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Economic Stressors and the Demand for "Fattening" Foods

By

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Economic Stressors and the Demand for "Fattening" Foods

The dramatic rise in obesity seen in the U.S. over the past few decades has spawned a vigorous body of economic research into possible causes (Finkelstein, Ruhm, and Kosa 2005). There are many candidate explanations for the secular trend: technological change in the workplace (Lakdawalla and Philipson 2009), female labor force participation (Scholder 2008), availability of convenience foods (Cutler, Glaeser, and Shapiro 2003; Chou, Grossman, and Saffer 2004), lower relative prices of energy-dense foods (Cawley 2010), urban sprawl (Ewing et al. 2003), and a move toward sedentary leisure activities (French, Story, and Jeffery 2001). Many of these putative causes of the obesity epidemic can be circumstantially ruled out because the timing of the trends do not match up well with changes in obesity rates, which have risen most dramatically since 1980. Notable exceptions include the increase (since 1980) in the number of fast food and full-service restaurants, along with the simultaneous increase in carbohydrate consumption (mostly in the form of grains, french fries, and sweetened beverages) (Finkelstein, Ruhm, and Kosa 2005). These trends, of course, are market outcomes themselves, and could be driven by either supply-side factors (e.g., technological or labor market changes that affect the cost of food production) or by demand-side factors (e.g., changes in the opportunity cost of time); the evidence here is limited, though some have argued that the latter seems more likely (e.g., Cutler, Glaeser, and Shapiro 2003; Chou, Grossman, and Saffer 2004).

This article proposes an alternative demand-side driver of obesity that is consistent with larger trends. I will argue that the upward trend in economic insecurity faced by

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households in the U.S. since 1980 could plausibly have caused much of the concurrent increase in obesity, and that greater consumer demand for "fattening" (especially carbohydrate-rich) foods would be consistent with this view. Because evidence on changes in economic insecurity and the link between economic insecurity and obesity have been reviewed elsewhere (Hacker 2006; Smith 2009; Wisman and Capehart 2010), only brief overviews are provided below; the proposed link to carbohydrate demand is novel and the focus of this article.

Economic Insecurity and Obesity

The notion that economic insecurity--defined roughly as idiosyncratic risk to financial well-being--might be an important demand-side determinant of obesity draws inspiration from "optimal fattening" theories in behavioral ecology (Smith 2009). Just as foraging animals everywhere exhibit tendencies to fatten as a hedge against starvation, evolutionary history is likely to have similarly linked the physiological and behavioral mechanisms governing human body weight with environmental indicators of impending food shortage. This is consistent, for example, with evidence suggesting that weight gain increases with the probability of job or income loss (Smith, Stoddard, and Barnes 2009), and that food insecurity is sometimes associated with obesity (Gundersen et al. 2008), Gundersen and Kreider 2009).

Changes in economic insecurity over time are difficult to measure. Consider, for example, employment security. Official statistics regarding unemployment rates are easy to obtain, but say little about the multitude of factors any given worker would need to consider when assessing the likelihood of job loss. These might include the status of the

local or national economy, but also the stability and prospects for the industry or sector in which one works, length of employment, and even the nature of personal interactions with supervisors and co-workers.

A natural experiment stemming from the privatization of the UK's Property Services Agency (PSA) in the early 1990s provides a vivid illustration. As part of the ongoing Whitehall II study of British civil servants, PSA employees (along with other departments that were not privatized) were interviewed before, during, and after privatization. Because current employees were not guaranteed employment in the to-be-formed enterprise, one might imagine that the period of uncertainty surrounding this event might have been stressful for the workers involved, and that even those who ultimately stayed on had good reason to be concerned about employment security. This was reflected in the observed effect on both health-related behaviors and various measures of health, including body mass index (BMI): over the five years required to complete the privatization, male and female PSA employees gained an average of 0.3 and 0.6 BMI units, respectively, (approximately 2-4 pounds) relative to employees in other departments (Ferrie et al. 1998), and these effects were apparent even before privatization took place (Ferrie et al. 1995). Though the net effect of this action on total employment was negligible (and thus would not be evident in official unemployment statistics), the effects on the body weights of the affected individuals were pronounced.

There is, nevertheless, reason to believe that economic insecurity has increased in the U.S. since 1980. During this period, changes in federal law have caused both defined-benefit pensions and health insurance subscriptions to fall dramatically, trade

liberalization has made "outsourcing" a household word, and income instability has risen sharply (Hacker 2006, Gottschalk and Moffitt 2009).

Obesity as an Evolutionary Vestige

If obesity is the product of periodic food scarcity experienced over the course of human evolutionary history, we should expect body weight to be sensitive not only to real changes in material risk, but also to environmental cues that would have reliably predicted food scarcity in the pre-industrial world. The precise mechanisms by which this occurs, however, remain something of an open question. Fattening might be accomplished via an increase in appetite; or changes in mood or mental health that cause people to be less physically active; or even down-regulation of basal metabolism.

Disentangling the relationships between environmental cues (that trigger fattening) and the physiological and behavioral responses to particular cues is beyond the scope of this article. But the possible roles played by carbohydrate-rich foods in an "optimal fattening" framework are intriguing. First of all, our peculiar physiological reaction to carbohydrate (the "cellular starvation" response, described below) suggests that the ingestion of carbohydrate-rich foods is interpreted by our bodies as an indicator of food scarcity. This naturally leads one to ask why or how such a response might have evolved. This question, in turn, can be tentatively answered with suggestive evidence from anthropology (also reviewed below) that carbohydrate-dense, nutrient-poor foods were consumed primarily during times of extreme food scarcity in the pre-industrial world. So perhaps it makes sense that a carbohydrate-rich diet is, as some argue, uniquely fattening: our bodies interpret starchy (and, to a lesser extent, sweet or fatty) foods as indicative of

extreme food scarcity, because this is what such a diet would have indicated over the course of human evolutionary history.

The preceding discussion would seem to suggest a simple causal relationship between carbohydrate-rich foods and obesity: an exogenous increase in either the demand for (driven, for instance, by a public health campaign promoting low-fat diets) or in the supply of (driven, for instance, by crop-specific agricultural subsidies or production technologies) such foods should be expected to cause weight gain. But here, intriguingly, the theory (and the evidence) bends back on itself. There is also evidence (reviewed below) that a variety of weight-gain-inducing environmental cues or "stressors" appear to cause increases in appetite *specific to carbohydrate-rich foods*. It is as if, over the course of our evolutionary history, we first evolved the tendency to fatten when eating carbohydrate, then later made use of this tendency by adjusting dietary composition (toward "fattening" foods) when presented with threats to food security. Thus we arrive back at a demand-side explanation for the observed trends in both obesity and dietary composition, with exogenous increases in economic insecurity promoting weight gain via the stimulation of demand for carbohydrates.

Insulin and Cellular Starvation

Dietary carbohydrate has very specific short-term effects on physiology and behavior. The human digestive system takes dietary sugars and starches and chemically breaks them down into simple sugars (primarily glucose and fructose), which are released into the bloodstream. In response to increased blood glucose (known as the postprandial *glycemic effect*), the pancreas secretes insulin, which triggers a cascade of biochemical

and physiological reactions that have the collective effect bringing blood glucose back down to normal levels (Ludwig 2002). Curiously, at the cellular level the insulin response looks a lot like anticipated starvation: blood glucose is quickly taken up and sequestered by the liver and the skeletal muscles while the production and release of glucose by the liver is down-regulated, and--importantly--free fatty acids in the blood are rapidly taken up and stored as body fat. If the glucose shock is large enough (as occurs when easily digestible carbohydrate or "high-glycemic" foods are ingested), subjects may actually experience a blood sugar "crash" to *below* normal levels, along with consequent feelings of hunger and carbohydrate craving. These effects are not observed when experimental subjects are instead fed low-glycemic foods with comparable caloric content (Brand-Miller et al. 2002). Interestingly, some believe that pairing glucose-rich (including starchy) foods with foods containing fructose or fat may be uniquely fattening. The reason is that fructose is converted by the liver directly into fatty acid; when this occurs in the presence of elevated insulin levels, the caloric energy from fructose (or from dietary fat) will be shunted directly into storage as body fat (Shafrir 1991). This could explain, for example, why fast food meals and mass-market snack foods (both of which are typically high-glycemic in combination with either high-fructose or high-fat) seem to be uniquely efficacious at stimulating weight gain (Isganaitis and Lustig 2005).

Famine Foods in Natural History

Foods eaten primarily during times of famine or seasonal food shortage are known as "famine foods" or "fallback foods." The ability to survive on such foods is thought to have been an important limiting factor in primate evolution (Marshall and Wrangham

2007), including that of the modern human lineage (Laden and Wrangham 2005). Fallback foods are typically protein- and nutrient-poor and undesirable due to factors such as bitter taste (often indicative of toxicity) and the need for intensive processing prior to consumption; to the extent that digestible calories are present, they typically come in the form of starchy carbohydrate. For example, in a survey of North American hunter-gatherer cultures, Huss-Ashmore and Johnston (1995) list roots, leaves, fruits, bark, and seeds as famine foods; a list that is remarkably similar to that reported by Irvine (1952) in a survey of groups foraging on the West African savanna. Of these, tubers or "underground storage organs" are thought to have played a critical role in early human life on the savanna, as evidenced by their widespread availability, the co-existence in the archeological record of early human fossils with those of root-eating rodents, and certain aspects of early human dental morphology (Laden and Wrangham 2005). Seasonal changes in the quality and breadth of diet are common among modern hunter-gatherer groups. The Hadza of Tanzania, for example, subsist primarily on five categories of foods: tubers, berries, meat, baobab, and honey. The availability of foods the Hadza claim to prefer most (honey, meat, and berries) fluctuates greatly over the course of the year, forcing them to rely for extended periods on the least-preferred fallback food, the fibrous tuber (Marlowe and Berbesque 2009). Even groups that have adopted subsistence agriculture, such as the Nangodi tribes of the West African savanna, exhibit average annual fluctuations in body weight of up to 8% and similarly subsist almost entirely on starchy carbohydrate-rich foods (stored grain) during the "hungry season" between December and July (Hunter 1967).

Though most research on famine foods has emphasized starchy plant materials as the near-exclusive source of calories, it is tempting to conjecture that two other types of foods would have reliably predicted the "hungry season": ripe fruits and fatty meats. The former, after all, typically matures and sweetens only at the end of the growing season, while the latter--if found in the wild--would almost certainly come from a foraging animal preparing for winter. This could explain why (as explained in the previous section) the presence of fructose (typically found in fruit) and fats seem to exacerbate the fattening effect of glucose in the diet.

Given our collective natural history of seasonal scarcity and the apparently reliable coincidence of food scarcity with a predominance of nutrient-poor starchy foods (and to a lesser extent, sugars and fats) in the diet, it seems plausible that the physiological and cellular responses to glucose in the blood could be an evolved adaptation.

Stress and "Comfort Foods"

It has long been known that social stressors can stimulate appetite (Greeno and Wing 1994). There is also a growing body of evidence suggesting that stress increases the desire for specific classes of food: sweets and starches (i.e., carbohydrates) and, to a somewhat lesser extent, fats. Oliver and Wardle (1999), for instance, report that in a sample of college students, self-reported changes in diet during periods of stress included decreases in "healthy" categories such as fruit/vegetables and fish/meat, and increases in cakes/biscuits, sweets/chocolate, and savory snacks, with the strongest effects observed in the two sweet categories. Similarly, Laitinen, Ek, and Sovio (2002) surveyed more than 2,000 individuals in Northern Finland, and found that self-described "stress-driven

eaters" report consuming greater proportions of chocolate, hamburgers/pizza, and sausages than those who reported no effect of stress on diet. In a laboratory setting, Epel et al. (2001) used a physiological measure of stress reactivity (cortisol levels before and after completing a series of stressful tasks), and allowed subjects to choose from four snack items (categorized as sweet-fat, sweet-lowfat, salty-fat, salty-lowfat) on separate days. On the day in which subjects were required to complete stressful tasks, the high-reactors preferentially snacked on the two sweet items, and ingested more calories than on the control day.

An enhanced preference for "fattening" foods is consistent with an optimal fattening perspective only if the preference is coincident with a situation in which fattening is in some sense optimal. Among socially foraging animals (including, in evolutionary history, humans), the presence of a "social stressor" such as those applied in the aforementioned laboratory studies might well constitute a threat to food security. But there are other situations that could be viewed as conducive to fattening in this sense, and that have been similarly shown to induce changes in diet. Three such cases are discussed below.

Seasonal Variation in Food Cravings

For many foraging animals, one of the best predictors of food scarcity in the wild is season. Our human ancestors almost certainly experienced seasonal food shortages with some regularity, and today even people living in the wealthiest corners of the modern world gain weight in the winter months, only to lose it in early spring (Yanovski et al. 2000). Individuals for whom this effect is most pronounced typically suffer from a host of other symptoms (including depression or dysphoria, decreased energy, fatigue, social

withdrawal, increased appetite, and hypersomnia), and are said to suffer from s*easonal affective disorder* (SAD) (Mercer, Adam, and Morgan 2000). Like the stress-induced appetite disturbances discussed above, patients suffering from seasonal affective disorder often report carbohydrate cravings (Wurtman et al. [1989], for example, describe as typical the case of a SAD patient who confesses to a weakness for bread and pasta), which can reportedly be ameliorated (along with the other symptoms of SAD) with prolonged exposure to artificial light (O'Rourke et al. 1989). Seasonal affective disorder is thought to be mediated by the serotonergic system in the brain (Wurtman and Wurtman 1989), and administration of the selective serotonin reuptake inhibitor *d-fenfluramine* has been shown (in a double-blind, placebo-controlled laboratory study) to be effective at eliminating the symptoms of SAD, including weight gain and carbohydrate craving (O'Rourke et al. 1989).

Food Cravings and the Menstrual Cycle

Given the centrality of reproductive success in the Darwinian calculus, it should not be surprising that dietary behavior also varies across the menstrual cycle in accordance with nutritional needs. Women typically gain weight throughout the cycle, then lose it during menstruation. Though this change is partly attributable to water retention, it also partly represents an increase in energy stores, and likely would have had adaptive value (over the course of human evolutionary history) as a hedge against the energetic requirements of pregnancy (Vieira 2009). The most extreme cyclical variation in body weight is typically accompanied by depressive symptoms, and is known as *premenstrual syndrome* PMS) or *premenstrual dysphoric disorder* (PMDD). In a laboratory study of 19 PMS

patients and 9 non-PMS controls tested during both the early follicular phase (immediately after menstruation) and the late luteal phase (just before menstruation), the patients suffering from PMS exhibited higher caloric intake (when offered snacks in the laboratory) and preferentially chose high-carbohydrate snacks (cookies, candy) over high-protein alternatives (Wurtman et al. 1989). Similarly, Yen et al. (2010) interviewed 60 PMDD patients and 59 controls during both the follicular and luteal phases, and asked a detailed series of questions about dietary cravings at each interview. Reported food categories included salted-fat, sweet-fat, carbohydrate/starch, and fast food; all of these exhibited small craving effects by both menstrual phase and by PMDD diagnosis (i.e., more frequent cravings in luteal phase and PMDD subjects), but the effects were strongest for sweet-fat foods. Again, this is a phenomenon that seems to be mediated by the serotonergic system in the brain, as evidenced by the work of Brzezinski et al. (1990), who showed (in a double-blind, placebo-controlled laboratory study) that administration of *d*-fenfluramine can suppress luteal phase appetite and reduce luteal phase depression scores in patients suffering from PMS.

Nicotine Withdrawal and Food Cravings

A final situation in which food cravings and weight gain seem to reflect an "optimal fattening" strategy in evolutionary history is smoking cessation. The putative pathway in this case is somewhat less direct: tobacco smoking is a relatively recent innovation in the history of our species, so it probably represents an "evolutionary mismatch" (i.e., a maladaptive behavior made possible by modern technology) rather than a behavior with significant adaptive value in human behavioral ecology. But nicotine is known to activate

the serotonergic system in the human brain, and there is evidence that some people may smoke as a "self-medicating" defense against economic insecurity (Barnes and Smith 2009). This could explain why smoking cessation is reliably accompanied by weight gain and carbohydrate cravings (Wurtman and Wurtman 1995). In a study by Spring et al. (1991), for instance, 31 obese female smokers were treated with either *d*-fenfluramine or placebo upon cessation of smoking, and followed for 28 days. The placebo group exhibited dysphoria, increased food intake by 300 calories per day--due almost exclusively to an increase in carbohydrate consumption (protein and fat intake remained constant)--and gained an average of 3.5 pounds. The *d*-fenfluramine group avoided dysphoria but initially increased carbohydrate intake, then returned to baseline dietary pattern and by the end of the four-week study period had lost an average of 1.8 pounds.

Conclusion

The obesity epidemic in the United States is almost certainly a product of changes in the economic environment over the last few decades. In this article, I have proposed a theory of endogenous preferences (derived from events in human evolutionary history) that could account for much of the observed trend.

In this view, it is no coincidence that obesity has increased in parallel with the rise in economic insecurity in the U.S. and around the world, and that consumption of fast foods and sweetened beverages--in which caloric density is paired with a strong glycemic effect--has increased at the same time. One advantage of building an economic theory of obesity on evolutionary foundations is that testable hypotheses are generated at many different levels of causation (Smith 2009). This article has, accordingly, reviewed

supporting evidence for insecurity-driven demand shifts from behavioral psychology, from nutritional endocrinology, and from anthropology.

The causal pathway implied by the foregoing argument--i.e., household-level economic insecurity induces demand for specific types of foods, which then induce weight gain--may be well-supported by ancillary evidence, but it is nevertheless only one of many plausible conjectures about the root economic causes of recent increases in the prevalence of obesity. In principle, a structural econometric model of obesity that identifies the effects of carbohydrate supply and demand--along with both direct and indirect effects of economic insecurity--could help to sort out the magnitudes of the various causes of weight gain, but the data requirements are daunting. Nevertheless, given the significant public health implications of the subject matter, this should become a priority of the research community.

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