School of Economic Sciences

Working Paper Series
WP 2006-4

Reconciling Psychology with Economics: Obesity, Behavioral Biology, and Rational Overeating

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May 31, 2006

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Abstract

The modern phenomenon of obesity is an archetypal example of a behavior whose explanation simultaneously falls within the purview of psychology, economics, and the biological sciences. While psychologists and advocates of public health have long viewed overeating as a weakness or disease in need of treatment, economists have pointed out that–like any other consumer behavior–choices about diet and exercise can be viewed from the perspective of rational decision theory, subject to the influence of variation in price and income but not necessarily as a problem in need of a solution. Recent advances in our understanding of the physiological mechanisms by which genes influence behavior in modern socioeconomic environments have begun to point the way to a resolution to this debate. Drawing inspiration from the scientific literature on the neuroendocrinology of energy homeostasis, this paper reviews the empirical determinants of obesity in light of the biologist's notion that humans and other animals evolved the ability to store body fat as an optimal response to the presence of starvation risk. This approach yields a powerful theoretical foundation, capturing such features of obesity as dynamic inconsistency, genetic variation, susceptibility to pharmaceutical intervention, and variation by season, socioeconomic status, and degree of financial security. It also provides a framework for reconciling the conflict between behavioral (descriptive) and neoclassical (prescriptive) economics.

Key Words: evolution, behavioral ecology, neuroeconomics, self-control, serotonin, nicotine, MDMA

*The author thanks Ted Bergstrom, Ray Huffaker, Jill McCluskey, Dottie Smith, and Chris Stoddard for helpful comments and discussions. Parts of the research for this study were performed while the author served as a fellow in the Deutsche Forschungsgemeinschaft, Graduiertenkolleg (DFG) 629 at the University of Bonn and in the UCLA International Institute, and financial support from these organizations is gratefully acknowledged.

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JEL: D01 D83 D91 I12

1 Introduction

The common laboratory mouse, when subjected to starvation, consistently exhibits a number of characteristic symptoms, including decreased body temperature, hyperphagia (a tendency to eat voraciously when food is available), decreased physical activity, diminished immune function, and infertility. These physiological and behavioral responses to severe calorie deficit have the collective effect of conserving available energy for vital functions, and (in the case of hyperphagia) hastening the restoration of energy reserves when the opportunity arises. For many years, scientists have also known about a strain of mouse (aptly dubbed the *obese*-type mouse) that carries a recessive gene that induces morbid obesity.

The funny thing is, the *obese* mouse also exhibits *every one of the above-mentioned symptoms of starvation.*

Though the notion of an enormous overfed mouse exhibiting the physiological symptoms and behavioral manifestations of starvation might seem paradoxical at first blush, there is a simple explanation: the *obese* mouse, we now know, is born with a mutant form of the *obese* gene that– in normal mice–codes for *leptin*, a peptide hormone secreted by fat cells. In normal mice, the concentration of leptin in the blood is proportional to the amount of energy stored as body fat, and the leptin "signal" (detected by leptin *receptors* in the hypothalamus) is used by the rodent's central nervous system to regulate physiology and behavior accordingly (Zhang *et al.* 1994). Their *obese* cousin, on the other hand, lacks circulating leptin, and acts as if energy reserves are (always) dangerously low.

The *obese*-type mouse might be nothing but a biological oddity (and thus of little interest to economists and other social scientists) were it not for a subsequent discovery: humans have been identified with the same disease. The normal form of the *obese* gene is now known to encode for leptin in humans as well as mice, and though genetically inherited defects in the *obese* gene are exceedingly rare in human populations, a few cases have been documented. In one such case, a pair of cousins whose genetic endowment prevented leptin synthesis were of normal weight at birth but rapidly gained weight in infancy. Their parents were emphatic about the behavioral manifestations of the disease, describing both children as "constantly hungry, demanding food continuously and eating considerably more than their siblings" (Montague *et al.* 1997) . Injections of a synthetic version of leptin have a dramatic negative effect on appetite and body weight in such patients (Farooqi *et al.* 1999).

It is customary among practitioners of the science of economics to begin any study of consumer behavior with a presumption of *rationality.* Granted, the human animal might at times tend to the quirky or the capricious, but a productive social science, the argument goes, must focus on the systematic (rather than the idiosyncratic) aspects of behavior if it is to have any hope of generating a

theory with substantial predictive power. In practice, of course, the rationality rule is not much of a restriction: if one is willing to be flexible in making postulations about human motivation and desire, virtually any pattern of behavior can be modeled as the solution to an optimization problem and therefore "rational". Nor does the more conventional meaning of the word seem entirely inconsistent with everyday experience. We all make decisions every day, after all, and when asked we can usually come up with reasonable explanations for our behavior–explanations that are readily translatable into the language of constrained optimization.

But there are also instances of consumer behavior that stretch even the economist's conception of rationality. The phrase "rational overeating" in the title of this paper is offered, in part, as a challenge to this conception: in what sense can *over*eating be optimal? I will argue in what follows that the answer to this question–and perhaps to other behavioral peculiarities at the intersection of psychology and economics–lies in a deeper understanding of behavioral biology. Which brings me back to leptin and the *obese*-type mouse.

Why is leptin relevant to economic analysis? There are two reasons. First of all, the striking and direct relationship between consumer behavior and physiology at the *molecular* level of both DNA (i.e., a particular variant of the *obese* gene) and endocrine hormones (i.e., the concentration of leptin in the blood) ought to give pause to those who take for granted that cognition and reason dominate the human condition. To be sure, it is appropriate to take an individual's genetic makeup as exogenous and (given the limitations of current technology) immutable. But modern science tells us that the human genome is the product of an age-old evolutionary process, and–as a closer look at the evidence will demonstrate–the behavioral algorithms it encodes are both more flexible and more *in*flexible than might be predicted by a (biologically naïve) theory of rational choice.

The second reason economists should pay heed to the leptin example is that it suggests a very specific decision-theoretic interpretation. Evolutionary biology offers a simple explanation for obesity: the process of natural selection has endowed humans and other animals with the ability to store energy as body fat in order to survive periodic famines. Leptin is one component of the sophisticated molecular system that helps us solve this "energy homeostasis" problem, and its influence is subject both to our genetic endowment at birth and (among other things) the amount of body fat we have in storage at a given point in time. The message leptin conveys to the brain–evident, as noted above, in both its behavioral and physiological manifestations–facilitates the maintenance of energy homeostasis. In other words, the concentration of leptin flowing in my veins should be viewed, in the language of decision theory, as an *information state*. The *obese*-type mouse, in turn, could be thought of as having particularly strident *prior beliefs* about its caloric needs, beliefs that–in the absence of pharmaceutical intervention–remain unchanged throughout its life. This particular view of leptin–that is to say, this translation of an observable molecular signal into a theoretical

abstraction–is likely to find application in economics beyond the study of obesity.

In a widely read article penned more than a century ago, economist Thorstein Veblen admonished his profession for failing to incorporate the methods and findings of the natural sciences, and for focusing on description at the expense of questions about ultimate causation that naturally flow from an evolutionary perspective:

> ...economic science is living over again in its turn the experiences which the natural sciences passed through some time back. In the natural sciences the work of the taxonomist was and continues to be of great value, but the scientists grew restless under the régime of symmetry and system-making. They took to asking why, and so shifted their inquiries from the structure of the coral reefs to the structure and habits of life of the polyp that lives in and by them...All the talk about cytoplasm, centrosomes, and karyokinetic process means that the inquiry now looks consistently to the life process, and aims to explain it in terms of cumulative causation. (Veblen 1898, p. 384)

It surprising to note, given the dramatic advances in the biological sciences in the twentieth century, that economic science has for the most part ignored the advice of Professor Veblen (Zak and Denzau 2001).[1] Just as a better understanding of the polyp leads to a richer theory of the reef, this paper will attempt to translate a better understanding of the biological underpinnings of obesity and "overeating" into a richer economic theory of self-control.

A word on the organization of the paper is in order. Although–as will be argued below–economics offers a framework that is in many ways uniquely well-equipped to ask and answer questions about the determinants of obesity, the very nature of the question being asked herein precludes a narrowly focused review of the writings of economists as they relate to obesity and the psychology of self-control. Accordingly, after briefly reviewing the empirical obesity literature (Section 2.1) and the economic theory of self-control (Section 2.2), I venture forth into the realm of behavioral biology. The proper way to do this is to begin with a statement of the adaptive problem to which obesity is (or was) the solution (Section 2.3), after which I feel compelled to dispense with some common misconceptions about how the physical substrate of biological evolution (i.e., genes) can–and cannot–influence behavior (Section 2.4). Section 3 discusses how the particulars of behavioral biology translate into economic decision theory, and offers four specific conjectures about the incidence of obesity in human populations (supporting evidence for which is considered in Section 4).

Because evolutionary biology is largely (though not exclusively) an historical science, necessarily reliant on indirect evidence in the testing of alternative hypotheses, it is necessary (for credibility's sake) to cast a wide net when reviewing the relevant empirical evidence. The hypothesis of interest

[1] For notable exceptions, see Hirshleifer (1977, 1985), Rogers (1994), Bergstrom (1996), and Robson (2001).

here–that human obesity has its origins in prehistoric starvation risk–can be verified in a number of ways: by looking at the degree of food security in traditional subsistence economies (Sections 2.3 and 4.3.2); by examining the function of and variation in the many "obesity genes" known to modern science (Sections 2.4.2, 4.2, and 4.3.1); by identifying fattening strategies in non-human animals and looking for parallels in human behavior (Sections 4.1 and 4.3); and by demonstrating that the incidence of obesity varies with indicators of food insecurity (Sections 4.1 and 4.3). Moreover, the empirical relevance of the molecular underpinnings of obesity is underscored both by the growing body of research pointing to a little-known group of viruses as a causal factor in some cases of obesity (Section 4.4.1) and by the surprising psychosocial determinants of self-medication with drugs that induce weight loss (Section 4.4.2). Section 5 concludes.

2 Background

2.1 Obesity Research

Behavioral scientists of all stripes study obesity. Historically, overeating has been viewed as a personality disorder, a symptom of "weakness of will," and the response of the medical community to the afflicted was limited to formulaic lifestyle advice ("Exercise more!"; "Eat less!"; etc.) or referral to psychoanalysis, neither of which has proven particularly effective at producing thinner patients (Goodrick and Foreyt 1991, Brownell and Rodin 1994). As it has become clear that obesity is consistently associated with a host of health problems–including hypertension, diabetes, heart disease, and cancer–and that certain demographic groups (e.g., the poor) are at higher risk, obesity has increasingly been viewed as a public health problem. Epidemiologists studying the phenomenon have typically utilized prospective studies or large-sample surveys, mining the data for "risk factors" (usually chosen from the popular explanations for obesity: TV, sedentia, fatty foods, etc.) (e.g., Dietz and Gortmaker 1984, Ching *et al.* 1994). The ultimate aim of these studies is to produce an effective treatment: whatever culprit they point to–be it high-fructose corn syrup or hours spent in front of the television–once the "cause" of obesity in the target group is identified, a corresponding anti-obesity message can then be disseminated by medical practitioners and public health officials.

Perhaps not surprisingly–given the historical emphasis of the science of epidemiology on communicable disease–epidemiological studies of obesity have paid little attention to *economic* determinants. While demographic marker variables such as income, education, or race are often included in such analyses, the effect of relative *prices*–of calorie-dense foods, for example–have for the most part been neglected by the public health community. This fact has not gone unnoticed by economists, and a small literature on the subject has recently developed. Cutler, Glaeser, and Shapiro

(2003), for example, reviewed survey data monitoring changes in the incidence of obesity over time and concluded that in particular, decreases in the *time cost* of food preparation seem to be driving recent increases in the prevalence of obesity; Chou, Grossman, and Saffer (2004), on the other hand, have found support for the hypothesis that both the price and availability of fast food and increases in the price of tobacco have resulted in much of the observed gains in weight; while Lakdawalla and Philipson (2002) argue that the transition from active to sedentary employment has had a large effect. It is important to note that while data limitations naturally preclude the simultaneous estimation of *every* possible determinant of a phenomenon as complex and multifactorial as human obesity, economics provides a theoretical framework broad enough to encompass the many so-called risk factors from epidemiology, and adds important insights by suggesting interactions between these factors and such conventional economic measures as the wage rate or the price of butter (Cawley 2004).[2]

But when the discussion shifts from descriptive analysis to the implications for public policy, it quickly becomes clear that this is a case in which fundamental tenets of welfare economics are being put to the test. After all, if consumers are freely choosing to eat more (or exercise less) because of technological advances that make calories more affordable (or exercise less affordable), who are we to presume that government intervention is necessary? The usual sources of market failure (incomplete markets arising from high transaction costs or asymmetric information) don't seem to apply here. It could be argued, of course, that the average consumer lacks sufficient knowledge of nutrition science, or that the moral hazard induced by health insurance results in a socially inefficient disregard for the future health consequences of gluttony. But many consumers profess awareness of the health consequences of overeating and overeat nonetheless; and the health consequences of being overweight can take decades to develop, so the present discounted value of any distortion would presumably be small. This would seem to place economic theory squarely in opposition to the consensus view of the public health community that obesity is a problem in need of a solution. It has therefore been argued that the strongest *economic* justification for policy intervention in the realm of obesity comes, oddly enough, from *psychology*.[3]

[2]Unfortunately, for the most part economic studies of obesity published to date have failed to seriously consider the possibility that changes in the *quality* of diet (i.e., we're all eating more "fattening" foods) could be driving the increase in obesity. Although the widely accepted popular view that dietary quality is an important determinant of body weight has increasing support in the biomedical literature, the subject is–alas–also beyond the scope of this paper, and will be left for future research. For a discussion of the endogeneity of dietary preferences and an exposition of the equilibrium selection problem posed by dietary choice, see Smith (2004b).

[3]See, for instance, Cutler *et al.* (2003) or Camerer *et al.* (2003).

2.2 Economics and Self-Control

Many economic models of self-control have been proposed, and most emphasize the dynamic nature of the self-control problem. In the earliest complete exposition of the problem, Robert Strotz (1956) hypothesized that consumers might discount future consumption not only according to the calendar date at which consumption is to occur, but also according to the instantaneous distance of the future date from the present.[4] 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. The consumer, for example, might make a plan to place 10% of his paycheck in a retirement account, starting next year. But if his preferences are dynamically inconsistent, he might–even in the absence of new information–change his plan come New Year's Day, and decide to 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.

This problem might, of course, be anticipated by the individual making the original plan, and Strotz and others have suggested that if it is possible to pre-commit future behavior, then individuals might willingly incur a cost in the present in order to restrict the set of choices available in the future. Indeed, most testable hypotheses stemming from dynamic models of self-control are related to pre-commitment.[5] Gul and Pesendorfer (2001) make use of this fact, capturing the self-control phenomenon with a model in which the preference domain includes not only the objects of choice but also the choice sets from which objects are selected. Their model allows, as do models of dynamic inconsistency, for the possibility that utility might be increased by pre-committing to a strictly smaller choice set.

The economic 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 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. 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

[4] More recent studies of this phenomenon include Thaler and Shefrin (1981), Ainslie (1991), Laibson (1997), Sozou (1998), and Gul and Pesendorfer (2001).

[5] 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 patterns of consumer behavior consistent with a lack of self-control (Shefrin and Thaler 1988, Laibson 1997, Levin 1998).

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 (at least for this exceedingly simple choice set) that no reasonable person would deny them, and the prescribed outcome (consistent behavior that varies 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 expanding the domain of preferences to include the presence of the additional temptation in the freezer, most people are likely to insist that it is still the *outcomes*–ice cream and waistline–that they really care about.[6] So the axiomatic approach loses much of its appeal when the self-control problem is admitted.

The lack of a compelling normative foundation has not precluded the development of an economic theory of self-control, and it has enjoyed considerable success. But by adopting a strictly positivist approach–i.e., making note of the odd behavior and positing odd preferences in order to accommodate it–the obvious question of *why* people might behave this way, or *why* they might have such preferences, goes unanswered. The remainder of this paper is an attempt to explain–in light of our rapidly advancing knowledge of the behavioral biology of obesity–why this discrepancy between long-term goals and short-term behavior has arisen, and to demonstrate how the answer to this etiological question can find application in economic analysis.

2.3 Evolution of Thrifty Genes

It was noted in the opening paragraphs of this paper that when viewed through the lens of evolutionary biology, body fat serves a vital function: it helps foraging animals solve the *energy homeostasis* problem[7] that arises when the food supply is subject to disruption. Evolution in the abstract is an

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

[7] *Homeostasis* is the ability or tendency of higher animals to maintain relatively stable internal physiological conditions under fluctuating environmental conditions. Among the processes under homeostatic control that depend critically on a reliable supply of caloric energy are body temperature, the beating of the heart, respiration (breathing), and the flow of glucose (blood sugar) to the brain. In the event of a temporary disturbance of any one of these processes, death can come quickly. For this reason, our bodies come equipped with the ability to consume themselves during periods of fasting–first by utilizing glycogen stores in liver and muscle tissue, then by catabolizing body (depot) fat and finally by breaking down proteins in the peripheral musculature. While stored glycogen and skeletal muscle have alternative functions (e.g., short-term energy homeostasis and locomotion, respectively), depot fat is of little use

optimization process, the objective being the maximization of "Darwinian fitness" or–to give but one definition–the expected number of progeny surviving in the long run. In applying this abstraction to the real world, practitioners of behavioral ecology[8] typically identify an easily measurable *proximate currency*–a proxy for fitness–such as the probability of surviving the winter, or the amount of time devoted to search, or the ratio of energy gained to energy spent.[9] The use of proximate currencies is what brings behavioral ecology into the realm of economic consumer theory: just as the economist posits that consumers maximize some function of the choice set, the behavioral ecologist posits that foraging animals maximize some function of the proximate currency. Proximate currencies relevant to the storage of body fat might include, for example, the likelihood of starvation, the deleterious health effects of obesity, or the effect of excess fat stores on the aerodynamics of predator evasion (Kullberg 1998). If we are to work from the hypothesis that *people* living in the modern world bear the mark of an evolutionary past in which caloric deficiency was a significant problem, it seems reasonable to ask whether the available evidence supports this notion.

For starters, there can be little doubt that peoples solely reliant on foraging and subsistence agriculture–i.e., the lifestyle presumably representative of the human condition for most of our collective evolutionary history–were faced with an uncertain food supply. The most authoritative study on this subject was conducted by Marjorie Whiting in 1958 . Whiting's broad survey of 118 traditional societies representing all the major cultural and geographic areas of the world revealed that *every single group* studied reported periodic shortages of food: in 29 percent, shortages were rare (every ten to fifteen years); in 25 percent, occasional (every two to three years); in 23 percent annual ("a few weeks preceding harvest, anticipated and expected, recognized as temporary"); and in 23 percent, more than once a year. In general frequent shortages were mild and occasional ones more severe. In 29 percent of these cultures, shortages were reportedly severe ("comparable to a famine, deaths occur...many persons desperate for food, emergency foods are exhausted"); in 34 percent, shortages were moderate ("real suffering and deprivation, a few persons are hungry and incapacitated, weight loss may be considerable, food stores exhausted, emergency foods sought"); and in 37 percent shortages were mild ("fewer meals per day than usual...less activity, no great hardship experience, people may lose weight, food stores are used"). While the sample of peoples still reliant on foraging and subsistence agriculture as late as 1958 might not be fully representative of Paleolithic conditions–such cultures might be expected, for example, to have been relegated to the least productive regions of the planet in the face of the advance of industrial agriculture–the uniformity of their experience is impressive.

(possibly excepting its role as an insulator in cold climates) other than as a store of emergency energy.

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

[9]See, e.g., Krebs and Davies (1997).

Suppose it is true that food insecurity was an important part of life for prehistoric humans. Why should this have anything to do with the behavior of consumers today, when death by starvation is exceedingly rare? If we are to take seriously the notion that knowledge of human evolutionary history can inform the study of human behavior in the 21st century, it is important to be explicit about the mechanism by which evolution proceeds. While it is possible to apply evolutionary theory to the cultural transmission of knowledge and societal norms of behavior (Richerson and Boyd 2005), the persistence, universality, and heritability of many human behaviors can only be explained by transmission at the level of the gene.

2.4 Genes and Behavior

2.4.1 Heritability and Genes

The proposition that modern human populations are genetically maladapted to the modern diet and lifestyle was first set forth by human geneticist James Neel in 1962. Neel observed that the metabolic disorder known as diabetes (now recognized as a complex of disorders linked by the shared symptoms arising from elevated blood sugar) might have served a useful purpose in an environment in which food was scarce. Individuals with a quick "insulin trigger," Neel argued, would be much more efficient at harvesting caloric energy when it was available, giving them an advantage during famines that might–on balance–offset the handicap of diabetic symptoms during times of plenty. Support for this argument can be found in the observation that while many diabetics suffer from a number of detrimental health problems, symptoms decline or disappear during periods of starvation–as they did, for instance, under the food rationing imposed during the 1870-71 siege of Paris (Zimmet 1997). Neel's "thrifty genotype" hypothesis has gained growing acceptance among the scientific community as supporting evidence accumulates (Neel 1999).

Obesity has long been known (as is now widely accepted) to have a strong "genetic component". The influence of genes on body mass index[10] can be inferred from the comparison of siblings raised together with siblings raised apart, of monozygotic (identical) with dizygotic twins, and of adopted with biological parent-offspring pairs. Numerous 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 differences), which range from 33% to 90%. The robustness of the finding that genes have a discernible (and sometimes dominant) influence on the likelihood of becoming obese makes it hard to deny that genes play a role in this particular consumer decision problem.[11] But what does it mean to call a trait "genetic"? To be more precise, in what sense can human decisions about diet

[10] Body mass index (BMI), the anthropometric measure of choice among obesity researchers, is calculated by dividing an individual's weight in kilograms by the square of his height in meters.

[11] For reviews, see Barsh *et al.* 2000 and Bouchard *et al.* 1998.

and exercise be *genetic*? At least two common misconceptions typically arise in discussions of the relationship between genes and behavior; each merits a few words here.

In the popular lexicon, calling a trait "genetic" implies that it is inherent, hardwired, predetermined–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). Consider, for example, the sunflower. Suppose I take a sunflower seed, place it in soil and provide it with water, sunshine, and fertilizer, and maintain the local ambient temperature within a specified range. Suppose further that as a result of my actions, a fully grown sunflower plant develops in the spot where I planted the seed. Clearly, if any one of the environmental parameters necessary for growth had been deficient, the plant would have failed to prosper. And just as clearly, without the intricate developmental program encoded in the sunflower genome, my unassuming little sunflower seed would never have become a sunflower. So is the mature plant the result of genes or of environment? Clearly, the answer is both. This is not to discount the importance or legitimacy of "heritability" measures. Given a large *population* of sunflowers of varying size, the question of the source of the variation–genetic or environmental–is perfectly legitimate, and perfectly amenable to inquiry by the scientific method.

This logic, of course, applies equally well to human obesity. Like other complex behavioral traits, obesity is often described as an instance of *heritable susceptibility*, which only manifests itself when the right conditions prevail. Indeed, the heritability estimates discussed above are in some sense just a measure of the cultural homogeneity of the population from which the sample is drawn: a culturally homogeneous but genetically heterogeneous sample would presumably yield a heritability estimate of 100%.

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 seek out and consume food after periods of fasting is *universal*. Similarly, the genes that govern the growth and development of five fingers on each hand, or two eyes, or two lungs, are universal. Some people might have smaller hands than others, or poorer eyesight, or a bigger lung capacity, and the source of the variation (genes or environment) can be debated. But in many cases the variation will be unimportant or due entirely to environmental factors (e.g., nutrition during development, years spent reading books in poor light, time devoted to physical exercise).

2.4.2 Molecules of Hunger

As the example of leptin and the *obese* gene illustrates, we now know much more about the role of genes in regulating behavior than can be inferred from heritability studies. By all accounts, the discovery of leptin in 1994 stimulated a flurry of research into the biochemistry and genetics of mammalian feeding behavior[12], so much that an "obesity gene 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, and some 90% of genes in mice have homologous[13] 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).

As of October 2004 the obesity gene map included more than 400 genes associated with feeding behavior in humans (Perusse *et al.* 2005). Nearly all of these genes are associated with hormone-like molecules that, like leptin, are thought to communicate information about the nutritional needs of the body. The secretion of the leptin molecule into the bloodstream, for example, is ascribed to a gene located on chromosome 7 that is expressed primarily 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 primarily 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)—secreted by the small intestine after a large meal–and its receptor in the brainstem; and a number of neurotransmitters found primarily in the brain (and apparently specific to the regulation of bodily energy) with names like neuropeptide Y, melanocyte-stimulating hormone, and thyrotopin-releasing hormone (Stryer 1981, Woods *et al.* 1998, Schwartz *et al.* 2000, McMinn *et al.* 2000, Halford *et al.* 2004).

There are a number of ways in which variations in genes might "calibrate" the molecular machin-

[12]Interest was no doubt stimulated in part by Amgen Inc.'s well-publicized $20 million purchase of the commercial drug development rights to leptin in May 1995 (Chicurel 2000). 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, implying a resistance to leptin's effects much like the resistance to insulin that characterizes type 2 diabetes (Considine *et al.* 1996).

[13]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.

ery that regulates energy homeostasis. Section 1 described the *obese* mutation, which completely inactivates the leptin signal and generates morbid obesity; but it is possible that other, milder mutations in the genes for ligands (hormone-like signals) or their receptor systems might simply reduce the amount of time the ligand remains bound to the receptor, or reduce the metabolic half-life of the ligand (i.e., reduce the amount of time the ligand remains in the bloodstream before being reabsorbed or decomposing to waste products; leptin, for example, has a much longer half-life than insulin). Other factors affecting the "strength of the signal" are the number of ligands secreted in response to a given stimulus, the number of receptors expressed in the hypothalamus or elsewhere, and the extent and nature of neural connectivity to the rest of the brain. Although the particulars of these molecular processes are not yet fully understood, each is certainly mediated by genes, and thus a potential way in which genetic variation might serve to calibrate the physiological and behavioral maintenance of energy homeostasis (see, e.g., Pinker 1997, Gazzaniga *et al.* 1998, Marcus 2004).

Before the "modern synthesis" of evolutionary biology pioneered by Ronald Fisher, J.B.S. Haldane, and Sewall Wright in the 1930s, biologists had difficulty reconciling Mendelian genetics (which takes genes as discrete units passed from parent to offspring) with the apparently continuous variation of certain heritable physical traits (height, weight, beak size, etc.) observed in real-world populations.[14] The resolution of this paradox came from the following insight: continuously varying characteristics are typically under the control of genes at *many loci* (i.e., locations in the genome), each of which has only a small effect on the observed outcome. So if there are some 400 genes controlling body mass index in a given human population, any given individual would be born with some combination of "thinness" and "fatness" variants of these genes, and a relatively smooth distribution of BMIs would be observed in the adult population. The polygenic control of quantitative traits is one of the mechanisms by which genetic variation is maintained in populations: even during times when, for instance, thinness is maladaptive, genes for thinness might nevertheless survive in some individuals for many generations, thanks to the counteracting effects of genes that promote fatness. Genetic variation is, of course, one of the prerequisites for the mechanism of natural selection, and under the influence of selective pressures on the trait in question, the dynamics of this process are not difficult to envision: in the presence of conditions (e.g., frequent famines) that favor survival and reproduction by only the fattest of individuals, we would expect the frequency of genes that promote a "thrifty" metabolism to increase over time.

2.4.3 The Fixed Nature of Genetic Traits

To most people "evolution" implies a dynamic, ever-changing process. But if the evolutionary process in question is one of biological evolution, in which genes encoded in DNA are altered by the

[14] For an accessible history and exposition of the modern synthesis, see Boyd and Silk (2002).

process of natural selection, evolution can be exceedingly slow. Modern science tells us that for an individual human being, DNA can be modified only at birth, and then only by rare mutations and the process of recombination (e.g., Wessels and Hopson 1988). It is possible that, at the population level, certain genes might increase (or decrease) in frequency over a period of decades, but such changes are most pronounced in small or decreasing populations, and in the presence of strong selective pressure.

Because of this constraint on the dynamics of genetic evolution, it is unlikely that gene frequencies observed in the modern human population would reflect the contemporary security of the food supply. Not only has the global population been increasing for thousands of years (Harpending *et al.* 1998), the risk of death from starvation has only been eliminated in the last century or two (and even today, only in wealthy nations).[15] While it is possible (and indeed evident, as discussed in Section 4.2 below) that small, isolated human populations might have experienced measurable changes in the frequency of obesity genes in modern times, it seems unlikely, for example, that there could have been significant changes in global gene frequencies in the few decades in which the modern obesity epidemic has occurred.

3 Subjective Decision Theory and Implicit Beliefs

> 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)

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

[15] Archaeological evidence tells us that humans began the transition from foraging to subsistence agriculture around 10,000 years ago (see, e.g., Boyd and Silk 2002), and this may well have represented an increase in food security. But small-scale agriculture in the absence of inter-regional trade was still subject to devastating climatic fluctuations, and historical records bear this out. Inhabitants of Western Europe, for example, were subjected to widespread famine as recently as 1849, when the Irish Potato Famine resulted in more than a million deaths (approximately 12% of the population); and this was not an anomaly of history: the population had previously survived famine events in 1816-18 (death rates rose by 50%), 1740, 1693-94 (10% of Louis XIV's subjects died in France, and as much as one-third of the population in other regions), and 1315-21 (the so-called Great Famine, which resulted in the death of as much as 10% of the population) (Fagan 2000). Even in the wealthiest nations today, the realities of geography and climate result in seasonal variation in the price and availability of fresh produce–and hence nutrition–so perhaps we still cannot call our own food supply "constant."

combination of the two. Savage's theory is liberating for behavioral scientists because it allows for the analysis of behavior (including the inference of a subject's "beliefs" about the world) without reference to the goings-on in the "black box" of the mind.

But the goings-on in that black box are gradually being illuminated by the findings of modern neuroscience, and one recurrent finding is that conscious experience is a poor guide to human motivation.[16] In particular, we know that much of the regulation of energy homeostasis takes place below the level of consciousness, with, for instance, metabolic rate and body temperature playing central roles in the determination of fat stores (Rising *et al.* 1992, Zurlo *et al.* 1992, Spraul *et al.* 1993).

Given variation (at the population level) in the genes conveying a propensity to become obese, together with what is known about the adaptive problem these genes seem designed to solve, it would seem that there is a simple decision-theoretic interpretation: each of us is born with our own unique set of prior beliefs about starvation risk.[17] Indeed, upon reflection, the preference relation described by Savage seems an apt description of the molecular system of genes and hormones that influence human behavior. The evolutionary process that generated the human genome may well be driven by statistical links between causes and effects[18], but the "feelings" (e.g., hunger, satiety, satisfaction, outrage) that guide our actions need not come packaged with explicit, conscious knowledge of likely future consequences. Our emotions help us make decisions in an uncertain world, and for most of our evolutionary history the "information" they provided served us well.[19]

[16] Among the more notorious of these findings is the work of Michael Gazzaniga on split-brain patients. He argues convincingly that the behavior of these subjects in controlled experiments suggests that certain brain structures actively "rationalize" behavior, essentially concocting a coherent explanation for one's actions *after* they take place (Gazzaniga 2000).

[17] Two interesting corrollaries to this view of a molecular basis for subjective "beliefs" governed by polygenic inheritance (discussed in Section 2.4.2 above) are that i) for any given level of background risk, much of the population will either over- or under-estimate that risk, and ii) beliefs can evolve beyond the support of past environments. Consider, for example the extremes in size acheived by the selective artificial breeding of dogs. Though DNA analysis has confirmed that all dogs are descended from wolves, no wolf observed in nature is as big as a Great Dane nor as small as a Pekinese. This is because body size is a trait influenced by many genes, and though the various "smallness genes" that make a Pekinese might all be present in any given population of wolves, they are presumably infrequent enough in nature that no individual wolf would ever be so unlucky as to inherit them all from his parents. Only with strong selective pressure (induced either by a human handler who repeatedly mates the smallest of wolf-pairs generation after generation, or by conditions in nature that favor small stature in the game of survival and reproduction) can such a transformation occur (Smith 1998).

[18] This is not to say that environmental variables are not guided in general by *deterministic* processes. Indeed, it has been argued that when formal models specify stochastic processes, they necessarily omit more explicit causative factors (Horan 1994, Huffaker 1998).

[19] The role of emotions in strategic behavior has been emphasized by Hirshleifer (1987) and Frank (1988). Just as the "emotional states" of hunger and satiety (it is argued here) help consumers solve the energy homeostasis problem, these authors emphasize the value of emotional states such as rage, jealousy, and vengeance in helping people solve

Of course, in a modern world in which industrialized agriculture and international trade have effectively eliminated (for most of us) the periodic famines that characterized most of human evolutionary history, our genes no longer serve the adaptive purpose they once did. What we are left with is the psychology of self-control. While it is possible, as noted above, to model self-control as the product of a declining rate of time preference, or as an intertemporal game between "multiple selves" or as the manifestation of a preference domain that includes "temptation," a more parsimonious explanation is suggested by the confluence of behavioral biology and subjective decision theory: when we overeat, we are behaving *as if* a famine were imminent; it should not be surprising, given the mismatch between modern technology and our ancient genes, that we find ourselves perpetually regretting our indulgences. Moreover, our knowledge of the differences between modern and pre-industrial environments can provide a basis for making *ex ante* predictions about the conditions most likely to induce self-control problems. In what follows, I will argue that many of the particulars of the incidence of obesity are consistent with this hypothesis. In particular, I will consider the following conjectures:[20]

Conjecture 1 *Individuals faced with a higher objective risk of food shortage are more likely, ceteris paribus, to be obese.*

Conjecture 2 *Genetically isolated sub-populations subject to higher historical famine risk will, ceteris paribus, exhibit higher rates of obesity.*

Conjecture 3 *Environmental cues that would have reliably predicted food shortages in the past may trigger fattening in modern environments, even if the association no longer holds.*

Conjecture 4 *The internal "information state" with respect to starvation risk is subject to intervention at the molecular level (e.g., by pharmaceuticals). The level of body fat can be expected to vary accordingly.*

The next section reviews supporting evidence for each of these propositions.

bargaining problems.

[20] A disclaimer: my exclusive focus on the role of information and uncertainty in proposing an endogenous psychology of overeating should not be taken to imply that deterministic material constraints are unimportant. Rather, my intention is to point out that optimal (or sub-optimal) fattening strategies are likely to be a function not only of relative prices, but also of a variety of social and economic factors that are commonly omitted in conventional economic analyses.

4 Obesity and Subjective Risk: Evidence

4.1 Food Insecurity

"Does hunger cause obesity?" William Dietz posed this seemingly paradoxical question in a 1995 case study of an obese 7-year-old girl. An interview with the girl's mother revealed that the family experienced periods of hunger each month during the days just before the welfare check arrived. Dietz, noting that artificial feast-famine cycles can induce fattening in laboratory rats (Szepesi *et al.* 1975 , Levin 1994, Kochan *et al.* 1997), hypothesized that the girl's obesity might be an adaptive response to periodic food shortages. Although death by starvation is a rare event in the U.S., a surprisingly large proportion of households experience periodic food shortages. According to a recent USDA report, 11.2 percent of U.S. households were "food insecure" in 2003, where food insecurity is measured by survey responses indicating the extent to which resource constraints caused hunger (Nord *et al.* 2004). Dietz's phenomenon has subsequently been noted in large-sample food insecurity surveys: food insecurity is in some cases associated with an increased risk of obesity.[21] The effect is strongest among mothers experiencing mild to moderate food insecurity (many of whom reportedly forgo an occasional meal in order to ensure that her children can eat), and the effect remains after controlling for income and other demographic variables.[22] Although the association of food insecurity with obesity has indeed been labeled a "paradox" by the economists and epidemiologists studying the problem, it is very much in accordance with the view that obesity is a natural phenomenon likely to be exacerbated by an increased risk of food shortages (Olson 1999, Townsend *et al.* 2001, Basiotis and Lino 2002, Adams *et al.* 2003 , Olson and Strawderman 2004).

4.2 Island Populations

As noted in Section 2.4.3 above, the rapid rise in obesity in the United States in the last few decades has happened much too quickly to have been driven by changes in the genetic makeup of the population. But there have been documented cases of small, isolated populations experiencing catastrophic famine, and the survivors and their descendants typically show a markedly increased susceptibility to obesity and type 2 diabetes when food is once again plentiful. Perhaps the most

[21] There is also evidence from laboratory studies that the thermic effect of food (i.e., the increase in core body temperature that typically follows a meal) is diminished in humans when the timing of meals is irregular (Farshchi *et al.* 2004). This is consistent with an adaptive response to uncertainty–that is, it appears that our bodies adapt to irregular feedings by conserving the energy necessary to generate the thermic effect. For a review of the literature on adaptive thermogenesis in humans, see van Baak (2004).

[22] Interestingly, although an association of food stamp program participation with obesity has been reported (Gibson 2003), it has been suggested that when food insecurity is controlled for, program participation can actually *decrease* the risk of becoming obese (Jones *et al.* 2003).

striking (and well-documented) example of such a population is found in the inhabitants of the Micronesian island of Nauru. The traditional lifestyle on isolated Nauru, dating back to prehistoric times, was based on agriculture and fishing, and was subject to frequent episodes of starvation due to droughts and the island's poor soil. During World War II, Japanese forces occupied the island, reduced food rations to half-a-pound of pumpkin per day, then deported most of the population to Truk, where half of them died from starvation. When the survivors returned, they benefited from a wealth of phosphate royalties and adopted a sedentary lifestyle and Western-style diet of calorie-rich processed foods (Rubinstein and Zimmet 1993). By 1990 some 40% of the adult population on Nauru suffered from diabetes, the highest prevalence in recorded history (Zimmet 1997).

The experience of Nauruans is not unique: other examples of populations in which a sudden transition to the Western lifestyle has induced a sudden (within two decades) increase in the prevalence of diabetes include the Pima Indians in the U.S., the Wanigela of Papua New Guinea, the Yemenite Jews airlifted to Israel, Japanese emigrants in the U.S., groups of Asian Indians in Fiji, Mauritius, Singapore, Tanzania, the U.S., and Britain, and Chinese emigrants in Hong Kong, Mauritius, Singapore, and Taiwan. Indeed, Jared Diamond has argued (from an impressive base of empirical support) that the sole exception to this pattern of susceptibility to environmentally-induced type 2 diabetes are peoples of European descent (Diamond 2003). Diamond hypothesizes that one explanation for European "resistance" to this particular disease might be that–because the transition to a high-calorie, low-exercise lifestyle occurred in Europe gradually over the course of several centuries–individuals who were genetically susceptible to type 2 diabetes might have been at a reproductive disadvantage before the advent of modern medicine. Indeed, Diamond notes, even with all the advantages of modern medicine the prevalence of diabetes in Nauru has actually *decreased* in the past decade, possibly because (as has been documented) the most severe cases resulted in death before reproductive age.

The distributional effects of the genetic variation in susceptibility to obesity and type 2 diabetes are hard to ignore. The ancestry of Mexican-Americans, for example, is on average around 35% indigenous, and that of African-Americans averages 80% African, possibly explaining–at least in part–the repeated finding that these ethnic groups are at higher risk for obesity, even after controlling for observable demographic factors (Hanis *et al.* 1991, Collins-Schramm *et al.* 2002).[23] It is also consistent with reports that, for example, Polynesians and African-Americans have lower resting metabolic rates (as measured by oxygen consumption) than European-Americans, and that Pima Indians have a lower core body temperature during sleep than their Caucasian counterparts (Rising

[23]More direct evidence for the relationship between obesity and ancestry comes from Grandinetti *et al.* (1999), who show (in a sample of 567 Native Hawaiians) an association between percentage of indigenous ancestry and body weight.

et al. 1995 , Rush *et al.* 1997, Forman *et al.* 1998). It is still likely, of course, that some part of the variation in obesity commonly attributed to ethnicity in analyses of large-sample survey data is due not to genetic variation but rather to unobserved socioeconomic factors (such as those discussed in Section 4.1 above and Section 4.3.2 below) distributed inequitably across ethnic groups. But the fact that members of some groups are evidently born with an elevated susceptibility to obesity is likely to raise issues of social justice when implementing (or failing to implement) obesity-related programs aimed at improving the health of the populace.

4.3 Environmental Cues

A strong argument can be made that humans and other animals avoid both poisoning and micronutrient deficiency by making use of environmental signals or cues when choosing particular foods. Sugar, for example, is found in nature only in ripe fruit, raw honey, and mother's milk, all perfectly reliable sources of valuable nutrition; while bitter and sour substances are often neurotoxins or signs of spoilage. So it should not be surprising that humans and other foraging animals are endowed with specific receptors for these substances on the tongue, as well as with innate tendencies to seek out the former and avoid the latter. But more than a millennium after the advent of refined sugar, we still respond to these cues even in the face of mounting evidence that doing so can be damaging to our health.[24]

This section will consider two instances of environmental cues that appear to trigger fattening in some humans. In each case it is apparent that while the cue was likely a strong signal of starvation risk in ancestral environments, the strength of the association is greatly diminished in the modern world.

4.3.1 Seasonality, SAD, and Siberian Hamsters

It is common in natural environments to observe seasonal patterns in the amount of body fat held in reserve by foraging animals (e.g., Butler and Woakes 2001, Schleucher 2004). These patterns, when observed in the field, tend to reflect local variations in the availability of food: animals generally fatten during times of relative plenty and shed fat during times of relative scarcity. While this observation may seem perfectly intuitive and unsurprising, it does not immediately explain how animals "know" when a food shortage is imminent. It might be, for example, that food availability is the only cue needed to trigger a fattening episode; on the other hand, a given species might rely on more informative environmental cues (such as, for example, population density, ambient

[24]For a more complete exposition of this phenomenon, see Smith (2004b). For a survey of the neurobiological underpinnings of dietary choice and an application to the economic theory of habit formation, see Smith and Tasnadi (2003).

temperature, or recent weather events) that signal *future* scarcity. 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 the most important determinant of seasonal 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 at the summer solstice. The most thoroughly studied rodent model of photoperiodic regulation is the Siberian hamster, which has the demonstrated ability to regulate its body mass progressively and continually according to its photoperiodic history–independently of the amount of food provided in the lab. Further investigation has shown the pineal hormone melatonin plays an important role in photoperiodic regulation of body fat in mammals (Mercer *et al.* 2000).

There is intriguing evidence that our own species also utilizes photoperiod as a seasonal fattening cue. The condition known as *seasonal affective disorder* (SAD) is characterized in humans by depression, hypersomnia (excessive sleeping), hyperphagia (excessive eating), and weight gain. Seasonal affective disorder derives its name from the fact that it typically occurs in winter, and heritability studies have shown that incidence can be at least partly explained by genetic variation. The most effective (and most commonly prescribed) treatment for seasonal affective disorder is prolonged daily exposure to intense artificial light. At the opposite end of the seasonal spectrum, there is another condition known as *summer depression*–also demonstrably heritable–the victims of which tend to suffer from insomnia, decreased appetite, and weight loss (Allen *et al.* 1993, Madden *et al.* 1996).

There is also evidence of a seasonal trend in weight gain among the general population: according to one recent study, the average adult American gains one pound between September and January, which is partially offset by an ensuing loss between January and March (Yanovski *et al.* 2000).[25] The conventional wisdom, of course, is that seasonal variation in diet and opportunities for exercise are responsible for the "holiday weight gain" phenomenon, but this notion has yet to be confirmed in controlled studies. The dependable coincidence of rich holiday desserts and cozy firesides with the winter solstice makes it difficult to draw conclusions about the relationship between photoperiod and body fat based on data collected from human populations, but the parallels to animal seasonality and the light-sensitivity of seasonal affective disorder are suggestive.

[25]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; Dzien and Dzien-Bischinger (2003) report an annual change of 0.8 BMI units in both men and women in Austria, with peak BMI occurring in early spring; Dietz and Gortmaker (1984) report seasonal effects on childhood obesity.

4.3.2 Income Effects, Willow Tits, and Psychosocial Determinants of Obesity

The willow tit is a small insectivorous bird who spends his days flitting about the canopies of Northern European forests, and his propensity to become entangled in strategically placed netting has been a boon for field biologists. Among the topics addressed by tit ecologists are the relationship between food security and overwinter fattening. In the course of these studies, a peculiar phenomenon was noted. At any given field station, the local dominance hierarchy quickly becomes apparent: in any pairwise matching the dominant tit gets first access to the best foraging sites, while the subordinate tit must expend additional effort to obtain a given caloric reward, often at increased risk of predation. But here is the strange part: subordinates, in spite of their restricted access to food, tend to have *more* body fat than dominants (Ekman and Lilliendahl 1993).[26]

Clark and Ekman (1995) offer a theoretical explanation for this observation: in a dynamic programming framework in which the objective is the minimization of the probability of starvation, it can be demonstrated (for parameterizations approximating conditions observed in natural settings) that a dominant will choose to store less body fat than his subordinate counterpart because–by virtue of his dominant status–he has at his disposal a relatively reliable supply of food. Interestingly, Clark and Ekman also show (1995, Fig. 1a) that the dominance rank–body fat relationship is *reversed* when food is particularly scarce. That is, when times are tough, the dominant faces an increased threat of food shortage–causing fattening to become more attractive–while the subordinate would like to fatten but cannot obtain enough to eat.

This last prediction–which has been confirmed in subsequent field studies (Brodin and Lundborg 2003)–is so interesting because of its striking parallel with the incidence of obesity in humans. As noted above, low-income Americans reporting food insecurity are at increased risk of obesity, even after controlling for income. Perhaps not surprisingly, this risk appears to go away when poverty and food insecurity become more severe: there simply isn't enough food in the best of times to enable the extreme poor to become obese (Townsend *et al.* 2001). The same pattern is seen in international data relating income to obesity: in the industrialized countries, the poor tend to be fat while the rich are thin; but in the third world the opposite is true (Monteiro *et al.* 2004). This is not to say that poor people in wealthy societies consciously contemplate the possibility of starvation. Rather, if the metaphor of the willow tits proves apt, we would expect that certain endocrine signals are triggered by the state of being poor, which in turn trigger the physiological and behavioral effects that result in obesity. Individuals with low social status or lacking a robust social support network might be more likely, for example, to exhibit elevated levels of "stress hormones" such as cortisol

[26] A similar phenomenon has been noted in nonhuman primates: Shively and Wallace (2001), for instance, have reported an association between low social rank and abdominal obesity in field studies of cynomolgus monkeys.

and decreased levels of the neurotransmitter serotonin.[27] This altered blood chemistry might then trigger increases in body fat independently of any real or perceived threat of starvation.

Could it be that observed variation in modern obesity by income or social status is the manifestation of a human psychology generated in a (distant past) environment in which food security was a function of social relationships? Serious consideration of this hypothesis should properly begin by asking whether food security *was*, in fact, a function of social relationships in human evolutionary history. While field studies of birds and monkeys are suggestive, social behavior is one realm in which there are few good substitutes for our own species. Though studies of body fat among modern hunter-gatherers are rare, anthropological data have much to say about social aspects of food security. Among nomadic foragers dependent on hunting such as the Aché of Paraguay, for example, individual families compensate for the high daily variability in hunting success by sharing meat whenever it becomes available. That variability motivates sharing is evidenced not only by the dearth of sharing among sedentary tribes that can more easily store food (Binford 1978, Gould 1981), but also by examination of which foods get shared the most: the more variable the food type, the more it is shared across family groups (Binford 1978, Kaplan and Hill 1985, Cashdan 1989). Although there are many reports of egalitarian distribution of food in groups where sharing is the norm, a closer examination shows that all is not equal: group members are reportedly acutely aware of the identity of the most productive hunters, and the best hunters typically have more offspring, though the mechanism is not known (E.A. Smith 2004). The question of whether the best hunters are in fact protected from the threat of starvation by virtue of their prestige and the informal debt owed them by their peers cannot easily be answered by studies of extant foraging groups, many of which are somewhat protected by support (e.g., from missionaries) from the developed world. But it seems reasonable to presume an individual living in the ancestral human environment held in high esteem by his peers would have had an advantage during extreme famine events, by virtue of the facts that i) he would likely be a skilled provider of foodstuffs in his own right, ii) he would be well-positioned to draw on support from peers (if any were in a position to help) and iii) he would likely have (if he lived in an environment in which storage of food or assets was possible) significant food stores or other forms of wealth that could be traded for food.

The picture painted by these anthropological studies suggests a number of likely correlates of individual food security in human evolutionary history: productive ability, a strong social support

[27] 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" that "might lead to the development of abdominal obesity." For a discussion of serotonin's evolutionary origins and role in the regulation of body fat, see Section 4.4.2 below.

network, and stored food or assets (which might have included, for example, livestock or land rights). These are perhaps not so different from the determinants of individual economic security in the modern world, such as education, a dependable job, family support, and financial assets. And though I know of no systematic study of the empirical relationship between obesity and economic security, a number of investigators (often in pursuit of a more general relationship between health and "stress" or "psychosocial" factors) have stumbled across just such a relationship:

- Morris *et al.* (1992) studied a sample of 6,057 British men who had been continuously employed for five years prior to an initial interview. Five years later, the 1,645 men who had *subsequently experienced some non-employment* had gained a significantly greater percentage of body weight (7.5%, vs. 5.0% for those who had maintained continuous employment).

- A 7-year prospective study of 438 male firefighters found that those who reported *worrying about financial security* gained, on average, four pounds more than non-worriers (Gerace and George 1996).

- It has long been known that body mass index is a decreasing function of *education*–an effect whose reported magnitude is often larger than that of income (e.g., van Lenthe *et al.* 2000).

- Impulsive dietary behavior is sometimes said to be "stress-induced" (Greeno and Wing 1994). Laitinen *et al.* (2002) use data from a longitudinal, population-based study in Northern Finland to examine this phenomenon more closely. In a sample of 2,359 31-year-old men, the best predictors of (self-reported) stress-related eating included being *single or divorced,* having a long *history of unemployment,* and having a *low level of occupational education.* Among the 2,791 women in the cohort, the only statistically significant predictor of stress-related eating was "a *lack of emotional support.*"

- Zagorsky (2004) used fifteen years (1985-2000) of data from the National Longitudinal Survey of Youth to examine the relationship between body mass index and *net worth*, and found that–holding income and other demographic variables constant–a one-point increase in body mass index (e.g., an increase of approximately seven pounds for a 6-foot, 200-lb. male) corresponded to a $1,000 decrease in net worth. Interestingly, a closer look at the data led the author to conclude that *inheritances* appeared to be driving this relationship.

- Emdad *et al.* (1998) surveyed a sample of 69 professional drivers and reported that one of the primary correlates of body mass index was "*availability of social attachment* outside work," where a lack of social attachment was associated with a high BMI.

- Hannerz *et al.* (2004) found in a 5-year prospective study of 1,980 Danish males that *job insecurity* significantly increased the likelihood of weight gain during the study period.

- It has long been known that male preferences for female body weight follow a consistent cross-cultural pattern: in cultures with scarce resources, heavier women are preferred; while in cultures with abundant resources, thinner women are preferred (Furnham and Baguma 1994). In a study of 1,176 U.S. undergraduate students, Nelson and Morrison (2005) confirmed this relationship at the individual level: on average, men who expressed dissatisfaction with their own *financial situation* or who *felt hungry* at the time of the interview preferred heavier women than men who felt financially secure or who had a full stomach.

4.4 Molecular Interventions

The fourth (and last) conjecture offered in Section 3 above might seem an odd one: I suggested there that external manipulation of a consumer's blood chemistry can be usefully viewed as altering his subjective "beliefs" about the world. In this section I will argue that such hypothesizing is useful not only because it provides a more richly descriptive model of obesity and than might be provided by a conventional rational choice framework, but also because investigation of the molecular underpinnings of a behavioral phenomenon can provide important insights into the behavior in question. In what follows I begin by reviewing the (indisputable) evidence that viral infections can cause obesity in animals and also the (suggestive) evidence that they can (and do) do the same thing to people. I then proceed to the question of self-medication: the manipulation of body weight via the administration of pharmaceutical substances. Rather than providing a review of the latest weight-loss drugs or an economic analysis of the market for such drugs, I focus instead on two related classes of drugs and their neurological targets in the brain. This narrow focus facilitates a discussion of the evolutionary origins of the neurological systems that regulate weight gain, which in turn sheds light on the psychosocial conditions (seemingly unrelated to weight gain) that induce consumers to choose these drugs. Ultimately, this approach not only provides surprising corroboration of the discussion in Section 4.3.2, but also speaks directly to issues of causation in both drug use and weight gain.

4.4.1 Obesity as Infectious Disease

Though epidemiologists have in recent years begun to move away from their traditional narrow focus on infectious agents, expanding their models to include social and economic factors long the exclusive domain of the social sciences, there has been much less attention paid by social scientists to the impact of infectious disease on social and economic behavior. In particular, it might seem unlikely that the choices of apparently healthy individuals (making apparently rational economic decisions about diet and exercise) could be influenced by an unknown or inactive (past) viral infection. But this is exactly what is suggested by an intriguing but little-noted series of studies by

Nikhil Dhurandhar and colleagues. Beginning from the observation that viral infections can cause fattening in chickens (Dhurandhar *et al.* 1990, 1992), Dhurandhar has subsequently extended his results to mice and non-human primates (Dhurandhar *et al.* 2000, 2002). Though the mechanism of action is unknown, some obesity viruses are known to cause lesions in the hypothalamus (the brain region responsible for maintenance of energy homeostasis in animals) and the damage appears in many cases to be permanent: that is, even when the animal recovers from the viral infection, the obesity remains (Bernard *et al.* 1999). There are now at least seven viruses known to cause obesity in animals, two of which have been implicated in human obesity (Dhurandhar 2004). The first, known as the SMAM-1 avian adenovirus[28], is thought to be responsible for an epidemic that killed thousands of poultry in India in the early 1980s. Because experimental infection of human subjects with such a virus would be ethically impermissible, Dhurandhar *et al.* (1997) instead collected blood samples from 52 obese subjects and tested for antibodies to SMAM-1, an indicator of previous infection. The antibody-positive group (10 subjects) had significantly greater body weight and body mass index compared to the antibody-negative group, and blood chemistry (abnormally low serum cholesterol and triglycerides) similar to that found in animals known to be infected with SMAM-1.

The second virus implicated in human obesity is known as Adenovirus-36 (Ad-36) and was first identified in 1978 in the feces of a 6-year-old girl suffering from diabetes and enteritis (Wigand *et al.* 1980). Though Ad-36 is a human virus, it has now been shown to induce fattening in animals under experimental conditions (Dhurandhar and Atkinson 1996, Dhurandhar *et al.* 2000, 2002), and in a sample of 502 obese and non-obese human subjects, significantly more of the obese (30%) than the non-obese (11%) tested positive for Ad-36 antibodies, and again blood chemistry followed the pattern seen in animal studies. In addition, 89 pairs of twins were assayed for Ad-36 antibodies; among twin pairs in which only one twin tested positive for Ad-36, the Ad-36 positive twins had significantly higher body mass index and percent body fat (Atkinson *et al.* 2005).

It is important to note that the human studies cited here are limited in scope and subject to the usual disclaimer that correlation does not imply causation. On the other hand, the parallels with the outcomes of controlled animal experiments are highly suggestive, and further attention to the role of infectious agents in the etiology of obesity seems warranted.

4.4.2 Self-Medication

Can a diet pill be viewed, like the length of the day or the threat of job loss, as altering one's internal subjective assessment of starvation risk? Unfortunately, for such speculation to be a useful

[28] Adenoviruses are a family of DNA viruses shaped like a 20-sided polyhedron and known to cause conjunctivitis and upper respiratory tract infections in humans.

exercise requires one to know both the biochemical mechanisms by which a weight-loss drug works and the adaptive function (i.e., the evolutionary origins) of the biochemical mechanisms in question. While in many instances a rudimentary understanding of the molecular action of pharmaceuticals is available, the question of the naturalistic adaptive function of specific endocrine signals has been mostly neglected by the biomedical community. Nevertheless, in light of the evidence discussed above, it seems reasonable to ask whether a naturalistic perspective might lend insight into the use of behavior-altering drugs. Two well-known examples of substances known to induce weight loss will be discussed here: serotonin and nicotine.

Serotonin The mammalian brain processes information via a combination of chemical and electrical processes. The chemical messengers it relies upon to activate patterns of electrical activity in neural networks are known as neurotransmitters. The three primary neurotransmitters in the human brain are dopamine, norepinephrine, and serotonin, all members of the class of compounds known as monoamines. Though each is found throughout the brain, individual nerve cells (neurons) tend to specialize: hence the terms dopaminergic system, serotonergic system, etc. when referring to particular networks of neurons. In addition to the modulation of mood, emotion, and sleep, the serotonergic system has long been known to play a central role in the regulation of body weight (Blundell 1977). While much of our knowledge of the role serotonin plays in appetite regulation is due to animal experiments (for instance: the report by Steffens *et al.* [1997] that the direct injection of serotonin into the brains of pigeons dramatically reduces food intake and increases time to initiation of feeding), we are also finding that many of today's most effective weight loss drugs act primarily on the serotonergic system in the human brain (Halford *et al.* 2005).

It was noted in Section 4.3.1 above that seasonal mood disorders can play a role in weight gain and weight loss. In a review of the most rigorous clinical and community studies published in the past four decades, McElroy *et al.* (2004) find a more general relationship between "mental health" and body weight: major depression is associated with weight gain in women, children and adolescents; patients with bipolar disorder tend to have elevated rates of overweight, obesity, and abdominal obesity; obese persons seeking weight-loss treatment tend to have elevated rates of depressive and bipolar disorders; and abdominal obesity is associated with depressive symptoms in both men and women.[29] Moreover, the authors note the strong parallels in the phenomenology, comorbidity, family

[29]It is important to note that "mood disorders" vary widely, and some are known to cause weight *loss* rather than weight gain. For example, a distinction is sometimes made in the psychology literature between "typical" and "atypical" depression. The latter (which is in fact quite common, especially in women) is diagnosed in part by a patient's report of recent weight gain. Similarly, the physical location of fat deposition (e.g., around the abdomen) can vary considerably, and might well be a function of the psychosocial causes of the condition. Unfortunately, standardized diagnostic methods often pay little attention to such subtleties (American Psychiatric Association 2000).

history, biology, and pharmacologic treatment responses between mood disorders and obesity.

Since the introduction of fluoxetine (a.k.a. Prozac) in the United States in 1988, the use of prescription antidepressant medication has skyrocketed. According to data collected in the 1999-2000 wave of the National Health and Nutrition Examination Survey, 7 percent of U.S. adults reported using one or more of these medicines within the past month–up from 3 percent in the 1988-94 survey period. During this period, antidepressant use among adults in all age groups doubled or tripled, with prevalence in one group (women between the ages of 18 and 44) reaching 13 percent. Of these drugs, 62% of those reported in the most recent survey fall in the class known as *selective serotonin reuptake inhibitors* (SSRI's), and the majority of the others (monoamine oxidase inhibitors, tricyclics, etc.) are thought to work–as are the SSRI's–at least in part by increasing the amount of available serotonin in the brain (Schloss and Williams 1998, National Center for Health Statistics 2004).

It should not be surprising, given the many commonalities between mood disorders and obesity, that Prozac causes weight loss, or that at least one SSRI (sibutramine) is now being marketed explicitly as a weight loss drug, or that other weight loss drugs (e.g., d-fenfluramine) target the serotonergic system, or that synthetic compounds that mimic serotonin (e.g., mCPP and 5-HTP) have been shown to induce weight loss in obese patients (Halford *et al.* 2005). But what are we to make of this serotonin connection? If this multi-faceted serotonergic system in our brains is the product of biological evolution, what could its original purpose have been?

There is an expansive literature describing the role played by serotonin in animal behavior. Most notorious among these studies are the many experimental treatments of nonhuman primates, in which social rank is readily determined through observation of stable dominance relationships.[30] A recurrent finding in these experiments is that serotonin levels are an increasing function of social rank: when an individual, for example, is elevated to dominant (e.g., "alpha-male" or "alpha-female") status, his or her serotonin levels shoot up; and not until that same individual is demoted (usually by conflict) to his or her previous subordinate status do serotonin levels return to normal (Raleigh *et al.* 1991). Serotonin might therefore be viewed as an internal indicator of "social success," a conclusion underscored by the finding that an abnormally low level of serotonin by-products in the cerebrospinal fluid of humans is a strong predictor of suicidal behavior (Asberg and Forslund 2000).[31]

[30]See, for example, McGuire *et al.* (1984) for a review of some early findings relating serotonin to social rank in captive colonies of vervet monkeys.

[31]Though suicide would seem counterproductive for an individual seeking to maximize his own chances of survival and reproduction, biologists have suggested a resolution to this paradox by noting that natural selection would favor individuals whose actions take into account the well-being of (genetically related) family members: in particular, if the (Darwinian) benefits that my death would bring to my family exceed the (Darwinian) costs to me, I should–by

The sensitivity of the serotonergic system to the subtleties of the social environment has been demonstrated again and again: Krotewicz and Romaniuk (1995) show that patterns of serotonergic activation in cats vary predictably according to the presence or absence of a second cat and the dominance rank (dominant or subordinate) in a given cat pair; McKittrick *et al.* (1995) show that upon establishment of a stable dominance hierarchy in a rat colony, binding to serotonin receptors is reduced among the (severely stressed) subordinate members; Majercsik *et al.* (2003) show that efficacy of buspirone (an anti-anxiety drug that works by stimulating certain serotonin receptors) is a function of individual vs. group housing in male rats and mice, and of self-reported "social support from non-relatives" in male humans; and Haller *et al.* (2004) review evidence that SSRI's are more effective among patients who report maltreatment in childhood.

In a provocative article, Deakin (2003) explores the puzzle posed by the fact that two very different psychiatric disorders–depression and antisocial personality disorder–are both characterized by impaired serotonin functioning. Deakin hypothesizes the disorders stem from the selective impairment of two groups of serotonergic neurons that "mediate adaptive responses to future and current adversity." In this view, serotonergic projections from one brain region (the dorsal raphe nucleus) "...oppose the action of dopamine and mediate avoidance of threats," while serotonergic projections from another region (the median raphe nucleus) "...suppress memory and awareness of current and past adversity." Impairment of the former results in "impulsivity and drug addiction," (i.e., anti-social personality disorder) whereas impairment of the latter results in "low mood, low self-esteem, hopelessness and pessimism" (i.e., depression). While Deakin acknowledges that this interpretation of the evidence is somewhat speculative, he also reports that modern imaging techniques are providing startling corroboration.

It would seem, given the evidence from these many disparate human and animal studies, that the answer to the question of the "original purpose" of serotonin in the human brain is related to the discussion of social dominance and food security discussed in Section 4.3.2 above. In particular, it appears that one general function of serotonin in human central nervous systems is to mediate behavioral responses to one's social environment. And if the parable of the willow tits holds true, it would make sense that we'll put on the pounds when we live in a world without a "safety net," and shed them when we are fortunate enough to find ourselves surrounded by a dependable network of friends and family. Serotonin makes this happen.

Nicotine Tobacco smoking has long been used as one way of maintaining a lower body weight (Li *et al.* 2003). One empirical implication of this observation is that tobacco and body fat should meet the economic definition of "substitute goods," as confirmed in the recent work of Chou, Grossman,

this logic–choose death (Decatanzaro 1980). In this view, the link between low serotonin and suicide fits neatly with the interpretation of serotonin levels as a measure of "social success."

and Saffer (2004).[32] But if tobacco–or more precisely, tobacco's "active ingredient," nicotine–is viewed as a drug that alters the consumer's internal "information state," a much richer picture of the relationship between smoking and obesity emerges.

When nicotine enters the bloodstream, it binds to nicotinic receptors in the brain, augmenting the release of serotonin and other neurotransmitters (Quattrocki *et al.* 2000). Given the relationship between nicotine and body fat, it seems reasonable to ask whether the complex relationships between tobacco use and the socioeconomic environment might be partially explained by correlates of economic *uncertainty*. That is to say, if nicotine's neurobiological effects are to be interpreted as increasing the smoker's subjective perception of economic security, we might expect that a consumer exposed to threats to his economic well-being will be more likely to take up smoking. Although–like the relationship between body fat and economic security reviewed in Section 4.3.2–this hypothesis has apparently never been investigated directly, a number of authors have reported suggestive findings:

- In a longitudinal study of the effects of changes in marital status on health-related behavior in a sample of 80,944 women, Lee *et al.* (2005) found that among non-smokers and past smokers, those who had *divorced* or been *widowed* during the 4-year study period had more than a twofold increased risk of relapsing or starting smoking than women who stayed married.

- A number of reports in the medical literature (reviewed in Quattrocki *et al.* 2000) indicate that *antidepressants* improve the success of smoking cessation programs.

- Hammarstrom and Janlert (1994) found in a 5-year longitudinal study of 1,080 graduating secondary school students in northern Sweden that–after controlling for demographic factors and prior smoking habits–those who had experienced an extended period of *unemployment* were significantly more likely to start smoking.

- Siahpush (2004) shows in cross-sectional data from the Australian National Health Survey and National Drug Strategy Survey that even after controlling for factors such as socioeconomic status, mental health, proportion of friends who smoke, and age of smoking initiation, the odds of being a smoker among *single mothers* were still twice those of mothers with partners.

- LaRosa *et al.* (2004) report the results of a survey of 2,315 French adults that collected information about demographic and socioeconomic factors as well as social and family environment. In a comparison of smokers to non-smokers, they found that "smokers were characterized by a way of life marked by *financial problems*, *reduced social contacts*, and a higher frequency, before

[32] But see also Gruber and Frakes (2005).

the age of 18, of *divorce or separation of the parents*, *frequent parental quarrels*, or *separation from the parents*."

- Kirby (2002), in an analysis of a nationally representative sample of American adolescents interviewed at two points in time, finds that *parental separation* increases the likelihood that adolescents will start smoking.

- One of the most consistent findings of research on smoking cessation therapy is that *social support* is a critical determinant of success (Fiore *et al.* 2000).

- Tsutsumi *et al.* (1998) use a survey of 597 Japanese adults to examine the relationship between health-related behaviors, social support, and attitudes towards one's community. They report, among other things, a negative association between smoking and *family support*.

- In an investigation of the impact of social relationships on health-damaging behaviors, Broman (1993) finds that those describing themselves as "*spouse*," "*friend*," or "*organization member*" smoke less.

- In a review of the public health literature, Wilson and Walker (1993) note that smoking is often *increased after the onset of unemployment*.

- In a random household survey of 1,137 African-American adults in California, Romano *et al.* (1991) found that women with *poor social networks* were more likely to smoke.

- Isohanni *et al.* (1991) report the results of an investigation of the social and family determinants of smoking in a cohort of 12,058 teenage subjects in northern Finland. They find an elevated risk of smoking in those with *one or more parents absent* for at least part of the child's upbringing, among adolescents who had experienced the *death of their father or divorce of their parents*, and among girls who had experienced the *death of their mother*.

- McChargue *et al.* (2004), using survey responses from a sample of 208 undergraduate smokers, report that "*the inability to bond with peers*" appears to promote nicotine dependence among female students.

- In studies of the function of the nicotinic receptor in non-human animals, an impact on "social" functioning has been noted. For example, it is known that social recognition in rats and sheep are mediated by nicotinic receptors (van Kampen *et al.* 2004, Ferreira *et al.* 2003), as is socially-induced stress in mice and rats (Bugajski *et al.* 2002, Salas *et al.* 2003).

Another drug known to act at least in part via both the nicotinic receptor and the serotonergic system is 3,4-methylenedioxymethamphetamine, popularly known as "ecstasy" or MDMA (Salzmann

et al. 2004, Herin *et al.* 2005). Originally intended to be a weight loss drug, MDMA (first synthesized by Merck Pharmaceuticals in 1912) was never marketed because of its adverse side effects. MDMA induces weight loss via both physiological (hyperthermia) and behavioral (peripheral hyperactivity, loss of appetite) effects, while also inducing such side effects as feelings of "euphoria and empathy" as well as of "extreme comfort, belonging, and closeness to others," which can result in "inappropriate and/or unintended emotional bonding." Though declared a Schedule I controlled substance by the U.S. Drug Enforcement Agency in 1985, ecstasy has become increasingly popular among U.S. teens and young adults (5%–7% of high school seniors surveyed in 2003 reported use within the past year; 60% said they could easily obtain the drug from illicit sources), reportedly in part due to its ability to enable partygoers to dance all night (National Institute on Drug Abuse 2004). The powerful anorectic activity of MDMA, together with the specificity of its psychotropic effects would seem to bolster the hypothetical naturalistic role of serotonergic and nicotinic receptors in regulating stores of body fat in inverse proportion to the strength of an individual's network of social support.

5 Conclusions

5.1 Rational Overeating

> All circumstances increasing the probability of the provision we make for futurity being enjoyed by ourselves or others, also tend to give strength to the effective desire of accumulation. Thus a healthy climate, or occupation, by increasing the probability of life, has a tendency to add to this desire. When engaged in safe occupations, and living in healthy countries, men are much more apt to be frugal, than in unhealthy, or hazardous occupations, and in climates pernicious to human life. Sailors and soldiers are prodigals. In the West Indies, New Orleans, and the East Indies, the expenditure of the inhabitants is profuse. The same people, coming to reside in the healthy parts of Europe, and not getting into the vortex of extravagant fashion, live economically. Wars and pestilence, have always waste and luxury, among the other evils that follow in their train. (Rae 1834, p. 123)

Is overeating a rational act? In October 2001, four weeks after terrorists destroyed the World Trade Center towers in New York City, *The Wall Street Journal* reported that Manhattanites were responding to the sudden loss of perceived security by–you guessed it–gaining weight.[33] In one

[33]Unfortunately, the phenomenon was not limited to Manhattan: a spokeswoman for Jenny Craig Inc. (a weight-loss company with 655 locations world-wide) reported a "sharp wave" of cancellations after the attacks, and in an August 2002 survey commissioned by the American Institute for Cancer Research, some 9% of Americans reported having

sense, the evidence reviewed herein suggests that individuals who overeat in response to such events are *more* rational (i.e., their behavior is *more* sensitive to variation in material constraints) than many economic theorists have given them credit for: by responding not only to contemporary price and income levels, but also to indicators of future scarcity, many "at risk" consumers would appear to be following something like optimal fattening strategies when they ingest and retain more calories than are needed to cover short-term energy expenditures.

But on the other hand–given the demonstrable reliability of the modern food supply–many people evidently choose obesity in the absence of any appreciable starvation risk. Are these consumers "rational"? The answer to this question, whether derived from the perspective of economic theory or from that of behavioral biology, would seem to be "it depends." In economics, the appropriate test is that of *time consistency*: the consumer's choices are presumed to maximize some function of his present and future well being, as long as the psychology of self-control (i.e., a non-stationary intertemporal utility function) doesn't get in his way. This is, of course, equivalent to the test suggested by biology: individuals whose excessive weight can, in light of our collective natural history, be attributed to demonstrably false subjective beliefs about food security, can also be expected to make choices that fail to maximize personal well being, in a manner that is observationally equivalent to time inconsistency.

One advantage of the naturalistic perspective is that it generates precise *ex ante* predictions about when to expect self-control problems to arise. An individual who becomes obese because his genes (being implicitly calibrated to a high level of background risk) predispose him to a high body mass index is unlikely (in the absence of education in matters relating to the impact of lifestyle on health) to make choices that maximize his long-term well being. The same could be said of an individual who becomes obese in response to environmental cues (e.g., the length of the day) that no longer predict scarcity. But this perspective also makes clear that systematic lapses in self-control are by no means prerequisite to becoming obese: cases of obesity attributable to changes in relative prices that have lowered the cost of getting fat may well be consistent with an optimal fattening strategy even when the probability of a food shortage approaches (but does not reach) zero.

5.2 Toward a Unified Theory of the Reef

As noted above, it is necessary when engaging in evolutionary theorizing about economic behavior to cast a wide evidentiary net. This should be viewed not as a burden but as an opportunity. The increasing accessibility of the many disciplines of behavioral biology makes it possible not just to reconcile economic models of optimizing behavior with the realities of human psychology, but

gained weight in the wake of September 11th (Barnes and Petersen 2001, American Institute for Cancer Research 2002). Interestingly, smoking and antidepressant use also increased (Vlahov *et al.* 2004, Boscarino *et al.* 2004).

it also opens the door to confirmation (or refutation) by evidence from a wide variety of scientific literatures: given a hypothesis about the natural history of a given behavioral phenomenon (e.g., obesity is a response to starvation risk), testable implications immediately follow for anthropology (modern hunter-gatherers should face the adaptive problem in question), for animal behavior (species faced with the same adaptive problem in the wild should respond accordingly), for heritability (the behavior should be heritable even if specific genes are unknown; genetically isolated sub-groups should vary in a manner consistent with the governing hypothesis), for neurobiology (the controlling ligands should have "side effects" consistent with their putative informational role), and even for the etiology of drug use (it should be consistent with the putative adaptive function of the governing neuroendocrine system).

Perhaps more importantly, the example of obesity serves to illustrate how an empirically grounded biological foundation for a theory of economic behavior can yield benefits both in descriptive power and in its ability to speak to issues of causation. To the uninformed observer, it might not be obvious that our feelings of hunger and lack of self-control at the dinner table are the product of some prehistoric famine, or that our metabolisms regulate themselves up and down in response to indicators of food scarcity. But these insights have surprisingly strong empirical support, and there is no *a priori* reason to think that traditional economic explanations (e.g., relative prices) will prove more important in explaining the incidence of obesity than the more "psychological" explanations (e.g., financial insecurity) inspired by a naturalistic perspective.[34]

Just as Veblen's industrious reef scientists expanded their line of inquiry to include multiple levels of causation, from the structure of coral reefs to the "structure and habits of life of the polyp that lives in and by them" to the cellular and molecular processes that underlie the life process, it is now–more than ever–possible to envision a similar expansion of economic science. As it becomes increasingly feasible to measure not just an individual's demographic background but also his genetic makeup, endocrine state, and even brain activity[35], and to organize and interpret these disparate measures with the principles of and evidence from behavioral biology, an increasingly fruitful science of economic behavior is certain to emerge.

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[34]For a preliminary answer to this question, see Smith, Stoddard, and Barnes (2005).

[35]See Camerer, Loewenstein, and Prelec (2005) for a review of the current state of brain imaging technology and its potential uses in the study of economic behavior.

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