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Social Dynamics of Obesity

Mary Burke and Frank Heiland

Abstract:
In order to explain the substantial recent increases in obesity rates in the United States, we consider the effect of falling food prices in the context of a model involving endogenous body weight norms and an explicit, empirically grounded description of human metabolism. Unlike previous representative agent models of price-induced gains in average weight, our model, by including metabolic heterogeneity, is able to capture changes in additional features of the distribution, such as the dramatic growth in upper-quartile weights that are not readily inferred from the representative agent setting. We calibrate an analytical choice model to American women in the 30-to-60-year-old age bracket and compare the model's equilibrium weight distributions to data from NHANES surveys spanning (intermittently) the period from 1976 through 2000. The model predicts increases in average weight and obesity rates with considerable accuracy and captures a considerable portion of the relative growth in upper-quantile weights. The differential response to price declines across the distribution depends on the fact that human basal metabolism (or resting calorie expenditure) is increasing and yet concave in body weight, and therefore food price effects on weight tend to be larger for individuals who are heavier initially. The lagged adjustment of weight norms helps to explain recent observations that obesity rates have continued to rise since the mid 1990s, despite an apparent leveling off of price declines. The predicted increase in body weight aspirations agrees with an observed trend in self-reported desired weights, and it defies the conventional wisdom that thinness has been a growing obsession among American women in recent decades.

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Mary Burke is an Economist at the Federal Reserve Bank of Boston. Her e-mail address is: mary.burke@bos.frb.org. Frank Heiland is an Assistant Professor of Economics at Florida State University . His email address is fheiland@fsu.edu.

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1 Introduction

The startling growth rates of average weight and obesity prevalence in the United States over the past 20 to 30 years have received widespread media attention for several years running. Obesity has become an object of grave concern among public health officials and has spawned voluminous research in the fields of medicine, public health, and, increasingly of late, economics. Concern has focused on identifying the causes of obesity and on enumerating the costs of obesity-related morbidity and mortality, in both economic and human terms (Hassan et al. 2003, Himes 2000, Hamermesh and Biddle 1994, Cawley 2004, Pagan and Davila 1997).[1] Not only has the weight distribution in the United States made a considerable shift to the right—average adult female weight, for example, increased by 20 pounds, or 13.5 percent, between 1976–80 (NHANES II) and 1999–2000 (NHANES 99)—but the upper tail has experienced disproportionate growth: For women over the same time span, 95th-percentile weight grew 16.7 percent, from 215 to 251 pounds, and 99th-percentile weight increased 18.2 percent, from 258 to 305 pounds, as shown in Table 1 and Figure 1.[2,3] The official definition of obesity employed by the Centers for Disease Control (CDC) and by the World Health Organization (WHO) is a body mass index (BMI) value of 30 or greater, where BMI is the ratio of weight, measured in kilograms, to squared height, measured in meters. A 5'4" woman who weighs 175 pounds or more is classified as

[1]The level of alarm has become so pronounced as to have already spawned a backlash by players in the food industry (see www.consumerfreedom.com) seeking to forestall regulatory interventions and by scientists claiming that the health risks of overweight and obesity have been overstated (Oliver 2005). Some have also questioned the CDC's classification of obesity based on body mass index (BMI), but few have disputed the magnitude of the changes in the weight distribution.

[2]Changes of similar magnitude are observed in the BRFSS data between 1990 and 2002 as shown in Table 1. For men, 95th (99th) percentile weight increased from 230 (264) to 277 (338) pounds, and the average increased from 177 to 192 pounds between NHANES II and NHANES 99 as shown in Table 1.

[3]The empirical findings on body weight presented in this paper are based on samples of 30-to-60-year-old Americans from two surveys administered by the Centers for Disease Control and Prevention: The Behavioral Risk Factor Surveillance System (BRFSS) and waves II, III, and 99 of the National Health and Nutrition Examination Survey (NHANES II, III, and 99). The BRFSS is an exceptionally large random sample of the resident population 18 years and older in participating states of the U.S. Self-reported information on actual weight, desired weight, and demographic characteristics is gathered in cross-sections between 1990 and 2002 (1994–2002 for desired weight). We correct for potential bias of self-reported weights (see Villanueva 2001), following the approach of Chou et al. (2004), using NHANES III data for the 30-60-year olds. NHANES II, III, and 99 collect information from medical examinations on weight and health status of a cross-section of the U.S. population in 1976–1980, 1988–1994, and 1999–2000. Combining the data from these two sources allows us to track changes in the distribution of weight and BMI by demographic characteristics between 1976–80 and 2002.

obese, and the obesity threshold for a 5'9" man is 203 pounds.[4]

A number of papers in economics have sought to explain the increase in obesity among American adults over varying time spans of recent history. The explanations have focused on standard economic influences, such as falling food prices and preparation time costs as well as reductions in physical labor on the job (Chou et al. 2004; Cutler et al. 2003, henceforth CGS; Philipson and Posner 1999; Lakdawalla and Philipson 2002). The theoretical models offered study representative agents and speak primarily to secular trends in average weight. Although the model of CGS, which emphasizes self-control problems, can predict growth in upper-quantile weights relative to the mean, the prediction is sensitive to the empirical variation in self-control over food intake—variation that is neither well understood nor readily observed. The prior works abstract from genetic heterogeneity, known to be a major factor in weight variation (Cawley 1999, Chou et al. 2004) and either ignore or hold fixed the social influences on weight determination. In this paper, we argue that social and biological determinants of weight gain—interacted with falling food prices—contribute substantially to our understanding of the various changes in the weight distribution over the past 30 years.

In the choice model, utility depends on food and nonfood consumption, and on how individual weight compares with a social weight standard or norm—construed as the weight to which individuals aspire. Individuals differ in their respective genetic endowments of (resting) metabolic capacity, but they are otherwise identical. The social weight standard is endogenous, however, and depends on the aggregate behavior of the heterogeneous population. Through analytical results and calibrated simulations, we illustrate how food price declines affect the entire weight distribution, and we describe explicit adjustment dynamics across long-run equilibria. The metabolic model and simulations are calibrated to American women ages 30 to 60. This demographic restriction enhances the calibration's precision, because the physiological and social processes we consider are gender and age-group specific. Consistent

[4]BMI values between 18.5 and 24.9 are considered "healthy," BMI less than 18.5 is "underweight," and BMI between 25 and 29.9 is "overweight" but not obese. BMI thresholds of 35 and 40 are used to classify increasingly severe degrees of obesity. The thresholds are based on correlations with morbidity and mortality risk (Kuczmarski and Flegal 2000). Several websites offer simple BMI calculators. See, for example, http://www.cdc.gov/nccdphp/dnpa/bmi/.

with the data for this demographic group, observed in the NHANES II, NHANES III, and NHANES 99 studies, we predict large increases in mean weight, and even larger gains in upper-quantile weights, as the food price falls. For simulated price declines that are pinned to independently estimated trends in the full price per calorie of food, including both the money price and time costs, the predictions match the quantitative changes in average weight and the obesity rate for this group with considerable accuracy. Depending on how rapidly the weight aspiration adjusts to changing behavior, the dynamic analysis shows that equilibrium adjustments may occur with a substantial lag, helping to explain the observations, over the past 10 years, that food prices have been roughly flat and yet average weight and obesity rates have continued to rise.

Among a number of genetically influenced physiological factors known to affect weight and BMI, the basal metabolic rate (BMR)—the calories expended per day in the maintenance of involuntary bodily functions with the body at rest—is arguably the most important, and it is relatively easy (albeit expensive) to measure (Schofield et al. 1985, Cunningham 1991). Using a well-known data set containing direct observations of basal metabolic rates, we estimate a parametric model of metabolism in relation to body weight, a model that includes an idiosyncratic component. While previous economic models of obesity have assumed that metabolism is linear in weight, we find—consistent with the most recent research on metabolism (Horgan and Stubbs 2003, Cunningham 1991)—that models in which BMR is strictly concave in weight fit the data better. By embedding the metabolic model into the economic choice model, we can describe complete weight distributions at each food price value. More important than its ability to capture cross-sectional weight variation (and metabolic variation appears capable of explaining a substantial portion of this variation) are the surprising consequences the metabolism model predicts for the evolution of the distribution over time as prices fall. The marginal effect of calorie consumption increases, on average, as average weight grows, even with no change in the distribution of genetic endowments. This result does not arise when metabolism is assumed to be linear in weight, and it contributes significantly to growth in upper-tail weights relative to the mean. The analysis shows that the specifics of the metabolism model matter considerably for prediction and

policy analysis.

We also illustrate the distributional implications of alternative explanatory models of obesity growth, including those of CGS and of a rational addiction model adapted from Cawley (1999). We introduce heterogeneity in the respective explanatory factors described in these models—self-control over food intake and addictive propensity—and describe the resulting weight distributions at different price levels. (In the case of CGS, we vary the time cost of food and its money price separately, in keeping with their framework.) We find that, when food prices fall, variation in these behavioral traits can lead to greater weight gains in the upper tail of the distribution than at the mean. However, these predictions rely on as-yet-unverifiable assumptions about the distributions of self-control and addictive propensities in the population. We therefore view these alternative frameworks as complementary to our own and argue that a better understanding of the severity and prevalence of self-control and addiction problems concerning food is needed in order to assess their contributions properly.

Social comparison in the model implies that individuals aspire to weigh less (by some fraction) than the average weight in the population at a given time. This endogenous weight aspiration increases as food prices fall, because price declines cause average weight to increase. Although it is difficult to observe weight norms directly, this prediction agrees with the observation that the self-reported "desired weights" of Americans increased significantly between 1994 and 2002, complicating the conventional wisdom that media images emphasizing thinness perfectly dictate weight aspirations. The evidence on desired weight comes from the CDC's Behavioral Risk Factor Surveillance System (BRFSS), which contains data on self-reported desired weights and actual weights for the same individuals.[5] While the data are not longitudinal, observations from different survey years suggest the overall trends. In 1994, the average weight for an American woman was 147 pounds, while the average *desired* weight was 132

[5]Self-reported weight data are known to be biased, and corrections are suggested in Chou et al. (2004), among others. However, self-reported desired weights must be taken at face value, since they cannot be checked against their "true" or revealed values. Accordingly, in comparing actual weight with desired weight (or actual BMI with desired BMI), we use self-reported values of weight, desired weight, and height. These are reported in Figures 2 and 3. Alternatively, we can correct both values according to the same algorithm, and these figures are available on request. Either way, desired weight increases with actual weight over time.

pounds. By 2002, the average had increased to 153 pounds, and average desired weight had increased to 135 pounds. These data—which follow similar patterns expressed in terms of BMI—suggest a reduction in (implicit or explicit) social pressure to maintain lower weights. At the same time, medical innovations reduced obesity-related morbidity and mortality considerably over the period (Flegal et al. 2005). Although we do not model medical incentives explicitly, the "moving norm" model is consistent with these developments as well.

The remainder of the paper is organized as follows. Section 2 describes the theoretical model. Section 3 analyzes the comparative static effects of price on equilibrium weights, the weight norm, and welfare. In Section 4, we simulate equilibrium weight distributions under three different price levels and simulate the dynamic adjustment paths across equilibria. We compare our results with benchmark models involving weight-linear metabolism, a fixed weight norm, and forward-looking (as opposed to myopic) behavior. In Section 5 we evaluate explanations for the evolution of the weight distribution (again under falling food prices) based on variation in self-control and addiction to food. The concluding section discusses policy implications and predictions about the future of obesity.

2 Theoretical Framework

2.1 Agent-based model

The theoretical model takes an agent-based approach, positing genetically heterogeneous individuals interacting within a social group. The nature of the interaction is that each individual compares her own weight to the group's commonly-held norm or "desired" weight, and this comparison enters her optimization problem as described below. The existence of weight standards may seem an obvious social fact, but there is no scientific consensus on how these standards are formed. Desired weight is defined as a fraction, less than one, of average weight in the group, and is therefore subject to change over time. This specification, in which people aim to be thinner than the average person in the refer-

ence population, combines two basic assumptions: 1) in contemporary Western society, thinness (up to a point) is prized (Garner et al. 1980, Mazur 1986), and (2) individuals assess themselves in relation to others rather than against an absolute scale. The latter assumption follows the social interactions literature in economics, as well as longstanding traditions in sociology and social psychology, in stressing the notion that people are concerned with being normal in relation to their peers (see, for example, Bernheim 1994, Brock and Durlauf 2001, Becker and Murphy 2000, Bandura 1986, and Dwyer et al. 1970, among many others). This specification creates room for gaps between the prevailing white Western ideal of thinness and the *de facto* standards to which individuals aspire (or against which they are judged); consequently, ours is not a model of the evolution of media ideals.

The assumption of a common (relative) weight norm is admittedly highly stylized, and we recognize that individual weight aspirations are likely to exhibit idiosyncratic variation. In the BRFSS data for 30-to-60-year-old women, the coefficient of variation of desired weight is 13.9 percent. However, the coefficient of variation of actual weight is significantly greater, at 23.1 percent.[6] Figure 2 illustrates this discrepancy by overlaying the distributions of actual and desired BMI. In addition, race is a significant explanatory factor in desired weight for this sample. (Figure 3 plots mean desired weights against mean actual weights for various demographic groups.) These facts suggest the presence of a social component as well as an individual component in the formation of weight aspirations. Our model emphasizes the social component by assuming a uniform weight norm for all American women between 30 and 60 years of age. This assumption prevents us from using *ad hoc* variation in norms within this demographic group as an explanatory tool, and therefore results in a conservative test of the influence of social weight norms.

Equilibrium for the system is defined as a weight distribution and a weight norm that are mutually consistent. Each individual maximizes a myopic utility function over short-term food and nonfood consumption, taking the reference weight and prices into account.[7] Food and nonfood consumption are

[6] For men, the corresponding figures are 13.7 percent and 18.6 percent, respectively.

[7] We will refer to the reference weight alternatively as the norm.

both goods, but deviation from the reference weight is a bad. A general expression of the one-period utility model is as follows:

$$U_{it}[F_t, C_t | W_{t-1}] = G_i[F_{it}, C_{it}] - J(W_{it}[F_{it}, W_{i,t-1}, \varepsilon_i] - M_{t-1})^2. \quad (1)$$

F_t and C_t represent food and nonfood consumption, respectively, for period t. W_{t-1}, representing weight at the end of period $t-1$, is a product of past actions. Individual heterogeneity is captured by ε_i, which is a stationary shock to basal metabolism, as described below. G_i is the norm-independent component of utility: It is strictly increasing and strictly concave in C and strictly concave but not necessarily monotonic in F. The term beginning with J gives the social-interaction component, which represents the cost of deviating from the reference weight, M. The subscript on M indicates that agents observe the value of M at the end of period $t-1$ and take this as fixed in the optimization; in particular, they do not forecast the value of M that will emerge as a consequence of aggregate behavior in period t. The coefficient J gives the strength of the social interactions, which is held constant across individuals. The presence of a norm has the intuitive effect of lowering the variance of weight in the population, even though not all population members conform to the norm exactly.

The individual correctly anticipates her own end-of-period weight as a function of food intake and so takes into account the effect of current food consumption on the cost of deviating from the reference weight. This cost is symmetric—it is just as undesirable to be underweight relative to the norm as to be overweight—and is meant to capture several known types of sanctions. The stigmatizing of overweight (and underweight) individuals is well-documented (Myers and Rosen 1999) and may entail, for example, teasing, ostracism, and discrimination in hiring. Peer pressure and contagion regarding eating behavior have also been observed, particularly among adolescent girls (Crandall 1988). Ross (1994) has emphasized depression as a consequence of overweight. She identifies three causes of depression among the overweight, two of which relate directly to the presence of socially derived weight norms. She finds that some overweight individuals become depressed as a direct result of

negative self-perception and that these individuals tend to belong to social groups with a low incidence of overweight. Graham and Felton (2005) reinforce these findings in two ways: They find that, in general, obesity contributes to depression among Americans (and they reject the reverse causality), but they also find that obesity does not raise depression risk significantly among African-American women, a group with one of highest obesity rates in the United States.

In addition to mental health costs, extreme overweight and underweight entail significant physical health consequences. Several studies have shown, for example, that the risks of diabetes, heart disease, osteoarthritis, and other health conditions accelerate with increases in body mass index (for example, Must et al. 1999). In addition, mortality exhibits a U-shaped relationship to BMI among men in the United States, indicating that underweight imposes similar mortality risks as overweight (Troiano et al. 1996). Evidence from developing countries, where underweight is much more prevalent than in the United States, indicates substantially elevated disease incidence among low-weight (BMI below 20) individuals (Ezzati et al. 2002). A model with deviation costs that depend on a mutable norm will capture these health costs only when the value of the norm lies within the medically recommended range. In the parameterizations we consider, the emergent norms do, in fact, fall within this range, but in general the model does not constrain them to do so. The health costs of obesity in particular are partly reflected in the increased per-capita health spending among the obese relative to the normal-weight population (Thorpe et al. 2004).

In addition to psychological and physical costs, there are direct economic costs associated with overweight and obesity. For example, among younger white females (age 16–44) in the United States, an increase in weight of two standard deviations has been shown to reduce the average wage by 9 percent (Cawley 2004).[8] Marriage-market penalties for overweight and obesity among women, which may involve both economic and psychic costs, have been documented by Averett and Korenman (1996).

Successive optimization of the one-period problem implies convergence to a stable weight for any

[8]The results in Cawley (2004) and in Averett and Korenman (1996), based on recent U.S. samples, show weight-related earnings penalties only for overweight and obese individuals. Since the incidence of underweight in the U.S. is limited, these findings do not rule out the possibility of equivalent economic costs among underweight subjects.

given value of M. This weight does not, in general, coincide with the stable weight that optimizes a dynamic programming problem in which one-period utility is given by $U[.]$. The myopic specification may be taken to imply some lack of self-control, although we do not explicitly model a time inconsistency problem, as do CGS. The model does not imply that individuals ignore the future altogether, since they take into account the near-term effect of calorie consumption on weight and factor in the social cost (or benefit) of the weight change. This assumes that individuals correctly perceive their net energy intake.[9] We examine the robustness of our predictions to forward-looking specifications and compare the power of our model to that of other models that emphasize intertemporal concerns. As discussed in Section 5, we find that the myopic specification does not drive the central qualitative results, but has an advantage in explaining the quantitative response to price changes and, in some cases, alters the welfare analysis.

For purposes of simulation and calibration, we specify the maximization problem as follows:

$$Max_{\{F_t,C_t\}} U_{it}[F_t, C_t | W_{i,t-1}, \alpha, \delta, \beta, J, \gamma, \rho, \varepsilon_i, M_{t-1}] = \tag{2}$$

$$\alpha F_{it} - \delta F_{it}^2 + \beta log[C_{it}+1] - J(W_{i,t-1} - (7/3500)BMR(\gamma, \rho, \varepsilon_i, W_{i,t-1}) + .9F_{it} - M_{t-1})^2,$$

$$s.t. \quad p_t F_t + C_t \leq Y.$$

Within a single period, calibrated to one week, the marginal utility of food, F, declines and eventually becomes negative. The expression inside the parentheses following J just amounts to the difference between end-of-period weight, W_t, and the norm, M, as of time $t-1$, as in equation (1).[10]

Aside from the calories burned in digestion, we assume for simplicity that calorie expenditure is limited to the basal metabolic rate (BMR), or the calories needed only to sustain basic bodily functions, such as lung and heart activity, with the body at rest. The advantage of this assumption is that BMR has

[9] There is evidence that people systematically underestimate their caloric intake (Wansink 2004), but we ignore this problem in the current paper.

[10] This social interaction term is similar to those in Glaeser and Scheinkman (2002), Brock and Durlauf (2001), and Burke and Prasad (2005), among others.

a strong exogenous (that is, genetic) component that has been measured in numerous studies (discussed below). Of course, physical activity induces calorie expenditure over and above BMR in the short run, and exercise may alter BMR in the long run via its effect on the quantity of fat-free body mass (Cunningham 1991). However, Black et al. (1996) find, using extensive data from affluent societies, that BMR is strongly correlated with total energy expenditure (TEE), the former accounting for 60 percent to 65 percent of the latter, on average. They also find that physical activity level (measured as the ratio of TEE to BMR) is orthogonal to BMR. Variation in BMR is therefore a good predictor of variation in total calorie expenditure, with the advantage of isolating the persistent genetic component of heterogeneity and the disadvantage of underestimating the variance of TEE.

A large literature, spanning the fields of public health and clinical nutrition, has concerned itself with estimating predictive equations for BMR in order to help determine caloric needs based on readily measured variables such as weight, height, age, and sex. Although consensus on a single best model has not been reached, taken together the studies indicate broad agreement on a number of issues: (1) body weight is a significant (positive) factor in BMR, and knowledge of the weight-BMR relationship is crucial for estimating energy needs (Cruickshank 1999); and (2) there is significant idiosyncratic variation in BMR around the predicted values. While Cunningham (1991), Weigle et al. (1988), Pullicino et al. (1996), and others have argued that lean body mass is a better predictor of BMR than weight (and that use of this measure eliminates needs for adjustments based on age, sex, and height), the significant relationship between weight and lean body mass implies a concave relationship between weight and BMR (Cunningham 1991) that can be used when lean body mass cannot be readily measured.

While some studies have found that the body responds to short-term weight gain or loss with compensatory changes in metabolism that seek to "defend" the initial equilibrium weight (Wilson 1999, Labayen et al. 2004), the duration of these effects is unknown. While we do not dispute the possibility of metabolic path dependence and other complexities at the individual level, two relatively recent studies (Martin et al. 2004, Arciero et al. 1993) have found significant predictive power for a model of BMR first advanced by Harris and Benedict (1919), indicating robustness of the weight-metabolism relation-

ship (over a given weight range) to secular changes, such as increased average calorie consumption, and group-level differences in the populations under study.[11]

One model often employed for predictive purposes is the weight-linear model of Schofield et al. (1985). Both CGS and Lakdawalla and Philipson (2002) adopt linear specifications, and CGS employ specific coefficients from the Schofield study. However, our own analysis of the Schofield data, as well as a number of other prominent studies, questions the accuracy of the linear model. For example, Horgan and Stubbs (2003) have shown that the Schofield equations substantially overestimate BMR for the obese, a problem due in part to the dearth of obese subjects in the Schofield data. The Horgan and Stubbs findings, as well as the reduced form of the Cunningham model (1991) and other models based on lean body mass, imply declining marginal effects of body weight on BMR. The effect arises because excess weight tends to come disproportionately in the form of fat, which burns far fewer calories per pound than does lean mass. In addition, there is evidence of heteroskedasticity in the error term. Studies that have found the disturbances to be positively correlated with weight include Leibel et al. (1995) and Rand (1982).

Our own analysis of the Schofield data, using maximum likelihood estimation, finds that (1) a weight-log-linear model of (expected) BMR fits the data better than a weight-linear model and (2) the error term is heteroskedastic, with error variances increasing in weight. To illustrate the importance of the metabolic specification, we generate simulated equilibrium weight distributions under both of our estimated models (log-linear and linear, respectively). Both models involve the same, heteroskedastic, error structure. Of the two models, only the log-linear specification generates strongly asymmetric equilibrium weight distributions with long upper tails, in strong agreement with the distributional features of the BRFSS and NHANES data. The concavity of the log-linear model also contributes to large weight growth in the upper tail over time, a pattern that does not emerge strongly under the linear model. (Results are discussed in more detail in Sections 4 and 5.)

[11] Evidence on racial and ethnic influences on BMR is mixed, depending on definitions of ethnicity and other methodological factors (Martin et al. 2004, Hayter and Henry 1994, Soares et al. 1998, Cruickshank 1999).

The linear heteroskedastic specification is as follows:

$$BMR(kcal\,per day) = \gamma + \rho W + \varepsilon_i log(W). \tag{3}$$

The shock, ε_i, is idiosyncratic and permanent; it is normally and identically distributed with mean zero and standard deviation σ_ε. In expectation, then, the relationship between BMR and weight is linear in this model, but for a given nonzero value of ε_i, metabolism deviates from the linear relationship in proportion to log weight. For the log-linear specification, expected BMR is log-linear in weight, and the error structure is the same as above. That is, we simply replace weight with the natural log of weight in the second right-hand-side term in (3). The metabolic equation in (3) implies the following (one-week) relationship between food intake and weight:

$$W_t = W_{t-1} - (7/3500)(\gamma + \rho W_{t-1} + \varepsilon_i log(W_{t-1})) + .9F_t. \tag{4}$$

The term $.9F_t$ represents the thermic effect of digestion, that is, the fact that digestion consumes, on average, 10 percent of calories consumed. Weight is measured in pounds, while metabolism is measured in (kilo)calories per day. Accordingly, the latter must be converted into pounds of body weight lost over one week. The conversion factor of $7/3500$ is the ratio of the number of days in a week to the number of calories (3500) per pound of body weight. Food is measured directly in pounds of body weight added per week, which can easily be converted back to calories by multiplying by 3500.

For simplicity, we do not model height variation. There are a number of reasons why we do not expect height variation, either cross-sectionally or over time, to contribute much explanatory power to the weight distribution. Among U.S. women, average height has increased by less than 2 percent over the past four decades (see for example, Komlos and Baur 2003); observed weight increases over that period have far exceeded the weight gains that would have held BMI constant. As for cross-sectional differences, the Cunningham (1991) study indicates that height does not add much explanatory power

to the model of BMR based on lean body mass. Also, Schofield et al. (1985) argued that height was secondary to weight in predicting basal metabolism. Using the Schofield data for women ages 30 and older, a linear regression of BMR on height and weight has an R-squared value of .339, an improvement of just .006 over a linear model that includes weight alone. A linear regression of BMR against body mass index (BMI), a measure that incorporates height, yields an R-squared of .235 for the same demographic group.

Height could affect weight outcomes for non-metabolism-related reasons, however. For example, we might expect weight aspirations to be height-specific, as in government tables indicating healthy BMI values rather than healthy weights. However, we express the common norm as a weight value rather than as a body mass index (BMI) value. While the BRFSS data do indicate variation in desired weights with height (as well as with actual weight), such variation does not render desired values of BMI constant across individuals. In fact, the desired BMI values implied by the BRFSS data decrease systematically in height, suggesting that women do not adequately adjust for height differences in setting weight aspirations. Therefore, the common weight norm assumption, while stylized, is not necessarily less realistic than one involving a common norm for BMI. Anecdotal evidence suggests that weight values are more salient than the less-readily-observed BMI values.

3 Equilibrium and Comparative Statics

3.1 Equilibrium definition

Individuals in the population are identical in all the parameters of the utility function, α, β, ρ, γ, J, M; have identical incomes; and face the same prices. The only explicit source of heterogeneity is the idiosyncratic metabolic shock, ε_i. The full equilibrium conditions under the linear metabolism model

can be expressed as follows:[12]

$$\alpha - 2\delta F_i^S - 1.8J(W_i^S - M^S) = \lambda p, \tag{5}$$

$$F_i^S = (1.11)(7/3500)(\gamma + \rho W_i^S + \varepsilon_i log(W_i^S)), \tag{6}$$

$$M^S = \zeta(\frac{1}{N}\sum_i W_i^S), \tag{7}$$

$$\frac{\beta}{C_i^S + 1} = \lambda, \tag{8}$$

$$pF_i^S + C_i^S = Y_i. \tag{9}$$

The conditions apply to an interior equilibrium, in which stable food intake, F_i^S, stable weight, W_i^S, and stable non-food consumption, C_i^S, are all strictly positive. M^S is the equilibrium weight norm, which, according to equation (7), is some fraction, ζ, of the average stable weight that arises under this norm. Equation (5) gives the first-order condition on food consumption, where λ is the Lagrange multiplier. Equation (6) guarantees that per-period food intake maintains weight at the level W_i^S. Equations (8) and (9) are, respectively, the first-order condition on non-food consumption and the budget constraint. The equilibrium norm depends on the relative price of food, the distribution of individual shocks, and the magnitude of J, because these determine the stable individual weights and consumption levels for any fixed M. The equilibrium norm (and therefore the weight distribution) also depends on ζ, which we will set at .88 in various simulations. Equilibrium depends on income levels and the remaining parameters as well, but we hold these fixed throughout the analysis.

Assuming the shocks are normally distributed, the expected value of the equilibrium norm is defined implicitly as a function of prices by the following equation, in which $\phi(.)$ represents the standard normal

[12] Equilibrium conditions for the log-linear metabolism model are equivalent but analytically less transparent, so we use the linear specification here for ease of exposition.

density function:

$$M^S(p) = \zeta \int_{-\infty}^{\infty} W_i^S(M^S(p), p, \varepsilon_i)\phi(\varepsilon_i/\sigma_\varepsilon)d\varepsilon. \tag{10}$$

It should be noted that because the absolute shocks are heteroskedastic in weight, the expected average weight in equilibrium does not correspond to the stable weight for the individual who draws $\varepsilon_i = 0$.

Under our functional form and parameters, an interior equilibrium exists and is unique for each realization of the metabolic shocks. The existence and uniqueness of a stable equilibrium norm follows from two (necessary and sufficient) properties of the model: (1) each individual has a unique stable weight for every possible value of M; and (2) the rate of change of the stable weights with M is positive and less than one. The existence and uniqueness of a stable weight for a given M and ε_i depend in turn on three necessary and sufficient conditions: (1a) a unique solution to the one-period problem exists for each starting weight; (1b) optimal caloric intake decreases (increases) as one's initial weight gets farther above (below) the target weight or norm; and (1c) the total number of calories burned per day is strictly increasing in weight for each individual, at a rate less than one. The stable weight solves the one-period problem when the initial weight happens to be the stable value, but it is not in general the individual's optimal stable weight.[13] From any initial state of the system, both convergence to the stable weight for any value of M and convergence to the equilibrium M for given parameters are guaranteed. We provide verification of these assertions in the mathematical appendix.

3.2 Comparative statics

3.2.1 Price effects and the social multiplier

First, we consider the effect of a change in the full price per food calorie on equilibrium outcomes. We have in mind a price decrease caused by an outward shift in the food supply curve, reflecting a decline in food production and preparation costs (as in Philipson and Posner 1999 and CGS). Price has both

[13] The optimal stable weight would maximize one-period utility, subject to the constraint that weight be unchanged during the period.

direct and indirect effects on body weight. The direct effect is the change in stable weight, holding the norm fixed. However, given that each individual adjusts her weight in response to the price change, the norm must be updated. The norm change in turn sets off additional changes in weight and a further adjustment of the norm, and so on, until a new equilibrium is reached.[14] The latter is an example of a "social multiplier" effect, as in Becker and Murphy (2000), Glaeser and Scheinkman (2002), Brock and Durlauf (2001), and Burke and Prasad (2005). The total effect is expressed as the decomposition of these two effects as follows:

$$\frac{dW_i^S}{dp} = \frac{\partial W_i^S}{\partial p} + \frac{\partial W_i^S}{\partial M}\frac{dM^S}{dp}, \tag{11}$$

where the expression $\frac{dM^S}{dP}$ refers to the change in the equilibrium norm caused by the price change. The first term is negative: It is optimal to eat more, and therefore weigh more, the cheaper is food, *ceteris paribus*. As weights rise, so does any positive function of the average, and weight always moves directly with the target weight M (that is, $\frac{\partial W_i^S}{\partial M}$ is strictly positive). Therefore, the social multiplier effect reinforces the price effect, guaranteeing that the equilibrium weights and the equilibrium norm are decreasing in price, that is, $\frac{dW_i^S}{dp} < 0$ and $\frac{dM^S}{dP} < 0$.

We can decompose the price effect on the equilibrium norm as follows:

$$\frac{dM^S}{dp} = \frac{\zeta}{N}\sum_i\left(\frac{\partial W_i^S}{\partial p} + \frac{\partial W_i^S}{\partial M}\frac{dM^S}{dp}\right) = \frac{\frac{\zeta}{N}\sum_i \frac{\partial W_i^S}{\partial p}}{1 - \frac{\zeta}{N}\sum_i \frac{\partial W_i^S}{\partial M}}. \tag{12}$$

The numerator in the last expression on the right represents the average partial price effect on individual weight, multiplied by the factor ζ. The denominator, which is strictly less than one under our assumptions, acts to amplify the partial price effects in equilibrium, leading to a greater change in average weight than would occur if the norm were fixed. The social multiplier can be expressed as the factor by which the average partial-price effect gets multiplied to yield the average equilibrium-price effect. This

[14] Convergence to a unique equilibrium for any given price requires that the social influence on weight not be too strong—specifically, the partial derivative of individual weight with respect to the weight norm must be less than one. See the mathematical appendix for further discussion.

factor amounts to the quantity $1/(1-m)$, where $m = \frac{\zeta}{N}\sum_i \frac{\partial W_i^S}{\partial M}$ represents the average partial effect of a norm change on stable weight, multiplied by ζ. We assume that this latter partial effect is strictly positive and yet not too large, that is, strictly less than one. This assumption guarantees that the multiplier is finite and yet strictly greater than one (Burke 2006).[15]

We have assumed that the initial price decline is exogenous to the model. However, the social multiplier effect implies an outward shift in the (norm-constant) food demand curve. To restore market equilibrium following such a shift, the price per calorie would have to move back up, but we do not derive this price movement in the current framework. Relative to a framework with a fixed weight norm, an increase in food supply in the endogenous-norm model results in a smaller price decline and a greater weight gain in equilibrium. The social multiplier effect thus acts to increase the price elasticity of calorie demand. If weight norms shift with a lag, rather than simultaneously with changes in average weight, the price elasticity of calories should be greater in the long run than in the short run.

3.2.2 Welfare effects of food price changes

Consumer welfare in our model depends only on weight relative to the flexible social norm, regardless of how this norm compares with a healthy weight standard. Thus, the welfare effects of price changes are potentially quite different than welfare effects for a consumer who compares her weight to a fixed health standard. We assess this difference within our framework by decomposing the welfare effects of a price decline into two components: the portion of the welfare effect induced by the price decline only, holding the weight norm fixed, and the portion of the welfare effect induced by the change in the equilibrium norm. The decomposition is as follows:

$$\frac{dV^S}{dp} = \left[\left(U_W + U_F\frac{dF^S}{dW^S}\right)\frac{\partial W^S}{\partial p} + U_C\frac{\partial C^S}{\partial p}\right] + \left[\left(U_W + U_F\frac{dF^S}{dW^S} - pU_C\frac{dF^S}{dW^S}\right)\left(\frac{\partial W^S}{\partial M}\frac{dM}{dp}\right) + 2J\left(W^S - M\right)\frac{dM}{dp}\right], \tag{13}$$

[15] Becker and Murphy (2000) define the social multiplier as the term $m = \frac{\zeta}{N}\sum_i \frac{\partial W_i^S}{\partial M}$, but the thrust of the results is the same in either case.

where V^S refers to the agent's utility in equilibrium and $\frac{dF^S}{dW^S}$ represents the increase in food consumption required to maintain a higher stable weight value.[16] U_W refers to the marginal utility of an increase in the stable weight, which is the same as the marginal utility of an increase in final weight within a given period. The terms inside the first set of square brackets represent the welfare effects of the price change, holding the norm fixed. The terms inside the second set of square brackets capture the additional impact on welfare prompted by the change in the equilibrium norm. Assume that in the initial equilibrium the consumer weighs more than the norm, in which case the marginal utility of weight gain, U_W, is negative and the marginal utility of food, U_F, is positive. Holding the norm fixed, a food price decline may or may not make the (myopic) consumer better off. Welfare will improve only if the benefits of added consumption (of both food and nonfood goods) outweigh the costs of weight gain relative to the fixed norm.

The social multiplier effect set off by the price drop induces additional weight gain and food consumption, but a decrease in nonfood consumption (to satisfy the budget constraint holding income constant—this need not imply lower nonfood consumption on net when price effects are included). Also, in the aggregate it leads to an increase in the value of the weight norm. Again, the welfare effect is ambiguous. The benefits are that the consumer eats more and, ignoring the price effects on weight, her own weight moves closer to the norm. The latter result holds (for an initially overweight individual) because the increase in stable weight caused by the norm change is less than the increase in the norm. The cost is that she gives up some nonfood consumption.

Given the ambiguity in both components of the welfare change, the net welfare effects of a price change are ambiguous.[17] The indeterminacy applies to initially overweight as well as to initially underweight individuals. (Simulated welfare effects are discussed in Section 4.) All else being equal, the net welfare effect of a food price decline for a given individual depends on whether weight norms are

[16] In a linear metabolism model this number would be a constant that depends on the individual's shock; with log-linear metabolism this value still depends on the idiosyncratic shock, but it also decreases in stable weight.

[17] Welfare effects in a model with forward-looking consumers and a fixed norm, as in Philipson and Posner (1999), are unambiguously positive. CGS raise the possibility of welfare losses for individuals with imperfect self-control, but they estimate that the costs of weight gain have likely been less on average than the benefits of time savings in food preparation.

fixed or flexible. In the simulations, for example, we find that many consumers would receive greater welfare gains from a price decline (or smaller losses) in a model with flexible, as opposed to fixed, weight norms, despite the fact that they would weigh more in the flexible-norm case.

4 Main Simulation Results

We use computational experiments to assess the model's ability to explain both the general shape of the empirical weight distribution and the growth in its upper tail since the mid 1970s. The calibration targets the weight distributions for American women in the 30–60-year age bracket, observed between 1976–80 and 2000 (see Figure 1 and Table 1). This specificity assists the precision of the calibration, but the patterns for this group are representative of the overall U.S. trends during the same period. We describe the equilibrium weight distributions for a series of three prices, a series meant to approximate (roughly and discretely) the (full) food price declines observed in the United States between 1976 and 2002. We also describe the dynamic evolution of the distribution, at points both in and out of equilibrium, in response to a more gradually declining price path. We compare results under our model of endogenous norms and non-linear metabolism with results under alternative models based on competing frameworks. For some features of the distribution—for example, pronounced rightward skewness—our model offers clearly superior explanatory power. In addition, our model offers a closer match for the quantitative changes in the mean, median, 95th-, and 99th-percentile weights over the time period. Another desirable feature of our model is that it places more constraints on the calibration relative to alternative frameworks, because in the latter, the parametric form of the heterogeneity is unknown. The results of the experiments are summarized, using descriptions of the key distributional features, in Table 1.

4.1 Calibration

In each experiment, we draw 50,000 values from the shock distribution; these values are held fixed across the experiments to prevent noise from clouding the effect of changes to the model. We calibrate the model to women ages 30 to 60, setting an initial list of parameters to roughly match average weight for this group observed in the 1976–80 NHANES data. All parameters are identical across individuals, except for the idiosyncratic metabolic shock. To calculate obesity rates, we measure the percentage of women who weigh more than 174.5 pounds. For a woman of average height, approximately 64 inches in the United States for the relevant age group, this weight implies a body mass index of 30, which is the official obesity threshold established by the Centers for Disease Control. The body weight norm is defined as 88 percent of the realized average weight in equilibrium, a figure based on the relationship between desired weights and actual weights in the BRFSS data for women, as shown in Figure 3.

The metabolism models are estimated directly from the original Schofield data (see Schofield et al. 1985 for details on these data).[18] For the weight-linear metabolism model, using the maximum likelihood method, we estimate a weight coefficient of $\hat{\rho} = 3.19$ (t-value of 15.5) based on the data's subsample of 411 women aged 30 to 60. Using the same method and data, we estimate a BMR model that is log-linear in weight (in pounds) with a coefficient on the natural logarithm of weight of $\hat{\rho} = 447.6$ (t-value of 15.1). Our estimates for the linear model are comparable with those reported in Schofield et al. (1985). Using the information on the 199 American women in the sample, we estimate that the constant term for the average U.S. woman in that age group is $\hat{\gamma} = 928.9$ for the log-linear model. As discussed above, to our knowledge, no previous estimates of parametric non-linear BMR models based on the Schofield data exist (for a non-parametric estimate, see Horgan and Stubbs 2003), and the previous economic literature has employed non-stochastic linear models of metabolism. Sample likelihood comparisons suggest that the log-linear model fits the data better than the weight-linear model, consistent with recent research suggesting a concave relationship between weight and BMR

[18] We thank Graham Horgan of Biomathematics and Statistics Scotland (BIOSS) for providing us with the data.

(Horgan and Stubbs 2003, Cunningham 1991). Given that the Schofield data contain relatively few obese individuals (the obesity rate in the sample of 411 women used here is 13.6 percent), our BMR model may understate the degree to which metabolism slows among overweight and obese women.

Recall that ε_i represents the idiosyncratic metabolic parameter for individual i, the main source of heterogeneity in our model. The parameter is fixed longitudinally for a given individual, but the actual deviation from the expected metabolic relationship at any point in time is given by $\varepsilon_i w_{it}$, where w_{it} is the individual's prevailing weight. Assuming ε_i is normally distributed with mean zero, we obtain estimates, denoted $\hat{\sigma}_\varepsilon$, for its standard deviation from the maximum likelihood estimations described above. For the weight-linear metabolism model, we estimate a value for $\hat{\sigma}_\varepsilon$ of 24.18 (t-value of 35.1), and for the weight-log-linear model, we get $\hat{\sigma}_\varepsilon = 24.1$ (t-value of 35.0).

Under the calibration, the marginal utility of the first unit of food in a week exceeds the marginal utility of the first unit of non-food consumption by 24 percent. (The coefficients on the utility function in equation (2) are $\alpha = 6.2$, $\delta = 0.9$, and $\beta = 5$.) The parameter J, representing the strength of social interactions, is set at 0.0018. This value, together with prices and the other utility parameters, determines the response of individual weight to an exogenous change in the weight norm. On average across individuals and prices, the magnitude of this effect is about 0.2, meaning that individual weight increases by one-fifth of a pound for every one-pound increase in the norm.

The model's price represents the full price, including both food inputs and time costs, of 3500 calories (the caloric equivalent of one pound of body weight). We experiment with this price at \$50, \$40, and \$32 to roughly match the decline in the real full price of calories over the past three decades. At the initial full price (\$50), the cost of the calories needed (1556) to cover basal metabolism and digestion for a 140-pound woman amounts to \$22.41 per day. At the lowest full price, this cost comes to \$14.81. Income is set at \$600 per week or \$31,200 per year, implying a gross hourly wage of \$15.[19] Our values for income and the full price of food imply that in the initial equilibrium (at the highest full

[19]This is consistent with hourly average U.S. wages in 2000, using 2000 dollars. In 1982 dollars, the average hourly wage in the U.S. in 2000 was about \$8, roughly the same as in 1980.

price of calories) the average person is spending about 26 percent of her income on food expenses.[20] CGS report that the time costs of food (including meal preparation and clean-up, but not shopping or transportation) fell by 42 percent between 1975 and 1995, due to greater availability of restaurants (in particular, fast food and take-out) and technological advances in food processing, storage, and preparation. Since time cost declines appear to have leveled off considerably following 1995 (Aguiar and Hurst 2006), the overall time cost decline between 1975 and 2000 should not be much different from 42 percent. With time costs valued at the assumed hourly rate of income, the assumed 36-percent overall decline in the full food price implies a real decline in the purchase price per calorie of 23 percent from its initial value.

Note that the 23-percent decline estimate lies between the relative decline in the overall food CPI and the relative price declines of several important food commodities. (As shown in Figure 4, the prices of ground beef, chicken, eggs, and lettuce have fallen 50 percent relative to the overall CPI since 1980.) As seen in Figure 5, between 1978 and 2002, the overall food-at-home price index fell by 15 percent relative to the CPI, and the food-away-from-home index fell by 6 percent. The declines for individual items should qualify the (smaller) estimated declines for the overall food CPI, since the latter is likely to be a less reliable measure of the price per calorie of food. In fact, the food CPI is likely to have underestimated the decline in the price per calorie over the period of interest: Changes were made in the list of included items to reflect changing consumer expenditure patterns (see the BLS Handbook of Methods, http://www.bls.gov/opub/hom/pdf/homch17.pdf), and these expenditures must have embedded the increases in calorie consumption observed over the period. USDA consumption data show that per capita calorie consumption (adjusted for losses) has increased by about 22 percent since 1975 (see Putnam et al. 2002). However, our chicken and beef prices refer, respectively, to the price per pound of whole fresh chicken and the price per pound of ground chuck (and not to the composite “beef and veal” and composite “chicken” category prices) and so should be relatively reliable

[20] Abstracting from time and preparation costs, Huang (1993) estimates that the average food budget share between 1953 and 1990 in the U.S. was about 18 percent.

indicators of per-calorie prices.[21]

To illustrate further, consider McDonald's Big Mac sandwich, which contained 590 calories consistently over the period under consideration (and still does). Our assumed highest and lowest full price levels imply, respectively, high and low full prices for the Big Mac of $8.40 and $5.30. Based on information from McDonald's and *The Economist* magazine, we calculate an average list price of a Big Mac in the United States of about $2 for the year 2000. The difference between $5.30, our lowest estimated full price, and $2 amounts to $3.30 (in year 2000 $), which is the value of approximately 13 minutes of the individual's time. Thus, our lowest price estimate seems roughly appropriate to capture the current full price of fast-food calories. Adjusting Big Mac list prices for inflation, we calculate that the real price of a Big Mac in 1980 was $2.24 (again in 2000 $), implying that the real list price of the Big Mac declined by approximately 12 percent between 1980 and 2000 and that its total time cost in 1980 was 25 minutes. These estimates imply that the purchase price per calorie for the Big Mac fell substantially less than the average purchase price per calorie of food in general, but that the Big Mac's time costs fell by a greater percentage than did overall time costs. These findings seem consistent with evidence that both the supply of and demand for fast food increased relative to other foods over the time period (Chou et al. 2004).

4.2 Linear vs. log-linear metabolism

Previous economic analyses of obesity involving models of metabolism have adopted equations that express (exogenous) calorie expenditure as a linear function of weight: In Lakdawalla and Philipson (2002), calories are perfectly proportional to weight, and in CGS the linear model (taken from Schofield et al. 1985) also contains a positive constant term. As discussed above, recent studies have shown that the marginal increase in basal metabolism with body weight declines with body weight, both

[21] It is possible that the average fat content per pound of fresh whole chicken or per pound of beef ground chuck changed over the period of observation, leading to changes in the calories per pound for either item. However, ground chuck generally conforms to a *de facto* standard of 80 percent lean content, and the BLS measures separate prices for other (leaner or fattier) grades of ground beef and also for other specific cuts of both chicken and beef.

longitudinally and cross-sectionally (Cunningham 1991, Pullicino et al. 1996, Horgan and Stubbs 2003). Confirming these findings, we obtain a superior fit of the Schofield data, using a metabolism model that is linear in the log of weight, rather than linear in weight itself. The concave specification of metabolism turns out to hold significantly different implications for obesity growth as prices fall than does the linear model, and it captures a greater portion of the observed increase in upper-quantile weights relative to the mean between 1976 and 2000.

The log-linear model's predictions at the $50 price match the 1976–1980 NHANES II data quite well, although parameters were selected only to match average weight at this price. (See Table 1 and Figure 6 for details.) At the same price, the linear model also provides a good match for the observed mean weight for NHANES II, but its predicted values for 95th- and 99th-percentile weights fall farther short of the actual values than under the log-linear model, because the linear (heteroskedastic) model produces a much less asymmetric distribution. (See Table 1 and Figure 7 for details.) The differences between the predictions of the respective models become even greater at lower prices. For the full price drop, from $50 to $32, the linear model implies a parallel shift of the weight distribution: Mean weight, 95th-percentile weight, and 99th-percentile weight all increase by about 18 pounds. The log-linear model predicts respective increases of 20.2, 27, and 31 pounds. Between NHANES II and NHANES 99, these values increase, respectively, by 20, 36, and 47 pounds.

Considering these movements in percentage terms, we see that the data exhibit greater percentage weight gains in the upper percentiles than at the mean: The ratio of the respective percentage changes in 95th-percentile weight and mean weight is 1.22 in the data, 0.96 in the log-linear model, and 0.79 in the linear model. (Percent changes are computed with respect to the average of initial and final weight; the figures do not change much if percent changes are computed from initial weights.) Although the simulated log-linear model does not generate disproportionate weight gains, its predicted gains are greater in absolute terms in the upper tail, and they are much greater than in the linear model.

The logic behind these results depends on two effects: the price elasticity of calorie consumption, and the calorie elasticity of weight (that is, the percent change in stable weight for a permanent mar-

ginal percent change in calorie consumption). The latter elasticity increases in body weight under the log-linear model and decreases in body weight under the linear model. Thus, for a given percentage increase in calories, the log-linear model of metabolism predicts greater percentage gains in weight for initially heavier individuals compared with lighter ones, and vice-versa for the linear model (all effects are evaluated along the metabolism regression lines). However, the price elasticity of calories is not, in general, constant in weight. In the log-linear model, the price elasticity of calories decreases with weight: This occurs because we assume that individuals correctly forecast the marginal effect of calories on weight for the current week and adjust consumption accordingly. We also assume that the marginal utility of food is constant in weight, that is, calories are not addictive. (Compare Section 2.1.) As a result of these assumptions, initially heavier people consume fewer additional calories in response to a price decline. Still, despite their lower caloric response they gain more weight than others, and larger percentage gains are possible, depending on how slowly the price elasticity of calories declines with weight. For the linear model, one would have to assume that calories were addictive, and to a sufficient degree, in order to get larger price-induced weight gains for heavier people, even in absolute terms.

Our analysis suggests that a better understanding of metabolic relationships, both cross-sectionally and longitudinally, is crucial for the formation of appropriate dietary recommendations. Although genetics cannot have changed much over the past 30 years, our analysis shows that the nature of metabolism itself, for a fixed gene pool, implies that a rightward shift in the weight distribution (caused by economic and social forces) results in greater average marginal effects of calories on weight, a type of "positive feedback" effect that does not occur under the linear specification.

4.3 Exogenous vs. endogenous weight norms

In contrast with our endogenous or evolving norm specification, other models that include a weight norm (Philipson and Posner 1999, Levy 2002) have treated the norm as exogenous. While the exact

basis for the norm is not specified in these models, such fixed norms may be meant to reflect official health recommendations or perhaps genetically hard-wired preferences.[22] Even permitting norms to vary in the long run, the fixed-norm case captures the short run if norms adjust with some lag. The fixed-norm case simply holds M constant at some arbitrary level. Individual optimization conditions do not change, but the norm-consistency condition is no longer relevant. Any given value of M results in a unique distribution of stable weights at each price, but the fixed value of M bears no necessary relationship to the emergent average weight in the population.

Adopting the log-linear model of metabolism, equation (3), and employing the same set of metabolic shocks across the cases as in the experiments above, we compare the effect of price declines between the fixed and endogenous-norm models. In the fixed-norm model, we set the norm equal to 130.6 pounds, a figure that corresponds to the equilibrium norm in the endogenous-norm model at the price of $50. Therefore, when the food price is $50, the distributions are identical across the two models. When the price falls to $40 and the norm is held fixed, the weight changes reflect only the partial effects of price, represented by the first term on the right-hand side of equation (11). In response to the price drop, the mean, median, standard deviation, 95th-percentile weight, 99th-percentile weight, and the obesity rate all increase. As expected, the increases are consistently smaller than they are under the endogenous-norm model, and the predictions get farther apart as price falls further.

By comparing the price effects under the fixed-norm model with price effects under endogenous norms, we get an estimate of the social multiplier. Recall from above that our social multiplier represents the ratio of the change in the equilibrium weight norm (in the endogenous model) to the average (multiplied by 0.88) of the partial (norm-constant) effects of price on weight. See equation (12) for details. We can estimate this value using the ratio of the price-change-induced change in the endogenous norm to the average (multiplied by .88) partial effect of the price change on weight observed in the fixed-norm model. For the price change from $50 to $40, the estimated social multiplier is 1.24.

[22] However, health recommendations also vary with scientific knowledge and are currently a matter of significant debate, based on recent findings relating overweight, but not obesity, with reduced mortality risk.

This value implies that the price-induced increase in average weight in the endogenous-norm model will be 24 percent greater than it would be in the fixed-norm case. Measured over the $40-to-$32 price interval, the value of the multiplier is 1.26.

We also track the welfare effects of the price changes in the contexts of the fixed and moving-norm models. With an endogenous norm, we find that the initial price change, from $50 to $40, leaves most individuals, 71 percent of the population, marginally better off. The greatest welfare gains accrue to those closest to the initial weight standard. Gains decline with initial differences between weight and the norm, eventually becoming negative. Welfare gains (losses) are not symmetric in the metabolic shock, however, given the concavity of the metabolic function, and very low metabolism individuals suffer the greatest losses. When price falls from $40 to $32, the changes are very similar, and 70 percent of the population are made better off.

If norms are held fixed, however, the initial price change improves welfare for only about 40 percent of the population—specifically, those in the upper half of the metabolic distribution (those with a relatively fast metabolism). Welfare gains (declines) are slightly smaller (greater) for the second price decline, and only 32 percent of the population are made better off. Therefore, a substantial portion of the population fare better in a society with flexible norms than in one with rigid standards. The model suggests that a certain amount of "fat acceptance" may improve welfare for many individuals. However, we have not taken into account the potential impacts on health (or on medical technology) as the norm moves out of the range of medically advised weight, nor potential externalities imposed on non-obese individuals, as, for example, occur when the increased cost of treating obesity-related disorders is not borne only by the obese (Bhattacharya and Sood 2005).

4.4 Forward-looking vs. myopic decision-making

Although our model assumes myopic consumers, our central qualitative results do not depend strongly on this assumption. To illustrate this point, we consider conventionally rational, forward-looking con-

sumers with no self-control problem or addiction problem with respect to food (we will deal with these alternative models in Section 5).

Using the same per-period utility function as in our myopic model, we simulate a two-period version with perfect foresight, a zero discount rate, and a fixed norm. This model closely resembles the one in Lakdawalla and Philipson (2002), which attributes the long-run rise of obesity to falling food prices and the increasingly sedentary nature of work. These authors adopt a linear, non-stochastic metabolism, but we add a heteroskedastic metabolic shock in order to generate cross-sectional variation (the specification of metabolism is as in (3), with coefficients as in Section 4.1). As shown in Table 1, the forward-looking model predicts lower average weights at each price than those predicted by our myopic model, because individuals take the full future costs of current food consumption into account. While the fixed norm also contributes to lower weights in this alternative model, weights are lower in the forward-looking model even in comparison with weights in a myopic model with the same fixed norm. Not surprisingly, the long-term price decline (from $50 to $32) causes smaller weight gains in the forward-looking model (with a fixed norm) than in our myopic model with endogenous norms, and so underestimates the actual weight gains over the period of interest. Even if we were to add this type of forward-looking behavior to our framework, we would find it harder to explain the observed weight gains, for given price changes, in a model with fixed norms than in one with changing norms.

4.5 Dynamic weight adjustment

So far we have analyzed long-run equilibria under a set of three discrete prices and compared the predicted outcomes to the NHANES data from three survey periods. However, we cannot be sure that any single NHANES snapshot (even one encompassing up to four years' worth of data) represents a long-run equilibrium weight distribution. Weight adjustments across equilibria must occur in "real time," because individuals cannot alter body weight instantaneously. Furthermore, even without observing such prices directly, we can be fairly confident that full food prices did not fall in a small number of

large discrete steps over the period of interest, but rather fell more smoothly. In contrast, we expect that body weight aspirations may adjust with a lag, since it takes time for individuals to observe and internalize increases in the average population weight. (Further below, we discuss other mechanisms that may signal a change in weight norms.) Depending on the relative speeds of price changes and weight adjustments, and depending also on the speed of adjustment of the social weight norm, the empirical weight distribution may spend much of its time out of long-run equilibrium. Our framework allows us to describe out-of-equilibrium dynamics and to predict the timing of weight changes in relation to the timing of changes in fundamentals.

We simulate the weight adjustment process for a decline in the average full price of 3500 calories, from $50 to $32, between 1976 and 1993. We assume that, as of 1976, the population of U.S. women in the 30-to-60-year age bracket was in the long-run (endogenous norm) equilibrium corresponding to the $50 food price. Adopting the log-linear metabolism model, we generate 50,000 individuals (metabolic shocks) and solve for this initial equilibrium distribution and its corresponding norm. Beginning from this equilibrium, we impose a series of discretely-timed price and norm changes, tracing the real-time adjustment of the weight distribution by solving the one-week optimization problems repeatedly over the interval. We update the norm annually in the first experiment, and every five years in a second experiment. We reduce the food price at the beginning of the year by 3 percent per year until 1993. After 1993, price is kept constant at $32, and we describe the subsequent time path of convergence to the final long-run equilibrium at the $32 price.[23]

We hold price constant beginning in 1993, based on several facts: (1) the overall food CPI did not fall beyond 1993, but rather showed a net gain of about one percentage point (relative to the overall CPI) by 2004; (2) the relative price of chicken was roughly flat since 1993; (3) the price of beef continued

[23]The price time-path in this simulation does not agree exactly with the price time-path implied by our prior equilibrium simulations. The discrepancy can be partly resolved by noting that the previous simulations assume that a given empirical snapshot represents the equilibrium weight distribution for the contemporaneous price. However, the model's adjustment process actually implies that, if prices change continually, the system is never in equilibrium. The current illustration, in which the price falls over a compressed time period and then remains constant for several years, serves as a qualitative demonstration of the adjustment dynamics.

to fall until 1998, then rose again, and ended up close to its 1993 level by 2003; (4) while weekly time costs (for food preparation and clean-up) fell 11 minutes per year on average between 1985 and 1994, the same measure fell only 1.9 minutes per year between 1994 and 2003. These facts, taken together, suggest that the full price per calorie has been roughly constant since 1993.

The panels in Figure 8 illustrate the time paths of average weight, 95th-percentile weight, and the social weight norm under these experiments. We see that, even though prices and weight norms change infrequently and discretely, weight increases occur gradually. Two factors contribute to this effect: (1) under myopia, calorie consumption takes multiple periods to reach its new stable value following a price or norm change, and (2) for any discrete change in calorie consumption, weight may take several periods to reach a new stable level. After the food price levels off, average weight continues to increase, by more than 3 pounds, resulting in an increase of 20 pounds over the entire interval. Under annual norm updating, average weight gets within one pound of its predicted final long-run equilibrium level of 168.6 pounds (see Table 1) by 2001. Under five-year norm updating, average weight does not reach this threshold until 2004, more than 10 years after the price levels off. Endogenous norm changes exaggerate the effect of a price decline on weight. However, when norms adjust with a lag, this extra effect may not occur until several years after the price change. That is, the long-run price elasticities of food consumption and body weight will be greater in the long run than in the short run. If the simulated price time-series is accurate, our dynamics help to explain the continuing increase in average weights over the past decade in the face of relatively flat food prices.[24] Although a model of rational food addiction can yield this same elasticity result (Cawley 1999), the probable length of the long run in the addiction context is unknown.

In our dynamic simulations, we assume that the weight norm changes infrequently (either annually

[24]More specifically, the delayed social multiplier effect can help explain simultaneous observations of a non-monotonic food price path (looking solely at the food CPI) with a monotonic (increasing) weight path. Out of equilibrium, a norm shift causes an outward shift of the food demand curve. In a general equilibrium model, this shift would push price back up, moderating the initial (supply-induced) price declines. The norm increase raises weight, the norm-induced price increase lowers weight, and the net effect could be positive. Over this adjustment process, we would witness an initial price decline, followed by a subsequent price increase, together with a monotonic increase in average weight.

or quinquennially), discretely, and simultaneously for all individuals, but we have not specified a social mechanism that might justify these assumptions. Our calculations from the 1994 through 2002 BRFSS data (repeated cross-sections) suggest that average desired weight has increased in most years since 1994. The increases are fairly gradual—about one-half pound per year—consistent with a gradual adjustment of weight norms (at least at the population level). One social signal of weight norms that may be salient among women is dress size. Interestingly, the dimensions corresponding to a given women's size have increased over time, such that, according to one estimate, a size-12 dress from the 1950s would be labelled a size 6 today (Simmons 2002). According to these same data, nearly all of this change occurred after 1970 (Simmons 2002). While some part of this change may reflect increases in women's height and overall skeletal dimensions, there is evidence that "vanity sizing"—sizing to flatter increasingly heavy customers—has been on the rise in recent years (Bold 1997, Helser 2004, Gebhart 2005). The use of such sizing strategies suggests that women care about the nominal size of garments, and that they may aspire to a fixed nominal size (a "perfect" size eight is one focal value), regardless of whether they are aware of re-sizing practices (Gebhart 2005). Clothing sizes provide signals of what is considered small, medium, and large relative to the target market, and under such conditions, nominal size norms act much like the moving weight norms of our model. Since size relabelling also entails costs, however, we would expect it to occur infrequently, rather than continuously. Furthermore, competition across retailers lends an impetus for such changes to occur in a coordinated fashion (at least within a given market segment): A consumer is apt to prefer a brand in which she wears a size eight to a brand in which she wears a size ten, all else being equal.

5 Simulation of alternative models

We showed in Section 4.2 that, in the context of our behavioral model, the linear metabolic specification generates distributions that are less skewed relative to the log-linear model and predicts that there will be virtually no difference between increases in average weight and increases in 95th-percentile weight

in response to price declines. Section 4.4 showed that forward-looking behavior does not change this result: The forward-looking model with linear metabolism does not look qualitatively much different from the myopic model with linear metabolism (under either fixed or variable norms). Therefore, myopia is not central to our qualitative predictions.

In order to capture the stylized facts of interest, a forward-looking model must contain some additional features. Aside from non-linear metabolism and endogenous norms, the two features suggested in the obesity literature are lack of self-control and susceptibility to food addiction. In this section, we compare our model to two prominent alternative theories based on, respectively, variation in the degree of self-control over food intake (CGS) and variation in the propensity for (rational) food addiction (Cawley 1999). While food addiction and lack of self-control over food may sound like similar phenomena, they are modelled differently within economics, and they may result in different predictions. The following analysis simulates these alternatives and compares them to our model with endogenous norms and non-linear metabolism. We conclude that the alternative explanations most likely complement our own model, rather than contradicting or pre-empting it.

5.1 The self-control hypothesis

CGS argue that variation in self-control can explain the disproportionate weight growth in the upper tail of the distribution over the past 20 years. In their model, consumers engage in hyperbolic discounting to varying degrees. Their hyperbolic consumers are more sensitive to a decline in the time cost of food than they are to a decline in the money cost. Further, the authors argue that the decline in food's time cost has been much greater than the decline in its money price. An individual with relatively poor self-control would likely have weighed more at the initial prices, *ceteris paribus*, and would have gained more weight than others in response to declining time costs.

However, the authors do not prove these claims, and we find that such proof requires additional model restrictions. For example, in order to ensure that low-self-control individuals gain more weight

than others in response to a reduction in food's time cost, the instantaneous utility of food consumption (that is, the pure pleasure of eating at a given instant in continuous time) must have a strictly positive third derivative. Assuming (as both they and we do) diminishing marginal utility of food, this condition means that the marginal utility of food declines less steeply (in absolute terms) with each additional unit of instantaneous consumption. This condition is necessary (but not sufficient) for non-satiation in food consumption, a condition that is implicit in their analysis. While non-satiation is a standard economic assumption, it is questionable in the context of instantaneous (or near-term discrete-time) food consumption. Even granting the condition, the effect of self-control on the response to changes in the full food price—and any time-cost change implies a change in the full price—is ambiguous: High-self-control types may gain more weight than low-self-control types when the full food price falls. Our own model assumes a zero third derivative, but our results on disproportionate growth in the upper tail do not depend on this assumption.

Despite these ambiguities, we recognize that the self-control hypothesis has considerable intuitive appeal. We do not doubt that imperfect self-control routinely influences food consumption choices—recall that in our model all consumers are myopic. Indeed, evolutionary biologists argue that humans are hard-wired to take advantage of available food in the present, given that for much of evolutionary history we faced scarce and unpredictable food supplies. To get a sense of the model's potential empirical reach, we estimate the effects of variation in self-control on weight levels and price effects for a parametric model based on the assumptions of CGS. In the CGS model, the degree of self-control is captured by the parameter, γ, which determines the discrepancy between near-term and long-term discount rates. Maximum self-control corresponds to $\gamma = 1$, and minimum self-control corresponds to $\gamma = 0$. In the former case, the consumer behaves according to the standard rational choice model, with purely exponential discounting, and in the latter case the consumer is perfectly myopic. While self-control may have a genetic foundation (Gale et al. 2004, Spiegelman and Flier 2001), we know of no attempt to describe its empirical distribution. We proceed with the naive assumption that the self-control parameter described above, γ, is uniformly distributed on the closed interval $[0,1]$. We use the

first-order condition in CGS's equation (4) to describe the weight distribution induced by variation in γ, at varying time-cost values. The calibration respects the explicit and implicit assumptions in CGS, including those pertaining to the derivatives of instantaneous food utility, and isolates the key differences between the CGS model and our own. For metabolism, we adopt the linear, non-stochastic model for women cited in CGS, which they attribute to Schofield et al. (1985).

We set the parameter λ, which affects the importance of hyperbolic discounting, to 1000, the value suggested in the text as a reasonable lower bound. We experiment with three different values for the food time cost, denoted τ, following their hypothesis that declines in time cost were the primary factor in the rise of obesity. The CGS model does not specify values for τ, but the parameter loosely corresponds to the average time needed to prepare a meal or snack. Accordingly, we consider values (in minutes) of 20, 10, and 5. For the decline from 20 to 10 minutes, the average weight gain is small, about 2.5 pounds, from an initial average of 148. However, weight gains are larger in the upper tail: The 95th-percentile weight increases by 9 pounds, and the distribution has a large positive skew. The time-cost decline from 10 minutes to 5 produces much more dramatic weight gains, counter to intuition. Average weight increases from 150.5 to 223.5 pounds, while 95th-percentile weight climbs from 157.5 to 494.6 pounds. At any price level, the simulated distributions are much more skew than the empirical distributions observed in the NHANES surveys, but this outcome depends on the assumed distribution of the self-control parameter. The fact that the sensitivity to time-cost changes moves inversely with time costs does not, however, depend on the calibration parameters. In contrast, our model predicts that price sensitivity varies directly with the price level. Therefore, the CGS model predicts accelerating growth in obesity as food time costs continue to fall, whereas our framework predicts that obesity will continue to grow, but at a slower pace.

5.2 Rational food addiction

Cawley (1999) argues that calorie consumption exhibits properties consistent with a rational addiction model. He also cites evidence that the propensity toward addiction to specific substances may vary across individuals, based on genetics. To determine the potential contribution of addiction to changes in the shape of the weight distribution over time, we simulate weight distributions for a population of individuals with varying propensities for food addiction. The model we adopt builds on the standard, two-period, forward-looking model with zero discounting, discussed above in Section 4.4. Following Becker and Murphy (1988) and Cawley (1999), we model food addiction by letting the linear coefficient on the utility of current food consumption, denoted α in our prior descriptions, be an increasing function of beginning-of-period body weight, where the function varies across individuals. That is, we replace α with the function $\alpha_i(W_{i,t-1}) = \alpha + \beta_i W_{i,t-1} =$. For $\beta_i > 0$, this model captures the reinforcement aspect of addiction: The greater the stock of past food consumption (a stock embodied, literally, in weight), the greater the marginal utility of food. For $\beta_i \leq 0$, the marginal utility of food is non-increasing in weight, and food is not addictive. Our model assumes $\beta_i = 0$ for all individuals. We vary the propensity to food addiction by letting β_i follow a normal distribution with mean zero and standard deviation of $\frac{1}{425}$, such that half the population is prone to food addiction to varying degrees. We let metabolism vary linearly with weight according to our fit of the Schofield data, but we suppress idiosyncratic metabolic variation in order to isolate the effects of the propensity to addiction.

In the simulation results shown in Table 1, we see that the distribution does exhibit positive skewness. Individuals with a greater propensity to addiction weigh more than others at a given price, *ceteris paribus*, and they gain more weight than others in response to a price decline. In order to avoid corner solutions (specifically, zero food consumption), the standard deviation of β_i must be restricted, and as a result, the predicted variance of weight is very low relative to the true value. All of these results depend on the assumed distribution of the addiction parameter, a distribution that has not been empirically estimated. The rational addiction model also predicts that the price elasticity of demand for addictive

goods will be greater in the long run than in the short run. However, this prediction depends on whether calories are addictive for the representative individual, and the empirical evidence on this question is not conclusive.

6 Conclusion

This paper presents a new framework for relating the recent increases in obesity rates to falling food prices. We focus on explaining changes in the shape of the weight distribution and, in particular, the disproportionate growth in the distribution's upper tail. We explain a substantial portion of this growth, using a model that interacts the effects of economic change, in the form of falling food prices, with social and physiological processes. In the social process, the body-weight standard becomes more relaxed as average weight increases in response to a food price decline; the relaxed standard then leads to further weight increases. At the same time, metabolism is concave in body weight, such that as weight increases, a given increase in calorie consumption leads to greater weight gain. The aspiration to weigh less than the average individual in the population, together with the concave metabolic function, predicts a right-skewed weight distribution as well as greater price-induced weight gains for initially heavier individuals. The simulations show that the model does a very good job of capturing both the shape and the movement of the distribution over time.

We find evidence of shifting norms in the BRFSS data on "desired weights," as well as in the documented increases in the average dimensions of given nominal sizes of women's clothing. If such shifts occur with a lag, the dynamic analysis shows that the adjustment to a new long-run equilibrium, following a price decline, may take years. We find support for this lagged effect in the recent evidence that average weight and obesity rates are continuing to increase despite the fact that food price declines (including preparation costs) appear to have levelled off since the mid 1990s.

It has not been our primary goal in this paper to explain cross-sectional variation in weight levels, and we have deliberately ignored many important sources of weight variation. However, our findings

suggest that metabolic variation alone induces substantial weight variation across individuals and that the weight distributions derived from an empirically grounded metabolism model strongly mirror the persistent qualitative features of the observed weight distributions. The growth of obesity has been too dramatic and has occurred too rapidly to be explained by changes in the gene pool. Yet our findings suggest that biological processes have played a role in this growth: Given the concave relationship between body weight and metabolism, the rightward shift of the weight distribution means that the realized marginal effects of calorie consumption on weight are now greater on average than in the past, even with no genetic change.

The concave metabolism model has further implications. An individual who predicts her future basal metabolic rate at a higher weight, based on the curve's slope at her current weight, will systematically overestimate it, and therefore will underestimate the long-term weight gain associated with a permanent (non-marginal) increase in calorie consumption. Scientific estimates of the metabolism-weight relationship from samples lacking overweight individuals have done exactly this—and have found a linear relationship that overestimates BMR at out-of-sample weights. Lacking complete knowledge of the weight-metabolism curve, even forward-looking individuals may experience regret over past eating decisions, as myopic individuals do. Even if people learn the model eventually (through revealed weight gains, for example), it may be more difficult to reverse such gains than originally anticipated. Under these constraints on rationality, even forward-looking individuals may be made worse off by a food price decline. These findings suggest a need for better public education, as well as better medical counselling, concerning the relationship between body weight, body composition, and calorie-burning.

Both a rational addiction framework and a framework involving variation in self-control can generate qualitative predictions of disproportionate growth in the upper tail of the weight distribution in response to price declines. However, these alternative explanations appear less robust than ours, because they rely more heavily on assumptions about utility functions. In addition, it is difficult to assess the quantitative contributions of self-control and addiction to variation in weight gain, because the distributions of self-control and propensity to addiction are not well understood. We look forward to more

research into the relative contribution of physiological, social, and economic forces to changes in the weight distribution over time.

Our model of endogenous norms predicts that population weights and obesity rates should continue to grow if food prices continue to fall, but that marginal price effects on calorie consumption should be smaller, the lower the initial price level. In this framework, the limits on weight and obesity growth depend on the slope of the weight-metabolism curve at high weight levels. If the curve continues to follow our fitted model, and provided calories are not addictive, the increases in average weight and the obesity rate should eventually level off, even if norms are flexible and prices continue to fall. If instead the metabolism curve becomes flat above a given (humanly feasible) weight threshold, calorie consumption above the maximal BMR value would cause unbounded increases in weight. Existing research on metabolism suggests the possibility of such thresholds (Cunningham 1991, Horgan and Stubbs 2003), but results are inconclusive, and further research on metabolism among obese subjects is clearly warranted.

Thinking beyond the model, are there forces or policy interventions that might be expected to lead to a slowing or reversal of current trends? In the case of smoking, increases in taxes and a shift in the social judgment of smoking led to significant declines in consumption. Food taxes, even if justified by bounded rationality or cost externalities, are likely to be politically infeasible, given that food, unlike tobacco, is a necessity. As for social acceptance, the trend has been toward fat acceptance and accommodation of obesity, rather than censure, consistent with our norms hypothesis. Hospitals have added larger beds and other specialized equipment for obese patients and the visibility of plus-size models has increased (Henderson 1997). Norms restricting the proper times and places for eating have also broken down. Recent scientific research even suggests that the medical definition of "healthy" BMI may need to be adjusted upward to accurately reflect the relative mortality risks within different ranges of BMI, risks that appear to have shifted, in part, as a result of advances in the treatment of obesity-related disorders (Flegal et al. 2005).[25]

[25]These findings have not been universally accepted. For example, a symposium at the Harvard School of Public Health,

The latter evidence suggests that there has been technological adaptation, in addition to social adaptation, to the shift in the weight distribution. That is, the increase in average weight has emerged as a possibly permanent and relatively benign development. As for a reversal of the growth of extreme obesity, a condition that still entails high morbidity and mortality risks, a medical breakthrough is more likely to be the catalyst than is behavior modification. The social and economic factors promoting obesity growth are likely to persist (and appear to be emerging in countries outside the United States as these countries follow a similar development path), and increasingly stern public health warnings are unlikely to have a significant impact. Furthermore, individuals in the upper tail of the BMI distribution are likely to be at a genetic disadvantage and therefore less likely than others to achieve weight loss through behavioral change. In addition, we expect the social stigma associated with extreme obesity, at least, to remain sufficiently high for the foreseeable future to sustain a considerable demand for weight-loss technologies in addition to treatments for obesity-related disorders.

in 2005, argued that the results suffered from biases due to reverse causality (for example, illness-causing weight loss) and residual confounding (for example, the correlation of smoking status, an omitted variable, with both lower body weight and higher mortality risk). However, the Flegal team has found its original results to be largely robust to these criticisms. See http://www.cdc.gov/nchs/products/pubs/pubd/hestats/excess deaths/excess deaths.htm.

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Mathematical Appendix

In this appendix we provide brief verification of the assertions of the existence and convergence statements of individual stable weight laid out in Section 3.

The optimal one-period choice of food and non-food consumption, beginning from any initial weight value, must satisfy equations (5), (8), and (9), as well as second-order conditions. The three equations can be combined and rewritten as the following optimality condition on end-of-period weight, W_t, for any initial weight W_{t-1}.

$$U_F(F(W_t^*|W_{t-1}))\frac{dF}{dW} - pU_C(C(W_t^*|W_{t-1}))\frac{dF}{dW} = 2J(W_t^* - M). \tag{14}$$

The left-hand side of equation (14) represents the marginal effect on one-period utility of end-of-period weight, deriving from the changes in food and non-food intakes consistent with a marginal change in the final weight. The right-hand side represents the marginal effect of end-of-period weight on the cost of deviating from the norm. The partial derivative U_F is evaluated at the food level consistent with the beginning and ending weights, where this food level is denoted $F(W_t|W_{t-1})$. The partial derivative U_C is evaluated similarly. The expression $\kappa := \frac{dF}{dW} > 0$ represents the increase in food consumption needed to achieve a marginal weight gain, holding basal metabolism fixed. This is an identical constant for all individuals, representing the conversion rate of calories into body weight, netting out the calories consumed in digestion. Values for the left-hand side of (14) depend on both the initial weight and the final weight, but values for the right-hand side depend only on the final weight.

Existence of a stable weight

A stable weight must satisfy the equation above as well as the condition that $W_t^* = W_{t-1}$. To check for existence of such a weight, impose the condition that $W_t = W_{t-1}$ and determine whether (14) has a solution. Consider the values $[U_F(F(W|W)) - pU_C(C(W|W))]\kappa$. The expression gives the net marginal

benefit of weight gain beginning from any weight, W, where each W is potentially an initial weight value at some point in time. If, starting from some initial weight, the net marginal benefit of weight gain happens to be equal to the marginal cost of weight gain, it is optimal to maintain the initial weight. Since any stable weight must satisfy this property, the locus of these net marginal benefits will be termed the "sustainability locus," and it will determine stable weight for any M. Values on the locus are initially positive, given the high marginal utility of food at the food intake consistent with just maintaining a very low weight, and the low marginal utility of non-food consumption. The function decreases in weight, eventually becoming negative. This decline occurs because metabolism increases in weight, and therefore the food required to maintain weight is increasing; as required food intake increases, the marginal utility of food decreases, and the marginal utility of non-food consumption increases. The right-hand side is initially negative (assuming M is greater than some minimal viable adult weight), but becomes positive as weight gets above M. Thus, there is a unique weight, $W^S(M)$, characterized by

$$[U_F(F(W^S|W^S)) - pU_C(C(W^S|W^S))]\kappa = 2J(W^S - M). \tag{15}$$

Convergence to a stable weight

To show convergence to this stable weight, consider one-period optimization from any initial weight, W_0, as illustrated in Figure 9. Consider the marginal net benefit of end-of-period weight as seen in the diagram. Values on this curve are again initially positive, and become negative as final weight, W_1, increases. This marginal benefit curve, however, has a steeper negative slope than the sustainability locus. This is because, as weight increases from W_0 to some W_1, the marginal net benefit of further weight gain evaluates U_F at $F(W_1|W_0)$, whereas the locus evaluates U_F at $F(W_1|W_1)$. Since the former food quantity is greater than the latter, the net marginal benefit of food (and of weight gain) is less than the value on the sustainability locus at W_1. The difference in these food levels arises because metabolism, measured in pounds of weight burned per week, grows less than proportionally to weight

itself. For the same M the marginal deviation cost behaves exactly as above. Thus, for any given initial weight there is a unique intersection point that determines $W_1^*(W_0)$. Second-order conditions are satisfied at this value, given the strict concavity of the optimization problem in F and C. If W_0 lies below the stable weight defined by (15), the analysis in the previous paragraph implies that, beginning at W_0, a final weight of W_0 cannot be optimal. At this point, the marginal benefit of weight gain exceeds the marginal cost, so it is optimal to gain weight. However, the optimal weight gain is less than the difference between W_0 and the stable weight. This also follows from the fact that the marginal benefit of within-period weight gain declines more rapidly than the values on the sustainability locus. The diagram depicts the relationship between these two curves, indicating that $W_1^* < W^S$.

Referring to Figure 9, beginning from weight W_0, let the optimal ending weight be W_1, where the latter satisfies $[U_F(F(W_1|W_0)) - pU_C(C(W_1|W_0))]\kappa = 2J(W_1 - M)$. To iterate, let the individual begin at W_1 and again solve the one-period problem. Now the individual evaluates the marginal benefit of weight gain relative to this new initial weight. For the first increment this is just the value on the sustainability locus at W_1. Thus, we have a new within-period net marginal benefit curve intersecting the sustainability locus at this point, where the new curve lies to the right of that for the previous (lower) initial weight. Therefore, when the individual wakes up at weight W_1, the marginal benefit of weight gain once again exceeds the marginal cost, and additional weight gain is optimal within the new period. However, since W_1 is greater than W_0, the marginal benefit of weight gain beginning from W_1 is less than that beginning from W_0, and the marginal cost is higher, implying a smaller optimal weight gain from W_1 to W_2, as shown in the diagram. Again, the individual winds up below the stable weight. The same logic applies at the next iteration, during which the individual again gains weight, but less than in the previous period. Weight gain occurs as long as $W < W^S$, yet converges to zero and ceases when W reaches the stable value. Had the initial weight been greater than the stable weight value, it can be shown similarly that the individual would optimally have lost weight period by period until converging to the stable weight.

Existence of and convergence to a stable weight norm

The graphical analysis in Figure 10 illustrates the relationship between the value of M and the value of stable weight. We can identify the stable weight for a given value of M by plotting, against W, each of the functions $[U_F(F(W|W)) - pU_C(C(W|W))]\kappa$ and $2J(W-M)$, and finding the value of W, denoted $W^{S*}(M)$, for which they intersect. The diagram illustrates the respective stable weight values for weight norm values labelled M and M'. The greater the value of M, the smaller the value of $2J(W-M)$ for each value of W, and the greater the stable weight. By inspection, we find that the rate of increase in stable weight with an increase in M is less than unity, because the sustainability locus has a negative slope. This less-than-proportionate increase in stable weight with an increase in M implies that the model satisfies the "moderate social influence" condition (Glaeser and Scheinkman 2002), a condition that guarantees the existence of an equilibrium weight norm. The value of dW^S/dM approaches 1 from below as J approaches infinity, so the existence condition is satisfied for all finite values of J. Simulations using FORTRAN90 code confirm uniform convergence to equilibrium.

Table 1: Summary of Weight Distributions

Distribution	**Mean (SD)**	**Min**	**Max**	**Median**	**95th**[a]	**99th**[b]	**Skewn.**[c]	**Obese**[d]	**Norm**[e]
				Empirical Distribution					
				Women (age 30-60)					
NHANESII, 1976-1980	148.4 (34.0)	80	360	141.0	215	258	1.356	18.9	
NHANESIII, 1988-1994	157.4 (39.5)	77	470	149.6	231	290	1.207	28.0	
NHANES99, 1999-2000	168.4 (45.6)	84	420	159.7	251	305	1.178	35.7	
BRFSS, 1990	148.4 (31.6)	73	434	142.9	205	256	1.429	14.0	
BRFSS, 2002	161.0 (38.6)	56	603	153.2	236	288	1.425	24.7	
				Men (age 30-60)					
NHANESII, 1976-1980	177.3 (29.8)	100	350	174.3	230	264	0.615	13.7	
NHANESIII, 1988-1994	185.4 (37.7)	90	532	180.2	251	317	1.476	21.6	
NHANES99, 1999-2000	191.9 (43.4)	94	425	184.4	277	338	1.183	27.3	
BRFSS, 1990	182.6 (31.7)	69	433	179.1	241	283	1.017	15.3	
BRFSS, 2002	194.5 (39.7)	49	629	189.4	267	325	1.289	26.3	
				Simulated Distribution (Women age 30-60)					
Moving Norm (Linear, P=$50)	148.0 (30.0)	51	273	146.9	199	223	0.240	18.6	130.2
Moving Norm (Linear, P=$40)	157.2 (30.4)	59	284	157.2	210	234	0.226	28.7	138.3
Moving Norm (Linear, P=$32)	166.0 (30.6)	64	292	164.9	218	241	0.217	37.6	146.1
Moving Norm (Log, P=$50)	148.4 (32.1)	67	332	144.6	207	241	0.772	18.9	130.6
Moving Norm (Log, P=$40)	159.8 (34.4)	72	354	155.6	222	258	0.756	29.4	140.6
Moving Norm (Log, P=$32)	168.6 (36.2)	76	370	164.4	234	272	0.743	38.6	148.4
Fixed Norm (Log, P=$50)	148.4 (32.1)	67	332	144.6	207	241	0.772	18.9	130.6
Fixed Norm (Log, P=$40)	157.6 (34.0)	71	349	153.7	219	255	0.758	27.2	130.6
Fixed Norm (Log, P=$32)	164.6 (35.3)	74	362	160.3	229	266	0.748	34.3	130.6
Forward-Looking (Linear, P=$50)	145.0 (25.1)	61	248	144.2	144	188	0.203	12.4	130.6
Forward-Looking (Linear, P=$32)	157.4 (25.4)	71	261	156.7	200	219	0.185	24.4	130.6
Forward-Looking (Log-Linear, P=$50)	144.5 (25.8)	72	274	142.2	191	215	0.557	12.4	130.6
Forward-Looking (Log-Linear, P=$32)	157.5 (27.5)	79	294	155.1	206	232	0.528	24.4	130.6
Rational Addiction (Linear[f], P=$50)	145.9 (18.8)	98	276	143.6	180	201	0.820	7.5	130.6
Rational Addiction (Linear[f], P=$32)	158.8 (20.8)	106	304	156.2	196	220	0.839	20.2	130.6

Note: [a]95th Percentile. [b]99th Percentile. [c]Skewness:= $\frac{\Sigma_i\left[(X_i-\mu)^3\right]}{(N-1)\sigma^3}$ for univariate data $X_1, X_2, ..., X_N$ where μ and σ denote mean and standard deviation. [d]Percentage with BMI of 30 or above (more than 174.5 pounds for women of average height in the simulations). [e]Population Weight Norm, see text for details. [f]Linear homoskedastic metabolism.

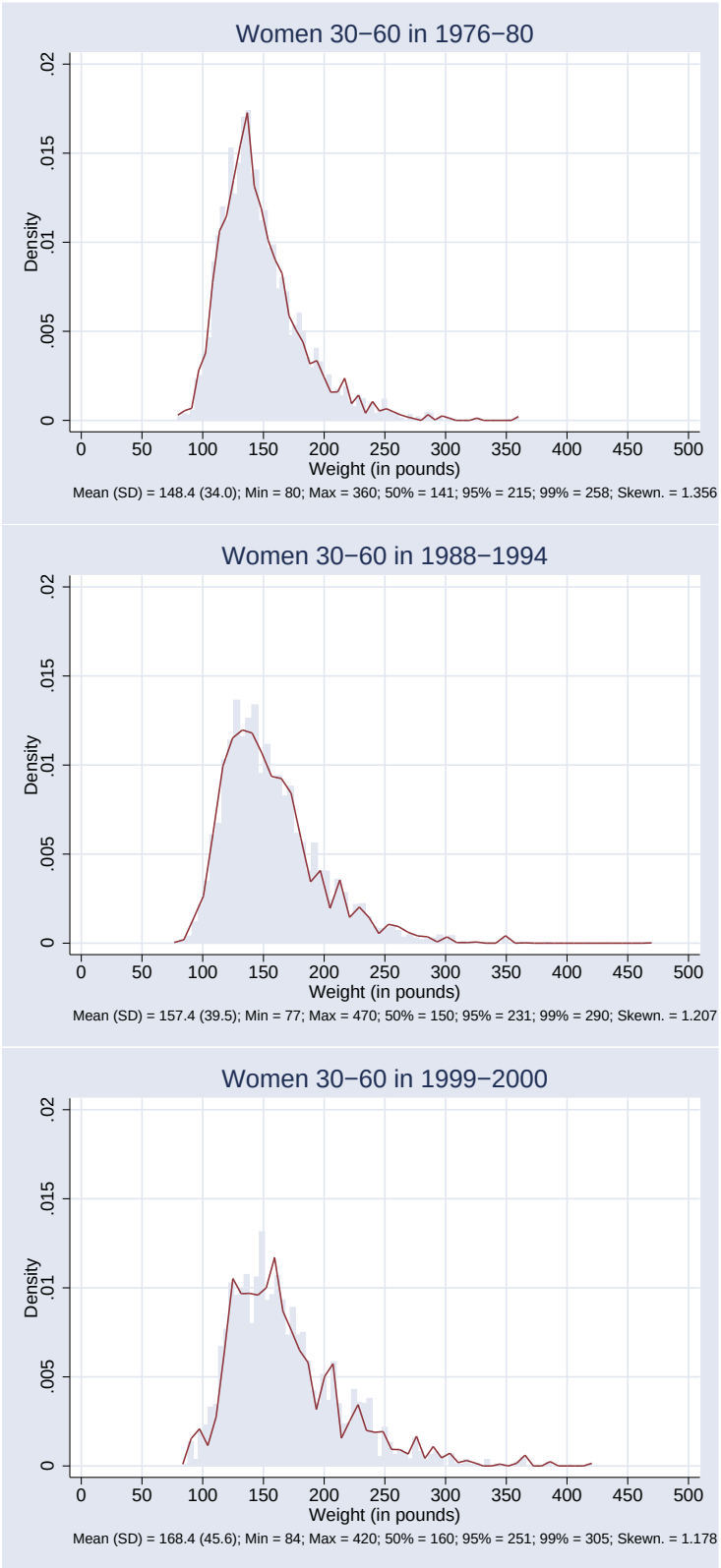


Figure 1: Female Weight Distribution 1976-1980, 1988-1994, and 1999-2000, with Kernel density estimate plot (Source: NHANES II, III, and 99)

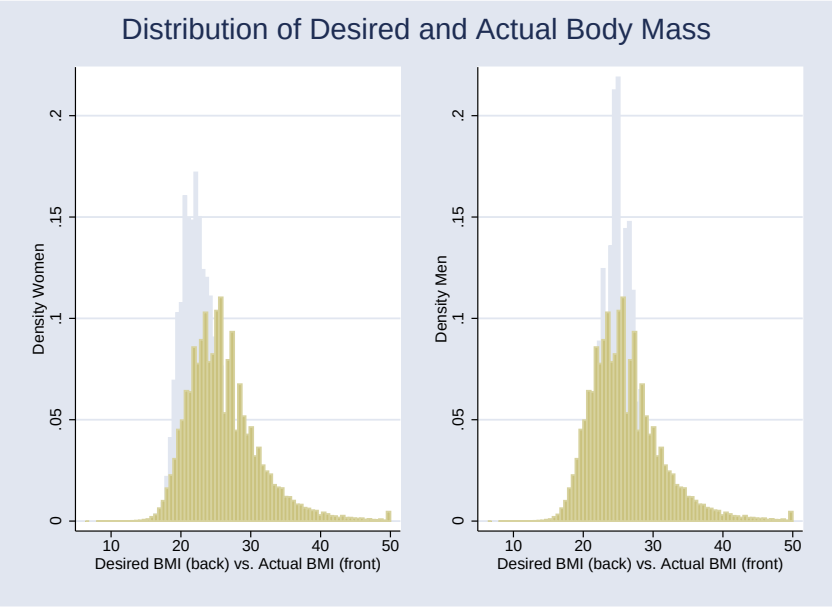


Figure 2: Actual and Desired BMI by Gender (Source: BRFSS various years; BMI>50 set to 50)

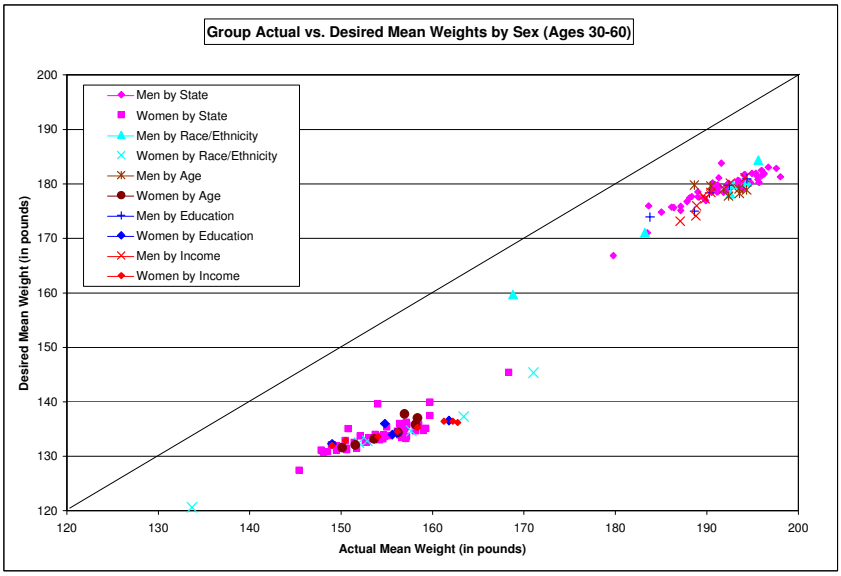


Figure 3: Relationship between Desired and Actual Weight by Groups (Source: BRFSS various years)

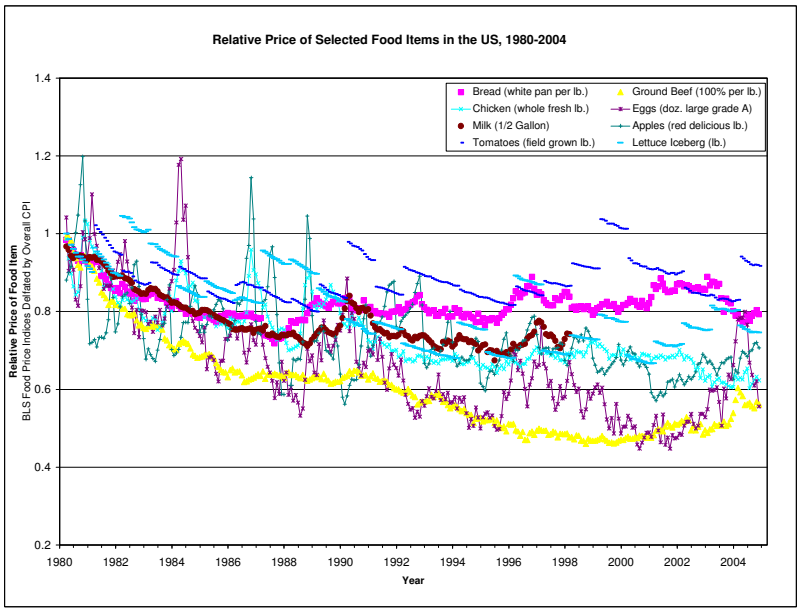


Figure 4: Relative Prices of Selected Consumer Food Items (1980=1; Source: Bureau of Labor Statistics)

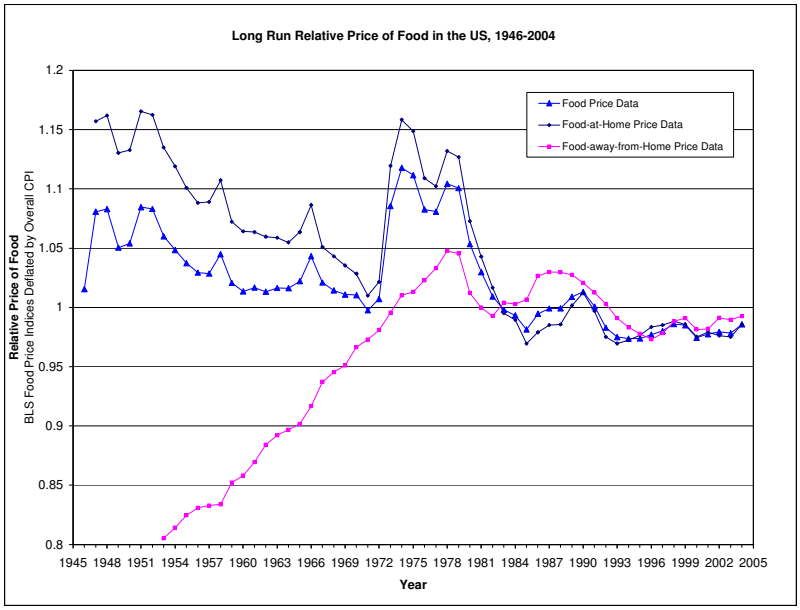


Figure 5: Long-Run Trend in Consumer Food Prices (1982-84=1; Source: Bureau of Labor Statistics)

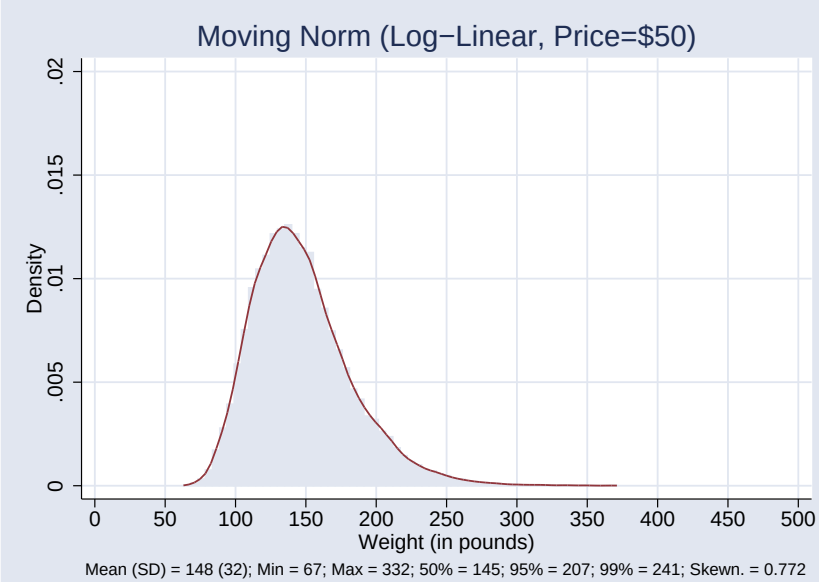


Figure 6: Moving Norm, Log-Linear Metabolism (with Kernel density estimate plot)

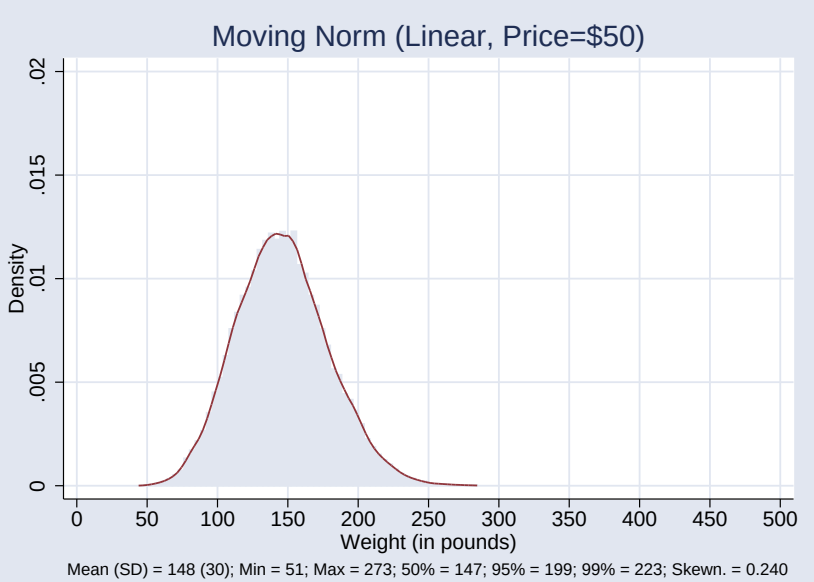
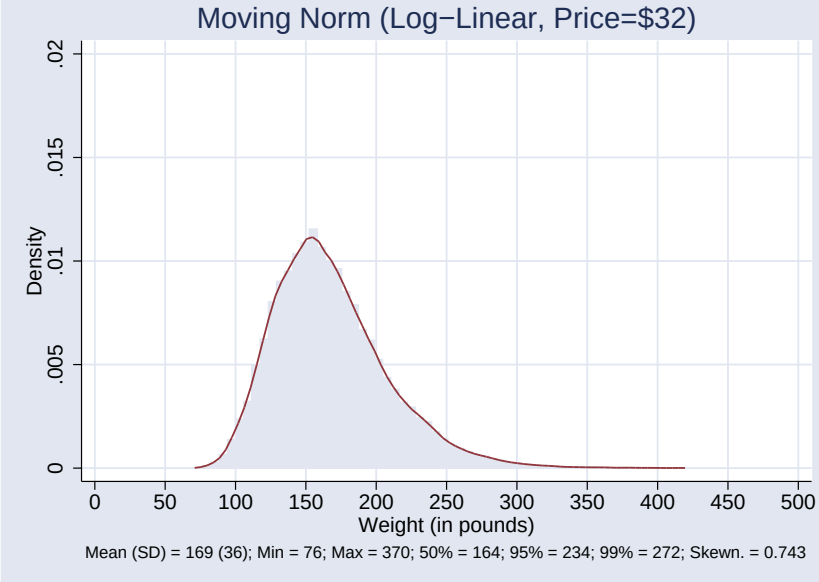
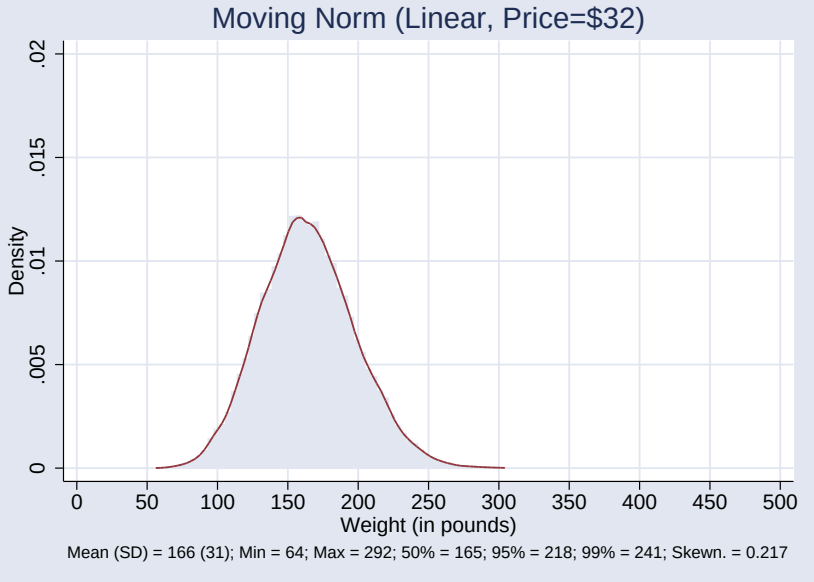


Figure 7: Moving Norm, Linear Metabolism (with Kernel density estimate plot)





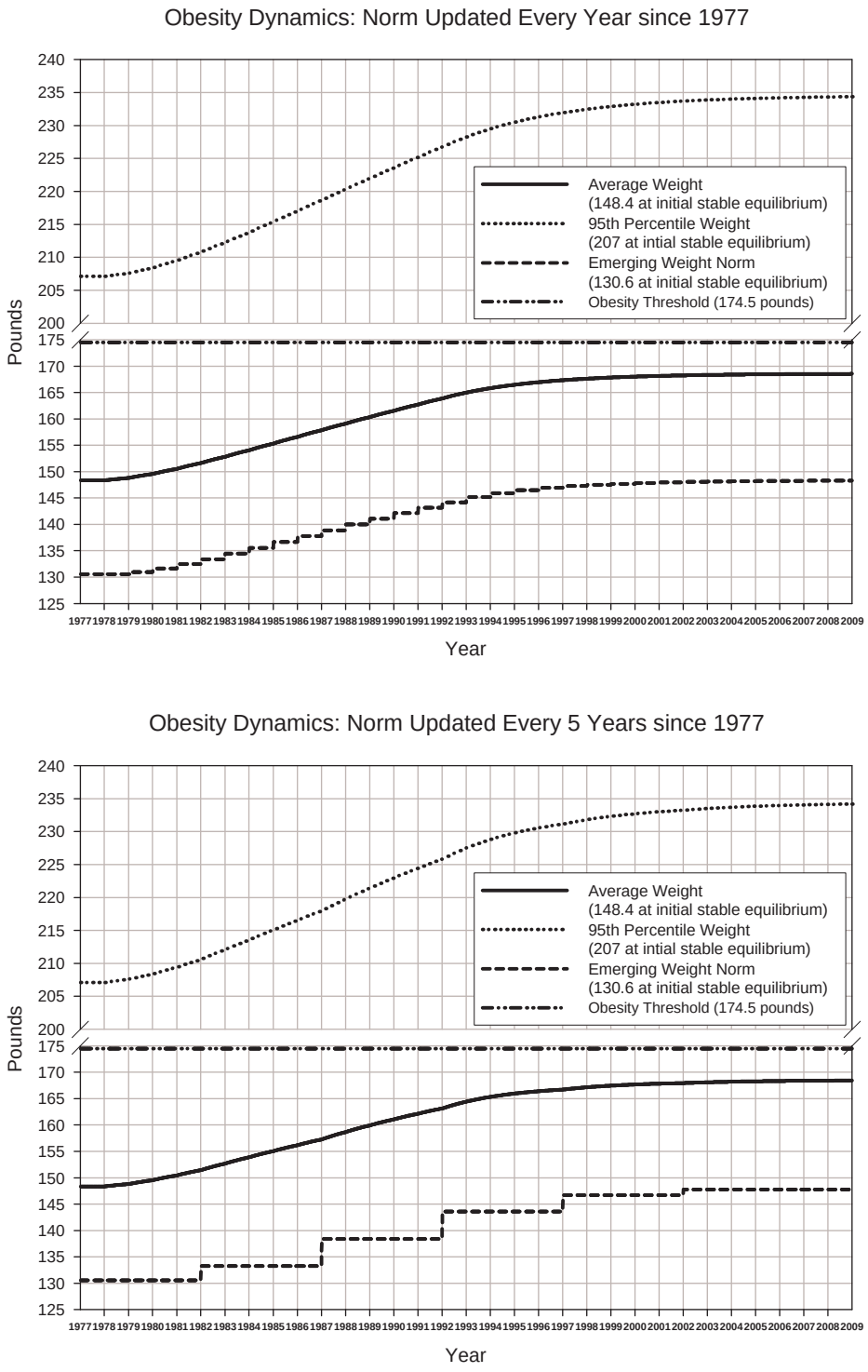


Figure 8: Obesity Dynamics: Annual vs. Five-Year Norm Updating, Log-Linear Metabolism (Gradual Price Decline 1977-1993)

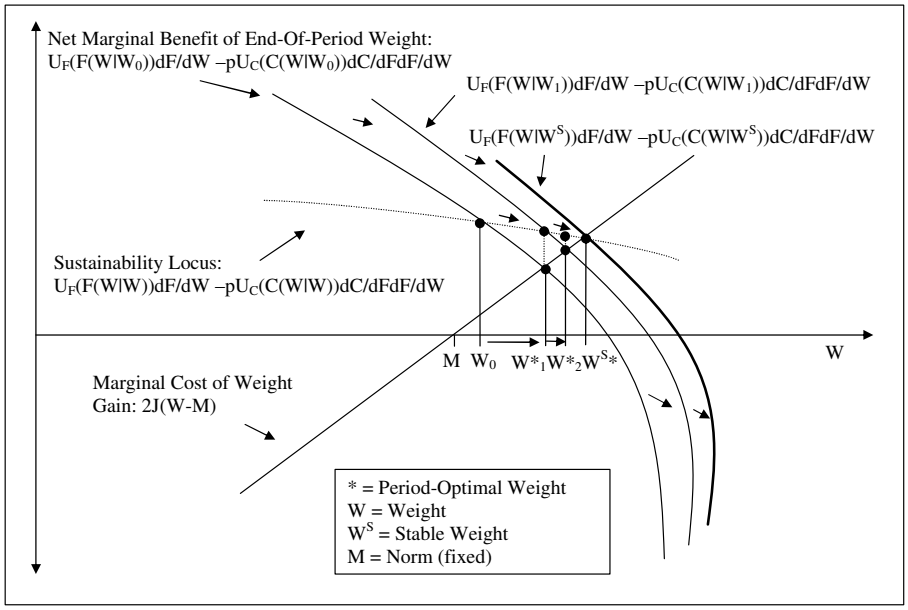


Figure 9: Illustration of Convergence to Stable Period-Optimal Weight (Fixed Weight Norm)

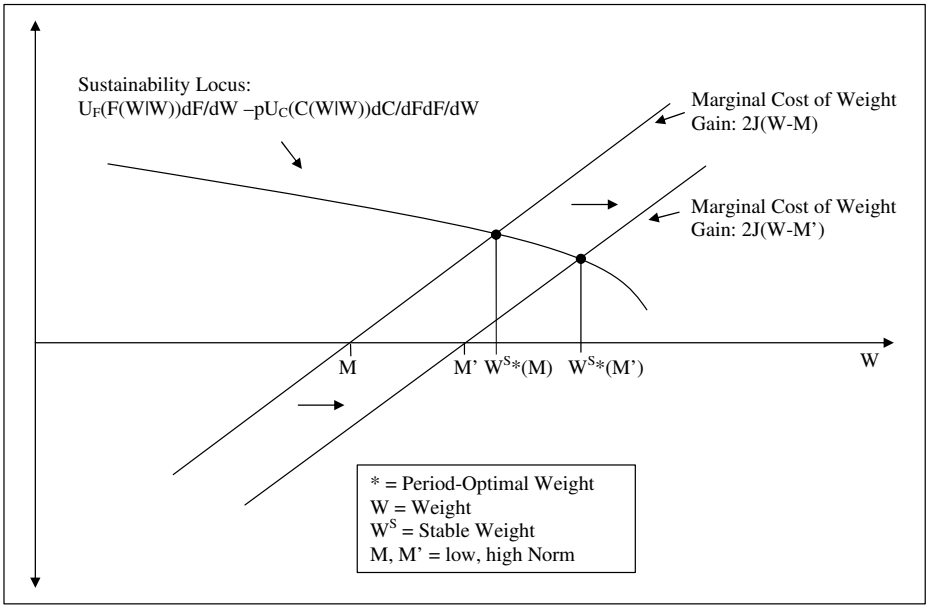


Figure 10: Illustration of Stable Period-Optimal Weight under a rising Weight Norm