OBESITY AND NATURE'S THUMBPRINT

How Modern Waistlines Can Inform Economic Theory

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Abstract

The modern prevalence and negative consequences of obesity suggest that many people have a tendency to eat more than is optimal. This paper 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.

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1. Introduction: Obesity as a Behavioral Puzzle

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 do miscalculate—does it tend to be in the direction of *over*eating? 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.

The main contributions of this paper are twofold: First, I interpret recent findings in the natural sciences and their relevance to economic decision theory; and second, I offer a synthesis of theory and evidence that suggests conditions under which people will fail to act in their own best interest. The broader implications for the modern theory and practice of welfare economics are also considered.

1.1 The Personal Costs of Obesity

The consequences of being overweight can be severe. Children as young as 6 years of

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.

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 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 reducing caloric intake to just *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 care[3]; 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, and it is easy to apply neoclassical principles to the problem. For example, the aforementioned attributes of the Western lifestyle might be interpreted as the result of lower implicit *prices* of fattening foods and easy-chair recreation (Figure 1).[4] Pending empirical confirmation of the purported price

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[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. It is thought that these levels are high enough to be a good indicator of adiposity for the general population.

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

[4] The private cost of achieving caloric surplus should properly include much more than just

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 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 examples in which people claim to have difficulty with self-control, and maintaining a dietary regimen is a leading example. But in considering the general problem faced by an individual when allocating consumption and savings over time, a

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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 Philipson and Posner (1999) for an economic analysis of obesity along these lines.

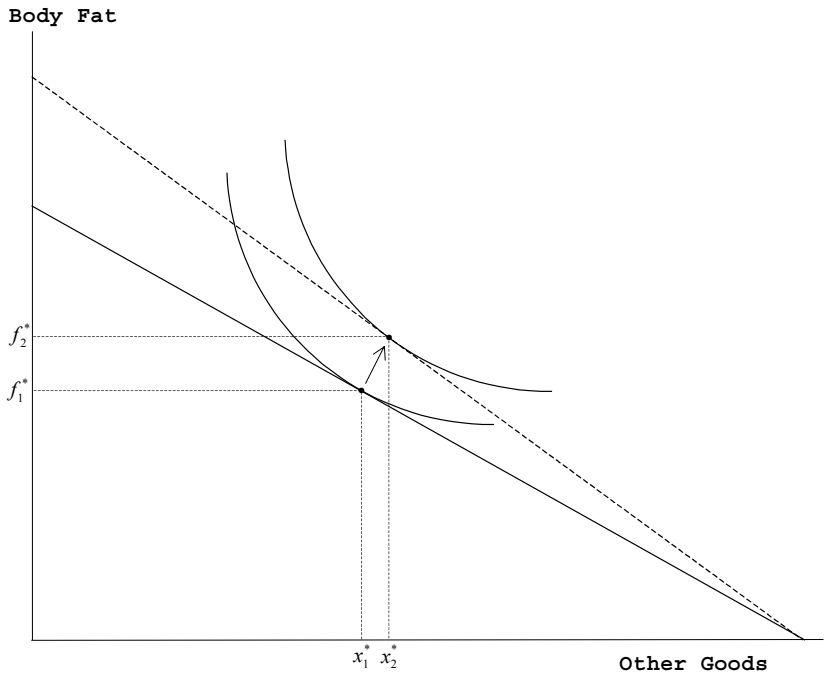

Figure 1: Body Fat and Neoclassical Choice

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.

problem with self-control implies that preferences are *dynamically inconsistent*: that is, a consumption plan made at time *t* may be altered or constrained at time *t+1* because the individual lacks the "self-control" necessary to carry out the plan. So I might make a plan to place 10% of my paycheck in a retirement account, starting next year. But if my preferences are dynamically inconsistent, I might—*even in the absence of new information*—change my plan come New Year's Day, and decide to 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).[5]

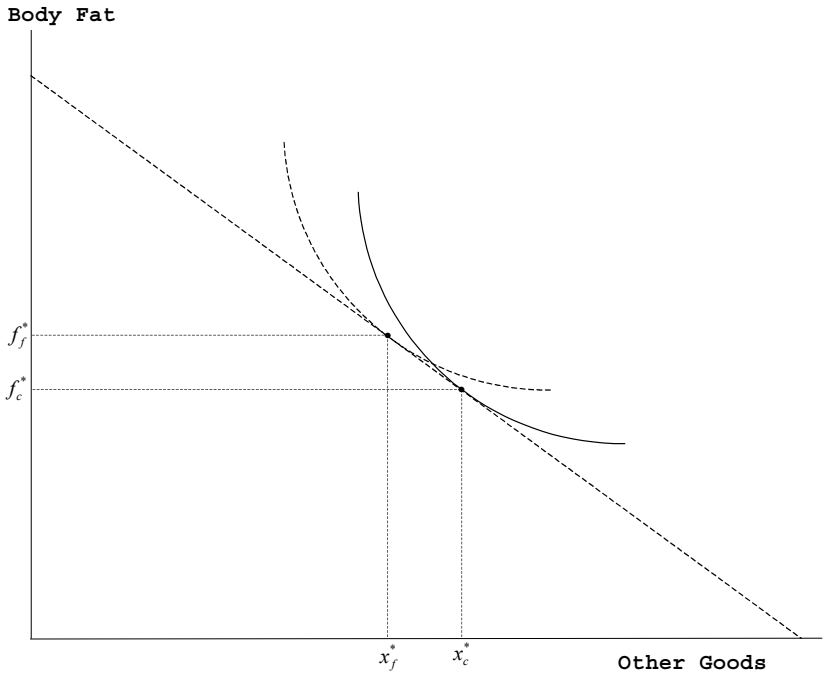

Figure 2: Diet and the Economic Theory of Self-Control

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.

There are a number of ways to formalize the self-control problem. Many build on Samuelson's (1937) discounted utility model by positing a discount rate on future utilities that changes as the date of consumption approaches. In a foundational paper, Robert Strotz (1956) hypothesized that consumers might discount future consumption according to both the

[5] 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.

calendar date(s) at which consumption takes place (as in the constant discounting model) and the *time distance* of the future date from the present. 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.[6] This problem might, of course, be anticipated by the individual making the original plan, and Strotz suggests 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. Subsequent studies have expanded on the self-control model: Laibson (1997) and Barro (1999) posit a quasi-hyperbolic functional form[7] for the consumer's discount function over future utilities. Becker and Mulligan (1997) model a consumer's efforts to "reduce the discount on future utilities" by incorporating this effort into the agent's utility function, so that the agent may—for the price of effort—influence his future behavior. Thaler and Shefrin (1981) are more explicit about the dichotomy between planned and actual behavior, positing a two-part decision process in which a "planner" attempts to influence the behavior of a "doer."

Most testable hypotheses that stem from these models of dynamic inconsistency 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.[8] Gul and Pesendorfer (forthcoming) 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.

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[6] Rogers (1994, p. 473) correctly points out that a varying rate of discount on future utilities does not imply dynamic inconsistency if the rate varies with *age* rather than with delay.

[7] The quasi-hyperbolic discount function implies a higher rate of discount in the first period, and a constant, lower rate in subsequent periods. The name derives from the generalized hyperbolic discount function, in which the discount factor is approximately proportional to the time distance between choice and consumption (Ainslie, 1991).

[8] 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).

2.2 Empirical Evidence

> *"…a majority of adults report that they would rather have $50 immediately than $100 in 2 years, but almost no one prefers $50 in 4 years over $100 in 6 years, even though this is the same choice seen at 4 years' greater distance…" (Ainslie, 1991)*

In the nearly five decades since it was proposed, evidence has accumulated that seems to lend credence to the Strotz model of self-control. By varying the size and timing of small rewards in the laboratory, both human and animal[9] subjects have demonstrated a systematic tendency to employ a *declining* rate of discount (i.e., they are quite impatient initially, but become more patient as the payoffs become further removed from the time at which the choice is made) (Green *et al*, 1981; Thaler, 1981; Benzion *et al*, 1989; Cropper *et al*, 1992, 1994; Horowitz, 1992). Expenditures on pre-commitment devices is another often-cited source of evidence; examples include weight loss programs, smoking cessation programs, Alcoholics Anonymous, "check cards" (credit cards requiring full payment at the time of purchase), inflexible home mortgage payment schedules, and voluntary payroll deduction programs (Rachlin and Green, 1972, Schelling, 1978, Thaler and Shefrin, 1981, Becker and Mulligan, 1997).

2.3 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 forgo much of the normative appeal of more conventional

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[9] Though some might question the applicability of animal behavior to the study of economics, there are in fact many instances in which animal behavior has been shown to be consistent with economic theory, and a few cases in which economic explanations seem to be quite superior to leading behavioral models from psychology and biology (Kagel *et al*, 1995). In addition to the obvious advantages of experimentation with animal rather than human subjects (lower wages and looser ethical standards, to name just two) Kagel *et al* suggest that "…a theory that works well across species has a greater likelihood of being valid than one that works well with only one, or a limited set of, species." (1995, p.4).

Perhaps the greatest benefit of using animals to test theories of economic behavior is that, while confirming many of the central predictions of economic choice theory, animal experiments often identify situations in which the more precise implications of economic theory are *not* consistent with the data, thus pointing to areas of economic theory which might deserve closer examination. An example of such an instance can be found in the study

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,[10] 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 Sections 2.1 and 2.2 above makes clear, and it has enjoyed considerable success. But by adopting a strictly positivist approach—i.e.,

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of self-control.

[10] Gul and Pesendorfer (forthcoming) introduce just such an axiom, which they refer to as *set*

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 economics.

Recall, if you will, the material question with which this investigation began: How much to eat. How people solve this problem. Why they often err on the side of excess. Because eating is an activity 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. Sections 4 and 5 consider the relevance of the rodent metaphor to human behavior.

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 ecologists[11], 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).

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betweenness.

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

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 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.[12] 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 relative abundance of food $\theta \in \{p, s\}$, where p denotes relative plenty (or "feast") and s denotes relative scarcity (or "famine"). Relative abundance is determined by a

[12] 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.

stationary two-state Markov process with transition matrix $\mathbf{P} = \begin{matrix} \\ p \\ s \end{matrix} \begin{matrix} \begin{matrix} p & s \end{matrix} \\ \left\| \begin{matrix} P_{pp} & P_{ps} \\ P_{sp} & P_{ss} \end{matrix} \right\| \end{matrix}$, $\sum_{\theta \in \{p,s\}} P_{\theta'\theta} = 1$,

where $P_{\theta'\theta}$ denotes the probability of state θ, given that the state was θ' in the previous period.

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 relative abundance in the current and the previous periods $(x = x_{\theta'\theta})$, while the choice of reserves f is a function only of relative abundance in the current period ($f = f_\theta$).[13] 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 and twice differentiable) function $r(f_{\theta'}, \theta; x_{\theta'\theta}, f_\theta)$ that measures the expected number of offspring (including the parent, if the parent survives to the next period) produced during the current period, given the state variables $f_{\theta'}$ and θ and 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*.

Under these conditions, and assuming P_{ps},P_{sp}>0, it can be shown that the genotype that will come to dominate the population in the long run will employ the strategy that maximizes the following expression:

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

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

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 state being *p* or *s*, respectively in the long run (Taylor and Karlin, 1998, p. 137), so this expression is an example of the more general result that in the presence of environmental fluctuations (i.e., risks that affect the entire population) evolution will tend to select agents that maximize the geometric mean fitness (see, e.g., Houston and McNamara, 1999; Bergstrom, 1997; or Robson, 1996).[14]

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 $r(\cdot)$). 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 ($\frac{\partial g}{\partial f_\theta} > 0$) 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 decreasing in f_θ ($\frac{\partial g}{\partial f_\theta} < 0$) for $f_\theta > f^*$ (presumably because excessive body fat generates the

[14] 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 $r_1(f_{\theta'}, s; x_{\theta's}, f_s) = 0$) and a fitness of *r*=2.1 during feasts $\left(r_1(f_{\theta'}, p; x_{\theta'p}, f_p) = 2.1\right)$; Strategy 2 yields a fitness of *r*=1 each season $\left(r_2(f_{\theta'}, \theta; x_{\theta'\theta}, f_\theta) = 1\right)$. Note that Strategy 1 maximizes the arithmetic mean $\left(a_1 = (0 + 2.1)/2 = 1.05 > 1 = a_2\right)$ number of offspring while Strategy 2 maximizes the geometric mean $\left(g_1 = \sqrt{(0)(2.1)} = 0 < \sqrt{(1)(1)} = 1 = g_2\right)$. 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. 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 number of offspring will come to dominate the population in the long run.

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

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

subject to

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

where

$$g\left(x_{ps}, f_s, x_{sp}, f_p\right) = r\left(f_p, s; x_{ps}, f_s\right) \cdot r\left(f_s, p; x_{sp}, f_p\right)$$

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

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

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 season is endogenously determined by the choice of f in the previous season, and (assuming strict concavity of $g(\cdot)$ and $\frac{g_3}{g_1} < \frac{c_s}{c_p} \leq 1$, where g_i denotes the partial derivative of $g\left(x_{ps}, f_s, x_{sp}, f_p\right)$ with respect to its i^{th} argument)[15] Nature finds it optimal to choose

[15] If the variables x and f are interpreted strictly as measures of food energy (so that c_s and c_p are rates of physiological conversion) then the requirement that $\frac{c_s}{c_p} \leq 1$ is akin to the Second

$f_p > f^*$ and $f_s < f^*$. Intuitively—as can be seen from the first-order conditions—an excess (dearth) of body fat is generated during feasts (famines), up to the point at which the marginal fitness *loss* (*gain*) from an additional unit of f_p (f_s) together with the marginal fitness *gain* from the additional units of x_{ps} (x_{sp}) that can be "purchased" during famines (feasts) just offset the marginal fitness *loss* from the corresponding decrease in x_{sp} (x_{ps}).

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).

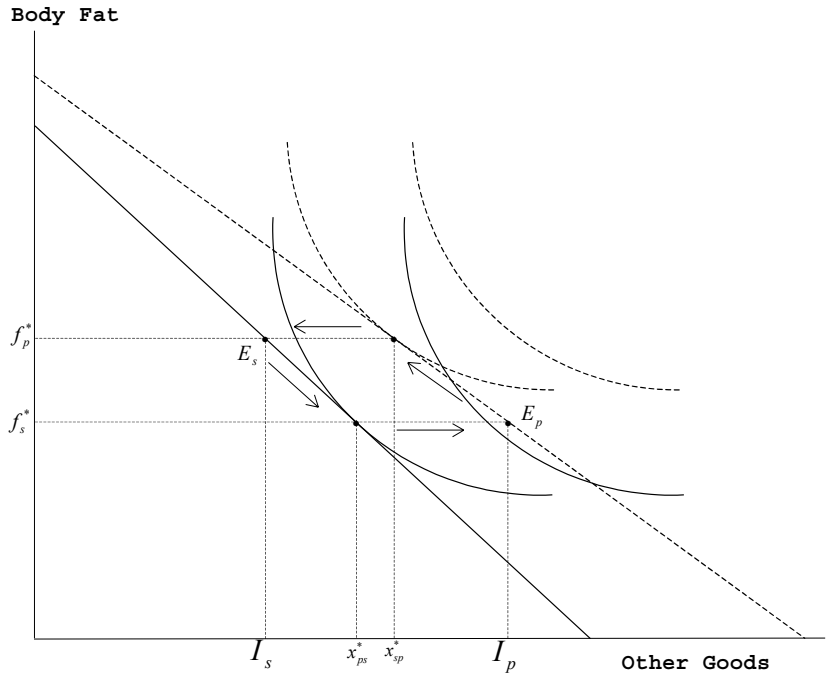

Figure 3: Evolutionary Equilibrium with Seasonal Scarcity

In evolutionary equilibrium the food supply is variable, shown here by a limited energy budget in the scarcity 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).

Law of Thermodynamics, which requires that the entropy of a closed system never decreases.

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.

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 phenotypic polymorphism in a trait 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.[16] 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 support for such strategies

[16] 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

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.[17] 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.[18] More generally, the polymorphism will be stable whenever the arithmetic mean fitness of the recessive is less than that of the dominant, while geometric mean fitness of the recessive is greater than that of the dominant (Maynard Smith, 1998).[19]

3.3.2 Social Dominance

The third explanation for a phenotypic polymorphism—assymmetric information or

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randomizing gene choose to hoard, and eventual extinction would be certain.

[17] 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.

[18] 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.

[19] As shown in Footnote 14, these conditions are satisfied for the two strategies considered

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, both in times of plenty (where less food favors a positive relationship between rank and body fat) and in times of scarcity (where less food favors a negative relationship between rank and body fat).

The social dominance phenomenon could be incorporated into the framework of the stationary seasonality model (Section 3.2) by positing two additional state variables, one for social status and one for absolute abundance. In accordance with the narrative of the previous paragraph, choice sets might vary with these states as well as with season, for example by affecting the periodic income I_θ. For example, where food is abundant, incomes might be $I_p \approx I_s >> 0$ for the dominant and $I_p >> I_s \approx 0$ for the subordinate; while where food is scarce incomes might be $I_p >> I_s \approx 0$ for the dominant and $I_p \approx I_s \approx 0$ for the subordinate. Behavior in evolutionary equilibrium might then be conditioned on all three determinants (relative abundance, rank, and absolute abundance) of expected future food supplies.

This conjecture is consistent with the comparative statics of the stationary scarcity model: If $g(\cdot)$ is strictly concave, $\frac{g_3}{g_1} < \frac{c_s}{c_p} \leq 1$, and $r(f_{\theta'}, \theta; x_{\theta'\theta}, f_\theta)$ is separable in $x_{\theta'\theta}$ and f_θ (so that $\frac{\partial^2 r(f_{\theta'}, \theta; x_{\theta'\theta}, f_\theta)}{\partial x_{\theta'\theta} \partial f_\theta} = 0$, $\theta, \theta' \in \{p, s\}$, $\theta \neq \theta'$), then body fat in times of plenty is

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here.

increasing in I_p $\left(\frac{\partial f_p^*}{\partial I_p} > 0\right)$, and the net seasonal change in body fat is decreasing in I_s $\left(\frac{\partial\left(f_p^* - f_s^*\right)}{\partial I_s} < 0\right)$.[20]

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 model 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. This causes no problem for consumer theory, as behavior is assumed to be flexible with respect to prices and income, and an analogous assumption is made implicitly in the specification of Section 3.2. But supposing that flexibility with respect to transformation rates and income is optimal, I have implicitly assumed that the *costs* of flexibility are not important. This is certainly not always an appropriate assumption in models of evolutionary equilibrium—incorporating flexibility into an agent's behavioral algorithm is likely to require time for information processing, not to mention the construction and maintenance of the associated cognitive and perceptual machinery. Clearly, however, there are many examples to be found in nature in which animal behavior is responsive to information inputs, and just as responsiveness to price environments is commonly assumed to be optimal in economic models of behavior, so too will responsiveness to seasonal variation here.

If there is also variability over time with respect to the frequency of food shortages, we might expect Nature to design some flexibility into the behavioral algorithms embodied in the first-order conditions. In other words, behavior might be sensitive to new information regarding the risk of food shortages. This will require a weakening of the assumption of

[20] Proofs are provided in the Appendix.

strong forward convergence, as the optimal strategy will become a function of age and experience.

Specifically, consider now the special case in which the transition matrix for relative abundance is given by $\mathbf{P} = \begin{Vmatrix} 1-\pi & \pi \\ 1-\pi & \pi \end{Vmatrix}$, 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 abundant), and that the probability of famine is very low, so that the probability of two successive famines is negligible $\left(\pi^2 \approx 0\right)$. This reduces the intertemporal choice problem to one of storing sufficient body fat during times of plenty to survive an occasional famine. Specifically, given that food is plentiful at time *t*, the fitness maximizing choice of f_t will be given by:

$$\max_{f_p} A\, r_{t|p}\left(f_{\theta'}, p; f_p\right) r_{t+1|s}\left(f_p, s; x_{ps}, f_s\right)^{\pi}$$

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

$$\pi\, r_{t|p}\, r'_{t+1|s} + r'_{t|p}\, r_{t+1|s} = 0$$

or equivalently,

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

where $\eta_t = \frac{f_t}{r_{t|p}} r'_{t|p}$ and $\eta_{t+1} = \frac{f_t}{r_{t+1|s}} r'_{t+1|s}$ 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_t. 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.

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 the 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, 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, and will be crucial to the discussion of evolutionary dynamics to follow: Parental traits are passed to offspring *genetically*; 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 satiation), 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 intertemporal independence to be important, 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 3.4 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 payoff function 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 $\{P[\pi_i]\}_{i=1}^{N}$.[21] 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

[21] The notion that simple behavioral algorithms can (and should) take advantage of regularities in the decision environment has been called *ecological rationality* (Gigerenzer *et*

constant (i.e., $\pi = 0$). In the absence of sufficient time (recall that the preferences 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 summer 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 $\pi_{\min}$ (the distribution with 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 value of π 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:

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

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 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:

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

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

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al, 1999).

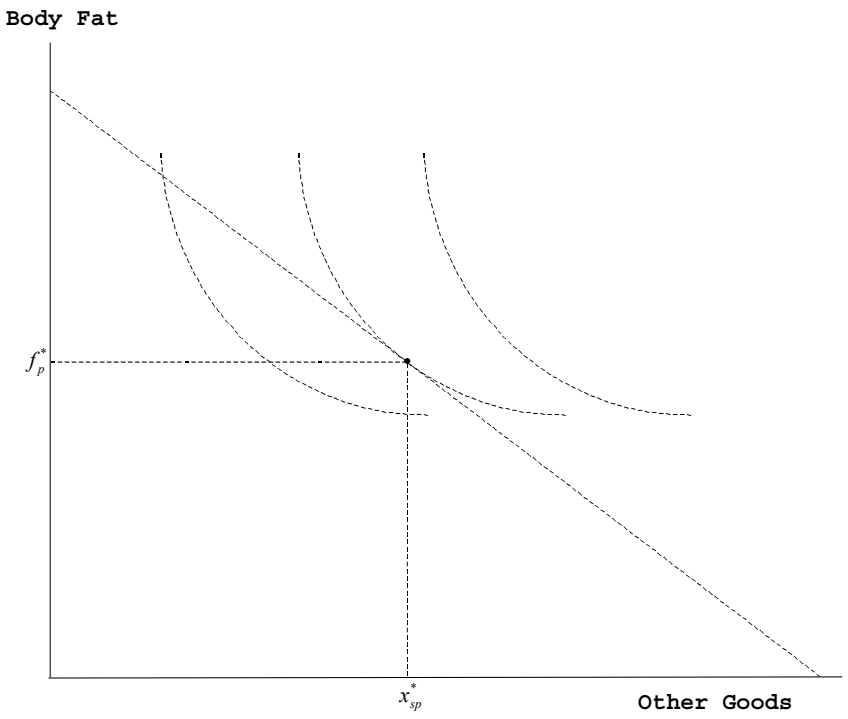

Figure 4: A Stable Food Supply and Evolutionary Disequilibrium

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.

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 $\hat{P}[\pi_j]_{j=1}^{\hat{N}}$ 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 *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.

An observer unschooled in our rodent population's evolutionary history might observe the high incidence of obesity in these animals and conclude that it was the "high-fat diet and sedentary lifestyle" of the laboratory environment that induced the disease. An animal rights advocate interested in the welfare of the animals might conclude that—since they are not forced to overeat, and there are ample opportunities for exercise within the laboratory walls—the rodents are much better off in the laboratory than in the wild, where there are risks of predation and food supplies are not reliable. The former conclusion is probably true but perhaps trivial; the latter conclusion, though perhaps less compelling, is consistent with a basic tenet of consumer choice theory—that expanding the choice set of an individual cannot make him worse off. But this latter conclusion, if extended to humans, is exactly what is being called into question by the economic theory of self-control described in Section 2 above. Viewed in the context of the evolutionary history of the species, such behavior is no longer puzzling and it becomes clear that there is more to the phenomenon than dynamic inconsistency or non-conventional preference domains.

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]

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. If, however, the Bayesian prior is genetically encoded, sudden elimination of risk from the food supply will result in chronic obesity. Applied to human behavior, two of the assumptions used in reaching this conclusion are unconventional and require elaboration: (i) that the Bayesian prior is *genetically encoded*, and (ii) that there has been a *sudden* shift in the degree of uncertainty in

[22] The idea that contemporary economic behavior reflects in some way preferences generated by evolution in the distant past is by no means new to the economics literature. See, for

the human food supply. Sections 4 and 5 below offer empirical evidence supporting these two propositions, respectively.

4. How Genes Influence Behavior

Over the course of a decade the average adult consumes approximately 10 billion 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 *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

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example, Hirshleifer (1977), Rogers (1994), Bergstrom (1996), and Robson (2001)

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 guide 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 variations 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). The topic of the universality of the human genome will be addressed more fully in Section 5 below.

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 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

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[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).

genes can be inferred from the comparison of the body mass index 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. In other words, there appear to be genes (discussed further in Section 4 below) that cause individuals to be *susceptible* to obesity, if the conditions are right. 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, decreased immune function, and infertility. These reactions 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*.

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[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

Early experiments using parabiosis[26] 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—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 research[29] into the

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error.

[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).

[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).

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 homologous[31] 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). http://www.obesityresearch.org/cgi/content/abstract/10/3/196 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 according to their parents, both children were reported to be "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-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 2000 the obesity genome map included more than 250 genes associated with feeding behavior in humans (Pérusse *et al*, 2001). Nearly all of these genes are

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[30] Base pairs are the molecular "letters" of DNA which 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.

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 expressed[32] 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" *cholecystokinin (CCK)* in 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 *thyrotopin-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 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 satiation such as

[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.

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) (Stryer, 1981; Pert, 1997). The activated receptor, in turn, induces cellular changes, often with magnitudes proportional to the amount or concentration of ligand present. Leptin, for example, circulates in the bloodstream until it comes in contact with leptin receptors in hypothalamic neurons in the brain. These neurons, upon activation by the leptin ligand, transmit this information (regarding the concentration of leptin in the bloodstream) to other parts of the brain that collect and process information about the nutritional state of the body (DeFalco *et al*, 2001). When leptin and the other signals of bodily energy reserves are absent or low in concentration, this is presumably perceived as a feeling of hunger, and the behavioral consequence is to take steps to achieve energy surplus (e.g., by reducing body temperature, eating more, and limiting physical activity) (Schwartz *et al*, 2000; McMinn *et al*, 2000; Woods *et al*, 1998).

There are a number of ways in which variations in genes might "calibrate" the molecular machinery that regulates energy homeostasis. The strength of the "signal" received and transmitted by the leptin receptor, for example, is a function of not just the concentration of leptin in the blood supply to the hypothalamus, but also the amount of time leptin remains bound to the leptin receptor—the *residence time*—before being re-released to the bloodstream. 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 leptin and/or the leptin receptor might simply reduce the residence time of 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).

ligand in the receptor, in effect lowering the strength of the signal received by the brain. Another important quality is the *stability* of the ligand; for example, leptin, once produced, remains in the bloodstream much longer than does insulin before decomposing into waste products. If a variation in the leptin gene reduced the stability of leptin, the concentration of leptin in the blood would be reduced for any given level of adipose tissue, again lowering the effectiveness of the message received by the brain. 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 (Gazzaniga, 2000; Gazzaniga *et al*, 1998; Pinker, 1997).

Ligand/receptor systems are the body's method of transmitting information from one cell to another—even, in the case of endocrine hormones like leptin, if those cells are located in entirely different organs—and are characterized by specificity (the ligand "key" will only activate one kind of receptor "lock"). The strength of the informational signal can be calibrated by genes.

The strong evidence that leptin and other genetically modulated signaling molecules play a role in regulating feelings of hunger and satiation—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 satiation tell them to do so under certain conditions (e.g., those conditions prevalent in today's Western societies).

This is not to say that obesity 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 convention in economic theories of choice is to take *feelings* as (exogenously determined) inputs to the decision process. The point here is that feelings of hunger and satiation are generated by a molecular process that begins with our genes. As such, we might reasonably expect the strength of these "feelings" to vary from person to person. More importantly, acknowledging the link between genes and behavior allows us to ask questions about the evolutionary process that generated these genes, and then to ask what

this means for economic theory.

Because genes are the product of biological evolution, any explanation of non-equilibrium effects requires some knowledge of the dynamics of the evolutionary process. Fortunately, recent advances in the biological sciences provide insight here as well, and are the subject of the next section.

5. Technology Has Outpaced Human Evolution

To most people "evolution" implies a dynamic, ever-changing process. Those who have studied a bit of biology are likely to know that the process of biological evolution can operate on glacial time scales, making direct observation of the process nearly impossible. Both aspects were emphasized by Charles Darwin (1859), with the malleability of biological form and function comprising (at the time) the most revolutionary part of his theory, while the exceedingly slow and gradual nature of the process helped to excuse his lack of direct evidence.[34] But how slow is slow? I will argue here that the appropriate time scale for the evolution of modern humans is certainly more than the scale of years or decades in which technology alters the modern diet and lifestyle.

If we are going to apply the "stable disequilibrium" concept of Section 3.5 to modern human behavior, we need to know something about the dynamics of the process by which the genetic makeup of the human race changes. More specifically, if we accept the proposition that human genes in the past became adapted to an environment in which the food supply was at risk, how do we know that these genes have not been re-shaped by modern advances in agriculture, or that they are not in the process of adapting, heading toward a new equilibrium?[35] Fortunately there is a considerable amount of evidence available today that

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[34] Darwin's theory, of course, eventually succeeded on the strength of its internal logic and the available indirect evidence, both from the natural world (e.g., geologic stratification, the fossil record, locally adapted living populations of plants and animals) and from the spectacular success of artificial selection in domesticated animals and plants (Weiner, 1994).

[35] Unfortunately the more popular models of evolutionary game theory and evolutionary economics are of little help here. The equilibrium concept I have employed, which might be viewed as a special case of the evolutionary equilibrium, or *evolutionarily stable strategy* of Maynard Smith and Price (1973; see also Maynard Smith, 1982), has nothing to say about the dynamics by which equilibrium obtains. The widely used *replicator dynamics* (Taylor and Jonker, 1978; Weibull, 1997) is suitable for answering questions of equilibrium selection, stability, and dominance, but its employment of a continuous time setting and a stable payoff

can help to answer these questions.

The first line of evidence provides information about ancient demography, including the size and location of populations of the prehistoric ancestors of all human beings alive today. Human DNA can be modified over time only at the time of reproduction, and then only by rare mutations and the process of recombination (Wessels and Hopson, 1988). Because some DNA does not code for genes (and therefore is not subject to the judgment of natural selection) and some non-coding DNA is not subject to recombination, it acts as a sort of "evolutionary clock" that can be used to reconstruct ancient human demography. This is accomplished by measuring the mutation rate of the DNA in question, collecting DNA samples from living human populations, and drawing inferences about prehistoric population dynamics (Rogers and Harpending, 1992). Such methods provide strong evidence that all humans alive today are descended from an ancestral population on the order of 10,000 individuals living 50,000 to 100,000 years ago. In other words, DNA studies tell us that the ancestral human population passed through a "bottleneck" or perhaps an extended period of stasis, then underwent a massive expansion to reach the numbers observed in the last few thousand years (Harpending *et al*, 1998; Boyd and Silk, 2000).

One consequence of this prehistoric population expansion is that there is much less genetic variation present in contemporary human populations than would be expected in equilibrium. This has been confirmed to some extent by genetic demographers, who have analyzed DNA samples in human populations all over the world. The most striking finding in these studies has been the degree to which *gene frequencies remain constant across populations that have remained genetically isolated for many thousands of years.* In fact, by some measures, observed genetic variation *within* populations is much greater than observed genetic variation *across* populations (Cavalli-Sforza *et al*, 1994). It would almost certainly be an overstatement to say that human evolution ended with the ancestral human population of 50+ millennia ago, but the evidence seems to suggest that human genes present today are, for the most part, drawn from the pool of genes present in this ancestral group.

So what evolutionary changes might we expect to have taken place as *homo sapiens* moved from foraging to agriculture to industry? A strong argument can be made in favor of

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function tends to emphasize the differential fitness of various strategies without generating

the conservation of *complex adaptations* such as the internal organs or the cognitive structure of the brain (Tooby and Cosmides, 1990). This argument certainly applies to the set of molecular mechanisms that regulate energy homeostasis: though there may be some genetic variation in individual susceptibility to obesity, the biochemical details appear to be universal in humans. But body fat is a *polygenic quantitative trait*—that is, it varies continuously across individuals in the population, and is controlled by many genes—and is therefore susceptible to change at the population level in the presence of selective forces. In other words, if individuals with excess body fat in a given generation survive and reproduce more (less) successfully, on average, than those with little body fat, then the population in subsequent generations will have, on average, more (fewer) "thrifty genes" that promote the storage of body fat. Unfortunately, this does not provide a conclusive answer to the question of the *rate* at which gene frequencies might be expected to change.

Another angle from which to consider the problem of the effect of improving food security on the gene frequencies is to consider the extent to which the food supply has in fact been constant in modern times. Archaeological evidence tells us that humans began the transition from foraging to subsistence agriculture around 10,000 years ago (Boyd and Silk, 2000), and this is almost certain to 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."

It seems implausible that a century or two of food security would be sufficient to re-

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realistic predictions about the rate at which we might expect equilibrium to be reached.

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 is significant adaptation 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.[36]

6. 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. But an important measure of any theory that purports to describe human behavior is its predictive value. To paraphrase Milton Friedman's famous 1953 essay, the proper test of this 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 a lack of 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

[36] 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, 2000). 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.

environmental cues, and condition behavior accordingly.[37] So any technological change that removes the probabilistic link between cues (whatever they might be) and outcomes is a potential source of evidence of the influence of a "Nature's Thumbprint" effect on behavior. A few examples of such evidence are offered below.

6.1 Isolated Human Populations and Rapid Cultural Change

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). As noted in Section 4.2 above, the rapid rise in obesity in the United States in the last few decades has happened much too quickly to have been the result of 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 in the form of a case study of the Pima Indians of Southern Arizona and Northern Mexico (Ravussin *et al*, 1994). Though circumstance separated these two groups less than 1000 years ago (too little time, it has been suggested, for the two populations to diverge genetically), one continues to live a traditional lifestyle, while the other has been exposed to modern American culture. David Heber and Susan Bowerman summarize and interpret the findings:

> *"The Pima Indians of Northern Mexico and Southern Arizona demonstrate the impact of nutrition on genetic expression of chronic diseases such as obesity and (type 2 diabetes). Pima Indians in Arizona have the highest reported prevalence of obesity and (type 2 diabetes), whereas the incidence is low in Pima Indians living in Northern Mexico. Pima Indians living in the mountains of Northern Mexico were separated some 700 to 1000 years ago from the Pima Indians living today in Arizona and continue to live a traditional lifestyle. They eat a diet with much less animal fat and more complex carbohydrates, and have higher activity levels than do the Pima Indians in Arizona. This population demonstrates increased body weight, height (which is a biomarker of prepubertal nutrition), increased body mass index, increased cholesterol, and increased incidence of (type 2 diabetes). The 'thrifty gene' hypothesis states that genes for conserving energy, while adaptive in a traditional environment, are responsible for the high prevalence*

[37] For examples of the sensitivity of humans to behavioral cues, see Laibson (2001) and Samuelson and Swinkels (2000).

of obesity and diabetes in persons exposed to overnutrition and underactivity." (2000, p. 15)

Another striking example from the other side of the globe is described by Jared Diamond (1992):

"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."

6.2 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 or relative food abundance, but also of social status and absolute food abundance. 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 similar to that explained in Section 3.3.2 above. A summary follows.

In choosing where to search for food, willow tits, like many foraging animals, face a trade-off between caloric intake and predation risk. 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; when food is abundant, however, the dominant chooses a lower predation risk and lower level of body fat, taking advantage of the fact that in times of need he will still be able to obtain sufficient food supplies. So when food is scarce, high-status individuals are fatter, but when food is abundant, low-status individuals are fatter (Clark and Ekman 1993, Figure 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.[38,39] This altered blood chemistry might well trigger increases in 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.[40]

6.3 Seasonality and Body Fat

It is common among foraging animals in natural environments to observe seasonal

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[38] 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 vervet monkeys.

[39] 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."

[40] 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

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 abundance, 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 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 triggering 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).

This also turns out to be another case in which there are striking parallels between human and animal behavior. 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 twin studies have shown that incidence can be at least partially 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*, the victims of which tend to suffer from insomnia,

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antidepressants are also SSRIs.

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)[41]. 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 by controlled studies. The dependable simultaneity 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.

6.4 Birth Weight as a Conditional Event

Another reliable signal of an unstable food supply in natural environments is malnutrition early in life. There is some evidence that this may be another important determinant of bodily energy reserves, though not necessarily in the consciously rational sense that economists commonly look for—rather, the evidence stems from the simple fact that fetal malnutrition is a risk factor for obesity later in life (Barker *et al*, 1989; Barker, 1995; Petry and Hales, 2000). In a review article, Peter Kopelman argues that this might be an evolved adaptation:

> *Evidence indicates that undernutrition of the fetus during intrauterine development may determine the later onset of obesity, hypertension and type 2 diabetes independent of genetic inheritance. Such a phenomenon suggests the possibility of long-term programming of genetic expression as a consequence of altered intrauterine growth. Barker has emphasized that an adverse nutritional environment* in utero *causes defects in the development of body organs leading to a 'programmed' susceptibility that interacts with later diet and environmental stresses to cause overt disease many decades later. In support of the hypothesis is the finding of an inverse relationship between birthweight and systolic blood pressure in both men and women in later life, with the highest mean systolic blood pressures being observed in those with the lowest birthweight and highest current weight.*

[41] 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 *et al* (1984) report seasonal effects on childhood obesity.

> *Central to the ('thrifty phenotype') hypothesis is the view that a predisposition to type 2 diabetes and other conditions, including adult obesity, is an adaptation to malnutrition by the developing fetus. Low birthweight is a proxy for a variety of intrauterine influences but predominantly is caused by maternal malnutrition. It is suggested that the fetus adapts its growth and metabolism to the expectation of poor availability of nutrition postnatally. This may have survival advantages* in utero *by targeting available nutrients to essential organs and, in later life, by increasing the ability to store energy as fat to provide energy reserves for use when food is scarce. These adaptations are detrimental when there is a constant supply of nutrition… (2000, pp. 638-639)*

7. Conclusions

7.1 Related Work

The evidence presented above suggests that—at least with respect to diet—the self-control problem has been written into our genes in the course of human evolution. An individual might "plan" to eat less in the future, but because the molecular basis for sensations of hunger and satiation is effectively immutable, such plans are often doomed to failure. Because humans evolved in an environment in which there was zero probability of a certain future food supply, this zero probability is written into our genes, and systematic overeating results. Thus a phenomenon that has been perceived as a problem of self-control can instead be viewed simply as a case of a *false prior distribution*. As a purely descriptive theory of behavior, the self-control model is, broadly speaking, observationally equivalent to the model of stable disequilibrium I suggest. But in addition to the specific predictions of the stable disequilibrium theory (which are borne out in the empirical phenomena described in Section 6 above), the various theories of self-control differ on some finer points, which deserve comment here.

Becker and Mulligan (1997) offer a conjecture as to how their model (in which consumers expend effort to "reduce the discount of future utilities") might be extended as a pure theory of costly information:

> *A pure "information gathering" model of the formation of time preference should be a subject of future research; it is likely that the predictions of such a model will differ from those of our model. We expect, for example, that on average new information should neither be "good" nor "bad." The gathering of more information may increase utility, but gathering might* ***decrease*** *the weight placed on future consumption when one learns that future resources will be less effective at producing utility than one had believed. In contrast,*

> *more future-oriented resources must increase the weight on future consumption in our setup. (p. 735)*

The model I have proposed is indeed an "information gathering" model of the formation of time preference: Consumers in this model are born with a Bayesian prior distribution with respect to the variability of food resources, and adjust their beliefs (and thus their behavior, or "time preference") as they gain experience. However, the fact that this prior distribution is systematically biased is not consistent with the authors' conjecture that on average information will be neither "good" nor "bad"; because of the evolutionary bias in the prior, new information will be, on average, "good." In this sense, Becker and Mulligan may have been correct in their original assumption, that there is value in expending effort to limit one's consumption. The fact that the prior distribution can limit both the speed and the extent of learning, however, raises difficult questions about the welfare implications of the model of human preferences presented in Section 3. These will be discussed in Section 7.3 below.

Samuelson and Swinkels (2000) provide an explicit model of self-control as a problem of costly information. They employ an evolutionary setting in which Nature chooses the optimal degree of flexibility (i.e., sensitivity to new information) by attaching both "direct utilities" (payoffs in terms of immediate pleasure or pain) and "indirect utilities" (payoffs derived from the agent's cognition or beliefs), given that information and cognition are costly. One interpretation of this approach, which emphasizes the evolutionary equilibrium solution, is that the underlying probability distribution—the Bayesian prior—limits the degree of behavioral flexibility.

Paul Romer (2000) considers a similar problem when he draws a distinction between *autonomous* behavioral mechanisms, *feeling-based* behavioral mechanisms, and *thinking-based* behavioral mechanisms. Romer points out that both autonomous and feeling-based mechanisms are presumably products of human evolution, and that "mistakes" in these mechanisms often correspond to modern situations that did not arise in the history of human evolution. He concludes that "In terms of the traditional language of welfare economics, this mistaken behavior will be welfare-maximizing given the existing set of feelings."

Sozou (1998) describes a simple way in which Bayesian updating can generate behavior consistent with hyperbolic discounting over consumption streams. 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 the model of 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.

7.2 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 the first quotation, 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 conventional view in the economic theory 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 is subject to change in response to changes in prices, or to (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 emotion of interest here being "hunger") is fully consistent with the model offered herein. Nothing in the formulation of the model in Section 3 implies 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.[42] But there are important consequences to 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 probability of starvation and the probability implied by our genes.

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

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 implicitly incorporate assumptions about the probabilities of outcomes. His theory of decision-making under conditions of uncertainty has been widely acclaimed as the foundation of modern decision theory. Savage's theory is also consistent in many ways with the conclusions being drawn in this essay, and as such it deserves elaboration.

Savage's accomplishment was to demonstrate that a few reasonable axioms could be shown to imply that when outcomes are uncertain, decisions should be made according to the expected utility property, *even if probabilities are not objectively known*.[43] The expected utility property implies that the optimal choice is that which maximizes the weighted sum of the utilities of all possible outcomes, with the weights being the probabilities of the associated outcome. In the Savage formulation, it is necessary to define an exhaustive and mutually exclusive set S of *states* of the world, the set Z of all *prizes* or consequences of concern to the decision-maker, the set F of *actions* that map from the S to Z, and a *preference relation* $\succ$ over actions. *Probabilities* of the states $p[s]$ are *subjective* (i.e., they arise from the preference relation), as are utilities. Although Savage had in mind a static setting that doesn't allow for Bayesian updating, it is informative to consider the various ways in which his model of subjective probabilities and expected utility might relate to the approach offered in Section 3.

In a standard application of expected utility theory to the problem of obesity, we might specify the states to be {*famine, feast*}, but note that the state {*famine*} is so unlikely in an industrial economy that any reasonable subjective probability of this state must be so small as to be negligible. Therefore, the choice of how much energy to devote to body fat and how much to physical activity or other goods would be best represented by a model of decision-making under conditions of certainty (Figure 1). Accordingly, if people express difficulty maintaining a diet plan, this would suggest a self-control problem (Figure 2).

In an environment where famine is a regular event, however, it is clear that a model of

[43] An alternative to the axiomatic approach, more akin to the approach adopted in Section 3, is to show the conditions under which the expected utility property is a necessary condition

uncertainty is appropriate. A very simple application of expected utility then might specify the prize space to be {*fat, thin, dead*}, the action space to be {*gorge, nibble*}, and the subjective probabilities of the two states {*famine*} and {*feast*} to correspond closely with empirically observed probabilities. This reduces the problem of summertime consumption in Figure 3 to a binary choice between {gorge} and {nibble}, where the consequences are $f[gorge] = \begin{cases} thin \text{ if } famine \\ fat \text{ if } feast \end{cases}$ and $f[nibble] = \begin{cases} dead \text{ if } famine \\ thin \text{ if } feast \end{cases}$. Assuming the preference relation over certain outcomes is $thin \succ fat \succ dead$ (so that $u[thin] > u[fat] > u[dead]$), consumers will choose the action *gorge* (*nibble*) if and only if the probability of *famine* (*feast*) is large enough that

$$p[famine] \cdot u[thin] + p[feast] \cdot u[fat] \geq p[famine] \cdot u[dead] + p[feast] \cdot u[thin]$$

(and vice versa). In the context of the evolutionary equilibrium of Section 3.2, the preference relation $\succ$ is endogenous, such that the subjective probabilities will correspond to actual probabilities and the function $u[\cdot]$ corresponds to $\ln r(\cdot)$.

In the context of the disequilibrium described in Section 3.5, actual probabilities will have changed, but $\succ$ will be exogenous and of the form determined by the preceding equilibrium. Therefore, it will no longer necessarily be true that subjective probabilities $p[\cdot]$ correspond to actual probabilities, or that $u[\cdot]$ corresponds to $\ln r(\cdot)$ (Figure 4).

Upon reflection, the preference relation described by Savage, with its implicit assignment of both utilities and subjective probabilities to future outcomes, seems an apt description of the molecular system of genes and hormones that influence cognitive processes in the human brain. The evolutionary process that generated the human genome is by definition a probabilistic one, and the "feelings" (e.g., hunger, satiation, satisfaction, outrage) it assigns to various future outcomes seem akin to what Savage had in mind when speaking of "utilities".

7.3 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*

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for maximizing Darwinian fitness (e.g., Robson, 1996).

> *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)*

Though this essay began as a modest inquiry into the nature of the behavioral puzzle posed by obesity and self-control, the conclusions reached here have profound implications for the modern theory and practice of economics. This essay has offered evidence that our preferences (with respect to the storage of body fat) are to some extent written into our genes, and that the probability distributions implied by these preferences do not correspond to the actual probability distributions observed in the modern world. In other words, *there is a well-defined sense in which people fail to act in their own self-interest*. This phenomenon is most apparent in cases such as obesity in which the negative consequences of one's actions become apparent over time and lead to an awareness of a "self-control" problem.

Taken to it's logical extreme, this finding has the troubling consequence of undermining a foundational tenet of welfare economics. To challenge the inviolable assumption of 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 (see, e.g., Stiglitz, 2000) 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. [44]

The economic theory of self-control, in allowing preferences to be defined over choice sets as well as outcomes, adopts a more conventional and intermediate view: since markets for choice sets are not feasible, markets are incomplete, justifying the policy implications that follow.[45] But closer examination of the biological underpinnings of the self-control problem

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[44] Showing the theoretical implication of a behavioral phenomenon taken to its logical extreme does not, of course, answer the more important question of the economic *magnitude* of the phenomenon. This essay does not purport to provide an answer to this question, other than to note in Section 0 that the prevalence and health implications of obesity suggest it is a problem of considerable economic magnitude, and to argue in Sections 3 through 6 that obesity results from the behavioral phenomenon (mis-specified subjective probabilities) in question.

[45] This view is implied in the quotation of O'Donoghue and Rabin which opens this section.

suggests a more fundamental flaw in the Fundamental Theorem. Specifically, because preferences necessarily assume a probability distribution over outcomes, *a market economy will yield an efficient allocation only when the prevailing probability distribution is equivalent to that implied by the preferences of the agents*. Allowance might be made, on feasibility grounds, for some error on the part of agents, 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 be specific about how we define individual welfare. The main conclusion of the model of Section 3 is that in modern economies people fail to maximize individual *fitness* in the biological, Darwinian sense. But it is another thing entirely to suggest that people *should* try to maximize fitness, much less that welfare economists should encourage them to do so. Of primary interest to the welfare economist, and to anyone who cares about the human condition, are the very real experiences of satiation and hunger, success and failure, elation and despair. If we assume, as was suggested above, that these experiences constitute the "utilities" Nature has assigned to outcomes (independent of the probabilities of those outcomes), then the objective of welfare economics ought to be the maximization of some function of these utilities over time. That these utilities fail to correspond to "expected number of offspring" in modern environments is not important for economic analysis. But the mismatch between the probabilities implicit in human action and the actual probabilities in the modern world raises questions of the optimality of the economic environment: the prices, the probabilities, the behavioral cues. The modern obesity epidemic suggests that markets may in some cases fail to provide the welfare-maximizing environment—not even in the modest, Pareto sense. It is not immediately clear, however, what a welfare economist should do about it.

The policy implications that will flow from a more informed view of human nature are likely to be incremental and akin to successful policies already being implemented. There might be, for example, welfare-improving educational programs that hasten the speed at which behavioral adjustments are made through the process of Bayesian updating. But much will depend on determining the specific cues that trigger these adjustments. It was noted in Section 6, for example, that some evidence exists that malnutrition early in life increases the risk of obesity later in life. A "welfare-improving educational program" in this case might consist of the provision of proper nutrition to expectant mothers and at-risk infants. It has

also been argued that the nutritional composition of modern diets may be to blame for a host of "diseases of civilization," including not just obesity, but also heart disease, hypertension, arthritis, and cancer, to name a few (Eaton and Konner, 1985; Eaton *et al*, 1988; Nesse and Williams, 1994).[46,47,48] In this view, overeating is presumably the result of the general dearth of key nutrients in processed foods. The body, sensing deficiency in these nutrients, responds by increasing caloric intake—which in the distant past of human evolution would have provided proper relief. This would seem to imply a reliance on an ancient Bayesian prior with respect to the distribution of nutrients in available foods. In this case a "welfare-improving educational program" might seek to provide consumers with nutritional advice, or perhaps take some steps to re-establish the link between calories and essential nutrients.

Perhaps the 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.[49]

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[46] I argue elsewhere (Smith, 2002) that evolution has endowed the human race with dietary preferences that are remarkably effective at meeting nutritional needs in natural environments. In the presence of such modern innovations as processed food and television advertising, however, we continue to follow these ancient algorithms to ill effect.

[47] Indeed, the "evolutionary diet" has recently become the latest in fad diets (e.g., Eaton *et al*, 1988, Simopoulos, 1997, and Somer, 2000). As such, it should properly be viewed with skepticism, but there is some experimental evidence that dietary composition can affect appetite and weight gain (Lawton *et al*, 1993).

[48] Sapolsky (2000) tells of a "natural" experiment in which a wild group of baboons was exposed to the modern human diet. When a community in Kenya located its municipal garbage dump within the range of these baboons, the entire troop stopped eating its normal diet of fruits, leaves, and tubers, and fed exclusively on the decaying refuse found in the dump. Subsequent blood tests revealed extraordinarily high levels of cholesterol and insulin among members of the troop.

[49] The alternative view of the theory of self-control—that probabilities are known but preferences are defined over choice sets—allows, in theory, for the inference of welfare from behavior: Whenever the self-control problem arises, the economic costs of the problem and its various policy solutions could presumably be measured via the observation of situations in

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, and might now deserve the attention of economists (e.g., Kahneman, 1999; Larson and Fredrickson, 1999). But at least one conventional measure of economic welfare holds some promise: expenditures on behavioral drugs. Presumably, expenditures on behavior-altering drugs provide a lower bound on the economic magnitude of the self-control phenomenon. The menu of such drugs, from appetite suppressants to antidepressants, grows larger every year, and each user of caffeine, alcohol, Prozac, or Viagra is in effect paying for a new set of preferences.

Since it was the magnifying glass of molecular biology that led to this quandary, perhaps there is also hope that the same technology will also provide a solution. The day is rapidly approaching when we will be able to say with some confidence whether an individual with a given genetic profile 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).

Although this paper has been limited in its scope to eating habits and obesity, the method applied here clearly has application to other realms of human behavior. Modern science is making it clearer every day that genes influence behavior: It is now known, for example, that aggression is heritable (Coccaro *et al*, 1993) and some of the genes that regulate irritability, anger, and aggression have been identified (Manuck *et al*, 2000). The evolutionary psychology literature is rich with examples of human behaviors that bear the mark of the distant evolutionary past, from gossip (Barkow, 1992) and dating (Wright, 1994) to social exchange (Cosmides and Tooby, 1992) and coalition formation (de Waal, 1998), all of which are amenable to the sort of model I have employed here. There are also many behaviors other than self-control, reported in the behavioral economics literature, which appear to be inconsistent with conventional normative theories of economic behavior and may be amenable to an "evolutionary disequilibrium" interpretation. These deserve attention in

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which choice sets can be explicitly chosen.

future research.

8. Mathematical Appendix

Proof that $\left(\frac{\partial f_p^*}{\partial I_p} > 0\right)$**:** Define the implicit functions $x_s^* = x_s^*\left(c_s, I_s, c_p, I_p\right)$, $f_s^* = f_s^*\left(c_s, I_s, c_p, I_p\right)$, $x_p^* = x_p^*\left(c_s, I_s, c_p, I_p\right)$, and $f_p^* = f_p^*\left(c_s, I_s, c_p, I_p\right)$ as solutions to the fitness maximization problem. Writing the Lagrangian of the problem as

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

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

$$g_1\left(x_s^*, f_s^*, x_p^*, f_p^*\right) - \lambda_s \equiv 0$$

$$g_2\left(x_s^*, f_s^*, x_p^*, f_p^*\right) - \lambda_s c_s + \lambda_p c_p \equiv 0$$

$$g_3\left(x_s^*, f_s^*, x_p^*, f_p^*\right) - \lambda_p \equiv 0$$

$$g_4\left(x_s^*, f_s^*, x_p^*, f_p^*\right) + \lambda_s c_s - \lambda_p c_p \equiv 0$$

$$I_s - c_s f_s^* + c_s f_p^* - x_s^* \equiv 0$$

$$I_p - c_p f_p^* + c_p f_s^* - x_p^* \equiv 0$$

where $g_i(\cdot)$ denotes the partial derivative of $g\left(x_s^*, f_s^*, x_p^*, f_p^*\right)$ 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 $g(x_s, f_s, x_p, f_p) = r(x_s, f_s) \cdot r(x_p, f_p)$, the cross-partials of $g(\cdot)$ can be expressed as functions of the first and second derivatives of $r(\cdot)$: $g_{11}=r_{11}r_p$, $g_{12}=g_{21}=r_{12}r_p$, $g_{13}=r_1r_3$, etc., where $r_\theta = r(x_\theta, f_\theta)$. 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 $\left(\frac{\partial(f_p^* - f_s^*)}{\partial I_s} < 0\right)$**:** 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) $-2 c_s r_2 r_4 r_{11} r_p$
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) $c_p r_1 r_3 r_{44} r_s$

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 $\mathbf{H}_{52}$-$\mathbf{H}_{54}$<0.

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