

OBESITY AND NATURE'S THUMBPRINT

How Modern Waistlines Can Inform Economic Theory*

Trenton G. Smith, Ph.D.

Department of Economics

Universität Bonn

e-mail: trent@wiwi.uni-bonn.de

16 April 2003

Abstract

The modern prevalence and negative consequences of obesity suggest that many people eat more than they should. This essay examines the biological underpinnings of mammalian feeding behavior in an attempt to reconcile the “self-control problem” with the normative tradition of neoclassical economics. Medical, genetic, and molecular evidence suggest that overeating is a manifestation of the fundamental mismatch between ancient environments—in which preferences for eating evolved—and modern environments. The phenomenon can be described with a simple optimal foraging model in which both the utility function and the Bayesian prior are generated endogenously in the distant past. The implied disparity between subjective probabilities and actual probabilities has potentially broad implications for welfare economics.

JEL Classification System codes: B41, D11, D60, D81, D91

Key Words: Self-Control, Evolution, Obesity

* Earlier versions of this paper were presented at the 16th Annual Congress of the European Economic Association, Lausanne, Switzerland, September 1, 2001; at the Economic Science Association North American Regional Conference, Tucson, Arizona, November 3, 2001; and at departmental seminars at Princeton University; the University of California, Irvine; the University of California, Santa Barbara; and California State University, Long Beach. Thanks are due to participants in these seminars and to Ted Bergstrom, Jack Hirshleifer, Bob Deacon, Ted Frech, Charlie Stuart, George Ainslie, Gary Becker, Kelly Bedard, Antonio Bento, Karl Englert, Anita Gantner, Mark Griffin, Darwin Hall, Henry Harpending, Daniel Kahneman, Bob Schillberg, Dottie Smith, Peter Sozou, Chris Stoddard, Attila Tasnadi, Thomas Tröger, and Bob Whitaker for helpful comments.

1. Introduction: Explaining Obesity

Have you ever tried to count calories? I have, and believe me it's not easy. Every ingredient in every morsel that goes into your mouth must be accounted for, its calorie density multiplied by its weight, the products summed and tallied. Even if all this information were readily available the task would be tedious and time-consuming. Most people don't find a careful accounting of bodily energy intake to be worth their while, and are nevertheless able to solve the problem of maintaining a healthy body weight.

Some people, on the other hand, do have a problem with weight. Obesity is widely acknowledged as a serious threat to public health in many countries, and is considered a social stigma in many cultures. Often those suffering from the disease express a desire to lose weight, but are unable to alter their behavior, attributing the problem to a "weakness of will" or lack of self-control.

This seemingly simple, one-dimensional decision problem—how much to eat—is the subject of this essay.¹ Specifically, I consider the two puzzles described above: *How* do people solve this problem of how much to eat and *why*—when they overdo it—do they so often declare an inability to mend their ways? While in a sense we are all experts on the subject, there is also a wealth of information to be gleaned from fields of study as diverse as behavioral endocrinology and nutritional anthropology. By drawing on such sources of empirical evidence, I show that the self-control phenomenon may be symptomatic of a more widespread phenomenon, with important implications for economic theory.

1.1 *The Personal Costs of Obesity*

The consequences of being overweight can be severe. Children as young as 6 years of age, when shown silhouettes of various body types, choose the phrases "cheats," "argues," "gets teased," "lazy," "lies," "ugly," and "stupid" to describe the obese figure (Staffieri, 1967). 9-year-olds associate an

¹ More precisely, the object of choice is net caloric intake—what I eat net of how much I use—or, equivalently, how much energy I store as body fat. The problem quickly grows in complexity as the dimensions of time and uncertainty are introduced.

overweight body shape with poor social functioning and impaired academic success (Hill and Silver, 1995). To be obese in adolescence is to be twice as likely to be held back a grade, to quit school, and to attempt suicide (Falkner *et al*, 2001). A prospective study found men overweight in adolescence 11 percent less likely to be married seven years later; for women the figure was 20 percent (Gortmaker *et al*, 1993).

Discrimination against the obese is not limited to the playground or the marriage market. In a 1969 survey of 77 physicians, obese patients were described as “weak-willed, ugly, and awkward” (Maddox and Liederman, 1969). More recent studies find evidence of discrimination on the job market, with obese men suffering a 5 percent wage penalty, while obese women on average make 12 percent less than their normal-weight counterparts (Register and Williams, 1990; Hamermesh and Biddle, 1994).

There are also severe effects on health and longevity. Obesity is strongly associated with (and in many cases thought to cause or aggravate) adverse medical conditions such as hypertension, diabetes, heart disease, and cancer, and (perhaps as a result of these related conditions) it has a strong negative effect on life expectancy (Friedman, 2000; Kopelman, 2000; Miller and Frech, 2002). One study, for example, found that gaining *one pound* increased the marginal risk of death within 26 years by 1% among individuals between the ages of 30 and 42 years, and by 2% between the ages of 50 and 62 years (Hubert, 1986). Animal studies suggest the effect may not be limited to individuals meeting conventional standards of “overweight”: Weindruch *et al* (1986) were able to extend the average lifespan of the common laboratory mouse by *sixty-five percent* (from 27.4 months to 45.1 months) by restricting caloric intake to one third of the free feeding level.

1.2 The Modern Prevalence of Obesity

In spite of the negative personal consequences, obesity appears to be on the rise. A recent report issued by the World Health Organization declared that “obesity...is now so common that it is replacing the more traditional public health concerns, including undernutrition and infectious disease, as one of the

most significant contributors to ill health” (1998, p. 1). Obesity has been increasing in prevalence over the last few decades, especially in Western countries. In the United States, for example, the prevalence of obesity among the adult (age 20-74) population increased from 12.8% in 1960-62 to 22.5% in 1988-94 (Flegal, *et al*, 1998)². A similar trend has been seen among children and adolescents, with the prevalence of overweight (6-17 years old) increasing from 5% in 1963-70 to 11% in 1988-94 (Troiano and Flegal, 1998). Today, more than half of all adults in the U.S. can be classified as overweight or obese (Must *et al*, 1999), and it has been estimated that some 300,000 deaths per year are attributable to obesity (Allison *et al*, 1999). Because of its association with numerous medical complications, a significant proportion of expenditures on health care can be attributed to obesity: The direct cost of obesity-related health care in the U.S. has been estimated at \$52 billion per year, or 5.7% of total expenditures on health care³; indirect costs due to lost productivity are thought to be of equal magnitude (Wolf and Colditz, 1998).

Experts often point to the modern high fat diet and sedentary lifestyle as fundamental causes of the recent rise in obesity (World Health Organization, 1998, p. xvi), but such proclamations fail to get to the heart of the question—*why* have people chosen such a lifestyle, if the dreaded disease of overweight is the result?

1.3 Is Obesity a Choice?

The trends and incidence data cited above suggest that obesity is a problem in need of a solution. But on the other hand, “overeating” would seem to be a matter of individual choice and as such, perhaps it is a problem best left to the individual to solve. The choice faced by the individual—what and how much to eat, given future health or social consequences—is well within the realm of neoclassical economic theory,

² This study, and most studies of this type, use body mass index (BMI) as an indicator of surplus adipose tissue. The BMI for a given individual is his weight (in kilograms) divided by the square of his height (in meters). There are obvious drawbacks to this measure, the most important being its inability to distinguish between fat and lean body mass. Flegal *et al* use the international benchmarks of BMI>25 for overweight and BMI>30 for obese.

³ This is consistent with international estimates of 2%-7% of health care expenditures (World Health Organization, 1998).

and it is easy to apply neoclassical principles to the problem. For example, the aforementioned attributes of the modern lifestyle can be viewed as lower implicit *prices* of fattening foods and easy-chair recreation (Figure 1).⁴ Pending empirical confirmation of the purported price environment, this might seem to be, for economics, the end of the story: technology has lowered the cost of corpulence, consumers have freely chosen it, and an efficient market outcome is the result. But this conclusion has an uneasy feel, given the overwhelming medical evidence suggesting that obesity is in many ways not an optimal outcome, and the insistence of most obese individuals that—given a choice—they would rather be thin (Bray, 1986; Hill and Silver, 1995).

The notion that people sometimes behave impulsively (i.e., in a manner that does not serve their long-term interests) has gained much attention within the behavioral economics literature. The next section briefly reviews the economic theory of self-control.

2. The Economic Theory of Self-Control

2.1 *Dynamic Inconsistency, Addiction, and Preference Domains*

Many people place a premium on the attribute of self-control. Individuals who have this capacity are able to stay on diets, carry through exercise regimens, show up to work on time, and live within their means. Self-control is so desirable that most of us complain that we do not have enough of it. (Laibson, 1997)

It is not hard to think of situations in which people claim to have difficulty with self-control, and maintaining a dietary regimen is a leading example. Many economic models of self-control have been

⁴ The private cost of achieving caloric surplus should properly include much more than just expenditures of *dollars* on Big Macs and La-Z-Boys, of course. As real wages rise, for example, the opportunity cost of the *time* required to prepare and eat freshly prepared, nutrient-rich foods rises, making calorie-rich fast food meals relatively less expensive. Alternatively, technology might lower costs in terms of the amount of *physical exercise* required to accomplish certain tasks. The advent of the automobile, for example, allowed many activities (banking, shopping, etc.) which previously required a walk across town to be accomplished by hopping in the car; today, the advent of the internet makes it possible to accomplish these same tasks without ever leaving one's desk. A fourth variable which might play a part is the *social or cultural context* in which the consumption decision takes place; a society that places a premium on thinness could be interpreted as placing a higher implicit "price" on an extra pound of body fat. See, for example, Philipson and Posner (1999) or Chou *et al* (2002) for economic analyses of obesity along these lines.

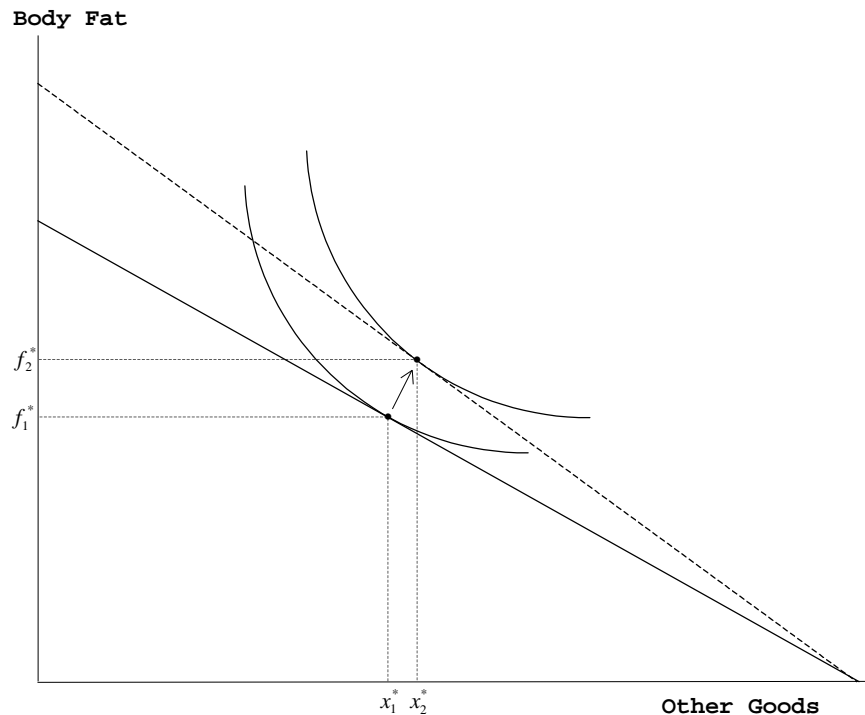


Figure 1: Body Fat and Neoclassical Choice

In the standard neoclassical treatment, preferences are well-defined and exogenous, represented here by indifference curves. In this diagram the price of body fat (Good f) is initially high relative to other goods (Good x), as indicated by the slope of the budget constraint (solid line), and the consumer's optimal choice is point (x_1^*, f_1^*) . When new technology lowers the price of body fat the consumer's budget set expands (dashed line), and the consumer's new optimal choice is point (x_2^*, f_2^*) , where body fat is greater.

proposed, and most emphasize the dynamic nature of the self-control problem. In the earliest complete exposition of the problem, Robert Strotz (1956) hypothesized that consumers might discount future consumption not only according to the calendar date at which consumption is to occur, but also according to the instantaneous distance of the future date from the present.⁵ This allowed Strotz to consider the problem of *dynamic inconsistency* and the “intertemporal tussle” in which a weakness of will precludes the execution of a consumption plan. So the consumer might make a plan to place 10% of his paycheck in a retirement account, starting next year. But if his preferences are dynamically inconsistent, he might—even in the absence of new information—change his plan come New Year's Day, and decide to

⁵ More recent studies of this problem include Thaler and Shefrin (1981), Ainslie (1991), Laibson (1997), Becker and Mulligan (1997), and Gul and Pesendorfer (2001).

put off the savings plan for another year. The problem of choosing a diet is, of course, quite similar—an individual whose long-term goal is to lose weight might go into a restaurant planning on having just a small salad, only to change his plan when the dessert cart rolls by (Figure 2).⁶

This problem might, of course, be anticipated by the individual making the original plan, and Strotz and others have suggested that if it is possible to “pre-commit” future behavior, then individuals might willingly incur a cost in the present in order to restrict the set of choices available in the future. That the consumer might anticipate the “dynamic” nature of his preferences is also emphasized in the theory of rational addiction proposed by Becker and Murphy (1988): the special case these authors refer to as “harmful addiction” is analogous to the self-control phenomenon discussed here.

Most testable hypotheses stemming from these models of self-control are related to pre-commitment: one way to identify dynamic inconsistency is to observe the consumer pre-committing by taking steps to limit the range of choices available in the future.⁷ Gul and Pesendorfer (2001) make use of this fact, capturing the self-control phenomenon with a model in which the preference domain includes not only the objects of choice but also the *choice sets* from which objects are selected. Their model allows, as do models of dynamic inconsistency, for the possibility that utility might be increased by pre-committing to a strictly smaller choice set.

2.2 *Adherence to Axioms vs. Weakness of Will*

The theory of self-control seems to capture real aspects of human behavior, and it also fits well with our intuition and personal experience. But it has an uneasy feel: Modeling behavior in this way seems to

⁶ The terms *consumption* and *savings* are somewhat confounded when diet is the object of choice: The decision to enjoy a caloric surplus today (i.e., to *consume* more) results in energy being *saved* (in the form of additional body fat) for future use. But an alternative meaning of the term consumption is to waste away, as in starvation. So an excess of consumption generates savings, while a dearth of consumption invites consumption. Fortunately, the conundrum is easily resolved by assuming that future utilities are increasing in wealth but decreasing in girth.

⁷ The level of commitment is not necessarily under the control of the consumer, of course: natural experiments such as the advent of the ATM machine or variations in the liquidity of wealth can also reveal a lack of self-control (Shefrin and Thaler, 1988; Levin, 1998; Laibson, 1997).

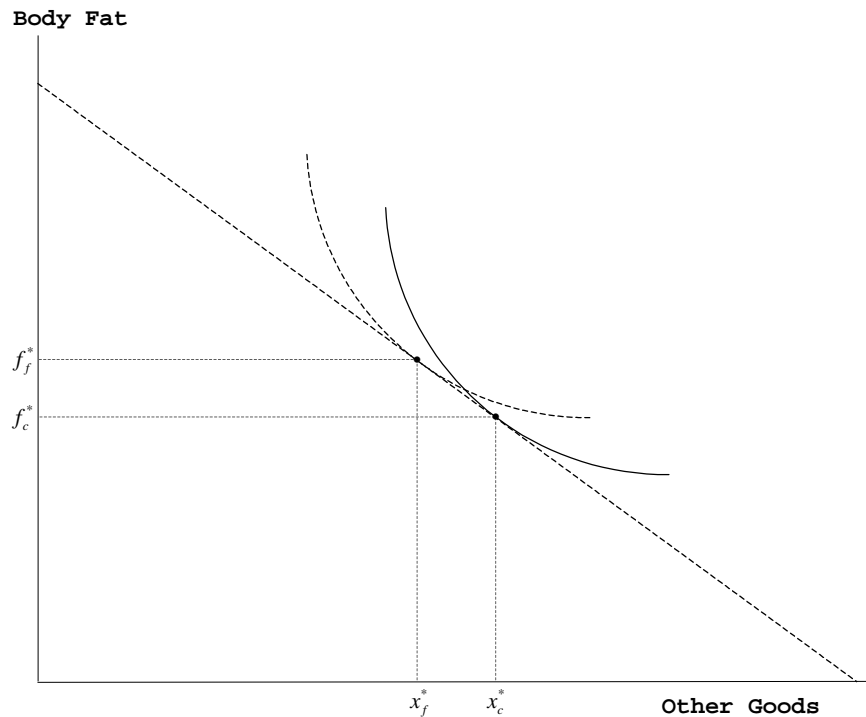


Figure 2: Diet and the Economic Theory of Self-Control

Here the theory of self-control is shown as it might be applied to caloric surplus. Implicit in variable f (body fat) is both a benefit (e.g., gustatory pleasure) to be enjoyed at the time of consumption and a cost (e.g., an expanded waistline) to be incurred at a later date. The “current self,” whose preferences are represented by the solid indifference curve, makes a consumption plan (i.e., plans his diet) and chooses his optimal bundle at point (x_c^*, f_c^*) . If his preferences are dynamically inconsistent (i.e., he lacks self-control) then the preferences of the “future self” (represented by the dashed indifference curve) might not coincide with current preferences. If the current self is unable to commit his behavior in advance then the future self chooses point (x_f^*, f_f^*) and the original diet plan is never carried out.

forgo much of the normative appeal of more conventional decision theory.

In the normative tradition of neoclassical economics, the foundation of any theory is the list of *axioms* or rules of behavior from which restrictions on behavior are derived (see, e.g., Savage, 1954; or Mas-Colell *et al*, 1995, Chapter 1). For example, if you agree that the outcomes you care about are the pleasure you get from eating ice cream and the size of your waistline, and you are able to rank the various combinations of these outcomes, then a normative theory will tell you that you *should* choose the highest-ranking combination from among those available. Here the axioms you have agreed to are *completeness* (what you care about) and *transitivity* (your ability to rank) and the restriction on behavior is that you will consistently choose how much ice cream to eat from the amount of ice cream available. No problem.

The axioms are sufficiently modest that no reasonable person would deny them, and the prescribed outcome (consistent behavior that varies only with constraints on the choice set) seems to describe actual behavior reasonably well.

But now suppose that, having decided the optimal combination of ice cream and waistline you would like at the prevailing price, you find that achieving the chosen outcome requires that you limit the amount of ice cream in your freezer. Problem. Because you have agreed that the outcomes you care about are ice cream and your waistline, the axiomatic approach of the previous paragraph requires that you be indifferent (i.e., the amount you eat is unchanged) between a freezer that contains just the right amount of ice cream and a freezer that contains more. While it is possible to introduce an additional axiom allowing the domain of preferences to include the presence of the temptation in the freezer,⁸ most people are likely to insist that it is still the *outcomes*—ice cream and waistline—that they really care about. So the axiomatic approach loses much of its appeal when the self-control problem is admitted.

This is not to say there is no value in a purely positive (i.e., descriptive, or behavioral) economic theory of self-control. The lack of a normative foundation has not precluded the development of the theory, as the discussion of Section 2.1 above makes clear, and it has enjoyed considerable success. But by adopting a strictly positivist approach—i.e., making note of the odd behavior and positing odd preferences in order to accommodate it—the obvious question of *why* people might behave this way, or *why* they might have such preferences, goes unanswered. The remainder of this essay is an attempt to explain *why* this discrepancy between long-term goals and short-term behavior has arisen, and what the answer implies more broadly for the modern theory and practice of welfare economics.

Recall, if you will, the material questions with which this investigation began: How people decide how much to eat, and why overeating is viewed as a problem of self-control. Because eating is an activity

⁸ Gul and Pesendorfer (2001) introduce just such an axiom, which they refer to as *set betweenness*.

common in animals as well as humans, it seems sensible to ask what biology has to say on the subject. Most animals, after all, also solve the problem of when and how much to eat, and they also sometimes overdo it when the opportunity arises. So perhaps a look at the biological underpinnings of feeding behavior will shed some light on the corresponding behavioral phenomenon in humans.

In the spirit of the laboratory scientist making use of mice and rats in his initial investigations of a phenomenon relevant to the human condition, I will begin by offering a simple model of energy allocation in rodents. Specifically, in the next section I consider the problem to be solved by natural selection (“Nature”) when a wild rodent population faces periodic food shortages, and what might be expected to happen if food security is suddenly improved. The relevance of the rodent metaphor to human behavior will be addressed in Section 4.

3. A Biological Theory of Energy Homeostasis

3.1 *Risk and Foraging*

Perhaps the most fundamental task faced by a foraging animal is that of meeting the twin goals of survival and reproduction while faced with an uncertain food supply. As such, the problem is well known among behavioral ecologists⁹, and it has received considerable attention in the “optimal foraging” literature. The model presented below, in which the optimal level of energy reserves is determined as a function of stochastic variations in food availability, is adapted from this literature (see, e.g., Houston and McNamara, 1999).

Generally speaking, a foraging strategy is *optimal*—and therefore constitutes a solution to the *fitness maximization problem*—if no other available strategy results in a greater number of descendants surviving in the distant future. The imprecision of this maddeningly vague definition is intentional: greater

⁹ Behavioral ecology is the study of the relationships between animal behavior and the (physical, biological, and social) environment in which the behavior evolved.

precision quickly invites more complexity than is needed at most levels of analysis. Acknowledgment of the phenomenon of sexual (as opposed to single-parent or asexual) reproduction, for example, necessitates an accounting not just for the number of descendants, but also for the degree of genetic relatedness of those descendants and for the interactions between paternal and maternal genes. In many cases, ignoring this complication will make the analysis tractable and still capture the essence of the problem in question.¹⁰ Being more specific about the time at which descendants are counted is also tricky: if “distant future” is defined as the end of the individual’s life, then it may be important to account for the age and quality of offspring at time of death; but if “distant future” is too far off, then the probability of extinction increases and must be considered.

Because the question to be addressed here is that of the optimal level of energy reserves in the presence of a fluctuating environment, several simplifying assumptions are appropriate. I will consider a population of rodents that reach adulthood after one period, and do not age (though they may die) in subsequent periods. This explicit dismissal of life cycle effects is equivalent to the twin conditions of strong forward convergence and strong backward convergence in dynamic programming; it implies that the actions specified by the optimal strategy will be independent of initial state and terminal reward. Initially, it is also assumed that information (with respect to the underlying probability distribution on environmental fluctuation) does not vary with time, so that the optimal strategy will also be independent of the time period in which the action is chosen. Reproduction is asexual and mutation is very rare.

Uncertainty in the food supply will be captured by an exogenous binary state variable representing the “season” or relative abundance of food $\theta \in \{p, s\}$, where p denotes relative plenty (or “feast”) and s denotes relative scarcity (or “famine”). Seasons are determined by a stationary two-state Markov process

¹⁰ An exception is discussed in Section 3.3.1 below, in which one explanation of an empirical phenomenon makes use of the fact that reproduction is sexual for the species in question.

with transition matrix $\mathbf{P} = \begin{matrix} & \begin{matrix} p & s \end{matrix} \\ \begin{matrix} p \\ s \end{matrix} & \begin{bmatrix} P_{pp} & P_{ps} \\ P_{sp} & P_{ss} \end{bmatrix} \end{matrix}$, $\sum_{\theta \in \{p,s\}} P_{\theta\theta} = 1$, where $P_{\theta\theta}$ denotes the probability of state θ ,

given that the state was θ' in the previous period. Risk is aggregate: in any given period, each member of the population faces the same value of θ .

An *action* is an allocation of energy in a given time period to energy reserves (body fat, $f \in [0, \infty)$) and other uses ($x \in [0, \infty)$). Attention will initially be restricted to stationary solutions of the fitness maximization problem such that the choice of x is a function only of season in the current and the previous periods ($x = x_{\theta\theta}$), while the choice of reserves f is a function only of season in the current period ($f = f_{\theta}$).¹¹ The choices f_{θ} and $x_{\theta\theta}$ are constrained by an exogenous endowment of energy income I_{θ} and the (constant) rate c_{θ} at which energy held in reserves from the previous period can be converted to other uses, (or conversely, if $f_{\theta} > f_{\theta'}$ the constant rate at which energy income can be converted to current-period reserves): $x_{\theta\theta} = I_{\theta} + c_{\theta}(f_{\theta'} - f_{\theta})$.

As is the practice in the optimal foraging literature, the analysis here will assume there exists a (continuous, non-negative, twice differentiable, and strictly concave) function $r(x_{\theta\theta}, f_{\theta})$ that measures the expected number of offspring (including the parent, if the parent survives to the next period) produced during the current period, given an action $(x_{\theta\theta}, f_{\theta})$. A *strategy* specifying actions for all possible contingencies is transmitted genetically to offspring; individuals following a given strategy constitute a *genotype*.

It is well known in the behavioral ecology literature that in the presence of environmental fluctuations (i.e., risks that affect the entire population) evolution will tend to select agents that maximize the

¹¹ The latter, an admittedly strong assumption, will be justified for each special case considered below.

geometric mean fitness (see, e.g., Houston and McNamara, 1999; Bergstrom, 1997; or Robson, 1996).¹²

In the present context (and assuming $P_{ps}, P_{sp} > 0$), this implies that the genotype that will come to dominate the population in the long run will employ the strategy that maximizes the following expression:

$$g = \left[r(x_{ss}, f_s)^{P_{ss}} r(x_{sp}, f_p)^{P_{sp}} \right]^{\frac{P_{ps}}{P_{ps} + P_{sp}}} \left[r(x_{pp}, f_p)^{P_{pp}} r(x_{ps}, f_s)^{P_{ps}} \right]^{\frac{P_{sp}}{P_{ps} + P_{sp}}}$$

Note that the exponents $\frac{P_{ps}}{P_{ps} + P_{sp}}$ and $\frac{P_{sp}}{P_{ps} + P_{sp}}$ denote the unconditional probabilities of a given season

being p or s , respectively in the long run.

A more complete treatment of the problem might endogenously generate the function $r(\cdot)$ that maps allocations to offspring. For example, the model might be expanded to include Nature's choice of our representative rodent's body size and shape, and the form of various internal organs, all of which might affect the rodent's ability to store energy as body fat (and hence alter the function $r(\cdot)$).¹³ In order to keep the model tractable, I will assume only that there exists some "optimal" level of body fat f^* such that in state θ , fitness is increasing in f_θ ($\frac{\partial r}{\partial f_\theta} > 0$) for $f_\theta < f^*$ (presumably because body fat is useful for

within-season energy regulation and other bodily functions such as insulation or shock absorption) and

¹² A simple example can illustrate this principle. Suppose feast and famine are equally likely, and there are only two feasible strategies: Strategy 1 yields a fitness of $r=0$ during famines (i.e., all individuals implementing the strategy die without reproducing, so that $r_1(x_{\theta_s}, f_s) = 0$) and a fitness of $r=2.1$ during feasts ($r_1(x_{\theta_p}, f_p) = 2.1$); Strategy 2 yields a fitness of $r=1$ each season ($r_2(x_{\theta_\theta}, f_\theta) = 1$). Note that Strategy 1 maximizes the arithmetic mean ($a_1 = (0 + 2.1)/2 = 1.05 > 1 = a_2$) number of offspring while Strategy 2 maximizes the geometric mean ($g_1 = \sqrt{(0)(2.1)} = 0 < \sqrt{(1)(1)} = 1 = g_2$). If the risk of famine for any given individual is uncorrelated with the risk faced by other members of the population, then the number of individuals employing Strategy 1 will increase, on average, by 5% each period, while the number of individuals following Strategy 2 remains, on average, constant. Thus in the presence of independent risks (and assuming the first genotype does not go extinct), the strategy that maximizes the arithmetic mean number of offspring will come to dominate the population in the long run. On the other hand, if the risk of famine is aggregate—so that all members of the population face either feast or famine in any given period—then the first occurrence of famine will eliminate Strategy 1 from the population, while Strategy 2 again maintains a stable population size. Thus in the presence of aggregate risk, the strategy that maximizes the geometric mean dominates in the long run.

¹³ Indeed, Section 5.4 below will offer evidence that such endogeneity may be empirically relevant.

decreasing in f_θ ($\frac{\partial r}{\partial f_\theta} < 0$) for $f_\theta > f^*$ (presumably because excessive body fat generates the physiological problems of obesity and incurs costs associated with greater body mass such as impaired agility or higher energetic costs of movement).

3.2 Evolutionary Equilibrium with Seasonal Scarcity

Many common sources of food scarcity in natural environments are cyclical, so an instructive special case of the general problem stated above is that of seasonal scarcity. Seasonality is implied by the

transition matrix $\mathbf{P} = \begin{bmatrix} 0 & 1 \\ 1 & 0 \end{bmatrix}$, which allows the fitness maximization problem to be stated as a simultaneous choice of seasonal allocations:

$$\max_{x_{ps}, f_s, x_{sp}, f_p} g(x_{ps}, f_s, x_{sp}, f_p)$$

subject to

$$x_{ps} = I_s + c_s(f_p - f_s) \text{ and } x_{sp} = I_p + c_p(f_s - f_p)$$

where

$$g(x_{ps}, f_s, x_{sp}, f_p) = r(x_{ps}, f_s) \cdot r(x_{sp}, f_p)$$

Assuming an interior solution, the fitness-maximizing allocation is characterized by the first-order conditions:

$$\frac{\partial g}{\partial f_s} = c_s \frac{\partial g}{\partial x_{ps}} - c_p \frac{\partial g}{\partial x_{sp}} \text{ and } \frac{\partial g}{\partial f_p} = c_p \frac{\partial g}{\partial x_{sp}} - c_s \frac{\partial g}{\partial x_{ps}}$$

which together imply $\frac{\partial g}{\partial f_p} = -\frac{\partial g}{\partial f_s}$. As shown in Figure 3, the energy budget in a given period is

endogenously determined by the choice of f in the previous period, and (assuming $\frac{g_3}{g_1} < \frac{c_s}{c_p} \leq 1$, where g_i

denotes the partial derivative of $g(x_{ps}, f_s, x_{sp}, f_p)$ with respect to its i^{th} argument, evaluated at the

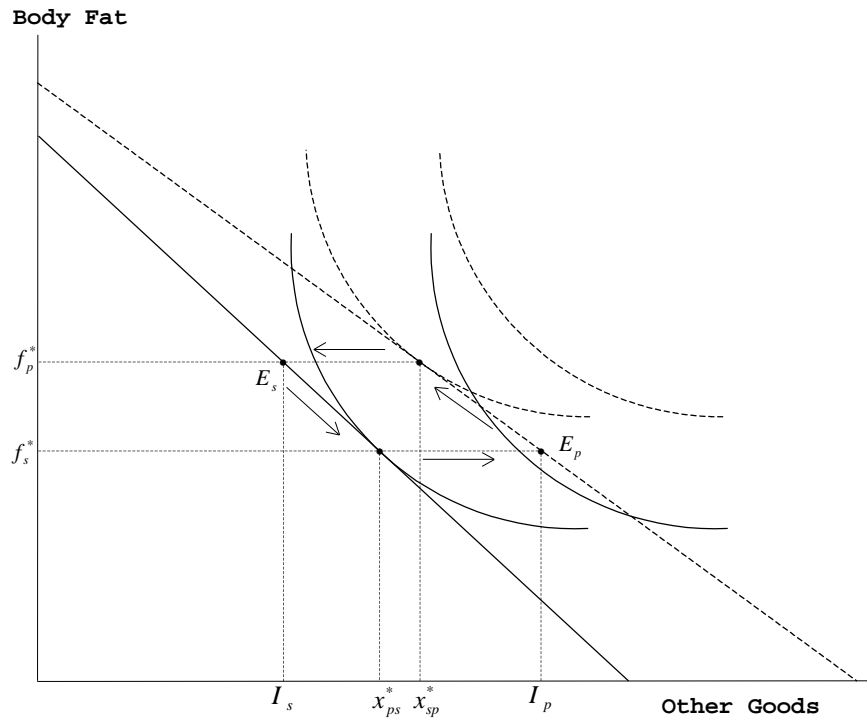


Figure 3: Evolutionary Equilibrium with Seasonal Scarcity

In evolutionary equilibrium the food supply is variable, shown here by a limited energy budget in the scarce season (solid line) and an expanded energy budget in the plentiful season (dashed line). In each season the consumer allocates current-season energy, taking the previous season's choice of body fat (f) as given. Note that the choice of f in times of plenty endogenously determines the endowment of f in times of scarcity, and vice versa (arrows). The fitness implications of the choice are different in times of plenty (when energy is abundant, and extra body fat can be stored for use in the subsequent scarcity) than in times of food scarcity (when energy is scarce, and previously stored body fat is allocated to other uses). Fitness in times of plenty, holding f_s constant, is represented here by dashed isofitness curves; fitness in times of scarcity, holding f_p constant, is represented by solid isofitness curves. In equilibrium, isofitness curves are observationally equivalent to indifference curves.

optimum)¹⁴ Nature finds it optimal to choose $f_p > f^*$ and $f_s < f^*$. Intuitively—as can be seen from the first-order conditions—an excess of body fat is generated during a feast up to the point at which the associated marginal fitness *cost* is just offset by the marginal fitness *benefit* of closing the “gap” (after

¹⁴ If the variables x and f are interpreted strictly as measures of food energy (so that c_s and c_p are marginal rates of physiological conversion) then the requirement that $\frac{c_s}{c_p} \leq 1$ is akin to the Second Law of Thermodynamics, which requires that the entropy

of a closed system never decreases. The requirement that $\frac{g_3}{g_1} < \frac{c_s}{c_p}$ ensures that $g(\cdot)$ is “concave enough” in x to make consumption smoothing optimal.

adjusting for transformation rates) between current consumption (x_{sp}) and consumption in the ensuing famine (x_{ps}). And likewise, the dearth of body fat during famine serves the same purpose—consumption smoothing—when food energy is scarce.

The circular nature of this problem is not unlike that actually faced by wild rodent populations in which the availability of food varies on an annual cycle, the life of any given individual spans a large, indefinite number of cycles, and energy stored in body fat is typically small relative to total energy intake (Vander Wall, 1990).

3.3 *Non-Uniqueness and Phenotypic Polymorphism*

The analysis thus far has implied a unique solution to the fitness maximization problem, suggesting an equilibrium population of uniformly plump rodents. Because real populations in fact exhibit considerable variation in this particular trait, some consideration should be given to the conditions under which such variety might arise. In biology, variation in a physical or behavioral trait within a single population is known as a *phenotypic polymorphism* (a *phenotype* being the observable characteristics of an organism, as determined by both genetic makeup and environmental influences).

In general, there are three ways in which a phenotypic polymorphism might arise: i) mixed strategies, or randomizing by individuals or genes; ii) the simultaneous occurrence of variants of the genes responsible for the trait, or *genotypic polymorphism*; and iii) asymmetric information or payoffs among groups within the population. It is possible that all three are responsible for the observed variation in body fat in rodents and other animals; each will be addressed briefly here.

3.3.1 *Mixed Strategies and Genotypic Polymorphism*

Bergstrom (1997) considers the problem of food hoarding among squirrels, and notes that if only pure strategies are adopted, all but the most risk-averse (high-hoarding) strategy will be eliminated during rare famine events; those squirrels who fail to save enough to survive the famine die and are eliminated from the gene pool. A superior strategy—from the point of view of the gene—is to randomize over hoarding

strategies, so that some carriers of the gene remain after a famine event; while during good times, fitness is increased because not all carriers of the gene hoard excessively.¹⁵ The randomizing device in such a case would presumably be an event in the early developmental stages of the animal, which would by definition be uncorrelated with the risk of famine. There is limited evidence of such strategies in nature, perhaps because such a randomizing device would be inherently difficult to find, but the theoretical prediction is strong enough that it would be surprising if evidence for the phenomenon does not materialize.

There are also a number of reasons to expect the evolutionary equilibrium to be characterized by *genetic* variation with respect to the amount of energy stored as body fat. One of the most compelling, described by Maynard Smith (1998, pp. 74-75) offers a partial solution to the weakness of pure strategies described in the previous paragraph. The reasoning is as follows: if there is cyclical variability in the environment over time (or variability with fixed probabilities), then a *dominant* gene employing the dominant pure strategy may coexist in a stable genotypic polymorphism with a *recessive* gene that employs a less risk-averse strategy.¹⁶ Consider, in the context of the stationary seasonality environment of Section 3.2, a fully recessive gene that employs a strategy yielding a fitness of $r=0$ when food is scarce (i.e., all recessives die without reproducing) and a fitness of $r=2.1$ when food is plentiful, while the dominant gene employs the pure strategy yielding a fitness of $r=1$ each season. This polymorphism is stable with frequency of the recessive gene equal to 0.49 in the wake of any given plentiful season.¹⁷ More generally, the polymorphism will be stable whenever the arithmetic mean fitness of the recessive is

¹⁵ Strictly speaking, this reasoning assumes that the size of the population is infinite. Otherwise there would be a positive probability of the event that *no* carriers of the randomizing gene choose to hoard, and eventual extinction would be certain.

¹⁶ Genes are often characterized as *dominant* or *recessive* because most sexually reproducing species have two copies of each chromosome, and therefore two copies of each gene. In a genotypic polymorphism, the possibility of *heterozygotes*—individuals with different genes at a given chromosomal locus—arises. Heterozygotes sometimes express traits intermediate between the two pure types, but often they favor one type over the other; hence the terms dominant and recessive.

¹⁷ This calculation implicitly assumes random mating, an infinite population, a generation length equal to one season, and the trait in question to be under monogenic control. Real populations are likely to meet few if any of these criteria, but the simplifications seem warranted for illustrative purposes.

less than that of the dominant, while geometric mean fitness of the recessive is greater than that of the dominant (Maynard Smith, 1998).¹⁸

3.3.2 *Social Dominance*

The third explanation for a phenotypic polymorphism—asymmetric information or payoffs—is the explanation given nearly exclusive attention in conventional economic analyses, and considerable attention elsewhere in this essay (footnote 4, for example), so my remarks here will be limited to a single example.

Rodents are social animals, and as such we might expect that within-group social stratification could lead to inequities in access to food supplies. A dominant high-status individual, for example, might (by virtue of his privileged rank) be better able to achieve caloric surplus and store energy as body fat. Thus we would expect adiposity to be increasing with rank. On the other hand, a dominant might (again by virtue of his privileged rank) be less at risk of starvation during times of food scarcity. Thus subordinates would have more incentive to accumulate body fat during times of plenty, and therefore adiposity might be *decreasing* with rank. The relationship between social status and body fat will therefore be a function of absolute levels of food abundance or “wealth”.

The social dominance phenomenon could be incorporated into the framework of the stationary seasonality model (Section 3.2) by allowing periodic income I_θ to vary not only with season, but also with social rank and with wealth. For example, where wealth is high, dominant individuals might enjoy a relatively stable food supply ($I_p \approx I_s \gg 0$) while subordinates face periodic shortages ($I_p \gg I_s \approx 0$); and where wealth is low, dominants might face periodic shortages ($I_p \gg I_s \approx 0$) while subordinates are faced with constant scarcity ($I_p \approx I_s \approx 0$). Behavior in evolutionary equilibrium might then be conditioned on

¹⁸ As shown in Footnote 12, these conditions are satisfied for the two strategies considered here.

all three determinants (season, rank, and wealth) of expected future food supplies. This conjecture is consistent with the comparative statics of the stationary scarcity model: If $g(\cdot)$ is strictly concave,

$$\frac{g_3}{g_1} < \frac{c_s}{c_p} \leq 1, \text{ and } r(x_{\theta\theta}, f_{\theta}) \text{ is separable in } x_{\theta\theta} \text{ and } f_{\theta} \text{ (so that } \frac{\partial^2 r(x_{\theta\theta}, f_{\theta})}{\partial x_{\theta\theta} \partial f_{\theta}} = 0, \theta, \theta' \in \{p, s\}, \theta \neq \theta'),$$

then body fat in times of plenty is increasing in $I_p \left(\frac{\partial f_p^*}{\partial I_p} > 0 \right)$, and the net seasonal change in body fat is

$$\text{decreasing in } I_s \left(\frac{\partial (f_p^* - f_s^*)}{\partial I_s} < 0 \right).^{19}$$

These examples should make it clear that intra-population variation in body fat, whether arising from mixing or from genetic or environmental variation in the equilibrium population, is fully consistent with the evolutionary equilibrium described in the preceding sections, and with genetic transmission of feeding strategies.

3.4 Bayesian Priors and Behavioral Flexibility

An important feature of the solution obtained above is prominence of the marginal rates of transformation (c_p and c_s) and incomes (I_p and I_s) as determinants of choice. These parameters are analogous to prices and income in models of consumer choice, and as such might be thought of as being subject to change from time to time. That is to say, the genetic transmission of fattening strategies specified by the first-order conditions in Section 3.2 does not preclude flexibility in the face of changing environmental constraints.

If there is also variability over time with respect to the *frequency* of food shortages, we might also expect behavior to be sensitive to new information regarding the risk of food shortages. This will require a weakening of the assumption of strong forward convergence, as the optimal strategy will become a

¹⁹ Proofs are provided in the Appendix.

function of age and experience.

Specifically, consider now the special case in which the transition matrix for season is given by

$\mathbf{P} = \begin{bmatrix} 1-\pi & \pi \\ 1-\pi & \pi \end{bmatrix}$, so that in any given time period food will be scarce with probability π and plentiful with

probability $1-\pi$, independent of which state obtained in the previous period. Assume furthermore that

the income constraint is non-binding in times of plenty (so that the rodent is satiated $\left(\frac{\partial g}{\partial x_{\theta p}} = 0 \right)$

whenever food is plentiful), and that the probability of famine is very low, so that the probability of two

successive famines is negligible ($\pi^2 \approx 0$). This reduces the intertemporal choice problem to one of storing

sufficient body fat during times of plenty in order to survive an occasional famine. Specifically, given

that food is plentiful at time t , the fitness maximizing choice of f_p will solve:

$$\max_{f_p} A r_{i|p}(f_p) r_{i+1|s}(x_{ps}, f_s)^\pi$$

subject to

$$x_{ps} = I_s + c_s(f_p - f_s)$$

where $r_{i|j}(\cdot)$ denotes the expected number of offspring in period $i+1$ given that $\theta = j$ in period i , and A

represents the geometric mean of future fitness (exogenous for the purposes of the period t decision). The

first-order conditions for the problem can be written:

$$\pi r_{i|p} r'_{i+1|s} + r'_{i|p} r_{i+1|s} = 0$$

or equivalently,

$$\pi = \frac{-\eta_t}{\eta_{t+1}}$$

where $\eta_t = \frac{f_p}{r_{i|p}} r'_{i|p}$ and $\eta_{t+1} = \frac{f_p}{r_{i+1|s}} r'_{i+1|s}$ might be referred to as the *fat elasticities of fitness*. Assuming

$r_t, r_{t+1} > 0$, $r'_t < 0$, $r'_{t+1} > 0$, and $r''_t, r''_{t+1} < 0$, the quantity $\frac{-\eta_t}{\eta_{t+1}}$ is increasing in f_p . In other words: the

higher the probability of lean times, the plumper our rodents during times of plenty.

Now suppose that for any given cohort of rodents the value of π is unknown at birth, but constant throughout life. Suppose furthermore that π can take on N discrete values with positive probability, where the probability that π takes value π_i is given by $P[\pi_i]$, where $\sum_{i=1}^N P[\pi_i] = 1$. The first-order

condition now becomes $\sum_{i=1}^N \pi_i P[\pi_i] = \frac{-\eta_t}{\eta_{t+1}}$ at the beginning of life, with the probabilities on the left-hand

side adjusting in a Bayesian manner as experience reveals information about the true value of π .

In practice, the details of the process of Bayesian updating are far from trivial. Not only must Nature choose the correct prior distribution $\{P[\pi_i]\}_{i=1}^N$ from the outset, she must also choose—from the multitude of events that comprise a rodent's life experience—a subset of events that provide information about the risk of famine. Each event must then be individually weighted according to the value of the information it provides about the likelihood of each value of π_i . Nature's method of solving this problem is quite elegant: over time, individual rodents are generated with a variety of prior distributions and a variety of sensitivities to new information, and only those rodents with the correct prior and the correct interpretation of new information survive in the long run. The mechanism by which this variety arises is well known to biology: Parental traits are passed to offspring *genetically*; and genetic variation in traits is generated via the processes of recombination (the “mixing” of maternal and paternal genes) and (much more rarely) random mutation. When the trait in question is a behavioral trait such as the propensity to overeat, the most successful rodents (i.e., those who survive in the long run) will in effect have the correct Bayesian prior *written into their genes*. To paraphrase evolutionary biologist Theodosius Dobzhansky: Natural selection is the process by which information is conveyed from environment to genome

(Dobzhansky, 1968, p. 248).

In effect, Nature generates agents who make the best use of regularities in the environment. If a young rodent observes, for example, that food is plentiful but his parents behave as though a famine were imminent, he might take this to heart and begin preparations himself. On the other hand, he might choose to ignore his parents and base his decisions entirely on the actual sequence of feasts and famines he observes over the course of his own lifetime. Thanks to the information provided by Nature (presumably via “feelings” of hunger and satiety), he need not directly assess the fitness implications of his actions; rather, the evolutionary process favors those individuals who behave *as if* they were aware of the implications: if parental behavior reliably predicts famines, for example, Nature will condition feelings of hunger on parental behavior. So our representative rodent need not make conscious calculations of the marginal fitness costs and benefits of his choice; he need only eat when he’s hungry.

At this point it is appropriate to comment on the role of *cultural transmission* of information from generation to generation. If the true value of π is serially correlated across generations then the behavior of parents might be expected to provide valuable information. On the other hand, if π is intertemporally independent from generation to generation, cultural transmission cannot increase fitness, and on the contrary will *strictly decrease* fitness in cases in which it provides information different from the true prior $\{P[\pi_i]\}_{i=1}^N$. We might expect some degree of intertemporal independence, for example, when food scarcity is a function of an uncertain population size: then, in a sense, a new random draw occurs at the beginning of each generation. Real conditions in nature are likely to lie somewhere between the two extremes, with the optimal solution being the use of both cultural transmission and own experience. The model of Section 3.5 will assume cultural transmission to be unimportant, but this assumption will not affect the main result.

There are important implications of assuming that Nature transmits information genetically in the form of a Bayesian prior. The price of this economy of Nature (i.e., leaving the computational heavy

lifting of fitness maximization to time and the process of natural selection) is that unprecedented changes in the decision environment are not anticipated. By taking advantage of regularities in the environment—which *is* the optimal thing to do, of course, as long as the regularities remain—our rodents leave themselves vulnerable to sudden changes in $g(\cdot)$ or $\{P[\pi_i]\}_{i=1}^N$.²⁰ This is because genes can only be altered when they are passed on to the next generation, and then at a (usually) glacial rate. This is the subject of the next section.

3.5 A Stable Disequilibrium

Now suppose that we take our equilibrium rodent population from the environment of the $\{P[\pi_i]\}_{i=1}^N$ regime and place it in a laboratory environment in which the food supply is constant (i.e., $\pi = 0$). In the absence of sufficient time (recall that the strategies that prevail in equilibrium are passed on genetically and thus cannot be altered within a single lifetime) and evolutionary pressure (more on this later) the inherited behavioral tendencies of our rodent population will continue to dictate the behavior that *in the past* was fitness-maximizing during the plentiful season (i.e., choosing $f > f^*$), as shown in Figure 4. In terms of our simple example, the rodents in the laboratory might—through the process of Bayesian updating—come to behave as though regime π_{\min} (the lowest probability of food scarcity) applies, but *further adaptation is limited by the extent of the distribution $\{P[\pi_i]\}_{i=1}^N$ found in the wild*, under which the evolutionary equilibrium obtained. This can be seen mathematically by considering the limiting values of $P[\pi_i]$ that can be reached after a long sequence of summers:

Consider an individual born in period 1 and define Z_t to be the event that no food shortages have occurred by period t . Bayes' Rule then gives:

²⁰ The notion that behavioral algorithms can (and should) take advantage of regularities in the decision environment has been called *ecological rationality* (Gigerenzer *et al*, 1999).

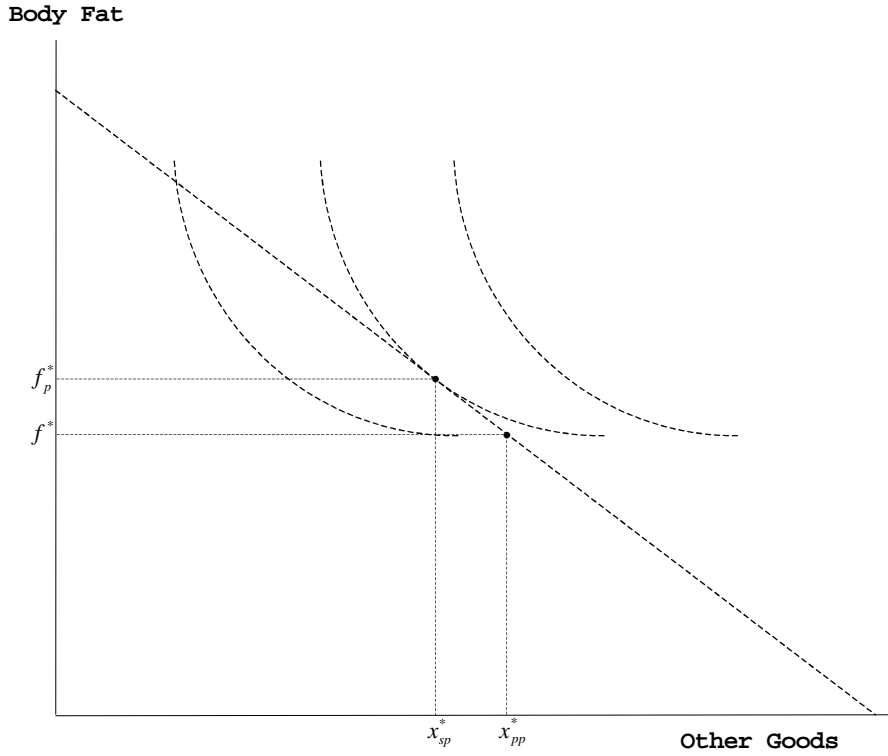


Figure 4: A Stable Food Supply and Evolutionary Disequilibrium

This diagram shows what happens when technology suddenly puts an end to food scarcity, with insufficient time for a new evolutionary equilibrium to obtain. Faced with an abundant food supply the consumer chooses a high level of body fat, as would have been optimal when food supplies were variable. The dashed “isofitness curves” no longer represent fitness tradeoffs, but can still be referred to as indifference curves, (or, perhaps, “Nature’s Thumbprint”) as they remain indicative of the consumer’s (now) exogenous preferences for body fat and other goods. Note that a self-control problem is indicated by the disparity between the chosen allocation (x_{sp}^*, f_p^*) and the hypothetical allocation (x_{pp}^*, f^*) , where the physiological complications of obesity would be avoided.

$$P[\pi_i | Z_t] = \frac{P[Z_t | \pi_i] P[\pi_i]}{\sum_{j=1}^N (P[Z_t | \pi_j] P[\pi_j])}$$

where $P[A|B]$ denotes the probability of the occurrence of event A, conditional on the occurrence of event B; in keeping with the previous treatment $P[A]$ denotes the prior ($t=1$) probability of event A, and π_i denotes the unique event that the true value of π is π_i . Dividing both numerator and denominator by $P[Z_t | \pi_i] P[\pi_i]$ and noting that $P[Z_t | \pi_i] = (1 - \pi_i)^t$ yields:

$$P[\pi_i|Z_t] = \frac{1}{\frac{(1-\pi_1)^t P[\pi_1]}{(1-\pi_i)^t P[\pi_i]} + \dots + \frac{(1-\pi_{i-1})^t P[\pi_{i-1}]}{(1-\pi_i)^t P[\pi_i]} + 1 + \frac{(1-\pi_{i+1})^t P[\pi_{i+1}]}{(1-\pi_i)^t P[\pi_i]} + \dots + \frac{(1-\pi_N)^t P[\pi_N]}{(1-\pi_i)^t P[\pi_i]}}$$

Note that the j th term in the denominator will approach zero as $t \rightarrow \infty$ if $\pi_j > \pi_i$, one if $\pi_j = \pi_i$, and infinity if $\pi_j < \pi_i$. Thus $\lim_{t \rightarrow \infty} P[\pi_i|Z_t] = 0$ for $\pi_i > \pi_{\min}$ and $\lim_{t \rightarrow \infty} P[\pi_{\min}|Z_t] = 1$ where $\pi_{\min} = \inf\{\pi_k\}_{k=1}^N$.

Assuming that food supplies had always been uncertain in the wild ($\pi_{\min} > 0$), the immediate consequence of placing the population in the secure laboratory environment is chronic obesity. This is because if a mutant had arisen during the evolutionary process whose behavior reflected an alternative prior distribution $\hat{P}[\pi_j]_{j=1}^N$ that included even a small probability that $\pi_{\min} = 0$, such a mutant would have strictly lower fitness than its counterparts, endowed with the true distribution $\{P[\pi_i]\}_{i=1}^N$, and the mutant would not survive in equilibrium.

The observed obesity in the laboratory constitutes a state of *disequilibrium* in the sense that preferences no longer serve to maximize Darwinian fitness, yet it is *stable* in the sense that preferences cannot be altered within a single generation.

3.6 False Priors and Self-Control

The feeding strategies employed in this stable disequilibrium fail to solve the fitness maximization problem: in making choices on the basis of a *false prior distribution*, our hypothetical rodent population will choose to store too much energy as body fat ($f_p > f^*$). But can it be said that the behavior in the laboratory constitutes a harmful self-control problem? A brief discussion of this issue is offered below.

First, it is important to note that Darwinian fitness is best defined in terms of long run outcomes (i.e., outcomes averaged over many generations), making any attempt to measure fitness in the short run (i.e., in one or just a few generations) questionable and potentially misleading. But fitness often acts through observable *proximate currencies*, such as health outcomes, and the physiological problems associated

with obesity (e.g., hypertension, heart disease, diabetes, decreased life expectancy) provide evidence that the fattening strategy is less than optimal.

This raises an interesting question. Given sufficient evidence that they would be healthier if they ate less, would our overweight rodents (assuming they could be made to understand the consequences of their actions) go on a diet, or would they continue to overeat, perhaps blaming the problem on the strength of their animal instincts? According to the model presented here, the answer is clear: genetically fixing preferences precludes the flexibility required to adapt to sudden change, even with a lifetime of learning.

To summarize: the initial result of this line of inquiry is a formal model of animal behavior in which food supplies are uncertain, preferences are generated endogenously and (in evolutionary equilibrium) correspond to biological fitness. Because strategies are transmitted genetically, sudden elimination of risk from the food supply will result in chronic obesity and a harmful self-control problem.²¹

This model of equilibrium and disequilibrium in the evolution of feeding behavior in rodents is obviously much too simple to describe the rich complexity of real behavior seen in mice and rats, much less humans. But the conclusions it draws nevertheless seem to capture the essence of what is observed.²² More importantly, if applied to human behavior, two of the assumptions driving the results are unconventional and require elaboration: (i) that strategies (including prior beliefs) are *genetically* encoded, and (ii) that there has been a *sudden* shift in the degree of uncertainty in the human food supply. The next section offers a defense of these two propositions.

²¹ Sozou (1998) describes a simple way in which Bayesian updating can generate the appearance of dynamically inconsistent behavior. Although the author clearly had in mind independent rather than aggregate risks, his *hazard rate* $h(\tau)$ is analogous to the probability of famine π in Section 3.4. Sozou shows that if the prior distribution on $h(\tau)$ has the exponential form then the consumer's discount function will be hyperbolic.

²² The idea that contemporary economic behavior reflects in some way preferences generated by biological evolution is by no means new to the economics literature. See, for example, Hirshleifer (1977, 1985), Rogers (1994), Bergstrom (1996), Romer (2000), Robson (2001), and Samuelson and Swinkels (2001).

4. How Genes Influence Behavior

Over the course of a decade the average adult consumes approximately 10 million calories of food. To account for the relatively modest changes in body weight typically observed, total food intake must be within 0.17% of total energy output (Weigle, 1994). This level of precision in energy homeostasis is no small feat, and recent medical research is beginning to provide the basis for understanding how it is achieved (Woods *et al*, 1998; Friedman and Halaas, 1998; Schwartz *et al*, 2000; McMinn *et al*, 2000). New knowledge of the molecular (i.e., genetic) mechanisms that regulate food intake and energy use provides an instructive example of the way in which genes can influence behavior, and offers a sense of the power of evolutionary forces in shaping human behavior.

4.1 *Nature's Thumbprint*

In the popular lexicon, calling a trait “genetic” implies that it is inherent, hardwired, pre-determined—that it is the result of “nature” rather than “nurture.” On the contrary, there is a strong argument to be made that *no* trait, whether physical or behavioral, can be viewed as the sole product of either nature (genes) or nurture (environment, in the broadest sense of the word). In the words of Leda Cosmides and John Tooby (1997):

Any developmental biologist knows that (nature vs. nurture) is a meaningless question. Every aspect of an organism's phenotype is the joint product of its genes and its environment. To ask which is more important is like asking, Which is more important in determining the area of a rectangle, the length or the width? Which is more important in causing a car to run, the engine or the gasoline? Genes allow the environment to influence the development of phenotypes.

In other words, nature vs. nurture is a false dichotomy. The only legitimate scientific question is the extent to which intra-population *variation* in a given trait can be attributed to genes or environment. The answer in the case of obesity is clearly both, as will be emphasized in Section 4.2 below: variation due to genetic inheritance can easily be established, but there are also many other determinants. Obesity, like other complex behavioral traits, is best viewed as a case of a heritable *susceptibility*, which only manifests

itself when the right conditions prevail.²³ For the purpose of establishing the results of Section 3 above, the sources of variation in modern human populations are unimportant. The simple observation that modern genes interact with modern environments to generate obesity is sufficient.

Another popular connotation of the word “genetic” when used in the context of heritable traits is that such traits vary importantly from individual to individual. This need not be true. While the magnitude of the tendency to accumulate body fat may vary from person to person, the tendency to feel hunger and to eat after periods of fasting is universal. Similarly, the genes that govern the growth and development of five fingers on each hand, or two eyes, or two lungs, are universal. Some people might have smaller hands than others, or poorer eyesight, or a bigger lung capacity, and the source of the variation (i.e., genes or environment) can be debated, but in many cases the variation will be unimportant or due entirely to environmental factors (e.g., nutrition during development, years spent reading books in poor light, time devoted to physical exercise).

4.2 Who Overeats?

While obesity is becoming increasingly common, it does not affect all groups equally. The poor tend to suffer its effects most dramatically in urban areas and developed countries (though the opposite seems to be true in developing countries and rural areas), and those with little education seem to be at risk, as are certain ethnic groups (World Health Organization, 1998; Seidell and Rissanen, 1998; Stunkard, 1996). Another risk factor may be the mere presence of obese family members (related or not) in the household (Garn *et al*, 1984).²⁴ It is clear that the rapid rise in the incidence of obesity in the United States cannot be the result of changes in the genetic makeup of the population, as much of the change has spanned less

²³ The authors of a review of case studies of identical twins raised apart have dubbed these inherited behavioral tendencies “Nature’s Thumbprint” (Neubauer and Neubauer, 1996).

²⁴ There is also some evidence to support the theory (yet to gain full support among the mainstream research community) that a virus may be responsible for some cases of obesity (Dhurandhar *et al*, 2000).

than a single generation. This evidence would seem to suggest a strong cultural influence on the prevalence of obesity.

But there is also evidence of a strong genetic influence on obesity. The influence of genes can be inferred from the comparison of the body mass indexes of twins raised together with twins raised apart, of monozygotic (identical) twins with dizygotic twins, and of adopted with biological parent-offspring pairs. Several studies have done this, and they typically provide estimates of the heritability of obesity (the amount of the variation in the sample explained by genetic influence), which range from 33% to 90%. Heritability estimates are in some sense just a measure of the cultural homogeneity of the population from which the sample is drawn (i.e., a culturally homogeneous but genetically heterogeneous sample would presumably yield a heritability estimate of 100%).²⁵ But the available studies nevertheless find that genes have a discernable (and sometimes dominant) influence on the likelihood of becoming obese (for reviews see Barsh *et al*, 2000 and Bouchard *et al*, 1998).

All together the empirical evidence suggests that it is the *interaction* of genetic background with environmental factors that results in the state of obesity. The conditions that induce obesity in susceptible individuals are no doubt numerous and complex, and the debate over which are most important is far from over.

4.3 *Leptin and the Obesity Genome*

4.3.1 *The ob/ob Mouse*

Common laboratory mice subjected to starvation consistently exhibit a number of characteristic symptoms, including decreased body temperature, hyperphagia (a tendency to eat voraciously when food is available), decreased physical activity, diminished immune function, and infertility. These reactions

²⁵ As noted in Section 3.3.1, an interesting exception to this would be a case in which a gene specified a *mixed strategy*—i.e., random variation in body fat among individuals with the gene. If mixing genes were a source of variation in body mass index in modern human populations, the studies cited above would tend to underestimate heritability, and a well-designed epidemiological study would presumably interpret such variation as measurement error.

have the collective effect of conserving available energy for vital functions, and (in the case of hyperphagia) to hasten the restoration of energy reserves when the opportunity arises. For many years, scientists have also known about a strain of mouse (now known as the *ob/ob* mouse) that carries a recessive gene that induces morbid obesity, *in addition to the above-mentioned symptoms of starvation*.

Early experiments using parabiosis²⁶ provided evidence that the *ob/ob* mouse lacked a hormone-like signaling compound that circulates in the blood, presumably for the purpose of communicating the level of adipose (fatty) tissue available for use; and furthermore that another strain of obese mouse (now referred to as the *db/db* mouse) lacked the ability to detect this signaling compound.²⁷ These hypotheses were confirmed when the *ob* and *db* genes were identified in mice (Zhang *et al*, 1994; Tartaglia *et al*, 1995).

The signaling compound responsible for regulating body fat and behavior in these mice is now known as *leptin*, a small protein molecule secreted by fat cells into the bloodstream. Leptin in the blood is then detected by *leptin receptors* in the brain, in effect communicating the amount of available body fat to the central nervous system.

It is now well established that while all normal mice have a gene that causes fat cells to secrete leptin, the *ob/ob* mice carry a mutant form of this gene that prevents leptin production; likewise, while all normal mice have a gene that causes the leptin receptor to be produced in the brain,²⁸ *db/db* mice carry a mutant form of this gene that prevents production of the leptin receptor. In both cases the signal received by the brain is “no fat stores are available!” and the behavioral and physiological reactions to this message—

²⁶ Parabiosis is the surgical linkage of two organisms so that their circulatory systems interconnect.

²⁷ The experiments went something like this: When the *ob/ob* mouse was joined to a normal mouse, the *ob/ob* mouse lost weight and stopped exhibiting the symptoms of starvation; but when the *db/db* mouse was joined to a normal mouse, the normal mouse lost weight. Although insulin was a well-known signaling compound at the time, both *ob/ob* and *db/db* mice were found to over-secrete insulin, so it was deduced that some other signaling compound must be responsible (Coleman and Hummel, 1969; Coleman, 1973).

²⁸ Specifically, the leptin receptor resides primarily in cells of the hypothalamus, which in turn has extensive neuronal connectivity with other brain regions (DeFalco *et al*, 2001).

mediated by the central nervous system—result in mice that become extremely obese while exhibiting the symptoms of starvation.

4.3.2 *Leptin in Humans*

By all accounts, the discovery of leptin in 1994 stimulated a flurry of research²⁹ into the biochemistry and genetics of mammalian feeding behavior, so much that an “obesity genome map” is now maintained by researchers in the field. Much of this work has been accomplished through the use of mice and rats in laboratory environments, in the hope that a better understanding of the molecular basis of rodent feeding behavior will lead to a better understanding of the molecular basis of human feeding behavior. This hope, it turns out, appears to be well founded. Mouse genes, like human genes, are encoded in some 3.2 billion base pairs,³⁰ and some 90% of genes in mice have homologous³¹ forms in humans (Malakoff, 2000; O’Brien *et al*, 1999). And thus far, every single obesity-related gene found in mice has led to the subsequent discovery of a homologous gene in humans (Barsh *et al*, 2000).

The normal form of the *ob* gene, for example, is now known to encode for leptin in humans as well as mice, and though the genetically inherited *ob/ob* disease is exceedingly rare in human populations, a few cases have been documented. In one such case, a pair of obese *ob/ob* cousins were of normal weight at birth, but rapidly gained weight in infancy, and their parents described both children as “constantly hungry, demanding food continuously and eating considerably more than their siblings” (Montague *et al*, 1997). Injections of a synthetic version of leptin have a dramatic negative effect on appetite in *ob/ob* patients (Farooqi *et al*, 1999).

There were high hopes initially that synthetic leptin might act as a wonder drug, providing the long-

²⁹ No doubt helped along by Amgen Inc.’s well-publicized \$20 million purchase of the commercial drug development rights to leptin in May 1995 (Chicurel, 2000).

³⁰ Base pairs are the molecular “letters” of DNA that comprise the genetic code.

³¹ Similar genes in different species are considered to be homologues if they are found in the same location on the homologous chromosome, and have nearly identical nucleic acid (DNA) sequences.

sought “cure” for obesity and overeating. While clinical tests are ongoing, early results have not been as promising as once hoped (Heymsfield *et al*, 1999). Further investigation has revealed that most obese persons have above-normal levels of leptin circulating in their blood, implying a resistance to leptin’s effects, much like the resistance to insulin that characterizes type 2 diabetes (Considine *et al*, 1996).

4.3.3 *Molecules of Hunger and Long-Term Energy Homeostasis*

As of October 2001 the obesity genome map included more than 250 genes associated with feeding behavior in humans (Rankinen *et al*, 2002). Nearly all of these genes are associated with hormone-like molecules that, like leptin, are thought to communicate information about the nutritional needs of the body. The secretion of the leptin molecule into the bloodstream, for example, is ascribed to a gene located on chromosome 7 that is expressed³² only in the cells of adipose tissue (i.e., body fat). The leptin receptor—the protein responsible for detecting the presence of leptin in the bloodstream—on the other hand, is due to a gene located on chromosome 19 that is expressed in certain cells of the hypothalamus in the brain, and at the blood-brain barrier. Other genes generate *insulin* (secreted by the pancreas in response to high levels of blood sugar) and its receptor (expressed in cells of the hypothalamus, liver, muscles, and adipose tissue); the “satiety hormone” *cholecystinin (CCK)*—secreted by the small intestine after a large meal—and its receptor in the brainstem; and a number of neurotransmitters found primarily in the brain (and apparently specific to the regulation of bodily energy) with names like *neuropeptide Y*, *α-melanocyte-stimulating hormone*, and *thyrotropin-releasing hormone* (Stryer, 1981; Schwartz *et al*, 2000; McMinn *et al*, 2000; Woods *et al*, 1998).

The picture emerging from this research into the biochemical minutia of mammalian body weight

³² Every cell in the body, including brain cells, fat cells, skin cells, etc., contains a *nucleus* which contains copies of the 23 pairs of chromosomes which comprise the human genome. The DNA sequence contained in each chromosome includes thousands of genes, but these genes are *expressed*—that is, made active—quite selectively. Expression implies the synthesis of a protein molecule or *peptide* with properties specific to the gene. Peptides have unique three-dimensional structures and can be employed, for example, as cellular machinery, structural material, or informational signals.

regulation is that of a system that maintains energy homeostasis through the manipulation of a number of physiological and behavioral variables, including feeding behavior. The *adiposity signals* leptin and insulin both circulate in the blood in proportion to the mass of adipose tissue, and trigger pathways in the brain that, *ceteris paribus*, decrease food intake and increase energy expenditure (via, for example, increased spontaneous physical activity and increased body temperature). These twin signals and the associated pathways in the brain serve as long-term regulators of body weight, and maintain stable levels of body weight through their interaction (admittedly in a manner not yet well-understood) with the short-term, meal-related regulators of hunger and satiety such as CCK.³³

4.4 Calibration of Ligand/Receptor Systems

Leptin provides an example of a *ligand/receptor system*, which is the body's primary tool for transmitting physiological information from one part of the body (in this case, adipose tissue) to another (in this case, the brain). Receptors and ligands are analogous to locks and keys, respectively, with the ligand (e.g., leptin) being a small molecule with unique chemical properties and three-dimensional structure that give it the unique ability to activate its receptor (e.g., the leptin receptor). The activated receptor, in turn, induces cellular changes, often with magnitudes proportional to the amount or concentration of ligand present (see, e.g., Stryer, 1981; Pert, 1997).

There are a number of ways in which variations in genes might "calibrate" the molecular machinery that regulates energy homeostasis. Section 4.3.1 described the *ob/ob* mutation, which completely inactivates the leptin signal and generates morbid obesity; but it is possible that other, milder mutations in the genes for ligand/receptor systems might simply reduce the residence time of the ligand in the receptor, or reduce the stability of the ligand (i.e., reduce the amount of time the ligand remains in the

³³ This biochemical model of the regulation of body weight is fully consistent with the notion that a virus might be responsible for some cases of obesity. Such viruses are thought to cause only mild symptoms during infection, but to inflict permanent damage on the cells of the hypothalamus specific to energy regulation (Dhurandhar *et al*, 2000; Bernard *et al*, 1988; Nagashima *et al*, 1992).

bloodstream before being reabsorbed or decomposing to waste products; leptin, for example, is much more stable than insulin). Other important factors are the *number* of leptin receptors expressed in the hypothalamus, and the extent and nature of *neural connectivity* to the rest of the brain; although the molecular basis for these two processes are not yet well-understood, both are certainly mediated by genes, and thus potential ways in which the strength of the leptin signal might be affected (see, e.g., Gazzaniga, 2000; Gazzaniga *et al*, 1998; Pinker, 1997).

The strong evidence that leptin and other genetically modulated signaling molecules play a role in regulating feelings of hunger and satiety—and that their effectiveness varies from person to person—casts the popular view of overeating as a problem of “weakness of will” in a new light. Rather, it would seem more appropriate to conclude that *people simply eat when they feel hungry*, that they stop eating when they feel full, and that some people become overweight because (genetically modulated) signals of hunger and satiety tell them to do so under certain conditions (e.g., those conditions prevalent in today’s Western societies).

This is not to say that overeating cannot be viewed as a choice. The evidence outlined here is entirely consistent with the (consciously) rational thought process we all know from experience. The point here is that *feelings* of hunger and satiety are generated by a molecular process that begins with our genes.

4.5 *The Fixed Nature of Genetic Traits*

To most people “evolution” implies a dynamic, ever-changing process. But if the evolutionary process in question is one of *biological* evolution, in which genes encoded in DNA are altered by the process of natural selection, evolution can be exceedingly slow. Modern science tells us that for an individual human being, DNA can be modified only at birth, and then only by rare mutations and the process of recombination (see, e.g., Wessels and Hopson, 1988). It is possible that, at the population level, certain genes might increase (or decrease) in frequency over a period of decades, but such changes are most pronounced in small or decreasing populations, and in the presence of strong selective

pressure.³⁴

Needless to say, these conditions are not likely to be met for the modern human population. Not only have global populations been increasing for thousands of years (Harpending *et al*, 1998), the risk of death from starvation has only been eliminated in the last century or two (and even today, only in wealthy nations).^{35,36} It seems implausible that 200 years of food security would be sufficient to re-calibrate the human genome to the new conditions of certainty, especially considering that such conditions are unprecedented (and hence not represented in the extant gene pool) in human evolutionary history. Even less likely are significant changes in gene frequencies in the few decades in which the modern obesity epidemic has occurred.

The evidence discussed here suggests a simple model of human behavior. If we take as given that the human genome is the product of the distant past, and that human preferences for food consumption are to some extent encoded in our DNA, then we should not expect these preferences to remain “optimal” in the face of rapid technological change.³⁷

³⁴ “Selective pressure” is a euphemism for conditions under which individuals with a given heritable trait die without reproducing.

³⁵ There can be little doubt that peoples solely reliant on foraging and subsistence agriculture were faced with an uncertain food supply. Consider the findings of a broad survey of traditional societies: in 29 percent, shortages were rare (every ten to fifteen years); in 25 percent, occasional (every two to three years); in 23 percent annual; and in 23 percent, more than once a year (Konner, 2002, p. 390).

³⁶ Archaeological evidence tells us that humans began the transition from foraging to subsistence agriculture around 10,000 years ago (see, e.g., Boyd and Silk, 2000), and this may well have represented an increase in food security. But small-scale agriculture in the absence of inter-regional trade was still subject to devastating climatic fluctuations, and historical records bear this out. Inhabitants of Western Europe, for example, were subjected to widespread famine as recently as 1849, when the Irish Potato Famine resulted in more than a million deaths (approximately 12% of the population); and this was not an anomaly of history: the population had previously survived famine events in 1816-18 (death rates rose by 50%), 1740, 1693-94 (10% of Louis XIV’s subjects died in France, and as much as one-third of the population in other regions), and 1315-21 (the so-called Great Famine, which resulted in the death of as much as 10% of the population) (Fagan, 2000). Even in the wealthiest nations today, the realities of geography and climate result in seasonal variation in the price and availability of fresh produce—and hence nutrition—so perhaps we still cannot call our food supply “constant.”

³⁷ There have been a number of authors who have employed an evolutionary approach to model human time preferences (e.g., Hansson and Stuart, 1990; Rogers, 1994) and self-control (Samuelson and Swinkels, 2001). These models generally rely on an equilibrium assumption (as opposed to the disequilibrium assumption I have employed), thus de-emphasizing differences between modern decision environments and those to which humans are presumably adapted.

5. Empirical Evidence for Thrifty Genes

The model of obesity offered here might seem counter-intuitive, and a bit unlikely—I have suggested that when food is abundant people are overeating in preparation for an admittedly phantasmic food shortage.³⁸ But an important measure of any theory that purports to describe human behavior is its predictive power. To paraphrase Milton Friedman’s 1953 essay, the proper test of a positive theory of feeding behavior is not whether people consciously overeat in order to prepare for a famine; rather, the proper test is whether they behave *as if* they were preparing for a famine. Indeed, I have taken Friedman’s defense of the *as if* approach one step further by attempting to show *why* we might expect limits to conscious control over the behavior: because in our evolutionary history such conscious control would have been at best useless, and at worst resulted in starvation. In the language of probability theory, the prior distribution we are born with precludes flexibility; such flexibility would have been harmful to our ancestors, and because of this the preferences they have passed on will continue to haunt us for generations to come.

But a lack of flexibility with respect to storage of body fat in the presence of rapidly improving food security is not the only implication of an evolutionary model of feeding behavior. Natural selection will tend to favor strategies that take advantage of valuable environmental cues, and condition behavior accordingly.³⁹ So any technological change that removes the probabilistic link between cues (whatever they might be) and outcomes is a potential source of evidence for the influence of “Nature’s Thumbprint” on behavior.

³⁸ The notion that modern human populations are genetically maladapted to the modern diet and lifestyle was first proposed in detail by human geneticist James Neel in 1962. Neel’s “Thrifty Genotype” hypothesis has gained growing acceptance among the scientific community as supporting evidence accumulates (Neel, 1962; 1999).

³⁹ For examples of the sensitivity of humans to behavioral cues, see Laibson (2001) and Samuelson and Swinkels (2001).

5.1 Seasonality and Body Fat

It is common among foraging animals in natural environments to observe seasonal patterns in the amount of energy stored as body fat. These patterns, when observed in the field, tend to reflect local variations in the availability of food: animals generally fatten during times of relative plenty, and shed fat during times of relative scarcity. While this observation may seem perfectly intuitive and unsurprising, it does not immediately explain how animals “know” when a food shortage is imminent. It might be, for example, that food availability is the only cue needed to trigger a fattening episode; on the other hand, a given species might rely on more dependable environmental cues (such as, for example, population density, ambient temperature, or recent weather events) to regulate and maintain an optimal level of energy reserves. Laboratory environments provide scientists with the opportunity to vary food supply and environmental cues independently, and thus to make inferences about the specifics of the evolved behavioral algorithm.

It turns out that in most mammalian species studied, *photoperiod*, or length of day, is one of the most important seasonal cues for fattening. Photoperiod is, of course, a very reliable indicator of season in natural environments, with the minimum photoperiod occurring annually at the winter solstice and the maximum occurring at the summer solstice. The most thoroughly studied rodent model of photoperiodic regulation is the Siberian hamster, which has the demonstrated ability to regulate its body mass progressively and continually according to its photoperiodic history—independently of the amount of food provided in the lab. Further investigation has shown the pineal hormone *melatonin* plays an important role in photoperiodic regulation of body fat in mammals (Mercer, *et al*, 2000).

There is some intriguing evidence that our own species also utilizes photoperiod as a seasonal fattening cue. There is a condition known as *seasonal affective disorder*, a mental illness characterized in humans by depression, hypersomnia (excessive sleeping), hyperphagia (excessive eating), and weight gain. Seasonal affective disorder derives its name from the fact that it typically occurs in winter, and

heritability studies have shown that incidence can be at least partly explained by genetic factors. The most effective (and most commonly prescribed) treatment for seasonal affective disorder is prolonged daily exposure to intense artificial light. At the opposite end of the seasonal spectrum, there is another condition known as *summer depression*—also demonstrably heritable—the victims of which tend to suffer from insomnia, decreased appetite, and weight loss (Allen *et al*, 1993; Madden *et al*, 1996).

There is also evidence of a seasonal trend in weight gain among the general population: according to one recent study, the average adult American gains one pound between September and January, which is partially offset by an ensuing loss between January and March (Yanovski, *et al*, 2000).⁴⁰ The conventional wisdom, of course, is that seasonal variation in diet and opportunities for exercise are responsible for the “holiday weight gain” phenomenon, but this notion has yet to be confirmed in controlled studies. The dependable coincidence of rich holiday desserts and cozy firesides with the winter solstice makes it difficult to draw conclusions about the relationship between photoperiod and body fat based on data collected from human populations, but the parallels to animal seasonality and the light-sensitivity of seasonal affective disorder are suggestive.

5.2 Social Dominance in Birds and Mammals

The model of Section 3.3.2 suggests conditions under which body fat might be a function not only of season, but also of social status and wealth. There is evidence that this is true in natural environments: an example is to be found in field studies of overwinter fattening among willow tits (small insectivorous birds) in Sweden (Ekman and Lilliendahl, 1993). Though it is well-known that dominant willow tits enjoy priority access to prime foraging sites, Ekman and Lilliendahl found that subordinates had greater stores of body fat. Clark and Ekman (1995) offer a theoretical explanation, based on an underlying logic

⁴⁰ Similar reports are available for other populations: Van Staveren *et al* (1986) report peak body weights in a sample of Dutch women in December and January, with minimum weights occurring in June and July; Dietz and Gortmaker (1984) report seasonal effects on childhood obesity.

similar to that explained in Section 3.3.2 above. A summary follows.

In a dynamic optimal foraging model, Clark and Ekman show that if food is scarce, the probability of starvation is high for both types, and the dominant will store more body fat by virtue of his privileged access to high-quality foraging areas; if food is plentiful, however, the dominant chooses a lower level of body fat, taking advantage of the fact that in times of need he will (by virtue of his dominant status) still be able to obtain sufficient food supplies. So the relationship between body fat and social status can be reversed, depending on local conditions of food availability or “wealth” (Clark and Ekman 1993, fig. 1a). The phenomenon is not limited to little birds: Shively and Wallace (2001) have studied social status and body fat in cynomolgus monkeys and found a strong association between low social status and abdominal obesity.

The parallel to human obesity is striking: as noted in Section 4.2 above, the poor are more likely to be obese than the rich in wealthy countries, while the opposite is true in poor countries. Given the observed patterns in animal behavior, it is tempting to conclude that the association of obesity with poverty is due to an innate tendency to accumulate fat stores when one’s social rank is low. Presumably this tendency would have served to maximize fitness if food security varied with social rank in ancient human societies, in the manner described in Section 3.3.2.

This is not to say that poor people in wealthy societies consciously contemplate the possibility of starvation. Rather, if the metaphor of the rodents and the willow tits proves apt, it would likely be the *endocrine state* associated with poverty that results in obesity: individuals with low social status might be more likely, for example, to exhibit elevated levels of the stress hormones such as cortisol, and decreased levels of the neurotransmitter serotonin.^{41,42} This altered blood chemistry might well trigger increases in

⁴¹ The evidence of a relationship between serotonin and social status derives primarily from experimental studies with nonhuman primates, in which social rank is readily determined through observation of stable dominance relationships. See, for example, McGuire, *et al* (1984), which reviews some early findings relating serotonin to social rank in captive colonies of

body fat independently of any real or perceived threat of starvation. There is accumulating evidence that this is in fact the case: weight loss is a well-known side effect of antidepressants such as Prozac, which increase serotonin levels in the brain, and at least one such drug is now being marketed explicitly for the purpose of weight loss.⁴³

5.3 Food Insecurity

Although death by starvation is a rare event in the U.S., a surprisingly large proportion of households experience periodic food shortages. According to a recent USDA report, 10.7 percent of U.S. households were “food insecure” in 2001, where food insecurity is measured by survey responses indicating the extent to which resource constraints caused hunger (Nord *et al*, 2002).

An interesting phenomenon has been noted in the food insecurity survey data: food insecurity is in some cases associated with an increased risk of obesity (Olson 1999; Townsend *et al*, 2001; Basiotis and Lino, 2002). The effect is strongest among mothers experiencing mild to moderate food insecurity (many of whom reportedly forgo an occasional meal in order to ensure that her children can eat), and the effect remains after controlling for income and other demographic variables. Although the association of food insecurity with obesity has been labeled a “paradox” by several authors (after all, the obese don’t exactly appear to be *starving*, do they?) it is very much in accordance with the view that obesity is a natural phenomenon likely to be exacerbated by an increased risk (i.e., the parameter π in the model of Section 3.4) of food shortages.⁴⁴ Again, this behavioral response to food insecurity need not be a conscious

vervet monkeys.

⁴² A recent report by Rosmond *et al* (2002) provides indirect support for this hypothesis. In a sample of 284 men, they found an association between a mutation in a serotonin receptor gene and both abdominal obesity and salivary cortisol. The authors hypothesize that those with “...genetic vulnerability in the serotonin receptor gene...” might be susceptible to “...stress factors that destabilize the serotonin-hypothalamic-pituitary-adrenal system...” which “...might lead to the development of abdominal obesity.”

⁴³ The drug, known as *sibutramine*, is among the class of drugs known as “selective serotonin reuptake inhibitors” or SSRIs (Bray and Tartaglia, 2000). Prozac and the family of related antidepressants are also SSRIs.

⁴⁴ In fact, even before population survey data on food insecurity were available, this hypothesis was offered by Dietz (1995) in

attempt to ward off starvation: the adaptive value of additional body fat is the same whether it is generated by consciously strategic behavior or by a subtle change in the biochemical signals that govern energy homeostasis in humans. And again, the phenomenon is not limited to our species: other mammals subjected to artificially induced fluctuations in food availability will also respond by increasing stores of body fat (Kochan *et al*, 1997).

5.4 Birth Weight as a Conditional Event

Another reliable signal of food scarcity in natural environments is malnutrition early in life. It has been hypothesized that malnutrition during development may “program” energy metabolism later in life:

The human baby responds and adapts to the nutrients it receives by altering its production of hormones and the sensitivity of its tissues to them, by changing its metabolism, and by redistributing its cardiac output to protect key organs... Slowing of growth is adaptive because it reduces the requirements for substrate... (P)hysiological adaptations...made during development tend to lead to life-long changes in the structure and function of the body—a phenomenon sometimes referred to as programming. It is as though the baby receives from its mother a forecast of the nutritional environment it will encounter after birth and changes its physiology and metabolism accordingly. (Barker 2001, p. 1)

The “fetal origins” or “thrifty phenotype” hypothesis was inspired by the empirical observation that low birth weight is associated with metabolic disease later in life (Barker *et al*, 1989; Hales and Barker, 1992). In this view, birth weight is a function of maternal nutrition, the level of which might have served (in natural environments) as a reliable signal of food scarcity. Many other studies, however, have shown a *positive* association between birth weight and adult obesity (Whitaker and Dietz, 1998; Gillman, 2002). By itself, the latter observation would not be puzzling: because birth weight is necessarily influenced by the genetic makeup of the fetus, one might expect an individual’s “fattening strategy” to be apparent from birth. But the fetal origins hypothesis would seem to predict just the opposite: if low birth weight is a signal of food insecurity, then it should increase, *ceteris paribus*, the incidence of obesity observed later

the context of a case study of an obese 7-year-old girl who regularly faced food shortages in the days before her mother received the monthly welfare check.

in life.

One way to reconcile this evidence with the theory of energy homeostasis described in Section 3 is to distinguish between the *level* and the *variability* of future food supplies. The latter was the focus of the analysis of Section 3.4, where it was shown that obesity should increase with increasing variability (as represented by the parameter π). But this analysis also assumed implicitly that an individual's body has been in some sense “designed” to function optimally with some intermediate level of fat stores (represented by the hypothetical “bliss point” f^*) at which the physiological complications of obesity (as well as those of starvation) are absent. One might expect that if a life event (such as low birth weight) served as a reliable signal of the future *level* of food availability (e.g., the expected proportion of one's lifetime spent in the obese state), then an optimal strategy would adjust one's physiological “expectation” of future stores of body fat. Indeed, this seems to be exactly what proponents of the fetal origins hypothesis are saying: “For so long as (the baby) continues to be poorly nourished during childhood and adult life, these adaptations (insulin resistance and accompanying changes in glucose and lipid metabolism) are beneficial. With increased food intake, decreased energy expenditure and the development of obesity, however, the adaptations are no longer beneficial.” (Barker 2001, p. 2) This view is also consistent with the observation that the most severe cases of metabolic syndrome are found among those who are born small but later become obese (Whitaker and Dietz, 1998; Gillman, 2002).

Unfortunately, it remains an open question whether the observed epidemiological pattern is indeed evidence of a “fetal origins” adaptation or simply the product of genetic variation: an individual whose genes imply a lower f^* would be expected to exhibit the same pattern (low birth weight coupled with inordinate sensitivity to the physiological complications of obesity) as an individual who adapted his fattening strategy in response to fetal malnutrition. Although some evidence favors the fetal origins hypothesis—for example: the heritability of birth weight appears to be quite low (Baird *et al*, 2001), and long-lasting health effects have been induced in laboratory animals whose mothers were deprived of

proper nutrition during pregnancy (Bertram and Hanson, 2001)—further research is needed.

5.5 Isolated Human Populations and Rapid Cultural Change

As noted above, the rapid rise in obesity in the United States in the last few decades has happened much too quickly to have been driven by changes in the genetic makeup of the population. More dramatic evidence can be found in the form of “natural” experiments in which small, previously isolated human populations are suddenly introduced to the Western diet and lifestyle. One such instance is to be found on the Micronesian island of Nauru, as described by Jared Diamond (1992) in the following passage:

One of the most serious (type 2 diabetes) epidemics is on Nauru, a remote atoll occupied by 5,000 Micronesians whose formerly energetic lifestyle depended on fishing and subsistence farming. Colonization by Britain, Australia and New Zealand, and income from phosphate mining, transformed Nauruans into one of the world’s wealthiest, most sedentary peoples. Virtually all food is now imported and energy-dense; calorie intake is more than double Australian recommended norms; and obesity is rampant. (Type 2 diabetes) used to be non-existent but a severe form striking many young adults reached epidemic frequencies after 1950 and now affects almost two-thirds of adults by age 55-64. The disease now contributes to most non-accidental deaths on Nauru, with the paradoxical result that wealthy Nauru has one of the world’s shortest human lifespans.

This is another instance in which it is unclear whether genes or environment generate the observed increase in obesity and the associated metabolic disease: it remains a matter of debate whether the changes in the local economic environment experienced by the Nauruans and others like them should be considered “sudden” in the sense that the local gene pool was adapted to historic conditions of scarcity, or “sudden” in the sense that mothers coming of age shortly after westernization passed (false) metabolic information about local conditions to their offspring. In either case, the fact that the most dramatic obesity “epidemics” occur in small, isolated populations with demonstrable histories of periodic famine provides additional support for the view that today’s obesity is a response to yesterday’s scarcity.

6. Discussion

6.1 Obesity, Emotions, and Conscious Deliberation

He who acts under an emotional impulse also acts. What distinguishes an emotional action from other actions is the valuation of input and output. Emotions disarrange valuations. Inflamed with passion, man sees the goal as more desirable and the price he has to pay for it as less burdensome than he would in cool deliberation. (von Mises, 1966, p. 16)

It is one of my fundamental tenets that any satisfactory account of probability must deal with the problem of action in the face of uncertainty. (Savage, 1954, p. 60)

In this excerpt from *Human Action: A Treatise on Economics*, Ludwig von Mises emphasizes the idea that conscious deliberation in the heat of the moment is, nevertheless, conscious deliberation. In so doing he gives voice to the view—implicit in most economic theories of choice—that emotions merely serve as *inputs* to the deliberation process, to be taken into account when considering the various possible outcomes of one’s choice of actions. Thus even “emotional” behavior can be influenced by changing prices, or by (as von Mises points out later in the same passage) a stiffening of criminal penalties.

It is not my intention to contradict this view. The emotions-as-inputs model of deliberate choice (the emotions of interest here being “hunger” and “satiety”) is fully consistent with the model offered herein. Nothing in the formulation of the model in Section 3 implies, for example, that for a given individual, the “choice” of consuming a surplus of caloric energy is independent of variation in the social or economic consequences of being overweight.⁴⁵ But there are important consequences of admitting emotions as inputs to deliberation. This essay has offered evidence that the strength of the “hunger” signal received by the brain is genetically encoded and has been calibrated by natural selection to ensure that starvation is avoided. But because human evolution has proceeded more slowly than recent advances in modern agricultural and transportation technology, there is now a distinct mismatch between the actual risk of

⁴⁵ Said consequences, not being the primary focus of the analysis, are assumed to be implicit in the parameters c_θ and I_θ .

starvation and the risk written into our genes.⁴⁶

The second quotation serves to emphasize that probabilities of future outcomes are necessarily implicit in the emotional signals flowing in our veins. As Leonard Savage showed so elegantly in 1954, there is a strong sense in which *actions* (guided in the Savage formulation by a binary *preference relation*) implicitly assign both subjective values (i.e., utilities) and subjective probabilities to outcomes. In other words, when I choose to take another bite of hamburger, or have another milkshake, my choice says something about how I feel about the ensuing consequences: both how I value them and how likely I believe them to be. This is true regardless of whether my choice is driven by emotion or by reason, or by some combination of the two.

Upon reflection, the preference relation described by Savage seems an apt description of the molecular system of genes and hormones that influence human behavior.⁴⁷ The evolutionary process that generated the human genome is necessarily a probabilistic one, but the “feelings” (e.g., hunger, satiety, satisfaction, outrage) that guide our actions need not come packaged with explicit, conscious knowledge of likely future consequences.⁴⁸ Our emotions help us make decisions in an uncertain world, and for most of our evolutionary history the “information” they provided served us well.

⁴⁶ The role of emotions in strategic behavior has been emphasized by Hirshleifer (1987) and Frank (1988). Just as the emotions hunger and satiety (it is argued here) help consumers solve the energy homeostasis problem, these authors emphasize the value of emotions such as rage, jealousy, and vengeance in helping people solve bargaining problems. Neither analysis emphasizes a “disequilibrium” problem, but the omission is perhaps justified, as the problem of predicting the actions of a human bargaining partner has not been drastically changed by modern technology.

⁴⁷ An important qualification: Savage did not explicitly model multiple-stage decision problems. The “consequences” relevant for the purposes natural selection, even if measured in a proximate currency such as health status, are necessarily spread over long periods of time. But a reasonable conjecture might be that in evolutionary equilibrium, the intensity of pleasure one experiences after taking an action (say, eating a hearty meal) would in some sense be proportional to the expected fitness of that action.

⁴⁸ Indeed, as the discussion of Section 4.3 above illustrated, much of the human body’s “reaction” to molecular signals such as leptin takes place below the level of consciousness: a disruption of the leptin signal triggers not only feelings of hunger, but also the physiology of starvation—including decreased body temperature, diminished immune function, and infertility. Nevertheless, it is hard to deny that genetic mutations that alter leptin’s potency appear to alter the extent to which an individual “believes” that a starvation event is imminent, at least as expressed in behavioral manifestations.

6.2 *Implications for Welfare Economics*

...suppose a person becomes fat from eating large quantities of potato chips. She may do so because of a harmful self-control problem, or merely because the pleasure from eating potato chips outweighs the costs of being fat. Both hypotheses are reasonable explanations for the observed behavior; however, the two hypotheses have very different normative implications. The former says people buy too many potato chips at the prevailing price; the latter says they buy the right amount. (O'Donoghue and Rabin, 1999)

For the average Joe living in the hypothetical ancient world of the evolutionary equilibrium, one might imagine that doing what *felt* right was indeed the right thing to do: he was not burdened with a genetic legacy that conveyed false information about the world; he was not burdened with a problem of self-control. But that world is gone, displaced by technological advances that have largely eliminated any real threat of starvation in our lives and drastically reduced the price of achieving caloric surplus. What can—or should—be done about this problem? A few thoughts will be offered here on the implications of the modern self-control problem for public policy and for the modern theory and practice of welfare economics.

The evidence supporting a biological basis for the self-control problem has the troubling consequence of undermining a foundational tenet of welfare economics. To question consumer sovereignty is to suggest that Adam Smith's (1776) invisible hand is misguided, for reasons not heretofore taken seriously by the economics profession. While economists familiar with the First Fundamental Theorem of Welfare Economics readily admit a role for government in providing solutions to problems such as air pollution that arise when markets are incomplete, they are much less wont to endorse the proposition that consumers might require protection from themselves when deciding how much to eat.

The economic theory of self-control adopts a more conventional and intermediate view: some people might have a problem with self-control, and policy interventions that mitigate the problem might make

those people better off.⁴⁹ This view is conventional in the sense that it implicitly points to a missing market as the cause of the problem: if consumers were allowed to make some consumption decisions in advance (i.e., if a market for pre-commitment existed) rather than in the heat of the moment, the problem would be solved. But closer examination of the biological underpinnings of the self-control problem suggests a more fundamental flaw in the Fundamental Theorem: markets may fail, in some cases, because consumers act on the basis of false beliefs. Allowance might be made, on feasibility grounds, for some error on the part of consumers, but when the error is systematic—as appears to be the case with obesity—then a market failure of a new sort is implied.

It is important in drawing conclusions about human welfare in light of evolutionary history to use caution in defining individual welfare. The “stable disequilibrium” of Section 3.5 implies that in modern environments, consumers may fail to maximize individual *fitness* in the biological, Darwinian sense. But it is another thing entirely to suggest that consumers *should* try to maximize fitness, much less that welfare economists should encourage them to do so. The same can be said about the relevant proximate currency of *health outcomes*: a consumer in the modern world may fail to choose a level of energy reserves that results in optimum health, but again it does not follow that he *should* do so.

But today’s consumer is faced with a very real trade-off between short-term pleasure and long-term health, and there is no *a priori* reason to expect that he should be particularly good at making this choice. Indeed, given the overwhelming body of evidence from modern medical science, it seems likely that the more knowledgeable the average consumer becomes in matters of diet and physiology, the more likely it is that he will make lifestyle choices that favor his long-term health and well-being. Of course, such an education would not come without cost, and it is hard to imagine a large fraction of the population achieving the level of expertise of the medical specialist. But there may be other, more feasible options.

⁴⁹ This view is implicit in the quotation of O’Donoghue and Rabin that opens this section.

It was noted in Section 5, for example, that obesity appears to be exacerbated by poverty, by food insecurity, by the winter months, and by malnutrition early in life. Improving the health and welfare of the general population might, in light of this evidence, consist of such unconventional anti-obesity measures as strengthening the social safety net, promoting the use of artificial light around the winter solstice, or providing prenatal care to expectant mothers. And there are many other possibilities: public education in matters of diet and nutrition, pedestrian-friendly urban infrastructures, and improved food-labeling laws (to name just three) could each go a long way toward helping consumers make choices that enable them to live long and happy lives.

It is interesting to note that although each of the policy measures mentioned above has been applied in one setting or another, many are difficult to justify within a conventional neoclassical framework. It would be difficult, for example, to convince an (otherwise well-trained) policy analyst unschooled in the evolutionary history of our species that the free market couldn't deliver the proper level of education (at least the "self-help" sort of education being discussed here), or that sidewalks and trails should be treated preferentially to roads and parking lots, or that poverty might actually *cause* obesity. But a more informed view of the peculiarities of modern behavior makes it clear that the self-control problem is real, and that it is likely to get worse as technology continues to transform the world in which we live.

Perhaps a more fundamental problem for welfare economics is the question of measurement. The equivalence between *individual choice* and *individual welfare* commonly assumed in welfare economics allows for the estimation and comparison of the impacts of various policies through the simple observation of human behavior. Weakening this equivalency—with the qualification that behavior is individually optimal only when subjective probabilities are equivalent to actual probabilities—poses a difficult problem for policy analysts.

The other social sciences have long been skeptical of the choice/welfare equivalence in economics, and a number of alternative measures of well-being have been devised that might now deserve the

attention of economists (e.g., Kahneman, 1999; Larson and Fredrickson, 1999). Another possibility is that advances in molecular biology and endocrinology will someday provide a solution. The day is approaching when we will be able to say with some confidence whether an individual with a given genetic profile and life history is hungry by measuring blood concentrations of compounds like leptin, insulin, and CCK. Likewise, it may someday be possible to obtain broader measures of well-being in an objective way by measuring blood concentrations of hormones like cortisol, produced during times of stress, or serotonin, which wards off depression (Flinn and England, 1995; Sapolsky, 1999a; 1999b; Ogilvie *et al*, 1996).

7. Concluding Remarks

The central claim of this short essay has been relatively modest: that overeating—the most prominent of self-control problems—is best viewed as a manifestation of the difficult problem of energy homeostasis faced by our ancestors. The support for this claim, I hope you will agree, is far-ranging: it is easily formalized with the mathematical tools (most of which will be familiar to the economic theorist) of behavioral ecology; it is supported directly by emerging knowledge of the human genome and the endocrinology of hunger and satiety; and it is supported indirectly (and in surprising ways) by the incidence of human obesity in the modern world. I have also claimed, in a somewhat more speculative manner, that my findings have important implications for the theory and practice of welfare economics. The unanswered question, if these claims are taken at face value, is whether this approach might be applicable to other realms of economic behavior. If in the end my thesis turns out to apply to body fat and little else, then what I have to offer is yet another anomaly (albeit a very interesting one) for the growing list of behavioral phenomena that don't comfortably fit into a neoclassical framework. But it seems likely that, upon further investigation, other examples will be found of cases in which people seem to be acting on the basis of biased subjective probabilities. The list of “anomalies” catalogued in the behavioral economics literature is long, after all, and the evolutionary psychology literature is rich with examples of

human behaviors that bear the mark of the distant evolutionary past.⁵⁰ And modern science is making clearer every day the myriad ways in which genes influence behavior.⁵¹

There is the danger that this line of research, by dropping conventional restrictions on subjective probabilities and prize spaces⁵², will generate a lot of “just so” storytelling about the forces driving human behavior. This is why it will be crucial to bring solid evidence into the debate every step of the way, and to take full advantage of the rich diversity of behavioral sciences that have long been ignored by the economics profession. It might be possible to generate a theory of overeating, for example, without taking into account evidence from the study of starvation in modern hunter-gatherer societies, or social behavior in birds, or the polymorphism at a particular locus on chromosome 7. But today’s theorist ignores such sources of empirical evidence at his peril, for they can generate surprising (but apparently valid) *a priori* predictions about human behavior and, perhaps, a new way of thinking about human welfare.

8. Mathematical Appendix

Proof that $\frac{\partial f_p^*}{\partial I_p} > 0$: Define the implicit functions $x_s^* = x_{ps}^*(c_s, I_s, c_p, I_p)$, $f_s^* = f_s^*(c_s, I_s, c_p, I_p)$,

$x_p^* = x_{sp}^*(c_s, I_s, c_p, I_p)$, and $f_p^* = f_p^*(c_s, I_s, c_p, I_p)$ as solutions to the fitness maximization problem.

Writing the Lagrangian of the problem as

$$\mathcal{L} = g(x_s, f_s, x_p, f_p) + \lambda_s (I_s - c_s f_s + c_s f_p - x_s) + \lambda_p (I_p - c_p f_p + c_p f_s - x_p),$$

⁵⁰ Examples of the latter have included gossip (Barkow, 1992), dating (Wright, 1994), social exchange (Cosmides and Tooby, 1992), and coalition formation (de Waal, 1998).

⁵¹ It is now known, for example, that aggressiveness is heritable (Coccaro *et al*, 1993) and some of the genes that regulate irritability, anger, and aggression have been identified (Manuck *et al*, 2000). Similarly, genes have been identified for risk-taking (Ebstein *et al*, 1995), shyness (Lesch *et al*, 1996), and longevity (Yu *et al*, 1996).

⁵² In practice, subjective probabilities are usually assumed to reflect actual probabilities, and the prize space is typically taken to be some intuitively plausible set of outcomes (e.g., the wealth from winning a lottery, or the pleasure from eating an ice cream cone) consistent with the conscious experience of the decision-maker.

the first-order conditions can then be expressed as the following six identities:

$$\begin{aligned}
g_1(x_s^*, f_s^*, x_p^*, f_p^*) - \lambda_s &\equiv 0 \\
g_2(x_s^*, f_s^*, x_p^*, f_p^*) - \lambda_s c_s + \lambda_p c_p &\equiv 0 \\
g_3(x_s^*, f_s^*, x_p^*, f_p^*) - \lambda_p &\equiv 0 \\
g_4(x_s^*, f_s^*, x_p^*, f_p^*) + \lambda_s c_s - \lambda_p c_p &\equiv 0 \\
I_s - c_s f_s^* + c_s f_p^* - x_s^* &\equiv 0 \\
I_p - c_p f_p^* + c_p f_s^* - x_p^* &\equiv 0
\end{aligned}$$

where $g_i(\cdot)$ denotes the partial derivative of $g(x_s^*, f_s^*, x_p^*, f_p^*)$ with respect to its i^{th} argument.

Differentiating the first-order conditions with respect to I_p yields the following system of equations:

$$\begin{bmatrix}
g_{11} & g_{12} & g_{13} & g_{14} & -1 & 0 \\
g_{21} & g_{22} & g_{23} & g_{24} & -c_s & c_p \\
g_{31} & g_{32} & g_{33} & g_{34} & 0 & -1 \\
g_{41} & g_{42} & g_{43} & g_{44} & c_s & -c_p \\
-1 & -c_s & 0 & c_s & 0 & 0 \\
0 & c_p & -1 & -c_p & 0 & 0
\end{bmatrix}
\begin{bmatrix}
\frac{\partial x_s^*}{\partial I_p} \\
\frac{\partial f_s^*}{\partial I_p} \\
\frac{\partial x_p^*}{\partial I_p} \\
\frac{\partial f_p^*}{\partial I_p} \\
\frac{\partial \lambda_s^*}{\partial I_p} \\
\frac{\partial \lambda_p^*}{\partial I_p}
\end{bmatrix}
=
\begin{bmatrix}
0 \\
0 \\
0 \\
0 \\
0 \\
-1
\end{bmatrix}$$

Solving for $\frac{\partial f_p^*}{\partial I_p}$ by Cramer's rule yields

$$\frac{\partial f_p^*}{\partial I_p} = \frac{\begin{vmatrix} g_{11} & g_{12} & g_{13} & 0 & -1 & 0 \\ g_{21} & g_{22} & g_{23} & 0 & -c_s & c_p \\ g_{31} & g_{32} & g_{33} & 0 & 0 & -1 \\ g_{41} & g_{42} & g_{43} & 0 & c_s & -c_p \\ -1 & -c_s & 0 & 0 & 0 & 0 \\ 0 & c_p & -1 & -1 & 0 & 0 \end{vmatrix}}{\mathbf{H}} = -\frac{\mathbf{H}_{64}}{\mathbf{H}}$$

where \mathbf{H} denotes the determinant of the coefficient matrix, and \mathbf{H}_{ij} denotes the determinant of the coefficient matrix, evaluated after deleting the i^{th} row and j^{th} column. \mathbf{H} is strictly positive by the strict concavity of g ; it remains to be shown that $\mathbf{H}_{64} < 0$.

Noting that $g(x_{ps}, f_s, x_{sp}, f_p) = r(x_{ps}, f_s) \cdot r(x_{sp}, f_p)$, the cross-partials of $g(\cdot)$ can be expressed as functions of the first and second derivatives of $r(\cdot)$: $g_{11} = r_{11}r_p$, $g_{12} = g_{21} = r_{12}r_p$, $g_{13} = r_{13}r_3$, etc., where $r_\theta := \{r(x_{\theta\theta}, f_\theta) : \theta \in \{p, s\}\}$. Furthermore, because $r_{12} = r_{34} = 0$ by the separability of $r(\cdot)$, \mathbf{H}_{64} can be expressed as the sum of the following ten terms:

- 1) $-c_p c_s r_1 r_2 r_3^2$
- 2) $c_p r_2^2 r_3^2$
- 3) $-c_s^2 r_1^2 r_3 r_4$
- 4) $2c_s r_1 r_2 r_3 r_4$
- 5) $-r_2^2 r_3 r_4$
- 6) $c_p c_s r_1 r_4 r_{33} r_s$
- 7) $-c_p r_2 r_4 r_{33} r_s$
- 8) $c_s^2 r_2 r_3 r_{11} r_p$
- 9) $c_s r_1 r_3 r_{22} r_p$

$$10) -c_p r_{22} r_{33} r_p r_s$$

Recalling that $r_2 = -r_4 \frac{r_s}{r_p}$ by the first-order conditions, the first five terms can be combined into the

following expression: $r_3 r_4 (r_2 - c_s r_1) \left(c_s r_1 - r_2 - c_p r_3 \frac{r_s}{r_p} \right)$, which is identically equal to zero by the first-

order conditions. Noting that $c_s, c_p, r_1, r_2, r_3, r_s, r_p > 0$ and $r_{11}, r_{22}, r_{33}, r_4 < 0$, 6) is the only remaining

positive term. But terms 6) and 7) can be rearranged into $c_p r_4 r_{33} r_s (c_s r_1 - r_2)$, where the expression inside

the parenthesis is negative by the first-order conditions, which require that $c_s r_1 - r_2 = -c_p r_3 \frac{r_s}{r_p}$. All

remaining terms are negative, so $\mathbf{H}_{64} < 0$.

Proof that $\frac{\partial(f_p^* - f_s^*)}{\partial I_s} < 0$: As above, differentiation of the first-order conditions with respect to I_s

yields the following expression for $\frac{\partial(f_p^* - f_s^*)}{\partial I_s}$:

$$\frac{\partial(f_p^* - f_s^*)}{\partial I_s} = \frac{\partial f_p^*}{\partial I_s} - \frac{\partial f_s^*}{\partial I_s} = \frac{\mathbf{H}_{52} - \mathbf{H}_{54}}{\mathbf{H}}$$

so establishing that $\mathbf{H}_{52} - \mathbf{H}_{54} < 0$ will establish that $\frac{\partial(f_p^* - f_s^*)}{\partial I_s} < 0$. $\mathbf{H}_{52} - \mathbf{H}_{54}$ can be expressed as the sum of

the following nine terms:

$$1) c_p r_1 r_2 r_3 r_4$$

$$2) c c_s r_1^2 r_4^2$$

$$3) -r_1 r_2 r_4^2$$

$$4) -2c_s r_2 r_4 r_{11} r_p$$

$$5) c_p r_1 r_3 r_{22} r_p$$

$$6) -r_1 r_4 r_{22} r_p$$

$$7) -c_s r_{11} r_{22} r_p^2$$

$$8) c_p r_{13} r_{44} r_s$$

$$9) -c_s r_{11} r_{44} r_p r_s$$

The first three terms can be combined into the following expression: $r_1 r_4^2 \left(c_s r_1 - r_2 - c_p r_3 \frac{r_s}{r_p} \right)$, which is

identically equal to zero by the first-order conditions. All remaining terms are negative, so $\mathbf{H}_{52}\text{-}\mathbf{H}_{54}<0$.

9. References

- Ainslie, George “Derivation of ‘Rational’ Economic Behavior from Hyperbolic Discount Curves,” *American Economic Review*, 81(2) 334-340, May 1991.
- Allen, Judith M., Raymond W. Lam, Ronald A. Remick, and Adele D. Sadovnick, “Depressive Symptoms and Family History in Seasonal and Nonseasonal Mood Disorders,” *American Journal of Psychiatry*, 150(3), 443-448, March 1993.
- Allison, David B., Kevin R. Fontaine, JoAnn E. Manson, June Stevens, Theodore B. VanItallie, “Annual Deaths Attributable to Obesity in the United States,” *Journal of the American Medical Association*, 282, 1530-1538, 1999.
- Baird, J., C. Osmond, A. MacGregor, H. Snieder, C.N. Hales, and D.I.W. Phillips, “Testing the Fetal Origins Hypothesis in Twins: the Birmingham Twin Study,” *Diabetologia*, 44, 33-39, 2001.
- Barker, D.J.P., *et al*, “Growth *in utero*, blood pressure in childhood and adult life, and mortality from cardiovascular disease,” *British Medical Journal*, 298, 654-657, 1989.
- Barker, D.J.P., “Preface,” *British Medical Bulletin*, 60, 1-3, 2001.
- Barkow, Jerome H., “Beneath New Culture is Old Psychology,” Chapter 18 in *The Adapted Mind: Evolutionary Psychology and the Generation of Culture* by Jerome H. Barkow, Leda Cosmides, and John Tooby, eds., Oxford University Press, 1992.
- Barsh, Gregory S., I. Sadaf Farooqi, and Stephen O’Rahilly, “Genetics of Body Weight Regulation,” *Nature*, 404(6778), 635-643, April 6, 2000.
- Basiotis, P. Peter, and Mark Lino, “Food Insufficiency and Prevalence of Overweight Among Adult Women,” *USDA Center for Nutrition Policy and Promotion: Nutrition Insights*, 26, July 2002.
- Becker, Gary S., and Kevin M. Murphy, “A Theory of Rational Addiction,” *Journal of Political Economy*, 96(4), 675-700, 1988.
- Bergstrom, Ted, “Economics in a Family Way,” *Journal of Economic Literature*, 34(4), 1903-1934, December 1996.

- Bergstrom, Ted, "Storage for Good Times and Bad: Of Rats and Men," University of California, Santa Barbara Working Paper, December 1997.
- Bernard, A., G. Zwingelstein, R.R. Meister, and T. Fabian-Wild, "Hyperinsulinemia induced by canine distemper virus infection of mice and its correlation with the appearance of obesity" *Comparative Biochemistry and Physiology Part A: Physiology*, 91B, 691-696, 1988.
- Bertram, Caroline E., and Mark A. Hanson, "Animal Models and Programming of the Metabolic Syndrome," *British Medical Bulletin*, 60, 103-121, 2001.
- Bouchard, Claude, Louis Pérusse, Treva Rice, and D.C. Rao, "The Genetics of Human Obesity," Ch. 10 in *Handbook of Obesity* by George A. Bray, Claude Bouchard, and W.P.T. James, eds., Marcel Dekker, 1998.
- Boyd, Robert, and Joan B. Silk, *How Humans Evolved*, 2nd edition, W.W. Norton, 2000.
- Bray, George A., "Effects of Obesity on Health and Happiness," Chapter 1 in *Handbook of Eating Disorders*, Basic Books, 1986.
- Bray, George A., and Louis A. Tartaglia, "Medicinal Strategies in the Treatment of Obesity," *Nature* 404, 672-677, April 6, 2000.
- Chicurel, Marina, "Whatever Happened to Leptin?" *Nature*, 404, 538-540, April 6, 2000.
- Chou, Shin-Yi, Michael Grossman, and Henry Saffer, "An Economic Analysis of Obesity: Results from the Behavioral Risk Factor Surveillance System," *NBER Working Paper 9247*, October 2002.
- Clark, Colin W., and Jan Ekman, "Dominant and subordinate fattening strategies: a dynamic game," *Oikos*, 72, 205-212, 1995.
- Coccaro, E.F., C.S. Bergeman, and G.E. McClearn, "Heritability of irritable impulsiveness: A study of twins reared together and apart," *Psychiatry Research*, 48(3), 229-242, 1993.
- Coleman, D.L., "Effects of Parabiosis of obese with diabetes and Normal Mice," *Diabetologia*, 9, 294-298, 1973.
- Coleman, D.L., and K.P. Hummel, "Effects of Parabiosis of Normal with Genetically Diabetic Mice," *American Journal of Physiology*, 217, 1298-1304, 1969.
- Considine, Robert V., Madhur K. Sinha, Mark L. Heiman, Aidas Kriauciunas, Thomas W. Stephens, Mark R. Nyce, Joanna P. Ohannesian, Cheryl C. Marco, Linda J. McKee, Thomas L. Bauer, and Jose F. Caro, "Serum Immunoreactive-Leptin Concentrations in Normal-Weight and Obese Humans," *New England Journal of Medicine*, 334(5), 292-295, February 1, 1996.
- Cosmides, Leda, and John Tooby, "Cognitive Adaptations for Social Exchange," Chapter 3 in *The Adapted Mind: Evolutionary Psychology and the Generation of Culture* by Jerome H. Barkow, Leda Cosmides, and John Tooby, eds., Oxford University Press, 1992.
- Cosmides, Leda, and John Tooby, *Evolutionary Psychology: A Primer*, manuscript, January 13, 1997.
- DeFalco, Jeff, Mark Tomishima, Hongyan Liu, Connie Zhao, XiaoLi Cai, Jamey D. Marth, Lynn Enquist, and Jeffrey M. Friedman, "Virus-Assisted Mapping of Neural Inputs to a Feeding Center in the Hypothalamus," *Science* 291(5513), 2608-2613, March 30, 2001.
- de Waal, Frans, *Chimpanzee Politics: Power and Sex among Apes*, Johns Hopkins University Press, 1998.
- Dhurandhar, N.V., B.A. Israel, J.M. Kolesar, G.F. Mayhew, M.E. Cook, and R.L. Atkinson, "Increased

- Adiposity in Animals Due to a Human Virus," *International Journal of Obesity*, 24, 989-996, 2000.
- Diamond, Jared M., "Diabetes running wild," *Nature*, 357, 362-363, June 4, 1992.
- Dietz, W.H., "Does Hunger Cause Obesity?" *Pediatrics*, 95, 766-767, 1995.
- Dietz, W.H. and S.L. Gortmaker, "Factors Within the Physical Environment Associated With Childhood Obesity," *American Journal of Clinical Nutrition*, 39, 619-624, 1984.
- Dobzhansky, Theodosius, "Teilhard de Chardin and the Orientation of Evolution: A Critical Essay," *Zygon*, 3(3), 242-258, 1968.
- Ebstein, R.P., O. Novick, R. Umansky, B. Priel, Y. Osher, D. Blaine, E.R. Bennett, L. Nemanov, M. Katz, and R.H. Belmaker, "Dopamine D4 Receptor (D4DR) Exon III Polymorphism Associated with the Human Personality Trait of Novelty Seeking," *Nature Genetics*, 12, 78-80, 1995.
- Ekman, J., and K. Lilliendahl, "Using priority to food access: fattening strategies in dominance-structured willow tit (*Parus montanus*) flocks," *Behavioral Ecology*, 4, 232-238, 1993.
- Fagan, Brian, *The Little Ice Age: How Climate Made History, 1300-1850*, Basic Books, 2000.
- Falkner, Nicole H., Dianne Neumark-Sztainer, Mary Story, Robert W. Jeffery, Trish Beuhring, and Michael D. Resnick, "Social, Educational, and Psychological Correlates of Weight Status in Adolescents," *Obesity Research*, 9, 32-42, 2001.
- Farooqi, I. Sadaf, Susan A. Jebb, Gill Langmack, Elizabeth Lawrence, Christopher H. Cheetham, Andrew M. Prentice, Ieuan A. Hughes, Mark A. McCamish, and Stephen O'Rahilly, "Effects of Recombinant Leptin Therapy in a Child with Congenital Leptin Deficiency," *New England Journal of Medicine*, 341(12), 879-884, September 16, 1999.
- Flegal, Katherine M., M.D. Carroll, Robert J. Kuczmarski, Clifford L. Johnson, "Overweight and Obesity in the United States: Prevalence and Trends, 1960-1994," *International Journal of Obesity*, 22, 39-47, 1998.
- Flinn, Mark V., and Barry G. England, "Childhood Stress and Family Environment," *Current Anthropology*, 36(5), 854-866, Dec. 1995.
- Frank, Robert H., *Passions Within Reason: The Strategic Role of the Emotions*, W.W. Norton, 1988.
- Friedman, Jeffrey M., "Obesity in the New Millennium," *Nature* 404, 632-634, April 6, 2000.
- Friedman, Jeffrey M., and Jeffrey L. Halaas, "Leptin and the Regulation of Body Weight in Mammals," *Nature*, 395, October 22, 1998.
- Friedman, Milton, "The Methodology of Positive Economics," Chapter 1 in *Essays in Positive Economics* by Milton Friedman, University of Chicago Press, 1953.
- Garn, Stanley M., Marquisa LaVelle, and Jeffery J. Pilkington, "Obesity and Living Together," *Marriage and Family Review*, 7, 33-47, 1984.
- Gazzaniga, Michael S., editor-in-chief, *The New Cognitive Neurosciences*, 2nd edition, MIT Press, 2000.
- Gazzaniga, Michael S., Richard B. Ivry, and George R. Mangun, *Cognitive Neuroscience: The Biology of the Mind*, W.W. Norton, 1998.
- Gigerenzer, Gerd, Peter M. Todd, and the ABC Research Group, *Simple Heuristics That Make Us Smart*, Oxford, 1999.
- Gillman, Matthew W., "Epidemiological Challenges in Studying the Fetal Origins of Adult Chronic

- Disease,” *International Journal of Epidemiology*, 31, 294-299, 2002.
- Gortmaker, Steven L., Aviva Must, James M. Perrin, Arthur M. Sobol, and William H. Dietz, “Social and Economic Consequences of Overweight in Adolescence and Young Adulthood,” *New England Journal of Medicine*, 329(14), 1008-1012, September 30, 1993.
- Gul, Faruk, and Wolfgang Pesendorfer, “Temptation and Self-Control,” *Econometrica*, 69(6), 1403-1435, November 2001.
- Hamermesh, Daniel S., and Jeff E. Biddle “Beauty and the Labor Market,” *American Economic Review*, 84(5), 1174-1194, 1994.
- Hansson, Ingemar, and Charles Stuart, “Malthusian Selection of Preferences,” *American Economic Review*, 80(3), 529-544, June 1990.
- Harpending, Henry C., Mark A. Batzer, Michael Gurven, Lynn B. Jorde, Alan A. Rogers, and Stephen T. Sherry, “Genetic Traces of Ancient Demography,” *Proceedings of the National Academy of Science (USA)*, 95, 1961-1967, February 1998.
- Heymsfield, Steven B., Andrew S. Greenberg, Ken Fujioka, Russell M. Dixon, Robert Kushner, Thomas Hunt, John A. Lubina, Janet Patane, Barbara Self, Pam Hunt, Mark McCamish, “Recombinant Leptin for Weight Loss in Obese and Lean Adults: A Randomized, Controlled, Dose-Escalation Trial,” *Journal of the American Medical Association*, 282(16), 1568-1575, October 27, 1999.
- Hill, A.J., and E.K. Silver, “Fat, friendless and unhealthy: 9-year old children’s perception of body shape stereotypes,” *International Journal of Obesity*, 19(6), 423-430, June 1995.
- Hirshleifer, Jack, “Economics from a Biological Viewpoint,” *Journal of Law and Economics*, 20, 1-52, 1977.
- Hirshleifer, Jack, “The Expanding Domain of Economics,” *American Economic Review*, 75(6), 53-68, 1985.
- Hirshleifer, Jack, “On the Emotions as Guarantors of Threats and Promises,” Ch. 14 in *The Latest on the Best: Essays in Evolution and Optimality*, John Dupré, ed., MIT Press, Cambridge, MA, 1987.
- Houston, Alasdair I., and John M. McNamara, *Models of Adaptive Behavior: An Approach Based on State*, Cambridge University Press, Cambridge, 1999.
- Hubert, H.B., “The importance of obesity in the development of coronary risk factors and disease: the epidemiological evidence,” *Annual Review of Public Health*, 7, 493-502, 1986.
- Kahneman, Daniel, “Objective Happiness,” Chapter 1 in *Well-Being: The Foundations of Hedonic Psychology*, by Daniel Kahneman, Ed Diener, and Norbert Schwarz, eds., Russell Sage Foundation, 1999.
- Kochan, Z., J. Karbowska, and J. Swierczynski, “Unusual increase of lipogenesis in rat white adipose tissue after multiple cycles of starvation-refeeding,” *Metabolism: Clinical and Experimental*, 46, 7-10, 1997.
- Konner, Melvin, *The Tangled Wing*, 2nd edition, Times Books, New York, 2002.
- Kopelman, Peter G., “Obesity as a Medical Problem,” *Nature*, 404(6778), 635-643, April 6, 2000.
- Laibson, David, “A Cue-Theory of Consumption,” *Quarterly Journal of Economics*, 116(1), 81-119, February 2001.
- Laibson, David, “Golden Eggs and Hyperbolic Discounting,” *Quarterly Journal of Economics*, 112(2),

443-477, May 1997.

- Larson, Randy J. and Barbara L. Fredrickson, "Measurement Issues in Emotion Research," Chapter 3 in *Well-Being: The Foundations of Hedonic Psychology*, by Daniel Kahneman, Ed Diener, and Norbert Schwarz, eds., Russell Sage Foundation, 1999.
- Lesch, Klaus-Peter, Dietmar Bengel, Armin Heils, Sue Z. Sabol, Benjamin D. Greenberg, Susanne Petri, Jonathan Benjamin, Clemens R. Muller, Dean H. Hamer, and Dennis L. Murphy, "Association of Anxiety-Related Traits with a Polymorphism in the Serotonin Transporter Gene Regulatory Region," *Science*, 274, 1527-1531, 1996.
- Levin, Laurence, "Are Assets Fungible? Testing the Behavioral Theory of Life-Cycle Savings," *Journal of Economic Behavior and Organization*, 36(1), 59-83, July 1998.
- Madden, Pamela A.F., Andrew C. Heath, Norman E. Rosenthal, and Nicholas G. Martin, "Seasonal Changes in Mood and Behavior: The Role of Genetic Factors," *Archives of General Psychiatry*, 53, 47-55, January 1996.
- Maddox, G.L., and V. Liederman, "Overweight as a social disability with medical implications," *Journal of Medical Education*, 44, 214-220, 1969.
- Malakoff, David, "The Rise of the Mouse, Biomedicine's Model Mammal," *Science*, 288, 248-253, April 14, 2000.
- Manuck, Stephen B., Janine D. Flory, Robert E. Ferrell, J. John Mann, and Matthew F. Muldoon, "A regulatory polymorphism of the monoamine oxidase-A gene may be associated with variability in aggression, impulsivity, and central nervous system serotonergic responsivity," *Psychiatry Research*, 95(1), 9-23, 2000.
- Mas-Collel, Andreu, Michael D. Whinston, and Jerry R. Green, *Microeconomic Theory*, Oxford University Press, 1995.
- Maynard Smith, J., *Evolutionary Genetics*, Oxford University Press, 1998.
- McGuire, M.T., M.J. Raleigh, and G.L. Brammer, "Adaptation, Selection, and Benefit-Cost Balances: Implications of Behavioral-Physiological Studies of Social Dominance in Male Vervet Monkeys," *Ethology and Sociobiology*, 5, 269-277, 1984.
- McMinn, J.E., Baskin, D.G., and Schwartz, M.W., "Neuroendocrine Mechanisms Regulating Food Intake and Body Weight," *Obesity Reviews*, 1, 37-46, 2000.
- Mercer, J.G., C.L. Adam, and P.J. Morgan, "Towards an understanding of physiological body mass regulation: Seasonal animal models," *Nutritional Neuroscience*, 3(5), 307-320, 2000.
- Miller, Richard D., Jr., and H.E. Frech, III, "The Productivity of Health Care and Pharmaceuticals: Quality of Life, Cause of Death and the Role of Obesity," *Working Paper in Economics #12-02*, University of California, Santa Barbara: Department of Economics, July 24, 2002.
- Montague, Carl T., I. Sadaf Farooqi, Jonathan P. Whitehead, Maria A. Soos, Harald Rau, Nicholas J. Wareham, Ciaran P. Sewter, Janet E. Digby, Shehla N. Mohammed, Jane A. Hurst, Christopher H. Cheetham, Alison R. Earley, Anthony H. Barnett, Johannes B. Prins, and Stephen O'Rahilly, "Congenital leptin deficiency is associated with severe early-onset obesity in humans," *Nature*, 387, 903-908, June 26, 1997.
- Must, Aviva, Jennifer Spadano, Eugenie H. Coakley, Alison E. Field, Graham Colditz, William H. Dietz, "The Disease Burden Associated With Overweight and Obesity," *Journal of the American Medical*

- Association*, 282, 1523-1529, 1999.
- Nagashima, K., J.B. Zabriskie, and M.J. Lyons, "Virus induced obesity in mice: Association with a hypothalamic lesion," *Journal of Neuropathology and Experimental Neurology*, 51, 101-109, 1992.
- Neel, James V., "Diabetes Mellitus: A 'Thrifty' Genotype Rendered Detrimental by 'Progress'?", *American Journal of Human Genetics*, 14, 353-362, 1962.
- Neel, James V., "The 'Thrifty Genotype' in 1998," *Nutrition Reviews*, 57(5) S2-S9, May 1999.
- Neubauer, Peter B., and Alexander Neubauer, *Nature's Thumbprint: The New Genetics of Personality*, Columbia University Press, 1996.
- Nord, Mark, Margaret Andrews, and Steven Carlson, "Measuring Food Security in the United States: Household Food Security in the United States, 2001," *USDA Food Assistance and Nutrition Research Report Number 29*, October 2002.
- O'Brien, Stephen J., Marilyn Menotti-Raymond, William J. Murphy, William G. Nash, Johannes Wienberg, Roscoe Stanyon, Neal G. Copeland, Nancy A. Jenkins, James E. Womack, and Jennifer A. Marshall Graves, "The Promise of Comparative Genomics in Mammals," *Science*, 286, 458-481, October 15, 1999.
- O'Donoghue, Ted, and Matthew Rabin, "Doing it Now or Later," *American Economic Review*, 89(1), 103-124, March 1999.
- Ogilvie, A.D., S. Battersby, V.J. Bubb, G. Fink, A.J. Harmar, G.M. Goodwin, C.A.D. Smith, "Polymorphism in serotonin transporter gene associated with susceptibility to major depression," *Lancet*, 347(9003), 731-733, March 16, 1996.
- Olson, Christine M., "Nutrition and Health Outcomes Associated with Food Insecurity and Hunger," *Journal of Nutrition*, 129, 521S-524S, 1999.
- Pert, Candace B., *Molecules of Emotion*, Scribner, 1997.
- Philipson, Tomas J., and Richard A. Posner, "The Long Run Growth in Obesity as a Function of Technological Change," John M. Olin Law & Economics Working Paper No. 78, University of Chicago, May 1999.
- Pinker, Steven, *How the Mind Works*, W.W. Norton, 1997.
- Rachlin, H., and L. Green, "Commitment, Choice, and Self-Control," *Journal of the Experimental Analysis of Behavior*, 17, 15-22, 1972.
- Rankinen, Tuomo, Louis Pérusse, S. John Weisnagel, Eric E. Snyder, Yvon C. Chagnon, and Claude Bouchard, "The Human Obesity Genome Map: The 2001 Update," *Obesity Research*, 10(3), 196-243, March 2002.
- Register, Charles A., and David R. Williams, "Wage Effects of Obesity among Young Workers," *Social Science Quarterly*, 71(1), 130-141, 1990.
- Robson, Arthur J., "A Biological Basis for Expected and Non-Expected Utility," *Journal of Economic Theory*, 68(2), 397-424, February 1996.
- Robson, Arthur J., "The Biological Basis of Economic Behavior," *Journal of Economic Literature*, 39(1), 11-33, March 2001.
- Rogers, Alan R., "Evolution of Time Preference by Natural Selection," *American Economic Review*, 84(3), 460-481, June 1994.

- Romer, Paul M., "Thinking and Feeling," *American Economic Review*, 90(2), 439-443, May, 2000.
- Rosmond, Roland, Claude Bouchard, and Per Björnthorp, "5-HT_{2A} Receptor Gene Promoter Polymorphism in Relation to Abdominal Obesity and Cortisol," *Obesity Research*, 10(7), 585-589, July 2002.
- Samuelson, Larry, and Jeroen Swinkels, "Information and the Evolution of the Utility Function," University of Wisconsin-Madison working paper, April 11, 2001.
- Sapolsky, Robert M., "Hormonal Correlates of Personality in Social Contexts: From Non-Human to Human Primates," Chapter 2 in *Hormones, Health, and Behavior*, by C. Panter-Brick and C.M. Worthman, eds., Cambridge University Press, 1999a.
- Sapolsky, Robert M., "The Physiology and Pathophysiology of Unhappiness," Chapter 23 in *Well-Being: The Foundations of Hedonic Psychology*, by Daniel Kahneman, Ed Diener, and Norbert Schwarz, eds., Russell Sage Foundation, 1999b.
- Savage, Leonard J., *The Foundations of Statistics*, John Wiley & Sons, 1954.
- Schwartz, Michael W., Stephen C. Woods, Daniel Porte Jr., Randy J. Seeley, and Denis G. Baskin, "Central Nervous System Control of Food Intake," *Nature*, 404, 661-671, April 6, 2000.
- Seidell, Jacob C., and Áila M. Rissanen, "Time Trends in Worldwide Prevalence of Obesity," Ch. 4 in *Handbook of Obesity* by George A. Bray, Claude Bouchard, and W.P.T. James, eds., Marcel Dekker, 1998.
- Shefrin, Hersh M., and Richard H. Thaler, "The Behavioral Life-Cycle Hypothesis," *Economic Inquiry*, 26(4), 609-643, October 1988.
- Shively, Carol A., and Jeanne M. Wallace, "Social Status, Social Stress, and Fat Distribution in Primates," Chapter 15 in *International Textbook of Obesity*, Per Bjorntorp, ed., John Wiley & Sons, 2001.
- Smith, Adam, *The Wealth of Nations*, 1776.
- Sozou, P.D., "On hyperbolic discounting and uncertain hazard rates," *Proceedings of the Royal Society of London B: Biological Sciences*, 265(1409), 2015-2020, October 22, 1998.
- Staffieri, J. Robert, "A Study of Social Stereotype of Body Image in Children," *Journal of Personality and Social Psychology*, 7(1), 101-104, 1967.
- Strotz, Robert H., "Myopia and Inconsistency in Dynamic Utility Maximization," *Review of Economic Studies*, 23, 165-180, 1956.
- Stryer, Lubert, *Biochemistry*, 2nd edition, W.H. Freeman, 1981.
- Stunkard, Albert J., "Socioeconomic Status and Obesity," pp. 174-187 in *The Origins and Consequences of Obesity*, edited by Derek J. Chadwick and Gail Cardew, Wiley, 1996.
- Tartaglia L.A., M. Dembski, X. Weng, *et al.*, "Identification and expression cloning of a leptin receptor OB-R," *Cell*, 83, 1263-1271, 1995.
- Thaler, Richard H., and Hersh M. Shefrin, "An Economic Theory of Self-Control," *Journal of Political Economy*, 89(2), 392-406, April 1981.
- Townsend, Marilyn S., Janet Peerson, Bradley Love, Cheryl Achterberg, and Suzanne P. Murphy, "Food Insecurity is Positively Related to Overweight in Women," *Journal of Nutrition*, 131, 1738-1745, 2001.

- Troiano, Richard P., and Katherine M. Flegal, "Overweight Children and Adolescents: Description, Epidemiology, and Demographics," *Pediatrics*, 101(3), 497-504, March 1998.
- Van Staveren, Wija A., Paul Deurenberg, Jan Burema, Lisette C.P.G.M. De Groot, and Joseph G.A.J. Hautvast, "Seasonal Variation in Food Intake, Pattern of Physical Activity and Change in Body Weight in a Group of Young Adult Dutch Women Consuming Self-Selected Diets," *International Journal of Obesity*, 10(2), 133-145, 1986.
- Vander Wall, Stephen B., *Food Hoarding in Animals*, 1990.
- Von Mises, Ludwig, *Human Action: A Treatise on Economics*, 3rd edition, Yale University Press, 1966.
- Weigle, D.S., "Appetite and the Regulation of Body Composition," *FASEB (Federation of American Societies for Experimental Biology) Journal*, 8, 302-310 March 1, 1994.
- Weindruch, Richard, Roy L. Walford, Suzanne Fligiel, and Donald Guthrie, "The Retardation of Aging in Mice by Dietary Restriction: Longevity, Cancer, Immunity and Lifetime Energy Intake," *Journal of Nutrition*, 116(4), 641-654, April 1986.
- Wessels, Norman K., and Janet L. Hopson, *Biology*, Random House, 1988.
- Whitaker, Robert C., and William H. Dietz, "Role of the Prenatal Environment in the Development of Obesity," *Journal of Pediatrics*, 132(5), 768-776.
- Wolf, Anne M., and Graham A. Colditz, "Current estimates of economic costs of obesity in the United States," *Obesity Research*, 6 (2) 97-106, 1998.
- Woods, Stephen C., Randy J. Seeley, Daniel Porte Jr., and Michael W. Schwartz, "Signals that Regulate Food Intake and Energy Homeostasis," *Science*, 280, 1378-1383, May 29, 1998.
- World Health Organization, *Obesity: Preventing and Managing the Global Epidemic, Report of a WHO Consultation on Obesity, Geneva, 3-5 June, 1997*, 1998.
- Wright, Charlotte M., Louise Parker, Douglas Lamont, and Alan W. Craft, "Implications of Childhood Obesity for Adult Health: Findings from the Thousand Families Cohort Study," *British Journal of Medicine*, 323, 1280-1284, 1 December 2001.
- Wright, Robert, *The Moral Animal: Evolutionary Psychology and Everyday Life*, Pantheon, 1994.
- Yanovski, Jack A., Susan Z. Yanovski, Kara N. Sovik, Tuc T. Nguyen, Patrick M. O'Neil, and Nancy G. Sebring, "A Prospective Study of Holiday Weight Gain," *New England Journal of Medicine*, 342(12), 861-867, March 23, 2000.
- Yu, Chang-En, Junko Oshima, Ying-Hui Fu, Ellen M. Wijsman, Fuki Hisama, Reid Alisch, Shellie Matthews, Jun Nakura, Tetsuro Miki, Samir Ouasis, George M. Martin, John Mulligan, Gerard D. Schellenberg, "Positional Cloning of the Werner's Syndrome Gene," *Science*, 272, 258-262, 1996.
- Zhang, Y., R. Proenca, M. Maffie, M. Barone, L. Leopold, and J. Friedman, "Positional Cloning of the Mouse *obese* Gene and its Human Homologue," *Nature*, 372, 425-432, 1994.