NBER WORKING PAPER SERIES

UNDERSTANDING OVEREATING AND OBESITY

Christopher J. Ruhm

Working Paper 16149
http://www.nber.org/papers/w16149

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
July 2010

I thank Stephen Holland, Charles Courtemanche, Thomas McGuire, Steven Stern and seminar participants at Harvard University, Korea University, University of Iceland, University of Pennsylvania, and University of Virginia for helpful suggestions. The views expressed herein are those of the author and do not necessarily reflect the views of the National Bureau of Economic Research.

Understanding Overeating and Obesity
Christopher J. Ruhm
NBER Working Paper No. 16149
July 2010
JEL No. I12,I18

ABSTRACT

The combination of economic and biological factors is likely to result in overeating, in the current environment of cheap and readily available food. This propensity is shown using a "dual-decision" approach where choices reflect the interaction between two parts of the brain: a "deliberative" system, operating as in standard economic models, and an "affective" system that responds rapidly to stimuli without considering long-term consequences. This framework is characterized by excess food consumption and body weight, in the sense that individuals prefer both ex-ante and ex-post to eat and weigh less than they actually do, with dieting being common but often unsuccessful or only partially successful. As in the standard model, weight will be related to prices. However, another potentially important reason for rising obesity is that food producers have incentives to engineer products to stimulate the affective system so as to encourage overeating. Data from multiple waves of the National Health and Nutrition Examination Surveys are used to investigate predictions of the dual-decision model, with the evidence providing broad support for at least some irrationality in food consumption.

Christopher J. Ruhm
Department of Economics
Bryan School, UNCG
P.O. Box 26165
Greensboro, NC 27402-6165
and NBER
chrisruhm@uncg.edu

The growth of obesity represents a major public health issue. Almost one-third (31.5%) of American adults (aged 20 to 74) were obese in 1999-2004 versus just 14.5 percent in 1976-80; if current trends continue, over two-fifths (41.8 percent) will be obese in 2020 (Ruhm, 2007). The prevalence of excess weight is particularly high in the United States but is growing rapidly throughout much of the world (World Health Organization, 1997; International Obesity Task Force, 2005). Obesity is associated with elevated mortality (Fontaine et al., 2003; Flegal et al., 2005; Franks et al., 2010) and high rates of diabetes, hypertension, asthma and other diseases (Must et al., 1999; Mokdad et al., 2001; Okoro et al, 2004). Severe obesity raises medical expenditures, stresses the health care system, reduces productivity (Thompson et al., 2001; Finkelstein et al., 2003; Andreyeva et al., 2004) and its growth threatens to reverse historical gains in life expectancy (Olshansky et al., 2005).

Eating and body weight are economic decisions, in that individuals presumably tradeoff the utility from current food intake against the associated monetary expense and disutility of future weight gains. In traditional economic models, consumers balance these tradeoffs to achieve a constrained optimum. Models of rational decision-making have been developed for a variety of health behaviors, including the consumption of addictive substances (Becker & Murphy, 1988), but might seem particularly well-suited to considering food choices because all adults are experienced eaters and should have little uncertainty about the consequences of eating for body weight.[1] With rational consumption, the role for government policy is limited to cases of externalities, public goods, or informational constraints. Other interventions reduce utility and so are undesirable.

It is questionable, however, to what extent the standard economic model can explain the rapid and continuing increase in obesity. Such explanations typically emphasize reductions in food prices and higher costs of expending calories as sources of the growth in body weight. As detailed below, food prices declined substantially from the early 1970s through the mid 1980s, when obesity began its rise, but changed little thereafter, while body weight continued to grow. Employment-related calorie *expenditures*

[1] By contrast, experience with some other addictive substance (like illegal drugs) will be limited or nonexistent, introducing uncertainty concerning the effects of consumption (e.g. how quickly addiction will occur).

almost certainly have fallen, as the economy has shifted towards more sedentary jobs (Philipson & Posner, 2003) but this long-run trend was largely complete by the middle 1970s, before obesity started to increase.[2] Perhaps most convincing is that many individuals act as if their eating patterns represent mistakes rather than planned behavior. Rational consumers should have little desire to lose weight and, when they do, be able to accomplish this relatively quickly and without large financial expenditures. Instead, the U.S. weight loss industry exceeds $50 billion dollars per year (MarketData Enterprises, 2009).[3] In the extreme, over 200,000 Americans obtain bariatric surgery annually (National Institute of Diabetes and Digestive and Kidney Diseases, 2008). This might be the ultimate pre-commitment device, since the size of the stomach is reduced making it almost impossible to consume large amounts of food.[4]

Limitations of the standard utility-maximizing model should not be surprising because eating decisions are not *only* economic, they also reflect biology. Humans have been genetically programmed over millions of years to eat, with the major concern until recently being to obtain sufficient calories for survival.[5] The combination of economic and biological factors is likely to result in overeating in the current environment of cheap and readily available food. This propensity is shown below using a "dual decision" model that builds on the insights of neuroscience and behavioral economics. Specifically, food consumption reflects the interaction between two parts of the brain: a "deliberative" system that makes decisions as traditionally modeled in economics and an "affective" system that responds rapidly to stimuli but does not account for long-term consequences.

The dual decision framework is characterized by overeating and excess weight, in that individuals would prefer ex-ante and ex-post to eat and weigh less than they actually do. Consumers are assumed to be sophisticated in understanding that they overeat and may take actions constraining their ability to do

[2] For instance, the share of the population in highly active occupations fell just three percentage points, from 45 to 42 percent, between 1980 and 1990 (Cutler et al., 2003).
[3] This excludes related medical expenditures and so dramatically understates total spending on weight loss.
[4] Bariatric surgery has many side-effects, some potentially life-threatening, and so would presumably be avoided by most rational consumers making optimal decisions.
[5] For example, Pi-Sunyer (2003, p. 859) writes: "The struggle for survival of the human species has been driven by a lack, not an excess, of food … The human body has developed over the years to defend actively against this threat. As soon as weight is lost, there is a powerful biological urge to regain it … In contrast, when there is an accretion of excess weight, the biological signals for reversing this are very muted … What is the wisdom of the body in times of deprivation becomes a foolishness in our modern environment."

so. As in the traditional model, body weight is influenced by food prices. But another potentially important reason for rising obesity is that producers have incentives to engineer foods to stimulate the affective system to increase eating. To the extent this engineering has advanced over time, individuals will feel that they have less control over their eating and that the resulting weight gains are mistakes.[6]

Predictions of the dual decision model are examined using data from multiple waves of the National Health and Nutrition Examination Surveys (NHANES) and selected years of the Behavior Risk Factor Surveillance System (BRFSS). The evidence broadly supports there being some irrationality in food consumption, leading to weight in excess of utility-maximizing levels. For instance, weight loss desires and attempts are positively related to body mass index and dieting has become more common as obesity has increased. Severely obese individuals are particularly likely to experience big weight gains and there is suggestive evidence that such persons consume relatively large amounts of foods engineered to stimulate affective system responses.

1. Background

In traditional economic models, agents maximize a utility function subject to a stable set of preferences. The resulting decisions are optimal, in that individuals do the best that they can given constraints on income, time and available information. The choices may come to be viewed as ex-post mistakes (e.g. when the outcomes of stochastic processes are revealed) but are correct ex-ante.

By contrast, much recent work in behavioral economics emphasizes the importance of systematic errors. The model detailed below focuses on eating and body weight and draws heavily on a more general framework developed by Lowenstein & O'Donoghue (2004). Their key insight is that many decisions are related to features of the human brain, with behavior resulting from the interaction of a utility-maximizing *deliberative* system and an *affective* system that is dominated by semi-automatic (but

---

[6] In a related vein, Laibson (1997) emphasizes the possibility that financial market innovation lowers utility, by reducing the ability of individuals to pre-commit to high savings levels.

potentially learned) responses that speed decision-making but lack the aspects of rationality generally focused upon by economists.[7]

This characterization reflects the following elements of brain anatomy.[8]

- The human brain evolved over millions of years with many new abilities being *added to* rather than *replacing* existing capabilities. It is often useful to characterize the brain as being divided into three structures. The oldest is the "reptilian" brain (the brain stem and cerebellum) which controls autonomic functions such as heartbeat and breathing.[9] Surrounding this is the limbic system (the amygdale, thalamus, hypothalamus and hippocampus), referred to here as the affective system, which coordinates sensory inputs to generate subjective feelings and emotional states like anger, pleasure and aggression. The outermost layer of the brain, which developed last, is the neocortex, consisting of the occipital, parietal, temporal and frontal lobes (that deal with sensory processing), and the prefrontal cortex which is the locus of abstract thinking, conceptualization and planning.
- The affective system responds to cues and specific stimuli and does not consider long-term effects of current actions. The deliberative system involves higher cognitive processes that account for both short-term and long-term consequences. The two systems, while connected, operate in parallel to yield differences in perception and memory. Unconscious emotional feelings thus exist independently of rational appraisals of them and many decisions involve an interaction of higher-order rational calculations with affective processes based on emotions, chemical responses, and feelings.
- Not only did evolutionary development of the affective system precede that of the deliberative system (rapid development of the prefrontal cortex took place only in the last 150,000 years) but the former

---

[7] Thaler & Sunstein (2009) refer to these as the "reflective" and "automatic" systems.
[8] These are elaborated on by Massey (2002), building on what MacLean (1973, 1990) refers to as the "triune brain". See Bernheim & Rangel (2004) or Loewenstein & O'Donoghue (2004) for further discussion of the supporting neuroscience evidence. Camerer et al. (2005) provide a detailed review of these issues and their consequences for modeling and decision-making in a variety of economic contexts. They also distinguish neural functions along two dimensions: cognitive versus affective and controlled versus automatic processes; the dual decision model largely focuses on the first of these.
[9] Brains of non-primates and non-mammalian species continue to evolve (Patton, 2008) but capabilities emerging after branching of the evolutionary tree leading to Homo sapiens are not incorporated into the human brain.

perceives and acts upon external stimuli *before* deliberative processes occur (LeDox, 1996). The number of neural connections running from the limbic system to the cortex also far exceeds the number going in the reverse direction, suggesting that emotional impulses frequently overwhelm rational cognitive processes.

Conflicts between the affective and deliberative systems are likely to be particularly salient for eating decisions that have large impulsive or automatic components. Berridge (1996) distinguishes between "liking" and "wanting", in the context of food, where the former roughly represents underlying taste parameters while the latter refers to potentially cue-conditioned affective responses.[10] Kessler (2009) provides a thorough discussion of strong endorphin and dopamine responses of the brain's "reward" (affective) system in the presence of foods, particularly those containing "supernormal stimuli" (Staddon, 1975), and how these can overwhelm the homeostatic feedback system through which the body ideally regulates eating to maintain weight.[11] Consistent with this, Wansink (2006) documents numerous examples of "mindless eating", where behavior is affected by environmental cues such as portion size, packaging and labeling, and sensory inputs like the sight and smell of food.[12]

2. The Dual Decision Model

The framework below focuses on decisions related to food intake and body weight. The deliberative system closely accords with traditional economic models and, under a specified set of assumptions, if operating in isolation it yields outcomes that are optimal in that deviations from them would be utility-reducing. However, the deliberative system instead interacts with the affective system

---

[10] He writes: "mesotelencephalic dopamine neurotransmitter systems and the central nucleus of the amygdale appear to participate more directly on wanting than in liking" (p. 21). Conversely, liking occurs in multiple parts of the brain, including in the "reptilian" brain (e.g. the brain stem). Berridge further points out that subjective reports may be inaccurate because "humans are not directly aware of many aspects of underlying psychological processes, motivational and otherwise, that control their behavior" (p. 19).

[11] Substantial research supports these conclusions (e.g. Rogers & Hill, 1989; Lambert, et al., 1991; Woods, 1991).

[12] For example, office workers ate one-third fewer chocolates if put in a desk drawer rather than on top of the desk, and less than half as many if placed six feet away. When asked why the latter placement reduced consumption, many answered that walking the six feet gave them time to think twice about whether they really wanted the chocolate. Thus, the extra time required was often sufficient for the deliberative system to influence decisions that would otherwise be dominated by the affective system.

where food consumption is present-oriented and resposive to specific stimuli. The resulting food and weight decisions are intermediate between what either system considers desirable.

The model incorporates a number of simplifications. Most importantly, dependence of current decision-making on past choices is ignored. This makes the mathematics transparent and illustrates the potential for suboptimal decisions without the need for addiction.[13] Food consumption is also assumed to be monotonically related to weight. This makes sense in a static model, when considering steady-state values, but implies that dynamic aspects of weight change are not examined. The model also abstracts from decisions about energy expenditure and so ignores complementary or compensating changes in physical activity. Unless fully offsetting, these will not alter the main model conclusions.

A natural consequence of dual decision-making is quasi-hyperbolic ($\beta - \delta$) discounting (Laibson, 1997). Specifically, the discount factor for future decisions, δ, is determined by the deliberative system, whereas current actions are also influenced by the impulsive affective system, yielding a lower discount factor (high discount rate), $\beta\delta$.[14] One outcome is that the degree of present-biasedness (β) differs across types of consumption, depending on the strength of affective system responses. Interaction of the deliberative and affective systems also provides a neuroscience-based explanation for models of "multiple selves", such as Sheffrin & Thaler's (1988) "planner-doer" model or Fudenberg & Levine's (2006) resolution of decision problems as the solution of a game between a long-run patient self and a series of short-run impulsive selves.[15] Moreover, it supplies a natural rationale for economic frameworks where restrictions on choices increase utility, such as Gul & Pesendorfer's (2001) model of temptation utility.

---

[13] Addiction increases the likelihood for such decision failures (see Bernheim & Rangel, 2004).

[14] Magnetic resonance imaging (MRI) of the brains of subjects choosing between immediate and delayed monetary rewards provides supporting evidence (McClure et al., 2004). The mesolimbic dopamine system and associated regions are involved only in choices with immediate outcomes, whereas the prefrontal cortex is always involved. When immediate payment is an option, relative activation of the two regions (prefrontal versus dopamine) is a significant predictor of the choices made. Conversely, Glimcher et al. (2007) obtain evidence for hyperbolic discounting without an indication of disparate activation of different areas of the brain.

[15] Sheffrin & Thaler (1988), postulate that individuals have coexisting but mutually inconsistent preferences: a long-run "planner" and a pathologically myopic "doer". Decisions of the planner reside primarily in the prefrontal cortex, those of the doer in the limbic system. The doer exercises direct control over decisions but can be constrained by the planner's use of (costly) self-control. In Fudenberg & Levine (2006), the long-run self chooses a self-control action that influences the utility function of the myopic self and is sometimes willing to incur short-run costs to reduce the future cost of self-control.

Sophisticated agents know that deliberative processes in later periods can be frustrated by affective system responses and so may take steps to reduce the future options available to the affective system.

A related "hot/cold" dual approach has been described by Metcalfe & Mischel (1999) and applied to addictive behaviors by Bernheim & Rangel (2004). Individuals in cold states act deliberatively but cued responses to stimuli can move them into "hot" states, where actions are reflexive and emotional, such that the deliberative system has little or no control. The dual decision approach is analogous except that deliberative processes are assumed to exert some influence in almost all cases. This model is also similar to cognitive-experiential self-theory, in psychology, which assumes that people process information using an "experiential" system and a "rational" system that operate in parallel and are interactive: the experiential system is automatic, preconscious, rapid, nonverbal and intimately associated with affect; the rational system is analytic, deliberative, slow and affect-free (Epstein, 2003).

2.1 The Deliberative System

The deliberative system is assumed to make food consumption choices according to the rational model developed by Philipson & Posner (2003) and elaborated upon by Lakdawalla & Philipson (2009). Utility (U) depends on weight (W), food intake measured as calories (f) and other consumption (c), and is maximized subject to a budget constraint. Weight is a function of f, p is the relative price of food, and I is income.[16] The optimization problem is characterized as:

(1) $$max_{f,c}\, U(W(f), f, c) \qquad \text{subject to} \qquad c + p\,f = I\ .$$

Individuals have an ideal weight (W^0), which they would choose if it were costless to achieve and food gave no direct utility. Therefore, $U_W(W^0) = 0$ and $U_{WW} < 0$, where subscripts represent partial derivatives. Food and other consumption are normal goods with diminishing marginal utility, so that U_f, $U_c > 0$ and U_{ff}, $U_{cc} < 0$; weight increases with food intake, implying that $W_f > 0$.

First-order conditions for utility maximization require choosing f and c to satisfy:

(2) $$U_W W_f + U_f = pU_c,$$

[16] Energy expenditure, which is ignored here, can easily be incorporated. In a model that does so, Lakdawalla & Philipson (2009) show that increased food intake raises body weight because it will be only partially offset by compensatory growth in energy expenditure.

implying equalization of utility from the last dollar spent on food and on other consumption. Food has both a direct positive effect on marginal utility (U_f) and an indirect effect ($U_W W_f$) that is positive (negative) at W less (greater) than W^0. The utility maximizing levels of W and f are decreasing in food prices: since a decrease in p lowers the right-hand side of (2), implying that reductions in c and increases in f (and so in W) are necessary to restore equality.

Next consider the relationship between utility-maximizing and ideal weight. Since U_f is positive, $U_W W_f$ needs to be negative at sufficiently low food prices for the first order conditions to be fulfilled. This requires that $W > W^0$. Therefore, rational consumers might state that they weigh more than would be ideal (referring to W^0), even while actually preferring their current weight, since achieving W^0 would require them to cut back on the amount of food consumed. On the other hand, it is difficult to imagine why such individuals would report attempting to lose weight through diets or other means, since such a drop in weight would be utility-reducing.

A primary implication is of rational decision-making is that public policies leading to lower average weight decrease utility and so should not be undertaken, unless there are negative externalities (like obesity-related medical costs) that food consumers do not account for. We abstract from such externalities to make the strongest case for standard economic framework. Even when doing so, two limitations of the model deserve attention. First, it is probably not appropriate to treat ideal weight, W^0, as an exogenous taste parameter if it is influenced by social norms, advertising or government policies. Such malleability creates a potential role for interventions. For instance, public health campaign stressing the undesirability of obesity might reduce W^0 and so lower utility-maximizing levels of body weight.

Second, the assumption that food consumption directly increases utility is not necessarily correct. For instance, individuals only gain utility from unappetizing food to the extent it provides needed calories. Interestingly, if food did not directly influence utility ($U_f = 0$), the first order conditions would require $U_W W_f = pU_c$, and individuals would choose to be *below* W^0 for any positive food price (since the marginal utility of weight would need to be positive).

The preceding discussion emphasizes the importance of considering why eating is pleasurable and the related consequences for food choices and body weight. This is accomplished by including preference shifters (z) that determine the utility obtained from calories and characterizing the utility-maximizing level of food (f^d) by:

(3) $$f^d = argmax_{f \in F} U(W(f), f, z),$$

where F is the feasible set of food options (incorporating biological and income constraints) and steady-state body weight is $W^d = W(f^d)$. Other consumption, c, is excluded from (3) and hereafter for expositional convenience but is implicit, since $c^d = I - pf^d$. The solution to this maximization problem is assumed to be optimal in that interventions causing food intake and weight to deviate from f^d and W^d reduce utility.

A key enhancement is that utility from food now depends on characteristics (z) like taste, texture, mouth-feel, speed at which it induces satiety, as well as other relevant factors such as visual cues and the social milieu of consumption. These are reflected by the cross-derivative U_{fz} and can often be manipulated by the food industry to make their products more attractive to consumers. Such innovations raise overall utility, even when they increase caloric intake and weight.[17]

2.2 <u>The Affective System</u>

There is also a separate affective system, where decisions are influenced by chemical reactions (e.g. dopamine responses) that occur quickly and automatically in response to cues and triggers. Affective system decisions do not account for not long-term consequences, like the relationship between food consumption and subsequent body weight. This system is influenced by food characteristics and stimuli, s, according to a "motivational function" $M(f, s)$.[18] Food characteristics influencing the affective and deliberative systems, s and z, are sharply distinguished here for illustrative purposes only. A more

[17] If food characteristics are structured to make eating pleasurable, $U_{fz} > 0$ and f^d and W^d increase. This occurs because, at given f, U_f and the left-hand-side of (2) rise, so that increased food intake is needed to restore equality.
[18] The effects of stimuli are also likely to depend on previous decisions. Laibson (2001) provides one example of a model emphasizing the role of such cue-contingent conditioned responses.

general model would allow such characteristics to influence both systems, although the strength or direction of the responses could vary.

The affective system optimal food intake is f^m where:

$$f^m = argmax_{f \in F} M(f, s). \tag{4}$$

Three points deserve mention. First, with pleasurable eating, it is likely that $f^m > f^d$ and $W^m > W^d$, because the affective system does not account for the consequences of current eating on future weight, whereas the deliberative system does. Second, producers have incentives to engineer into foods stimuli attractive to the affective system (those where $M_{fs} > 0$), if the increased revenue from doing so exceeds the associated costs. This supplies an additional reason for f^m to exceed f^d. Third, if $f^m > f^d$, consumers might prefer interventions that reduce the influence of the affective system.

2.3 Resolving Conflicts Between the Two Systems

Conflicts between the deliberative and affective systems, where $f^m > f^d$, are assumed to be resolved through intermediate levels of food intake, f^c, according to:

$$f^c = \phi(R) f^m + [1 - \phi(R)] f^d, \qquad 0 \leq \phi(R) \leq 1. \tag{5}$$

In (5), ϕ is a weighting function reflecting the relative power of the two systems and R is self-control, modeled here as a fixed endowment.[19] Greater self-control increases the relative strength of the deliberative system, $\phi_R < 0$. There are two extreme cases. With infinite self-control, the deliberative system dominates, such that $\phi = 0$ and $f^c = f^d$. This characterizes traditional economic models with full rationality. Alternatively, if self-control is completely lacking, $\phi = 1$ and $f^c = f^m$.

It is sometimes useful to rewrite (5) as

$$f^c = f^d + \phi(R)A, \tag{6}$$

[19] See Ozdenoren, Salant & Silverman (2006) for a more sophisticated approach where self-control is a depletable resource. Many processes could lead to the intermediate outcomes characterized by (5). For instance, assume the deliberative system has ultimate decision power but deviations from f^m impose a loss $L = L(|f - f^m|, R) = L(\Delta, R)$, for $\Delta = |f - f^m|$, where $L_\Delta > 0$ and $L_R < 0$. The first-order condition to be satisfied is $U_W W_f + U_f - L_\Delta \Delta_f = pU_c$ or, since $\Delta_f = -1$ when $f^d < f^m$, $U_W W_f + U_f + L_\Delta = pU_c$. At f^d, the equality (2) holds implying that the left-hand-side of the new first order condition exceeds the right-hand-side by L^d. To achieve equality, f must increase (and c decrease), lowering U_f, raising U_c, and implying that $f^c > f^d$.

where $A = (f^m - f^d)$ will be referred to as the strength of the affective system response.[20] Thus, overeating, defined as $f^c > f^d$, increases in A and decreases in R, both of which vary across individuals.

As detailed in appendix A, steady-state weight (W^*) can be approximated by the linear function:

$$W^* = af - b, \quad (7)$$

where a and b depend upon basal metabolism and physical activity. Substituting (6) into (7), the steady-state weight of individual i is:

$$W_i = a[f_i^d + \phi(R_i)A_i] - b. \quad (8)$$

(8) indicates that heavy individuals tend to have some combination of high utility-maximizing levels of food consumption (f^d), strong affective system responses (A) and weak self-control (R).[21]

2.4 Prices

Food prices are negatively related to consumption and body weight in the dual decision model, as in the standard framework, but several differences are worth mentioning. First, money prices have weaker effects because they do not influence the affective system. Second, price increases sometimes raise utility. This occurs since food consumption initially exceeds f^d (because of the affective system) and higher prices reduce eating, possibly bringing it closer to the utility-maximizing level.[22] Third, time prices are relatively more important. The intuition is that the affective system makes decisions more quickly than the deliberative system, so that higher time costs weaken its influence (i.e. reduce ϕ).[23]

2.5 Strategic Behavior

Persons aware that their affective systems cause overeating may act purposefully to reduce the excess consumption. Such behavior has been considered in the context of smoking (Gruber & Köszegi, 2001) or drug use (Bernheim & Rangel, 2004), and takes various forms. Many strategies voluntarily

---

[20] This is called "incentive salience" in the psychological literature (Berridge, 2007).

[21] Heavy persons may also have low basal metabolic rates and levels of physical activity.

[22] However, utility might fall instead, because of the negative income effect of price increases (which reduce c as well as f) or if the price rise is so large as to push eating sufficiently far below f^d.

[23] This can be seen by incorporating time prices (t) into the loss function in footnote (18) as $L = L(\Delta, t, R)$, where $L_{\Delta t} < 0$. Consider an increase in time prices that is offset by a reduction in money prices, such that the total price is unchanged. At f^c, the first order condition held initially but lower time prices reduce L_D lowering the left-hand-side of the equality but with no change in the right-hand-side (since p did not change). Food consumption must therefore fall (and in so doing raise U_f) to restore equality. Cutler et al. (2003) make a similar argument.

increase the money or time cost of eating. For instance, individuals may avoid bringing certain foods home, raising time costs since consumption requires a trip out of the house. Money prices may also increase if the food is purchased in more expensive outlets (e.g. restaurants instead of supermarkets) or in smaller quantities where the cost per unit is higher.

Individuals may limit their exposure to stimuli, s, that trigger affective system responses, for instance, by avoiding restaurants where overeating is likely to occur. They may also attempt to counteract affective system responses or reduce its time advantage over the deliberative system (e.g. by visualizing the consequences of eating energy-dense foods or establishing eating "rules"). These are designed to cut eating by decreasing f^m or ϕ. Efforts may also be made to directly increase self-control (reducing ϕ). Although modeled as an endowment above, self-control is more accurately characterized as an exhaustible resource that is depleted by stress, cognitive effort, and prior acts of self-restraint (Metcalfew & Mischel, 1999; Shiv & Fedorikhin, 1999; Ward & Mann, 2000). Stress-management may therefore conserve scarce self-control while formal diets reduce cognitive load. Joining weight-watchers or entering weight loss tournaments may be effective if public announcements of weight affect the loss function. Finally, medical procedures (like bariatric surgery) directly reduce f^m.

What is striking is that almost all of these strategies represent constraints that would cut the utility of fully rational consumers. Evidence of their frequent adoption therefore almost certainly indicates inadequacies of the traditional economic framework, where deviations from utility-maximizing food consumption and weight will be absent or short-lived. By contrast, dual decision-making predicts extensive and difficult to eliminate departures from f^d and W^d.

2.6 Food Engineering

Standard economic explanations for rising obesity emphasize falling food prices and increased costs of energy expenditure. Both factors operate in the dual decision model, with decreases in time costs being of particular importance, as discussed above and made more precise below.

Less attention has been paid to "food engineering", by which I mean the strategic manipulation of food characteristics to stimulate consumption. As mentioned, changes appealing to the deliberative system utility function are desirable, even if higher obesity results. However, food producers and marketers also have incentives to design their products to promote affective system responses, particularly since many standard constraints on consumption (e.g. concerns about future weight gain) are weak or nonexistent when the rapidly acting and semi-automatic affective system is stimulated.

Incentives to engineer food have always existed but the ability to do so has increased over time. Cutler et al. (2003) document the importance of technological advances transforming food production so that "preparation can now be done in restaurants and factories, exploiting technology and returns to scale" (p. 105). These innovations have been complemented and promoted by agricultural policies expanding the availability and reducing the cost of "program" crops, like soybeans and corn, that that have become major inputs into processed foods (Wallinga et al. 2009, Cawley & Kirwan, forthcoming). Both changes date from the 1970s, exactly when obesity began its rapid increase. The result is that we now primarily consume what Pollan (2008) describes as "edible foodlike substances" rather than food itself.

There are many components of food engineering.[24] Among the most important is finding the balance of (generally high levels of) fat, sugars and salt that maximize palatability (Drewnowski, 1995).[25] This is reflected in a 63 percent rise in the per capita consumption of added fats and oils, and the 19 percent increase in added sugars occurring between 1970 and 2005 (Wells & Buzby, 2008).[26] Engineering food also involves creating elaborately structured products with heightened complexity and multi-sensory effects. Food is designed to have the right combinations of flavor, aroma, oral and visual texture, and after-taste, usually making heavy use of refined products (often reducing the need for chewing, so that consumption occurs more quickly) and chemical flavorings. For eating outside the home, the environment is designed to be comfortable and stimulating, with attention paid to the variety of food on the plate and its packaging, as well as to cues like sound and lighting.

---

[24] This paragraph is based on the excellent in-depth investigation of these issues in Kessler (2009).
[25] Palatability refers to the capacity of food to stimulate appetite.
[26] The increase in fats and oils has been from vegetable (rather than animal) sources; the rise in sugars is due to corn sweeteners (e.g. high fructose corn syrup). As mentioned, both of these sources are mainly from program crops (like soybeans and corn) where production increases have been promoted by federal agricultural policies.

Advances in food engineering that are focused on the affective system are likely to cause broadly distributed but unequal increases in weight. As shown in equation (8), the changes will likely to be largest for those with strong affective system responses and low self-control. Since such persons would have tended to be relatively heavy, even with less sophisticated food engineering, the weight gains are expected to disproportionately affect those in the right tail of the distribution.

3. Data and Descriptive Patterns

I next empirically examine whether eating behavior and body weight are better described by standard utility-maximization or by frameworks, like the dual-decision model, where many individuals eat and weigh more than they consider optimal.[27] For brevity, these are sometimes labeled "rational" and "irrational" or "non-rational" eating although, even in the latter case, the deliberative system operates rationally (while the affective system does not).

Most data come from the second and third National Health and Nutrition Examination Surveys (NHANES 2, 1976-80; NHANES 3, 1988-94) and the first eight years (1999-2006) of the current continuously conducted NHANES (hereafter referred to as NHANES 99), with data from the first National Health Examination Survey (NHES, 1960-62) also used when considering longer-term trends in body weight. Each is a cross-sectional national survey conducted by the National Center for Health Statistics, Centers for Disease Control and Prevention and designed to provide prevalence estimates for selected diseases and risk factors, monitor trends in risky behaviors and environmental exposures, and to study the relationship between diet, nutrition, and health.[28]

NHANES data has several features that are useful for this project. Almost all respondents' complete health and laboratory examinations containing clinical measures of height and weight, obtained

---

[27] Deliberative system decision-making is treated as the appropriate locus determining welfare throughout, an assumption that can be questioned (e.g. Kahneman, 1994). However, the strong optimality conclusions resulting from traditional utility-maximization also disappear if this assumption is abandoned.
[28] See http://www.cdc.gov/nchs/nhanes.htm for additional information. Preliminary analysis also included NHANES 1 (1971-74). Since the BMI distribution was virtually identical to that in NHANES 2, these results are not shown.

using standardized procedures and equipment.[29] Such data avoid errors in self-reported height and weight that generally lead to underestimates of BMI, particularly for heavy individuals. Sample sizes are also reasonably large and the surveys contain 24-hour food diaries indicating the type, amount, and timing of foods eaten on the reference day. NHANES 3 and 99 include questions comparing current versus desired weight and the latter provides information on weight loss attempts and weight changes during the previous year.[30]

Supplemental data from the 1991, 1994, 2000 and 2003 waves of the Behavioral Risk Factor Surveillance System (BRFSS) are used to examine trends in weight loss attempts. The BRFSS contains large samples and consistent questions on such attempts (for the years analyzed). Height and weight are self-reported from telephone surveys, resulting in an understatement of BMI and obesity prevalence (Chou, et al., 2004) but there is no reason to believe that these errors vary over time.[31]

The analysis is restricted to 25-60 year olds. Persons under 25 are excluded to focus on adults with considerable experience managing the relationship between eating and body weight, for whom the rational model is presumably most applicable. Those over 60 are eliminated to reduce the effects of mortality selection and eating limitations induced by health problems. Most analysis is conducted separately for men and women, with selected estimates for subgroups stratified by age and education. Pregnant women are excluded. Sampling weights are incorporated throughout.

BMI is weight in kilograms divided by height in meters squared. Following national and international standards (World Health Organization, 1997; National Heart, Lung and Blood Institute,

[29] For the NHES, two pounds were subtracted from measured weight, because the examinee was partially dressed (unlike the other surveys where individuals wore only underwear) and the remaining clothing was estimated to weigh approximately two pounds (National Center for Health Statistics, 1981).
[30] NHANES 3 also includes questions on weight loss attempts but they are not comparable to those in NHANES 99.
[31] See http://www.cdc.gov/brfss/ for further information on the BRFSS. These years were chosen to provide comparable data and have similar economic conditions in the earlier and later periods: 1991 and 2003 were recession years; 1994 and 2000 were near the business cycle peak. Since Kansas, Nevada and Wyoming were excluded in 1991, observations from these states were dropped for the later survey years, to maintain comparability.

1998), "underweight", "healthy weight", "overweight", "mild obesity" and "severe obesity" are defined as BMI of <18.5, 18.0-24.9, 25.0-29.9, 30.0-34.9 and ≥35.0.[32]

Most econometric models include supplementary controls for: age and age squared, race/ethnicity (black, non-black Hispanic, other), education (high school dropouts, some college, college graduate), marital status (married, widowed, separated/divorced) and tobacco use (ever smoked, current smoker).[33] The results are generally not sensitive to the exact choice of covariates.

Figure 1 shows the BMI distribution in each of the four survey periods. Consistent with previous evidence (e.g. Flegal et al., 2002; Ruhm, 2007), body weight changed little between the early 1960s and late 1970s but increased rapidly thereafter. Median BMI was 24.5 in 1960-62 and 24.7 in 1976-80, versus 25.7 in 1988-94 and 27.3 in 1999-2006. The fraction of overweight/obese 25-60 year olds rose only slightly between 1960-62 and 1976-80 (from 45.4% to 48.1%) but increased to 56.0% in 1988-94 and 67.0% in 1999-2006.

Reductions money prices of food may explain some of the initial growth in obesity but are less relevant for the subsequent increases. Evidence for this is provided in Figure 2, which displays 1967-2007 trends in various components of prices, normalized so that 1967 values equal 100. The money cost of food is as the ratio of the food consumer price index (CPI) to the all-items CPI.[34] Eating time refers to the average hours spent by 25-60 year olds in meal preparation, consumption and cleanup; it is based on data from five time use studies (in 1965-66, 1975-76, 1985, 1992-94 and 2003) compiled and standardized by Aguiar & Hurst (2007).[35] Information on restaurants per capita is available at five-year

---

[32] BMI is the favored method of assessing excess weight since it is simple, rapid, and inexpensive to calculate. However, it does not account for variations in muscle mass or the distribution of body fat (e.g. intra-abdominal versus overall adiposity). Some researchers prefer alternative anthropometric measures such as waist circumference (Sönmez et al., 2003), waist-hip ratio (Dalton et al., 2003), or waist-height ratio (Cox and Whichelow, 1996). Cawley and Burkhauser (2008) recommend the use of Bioelectrical Impedance Analysis. Severe obesity combines the official definitions of class II and class III obesity.

[33] The reference group includes white, non-Hispanic, never married, high school graduates (without college) who have never smoked. Tobacco use is included because of empirical evidence suggesting its association with obesity, although the exact relationship is controversial (Gruber & Frakes, 2006; Chou et al., 2006; Courtemanche, 2009).

[34] CPI data are from Council of Economic Advisers (2009), Table B-60.

[35] The data were obtained from: http://troi.cc.rochester.edu/~maguiar/timeuse_data/datapage.html.

intervals beginning in 1967 from the Economic Census.[36] Eating time and the number or restaurants per capita are interpolated or extrapolated, using linear trends, for years without direct data.

These variables deserve further discussion. Food prices indicate the money price of food, relative to other consumption, but do not distinguish between food types (e.g. by energy density). A detailed analysis of U.S. price trends by Christian et al. (2009) suggests that this is not a major limitation. When considering time prices, it is important to distinguish between impulsive and planned eating. Many technological innovations decrease the time expense of planned consumption, whereas the more ubiquitous presence of food particularly reduces the time cost of unplanned eating. Consider microwavable meals, which cut the time needed to prepare planned meals at home. Dual decision makers might limit purchases of these products to reduce the risk of impulsive eating since, by doing so, a substantial time cost of going to the store would be required before such a meal could be prepared. Conversely, wider availability of food cuts the cost of unplanned consumption. For instance, the addition of convenience stores to service stations makes it easy to impulsively buy food when purchasing gas. Thus, where other researchers (e.g. Chou et al., 2004) use the number of restaurants as a general indicator of time prices, they are viewed here as to proxy food availability and so (mainly) as a measure of the time cost of unplanned eating. Conversely, eating time is assumed to be a better (albeit imperfect) indicator of the time cost of planned consumption.

Figure 2 demonstrates that the relative money price of food fell almost 13 percent between 1974 and 1985 but with no trend since then. Eating time similarly declined more than one-quarter from 1967 to 1993, but stabilized thereafter.[37] Thus these trends could explain some of the initial increase in obesity but

---

[36] These refer to establishments with payroll in SIC code 58 (Eating & Drinking Places) from 1967-1992 and NAICS code 722 (Food Services & Drinking Places) from 1997-2007, converted to capita by dividing by the U.S. resident population, and adjusted to make the SIC and NAICS codings comparable. See http://www.census.gov/econ/census07/index.html for further information on the Economic Census.

[37] Reductions in preparation and cleanup time explain more of the change between 1967 and 1993 than declines in actual eating. This reflects large decreases in meal preparation and cleanup by women, partially offset by increases among men. Ramey (2007) suggests that the drop through 1985 may be larger than that indicated in Figure 2, because most food consumed on the job was classified as "work" rather than "eating" in the 1965, 1975 and 1985.

not its continued growth since the early 1990s.[38] By contrast, restaurants per capita rose rapidly during the four decades: by 2%, 17%, 27% and 34% in 1977, 1987, 1997 and 2007, compared to 1967 – suggesting that time costs of unplanned eating could represent an important and continuing source of obesity growth, particularly in the context of models emphasizing impulsive consumption.

Table 1 shows how BMI is related to self-perceptions of weight, preferred weight, and weight loss attempts for the NHANES 99 cohort. Self-perceived weight is obtained from responses to whether the individual considers himself/herself to be "overweight", "underweight" or "about the right weight". Preferred weight refers to if he/she would like to weigh "more", "less" or "stay about the same". Weight loss attempts indicate individuals who have "tried to lose weight" during the past 12 months.[39]

Weight self-perceptions and preferences accord closely with national and international classifications using BMI. Almost all obese men and women consider themselves overweight and would prefer to weigh less, whereas a majority of healthy weight respondents claim to weigh about the right amount and most such men prefer to keep their weight about the same. A small (large) majority of men (women) classified as overweight, based on BMI, consider themselves too heavy and would like to lose weight; most of the clinically underweight think they weigh too little and would like to gain weight. Women are more likely than men to attempt weight loss at any given BMI.

4. Empirical Analysis

Four observations underlie the empirical strategies below. First, the dual decision model predicts that deviations from utility-maximizing weight will be ubiquitous and that weight loss attempts will be pervasive. Second, these eating mistakes will be concentrated among individuals with low self-control *(R)* or strong affective system responses *(A)*. Since, such persons tend to have high BMI, overeating and

[38] In a more formal analysis, Chou et al. (2004) estimate that food price trends explain around 13 percent of the increases in BMI and obesity occurring from 1984 to 1999. Just (2006) provides more general evidence of the limited price-responsiveness of food consumption. Goldman et al. (2009) show very small short-run effects of food prices on weight: a 10 percent increase in the money price is predicted to increase average BMI by just 0.6 percent (0.22 kg/m^2) after two years. They obtain substantially larger long-run elasticities but these depend on functional-form assumptions of model used, rather than being directly estimated.

[39] The relevant question was not asked of persons reporting that they intentionally *lost* more than ten pounds in the last year. Such individuals are coded as having attempted to lose weight.

its related consequences and behavioral responses will be positively related to body weight. Third, increased sophistication of food engineering provides one reason for secular increases in obesity. Fourth, advances in food engineering that are focused upon stimulating affective system responses will disproportionately affect relatively heavy individuals, for whom A tends to be high. As a result, BMI is likely to have become more strongly related (over time) to overeating, the consumption of engineered foods, and associated consequences such as weight loss attempts.

It is useful to compare these predictions to those obtained under the traditional economic model. Although rational consumers may exceed their "ideal weight" (W^0), they will not choose to cut energy intake because resulting utility loss exceeds the benefits of lower weight. Deviations of weight from utility-maximizing levels will typically be small, short-lasting and idiosyncratic: occurring, for example, because higher food prices reduce f^d, requiring a brief adjustment to reach the newly desired steady-state.[40] A corollary is that there is generally no reason to expect weight loss attempts to be related to BMI. Since falling food prices provide a major explanation for rising obesity, secular increases in weight should also slow or stop during periods when such prices are stable. Moreover, there are no clear predictions about where in the BMI distribution secular weight gains should be concentrated but, since such increases are utility-maximizing, they will *not* be accompanied by more frequent weight loss attempts. Finally, there are no unambiguous predictions about the relationship between BMI and the consumption of engineered foods, although with sufficient assumptions almost any pattern is possible.

Based on these observations, the empirical analysis will test four specific hypotheses.

Specific Hypothesis 1: Secular weight growth has disproportionately occurred in the right-tail of the BMI distribution, with skewed increases observed even during periods of relatively stable food prices.

Specific Hypothesis 2: Weight mistakes are common, particularly at high levels of BMI, and have increased over time. The strongest test for these errors comes from patterns of weight loss attempts.

[40] Since it is usually healthy to gain or loss one to two pounds per week, there is no biological constraint to gaining or losing fifty pounds or more during a single year.

Specific Hypothesis 3: Severe obesity results from uncontrolled eating, as evidenced by high rates of large (and presumably unintended) weight gains among the heaviest individuals.

Specific Hypothesis 4: Obese individuals consume a disproportionate share of engineered foods, particularly in recent years, with the increasing sophistication of food engineering.

4.1 Body Weight Trends

The dual decision model predicts that secular increases in weight should be concentrated in the upper tail of the BMI distribution, since it is populated by individuals with strong affective system responses and low self-control, who are most affected by advances in food engineering. Declining food prices could also result in an increasingly skewed BMI distribution, even with fully rational consumers, if the marginal utility of food diminishes more slowly for heavier persons. However, this should have slowed or stopped between NHANES 3 and 99, because prices changed little during that time.

To more confirm the preliminary evidence of increasing skewness of the BMI distribution presented in Figure 1, Table 2 summarizes the results of quantile regressions examining trends in the 10^{th}, 25^{th}, 50^{th}, 75^{th}, 90^{th} and 95^{th} percentiles of BMI, using data from NHANES 2, 3 and 99. The table shows predicted differences in 1988-94 and 1999-2006 versus the 1976-80 baseline. Specification (a) controls only for NHANES survey dummy variables; model (b) also holds constant the demographic covariates.

Three findings are noteworthy. First, body weight rose disproportionately in the upper tail of the distribution: the 90^{th} and 95^{th} percentiles male (female) BMI grew 4.5 and 5.5 (5.5 and 5.9) kg/m^2 from 1976-80 to 1999-2006, compared to 2.1 (3.1) kg/m^2 at the median. Second, the rate of increase was at least as fast between 1988-94 and 1998-2006 as from 1976-80 to 1988-94: median male (female) BMI rose 0.6 (1.2) kg/m^2 between NHANES 2 and 3, compared to 1.6 (1.9) kg/m^2 from NHANES 3 to 99. At the 90^{th} percentile, BMI growth was 2.0 (2.8) kg/m^2 in the earlier period and 2.5 (2.7) kg/m^2 in the later one. Third, the patterns are not much affected by controlling for demographic characteristics. Predicted growth between 1976-80 and 1999-2006 was 1.3, 2.0 and 4.3 kg/m^2, at the 25^{th}, 50^{th} and 90^{th} percentile for males, with controls (specification b), versus 1.5, 2.1 and 4.5 kg/m^2 without them (model a). For women, expected BMI growth was 1.6, 2.9, and 5.7 kg/m^2 with the additional covariates and 1.8, 3.1 and

5.5 kg/m^2 without. The inclusion of a still more comprehensive set of covariates (not shown) had little effect on the findings, often resulting in parameter estimates closer to those in model (a) than (b).[41]

4.2 Weight Preferences and Weight Loss Attempts

Skewed weight growth during periods of stable food prices suggests non-rational eating but is possible under the traditional model. For example, the costs of being (as opposed to becoming) heavy may have declined due to improvements in medical treatments or reductions in the social stigma of obesity (Gregg et al., 2005; Flegal et al, 2007). Under such explanations, utility-maximizing levels of weight (W^d) may have increased but rational consumers should rarely be trying to *lose* weight. By contrast, if the affective system causes overeating, weight loss attempts will be pervasive, positively related to BMI, and increasing over time as advances in food engineering stimulate excess consumption.

Table 1 demonstrated that most overweight and obese individuals consider themselves too heavy and desire to weigh less. If these questions are answered relative to utility-maximizing weight, they provide strong evidence in irrationality. However, such responses could be consistent with rational eating if the responses are provided in relation to ideal rather than utility-maximizing weight (W^0 rather than W^d). Much harder to reconcile with the standard model is that weight loss attempts are frequent and highly correlated with BMI. More than one-third of men and half of women had tried to lose weight during the previous year, as did 55 (70) percent of obese males (females).

I next use regression analysis to more fully explore these relationships. The initial investigation focuses on NHANES 99 and dichotomous outcomes indicating whether preferred weight is less than current weight and the prevalence of weight loss attempts during the prior year. The patterns are allowed to vary in a highly flexible manner by including BMI dummy variables for: < 20, single point ranges between 20 and 30 (e.g. 20.0-20.9), two point categories from 30 to 40 (e.g. 30.0-31.9), and ≥40.[42] Probit models are estimated, with the inclusion of demographic controls. Predicted values are calculated for each

[41] The additional covariates were veteran status, foreign birth, income-to-poverty ratio, and housing crowding defined, following Melki et al. (2003), as number of residents divided by rooms in the household excluding the kitchen and bathrooms (with regression controls indicating values < 1 or > 1.5, with 1-1.5 the reference group).

[42] Two point BMI categories are used above 30 since the right-tail of the distribution is less densely populated.

sample member at specified BMI levels, by "switching on" the corresponding BMI dummy variable and "switching off" all others. Sample averages of the predicted values are calculated and summarized in Figure 3A, where "Weigh Less" refers to the first dependent variable and "Diet" to the second.

The top two lines of the figure confirm that desires for weight reduction rise monotonically with BMI and are always higher for females than males: 16.1, 77.1 and 95.0 percent of women with a BMI of 20, 25, and 30 are predicted to prefer to weigh less than they actually do, compared to 3.6, 30.0 and 80.4 percent of corresponding men. At the median BMI (27.5 for men and 27.0 for women), 58 percent of males and 86 percent of women think they weigh too much.

The bottom two lines of Figure 3A show that weight loss attempts also increase with BMI and are more common among women than men. At BMI of 20, 25, and 30, weight loss attempts are predicted for 17.9, 56.2 and 68.3 percent of females versus 4.0, 26.0 and 44.6 percent of men. Attempted weight loss is relatively stable for women, once the obesity threshold is reached, but continues to rise with BMI for men.[43] As discussed, a positive relationship between weight loss attempts and BMI is predicted by the dual decision model but harder to explain with rational eating.

Figure 3B examines whether the connection between BMI and weight preferences changed between 1988-94 and 1999-2006.[44] There is remarkable congruence across the two time periods, with the curves being nearly on top of each other at most BMI levels. Since body weight trended rapidly upwards, this indicates that adults have become increasingly likely to feel that they weigh too much.[45]

A sharper test between rational and irrational eating involves examining whether weight loss attempts have become more common over time. With rational consumption, the observed growth in weight reflects increases in W^d and the frequency of dieting should not have grown. Conversely, with impulsive eating, much weight gain will have been unintentional and weight loss attempts will have risen. Since questions on weight loss attempts are not comparable between NHANES 3 and 99, this issue is

---

[43] The results are similar for a dichotomous dependent variable indicating both weight loss attempts and desires.
[44] The analysis cannot be extended back to 1976-80 because the necessary questions are not included in NHANES 2.
[45] Probit models predict that the probability of preferring to weigh less are about 0.9 percentage points greater for males and 1.2 points higher for females in NHANES 99 than NHANES 3, although the estimates are imprecise.

investigated using the 1991, 1994, 2000 and 2003 years of the *Behavioral Risk Factor Surveillance System*, containing the common question "Are you now trying to lose weight?" To make the analysis as comparable as possible to that using NHANES 3 and 99, 1991 and 1994 are grouped together, as are 2000 and 2003. The econometric model is the same as in Figures 3A and 3B, except that the top BMI category is ≥36 (rather than ≥40), reflecting the known understatement of BMI when using self-reported data.[46]

Figure 3C confirms that weight loss attempts increase monotonically with BMI and are more common for females than males. However it adds evidence that the probability of dieting, conditional on BMI, remained essentially unchanged from 1991/94 to 2000/03. With rational eating, weight loss attempts at given BMI levels should have become *less* common in later years, since utility would be maximized at higher levels of weight. Instead, W^d appears not to have changed, suggesting that the secular rise in BMI reflects mistakes, and that the unconditional (on BMI) probability of weight loss attempts will have become much more common over time. Probit analysis confirms this. This probability is predicted to be a highly significant 5.5 (4.4) percentage points greater for males (females) in 2000/2003 than the 1991/94 baseline of 29.6 (45.1) percent.

4.3 <u>Weight Changes</u>

In the dual decision model, uncontrolled eating that leads to rapid weight gains is likely to be particularly common at high levels of BMI because heavy persons often have strong affective system responses or weak self-control. Such individuals might also relatively often drop substantial weight, if their frequent weight loss attempts are even partially successful (at least temporarily). By contrast, weight variations by utility-maximizing consumers are planned, should generally be of small size (since W^d will usually not change much in a single year), and probably uncorrelated with or negatively related to BMI.[47] These predictions are explored using information data from NHANES 99.[48]

Table 3 provides descriptive information on the probability of weight gain and increases of at least 5 or 10 pounds and 5 or 10 percent of body weight during the last 12 months. Obese and overweight

---

[46] For example, the 95th percentile of BMI is 42.4 for NHANES 99 females versus 38.7 in the 2000/2003 BRFSS.
[47] For example, large gains might be concentrated among individuals who previously lost weight due to illness.
[48] These results must be interpreted with caution since self-reports of weight changes may contain errors.

persons are somewhat more likely, than their counterparts, to have added weight but much more frequently gained large amounts: 31.1 and 24.7 (33.3 and 34.0) percent of severely and mildly obese men (women) gained at least 10 pounds in the prior year, compared to 10.7 (13.8) percent of healthy weight persons. Obese individuals are also much more likely to have added ≥10 percent of body weight.

Probit models confirm that these patterns persist after controlling for demographic characteristics. Severely obese men are 15.6 (21.7) percentage points more likely than those of healthy weight to have gained 5 (10) or more pounds during the last year and 13.4 points more probable to have a ≥10 percent increase; corresponding differentials for severely obese women are 11.5, 18.9 and 10.2 percentage points (see Table 4). High BMI individuals tend add large amounts of weight, when gaining any, which is again consistent with uncontrolled eating. Conditional on some weight increase, severely obese men (women) are 15.2 and 45.1 (20.6 and 43.8) percentage points more likely than their healthy weight counterparts to add at least 5 and 10 pounds, and 19.8 (19.5) points more probable to have a ≥10 percent weight increase.

Weight is also more variable at high BMI: standard deviation of the one-year change is 9.3, 11.9, 14.3, and 22.0 pounds for healthy weight, overweight, mildly obese and severely obese men and 10.1, 15.5, 16.9 and 22.7 pounds for corresponding women. This is consistent with heavy individuals being more likely to have periods of uncontrolled eating, combined with repeated efforts to lose weight.

Weight loss attempts are used throughout as an indicator of eating mistakes. However, it only makes sense for the deliberative system to undertake such efforts if they meet with some success. Table 5, which displays predicted weight changes as a function of BMI 12 months earlier and the presence or absence of weigh loss attempts, shows that this appears to occur.[49] Severely obese men (women) trying to cut weight lost 11 (8) pounds during the previous 12 months, which is 9 (8) pounds more than their counterparts not attempting to do so. Dieting was associated with a 6 pound decrease for mildly obese men, versus a one pound gain for the comparison group; corresponding women did not lose weight but

[49] These estimates are from regression models that include demographic covariates and a full set of interactions between weight class and weight loss attempts. Lagged weight class is used so that the key conditioning variable is evaluated prior to attempted weight loss.

were able to avoid the gains experienced by their non-dieting counterparts. Weight loss attempts did not have a statistically significant effect for overweight or healthy weight persons.

4.4 Food Consumption

The dual decision model predicts that heavy individuals will consume disproportionate amounts of engineered foods, particularly in recent years as the latter has become more sophisticated. A preliminary investigation of this issue is provided using the 24-hour food diaries to examine patterns of macronutrient intake for NHANES 99 and corresponding changes occurring between NHANES 2 and 99. An obvious problem is in how to define engineered foods. Since such products tend to be high in salt and invisible fats, I investigate how the consumption of sodium and fat, particularly saturated fat, varies with BMI. A more complete examination of specific food products is beyond the scope of the current study.[50]

Table 6 summarizes regression estimates for NHANES99 respondents that control for day-of-the-week, as well as the demographic covariates. Consumption of fats and saturated fats are measured as a percentage of total calories and salt intake as milligrams per calorie.[51] Baseline estimates, reported in the last row of each panel, present predicted values for healthy weight persons with other regressors evaluated at the sample means.

The results provide strong evidence that obese individuals consume disporportionate amounts of salt and fat. The predicted energy share from fats is 3.4 (1.8) percentage points higher for severely obese males (females) than the healthy weight baseline of 31.5 (32.3) percent, an 11 (6) percent difference. Corresponding differentials for mildly obese men (women) are 2.3 (1.7) percentage points or 7 (5) percent; those for overweight persons are positive but small and insignificant. The differentials for

[50] Total energy intake is not focused upon because previous research suggests that it tends to be under-reported, particularly by heavy individuals (Briefel et al. 1997; Hill & Davies, 2001), and because obese individuals are frequently dieting. I examined and obtained evidence that the number of daily eating occasions was negatively related to BMI. Mancino & Kinsey (2008) predict and provide empirical evidence that caloric intake increases with the length of the interval between meals (corresponding to fewer eating occasions). Hamermesh (2010) also finds that the frequency of eating occasions is negatively correlated with BMI. Appendix Table B.1 supplies additional descriptive information on how eating patterns differ by weight class.

[51] Obese individuals probably understate the consumption of fats and carbohydrates by more than proteins (Heitmann et al., 2000; Lafay et al., 2000), biasing the results against the maintained hypothesis that the energy share of calories from fats increases with BMI. Time trends will be unaffected if the reporting errors remain constant across periods.

saturated fats are even larger in percentage terms: 14 (8) percent higher for severely obese men (women) and 10 (6) percent greater for the mildly obese. Finally, severely obese males (females) are predicted to consume .08 (.10) mg/kcal or 5 (11) percent more salt than the reference group; the mildly obese consume .08 (.06) mg/kcal or 7 (4) percent more. (Overweight also females consume more sodium than their healthy weight counterparts; but corresponding males do not).

I next investigate whether the positive relationship between BMI and the consumption of fat or sodium strengthened over time, using data from NHANES 2 and 99.[52] In addition to demographic covariates, the regressions control for a fourth-order polynomial of BMI.[53] Predicted values are expressed as sex-specific and survey-specific differences relative to a BMI of 20. This normalization automatically accounts for methodological changes in the 24-hour food diaries.[54]

There was no association between BMI and the share of energy from fat in 1976-80 but a strong positive relationship had emerged by 1999-2006 (Figure 4A).[55] For example, there was no difference in the percentage of calories from fat for persons with a BMI of 35 versus 20 in the earlier period but the share was predicted to be 3 (2) percentage points higher for the heavier men (women) in the later one. Similarly the energy share from saturated fats was unrelated to male body mass and possibly weakly increasing with female BMI in 1976-80, whereas a strong positive relationship existed in 1999-2006, particularly for men (Figure 4B).[56] Finally, sodium intake was uncorrelated with or negatively related to body weight in 1976-80, whereas a strong positive correlation existed in 1999-2006: predicted

---

[52] Patterns for 1988-94 (not shown) are closer to those for 1999-2004 than 1976-80.
[53] Similar results are obtained using the same discrete BMI dummy variables as in Figures 3A and 3B, except that the curves are less smooth and the relationships not as visually apparent.
[54] The reported average share of calories from fat declined from 37 percent in 1976-80 to 34 percent in 1999-2006, consistent with other evidence (Centers for Disease Control and Prevention, 2004). This may reflect greater under-reporting of fat consumption (Heitmann, et al., 2000) and reductions in the proportion of energy from fats. Popkin et al. (2001) emphasize that there has probably been a decrease in the consumption of "visible" fats, such as those in meat, even while more "invisible fats" (incorporated in engineered food products like pizza, burritos, pasta and luncheon meats) are being eaten.
[55] The p-value on the joint test of significance of the BMI coefficients exceeded 0.8 for both sexes in 1976-80 but was less than 0.01 for both in 1999-2006.
[56] The p-value on the joint test of the BMI coefficients was 0.66 for men and 0.37 for women in 1976-80 but below 0.01 for both in 1999-2006.

consumption was 0.10 (0.13) mg/kcal higher at 35 than 20 kg/m^2 for men (women) in NHANES 99 compared to 0.01 (-0.05) mg/kcal in NHANES 2 (Figure 4C).[57]

4.5 Age and Education Differences

Persons aged 25 and over have been assumed to have a good understanding of the connection between food consumption and body weight. However, some adults may fail to fully comprehend this relationship and so make erroneous, but still rational, eating decisions. Circumstances might also alter so as to induce undesired weight gains that can only be eliminated through lifestyle changes that are outside of the previous experience, requiring lengthy experimentation to restore utility-maximizing weight levels.

For instance, older individuals could inadvertently consume excess food because they are unaware that basal metabolic rates (BMR) decline with age (Henry, 2005; Frankenfield et al., 2005).[58] Overeating and its associated consequences might similarly reflect a lack of information. This would presumably be less of an issue for the highly educated, who have the best information and ability to process it.

These possibilities are examined by comparing results for subsamples stratified into “young adults” (25-42 year olds) versus “mature” individuals (aged 43-65) and the “college educated” compared to those with “no college” (high school dropouts or graduates). Disproportionate secular weight growth for heavy individuals is investigated by estimating predicted trends between 1976-80 and 1999-2006 at the 50th and 90th BMI percentiles. Next, BRFSS data are used to determine changes from 1991/4 to 2000/03 in the unconditional (on BMI) probability of weight loss attempts. Finally, consumption of engineered foods is examined by comparing the ratio of predicted intake of salt and fat (in 1976-80 and 1999-2006) for persons with BMIs of 35 and 20 – hereafter referred to as “35-20” ratios. The analysis methods are the same as above. Results for males are summarized in Table 7 and those for females in Table 8.

---

[57] The BMI coefficients were jointly significant at the 0.01 level for NHANES 99 males and females but the p-values were 0.95 and 0.79 for their NHANES 2 counterparts.
[58] Such forecast errors become more plausible if age-related decreases in BMR are non-linear. The available evidence is limited but existing BMR equations incorporating age usually assume a linear relationship.

There is little indication that overeating occurs because of unawareness of or difficulty in accounting for age-related declines in basal metabolism. Were this the case, skewness of the BMI distribution, growth in weight loss attempts, and the consumption of engineered foods by heavy individuals would likely have risen faster over time for mature than young adults. BMI does grow marginally faster for the older group – e.g. by 4.5 (5.5) kg/m^2 for mature men (women) at the 90th percentile versus 4.0 (5.4) kg/m^2 for corresponding persons younger than 43 – but the small absolute differences do not approach statistical significance and reflect the higher baseline BMI of mature adults. Engineered foods account for a rising share of the diets of heavy individuals but with no evidence of a more rapid increase for older adults. The 35-20 ratio for fat consumption is 1.00 (1.01) for young (mature) males in 1976-80 and 1.09 (1.10) in 1999-2006; the 35-20 ratio for sodium intake rises more slowly for mature than young men (from 1.07 to 1.09 versus 0.97 to 1.05). Both ratios grow less over time for mature than young women. The 6.0 percentage point increase in the predicted weight loss attempts of older males does exceed the 4.8 point rise for young men (and starting from a lower baseline) but this difference is neither statistically significant nor replicated among females.

The data strongly refute the possibility that secular increases in overeating and weight loss mistakes are concentrated among the less educated. Growth in BMI is similar for college and non-college educated males at both the 50th and 90th percentiles, with lower baseline values suggesting faster relative increases for the highly educated. Skewness of the BMI distribution increased more for college than non-college females: the predicted rise at the 90th BMI percentile was 6.4 kg/m^2 for the former versus 4.7 kg/m^2 for the latter. The 6.7 (5.5) percentage point predicted growth in weight loss attempts, between 1991/94 and 2000/03, for college educated males (females) exceeds the 3.8 (3.0) point increase for those without college. Finally, the 35-20 ratios generally increased more between 1976-80 and 1999-2006 for the highly educated. For fat consumption, it rose from 0.97 to 1.12 (1.01 to 1.09) for college educated men (women) versus from 1.02 to 1.09 (1.01 to 1.02) for those with less schooling. For sodium intake the ratio increased substantially for highly educated males (from 0.94 to 1.08) while remaining unchanged for the less educated. This ratio may have increased faster for college than non-college educated females but the baseline values are imprecisely measured.

5. Discussion

In the standard economic framework, eating and body weight decisions reflect utility-maximization by fully rational individuals. However, such models fail to account for the role of biology, where cognitive decision-making centered in the pre-frontal cortex is accompanied by emotional and impulsive responses occurring in more primitive parts of the brain.

As an alternative, I develop a "dual decision" model where eating behaviors reflect the combined influences of a utility-maximizing deliberative system and an affective system that responds quickly and often impulsively to external stimuli, without accounting for the long-term consequences. The resulting food consumption generally deviates from the utility-maximizing optimum that would occur if the deliberative system operated in isolation. Dual decision-making is consistent with and provides a reason for models emphasizing self-control problems and self-perceived mistakes, such as quasi-hyperbolic discounting or cue-triggered consumption. An important implication is that advances in food engineering may be responsible for some of the growth in obesity occurring since the late 1970s.

The empirical analysis indicates that energy intake and body weight frequently exceed utility-maximizing levels. Weight loss attempts and the probability of large weight gains are strongly positively related to BMI and dieting has increased over time. Such patterns are consistent with eating "mistakes" caused by dual decision-making. Food engineering is difficult to study but the emergence of a positive relationship between BMI and sodium intake and the energy share of fats is consistent with a role for it.

By contrast, the results are hard to reconcile with the standard utility-maximization. In that model, individuals will usually be at or near their desired body weight, with changes in the latter generally being small (in response to fluctuations in determinants like prices) and rapidly achieved. There is no reason for weight loss attempts to be strongly related to BMI or to have become more common over time. Nor are there obvious relationships between BMI and the consumption of engineered foods. Falling food prices and increased costs of energy expenditure play a key role in explaining weight trends in this model, but neither changed dramatically after the late 1980s or early 1990s, while obesity continued to rise.

The analysis of subgroups highlights at least two disparities deserving mention. First, overeating may be a more serious problem for women than men: females had larger secular increases in BMI (particularly in the right-tail of the distribution) and were more likely to experience large weight gains (during a 12 month period), state that they weigh too much, and attempt to lose weight. Second, college educated individuals probably had fewer overeating problems than their less educated counterparts in the 1970s, but these differentials may have narrowed over time.

Although the current analysis does not identify sources of these disparities, or definitively confirm their occurrence, it suggests some fascinating possibilities. For instance, if affective system responses increase *absolute* rather than *relative* caloric intake, bigger effects would be expected for women than men, since the former have lower average basal metabolisms. The narrowing education gaps are consistent with a situation where highly informed and cognitively sophisticated consumers were better able than their counterparts to avoid excessive food consumption during the 1970s but with subsequent advances in food engineering overwhelming the self-control mechanisms of these individuals. Future research could fruitfully study these issues, as well as others, such as the degree to which individuals compensate for affective system induced overeating by increasing physical activity.

Verification of dual decision-making would have important implications for policy. Although individuals respond to higher prices by reducing consumption, as with standard utility-maximization, this framework emphasizes the role of time prices, which weaken the influence of the rapidly operating affective system. Taxation has been proposed to curtail the consumption of energy-dense foods and beverages (Jacobson & Brownell, 2000; Brownell et al., 2009) but the dual decision approach suggests that policies selectively constraining the availability of such products (and so raising time prices) may be more effective.[59] An example is the elimination of energy-dense commodities from school cafeterias and vending machines. Behavioral economists have recently advocated these and other interventions, like

[59] Empirical evidence also suggests the limited effectiveness of taxation in this context (e.g. Chouinard et al., 2007).

designing defaults options (e.g. smaller portion sizes), to exploit systematic decision errors generally leading to overeating so as instead to reduce energy intake (Just, 2006; Lowenstein et al., 2007; Downs et al., 2009). The traditional model includes a potential government role in the providing information about food characteristics (or requiring the private sector to do so) since consumers need to be informed in order to make good decisions. Such information may be less useful for dual decision-makers, to the extent that errors reflect affective system responses and the food engineering strategies designed to trigger them.

More fundamentally, absent public goods, externalities, or information imperfections, most policy interventions have negative effects with rational eating, since they distort eating decisions away from utility-maximizing levels. By contrast, mistakes are a central feature of dual decision-making, implying a wider potential role for policy. That said, the specific interventions will often be complicated and, if poorly implemented, will reduce rather than increase utility. The general reluctance of economists to engage in policy activism therefore retains merit.

Appendix A: Food Intake and Steady-State Weight

The energy accounting framework developed by Cutler et al. (2003) is useful for determining the relationship between food intake and steady-state weight. Energy is expended on basal metabolism (*B*), the "thermic effect" (*T*) – the energy used to process food – and calories expended on physical activity (*P*). *B* and *P* are related to weight according to:

(A.1) $$B = \alpha + \beta W \quad \text{and}$$

(A.2) $$P = EW,$$

where *E* is the level of physical activity. The thermic effect is directly proportional to energy intake:

(A.3) $$T = \gamma f,$$

so that total energy expenditure (*N*) is:

(A.4) $$N = \alpha + (\beta + E)W + \gamma f.$$

Weight is in steady-state (*W**) if calorie intake (*f*) and expenditure (*N*) are equal. This requires:

(A.5) $$W^* = af - b,$$

where $a = (1-\gamma)/(\beta+E)$ and $b = \alpha/(\beta+E)$. (A.5) implies that $\partial W^*/\partial f = a$, so that steady-state weight is linear in caloric intake. Cutler et al. provide the following values for the parameters determining a and b for men (women), with weight measured in kilograms: $\alpha = 879$ (829); $\beta = 11.6$ (8.7); $\gamma = 0.1$; and $E =$ 12.6 (11.3) for 70 kg men (60 kg women). Using these values, $a = .0372$ (.0450) and $b = 36.32$ (41.45), implying that a 100-calorie increase in daily energy intake raises the steady-state weight by 3.7 (4.5) kg or 8.2 (9.9) pounds.

Appendix B: Additional Results

Table B.1 Eating Patterns of 25-60 Year Olds By Sex and Weight Class

	Full Sample	Weight Class			
		Healthy Weight	Overweight	Mild Obesity	Severe Obesity
Males					
Total Energy (kcal)	2795 (21)	2816 (37)	2769 (33)	2829 (45)	2781 (60)
% of Calories From					
Fat	33.1 (0.2)	31.7 (0.3)	32.6 (0.3)	34.6 (0.4)	35.7 (0.5)
Saturated Fat	10.8 (0.1)	10.3 (0.1)	10.7 (0.1)	11.4 (0.2)	11.8 (0.2)
Carbohydrates	47.1 (0.2)	48.3 (0.4)	47.3 (0.3)	45.9 (0.4)	45.9 (0.6)
Protein	15.2 (0.1)	14.8 (0.1)	15.3 (0.1)	15.7 (0.2)	15.7 (0.3)
Alcohol	4.5 (0.2)	5.2 (0.3)	4.8 (0.2)	3.8 (0.3)	2.6 (0.4)
Sodium Intake (mg/kcal)	1.57 (0.01)	1.54 (0.02)	1.54 (0.02)	1.63 (0.02)	1.63 (0.01)
# Eating Occasions/Day	4.88 (0.05)	4.88 (0.05)	4.96 (0.06)	4.80 (0.07)	4.74 (0.09)
Females					
Total Energy (kcal)	1903 (13)	1902 (21)	1860 (28)	1900 (29)	1958 (31)
% of Calories From					
Fat	33.4 (0.2)	32.7 (0.3)	33.3 (0.3)	34.3 (0.4)	34.5 (0.4)
Saturated Fat	11.0 (0.1)	10.7 (0.1)	10.9 (0.1)	11.2 (0.2)	11.4 (0.2)
Carbohydrates	48.9 (0.2)	49.0 (0.3)	48.9 (0.4)	48.8 (0.5)	48.7 (0.4)
Protein	15.2 (0.1)	15.0 (0.2)	15.4 (0.2)	15.2 (0.2)	15.5 (0.2)
Alcohol	2.4 (0.1)	3.3 (0.2)	2.3 (0.2)	1.7 (0.2)	1.3 (0.2)
Sodium Intake (mg/kcal)	1.61 (0.01)	1.59 (0.02)	1.63 (0.02)	1.62 (0.02)	1.66 (0.01)
# Eating Occasions/Day	4.88 (0.04)	5.04 (0.06)	4.86 (0.07)	4.86 (0.07)	4.64 (0.07)

Note: Data are from NHANES 99. The sample includes 4,831 males and 4,709 females. Food intake is measured over a 24-hour observation period. Eating occasions are separated by at least of 30 minute period without eating or drinking. Observations are weighted and robust standard errors, clustered by NHANES survey, PSU, and strata are shown in parentheses.

References

Aguiar, Mark and Erik Hurst. 2007. "Measuring Trends in Leisure: The Allocation of Time Over Five Decades" *Quarterly Journal of Economics* 122(3), August, 969-1006.

Andreyeva, Tatiana, Roland Sturm, and Jeanne S. Ringel. 2004. "Moderate and Severe Obesity Have Large Differences in Health Care Costs" *Obesity Research* 12(12), December, 1936-1943.

Becker, Gary S. and Kevin M. Murphy. 1988. "A Theory of Rational Addiction" *Journal of Political Economy*, 96(4), August, 675-700.

Bernheim, B. Douglas and Antonio Rangel. 2004. "Addiction and Cue-Triggered Decision Processes" *American Economic Review* 94(5), December, 1558-1590.

Berridge, Kent C. 1996. "Food Reward: Brain Substrates of Wanting and Liking" *Neurosciences and Biobehavioral Reviews* 20(1), 1-25.

Briefel, Ronette R., Christopher T. Sempos, Margaret A McDowell, Sandy (Chia-Ying) Chien, and Katherine Alamio. 1997. "Dietary Methods Research in the Third National Health and Nutrition Examination Survey: Underreporting of Energy Intake" *American Journal of Clinical Nutrition* 65(4s), April, 1203s-1209s.

Brownell, Kelly D., Thomas Farley, Walter C. Willett, Barry M. Popkin, Frank J. Chaloupka, Joseph W. Thompson and David S. Ludwig. 2009. "The Public Health and Economic Benefits of Taxing Sugar-Sweetened Beverages" *New England Journal of Medicine* 361(16), October 15, 1599-1605.

Camerer, Colin, George Loewenstein, and Drazen Prelec. 2005. "Neuroeconomics: How Neuroscience Can Inform Economics" *Journal of Economic Literature* 43(1), March, 9-64.

Cawley, John and Barrett Kirwan. Forthcoming. "Pour Some Sugar on Me: The Contribution of U.S. Agricultural Policy to Obesity" in John Cawley (ed.) *Handbook of the Social Science of Obesity*. New York: Oxford University Press.

Cawley John and Richard V. Burkhauser. 2008. "Beyond BMI: The Value of More Accurate Measures of Fatness and Obesity in Social Science Research" *Journal of Health Economics* 27(2), March, 519-529.

Centers for Disease Control and Prevention. 2004. "Trends in Intake of Energy and Macronutrients – United States, 1971-2000. *Morbidity and Mortality Weekly Report* 53(4), Feburary 6, 80-82.

Chou, Shin-Yi, Michael Grossman, and Henry Saffer. 2004. "An Economic Analysis of Adult Obesity: Results from the Behavior Risk Factor Surveillance System" *Journal of Health Economics* 23(3), May, 565-587.

Chou, Shin-Yi, Michael Grossman, and Henry Saffer. 2006. "Reply to Jonathan Gruber and Michael Frakes" *Journal of Health Economics* 25(2), March, 389-393.

Chouinard, Hayley H., David E. Davis, Jeffrey T. LaFrance and Jeffrey Perloff. 2007. "Fat Taxes: Big Money for Small Change" *Forum for Health Economics and Policy (Obesity)* 10(2), Article 2.

Council of Economic Advisers. 2009. *Economic Report of the President, 2009*. Washington DC: U.S. Government Printing Office.

Courtemanche, Charles. 2009. "Rising Cigarette Prices and Rising Obesity: Coincidence or Unintended Consequence" *Journal of Health Economics* 28(4), July, 781-798.

Cox, Brian D., and Margaret J. Whichelow. 1996. "Ratio of Waist Circumference to Height is Better Predictor of Death than Body Mass Index" *British Medical Journal* 313(7070), December 17, 1487-1488

Christian, Thomas and Inas Rashad. 2009. “Trends in U.S. Food Prices, 1950-2007” *Economics and Human Biology* 7(1), March, 113-120.

Cutler, David M., Edward L. Glaeser and Jesse M. Shapiro. 2003. “Why Have Americans Become more Obese?” *Journal of Economic Perspectives* 17(3), Summer, 93-118.

Dalton, M., A. J. Cameron, P. Z. Zimmet, J. E. Shaw, D. Jolley, D. W. Dunstan, et al. 2003. “Waist Circumference, Waist-Hip Ratio and Body Mass Index and Their Correlation with Cardiovascular Disease Risk Factors in Australian Adults” *Journal of Internal Medicine* 254(6), December, 555-563.

Downs, Julie S., George Lowenstein and Jessica Wisdom. 2009. “Strategies for Promoting Healthier Food Choices” *American Economic Review: Papers and Proceedings* 99(2), May, 159-164.

Drewnowski, Adam. 1995. “Energy Intake and Sensory Properties of Food” *American Journal of Clinical Nutrition* 62(5), November, 1081S-1085S.

Epstein, Seymour. 2003. “Cognitive-Experiential Self-Theory, An Integrative, Psychodynamic Theory of Personality” *Bulletin of the American Academy of Clinical Psychology* 9(1), Summer, 5-10.

Finkelstein, Eric A., Ian C. Fiebelkorn, and Guijing Wang. 2003. “National Medical Spending Attributable to Overweight and Obesity: How Much, and Who’s Paying” *Health Affairs* May 14; web exclusive, w3.219-w3.226.

Flegal, Katherine M., Margaret D. Carroll, Cynthia L. Ogden, and Clifford L. Johnson. 2002. “Prevalence and Trends in Obesity Among US Adults, 1999-2000” *Journal of the American Medical Association* 288(14), October 9, 1723-1727.

Flegal Katherine M., Barry I. Graubard, David F. Williamson, and Mitchell H. Gail. 2005. “Excess Deaths Associated with Underweight, Overweight, and Obesity” *JAMA* 293(15), April 20, 1861-1867.

Flegal Katherine M., Barry I. Graubard, David F. Williamson, and Mitchell H. Gail. 2007. “Cause-Specific Excess Deaths Associated with Underweight, Overweight, and Obesity” *JAMA* 298(17), November 7, 2028-2037.

Fontaine, Kevin R., David T. Redden, Chenxi Wang, Andrew O. Westfall, and David B. Allison. 2003. “Years of Life Lost Due to Obesity” *JAMA* 289(2), January 8, 187-193.

Frankenfield, David, Lori Roth-Yousey, Charlene Compher, for the Analysis Working Group. 2005. “Comparison of Predictive Equations for Resting Metabolic Rate in Healthy Nonobese and Obese Adults: A Systematic Review” *Journal of the American Dietetic Association* 105(5), May, 775-789.

Franks, Paul W., Robert L. Hanson, William C. Knowler et al. 2010. “Childhood Obesity, Other Cardiovascular Risk Factors, and Premature Death” *New England Journal of Medicine* 362(6), February 11, 485-493.

Fudenberg, Drew and David K. Levin. 2006. “A Dual-Self Model of Impulse Control” *American Economic Review* 96(5), December, 1449-1476.

Glimcher, Paul William, Joseph Kable and Kenway Louie. 2007. “Neuroeconomic Studies of Impulsivity: Now or Just as Soon as Possible” *American Economic Review* 97(2), May, 142-147.

Goldman, Dana, Darius Lakdawalla, and Yuhui Zheng. 2009. “Food Prices and the Dynamics of Body Weight” *National Bureau of Economic Research Working Paper No. 15096*, June.

Gregg, Edward W., Yiling J. Cheng, Betsy L. Cadwell, Giuseppina Imperatore, Desmond E. Williams, Katherine M. Flegal, et al. 2005. “Secular Trends in Cardiovascular Disease Risk Factors According to Body Mass Index in US Adults” *Journal of the Medical Association* 293(15), April 20, 1868-1874.

Gruber, Jonathan and Michael Frakes. 2006. “Does Falling Smoking Lead to Rising Obesity” *Journal of Health Economics* 25(2), March 183-197.

Gruber, Jonathan and Botond Köszegi. 2001. “Is Addiction “Rational”? Theory and Evidence” *Quarterly Journal of Economics* 116(4), November, 1261-1303.

Gul, Faruk and Wolfgang Pesendorfer. 2001. “Temptation and Self-Control” *Econometrica* 69(6), November, 1403-1435.

Hamermesh, Daniel S. 2010. “Incentives, Time Use and BMI: The Roles of Eating, Grazing and Goods” *Economics and Human Biology*, 8(1), March, 2-15.

Heitmann, B.L., L. Lissner and M. Osler. 2000. “Do We Eat Less Fat, or Just Report So” *International Journal of Obesity*, 24(4), April, 435-442.

Henry, CJK. 2005. “Basal Metabolic Rate Studies in Humans: Measurement and Development of New Equations” *Public Health Nutrition* 8(7A), October, 1133-1152.

Hill, R.J. and P.S. W. Davies. 2001. “The Validity of Self-Reported Energy Intake as Determined by Using the Doubly Labelled Water Technique” *British Journal of Nutrition* 85(4), April, 415-430.

International Obesity Task Force. 2005. *EU Platform on Diet, Physical Activity and Health*. Brussels, International Obesity Task Force.

Jacobson, Michael F. and Kelly D. Brownell. 2000. “Small Taxes on Soft Drinks and Snack Foods to Promote Health” *American Journal of Public Health* 90(6), June, 854-857.

Just, David R. 2006. “Behavioral Economics, Food Assistance and Obesity” *Agricultural and Resource Economics Review* 35(2), October, 209-220.

Kahneman, Daniel. 1994. “New Challenges to the Rationality Assumption” *Journal of Institutional and Theoretical Economics* 150(1), 18-36.

Kessler, David A. 2009. *The End of Overeating*. New York: Rodale.

Lafay, L., L. Mennen, A. Basdevant, M.A. Charles, J.M. Borys, E. Eschwège, M. Romon and the FLVS Study Group. 2000. Does Energy Intake Underreporting Involve All Kinds of Food or Only Specific Food Items? Results from the Fleurbaix Laventie Ville Santè (FLVS) Study” *International Journal of Obesity*, 24(11), November, 1500-1507.

Laibson, David. 1997. “Golden Eggs and Hyperbolic Discounting” *Quarterly Journal of Economics* 112(2), 443-477.

Laibson, David. 2001. “A Cue-Theory of Consumption” *Quarterly Journal of Economics* 116(1), February, 81-119.

Lakdawalla, Darius and Tomas Philipson. 2009. “The Growth of Obesity and Technological Change” *Economics and Human Biology* 7(3), December, 283-293.

Lambert, Kelly Gurley, Tara Neal, Jill Noyes, Conway Parker and Pamela Worrel. 1991. “Food-Related Stimuli Increase Desire to Eat in Hungry and Satiated Human Subjects” *Current Psychology: Research & Reviews* 10(4), December, 297-303.

Loewenstein, George F. and O'Donoghue, Ted. 2004. “Animal Spirits: Affective and Deliberative Processes in Economic Behavior” Working Paper 04-14, Center for Analytic Economics, Cornell University.

Lowenstein, George F., Troyen Brennan and Kevin G. Volpp. 2007. “Asymmetric Paternalism to Improve Health Behaviors” *Journal of the American Medical Association* 298(20), November 28, 2415-2417.

MacLean, Paul D. 1973. *A Triune Concept of the Brain and Behavior*. Toronto, Ontario: University of Toronto Press.

MacLean, Paul D. 1990. *The Triune Brain in Evolution: Role in Paleocerebral Functions*. New York: Plenum.

Mancino, Lisa and Jean Kinsey. 2008. *Is Dietary Knowledge Enough? Hunger, Stress, and Other Roadblocks to Healthy Eating*, ERR-62, U.S. Department of Agriculture, Economic Research Service, August.

MarketData Enterprises. 2009. *The Weight Loss & Diet Control Market (10th Edition)*. Farmington, CT: MarketData Enterprises, Inc.

Massey, Douglas S. 2002. "A Brief History of Human Society: The Origin and Role of Emotion in Social Life" *American Sociological Review* 67(1), February, 1-29.

McClure, Samuel M., David I. Laibson, Goerge Loewenstein and Jonathan D. Cohen. 2004. "Separate Neural Systems Value Immediate and Delayed Monetary Rewards" *Science* 306, October 15, 503-507.

Melki, I.S., H.A. Beydoun, M. Khogali, H. Taminm, K.A. Yunis, for the National Collaborative Perinatal Neonatal Network. 2004. "Household Crowding Index: A Correlate of Socioeconomic Status and Inter-Pregnancy Spacing in an Urban Setting" *Journal of Epidemiology and Community Health* 58(6), June, 476-480.

Metcalfe, Janet and Walter Mischel. 1999. "A Hot/Cool – System Analysis of Delay of Gratification: Dynamics of Willpower" *Psychological Review* 106(1), 3-19.

Mokdad, Ali H., Barbara A. Bowman, Earl S. Ford, Frank Vinicor, James S. Marks, and Jeffrey P. Koplan. 2001. "The Continuing Epidemic of Obesity and Diabetes in the United States" *JAMA* 286(10), September 21, 1195-1200.

Must, Aviva, Jennifer Spadano, Eugenie H. Coakley, Alison E. Field, Graham Colditz, and William H. Dietz. 1999. "The Disease Burden Associated With Overweight and Obesity" *JAMA* 282(16), October 27, 1523-1529.

National Center for Health Statistics. 1981. *Public Use Data Tape Documentation, Physical Measurements 1 Tape Number 1003, National Health Examination Survey, 1959-1962*, May; accessed from, http,//www.cdc.gov/nchs/data/nhanes/nhes123/1003.pdf.

National Heart, Lung, and Blood Institute. 1998. *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report*. Washington D.C.: US Government Printing Office.

National Institute of Diabetes and Digestive and Kidney Diseases. 2008. "Longitudinal Assessment of Bariatric Surgery (LABS)", *NIH publication No. 04-5573*, November.

Okoro, Catherine A., Jennifer M. Hootman, Tara W. Strine, Lina S. Balluz and Ali H. Mokdad. 2004. Disability, Arthritis, and Body Weight Among Adults 45 Years and Older. *Obesity* 12(5), May, 854-861.

Olshansky S. Jay, Douglas J. Passaro, Ronald C. Hershow, Jennifer Layden, Bruce A. Carnes, Jacob Brody J, et al. 2005. "A Potential Decline in Life Expectancy in the United States in the 21st Century" *New England Journal of Medicine*, 352(11), March 17, 1138-1145.

Ozdenoren, Emre, Stephen Salant, and Dan Silverman. 2006. "Willpower and the Optimal Control of Visceral Urges" *National Bureau of Economic Research Working Paper No. 12278*, May.

Patton, Paul. 2008. "One World, Many Minds: Intelligence in the Animal Kingdom" *Scientific American Mind*, 19(6), December, 72-79.

Philipson, Tomas J. and Richard A. Posner. 2003. “The Long-Run Growth in Obesity as a Function of Technological Change” *Prospectives in Biology and Medicine* 46(3), Summer, S87-S107.

Pi-Sunyer, Xavier. 2003. “A Clinical View of the Obesity Problem” *Science* 299(5608), February 7, 859-860.

Pollan, Michael. 2008. *In Defense of Food*. New York: Penguin Books.

Popkin, Barry M., Anna Maria Siega-Riz, Pamela S. Haines, and Lisa Jahns. 2001. “Where’s the Fat? Trends in U.S. Diets 1965-1996” *Preventive Medicine*, 32(3), March, 245-254.

Ramey, Valerie. 2007. “How Much Has Leisure Really Increased Since 1965”, mimeo, University of California at San Diego, January.

Rodgers Peter J. and Andrew J. Hill. 1989. “Breakdown of Deitary Restraint Following Mere Exposure to Food Stimuli: Interrelatinships Between Restraint, Hunger, Salivation, and Food Intake” *Addictive Behaviors* 14(4), 387-397.

Ruhm, Christopher J. 2007. “Current and Future Prevalence of Obesity and Severe Obesity in the United States” *Forum for Health Economics and Policy (Obesity)* 10(2), Article 6, 1-22.

Sheffrin, Hersh M. and Richard H. Thaler. 1988. “The Behavioral Life-Cycle Hypothesis” *Economic Inquiry* 26(4), October, 609-643.

Shiv, Baba and Alexander Fedorkikhin. 1999. “Heart and Mind in Conflict: The Interplay of Affect and Cognition in Consumer Decision” *The Journal of Consumer Research* 26(3), December, 278-292.

Sönmez, K., M. Akcakoyun, A. Akcay, D. Demir, N. E. Duran, M. Gencbay et al. 2003. “Which Method Should be Used to Determine the Obesity, in Patients with Coronary Artery Disease? (Body Mass Index, Waist Circumference, or Waist-Hip Ratio)” *International Journal of Obesity* 27(3), March, 341-346.

Staddon, J.E.R. 1975. “A note on the Evolutionary Significance of ‘Supernormal’ Stimuli” *The American Naturalist* 109(969), September-October, 541-545.

Thaler, Richard H. and Cass R. Sunstein. 2009. *Nudge*. New York: Penguin Books.

Thompson, David, Jonathan B. Brown, Gregory A. Nichols, Patricia J. Elmer and Gerry Oster. 2001. “Body Mass Index and Future Healthcare Costs, A Retrospective Cohort Study” *Obesity* 9(3), March, 210–218.

Wallinga, David, Heather Schoonover and Mark Muller. 2009. “Considering the Contribution of US Agricultural Policy to the Obesity Epidemic: Overview and Implications” *Journal of Hunger and Nutrition* 4(1), January, 3-19.

Ward, Andrew and Traci Mann. 2000. “Don’t Mind if I Do: Disinhibited Eating Under Cognitive Load” *Journal of Personality and Social Psychology* 78(4), 753-763.

Wansink, Brian. 2006. *Mindless Eating*. New York: Bantam Dell.

Wells, Hodan Farah and Jean C. Buzby. 2008. *Dietary Assessment of Major Trends In U.S. Food Consumption, 1970-2005*, Economic Information Bulletin No. 33. Economic Research Service, U.S. Department of Agriculture, March.

Woods, Stephen C. 1991. “The Eating Paradox: How We Tolerate Food” *Psychological Review* 98(4), October, 488-505.

World Health Organization. 1997. Obesity: *Preventing and Managing the Global epidemic. Report of a WHO Consultation on Obesity*. Geneva, Switzerland: World Health Organization.

Table 1. Weight Perceptions, Preferences and Weight Loss Attempts of 25-60 Year Olds, By Weight Class and Sex, 1999-2006

	Full Sample	Underweight (BMI<18.5)	Healthy Weight (BMI: 18.5-24.9)	Overweight (BMI: 25.0-29.9)	Mild Obesity (BMI: 30.0-34.9)	Severe Obesity (BMI ≥ 35.0)
Males						
Weight Self-Perception						
Underweight	6.0 (0.4)	73.6 (7.4)	17.0 (1.1)	1.5 (0.3)	0.5 (0.3)	0.0 (0.0)
About Right	42.6 (0.9)	26.4 (7.4)	74.5 (1.3)	45.8 (1.2)	14.0 (1.2)	4.6 (1.0)
Overweight	51.4 (1.0)	0.0 (0.0)	8.5 (0.9)	52.7 (1.3)	85.5 (1.2)	95.4 (1.0)
Preferred Weight						
More	8.9 (0.5)	83.4 (5.6)	24.9 (1.3)	3.0 (0.4)	0.8 (0.3)	0.0 (0.0)
About the Same	33.4 (0.9)	16.6 (5.6)	60.9 (1.5)	34.9 (1.2)	10.3 (1.1)	3.3 (0.9)
Less	57.6 (0.9)	0.0 (0.0)	14.2 (1.2)	62.1 (1.3)	88.9 (1.1)	96.7 (0.9)
Attempted Weight Loss	35.9 (0.9)	0.0 (0.0)	11.5 (0.9)	38.5 (1.5)	50.7 (1.8)	63.0 (2.0)
Population Share	100.0	0.9	27.2	41.0	20.3	10.7
Females						
Weight Self-Perception						
Underweight	2.5 (0.2)	58.0 (5.7)	3.3 (0.4)	0.2 (0.1)	0.2 (0.1)	0.0 (0.0)
About Right	28.0 (0.9)	40.6 (5.9)	60.2 (1.3)	17.7 (1.3)	4.0 (0.5)	1.4 (0.4)
Overweight	69.5 (0.9)	1.4 (1.4)	36.5 (1.3)	82.0 (1.3)	95.8 (0.5)	98.6 (0.4)
Preferred Weight						
More	2.6 (0.3)	51.1 (6.1)	3.7 (0.5)	0.4 (1.5)	0.1 (0.1)	0.0 (0.0)
About the Same	19.7 (0.8)	47.4 (6.4)	41.9 (1.4)	11.0 (1.0)	3.1 (0.5)	1.6 (0.5)
Less	77.7 (0.8)	1.4 (1.4)	54.4 (1.5)	88.6 (1.0)	96.9 (0.5)	98.4 (0.5)
Attempted Weight Loss	56.1 (1.0)	6.9 (2.9)	39.1 (1.7)	64.1 (1.4)	69.1 (1.9)	71.5 (1.7)
Population Share	100.0	2.2	35.7	24.4	17.8	17.9

Note: Data are from NHANES 99. Table shows percentages, with observations weighted so as to be nationally representative. Robust standard errors, clustered by NHANES survey, PSU, and strata, are shown in parentheses. Body Mass Index (BMI) is weight in kilograms divided by height in meters squared. Attempted weight loss refers to the last 12 months. The male sample size is 5,078 consisting of 51, 1,362, 2,100, 1,022 and 543 underweight, healthy weight, overweight, mildly obese and severely obese individuals. Corresponding sample sizes for females are 4,946, 89, 1,522, 1,385, 983 and 967.

Table 2. Trends in BMI at Different Points in the Distribution by Sex, 25-60 Year Olds

Percentile	Baseline BMI (1976-80)	Change in BMI Since Baseline 1988-94 (a)	1988-94 (b)	1999-2006 (a)	1999-2006 (b)
Males					
10	21.2	0.60 (0.15)	0.33 (0.12)	1.00 (0.13)	0.80 (0.11)
25	23.1	0.70 (0.15)	0.55 (0.14)	1.50 (0.13)	1.34 (0.12)
50	25.4	0.60 (0.18)	0.66 (0.12)	2.10 (0.15)	1.97 (0.10)
75	27.9	1.20 (0.23)	1.08 (0.21)	3.10 (0.19)	2.90 (0.19)
90	30.7	2.00 (0.30)	1.90 (0.32)	4.50 (0.27)	4.30 (0.28)
95	33.3	2.40 (0.64)	2.34 (0.54)	5.50 (0.54)	5.41 (0.48)
Females					
10	19.5	0.40 (0.15)	0.37 (0.12)	1.20 (0.14)	0.84 (0.11)
25	21.3	0.50 (0.15)	0.61 (0.13)	1.80 (0.13)	1.62 (0.13)
50	23.9	1.20 (0.17)	1.35 (0.20)	3.10 (0.15)	2.92 (0.19)
75	28.0	2.30 (0.35)	2.32 (0.29)	4.90 (0.32)	4.75 (0.27)
90	33.1	2.80 (0.42)	3.04 (0.49)	5.50 (0.39)	5.74 (0.45)
95	36.5	2.90 (0.62)	2.60 (0.63)	5.90 (0.55)	5.51 (0.57)
Additional Controls		No	Yes	No	Yes

Note: Table shows the predicted difference in BMI, at specified percentiles of the distribution, relative to the 1976-80 baseline. These estimates are obtained using quantile regression. Observations are weighted with robust standard errors, clustered by survey, psu and strata, shown in parentheses. The "additional controls" in specification (b) include: age, age squared, sex, race/ethnicity (3 variables), education (3 variables), marital status (3 variables), and tobacco use (2 variables). Sample sizes are 12,595 for males and 13,357 for females.

Table 3. Weight Gain of 25-60 Year Olds During Last Year By Weight Class and Sex, 1999-2006

Weight Gain	Full Sample	Healthy Weight	Overweight	Mild Obesity	Severe Obesity
Males					
Any Gain	31.6	26.2	31.5	36.8	37.7
	(0.8)	(1.4)	(1.3)	(2.0)	(2.2)
≥5 lbs	28.2	21.9	27.9	34.2	35.8
	(0.8)	(1.3)	(1.3)	(1.9)	(2.3)
≥10 lbs	17.9	10.7	16.2	24.7	31.1
	(0.7)	(0.8)	(1.0)	(1.7)	(2.3)
≥5%	27.6	23.4	28.2	30.4	32.8
	(0.8)	(1.3)	(1.3)	(1.7)	(2.4)
≥10%	17.8	13.1	17.2	22.1	24.9
	(0.7)	(0.9)	(1.1)	(1.6)	(2.3)
Females					
Any Gain	38.6	34.0	41.8	45.5	39.1
	(0.9)	(1.4)	(1.4)	(1.8)	(1.8)
≥5 lbs	34.4	26.8	38.7	42.3	38.6
	(0.9)	(1.3)	(1.3)	(1.7)	(1.8)
≥10 lbs	24.3	13.8	28.0	34.0	33.3
	(0.8)	(1.0)	(1.3)	(1.7)	(1.8)
≥5%	36.0	30.6	39.7	42.7	37.6
	(0.9)	(1.5)	(1.4)	(1.7)	(1.8)
≥10%	27.3	19.9	31.4	35.8	30.3
	(0.8)	(1.2)	(1.2)	(1.7)	(1.7)

Note: Data are from NHANES 99. Weight changes are measured over a one year period. Table shows percentages, with observations weighted so as to be nationally representative. Robust standard errors, clustered by NHANES survey, PSU, and strata, are shown in parentheses. The sample includes 4,984 males and 4,818 females.

Table 4. Econometric Estimates of Weight Gain of 25-60 Year Olds During Last Year By Weight Class and Sex, 1999-2006

Weight Class	Unconditional				Conditional on Some Weight Gain		
	Any Gain	≥5 lbs	≥10 lbs	≥10%	≥5 lbs	≥10 lbs	≥10%
Males							
Severe Obesity	.127	.156	.217	.134	.152	.451	.198
	(.028)	(.028)	(.029)	(.029)	(.019)	(.039)	(.052)
Mild Obesity	.120	.140	.154	.107	.129	.305	.151
	(.028)	(.027)	(.023)	(.022)	(.019)	(.035)	(.039)
Overweight	.060	.070	.063	.051	.077	.140	.083
	(.021)	(.019)	(.015)	(.016)	(.025)	(.039)	(.042)
Baseline	.256	.208	.102	.126	.791	.383	.477
Females							
Severe Obesity	.050	.115	.189	.102	.206	.438	.195
	(.023)	(.023)	(.021)	(.021)	(.011)	(.024)	(.028)
Mild Obesity	.113	.152	.196	.158	.146	.333	.207
	(.023)	(.022)	(.021)	(.021)	(.020)	(.035)	(.033)
Overweight	.074	.115	.138	.113	.147	.271	.179
	(.019)	(.019)	(.018)	(.018)	(.015)	(.029)	(.027)
Baseline	.358	.286	.154	.216	.775	.410	.581

Note: Data are from NHANES 99. Table displays average marginal effects, compared to healthy weight individuals and obtained from probit models that also control for a quadratic in age, race/ethnicity, marital status, and tobacco use. Observations are weighted and robust standard errors, clustered by NHANES survey, PSU, and strata, are shown in parentheses. The "baseline" results indicate average predicted values for the counterfactual where individuals retain their own values of the supplementary covariates but are in the healthy weight range.

Table 5. Weight Change of 25-60 Year Olds by Weight Class Last Year, Weight Loss Attempts and Sex, 1999-2006

Weight Class Last Year	Males: No Weight Loss Attempt (a)	Males: Weight Loss Attempted (b)	Females: No Weight Loss Attempt (a)	Females: Weight Loss Attempted (b)
Severe Obesity	-2.10	-11.49	-0.14	-8.13
	(1.49)	(1.76)	(1.54)	(1.44)
Mild Obesity	1.11	-5.69	2.23	-0.38
	(1.04)	(1.18)	(1.43)	(1.20)
Overweight	1.66	0.19	3.40	2.63
	(0.86)	(0.95)	(1.15)	(1.08)
Healthy Weight	2.70	4.08	3.76	5.95
	(0.86)	(1.29)	(0.98)	(1.04)
Full Sample	1.87	-3.86	3.02	0.25
	(0.83)	(0.95)	(1.00)	(1.01)

Note: Data are from NHANES 99. Weight changes are measured over a one year period. Table shows predicted change (in pounds) for persons in the specified weight class and weight loss attempt status. Predictions are obtained from regressions that contain a full set of weight class and weight loss attempt variables as well as controls for a quadratic in age, race/ethnicity, marital status, and tobacco use. The top and bottom panels refer to results from separate regression models. Observations are weighted and robust standard errors, clustered by NHANES survey, PSU, and strata, are shown in parentheses. The sample includes 4,984 males and 4,818 females.

Table 6. Econometric Estimates of Food Consumption of 25-60 Year Olds by Weight Class and Sex, 1999-2006

Weight Class	% of Calories From		Sodium Intake (mg/kcal)
	Fat	Saturated Fat	
Males			
Severe Obesity	3.42 (0.52)	1.42 (0.21)	0.081 (0.030)
Mild Obesity	2.26 (0.50)	0.97 (0.19)	0.078 (0.027)
Overweight	0.46 (0.37)	0.30 (0.15)	-0.012 (0.022)
Baseline	31.5	10.0	1.53
Females			
Severe Obesity	1.78 (0.51)	0.85 (0.24)	0.103 (0.030)
Mild Obesity	1.73 (0.50)	0.66 (0.20)	0.058 (0.028)
Overweight	0.84 (0.47)	0.35 (0.19)	0.059 (0.24)
Baseline	32.3	10.5	1.55

Note: Data are from NHANES 99. The dependent variables refer to food consumption during the 24-hour observation period. Table displays regression estimates of predicted differences, compared to healthy weight individuals, for models that also control for a quadratic in age, race/ethnicity, marital status, tobacco use, and the day of the week during which food intake is measured. Observations are weighted and robust standard errors, clustered by NHANES survey, PSU, and strata, are shown in parentheses. The "baseline" results indicate the average predicted value for the counterfactual where individuals retain their own values of the supplementary covariates but are in the healthy weight range

Table 7. Subgroup Regression Estimates, 25-60 Year Old Males

Group	Change in BMI				Δ in Weight Loss Attempts	Food Intake Ratio: 35 kg/m² vs. 20 kg/m²			
	50th Percentile		90th Percentile			% Calories from Fat		Sodium Consumption (mg/kcal)	
	1988-94	1999-2006	1988-94	1999-2006		1976-80	1999-2006	1976-80	1999-2006
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)	(i)
Full Sample	0.66	1.97	1.90	4.30	.055	1.008	1.098	1.006	1.065
	(0.12)	(0.10)	(0.32)	(0.28)	(.006)	[.874]	[.000]	[.950]	[.001]
	{25.4}		*{30.7}*		*{.296}*				
25 - 42 Year Olds	0.44	1.77	1.38	3.96	.048	0.998	1.093	0.968	1.045
	(0.19)	(0.17)	(0.42)	(0.38)	(.005)	[.281]	[.000]	[.493]	[.003]
	{25.0}		*{30.4}*		*{.302}*				
43 - 60 Year Olds	1.10	2.30	2.44	4.47	.060	1.010	1.100	1.066	1.088
	(0.20)	(0.18)	(0.41)	(0.37)	(.010)	[.267]	[.000]	[.442]	[.019]
	{25.9}		*{31.1}*		*{.280}*				
No College	0.91	2.00	1.89	3.99	.038	1.021	1.085	1.058	1.059
	(0.21)	(0.19)	(0.41)	(0.37)	(.007)	[.937]	[.000]	[.706]	[.006]
	{25.8}		*{31.5}*		*{.290}*				
College Educated	0.52	1.92	1.69	4.12	.067	0.971	1.123	0.938	1.080
	(0.22)	(0.18)	(0.38)	(0.31)	(.012)	[.134]	[.000]	[.557]	[.013]
	{24.9}		*{29.8}*		*{.299}*				

Note: Columns (a) through (d) displays changes in the 50th and 90th percentiles of predicted BMI, relative to the 1976-80 baseline, using the same procedures as in Table 2. Column (e) shows the predicted change in weight loss attempts in 2000/03 versus 1991/94, without conditioning on BMI. Columns (f) through (j) show the predicted ratio of the percent of calories from fat or sodium intake (mg/kcal) for a BMI of 35 relative to 20, using the same methods as in figure 4. Observations are weighted and robust standard errors, clustered by NHANES survey, PSU, and strata, are shown in parentheses. Numbers in square brackets, in columns (f) through (i), show p-values for the null hypothesis that BMI has no effect on the dietary measure. Italicized numbers in curly brackets indicate baseline values for 1976-80 (columns a – d) or 1991/94 (column e). NHANES data are used, except in column (e), where data are from the BRFSS.

Table 8. Subgroup Regression Estimates, 25-60 Year Old Females

Group	Change in BMI: 50th Percentile 1988-94 (a)	Change in BMI: 50th Percentile 1999-2006 (b)	Change in BMI: 90th Percentile 1988-94 (c)	Change in BMI: 90th Percentile 1999-2006 (d)	Δ in Weight Loss Attempts (e)	Food Intake Ratio: 35 kg/m² vs. 20 kg/m²: % Calories from Fat 1976-80 (f)	% Calories from Fat 1999-2006 (g)	Sodium Consumption (mg/kcal) 1976-80 (h)	Sodium Consumption (mg/kcal) 1999-2006 (i)
Full Sample	1.35	2.92	3.04	5.74	.044	1.013	1.062	0.970	1.086
	(0.20)	(0.19)	(0.49)	(0.45)	(.005)	[.822]	[.000]	[.792]	[.000]
	{23.9}		*{33.1}*		*{.451}*				
25 - 42 Year Olds	1.00	2.71	2.93	5.37	.041	1.010	1.061	0.928	1.068
	(0.22)	(0.22)	(0.67)	(0.65)	(.006)	[.911]	[.001]	[.323]	[.032]
	{23.0}		*{32.3}*		*{.458}*				
43 - 60 Year Olds	1.87	3.12	2.23	5.57	.042	1.023	1.061	1.017	1.112
	(0.31)	(0.29)	(0.64)	(0.59)	(.006)	[.759]	[.002]	[.898]	[.000]
	{25.0}		*{33.8}*		*{.395}*				
No College	1.27	2.73	2.99	4.68	.030	1.014	1.023	0.934	1.077
	(0.25)	(0.25)	(0.56)	(0.56)	(.014)	[.868]	[.014]	[.163]	[.035]
	{24.5}		*{34.0}*		*{.478}*				
College Educated	1.40	3.09	2.67	6.44	.055	1.012	1.090	1.058	1.092
	(0.29)	(0.25)	(0.77)	(0.68)	(.011)	[.136]	[.001]	[.429]	[.001]
	{22.7}		*{30.2}*		*{.429}*				

Note: Columns (a) through (d) displays changes in the 50th and 90th percentiles of predicted BMI, relative to the 1976-80 baseline, using the same procedures as in Table 2. Column (e) shows the predicted change in weight loss attempts in 2000/03 versus 1991/94, without conditioning on BMI. Columns (f) through (j) show the predicted ratio of the percent of calories from fat or sodium intake (mg/kcal) for a BMI of 35 relative to 20, using the same methods as in figure 4. Observations are weighted and robust standard errors, clustered by NHANES survey, PSU, and strata, are shown in parentheses. Numbers in square brackets, in columns (f) through (i), show p-values for the null hypothesis that BMI has no effect on the dietary measure. Italicized numbers in curly brackets indicate baseline values for 1976-80 (columns a – d) or 1991/94 (column e). NHANES data are used, except in column (e), where data are from the BRFSS.

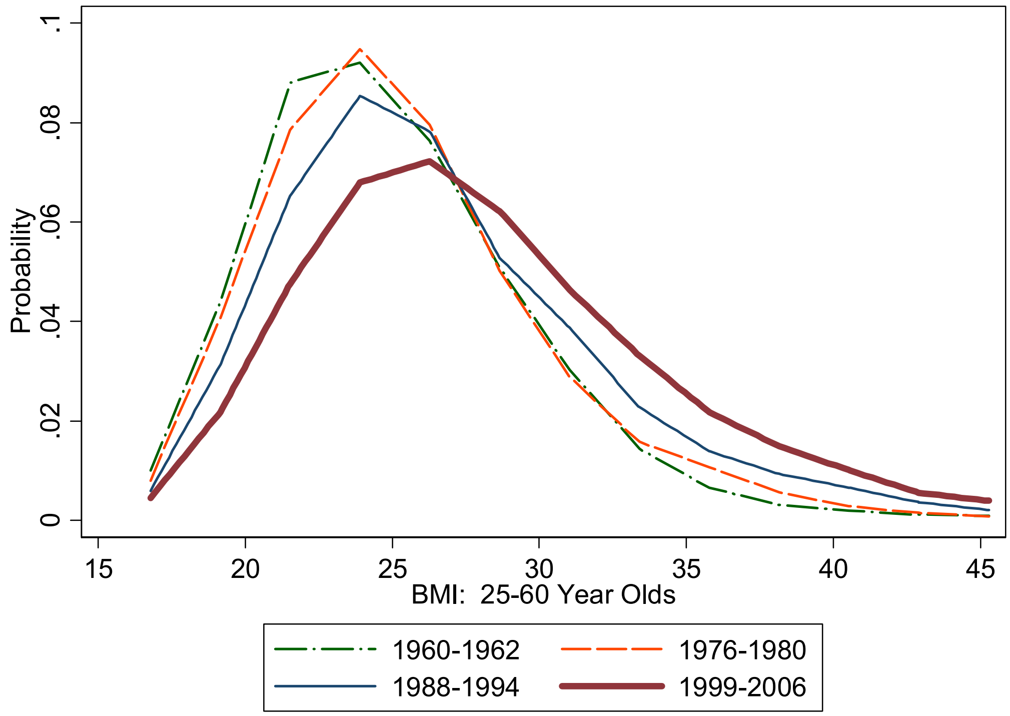

FIGURE 1. Trends in the Body Mass Index Distribution of 25-60 Year Olds

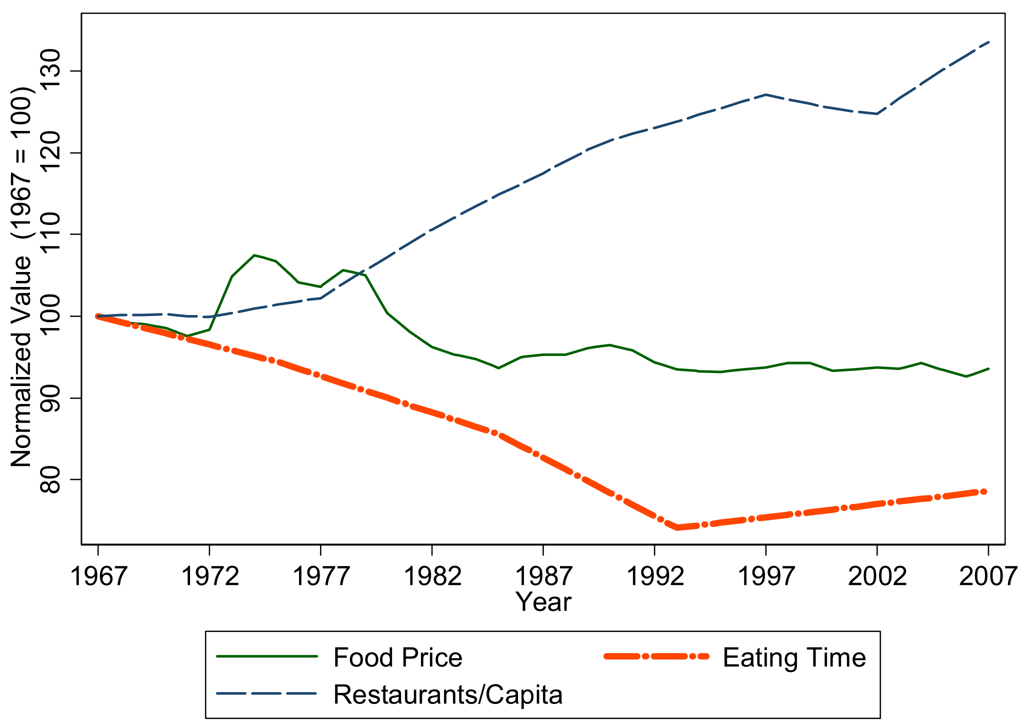

FIGURE 2. Trends in Food Prices, Eating Time and Number of Restaurants

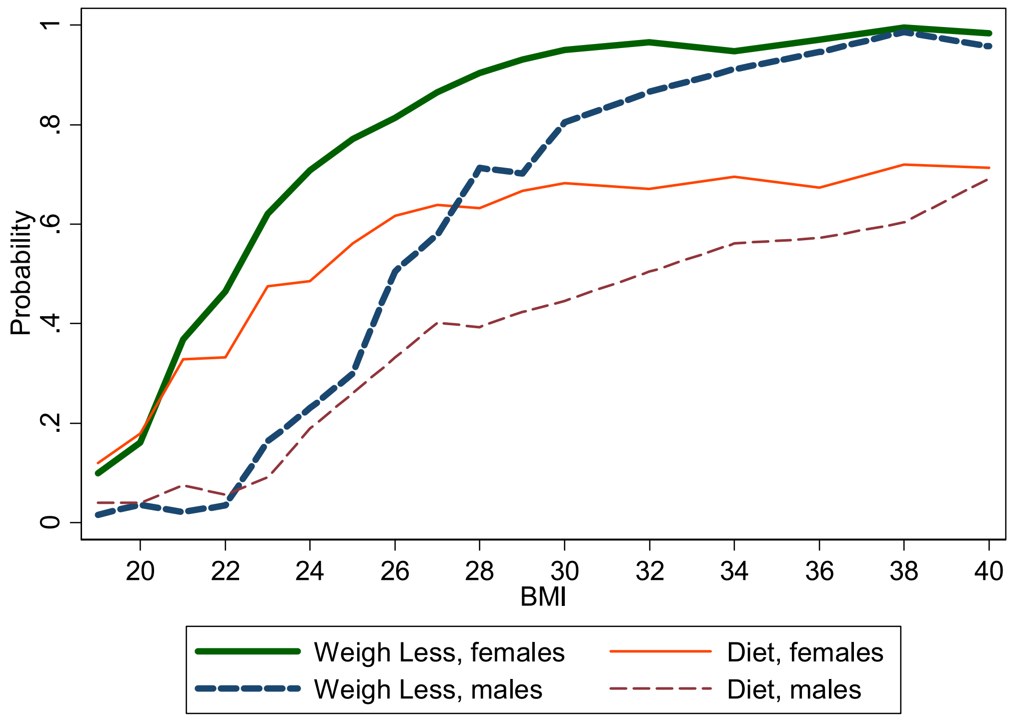

FIGURE 3A: Desired and Attempted Weight Loss By BMI and Sex, 1999-2006

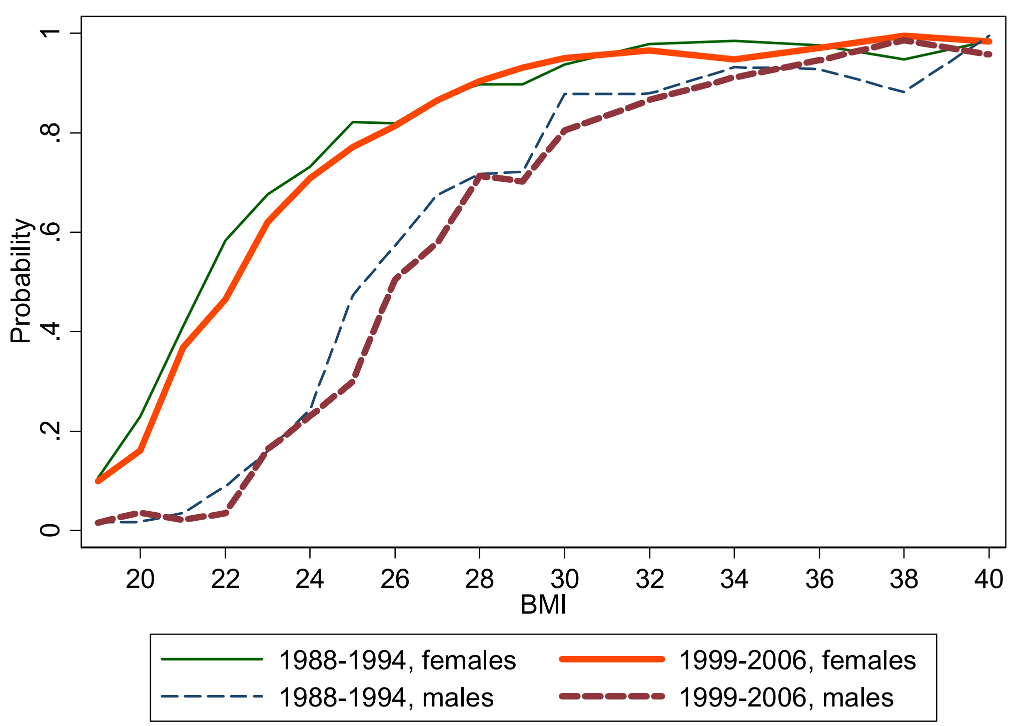

FIGURE 3B. Desired Weight Loss By BMI, Sex and Survey Year

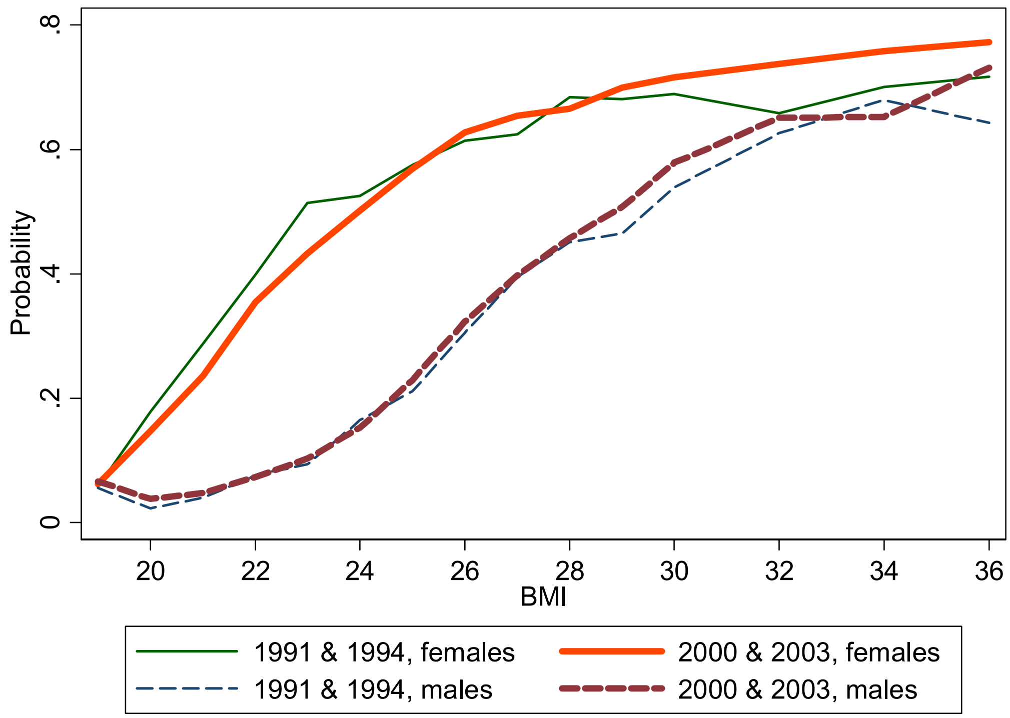

FIGURE 3C. Attempted Weight Loss By BMI, Sex and BRFSS Survey Year

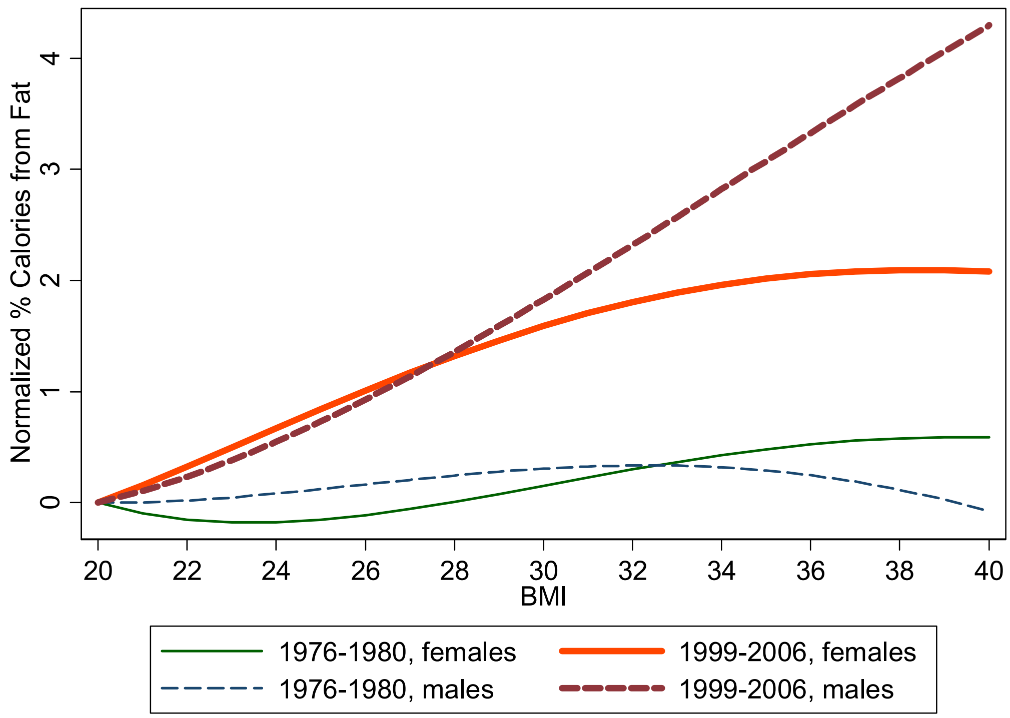

FIGURE 4A: Fat Consumption by BMI, Sex and Survey Year

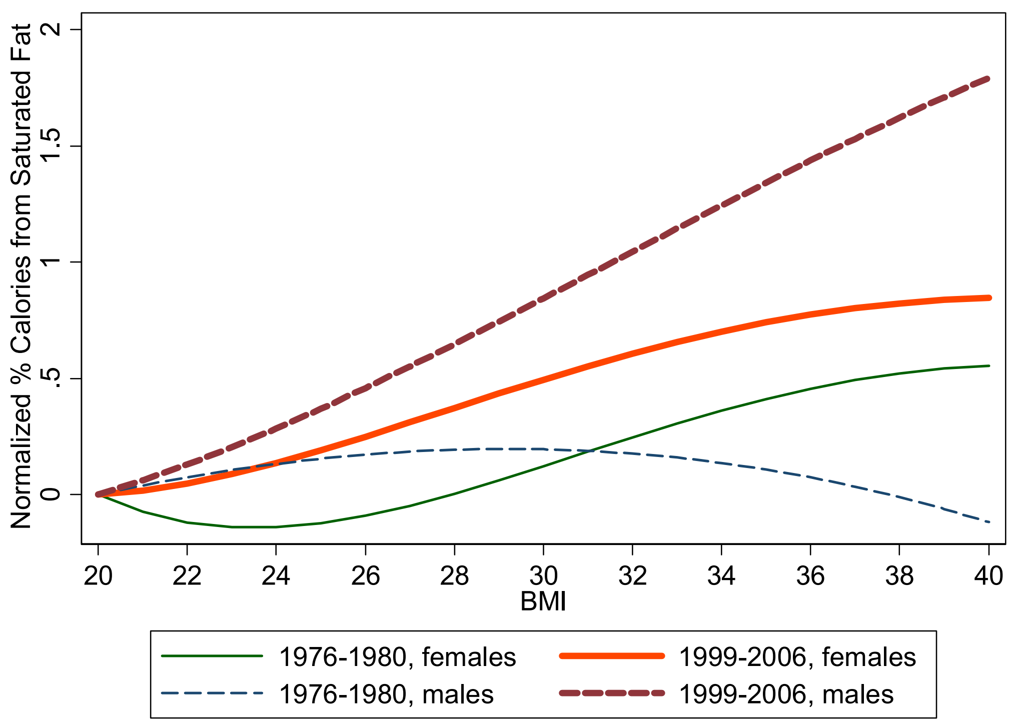

FIGURE 4B: Saturated Fat Consumption by BMI, Sex and Survey Year

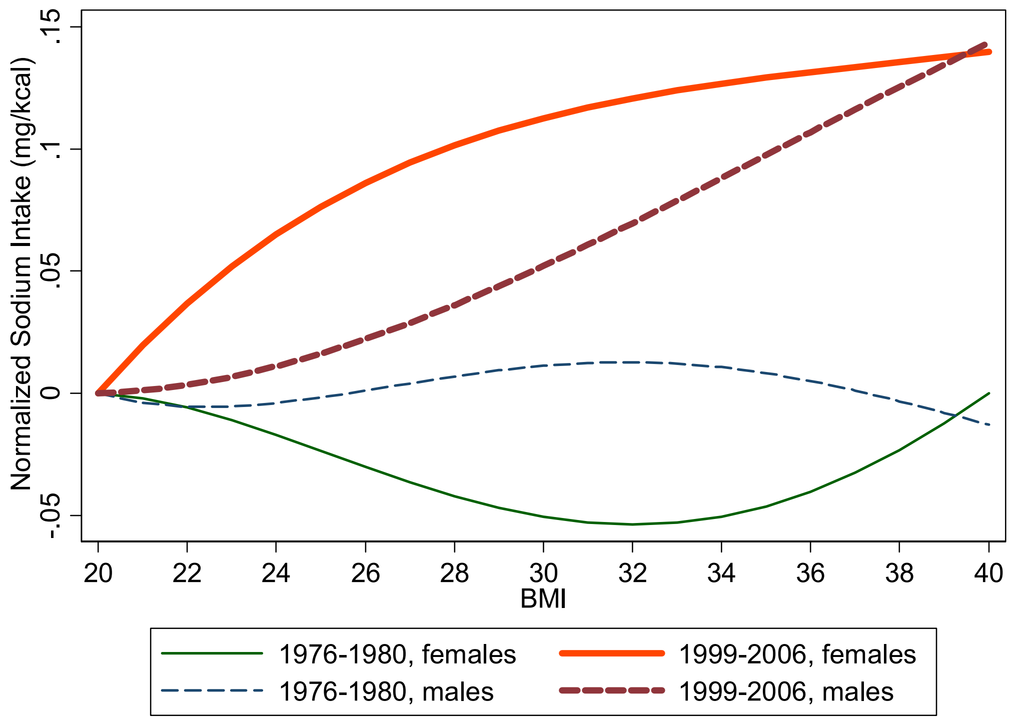

FIGURE 4C: Sodium Consumption by BMI, Sex and Survey Year