproductive effort during pregnancy and lactation is thus buffered from environmental energetic constraints (Ellison 2003).

Human genetics are likely to have undergone selection for traits that promote energy intake and storage and minimize energy expenditure (Rosenbaum & Leibel 1998). There is great diversity in obesity-related genotypes (Perusse et al. 2005). However, the vast majority of obesity is related to more than one locus, each accounting for only part of the phenotypic variance (Comuzzie 2002). Because all aspects of metabolism are under genetic control, and the expression of obesity phenotypes is much more limited than the expression of peptides that regulate metabolism, natural selection for the capacity to save and store energy is likely to have taken place for different genes with the same phenotypic result (Lev-Ran 2001), perhaps ultimately to defend the energy needs of large brain size. Neel et al. (1998) argued that many different genes underwent such selection in different populations and geographic areas and under different kinds of environmental pressure. Against this microevolutionary scenario, that most mammals are able to overeat to high levels of body fatness suggests the genetic basis for the majority of human obesity lies in deeper evolutionary time, although the greater normative level of body fatness of humans relative to other primates and most mammals is likely to have evolved with the encephalization that took place with *H. erectus*.

Seasonality of food availability likely was a major environmental pressure, given that it occurred during hominid evolution (Foley 1993) and is common in primate (Hladik 1988) and human subsistence ecologies of all kinds (de Garine & Harrison 1988). Human genotypes for obesity, however, are not incompatible with present environments of good food security and sedentary lifestyle, given that the almost-worldwide increases in survivorship and longevity have taken place often in tandem with the emergence of obesity (Eaton et al. 1998).

Because energy stores are vital to survivorship and reproduction, the ability to conserve energy as adipose tissue would have conferred selective advantage to *Homo sapiens*. Neel (1962) suggested the existence of thrifty genotypes that code for efficient and potentially excessive energy accumulation. This formulation has undergone modification, with alternative terms proposed for conditions associated with genes for diabetes, obesity, and hypertension considered to have been adaptive in the remote past but now compromised by changed environments. These terms include syndromes of impaired genetic homeostasis, civilization syndromes, and altered lifestyle syndromes (Neel et al. 1998). Genes corresponding to such syndromes may be called stockpiling (Garrow 1993), greedy, or acquisitive instead because little obesity is caused by thrifty metabolism (Lev-Ran 2001).

The biological drives of feeding, hunger, and the dietary regulation of macronutrient intake may have shared physiological and behavioral bases with other animals (Ulijaszek 2002b, Berthoud 2004). Various mammals are susceptible to overeating and increased body-fat deposition when presented with diets that are plentiful, palatable, and/or high in fat, indicating that the tendency to overeat in response to food-portion size, palatability, energy density and to overeat fat passively are general mammalian evolutionary traits. Furthermore, social aspects of feeding are far from unique to humans; the social facilitation of food intake (in which social interaction during eating increases food intake) has long been known to take place among animals from chickens to primates.

Body size in the genus *Homo* was greater than that of most australopithecines, and although meat and nutritionally dense plant foods were the major dietary components most likely to have fueled body-size increase

and encephalization in *Homo* (Plummer 2004), decreased taste sensitivities associated with greater body size would have also favored increased diet breadth. Low sweetness and bitterness sensitivities allow larger primate species to find food sources of lower-energy density palatable and to eat them more frequently than among primate species of small body size (Simmen & Hladik 1998). Furthermore, basal metabolic rate per unit of body mass scales negatively with body weight among primates (Martin 1993), making the energy requirement per unit of body size lower in large primates than in small ones.

Human eating behavior differs from other mammalian species in the extent to which (*a*) food availability is controlled, (*b*) social and cultural norms of diet and eating exist, and (*c*) personal feeding constraints operate (Ulijaszek 2002b). Social and cultural norms of diet and eating are likely to have increased in complexity only with the emergence of complex symbolic behavior among *H. sapiens* by 75,000 years ago (Henshilwood et al. 2004). With cooking, the emergence of cuisine, and increased complexity of food use, great diversity in the social patterning of feeding has taken place. Social feeding may have been a behavioral adaptation of early *Homo* that has continued to have implications for the energy balance of contemporary human populations.

GENETICS OF OBESITY

Various types of evidence have been used to identify genetic contributions to human obesity. These include (*a*) familial clustering of body fatness (Allison et al. 1996), (*b*) estimates of heritability for obesity and fatness phenotypes in twin studies (Stunkard et al. 1986, Keller et al. 2003), (*c*) identification of monogenic severe early-onset obesity (Flier 2004), and (*d*) genotyping of polygenic obesity (Clement et al. 2002).

The heritability of human adiposity (estimated in most studies from BMI, but also from skinfolds) varies from 0.49 to 0.93 (Keller et al. 2003). Heritability of food intake is lower, generally varying between 0.11 to 0.65 (Keller et al. 2003). With respect to eating behaviors and styles, heritabilities of 0.44 and 0.65 have been reported for meal frequency and size, respectively (de Castro 1999), 0.40 and 0.45 for disinhibition (Neale et al. 2003, de Castro & Lilenfeld 2005), 0.44 for cognitive restraint, and 0.24 for perceived hunger (de Castro & Lilenfeld 2005).

Over 600 genes, markers, and chromosomal regions have been associated with human obesity phenotypes (Perusse et al. 2005), and the numbers continue to grow. By October 2004, 173 human obesity cases owing to single-gene mutations in 10 different genes had been reported, whereas 49 loci related to Mendelian syndromes relevant to human obesity had been mapped to a genomic region, with causal genes or strong candidate genes identified for most of them. Furthermore, 204 quantitative trait loci (single genes with large effects on a given quantitative trait) for obesity-related phenotypes had been identified from 50 genome-wide scans (Perusse et al. 2005). Several single-point mutations have also been associated with various obesity phenotypes (Bouatia-Naji et al. 2006, Wilson et al. 2006). At the population level, obesity is mainly polygenic, with genetic variations influencing metabolism. There are interactions of different obesity genes, and gene-dosage effects in heterozygotes of obesity genotypes, such that intermediate phenotypes are less extreme than homozygotes (Chung & Leibel 2005). Success in the identification of polygenic determinants of obesity across human populations has been limited (Comuzzie 2002), although genome-wide scans in different populations have localized major obesity loci on chromosomes 2, 5, 10, 11, and 20 (Clement et al. 2002). The study of polygenic obesity requires the analysis of genotype-phenotype associations while taking into account the influence of environmental factors such as diet and sedentary lifestyle (Clement 2005). However, such an integrated approach requires large samples and the expansion of biocomputing tools for the analysis of

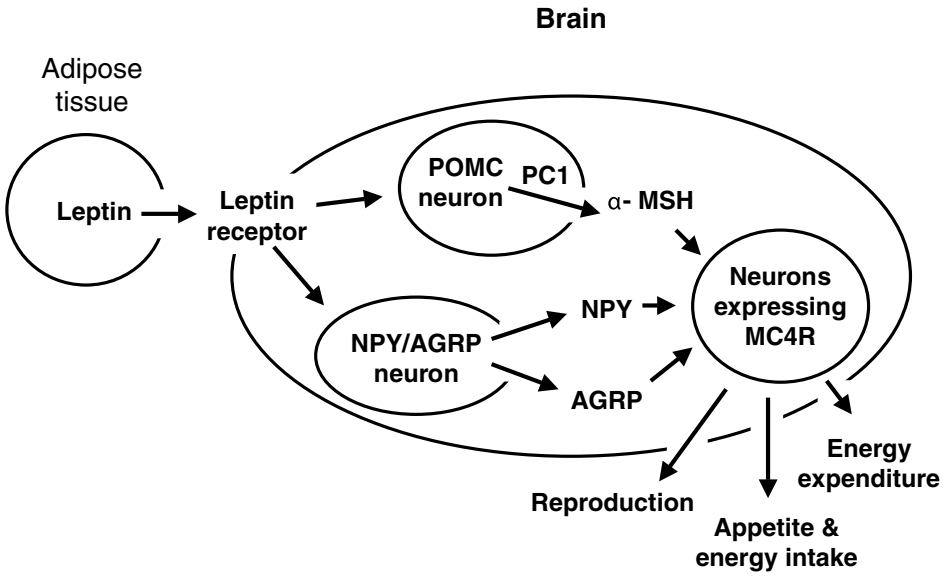

Figure 2

Genetic regulation of energy balance by way of leptin signaling and agouti related protein (AGRP). Modified from Mizuno et al. 2003 and Flier 2004. α-MSH, α-melanocyte stimulating hormone; MC4R, melanocortin 4 receptor; NPY, neuropeptide Y; PC1, prohormone convertase 1; POMC, pro-opiomelanocortin.

multiple interactions with no a priori hypotheses (Clement 2005).

For over 50 years, monogenic rodent models of obesity have been used widely in attempts to understand body-weight regulation in humans. Linkages between the genetic and physiological study of obesity became possible with the identification of (*a*) the *ob* gene product leptin (Zhang et al. 1994) and the leptin receptor (Tartaglia et al. 1995), and (*b*) the *agouti* gene and agouti related protein (AGRP) (Zemel et al. 1995, Mizuno et al. 2003). Leptin is a cytokine secreted from adipose tissue at a rate proportional to the size of body-fat stores and is the principal physiological indicator of nutritional state and fatness. AGRP, along with neuropeptide Y (NPY) and α-melanocyte stimulating hormone (α-MSH), is a central mediator leptin action. The melanocortin 4 receptor (MC4-R) plays a central regulatory role in the action of leptin and is influenced by levels of α-MSH, NPY, and AGRP. The signaling pathways involving leptin and MC4-R influence the regulation of all aspects of energy balance (Barsh & Schwartz 2002, Fan et al. 2005). Several types of monogenic human obesity owing to genetic disruption of the leptin-signaling pathway identified more recently include deficiency syndromes of leptin, leptin receptor, pro-opiomelanocortin (POMC), MC4-R, and prohormone convertase 1 (Farooqi & O'Rahilly 2005). These peptides function in a central nervous system pathway for energy-balance regulation (**Figure 2**) (Mizuno et al. 2003, Flier 2004). Although the mechanism whereby MC4R influences food intake is reasonably elaborated, its influence on energy expenditure is not. However, in the Quebec Family Study, Loos et al. (2005) have identified a DNA-sequence variation at the MC4R gene locus that may contribute to physical inactivity. Although the major leptin-regulatory arm of this model accounts for less than 4% of severe early-onset obesity, quantitative differences in the expression or function of these same genes, either alone or in combination with one another, may underlie the more-common and genetically complex forms of human obesity (Flier 2004).

PHYSIOLOGY AND BEHAVIOR

Physiologically, obesity can only develop if food consumption is high and/or energy expenditure is low, resulting in positive energy

balance across months or years. A positive energy balance of 10% can lead to approximately a 13.5-kg increase in body weight within a year (Bray 1987). The increasing rates of obesity among adults in many industrialized populations across the second half of the twentieth century are a result of a disregulation of energy balance of less than 1% per year. The physiology of energy accumulation is that of neuroendocrine, gut- and adipose-tissue regulation of energy balance, the maintenance of which involves coordinated and physiologically linked changes in energy intake and expenditure (Moore 2000). Both leptin- and melanocortin-signaling pathways are upstream of nervous system–effector mechanisms that regulate both appetite and energy expenditure (Flier 2004).

Much less is known about neurophysiological mechanisms by which reduced energy expenditure influences energy balance than of altered appetite and energy balance (Flier 2004). There is no dominant physiological pathway of desire to eat in relation to nutritional requirement or environmental constraint of intake through availability. Rather, a range of physiological signals regulates intake. Over 60 obesity-related peptides are known, many identified as either promoting increased or decreased energy intake. Various models have been put forward that link energy-balance endocrinology of the gut, pancreas, adipose tissue, the central nervous system, and the brain (Tschop et al. 2000, Schwartz & Morton 2002, Flier 2004), and **Figure 3** gives a simplified consensus view.

In the absence of food (**Figure 3*a***), there are falling levels of leptin from adipose tissue, insulin from the pancreas, and peptide YY-36 from the gut, as well as increasing levels of ghrelin from the gut and upregulated NPY/AGRP neurons in the hypothalamus, the physiological system that links the nervous and endocrine systems. Upregulated NPY/AGRP neurons cause the release of NPY and AGRP that inhibit downstream neurons in the paraventricular nucleus of the hypothalamus and the ventromedial hypothalamus (often thought of as the satiety center of the brain), stimulating appetite. Stimulation of NPY/AGRP neurons also causes cellular and tissue growth and appetite-stimulating effects in the lateral hypothalamus, which in turn upregulate energy intake and downregulate energy expenditure. Decreasing leptin and insulin concentrations also lead to reduced stimulation of POMC neurons, resulting in lowered secretion of α-MSH in the pituitary. α-MSH is a secondary peptide product of the POMC gene, one of eight yielded by differential processing of the primary peptide product of this gene. This in turn lowers its stimulation of the downstream neurons in the hypothalamus. Inhibition of these downstream neurons reduces metabolic breakdown of larger molecules to smaller ones. The NPY/AGRP neurons are stimulated by starvation but are not significantly affected by overfeeding (**Figure 3*b***), whereas POMC neurons are affected by both starvation and overfeeding. Thus there would have been stronger natural selection against starvation than overfeeding.

Other gut peptides involved in appetite regulation include cholecystokinin (CCK), glucagon-like peptide 1, and bombesin-like peptides (similar in structure to a family of short peptides widely distributed among mammals with potent physiological effects on feeding and satiety). Bombesin-like peptides include gastrin-releasing peptide, originally named so for its ability to release gastrin in the gut but also expressed in the pituitary gland, and neuromedin B, whose physiological effects include the regulation of feeding, blood pressure, blood glucose, body temperature, and cellular growth. CCK is involved in the control of the amount of food eaten at any time and is released from the small intestine into the circulation in response to nutrients in the lumen of the gut, such as fatty acids (Moran 2000). Glucagon-like peptide 1 is released after feeding and produces loss of appetite (Yamamoto et al. 2002). As well as its involvement in the regulation of energy balance, MC4R has been implicated in the regulation

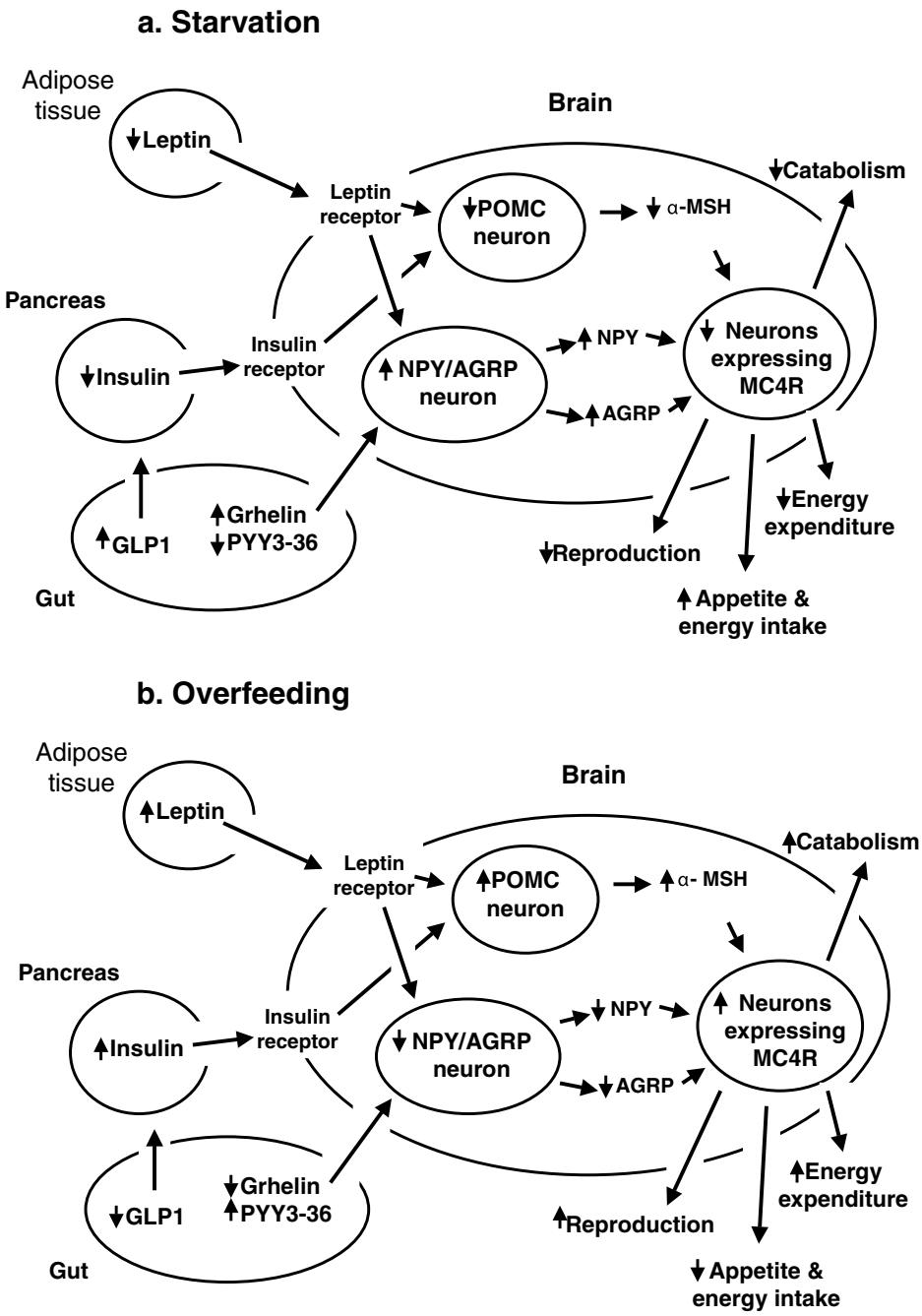

Figure 3

Neuroendocrine regulation of energy balance. Modified from Schwartz & Morton 2002, Tschop et al. 2000, and Flier 2004. AGRP, agouti related protein; α-MSH, α-melanocyte stimulating hormone; GLP1, glucagon-like peptide 1; MC4-R, melanocortin 4 receptor; NPY, neuropeptide Y; POMC, pro-opiomelanocortin.

of reproduction by way of the hypothalamo-pituitary-gonadal axis (Schioth et al. 2003).

The neurophysiological system described in **Figure 3** strongly controls feeding and reduced energy expenditure when energy reserves are depleted and fuel security is low. However, it can only exert weak control on reducing food intake and increasing energy expenditure when energy stores are replete and food security is high (Ahima et al. 1996).

Environmental factors associated with modern life and good food security create constant pressures on food intake that are not compensated by equivalent increases in energy expenditure, with surplus energy stored overwhelmingly in adipose tissue (Berthoud 2004). This model is a partial one, however, because food intake is driven by additional cognitive and environmental factors (Berthoud 2004). At the low end of the BMI distribution, considerable research has been conducted on low food intake and adaptation to low energy intakes (Waterlow 1990, Shetty 1993), showing energy balance at low intakes to be strongly defended. The major components are feedforward mechanisms between the brain and gut that anticipate the nutritional needs of the body (Myers & Sclafani 2003) by responding to the abundance of food cues in food-secure and socially enhanced environments (de Castro & Stroebele 2002, Ulijaszek 2002b, Rolls 2003). Such cues include perceived qualities of potential foods, including (*a*) smell; (*b*) associations with pleasure, displeasure, or disgust; (*c*) expectations from foods; and (*d*) sensory properties while eating. Thus the neuroendocrine pathways regulating energy balance must intersect with systems regulating pleasure and reward (Saper et al. 2002). Leptin is known to inhibit sweet-sensitive taste cells in the tongue (Kawai et al. 2000). Thus, when leptin levels increase (as in overfeeding), perception of the sweetness of food declines and with it the range of food intake because of the reduced palatability of foods with sweetness as a component of their flavor. Furthermore, the gut peptide CCK is a physiological satiety factor in humans. However, little else is known of the links between the pathways for pleasure and reward and those regulating hunger and satiety (Flier 2004).

Food cues involve learning because there are few unlearned sensory preferences for foods and taste among humans (Mela & Catt 1996). Although infants have innate preferences for sweetness in food and aversions to sour and bitter tastes, food preferences are molded from birth, both culturally and behaviorally. At any given time they are highly stable, but are heavily determined by social contexts of eating, in addition to expectations from foods prior to eating (Mela 1999). In the absence of food limitation in either volume, weight, or energy, the most-powerful behavioral influences on the amounts of food eaten by humans include (*a*) other individuals at a meal (de Castro 1999, Bell & Pliner 2003, Wansink 2004), (*b*) television viewing (Stroebele & de Castro 2004), (*c*) the size of food packages and portion sizes (Rolls et al. 2004, Wansink 2004), (*d*) palatability (Spitzer & Rodin 1981), (*e*) the energy density of food (Stubbs 1998), and (*f*) the consumption of caloric beverages with a meal (Della Valle et al. 2005). In all cases, the greater the size or dose of the influence, the more is eaten. Influences on the energy intake and density of food eaten in societies where there is food insecurity include (*a*) economic constraints (Darmon et al. 2002, 2003; Drewnowski & Darmon 2005), (*b*) consumption of energy-containing beverages (Bray et al. 2004), (*c*) passive overconsumption of fat in high-fat diets (Blundell & Stubbs 1997), and (*d*) social and cultural mores (de Garine & Pollock 1995). There remains, however, great within-population variation in food-consumption behavior not explained by these factors. Both food novelty and palatability play to powerful behavioral influences on the size of food intake. Many industrial food products appeal to the palate as well as being energy dense, whereas the range of novel food items marketed in the industrialized world has increased dramatically in the past 20 years. Furthermore, technology in such societies has now divorced the sensory and nutritional attributes of foods in a way that prevents learning by associative conditioning of palatability, appetite, and satiety (Stubbs & Whybrow 2004). The extent to which this uncoupling between sensory and nutritional properties of food is responsible for overconsumption is not known, however (Stubbs & Whybrow 2004).

Two theories have been proposed to explain between-individual differences in food consumption behaviors and the tendency to

overeat. The first, that of externality, postulates that obese people are more reactive to external cues (such as the time of day, presence of food, and situational effects) and less sensitive to internal physiological hunger and satiety signals than are lean subjects (Schachter & Rodin 1974). According to this view, high reactivity to external cues encourages overeating and the development of obesity in environments in which highly palatable food is readily available. However, evidence confirming such an eating style among obese subjects is not conclusive (Brownell & Wadden 1991). If it exists, externality may be an antecedent of obesity and not a consequence or correlate of overeating (Mela 1996). The second theory, that of psychosomatic feeding, focuses on emotional eating (Allison & Heshka 1993). The usual response to arousal (such as fear, anxiety, or anger) is loss of appetite. However, some individuals respond by overeating. This may be a result of an inability to differentiate between the need for food and other uncomfortable sensations and feelings, probably as a result of early learning experiences. The balance of conflicting evidence suggests relative overeating in obese individuals during negative emotional states (Geliebter & Aversa 2003). However, people can counteract their externally or emotionally induced tendency to overeat by cognitive restraint of their food intake. Obese people are less likely to practice restraint in eating, however (Herman & Mack 1975), regardless of any psychological reason as to why they might overeat.

CULTURAL, BEHAVIORAL, AND ECONOMIC PERSPECTIVES

Although good food security and sedentization of life are central to the generation of obesogenic environments, various other factors also contribute to them. The maintenance of parsimonious food behaviors, involving avoidance of waste or getting the best value for money, becomes maladaptive in the context of good food security. Indirect evidence for waste avoidance comes from studies in the United States and Mexico that show humans are largely blind to portion size (Rolls et al. 2004, Mrdjenovic & Levitsky 2005, Levitsky et al. 2005, Wansink & Cheney 2005), with increased portion size predicting increased energy consumption (Fisher et al. 2003, Dilberti et al. 2004, Levitsky & Youn 2004, McConahy et al. 2004, Rolls et al. 2004, Wansink & Cheney 2005). In the United States, portion sizes of prepackaged and restaurant-prepared foods have increased greatly across the past two decades (Harnack et al. 2000, McConahy et al. 2002, Young & Nestle 2002, Smiciklas-Wright et al. 2003, Kral & Rolls 2004). Increased snacking and decreased structure of meals have also taken place in many industrialized (Decarli et al. 2000, Samuelson 2000, Jahns et al. 2001, Zizza et al. 2001, Crooks 2003, St-Onge et al. 2003) and industrializing nations. Children in the United States eat more food away from home, drink more soft drinks, and snack more frequently than 20 years ago (Jahns et al. 2001, Nielsen et al. 2002, St-Onge et al. 2003). Snack foods are often densely caloric, prepared, processed, and packaged foods (Nielsen et al. 2002). Both adults (Tucker & Friedman 1989, Tucker & Bagwell 1991, Jeffrey & French 1998) and children (Dietz & Gortmaker 1985, Del Toro & Greenberg 1989, Renders et al. 2004, van den Bulck & van Mierlo 2004,) often snack without feeling physically hungry, especially when distracted by an external stimulus, such as watching television (Stroebele & de Castro 2004). It is more difficult for humans to accurately monitor how much they have eaten when distracted (Wansink 2004). Television also increases exposure to the commercial marketing of energy-dense foods. In the 1990s, children in the United States watched on average approximately 10,000 television advertisements for food each year, 95% of which were for foods in one of four categories: sugared cereals, sweets/chocolate, fast food, and soft drinks (Brownell 2002). However, although both time spent watching television and rates of obesity increased concurrently in the United States since the 1960s, a causal link

between the two has yet to be demonstrated (Gorely et al. 2004).

Fast food has characteristics that favor the development of obesity, including its high-energy density, fat, and fructose content (Isganaitis & Lustig 2005), and Jebb (2003) has proposed a possible mechanistic link between fast foods, energy density, and obesity. However, Bandini et al. (1999) found that obese adolescents in the United States eat no more fast food than nonobese adolescents, emphasizing that excess energy intake may come from a variety of food sources and not solely from energy-dense snack foods. Although the globalization of fast food is beginning to affect children's eating patterns in many countries undergoing nutrition transition, the contribution of fast food and soft drinks to children's diet remains relatively small compared with the United States (Adair & Popkin 2005).

Increasing time constraints on home cooking in food-secure nations also likely contribute to obesity rates because of high female engagement in the workforce (St-Onge et al. 2003). A consequence of this has been the emergence and rise in demand for prepackaged convenience foods with short preparation times (Schluter & Lee 1999) and of food consumption away from the home (Lin et al. 1996, McCrory et al. 1999, French et al. 2001, Nielsen et al. 2002, Critser 2003, St-Onge et al. 2003). Both phenomena have increased dependence on industrialized food in many countries. Other time-saving devices (including drive-through, 24-hour, take-away, and home-delivery food services) have helped make food ubiquitous in everyday life in the United States (Brownell 2002) and increasingly elsewhere.

In the United States, Sweden, United Kingdom, Poland, and other industrialized nations, the inverse relationship between socioeconomic status and obesity has been explained by class differences in obesity-relevant health behaviors that have persisted, with people of higher social class eating diets with lower fat content, exercising more, and being more likely to diet to control weight (Jeffrey et al. 1991, Molarius 2003, Bielicki et al. 2005, Stamatakis et al. 2005). It has also been linked to dietary energy density and energy cost (Darmon et al. 2002, French 2003). In the United States, the price of fresh fruit and vegetables has increased as a proportion of disposable income across time, whereas the price of refined grains, sugars, and fats has declined (Sturm 2005). Diets that are more energy-dense are associated with lower daily food consumption costs (Drewnowski & Darmon 2005). However, they also have lower effects on satiety and can result in passive overeating and weight gain (Prentice & Poppitt 1996). Obesity may thus be linked with disparities in food choice because affordability and accessibility to foods recommended or seen as healthy may be limited by financial constraints in low-income groups. In an ecological study of obesity in 21 developed nations, Pickett et al. (2005) found income inequality to be positively associated with energy intake and obesity.

Low levels of physical activity are associated with an increased risk of obesity (Erlichman et al. 2002), and obesogenic environments not only discourage physical activity but also encourage inactivity both occupationally and during leisure time (Hill & Peters 1998, Brownell 2002, Hill & Wyatt 2005). There has been a great decline in occupationally related activity since the turn of the twentieth century (Popkin et al. 2005). In industrialized nations and urban areas of developing countries, jobs requiring heavy manual labor have been largely replaced by jobs in service and high technology sectors, which require minimal physical exertion (French et al. 2001). The increased use of automobiles and public-transportation systems encourages inactivity, whereas increased time spent watching television, playing electronic games, and/or using computers has increased sedentary behavior of both adults and children (Hill & Peters 1998, Jeffrey & French 1998, Brownell 2002). Obesity is uncommon among occupational groups that undertake high

levels of physical activity during working hours. In one population with high levels of obesity, Keighley et al. (2006) found that adults in American Samoa engaged in farm work had lower BMIs than those not engaged in such work. In the United States and elsewhere, children participate less in physical activity at school (Hill & Peters 1998), whereas unsafe neighborhoods and limited access to recreation areas in some urban environments discourage leisure-time physical activity (Pucher & Dijkstra 2003).

Cultural variations of appropriate and preferable body image (de Garine & Pollock 1995) also may have contributed to obesity rates. In some societies, larger body size has traditionally been seen as attractive and indicative of attributes such as health, fertility, beauty, wealth, and power. In a cross-cultural comparison of appropriate body size in different traditional societies, Brown (1991) found that the vast majority favored plumpness as being attractive. Such societies include ones in Nauru, Samoa, and Malaysia (de Garine & Pollock 1995). Various societies across the world practice or have practiced ritual fattening to promote fertility, marriageability, and embodied social status. These include groups in Africa, Central and North America (Mexican Americans and African Americans in particular), Japan, and the Pacific (de Garine & Pollock 1995). Among these, only populations in the Pacific now experience widespread obesity.

Relationships between obesity and perceived attractiveness vary among communities and societies. African American women prefer body size that is larger, on average, than similar groups of European American women (Stevens et al. 1994, Flynn & Fitzgibbon 1998, Becker et al. 1999, Fitzgibbon et al. 2000). Furthermore, overweight and obese African American women perceive themselves as healthier, more attractive, and more attractive to the opposite sex than white women of similar weight and age (Stevens et al. 1994, Flynn & Fitzgibbon 1998, Becker et al. 1999), whereas European Americans experience dissatisfaction with their own body size at lower BMIs than either Hispanic Americans or African Americans (Fitzgibbon et al. 2000).

Studies showing an increased value of thinness and increased awareness of the risk factors associated with overweight and obesity suggest that sociocultural factors, such as participation in the global economy and exposure to western ideas, may influence body-image perceptions worldwide. A number of communities and societies in which obesity has risen in recent decades and that previously were shown to desire and/or accept larger bodies and obesity now prefer thinner bodies (Madrigal et al. 2000, Anderson et al. 2002, Tur et al. 2005). This has been observed among African American girls (Katz et al. 2004) and women with diabetes (Anderson et al. 1997, Lieberman et al. 2003), British Bangladeshis with diabetes (Greenhalgh et al. 2005), Turkish adolescents (Canpolat et al. 2005), Pacific Islanders (Wilkinson et al. 1994, Craig et al. 1996, Brewis et al. 1998, Becker et al. 2005), the Ojibway-Cree in Canada (Gittelsohn et al. 1996), urban Native American youth (Rinderknecht & Smith 2002), and Korean children (Lee et al. 2004). Among Europeans, the desire for thinner body size is increasingly observed in children and adolescents and is not confined to females of upper-socioeconomic status (Story et al. 1995, Katz et al. 2004, Lee et al. 2004, Canpolat et al. 2005). Although high cultural valuation of body fatness may contribute to the emergence of obesity, it may possibly cease to be an important contributor in subsequent generations.

CONCLUSIONS

Increasing rates of obesity across the world are broadly attributed to environments that are obesogenic, against an evolutionary heritage that is maladaptive in these new contexts. The extensive emergence and rise of obesity among most of the world's populations indicate that the ability to become obese is universal, whereas great variation in obesity rates

across geographical regions indicates possible population differences in genetic susceptibility to obesity. Human genetics are likely to have undergone selection for traits that promote energy intake and storage and that minimize energy expenditure, and there are a great many obesity-related genotypes. Current models linking genotypes with physiology and feeding behavior are only able to explain a small proportion of all obesity, but they may underlie the more-common and genetically complex forms of human obesity. Studies of the regulation of energy balance show that human physiology exerts strong control of feeding under conditions of fasting or food shortage, but only weak control on reducing food intake and increasing energy expenditure under conditions of replete energy stores and good food security. Thus it is almost inevitable that obesity should have emerged as a major human biological phenomenon in the environments that have been constructed in industrialized nations over the past 60 years and that have been transferred across the world with modernization since.

Because of the diverse contexts in which obesity has emerged and the complex environments in which it persists, a ubiquitous reversal in the prevalence of obesity at any stage is unlikely in the near future. Any ceiling on the potential for obesity in the majority of the world's populations is clearly far from reached. However, we should avoid the danger of extrapolating from the recent past into the future because present obesity patterns are outcomes of conjoining forces: (*a*) a continuing economic development with comparatively few serious setbacks; (*b*) an increased food security for much of the world's populations, yet unchanged or significantly declined food security for the rest; (*c*) the penetration of the world food system into the remotest parts of the world; (*d*) declining prices for energy-dense foods; (*e*) the progressive mechanization of the vast majority of labor-intensive tasks; (*f*) the urbanization and sedentization of work in the form of service-oriented jobs as replacements for labor-intensive production jobs; (*g*) the mechanization of transport; and (*h*) the sedentization of leisure time. That humans have biological tendencies to maximize food intake and use it efficiently is clear and increasingly elaborated by physiologists, but the picture is still far from complete. The human tendency to minimize energy expenditure where possible is well-known but even less well understood biologically than food intake. However, many of the economic trajectories of the second half of the twentieth century may be environmentally unsustainable (Parker 1993, Kasun 1999, von Geibler et al. 2004) because they rely on ever-increasing consumption of global resources. Economic systems create and respond to markets, and these will change as sustainability issues become more important and new technologies emerge. These undoubtedly will be reflected in changes in human body size and nutritional status into the future. If food security continues to improve across the twenty-first century, obesity rates possibly may at least stabilize in the richer nations, while emerging and increasing at greater rates in nations that emerge economically.

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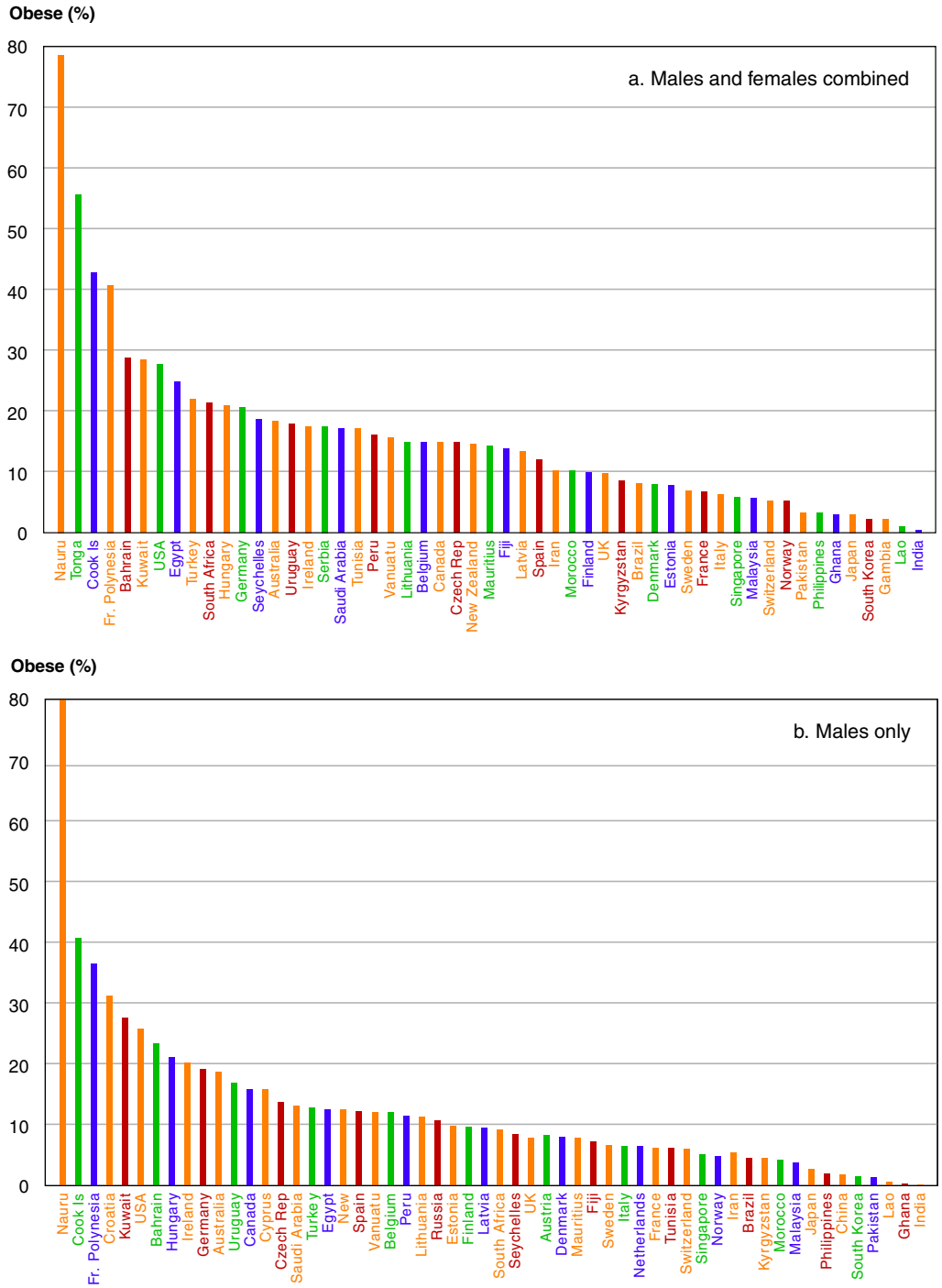

Figure 1

Obesity rates by nation (body mass index greater than 30 kg/m^2). Figure created using data from Nishida & Mucavele 2005.

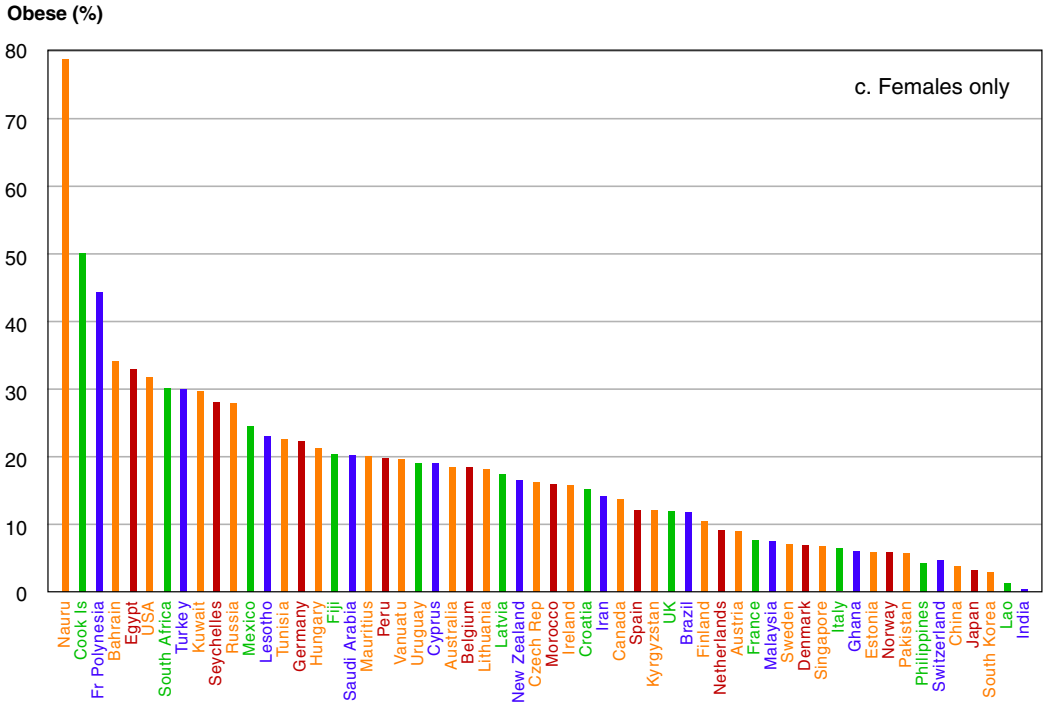

Figure 1

(*Continued*)