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THE ECONOMIC CAUSES OF OBESITY: A SURVEY

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Abstract. Obesity rates have increased dramatically over the last 30 years. Rising obesity has developed into a considerable worldwide public health problem with significant economic and social consequences. Accordingly, obesity has become a subject of economic research. This paper provides a broad survey of the recent and growing economic literature on the causes of obesity. The literature, both theoretical and empirical, overall aims to explain the rising obesity trend and to identify the factors that contribute to obesity. Also surveyed are non-economic causes and consequences of obesity and the interconnection with economic influences. An understanding of the causes of obesity allows policies to be considered that might stem the global increase in obesity.

Keywords. Obesity; Overweight; Body mass index

1. Introduction

The obesity epidemic ranks among the leading 10 global public health problems (WHO, 1998). Poor diet and physical inactivity were the second leading cause of death in the USA in 2000 and may soon overtake tobacco as the leading cause of avoidable death (Mokdad *et al.*, 2004). Obesity is a complex medical condition, which has social and psychological dimensions and some major economic aspects. It affects people of all ages and socio-economic groups, and of both genders, and is not restricted to developed countries (WHO, 2000).

Worldwide obesity rates have increased dramatically over the last 30 years. For the first time in human history, the number of overweight people, who consume more food than they need, rivals the number of underweight people, who suffer from malnutrition and hunger – there are an estimated 1.1 billion of each in the world. Both the overweight and the underweight share high levels of serious chronic diseases and disability, reduced quality of life, shortened life expectancies and lowered levels of labor productivity – each of which is an impediment to a country's growth and development (Gardner and Halweil, 2000).

Because of its significance, obesity has become the subject of economic research. Most of the growing literature on obesity has been published since 2003. Much of the economic literature has focused on trying to identify the factors which contribute to adult and childhood obesity, in order to address the questions of why

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people overeat, and what has upset the balance between energy intake and energy expenditure in recent decades. Other issues addressed in the economic literature are the consequences of obesity and the different strategies aimed at stemming the global increase in obesity rates.

The purpose of this paper is to survey the economic literature on the causes of obesity. Included in the survey are also results of research outside of economics that provide background for the economic issues associated with obesity.

The paper is structured as follows. Section 2 provides background on the classification of obesity, its worldwide prevalence, health consequences and economic consequences. This background relates to both adults and children. Section 3 organizes and covers the existing theoretical and empirical work on various biological, behavioral and environmental explanations of the rise in obesity rates. Section 4 concludes.

2. Background

2.1 *Classification of Degrees of Obesity*

The common and accepted classification of weight is based on body mass index (BMI), calculated as the weight in kilograms divided by the square of the height in meters (kg/m^2). The standard categories of BMI in adults are underweight (BMI below 18.5), normal (BMI from 18.5 to 24.9), overweight (BMI from 25 to 29.9, also called pre-obese) and obese (BMI 30 and above). In some studies, the obese category is divided to sub-categories: moderately obese (BMI from 30 to 34.9, also called obese class I), severely obese (BMI from 35 to 39.9, also called obese class II) and extremely obese (BMI of at least 40, also called obese class III). Sturm *et al.* (2004) highlight the need to distinguish moderate and more severe levels of obesity, especially since the growth rate of extreme obesity between 1985 and 2000 has been twice that of moderate obesity.

BMI does not measure body fat level directly but is considered a reliable proxy for total body fat for the majority of adults (Martin *et al.*, 2000). BMI has limitations as a measure in that it ignores body composition. Muscular persons, such as athletes, may be misclassified as obese using BMI alone.[1]

The definition of being overweight among children is statistical, based on growth charts. Being overweight is defined as at or above the 95th percentile of BMI-for-age. At risk for becoming overweight is defined as at or above the 85th percentile, but less than the 95th percentile of BMI-for-age. There are no BMI-for-age references for children younger than 2 years; therefore being overweight for children in this age group is defined as at or above the 95th percentile of weight for length (Ogden *et al.*, 2002).

2.2 *The Prevalence of Obesity*

The prevalence of overweight, obesity and extreme obesity in the USA has increased dramatically over several decades. Results from the recent NHANES[2]

Table 1. Trends in Body Mass Index and the Percentage of Obese Adults (18 Years of Age and Older).

Survey	Period	BMI	Percentage obese
NHES I	1959–1962	24.91	12.73
NHANES I	1971–1975	25.14	13.85
NHANES II	1976–1980	25.16	13.95
NHANES III	1988–1994	26.40	21.62
NHANES 99	1999–2000	27.85	29.57

Source: Chou *et al.* (2004), based on NHANES data.

survey indicate that an estimated 66% of adults in the USA were either overweight or obese in 2003–2004. The increase was concentrated in the obese category, which has more than doubled between 1980 and 2004, from 15% to 32% (Ogden *et al.*, 2006). Trends over time in American adult BMI and obesity are presented in Table 1.

However, obesity is not mostly a US problem but exists in other developed countries as well. European obesity levels have risen dramatically (see Sanz-de-Galdeano, 2005). The increase in obesity in the UK is similar to that of the USA, although it starts from a lower level. Britain's obesity problem is ranked as the worst in Europe and the third-worst in the world, behind Mexico and the USA (Howard, 2004). In Britain, most adults are overweight, and almost one in four is obese (Vlad, 2003). Figure 1 shows data on obesity in OECD countries. There has been a marked increase in the prevalence of obesity among Eastern Europeans since the region began its transformation to democracy in 1989. The level of obesity has doubled since then. Romania and the former Yugoslavia are leading the obesity epidemic in East Europe (Spritzer, 2004).

About one-fifth of the overweight people in the world are Chinese. China, who was once considered to have one of the leanest populations, is experiencing a rapid increase in the prevalence of overweight and obesity. A total of 14.7% of the Chinese were overweight and another 2.6% were obese in 2002.[3] Although the prevalence of obesity in China is relatively low compared with Western countries, the transition has occurred in a remarkably short time (Wu, 2006).

Worldwide childhood obesity is also on the rise. The prevalence of overweight children and adolescents in the USA rose by about threefold since 1980. Among American children aged 2–19 years in 2003–2004, 17.1% were overweight (Ogden *et al.*, 2002, 2006). Kim *et al.* (2006) estimated that the prevalence of preschool-aged overweight children increases by 1.21% per decade. Trends over time in American childhood BMI and overweight are presented in Tables 2 and 3.

One-third of all British children between 2 and 15 years old are overweight or obese. As many as 8.5% of 6 years olds, and 15% of 15 year olds, are clinically obese (Vlad, 2003; Howard, 2004). Rates of childhood obesity in Canada

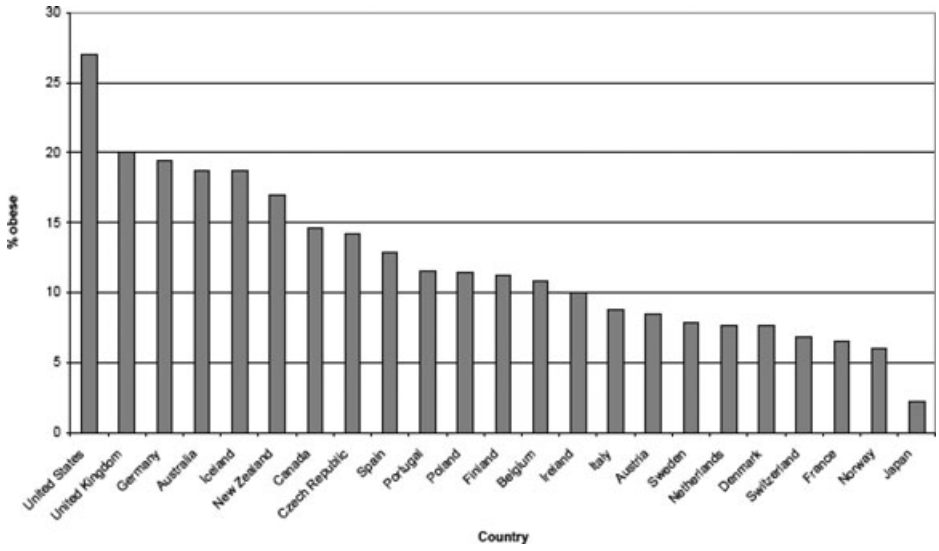

Figure 1. Obesity in International Perspective.

Source: Cutler *et al.* (2003), based on OECD Health Statistics (2000).

Table 2. Trends in Body Mass Index and the Percentage of Overweight Children (Age 3 to 11).

Survey	Period	BMI	Percentage overweight
NHES II	1963–1965	16.63	4.24
NHANES I	1971–1974	16.44	5.33
NHANES II	1976–1980	16.64	7.33
NHANES III	1988–1994	17.15	10.59
NHANES 99	1999–2000	17.37	14.26

Source: Chou *et al.* (2005), based on NHANES data.

have almost tripled over the past 20 years (Spurgeon, 2002). The prevalence of overweight and obesity among Australian children and adolescents nearly doubled since 1980. Almost a quarter of them are overweight or obese (DAA, 2003). Lissau *et al.* (2004) investigated adolescents in 15 countries in 1997–1998, and found that the countries with the heaviest youngsters are the USA, Ireland, Greece, Portugal, Israel and Denmark. Lithuania had the lowest obesity rates in this study. Among Lithuanian 15-year-olds, about 2% of girls and 0.8% of boys were obese, and 8% of girls and 5% of boys were overweight.

Table 3. Trends in Body Mass Index and the Percentage of Overweight Children (Age 12 to 18).

Survey	Period	BMI	Percentage overweight
NHES I, III	1959–1962, 1966–1970	20.61	4.45
NHANES I	1971–1974	20.97	6.82
NHANES II	1976–1980	21.03	5.63
NHANES III	1988–1994	22.11	10.62
NHANES 99	1999–2000	22.82	14.75

Source: Chou *et al.* (2005), based on NHANES data.

2.3 *Related Health Problems*

Obesity is a major risk factor for many chronic conditions, including type-2 diabetes,[4] cardiovascular disease (CVD), hypertension, hypercholesterolemia, certain types of cancer (mainly colon, breast, endometrial, liver, esophageal and kidney), stroke, asthma, sleep apnea, musculoskeletal diseases (arthritis and chronic back problems), stomach ulcer, gallbladder diseases (gallstones) and chronic liver disease.[5] Most of the chronic conditions are preventable. Out of these conditions, diabetes may be most closely linked to obesity (Flegal *et al.*, 2002).

The full long-run consequences of increased obesity rates at the aggregate level are not yet visible (Sturm, 2002). Most studies clearly show, however, an increase in mortality rates associated with obesity, particularly with higher levels of obesity. Obese people have a 50%–100% increased risk of death from all causes compared with normal weight individuals (Mokdad *et al.*, 2004; Flegal *et al.*, 2005). Obesity has approximately the same association with chronic health conditions as 20 years of aging; this greatly exceeds the associations of smoking or problem-drinking (Sturm, 2002). Fontaine *et al.* (2003) have estimated the expected number of years of life lost (YLL)[6] due to being overweight across the life span of an adult and found that obesity appears to lessen life expectancy markedly, especially among young adults. For severely obese white men aged 20–30 years, this suggests a 22% reduction (13 years) in expected remaining life span.

Sturm *et al.* (2004) estimated the association between obesity and disability. By analyzing trends between 1985 and 2002 and extrapolating to 2020, they showed that if current trends in obesity continue (holding everything else constant), the effects of unhealthy weight gain can reduce or even reverse past improvements in health among the elderly, unless other societal changes or future medical advances compensate. As Sturm (2002) has noted, even though the medical complications of overweight (pre-obese category) are smaller than complications of obesity, they apply to a very large population and therefore have significant impact.

In addition to the medical problems, in many cultures there is a social stigma attached to being overweight. Women in particular experience great social

and psychological pressure with respect to body size (Averett and Korenman, 1996).

Increasing childhood obesity is related to increasing adult obesity. Obesity even in very young children is correlated with higher rates of obesity in adulthood (Anderson and Butcher, 2006a). In addition to the long-term risks, there are also immediate medical and psychological consequences of being overweight in children and adolescents.[7] Overweight children tend to suffer from cardiovascular problems, metabolic disorders and generally from a significantly lower health-related quality of life. Many obesity-related health conditions once thought applicable only to adults are now being seen in children and with increasing frequency (Daniels, 2006).

2.4 *The Economic Burden of Obesity*

Obesity has major economic aspects. Obesity affects demand for and supply of health care. It has relations to demand and supply in markets for food, fast food, restaurants, advertising, physical exercise and dieting. The epidemic of obesity absorbs increasingly greater health care budgets and attracts increasing concern from governments. Western governments spend a large share of their expenditures on health, and this share is rising over time.[8] Health spending has outpaced economic growth in many countries, putting pressure on government budgets (Pear, 2004). Health expenditure (as a share of GDP) in the USA was 15.2% in 2003.[9] Obesity and being overweight are contributing noticeably to the increases in health expenditures. The annual medical expenditures attributable to people being overweight and obese rival the medical expenses attributable to smoking (Finkelstein *et al.*, 2003). Obesity accounts for between 2% and 9% of the total health budget in high-income countries. The true costs are greater, as not all obesity-related conditions are included in these calculations.

The overall economic burden of obesity consists of different costs borne by governments, employers, health care organizations, insurance companies and the obese individuals themselves (Komlos *et al.*, 2004; Bhattacharya and Bundorf, 2005). The costs are direct medical treatment (surgery, pharmaceutical treatments); medical treatment of the complications and chronic diseases associated with obesity; indirect costs (lost productivity due to morbidity and mortality); cost of weight loss programmes (often combined with health club membership and sales of exercise equipment); the cost of the excess food consumed; marketing and advertising to encourage over-consumption of food; and other costs such as upsizing of public seats. The economic cost of obesity in the USA was some $117 billion in 2000 (US Surgeon General's Office, HHS, 2001). Of these costs, half consisted of medical expenditures.

Obese adults in the USA under age 65 incur annual medical expenditures that are 36%–37% higher than adults of normal weight incur, primarily because of prescription drugs (Sturm, 2002; Finkelstein *et al.*, 2003; Raebel *et al.*, 2004). Medical treatments of chronic diseases tend to be very expensive. Treatments of diabetes, CVD and stroke are especially expensive.

Obesity also has an impact on the labor market. Averett and Korenman (1996) find that obese women have lower family incomes than normal-weight women, even after controlling for family background differences. The lower economic status of obese women is explained mostly by differences in the marriage market (marriage probabilities and spouse's earnings) and partly by labor market discrimination against obese women. Cawley and Danziger (2005) bring some evidence on weight-based discrimination of women in wages and employment.[10]

3. Literature Review

The reason for being overweight is a positive balance of calories: calorie consumption exceeds calorie expenditure. WHO (1998) views the fundamental causes of obesity as sedentary lifestyles and high-fat, energy-dense diets.

Unlike cigarettes or drugs, food is an essential good. A minimal level of food consumption is required for living and for maintaining body weight but why do people overeat? What has upset the balance between energy intake and expenditure in recent decades? In the non-economic literature, blame has been largely attributed to our genes (Comuzzie and Allison, 1998; Barsh *et al.,* 2000), advertisements on television (Taras and Gage, 1995; Gore *et al.*, 2003), super-sizing at restaurants (Young and Nestle, 2002), consumption of fast food (Pereira *et al.*, 2005), driving cars (Hinde and Dixon, 2005) and marketing of foods directly to children (Nestle, 2006). Economists have studied the determinants of BMI and have modeled various explanations of obesity, which relate to genetic, behavioral and environmental factors that affect energy intake and energy expenditure.[11] These explanations are presented in this paper.

3.1 *Genetics*

Being overweight among children and adolescents[12] could be explained by parents' being overweight. Perhaps youth with fatter parents are able to produce more fat tissue from given calorie intakes. Obviously, the genetic component is not influenced by economic incentives. Stunkard *et al.* (1990) assessed the relative importance of genetic and environmental effects on the BMI of twins who were raised apart and together, and found that genetic influence on BMI is substantial. Their findings support earlier studies of twins and adoptees. Bouchard and Tremblay (1997) found differences in susceptibility to chronic overfeeding or in sensitivity to negative energy balance which seem to be largely explained by genetic factors. Classen and Hokayem (2005) found strong evidence that a higher degree of obesity among mothers leads to a significantly increased likelihood that they will raise an obese youth. Coate (1983) presented empirical evidence on the determinants of obesity in youth in the USA, with emphasis on isolating effects of diet and genetic influences that correlate with parental fatness. In his multivariate statistical analysis, obesity, weight growth and diet were endogenous or mutually determined within the model. Obesity and weight growth depended on diet, parent's fatness, age, race and sex. The choice of diet depended on weight, obesity status, family

income, mother's education, family size and sex. Coate found that calories, parental fatness, and age and race differences between obese and non-obese youth explain less than one-half of the overweight growth differential.[13] Socio-economic effects (family income, mother's education or family size) on diet were significant for older children (10–16 years) but not for preschool children (less than 3 years).

Philipson and Posner (1999) and Philipson (2001) point out that the genetic component of obesity may explain cross-sectional differences but cannot explain the rapid change over time in the extent of obesity. Such a change would be much slower than is observed, if the reasons were genetic transmission.

The gene pool does not change rapidly enough for a change in genes to explain the recent increase in childhood overweight and obesity. But it does appear that certain people may have a higher genetic susceptibility to weight gain. Bouchard and Tremblay (1997) conclude that there are individuals at risk of gaining weight and body fat or who are resistant to weight loss. Anderson and Butcher (2006a) explain that parents may pass along to their children a susceptibility to overweight in the presence of energy imbalance. Changes in the environment, that affect energy intake or expenditure, could then trigger weight gain in this susceptible population. Anderson and Butcher also clarify that it is difficult to differentiate the parents' influence between genetics and behavior. Parents influence children's food selection. Children can gain weight in households where more energy-dense foods are available. In addition, children's physical activity can be affected by how active their parents are.

It seems that because of its rapid development in genetically stable populations, genetics alone cannot explain the increases in obesity in recent decades. It would be more reasonable to attribute the global phenomenon of obesity to other factors affecting diet or physical activity level, combined with genetic susceptibility.

3.2 *Biological Basis*

Overeating might have a biological basis, arising from the will to survive. Our eating preferences evolved in former environments in which risk aversion (to death from starvation) led to a natural equilibrium of food consumption. If eating preferences are genetically fixed and therefore unable to adapt to rapid changes of modern environments, overeating is a manifestation of a fundamental mismatch between ancient environments and modern environments.

Food supply had always been unstable and uncertain in natural environments. Smith (2002) proposes that, in order to survive, one (an animal or a human being) had to maintain an optimal level of energy reserves, which entailed accumulating body fat during seasons of relative plenty and using the stored fat during times of relative scarcity. The fitness maximization problem is posed as simultaneous choice of seasonal allocations of energy reserves:

$$g(x_{ps}, f_s, x_{sp}, f_p) \tag{1}$$

where f is body fat and x denotes other uses of energy reserves, in different states of plenty (p) and scarcity (s). x_{ps} indicates that the current state is scarcity but there

was plenty in the previous period. The constraints are

$$x_{ps} = I_s + c_s(f_p - f_s) \tag{2}$$

and

$$x_{sp} = I_p + c_p(f_s - f_p) \tag{3}$$

where I is an exogenous endowment of energy income and c is a constant rate at which energy reserves from the previous period can be converted to other uses (or conversely from energy income to current-period reserves). As revealed by first order conditions, an excess of body fat is generated during seasonal plenty, up to the point at which the associated marginal fitness cost is just offset by the marginal fitness benefit of closing the gap caused by food shortages. In other words, consumption smoothing is taking place.

In environments where food supply is abundant and stable, consumers may still follow ancient embedded preferences of accumulating energy reserves for an uncertain future. They thus fail to maximize individual fitness, in the biological, Darwinian sense, and find themselves in a stable disequilibrium of chronic overeating. Smith states that obesity appears to be exacerbated by poverty, by food insecurity, by the length of winter months, and by malnutrition early in life. Extensive medical, genetic and molecular evidence is presented to support his hypothesis.

Smith and Tasnadi (2003) propose a theory of natural addiction in which consumption of sweetened foods causes an immediate release of β-endorphin in the brain. Acute exposure to sweets reduces pain. The feeling is similar to that associated with extended physical activity ('runner's high'). In natural settings, sugar was conventionally associated with valuable nutrition because it was found only in ripe fruit and raw honey. The 'belief' embedded in our genes is that foods containing simple carbohydrates (sugar) are nearly always nutritionally valuable. The advent of commercially viable sugar refining technology early in the twentieth century changed this association dramatically. Today, foods with the highest sugar content often have no nutritional value whatsoever, and provide 'empty calories'. However, the biochemical system upon which we rely when choosing our foods has not changed; it still reacts to sweet foods as if they have remained a rare and valuable commodity.

The objective in this model is to minimize the odds of death by malnutrition, or equivalently, maximize the probability of survival, which is

$$P(C_x x + C_a a \geq k) \tag{4}$$

where x and a are two foods with nutrient densities of 1 and $1/p$, and k is a minimum quantity of nutrients needed to survive. C_x and C_a are independent random variables of the nutrient concentrations in foods x and a. The budget constraint is

$$x + pa \leq m \tag{5}$$

where m is a limited capacity. This model implies a discrepancy between the behavior of the agent and the maximization of the objective utility function. The

agent expects a large benefit from his or her activities (i.e. eating sweets). When the expected benefits are not realized, the agent constantly regrets past actions, just as in addiction theories.

Smith (2004) hypothesized that people choose foods not just for flavor or by habit but also by caloric density as measured by post-ingestive feelings of satiety. Evolution seems to have conditioned human dietary choice: people used to prefer foods that were eaten by others, tasted sweet or salty, and were associated with post-ingestive satiety; all these were social and chemical signals that meant that the foods were safe and nutritionally valuable. Current food producers and advertisers have learned to isolate these signals. Fast food restaurants have featured a variety of sweetened, salty and calorie-dense foods. Thus, food advertising provides information that once served as a signal of nutritional value and product quality, even if no descriptive information is conveyed about the product being advertised. In fact, the information conveyed in television advertising is in a sense misinformation, with most food advertisements during children's television programming fitting into this category.[14] Children who choose these advertised foods are unconsciously following an ancient algorithm for choosing a healthy diet. Because human preferences cannot be altered within the span of a single lifetime, we are no longer able to solve the diet problem in an optimal way.

3.3 *Rational Addiction*

Obesity could be explained as a consequence of a widespread addictive behavior of overeating. The definition of addiction to a good is a strong positive effect of past consumption of the good on current consumption. An addiction to food means that increased current eating raises both future weight and the desire to eat more in the future. Addiction to food presents a logical explanation for why consumers persist in purchasing and consuming more food than is necessary for survival.

Becker and Murphy (1988) develop a theory of rational addiction in which rationality is modeled as dynamic maximization of utility from stable preferences. Utility is defined as

$$u(t) = u[y(t), c(t), S(t)] \tag{6}$$

where c and y are two goods (c is the addictive good), and S is the stock of 'consumption capital' (past consumption of the addictive good only). A rational person maximizes the following utility function subject to a constraint on his expenditures:

$$\int_0^T \mathrm{e}^{-\sigma t} u[y(t), c(t), S(t)]) \, \mathrm{d}t \tag{7}$$

where T is length of life and σ is a constant rate of time preference. A person is addicted if an increase in his or her current consumption increases both future consumption and marginal utility from future consumption. Such complementarities cause some steady states to be unstable. That is, the addicted person reaches an

unstable steady state of growing consumption over time. This theory can explain a wide variety of addictive behaviors, including overeating.

Rational addiction to food, in the sense of Becker and Murphy, means that consumers take costs and benefits into account and do not overeat out of some pathological obsession. Therefore, price-based policies can in principle be effective in modifying behavior.

Cawly (1999) applied the rational addiction model to caloric intake and found support for the hypothesis that consumption of calories is addictive. Richards *et al.* (2004) hypothesized that calories from different sources may differ in their addictive properties. They found evidence of rational addiction to specific food nutrients (fat, protein, carbohydrates and sodium) with especially strong addiction to carbohydrates.

As opposed to this empirical evidence, Auld and Grootendorst (2004) demonstrated rational addiction even for consumption of non-addictive goods (such as milk and eggs). They showed that time series data will often be insufficient to differentiate rational addiction from serial correlation. Their criticism demonstrates the difficulty in implying that some half of the population in the developed countries became addicted to food in recent decades.

3.4 *Satisfaction from Food*

Food consumption may be neither addictive nor habit forming. Rational consumers may rather maximize their satisfaction from food subject to their personal constraints (see Cawley, 2004b). Levy (2002b) has set out a dynamic model in which consumers are rational lifetime planners. People rationally balance the marginal satisfaction from food consumption against marginal deterioration of health. That is, they reduce consumption of food when physical health and appearance become critically inadequate and increase it when physical health and appearance are improved. Consumers maximize their expected lifetime utility from food consumption:

$$\int_0^T p(W(t) - W^*)^2 \left\{ \int_0^t \mathrm{e}^{-\rho\tau} u(c(\tau))\,\mathrm{d}\tau \right\} \mathrm{d}t \tag{8}$$

where T is consumer's life expectancy ($0 \leq t \leq T$) and ρ is rate of time preference. Weight influences life expectancy: the probability of dying at time t, $p(t)$, rises with the quadratic deviation of $W(t)$, weight at time t, from the physiologically optimal weight (W^*). Weight is gained by consuming food and lost through burning calories. Utility is derived from the individual's instantaneous satisfaction from eating, subject to

$$\dot{W} = c(t) - \delta W(t) \tag{9}$$

where δ is a positive scalar indicating the marginal effect of weight on burning calories. Levy uses a specific utility function in order to reach the optimal solution. Although a deviation from the physiologically optimal weight increases

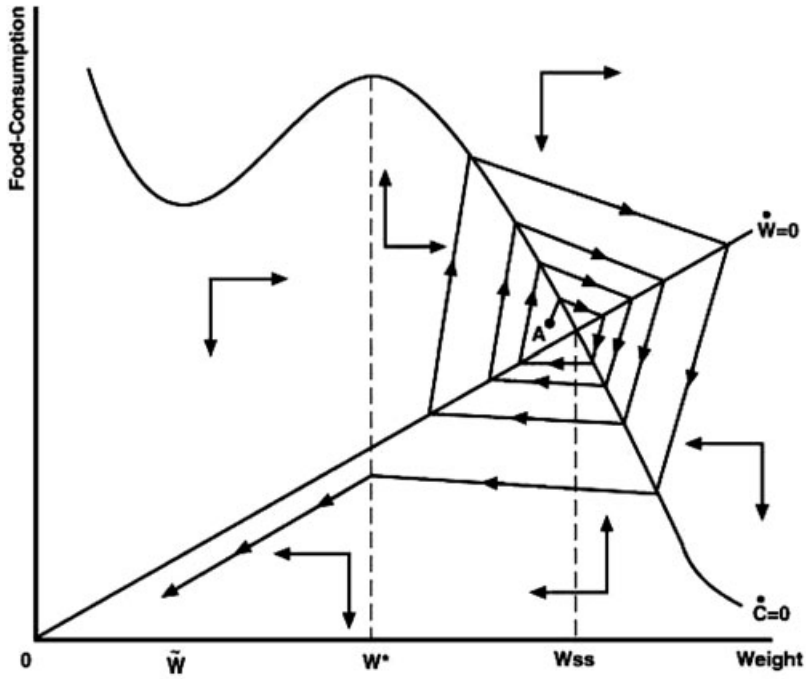

Figure 2. Phase Plane Diagram of Food Consumption and Weight.
Source: Levy (2002a).

the probability of dying, the anticipated stationary state for an expected rational lifetime-utility maximizer is a state of being overweight. Figure 2 illustrates the steady state of overweightness and the possibility of chronic loss of weight in a late stage of life (starting at point A). The rationally optimal level of weight increases with the individual's rate of time preference and declines with the rate of calorie burning and the rate of decline of the probability of continuing to live. Levy also incorporates socio-cultural norms into the basic model and shows that when there exists a socio-cultural norm of appearance, the rationally stationary weight of fat people is lower than otherwise and the rationally stationary weight of lean people is greater than otherwise.

Levy (2002a, 2003) has set out another dynamic model in which consumers maximize their expected lifetime utility from two types of food, junk food and health food:

$$\int_0^t \mathrm{e}^{-\rho\tau} u(\tau)\,\mathrm{d}\tau \tag{10}$$

where t is consumer's life expectancy and ρ is rate of time preference. Utility

is derived from the individual's instantaneous satisfaction from eating junk food:

$$u(t) = u(c_j(t), c_h(t)) \tag{11}$$

where c_j is consumption of junk food and c_h is consumption of health food. It is assumed that junk food is cheaper and also tastier than health food, and the individual's instantaneous income is fully spent on buying junk food and health food. In this model, consumers can thus rationally balance the marginal satisfaction from junk-food consumption against marginal deterioration of health. That is, they reduce consumption of junk food when physical health and appearance become critically inadequate and increase it when physical health and appearance are improved. The results are that junk-food consumption over time is affected by aging, the rate of time preference and the relative price of junk food.

3.5 *Long-term and Short-term Inconsistencies*

In Western societies there are claims that people are eating better and improving their understanding of the benefits of a healthy lifestyle but nonetheless they are becoming heavier and increasing their risk of suffering from diet related illnesses. For example, while the incidence of obesity has been steadily increasing in the USA, there has been a parallel increase in sales of organic and low-fat foods. Mancino (2003) points to this inconsistency and suggests that individuals attempt to incorporate beliefs about healthy eating into their food choices but also forgo good intentions for more immediate gratification because of situational factors such as time pressure, hunger and the need for convenience. In this model, an individual makes consumption decisions on a per-meal basis (m) over a finite planning period, and maximizes utility:

$$U_m = U(F_m, NF_m, TL_m, H_m) \tag{12}$$

where F is a vector of food goods, NF is a vector of non-food goods, TL is leisure time and H is the individual's perceived health status. Within each planning period, an individual decides whether to spend income on food or non-food, and faces the per-period budget constraint

$$P_F F_m + P_{NF} NF_m \leq y \cdot TY \tag{13}$$

where P_F and P_{NF} are the prices of the food and non-food goods, y is the wage rate and TY is working time. Within each decision period, an individual decides how to divide available time between working, preparing food and leisure, and faces the per-period time constraint

$$TY_m + Tf_m \cdot F_m + TL_m = TT_m \tag{14}$$

where Tf_m is preparing-food time and TT_m is total available time. There is also a vector of exogenous hunger-related factors, α_m, experienced at the time the individual makes his consumption decision, which influences only the utility derived from food and leisure. The solution yields a demand function for calories

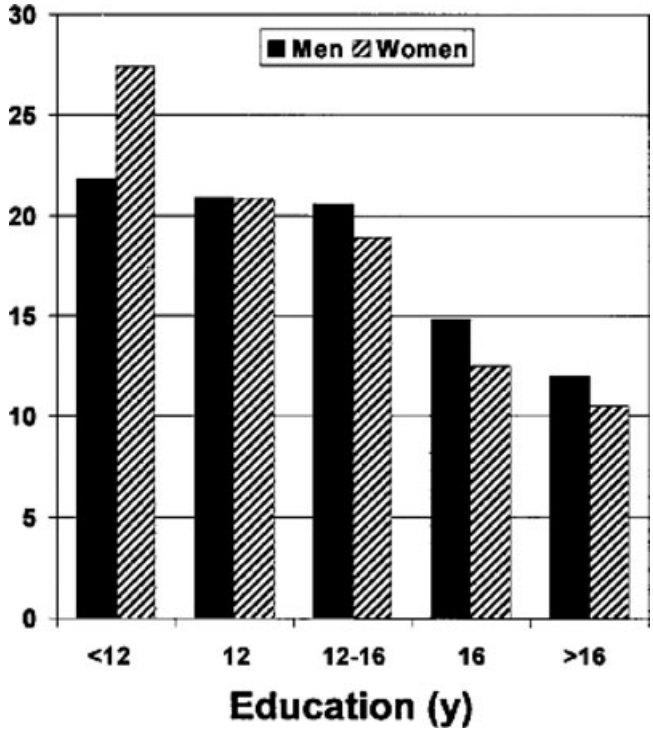

Figure 3. Obesity Rate as a Function of Education Level.
Source: Drewnowski and Specter (2004).

as a function of food prices, price savings, income, situational factors (like hunger), health information, physical activity and body weight. The equilibrium conditions reveal elements that induce actual behaviors contrary to personal long-term health objectives and self-interest: consuming food prepared away from home, less knowledge about health and nutrition, high opportunity cost of time, and low price savings from food preparation. Empirical results are viewed as validating the theoretical model.

3.6 *Education and Schooling*

Education can be health promoting through knowledge. Figure 3 presents obesity rates as a function of education levels and shows how schooling can be linked to behavior affecting health and weight. Nayga (2000) has empirically examined whether the effects of schooling on obesity are due to differences in individual health knowledge. Households produce commodities such as health of family

members and maximize the joint utility function

$$U = U(X_i, H) \tag{15}$$

where X is a vector of goods and H is health outcome (i.e. obesity). The production of a household member's health is described by the production function

$$H = h(K, D_j, u) \tag{16}$$

where K is health input (i.e. diet-disease knowledge), D is a vector of individual characteristics (including schooling) and u represents unobservable determinants of health. Utility is maximized subject to technology and income constraints. Income and prices are exogenous. Health input and health outcome are treated as endogenous. The results confirm that health knowledge decreases the probability of an individual's being obese. Loureiro and Nayga (2004) have also estimated that educational policies have a significant effect on decreasing obesity rates.

Schools have a role as a location in which food is consumed. Some three-quarters of school children in the USA eat lunch at school[15] and consume about one-third of the their total calories from this meal. Few school lunches meet the guidelines of nutrition requirements. Whitmore (2004) assessed whether school lunches affect the incidence of childhood overweight. She followed a cohort of children over time starting at school entry, and found that children who eat school lunches are about 2 percentage points more likely to be obese than children who bring lunch prepared at home, all else equal. The reason is that school lunch eaters consume more calories at lunch. Both groups enter school with the same obesity rates and consume the same amount of calories during the rest of the day. Although the results explain a relatively small part of the overall obesity rate, there is a policy dimension through centralized control of the content of school lunches.

Anderson and Butcher (2006b) note that schools in the USA have often adopted unhealthy food policies and have given students greater access to junk foods and soda pop, using proceeds from sales to fund school programmes. They examined whether students' weight is higher where schools allow such food policies and found an average small effect of access to junk food on increase of BMI but this average effect was entirely driven by adolescents who had an overweight parent. That is, only adolescents with a genetic or family susceptibility to obesity were affected by the school food environment. This finding is consistent with the fact that people in the right hand tail of the BMI distribution have been putting on weight more rapidly than those in the rest of the distribution. There therefore appears to be a portion of the population that is susceptible to obesity, and school food policies are part of the environment that encourages the propensity of this part of the population to gain weight.

Also in the context of schools, Cawley *et al.* (2005) investigate the impact of decreased physical education (PE) classes in schools on youth BMI or the probability that a student is overweight. They are unable to detect any effect and they conclude that, while raising PE requirements may make students more active by some measures, currently there is no basis for increasing PE in order to prevent or reduce childhood overweight.[16]

Table 4. Trends in Childhood Obesity Rates and Mothers' Working Hours, by Family Income, Based on US Current Population Survey and NHANES Data.

Income quartiles	Rates of obesity		Average work hours/week	
	NHANES I (1971–1974)	NHANES II (1988–1994)	March 1976 CPS	March 1995 CPS
All	4.5	10.3	17.9	23.9
First quartile	5.7	14.9	15.3	17.2
Second quartile	4.2	9.6	17.4	24.6
Third quartile	5.6	8.8	18.6	26.5
Fourth quartile	2.1	9.9	20.1	27.2

Source: Anderson *et al.* (2003a).

3.7 *Working Mothers*

The prevalence of both overweight children and working mothers has risen dramatically over the past few decades, although these parallel trends may be coincidental. Table 4 shows the changes in childhood obesity and in average weekly working hours for mothers. Anderson *et al.* (2003b) investigated whether a child was more likely to be obese if the mother worked more intensively over the child's life. They found that the increased hours worked per week among mothers between 1975 and 1999 were associated with an about 0.4–0.7 percentage point increase in overweight children, which is a small share of the overall increase.

The hypothesis that female labor participation contributes considerably to the incidence of obesity has been rejected in other studies (see, for example, Cutler *et al.*, 2003; Loureiro and Nayga, 2004).

3.8 *Relative Prices*

Variations in relative prices over time can underlie variations in weight. French *et al.* (1997, 2001)[17] bring some experimental evidence from vending machines, showing that price reduction strategies which change the price differentials between high-fat and low-fat snack substitutes may cause people to alter their consumption behavior.

Chou *et al.* (2004) empirically examined consequences of changes in relative prices and measures of the per capita number of restaurants. Prices examined were for meals in fast-food and full-service restaurants, food consumed at home, and cigarettes and alcohol. Obesity was specified to depend on working hours, family income, relative prices, schooling, and marital status. Prices had expected effects on obesity and explained a substantial amount of its trend. The study controlled for age and race. Weight outcomes rose when the relative prices of food at home declined. The demand for convenience food and for unhealthy fast food was to

Figure 4. Trends in Smoking Rates and Obesity Rates.
Source: Gruber and Frakes (2006).

a large extent a response to expanded labor market opportunities for women that increased the value of household time. Increases in the relative price of cigarettes reduced smoking and contributed to increased average weight.

Rashad *et al.* (2006) also examined the effects of relative prices on BMI and obesity. An increase in the per capita number of restaurants increased obesity, and female BMI was responsive to changes in cigarette taxes. Their conclusion is that the rapid increase in obesity over time, especially during the 1980s, is due in part to the great increase in the per capita number of restaurants, and is partly an unintended consequence of the campaign to reduce smoking. Figure 4 presents the opposite trends over time of obesity and smoking.

Also in the context of relative prices, Gruber and Frakes (2006) investigated the link between smoking and obesity rates in the USA, using the cigarette tax rather than the cigarette price as an explanatory variable. They were unable to detect any evidence that higher cigarette taxes are related to higher obesity rates, but do not rule out a small weight effect from cessation of smoking.

3.9 *Urbanization*

Socio-demographic factors such as urbanization could have a role in the growth of obesity. Sallis and Glanz (2006) explain how various changes in the US built environment have promoted sedentary lifestyles and less healthful diets. Ewing *et al.* (2003) examined the relationship between urban sprawl and physical activity, obesity, and common chronic diseases, and found that urbanization increased weight. An investigation by Loureiro and Nayga (2004) of cross-country differences in obesity rates in OECD countries considered urbanization processes, dietary habits

and labor market changes. They estimated a regression in which the percentage of overweight (and obese) people depends on average caloric intake, percentage of urban population, percentage of females participating in the labor force, budget spent on preventive medicine, budget spent on education, percentage of smokers and percentage of total working population. The results showed that urbanization is strongly related to growth of obesity rates. On the contrary, Eid *et al.* (2006) found no evidence that urban sprawl causes obesity. They showed that previous findings most likely reflect a failure to properly control for the fact that individuals who have a propensity to be obese choose to live in sprawling neighborhoods.

3.10 *Technological Change*

People in developed countries might have gained weight because of technological changes. There has been a secular shift over time in employment from manufacturing and mining to services and sedentary jobs that involve less on-the-job exercise. Caloric expenditures in household work have also been reduced due to labor-saving devices. Overall physical activity has declined despite the rise in recreational exercising.

Philipson and Posner (1999) state that technological change has lowered the cost of calories (food) through agriculture innovation. Technology has also raised the cost of expending calories – people must pay (mostly in terms of leisure) for undertaking physical activity, instead of expending calories at work. More calories are thus consumed but fewer calories are expended. Utility is defined as

$$U = U(W(F, S), F, C) \tag{17}$$

where W is weight, F is food intake, S denotes the calories used in physical activity and C is alternative consumption. Weight is affected by the intake and expenditure of calories. The utility function has an inverted U-shape over weight. Utility is maximized under the budget constraint

$$C + pF \leq I \tag{18}$$

where p is the relative price of food and I is income. The necessary condition for an interior choice of calories balances the joy of eating plus the effect of the weight change induced by eating against the forgone consumption of alternative goods. The inverse U relation between income and weight follows from complementarity between consumption and weight. Increases in income promote food demand but also depress food demand as people like to be thin and healthy. Thus, in poor or early societies the obese are relatively wealthier but in wealthy societies the obese are relatively poorer. The model demonstrates that obesity is technologically induced, but also predicts that the growth in obesity is self-limiting. As technological change lowers the price of food and thereby frees up time to raise income by other forms of production, weight will not continue to grow indefinitely. Beyond a certain caloric intake level, F_M, there is a marginal cost of consuming more calories even if they are free because the utility loss from gaining weight

dominates the joy of eating. More precisely, under fairly weak regularity conditions

$$\lim_{p\to 0} W = \lim_{I\to\infty} W = W_M \tag{19}$$

which yield an upper bound on weight, $W_M = W(F_M)$. The model shows that obesity may also be increased by anti-smoking interventions (because smoking suppresses appetite) and by programmes such as food stamps. In an extension of the model, when physical activity is endogenous and income is defined as *I(S)*, if work is less strenuous, people eat less. Effects through the allocation of time show that on-the-job exercise is substituted by leisure devoted to off-the-job exercise. That is, the jogging and gym 'revolution' is a substitution brought about by technological change at work and may offset the rise in obesity due to work-related technological change. Fast food is a consequence of these technological changes rather than an independent explanation of obesity, which implies that the fast-food industry cannot be held accountable for the rise in obesity.

Lakdawalla and Philipson (2002)[18] decomposed the increase in weight that has resulted from technological changes over the last few decades into supply and demand components and concluded that about 40% of the increase in weight was due to expansion in the supply of food through agriculture innovation that has lowered food prices, while 60% was due to demand factors through more sedentary market and household work.

A direction of causality is that technological change resulted in an increase in food consumption rather than reduced exercise (Cutler *et al.*, 2003). In the 1960s, the bulk of food preparation was done by families cooking and eating food at home. Due to technological innovations of food processing, food packing and kitchen appliances like microwaves, there has been a switch from individual to mass preparation of food. Manufacturers cook food centrally and the food is provided to consumers for ready consumption, thereby taking advantages of scale economies in food preparation. The result of these technological innovations is a significant reduction in the time spent cooking and cleaning at home. This revolution lowered the time cost of food and led to a more frequent food consumption of greater variety, and thus to greater weight. Cutler *et al.* empirically reject explanations for the trend of rising obesity based on commuting patterns (driving to work) and occupational structure. These factors mostly changed before 1980 and therefore cannot explain the growing obesity rates since 1980, and cannot explain children's obesity either. Consumption of processed food has increased, however, in the past two decades. The increase in caloric intake is moreover mainly from snacks consumed at home, and not from main meals. These conclusions counter other food-related explanations for rising obesity, e.g. fast-food restaurants and increased portion sizes. The model is based on the division of labor in food preparation and uses hyperbolic discounting, which is an aspect of time preference. The instantaneous utility function is

$$U_t = C_t + U(K_t) - h \cdot Weight_t \tag{20}$$

where C is consumption of durable composite commodity and K is caloric intake. Utility is derived from consumption, while utility is lost from being overweight,

because of health and social consequences. The costs that underlie utility loss are linear with slope h. A rational consumer will consume food until the marginal consumption benefit is equal to the marginal cost. Technological innovation that allows mass preparation of food affects consumption through two variables: the price P of food (including both time and money cost) and the time delay before consumption, τ, which is the time taken to prepare food. Thus, the benefits of consumption are discounted for that interval of time. Groups (especially married women) that experienced a large reduction in the time spent preparing food also had large increases in BMI. This factor does not explain all of the increase in obesity but does explain a significant share. Obesity across countries is thus correlated with access to new food technologies and to processed food. The dramatic time savings in food preparation could represent a pure economic benefit but for the presence of self-control problems that make it possible that the changes have been welfare reducing. Weight gain from a reduction in time delay is particularly important for those people who are hyperbolic discounters. The greater is hyperbolic discounting, the more food consumption increases and the greater the weight gain.

3.11 *Time Preference*

An increase in the marginal rate of time preference may be a contributing factor in the rise of obesity. Time preference is the rate at which people are willing to trade current utility for future benefit. Various social, cultural and psychological factors influence a person's time preference. Time preference influences current food consumption decisions since immediate gratification from eating is forgone in order to gain future potential health benefits. A higher rate of time preference could reduce investment in exercise and increases caloric intake and therefore increases weight. Komlos *et al.* (2004) build a model in which individuals maximize lifetime utility:

$$\int_0^T \mathrm{e}^{-\sigma t} U(C_t, H_t(H_{t-1}, I_{t-1}))\,\mathrm{d}t \tag{21}$$

where C is consumption of goods and services, T is expected lifetime and σ is marginal rate of time preference. Health status, H, depends on past investments in health, I, and past levels of health. The lifetime budget constraint is the present value of lifetime income:

$$\int_0^T \mathrm{e}^{-rt}(P_{ct}C_t + P_{It}, I_t)\,\mathrm{d}t \tag{22}$$

where r is the market interest rate, P_c is the price of consumption and P_I is the price of health investment. The model shows that individuals with high rates of time preference prefer current utility to future potential health benefits and therefore consume more high-calorie foods and invest less in physical exercise, at the expense of lower levels of health and utility in the future. With the saving rate and consumer

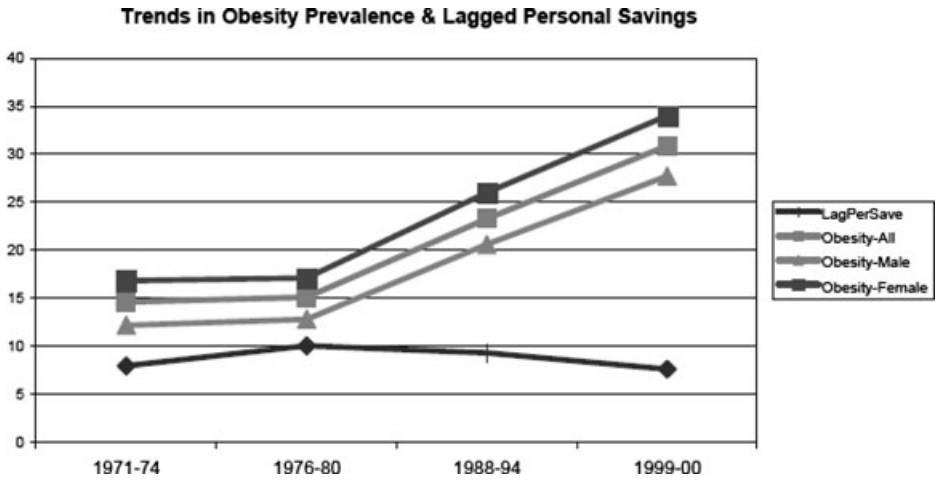

Figure 5. Trends in Obesity Prevalence and Lagged Personal Savings.
Source: Komlos *et al.* (2004), based on NHANES data.

debt as indicators of the rate of time preference, evidence from the USA as well as international evidence suggests that the link between obesity and the marginal rate of time preference is plausible. Figure 5 presents trends over time in obesity prevalence and saving rates.

Smith *et al.* (2005) find some evidence of a positive link between time preference and BMI among American youth. When they examine the data by gender, they find that higher time preference is associated with greater mean body weight among men, and to a lesser extent among women. When they break the data down by both gender and ethnicity they find that time preference is positively associated with BMI among black and Hispanic men and black women. Their results rely on the assumption that the savings data are good proxies for time preference.

Borghans and Golsteyn (2006) find no evidence for a change in the proxies for discounting rate. Their main conclusion is that overweight might be related to the way people discount future health benefits, but the increase in BMI is more likely explained by shifts in other parameters that determine the intertemporal decisions regarding the trade-off of current and future health and satisfaction.

3.12 *Poverty*

It is well known that poverty undermines health. Many health disparities are linked to inequalities in income. Obesity as well is not evenly distributed across socio-demographic groups. In richer countries, certain population groups, especially those with a lower socio-economic status or minority groups, have higher rates of obesity. Obesity often coexists with food insecurity and even with undernutrition.

Shahar *et al.* (2005) present evidence on poor diet quality and high prevalence of obesity among low socio-economic populations, compared to high socio-economic populations.[19] Wilde *et al.* (1999) find that even American children who attend government programmes for low-income or undernourished children, e.g. the National School Lunch Program (NSLP) and Food Stamp Program, do not meet recommendations for pyramid groups.[20]

The association between food insufficiency and being overweight seems to be a contradiction, since a person with insufficient food is seemingly eating too much. Basiotis and Lino (2002) propose the explanation that food-insufficient people consume cheaper foods, which are more calorie-dense. Ranney and McNamara (2002) investigated the relation between household food expenditures and the extent to which individuals meet the Food Guide Pyramid recommendations. They measured the consumer willingness-to-pay for dietary quality. They found that moving to a healthier diet that meets the dietary guidelines implies increasing food expenditures. Even though the increase in spending on food is quite small for most people, at $5–$10 per month per individual, this expense may be significant for the low-income households.

Drewnowski and Specter (2004) have drawn attention to the following points. First, as presented in Figure 6 (and Figure 3), the highest rates of obesity occur among population groups with the highest poverty rates (and the least education). Second, there is an inverse relation between energy density and energy cost, such that energy-dense foods may represent the lowest-cost option to the consumer. Third, palatability of sweets and fats is associated with higher energy intakes. Fourth, poverty and food insecurity are associated with lower food expenditures, low fruit and vegetable consumption, and lower-quality diets. Diets based on refined grains, added sugars and fats are more affordable than diets based on lean meats, fish, fresh vegetables and fruits.

Another aspect of poverty is limited accessibility to physical activity. Sallis and Glanz (2006) show that not only do low-income neighborhoods have fewer supermarkets with fresh fruits and vegetables, but they also have fewer parks, sports fields, fitness clubs and trails that create an accessible environment for physical activity.

In addition to the evidence on poverty promoting overweight, there are studies that show increasing overweight in other socio-economic groups. Kim *et al.* (2006) found evidence of increases in the prevalence of overweight and risk of being overweight in preschool-aged children, even infants, among American middle-income families enrolled in a health maintenance organization.

3.13 *Information*

Obesity could be explained by a lack of credible nutritional information on the nutritional value of food, or information on the health consequences of poor eating habits. Children are less likely to have this kind of information. Cawley (2006) notes that consumers typically have less information about the calorie content of foods they eat away from home. Variyam *et al.* (2001) estimated the degree of

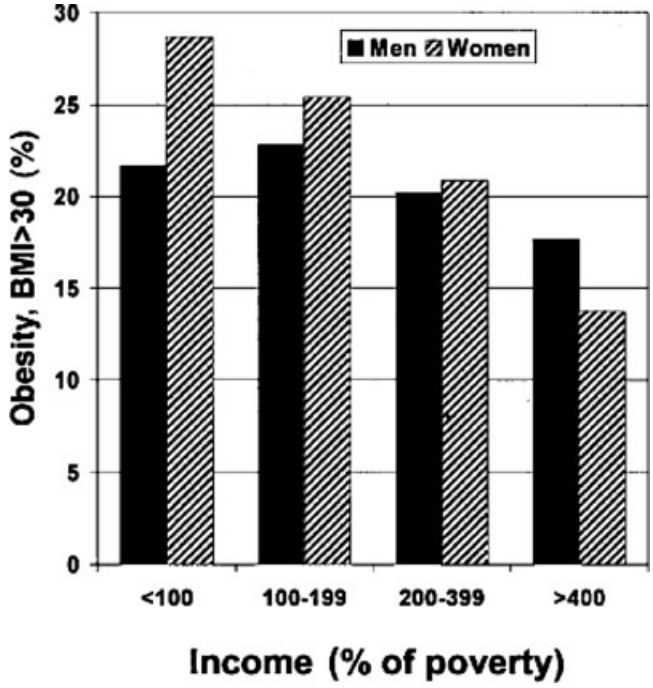

Figure 6. Obesity Rate as a Function of Income.
Source: Drewnowski and Specter (2004).

consumer misperception regarding one's own diet quality, and found that 40% of the population of household meal planners/preparers were optimists who perceived the quality of their diets to be better than their actual diet quality.

The introduction of food labeling imposed significant changes in the information about calories and nutrients that manufacturers of packaged foods must provide to consumers. Nutrition labeling is a way to meet the consumers' need for accurate, standardized and comprehensible information, and the labeling regulations are expected to help consumers choose more healthful diets. Variyam and Cawley (2006) tested whether the release of this information impacted body weight and obesity among American adults. They compared the change in body weight before and after the implementation of food labeling in the USA in 1990, among those who use labels when food shopping and those who do not use labels. The results were that the implementation of the nutritional labels was associated with a decrease in body weight and in the probability of obesity among label-reader white women.

Despite the evidence on positive correlation between label use and certain dietary characteristics, the trend of rising obesity has continued unabated even after food labels were mandated. Even though a growing number of countries are

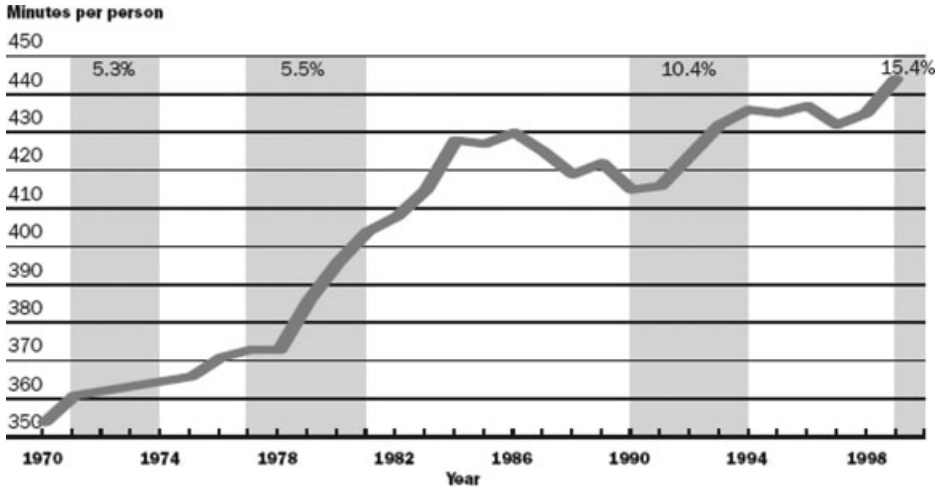

Figure 7. Trends in Average Daily Minutes of Television Watching and Childhood Obesity Rates, Based on Nielson Media Research (2000) and NHANES Data.

Source: Anderson and Butcher (2006a).
Notes: Shaded areas represent years over which BMI measures are available. The percentage of children overweight in those years is shown.

implementing mandatory nutrition labeling regulations, most demographic groups do not seem to benefit (at least in terms of body weight) from these regulations. This does not imply that the food labeling has been ineffective.

The effects of information include advertising. People are exposed to commercial advertisements for food, in particular high-calorie low-nutrient food that is highly profitable for food sellers. Children too young to be able to identify untrue or misleading claims are exposed to junk-food advertising (see Kotz and Story, 1994; Taras and Gage, 1995), which may contribute to unhealthy food choices and weight gain. Taras *et al.* (1989) found that parents perceive that television influences family purchasing patterns and eating patterns through children's requests for what they see on television advertisements. Figure 7 shows the trends in average daily minutes of television watching and in children's obesity rates. Viewing appears to be continuing to increase, as is obesity.[21]

Chou *et al.* (2005) estimated the effects of fast-food restaurant advertising on children and adolescents being overweight. Their results show a strong positive effect of exposure to fast-food restaurant advertising on BMI of children. An increase in advertising increases both BMI and the probability of being overweight in children and adolescents. The effect is more pronounced for males than for females.

3.14 *Weight as a Signal*

Under asymmetric information, weight could be a signal about wealth (Philipson and Posner, 1999; Philipson, 2001), reflecting cultural beliefs and attitudes about body image and body fatness. In the nineteenth century, when there was hunger in Western societies, thinness was a signal of malnutrition and sickness, while fatness was a signal of wealth, prosperity and high status. Signaling theory predicts that in contemporary times obesity should decline with wealth, because of the negative signal regarding health. Being thin can also be valued as a signal of self-discipline and willpower whereas excess weight can signal lack of self-discipline. These remarks pertain to contemporary Western societies. There are populations (mostly in poorer countries) that still value fatness.

4. Conclusion

The global increase in the incidence of obesity has personal and social consequences. There are also economic consequences. Obesity is a complex phenomenon. This survey has reviewed the growing economic literature on the causes of the obesity epidemic. The models and empirical research surveyed in this paper indicate that obesity is not solely, or perhaps primarily, a medical problem. Diverse factors have been identified in the literature as possible contributors to rising obesity over time: biological characteristics (for example, genetic susceptibility), behavioral aspects (for example, addiction or time preference) and environmental influences (for example, urbanization and technological change). The explanations of the striking increase in the prevalence of obesity remain under debate and are still open to further research.

An understanding of the causes and consequences of obesity is essential for policy responses. Decisions are required about whether government intervention is at all justified. This requires forecasts as to whether obesity rates are likely to continue to rise or to reverse in the future without government intervention. The answers determine the role of government in reducing obesity levels and provide input for evaluating the feasibility and cost-effectiveness of different policy instruments that might be used, including policy responses through prices (taxes and subsidies) or through information (advertising and education).

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Notes

1. Waist circumference and waist–hip ratio are other simple measures and good indicators of abdominal fat (which is a predictor of risk for heart diseases and other diseases). Additional methods of assessing obesity are underwater

weighing and skin-fold thickness. More accurate measurements of body fat are computerized tomography and magnetic resonance imaging. Most of these methods are expensive and not suitable for routine clinical use. For more details on BMI, see WHO (2003) or National Task Force on the Prevention and Treatment of Obesity (2000). See also Cawley and Burkhauser (2006) on classification and rates of obesity when measured by more accurate measures of fatness.

2. NHANES is a series of National Health and Nutrition Examination Surveys, which are conducted in the USA. These surveys include information from physicians who measure weight and height directly, so obesity calculations are as exact as possible (in self-reporting surveys overweight participants tend to under-report their weight, and all participants tend to over-report their height). Such surveys are also conducted in other Western countries. For the recent results of NHANES, see www.cdc.gov/nchs/products/pubs/pubd/hestats/obese03_04/overwght_adult_03.htm.
3. These figures were estimated according to the cut-off points, which are derived from white populations and hence may not be applicable to Asians. Using lower cut-offs would increase the prevalence of overweight and obesity significantly.
4. Previously known as non-insulin-dependent diabetes or adult-onset diabetes.
5. See, for example, National Task Force on the Prevention and Treatment of Obesity (2000), Sturm (2002), Finkelstein *et al.* (2003).
6. YLL is defined as the difference between the number of years a person would be expected to live if he/she were not obese and the number of years expected to live if the person were obese.
7. See also Johnson *et al.* (2006).
8. See The World Health Report, 2006, www.who.int/whr/2006/annex/06_annex2_en.pdf.
9. On growth of health care expenditure in OECD countries, see Kotlikoff and Hagist (2005).
10. For more on the links between weight and wages, see, for example, Baum and Ford (2004), Cawley (2004a). On the relationship between weight and wealth, see Zagorsky (2004, 2005).
11. See also Anderson *et al.* (2003a), Anderson and Butcher (2006a), Cawley (2006), who have surveyed causes of childhood obesity. See also the review of Finkelstein *et al.* (2005).
12. Apart from rare obesity-associated gene disorders, such as Prader–Willi syndrome (PWS) or Barder–Biedl syndrome (see Kopelman, 2000).
13. In this study, skin-fold thickness measurements were used as indicators of obesity. There is a rather large variation in adipose (fat) tissue among children of the same weight, and skin-fold thickness is considered a reliable indicator of total body fat.
14. See, for example, Kotz and Story (1994), Taras and Gage (1995).
15. The US National School Lunch Program is partly financed by the government. Almost half of the participants obtain their lunch free. Children from non-poor families pay full price. Others pay a reduced price.
16. For more on the role of school regarding childhood obesity, see Story *et al.* (2006).
17. See also French (2003).
18. See also Lakdawalla *et al.* (2005).
19. See also, for example, Cutler *et al.* (2003), Drewnowski (2003), Anderson and Butcher (2006a).

20. The Food Guide Pyramid is a general guide for daily eating. It calls for eating a variety of foods to obtain needed nutrients and at the same time the right amount of calories. For more details, see www.nal.usda.gov/fnic/Fpyr/pyramid.html.
21. Television watching contributes to weight gain not only through exposure to advertisements, but also by being a sedentary behavior and a cue for snacking (see, for example, Gore *et al.*, 2003).

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