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INSECURITY, INEQUALITY, AND OBESITY IN AFFLUENT SOCIETIES

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Chapter 4

Behavioural Biology and Obesity

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Introduction

It is sometimes said that the obesity epidemic has its roots in human evolutionary history, and that fattening is the inevitable result of the abundance of cheap food available in supermarkets. But not everyone gets fat upon exposure to the modern marketplace: obesity rates vary geographically, over time, and by social class. In this chapter, I will argue that the observed variation in human obesity is easier to understand from the perspective of behavioural biology. A deeper understanding of the biological underpinnings of human obesity also points suggestively to weakening social welfare policies as a potentially important cause of recent increases in obesity—in other words, it suggests the welfare regime hypothesis outlined in the Introduction, Chapter 2, and Chapter 11.

Optimal fattening in the natural world

If obesity is indeed a product of biological evolution—and there is every reason to believe that it is—then it makes sense to look first to the factors that drive fattening in the natural world. Ecologists generally agree that the primary adaptive function of stored (depot) body fat in foraging animals is to serve as a physiological buffer against periods of food shortage. But offsetting the survival benefits, there are also costs: carrying excess body fat, for example, can increase metabolic requirements, might hamper mobility, and may make an individual more susceptible to predation or less attractive to mates. This tension between benefits and costs means that more fat is not always better, and, moreover, that it might make sense to fatten more when the benefits are greater. In particular, the natural history of this phenomenon would suggest that an optimal fattening strategy should depend not just on current availability or abundance of food, but

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also on the extent to which future food supplies are *uncertain*. But if this is true, a natural question arises: how do animals know that food supplies are at risk?

Like humans, individual foraging animals within a given species vary greatly in the extent to which caloric energy is stored as body fat. A great deal of evidence on this subject is available in the scientific literature, and behavioural ecologists have noted an overarching regularity. In natural settings, fattening is typically triggered by the presence of reliable environmental precursors to food shortage [1]. One of the most important and widely studied triggers is *photoperiod*, or length of day. The progressive shortening of the day as the winter solstice approaches is a reliable predictor of seasonal (winter) food shortages in many natural environments, and so perhaps it is unsurprising that many species have evolved a tendency to fatten during this time, before food scarcity becomes a constraint.

Seasonal fattening observed in the wild does not—in and of itself—necessarily imply that length of day is the critical environmental trigger. It might be, for instance, that the behaviour instead depends significantly on weather events, or on experience in previous years, or on increasingly scarce food supplies. But these various hypotheses have been tested in laboratory settings under controlled conditions and—again and again—largely ruled out [2]. Evolutionary forces appear to have settled on photoperiod, the most reliable of seasonal regularities, as an indicator of impending food scarcity for a wide variety of species—including our own. Though technology and trade have greatly diminished the extent to which food becomes less available in the winter months, seasonal variation in body fat is still observed in modern human populations [3–5]. And though there are many possible explanations, there is suggestive evidence that photoperiod still plays a role. The affliction known as *seasonal affective disorder*, or winter depression, for instance, is distinguished from other forms of depression in part by the realization of weight gain, and other symptoms—increased appetite, excessive sleeping, and torpor—closely parallel those exhibited by laboratory animals whose fattening episodes are triggered by shortened days. Despite the best efforts of the pharmaceutical industry, the most reliable treatment for seasonal affective disorder today is prolonged exposure to artificial light. Moreover, another mental illness—*summer depression*—has also been documented. It typically occurs in early summer, and is characterized by insomnia, loss of appetite, and weight loss [6].

Though seasonality is important and serves as an instructive example, the security of food supplies in the natural world can vary for other reasons. For many species, social cooperation or competition is key to securing food. For example, willow tits—small insectivorous birds—often share foraging sites, and clear dominance hierarchies typically emerge. At a given foraging site, the dominant bird has first access to the most productive feeding areas, while subordinates are forced to flit about the periphery in search of prey. In field studies, an intriguing paradox has emerged: in spite of their limited access to food, subordinate tits are typically found to be *fatter* than dominants [7]. The paradox is resolved by looking beyond *availability* and instead considering *risk*. Food may be less available for the subordinate, but the risk of food shortage is even greater (in other words, when times get tough, the dominant will secure what little food is available, while the subordinate is left to starve). Though the two factors at work here—availability and risk—work in opposite directions for the subordinate (with limited availability favouring thinness, while high risk favours fattening), the latter appears—at least in this instance—to have a greater impact on observed outcomes. This is not to say that food availability is unimportant; indeed, it has also been shown that if food becomes scarce enough, the dominance-fattening relationship reverses, with the subordinates (who in this case cannot secure enough food to build up stores of fat) becoming thinner than dominants [8, 9].

Thinking of fattening as a strategy favoured by evolution necessarily leads to consideration of the interaction between current availability and future risk. This suggests that the popular view—that evolution never prepared us for 'cheap food'—may be only half right. Cheap food (i.e., ease of availability) by itself will not necessarily be a significant driver of human obesity in the absence of risk—or at least, in the absence of environmental triggers that would have indicated risk over the course of human evolutionary history.

Information molecules and emotional eating

At first blush, it might seem implausible to apply an ecological model to human obesity. After all, how often do people in the modern world calculate the probability of a starvation event when deciding how much to eat, or how much to exercise? But it is important to remember that biological evolution is, at its core, a molecular process, in which the biochemical building blocks of life (e.g., DNA, along with epigenetic materials) are passed from generation to generation, and shaped by selection events in our collective evolutionary history. While this molecular heritage is incredibly rich in complexity, there is no reason to expect that it comes packaged

with a conscious awareness of the original reasons our bodies do the things they do. It seems unlikely, for example, that patients afflicted with seasonal affective disorder spend a lot of time worrying about whether food supplies will last the winter. All such patients are likely to know is that they feel hungry, or they feel tired, or they just don't feel like getting up to go to the gym.

Thanks to modern molecular biology, the mechanisms by which our genes influence our behaviour are now much more than mere speculation. Consider, for example, the peptide hormone known as *leptin*. Secreted by fat cells, leptin circulates in the bloodstream roughly in proportion to the amount of body fat stored. The concentration of leptin in the blood is monitored by leptin-specific receptors in the brain and elsewhere, with specific physiological and behavioural effects. These effects are perhaps most stark—and thus revealing—in animals which have been bred or engineered to completely lack the ability to synthesize leptin. The *obese*-type mouse is one such creature. The *obese* mouse is typically of normal weight at birth, but rapidly gains weight while—even after becoming extremely obese, and remaining under free-feeding conditions—exhibiting symptoms of extreme starvation: decreased body temperature, overeating, torpor, diminished immune function, infertility, and even an enhanced ability to detect sugar in food [10, 11]. These traits would all be adaptive under conditions of actual starvation—that is to say, in a normal mouse in which an absence of leptin actually signalled a complete lack of fat stores, these physiological and behavioural reactions would help to ensure survival. Though human analogues of the *obese* mouse are exceedingly rare, it may be worth noting that medical reports on two such patients (whose symptoms were relieved with the injection of synthetic leptin) fail to report deficiencies in dietary quality or concerns about body image, or even an imminent famine, but rather note merely that the two were 'constantly hungry'.

Genes for leptin and the leptin receptor have been identified in humans and many other species, and though the basic functionalities are constant within a given species, slight variations from individual to individual can 'calibrate' the potency of the leptin signal up or down. Leptin is only one of dozens of circulating molecules known to regulate energy homeostasis in humans, and the interactions between these factors (sometimes referred to as 'information molecules') is complex and not yet well understood [11]. But leptin nevertheless offers a view into the manner in which evolutionary forces can influence human behaviour: via genes, which calibrate the molecular signals that regulate

metabolism and are perceived as emotional states such as hunger, satiety, depression, stress, and the palatability of food.

Obesity-related stressors today

It may be counter-intuitive to apply a theory of optimal fattening to human obesity, but given the suggestive evidence and plausible biochemical mechanisms reviewed above, it is tempting to try. What might we find if we were to take a step back, and look at obesity in our own species from the perspective of behavioural ecology? The first task would be identification of the kinds of environmental cues that might have served as indicators of impending food shortage over the course of human evolutionary history. These probably would have included photoperiod and other indicators of season, as discussed above, but socio-economic interactions have also been important for many thousands of years. Reciprocal food sharing is a nearly universal feature of hunter-gatherer societies, and seasonal food storage is practised by even the most simple of human cultures [12–14]. This reliance on cooperation and storage (along with the incidental threat of confiscation) would have made the strength of one's social entitlements among the most important determinants of food security. The implications of this history for obesity in the modern world are varied. It might be that the quality of social bonds and of the respect and trust of one's peers persist as determinants of the physiology of fattening. But it is certainly also true that threats to financial or economic security today are often determined by economic forces and state-level public policies beyond the control of the individual or the household. It is reasonable to ask whether or not these larger forces might also induce weight gain (or loss).

Economic insecurity as a stressor

It has long been known that 'stress' can cause weight gain [15–19]. This can be seen even at the molecular level, as in the observed strong interactions between glucocorticoids (the 'stress hormones') and leptin [20–22]. Uncertainty about future financial or economic well-being is a very specific type of 'stressor', and there have been reports in the scientific literature suggesting a link between obesity and various forms of economic insecurity. Townsend *et al.* and others, for example, have noted that individuals who report episodes in which they did not have enough money to buy food (as measured by the US Department of Agriculture's food

insecurity survey instrument) are often at greater risk of weight gain [23]. Similarly, Gerace and George interviewed 438 male firefighters, and found in a seven-year follow-up that those who had initially reported worrying about financial security subsequently gained an average of 4 lbs more than those who did not [24]. Smith *et al.* examined the relationship between weight gain and income security in 2,500 working-age males over a twelve-year period [25]. The authors used a statistical estimation technique (instrumental variables) to isolate episodes of unemployment or income loss that occurred over this period due to forces beyond the individuals' control, and found that a 1 per cent increase in the probability of job loss caused weight gain of around 0.6 lbs, while each episode in which annual income decreased by 50 per cent or more triggered a weight gain of 5 lbs.

There are other phenomena in the literature on obesity that are consistent with the economic insecurity hypothesis. Several recent studies, for example, have reported an 'obesity contagion' phenomenon in which individual weight gain appears to induce weight gain in one's friends and family [26–29]. Though many hypotheses have been offered as to the reasons this pattern might have emerged, from the propagation of body-weight norms to the spread of unhealthy eating habits, it is also possible that the underlying mechanism is economic insecurity. Family and close friends can be an important source of financial risk sharing and income pooling, so there is every reason to expect that perceived economic insecurity would be affected by financial or economic events in the lives of one's peers [30–32]. Barnes *et al.* provide some evidence in support of this mechanism by showing that cohabitation with working (but not non-working) individuals appears to be protective against weight gain [33].

Obesity around the world

As the incidence of obesity has risen around the world, many countries have begun to monitor the problem by periodically conducting national surveys. The reported differences in obesity prevalence both between countries and over time is striking (Table 4.1). The United States has the highest obesity rates in the developed world (32.6 per cent as of 2005) as well as the greatest absolute increase in recent years. Other countries—New Zealand and Iceland being particularly notable—have also experienced large increases, while some countries—such as Japan and Switzerland—have been relatively unaffected by this global epidemic. Many explanations have been offered for the trend, but some of the most obvious—such as income growth, or decreases in the relative price of food (Figures 4.1 and 4.2)—do not match up well with the data. Recent economic history, however, may provide some clues. New Zealand and Iceland have been referred to as 'economic miracles' in the past two decades, for their leadership in the aggressive pursuit of so-called 'neoliberal' economic policies. These typically include deregulation, privatization of public service agencies, free trade, tax structures that favour capital accumulation over redistribution, weakening of both the social safety net and labour protections, and macroeconomic policies that aim for price stability rather than full employment [34, 35]. While it can be difficult to compare policies across countries over time, the Fraser Institute publishes an index of economic liberalization for many countries. Figure 4.3 plots changes in this index for OECD countries against changes in obesity rates. At least at this superficial level, it would appear that economic liberalization (arguably a root cause of household-level economic insecurity in many countries) has much more explanatory power than either per capita income or the relative price of food. A recent paper by Offer *et al.* (and Chapter 11) come to a similar conclusion with more rigorous methods, examining the effects of economic insecurity, income inequality, and

Table 4.1 Obesity rates in OECD countries, 1990-2005

	Obesity Rate (%)		
	1990*	2005	Rate of Change (% per year)
United States	20.0	32.6	0.84
New Zealand	13.5	25.8	0.82
Iceland	9.4	20.2	0.72
Australia	10.9	20.0	0.61
England	13.8	22.7	0.59
Spain	9.7	17.8	0.54
Finland	10.3	17.3	0.47
Denmark	8.0	14.2	0.41
Sweden	7.2	13.2	0.40
Netherlands	7.1	12.7	0.37
France	7.2	12.4	0.35
Austria	10.0	14.8	0.34
Canada	15.1	18.6	0.32
Italy	8.7	11.9	0.29
Switzerland	6.7	9.9	0.25
Japan	2.2	3.8	0.11

Notes: Obesity data are from OECD Health 2009, corrected for differences in demographics and reporting method using correction factors from NHANES III and interpolated where needed (author's calculations).
*Because 1990 obesity rates for four countries (Austria, Canada, Italy, and Switzerland) are not available, initial values for these countries are taken from national surveys in 1991, 1994, 1994, and 1992 respectively.

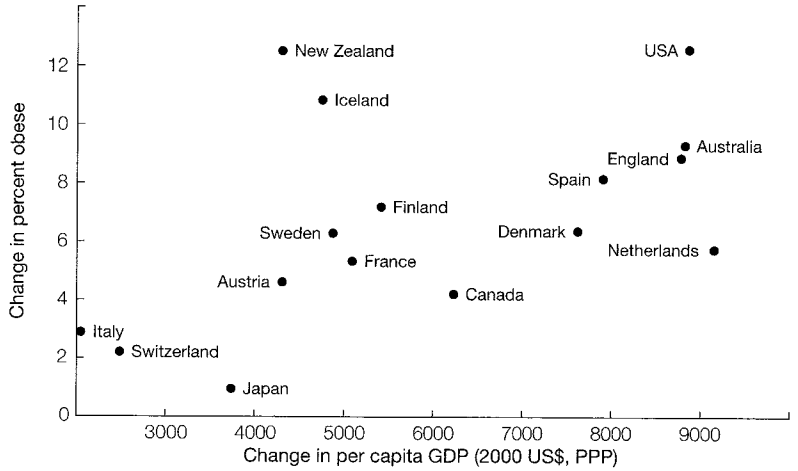

Figure 4.1: GDP and obesity in OECD countries, 1990–2005.

Notes on Figures 4.1–4.3: Obesity data from *OECD Health 2009*, corrected for differences in demographics and reporting method using correction factors from the third National Health and Nutrition Examination Survey (*NHANES III*) and interpolated where needed (author's calculations). Because 1990 obesity rates for four countries (Austria, Canada, Italy, and Switzerland) are not available, data for these countries cover only 1995–2005. Gross domestic product (GDP) and price indices from *OECD Stat* (accessed April 2009 and July 2009, respectively). Index of Economic Liberalization is drawn from *Economic Freedom of the World: 2008 Annual Report*, http://www.freetheworld.com.

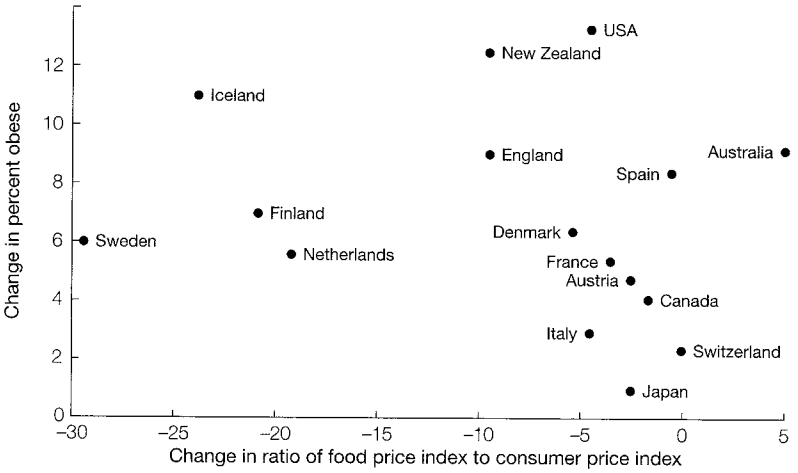

Figure 4.2: Food prices and obesity in OECD countries, 1990–2005.

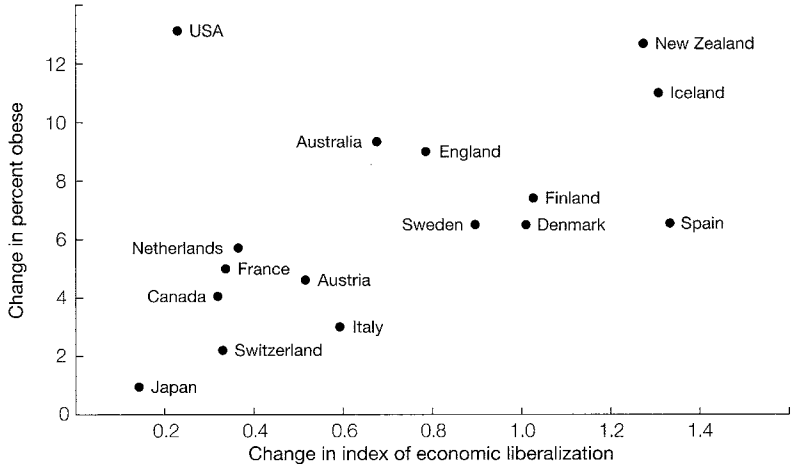

Figure 4.3: Economic liberalization and obesity in OECD countries, 1990–2005.

fast-food prices on obesity rates in a large sample of countries; again, the authors report that economic insecurity seems to dominate [36].

Economic insecurity in the USA, 1970 to the present

The anomaly in Figure 4.3 is the United States. While it is true that the USA already had a very high economic liberalization score in 1990 (and thus perhaps little opportunity for subsequent increase), it may be informative to ask whether or not there is any evidence that household-level economic insecurity changed over this period there. In fact, obesity rates have been rapidly increasing in the USA since the late 1970s, so perhaps earlier events will be relevant as well [37].

There are a number of dimensions along which public policies enacted over this period likely affected household-level economic insecurity. Among the first was the US 1974 Employee Retirement Income Security Act, which exempted employers from all state regulation of health insurance if they chose to self-insure rather than contracting with insurance companies. An unfortunate side effect of this provision was that it withdrew most of the large American corporations—employers of some of the healthiest workers—from the health insurance market, triggering a progressive degradation of the risk pool that left some 38 million Americans without health insurance—and thus at risk of financial ruin—by 1992 [38].

Another blow came in 1981, when a Reagan administration ruling on an obscure provision in a 1978 law legalized the 401(k) plan. Unlike traditional defined-benefit pensions, 401(k)s are individual retirement accounts in which the risk associated with investment returns are borne by each worker individually rather than pooled across all employees. 401(k)s also made it possible for firms to decrease contributions to employee pensions, with average contributions falling from 3.5 per cent of worker payrolls in the late 1970s to 1.5 per cent in the late 1980s [39]. It has also been suggested that the very nature of the employee-employer relationship has changed, thanks in part to the aggressive pursuit of international trade (e.g., the ratification of the North American Free Trade Agreement—source of much blue-collar angst about job loss—in 1994), and in part to changes in the enforcement of American antitrust and corporate governance laws since 1980 [40–42]. At the same time, the welfare reforms enacted in 1996 significantly weakened an important part of the US social safety net, by placing strict lifetime limits on subsistence payments for the indigent [43].

Looking more broadly at income trends in recent decades, one of the most dramatic is the increase in income inequality in the USA [44]. But income inequality as usually measured—as a cross-section or 'snapshot' of incomes at a point in time—is an imperfect measure of the experience of individual households. A highly unequal income distribution might indicate, as is usually presumed, that a subset of households experience persistent poverty, while others enjoy persistent wealth. Alternatively, it could be indicative of income *instability*, or the extent to which the income of a given household will tend to fluctuate from year to year. Gottschalk and Moffitt studied the trend in inequality using longitudinal data from 1970 to 2004, and found that income instability increased sharply in the late 1970s and early 1980s, and has remained more or less at that level ever since [45]. By their estimates, rising instability can account for roughly half the increase in cross-sectional inequality through the late 1980s.

Political scientist Jacob Hacker has dubbed the multifaceted rise in economic insecurity in the USA over the past three to four decades 'The Great Risk Shift' [43]. Proponents of the public policies that have resulted in the transfer of risk (of health care expenditures, of poverty in retirement, of job loss) from the state to the individual argue that such changes improve allocative efficiency in the economy. But it is possible that any such gains come at the cost of an expansion of the American waistline.

Conclusion

Evidence for the evolutionary origins of human obesity is quite literally written into our genes. This natural history, together with a broad array of supporting evidence, suggests that the presence of economic risk at the household level is very likely a key determinant of body fat today. It also suggests that obesity may be an unintended side effect of public policies that affect the extent to which economic risks are borne by the modern household.

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