SOCIAL DYNAMICS OF OBESITY

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We explain the recent increases in obesity in the United States with a model involving falling food prices, endogenous social body weight norms, and heterogeneous human metabolism. Calibrating an analytical choice model to American women in the 30- to 60-yr-old age bracket, we compare the predicted weight distributions to National Health and Nutrition Examination survey data spanning (intermittently) the years 1976–2000. The model, the first to describe explicitly complete weight distribution dynamics for this group, predicts average weights and obesity rates with considerable accuracy and captures a significant portion of the recent growth in upper quantile weights. (*JEL* D11, I12, Z13)

I. INTRODUCTION

The startling growth rates of average weight and obesity prevalence in the United States over the past 20–30 yr 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. The weight distribution in the United States has not only made a considerable shift to the right—average adult female weight, for example, increased by 20 pounds, or 13.5%, between 1976–1980 (National Health and Nutrition Examination Survey [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%, from 215 to 251 pounds, and 99th-percentile weight increased 18.2%, from 258 to 305 pounds, as shown in Table 1 and Figure 1.[1,2] The official definition of obesity employed by the Centers for Disease

1. 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.

2. The empirical findings on body weight presented in this paper are based on samples of 30- to 60-yr-old Americans from two surveys administered by the CDC : BRFSS and waves II, III, and 99 of the NHANES (NHANES II, NHANES III, and NHANES 99). The BRFSS is a large random sample of the resident population 18 yr and older in participating states of the United States. Self-reported information on 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 following the approach by Chou, Grossman, and Saffer (2004) using NHANES III data for the 30- to 60-yr-olds. NHANES II, NHANES III, and NHANES 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.

ABBREVIATIONS

BMI: Body Mass Index
BMR: Basal Metabolic Rate
BRFSS: Behavioral Risk Factor Surveillance System
CGS: Cutler, Glaeser, and Shapiro (2003)
CDC: Centers for Disease Control and Prevention
CPI: Consumer Price Index
NHANES: National Health and Nutrition Examination Survey
TEE: Total Energy Expenditure

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TABLE 1
Summary of Empirical Weight Distributions

Distribution	Mean[a]	Minimum	Maximum	Median	95th[b]	99th[c]	Skewness[d]	Obese[e]
Women (aged 30–60 yr)								
NHANES II, 1976–1980	148.4 (34.0)	80	360	141.0	215	258	1.356	18.9
NHANES III, 1988–1994	157.4 (39.5)	77	470	149.6	231	290	1.207	28.0
NHANES 99, 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 yr)								
NHANES II, 1976–1980	177.3 (29.8)	100	350	174.3	230	264	0.615	13.7
NHANES III, 1988–1994	185.4 (37.7)	90	532	180.2	251	317	1.476	21.6
NHANES 99, 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

[a]Standard deviation in parentheses.

[b]95th Percentile.

[c]99th Percentile.

[d]Skewness : $= \sum_i[(X_i - \mu)^3]/((N-1)\sigma^3)$ for univariate data $X_1, X_2, \ldots, X_N$ where μ and σ denote mean and standard deviation, respectively.

[e]Percentage with BMI of 30 or above (more than 174.5 pounds for women of average height in the simulations).

Control and Prevention (CDC) and by the World Health Organization 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. For a 5′4″ woman, 175 pounds or greater classifies as obese, and for a 5′9″ man, the obesity threshold is 203 pounds.[3]

A number of papers in economics have sought to explain obesity growth 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 and reductions in physical labor on the job.[4] The theoretical models offered study representative agents and speak primarily to secular trends in average weight. Although the model of Cutler, Glaeser, and Shapiro (2003) (henceforth CGS), emphasizing 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 not well understood. The prior works abstract from biological heterogeneity—acknowledged by Cawley (1999) and Chou, Grossman, and Saffer (2004) as a major factor in weight variation—and either ignore or hold fixed social influences on weight determination. In this paper, we argue that a richer description of the social and biological determinants of weight gain—interacted with falling food prices—contributes substantially to a more complete understanding of the evolution of the weight distribution over the past 30 yr.

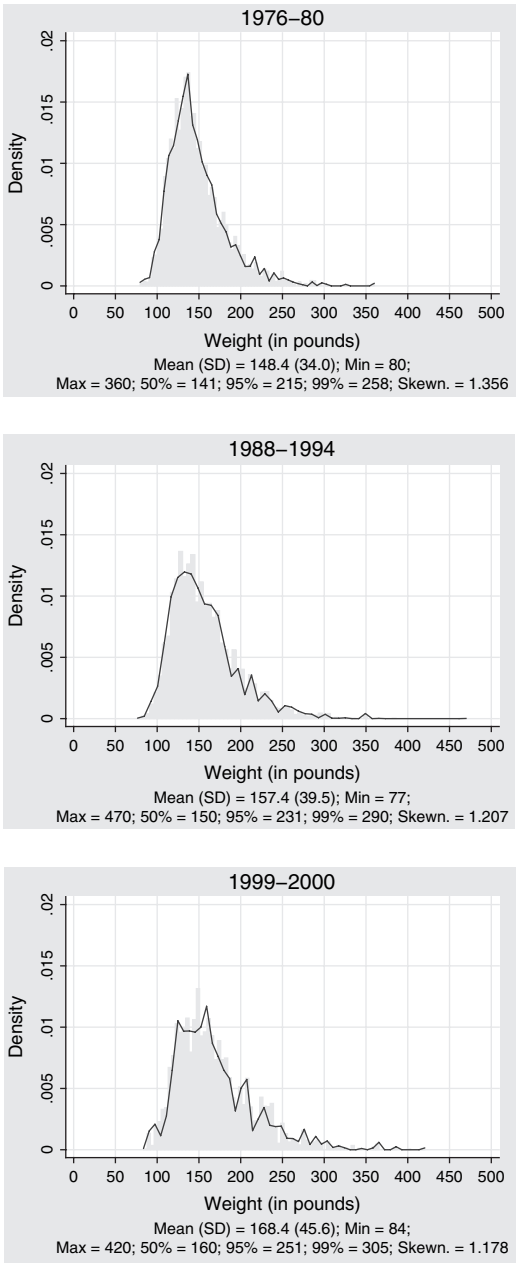
In our choice model, utility depends on food and nonfood consumption and on how individual weight compares with a social weight standard or norm. Extensive research in the fields of social psychology and sociobiology asserts that standards of physical appearance are powerful motivators of human behavior, although these disciplines may disagree on the forces that determine the content of such standards.[5] Previous models of weight determination have incorporated

3. 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 risks, as explicated by Kuczmarski and Flegal (2000). Several Web sites offer simple BMI calculators. See, for example, http://www.cdc.gov/nccdphp/dnpa/bmi/.

4. The relevant papers include Chou, Grossman, and Saffer (2004), CGS, Philipson and Posner (1999), and Lakdawalla and Philipson (2002).

5. Relevant papers from social psychology are Garner et al. (1980) and Mazur (1986), and for those from sociobiology, see Pinker (1999).

FIGURE 1
Historical Female Weight Distributions (Age 30–60 yr), with Kernel Density Estimate Plots



Source: NHANES.

weight standards into the utility function via various exogenous constructs: Lakdawalla and Philipson (2002) refer to an ''ideal weight''; Levy (2002) posits a ''sociocultural weight norm''; and CGS posit a social- and health-related cost of weight gain. In contrast, we posit an endogenous social weight standard that depends on aggregate behavior in the social group. This specification generates a number of predictions that differ from those that arise when norms are held fixed, and we find that the data provide stronger support for the model with flexible, socially determined norms.

Through analytical results and calibrated simulations, we illustrate how food price declines affect the entire weight distribution and describe explicit adjustment dynamics across long-run equilibria. The metabolic model and simulations are calibrated to American women aged 30–60 yr. This demographic restriction enhances the calibration's precision, because the physiological and social processes we consider are gender and age-group specific. Consistent 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 based on independently estimated trends in the full price per calorie of food, including both the money price and the time costs, the predictions match the quantitative changes in average weight and the obesity rate for this group with considerable accuracy. Counterfactual simulations in which weight norms are fixed do not explain the data as well. 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 yr, 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 is relatively easy (yet expensive) to measure. Using a well-known data set containing direct observations of BMRs, we estimate parametric models of metabolism in relation to body weight, including a description of its idiosyncratic component. By embedding the metabolic model into the economic choice

model, we can describe complete weight distributions at each food price. More important than capturing cross-sectional weight variation (and metabolic variation appears capable of explaining a substantial portion of the latter), the metabolism model holds nonobvious consequences 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.

Social comparison in the model implies that individuals aspire to weigh less (by some fraction) than average weight in the population at a given time. This endogenous weight aspiration therefore increases as food prices fall, because the price decline causes 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 dictate weight aspirations. The data on desired weight come from the CDC's Behavioral Risk Factor Surveillance System (BRFSS), which contains self-reported desired weights and actual weights for the same individuals.[6] While the data are not longitudinal, observations from different survey years are instructive of overall trends. In 1994, average weight for an American woman was 147 pounds, and the average desired weight was 132 pounds. By 2002, the average had increased to 153 pounds, and average desired weight had increased to 135 pounds. These figures—which follow similar patterns expressed in terms of BMI—suggest a reduction in (implicit or explicit) social pressure to maintain lower weights. The recent survey of weight perception by Rand and Resnick (2000) finds that 87% of Americans, including 48% of obese Americans, believe that their body weight falls in the "socially acceptable" range.

6. Self-reported weight data are known to be biased, and corrections are suggested in Chou, Grossman, and Saffer (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. This is reported in Section II. 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.

The remainder of the paper is organized as follows. Section II describes the theoretical model. Section III analyzes the comparative static effects of price on equilibrium weights, the weight norm, and welfare. In Section IV, 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 V, 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. Section VI discusses policy implications and predictions on the future of obesity.

II. THEORETICAL FRAMEWORK

Agent-Based Model

The theoretical model takes an agent-based approach, positing that genetically heterogeneous individuals interact 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. Desired weight is defined as a fraction, less than 1, 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 reference population, combines two basic assumptions: (1) in contemporary Western society, thinness (up to a point) is prized 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.[7] This specification creates room for gaps between the prevailing white Western ideal of thinness and the de facto standards to which individuals aspire, and ours is consequently not a model of the evolution of the media ideals themselves.

7. See, for example, Bernheim (1994), Brock and Durlauf (2001), Becker and Murphy (2000), and Bandura (1986), among many others.

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-yr-old women, the coefficient of variation of desired weight is 13.9%. However, the coefficient of variation of actual weight is significantly greater, at 23.1%.[8] In addition, race is a significant explanatory factor in desired weight for this sample. (Figure 2 plots mean desired weights against mean actual weights for various demographic groups.) These facts suggest the presence of a social component in the formation of weight aspirations. In assuming that the weight target is uniform across individuals, the model takes the stylized view that this demographic constitutes a coherent social group.[9] Thus, the model is likely to generate less variation than one with idiosyncratic preference shocks or multiple subgroup-specific weight targets. This approach therefore constitutes a conservative test of the explanatory power of social weight norms.[10]

Equilibrium is defined as a weight distribution and a 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.[11] Food and nonfood consumption are both good, but deviation from the reference weight is bad. A general expression of the one-period utility model is as follows:

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

F_t and C_t represent food and nonfood consumption, respectively, for period t. W_{t-1} is weight at the end of period $t-1$ and is a product of past actions. Individual heterogeneity is captured by ε_i, which is a stationary shock to basal metabolism, 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 $J(W_{it}[F_{it}, W_{i,t-1}, \varepsilon_i] - M_{t-1})^2$ gives the social interaction component, which is the cost of deviating from the reference weight, M. The subscript on M indicates that agents observe the value of the reference weight (M) at the end of the period $t-1$ and take this as fixed in the optimization; in particular, they do not forecast the value of the reference weight (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 everyone conforms 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. Stigmatization of overweight (and underweight) individuals has been documented by Myers and Rosen (1999), among others, and may entail teasing, ostracism, and discrimination in hiring. Contagion regarding eating behavior among adolescent girls has been observed by Crandall (1988). Ross (1994) 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) also find that obesity contributes to depression (while rejecting the reverse causality); however, they find that obesity does not raise depression risk significantly among African American women, a group with one of the highest obesity rates.

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

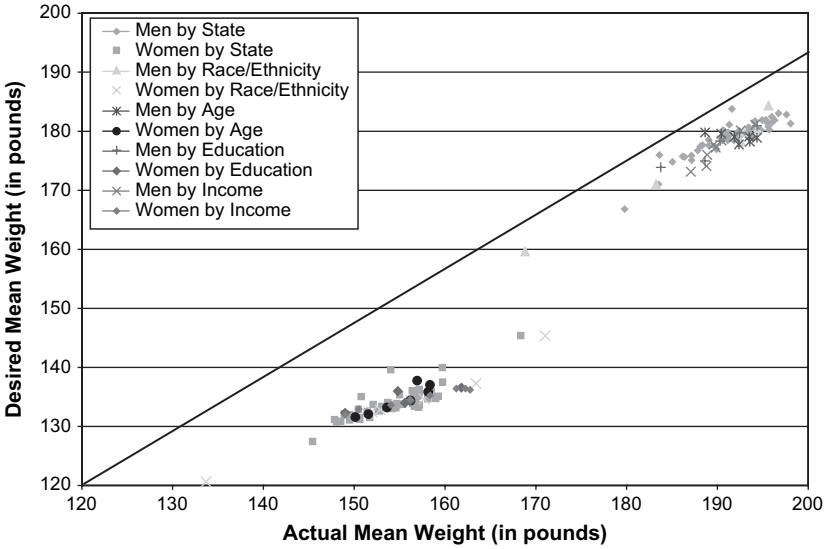
8. For men, the corresponding figures are 13.7% and 18.6%, respectively.

9. Note that we adopt a uniform weight target, rather than a BMI target. Under a uniform BMI target, weight aspirations would increase with height. However, the BRFSS data indicate that desired BMI values decrease systematically in height, suggesting that women do not fully adjust for height differences in setting weight aspirations. In addition, anecdotal evidence suggests that weight values are more salient than less-readily-observed BMI values. These facts, together with others discussed below, make it reasonable to abstract from height variation in the model.

10. In two related papers, Burke and Heiland (2006a) and Burke and Heiland (2006b), we explore the causes and implications of variation in weight norms across groups of women by educational attainment and race, respectively.

11. We will refer to the reference weight alternatively as the norm.

FIGURE 2
Group Actual vs. Desired Mean Weight by Demographics (Age 30–60 yr)



Source: BRFSS, 1990 and 1994–2002.

diabetes, heart disease, osteoarthritis, and other health conditions accelerate with increases in BMI (e.g., Must et al. 1999). In addition, mortality exhibits a U-shaped relationship to BMI among both men and women in the United States, indicating that underweight imposes similar mortality risks as overweight.[12] 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.

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 yr) in the United States, Cawley (2004) finds that an increase in weight of two standard deviations reduces the average wage by 9%.[13] 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 given value of the reference weight (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 $U[\cdot]$. The myopic specification may be taken to imply some lack of self-control, although we do not explicitly model a time inconsistency problem, as CGS do. 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

12. Two prominent studies are Troiano et al. (1996) and Flegal et al. (2005). However, Flegal et al. (2005) find that the lowest mortality rate is obtained for the group of individuals classified as overweight but not obese (BMI between 25 and 30) and that mortality rates rise as BMI gets either above 30 or below 25.

13. The results in Cawley (2004) and 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 United States is limited, these findings do not rule out the possibility of economic costs among underweight subjects.

their net energy intake.[14] We examine the robustness of our predictions to forward-looking specifications in Section V.

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

$$\max_{\{F_t,C_t\}} U_{it}[F_t, C_t | W_{i,t-1}, \alpha, \delta, \beta, J, \gamma, \rho, \varepsilon_i, M_{t-1}] = \alpha F_{it} - \delta F_{it}^2 + \beta \log[C_{it} + 1] - J(W_{i,t-1} - (7/3,500)\mathrm{BMR}(\gamma, \rho, \varepsilon_i, W_{i,t-1}) + 0.9F_{it} - M_{t-1})^2, \quad s.t.\ p_t F_t + C_t \le Y. \tag{2}$$

Within the single period, calibrated to 1 wk, the marginal utility of food, F, declines and eventually becomes negative. The expression $W_{i,t-1} - (7/3,500)\mathrm{BMR}(\gamma, \rho, \varepsilon_i, W_{i,t-1}) + 0.9F_{it} - M_{t-1}$ just amounts to the difference between end-of-period weight, W_t, and the given norm, M, as of time $t-1$, as in Equation (1).[15] In the budget constraint, p_t represents the price per unit of food, where the size of the unit is defined below and the price of nonfood consumption is normalized to 1; Y represents weekly income.

Aside from the calories burned in digestion, we assume for simplicity that calorie expenditure is limited to 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 a strong exogenous (i.e., genetic) component that has been measured in numerous studies (discussed below). Of course, physical activity is an important component of the energy balance equation, inducing calorie expenditure over and above BMR in the short run and possibly altering BMR in the long run by increasing the quantity of fat-free body mass. For our purposes in this paper, however, abstracting from the choice of physical activity level is a reasonable strategy, for several reasons. First, we note that a number of other prominent papers in the literature, including CGS, Levy (2002), and Philipson and Posner (1999), also treat calorie burning as exogenous. Second, there is evidence that overall physical activity levels in the United States have not changed much since 1975, while average calorie consumption has increased significantly since then, as shown by evidence in CGS and Robinson and Godbey (1997). Third, our predicted relationship between the metabolic endowment and body weight is likely to be qualitatively robust to the choice of exercise. Black et al. (1996) find, using extensive data from affluent societies, that BMR is strongly correlated with total energy expenditure (TEE) and that physical activity level, measured as the ratio of TEE to BMR, is orthogonal to body weight. These findings imply that, in expectation (and controlling for height), TEE is the same multiple (about 1.685) of basal metabolism for all individuals in our demographic group.

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 (for weight maintenance, loss, or gain) based on readily measured variables such as weight, height, age, and gender. One model that is often employed for predictive purposes is the weight-linear model of Schofield, Schofield, and James (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) showed that the Schofield equations substantially overestimate BMR for the obese individuals. The findings of Horgan and Stubbs, as well as the reduced form of the Cunningham (1991) model, 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

14. Wansink (2004) argues that people systematically underestimate their caloric intake, but we ignore this problem in the current paper.

15. This social interaction term is similar to those in Glaeser and Scheinkman (2003), Brock and Durlauf (2001), and Burke and Prasad (2005), among others.

than lean mass. In addition, there is evidence of heteroskedasticity in the error. Studies that found that the disturbances are positively correlated with weight include Leibel, Rosenbaum, and Hirsch (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. 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. (The quantitative results are discussed in Sections IV and V.)

To simplify analytic exposition, we present the linear heteroskedastic specification in the equations that follow:

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

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 Equation (3). The metabolic equation (3) implies the following (1-wk) relationship between food intake and weight:

$$\text{(4)}\quad W_t = W_{t-1} - (7/3,500)(\gamma + \rho W_{t-1} + \varepsilon_i \log(W_{t-1})) + 0.9F_t.$$

The term $0.9F_t$ represents the thermic effect of digestion, that is, the fact that digestion burns, on average, 10% 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 1 wk. The conversion factor of 7/3,500 is the ratio of the number of days in a week to the number of calories (3,500) per pound of body weight. Food is measured in pounds of body weight added per week, which can be easily converted back to calories by multiplying by 3,500.

Notice that we abstract from height variation, both cross-sectionally and longitudinally. The evidence of Schofield, Schofield, and James (1985) and Cunningham (1991) indicates that, once basal metabolism is taken into account, height does not explain much additional cross-sectional variation in weight. In addition, increases in average height in recent decades among women in the United States have been well outstripped by increases in average female weight, resulting in significant increases in mean BMI. Observing women aged 30–60 yr in each of the NHANES surveys, we find that average height increased by just 0.56% between 1976 and 2000.[16] To maintain a constant mean BMI, average weight would have had to increase by only 1.1%, whereas the actual weight increase was 13.5% for this age group over this period.

III. EQUILIBRIUM AND COMPARATIVE STATICS

A. Equilibrium Definition

Individuals in the population are identical in all of the parameters of the utility function, α, β, ρ, γ, J, and M, have identical incomes, and face the same prices. The only explicit source of heterogeneity is the idiosyncratic metabolic shock, ε_i. The full (interior) equilibrium conditions under the linear metabolism model can be expressed as follows:[17]

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

$$\text{(6)}\quad F_i^s = (1.11)(7/3,500) \times (\gamma + \rho W_i^s + \varepsilon_i \log(W_i^s)),$$

16. This estimate is based on sample-weighted averages of the examined survey subjects, that is, those subjects whose height was measured by the survey takers.

17. 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.

$$M^{S} = \zeta\left((1/N)\sum_{i} W_i^s\right), \tag{7}$$

$$\beta/(C_i^s + 1) = \lambda, \tag{8}$$

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

The conditions apply to an interior equilibrium, in which stable food intake, F_i^S, stable weight, W_i^S, and stable nonfood 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 stable weight level, W_i^S. Equations (8) and (9) are, respectively, the first-order condition on nonfood consumption and the budget constraint.

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 $\phi(\cdot)$ represents the standard normal density function:

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

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. We verify these assertions in a mathematical appendix (available on request).

B. Comparative Statics

Price Effects and the Social Multiplier. First, we consider the effect of a change in the full price per 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 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 weights and a further adjustment of the norm, and so on until a new equilibrium is reached.[18] The latter is an example of a "social multiplier" effect, as in Becker and Murphy (2000), Glaeser and Scheinkman (2003), Brock and Durlauf (2001), and Burke and Prasad (2005). The total effect is expressed as the decomposition of these two effects as follows:

$$dW_i^s/dp = \partial W_i^s/\partial p + (\partial W_i^s/\partial M)(dM^{S}/dp), \tag{11}$$

where the expression dM^S/dp refers to the change in the equilibrium norm caused by the price change. The first term on the right-hand side of the equation 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 (i.e., $\partial W_i^s/\partial M$ 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, $dW_i^s/dp < 0$ and $dM^S/dp < 0$.[19]

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 factor amounts to the quantity $1/(1 - m)$, where $m = (\zeta/N)\sum_i \partial W_i^s/\partial M$ 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 strictly less than 1. This assumption guarantees that the multiplier is strictly greater than 1 and yet finite (Burke 2007).[20]

18. 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 1.

19. It is possible, as a referee has pointed out, that a decrease in the relative price of "low-quality" energy-dense food could result in a net improvement in dietary quality. For such a response to result in weight loss, however, both low-quality calories and overall calories would have to be Giffen goods. We find these conditions unlikely to hold for the typical person, and the evidence in Huang (1993) and Popkin (2001) fails to support them.

20. Becker and Murphy (2000) define the social multiplier as the term $m = (\zeta/N)\sum_i \partial W_i^s/\partial M$, but the thrust of the results is the same in either case.

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. Assume, in an initial equilibrium, that the consumer weighs more than the norm, in which case the marginal utility of weight gain is negative and the marginal utility of food 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 induces additional food consumption (and hence further weight gain) and raises the value of the weight norm. Again, the welfare effect is ambiguous. The benefits are that the consumer eats more and that her weight moves closer to the norm. The cost is that she gives up some nonfood consumption. Given the ambiguity in both components of the welfare change, the overall welfare effects of a price change are ambiguous.[21] The simulations indicate that, under our calibration parameters, individuals with low metabolism are more likely than others to experience welfare losses as a result of price declines and that individuals are more likely to experience welfare losses when norms are fixed than when they are flexible.

IV. 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- to 60-yr age bracket observed between 1976–1980 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 nonlinear metabolism with results under alternative specifications, representing counterfactual scenarios and competing frameworks. For some features of the distribution—for example, pronounced rightward skewness—our model offers clearly superior explanatory power relative to the alternatives. The counterfactual analysis indicates that, suppressing either the social multiplier effects or the nonlinear metabolic relationship (in favor of a linear model), it becomes much harder to match the changes in the mean, median, 95th-, and 99th-percentile weights that occurred over the time period. Another desirable feature of our model is that it places more constraints on the calibration relative to alternative frameworks, for in the latter, the parametric form of the heterogeneity is unknown. The results from the experiments are summarized, using descriptions of the key distributional features, in Table 2.

A. Calibration of the Model

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 aged 30–60 yr, setting an initial list of parameters to roughly match average weight for this group observed in the 1976–1980 NHANES data. All parameters are identical across individuals, except for the idiosyncratic metabolic shock. To calculate obesity rates, we measure the percentage of people 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 BMI of 30. The body weight norm is defined as 88% of the realized average weight in equilibrium, a figure based

21. 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.

TABLE 2
Summary of Simulated Weight Distributions Women (aged 30–60 yr)

Distribution	Mean[a]	Min	Max	Median	95th[b]	99th[c]	Skewness[d]	Obese[e]	Norm[f]
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[g], P = \$50)	145.9 (18.8)	98	276	143.6	180	201	0.820	7.5	130.6
Rational addiction (linear[g], P = \$32)	158.8 (20.8)	106	304	156.2	196	220	0.839	20.2	130.6

[a]Standard deviation in parentheses.
[b]95th Percentile.
[c]99th Percentile.
[d]Skewness : $= \sum_i[(X_i - \mu)^3]/((N-1)\sigma^3)$ for univariate data $X_1, X_2, \ldots, X_N$, where μ and σ denote mean and standard deviation, respectively.
[e]Percentage with BMI of 30 or above (more than 174.5 pounds for women of average height in the simulations).
[f]Population weight norm, see text for details.
[g]Linear homoskedastic model of metabolism.

on the relationship between desired weights and actual weights in the BRFSS data for women, as seen in Figure 2.

The metabolism models are estimated directly from the original Schofield data (see Schofield, Schofield, and James [1985] for details on these data).[22] 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 30–60 yr. 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 weight of $\hat{\rho} = 447.6$ (t value of 15.1). 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.

Recall that ε_i represents the idiosyncratic metabolic parameter. 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 consumption in a week exceeds the marginal utility of the first unit of nonfood consumption by 24%. (The coefficients of 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

22. We thank Graham Horgan of Biomathematics and Statistics Scotland for providing us with the data.

that individual weight increases by one fifth of a pound for every 1-pound increase in the norm.

The model's price represents the full price, including both food inputs and time costs, of 3,500 calories (the caloric equivalent of 1 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, respectively. At the initial full price (\$50), the cost of the calories needed (1,556) to cover basal metabolism and digestion for a 140-pound woman amounts to \$22.41/d. At the lowest full price, this cost comes to \$14.81. Income is set at \$600/wk or \$31,200/yr, implying a gross hourly wage of \$15.[23] Our values for income and the full price of food imply that in the initial equilibrium (at the highest full price of calories) the average person is spending about 26% of her income on food expenses.[24]

To illustrate our price assumptions more vividly, 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 dollars), which is the value of approximately 13 min 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 dollars), implying that the real list price of the Big Mac declined by approximately 12% between 1980 and 2000 and that its total time cost in 1980 was 25 min. These estimates imply that the purchase price per calorie for the Big Mac fell by substantially less than our simulated average food price per calorie, but that the Big Mac's time costs fell by a greater percentage than did observed meal preparation and clean-up costs. These findings seem consistent with the evidence, supported by Chou, Grossman, and Saffer (2004), that both the supply of and the demand for fast food increased relative to other foods over the time period.

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

24. Abstracting from time and preparation costs, Huang (1993) estimates that the average food budget share between 1953 and 1990 in the United States was about 18%.

B. 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. 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 3 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 4 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-, and 99th-percentile weights 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 increased, 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

FIGURE 3
Equilibrium Weight Distributions: Moving Norm, Log-Linear Metabolism, with Kernel Density Estimate Plot

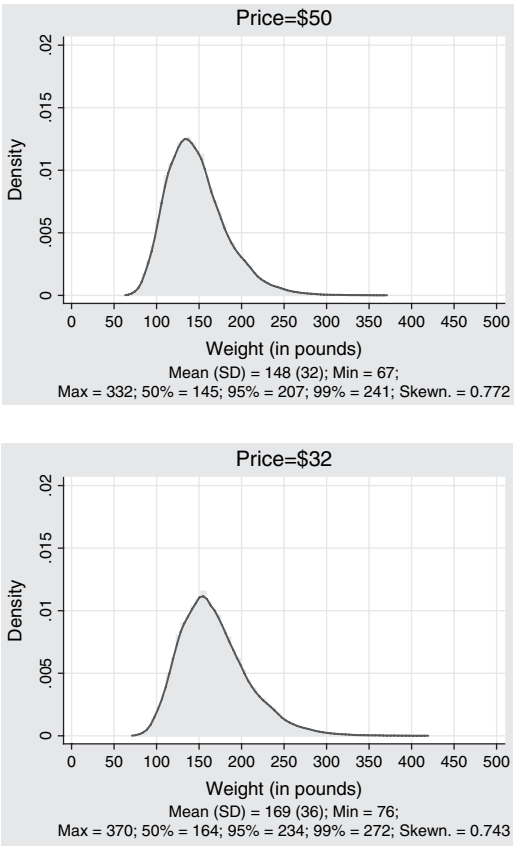
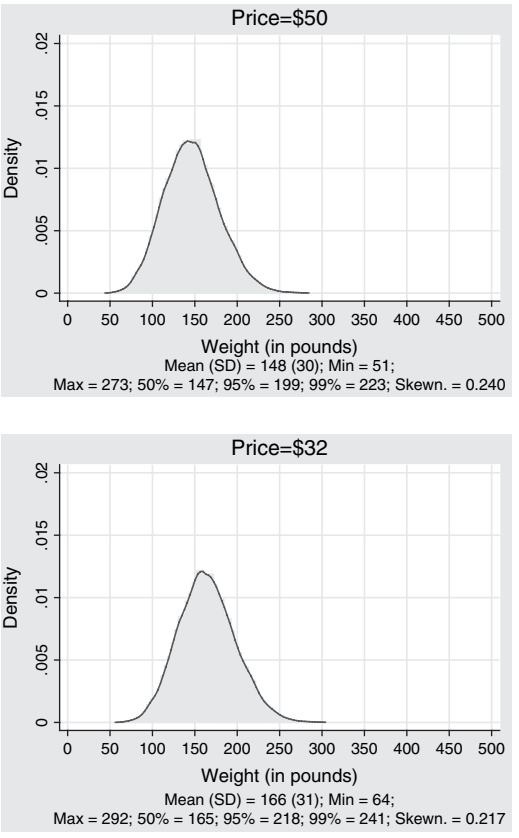


FIGURE 4
Equilibrium Weight Distributions: Moving Norm, Linear Metabolism, with Kernel Density Estimate Plot



with respect to the average of initial and final weight; the figures do not change much if percents 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 (i.e., the percent change in stable weight for a permanent marginal 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 decreases with weight in the log-linear model. This occurs because we assume that individuals correctly forecast the marginal effect of calories on weight for the current week and adjust consumption accordingly and because we assume that calories are not addictive. Consequently, initially heavier people consume fewer additional calories in response to a price decline. Despite their lower caloric response, they gain more weight than others, and larger percentage gains are possible depending on the rate at which 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 yr, 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.

C. Exogenous vs. Endogenous Weight Norms

In contrast with our endogenous or evolving norm specification, other models that include a weight norm, such as Philipson and Posner (1999) and 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 hardwired preferences.[25] 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 described above, 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 the 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. For the price change from \$50 to \$40, the estimated social multiplier is 1.24. This value means that, on average over this price interval, price effects on equilibrium mean weight will be 24% greater in magnitude in the endogenous-norm model than in the fixed-norm model. Measured over the \$40–\$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% 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% of the population are made better off.

If norms are held fixed, however, the initial price change improves welfare for only about 40% 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% of the population are made better off. Therefore, a substantial portion of the population fares 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 the potential externalities imposed

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

on nonobese individuals. Bhattacharya and Sood (2005) find, for example, that the cost of treating obesity-related disorders is not borne only by obese individuals.

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

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). The latter authors adopt a linear nonstochastic metabolism, but we add a heteroskedastic metabolic shock in order to generate cross-sectional variation (the specification of metabolism is as in Equation (3) with coefficients as in the section "Calibration of the Model"). As shown in Table 2, the forward-looking model predicts lower average weights at each price than does our myopic model. 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 (also with a fixed norm) and 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 the given price changes, in a model with fixed norms than in one with changing norms.

E. 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 4 yr' worth of data) represents a long-run equilibrium weight distribution. Weight adjustments across equilibria must occur in "real time," because individuals cannot instantaneously alter body weight. 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 might adjust with a lag, since it takes time for individuals to observe increases in the average population weight. 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 3,500 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-yr 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 1-wk optimization problems repeatedly over the interval. We update the norm annually in the first experiment and every 5 yr in a second experiment. We reduce the food price at the beginning of the year by 3%/yr 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.[26]

26. 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.

We hold price constant beginning in 1993, based on several facts: (1) the overall food consumer price index (CPI) did not fall beyond 1993 but rather showed a net gain of about 1 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 to fall until 1998, then rose again, and ended up close to its 1993 level by 2003; and (4) while weekly time costs (for food preparation and cleanup) fell 11 min/yr on average between 1985 and 1994, the same measure fell only 1.9 min/yr between 1994 and 2003.[27] These facts, taken together, suggest that the full price per calorie has been roughly constant since 1993.

The panels in Figure 5 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 1 pound of its predicted final long-run equilibrium level of 168.6 pounds (see Table 2) by 2001. Under 5-yr norm updating, average weight does not get within this threshold until 2004, more than 10 yr after the price levels off. This exercise illustrates the fact that, when norms adjust with a lag, the price elasticities of food consumption and body weight are 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. Although a model of rational food addiction, such as Cawley (1999), can yield this same elasticity result, we know of no attempt to simulate explicit adjustment dynamics for such a model and so the probable length of the long run in the addiction context is unknown.

27. The data for 1985 come from the *Americans' Use of Time Survey*, for 1994 from the *National Human Activity Pattern Survey*, and for 2003 from the *American Time Use Survey*. For additional information on these surveys, see Aguiar and Hurst (2006).

V. SIMULATION OF ALTERNATIVE MODELS

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 and variation in the propensity for food addiction. While addiction and lack of self-control may sound like similar phenomena, they are modeled 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 nonlinear metabolism. We conclude that the alternative explanations most likely complement our own model, rather than contradicting or preempting it.

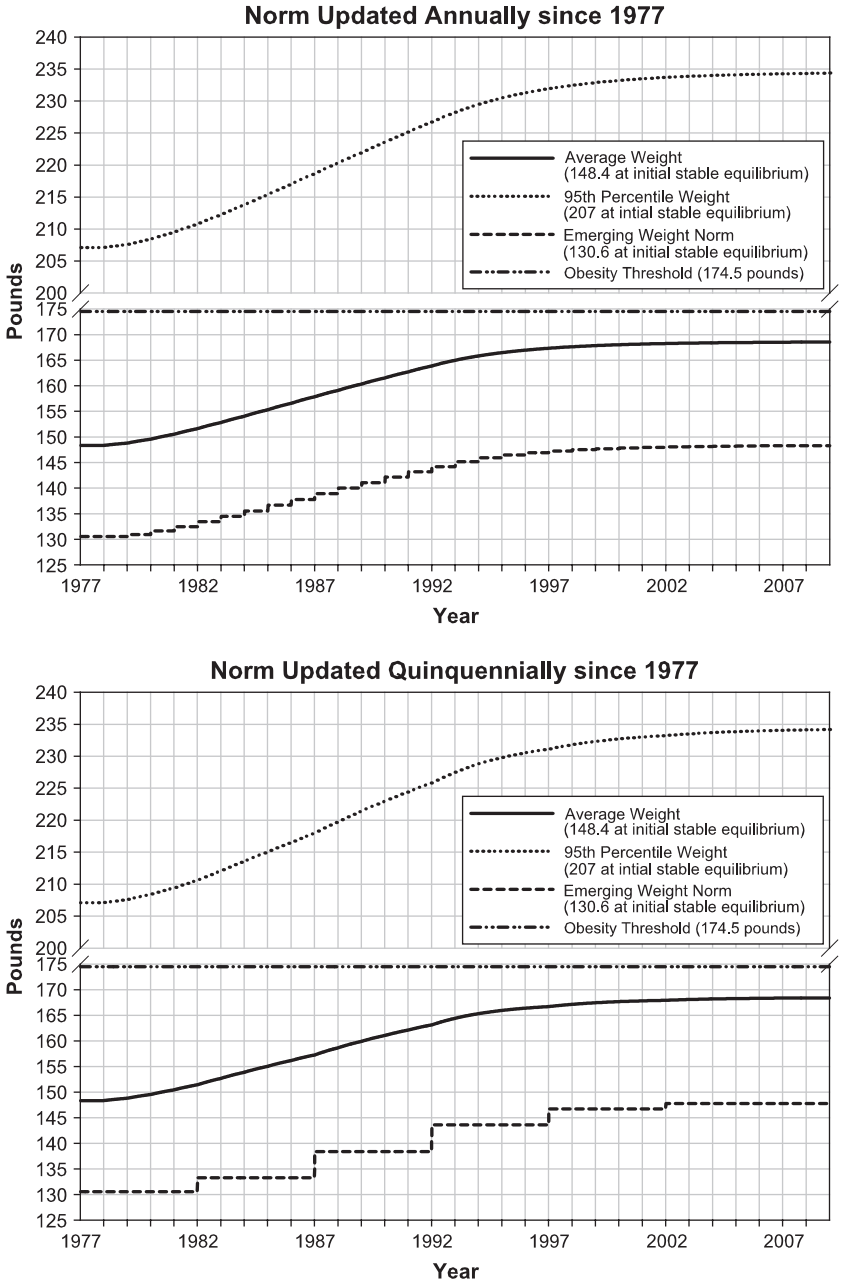
A. The Self-Control Hypothesis

CGS argue that variation in self-control can explain the disproportionate weight growth in the upper tail of the distribution over the past 20 yr. 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. The paper asserts that 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.

We find that the latter claim does not necessarily hold without additional model restrictions not specified in the original text, restrictions that are not necessarily realistic in the context of food consumption. Despite this theoretical weakness, 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 hardwired to take advantage of available food in the present, given that for much of evolutionary history, we faced scarce and unpredictable food supplies.

We estimate the effects of variation in self-control on weight levels and price effects for a parametric model based on the assumptions

FIGURE 5
Weight Dynamics: Annual vs. Quinquennial Norm Updating, Log-Linear Metabolism; Gradual Price Decline 1977–1993



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 Gale, Castracane, and Mantzoros (2004) suggest that self-control may have a genetic foundation, we know of no attempt to describe the empirical distribution of self-control. We proceed with the naive assumption that the self-control parameter, 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 the self-control parameter, at varying time-cost values. The calibration respects the explicit and implicit assumptions in CGS. For metabolism, we adopt the linear nonstochastic model for women cited in CGS, which they attribute to Schofield, Schofield, and James (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 the food time cost, but the parameter loosely corresponds to the average time needed to prepare a meal or snack. Accordingly, we consider values (in min) of 20, 10, and 5. For the decline from 20 to 10 min, 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 to 5 min produces much more dramatic weight gains. 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.

B. 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 the section "Forward-Looking vs. Myopic Decision Making". 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, to be an increasing function of beginning-of-period body weight, where the function varies across individuals. We vary the propensity to food addiction according to a normal distribution, such that half the population is prone to food addiction to varying degrees and the other half does not experience food addiction. 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 2, we see that the distribution exhibits positive skewness. Individuals with a greater propensity to addiction weigh more than others at a given price, ceteris paribus, and 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. However, 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.

VI. 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 with social and physiological processes. In the social process, the body weight standard becomes more relaxed as average weight increases in response to price declines; the relaxed standard then leads to further weight increases. 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 model does a 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 leveled off since the mid-1990s.

It has not been our primary goal 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 BMR at a higher weight, based on the curve's slope at her current weight, will systematically overestimate it and will underestimate the long-term weight gain associated with a permanent (nonmarginal) 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 were to learn the model eventually through revealed weight gains, it may prove more difficult than anticipated to reverse such gains. Under these constraints on information, 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 counseling, 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 contributions 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 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. Norms restricting the proper times and places for eating have also broken down. Recent research by Flegal et al. (2005) 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.[28]

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—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.

28. These findings have not been universally accepted. A symposium at the Harvard School of Public Health, in 2005, argued that the results suffered from biases due to reverse causality (e.g., illness-causing weight loss) and residual confounding (e.g., 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%20deaths/excess%20deaths.htm.

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