

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-year-old age bracket, we compare the predicted weight distributions to NHANES survey data spanning (intermittently) the years 1976 through 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.

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I. 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. 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 (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 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. 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 et al. (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 et al. (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 years.

In our choice model, utility depends on food and non-food 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 weight standards into the utility function via various exogenous constructs: Lakdawalla and Philipson (2002) refer to an “ideal weight”; Levy posits a “socio-cultural 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 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 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 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 ten 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 is relatively easy (yet expensive)

to measure. Using a well-known data set containing direct observations of basal metabolic rates, 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 non-obvious 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.

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 on the future of obesity.

II. THEORETICAL FRAMEWORK

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. 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 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.

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 30–60-year-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 percent.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 so-

cial 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 non-food consumption, taking the reference weight and prices into account.¹¹ Food and non-food consumption are both goods, but deviation from the reference weight is a bad. A general expression of the one-period utility model is as follows:

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

F_t and C_t represent food and non-food 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 beginning with J 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 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 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 diabetes, heart disease, osteoarthritis, and other health conditions accelerate with increases in body mass index (e.g., Must et al. 1999). In addition, mortality exhibits a U-shaped relationship to BMI among both men and women in the U.S., 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) 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 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.¹⁴ We examine the robustness of our predictions to forward-looking

specifications in Section 5.

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

$$\begin{aligned} \text{Max}_{\{F_t, C_t\}} U_{it}[F_t, C_t | W_{i,t-1}, \alpha, \delta, \beta, J, \gamma, \rho, \varepsilon_i, M_{t-1}] = & \quad (2) \\ \alpha F_{it} - \delta F_{it}^2 + \beta \log[C_{it} + 1] - J(W_{i,t-1} - (7/3500)BMR(\gamma, \rho, \varepsilon_i, W_{i,t-1}) + .9F_{it} - M_{t-1})^2, \\ \text{s.t. } p_t F_t + C_t \leq Y. \end{aligned}$$

Within the 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).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 non-food consumption is normalized to one; Y represents weekly income.

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 a strong exogenous (that is, 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 U.S. have not changed much since 1975, while average calorie consumption has increased significantly since then, as shown by evidence in CGS and in 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), total energy expenditure 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 sex. One model that is 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) showed that the Schofield equations substantially overestimate BMR for the obese. The Horgan and Stubbs findings, 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 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 et al. (1995) and Rand (1982).

Our own analysis of the Schofield data, using maximum likelihood estimation, finds that (i) a weight-log-linear model of (expected) BMR fits the data better than a weight-linear model and (ii) 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 4 and 5.)

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

$$BMR(kcalperday) = \gamma + \rho W + \varepsilon_i \log(W). \quad (3)$$

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:

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

The term $.9F_t$ represents the thermic effect of digestion, that is, the fact that digestion burns, 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 in pounds of body weight added per week, which can be easily converted back to calories by multiplying by 3500.

Notice that we abstract from height variation, both cross-sectionally and longitudinally. The evidence of Schofield (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 U.S. have been well outstripped by increases in average female weight, resulting in significant increases in mean BMI. Observing women ages 30 to 60 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

Equilibrium Definition

Individuals in the population are identical in all of 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 (interior) equilibrium conditions under the linear metabolism model can be expressed as follows:¹⁷

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

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

$$M^S = \zeta((1/N) \sum_i W_i^S), \quad (7)$$

$$\beta/(C_i^S + 1) = \lambda, \quad (8)$$

$$pF_i^S + C_i^S = Y_i. \quad (9)$$

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 stable weight level, W_i^S . Equations (8) and (9) are, respectively, the first-order condition on non-food 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:

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

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.

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 (2002), Brock and Durlauf (2001), and Burke and Prasad (2005). The total effect is expressed as the decomposition of these two effects as follows:

$$dW_i^S/dp = \partial W_i^S/\partial p + (\partial W_i^S/\partial M)(dM^S/dp), \quad (11)$$

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 (that is, $\partial W_i^S/\partial M$ 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 one. This assumption guarantees that the multiplier is strictly greater than one and yet finite (Burke 2006).²⁰

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 non-food 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 non-food 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 30–60-year 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 U.S. 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 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 non-linear 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.

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 ages 30 to 60, 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 U.S. for the relevant age group, this weight implies a body mass index of 30. The body weight norm is defined as 88% 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 seen in Figure 2.

The metabolism models are estimated directly from the original Schofield data (see Schofield et al. (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 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 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 maxi-

mum 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%. (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 \$15.²³ 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 \$), 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 the evidence, supported by Chou et al. (2004), that both the supply of and demand for fast food increased relative to other foods over the time period.

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-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 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 (that is, 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 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.

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 hard-wired 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, 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. For the price change from \$50 to \$40, the estimated social multiplier is 1.24. This value implies that the price-induced increase in average weight in the endogenous-norm model will be 24% 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% 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 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. Bhattacharya and Sood (2005) find, for example, that the cost of treating obesity-related disorders is not borne only by the obese.

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 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). The latter 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 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 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.

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 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 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% 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.²⁶

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 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 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 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 one pound of its predicted final long-run equilibrium level of 168.6 pounds (see Table 2) by 2001. Under five-year norm updating, average weight does not get within this threshold until 2004, more than 10 years 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.

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 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.

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. 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 which are not necessarily realistic in the context of food consumption (see Appendix). 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 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.

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 Gale et al. (2000) suggests 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, including those pertaining to the derivatives of instantaneous food utility. 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 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 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.

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. 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 levelled 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 basal metabolic rate 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 (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 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 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, 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.²⁷

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.

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Captions:

Figure 1: Historical Female Weight Distributions (age 30-60), with kernel density estimate plots. (Source: NHANES)

Figure 2: Group Actual vs. Desired Mean Weight by Demographics (age 30-60). (Source: BRFSS, 1990 and 1994-2002)

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.

Figure 5: Weight Dynamics: Annual vs. Quinquennial Norm Updating, Log-Linear Metabolism; gradual price decline 1977-1993.

Footnotes.

*We are grateful for the comments of Alberto Bisin, Kislaya Prasad, Peyton Young, Carol Graham, Bill Dickens, Farasat Bokhari, Giorgio Topa, Inas Rashad, and seminar participants at Florida State University, Royal Holloway-University of London, Western Washington University, the Federal Reserve Bank of Boston, the Federal Reserve Bank of New York, and the University of Miami. We also thank two anonymous referees.

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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 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-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 a large random sample of the resident population 18 years and older in participating states of the U.S. 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 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.

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 risk, as explicated by Kuczmarski and Flegal (2000). Several websites offer simple BMI calculators. See, for example, <http://www.cdc.gov/nccdphp/dnpa/bmi/>.

4. The relevant papers include Chou et al. (2004); Cutler et al. (2003); Philipson and Posner (1999), Lakdawalla and Philipson (2002).

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

6. 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 can’t 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 2. 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.

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

8. For men the corresponding figures are 13.7 percent 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.

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

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 obtains 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 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 economic costs among underweight subjects.

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 (2002), Brock and Durlauf (2001), and Burke and Prasad (2005), among others.

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.

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 one.

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.

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.

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

23. 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.

24. 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%.

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.

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.

27. 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 (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](http://www.cdc.gov/nchs/products/pubs/pubd/hestats/excess%20deaths/excess%20deaths.htm).

Abbreviations:

BMI: Body Mass Index

BMR: Basal Metabolic Rate

BRFSS: Behavioral Risk Factor Surveillance System

CGS: Cutler et al. 2003

CDC: Centers for Disease Control

CPI: Consumer Price Index

NHANES: National Health and Nutrition Examination Survey

TEE: Total Energy Expenditure

WHO: World Health Organization

TABLE 1
Summary of Empirical Weight Distributions

Distribution	Mean ^a	Min	Max	Median	95th ^b	99th ^c	Skewness ^d	Obese ^e
<i>Women (age 30-60)</i>								
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
<i>Men (age 30-60)</i>								
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

Notes: ^aStandard deviation in parentheses. ^b95th Percentile. ^c99th Percentile. ^dSkewness:= $\sum_i [(X_i - \mu)^3] / ((N - 1)\sigma^3)$ for univariate data X_1, X_2, \dots, X_N where μ and σ denote mean and standard deviation. ^ePercentage with BMI of 30 or above (more than 174.5 pounds for women of average height in the simulations).

TABLE 2
Summary of Simulated Weight Distributions

Distribution	Mean ^a	Min	Max	Median	95th ^b	99th ^c	Skewness ^d	Obese ^e	Norm ^f
<i>Women (age 30-60)</i>									
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

Notes: ^aStandard deviation in parentheses. ^b95th Percentile. ^c99th Percentile. ^dSkewness:= $\sum_i [(X_i - \mu)^3] / ((N - 1)\sigma^3)$ for univariate data X_1, X_2, \dots, X_N where μ and σ denote mean and standard deviation. ^ePercentage with BMI of 30 or above (more than 174.5 pounds for women of average height in the simulations). ^fPopulation Weight Norm, see text for details. ^gLinear homoskedastic model of metabolism.

FIGURE 1

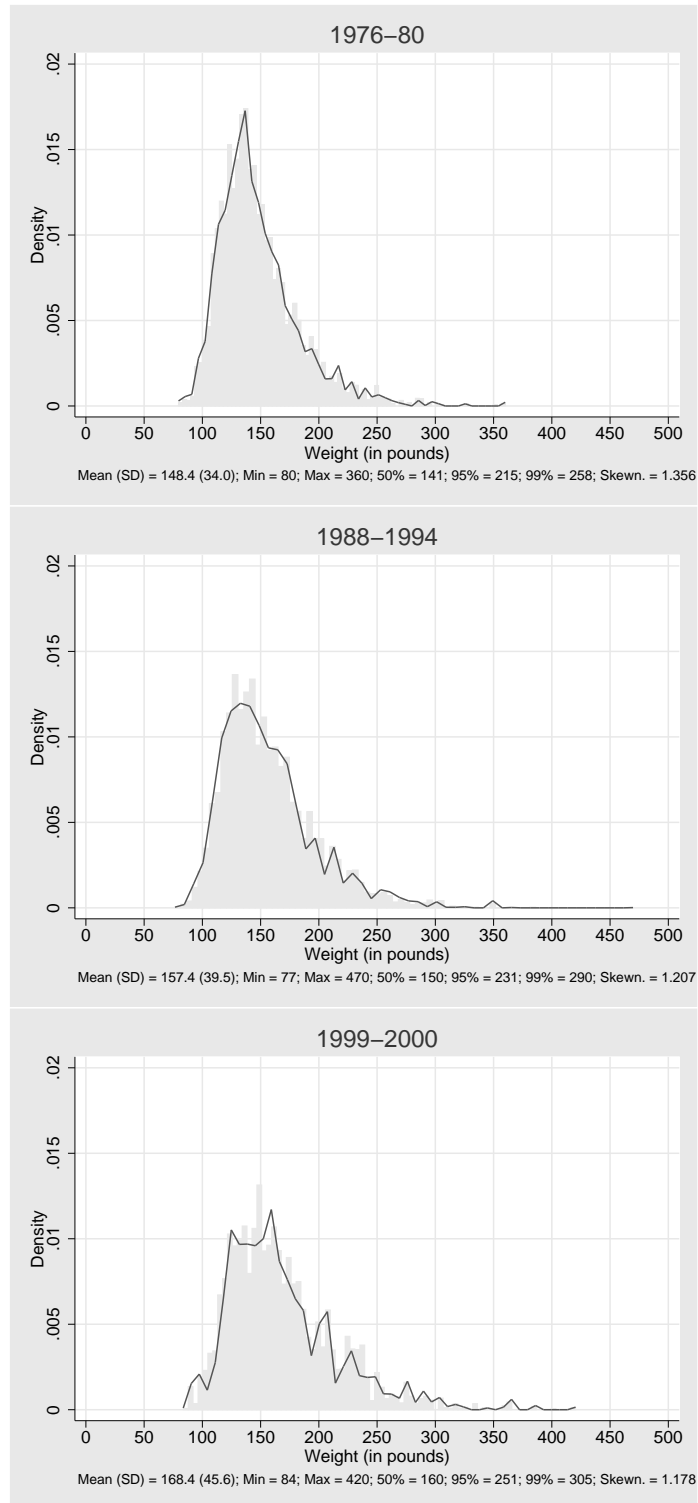


FIGURE 2

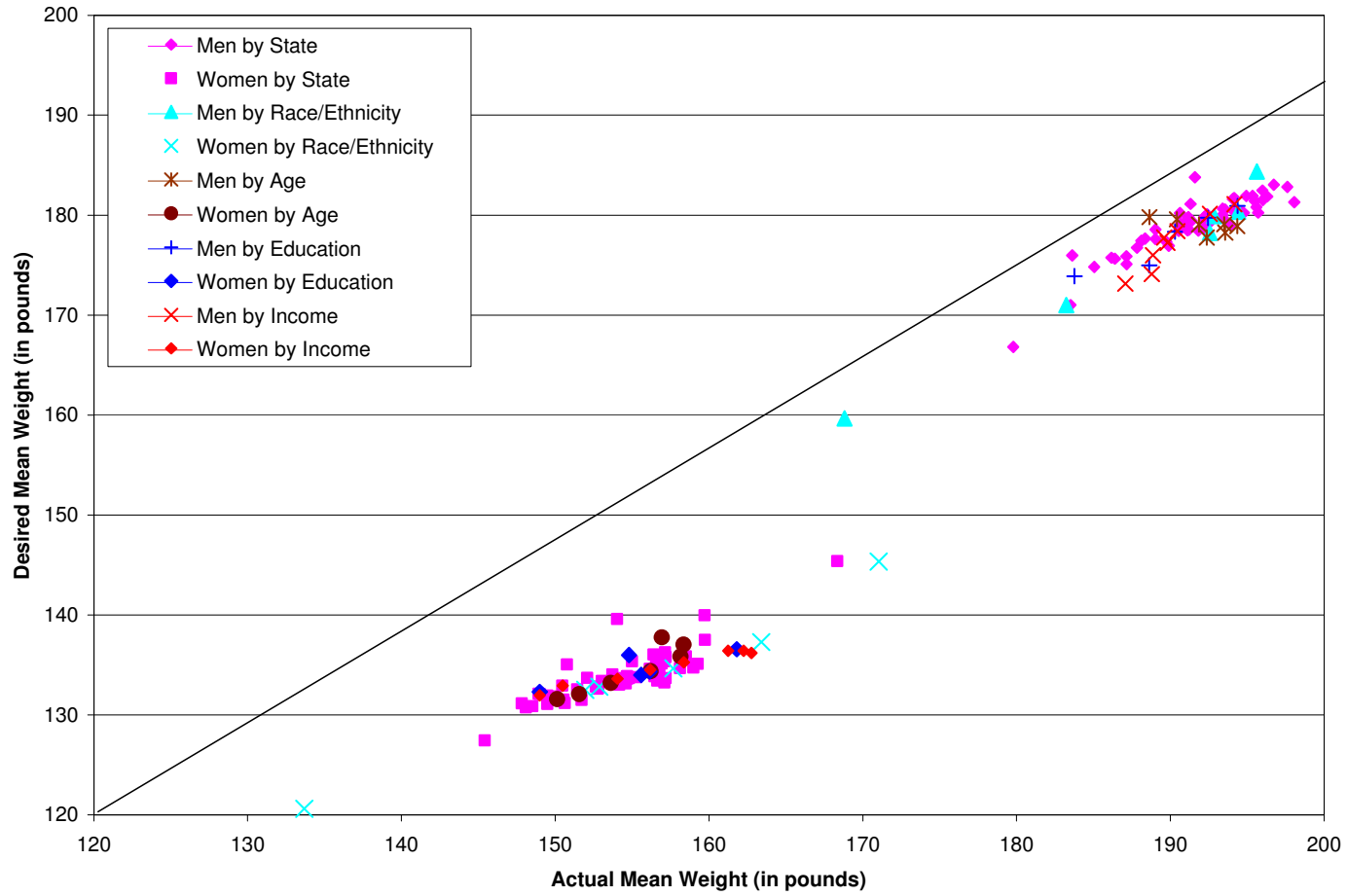


FIGURE 3

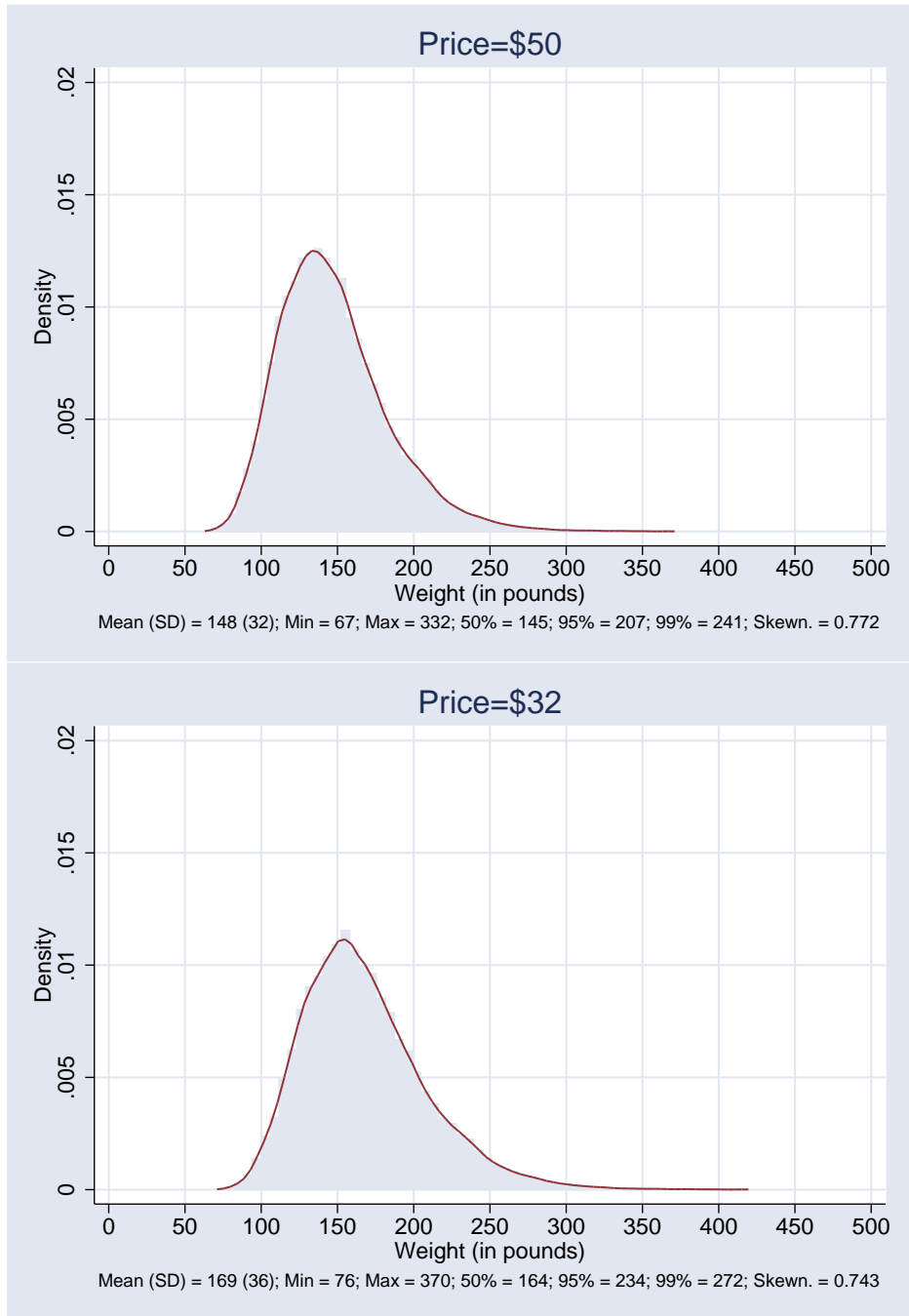


FIGURE 4

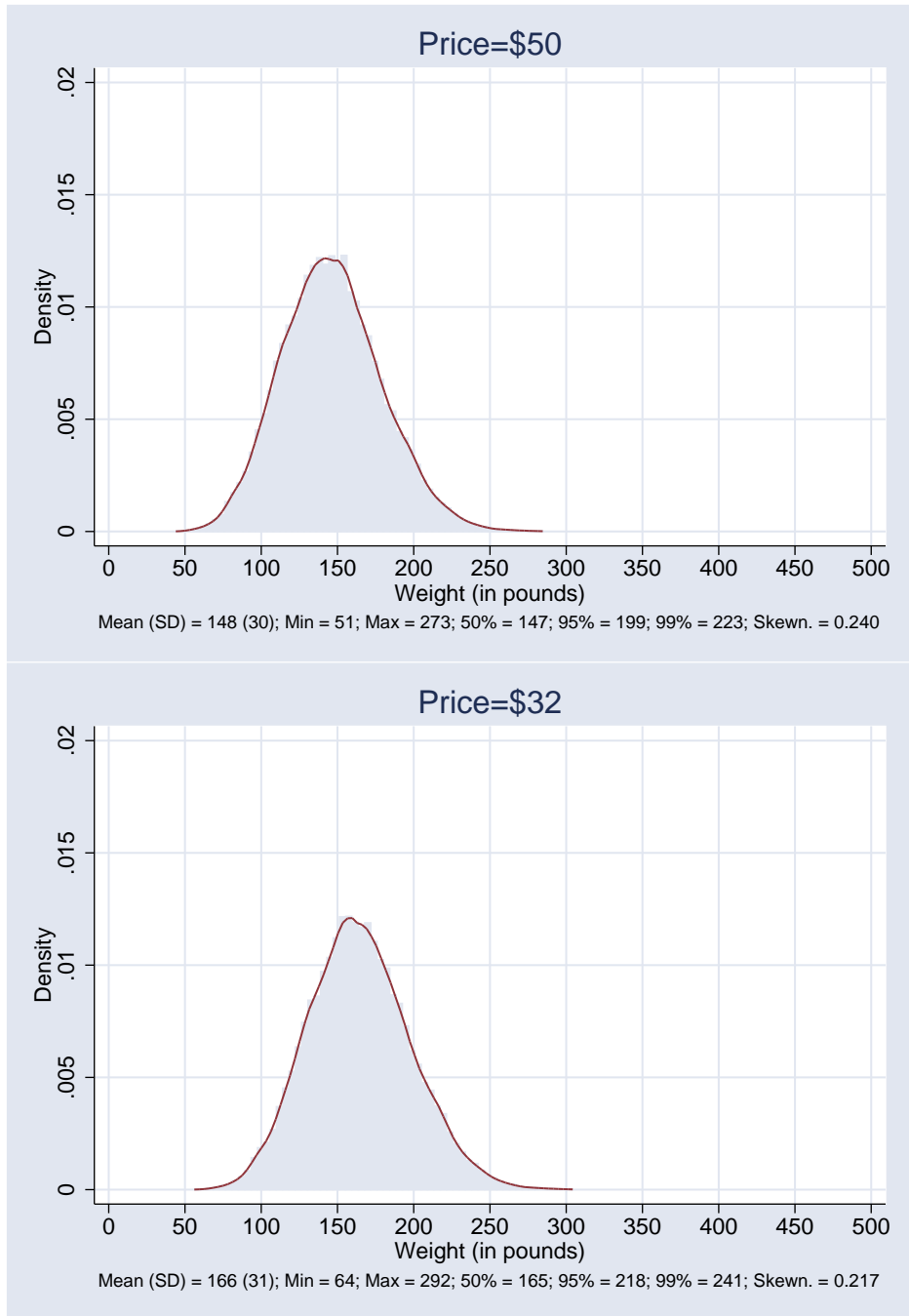
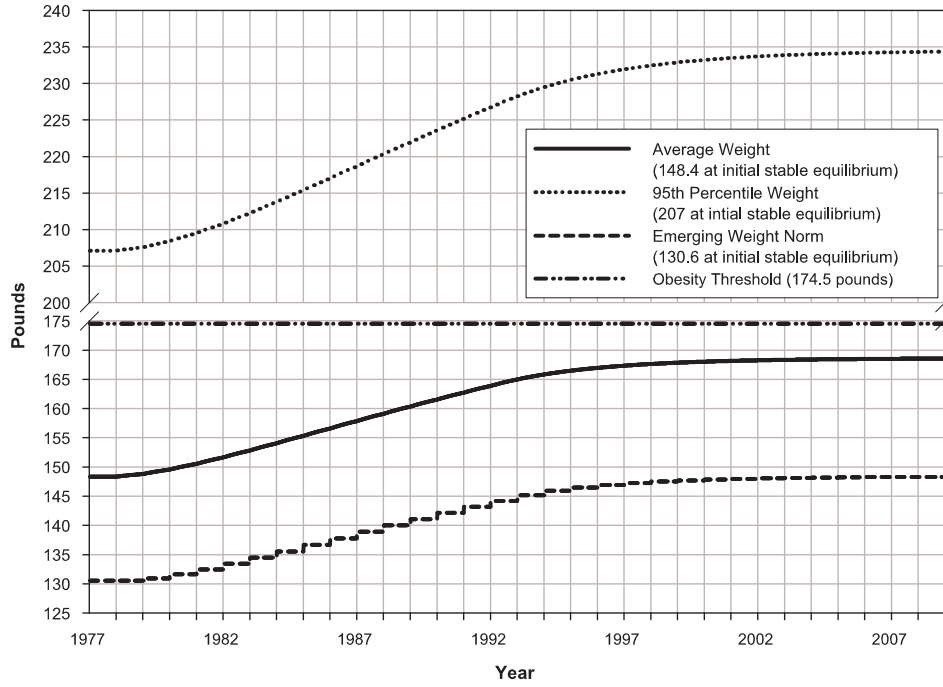


FIGURE 5

Norm Updated Annually since 1977



Norm Updated Quinquennially since 1977

