

Neighborhood and Metropolitan-Level Determinants of Adult Overweight and Obesity in the United States

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Abstract: We employ multi-level statistical methods to 1993-2002 Behavioral Risk Factor Surveillance Survey, 1990 Census, and other data to estimate how neighborhood and metropolitan-level economic, physical and social factors influenced weight among non-elderly adults residing in the United States controlling for individual characteristics and behaviors. Several individual-level characteristics are estimated to have been positively (non-Latino black, Latino, low income, age) and negatively (married, employed, female, Asian, college graduate) associated with height-adjusted weight; and ethno-racial and economic segregation and the density of fast-food restaurants positively, and the price of fast-food negatively, influenced weight – even after controlling for diet, exercise, and smoking. These three behaviors explain more than 15% of the variation in weight, but were also influenced by several area-level factors. Thus, while individual characteristics and behaviors influenced body weight, area-level factors both directly (physiologically) and indirectly (psychologically) did also. We conclude by discussing how housing, labor market, and food and drug administration policies may counter the obesity epidemic in the United States in light of our empirical results.

August 2004

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1. Introduction

Demographic and Geographic Corpulence Trends and Consequences

Although the accumulation of excess adipose tissue (body fat) has risen more rapidly in Australia and the United Kingdom since 1980 and evidence suggests the epidemic is spreading to developing countries (Friedrich, 2002), the United States is the most obese nation in the world (Critser, 2003; Organization for Economic Cooperation and Development, 2003). Obesity is defined as a body mass index (BMI) of more than 30 (kilograms/meters²) or about 20 percent above “ideal” body weight for a given height.¹ Compared to most of the twentieth century when weights were generally lower than recommended to maximize longevity (Fogel, 1994), recent obesity levels in the United States are estimated to have risen dramatically – from approximately 15 percent in the early 1980s to almost 23 percent in the mid-1990s according to analyses of National Health and Nutrition Examination Survey data which measure weight and height directly (Flegal, Carroll, Kucumarski, & Johnson, 1998) and from about 12 percent in the early 1990s to 18 percent in the late 1990s by those employing self-reported Behavioral Risk Factor Surveillance Survey (BRFSS) data (Mokdad et al., 2001; Mokdad et al., 1999).² Today, however, between 20 and 25 percent of adults in the United States are obese and more than half are overweight ($25 \text{ kg/m}^2 \geq \text{BMI} < 30 \text{ kg/m}^2$) despite almost 73 percent reporting having changed their diets and 60 percent claiming to have increased physical activity to achieve their desired weight (Serdula et al., 1999). Consequently, excessive weight appears neither a contemporary phenomenon nor relatively easy to avoid through behavioral change.

There are, of course, some demographic group differences in weight outcomes. Higher proportions of women, blacks, American Indians, Alaska Natives, Native Hawaiians and some Latino populations (e.g., Mexican Americans) are obese compared to other ethno-racial groups, and although Children in the United States are estimated to be only one-third as likely to be overweight or obese as adults (Jolliffe, 2004; Troiano & Flegal, 1998), recent evidence suggests that childhood overweight and obesity is a robust predictor of adult obesity (U.S. Preventative Services Task Force, 2003).³ Perhaps most striking, however, is that similar increases in obesity rates are estimated to be occurring across diverse demographic groups (Flegal & Troiano, 2000).⁴ This is generally consistent with Geoffrey Rose’s hypothesis that increases in the prevalence of an elevated level of some deviant characteristic will be associated with higher levels for the entire population rather than with higher levels among those in the upper tail of a distribution only (Flegal & Troiano, 2000; Rose, 1985, 1992; Rose & Day, 1990).

Obesity’s rapid geographic dispersion has been no less alarming than its wide demographic diffusion. Whereas only four of the states participating in BRFSS are estimated to have had at least 15 percent of their adult populations being obese in 1991, by 2000 all but one state (Colorado) was (Mokdad et al., 2001). Still, there are significant geographic differences in the prevalence of obesity across states. In 2001, for instance, Colorado remained the only state with less than 15 percent of its adult population obese (14.4 percent) and Mississippi had the highest proportion with 25.9 percent (Mokdad et al., 2003).

¹ BMI may also be computed by multiplying weight in pounds by height in inches twice then multiplying by 704.5.

² This is not to suggest that growth in height-adjusted weight did not occur earlier in the United States. Between 1864 and 1961, for instance, the BMI of men aged 19 years rose from just under 22 to almost 23, and among men aged 45 years from about 23 to almost 26 (Costa & Steckel, 1995).

³ Subcutaneous fat accumulation is most robust and increasing in early life from about 34 weeks postmenstrual age to about 9 months, and then slackens until about age 6 to 8, at which time it resumes (Tanner, 1978 [1990]).

⁴ This claim is less true for children – particularly young children – and to some extent for adolescents and young men, for whom the main BMI increases appear to be in the upper tail of the distribution (Flegal & Troiano, 2000).

These population and spatial trends – if left unchecked for a few generations – would lead to the entire U.S. adult population being overweight and obese (Foreyt & Goodrick, 1995). Despite such concerns, weak evidence that individual willpower is more important than extra-individual environmental or intra-individual physiological factors in determining the probability of being overweight or obese (Stein, 2004), and a recent interest in the geographically-specific socioeconomic determinants of morbidity and mortality among epidemiologists (Cawley, 2002; Kawachi & Berkman, 2003; Link & Phelan, 1995; O'Campo, 2003; Ruhm, 2003; Sobal & Stunkard, 1989) and economists (Cawley, 2002; Chou, Grossman, & Saffer, 2004; Cutler, Glaeser, & Shapiro, 2003; Lakdawalla & Philipson, 2002; Rashad & Grossman, 2004; Ruhm, 2003), very little work on whether and how home, work, neighborhood and metropolitan, school, and other environments influence body weight has been undertaken. Indeed, the mainstream economic and public view suggests that overweight is “an avoidable state, which can be adjusted through diet and behavioral modification” (Philipson, 2001). Latterly albeit laggardly scholarly efforts to estimate the independent and joint influence of intra-individual physiological or somatic, extra-individual contextual or environmental, and individual behavioral or psychosocial, sources of the American overweight epidemic have implications that stretch beyond those presently affected either fiscally or physically, and into the political arena and the nation’s future overall well-being (Diez-Roux, 2002).

Health and Socioeconomic Effects

Morbidity and mortality are positively associated with overweight and obesity, but there is evidence that the relationship persist even within a “healthy” range of body weight. Women with a body mass index as low as 17, for example, have been found to die at slightly lower rates than those with BMIs below the overweight threshold (e.g., between 21 and 25), and at the lower end of this range women tend to have higher rates of diabetes and hypertension than those with even lower BMIs (Willet, 2001). Claims that obesity has recently approached or surpassed smoking as the leading cause of death in the United States, consequently, offer a lower-bound estimate of the impact of body weight on death and illness. Younger adults are presently more severely affected in the United States (Fontaine, Redden, Wang, Westfall, & Allison, 2003), and more than 300,000 adults of all ages are estimated to die annually from obesity-related causes compared to approximately 400,000, 100,000 and 20,000 from tobacco, alcohol and illicit drugs (Allison, Fontaine, Manson, Stevens, & VanItallie, 1999; Fontaine et al., 2003; McGinnis & Foege, 1993; Mokdad, Marks, Stroup, & Gerberding, 2004).

Overweight and obesity also reduce overall quality of life (Fontaine & Bartlett, 1998; Fontaine et al., 2003). Because rates of diabetes and gallbladder disease, cardiovascular disease, stroke, disability, anxiety, asthma, depression, hypertension, osteoarthritis (e.g., hip fracture), certain cancers (e.g., colon, kidney, postmenopausal breast, endometrial), and lost productivity due to absenteeism and premature retirement tend to be higher as body weight rises (Cruickshank et al., 2001; Ferraro & Kelley-Moore, 2003; Friedrich, 2003; Mokdad et al., 2003; Must et al., 1999; Sturm, Ringel, & Andreyeva, 2004), conservative estimates suggest weight-related comorbidities accounted for five to seven percent of annual medical care costs in the 1980s (Colditz, 1992) and 1990s (Colditz, 1999; Thompson & Wolf, 2001; Wolf & Colditz, 1996; Wolf & Colditz, 1998) or \$75 billion in 2003 dollars (Finkelstein, Fiebelkorn, & Wang, 2004).⁵ Indeed, some scholars contend that these costs are likely to be more substantial than those for any other primary disease for the current generation (Martin, Robinson, & Moore, 2000).

⁵ Research employing data from the early 1980s placed the lifetime subsidy from others to those with a sedentary lifestyle at \$1,900 (Keeler, Manning, Newhouse, Sloss, & Wasserman, 1989).

Evidence that “unattractive” men and women generally, and overweight and obese persons in particular, experience employment and earnings penalties partly due to employer discrimination has also been mounting (Averett & Korenman, 1996; Baum II & Ford, 2004; Cawley, 2004; Cawley & Danziger, 2004; Crandell, 1994; Hammermesh & Biddle, 1994; Loh, 1993; Pingitore, Dugoni, Tindale, & Spring, 1994).

While these economic impact estimates are a key component of documenting the severity of the overweight epidemic in the United States, the most important contribution an economic analytical approach offers is a systematic statistical evaluation of the relative influence of potential causes and their policy implications (Roux & Donaldson, 2004). Below we draw upon recent studies in economics and social epidemiology to outline a multifacorial theoretical framework (section two), build a multi-level model of body weight (section three), and estimate and interpret the relative contributions of various physiological, tropismatic, and environmental factors employing 1993-2002 Behavioral Risk Factor Surveillance Survey (BRFSS), 1990 Summary Tape File (STF3) Census, American Chamber of Commerce Research Association (ACCRA), and xx data linked by zzz metropolitan areas. A fifth and final section highlights the contributions of our findings as well as qualifies them in light of data and methodological limitations, and concludes with some suggestions for future research.

2. A Tripartite Theoretical Framework

Contemporary research on corpulence may be separated into three conceptually crude yet useful camps spanning a micro-to-macro (or “downstream-to-upstream”) continuum – physiological, tropismatic, and environmental.⁶ Although our third (contextual-level) category is identical to what others adopting a multi-causal etiology of overweight or obesity employ, our first two organism-level categories have alternatively been termed “biological” and “individual” (Horgen & Brownell, 2002) or “biological” and “social” (Chesney, Thurston, & Thomas, 2001).⁷ We prefer “physiological” and “tropismatic” because humans are fundamentally biological, psychological and social – and for purposes of the present study we wish to distinguish behaviors contributing to net energy balance (e.g., eating, exercising) which may be influenced by environmental factors filtered through conscious *reflection* (e.g., geographic density of fast-food restaurants, climate, advertising) from those homeostatic functions that are largely genetic (e.g., metabolic functioning, nutrient oxidation, sympathetic nervous system activity) and relatively *reflexive* or subconscious (Price, 2002; Tataranni & Ravussin, 2002).⁸ In short, we wish to separate those tropismatic influences about which individuals are relatively sentient and therefore over which they may exert some self-control (e.g., not eating fast

⁶ Historically, examples of all three categories exist – often simultaneously (Bray, 1990; Shell, 2002).

⁷ The latter team of authors (Chesney et al., 2001) offer an even more fine-grained categorization, splitting what we are terming “tropismatic” (and they term “social”) into *psychological* and *behavioral*, and splitting what we and they term “environmental” into *proximal* and *distal* subcategories. McKeown, in his discussion of the paradox of why infectious diseases rose with initial human agricultural settlement some 10,000 years ago and then began to decline at the dawn of the first industrial revolution (when population density increased still more), contends that one must simultaneously consider biology, host response and environment (McKeown, 1979).

⁸ We are not suggesting that these three categories are in fact strictly discontinuous – only conceptually and to varying degrees (Veblen, 1914 [1941]). Certainly the adverse metabolic effects of consuming high-fructose corn syrup or palm oil, for instance, are relatively unknown and therefore subconscious, but also conditioned on their availability in contemporary processed foods and the pecuniary and non-pecuniary cost of obtaining or avoiding them (Critser, 2003). Nor do we claim that all subconscious processes contributing to weight gain are physiological. Even when individuals are educated about the dangers associated with being overweight they often make, according to economist Tomas Philipson, “an unconscious decision to accept weight gain as a by-product of lower food prices coupled with higher paying, but sedentary, work” (Mitka, 2003).

food despite its relatively low immediate cost) from those physiological and environmental influences about which they are less aware and consequently over which self-control is lacking or impossible (e.g., metabolic functioning, existence of misinformation about the nutrient value of foods from industry advertising and government recommendations).

Physiological Mechanisms

Modern work on the reflexive physiological aspects of overweight and obesity began with Swiss biologist Albrecht von Haller's observation in 1777 that hunger is associated with the excitation of stomach nerves – a theory that would remain widely accepted but empirically unproved until Walter Cannon's and Anton Carlson's research in the early 1900s (Mayer, 1968). Major advances linking these abdominal urgings to brain activity were given substantial momentum with anatomical confirmation of the so-called “satiety” and “feeding” centers in the hypothalamus area of the brain by Harvard University's Jean Mayer in 1967. This generated several new hypotheses at the time regarding how brain-to-stomach communication occurs, but almost four decades later the molecules and cell types dedicated to regulating food intake remain only partially known (Chua & Leibel, 2002). In other words, although genes that mediate the consumption of sweet-tasting carbohydrates have been mapped and there is evidence that a preference for fat is genetically determined to some degree (Reed, Bachmanov, Beauchamp, Tordoff, & Price, 1997); relatively few obese individuals have been found to be leptin-deficient or to lack a functional leptin receptor (Shell, 2002), and a recent world-wide search among obese individuals by many investigators uncovered only 50 individuals with 19 major mutations in 6 genes (Perusse et al., 2001). This disappointing search among human subjects combined with the fact that leptin plays numerous physiological roles and is generated by multiple organs has nudged researchers away from the leptin-generating system (Trayhurn, Hoggard, Mercer, & Rayner, 1999) and toward leptin-receptor research (Lee et al., 1996; Tartaglia et al., 1995). Most scholars currently attribute 25-50 percent of point-in-time body weight variability to biology (Bouchard, 1995; Comuzzie & Allison, 1998; Yanovski & Yanovski, 1999), and from 23 percent (Korkeila, Kaprio, Rissanen, & Koskenvuo, 1995) to 70-86 percent (Austin et al., 1997; Fabsitz, Sholinsky, & Carmelli, 1994) of weight variance within people over time to genes. Still, the most effective weight-reducing drugs presently available (e.g., Xenical or Olistat) have produced only modest effects when combined with diet and exercise (O'Connor & Grady, 2003).

Tropismatic Psychosocial Aptitudes

The alternative conventional view that individuals who are overweight or obese are at best psycho-pathological, or at worst lazy or sinful, also has a lengthy history. From Greece (Hippocrates warned of higher likelihood of sudden death among those who are relatively fat) to Sparta (where the corpulent were exiled) to the Roman Empire (where wives starved themselves, sometime to death, to please their husbands) to Medieval Christian Europe (where gluttony was pronounced a deadly sin) to early 20th-century America (where religious condemnation, industrial capitalism's quest for greater efficiency, and Freudian oral-fixation theory combined to attribute individual culpability to those who were obese) to the present in the United States (where individual diet and exercise are viewed as the main solution to excessive weight) – relatively reflexive biological and environmental factors have been overshadowed by comparatively reflective individual behaviors as the main determinant of body weight (Shell, 2002). Such a monomaniacal perspective has unsurprisingly resulted in research and treatments that focus primarily on changing individual psychology (e.g., negative affect) and behavior (e.g., poor diet, sedentariness) rather than social (e.g., familial or group norms) and environmental (e.g., availability of affordable healthy food) factors that are likely to

influence body weight both physiologically and behaviorally.⁹ We do not wish to suggest that psychology as a profession has historically ignored the importance of extra-individual factors influencing behavior through cognition (Slater, 2004) or that their work has not pointed away from intention and conscious choice as primary explanations for differential weight outcomes (Holmes et al., 1998). Indeed, environmentally-generated stress has received considerable attention within the discipline (Greeno & Wing, 1994) with some highlighting the failure of individual-level interventions and arguing forcefully for a multi-level, population-based, preventative approach for solving the obesity epidemic (Chesney et al., 2001; Visscher & Seidell, 2001). In sum, weight is a function of relatively subconscious physiological food intake and processing mechanisms *and* relatively sentient tropismatic responses to extra-individual stimuli. The prevalence of individual weight differences within similar contexts testifies to the obvious influential role of individual genes and behaviors (Comuzzie & Allison, 1998; Hill & Peters, 1998).

Environmental Factors

The finding that neither individual biology nor behavior changes easily or rapidly has led other students of human growth to highlight the stability of our genetic constitution during the past several centuries and to argue that while physiology and psychology clearly influence individual energy intake and expenditure and may help explain individual- and group-level weight differences, they cannot explain the population-wide increase in body weight or stature (Fogel & Costa, 1997).

Some of the most compelling early evidence for the environmental hypothesis of disease came from epidemiological studies of international migration (Kasl & Berkman, 1983). The fact that the Japanese, for instance, have low serum cholesterol levels and a very low occurrence of heart disease and that Japanese migrants to the United States following World War II experienced rapid increases in the level of coronary disease that were positively associated with geographic distance from Japan suggests cultural surroundings and diet influence health (Marmot & Syme, 1976).¹⁰ Similarly convincing work has compared genetically similar Pima Indians residing in Mexico and in Arizona, with those in the United States reported to have weighed 44 pounds more on average (Ravussin et al., 1994). The basic message is that humans are evolutionarily predisposed toward consuming and storing energy in relatively food-scarce environments, and when the environment becomes more conducive to energy intake or dissuasive of energy expenditure weight rises. But even if “biology permits obesity . . .” and “the environment causes obesity” (Horgen & Brownell, 2002), which environmental characteristics are potentially obesogenic in the United States?¹¹

Context-explicit research has generally incorporated either pecuniary (e.g., deprivation, income inequality, unemployment, food prices) or physical (e.g., sidewalk safety, suburban residence) environmental factors as extra-individual explanatory variables. One econometric exploration of state-level factors contributing to mortality in the United States in 1960 – offering an early multicausal answer – found environmental factors to be more important than medical care (McKeown, 1979), and hypothesized that an estimated (but counterintuitive) positive income-mortality relationship resulted from unfavorable diet and exercise (tropismatic) as well as stress

⁹ Several recent reviews illustrate how economists, psychologists, and sociologists have approached the role of biology, emotion, and rationality in human behavior and intimate possibilities for future consilience (Kahneman, 2003; Link & Phelan, 1995; Massey, 2002; McEwen, 2001; Robson, 2001; Syme, 2004).

¹⁰ Considerable subsequent research has provided similar evidence employing data from various sending and receiving nations (Bhatnagar et al., 1995; Jasso, 2003; Marmot, Adelstein, & Bulusu, 1984; Ravussin, Valencia, Esparza, Bennett, & Schulz, 1994; Wandel, 1993; Ziegler et al., 1996; Zigler et al., 1996).

¹¹ Obesogenic (and its opposite, leptogenic) are relatively new adjectives and refer to conditions that foster excessive weight gain or weight loss respectively.

(physiological) factors accompanying higher income which may have offset the benefits of medical care (Auster, Levenson, & Saracheck, 1969). Almost three decades later – and building on research suggesting area-level stressors (as opposed to individual-level stress) may adversely affect various health outcomes (Kawachi & Kennedy, 1997; Macintyre, MacIver, & Sooman, 1993) – the first direct estimate of how a specific environmental factor in the United States may influence weight was made. The study employed cross-sectional 1992 American Cancer Society Nutrition Survey and 1990 Census data, and reported that state-level income inequality independently augmented abdominal weight among relatively well-educated non-Latino white men aged 50 to 64 (but not among women) residing in 21 states (Kahn, Tatham, Pamuk, & Heath, 1998). Admitting the limited age, ethno-racial, and geographic nature of their data and that environmental stressors may be better captured at the intra-state level, the authors cite others' work showing a causal link between environmentally-generated stress (i.e., from inequality, unemployment, crime), the internal release of various hormones likely to augment weight (i.e., adrenaline, norepinephrine, cortisol), and increased abdominal weight (Bjorntorp, 1996; Brunner, 1997; Brunner & Marmot, 1999; Bujalska, Kumar, & Stewart, 1997; Hautanen, Raikkonen, & Adlercreutz, 1997; Jayo, Shively, Kaplan, & Manuck, 1993; Marin & Bjorntorp, 1993; Pasquali et al., 1996; Pedersen, Jonler, & Richelsen, 1994; Rebuffe-Scrive, Bronnegard, Nilsson, Gustafsson, & Bjorntorp, 1990; Resmond, Lapidus, Marin, & Bjorntorp, 1996; Shively & Clarkson, 1988). This finding in the context of a complex causal chain motivated one leading scholar in the field to term fat “the missing link” between psychosocial factors and various health outcomes (Bjorntorp, 1991), but the biological plausibility of whether infrastructural and socioeconomic characteristics of one's neighborhood or the larger metropolitan area independently influence the probability or prevalence of obesity through physiological or tropismic pathways is largely an untested hypothesis.

A second recent study used cross-sectional Behavioral Risk Factor Surveillance Survey (BRFSS) data from 44 states in 1990 as well as Census data to examine directly whether height-adjusted body weight and other disease risk factors among adults were associated with state-level income inequality, and found that inequality was positive associated with sedentariness and body weight, but only among women and especially for those at the bottom of the income distribution (Diez-Roux, Link, & Northridge, 2000). The apparent contradictory inequality-weight estimates by gender of these first two recent U.S.-based studies are likely artifacts of different outcome variables, as well as the more comprehensive age and geographic coverage in the second study. Still, if environmentally-generated stress has an independent physiological effect on the accumulation of body fat, one would expect it to function among both males and females as these studies jointly suggest.

While it appears that state-level income inequality may have adversely affected adult weight net of individual-level characteristics and behaviors in the early 1990s, inequality is only one area-level factor that may influence weight, and analysis of cross-sectional data cannot (by design) explain trends in overweight and obesity. Furthermore, whereas a third recent study estimates an inverse relationship between MSA-level income inequality and individual body weight among all adults using 1996-1998 BRFSS and 1990 Census data after controlling for individual- and other MSA-level factors (Chang & Christakis, 2004), a fourth finds that census tract-level income inequality augmented individual weight among black women in the 1980s (Robert & Reither, 2004). These initial efforts during the past decade thus focused on the important but limited task of detecting specific area-level correlates at particular points in time. Because results have been mixed, however, one should be cautious when considering speculations about how inequality influences behaviors, or produces physiological changes, likely to increase net energy balance.

The first longitudinal study to estimate the relationship between economic and weight trends analyzed state-level 1987-1995 BRFSS data and average annual unemployment rates from Bureau of Labor Statistics (BLS) data. Although economic upturns were estimated to positively affect most

health outcomes, a stronger economy was found to augment obesity partly by stimulating unhealthy diet and exercise behaviors (Ruhm, 2000). Subsequent work by the same author employing five additional years of the BRFSS data confirms this latter result (Ruhm, 2003), and we are left with a story suggesting that although income inequality may adversely affect weight so too might economic growth manifested as an employment gain.

Several other recent longitudinal studies by economists investigate area-level factors other than inequality and labor market conditions that are likely to alter energy intake and expenditure behavior. One (Lakdawalla & Philipson, 2002), for example, estimates that approximately 40 percent of the 1981-1994 rise in BMI is attributable to improved agricultural technologies and associated declining metropolitan-area food prices, and some 60 percent to declining physical activity resulting from technological advances reducing home and work effort. Although this study controls for unmeasured time effects, it unfortunately fails to do so for unmeasured area effects shown by others to be potentially significant (e.g., inequality, economic growth).¹² Nonetheless, if one defines unemployment as a drastic decline in work strenuousness, the larger estimated negative influence of energy expenditure on weight gain due to less physical effort at home and work simply offers a less acute version of the unemployment story (Ruhm, 2000, 2003). Whether in its extreme or mild form, the energy expenditure hypothesis is consistent with those who argue that caloric intake has remained remarkably constant during the last two or three decades (Hill & Melanson, 1999).

Another economist recently estimated that falling regional grocery store food prices augmented weight among young adults between 1981 and 1998 in the United States (Cawley, 2002), but others argue that falling work strenuousness and unemployment as well as grocery store prices (supply-side factors) are less important for understanding the rise in overweight and obesity since the 1970s than economic factors influencing eating (demand-side) behavior.¹³ David Cutler and associates, for instance, provide evidence from the United States intimating that adult physical activity has changed very little over the past three decades and some combination of rising caloric intake, nutrient composition, and meal frequency – resulting from more efficient food preparation and preservation technologies – are the main culprits in the American obesity epidemic (Cutler et al., 2003). Although this research does not econometrically estimate how changing food prices influenced weight in the United States, results from regression analysis using international data suggest a negative relationship between fast food prices and percent obese. Various state-level political-economic changes in the United States between 1984 and 1999 likely to influence food consumption (e.g., declining alcohol and food prices, rising tobacco prices; higher restaurant densities), furthermore, are estimated to have increased BMI and the probability of being obese (Chou et al., 2004). One useful summary of how these socioeconomic changes have contributed to greater obesity during the past two decades suggests that up to 66 percent results from increased eating out (mainly at fast food restaurants) and another 20 percent emanates from higher cigarette prices (Rashad & Grossman, 2004). It would appear therefore that there are important trade-offs between certain social goals such as lower calorie prices, higher nicotine prices, and greater female labor force participation, and others such as lower levels of overweight and obesity and associated costs.

Perhaps complementing epidemiologists' and economists' foci on how area-level income inequality, unemployment, and food prices influence excessive weight accumulation, a divergent strand of research mainly pursued by epidemiologists and urban planners investigates how the built or physical environment, as well as perceptions of these, influence body weight. Employing self-

¹² Others have also emphasized the “declining work effort” hypothesis (Philipson, 2001; Philipson & Posner, 1999).

¹³ “Food at Home Prices,” or what are more commonly known as grocery store prices, are considered a supply-side factor because food purchased in a grocery store (unlike that purchased in a restaurant) requires some effort to obtain, prepare and serve.

reported data from more than 4,000 women residing in 16 southern states in 1993, for instance, one study finds that differences in obesity were related to place of residence. Specifically, higher proportions of women in rural areas were impoverished and obese compared to those residing in urban areas, and higher proportions of those residing in urban areas were poor and obese relative to those living in suburban areas (Ramsey & Glenn, 2002). Drawing from correlation analysis only, the authors conclude that the weight impact of one's neighborhood worsens as one "moves" from a suburban to an urban, and from an urban to a rural area, due to increasing poverty.

A second study focusing on the physical and social aspects of one's environment examines how community infrastructure, perceptions of this, and worksite infrastructure influenced overweight among adults in Missouri using cross-sectional data obtained from almost 2,400 interviewees in 1999-2000 (Catlin, Simoes, & Brownson, 2003). Adjusting for a host of individual characteristics and behaviors, negative perceptions of one's self-defined "community or neighborhood" and the absence of outdoor exercise facilities, sidewalks and shoulders were estimated to be positively associated with the probability of being overweight or obese. Conversely, workplace policies such as permitting time for physical activity or the availability of facilities for such were not found to be associated with excessive weight. These results seem to be at odds with the study summarized immediately above given that suburban neighborhoods are less likely than urban neighborhoods to have exercise facilities, sidewalks and shoulders – but data are from different geographies and years.

A third recent and related analysis that employs pooled 1998-2000 BRFSS and various other data finds that urban sprawl had a small significant association with walking, obesity, and hypertension across 448 counties in the United States after controlling for various demographic and behavioral covariates, but only with walking across 83 metropolitan areas (Ewing, Schmid, Killingsworth, Zlot, & Raudenbush, 2003; McCann & Ewing, 2003).

As this literature review intimates, the etiology of overweight and obesity is not a new area of research, and cause is both multifactorial and multilevel (Weinsier, Hunter, Heini, Goran, & Sell, 1998). Physiological, tropismatic, and environmental influences have received disproportionate attention over time, however. For instance, despite a long history of disappointed personal weight-control efforts partly consequent of conflicting official dietary advice (Willet, 2001), misperceptions of one's weight status (Chang & Christakis, 2003), differential levels of obesity health risk knowledge (Kan & Tsai, 2004), the trade-off between smoking and weight gain (Flegal, Troiano, Pamuk, Kucumarski, & Campbell, 1995; Kawachi, Troisi, Rotnitzky, Coakley, & Colditz, 1996), adverse effects of more efficient food processing and preservation technologies on self-control (Cutler et al., 2003), and almost half a century of evidence suggesting physiological (e.g., central nervous system, endocrine system) psychological, and environmental factors influence caloric intake and metabolism (Greeno & Wing, 1994; Mayer, 1968) – *individual* eating and exercise behavioral choices rather than *intra-individual* genetic or *extra-individual* socioeconomic factors have often been regarded as the primary determinants of overweight and obesity in the United States (Brownell & Horgen, 2004; Critser, 2003; Nestle, 2002; Shell, 2002). As the fourteen studies summarized in some detail above suggest; however, excessive weight is increasingly being considered a consequence of environmental or extra-individual factors.

The availability and favorable price of easily digested foods (e.g., potatoes, white bread or rice, candy, pasta) compared to those that are digested less rapidly (e.g., whole grains, beans, most fruits and vegetables), for instance, provides an example where both extra- and intra-individual forces influence weight. Environmentally, between 1972 and 1999 the per capita number of fast-food restaurants doubled in the United States (Chou et al., 2004). The physiological effect is simply that foods that are digested faster accelerate the production of blood sugar (glucose) and subsequently a hormone (insulin) which delivers glucose to our muscles but then promptly stimulates more hunger (Willet, 2001). In other words, the increased availability of relatively inexpensive unhealthy foods

that are more quickly digested and have a bumpier glucose-insulin production cycle which keeps hunger at bay for a shorter period of time immediately following consumption raises overall caloric intake. All of this suggests that increasing overweight and obesity are partly determined by the interaction between environment and genes, and that one ought to examine how environments differ and have changed to alter energy-enhancing behaviors if interested in reversing the trend (Hill & Peters, 1998).

Some attribute this secular shift of attention to relatively fixed cultural conventions placing fault on overweight individuals and to public health institutions focusing historically on infectious rather than chronic disease (Horgen & Brownell, 2002), but whatever the cause such oversimplification and aversion to multi-causal approaches is understandable when weight is viewed only as individual net energy (or calorie) balance (Mayer, 1968).¹⁴ This is no longer the case, and below we offer a multi-level model that permits us to estimate how various local economic, physical and social factors influenced individual weight outcomes during the past decade in the United States.

3. Data and Empirical Model

Data

The primary data employed in this study are from the Behavioral Risk Factor Surveillance Survey (BRFSS). The BRFSS was first administered in 1984 by the National Center for Chronic Disease Prevention and Health Promotion of the Centers for Disease Control and Prevention (CDC) for 15 states. The main objective of the BRFSS monthly telephone interviews is to collect data on preventative health efforts (e.g., exercise, fruit and vegetable consumption, medical care, seatbelt use) and risky behaviors (e.g., alcohol consumption, smoking) that are associated with the nation's top ten adult diseases and injuries in an effort to provide information that may help reduce these. Specifically, data for one adult is collected from each randomly selected household, and in addition to self-reported height and weight – which are used to compute whether one is overweight or obese – information about conventional demographic characteristics as well as various behaviors and factors that may impact health outcomes are included.¹⁵ All states (including the District of Columbia) are represented in the BRFSS, and although county-level data existed for most states by 1993 this was not the case for Wyoming, Rhode Island and Washington, D.C. until 1996.¹⁶ Data at the level of Metropolitan Statistical Area (MSA) became available for the first time in some states in 1998.¹⁷

¹⁴ One calorie is about the amount of energy a 150-pound person burns each minute while sleeping or, technically, the amount of heat needed to raise the temperature of a liter of water from 14.5 to 15.5 degrees Celsius (Willet, 2001). One pound is equal to 3,500 calories.

¹⁵ Specifically, *underweight* is defined as BMI < 18.5 kg/m², *normal weight* as BMI ≥ 18.5 to 24.9 kg/m², *overweight* as BMI = 25 to 29.9 kg/m², and *obese* as BMI ≥ 30 kg/m² by the National Institutes of Health.

¹⁶ BRFSS data for Wyoming are unavailable for 1993, and Rhode Island data are not available from the CDC for 1994 because the state decided to collect them during a six-month, rather than the 12-month, period recommended by the CDC. Data for Washington, D.C. were not collected in 1995. Although we were able to obtain the 1994 Rhode Island data directly from the state, we exclude them from the analysis below for the same reason the CDC does not offer them publicly.

¹⁷ MSA identifiers were only included if at least 500 interviews were obtained, and within MSAs, only counties for which sufficient data were collected to permit weighting (at least 50 interviews) were included. In the 2002 Atlanta data, for instance, only three of 28 counties are included due to insufficient sample sizes according to Michele Sussman Walsh of the CDC. Others (Ewing et al., 2003) have employed the 1998-2000 data at the county and MSA level to investigate the relationship between sprawl and obesity.

Our second source of data is the U.S. Census Bureau's 1990 Summary Tape File 3 (STF3), which we use to compute various "neighborhood-level" (census tract) variables (e.g., income inequality, segregation) summarized by MSA as defined by the Office of Management and Budget's (OMB) December 2003 "core based statistical area" (CBSA) specifications, as well as other metropolitan-level factors such as population density, mean household income, median rent and household value, and residential crowding. The CBSA specifications define each metropolitan area neatly by county boundaries and thereby eliminate difficulties involved in trying to distinguish discrete metropolitan areas in the past (e.g., the Boston MSA was defined by parts of census tracts, counties, and cities before 2003) as well as permit analyses in which several data sources may be linked by metropolitan area for the entire nation over time.¹⁸

Several additional data sources provide, or permit one to produce, metropolitan-specific information about the pecuniary and non-pecuniary (e.g., acquisition and preparation) costs of calorie consumption.¹⁹ The U.S. Census Bureau's Census of Retail Trade (CRT) provides data on the number of fast-food and full-service restaurants by metropolitan area for various years between 1982 and 2000, with the distinction mainly a function of whether the establishment offers limited lines of refreshments and prepared food for proximate or take-home consumption and limited on-premises service (fast-food), or a full menu of prepared food in a setting that seats at least 15 people and has waitpersons (full-service). Because the distinction between these two types of restaurant is not always clear (e.g., full-service establishments sometimes offer high-caloric inexpensive food) others have grouped them into one variable to capture the effect of the number of restaurants in general on weight outcomes (Chou et al., 2004). In the analysis below we employ only the 1992 data because this is the year that immediately precedes our individual-level weight data and there is little reason to suspect that food prices rose disproportionately by metropolitan area during the past decade; and we keep these two restaurant types separate because we estimate that they differentially influenced eating behavior and height-adjusted weight.

Fast-food, full-service, and grocery food prices are obtained from two sources. First, the average cost of a full-service restaurant meal by metropolitan area is obtained from 1992 CRT price category data. Specifically, midpoint prices are assigned to meals that fall within a certain price range (e.g., \$3.00 for a meal that is recorded in the "\$2.00-\$4.99" category) and reasonable prices are assigned to the low (e.g., \$1.50 for the "below \$2.00") and high (e.g., \$45 for the "\$30 or above") open-ended categories. Second, averaged quarterly city-level Cost of Living data from the 1992 American Chamber of Commerce Researchers Association (ACCRA) are used to compute the average cost of a fast-food meal and of a bundle of grocery-store food by metropolitan area. The fast-food prices include those of a McDonald's Quarter-Pounder with cheese, a thin-crust cheese pizza at Pizza Hut or Pizza Inn, and fried chicken at Kentucky Fried Chicken or Church's; and the grocery store or "food-at-home" prices include those for 13 specific items from households whose heads have a mid-management occupation – including bacon, bananas, bread, eggs, chicken, ground beef, milk, lettuce, margarine, Parmesan cheese, potatoes, steak, and tuna (Chou et al., 2004).

¹⁸ There are two county definitions that have changed since 1990 that required some attention. First, Clifton Forge City became a part of Alleghany County in Virginia and is defined simply as census tract 701. Because this space is relatively small, has a small population, and is completely enveloped by Alleghany County, we ignore this change. Second, Broomfield County, Colorado was created from census tracts and parts of census tracts from four surrounding counties. Because census tracts changed between 1990 and 2000, we define Broomfield County crudely according to the census tract outline maps located at http://ftp2.census.gov/geo/maps/trt1990/st08_Colorado/. Specifically, we define the county as census tracts 85.13, 85.15, 85.16, and 85.17 in Adams County, tracts 131.02-131.05 in Boulder County, and tracts 98.16 and 98.20 in Jefferson County. We exclude census tract 20 from Weld County because an extremely small area and population of Broomfield City was in this tract.

¹⁹ We are grateful to Mike Grossman and Inas Rashad for providing us these data.

Apart from several census-generated neighborhood- and metropolitan-level variables that may alter incentives to engage in physical activity (e.g., economic segregation, population density, median household value), it is plausible that climate also influences the availability and type of outdoor infrastructures and facilities, and thus the probability of expending energy either through athletic exercise or daily activities such as traveling to work, to take children to school, or shop. City-level climatic data (temperature and rainfall) are obtained from the 1994 County and City Data Books of the U.S. Bureau of the Census and matching largest city to its corresponding CBSA is accomplished using the University of Missouri's MABLE/Geocorr2K system.²⁰

The 2003 OMB county-defined metropolitan areas permit one to connect census tract-level information about population density, housing and rental prices, the age of the housing stock, economic and ethno-racial segregation, income inequality and other area-level variables from the 1990 STF3 – as well as city- and metropolitan-level information about restaurants, grocery stores, food prices, and climate from CRT, ACCRA, and National Climatic Data Center (NCDC) data – with individual-level BRFSS behavioral, demographic and economic data.²¹ In short, we are able to analyze the influence of (1) individual-level behavioral, demographic and economic characteristics, (2) neighborhood- and metropolitan-level infrastructural and socioeconomic context, (3) food availability and prices, and (4) local weather conditions on overweight and obesity in the United States between 1993 and 2002 because our data may be linked at the 2003 OMB-defined metropolitan level.

Our reason for selecting metropolitan area rather than a smaller (e.g., census tract, county) or larger (e.g., state, region) geography extends beyond the ability to integrate data permitting one to construct and analyze factors likely to influence weight, however. Indeed, the data could be connected at the state or county level also. But because overweight and obesity have been rising across the entire U.S. population rather than among select demographic groups, ideally we would like to analyze changes in weight within a geographic space that incorporates a large proportion of

²⁰ Climate data are available at <http://fisher.lib.virginia.edu/collections/stats/cdb/> and the geographic matching software is located at <http://mcdc2.missouri.edu/websas/geocorr2k.html>.

²¹ We employ the 2003 OMB county-defined CBSAs to generate 370 (from an initial 389) metropolitan areas through which we attempt to connect the 1990 STF3, 1993-2002 BRFSS, and 1992 ACCRA and CRT data. In a first step, individuals in BRFSS are assigned a metropolitan area identifier by linking individuals through BRFSS counties to OMB-defined CBSAs. Approximately 39 percent of all individuals in the 1993-2003 BRFSS (404,085 of 1,031,892) had responses for all relevant variables and were located in one of the 343 CBSAs (93 percent of the generated 370) – 481 persons or 0.1 percent resided in counties used to define a CBSA but that were not included in the BRFSS survey, and 114,789 or 78 percent resided in non-CBSA counties that were in the BRFSS data but not used in the OMB definitions. In a second step – during which we matched individuals in the BRFSS data to tract-level data from 1990 STF3 by metropolitan area through county boundaries – all individuals were matched and resided in 79 percent or 48,412 of the 61,258 census tracts in the 1990 STF3. A third step required matching 1990-defined MSAs with 2003-defined CBSAs in order to link BRFSS and CRT data – resulting in 294,545 (or 73 percent of 404,085) individuals residing in 244 (or 71 percent of the previous 343) CBSAs. In a fourth step we matched most populated ACCRA city (place) with its corresponding 2003-defined CBSA in the BRFSS, which resulted in 287,899 (or 71 percent of 404,085) individuals residing in 214 (62 percent) of 343 CBSAs. Alternatively, matching most populated ACCRA city (place) with its corresponding 2003-defined CBSA in the BRFSS data remaining after having matched them with the CRT data produced a file containing 222,973 (or 77 percent of 287,899) individuals, and 174 (or 81 percent) of 214 CBSAs. A final tripartite step was undertaken to incorporate climate data publicly available at <http://www.ncdc.noaa.gov/oa/ncdc.html>. Specifically, using the same algorithm employed above to connect ACCRA and BRFSS data, we merged National Climatic Data Center (NCDC) and our three BRFSS files resulting from the three previous matches and obtained final samples of 280,717 individuals and 226 CBSAs, 286,037 individuals and 209 CBSAs, and 221,649 individuals and 172 CBSAs. Overall, our final matched sample represents 21 percent of all individual adults in the unmatched 1993-2002 BRFSS data and 46 percent of the 270 CBSAs we created from the original 389 OMB 2003 definitions.

the entire population and wherein people are integrated economically and socially. Metropolitan area, formally defined as one “containing a recognized population nucleus and adjacent communities that have a high degree of integration with that nucleus” (U.S. Office of Management and Budget, 2000), is arguably best suited for this purpose given that populations interact socio-economically more at this geographic level compared to county or state levels.²² Earlier studies, for instance, claim that an ecological area of an entire state is “unusually large” for purposes of investigating how residential context may influence weight outcomes (Kahn et al., 1998), and that interpersonal contact potentially influencing eating and exercise patterns are “certainly more likely across smaller areas” (Chang & Christakis, 2004). And the number of recent investigations having narrowed the geographic scope to the metropolitan level and proceeding with either cross-sectional or pooled data (Chang & Christakis, 2004; Ewing et al., 2003) or longitudinal data (Lakdawalla & Philipson, 2002) is on the rise.

Empirical Model and Implementation

We build directly on a behavioral model of the determinants of excessive weight (Chou, Grossman, & Saffer, 2002; Chou et al., 2004) using standard economic tools and assuming that overweight or obesity is a byproduct of other more fundamental goals in the context of a household production function model of consumer behavior (Becker, 1965). Our main contribution is that we include several social, economic, and climatic factors at the metropolitan level which may have both subconscious physiological and sentient behavioral influences on weight (e.g., income inequality, ethno-racial segregation, residential crowding) in addition to the food availability and price (Cawley, 2002; Chou et al., 2004; Cutler et al., 2003; Lakdawalla & Philipson, 2002) and labor market condition (Ruhm, 2000, 2003) variables that have been employed in recent longitudinal multi-level research by economists. Others (Catlin et al., 2003; Chang & Christakis, 2004; Diez-Roux et al., 2000; Ewing et al., 2003; Kahn et al., 1998; Ramsey & Glenn, 2002; Robert & Reither, 2004) have estimated the body weight effects of some of the area-level factors we include in our model but only employing one or two years of cross-sectional data, and none simultaneously considers climate and food market conditions. Both are likely to influence behaviors affecting weight as well as subconscious physiological processes. In short, this is the first study to estimate how market and non-market area-level factors influence height-adjusted weight physiologically and behaviorally over time – thus simultaneously considering the three main categories of weight determinants.

At the organism or physiological level, post-puberty primate body weight is a cumulative function of caloric intake minus caloric expenditure that varies positively with age at a decreasing rate until about age 55, at which time weight begins to decline (Tanner, 1978 [1990]). In addition to age, other exogenous physiological characteristics such as gender and ethno-racial group uniquely influence weight through food intake and metabolic functioning. Thus, because recent evidence suggests that the entire distribution of body weight has shifted across all demographic groups in the United States (Flegal & Troiano, 2000) and dichotomous variables constructed from continuous variables may conceal policy-relevant information about this measure of well-being (Joliffe, 2004), in a first stage of our analysis we regress one continuous (BMI) and two dichotomous (OVERWEIGHT, OBESE) variables on AGE, FEMALE, BLACK, LATINO, and OTHER non-white to estimate (or at least control for) the influence of physiology on height-adjusted weight. Table 1 defines and provides means and standard deviations for all variables used in our analysis.

²² Populations within most states cluster in multiple metropolitan areas, for instance, and counties are demographically homogenous compared to metropolitan areas. More important for our purposes, however, metropolitan areas represent an area defined by travel patterns and economic activity rather than political boundaries.

While this vector of individual genetic traits may predispose someone toward excess weight, the behavioral model employed here offers a wider explanatory framework because it recognizes that consumers combine purchased goods and services with leisure time to produce more fundamental commodities such as health, tasty food, and entertainment that enter their ulterior utility function – and that this mix is influenced by extra-individual, area-specific socioeconomic factors.

Energy consumption and expenditure are also influenced by other individual characteristics and behaviors that are relatively tropismatic. In other words, some individual-level behaviors and characteristics are influenced by human reflection that is conditioned on environmental context rather than primarily being physiologically reflexive and subconscious. Furthermore, some may be influenced by weight in addition to partly determining it. Educational attainment and labor market outcomes are two examples. Consequently – controlling for age, sex, and ethno-racial group – calories consumed in a given time period will be a function of hours worked outside the home, household income, food prices, educational attainment, marital status, diet, exercise, smoking, and one's preference for time. Self-control, of course, underlies some of these variables, but even this may be a function of one's socioeconomic environment. On the right-hand side of the energy balance equation, energy expenditure is a function of calories lost while at work, while doing chores at home, and while engaging in leisure activities. The impact of one's occupation on caloric loss necessarily depends on the physical strenuousness of the work undertaken (Lakdawalla & Philipson, 2002) as well as the number of hours worked. Working more hours will theoretically raise the value of one's time and result in substituting market goods and services for household chores and leisure activities, and a number of other factors (e.g., household income, educational attainment, marital status) will likewise influence energy expenditure (Chou et al., 2002). Finally, as was the case regarding caloric intake, proximate factors influencing caloric loss (work, chores, leisure) may be conditioned by one's social environment in addition to individual characteristics and local food prices.

4. Results

To be added (but see table 2 for preliminary results).

5. Conclusion

To be added.

References

- Allison, D., Fontaine, K., Manson, K., Stevens, J., & VanItallie, T. (1999). Annual Deaths Attributable to Obesity in the United States. *Journal of the American Medical Association*, 282(16), 1530-1538.
- Auster, R., Levenson, I., & Saracheck, D. (1969). The Production of Health, and Exploratory Study. *The Journal of Human Resources*, 4(4), 411-436.
- Austin, M. A., Friedlander, Y., Newman, B., Edwards, K., Mayer-Davis, E. J., & King, M.-C. (1997). Genetic Influences on Changes in Body Mass Index: A Longitudinal Analysis of Women Twins. *Obesity Research*, 5(4), 326-331.
- Averett, S., & Korenman, S. (1996). The Economic Reality of the Beauty Myth. *The Journal of Human Resources*, 31(2), 304-330.
- Baum II, C. L., & Ford, W. F. (2004). The Wage Effects of Obesity: A Longitudinal Study. *Health Economics, Forthcoming*.
- Becker, G. S. (1965). A Theory of the Allocation of Time. *Economic Journal*, 75(299), 493-517.
- Bhatnagar, D., Anand, I. S., Durrington, P. N., Patel, D. J., Wander, G. S., Mackness, M. I., Creed, F., Tomenson, B., Chandrashekar, Y., Winterbotham, M., Britt, R. P., Keil, J., E., & Sutton, G. C. (1995). Coronary Risk Factors in People from the Indian Subcontinent Living in West London and Their Siblings in India. *Lancet*, 345(February 18th), 405-409.
- Bjorntorp, P. (1991). Visceral Fat Accumulation: The Missing Link Between Psychosocial Factors and Cardiovascular Disease? *Journal of Internal Medicine*, 230, 195-201.
- Bjorntorp, P. (1996). The Regulation of Adipose Tissue Distribution in Humans. *International Journal of Obesity and Related Metabolic Disorders*, 20(291-302).
- Bouchard, C. (1995). Genetic Influences on Body Weight and Shape. In C. G. Fairburn (Ed.), *Eating Disorders and Obesity: A Comprehensive Handbook* (pp. 21-26). New York, NY: Guilford Press.
- Bray, G. A. (1990). Obesity: Historical Development of Scientific and Cultural Ideas. *International Journal of Obesity*, 14, 909-926.
- Brownell, K. D., & Horgen, K. B. (2004). *Food Fight: The Inside Story of the Food Industry, America's Obesity Crisis, and What We Can Do About It*. New York, NY: McGraw-Hill.
- Brunner, E. (1997). Stress and the Biology of Inequality. *British Medical Journal*, 314, 1472-1476.
- Brunner, E., & Marmot, M. (1999). Social Organization, Stress, and Health. In R. G. Wilkinson (Ed.), *Social Determinants of Health* (pp. 17-43). New York, NY: Oxford University Press.
- Bujalska, I. J., Kumar, S., & Stewart, P. M. (1997). Does Central Obesity Reflect "Cushing's Disease of the Omentum?" *Lancet*, 349, 1210-1213.
- Catlin, T. K., Simoes, E. J., & Brownson, R. C. (2003). Environmental and Policy Factors Associated with Overweight among Adults in Missouri. *American Journal of Health Promotion*, 17(4), 249-258.
- Cawley, J. (2002). *Addiction and the Consumption of Calories: Implications for Obesity*. Unpublished manuscript, Cambridge, MA.
- Cawley, J. (2004). The Impact of Obesity on Wages. *The Journal of Human Resources*, 34(2), 451-474.
- Cawley, J., & Danziger, S. (2004). *Obesity as a Barrier to the Transition from Welfare to Work*. Cambridge, MA: NBER Working Paper #10508.
- Chang, V. W., & Christakis, N. A. (2003). Self-Perception of Weight Appropriateness in the United States. *American Journal of Preventative Medicine*, 24(4), 332-339.
- Chang, V. W., & Christakis, N. A. (2004). Inequality and Weight Status. *Social Science & Medicine*, ??(?), ??-??

- Chesney, M. A., Thurston, R. C., & Thomas, K. A. (2001). Creating Social and Public Health Environments to Sustain Behavior Change: Lessons from Obesity Research. In J. H. Gentry (Ed.), *Integrating Behavioral and Social Sciences with Public Health* (pp. 31-50). Washington, D.C.: American Psychological Association.
- Chou, S.-Y., Grossman, M., & Saffer, H. (2002). *An Economic Analysis of Adult Obesity: Results from the Behavioral Risk Factor Surveillance System*. Cambridge, MA: NBER Working Paper #9247.
- Chou, S.-Y., Grossman, M., & Saffer, H. (2004). An Economic Analysis of Adult Obesity: Results from the Behavioral Risk Factor Surveillance System. *Journal of Health Economics*, 23, 565-587.
- Chua, J., Streamson C., & Leibel, R. L. (2002). Body Weight Regulation: Neural, Endocrine, and Autocrine MEchanisms. In A. J. Stunkard (Ed.), *Handbook of Obesity Treatment* (pp. 19-41). New York, NY: The Guilford Press.
- Colditz, G. (1999). Economic Costs of Obesity and Inactivity. *Medicine and Science in Sports and Exercise*, 31(11), S663-S667.
- Colditz, G. A. (1992). Economic Costs of Obesity. *American Journal of Clinical Nutrition*, 55, 503S-507S.
- Comuzzie, A. G., & Allison, D. B. (1998). The Search for Human Obesity Genes. *Science*, 280, 1374-1377.
- Costa, D. L., & Steckel, R. H. (1995). *Long-Run Trends in Health, Welfare, and Economic Growth in the United States*. Cambridge, MA: NBER Working Paper #76.
- Crandell, C. S. (1994). Prejudice Against Fat People: Ideology and Self-Interest. *Journal of Personality and Social Psychology*, 66(5), 882-894.
- Critser, G. (2003). *Fat Land: How Americans Became the Fattest People in the World*. Boston, MA: Houghton Mifflin Company.
- Cruickshank, J. K., Mbanja, J. C., Wilks, R., Balkau, B., McFarlane-Anderson, N., & Forrester, T. (2001). Sick genes, sick individuals or sick populations with chronic disease? The emergence of diabetes and high blood pressure in African-origin populations. *International Journal of Epidemiology*, 30, 111-117.
- Cutler, D. M., Glaeser, E. L., & Shapiro, J. M. (2003). Why Have Americans Become More Obese? *Journal of Economic Perspectives*, 17(3), 92-118.
- Diez-Roux, A. V. (2002). Invited Commentary: Places, People, and Health. *American Journal of Epidemiology*, 155(6), 516-519.
- Diez-Roux, A. V., Link, B. G., & Northridge, M. E. (2000). A Multilevel Analysis of Income Inequality and Cardiovascular Disease Risk Factors. *Social Science & Medicine*, 50(5), 673-687.
- Ewing, R., Schmid, T., Killingsworth, R., Zlot, A., & Raudenbush, S. W. (2003). Relationship Between Urban Sprawl and Physical Activity, Obesity, and Morbidity. *American Journal of Health Promotion*, 18(1), 47-57.
- Fabsitz, R. R., Sholinsky, P., & Carmelli, D. (1994). Genetic Influences on Adult Weight Gain and Maximum Body Mass Index in Male Twins. *American Journal of Epidemiology*, 140(8), 711-720.
- Ferraro, K. F., & Kelley-Moore. (2003). Cumulative Disadvantage and Health: Long-Term Consequences of Obesity? *American Sociological Review*, 68(October), 707-729.
- Finkelstein, E. A., Fiebelkorn, I. C., & Wang, G. (2004). State-Level Estimates of the Annual Medical Expenditures Attributable to Obesity. *Obesity Research*, 12(1), 18-24.
- Flegal, K. M., Carroll, M. D., Kucmarski, R. J., & Johnson, C. L. (1998). Overweight and Obesity Trends in the United States: Prevalence and Trends, 1960-1994. *International Journal of Obesity Related Metabo . . .* 22, 39-47.
- Flegal, K. M., & Troiano, R. P. (2000). Changes in the Distribution of Body Mass Index of Adults and Children in the US Population. *International Journal of Obesity*, 24, 807-818.

- Flegal, K. M., Troiano, R. P., Pamuk, E. R., Kucumarski, R. J., & Campbell, S. M. (1995). The Influence of Smoking Cessation on the Prevalence of Overweight in the United States. *New England Journal of Medicine*, 333(18), 1165-1170.
- Fogel, R. W. (1994). Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the Making of Economic Policy. *American Economic Review*, 84(3), 369-395.
- Fogel, R. W., & Costa, D. L. (1997). A Theory of Technophysio Evolution, with Some Implications for Forecasting Populations, Health Care Costs, and Pension Costs. *Demography*, 34, 49-66.
- Fontaine, K. R., & Bartlett, S. J. (1998). Estimating Health-Related Quality of Life in Obese Individuals. *Disease Management Health Outcomes*, 3(2), 61-70.
- Fontaine, K. R., Redden, D. T., Wang, C., Westfall, A. O., & Allison, D. B. (2003). Years of Life Lost Due to Obesity. *Journal of the American Medical Association*, 289(2), 187-193.
- Foreyt, J., & Goodrick, K. (1995). The Ultimate Triumph of Obesity. *Lancet*, 346, 134-135.
- Friedrich, M. J. (2002). Epidemic of Obesity Expands Its Spread to Developing Countries. *Journal of the American Medical Association*, 287(11), 1382-1385.
- Friedrich, M. J. (2003). Researchers Explore Obesity-Cancer Link. *Journal of the American Medical Association*, 290(21), 2790-2791.
- Greeno, C. G., & Wing, R. R. (1994). Stress-Induced Eating. *Psychological Bulletin*, 115(3), 444-464.
- Hammermesh, D. S., & Biddle, J. E. (1994). Beauty and the Labor Market. *American Economic Review*, 84(5), 1174-1194.
- Hautanen, A., Raikkonen, K., & Adlercreutz, H. (1997). Associations between Pituitary-Adrenocortical Function and Abdominal Obesity, Hyperinsulinemia and Dyslipidaemia in Normotensive Males. *Journal of Internal Medicine*, 241, 451-461.
- Hill, J. O., & Melanson, E. L. (1999). Overview of the Determinants of Overweight and Obesity: Current Evidence and Research Issues. *Medicine and Science in Sports and Exercise*, 31(11), S515-S521.
- Hill, J. O., & Peters, J. C. (1998). Environmental Contributions to the Obesity Epidemic. *Science*, 280, 1371-1374.
- Holmes, M. D., Stampfer, M. J., Wold, A. M., Jones, C. P., Spiegelman, D., Manson, J. E., & Colditz, G. A. (1998). Can Behavioral Risk Factors Explain the Difference in Body Mass Index Between African-American and European-American Women? *Ethnicity and Disease*, 8(Autumn), 331-339.
- Horgen, K. B., & Brownell, K. D. (2002). Confronting the Toxic Environment: Environmental and Public Health Actions in a World Crisis. In A. J. Stunkard (Ed.), *Handbook of Obesity Treatment* (pp. 95-106). New York, NY: The Guilford Press.
- Jasso, G. (2003). Migration, Human Development, and the Life Course. In M. J. Shanahan (Ed.), *Handbook of the Life Course* (pp. 331-364). New York, NY: Kluwer Academic/Plenum Publishers.
- Jayo, J. M., Shively, C. A., Kaplan, J. R., & Manuck, S. B. (1993). Effects of Exercise and Stress on Body Fat Distribution in Male Cynomolgus Monkeys. *International Journal of Obesity and Related Metabolic Disorders*, 17, 597-604.
- Jolliffe, D. (2004). Continuous and Robust Measures of the Overweight Epidemic: 1971-2000. *Demography*, 41(2), 303-314.
- Jolliffe, D. (2004). Extent of Overweight among US Children and Adolescents from 1971 to 2000. *International Journal of Obesity*, 28, 4-9.
- Kahn, H. S., Tatham, L. M., Pamuk, E. R., & Heath, C. W. J. (1998). Are Geographic Regions with High Income Inequality Associated with Risk of Abdominal Weight Gain? *Social Science & Medicine*, 47(1), 1-6.

- Kahneman, D. (2003). A Psychological Perspective of Economics. *American Economic Review*, 93(2), 162-168.
- Kan, K., & Tsai, W.-D. (2004). Obesity and Risk Knowledge. *Journal of Health Economics*, ??(??), ??-??
- Kasl, S. V., & Berkman, L. B. (1983). Health Consequences of the Experience of Migration. *Annual Review of Public Health*, 4, 69-90.
- Kawachi, I., & Berkman, L. B. (Eds.). (2003). *Neighborhoods and Health*. New York, NY: Oxford University Press.
- Kawachi, I., & Kennedy, B. P. (1997). Health and Social Cohesion: Why Care about Income Inequality? *British Medical Journal*, 314, 1037-1040.
- Kawachi, I., Troisi, R. J., Rotnitzky, A. G., Coakley, E. H., & Colditz, G. A. (1996). Can Physical Activity Minimize Weight Gain in Women After Smoking Cessation? *American Journal of Public Health*, 86(7), 999-1004.
- Keeler, E. B., Manning, W. G., Newhouse, J. P., Sloss, E. M., & Wasserman, J. M. A. (1989). The External Costs of a Sedentary Lifestyle. *American Journal of Public Health*, 79(8), 975-981.
- Korkeila, M., Kaprio, J., Rissanen, A., & Koskenvuo, M. (1995). Consistency and Change of Body Mass Index and Weight: A Study of 5967 Adult Finnish Twin Pairs. *International Journal of Obesity*, 19, 310-317.
- Lakdawalla, D., & Philipson, T. (2002). *The Growth of Obesity and Technological Change: A Theoretical and Empirical Investigation*. Cambridge, MA: NBER Working Paper 8965.
- Lee, G.-H., Proenca, R., Montez, J. M., Carroll, K. M., Darvishzadeh, J. G., Lee, J. I., & Friedman, J. M. (1996). Abnormal Splicing of the Leptin Receptor in *Diabetic Mice*. *Nature*, 379(February 15th), 632-635.
- Link, B. G., & Phelan, J. (1995). Social Conditions as Fundamental Casues of Disease. *Journal of Health and Social Behavior*, 35, 80-94.
- Loh, E. S. (1993). The Economic Effects of Physical Appearance. *Social Science Quarterly*, 74(2), 420-438.
- Macintyre, S., MacIver, S., & Sooman, A. (1993). Area, Class and Health: Should We be Focussing on Places or People? *Journal of Social Policy*, 22, 213-234.
- Marin, P., & Bjorntorp, P. (1993). Endocrine-Metabolic Pattern and Adipose Tissue Distribution. *Hormone Research*, 39(Supplement 3), 81-85.
- Marmot, M. G., Adelstein, A. M., & Bulusu, L. (1984). Lessons from the Study of Immigrant Mortality. *Lancet*(June 30th), 1455-1457.
- Marmot, M. G., & Syme, S. L. (1976). Acculturation and Coronary Heart Disease in Japanese-Americans. *American Journal of Epidemiology*, 104(3), 225-247.
- Martin, L. F., Robinson, A., & Moore, B. J. (2000). Socioeconomic Issues Affecting the Treatment of Obesity in the New Mellennium. *Pharmacoeconomics*, 18(4), 335-353.
- Massey, D. S. (2002). A Brief History of Human Society: The Origin and Role of Emotion in Social Life. *American Sociological Review*, 67(February), 1-29.
- Mayer, J. (1968). *Overweight: Causes, Cost, and Control*. Englewood Cliffs, NJ: Prentice-Hall, Inc.
- McCann, B. A., & Ewing, R. (2003). *Measuring the Helath Effects of Sprawl: A National Analysis of Physical Activity, Obesity and Chronic Disease*. Washington, D.C.: Smart Growth America.
- McEwen, B. S. (2001). From Molecules to Mind: Stress, Individual Differences, and the Social Environment. *Annals of the New York Academy of Sciences*, 935, 42-49.
- McGinnis, M., & Foege, W. (1993). Actual Causes of Deaths in the United States. *American Journal of Public Health*, 270(18), 2207-2212.
- McKeown, T. (1979). *The Role of Medicine: Dream, Mirage, or Nemesis?* Princeton, NJ: Princeton Univeristy Press.

- Mitka, M. (2003). Economist Takes Aim at "Big Fat" US Lifestyle. *Journal of the American Medical Association*, 289(1), 33-34.
- Mokdad, A. H., Bowman, B. A., Ford, E. S., Vinicor, F., Marks, J. S., & Koplan, J. P. (2001). The Continuing Epidemics of Obesity and Diabetes in the United States. *Journal of the American Medical Association*, 286(10), 1195-1200.
- Mokdad, A. H., Ford, E. S., Bowman, B. A., Dietz, W. H., Vinicor, F., Bales, V. H., & Marks, J. S. (2003). Prevalence of Obesity, Diabetes, and Obesity-Related Health Risk Factors, 2001. *Journal of the American Medical Association*, 289(1), 76-79.
- Mokdad, A. H., Marks, J. S., Stroup, D. F., & Gerberding, J. L. (2004). Actual Causes of Death in the United States. *Journal of the American Medical Association*, 291(10), 1238-1245.
- Mokdad, A. H., Serdula, M. K., Dietz, W. H., Bowman, B. A., Marks, J. S., & Koplan, J. P. (1999). The Spread of the Obesity Epidemic in the United States, 1991-1998. *Journal of the American Medical Association*, 282(16), 1519-1522.
- Must, A., Spadano, J., Coakley, E. H., Field, A. E., Colditz, G., & Dietz, W. H. (1999). The Disease Burden Associated with Overweight and Obesity. *Journal of the American Medical Association*, 282(16), 1523-1529.
- Nestle, M. (2002). *Food Politics: How the Food Industry Influences Nutrition and Health*. Berkeley, CA: University of California Press.
- O'Campo, P. (2003). Invited Commentary: Advancing Theory and Methods for Multilevel Models of Residential Neighborhoods and Health. *American Journal of Epidemiology*, 157(1), 9-13.
- O'Connor, A., & Grady, D. (2003). F.D.A. Moves to Let Drug Treat Obese Teenagers. *New York Times*, pp. A27.
- Organization for Economic Cooperation and Development. (2003). *Health at a Glance: OECD Indicators 2003*. Retrieved, from the World Wide Web: http://www.oecd.org/document/11/0,2340,en_2649_34487_16502667_1_1_1_37407,00.html
- Pasquali, R., Anconetani, B., Chattat, R., Biscotti, M., Spinucci, G., & Casimirri, F. (1996). Hypothalamus-Pituitary-Adrenal Axis Activity and Its Relationship to the Autonomic Nervous System in Women with Visceral and Subcutaneous Obesity: Effects of the Corticotropin-Releasing Factor/Arginine-Vasopressin Test and of Stress. *Metabolism*, 45, 351-356.
- Pedersen, S. B., Jonler, M., & Richelsen, B. (1994). Characterization of Regional and Gender Differences in Glucocorticoid Receptors and Lipoprotein Lipase Activity in Human Adipose Tissue. *Journal of Clinical Endocrinology and Metabolism*, 78, 1354-1359.
- Perusse, L., Rice, T., Chagnon, Y. C., Despres, J. P., Lemieux, S., Roy, S., Lacaille, M., Ho-Kim, M. A., Chagnon, M., Province, M. A., Rao, D. C., & Bouchard, C. (2001). A Genome-Wide Scan for Abdominal Fat Assessed by Computed Tomography in the Quebec Family Study. *Diabetes*, 50(3), 614-621.
- Philipson, T. (2001). The World-Wide Growth in Obesity: An Economic Research Agenda. *Health Economics*, 10, 1-7.
- Philipson, T. J., & Posner, R. A. (1999). *The Long-Run Growth in Obesity as a Function of Technological Change*. Cambridge, MA: NBER Working Paper #7423.
- Pingitore, R., Dugoni, B. L., Tindale, R. S., & Spring, B. (1994). Bias Against Overweight Job Applicants in a Simulated Employment Interview. *Journal of Applied Psychology*, 79(6), 909-917.
- Price, R. A. (2002). Genetics and Common Obesities: Background, Current Status, Strategies, and Future Prospects. In A. J. Stunkard (Ed.), *Handbook of Obesity Treatment* (pp. 73-94). New York, NY: The Guilford Press.

- Ramsey, P. W., & Glenn, L. L. (2002). Obesity and Health Status in Rural, Urban, and Suburban Southern Women. *Southern Medical Journal*, 95(7), 666-671.
- Rashad, I., & Grossman, M. (2004). The Economics of Obesity. *The Public Interest*, 156(Summer), 104-112.
- Ravussin, E., Valencia, M. E., Esparza, J., Bennett, P. H., & Schulz, L. O. (1994). Effects of a Traditional Lifestyle on Obesity in Pima Indians. *Diabetes Care*, 17(9), 1067-1074.
- Rebuffe-Scrive, M., Bronnegard, M., Nilsson, A., Gustafsson, J. A., & Bjorntorp, P. (1990). Steroid Hormone Receptors in Human Adipose Tissue. *Journal of Clinical Endocrinology and Metabolism*, 71, 1215-1219.
- Reed, D. R., Bachmanov, A. A., Beauchamp, G. K., Tordoff, M. G., & Price, R. A. (1997). Heritable Variation in Food Preferences and Their Contribution to Obesity. *Behavioral Genetics*, 27(4), 373-387.
- Resmond, R., Lapidus, L., Marin, P., & Bjorntorp, P. (1996). Mental Distress, Obesity and Body Fat Distribution in Middle-Aged Men. *Obesity Research*, 4, 245-252.
- Robert, S. A., & Reither, E. N. (2004). A Multilevel Analysis of Race, Community Disadvantage, and Body Mass Index among Adults in the US. *Social Science & Medicine*, 58(1), 1-12.
- Robson, A. J. (2001). The Biological Basis of Economic Behavior. *Journal of Economic Literature*, 39(March), 11-33.
- Rose, G. (1985). Sick Individuals and Sick Populations. *International Journal of Epidemiology*, 14, 32-38.
- Rose, G. (1992). *The Strategy of Preventative Medicine*. New York, NY: Oxford University Press.
- Rose, G., & Day, S. (1990). The Population Mean Predicts the Number of Deviant Individuals. *British Medical Journal*, 301, 1031-1034.
- Roux, L., & Donaldson, C. (2004). Economics and Obesity: Costing the Problem or Evaluating Solutions? *Obesity Research*, 12(2), 173-179.
- Ruhm, C. J. (2000). Are Recessions Good for Your Health? *Quarterly Journal of Economics*, 115(2), 617-650.
- Ruhm, C. J. (2003). *Healthy Living in Hard Times*. Unpublished manuscript, NBER Working Paper Series #9468, Cambridge, MA.
- Serdula, M. K., Mokdad, A. H., Williamson, D. F., Galuska, D. A., Mendlein, J. M., & Heath, G. W. (1999). Prevalence of Attempting Weight Loss and Strategies for Controlling Weight. *Journal of the American Medical Association*, 282(14), 1353-1358.
- Shell, E. R. (2002). *The Hungry Gene: The Inside Story of the Obesity Industry*. New York, NY: Grove Press.
- Shively, C. A., & Clarkson, T. B. (1988). Regional Obesity and Coronary Artery Atherosclerosis in Females: A Non-Human Primate Model. *Acta Medica Scandinavica*, 723(Supplement), 71-78.
- Slater, L. (2004). *Openign Skinner's Box: Great Psychological Experiments of the Twentieth Century*. New York, NY: W.W. Norton & Company.
- Sobal, J., & Stunkard, A. J. (1989). Socioeconomic Status and Obesity: A Review of the Literature. *Psychological Bulletin*, 105(2), 260-275.
- Stein, J. (2004). Adults Acknowledge Weight Issues. *Los Angeles Times*, pp. F4.
- Sturm, R., Ringel, J. S., & Andreyeva, T. (2004). Increasing Obesity Rates and Disability Trends. *Health Affairs*, 23(2), 199-205.
- Syme, S. L. (2004). Social Determinants of Health: The Community as an Empowered Partner. *Preventing Chronic Disease: Public Health Research, Practice, and Policy*, 1(1), 1-5.
- Tanner, J. M. (1978 [1990]). *Foetus into Man: Physical Growth from Conception to Maturity*. Cambridge, MA: Harvard University Press.
- Tartaglia, L. A., Dembski, M., Weng, X., Deng, N., Culpepper, J., Devos, R., Richards, G. J., Campfield, L. A., Clark, F. T., Deeds, J., Muir, C., Sanker, S., Moriarty, A., Moore, K. J.,

- Smutko, J. S., Mays, G. G., Woolf, E. A., Monroe, C. A., & Tepper, R. I. (1995). Identification and Expression Cloning of a Leptin Receptor, OB-R. *Cell*, 83(December 29th), 1263-1271.
- Tataranni, P. A., & Ravussin, E. (2002). Energy Metabolism and Obesity. In A. J. Stunkard (Ed.), *Handbook of Obesity Treatment* (pp. 42-72). New York, NY: The Guilford Press.
- Thompson, D., & Wolf, A. M. (2001). The Medical-Care Cost Burden of Obesity. *Obesity Reviews*, 2, 189-197.
- Trayhurn, P., Hoggard, N., Mercer, J. G., & Rayner, D. V. (1999). Leptin: Fundamental Aspects. *International Journal of Obesity*, 23(Supplement 1), 22-28.
- Troiano, R. P., & Flegal, K. M. (1998). Overweight Children and Adolescents: Description, Epidemiology, and Demographics. *Pediatrics*, 101(3), 497-504.
- U.S. Office of Management and Budget. (2000). *Standards for Defining Metropolitan and Micropolitan Statistical Areas; Notice*. Washington, D.C.
- U.S. Preventative Services Task Force. (2003). *Screening for Obesity in Adults: Recommendations and Rationale*. Retrieved February 20, 2004, from the World Wide Web: www.ahcpr.gov/clinic/uspstfix.htm
- Veblen, T. (1914 [1941]). *The Instinct of Workmanship*. New York, NY: W.W. Norton & Company, Inc.
- Visscher, T. L. S., & Seidell, J. C. (2001). The Public Health Impact of Obesity. *Annual Review of Public Health*, 22, 355-375.
- Wandel, M. (1993). Nutrition-Related Diseases and Dietary Change among Third World Immigrants in Northern Europe. *Nutrition and Health*, 9(2), 117-133.
- Weinsier, R. L., Hunter, G. R., Heini, A. F., Goran, M. I., & Sell, S. M. (1998). The Etiology of Obesity: Relative Contribution of Metabolic Factors, Diet, and Physical Activity. *American Medical Journal*, 105, 145-150.
- Willet, W. C. (2001). *Eat, Drink, and Be Healthy*. New York, NY: Simon & Schuster.
- Wolf, A. M., & Colditz, G. A. (1996). Social and Economic Effects of Body Weight in the United States. *American Journal of Clinical Nutrition*, 63(3), 466S-469S.
- Wolf, A. M., & Colditz, G. A. (1998). Current Estimates of the Economic Cost of Obesity in the United States. *Obesity Research*, 6(2), 97-106.
- Yanovski, J. A., & Yanovski, S. Z. (1999). Recent Advances in Basic Obesity Research. *Journal of the American Medical Association*, 282(16), 1504-1506.
- Ziegler, R. G., Hoover, R. N., Nomura, A. M. Y., West, D. W., Wu, A. H., Pike, M. C., Lake, A. J., Horn-Ross, P. L., Kolonel, L. N., Siiteri, P. K., & Fraumeni Jr., J. (1996). Relative Weight, Weight Change, Height, and Breast Cancer Risk among Asian-American Women. *Journal of the National Cancer Institute*, 88(10), 650-660.
- Zigler, R. G., Hoover, R. N., Nomura, A. M. Y., West, D. W., Wu, A. H., Pike, M. C., Lake, A. J., Horn-Ross, P. L., Kolonel, L. N., Siiteri, P. K., & Fraumeni Jr., J. (1996). Relative Weight, Weight Change, Height, and Breast Cancer Risk among Asian-American Women. *Journal of the National Cancer Institute*, 88(10), 650-660.

Table 1: Variable definitions, means, and standard deviations^a

<i>Variable</i>	<i>Definition</i>	Mean	S.D.
<i>Dependent^b</i>			
Body Mass Index (BMI)	Weight in kilograms divided by height in meters squared	26.052	(5.093)
Overweight	Dichotomous variable = 1 if BMI >= 25	0.535	(0.499)
Obese	Dichotomous variable = 1 if BMI >= 30	0.173	(0.379)
<i>Physiological^b</i>			
Age	Years since respondent's birth	44.050	(16.815)
Age squared	Square of Age	2,223.155	(1654.161)
Female	Dichotomous variable = 1 if sex is female	0.483	(0.500)
Black	Dichotomous variable = 1 if respondent's ethno-racial group is non-Latino black	0.108	(0.311)
Latino	Dichotomous variable = 1 if respondent's ethno-racial group is Latino regardless of race	0.113	(0.317)
Other	Dichotomous variable = 1 if respondent's ethno-racial group is non-Latino Asian or other race	0.050	(0.219)
<i>Tropismatic^b</i>			
High school graduate	Dichotomous variable = 1 if respondent completed 12-15 years of formal schooling	0.572	(0.495)
College graduate	Dichotomous variable = 1 if respondent graduated from college	0.322	(0.467)
Married	Dichotomous variable = 1 if respondent is married or cohabiting	0.606	(0.489)
Employed	Dichotomous variable = 1 if respondent is employed	0.676	(0.468)
Household income	Respondent's real annual household income in thousands of 1993 Dollars	40.285	(25.427)
Exercise	Dichotomous variable = 1 if participated in physical activity outside of work during past month	0.245	(0.430)
Three or More Servings of Fruits and Vegetables Daily	Dichotomous variable = 1 if respondent eats 3 or more servings of fruit and vegetables daily	0.751	(0.432)
Smoke	Dichotomous variable = 1 if respondent currently smoke cigarettes	0.227	(0.419)
<i>Environmental^c</i>			
Residential Segregation	Proportion of non-Latino black residents in a metropolitan area that would need to move across census tracts to achieve an even distribution	0.618	(0.107)
Income Inequality	Metropolitan area Gini coefficient	0.394	(0.023)
Poverty Concentration	Proportion of poor residing in extremely poor (defined as at least 40 percent of the population) neighborhoods by metropolitan area	0.126	(0.095)
Population Density	Number of people per square mile residing in a metropolitan area	353.948	(243.332)
Metropolitan Income	1990 median household income in thousands of dollars by metropolitan area	30.471	(5.572)
Number of fast-food restaurants	Number of fast-food restaurants per 10,000 residents in respondent's metropolitan area in 1992	814.615	(622.966)
Number of full-service restaurants	Number of full-service restaurants per 10,000 residents in respondent's metropolitan area in 1992	767.246	(590.789)
Price of a fast-food meal	Mean real price of a 1992 fast-food restaurant meal in respondent's metropolitan area (1993 dollars)	4.375	(0.287)
Price of a full-service meal	Mean real price of a 1992 full-service restaurant meal in respondent's metropolitan area (1993 dollars)	8.187	(2.136)
Food at home price	Mean real price of 13 food items in 1992 in respondent's metropolitan area (1993 dollars, see text for details)	1.737	(0.140)
Rain	Inches of rain in metropolitan area's largest city in 2000	35.516	(14.592)
Temperature	Thousands of 2000 heating degree days in metropolitan area's largest city	3.972	(2.197)

^aStatistics produced with data available for each category of variables. See text for explanation of observations lost due to merging of various data sources.

^bSample size is 404,085 adults within 343 metropolitan areas.

^cSample size is 221,649 adults within 172 metropolitan areas.

Table 2: Determinants of Body Mass Index, Overweight, and Obesity among Adults in Metropolitan Areas of the United States: 1993-2002^a

Independent Variable	Body Mass Index		Overweight		Obese	
	(1)	(2)	(3)	(4)	(5)	(6)
Physiological						
Age	0.335 (0.005) ^{\$}	0.343 (0.007) ^{\$}	0.118 (0.002) ^{\$}	0.119 (0.002) ^{\$}	0.133 (0.002) ^{\$}	0.133 (0.003) ^{\$}
Age squared	-0.003 (0.000) ^{\$}	-0.003 (0.000) ^{\$}	-0.001 (0.000) ^{\$}	-0.001 (0.000) ^{\$}	-0.001 (0.000) ^{\$}	-0.001 (0.000) ^{\$}
Female	-1.354 (0.039) ^{\$}	-1.361 (0.043) ^{\$}	-0.868 (0.013) ^{\$}	-0.856 (0.019) ^{\$}	-0.125 (0.013) ^{\$}	-0.112 (0.016) ^{\$}
Black	1.510 (0.056) ^{\$}	1.482 (0.057) ^{\$}	0.656 (0.014) ^{\$}	0.687 (0.026) ^{\$}	0.548 (0.016) ^{\$}	0.544 (0.027) ^{\$}
Latino	0.486 (0.080) ^{\$}	0.683 (0.117) ^{\$}	0.217 (0.022) ^{\$}	0.280 (0.027) ^{\$}	0.042 (0.027) ^{\$}	0.140 (0.034) ^{\$}
Other	-1.232 (0.081) ^{\$}	-0.905 (0.129) ^{\$}	-0.382 (0.023) ^{\$}	-0.289 (0.039) ^{\$}	-0.357 (0.030) ^{\$}	-0.213 (0.052) ^{\$}
Tropismatic						
High school graduate	-0.511 (0.063) ^{\$}	-0.507 (0.059) ^{\$}	-0.168 (0.012) ^{\$}	-0.157 (0.016) ^{\$}	-0.233 (0.015) ^{\$}	-0.228 (0.018) ^{\$}
College graduate	-1.376 (0.059) ^{\$}	-1.289 (0.062) ^{\$}	-0.487 (0.018) ^{\$}	-0.424 (0.025) ^{\$}	-0.582 (0.019) ^{\$}	-0.541 (0.025) ^{\$}
Married	0.075 (0.030) ^{\$}	0.072 (0.041) ^{\$}	0.131 (0.009) ^{\$}	0.139 (0.011) ^{\$}	0.024 (0.010) [~]	0.033 (0.015) [~]
Employed	-0.066 (0.047) ^{\$}	-0.033 (0.049) ^{\$}	0.058 (0.010) ^{\$}	0.063 (0.013) ^{\$}	-0.072 (0.013) ^{\$}	-0.060 (0.016) ^{\$}
Household income	-0.012 (0.001) ^{\$}	-0.012 (0.001) ^{\$}	-0.005 (0.000) ^{\$}	-0.005 (0.000) ^{\$}	-0.008 (0.000) ^{\$}	-0.008 (0.000) ^{\$}
Exercise	-0.757 (0.045) ^{\$}	-0.761 (0.055) ^{\$}	-0.234 (0.009) ^{\$}	-0.230 (0.012) ^{\$}	-0.457 (0.011) ^{\$}	-0.452 (0.015) ^{\$}
Three or More Servings of Fruits and Vegetables Daily	-0.242 (0.035) ^{\$}	-0.259 (0.038) ^{\$}	-0.131 (0.008) ^{\$}	-0.122 (0.011) ^{\$}	-0.125 (0.012) ^{\$}	-0.136 (0.018) ^{\$}
Smoke	-1.085 (0.049) ^{\$}	-1.211 (0.056) ^{\$}	-0.421 (0.011) ^{\$}	-0.435 (0.016) ^{\$}	-0.502 (0.015) ^{\$}	-0.520 (0.023) ^{\$}
Environmental						
Residential Segregation		0.403 (0.325)		-0.067 (0.119)		0.179 (0.127)
Income Inequality		-2.866 (2.306)		-2.019 (1.115) [#]		-2.094 (1.055) [~]
Poverty Concentration		0.937 (0.364) ^{\$}		0.329 (0.138) [~]		0.315 (0.138) [~]
Population Density		0.000 (0.000)		0.000 (0.000) [~]		0.000 (0.000)
Metropolitan Income		-0.015 (0.009) [~]		-0.010 (0.005) [~]		-0.010 (0.004) [~]
Number of Fast-food restuarants		0.488 (0.360)		0.266 (0.176) [#]		0.366 (0.163) [~]
Number of Full-service restuarants		-0.504 (0.355)		-0.284 (0.169) [#]		-0.372 (0.157) [~]
Price of a Fast-food meal		-0.245 (0.115) [#]		-0.085 (0.044) [~]		-0.086 (0.049) [#]
Price of a Full-service meal		-0.032 (0.011)		-0.013 (0.006)		-0.006 (0.007)
Food at home price		0.090 (0.183)		-0.050 (0.098) [~]		0.079 (0.077)
Rain		0.001 (0.003)		0.002 (0.001) [#]		0.001 (0.001)
Temperature		0.041 (0.016)		0.022 (0.008) [#]		0.017 (0.008) [~]
Summary Statistics						
R ² (or Psuedo-R ² for Logit Regressions)	0.1036	0.1049	0.0731	0.0732	0.0539	0.052
F-statistic	805.66	463.87				
Percent Concordant Pairs			0.6371	0.6378	0.8204	0.8212
Probability > Chi-Squared			0.0000	0.0000	0.0000	0.0000
Sample Size (Unweighted)	404,085	221,649	404,085	221,649	404,085	221,649

^aStatistics produced with data available for each category of variables as explained in Table 1. Estimated coefficients for intercepts and year dummy variables not shown. Robust standard errors (Huber 1967) are in parentheses and are generated allowing for 2003 OMB county-defined core based statistical area (CBSA) and year clustering. All results are produced employing BRFSS sample Note: Statistically significant at the 99% (\$), 95%(~), and 90% (#) level