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Food insecurity as a driver of obesity in humans: The insurance hypothesis

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Abstract: Integrative explanations of why obesity is more prevalent in some sectors of the human population than others are lacking. Here, we outline and evaluate one candidate explanation, the insurance hypothesis (IH). The IH is rooted in adaptive evolutionary thinking: The function of storing fat is to provide a buffer against shortfall in the food supply. Thus, individuals should store more fat when they receive cues that access to food is uncertain. Applied to humans, this implies that an important proximate driver of obesity should be food insecurity rather than food abundance per se. We integrate several distinct lines of theory and evidence that bear on this hypothesis. We present a theoretical model that shows it is optimal to store more fat when food access is uncertain, and we review the experimental literature from non-human animals showing that fat reserves increase when access to food is restricted. We provide a meta-analysis of 125 epidemiological studies of the association between perceived food insecurity and high body weight in humans. There is a robust positive association, but it is restricted to adult women in high-income countries. We explore why this could be in light of the IH and our theoretical model. We conclude that although the IH alone cannot explain the distribution of obesity in the human population, it may represent a very important component of a pluralistic explanation. We also discuss insights it may offer into the developmental origins of obesity, dieting-induced weight gain, and anorexia nervosa.

Keywords: Obesity, overweight, meta-analysis, food insecurity, weight regulation, hunger-obesity paradox, behavioural ecology, eating disorders

1. Introduction

The prevalence of obesity and overweight is increasing across almost all countries of the world (NCD Risk Factor Collaboration 2016; Wang et al. 2011). This is considered to constitute a major global public health challenge. Despite the societal importance of the topic, there is a dearth of well-developed explanatory theories for why some people become obese. Weight gain occurs when individuals habitually consume more energy than they use. Thus, decision making – in particular, decision making about how much and which foods to eat – is a central and necessary node on the causal pathway to weight gain. Decision making must, in turn, be underlain by decision-making mechanisms. It is the operating principles of these mechanisms that we need to understand: Under what circumstances will individuals recurrently make decisions that lead to their habitual consumption of more calories than they immediately require?

In this article, we advance and review one particular hypothesis concerning obesity. We will call this the insurance hypothesis (IH). We will lay out the hypothesis and its predictions over the course of the article, but it is worth stating its main constituent claims up-front:

- Storage of body fat is an adaptive strategy used by many vertebrates, including humans, to buffer themselves against periods during which food is unavailable.
- Fat storage also has costs.
- The optimal level of body fat to store, therefore, depends on security of access to food: If food is guaranteed to be always available, relatively little fat storage is necessary, but as the risk of temporary unavailability of food increases, the amount of fat the individual should optimally store also increases.
- Humans and other vertebrates possess decision-making mechanisms that adaptively regulate their fat storage. These mechanisms cause them to increase their energy intake above their level of energy expenditure when they receive cues from their environment that access to food is insecure, and reduce their energy intake

to close to their expenditure when they receive cues that access to food is secure.

- A major driver of obesity and overweight among contemporary humans is exposure to cues that, over evolutionary time, would have reliably indicated that access to food was insecure. Exposure to these cues engages evolved decision-making mechanisms and leads to increased food consumption relative to expenditure, thus resulting in greater fat storage and higher body weights.

It is the final claim that constitutes the IH for the distribution of obesity in contemporary humans. However, the plausibility of the final claim depends logically on establishing each of the earlier points. Thus, in this article, we will consider each of the earlier points before reviewing the evidence supporting the final one.

We must stress that the IH does not originate with us. The adaptive ideas underlying it were developed within behavioural ecology more than two decades ago (see sect. 3) and have been most thoroughly tested empirically in birds (see sect. 4). There is already an extensive human social science literature on the relationship between obesity and food insecurity (see sect. 5); here, the idea tends to be known by such names as the food-insecurity hypothesis or hunger-obesity paradigm. However, this human literature makes no reference to the adaptive ideas from behavioural ecology and little reference to the empirical evidence from non-human animals. Thus, our goal in this article is to bring together the models from behavioural ecology, the non-human findings, and the empirical evidence from humans to provide an integrative statement and assessment of the IH, including its strengths, its limitations, and its possible extensions and applications.

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2. Existing approaches to the psychology of human obesity

The IH is fundamentally a psychological hypothesis, given that it concerns mechanisms, presumably in the brain, that sense cues in the individual's experience and use those cues to regulate energy intake and/or expenditure. Before turning to the IH, then, we will examine some of the other psychological approaches to obesity that have been proposed. A first influential idea is the evolutionary mismatch hypothesis (e.g., Nesse & Williams 1995, p. 48): Roughly speaking, the idea that human decision-making mechanisms are optimized for ancestral environments where calories were usually scarce. In contemporary environments, these mechanisms produce overconsumption, especially of energy-dense foods. Obesity in contemporary populations is thus the by-product of a mind evolved to deal with frequent scarcity living now in constant abundance (for a recent version of this argument, see McNamara et al. 2015). A variant of the evolutionary mismatch hypothesis states that it is energy expenditure, rather than food supply, in modern environments that falls outside of the ancestral range (Prentice & Jebb 1995). Because ancestral energy expenditure was always high, we do not down-regulate food intake sufficiently when this is not the case.

Consistent with the evolutionary mismatch hypothesis is the overwhelming evidence that mean body weight increases as the population's lifestyle comes to resemble that of the urban developed world (NCD Risk Factor Collaboration 2016). However, the evolutionary mismatch hypothesis alone is incomplete, because it fails to account for the patterned variability in the incidence of obesity. If, as a species-typical fact, humans lack mechanisms to appropriately limit their intake of energy-dense foods when these foods are constantly abundant, then more or less *all* humans living under conditions of affluence should be overweight or obese. This is not the case. In countries such as France, Italy, Spain, Austria, Canada, and Korea, the majority of people have body mass indexes (BMIs) of less than 25, the conventional cutoff for classification as overweight (Wang et al. 2011). Even in the United States, which has very high rates of obesity, around one third of adults are neither overweight nor obese (Wang et al. 2008). Moreover, the evolutionary mismatch hypothesis provides no account of why there should be such dramatic differences between affluent countries in obesity prevalence. Widespread obesity is concentrated in countries with relatively high levels of economic inequality (Pickett et al. 2005), or (relatedly) where large numbers of individuals face economic insecurity (Offer et al. 2010). For example, whereas the 2014 rate of adult female obesity (BMI $\geq$ 30) is 34.9% in the unequal United States, it is only 3.0% for Japan and 17.3% for Switzerland (World Health Organization 2015). Yet it would be hard to argue that most people in Japan or Switzerland lack access to abundant energy-dense food if they want it.

Just as the evolutionary mismatch hypothesis alone fails to predict the between-country variation in obesity prevalence, it also fails to predict the within-country variation, too. Within high-income countries, obesity has been

consistently linked to a low socioeconomic position, especially in women, whether this is defined by income, education, or occupation (McLaren 2007; Sobal & Stunkard 1989). Living in a disadvantaged community increases the risk of obesity above and beyond the effects of individual-level socioeconomic status (Black & Macinko 2008). The simplest rendering of the evolutionary mismatch hypothesis would predict that the more financial resources people have, the more they would be able to satisfy their evolved food motivations, and the fatter they would be. In fact, the opposite is true: It is those social groups with the greatest constraints on available resources to spend on food that carry the most body fat. Thus, while the evolutionary mismatch hypothesis correctly draws attention to the obesogenic potential of the food landscape in developed countries, it needs augmenting to account for the fact that obesity is concentrated under particular types of social conditions.

A separate literature links obesity to a group of related psychological traits such as impulsivity, inhibitory control, or sensitivity to reward (e.g., Guerrieri et al. 2012; Nederkoorn et al. 2006; Weller et al. 2008). The central finding of this literature is that obese individuals are relatively impulsive (present-oriented, unable to delay gratification, sensitive to reward, etc.). Because this approach is rooted in the psychology of individual differences, it has greater potential to explain why some people become obese and others do not. However, when researchers have measured, in the same study, impulsivity for food and impulsivity in non-food domains, it is only the food-related impulsivity measure that is associated with obesity or food consumption patterns, not the more general measure (Dassen et al. 2015; Houben et al. 2014). Thus, the finding essentially comes down to the fact that people who are obese or eat unhealthily place a high motivational value on getting food soon. Although this is plausible, it fails to provide a very deep explanation: What is the cause of some people placing a higher motivational value on immediate food than others do?

In view of the foregoing discussion, it seems clear that our existing understanding of the drivers of obesity is incomplete. Any satisfactory approach needs to account for the strong ecological patterning of obesity and overweight (socioecological factors such as income inequality or individual poverty increase the risk), but also explain *why* people respond to these particular contexts by increasing their energy intake relative to their expenditure. In the next section, we return to evolutionary first principles of what fat storage is for, in order to develop the foundations of the IH.

3. A functional approach to fat storage: The insurance hypothesis

3.1. Background

Specialized lipid stores are found in the bodies of all well-nourished animals (McCue 2010). Lipid storage is an evolved adaptation that allows individuals to continue to survive and reproduce in the face of temporary shortfalls in energy intake from food (Higginson et al. 2012; 2014; Norgan 1997; Pond 1998). When glycogen reserves from immediate food intake become depleted, animals generate energy mostly through the oxidation of their lipid stores until food becomes available again, though they switch to the catabolism of protein when the level of adiposity drops low enough (McCue 2010). Lipid stores are thus beneficial to the organism and, other things being equal, the greater the extent of stored lipids, the longer the period of energy shortfall an individual is able to buffer.

However, storing lipids also has disadvantages. First, as body weight increases, so too do energy requirements. The positive scaling of energy requirement with body weight is well established across species (White & Seymour 2003), but energy requirements and body weight also co-vary within a species, including within humans (Garby et al. 1988; Johnstone et al. 2005; Leibel et al. 1995; Prentice et al. 1986). Not all of this evidence is correlational: Leibel et al. (1995) measured energy expenditure in human participants at baseline and then after a 10% weight gain or a 10% weight loss, and found that energy expenditure responded to changes in body weight. Thus, an individual storing more body fat will increase his or her ability to buffer periodic shortfalls, but do so at the cost of requiring greater energy intake to maintain his or her body weight.

Another consequence of increased body weight is reduced locomotor performance. In birds, for example, it is well established that extra mass impairs flight performance (Kullberg et al. 1996; O'Hagan et al. 2015; Witter et al. 1994). In terrestrial animals, too, the cost of locomotion increases with body mass, albeit following a decelerating function (Rubenson et al. 2007). The BMI distribution of successful human runners is sharply curtailed at the heavier side, and the more elite the selection of athletes, the lower the variance in BMI (Sedeaud et al. 2014). For running events of 3,000 meters and more, the BMI associated with maximal elite performance is around 20, which is towards the bottom end of the normal weight range. (Elite competitors in events shorter than 400 meters have higher BMI values, sometimes in the overweight range, but this is due to muscularity rather than adiposity.) Reduced locomotor performance is likely to affect fitness: For a prey species, locomotor abilities are central to escaping predators, whereas for predators, particularly cursorial predators like humans, locomotor abilities are central to getting enough to eat. Increased body weight also increases the risk of injury or death due to the forces and loads involved in maintaining a larger body (e.g., osteoarthritis; see Bray 2004; Felson 1988).

In view of the consequences of increased body weight, behavioural ecologists have long accepted that increased fat storage has benefits, in terms of enhanced ability to buffer shortfalls, as well as costs, in terms of increased energy requirements, health risks, and impairments to locomotion (e.g., Witter & Cuthill 1993). The optimal level of fat reserves to carry thus depends on how the beneficial aspects of increased adiposity trade off against the detrimental ones, and the shape of this trade-off will depend on the environment experienced by the individual. Beginning with Lima (1986), a series of theoretical papers has shown, using slightly different assumptions and approaches, that the optimal level of fat an animal should carry depends on the risk of shortfall in the food supply (Bednekoff & Houston 1994; Higginson et al. 2012; 2014; 2016; Lima 1986; McNamara & Houston 1990). If there is no risk of shortfall, the individual can maintain a minimal level of fat and need not incur the drawbacks of carrying any more than that. If the risk of shortfall is substantial, then the individual has to carry fat as insurance – insurance that is to be paid for in terms of the drawbacks of increased body weight. This

is the adaptive principle central to the IH and to this article.

3.2. An illustrative model

As the insurance principle is so fundamental to our claims, we wish to illustrate how it arises in quite a general way from principles of fitness maximization. We will therefore present a simple theoretical model here. The text presents the model in verbal form, and Online Appendix A provides the details. Our model uses an approach similar to several of the prior published ones, but sacrifices some realism in favour of generality and ease of exposition. Readers are referred to the papers on which we have built (Bednekoff & Houston 1994; Higginson et al. 2012; 2014; 2016; Lima 1986; McNamara & Houston 1990) for a sense of the elaborations that have been explored and, more importantly, for how similar results appear again and again in models set up in slightly different ways.

In our model, individuals must decide in each time period how much they will eat if they find food (from 0 to a maximum capacity of N energy units; N is always 10 for the results presented in this section). They have a metabolic requirement per time period, and anything they eat above this will be converted into fat and stored, increasing reserves but adding weight. (Weight and level of fat reserves are synonymous in our model.) The metabolic requirement is fixed at 1 unit per time period regardless of current body weight for the results presented here. (For the consequences of varying this, see Online Appendix A. Results are qualitatively unchanged by varying the metabolic requirement as long as that requirement remains substantially less than the amount an individual is able to eat in one time period, and unless it increases extremely

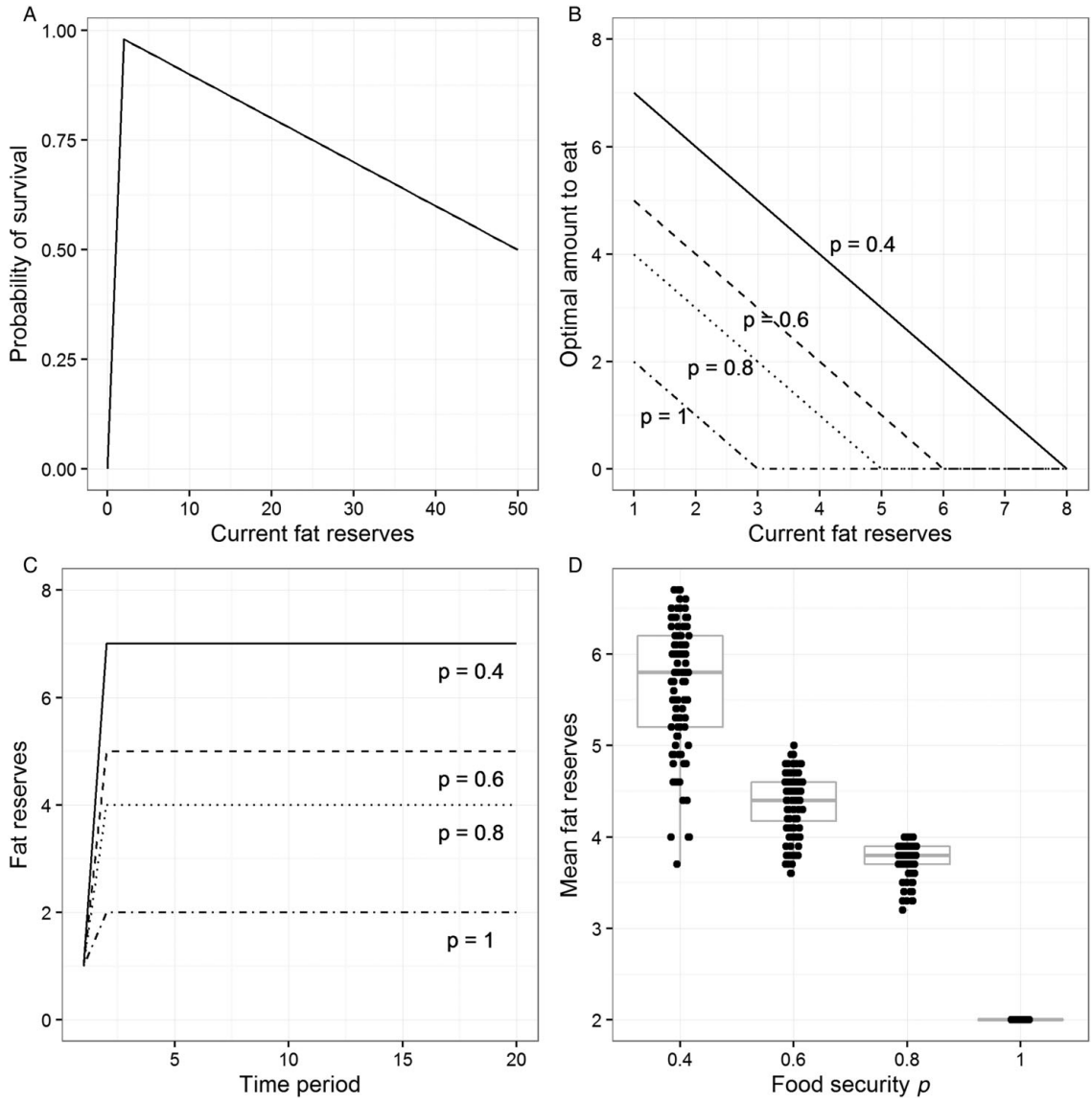

Fig. 1 - B/W online

Figure 1. Output from the model described in section 3 (for details, see Online Appendix A). (A) The assumed probability of survival against current level of fat reserves. (B) The optimal amount to eat for different levels of fat reserves and four values of the food security parameter p. (C) Fat reserves over 20 time periods for individuals who begin with reserves of 1 unit, follow the optimal eating policy for their level of food security, and find food in every period. (D) Mean fat reserves over 40 time periods for simulated individuals who find food each period with probability p and follow the optimal eating policy for their level of food security. Points have been jittered in the horizontal dimension to make them more visible.

steeply with increasing body weight. It is reasonable to assume that it should not do so, given that the main determinant of metabolic rate is lean mass; metabolic rate increases only slowly with additional fat mass.)

The individual may fail to survive the time period for two reasons. It may starve to death. As a default, we implement the probability of starvation as increasing very steeply as reserves approach zero: The probability is 0 at reserves of 2 units, 0.5 at reserves of 1 unit, and 1 (i.e., certainty) at reserves of 0 units. If the individual does not starve to death, then there is a probability of death from other causes such as predation or injuries. This probability increases by 1% for every extra unit of body weight. Thus, we are assuming an asymmetric survival function in relation to weight (Fig. 1A): There is a cliff-edge at the critically low threshold and a gentler slope with increasing weight, producing maximal survival just above the critical threshold. This asymmetric, inverted-V shape is biologically plausible and central to all of the theoretical models in this literature. Our model allows us to independently vary the location of the cliff-edge, its steepness, and the size of the fitness cost of each extra unit of reserves (see Online Appendix A and sect. 6.2 for the consequences of doing this).

Each time period, the individual finds food with probability p. We can think of p as the individual's level of food security. If p is 1, then access to food is totally secure, whereas if p is, say, 0.6, then access to food is very insecure: there is a 40% chance there will be no food. Given that we are concerned with computing optimal behaviour, we treat individuals as knowing the value of p for their environment perfectly. The question we set our model is: What is the best amount to eat if the individual does find food, given its level of food security p, and its current level of reserves? To find this optimal eating policy, we use a dynamic programming approach (Clark & Mangel 2000; Houston & McNamara 1999; Mangel & Clark 1988). This involves starting at the final time period in a long sequence and computing, for each value of p and possible level of reserves, what the probability of surviving beyond that period would be if the individual ate 0 units, 1 unit, 2 units, and so on (if food can be found). This produces a look-up table specifying for every level of reserves the amount to eat that maximizes the probability of survival. We then move to the previous period and ask, for every level of possible reserves the individual might have, and given that in the next period it will follow the already-calculated optimal policy for the reserves it will have at that point, what is the probability of survival associated with every possible eating decision? This in turn gives a look-up table linking reserves to the amount to eat for the penultimate period. The backwards iteration is repeated for 100 periods, and the output is the look-up table from the earliest time point. What we report as the optimal policy for each possible value of p thus represents the mapping between current reserves and amount to eat (if food can be found) that maximizes the probability of survival into the distant future.

Note that, although we have described the catastrophic fitness event that occurs when reserves fall below a critical threshold as death by starvation, and the maximand of the model as survival into the distant future, the catastrophic event could equally be thought of as loss of reproductive capacity; the maximand, the probability of successful reproduction. The computations and predictions would be the same under this interpretation. This is important because temporary energetic shortfall may lead to loss of reproductive capacity long before death by starvation is reached; this may be an equally important way in which energetic shortfall is detrimental to fitness. We return to this issue in section 6.2.

3.3. Model results

For all levels of p, the optimal policy produced by our model has the same basic form: If current reserves are very high, don't eat anything, and instead burn down some reserves. As reserves get lower, there comes a point where it is optimal to eat something and thus maintain or increase reserves (Fig. 1B). Both the level of reserves at which eating should begin and the optimal amount to eat when reserves are low depend on the level of food security p. When $p = 0.4$, for example, the individual should start to eat when reserves drop to 7 units, and when reserves drop to 1, the individual should take in 7 units per period. When $p = 0.8$, eating only kicks in when reserves drop to 4 units, and the most that the individual should ever eat is 4 units in a period. When $p = 1$, complete food security, the individual only eats when reserves drop to 2 units.

The optimal policies illustrated in Figure 1B amount to "trying" to maintain a constant fat buffer whose size is related to the level of food security p: The lower p is, the larger the buffer should be. We can illustrate this by simulating individuals who follow the optimal policies for different values of p and find food every time period (Fig. 1C). As Figure 1C shows, individuals initially eat more than their energetic requirements, then stabilize at a certain level of reserves. For $p = 1$, this is simply the level of reserves that maximizes survival in the current period (2 units), but for lower values of p, individuals carry more than this, and the lower p is, the more they carry.

Under food insecurity, by definition, individuals may not find food in every time period. Thus, a more realistic investigation is to simulate individuals who have a probability p of finding food each period and follow the optimal eating policy for that value of p. Given that this simulation has a stochastic component, no two individuals have exactly the same sequence of experiences or weights (as long as $p < 1$). We therefore simulate 100 individuals at each level of food security for 40 periods each. All individuals begin with 5 units of reserves, and individuals not surviving for 40 periods are excluded. Figure 1D plots individuals' mean body weights/fat reserves, removing the first 10 periods to eliminate initialization artefacts. As the figure shows, mean weights/reserves become higher as p becomes lower.

Thus, our very simple model recovers the insurance principle often described in the theoretical behavioural ecology literature. High levels of stored reserves ought to be found not among those whose access to food is assured, but exactly among those whose access to food is insecure. The more insecure this access is, the heavier their target weight should be, essentially because it is in their interest to bear the costs of some extra weight to insure themselves against the more catastrophic cost of possible starvation. The consequence of following this optimal policy is that individuals should in practice become heavier as their access to food becomes more insecure. This result is very robust to numerical variation in the parameters chosen (see Online Appendix A).

4. Non-human evidence that food insecurity causes weight gain

The insurance principle described in section 3 was well known in behavioural ecology at least as early as the publication of Lima (1986). Evidence consistent with it was available from observational comparisons both within and between species. For example, Rogers (1987) showed that bird species whose winter food supplies were unpredictable (insecure, in the language used in this article) carried more fat than those whose winter food supply was predicable (secure). More recent work confirms the basic effect of food security and demonstrates an additional effect of predation risk (Rogers 2015). Species facing higher predation risk, other things being equal, carry relatively less fat than those whose risk is lower. Because one of the major costs of additional fat in birds is the reduction in predator escape performance, this makes sense in the light of the theoretical literature: Birds trade off the risk of predation if they are fat against the risk of starvation if they are thin. Other early work showed that within bird species, fat storage increases at those times of year when insecurity of food supply is likely (for a review, see Witter & Cuthill 1993).

The real breakthrough arose when the experimental method began to be applied to fat storage. This allowed the unequivocal demonstration that fat storage was plastic within individuals and could be deployed strategically as a response to environmental experience. Ekman and Hake (1990) experimentally manipulated the food-access regime of captive greenfinches *Carduelis chloris*, by either giving them food *ad libitum* or an equal total quantity of food appearing intermittently at unpredictable times of the day. They found that 10 of 11 birds significantly increased their weight in response to the unpredictable regime; the lightest and leanest individuals showed the strongest response. Witter et al. (1995) subjected an experimental group of adult European starlings *Sturnus vulgaris* to unpredictable daily periods of food deprivation: Birds in this group increased their weight, while those in an *ad libitum* control group did not. This result was confirmed in a later experiment in juveniles (Witter & Swaddle 1997); here again, the largest response was seen in those individuals whose weight was lowest prior to the manipulation.

A related set of findings concerns the effect of dominance on weight regulation. Ekman and Lilliendahl (1993) showed in willow tits *Parus montanus* that it was subordinate individuals who carried the greatest fat reserves (for a related theoretical model, see Clark & Ekman 1995). Moreover, experiments in which dominant individuals were removed from flocks showed that this relationship was causal: Subordinates lost weight when the dominants were removed. Witter and Swaddle (1995) showed that in European starlings, too, subordinates carried more weight than dominants and lost weight when dominants were removed from their group. They also replicated the effect of imposing food insecurity on weight, but showed that the weight gain in response to insecurity was greatest among subordinates. Subordinate birds are, by definition, prone to being displaced or excluded from resources that are available. Thus, any insecurity in access to food is likely to fall particularly strongly on them, and so it is consistent with the IH that their levels of fat storage would be raised. This is a very interesting finding in light of the human epidemiological evidence that within affluent societies, it is the most disadvantaged social groups in which obesity is most common (Black & Macinko 2008; McLaren 2007; Sobal & Stunkard 1989).

Thus, the evidence from small birds shows that when individuals receive cues suggesting that their access to food is likely to be insecure – and hence that there might be periods of shortfall – they increase their stored fat reserves to provide insurance. Moreover, the use of experimental approaches demonstrates that the association between insecurity and fat storage is causal, and that individuals can dynamically increase or decrease stored fat in response to variation in their experience of the world. The implication is that birds have evolved psychological mechanisms that integrate information received concerning metabolic demands and likely security of access to food, and these mechanisms up-regulate levels of food consumption – or down-regulate energy expenditure – as perceived security of access to food decreases.

The evidence reviewed thus far is all from birds. The costs of excess mass might be particularly high in a small flying animal; terrestrial animals might thus tune their reserves less finely to their current expectations of shortfall. A recent experimental study showed that weight increased in mice whose food access was restricted, compared to a control group (Li et al. 2010). Thus, the insurance principle works in at least one species of mammal as well as birds. This does not, of course, guarantee that humans possess similar mechanisms. However, there is a large empirical literature on food insecurity and fatness in humans, and it is to this literature we now turn.

5. Empirical evidence for the IH

5.1. Background

In 1995, William H. Dietz published a paper in the journal *Pediatrics* with the title "Does hunger cause obesity?" (Dietz 1995). Dietz presented a case study of an obese young girl whose impoverished parents (also obese) received welfare assistance. They frequently lacked money to buy food in the period just before their welfare cheque arrived. They apparently compensated by consuming many calories whenever they could, leading to their high body weights. Dietz speculated that what was at work in this family might be "an adaptive response to episodic food insufficiency" (p. 766).

Dietz's empirical insight was followed up, but his adaptive logic was not. Hundreds of papers have subsequently been published on the association between food insecurity and high body weight in humans, as we shall see later in this section. Ironically, they often describe the association as paradoxical (e.g., Crawford & Webb 2011; Scheier 2005; Tanumihardjo et al. 2007). For example, Basiotis and Lino (2003, p. 57) asked, "How can a person report that in her household sometimes or often they do not have food to eat, yet be overweight? … A definitive solution to this paradox must await additional research." In fact, the association follows from the adaptive theoretical models developed years earlier in behavioural ecology. Unfortunately, not a single paper from the human social science literature that we have been able to find cites any of the theoretical models from behavioural ecology discussed in section 3.

The empirical studies that began to appear after Dietz's paper used either large, representative population surveys, or smaller opportunity samples of particular social groups, to investigate whether participants' reports of their food insecurity were associated with their body mass. Within this literature, food insecurity is defined as "limited or uncertain ability to acquire nutritionally adequate and safe food in socially acceptable ways" (Castillo et al. 2012; Dinour et al. 2007). It is typically measured using self-report questionnaires, of which the most widely used examples are the Radimer/Cornell Hunger and Food Insecurity Instrument (Kendall et al. 1995; Radimer et al. 1992) and its derivative, the U.S. Department of Agriculture's Core Household Food Security Module (Nord et al. 2009). These questionnaires address both the *experience* of sometimes having insufficient food (e.g., "The food that we bought just didn't last, and we didn't have money to buy more.") and also the *cognitive evaluation* that an episode of insufficient supply is likely ("We worried whether our food would run out before we got money to buy more."). Thus, what these instruments measure is some kind of running cognitive estimate of the variable p in our model: that is, the likelihood of a temporary shortfall in the food supply. Both questionnaires yield a continuous food insecurity score, although in practice this is often reduced to a food-secure versus food-insecure dichotomy, or a three-way – occasionally a four-way – classification.

The human literature on food insecurity and body weight has become so extensive that several reviews have appeared (Dinour et al. 2007; Eisenmann et al. 2011; Franklin et al. 2012; Laraia 2012; Larson & Story 2011; Morais et al. 2014). The general consensus of these reviews is that there is a positive association between food insecurity and high body weight in women, but the association is less clear or absent in men. This may well relate to the wider finding that low socioeconomic position is a more consistent predictor of overweight or obesity in women than in men (Sobal & Stunkard 1989). The previous reviews have also concluded that the relationship between food insecurity and high body weight may not be detectable in children, and that developing countries may not show the same pattern as the developed countries (especially the United States) from which most of the evidence comes.

5.2. Meta-analysis methods

Although the level of consensus within the existing review articles is fairly high, none has used meta-analytic techniques to estimate the overall strength of the association or examine potential moderators of association strength. Instead, they based their conclusions on tallying up which studies reported statistically significant associations and which ones did not. Because individual studies may have fairly low statistical power, this approach does not definitively answer the question of whether, for example, the association is significantly less strong in men and children than in adult women. We thus undertook a meta-analytic review of the human food insecurity–body weight literature to 2015. The full methods and results of the meta-analysis are presented as Online Appendix B. This and the next section provide a short summary.

We used PubMed and Scopus searches, enriched with all papers citing and cited by key previous reviews of the literature, to identify papers reporting quantitative data on an association between a measure of food insecurity and a measure of body weight. The initial candidate set identified by our searches was 173 papers. Review of the full text of these led to a final set of 125 papers included in the meta-analysis. The 48 excluded papers either did not present original data on a relevant association or did not present them in a form statistically comparable to the other studies. The standard measure of association used in this literature is the odds ratio (OR) or its logarithm (LOR) for high versus normal body weight for participants reporting food insecurity as compared to security. The exact definition of high body weight varies from association to association (e.g., for some associations it is obesity [BMI $\geq$ 30] versus normal weight, for others overweight [BMI $\geq$ 25] versus normal weight), as does the exact specification of the food-insecurity variable. In the majority of cases, ORs or LORs were provided directly by the study's authors. In the remaining cases, we converted correlations, frequencies, or means and standard deviations into LORs using standard transformations. Papers often presented multiple associations (e.g., separate comparisons for men and women, for obesity vs. normal weight and overweight vs. normal weight, or for severe food insecurity vs. security and moderate food insecurity vs. security). Thus, there were a total of 301 reported associations from the 125 papers. We dealt with the statistical non-independence of multiple associations from the same study using multilevel meta-regression.

As well as asking whether the evidence supports an association between food insecurity and high body weight overall, we explored the effects on association strength of a wide variety of moderating factors. These included aspects of the study design (longitudinal vs. cross-sectional, whether the authors controlled for co-variates such as socioeconomic position); the analysis (whether the high body-weight outcome was obesity or overweight, whether the predictor was continuous, dichotomous, or multinomial), and the participants (whether the sample was male, female, or mixed sex; adults or children; World Bank–defined high-income country or not). Full statistical results are presented in Online Appendix B. Here, we summarize the main findings qualitatively and illustrate them graphically in Figure 2 by showing central LOR estimates and their 95% confidence intervals, for a series of different subsets of the data.

5.3. Meta-analysis results

Overall, there was a positive association between food insecurity and high body weight (line 1 of Fig. 2). The central LOR estimate of 0.19 corresponds to an OR of 1.21 (95% CI [confidence interval] 1.14–1.29); the odds of high body weight are around 21% higher for food-insecure than food-secure participants. This estimate was almost unchanged when we restricted the analysis to just those associations where the OR or LOR had been stated in the original paper, rather than converted by us from other kinds of statistics (line 2 of Fig. 2). An important possibility is that this association is just a consequence of both food insecurity and obesity both being related to a common third variable, most obviously income or socioeconomic position (Gundersen et al. 2011a). If this was the mechanism producing the association, we would expect estimated associations from analyses that control for socioeconomic

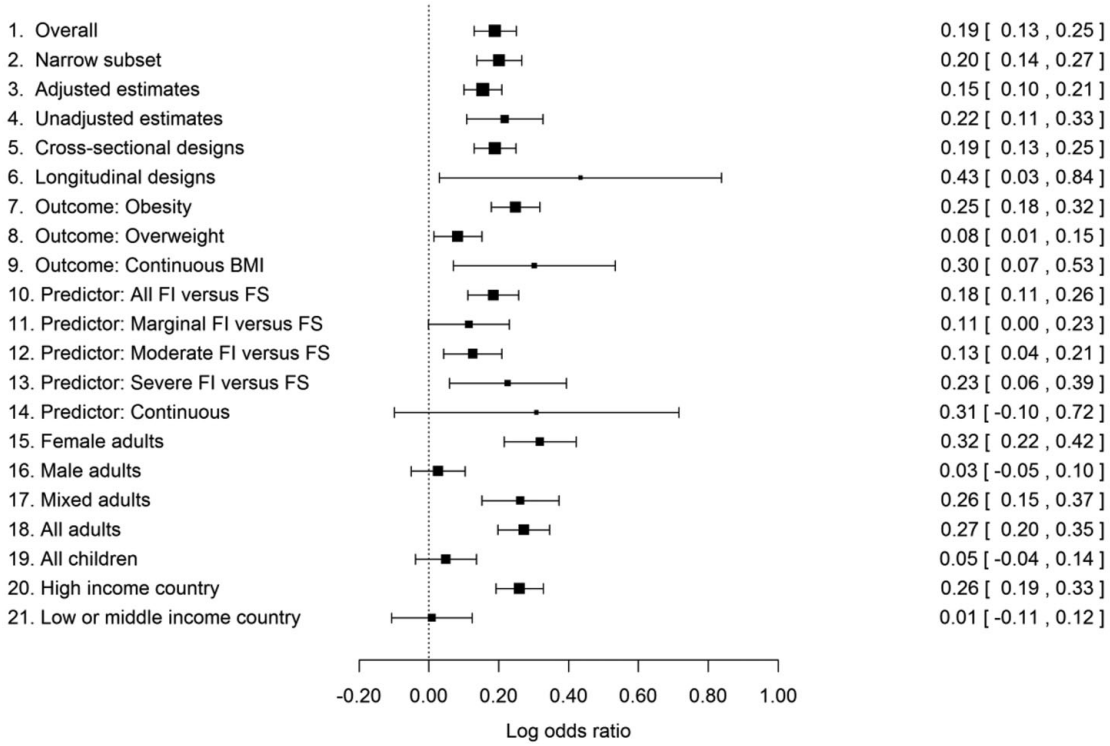

Fig. 2 - B/W online

Figure 2. Estimated log odds ratios (LORs) for high versus normal body weight in food-insecure versus food-secure individuals, plus their 95% confidence intervals, from the data set overall (line 1) and from various subsets of the data. Zero represents no association. The high body-weight outcome varies from association to association (e.g., obesity, overweight), as does the exact specification of the food-insecurity variable. For details, see section 5 and Online Appendix B.

and demographic factors to be substantially weaker than those from unadjusted analyses. This was not the case: The adjusted LORs in the data set were only slightly less strong than the unadjusted ones and still significantly greater than 0 (lines 3 vs. 4).

A previous narrative review suggested that longitudinal evidence for the association (which gives a stronger suggestion of causality) has not been as convincing as cross-sectional evidence to date (Larson & Story 2011). We found no evidence that longitudinal associations are any weaker than cross-sectional ones (lines 5 and 6). There are just many fewer longitudinal studies (seven that we were able to include, and several of these concerned the specific situation of longitudinal studies of pregnancy). Correspondingly, there is less precision in their estimate of the association. We note that most of the few longitudinal studies are only longitudinal in a partial sense: They examine change in body weight over time by food-insecurity status. We are aware of only one study employing the stronger "doubly longitudinal" approach, in which change in body weight is examined by *change in* food insecurity (Whitaker & Sarin 2007). Given that change in food-insecurity status may be relatively rare, such studies are difficult and require large samples. However, it is these designs that come as close to the experimental approaches used in birds as is possible with human participants. More longitudinal evidence, particular doubly longitudinal studies, is thus a priority.

There is considerable variation across studies in how the data are analysed. We found that associations are significantly stronger when the outcome variable is obesity (BMI $\geq$ 30) than when it is the less extreme outcome overweight (BMI $\geq$ 25; lines 7, 8, and 9 of Fig. 2). We found no significant differences in association strength according to exactly which predictor was used (lines 10–14 in Fig. 2). This is of note because in one of the most influential studies (Townsend et al. 2001), it was the milder but not the most severe levels of food insecurity where increased odds of obesity were found. Our analysis suggests that this is not a general pattern. However, the division points between marginal, moderate, and severe food insecurity are made in different ways by different authors, even those using the same measurement questionnaire. Thus, the lack of clear patterning of association strength by level of food insecurity may simply result from variation between studies in the definition of each level.

All-male adult samples showed significantly weaker associations than all-female or mixed-sex ones (which are often female-biased; lines 15–17 of Fig. 2). Moreover, the LOR in just the all-male adult samples did not differ significantly from zero. There has been a particular focus on women and girls in this literature, with 117 all-female associations reported compared to 41 male and 143 mixed-sex associations. However, this is likely to be a consequence of the sex difference in association – an influential early paper showed that food insecurity was particularly relevant to women's obesity (Townsend et al. 2001), and this inspired further research – rather than its cause. Forty-one papers still constitute a good sample size for detecting an association in men.

Child samples showed significantly weaker associations than adult ones. The LOR did not differ from zero in all children considered separately from the adults (lines 18

and 19). We also examined whether the age of children made any difference; overall, it did not, though there was some evidence that the sex difference in association characteristic of adults begins to be detectable in older children (see sect. B3.2 in Online Appendix B).

We also examined the moderating effect of the level of economic development of the study country. This made a significant difference to the association strength, with a positive LOR in high-income countries and an overall LOR close to zero in low- and middle-income countries (lines 20 and 21). The overall null effect in the low- and middle-income countries masks variability: Some individual studies have found significant positive associations in line with the high-income country evidence (e.g., Chaput et al. 2007 in urban Kampala), although there are several associations in the opposite direction (i.e., food insecurity reduces odds of overweight: Dubois et al. 2011; Isanaka et al. 2007) in children. The geographical coverage of the data set is very uneven: 209 of the 301 associations came from high-income countries, and 178 of these from the United States. More evidence is thus needed from different kinds of samples in the developing world and also from non-U.S. high-income countries.

A serious problem for the interpretability of meta-analytic results is publication bias. If significant positive associations are more likely to be published than null ones, then any data set assembled through a search of the literature will overestimate the true association. We examined whether publication bias was likely to be operative in two ways. First, we compared estimates from appropriate parts of our data set to those from two individual studies that used authoritative methods (Gundersen et al. 2009; Townsend et al. 2001). These both featured large, nationally representative samples (from the U.S. National Health and Nutrition Examination Survey) and high-quality measurement of both food insecurity and body weight. The results of Townsend et al. (2001) produced a combined LOR of 0.27 (95% CI 0.10–0.44) for U.S. women and an LOR not significantly different from 0 (exact value and CI unstated) for U.S. men. The aggregated studies from high-income countries in our data set give LORs of 0.42 (95% CI 0.29–0.55) for women and 0.03 (95% CI−0.05 to 0.10) for men. The individual LOR from Gundersen et al. (2009) for U.S. children (0.13, 95% CI −0.17 to 0.43, using the BMI-based measures) is extremely similar to the meta-analytic LOR for all children in high-income countries (0.11, 95% CI 0.01–0.21). Our aggregated estimates for high-income countries are thus broadly in line with the evidence from high-quality individual studies.

Second, we performed a standard statistical test for publication bias based on the asymmetry of the distribution of associations (Egger et al. 1997; see sect. B3.3 in Online Appendix B for details). The test was significant, suggesting publication bias might be operative. We then used the "trim and fill" method to impute the associations required to make the distribution symmetrical (Duval & Tweedie 2000). This procedure reduced the central estimate of the LOR by around one third, but it remained significantly different from zero (0.12, 95% CI 0.07–0.17). Moreover, the differences between women, men, and children, and between high-income and other countries, survive imputation of extra associations via the trim-and-fill procedure (see sect. B3.3 in Online Appendix B).

In summary, our meta-analysis of the literature leads to several conclusions. The large body of available evidence supports the view that food insecurity is a predictor of high body weight in humans. This is unlikely to be an artefact of food insecurity and high body weight both being associated with some third variable, such as socioeconomic position. However, the association is far from uniform. Specifically, the overall association is driven by adult women in high-income countries; it is weaker or absent in men, in children, and in low- and middle-income countries. These conclusions are largely consistent with those of previous reviews (Dinour et al. 2007; Eisenmann et al. 2011; Franklin et al. 2012; Laraia 2012; Larson & Story 2011; Morais et al. 2014). This is reassuring, given that we assembled a larger and more comprehensive data set than any previous reviews and used quantitative meta-analytic techniques for the first time. With the meta-analytic evidence in hand, we are now in a position to make an evaluation of the IH as an explanation for the distribution of obesity in the contemporary human population. That evaluation is presented in the next section.

6. Evaluating the IH as an explanation for human obesity

To begin evaluating the IH, it is worth restating exactly what its claims are. The hypothesis proposes that humans possess evolved mechanisms that respond to cues or experiences indicating that access to sufficient food is uncertain by increasing energy intake relative to expenditure, and hence storing more fat. Exactly how these mechanisms work at the proximate level (e.g., what the cues are, the relative contributions of increased intake and reduced energy expenditure, whether it is motivation for food overall or for energy-dense foods in particular that is affected) requires further specification. Note that the hypothesis does not need to claim that being obese is a *currently* adaptive strategy for people in food-insecure social groups. That is, it need not predict that in food-insecure social groups, fatter people have better survival than leaner people. Such a pattern would be very interesting in the light of the hypothesis, but the absence of such a pattern would not refute it. This is because the hypothesis claims psychological mechanisms that increase fat storage in response to cues of food security have, on average, been fitness-promoting over evolutionary time. It is agnostic on whether they still promote fitness in, say, the contemporary United States. For example, the mapping between cues of food insecurity and evolutionary fitness might be quite different in contemporary environments than in historical ones.

Although the evidence reviewed in section 5, taken overall, finds the association predicted by the IH, there are still important grounds for scepticism or at least qualification. Below we discuss some of these, before concluding with an overall evaluation.

6.1. Is the association strong enough?

To convincingly claim the IH was supported by the epidemiological data would require a strong association between food insecurity and high body weight. Our observed association, although statistically highly significant, is moderate: For adult women in high-income countries, the odds of high body weight are about 50% higher for food-insecure individuals compared to food-secure ones. To put this in context, it is larger than the increase in odds of high body

weight due to carrying a risk allele of the FTO gene (Frayling 2013; see sect. 6.5). Moreover, it is generally accepted that the existence of measurement error leads to the underestimation of associations. In classical psychometric theory, the best estimate of the true association is the observed association divided by the square root of the product of the reliabilities of the two measures, where reliability is the proportion of variation in the measure that reflects variation in the underlying quantity (Spearman 1910). Thus, if the reliabilities of the measures are 0.5, the true association is twice as strong as the observed association.

In the food-insecurity–obesity literature, there is likely to be considerable measurement error in both outcome and predictor. The limitations of BMI and its derivatives as measures of fatness are well known: They do not measure adiposity directly, and people of quite different body compositions can have the same BMI (Prentice & Jebb 2001). On the predictor side, the questionnaires used to assess food insecurity are unlikely to capture the required causal variable very accurately. The causal variable is presumably some implicit integration of multiple cues and experiences over an extended period of time. Questionnaires simply may not be able to capture this well; indeed, it may not be the kind of psychological variable that is available to explicit self-report with any precision. Thus, the relatively modest association strength does not, in our view, necessarily undermine the IH; rather, we are struck that any clear evidence emerges from such noisy measures.

6.2. Why is there a sex difference?

Our meta-analysis finds no association between food insecurity and high body weight in men. On the face of it, this is problematic for the IH, which should be generally applicable. In this section, we consider how differences between women's and men's life histories could explain why the predictions of the model described in section 3 are met in the one case but not the other. There is a clear sex difference in human adiposity, with fat representing around 27% of body weight in women to about 15% in men (Norgan 1997). The sex difference is generally attributed to the energetic requirements of reproduction for women (Norgan 1997; Zafon 2007). However, what we are concerned with here is not women's greater average adiposity, which appears readily explained by reproductive demands, but the greater responsiveness of their adiposity to food insecurity.

The best way to try to explain the sex difference within the model presented in section 3 is to make the shape of the function mapping reserves to fitness (Fig. 1A) different for men and women. The model allows three ways of doing this (see sects. A3.2–A3.4 in Online Appendix A). First, we can move the location of the fitness cliff-edge further to the right for women (Fig. 3A). This would make sense if the level of adiposity below which it is costly to drop is higher for them than for men, due to the need to be able to fund pregnancy and lactation. Moving the cliff-edge to the right increases steady-state adiposity at every level of p, and hence can account for women's greater adiposity overall (Fig. 3B). However, it does not increase responsiveness to food insecurity: The gradient of the relationship between p and steady-state fat reserves is unchanged by moving the cliff-edge to the right, as Figure 3B shows.

Second, we can make the probability of fitness loss increase in a more graded way as reserves become low, rather than the step-function used thus far (Fig. 3C). This is another way of capturing the intuition that for women there are costs of low reserves that manifest short of the point of death by starvation. A more graded diminution leads to individuals maintaining higher levels of fat reserves (this is because the effect of introducing the more graded function is to move the point of maximal survival in each period somewhat to the right; see Fig. 3C). However, it does not lead to greater responsiveness to changes in the level of food security p. On the contrary, a more graded survival function leads to fatter individuals who are somewhat *less* sensitive to the prevailing value of p (Fig. 3D). Thus, in our model, allowing women to have a greater minimal required level of adiposity, or a more graded relationship between low fat levels and reproductive success, correctly predicts that they will be fatter on average, but fails to shed any light on why they should be more sensitive to the experience of food insecurity.

The third way of altering the model is to make the slope at the right of the survival function steeper for men than for women. To recap, this slope represents the degree to which survival declines with each extra unit of weight. Steeper slopes (as shown in Fig. 3E) produce individuals who maintain lower average reserves and are also less responsive to the current level of food security p (Fig. 3F). This lack of responsiveness arises because with a heavy penalty for each extra unit of weight, it becomes too costly to carry a substantial buffer, regardless of the risks. Sexually differentiated foraging and mobility patterns are widely documented in hunter-gatherer societies and assumed to be typical of past human societies: Men range more widely, partly through pursuing more mobile prey (Marlowe 2007) and partly for other reasons (MacDonald et al. 1999). Men are also much more likely to be involved in intraspecific violent conflict, thought to be an important selection pressure in ancestral human societies (McDonald et al. 2012). Thus, one tentative possibility is that men's activities meant that the costs of extra body weight were more severe for them than for women over evolutionary time. If this were correct, our model would predict both lower average adiposity in men and reduced responsiveness to current food insecurity.

This explanation is not definitive, because one can imagine a differently implemented model leading to different conclusions. Furthermore, the sex differences in the mappings between body weight and fitness need to be established empirically. Nonetheless, it illustrates how principled refinement to the model presented here can generate hypotheses for further investigation. Our tentative suggestion on sex differences is at the very least incomplete, because the model parameter values required to make males insensitive to food insecurity also lead to them being extremely lean under all circumstances. Though men are leaner than women, globally, male body weights have increased just as steeply in recent years as female ones (NCD Risk Factor Collaboration 2016). This means that *something* in the environment can drive substantial increases in male body weight, even though that something is apparently not food insecurity. Recourse to candidate explanations other than the IH is required. Once we admit that other candidate explanations are important for men, the door is open to their invocation in women, too. Hence, the failure of the IH for men implies that our explanations for the contemporary distribution of obesity must be multifactorial, with food insecurity playing only a part.

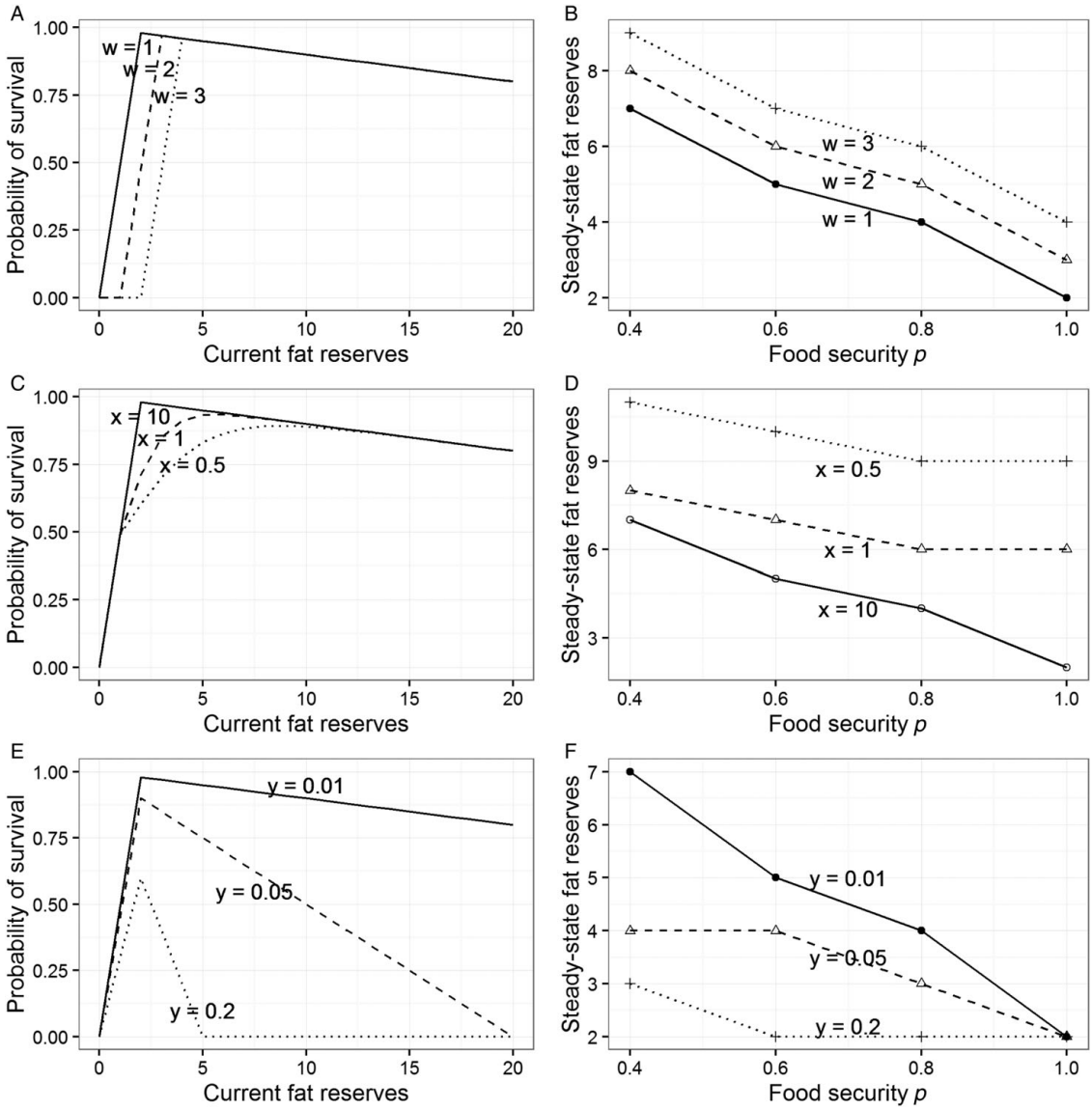

Fig. 3 - B/W online

Figure 3. Modifications to the model from section 3 to explore potential explanations for sex differences. See sections A3.2–A3.4 in Online Appendix A for full details. (A) Three different locations for the cliff-edge below which starvation becomes likely (controlled by parameter w). (B) Steady-state target levels of fat reserves at different values of p for the different cliff-edge locations shown in panel A. (C) Three different shapes of the left part of the survival function (controlled by parameter x). (D) Steady-state target levels of fat reserves at different values of p for the different shapes shown in panel C. (E) Three different slopes of the right part of the survival function, the cost of carrying each additional unit of weight (controlled by parameter y). (F) Steady-state target levels of fat reserves at different values of p for the slopes shown in panel E.

6.3. Why is the association found only in high-income countries?

Our meta-analysis showed that food insecurity predicts high body weight only in high-income countries. In low- and middle-income countries, the average association is zero. In high-income countries, the available diet generally has higher energy density than the food available in lower-income countries (Drewnowski & Popkin 1997). This means that food-insecure individuals will be able to consume high levels of calories in periods when they *do* have access to food, even if these periods are intermittent. In a low-income country, not only might food access be insecure, but also when food is available, it may not be energy-dense enough to allow the buildup of fat reserves before the next period of scarcity strikes.

In section A3.5 in Online Appendix A, we explore the consequences of low energy density of food in our model. We do this by placing a sharp constraint on N, the number of units of energy that can be consumed in one time period when food is available. Constraining N has interesting consequences; when p is low, the steady-state target level of reserves is higher when N is small than when it is large. On the other hand, in simulations, the actual body weights that individuals maintain are much more variable when N is low and are often well below the steady-state target (Fig. 4). This is because, under food insecurity, stochastic periods without food deplete individuals' reserves, and it takes them much longer to build those reserves back up again when food is available, because the amount by which their intake can exceed their expenditure in any one period is constrained. Essentially, in a low-p, low-N world, individuals should aspire to carry

Fig. 4 - B/W online

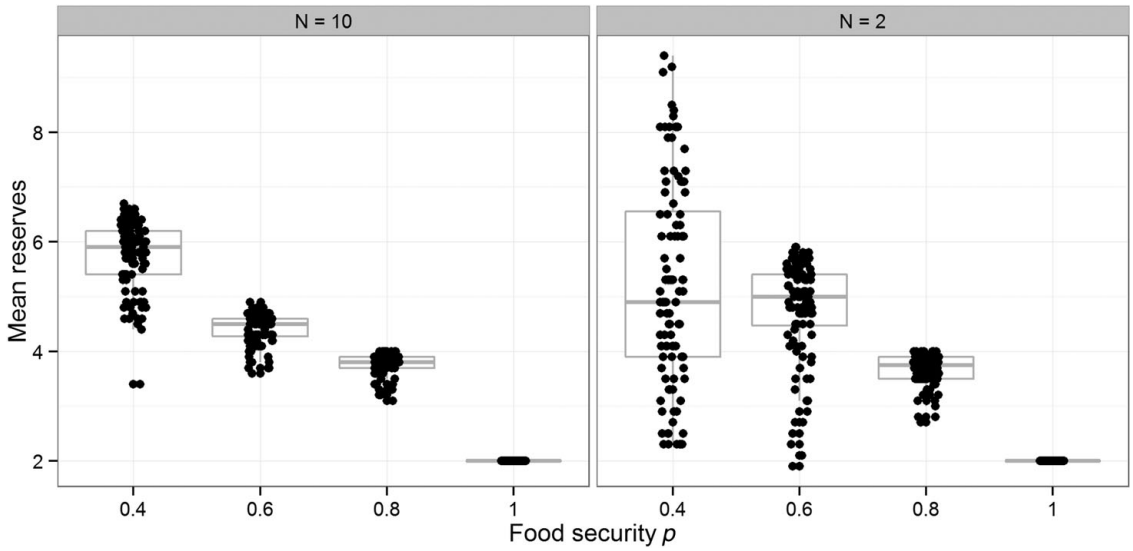

Figure 4. Mean level of reserves over 40 days for simulated individuals experiencing different levels of food security p, for two different values of the maximum energy available from food per period, N. When N is small, p becomes a poor predictor of body weight as the variability between individuals at the same level of p becomes greater. Points have been jittered in the horizontal dimension to make individual data points more visible.

high reserves, but are often unable to get as fat as they should want because their food supply is not energy-dense enough. This means that, when N is small, p becomes a relatively poor predictor of body weight. In the data underlying Figure 4, p predicts 77% of the variance in reserves in the left panel and only 20% of the variance in the right panel.

If the interaction between food insecurity and energy density of food is indeed an explanation for the lack of observed association in low-income countries, then interesting predictions follow. We should predict that food-insecure individuals in these countries would often wish to be fatter than they can actually manage to be. The existence of heavier body ideals in subsistence populations is well documented and stands in contrast to the ideals of thinness typical of high-income societies (Anderson et al. 1992; Tovee et al. 2006; Wetsman & Marlowe 1999). More specifically, Gulliford et al. (2006) found that food-insecure individuals in Trinidad and Tobago did not have higher BMIs than food-secure individuals. However, food-insecure study participants were more likely to report that they were trying to gain weight. Reporting trying to gain weight was quite common in Trinidad and Tobago, whereas it would presumably be very rare in a high-income country.

The idea that, under low-income conditions, the available food is insufficiently energy-dense for food-insecure individuals to maintain high body masses offers a reasonable explanation for why the association is restricted to high-income countries. In fact, we need to go further: The very high body weights seen in high-income countries probably represent the operation of decision-making mechanisms optimized to deal with food insecurity in energy-sparse ancestral food environments in contemporary environments where widely available foods are energy-dense. That is, rather than levels of food insecurity per se explaining the contemporary distribution of obesity, it is the combination of high levels of perceived food insecurity with historically unprecedented energy density of widely available foods; the IH needs to be synthesized with some form of evolutionary mismatch argument to explain the extent of contemporary obesity. Such a synthesis makes sense of why widespread obesity should be an epidemic of affluence, but particularly of affluent countries characterized by high levels of inequality and/or economic insecurity. It also predicts rapid increases in obesity as unequal developing countries make the transition to urban living and an industrialized food supply, as has indeed been observed (Drewnowski & Popkin 1997).

6.4. Why is there no association in children?

Our meta-analysis showed that the association between food insecurity and high body weight was not generally detectable in children, even older children. (In the child samples from high-income countries considered separately, the OR was just significantly different from 1 [1.11, 95% CI 1.01–1.24], though even here, it was significantly smaller than that for adults [1.41, 95% CI 1.30–1.53].) It is not clear how the IH as currently formulated could account for this. There are some methodological issues that may contribute to the absence of a detectable association. First, studies on children generally measure food insecurity through parental reports. Thus, the measure of food insecurity is even further removed from the causal psychological state than is true in studies in adults, weakening the ability to discover a relationship. Second, measurement of fatness in children is itself complicated by growth. Growth trajectories will be related to the food supply, with individuals with better food access tending to grow faster. Simple BMI-type measures may be particularly problematic for assessing adiposity in growing children (Freedman et al. 2005). However, explaining the weaker association in children stands as a challenge to the IH.

6.5. Why are there genetic influences on obesity?

There is abundant evidence of heritable genetic effects on body-weight adiposity (Maes et al. 1997), with a number of specific genetic loci having been implicated through

association studies (Frayling 2013; Locke et al. 2015). At first blush, this seems at variance with the IH, which gives causal primacy to environmental inputs in explaining who gets fat and who does not. However, we do not see the existence of heritable variation as a fundamental challenge to the hypothesis. Genetic variation is ubiquitous in all kinds of morphological and physiological traits. In terms of the IH, we should expect mutation to produce variation in the mechanisms governing weight regulation, such as those perceiving cues of food insecurity and governing the rate of fat storage in response to them (McNamara et al. 2015). In addition, there might be genetically based variation in such parameters as metabolic rate and the mobility costs of carrying extra fat. Thus, the framework outlined in this article not only allows, but also leads us to expect, that genetic variation in such traits would lead to variation in adiposity. When genetic variation is explicitly incorporated into adaptive dynamic models, the predicted outcome is often genotype by environment interactions (Thorpe et al. 1998). Thus, it would be of interest to investigate whether known obesity-proneness genetic variants increase obesity risk under all circumstances or especially where food insecurity is also present.

The genetic variability maintained in the mechanisms underlying weight regulation will be greater if the strength of selection against deviations from optimal weight regulation is relaxed. Such a relaxation of selection in recent human evolution, specifically an elimination of the fitness costs of carrying too much weight over the last two million years, is proposed by the "drifty genotype" hypothesis for human obesity (Speakman 2008). Contrary to Speakman (2008, p. 306), we find it implausible that the fitness costs of high weight have been completely abolished in humans. The claim that ancestral humans completely eliminated predation as a source of mortality cannot be sustained; moreover, the costs of high body weight arise not just from predation but from foraging, agonistic interactions, and many other sources (see Higginson et al. 2016, p. 6). However, it is possible that selection has been *relatively* relaxed in recent human evolution, and that, consequently, there is greater non-adaptive genetic variation in weight-regulation mechanisms in humans than other species. (We note also that weaker selection against carrying slightly too much fat than against carrying slightly too little fat is already integral to the model from sect. 2 and models like it, due to the asymmetry of the survival function shown in Fig. 1A. Thus, within the IH, there is already greater scope for genetic drift of variants that lead to reserves being a little too high than variants with the opposite effect; see Higginson et al. 2016.) In any case, relaxation of selection in recent human evolution would not completely abolish phylogenetically older weight-regulation mechanisms; the basic functioning of those mechanisms should still remain detectable on average, even if there is individual genetic variability in the response (a point on which Speakman 2004 concurs). The IH and the drifty genotype hypothesis could thus coexist in a multifactorial explanation of the contemporary distribution of obesity.

6.6. Overall evaluation

The IH is attractive because of the way it incorporates both the biological and social roots of obesity. It incorporates the biological roots by deriving from well-developed adaptive principles, positing species-typical evolved adaptations, and drawing on comparative evidence from other species. It incorporates the social roots by locating a key proximate cause of obesity in the social-structural factors that lead to some individuals being food insecure within societies that are very affluent overall. The empirical evidence that food insecurity predicts high body weight in adult women in high-income countries is clear, and a reasonable rationale can be given for why it is only in high-income countries that the association can be observed. On the other hand, the lack of an association in men, although potentially explicable, undermines any claim that the IH by itself is sufficient as an explanation for the current distribution of human obesity. The constellation of food insecurity and an energy-dense food landscape is an obesogenic one, but not all contemporary obesity can be explained by the presence of this constellation.

7. Further applications of the IH

In this section, we briefly discuss some possible extensions of the IH to explain other phenomena related to fatness and the management of body weight.

7.1. Understanding developmental influences on obesity

In recent years, it has become increasingly clear that experiences in early life can predispose individuals to maintaining high levels of body fat, not just as children but subsequently as adults. These experiences can include poor *in utero* nutrition (Law et al. 1992; although see Rogers 2003), childhood exposure to food scarcity (Olson et al. 2007), or psychosocial stress more generally (D'Argenio et al. 2009; Greenfield & Marks 2009; Gundersen et al. 2011b; Gunstad et al. 2006b). Such phenomena are not restricted to humans. We have recently found that European starlings *Sturnus vulgaris* made to compete hard for food as nestlings develop into adults with a "hungry phenotype": They are hyperphagic, indiscriminate about what they eat, and heavy for their skeletal size (Andrews et al. 2015; Bloxham et al. 2014). There are similar experimental findings from rats and monkeys (Kaufman et al. 2007; Qasem et al. 2012).

Rather than seeing these developmental phenomena as separate from the IH, we can see them as part of it. Under the IH, the individual's task is to build up an estimate of the likelihood of periodic shortfall in the food supply over its lifetime, so that it can maintain appropriate reserves. Early experience provides the first data contributing to such an estimate. How much importance it makes adaptive sense to give to early life relative to later experience in setting adult phenotype is a topic of active research (Fawcett & Frankenhuis 2015; Frankenhuis & Panchanathan 2011; Nettle & Bateson 2015; Nettle et al. 2013; Stamps & Krishnan 2014). It depends, among other things, on the temporal consistency of environmental conditions. Nonetheless, it is plausible to suggest that the empirically observed associations between early life adversity and later obesity reflect some initial calibration or prior setting of the mechanisms that estimate the dangers of starvation from food shortfall in adulthood.

7.2. Explaining dieting-induced weight gain

A number of studies suggest that restrictive dieting, as a strategy for weight loss, is not only ineffective but also

counterproductive in the majority of individuals (Mann et al. 2007; Pietiläinen et al. 2012; Siahpush et al. 2015). Most individuals who practice restrictive diet regimes regain more weight than they lose, increasing their risk of obesity in the long term. From the food-insecurity perspective, this makes sense. By following a restrictive diet, individuals are intentionally exposing themselves to restricted food availability. Thus, it is very likely that the effect of dieting episodes is to provide the mechanisms governing weight regulation with cues of food insecurity (Nesse 1984; Williams & Nesse 1991). Under the IH, weight gain as soon as food becomes available again is the predicted result.

7.3. Understanding anorexia nervosa

Although obesity is a major public health concern in affluent countries, about 1% of young people in these countries (mostly women) significantly impair their survival chances by maintaining low body weight in anorexia nervosa. Anorexia is defined by a low body mass index, as well as the sufferer imposing a low body mass target on themselves, above which they dread going and feel it would be inappropriate to do so (Bulik et al. 2005). Although a full discussion is beyond the scope of this article, the IH is potentially relevant to anorexia in two ways. First, in terms of aetiology, the hypothesis predicts that anorexia will occur where the person's estimate of their food security is unusually high. That is, if an individual has developed the perception that shortfalls will never occur, he or she should favour an extremely lean body and be motivated to maintain it. We have not been able to find any epidemiological studies of food insecurity in relation to anorexia, but we would predict that anorexia sufferers will be at the high-security end of the spectrum, diametrically opposite the obese. Some support for this prediction comes from the evidence that anorexia risk, in contrast to obesity risk, is highest in families of relatively high socioeconomic position (Goodman et al. 2014). Note that the IH is agnostic about why individuals might have unusually high perceptions of food security; thus, the hypothesis is not incompatible with a neuropsychological literature investigating general decision-making deficits in some anorexia sufferers (Danner et al. 2012). Given that anorexia shows substantial genetic heritability (Bulik et al. 2006), it could be that genetic factors affect the formation of food-insecurity estimates. The hypothesis merely predicts that low perceived food insecurity might be an important psychological mediator between anorexia risk factors and anorexia symptoms.

A second potential area of relevance is in anorexia treatment. If perceived food insecurity is causally important in promoting weight gain, as the IH asserts, then inducing some food insecurity, for example, by randomly varying feeding routines, might be useful in combating low body weight. This is a contentious proposal, because anorexia patients are at considerable risk of starving themselves to death, and the understandable caregiver response is to try to provide all kinds of foods at all times in the hope that the person will eat them. However, it might be that making at least some kinds of food unavailable at least some of the time is a better strategy for motivating long-term gains in body weight. Given that anorexia tends to have a chronic and disabling course, with a tendency of patients to defend and return to their weight-management practices (Abbate-Daga et al. 2013), the food-insecurity perspective deserves further, if cautious, exploration.

8. Implications of the IH

We conclude by considering the implications of the IH. Despite abundant research on human obesity, there is rather little evidence for effective, scalable interventions that prevent obesity or lead to weight loss that is maintained in the long term (Glenny et al. 1997). The IH does not in itself change this situation, of course. However, it ought to change our framing of the problem. If (adult female) obesity results from the psychological mechanisms posited by the IH fulfilling their evolved function, then there is no reason to expect simple information giving, food labelling, or explicit exhortation to be able to override them. Certain interventions, such as restrictive dieting, in fact look potentially counterproductive. Indeed, the IH suggests that the interventions most likely to work are the very antithesis of restrictive dieting: In the words of Dietz's original paper, the IH suggests that "the prevention of obesity in impoverished populations may require increased food supplementation rather than food restriction to achieve a more uniform pattern of food consumption" (Dietz 1995, p. 767).

Perhaps the major virtue of the IH is summed up in the following oxymoron. The IH is a hypothesis about individual decision-making mechanisms, but it ends up pushing the focus in terms of explaining obesity away from individual decisions and onto society. Surely, the key question is why, in countries of historically unprecedented affluence, there are millions of people who feel they might not have enough to eat. These people need not less food, but more: Better food access and less uncertainty in their lives. If the IH has any merit, then tackling these societal problems should lead to a melioration of the obesity epidemic.

Supplementary Material

To view supplementary material for this article, please visit https://doi.org/10.1017/S0140525X16000947.

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Open Peer Commentary

Eating and body image: Does food insecurity make us feel thinner?

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Abstract: Body image distortions are common in healthy individuals and a central aspect of serious clinical conditions, such as eating disorders. This commentary explores the potential implications of body image and its

distortions for the insurance hypothesis. In particular, we speculate that body image may be an intervening variable mediating the relationship between perceived food scarcity and eating behavior.

In the target article, Nettle et al. integrate different domains of research from epidemiology, animal studies, and human psychology to explain eating behavior in humans. We are especially intrigued by the speculations the authors make about the implications of the insurance hypothesis for understanding the etiology of eating disorders, particularly anorexia nervosa. Here, we wish to highlight research from the field of body representations, which we believe has interesting connections with, and implications for, the insurance hypothesis.

Given that it forms the core of the insurance hypothesis, Nettle et al. appropriately focus on the role of food insecurity in shaping eating behavior. However, their model includes one other important variable, which receives less attention, namely, the current fat reserves of the organism. Consider two scenarios. In the first scenario, the probability of finding food equals 0.5, and the organism has substantial current fat reserves (3 units). In the second scenario, the probability of finding food also equals 0.5, but the current reserves are now lower (1 unit). In the model proposed by Nettle et al., these two scenarios would result in a very different eating behavior, even when food insecurity (i.e., the probability of finding food) stayed the same. Thus, to decide whether fat storage is beneficial, an organism must be able not only to predict the food supply, but also to accurately evaluate its current reserves. In other words, to act optimally, the organism needs to know its own body size. It needs a body image.

The distinction between actual levels of fat reserves and the mental representation thereof would be largely academic if body representations were largely veridical. In fact, however, there is substantial evidence for large distortions of body representation in many neurological and psychiatric conditions, most pertinently in the present context in eating disorders such as anorexia (e.g., Bruch 1978; Cash & Deagle 1997), as well as in obesity (e.g., Powell et al. 2010). In the case of anorexia, moreover, body image distortions are a strong predictor of poor prognosis for recovery (Casper et al. 1979) and of relapse following recovery (Fairburn et al. 1993; Keel et al. 2005).

Recent research has suggested that even healthy people maintain highly distorted body representations (for review, see Longo 2017). For example, perceptual abilities such as tactile distance perception (Longo & Haggard 2011; Taylor-Clarke et al. 2004) and position sense (Longo & Haggard 2010; Longo et al. 2012) appear to rely on highly distorted representations of body size and shape. Similarly, distortions have also been found in explicit judgments of body part length (Longo & Haggard 2012) and even in judgments about the spatial configuration of body landmarks (Fuentes et al. 2013; Longo 2015). Further, a clear pattern of body image distortions has been shown in normal-weight adolescent girls, with a tendency to overestimate body width and underestimate body length (Halmi et al. 1977).

Thus, distorted body representations appear to be a normal part of human cognition, as well as being central to serious clinical conditions involving disordered eating. We believe these findings have interesting and important implications for the model proposed by Nettle et al. We speculate that a distorted body image may be an intervening variable mediating the relationship between perceived food scarcity and eating behavior. More specifically, distortions of body image may function to modulate eating behavior: Perceiving oneself as thin may motivate increased consumption, whereas perceiving oneself as fat may discourage consumption.

If both food insecurity and perceived fat reserves (body image) are important contributors to eating behavior, what is the relationship between these two factors? One possibility is that body image and food insecurity are independent of each other, have different causes, and affect eating behavior separately. In this case, body image may modulate the effect of food insecurity influence on fat storage. For example, people who perceive themselves as fat will not eat (or will eat less) even when the food supply is insecure. In the target article, Nettle et al. predict that anorexia occurs when a person's estimate of food security is unusually high, and they propose that introducing food insecurity may promote weight gain in anorexia patients. However, if body image is independent of perceived food insecurity, this proposed treatment may prove inadequate.

Alternatively, body image may be shaped by environmental cues and serve as an intervening variable mediating the relationship between perceived food scarcity and eating behavior. In this case, perceiving the supply of food as secure should lead people to perceive themselves as fat, whereas perceiving the supply of food as scarce should lead people to perceive themselves as thin. In this case, the treatment proposed by Nettle et al. would affect not only eating behavior but also body image. To our knowledge, no research has specifically investigated the relationship between body image and perceived food insecurity. This opens a new possible line of future research and provides a potential way of empirically testing the implications of the insurance hypothesis.

Mapping multiple drivers of human obesity

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Abstract: The insurance hypothesis is a reasonable explanation for the current obesity epidemic. One alternative explanation is that the marketing of high-sugar foods, especially sugar-sweetened beverages, drives the rise in obesity. Another prominent hypothesis is that obesity spreads through social influence. We offer a framework for estimating the extent to which these different models explain the rise in obesity.

The United States is well known as a generally high-income country, but what is less well known is the fact that over a third of Americans are classified as being obese (Ogden et al. 2014) – two-thirds if "overweight" is figured in – with more than 100,000 deaths per year attributed to obesity. Despite the intentions of many to lose weight, the problem has been exceptionally resilient at multiple scales, from individuals who try to change personal habits (DellaVigna & Malmendier 2006) to health or government organizations that address the problem at a population scale (Schroeder 2007). Given their different types and scales of analysis, different social sciences (economics, psychology, anthropology, sociology) tend to favor different explanations for the evolution of dietary habits.

In the target article, Nettle et al. propose a version of the standard evolutionary-psychology hypothesis that modern obesity is a result of high availability of food to Paleolithic hunter-gatherers, who stored calories as fat whenever famine loomed. Nettle et al.'s insurance hypothesis is quite reasonable (Shrewsbury & Wardle 2012) – indeed, storage against food uncertainty is the commonly understood purpose of fat – and falls comfortably alongside numerous other plausible hypotheses for obesity. In community medicine, for example, hypotheses about behavioral change center on information and supply (Guiteras et al. 2015). Regarding sugar, the supply side includes factors such as widespread marketing of inexpensive high-sugar foods, especially sugar-sweetened beverages, that may drive the rise in obesity (Johnson et al. 2007), diabetes (Basu et al. 2013), and heart disease (Kearns et al. 2016). Urban geography, furthermore, can bound food supply, creating high-sugar "oases" within food deserts, causing obesity and diabetes to disproportionately affect the poor.

On the information side is a deluge of food advertising, diet fads, conflicting medical advice, and social-media messaging

(Nestle 2016). If humans make boundedly rational decisions, some might figuratively reside in an *information desert*, where the true costs and benefits of their decisions are too far away and thus not transparent. Similarly, *present bias* favors the affordability and immediate gratification of sugar compared to its longer-terms risks, which are farther away. In this environment of information overload, evidence-based information may have an attenuated effect.

One important question in all this is the role that social learning plays in the rise in obesity. Dietary habits tend to be resilient, embedded as they are in religion, cultural traditions, and nutritional needs. Intergenerational, or vertical, learning, together with family economics, can have lasting effects on dietary choices and thus on obesity rates (Hernandez & Pressler 2014). Recently, a more horizontal social-learning effect has been proposed, the hypothesis being that obesity spreads through social influence (Christakis & Fowler 2013). This hypothesis has received sharp criticism, however, for not distinguishing between social influence and homophily, which can yield the same clustering of obesity in social or kin networks (Shalizi & Thomas 2010). In other words, the observation that a person is 57% more likely to be obese if a friend is obese (Christakis & Fowler 2007) could be the result of either social influence or homophily.

Recently, in a target article in *BBS* (Bentley et al. 2014), we proposed a parsimonious, data-driven heuristic map containing four quadrants that can be used to gauge the relative importance of social influence and transparency of payoffs for any human behavior (Bentley & O'Brien 2016) (see Fig. 1 here). In terms of those two variables with respect to diet, the insurance hypothesis corresponds to highly transparent payoffs and negligible social influence. Conversely, the contagion hypothesis comprises high social influence but little transparency about inherent costs and benefits of a behavior. We have subsequently parameterized our map to estimate the relative strength of these factors from real data (Brock et al. 2014; Caiado et al. 2016).

Putting examples into the quadrants, the insurance hypothesis would be in the upper left corner, characterized by individual choice and transparent payoffs. Cases of homophily would also occur in the upper left. Family dietary transitions would fall into the upper right quadrant, characterized by social learning with a transparent rationale. In this case, transparency is in terms of the prestige of the person from whom one is learning a behavior rather than in terms of the payoff of the behavior itself. In the lower right quadrant is contagion theory, which holds that obesity diffuses through social networks, including friends and family, because there is little distinction in from whom one learns dietary habits. In the lower left quadrant is information overload, where consumers may stand in front of a wall of sugary drinks at a gas station, or even a college-campus store, and just pick one. The position of a given case within one of these quadrants will be indicated by its pattern of behavioral data, through time and across the distribution of options.

Importantly, the different positions also carry different implications for intervention. The upper left would recommend "supply" interventions, such as introducing real grocery stores into an urban food desert. In the upper right, interventions might need to address family traditions, for example, and encourage teaching new dietary habits to children, especially given that older generations have been deliberately misinformed about certain foods. In the lower right, the social diffusion of obesity could be mitigated by somehow altering the social-network structure, which could include social-media approaches. Finally, information overload in the lower left would involve better messaging and communications campaigns that make the benefits of better diets more transparent against all background noise.

None of these strategies is always the case, but our point is they are very different, and without knowing which path to pursue, we'd be all over the map. In summary, we agree that the insurance hypothesis is powerful and valid, and knowing when and where it applies is crucial to making use of it.

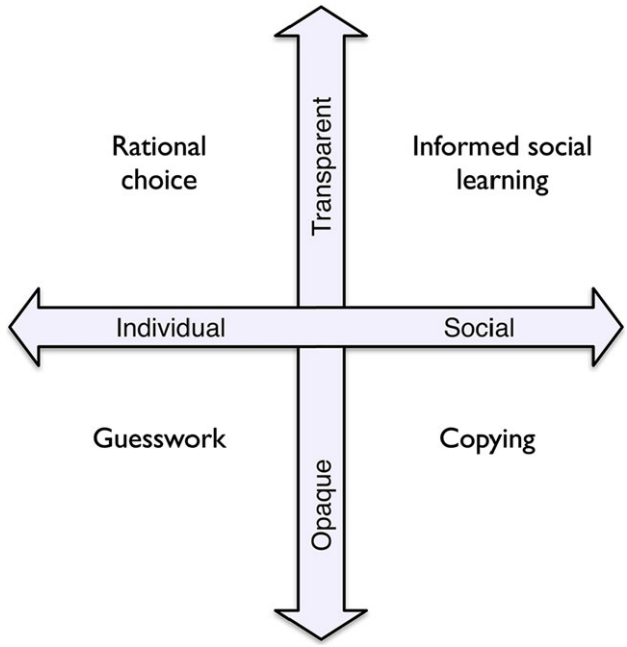

Fig. 1 - B/W online

Figure 1 (Bentley & O'Brien). A four-quadrant heuristic map for understanding different domains of human decision making, based on whether a decision is made independently or socially and on the transparency of options and payoffs. Source: Bentley et al. (2014, Fig. 1).

Expanding the insurance hypothesis of obesity with physiological cues

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Abstract: Food insecurity relates to fat storage, but cannot explain fat storage in excess of levels optimal for buffering – that is, obesity. However, factors related to food unpredictability in the past, including stress, disease, micronutrient content of food, and physical activity, may cue physiological processes that change intake or fat deposition even in the absence of actual food unpredictability.

The insurance hypothesis proposed by Nettle et al. is primarily presented as a psychological hypothesis. Yet, energy intake, hunger, and obesity are not just psychological, but are also physiological processes. Including a consideration of these physiological processes – and, in particular, the cues affecting them – can help expand the insurance hypothesis to relate food insecurity to obesity, even when food is secure, and to incorporate other causes of obesity, including stress, disease, dietary micronutrient composition, and sedentism.

Of these, stress bears the closest relationship to conscious perceptions of food insecurity. Activation of the hypothalamic-pituitary axis and the release of glucocorticoids can induce both increased energy expenditure and increased energy intake, particularly when stress is chronic (Tataranni et al. 1996; Torres & Nowson 2007). Through much of human history, glucocorticoids would have served as reliable cues of both an increase in energy need and an increase in energy unpredictability. Even psychosocial stress would have been related to changes in social

food-sharing networks and thus resource buffering and food security (Gurven et al. 2015). Among subsistence populations, food anxiety, psychological well-being, and actual caloric productivity are closely related (Stieglitz et al. 2014). However, for humans in post-industrial countries, there may often be little linkage between HPA activation and food unpredictability; stressors lead to physiological increases in energy uptake that are not accompanied by changes in expenditure and unpredictability, thus causing the accumulation of excess weight.

A second cue worth considering is immune activation. Immune function is energetically costly, thus frequent immune activation might be expected to serve as a cue signaling greater needs for preparatory energy storage, particularly when coupled with resource unpredictability. In contexts in which disease is actually more prevalent, these excess calories might be used (Blackwell et al. 2016; Gurven et al. 2016). However, if disease cues are not actually linked to disease, then excess energy might again be accumulated. This might be the case if disease is present at early ages but reduced at older ages, similar to other formulations of the thrifty phenotype hypothesis.

Immune activation may also affect intake by increasing the need for particular micronutrients (Cotter et al. 2011). Organisms do their best to optimize the micronutrient composition of their diets, but when micronutrients are limited, excess consumption can be necessary to meet micronutrient demands (Simpson et al. 2004). Internal processes likely monitor micronutrients, allowing food content to cue micronutrient unpredictability, even in the absence of caloric unpredictability. Such cues may also affect perceived unpredictability. For example, the nutritional content of foods affects both energy intake and the experiences of hunger (Fuhrman et al. 2010). Changes in the food supply with industrialization mean that foods are increasingly put together from constituent components, rather than prepared in ways that reflect natural associations between micronutrients present in animal and plant material (Cordain et al. 2005; Pollan 2006). Thus, physiological mechanisms that evolved to balance micronutrients might have a hard time motivating correct intake when the content of foods does not resemble the content of foods in the past.

Finally, physical activity may also serve as a physiological cue, not only to expected energy expenditures, but also to the expected costs of carrying excess weight. Carrying excess weight is only functionally costly when an organism must move to escape predators or acquire food. Through most of human history, mobility would have been critical for survival. Yet, the degree and type of mobility required would have varied between individuals. Thus, we might expect individuals to monitor how much physical activity they engage in and use this as a cue to the future need for activity and thus the expected costs of carrying weight. If such a mechanism exists, sedentism might lead to excess weight gain above and beyond changes in activity energy expenditure, by also affecting things like the rate of adipose deposition. For example, high-density lipoproteins (HDLs) may be part of a mechanism for regulating fat deposition in relation to physical activity, as HDLs are increased by physical activity (Warburton et al. 2006), and they also reduce the deposition of body fat by affecting lipolysis (Wei et al. 2014).

In the mathematical model described by Nettle et al., individuals have perfect knowledge about food security in their environments. Yet, in real life, knowledge is imperfect and might be particularly imperfect for stochastic food fluctuations. Thus, humans must regulate their intake based on inexact cues to multiple variables, including expected energy needs, expected unpredictability of food, and the expected costs of carrying extra weight. A consideration of inexact cues and the systems that interpret them is important because even though a general theory like the insurance hypothesis can describe why we see associations between food insecurity and obesity, it says little about why we see humans exceeding optimal levels of fatness.

Thinking about cues can also help us make clear predictions leading to novel interventions against obesity. Many of the cues discussed here may misfire in modern environments, leading to feelings of food insecurity in the absence of actual caloric insecurity, or conversely, affecting behavior without changing conscious assessments of food insecurity. For example, stress might be associated with obesity only in contexts where it affects perceived food security, but not actual fluctuations in food availability. Similarly, poverty and food insecurity lead people to choose high-calorie, low-micronutrient foods, but we might also predict that the micronutrient density of a person's diet could influence perceived food insecurity, creating positive feedback and exacerbating weight gain.

A complete explanation for the obesity epidemic should explain not only why food insecurity is associated with *increased* weight, but also why it is associated with *excess* weight – that is, why these mechanisms overshoot what might be expected to be adaptive levels. In short, thinking about the cues associated with expected energy needs, expected unpredictability of food, and expected costs of carrying extra weight can help link the insurance hypothesis to other theories of obesity.

Epidemiological foundations for the insurance hypothesis: Methodological considerations

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Abstract: Nettle et al. evaluate evidence for the insurance hypothesis, which links obesity with the perception of food scarcity. Epidemiological findings in this area have generally been weak and inconsistent. The present commentary examines three key methodological issues arising from the literature on the association between obesity and the perception of food scarcity in humans, with suggestions for future epidemiological research.

The target article by Nettle et al. evaluates the evidence for the insurance hypothesis, which posits that obesity in humans is linked to food insecurity, such that the presence of environmental cues that signal food shortage lead to an increase in fat storage. In environments where the presence of food shortage signals is chronic, this process may lead to increased levels of obesity in the population.

To examine this hypothesis, the authors present a review and meta-analysis of the epidemiological evidence of the linkage between food insecurity and obesity in humans. The analysis reveals a robust positive association, but one that is limited to females in high-income nations. The authors make some general comments concerning the limitations of the epidemiological research, but to fully evaluate the insurance hypothesis, it is critical to examine in detail these limitations of the meta-analysis and, in turn, to present an epidemiological approach that would be well equipped to examine this hypothesis.

The first critical limitation is that most of the studies conducted in this area have employed a cross-sectional design rather than a longitudinal design. Although cross-sectional designs allow for the ascertainment of associations and the development of hypotheses concerning the possible causal relationships between food insecurity and obesity, it is not possible to establish causality using studies of this nature. It should be noted that cross-sectional designs may be useful in determining the extent to which any associations between food insecurity and obesity generalize across populations, but these considerations are secondary in comparison to studies designed in a manner to test adequately a causal hypothesis.

A second critical limitation in the literature is that few of these studies have been able to adequately control for possible sources of confounding in the association between food insecurity and

obesity. Nettle et al. report that their meta-analysis did not detect a difference between estimates of association that were adjusted for confounding (socioeconomic status) and those that were not, suggesting that the existing studies may have failed to sufficiently control for confounding. Yet, controlling for confounding is a key factor in the ascertainment of causality; without adequate control for confounding, it is impossible to evaluate whether food insecurity plays a causal role in increasing obesity. The use of longitudinal designs, with repeated measures of both the perception of food insecurity and obesity, would allow the fitting of *conditional fixed-effects models* (Allison 2009; Hamerle & Ronning 1995), which account for all sources of non-observed fixed confounding that influence both food insecurity perception and obesity and which can be augmented by observed time-dynamic co-variate factors during the period of observation. Given the availability of repeated measures data, fixed-effects modeling provides a robust indication of possible causality. Nettle et al. note (in sect. 5.3) that they were able to find only one study that had repeated measures of both food insecurity and obesity (Whitaker & Sarin 2007).

In addition, although the opposite causal pathway (obesity causing food insecurity) seems implausible, it may be plausible to suggest that individuals who are obese have different *perceptions* of food availability than individuals who are not, and that individuals who are obese are perhaps more likely to perceive food sources as insecure rather than secure. To ascertain the direction of causality and rule out the possibility that obesity could be driving an increase in the perception of food insecurity, repeated measures data could also be used to test the likely direction of causality using structural equation modeling procedures (Fergusson et al. 2007; Fergusson et al. 2015) that compare the fit of models that represent (a) a causal pathway from perceptions of food insecurity to obesity, (b) a causal pathway from obesity to perceptions of food insecurity, and (c) a reciprocal causal pathway in which each plays a causal role in the other.

It is clear from the target article by Nettle et al. that an understanding of group differences plays an important role in our understanding of the role of food insecurity in the risk of obesity. However, a third critical limitation is that the studies reviewed have been unable to adequately test important group differences in the association between food insecurity and obesity. Nettle et al. report that their meta-analysis revealed evidence of gender differences, such that the association applied for females but not for males, but was unable to detect any differences related to age or other major group difference (such as ethnicity). Although it may be difficult to design a single study that can adequately test all plausible group differences in the association between food insecurity and obesity, the use of nested designs or multigroup analyses (Boden et al. 2015; Boden et al. 2016; Fergusson et al. 2008; Muthén & Muthén 1998–2012) allows the fitting of models across groups and permits tests of the equivalence of model fit across these groups, using only a single model. Multiple tests of groups' differences may be applied to a single model as well. The signal advantage of this modeling procedure is that it proves possible to directly compare parameter estimates across groups with the model without increasing standard error, thereby reducing model imprecision and increasing the sensitivity of the model to detect effects. A nested or multiple-group model approach can also be combined with the conditional fixed-effects and structural equation modeling approaches detailed above to provide a comprehensive approach to testing the robustness of the association to confounding, the direction of causality in the association, and the ascertainment of critical group differences in the association between the perception of food insecurity and obesity.

Nettle et al. provide a thorough review and analysis of the epidemiological literature concerning food insecurity and obesity. It is clear that, on the basis of this review, there are considerable methodological weaknesses that compromise the robustness of the observed associations. The application of a series of design improvements and modeling procedures would allow a much better understanding of the nature of these associations and provide further evidence to evaluate the insurance hypothesis.

Future research directions for the insurance hypothesis regarding food insecurity and obesity

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Abstract: The focus of this commentary is Nettle et al.'s insurance hypothesis linking food insecurity to a high body mass index (BMI). We discuss how the relationship between race/ethnicity and obesity in the United States is consistent with this hypothesis, then present potential ways forward to elucidate the validity of this hypothesis in humans through rigorous controlled trials.

Nettle et al. note that "high levels of stored reserves ought to be found not among those whose access to food is assured, but exactly among those whose access to food is insecure" (sect. 3.3, para. 4). We agree with this and commend the thorough analysis and discussion presented in the target article that builds on similar concepts presented previously (e.g., the economic insecurity hypothesis in Smith et al. 2007), the resource scarcity hypothesis (Dhurandhar 2016), and others (Kaiser et al. 2012). Herein, we extend the discussion of the insurance hypothesis in two areas given little or no attention by Nettle et al.: (1) the relationship between food insecurity and obesity among racial and ethnic minorities in the United States and abroad and (2) the role of randomized studies to test the hypothesis in humans.

First, patterns of poverty and food insecurity, which disproportionately affect racial and ethnic minorities in the United States and abroad, yield results consistent with the insurance hypothesis. In 2015, 24.1% of Blacks and 21.4% of Hispanics were in poverty, compared to 10.1% of non-Hispanic Whites (U.S. Census Bureau 2016). Correspondingly, food insecurity is higher in racial and ethnic minority populations (Coleman-Jensen et al. 2014; Nord et al. 2005). In 2013, an estimated 10.6% of non-Hispanic White households were food insecure compared with 26.1% of Black households and 23.7% of Hispanic households respectively. In line with the insurance hypothesis, rates of obesity are significantly higher in most racial and ethnic minority groups, though exceptions exist: The estimated prevalence of obesity among Asians (10.8%) is one-third that of non-Hispanic Whites (32.6%) (Ogden et al. 2014). This exception does not contradict the insurance hypothesis: 2015 median income among U.S. Asian families was 18% higher than in non-Hispanic White households (U.S. Census Bureau 2015). The authors also cite the well-documented existence of heavier body ideals in subsistence populations (Anderson et al. 1992; Tovée et al. 2006; Wetsman & Marlowe 1999), suggesting cultural adaptations that complement individual behavioral responses to energetic uncertainty. Similarly, body weight ideals are higher in Blacks and Hispanics relative to the ideals of thinness reported in non-Hispanic White counterparts

(Bennett & Wolin 2006; Chang & Christakis 2003). Although a multitude of factors contribute to racial and ethnic disparities observed in the United States and abroad, the insurance hypothesis suggests an additional contributing factor – energetic uncertainty and adaptations made in response to perceived food insecurity.

Given the complex etiology of human obesity, we wish to emphasize the value of randomized controlled studies in testing the insurance hypothesis and isolating perceived energetic uncertainty as a contributing factor. Although Nettle et al. argue that designs such as the "the stronger 'doubly longitudinal' approach … come as close to the experimental approaches used in birds as is possible with human participants" (sect. 5.3, para. 2), we are aware of at least two published studies that have experimentally manipulated some aspect of social status, a potential contributor to energetic uncertainty, in human populations and tested subsequent obesity-related eating behavior. Both found that experimentally induced lower social status in humans caused consumption of a significantly greater percent of daily calorie needs (Cardel et al. 2016) or total calories consumed (Bratanova et al. 2016). The Moving to Opportunity Study was a randomized social experiment with a voucher-based intervention that allowed the intervention group to move to a nicer neighborhood, and this intervention was sufficient to reduce mean body mass index (BMI) compared to the control group that received a traditional voucher (Ludwig et al. 2011). Further, the consumption of foods believed to be high in calories was higher in participants primed to the concept of environmental harshness (Laran & Salerno 2013). Although the aforementioned studies are clearly related to the insurance hypothesis, neither was a pure operationalization of energetic uncertainty. We believe that other cues of food insecurity, or food insecurity itself, could be manipulated and tested for their effect on food selection and eating behavior, and future research should strive to operationalize perceived energetic uncertainty such that it offers a more direct test of the insurance hypothesis.

Based on recent findings in human nutrition and obesity research, it's clear that simply providing access to more resources and/or education alone is not enough to attenuate the effect of food insecurity on the development of obesity. In some cases, the provision of financial resources, which *presumably* reduce food insecurity, have led to weight gain (Leroy 2013), particularly in individuals who were already overweight or obese. Additional research should identify the predictors of perceived food *security*, whether the experimental manipulation of these and other factors improves one's sense of control over perceived access to their food supply (e.g., gardening, financial planning, and budget-focused dietary interventions; Dhurandhar 2016) and their potential value to a population-level intervention strategy for obesity related to food insecurity in the "obesogenic" land of plenty in high-income countries.

The life history model of the insurance hypothesis

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Abstract: Nettle et al.'s explanation based on the insurance hypothesis applies only to the association between food insecurity and body weight among adult women, but not to the results about there being no such associations among adult men and children. These results may be best understood when the insurance hypothesis is integrated with the life history model.

The target article provides an evolutionary hypothesis for the relationships between perceived food insecurity and high body weight in humans. However, this hypothesis did not receive strong empirical support. That is, (1) the association between food insecurity and high body weight is restricted to adult women only, and (2) there is no such association in men and in children. Their hypothesis needs some adjustments to accommodate existing empirical evidence. In this commentary, we present a revised version of the insurance hypothesis within an evolutionary life history framework to explain these results.

The life history theory provides a theoretical model for understanding how individuals balance the trade-offs between different strategies associated with individuals' growth, development, and reproduction at different stages of life (Del Giudice et al. 2015; Kaplan & Gangestad 2005). An individual's life history strategy is enacted by the coordinated tuning of multiple physiological and psychological systems, which constantly assess environmental constraints and, accordingly, calculate the energetic allocations (Del Giudice et al. 2015). Individuals' life history strategies vary based on differences in their early-life environments. That is to say, early-life experiences may calibrate the life history strategies and, hence, may set life history speed. The different life history strategies may adaptively regulate individuals to cope with ecological challenges present in their local environments. Therefore, cues that access to food is uncertain in the early-life stage may influence individuals' contingent expression of life history strategies, which may subsequently play a role in their approaches to food and dieting in adulthood. We call this the life history model of the insurance hypothesis.

From the life history model of the insurance hypothesis, cues about food insecurity early in life may serve as an assessment indictor to judge the risk of starvation from food shortfall in adulthood. It may be ultimately be indicative of an extreme lack of resources or a hazardous environment where survival is at risk.

For women, these unfavorable cues in early life may promote more immediate reproduction and then induce some behaviors that may promote fat accumulation. This is because female body fat plays a key role in women's fertility regulation – a critical component of life history strategies (Hill et al. 2013; Hill et al. 2014). A woman who intends to give birth has to reserve a minimum store of body fat. Therefore, it is predicted that the unfavorable early-life environments with food insecurity may shape a "faster" life history developmental path, which leads to psychological and behavioral changes that promote body fat accumulation. Some experimental evidence, based on the life history model, support this. For example, women who had poorer childhoods responded to harshness cues in their adult environments by showing a greater desire for food and a diminished concern with calorie restriction and weight loss. Conversely, women who had wealthier childhoods responded to such cues by showing a lower desire for food and an elevated concern with calorie restriction and weight loss (Hill et al. 2013). In addition, a more recent study demonstrated that exposure to current environmental harshness led women from poorer childhoods to idealize a heavier female body size rather than the thin body size typically favored by Western women (Hill et al. 2014).

But for men, the same early-life cues about food insecurity, which promote more immediate reproduction, may prompt behaviors that promote status enhancement rather than fat accumulation (Archer 2009; Del Giudice 2009; Wilson & Daly 1985). This is because the fat accumulation for men may not be advantageous in such unfavorable environments. Conversely, status-relevant enhancement may help them attract more sexual partners and hence access more sexual reproduction opportunities (Betzig 1992; Buss 1989; Kruger 2008). This may explain why there is no association between food insecurity and high body weight in men, as shown in the meta-analysis in the target article.

The life history model of the insurance hypothesis elucidates that the resource-relevant environmental factors may have a

time effect. For example, father absence early in life shows its effect on women's life history strategy at puberty but not right away (i.e., childhood) (Ellis 2004; Ellis et al. 2003). Body overweight is the same. The life history model of the insurance hypothesis predicts that food insecurity experiences in early life do not influence obesity as children, but subsequently as adults. There is empirical support for this revised model. As found in the meta-analysis in the target article, there is no association between food insecurity and high body weight in children. However, ample studies have provided evidence for the link between unfavorable experiences early in life and body weight in adulthood. For example, growing up in a poor household was associated with increased risk of overweight and obesity in adulthood (Olson et al. 2007). In addition, a recent meta-analysis indicated that a harsh childhood environment, reflected in childhood maltreatment, was associated with an increased risk of obesity in adulthood (especially for women) but not in childhood and adolescence (Danese & Tan 2014).

In summary, the life history model of the insurance hypothesis would not predict body weight change in children but would predict it in adults, and the effect would be more visible in women. From this revised model, we can develop more specific research questions to test. For example, although growing up in environments with food insecurity may have an effect on increased risk of overweight and obesity in adult women, the effects may exist only in young adult women who are fertile but not in older adult women. For another example, future studies should test whether body weight is influenced by an early-life environment that is restricted only to shortfall in the food supply or one that is generally harsh or unpredictable.

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Appraising food insecurity

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Abstract: This commentary focuses on the mechanisms underlying the appraisal of food insecurity. I first describe what appraisal is and why it plays a major role in explaining how different individuals consider food supply as more or less secure. I then describe the potential reciprocal links between appraisal and obesity, based on the well-documented evidence that obesity can cause cognitive deficits.

According to Nettle et al., obesity could be partially explained by perceived food insecurity. Given the central nature of this notion in their hypothesis, one may wonder: (1) What are the mechanisms underlying the assessment of food (in)security? (2) Is this assessment sensitive to biases? (3) Does this assessment work similarly in individuals with different body mass indexes (BMIs) – that is, in obese individuals?

The assessment of food (in)security (and more generally, any situation or event), called *appraisal* in affective sciences, refers to the subjective evaluation of an event's significance (e.g., Coppin & Sander 2016). This appraisal process is based on specific criteria, such as relevance (How important is this event or situation?), predictability (How predictable is it?), implications (What are its consequences?), and coping potential (Can I handle it? How well?) (for more details, see Sander et al. 2005). This process could explain how different individuals can consider a similar food supply as more or less secure, and how the same individual can consider the same food supply as more or less secure at two different time points. If certain individuals appraise a given food supply as important and highly unpredictable, if they foresee a potential food shortage as obstructive to their needs, and do not feel like they can cope with the situation in a satisfactory manner, one would predict that these individuals have higher chances of becoming or being obese based on Nettle et al.'s work.

Besides being subjective and variable across individuals and time, appraisal is also sensitive to biases: that is, systematic tendencies to assess events or situations in particular ways. For instance, individuals suffering from particular types of depression tend to systematically underestimate their coping potential (e.g., Scherer 1987; see also Joormann & Siemer 2011). One can imagine similar potential biases in other groups of individuals. Researchers have consistently found lower socioeconomic status to be associated with biases in the appraisal of ambiguous situations, which are considered to be more threatening than individuals from higher social classes (e.g., Chen & Matthews 2001). It has also been shown that social identities influence the assessment of food-related smells (Coppin et al. 2016) and food items (Hackel et al., submitted). Altogether, this suggests that group affiliation could predispose to particular appraisal tendencies, including in the case of food-related items. In the even more specific case of food security, these biases may lead to a less secure assessment of food resources in particular groups. More specifically, appraisal may differ in men and women, and individuals from low- versus high-income countries, which could explain the specificity of the correlation reported by Nettle et al. to women in high-income countries.

Although appraisal often occurs in an automatic fashion (Sander et al. 2005), it depends on an individual's cognitive functions. In the case of obesity, cognitive deficits related to obesity-associated neuroinflammation (e.g., Miller & Spencer 2014) are well documented (e.g., Coppin 2016; Fitzpatrick et al. 2013). Thus, obese individuals tend to show deficits in executive functions, such as working memory and decision making (e.g., Coppin et al. 2014). Moreover, much comorbidity, such as depression, exists (e.g., Carey et al. 2014). Yet, as mentioned earlier, depression is associated with particular appraisal biases. Consequently, once individuals are obese, their appraisal of food security may also be changed by altered cognitive functions and potentially reinforced by biases.

In light of the evidence above, the following vicious circle could occur: Food would be appraised as less secure by some individuals (e.g., because of biases and/or individual and/or group predispositions), leading to increased food intake and weight, itself causing neuroinflammation and cognitive deficits, potentially creating or reinforcing preexisting cognitive biases, in addition to biases from comorbid conditions, leading to even higher BMI. Systematically investigating the appraisal of food security and its association with BMI (including in longitudinal studies) may deliver fundamental insights into cognitive and affective sciences, as well as in obesity research. Although this approach does not constitute directly applicable therapeutic interventions for clinical application, the expected research results may lay the necessary groundwork of knowledge from which such interventions can be derived. For instance, a finding that low predictability of food supply and low coping potential appraisal are associated with higher BMIs might be used by therapists by trying to modify such potentially dysfunctional appraisals in the current environment to reestablish a healthier BMI.

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Episodic memory as an explanation for the insurance hypothesis in obesity

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Abstract: In evaluating the insurance hypothesis as an explanation for obesity, we propose one missing piece of the puzzle. Our suggested explanation for why individuals report food insecurity is that an individual may have an impaired episodic ability to plan for the future.

According to the insurance hypothesis proposed by Nettle et al., "humans possess evolved mechanisms that respond to cues or experiences indicating that access to sufficient food is uncertain by increasing energy intake relative to expenditure, and hence storing more fat" (sect. 6, para. 1). However, given the current "obesogenic" environment of highly available energy-rich food, it is unclear why modern humans, at least in the Western world, might experience food insecurity. One possible explanation is that some individuals have a reduced ability to imagine and plan for the future, and that this may lead to feelings of insecurity regarding the future availability of food.

The ability to imagine and plan for the future is known as "episodic foresight" and is one half of an overarching ability to re-experience the personal past and imagine the personal future, known as "mental time travel" (Tulving 1993). There is now a general consensus that ability in episodic memory and episodic foresight are linked within the individual. They involve common mental processes (Schacter & Addis 2007), engage the same neural substrates (Buckner & Carroll 2007), and are impaired in the same patients (Hassabis et al. 2007; Tulving 1985). They also develop at the same time in children (Suddendorf & Busby 2003; 2005) and decline in the same way in the elderly (Addis et al. 2008). These abilities appear also to be linked evolutionarily. Animals that exhibit behaviour suggestive of episodic memory have also been found to plan for the future (Cheke & Clayton 2012; Clayton & Dickinson 1998; Correia et al. 2007; Raby et al. 2007).

Growing evidence also suggests that mental time travel ability influences and is influenced by obesity. The decision to eat a given food is controlled not only by homeostatic signals, but also influenced by higher neural systems – for example, the hippocampus (Berthoud 2011). The hippocampus is an integral brain region for mental time travel (Schacter et al. 2008; Simons & Spiers 2003), and this ability has been demonstrated to play a key role in consumption regulation (Higgs 2002; Higgs et al. 2008). Patients with bilateral hippocampal damage producing episodic amnesia (a condition that produces severe deficits in both episodic memory and foresight) appear to consume several consecutive meals without reporting satiety (Hebben et al. 1985; Rozin et al. 1998). Furthermore, in rats, selective lesions to the hippocampus produce increased food intake and weight gain relative to intact and sham-operated controls (Davidson et al. 2009).

Research in rodents has shown that both diet-induced and genetic models of obesity are associated with impaired memory function and hippocampal damage. For example, rats maintained on a high-fat, high-sugar diet display spatial memory deficits after only 72 hours, and non-spatial memory deficits after 60 days (Kanoski & Davidson 2010). In addition, rats fed a sucrose or fructose solution for four weeks showed a 40% reduction in hippocampal neurogenesis and increased hippocampal apoptosis compared to the water solution control group (van der Borght et al. 2011). Genetic models of obesity demonstrate similar memory deficits (Li et al. 2002; Winocur et al. 2005) often accompanied by hippocampal dysfunction (Li et al. 2002). Hippocampal dysfunction has also been shown in overweight humans (Jagust et al. 2005; Raji et al. 2010), and there is evidence to suggest this is associated with episodic memory deficits (Cheke et al. 2016; Cournot et al. 2006; Gunstad et al. 2006a). Although there is, to date, little research investigating episodic foresight in obesity, overweight/obese individuals have been shown to have problems with planning tasks, including the Tower of London task (Gunstad et al. 2007; Sweat et al. 2008).

This evidence suggests that obesity is associated with neural and psychological deficits consistent with impaired mental time travel. Such a reduction in the ability to learn from personal experience, and to extrapolate that experience to imagine the personal future, may lead to feelings of uncertainty and insecurity about the environment, and the availability of resources. As such, it may be that, although high-energy food is more available than ever before, psychological uncertainty may give the impression of uncertain resources, which the insurance hypothesis suggests may lead to increased energy storage and consequent body fat.

In summary, we suggest that impaired hippocampal function and mental time travel may be a key mechanism underpinning the insurance hypothesis, creating the perception of limited resources even in a plentiful environment. Furthermore, given evidence that deficits in mental time travel can both precede and follow the development of obesity, it may be that this forms part of a vicious cycle in which memory and planning deficits promote obesity, which itself reduces the ability to remember and plan.

Implicit attitudes, eating behavior, and the development of obesity

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Abstract: Nettle et al. describe increasing food intake (relative to energy expenditure) in response to food insecurity as a key contributor to obesity. I argue that a variety of implicit psychological mechanisms underlie this process to contribute to weight gain. The biobehavioral pathways and the social nature of food selection discussed here are importantly related to food selection and obesity.

Human food selection and its relation to the global obesity epidemic is a complex, multifaceted problem. As Nettle et al. discuss, this complicated public health challenge requires multiple explanations. The authors present a compelling case for food insecurity as a contributor to weight gain and increases in obesity, but note the need for specification as to how this mechanism operates. Here, I argue that a variety of unconscious psychological mechanisms either underlie the relation between food insecurity and weight gain or co-occur as an independent contributor to obesity, in addition to the authors' model. A growing body of research in social and developmental psychology highlights that people's explicit preferences do not always match up with their implicit associations – attitudes that people may not even realize they hold (Greenwald & Banaji 1995). In the strongest case, such attitudes even reflect stereotypes with which a person actively disagrees. For example, even those who explicitly endorse egalitarian views and believe that people from all backgrounds are equally American implicitly endorse connections between social group membership and national identity (i.e., "American = White?" by Devos & Banaji 2005). These associations are importantly related to behavior, including discrimination in hiring, heath care, and judicial decisions (Chapman et al. 2013;

McConnell & Leibold 2001; Rachlinski et al. 2009; Rudman & Glick 2001). Similarly, a variety of implicit processes may underlie human eating behavior or illuminate why food insecurity and weight gain are related. Just as implicit social attitudes may differ from people's explicit beliefs about social groups and stereotypes, conscious food-related goals or preferences may bear little resemblance to the unconscious mechanisms that influence what and how much people eat and the relation between food intake and weight gain.

For instance, living in chronically stressful environments alters regulation of the hypothalamic-pituitary-adrenal (HPA) axis. In turn, hormonal shifts are related to changes in appetite, preferences for comfort foods, and changes in metabolism that can contribute to weight gain (Knutson et al. 2007; Lumeng et al. 2014). These findings point to a complex biobehavioral network that affects not only what people choose to eat (e.g., a comforting food at the end of a stressful day), but also how the body processes those foods and signals the need to eat again by altering the balancing of appetite-inhibiting and appetite-signaling hormones. Chronically stressful environments, which are replete with psychosocial stress, are often also characterized by food insecurity. Therefore, weight gain in food-insecure environments may actually represent a complex network of stress-related reactions, in which food insecurity is just one symptom of a broader environment of stress. Additionally, some foods – specifically those that would contribute to weight gain and are readily available in high-income countries – may have addictive properties. In studies of rats, overconsumption of palatable high-fat foods triggered addiction-like behaviors and neural responses, and was not disrupted by conditioned electric shocks (Johnson & Kenny 2010). Foods that are similarly high in fat, sugar, and salt are readily available to humans, who demonstrate patterns of neural response to these food cues that are similar to neural circuitry observed in drug addiction (Gearhardt et al. 2011). In addition to an instinct to increase consumption when faced with food insecurity, the propensity to eat foods that contribute to weight gain is very difficult to overcome.

In addition to these biobehavioral pathways, eating is a highly social experience (Liberman et al. 2016; Rozin 2005; Shutts et al. 2013). From an early age, food consumption and preferences are influenced by social input. Children eat more of foods that are labeled with socially relevant symbols, including familiar brands and characters (Roberto et al. 2010; Robinson et al. 2007). From infancy through adulthood, people eat more when surrounded by other people or when receiving positive social feedback from caregivers (Lumeng & Hillman 2007; Lumeng et al. 2007; Salvy et al. 2007; 2012). Additionally, social modeling influences food selection across the life span, as people tend to prefer the foods their peers and social in-group members eat (Birch 1980; Cruwys et al. 2015; Frazier et al. 2012; Hendy & Raudenbush 2000; Shutts et al. 2009; 2010). These contextual influences may unconsciously guide adults and children toward particular food choices, including eating more food than they would otherwise or selecting foods that contribute to weight gain, for reasons beyond their own preferences or goals.

The social and biobehavioral pathways outlined here suggest that unconscious psychological mechanisms alter human eating behavior, as well as the body's response to food in the absence of any changes in food intake. Food insecurity is an important indicator that an individual lives in a stressful environment. Along with social influences on food selection, these environments play an important role in food selection, weight gain, and the development of obesity. Finally, implicit in many studies of obesity is that individuals are to blame for their outcomes. When Nettle et al. use phrases such as "decision-making mechanisms" (e.g., sect. 1, para. 1) to describe human eating behavior, this language implies that their model characterizes a deliberate, conscious process. Obesity is highly stigmatized – obese and overweight individuals face discrimination and negative social consequences (Carr & Friedman 2005; Puhl & Brownell 2001; Schwartz et al. 2003). In the case of childhood obesity, practice guidelines imply that parents are to blame not only for not adequately encouraging their children to eat healthy foods and avoid unhealthy foods, but also for improperly restricting their children's eating and pressuring their children to eat (Barlow 2007; Pesch et al. 2016). These issues highlight that people are often blamed for their own weight gain or their children's weight gain, despite the myriad unconscious processes that influence eating behavior and the body's response to food. To productively understand the development of obesity and effective prevention strategies, considering both conscious decisions *and* underlying psychological mechanisms is critical.

A life-history theory perspective on obesity

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Abstract: We extend Nettle et al.'s insurance hypothesis (IH) argument, drawing upon life-history theory (LHT), a developmental evolutionary perspective that documents downstream consequences of early-life exposure to unpredictable environments. We discuss novel evidence consistent with both IH and LHT, suggesting that early-life exposure to unpredictable environments is associated with reduced engagement in weight management behaviors and a greater probability of adulthood obesity.

Nettle et al. provide evidence in support of the insurance hypothesis (IH), which suggests that food insecurity is one key proximate driver of obesity in humans. We resonate with their arguments and agree that the IH likely serves as a partial explanation for obesity in humans. Here we extend their argument and incorporate evidence from another evolutionary theory – life-history theory (LHT; Kaplan & Gangestad 2005) – that is consistent with the IH and provides additional insight into both the proximate psychological processes as well as the more ultimate functional considerations that lead to obesity in humans.

LHT is an evolutionary theory that addresses the presence of adaptive mechanisms calibrated early in life to help people maximize their reproductive potential given contingencies in the local ecology. Work in the LHT tradition finds that, across a range of species, the degree of unpredictability in an organism's early-life environment influences behavior in a variety of domains throughout the life course (Simpson et al. 2012).

LHT is rooted in the idea that organisms have a finite energy budget that must be allocated in a way that maximizes their overall reproductive fitness. Just as IH highlights the key role of uncertainty in determining the prevalence of obesity, LHT emphasizes that an organism's fitness-maximizing strategy is adaptively calibrated to the level of unpredictability it encounters early in its development. A high level of unpredictability signals to the organism that the future is uncertain, which then increases the extent to which the organism invests in short-term pursuits tied to immediate reproduction. A low level of unpredictability, in contrast, signals that the organism can afford to adopt a reproductive strategy marked by a longer time horizon and greater investment in somatic effort over the long-term. These responses to unpredictability are referred to as fast and slow life-history strategies respectively. Thus, life-history strategies vary on a continuum from fast to slow and are designed strategically to optimize an organism's reproductive success given the level of unpredictability in the environment.

Faster life-history strategies are characterized by behaviors that increase immediate reproduction, such as having more sexual partners earlier in life and a higher number of offspring, investing relatively less in each offspring, and displaying a psychological orientation toward taking risks and seeking short-term rewards (e.g., Griskevicius et al. 2013). Conversely, slower life-history strategies are characterized by behaviors that emphasize long-term investment in fewer offspring, such as delaying reproduction until later in life, having fewer sexual partners, and delaying immediate gratification in favor of long-term rewards.

LHT also provides insight into adult eating behavior. Recent work provides evidence that low childhood socioeconomic status (SES) is associated with a tendency to eat even when nutrition is not needed, and that this tendency persists into adulthood (Hill et al. 2016; Olson et al. 2007). In humans, the conditions typically associated with low-SES environments include uncertainty and resource scarcity and thus reflect the type of harsh and unpredictable conditions that promote the development of a faster life-history strategy. Growing up in an environment in which access to an adequate food supply is relatively uncertain promotes eating whenever food is available, regardless of energy need. If the availability of food in the future is relatively uncertain, it may behoove one to consume as much as possible when one has access to it.

Although this strategy may be adaptive when embedded in unpredictable low-SES environments, it is less likely to be adaptive in high-SES environments, which tend to be more stable and characterized by greater access to nourishment. However, LHT suggests that the life-history strategy one develops early in life tends to persist into adulthood. One's life-history strategy is calibrated early in development and often continues to influence behavior across the life course, even if the qualities of one's current environment change (Simpson et al. 2012). Thus, even if individuals who grew up in a low-SES environment are able to improve their circumstances and transition into a more stable, higher-SES environment, their faster life-history strategy still tends to shape their behavior. This ongoing reliance on a faster life-history strategy may, in turn, lead to a continued tendency to eat regardless of level of energy need. This tendency would stand in contrast to that of their counterparts who grew up in higher-SES environments, who are more likely to calibrate their eating behavior to their current level of hunger.

Although initial evidence provides insight into how one's early-life environment can affect adult eating behavior, one limitation of existing work is that it does not test whether early developmental experiences associated with unpredictability are linked specifically to adulthood obesity.

Recent data from our lab speak directly to the link between early-life unpredictability and likelihood of obesity in adulthood. A sample of 400 community participants reported their level of early-life unpredictability, provided a measure of their life-history strategy, described their current engagement in weight management behaviors, and reported their current height and weight (from which we were able to calculate their body mass index). The data closely fit a serial mediation model in which exposure to unpredictability early in development led people to adopt a relatively fast life-history strategy, which in turn predicted reduced engagement in weight management behaviors and, ultimately, a higher likelihood of experiencing obesity in adulthood (Maner et al., under review). These findings suggest that early-life environmental unpredictability may play a key role in determining eating behavior and obesity in adulthood.

These data, as well as other recent work in the LHT tradition, provide evidence consistent with the hypothesis that many behaviors in adulthood – including those that pertain to eating – are adaptively calibrated to environments experienced earlier during development. These findings complement and extend the IH by shedding light on how early-life exposure to environmental unpredictability influences eating behavior and obesity in adulthood. We believe the IH and LHT can inform one another and together provide a richer portrait of life span influences on eating behavior and obesity.

Toward a mechanistic understanding of the impact of food insecurity on obesity

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Abstract: Nettle et al. provide a useful but incomplete analysis of the drivers of obesity. In this commentary, we argue that a dual-motives conceptualization of self-control, together with insights from the psychology of (perceived and actual) scarcity, might help advance a more fine-grained mechanistic understanding of the observed association between food insecurity and obesity.

The work of Nettle et al. on the drivers of obesity impressively integrates theory and data from multiple disciplines, including behavioral ecology, evolutionary biology, and differential psychology. It addresses a pressing public health issue, as the prevalence of obesity is rising worldwide (Ng et al. 2014). However, we think that Nettle et al.'s presentation of psychological theories of obesity is somewhat incomplete and that the explanation of the observed pattern of effects may benefit from a closer integration of current research on self-regulation and self-control.

For many people, restricting food intake represents a typical self-control dilemma. For example, a dieter might feel tempted to eat a delicious dessert although she is trying to lose weight. Thus, the dieter is torn between two motives (Fujita 2011): one that presses for a smaller, more concrete, and proximal reward (i.e., eating the dessert now), and the other that presses for a larger, more abstract, and remote reward (i.e., losing weight). Several studies have shown that conditions of uncertainty are highly relevant for self-control dilemmas such as the one described above, because uncertainty exacerbates temporal discounting – the tendency to prefer smaller immediate over larger delayed rewards (Kidd et al. 2013; Mischel 1961; Mischel & Grusec 1967). In fact, it has been argued that this decreased motivation for long-term rewards under conditions of uncertainty can be highly adaptive (De Ridder & De Wit 2006; Mischel 2014).

In addition to this motivational explanation of why people sometimes prefer immediate rewards over larger delayed rewards, there can also be situations in which people lack the ability to inhibit impulses (Hofmann et al. 2008). For example, Friese et al. (2008) have demonstrated that impulsive influences on eating behavior are particularly increased when cognitive load (i.e., the amount of mental effort being used in the working memory) is high. In a similar vein, cognitive load has been demonstrated to disinhibit dieters' eating behavior (Ward & Mann 2000) and to promote unhealthy choices (Shiv & Fedorikhin 2002). Thus, high cognitive load may undermine people's control capacities such that impulses that are normally held in check "break through" and have an increased impact on eating and drinking behavior (Hofmann et al. 2008; Wiers et al. 2010).

Importantly, cognitive load is often chronically high among people with scarce resources, and the cognitive load caused by poverty might directly explain its connection to unhealthy impulsive behaviors (Mani et al. 2013; Shah et al. 2012; Vohs 2013). By experimentally inducing concerns about financial decisions in a series of four laboratory experiments, Mani et al. (2013) found that thinking about financial challenges reduces cognitive performance among poor but not among rich participants. In a similar

vein, a field study by the same authors among Indian sugarcane farmers demonstrated diminished cognitive function of farmers in times of poverty (pre-harvest) compared to times of wealth (post-harvest). These results suggests that poverty (as well as other forms of scarcity) can cause cognitive costs, and poverty's *cognitive burden* – in terms of reducing available processing resources – takes a heavy mental toll on behavioral regulation (Vohs 2013).

The psychology of self-control, together with insights from the psychology of scarcity, may also explain why the food insecurity–obesity link is stronger for women in high-income countries as demonstrated by Nettle et al.: Globally, women suffer a greater burden of poverty than do men (Gornick & Boeri 2016; Pearce 1978). As a consequence, they also suffer a greater cognitive burden, which may undermine their control capacities for impulsive influences on eating behavior. Moreover, many high-income countries are characterized by the ubiquity of cheap, energy-dense foods (Drewnowski & Specter 2004; French et al. 2001; Hill & Peters 1998; Hill et al. 2003; Rao et al. 2013), and the relationship between the energy density and the energy cost of foods is often inverse, implying that healthier foods and diets are more expensive than less healthy options (Drewnowski & Specter 2004; Rao et al. 2013). The high availability and low costs of energy-dense foods, combined with the cognitive burden that poverty imposes on many people (especially women), may thus account for the observed pattern of increased energy intake. It is noteworthy that questionnaires on food insecurity (Kendall et al. 1995; Nord et al. 2009; Radimer et al. 1992) used in Nettle et al.'s meta-analysis seem to tap into these cognitive aspects (e.g., worry) of insufficient food supply, which explains why the inclusion of income or socioeconomic position does not change the results of their analysis.

Finally, we believe that the current methods for testing the food insurance hypothesis are limited. We agree that more longitudinal evidence is necessary to test whether changes in food insecurity lead to changes in body weight, as suggested by Nettle et al. Future research on food insecurity and human obesity would, however, also profit from more rigorous experimental tests of the underlying processes (e.g., cognitive load) of the food insecurity–obesity link. As in studies testing the basics tenets of self-control (e.g., Friese et al. 2008) and research on scarcity and cognitive functioning (e.g., Mani et al. 2013), controlled laboratory experiments and field studies are needed to test whether food insecurity is causally related to overconsumption and, eventually, obesity.

Potential psychological accounts for the relation between food insecurity and body overweight

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Abstract: We suggest two psychological mechanisms, temporal discounting and feeling of resource scarcity, for explaining the relation between food insecurity and body overweight. We demonstrate how Nettle et al.'s findings could be explained, post hoc, by each of these accounts, suggesting that their data are not rich enough to allow identification of mechanisms that underlie food insecurity and overweight relationship.

Nettle et al. propose the insurance hypothesis to explain the empirical relation between food insecurity and body overweight. The results of their meta-analysis suggest that this relation is significant only among women in high-income countries. The focus of the model on survival motivations falls short of explaining these empirical findings. More importantly, the insurance hypothesis might not explain the data better than alternative psychological mechanisms that are relevant to food consumption and health. To demonstrate this point, we suggest two mechanisms as potential alternative explanations: (1) temporal discounting and (2) cognitive implications of resource scarcity.

Temporal discounting (Frederick et al. 2002) explains seemingly maladaptive behaviors like gaining excess weight by an individual's tendency to be driven by immediate rather than by future costs and benefits (Hall & Fong 2007). Such a short-term view suggests, for example, that families living on welfare allowance might consume most of their budget right after receiving their food stamps, forcing them to shift later to consuming low-price food that is calorie laden and unhealthy (Drewnowski & Darmon 2005). A natural reason for this myopic view and future discounting is that the future is more uncertain than the present. In the food stamps example, unforeseen events might reduce their net benefits (e.g., decrease the stamps' purchasing power), so it might make sense to use them right away. Recent experimental studies have yielded empirical support for this effect of uncertainty on temporal discounting (Epper et al. 2011; Milkman 2012). In the context of Nettle et al.'s analysis, these observations suggest that food insecurity might affect body overweight by facilitating temporal discounting, rather than by activating an "insurance" mechanism against shortage in food supply. The temporal discounting hypothesis does not necessarily predict a gender effect in the data that showed the effect of food insecurity on overweight mostly among women (much like the insurance hypothesis, which requires additional assumptions for explaining this pattern). Yet perhaps a first clue can be suggested by recent findings showing that although obese women exhibit greater temporal discounting than healthy-weight women, this pattern is not observed among men (Weller et al. 2008).

In any event, the current empirical data cannot distinguish between these two very different, even somewhat opposing, mechanisms. Temporal discounting suggests that food insecurity facilitates a short-term view and future discount, whereas the insurance hypothesis suggests that it actually activates a long-term view (insurance against future shortage). More empirical data are needed to test which of the two mechanisms operate in the context of food insecurity.

We now turn to another alternative explanation for the food insecurity – obesity relationship: the psychology of scarcity. Recent evidence suggests that whenever people feel that resources are low relative to their needs, a scarcity mind-set emerges, and it changes the process of making decisions (Shah et al. 2012). Specifically, under conditions of scarcity, pressing needs capture attention (Aarts et al. 2001; Shah et al. 2012). For example, income deficit elicits greater focus on expenses, and hungry or thirsty individuals focus on food- and drink-related cues (Aarts et al. 2001; Radel & Clément-Guillotin 2012). The attentional focus on satisfying scarce resources, mainly those who are needed to assure functionality, might result in boomerang effects. For example, low-income individuals tend to take loans with high interest that help in meeting today's needs, focusing on the loan's benefit rather than on its costs. Yet paradoxically, taking these loans makes future expenses harder to resolve. Food insecurity could be described as a natural form of scarcity, which might lead people to focus on short-term benefits of eating (energy, taste), rather than on its costs (excessive weight), and consequently yield excessive food consumption. In addition, resource scarcity was found to yield poorer self-control (Mani et al. 2013), which also facilitates short-term behavior and excessive consumption.

Another form in which the feeling of scarcity might affect food consumption relates to time scarcity, which might act not only as a cognitive barrier, but also as an "objective" barrier to healthy

food consumption and exercise (as both typically require some investment of time; Jabs & Devine 2006). Given the lifestyle of modern society, time scarcity seems like a natural account for explaining why the food insecurity–obesity relationship was found mostly in high-income countries. In high-income countries, time scarcity coupled with the high availability of ready-to-eat food is associated with increased likelihood of obesity (Jabs & Devine 2006). Furthermore, one might also speculate that time scarcity might also yield the gender effect observed in the meta-analysis data, as women in modern societies tend to experience higher time scarcity than men (Bittman & Wajcman 2000; Mattingly & Blanchi 2003). Once again, the main point we would like to emphasize is that the current data are not rich enough to differentiate between the insurance hypothesis and the scarcity mind-set hypothesis, which suggest very different mechanisms.

Nettle et al.'s data are intriguing but are inadequate for differentiating between the alternative mechanisms we propose here and the insurance hypothesis. One could possibly think of other processes of relevance, such as the projection bias referring to the tendency to overpredict the degree to which future needs and tastes will resemble current ones (Loewenstein et al. 2003). It is also plausible that several processes operate in the current context and interact with environmental variables (e.g., culture, economy) and/or with individual differences (e.g., gender, age). We share the authors' enthusiasm regarding the intriguing relationship between food insecurity and overweight. Moreover, we see much potential in their evolutionary approach. Nevertheless, the question about the exact mechanisms that give rise to this relationship remains open to different interpretations. Additional data collection and experimental analyses are needed for better understanding of the actual forces that drive this interesting connection between food insecurity and overweight.

Towards a behavioural ecology of obesity

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Abstract: Addressing the obesity epidemic depends on a holistic understanding of the reasons that people become and maintain excessive fat. Theories about the causes of obesity usually focus proximately or evoke evolutionary mismatches, with minimal clinical value. There is potential for substantial progress by adapting strategic body mass regulation models from evolutionary ecology to human obesity by assessing the role of information.

Progress in understanding fat storage has followed from research on the adaptive use of energy reserves by non-humans (Wells 2009). We fundamentally agree with Nettle et al. that applying evolutionary thinking to human fattening dynamics will provide insights for understanding the incidence of obesity and other metabolic diseases. Nevertheless, we feel that the insurance hypothesis (IH), as formulated by Nettle et al., misses key nuances that limit its explanatory power unnecessarily and may underpin its failures to capture details of their data analysis. Here, we suggest how progress can be made from building on these foundations.

Missing from the IH is an explicit treatment of information (Dall et al. 2005): Why is it that people living in wealthy countries with social security – making it very unlikely that they will starve to death – store fat reserves as though starving to death is a distinct possibility? In the notation of their model, it is crucial to distinguish actual p (the probability that food is found) from perceived p, and understand how they can come to differ. For instance, Nettle et al. point out that disadvantaged people are more likely to be obese, but fail to consider why their perceived p should be differentially biased. Mismatch hypotheses for humans have recently come under fire. Rather than discard them completely, we suggest a refined IH, which would have to explicitly incorporate information dynamics driven by evolutionary mismatches. As the authors point out, if restriction of food during dieting is taken to influence perceived p, then target fat reserves should increase after dieting, which as we have shown (Higginson & McNamara 2016) is a contrast effect (McNamara et al. 2013).

In Nettle et al.'s model, food insecurity is taken to be $1-p$. Thus, under their model the maximum availability of food is inversely proportional to food insecurity. A refined IH would allow for both food insecurity and current food abundance such as in our models (Higginson & McNamara 2016; McNamara et al. 2015). The simplest way to do this is to vary both p and maximum meal size N. Low p and high N would be food insecure, and high p and low N would be food secure while having the same mean availability.

Human fattening patterns often involve "ratchetting" whereby any given stored fat level and/or body mass is associated with a metabolic profile that "defends" the steady state body condition (fat or lean) from short-term perturbations via compensatory metabolic processes (e.g., Leibel et al. 1995), even when differentially fat individuals share the same nutritional environments. Such dynamics are not captured in any IH model we are aware of and will require the incorporation of factors that have not yet been considered. Models show that gathering information about the environment may be neglected when energy insurance is necessary (Dall & Johnstone 2002), which could provide a mechanism for divergence of actual p and perceived p. Because the central nervous system is costly, natural selection will have exploited the fact that physiological states (such as fat stores) contain information about environmental conditions (Higginson et al., in preparation). Chronic obesity may result from an informational ratchet effect if current state is taken to provide information that in the current environment it is appropriate to store a large amount of fat.

Such information dynamics could underlie the differences we see among populations, not least the lack of effect among children, who may not yet have stable estimates of prevailing levels of food insecurity. On the other hand, perceived p may not be limited to what is experienced within a particular individual's lifetime. There is the possibility that children respond to the experiences of the mother during her life or during pregnancy (epigenetic effects). We expect selection on what the mother passes on and on how offspring respond (McNamara et al. 2016; Wells 2007a). Because different individuals (mothers and offspring) have different experiences, they would have different target body reserve levels. Models of offspring provisioning under the risk of starvation (Dall & Boyd 2002) could be developed for humans. Divergence of metabolic rates may lead to persistent differences among individuals (Mathot & Dall 2013).

Evolutionary ecology theory predicts that individuals with poor prospects should take more risks and discount the future, so there may be similarities in the cause of obesity and the causes of unsustainable debt (Shah et al. 2012), in that low-income people prioritise the present. Nettle et al. posit one hypothesis for why the IH is only supported for women in high-income countries. The behavioural ecology literature on hierarchies (e.g., among birds) points to another explanation: In patriarchal societies, women can be perceived to be in some sense "subordinate" (Acker 1989); they are more likely to suffer in difficult circumstances and so should store more fat.

Strategic body mass regulation theory makes few assumptions about how the adaptive body mass dynamics predicted in any

given scenario are controlled proximately. Most models assume that any decision-making system (hormones, cognition, etc.) is highly flexible such that it can be optimised (Fawcett et al. 2013). But it is likely that animals including humans actually have simple mechanisms that have evolved to perform sufficiently well in most conditions that have been experienced over evolutionary time (McNamara & Houston 2009). Having a highly specific and flexible rule may be costly, and this cost will be traded off against the cost of inaccuracy of decision making: Humans may have evolved inexpensive "rules" that perform well in most environments, but lead to overeating in rich environments (Higginson et al. 2015).

In summary, we need to develop human-specific evolutionary models of body mass regulation that take information use and physiological "rules" into account. We need to work with clinicians, psychologists, and physiologists, among others, which will help incorporate the human-relevant details to build better theory. This could elucidate what aspects of the environment drive overeating and weight gain and provide an evolutionarily informed solution to the obesity epidemic.

Predicting human adiposity – sometimes – with food insecurity: Broaden the model for better accuracy

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Abstract: The target article explores the role of food insecurity as a contemporary risk factor for human overweight and obesity. The authors provide conditional support for the insurance hypothesis among adult women from high-income countries. We consider the potential contribution of additional factors in producing variation in adiposity patterns between species and across human contexts.

In their article, Nettle et al. propose an evolution-based hypothesis to explain why obesity is more prevalent in some human populations than in others. According to their insurance hypothesis (IH), individuals should respond to food insecurity cues by experiencing psychological and behavioral changes that promote increased fat storage. Such changes are posited to be driven by the increased survival afforded by fat stores in buffering against energy shortfalls during times of food scarcity.

We agree with the authors' general supposition that food insecurity should promote increased energy storage, as it can buffer the organism from dropping below levels necessary for survival and/or reproduction if food becomes temporarily unavailable. Indeed, one of the primary selective forces that has granted organisms the ability to store energy in the first place has been the advantages it provides organisms when food is scarce, variable, or both. However, a good theory proves its mettle by parsimoniously accounting for existing patterns of data and generating new predictions, and the IH falls short in both regards. In particular, the IH seems to be missing important parameters that would allow it to account for (and make novel predictions about) (a) sex differences in the relationship between food insecurity and adiposity observed in some species, such as humans, but not in other species, such as birds, and (b) observed differences between developed and developing nations in the degree to which food insecurity predicts adiposity. We elaborate on these points below.

To evaluate their hypothesis, Nettle et al. present a theoretical model of optimal eating behavior under varying conditions of food security. The authors then reviewed evidence suggesting that food insecurity causes weight gain among species of birds. This is followed by a meta-analysis of research examining the association between food security and body weight in human populations. The relatively straightforward association between food insecurity and adiposity predicted by the authors' model accounted well for the pattern of results obtained in birds. Male and female birds demonstrated increased storage of adipose tissue when in resource-scarce conditions compared to when in resource-abundant conditions. In humans, however, the association between food insecurity and body weight was highly conditional. Specifically, the predicted association was only observed for women in high-income countries and did not emerge until after the post-pubertal transition.

One of the strengths of theoretical models in evolutionary biology is that they can be used to make nuanced predictions about differences one should expect to observe between species, between the sexes, and even between individuals within a species by considering the target organism's life history, mating system, ecology, obligatory investment in offspring, and so on. Unfortunately, the IH does not capitalize on these theoretical strengths. Instead, the authors describe an overly simplified model in which optimal eating behavior is determined largely by food security or availability, current fat reserves, and the probability of death by starvation or other causes. However, there are at least two factors known to play key roles in determining adiposity that have been excluded from the main theoretical model that, if added, would go a long way in accounting for the observed differences between men and women, humans and birds, and people living in high-income and low-income countries. In particular, we recommend that the authors more deliberately consider (1) the importance of body fat for reproduction, and (2) environmental and social factors impacting access to food among the poor.

Bearing on the first of these points, the authors need to consider the relative importance of body fat for successful reproduction in males and females. In the eyes of evolution, reproduction is at least as important as survival. Accordingly, an organism's energy regulation mechanisms should be finely tuned to ensure, specifically, that the organism is able to meet the energy requirements needed for reproduction. This requirement is likely to vary considerably depending on the sex and species that is being considered. For an internally gestating human female, for example, the energy demands of reproduction are far greater than those for the non-gestating, non-lactating, human male. Indeed, research finds that women's ovarian function, gestation length, offspring birthweight, and milk production are each sensitive to women's energy balance and are optimized when energy is readily available. The quantity and viability of men's sperm, on the other hand, are relatively unaffected by men's energy status unless they approach the starvation threshold (Ellison 2003; Fontana & Torre 2016).

Similarly, because successful reproduction imposes on internally gestating mammals vastly different demands than those that are imposed on an egg-laying avian species, we should expect cross-species differences in fat regulation mechanisms that reflect these differences. Perhaps reproduction is optimized among birds when both males and females possess a certain amount of body fat due to the role played by each in egg incubation. Incorporating the energy demands of reproduction into the IH model would undoubtedly go a long way in accounting for why the predicted effects apply selectively to human women, but generalize across male and female birds. It would also likely generate novel predictions about how males and females of other species adjust their fat stores in response to food insecurity, as well as make predictions about the species in which sex differences are expected and those in which they are not.

As a more minor point, we also encourage the authors to consider adding to their model a society-level parameter capturing access to calories among the poor. Access to food among the poor is higher in high-income countries than in middle- and low-income countries

(e.g., see Levine 2011). Accordingly, mechanisms that increase fat storage in response to resource scarcity are more likely to promote obesity in these high-income countries because their poor have the opportunity to overeat. Among those living in lower- and middle-income countries, these same mechanisms would not promote obesity because they would be operating in an environment similar to their selective context (Prentice 2001). Adding this parameter would likely lend additional flexibility to the authors' model, making it more predictive of observed obesity patterns.

Anti-fat discrimination in marriage more clearly explains the poverty–obesity paradox

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Abstract: The target article proposes the insurance hypothesis as an explanation for higher levels of obesity among food-insecure women living in high-income countries. An alternative hypothesis based on anti-fat discrimination in marriage can also account for such correlations between poverty and obesity and is more consistent with finer-grained analyses by marital status, gender, and age.

Nettle et al. suggest that there are few integrative explanations for disparities in obesity among humans. In response to this apparent gap, they propose the insurance hypothesis, arguing that the human body is adapted to store more fat when faced with cues of uncertainty in food supplies. An empirical keystone of their argument is the well-established observation in high-income countries that women are more likely to be obese in situations of food insecurity. Public health researchers often refer to this phenomenon as the poverty–obesity paradox. A positive association between obesity and food insecurity is consistent with the insurance hypothesis, but it is also consistent with a number of other explanations neglected by the target article.

Some of these neglected explanations for the poverty–obesity paradox share deep similarities with the insurance hypothesis by proposing that certain food environments drive people to store more fat. Different versions of such resource-driven theories argue that deprivation can lead people to choose energy-dense or protein-poor foods that are cheaper per calorie but that are also less satiating and thus more likely to cause people to consume excess calories (Hruschka 2012). Such explanations are popular in the fields of nutrition, public health, and a number of social sciences. However, as the target article concedes for the insurance hypothesis, they cannot easily account for the absence of the poverty–obesity paradox among certain groups – such as men or children.

By contrast, an alternative explanation based on female-biased anti-fat discrimination in marriage can better account for both the presence of the poverty–obesity paradox among women *and* its absence among men and children by positing that marital choices sort some obese individuals into poorer households (Hruschka 2017; Han & Hruschka, submitted). Importantly, this explanation does not require that people pursue and achieve different weight goals. As long as there is variation in obesity and wealth, if wealthy individuals choose thinner partners and thinner individuals choose wealthier partners, correlations between individual obesity and household poverty can arise. Recent studies of heterosexual marital preferences in the United States suggest that men, on average, have stronger preferences for thinner partners, and women have stronger preferences for wealthier partners (Oreffice & Quintana-Domeque 2010). In such situations, we should observe heavier women married to poorer men and thus living in poorer households. However, we should not expect heavier men married to women with lower incomes. This gendered prediction is fully consistent with the gender-specific correlations described in the target article. Moreover, if correlations of adult obesity and poverty arise through marriage, then there should be little association of childhood obesity with household income, except perhaps due to propensities inherited from parents.

Resource-driven theories (including the insurance hypothesis) and explanations based on anti-fat discrimination in marriage also make contrasting predictions about individuals who have never married. According to resource-driven theories, there is no a priori reason why the relationships between food insecurity and obesity should differ by marital status. By contrast, anti-fat discrimination in marriage would only predict positive correlations between food insecurity and obesity among those individuals sorted into households through marriage. Conversely, among individuals who have never married, we should observe no such correlation.

Only one study to our knowledge has explicitly tested whether the association of food insecurity and obesity varies by marital status (Hanson et al. 2007). As expected from theories of anti-fat discrimination in marriage, the study found an association among women who had entered marriage at least once in their lives, but not among never-married women (Hanson et al. 2007). We find similar results when looking at obesity and another proxy of poverty – household income – in nationally representative samples from both the United States and South Korea (Hruschka 2017; Han & Hruschka, submitted).

The target article also confirms prior work that the poverty–obesity paradox is largely confined to high-income settings, and that for the 80% of contemporary humanity living on less than $10 (USD) per day, increasing income leads to greater weight gain (Hruschka et al. 2014). A simple dual process model may be able to account for these varying associations of income and obesity (Hruschka 2017). The first underlying process is simply that people accrue more body fat as they encounter increasing abundance. However, as people become richer, any additional resources have less of an effect until the relationship between resources and body fat becomes effectively flat in the highest-income countries. The second process – sorting of heavier individuals into poorer households through marriage – is weaker and only becomes apparent in higher-income settings where the first process no longer creates a noticeable association between resources and obesity. Importantly, this second process will only take place when there are specific, gendered cultural preferences for thinness and wealth.

In sum, resource-driven theories positing that deprivation or uncertainty lead to greater obesity are inconsistent with findings from men, children, and never-married women, as well as the poorest 80% of contemporary humanity. On the other hand, the theory of anti-fat discrimination in marriage markets is consistent with demographic patterns in a number of high-income countries and is readily integrated into straightforward models of weight gain resulting from increasing resources.

Here we focus on the empirical keystone of the target article's argument – the poverty–obesity paradox in high-income settings. However, the authors also briefly describe additional puzzles that might be consistent with the insurance hypothesis. Some of these empirical patterns – the association between societal inequality and obesity – are not nearly as reliable or well-established as the poverty–obesity paradox. Meanwhile, other puzzles might be explained by the misuse of body mass index as a measure of adiposity across human populations when comparing, for example, Japan and the United States (Hruschka et al. 2014; Hruschka & Hadley 2016). Further work that lays out all potential hypotheses on a level playing field and identifies contrasting predictions to investigate with finer-grained data will lead to a better understanding of what best accounts for these puzzles in the distribution of obesity.

Committed to the insurance hypothesis of obesity

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Abstract: Can ideas about the regulation of body mass in birds be used to explain the breakdown of regulation associated with obesity and anorexia in humans? There is no evidence to think so. Medicine can always benefit from the application of evolutionary ecology ideas, but we must be prepared to dismiss these ideas when they just do not fit the data.

It is always inspiring when evolutionary ecology is applied to enduring problems in human health. Birds adaptively and carefully regulate their body mass. When food supplies are unpredictable, birds carry more fat reserves, just in case, but when food is regularly available, they remain lean and save themselves the cost and risk of flying around carrying all that extra weight. Nettle et al. use this paradigm about optimal body mass regulation in birds to attempt to explain the complete failure of body mass regulation associated with obesity in humans.

The authors first present the evolutionary mismatch hypothesis (EMH), which is based on the premise that humans evolved in, and are adapted to, conditions different from the conditions that we face today. The EMH applies to many aspects of human biology, not just to obesity. As related to obesity, the EMH has at least two variants: namely, the thrifty genotype hypothesis (Neel 1962) and the drifty gene hypothesis (Speakman 2008). These hypotheses are not mutually exclusive, and neither one is considered to be perfect or complete. Nevertheless, Nettle et al. dismiss the EMH – probably just Neel's formulation – because, they argue, if the hypothesis were complete, then "all humans living under conditions of affluence should be overweight or obese" (sect. 2, para. 2). Tobacco companies could use this logic to dismiss smoking as a cause for lung cancer because not everyone who smokes ends up developing cancer. Clearly, other factors are also involved.

The authors mention two other problems with the EMH: differences in obesity rates among countries and between the sexes. However, differences among countries could be easily explained by culture and diet (Dinsa et al. 2012; Shetty & Schmidhuber 2006). Similarly, the fact that in high-income countries obesity is more prevalent in women of low socioeconomic status could easily be explained by cultural and sociosexual factors (Brown & Konner 1987; Kanter & Caballero 2012). Neither EMH variant is perfect or complete, but both are closer to being so when other factors are considered. Nevertheless, Nettle et al. dismiss them and propose instead the insurance hypothesis (IH).

The IH posits that when food supplies are unpredictable, humans act like birds and carry more reserves. However, compared to our ancestral conditions when food supplies were indeed unpredictable, food supplies today are plentiful and dependable. Hence, the IH's most general prediction is that obesity today should be low compared to our ancestral conditions. This general prediction is not even mentioned, but it is clearly unsupported.

The authors present support for the IH from a variety of studies, but just cannot avoid the problem that a lack of regular access to food is always associated with a myriad of other socioeconomic factors. A comprehensive meta-analysis reveals that a positive relationship between food uncertainty and obesity occurs only in women (not in men, or children) and only in high-income countries (not in middle, or low-income countries). That is one case out of a possible six.

In section 6, Nettle et al. indicate that the IH proposes that humans evolved mass regulation mechanisms that might have no relevance in today's society, and that the hypothesis says nothing about obesity being currently adaptive. Hence, the hypothesis is non-adaptive, or even maladaptive, and despite what the authors suggest, not an alternative but, rather, another variant of the EMH. One difference is that the other hypotheses, even if based on ecology, have physiological and genetic aspects to them, whereas the IH is strictly an ecological hypothesis. Also, the other hypotheses are based on historical changes in food abundance and quality, whereas the IH is based on differences in the predictability of food supplies.

In section 6.2, the authors try to explain the aforementioned one-out-of-six results, specifically the lack of an effect in men. After proposing and rejecting two "strawman" explanations, they settle on the idea that because males needed to hunt and fight and females did not, carrying extra mass was more onerous for males than for females. Even if we accept this additional explanation, it could also be used to explain the problems with all versions of the EMH. Instead of rejecting the IH, the model is adjusted so that it now fits better with the data. Oddly enough, the modified model predicts "no effect" between food uncertainty and obesity in males. We are left with a gap. Given that obesity does occur in males, a completely different hypothesis is clearly necessary to explain obesity in men, but none is proposed.

In section 7.1, Nettle et al. point out that food scarcity early in life predisposes people to obesity later in life, and argue that this developmental effect is congruent with the IH. This extension of the IH confuses scarcity with predictability. The IH deals with food predictability, not scarcity, and scarcity early in life says nothing about predictability later in life. However, two other explanations of obesity, the thrifty phenotype hypothesis (Hales & Barker 1992) and the thrifty epigenome hypothesis (Stöger 2008), both posit that scarcity early in life predisposes people later in life for metabolic disorders, including diabetes and obesity.

Finally, the authors twist and contort the IH to try to explain anorexia nervosa, without considering more parsimonious and better-supported alternative evolutionary hypotheses (Abed 1998; Guisinger 2003; Lozano 2008; Surbey 1987). The IH specifically predicts that people who are certain about their food supply should maintain a relatively low fat load, but not that they should develop anorexia nervosa.

In summary, Nettle et al. begin and remain fully convinced of the validity of the IH. One wonders what evidence would have been sufficient for them to reject their hypothesis. The hypothesis that birds carefully regulate their body mass depending on the variability of food supplies is logical and well supported. Unfortunately, this hypothesis clearly cannot be extended to explain the complete failure of body mass regulation that leads to obesity in humans. It is promising when researchers try to make sense of human biology using the light of evolution, but disappointing when the light's brightness prevents them from seeing their own data.

Social nature of eating could explain missing link between food insecurity and childhood obesity

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Abstract: We suggest that social factors are key to explain the missing link between food insecurity and obesity in children. Parents and public institutions are children's nutritional gatekeepers. They protect children from food insecurity by trimming down their consumption or by

institutional support. To gauge children's food insecurity, evaluations across the different nutritional gatekeepers need to be integrated.

The insurance hypothesis offers an intriguing environment-based account of the global obesity crisis. Considering the mismatch between ancestral food scarcity and the unprecedented energy-density of contemporary food environments, it attributes socioeconomic differences in obesity to an evolved adaptive mechanism. Specifically, individuals are hypothesized to store more fat when cues indicate that access to food is uncertain, thus buffering against future shortages. The authors acknowledge the multicausality of obesity, with no single factor explaining all or most of the variance. Against this background, they also consider the role of genes and psychological factors such as impulsivity and inhibition in explaining the complex phenomenon of obesity. However, one important factor is missing from their account: the role of others, namely, nutritional gatekeepers.

Although Nettle et al. emphasize the role of society in explaining obesity, they depict food choice – perhaps the single most important behavioral act associated with obesity or lack thereof – as an individualistic decision. They thus overlook social dimensions that are crucial in explaining some of the perplexing patterns surrounding obesity. Eating is not a solitary intake of energy but often a social activity shaped by others' dietary behavior and choices (Herman et al. 2003). "Company" literally means "with bread" – company is those with whom we break bread. Few, if any, health-related behaviors are as closely embedded in the social context as eating – especially where children are concerned. By the age of 10, a child has eaten about 10,000 meals, most of them in the company of their family. Yet, children rarely enjoy autonomy in their food choices. Nutritional gatekeepers – not only parents and grandparents, but also institutions (e.g., kindergartens, schools, policymakers) determine the food choice architecture. One of the most important food contexts for children is the family, with nutritional gatekeepers determining more than 70% of what the family eats (Wansink 2006) through, for example, their economic resources (Keane et al. 2012), family mealtime practices (Dallacker et al., under review), nutritional knowledge, and numerical abilities, which are associated with comprehension of nutrition labels or portion size estimation skills (Dallacker et al. 2016; Mata et al. 2008).

Why do the authors not find a relationship between food insurance and obesity in children? We suggest that recognizing the social nature of eating – and, in particular, of children's food choices – can offer answers for this missing link. Not all participants in the shared activity of eating (e.g., Sobal & Nelson 2003) will be equally affected by (perceived) food insecurity. Despite eating at the same table, the last-born child is often less well-nourished than the first-born. For example, in a family of seven, the height for age of the last-born child is up to 2.5 standard deviations less than that of the first-born (Hertwig et al. 2002). Yet ethical norms and legal policies aim to protect children from malnutrition or starvation. For example, mothers report abstaining from food to ensure that their children are adequately nourished (McIntyre et al. 2003; Piperata et al. 2013). In wealthy societies, institutional settings such as daycare centers and schools often provide free lunches or free milk to children from impoverished families. In the United States, for example, 16 different food assistance programs were funded in 2002, and one in five Americans participated in them at one point during that year (Fox et al. 2004). Thus, relatively rich societies aim to protect children from the detrimental effects of food insecurity through social norms, welfare assistance, and institutionalized arrangements. Admittedly, despite these efforts, even high-income countries appear to suffer from "hidden hunger" and malnutrition caused by vitamin and mineral deficiencies that threaten to impair children's intellectual and physical development (Biesalski & Black 2016).

The authors suggest a methodological explanation for the missing link between food insecurity and body mass index: The studies included in the meta-analysis measured a child's food insecurity through parental reports, which are likely to differ from the child's perception (Connell et al. 2005; Fram et al. 2011). Importantly, this is not only a methodological, but also, again, a social explanation. As described above, wealthy societies aim to protect children from hunger and food poverty both within the family and beyond (Fox et al. 2004). As a consequence of the multiple individual and institutional nutritional gatekeepers involved in children's nutrition, parental perceptions – being just one piece of the social puzzle – may not be a veridical and integrative proxy of a child's food (in-)security – even more so when parents equate food security with lack of hunger and thus neglect the risk of malnutrition.

To conclude, eating was, has been, and will likely continue to be a shared activity – not always, but often. Any comprehensive model of obesity therefore needs to account for the social nature of food choice and consumption. This is particularly the case for children, whose food choice autonomy is restricted. The authors did not find a link between food insecurity and children's obesity. The reason could be that this link simply does not exist or is relatively weak because parents, institutions, and policymakers buffer children from food insecurity. Alternatively, a link may exist, but it may be moderated by who is competent to gauge children's experience of food security or lack thereof: the children, their parents, institutional settings, policymakers? A stringent test of the food insecurity hypothesis in children demands that proper attention be paid to the social dynamics of food choice and eating.

A game theory appraisal of the insurance hypothesis: Specific polymorphisms in the energy homeostasis network as imprints of a successful minimax strategy

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Abstract: The existence of specific polymorphisms in genes of key hormones of the energy homeostasis network that have been shown to predispose to obesity and the so-called metabolic syndrome provides further biological support for the proposed insurance hypothesis. In a broader sense, such polymorphisms can be understood as biological imprints of an evolutionarily successful minimax strategy employed by ancient *Homo sapiens* subpopulations in a one-player game against nature.

I congratulate Nettle et al. for their tour de force not only in describing the basic tenets of their new insurance hypothesis, but also in attempting to provide empirical evidence for the basic assumptions employed by this new theoretical paradigm. The existence of specific polymorphisms in genes of key hormones of the energy homeostasis network that have been shown to predispose to obesity and the so-called metabolic syndrome (Chang et al. 2016; Mărginean et al. 2016; Mora et al. 2015) provide further biological support for the proposed insurance hypothesis. In a broader sense, polymorphisms in the genes of leptin, adiponectin, resistin, peptide YY, and ghrelin (Meier & Gressner 2004), for example, can be understood as biological imprints of an evolutionarily successful minimax strategy employed by subpopulations of ancient *Homo sapiens* to optimize their energy homeostasis in the face of uncertainty regarding the future availability of food resources.

The minimax theorem was initially proposed by John von Neumann in 1928 (von Neumann & Morgenstern 1947). In basic terms, the minimax strategy, which is essentially risk-averse, focuses on avoiding the worst outcome, although it may not necessarily lead to the best possible outcome. In the so-called one-player games against nature (which will be the focus of this discussion), the terms *minimax* (minimizing the opponent's maximum payoff) and *maximin* (minimizing one's own maximum loss) can be used interchangeably as, by definition, nature has no payoffs.

Table 1. Top: *Payoff matrix for the proposed one-player game-against nature. The subjective expected utility is represented by cardinal payoffs (from 0 to 100U [utility units or Utils]).* Bottom: *The constructed "regret table," which can be used to estimate the optimal option according to the minimax regret principle.*

Payoff Matrix		*Food availability (nature's outcome)* **Food scarcity**	**Food abundance**
Fat-storage levels (player's decision)	**High storage (obese profile)**	90U	60U
	Low storage (skinny profile)	0U	100U
Regret Table		*Food availability (nature's outcome)*	
		Food scarcity	**Food abundance**
Fat-storage levels (player's decision)	**High storage (obese profile)**	0U	40U
	Low fat storage (skinny profile)	90U	0U

The minimax strategy stands in opposition to other possible rational alternatives, such as the maximax strategy, a risk-taking option that involves selecting the alternative that may lead to the maximum available payoff; the so-called minimax regret strategy, a *par excellence* risk-neutral strategy that focuses on minimizing the maximum regret; and the principle of expected utility maximization, which is usually employed when nature's probability distribution is known and which recommends choosing the highest expected payoff after weighting each contingency by its probability.

In a recent study (Chen & Ho 2014), it has been demonstrated that minimax strategies are widely used by complex biological networks to maintain a robust stability of the phenotypic trait of interest in the face of random genetic variations and unpredictable environmental disturbances. It has already been shown that evolutionary game theory can be successfully used to model the evolution of several biochemical systems (Pfeiffer & Schuster 2005; Schuster et al. 2008; 2011; Zhu et al. 2016). Alternatively, it is also possible to model the evolutionary behavior of individual biochemical systems as a whole, focusing on the desired phenotypic trait and using standard game theory approaches. In this sense, the scenario of interest in the target article can be represented as a one-player game against nature (Milnor 1951) (Table 1, top). In such a situation, in the face of uncertainty regarding the future availability of food resources, the minimax strategy would lead to the development of a robust energy-conserving phenotype focused on avoiding the worst outcome (i.e., starvation to death), although it could potentially lead to suboptimal general health outcomes due to obesity-induced diseases.

In the proposed one-player game against nature representing the food-availability versus fat-storage levels scenario (Table 1, top), the minimax principle would recommend that the player choose the option of high levels of fat storage (obese profile), which would possibly yield the best of the worst possible outcomes (Table 1, top – first row, second column: 60U). The maximax principle, conversely, would recommend that the player choose the option of low levels of fat storage (skinny profile), as it is the option that could possibly lead to the best outcome (Table 1, top – second row, second column: 100U). As it can be easily noted, although possessing some degree of rationality, the maximax principle (which is sometimes sarcastically described as "aim for the best, but get the worst") is inherently weak, as it disregards the probability distribution of nature's outcomes, being often adopted by naive decision makers such as young children. The maximum regret for each option can be calculated by first estimating the maximum profit for each one of nature's outcomes in the initial payoff matrix (i.e., 90U for food scarcity and 100U for food abundance). Then a new "regret table"' (Table 1, bottom) is constructed by subtracting the largest payoff obtained for that specific column by each cell's value. Ultimately, the value in each cell will represent the maximum regret for that specific outcome. In this case, the minimax regret principle would recommend the choice of high levels of fat storage (obese profile), as it is the one that minimizes the maximum regret (Table 1, bottom – first row, second column: 40U).

Taking into account the discussion above, I believe that instead of an exclusively psychological mechanism that, either consciously or subconsciously, leads to suboptimal prediction of the future availability of food resources, the insurance hypothesis proposed by Nettle et al. should be understood as a more deeply rooted mechanism involved in the evolutionary dynamics of human biology. In such a scenario, specific polymorphisms in the genes of key hormones of the energy homeostasis network seem to have provided an adaptive advantage to the subpopulations that harbored such traits. According to this framework, food insecurity, and not necessarily real alternated cycles of food abundance and famine (as required by the outdated thrifty genotype hypothesis; Neel 1999) or the necessity of elimination of the fitness costs for obesity (as required by the implausible drifty genotype hypothesis; Speakman 2008), should be understood as the pivotal driver for the evolutionary success of obesity. According to this framework, the deleterious health effects of obesity and its wide prevalence in modern society may ultimately represent the "winner's curse" (Thaler 1988) of an evolutionarily successful minimax strategy selected by subpopulations of ancient *Homo sapiens* individuals. In such early evolutionary stages, in the face of greater uncertainty regarding the future availability of food resources, subsistence and survival were the key objectives to be achieved by such an energy homeostasis system, and any obesity-related biological disadvantages were only minor uncertain side effects located in a future too remote to be taken into account.

"It's a bit more complicated than that": A broader perspective on determinants of obesity

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Abstract: The insurance hypothesis does not address important factors known to contribute to obesity levels in all persons, not just adult women in the industrialized world. These include psychological determinants of eating behaviours, the decline in physical activity leading to a negative energy balance, the dense built environment, pervasive food marketing, and the increased availability of energy-dense, nutrient-poor food.

The proposed insurance hypothesis is intriguing but seems to fall short of addressing a number of issues that contribute to obesity levels not only in adult women, but also in all persons from affluent and less affluent societies. For a start, the proposed hypothesis overlooks the role of psychological determinants of behaviours that lead to obesity such as taste, pleasure, mood, and habitual responses. There is compelling evidence that these factors impact eating behaviour and thus obesity (Gibson 2006). The insurance hypothesis suggests that food consumption is driven by survival, yet much research suggests that in humans, food is consumed because of taste and pleasure rather than survival (e.g., see Jansen et al. 2003). An additional factor that affects food consumption regardless of satiety consists of the habitual affordances of the environment. For example, Neal et al. (2011) found that people consumed significantly more stale popcorn in an environment that cued the behaviour (a cinema), compared to an environment where there was not the same cue to action (a classroom). This line of evidence would appear to be counter to the insurance hypothesis.

A second significant contributor to obesity not captured by the insurance hypothesis is the negative energy balance experienced by most people living in the industrialised world. For example, energy intake (kcal/day), as recorded in the National Health and Nutrition Survey by the Centers for Disease Control and Prevention from 1971 to 2000, shows no noticeable change across time in United States women ages 20–74 years. In contrast, levels of physical activity (occupational, household, and transportation) have declined substantially (Archer et al. 2013). In the United Kingdom, the Institute of Fiscal Studies (Griffith et al. 2016) found that total calories purchased from 1980 to 2013 have actually decreased. However, levels of obesity in the United Kingdom have increased because of substantial decreases in strenuous physical activity at work and in daily life. Such decreases in strenuous physical activity have been accompanied by substantial increases in sedentary behaviours; data indicate that adults in high-income countries spend most of their time sitting at work or watching TV (e.g., Matthews et al. 2008). Hence, reductions in energy expenditure contribute to obesity in addition to food availability and perceptions of food insecurity.

A third factor that contributes to the obesity problem, but not captured by the insurance hypothesis, is the built environment. Availability and accessibility of infrastructure for walking and bicycling, perceived safety, and aesthetic attributes have been found to predict levels of physical activity and obesity (Sallis et al. 2012). Thus, the built environment, in conjunction with the food environment, contributes to obesity. A food environment in which there is easy geographic access to fast-food outlets and convenience stores encourages individuals to consume foods that are high in energy and saturated fats. In fact, a recent literature review showed greater availability of fast-food restaurants in low-income neighbourhoods (Fraser et al. 2010). Pervasive food marketing exacerbates the effects of the obesogenic environment. In developing countries, where as much as 60% of household income is spent on food (Caballero 2007), marketing campaigns and price incentives for high-calorie products have a substantial impact on food-purchasing patterns.

Finally, energy-dense, nutrient-poor food is accessed easily worldwide and not just in high-income countries. Evidence shows that in low-income countries, such as India, Vietnam, Bolivia, and Nigeria, energy-dense nutrient-poor fast food is becoming increasingly popular (Rockefeller Foundation 2013). As an example, in India between 2007 and 2012, consumption of soft drinks increased by 70% and consumption of unhealthy foods by 110%. Counter to the proposal that the association between food insecurity and high body weight is restricted to adult women from high-income countries, women in the developing world often eat the least nutritious food for several reasons: lack of time, poverty, or because they are the last in the family to eat (Rockefeller Foundation 2013). Thus, data from the International Association for the Study of Obesity (Rokholm et al. 2010) show that high body mass index (BMI) rates greater than 30 in adult women can be found in both affluent (e.g., United States, England) and less affluent countries (e.g., Samoa, Egypt, Mexico). Conversely, current data from the United Kingdom suggest that increases in obesity have slowed (Sperrin et al. 2014) at a time when food insecurity is increasing (Loopstra et al. 2015), an observation that would appear to directly contradict the insurance hypothesis.

In summary, the insurance hypothesis seems insufficient to account for increases in obesity, as it ignores a number of important contributing factors, such as psychological factors affecting eating behaviour, the reduction of physical activity leading to a negative energy balance, and changes to the built environment in which we live. Moreover, this hypothesis seems incompatible with current data on obesity, particularly in low-income countries.

Anorexia: A perverse effect of attempting to control the starvation response

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Abstract: Starvation arouses evolved protective mechanisms including binge eating and increased metabolic efficiency and fat storage. When aroused by dieting, the experiences of out-of-control eating, increased appetite, and increased fat storage arouse greater fears of obesity, spurring renewed attempts to restrict intake severely. The resulting positive feedback cycle escalates into bulimia for many, and anorexia in a few.

Nettle et al. provide a long-needed integration of behavioral ecological research on the costs and benefits of fat storage with data on rates of obesity in humans. The logic is convincing, the model helps to specify the exact argument, and the data on birds document that experiencing unreliable access to food shifts the fat-storage set point upward. One study on a mammal is mentioned (Li et al. 2010), but supporting data are also available from multiple species (Dionne et al. 2016; Wilson & Cantor 1987). Nettle et al. note the unfortunate neglect of such behavioral ecological models in the social science literature, although there is some relevant work, especially from economists (Bellisari 2008; Smith 2009).

The authors' meta-analysis supports their insurance hypothesis, but I was surprised that the effect was not larger and more generalizable. Food insecurity increased the risk of obesity by only 21% and only for women in high-income countries. The authors are admirably restrained in their conclusions, emphasizing that factors other than the insurance hypothesis are also important. I wonder, however, if the limited strength of the results might be partly accounted for by measurement problems related to relying heavily on the questionnaire by Radimer et al. (1990) and its derivatives.

Examination of the specific questions on these questionnaires reveals that they measure only food insecurity resulting from lack of money, not from other causes. The first question is, "I worry that my food will run out before I get money to buy more." Out of the 13 questions, 12 refer explicitly to not having enough money for food. Not surprisingly, food security on this measure is achieved for 50% of families with incomes over $25,000 but less than 12% of families with household incomes

below $20,000 (Kendall et al. 1995). In short, these instruments measure levels of poverty severe enough to compromise food availability at times; such levels of poverty are, of course, highly correlated with social class, education, race, and sex. For people of middle-class and higher incomes in developed countries, the experience of hunger is rarely from lack of money. Dieting, medical conditions, and schedule constraints are more likely reasons for experiencing episodes of hunger. This makes the usual measures of food insecurity of limited utility, even while they remain valuable for studies of more diverse populations.

This target article has major implications for anorexia nervosa. As the article notes, substantial evidence supports the hypothesis that restrictive dieting causes long-term weight gain (Dulloo et al. 2015; Mann et al. 2007; Pietiläinen et al. 2012). Some of these studies, and those on rodents, also note that caloric restriction induces binge eating. The authors suggest that the insurance hypothesis predicts that anorexia should be more common when food security is high because that makes low body mass safe. That fits the epidemiologic evidence showing the high prevalence of anorexia in upper-class women in wealthy countries, but anorexia is vastly different from merely maintaining a low body mass when food is reliably available. Anorexia is not an adaptation; it is a disorder that arises from evolved eating regulation mechanisms that malfunction in some individuals when exposed to certain aspects of modern environments. People with anorexia nervosa do not maintain a low weight and go about life's business; they are obsessed and often deluded in their monomaniacal dedication to achieving thinness as a primary life goal. They are constantly hungry and preoccupied with food and weight. Many die.

A corollary of the insurance hypothesis offers an alternative explanation (Nesse 1999, p. 363). Almost all cases of anorexia begin with a stringent diet. The reliable result is an episode of out-of-control binge eating within a few days, behavior that would be life-saving in a famine. The experience of out-of-control eating combines with experiencing increased appetite, and observing increased fat storage, to arouse yet more intense fear of obesity, spurring yet more stringent efforts to control caloric intake, creating a vicious cycle that results in bulimia for many and anorexia in a few.

Nettle et al. have provided a valuable service by bringing principles from behavioral ecology together with those from obesity research. Related principles can also explain how exposure to modern media and other incentives to be thin can induce severe dieting that sets off adaptive responses that are useful during a famine, but prone to runaway positive feedback and disorder when aroused by dieting. Severe dietary restriction arouses responses adaptive during starvation – including binge eating, increased appetite, and increased fat storage – that motivate yet more intense efforts to control intake, in a vicious circle that spirals out of control to anorexia or bulimia. If correct, this explanation has obvious utility for prevention and treatment.

Using food insecurity in health prevention to promote consumer's embodied self-regulation

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Abstract: Health messages designed to address obesity are typically focused on the long-term benefits of eating healthy food. However, according to the insurance hypothesis, obese people are food insecure, and this causes them to be overly concerned about short-term consumption. As such, it is necessary to rethink public health messaging and consider how to reduce short-term insecurity by eating healthy food.

Although high-calorie foods are constantly available in contemporary environments, the evolutionary mismatch hypothesis suggests that people overconsume because their behaviour is optimized for the ancestral environment (e.g., McNamara et al. 2015; Nesse & Williams 1995; Prentice & Jebb 1995). Consumers can satisfy their immediate needs by making choices between different foods. Considering this might help explain why it is that health prevention messages are often focused on the long-term consequences of those choices. However, the recently developed insurance hypothesis by Nettle et al. leads to the suggestion that, even in contemporary environments, obese individuals may be living under the cloud of food insecurity. Although this hypothesis is certainly not the only explanation for the distribution of obesity in the population, it does at least provide a new perspective for understanding food behaviour with a view to changing health communication.

Nettle et al. start by discussing the shortcomings of the literature, which suggests that "people who are obese or eat unhealthily place a high motivational value on getting food soon" (target article sect. 2, para. 4; e.g., Guerrieri et al. 2012; Nederkoorn et al. 2006; Weller et al. 2008). According to the authors, this literature fails to describe the process(es) that lead(s) people to place a high motivational value on immediate consumption. According to Nettle et al., a plausible explanation for this is that obese people living under food insecurity would like to acquire food as soon as it becomes available. The insurance hypothesis might also help explain why it is that obese people are more sensitive to the expected pleasure of high-calorie food consumption (Pursey et al. 2014; for a review, see Spence et al. 2016). Indeed, the expected pleasure is generally associated with the likely inflow of nutrients that is higher for high-calorie foods (de Graaf 2012; Herman & Polivy 1983; Redden & Haws 2013). Thus, the *unhealthy=tasty intuition* (UTI), which might lead people to make unhealthy food choices and which could in turn affect their body mass index (BMI), could actually be attributable to the lack of nutritive expectations associated with healthy food consumption (Mai & Hoffmann 2015; Raghunathan et al. 2006). The UTI was initially tested on U.S.-American participants (Raghunathan et al. 2006). Interestingly, however, Werle et al. (2013) subsequently found that the French had a healthy=tasty intuition, due perhaps to their less utilitarian approach to eating (e.g., as compared to U.S.-Americans, see Rozin et al. 1999).

The nutritional aspect of consumption appears important for those individuals suffering from obesity. This interest can be justified by uncertainty in terms of acquiring nutritionally adequate foods in the future, as suggested by the insurance hypothesis. However, as pointed out by Block et al. (2011, p. 7): "No one sits down to eat a plate of nutrients." Thus, promoting the sensory pleasure (rather than nutritional quality) of eating healthy food might constitute a better way in which to reduce both food insecurity and the overconsumption of high-calorie foods (Petit et al. 2016b). This strategy would also be in keeping with an embodied vision of self-regulation, according to which "being more conscious of one's bodily states (and their simulation) in response to appetitive stimuli may be beneficial to pursuing healthy goals" (Petit et al. 2016a, p. 612). For instance, consumers should reduce their food intake when they feel a decline in enjoyment during consumption, signaling them that they will soon be full (de Graaf 2012; Herman & Polivy 1983; Redden & Haws 2013). Focusing their intention on the multisensory experiences (e.g., on the smell, taste, and mouthfeel of the food) while eating would inform the consumer's brain of the likely inflow of nutrients, thus reducing both their food insecurity and their consumption (de Graaf 2012; Ramaekers et al. 2014). By contrast, when consumers are more focused on health goals than on their physical sensations, they would be likely to underestimate the caloric content (Petit et al. 2016a). For instance, they are more sensitive to the health halo of fast-food restaurant health claims, leading

to overconsumption to compensate for the underestimated nutritional intake (Chandon & Wansink 2007; Chernev & Gal 2010).

The insurance hypothesis can also help explain why it is that those individuals with a higher (vs. lower) BMI are better able to make healthy food choices and exhibit more activity in those brain areas that are associated with gustatory inference (insula), reward value (orbitofrontal cortex), and self-control (inferior frontal gyrus) when they are focused on the pleasure of eating (vs. on health benefits; see Petit et al. 2016b). Indeed, Petit et al. explained their results by suggesting that people with a higher BMI are no less able to control themselves while making healthy food choices than those with a lower BMI but simply need a different valuation of those choices. By highlighting the pleasure (and thus nutrients) of eating healthy foods, public authorities and organizations in charge of promoting healthy lifestyles may be able to reduce food insecurity, and hence make healthy food choices more acceptable.

Recently, Petit et al. (2017) demonstrated that encouraging people to imagine the sensory experiences of eating a portion of food increases both the expected pleasure and the calorie estimation of smaller food portions, thus leading to a significant reduction of the portion size effect (i.e., generally, people tend to eat more when they are served a larger than a smaller portion of food). The fact that the calorie content of food portions is often underestimated helps explain why it is that people with food insecurity would be likely to select larger food portions and thus overeat (Wansink & Chandon 2006). Therefore, promoting the simulation of pleasant eating experiences would likely reduce the food insecurity highlighted by Nettle et al. by showing consumers that smaller food portions will satisfy their nutritional needs (Cornil & Chandon 2016; Petit et al. 2017).

Obesity as self-regulation failure: A "disease of affluence" that selectively hits the less affluent?

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Abstract: An effect of the long-term cycle of industrial and post-industrial global development is the increasingly generalized access to abundant and diversified food sources. This poses a substantial problem of self-regulation that mainly affects the less affluent and whose failures may play an important role in the explanation of the increasing social incidence of overweight and obesity problems.

I concur with the authors that the food insurance hypothesis (IH) may be relevant as an explaining factor for current obesity trends, and that a simple evolutionary mismatch (EM) logic is unable to explain inter- and intracountry differences in obesity rates. I think, however, that a more sophisticated version of the same EM hypothesis might be better defended against IH. In particular, I am thinking of obesity as a failure of self-control in the presence of ego-depletion phenomena (Baumeister et al. 2000; Baumeister & Vohs 2016). As shown by Dragone (2009), regulatory failures in eating behavior may even occur in the context of rational intertemporal decision making.

In a simple version of the EM, it would be implied that richer, better educated people should be more able to access food and thus be more obese than poorer, less educated ones, which is contrary to the evidence. But in a more sophisticated version that takes self-control issues into account, access to better material and educational resources provides individuals with a larger, relatively more fulfilling menu of alternative rewards to counter the constant ego-depleting pressure that is caused by resistance to the easy availability of tasty, highly caloric food that is currently guaranteed to almost everybody, even in case of modest spending capacity. This kind of self-control problem can be seen as a consequence of the generalized improvement of access to food sources caused by the constant improvements of material subsistence conditions along the long-term industrial and post-industrial development cycles (Ezzati et al. 2005). In conditions of substantial material scarcity, self-control issues concerning access to food were relatively exceptional, and even occasional opportunities of overfeeding could have been regarded as functionally (rather than dysfunctionally) related to long-term survival goals. But in the current context of material abundance, an almost constant exercise of self-control is needed to both stick to healthy food choices and resist unhealthy food temptations. Where effective countermeasures are lacking, ego depletion in food-related choices will eventually occur once the self-control muscle has entered the refractory phase from over-exercising (Muraven & Baumeister 2000), and the subject will eventually capitulate to unhealthy food choices.

As a matter of fact, relatively more affluent people have a much richer repertoire of choices and tools to counter unhealthy food craving including (a) the availability of tastier, more appealing (and more expensive) healthy food; (b) reliance upon personal trainers and consultants to support them in maintaining their self-control goals through implementation intention (Webb & Sheeran 2007), planned goal striving (Bayer et al. 2010), and self-awareness (Alberts et al. 2011); (c) seeking alternative rewards from a vast range of non-food-related sources, often inaccessible or less accessible to less well-off people (Sivanathan & Pettit 2010); and (d) having access to a richer range of positive affect stimuli to ease recovery after self-regulation (Tice et al. 2007). In addition, relatively more affluent people are exposed to stronger social incentives to exercise self-control over food choices to preserve their physical aspect: from the need to promote their public image, to the necessity of commanding others' respect and admiration to maintain leadership, to keeping up with success-related aesthetic role models, and so on. It is also to be remarked that consistently successful self-regulation generates intrinsic rewards that effectively moderate ego depletion, provided that substantial depletion states are strategically avoided (Vohs et al. 2012), so that subjects who are able to draw upon a richer repertoire of self-regulation tools and are exposed to stronger social incentives will tend to widen the regulatory gap with respect to less endowed and successful subjects (Muraven 2010).

In other words, more-privileged people have better chances than less-privileged ones of achieving a better regulatory balance by accessing superior healthy food alternatives and valuable, alternative non-food-related compensatory rewards, as well as of being driven by stronger self-control motives, with better consequences in terms of body weight management. Moreover, they can rely upon the ego-reparative capacity of money (Boucher & Kofos 2012). On the other hand, many different kinds of non-healthy food, *because* of their current abundance, are among the cheapest and more accessible sources of rewards – and in particular, of rewards that do not depend on social status, recognition, or education, unlike most others – and are therefore likely to be the option of choice for relatively poor, uneducated individuals (Orr et al. 2014). This is the case not only as a consequence of ego depletion from food-related self-control attempts themselves, but also *in addition* as an easily accessible compensation of negative affect from other forms of failure of self-control (e.g., poor concentration on study and school failure). These forms include an ego-depleting lack of autonomy in goal implementation (Muraven et al. 2008), social stigma (Inzlicht et al. 2006), resistance to peer group conformism (Renna et al. 2008), exposure to persuasive messages (such as advertising of unhealthy food; e.g., Wheeler et al. 2007), lack of countervailing strategies (Janssen et al. 2010), or ironically as an ego-depleting cross-effect

from *successful* self-control in other domains (e.g., refraining from addictive habits; e.g., Shmueli & Prochaska [2009]).

The often-stressed problem of physical accessibility of healthy food, or more generally of a varied menu of food choices, does not likewise seem to be a major explanatory factor for the social incidence of obesity. Even in poor neighborhoods, accessibility of sources of unhealthy food is generally matched by comparable accessibility of healthier food choices (Lee 2012), so that a stronger propensity for the former is more easily explained in terms of regulatory failure than in terms of lack of accessibility or food insecurity.

Therefore, a suitably extended EM formulation could provide an alternative, or at least complementary, explanatory route for obesity trends vis-à-vis IH. In particular, it would be especially interesting to investigate the actual (possibly culture-specific) mix of self-regulatory versus food insecurity factors at work in the widely observed social influence dynamics characterizing current obesity trends (Shoham et al. 2015).

Household-level financial uncertainty could be *the* primary driver of the global obesity epidemic

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Abstract: Evidence has accumulated in support of the notion that changes in household-level financial uncertainty (or "economic insecurity") may be an important fundamental cause of the global obesity epidemic. The timing and spatial/demographic incidence of the obesity epidemic suggest that economic policies aimed at expanding economic freedom may have inadvertently shifted risk to households, thereby generating a costly public health problem.

In their review of the empirical literature on obesity, Nettle et al. choose to limit their analysis to evidence derived from food insecurity surveys. Theory and evidence from behavioural ecology, as the authors rightly note, suggest that human obesity should be responsive to cues that predict – or would have predicted, in human evolutionary history – prospective food shortages. But the epidemiological literature on food insecurity relies, for the most part, on qualitative survey data (collected retrospectively) about perceived food scarcity. Measurement error alone could explain the observed gender differences (might men be less willing than women to report an inability to reliably feed their families?), confounding variables are numerous and often unobservable, and even enthusiastic proponents of what the authors refer to as the insurance hypothesis (myself included) should be surprised that a correlation with obesity is so reliably observed.

Had the authors expanded their purview to include household-level *economic* insecurity – that is, uncertainty with respect to financial well-being – they would have found a wealth of evidence supporting the link to obesity (for reviews, see Smith [2009] and Wisman & Capehart [2010]). This literature, which also draws on theory and evidence from behavioural ecology, typically employs econometric methods aimed at identifying causal relationships between economic insecurity and obesity. Smith et al. (2009), for example, show that working-age men in the United States gained more weight over a 12-year period when they faced a higher risk of job loss (regional unemployment rates were used to instrument for risk of job loss – effectively "selecting" those individuals who face said risk through no fault of their own), while Barnes et al. (2013) show that cohabitation with other adults can be protective against weight gain – provided those other adults have paying jobs.

One of the most compelling confirmations of this hypothesis appears to have been accidental: Ferrie et al. (1995; 1998) report on a natural experiment that occurred during the long-running Whitehall II Study of British civil servants. In the early 1990s one of Whitehall II's 20 civil service departments was threatened with privatisation (and subsequently, was indeed privatised). The timing of the Whitehall II survey dates was such that data were collected both before and after discussions of privatisation began, and then again after privatisation occurred. Although the authors clearly had no specific hypothesis about employment security and obesity in mind (Whitehall II being designed to test more general hypotheses about workplace stress and health outcomes), they nevertheless report strong evidence: Of the many health measures reported, the most significant differences (for both men and women) among those working in the threatened department were increased body mass index and increased likelihood of excessive sleep. These differences were noted both before and after privatisation actually occurred, and were independent of actual changes in employment status.

The timing and magnitude of the obesity epidemic has varied dramatically from country to country, but the largest increases have generally been observed in countries, such as the United States, the United Kingdom, Iceland, New Zealand, and Australia, that have aggressively pursued neoliberal economic policies (Ljungvall 2013; Egger et al. 2012; Smith 2012a; Vogli et al. 2014). These policies have typically included expansion of international trade, privatisation of public services, softening of regulatory restraints on industry, monetary policy emphasising price stability rather than full employment, and a weakening of labour protections and other aspects of the social safety net. Although these policies may have been well intended (typically being enacted with the stated purpose of expanding opportunities for mutually beneficial exchange), they also, arguably, have had the collective effect of increasing the burden of financial risk faced by households. In the United States there is growing evidence that both earnings volatility (Gottschalk & Moffitt 2009) and economic insecurity more generally (Hacker et al. 2014) have increased over the period that obesity rates have risen (roughly, 1980–present), and that obesity rates have risen most dramatically among demographic groups that have seen the largest increases in economic insecurity (Smith et al. 2016).

Even if it is true that economic liberalisation has helped cause the global obesity epidemic, there is an obvious alternative mechanism to be considered. One might expect that liberalisation, in addition perhaps to making life more risky for the individual citizen, also tends to reduce regulatory constraints on the food industry. This could result in increased obesity rates via either lower food prices or industry marketing practices (e.g., promotion, product formulation). In the most direct test of these competing hypotheses (insecurity vs. "fast food") of which I am aware, Offer et al. (2010) found, in a panel of developed countries, that insecurity appears to play a far larger role. I myself have offered a related conjecture, suggesting that the similarities in the physiological effects of modern fast food and what anthropologists call "famine foods" are not accidental. Rather, one might say that industry is simply serving up what humans have evolved to crave during periods of economic insecurity (Smith 2012b).

As a trained economist (and erstwhile biologist), I have always harboured a natural sympathy for the notion that the market mechanism is an elegant way of allocating societal resources. Give people incentives, the argument goes, and they will perform! But years of studying the role of insecurity in the etiology of obesity have given me pause. In the workplace, every opportunity to succeed necessarily comes paired with a threat of failure. This leads me to another core tenet of economics: Every benefit must be weighed against its cost. It seems that some of my profession's most cherished policy prescriptions may have come at the cost of a global obesity epidemic.

Children respond to food restriction by increasing food consumption

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Abstract: Consistent with the insurance hypothesis, research shows that when children experience restricted access to food, they display increased intake when restrictions are lifted. This effect appears more robust for girls compared to boys, and for children with lower levels of inhibitory control. The insurance hypothesis has potentially important implications for parental feeding practices.

Nettle et al. find limited evidence for an association between food insecurity and body mass index (BMI) in children and note that "explaining the weaker association in children stands as a challenge to the IH [insurance hypothesis]" (sect. 6.4). There are two important reasons why such a finding is unsurprising. First, as Nettle et al. note, food insecurity is typically assessed via parental report and will not necessarily reflect a child's experience. As described below, irrespective of food insecurity, many parents place restrictions on their child's access to food. Thus, in principle, even in affluent households children may feel they are not always able to choose the type and quantity of foods they eat. Second, in some instances these kinds of parental restrictions can actually help prevent excess weight gain among children (Rollins et al. 2014b). As with low-income countries, we may fail to find a relationship between food insecurity and BMI among children because even during times of increased availability, some children may not have access to sufficient quantities of energy-dense foods to enable them to store excess weight.

Nevertheless, a number of alternative lines of research do provide support for the insurance hypothesis among children. This research includes both laboratory-based manipulations of food restriction as well as measures of parental food restriction. Parental food restriction refers to parents' attempts to limit children's access to or consumption of certain types of food (typically those that are energy dense) for health or weight-related reasons. Such behaviour is generally assessed via parental or child questionnaires that ask about whether the child is allowed to eat certain foods, whether they have to ask permission before eating certain foods, and whether the parent restricts the quantity of foods consumed.

Laboratory studies have shown that restricting children's access to a particular food will increase their expressions of desire for and consumption of that food (Fisher & Birch 1999b; Jansen et al. 2007; 2008; Rollins et al. 2014a). Although such effects do not appear to extend beyond the immediate postrestriction period (Fisher & Birch 1999b; Rollins et al. 2014a), research on parental feeding practices suggest that when children are exposed to continued food restrictions, effects on intake may be sustained. For example, children whose parents restricted access to particular snacks ate greater quantities of these snacks in the laboratory setting (Fisher & Birch 1999a; Jansen et al. 2007; Rollins et al. 2014a; although see Jansen et al. 2008 for a null effect). Likewise, longitudinal research has found that girls who were exposed to greater parental food restrictions at age 5 were more likely to eat in the absence of hunger in the laboratory at ages 7 and 9 (Birch et al. 2003; Fisher & Birch 2002). Such effects are consistent with the insurance hypothesis.

An important finding from the meta-analysis conducted by Nettle et al. is that food insecurity is associated with BMI among women but not men. Although studies employing laboratory manipulations of food restriction among children have not tended to explore sex differences (Fisher & Birch 1999b; Jansen et al. 2007; 2008; Rollins et al. 2014a), there seems to be little evidence to indicate that boys and girls respond differently in these studies (Fisher & Birch 1999b). However, there is evidence to suggest they respond differently to parental food restriction; Fisher and Birch (1999a) found that both parent and child reports of maternal food restriction were associated with laboratory measures of intake among girls but not boys. Interestingly, this study also found that maternal and child reports of food restriction were correlated for girls but not boys, leading the authors to speculate that although boys and girls may be subject to similar restrictions, boys may be granted more autonomy and choice over what they eat, which may diminish the effects of restriction.

Other longitudinal research has looked at the association between parental food restriction and child BMI. This work suggests a complex relationship with some studies supporting the notion that parental restriction increases BMI (Anzman & Birch 2009; Faith et al. 2004; Francis & Birch 2005) and others showing that parental restriction occurs in response to concern over child weight (Rhee et al. 2009; Spruijt-Metz et al. 2006). Because certain personality traits have also been linked to a tendency to consume more energy-dense foods (e.g., Tapper et al. 2015), an additional possibility is that child BMI and parental food restriction are correlated only because they are both influenced by child trait variables. As noted above, a further complication is that parental restriction may sometimes prevent children from gaining excess weight (Rollins et al. 2014b).

More recently, research has shown that the effects of parental food restriction on intake are moderated by children's level of inhibitory control. In the laboratory, children with low inhibitory control respond to food restriction with increased intake, whereas children with high inhibitory control do not (Rollins et al. 2014a). Similarly, parental restriction is associated with greater increases in girls' BMI among those with low inhibitory control, but not among those with high inhibitory control (Anzman & Birch 2009). A similar pattern occurs for eating in the absence of hunger (Rollins et al. 2014b). Such effects are consistent with other evolutionary accounts of behaviour that describe how reduced levels of inhibitory control are adaptive for those who grow up in harsh, unpredictable environments (Griskevicius et al. 2013; Simpson et al. 2012).

Thus, research findings relating to child feeding are consistent with the insurance hypothesis. However, they also raise important questions that have yet to be answered. For example, what type of parental food restriction drives overeating? Is there a critical period in childhood that determines eating later in life? The insurance hypothesis could help guide research in such areas. Given that child feeding is an issue many parents struggle with (Moore et al. 2010), such research could also help inform the development of evidence-based advice and intervention.

Obesity is not just elevated adiposity, it is also a state of metabolic perturbation

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Abstract: Nettle et al. miss the crucial difference between adaptive models of storing energy and explanations for the pathological metabolic state of obesity. I suggest that the association of food insecurity with obesity in women from industrialized settings is most likely due to reverse causation: Poverty reduces agency to resist obesogenic foods, and this scenario is compounded by perturbations of insulin metabolism stemming from high adiposity and lipogenic diets.

Undoubtedly, multidisciplinary approaches are required to explain differential susceptibility to obesity, but this article

suffers from lack of reference to several key literatures, and I find the conclusions seriously flawed. In my own work, I have proposed and tested several adaptive hypotheses relating to adiposity (Wells 2006; 2009; 2010a; 2011a; 2012a; 2012d; 2012e; 2012f). Paleoclimate data indicate that hominins were exposed to growing ecological stochasticity in comparison with non-human apes and evolved many components of physiological flexibility in response (Wells 2012d), of which adipose tissue was particularly important. There is little evidence that starvation was the primary selective pressure; rather there are several "fitness functions" of adipose tissue (Wells 2010a) broadly connected to tolerating short-term or seasonal changes in energy balance. I have provided empirical support for body composition variability enabling adaptation to diverse stresses including climate, pathogen burden, and seasonality, while buffering health, growth, and reproduction (e.g., Wells 2012c; 2012g; Wells & Cortina-Borja 2013; Wells et al. 2010). Crucially, I restricted these analyses to populations in non-Western settings, and I have repeatedly emphasized that the adaptive biology of adipose tissue must be clearly differentiated from the pathological scenario of obesity and the impact of obesogenic environments (Wells 2010a; 2012e; 2012f). Nettle et al. are aware of this literature, having cited it elsewhere (Nettle et al. 2013), but have adopted the opposite approach: proposing that adaptive models explain obesity itself.

Nettle et al.'s approach posits that psychological mechanisms underlie "decisions" about "how much to eat," though they do not specify whether this involves conscious deliberation or not. They then argue that it is adaptive to assess food security (again, without specifying how) and select an appropriate level of fat-insurance to acquire. Regardless of how these cost-benefit decisions are made, their "appropriate level" model explicitly assumes that individuals must decide both *when to acquire fat* and *when to stop acquiring it*, as their model proposes rising fitness costs. But the fundamental problem of obesity, well known to all in the field, is that *fat individuals become even fatter*, and far from supporting their adaptive arguments, their meta-analysis directly contradicts them.

According to adaptive principles, food insecurity should predict the greatest weight gain in thin individuals. This is very evident in the bird literature they reviewed – for example, the experimental study of greenfinches where food insecurity promoted weight gain inversely in association with baseline weight (Ekman & Hake 1990). Paradoxically, however, food insecurity showed significantly stronger associations with excess weight in the meta-analysis for obesity than for overweight. From an allometric perspective, birds have adequate fat stores to buffer only short periods of starvation, whereas men and women of average U.K. height with a body mass index (BMI) of 30 kg/m^2 have sufficient energy reserves to endure total starvation for ~75 and ~88 days and to endure half-rations for 150 and 175 days, respectively. Larger individuals with BMI of 40 kg/m^2 could tolerate half-rations for over nine months simply by oxidizing fat reserves (though in practice for even longer, as lean mass also declines during extended weight loss). That food insecurity should predict excess weight more strongly *among those already with substantial energy stores* than among those with lower reserves contradicts their adaptive model. Their "positive" findings thus derive from high-income populations analogous to "fat greenfinches," whereas what we really need to know is whether food insecurity promotes weight gain in thin humans.

If their findings fit poorly with their energy insurance hypothesis for obesity, what other explanations are more likely? It is baffling that they make no reference to the neurobiology of appetite regulation; the role of key hormones such as insulin, leptin, and ghrelin; or the role of insulin resistance in driving hyperphagia among those obese. High circulating insulin levels in obese individuals make them highly susceptible to energy-dense diets, particularly those high in sucrose (Lustig 2006; 2008; Wells & Siervo 2011). This means that certain foods can themselves drive hunger and lethargy, helping explain why those who are already fat keep getting fatter (Lustig 2006; 2008; Wells & Siervo 2011).

A much simpler explanation for Nettle et al.'s finding, therefore, is reverse causation. Food-insecure individuals are also poorer, which shapes their dietary choices, and poorer groups are well established to have greater geographical exposure to junk food and other obesogenic factors (Block et al. 2004; Drewnowski & Specter 2004; Larson et al. 2009). Indeed, I have argued that poor and food-insecure groups have the least agency to resist commercial interests, and that this lack of agency is itself promoted by corporate manipulation of dietary quality and food availability (Wells 2016). This looks less like adaptive energy insurance acquisition and more like the interaction of perturbed metabolism with material deprivation and disempowerment – thus, from obesity to morbid obesity.

I have similar concerns over Nettle et al.'s perspective on the developmental origins of obesity. Their proposal that offspring acquire energy reserves in anticipation of future food insecurity reiterates the predictive adaptive response hypothesis (Gluckman & Hanson 2004), yet this has been extensively criticized specifically in relation to growth and metabolism (Wells 2010b; 2012b). Early growth patterns primarily predict later size and lean mass (Wells 2011a; 2011b) rather than adiposity, and associations of birth weight or infant weight gain with later obesity appear restricted to obesogenic settings (Wells et al. 2007). In Peru, early-life exposure to food insecurity following the 1998 El Nino event reduced childhood height and lean mass but had no effect on fat mass (Danysh et al. 2014).

Obesity is a serious public health issue because even when people strongly desire to lose weight, their metabolism overrides their intentions. This is most powerfully demonstrated by the effects of bariatric surgery: Long before any change has occurred in energy stores, profound alterations in hormone levels lead to reduced appetite and improved metabolic health (Rubino et al. 2004).

It would be interesting to test the food insecurity–energy insurance hypothesis in those of poorer nutritional status, but the authors have failed to provide evidence that it drives human obesity.

Authors' Response

Adaptive principles of weight regulation: Insufficient, but perhaps necessary, for understanding obesity

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Abstract: We reflect on the major issues raised by a thoughtful and diverse set of commentaries on our target article. We draw attention to the need to differentiate between ultimate and proximate explanation; the insurance hypothesis (IH) needs to be understood as an ultimate-level argument, although we welcome the various suggestions made about proximate mechanisms. Much

of this response is concerned with clarifying the interrelationships between adaptationist explanations like the IH, constraint explanations, and dysfunction explanations, in understanding obesity. We also re-examine the empirical evidence base, concurring that it is equivocal and only partially supportive. Several commentators offer additional supporting evidence, whereas others propose alternative explanations for the evidence we reviewed and suggest ways that our current knowledge could be strengthened. Finally, we take the opportunity to clarify some of the assumptions and predictions of our formal model.

R1. Introduction

We are grateful to our commentators for engaging with our target article and providing thought-provoking responses. In what follows, we discuss each of the major groups of issues raised. In many cases, these interconnect. The issues are varied. Some commentaries review additional sources of supportive evidence that we did not include in the target article. Some raise issues that turn on misapprehensions of what we claimed, misapprehensions that can be sorted out by defining terms and clarifying levels of analysis. Still others fill in ideas about proximate mechanisms, an issue not covered in our target article. Several commentaries offer alternative accounts of the empirical evidence we reviewed or draw attention to its limitations. Finally, many of the most difficult issues raised involve understanding how the IH relates to other, possibly better-known, explanatory approaches to obesity. The IH stems from reasoning about the normal functioning of evolved weight-regulation mechanisms. How does this articulate with literatures that see obesity as a metabolic dysfunction or regard overeating as a failure of self-control? We have not been able to answer every point of detail, but we have endeavoured to cover the overarching or recurrent themes.

R2. Proximate and ultimate explanations

A useful distinction can be made between ultimate and proximate explanations for a phenotypic or behavioural pattern (Scott-Phillips et al. 2011; Tinbergen 1963). Ultimate explanations are based on how the phenotypic or behavioural pattern contributes to survival and reproduction, and hence offer an account of why that particular pattern has been retained over generations as part of the organism's phenotypic repertoire. For example, for trees in temperate latitudes, having full leaves in winter is disadvantageous because the photosynthetic yield from foliage at this time of year is insufficient to outweigh the cost of maintaining the foliage and the increased likelihood of weather damage when a tree is in full leaf. Thus, many temperate trees have evolved a deciduous pattern; they grow full leaves in spring and drop them in autumn, thus balancing the benefits of photosynthesis when there is more sunshine and the costs of having leaves when there is not. This is an ultimate-level explanation. Likewise, the IH as we present it in the target article is an ultimate-level hypothesis; it seeks to explain why, in terms of benefits to evolutionary fitness, individuals might maintain higher fat reserves under conditions of insecurity than under conditions of security. Ultimate-level explanations are vital for explaining the ecological patterning of a phenotype in a satisfactory way.

Proximate explanations deal with the mechanisms by which the behaviour or phenotypic pattern of interest is triggered. In general, there are many possible (and perhaps non-exclusive) proximate mechanisms that could deliver any given ultimate-level function. For example, some deciduous trees are cued to sprout leaves by day length, while others are cued by temperature – two different proximate mechanisms for achieving the same ultimate function. Importantly, the proximate explanation need not involve the organism representing, at any level, what the ultimate function of the pattern is. Trees do not need to know or represent why it is they sprout leaves in spring in order to do so effectively. Rather, ancestral trees that sprouted leaves in spring and dropped them in autumn, however that was caused, have more descendants living today than trees that did not. Often, an ultimate function is delivered by a varied suite of different mechanisms operating in concert and with considerable redundancy; this is known to be true, for example, of animal navigation (Frost & Mouritsen 2006).

Although behavioural biologists debate the limits to which functional explanation can be pursued independently of understanding mechanisms and vice versa (Fawcett et al. 2013; McNamara & Houston 2009), all agree that an explanation in terms of a proximate mechanism is not an alternative to an explanation in terms of an ultimate function. This is important in the current case. For example, **Mullan, Ntoumanis, Thøgersen-Ntoumani, & Lipp** (**Mullan et al.**) point out that people's immediate motivations for eating are often to do with taste and pleasure, rather than gaining calories. This is quite true, but to include taste and pleasure in our formal model on the same basis as evolutionary fitness would be to confuse proximate and ultimate levels of analysis. The ultimate function of eating is to obtain energy and nutrients; the proximate mechanisms by which our evolved brains do this include making food tasty and eating pleasurable. These two statements are both true, not alternatives to one another.

Similarly, "culture" and "diet" (**Lozano**) are not alternatives to the IH, but pathways through which food insecurity could affect body weights. **Ert & Heiman** suggest that the findings of the empirical literature on food insecurity and body weight could be explained by the IH, but could equally well be explained by changes to temporal discounting or the feeling of scarcity brought on by food insecurity. Ert & Heiman describe these as "alternative psychological mechanisms" to the IH. However, we didn't propose any psychological mechanisms in the target article, so these obviously cannot be alternative ones! Rather than temporal discounting or the feeling of scarcity being alternatives to the IH, they could be part of a suite of psychological mechanisms that deliver increases in energy intake relative to expenditure under conditions of food insecurity, as the IH requires.

Our target article did not go into the question of proximate mechanisms at all, a fact that many commentators picked up on. We concur with them that the issue of proximate mechanisms is of critical importance. Its intentional absence was explained by limitations on what could be covered in one article. As behavioural ecologists often do, we began our enquiry from considering ultimate payoffs and hence determining the kinds of strategies that one would expect to see emerging via natural selection (and

the commentary by **Mattei** extends this strategic logic). We did so in a way that was agnostic about the mechanisms by which weight regulation is actually achieved.

This agnostic opening gambit does not mean we deny the existence or importance of such mechanisms. Understanding mechanism is crucial not only for a complete understanding of how something works, but also for particular relevance to understanding how phenotypes become maladapted. To return to our trees example, under rapid global warming, trees that use a temperature-based mechanism to cue coming into leaf will become progressively maladapted to actual patterns of available solar radiation (they will sprout leaves earlier and earlier), whereas trees that use day length will remain adapted. We thus fully concur with **Higginson, McNamara, & Dall (Higginson et al.)** that exploring the (no doubt multiple) mechanisms individuals have evolved to extract information from their environment about future energy need and energy availability will be critical to understanding why average body masses have become so unprecedentedly high in many modern populations, and why they become extremely high in some individuals (and, in eating disorders, extremely low in others; per **Nesse**). Understanding proximate cues also offers possibilities for understanding how it might be possible to intervene to change outcomes, short of changing the whole ecology of society **(Cardel, Pavela, Dhurandharc, & Allison [Cardel et al.]**; **Petit & Spence)**. Ultimate understanding can only be a starting point for these kinds of enquiry; where a phenotype becomes maladapted, you also need to understand the interaction between evolved mechanisms and current environmental inputs.

R3. Varieties of proximate mechanism

Both **DeJesus** and **Wells** suggested that the IH implies that humans consciously reason about the probability of future shortfall and the resulting need to carry fat reserves. We were surprised by this, as the target article makes no such claim, and the claim strikes us as implausible. If mice and greenfinches can solve problems of weight regulation under intermittent food supply (presumably) without the need for conscious reasoning, it would be strange to assume that humans require conscious reasoning to achieve the same ends. The problem may lie with the term "decision making" that we used; for us, this does not imply conscious deliberation, or even necessarily the involvement of the brain. It simply describes any kind of mechanism that maps some information from the environment onto some kind of phenotypic output. It may have different connotations for others, so we are glad to clarify.

On a related note, **Bentley & O'Brien** argue that the IH belongs to the class of explanations in the upper left corner of their explanation space. This is the quadrant where decisions are made individually by reasoning agents to whom the payoffs of the different alternatives are transparent. We don't agree with this characterisation. The IH no more requires the payoffs for storing more fat to be transparent to the mind than the existence of tanning requires that melanocytes can represent the payoffs to having a lighter or darker skin. In fact, in Western populations, there probably are no payoffs to carrying more fat when food insecure, transparent or otherwise. Rather, the IH contends that there are evolved mechanisms that, when presented with particular types of cue, respond with changes to food motivation and/or energy expenditure that result in more fat storage. They respond in this way because ancestors who responded in this way to ancestral food insecurity left more descendants than ancestors who lacked this response.

But what, then, can we say about the proximate mechanisms likely to be involved? We will not repeat at length the useful discussions on potential mechanisms by **DeJesus**; **Petit & Spence**; **Coppin**; **Blackwell**; and **Ambroziak, Azañón, & Longo (Ambroziak et al.)** as well as others. We agree that there are likely to be multiple, largely unconscious and automatic pathways by which experiences that in ancestral environments predicted the likelihood of future food shortfall lead to increases in food consumption, shifts in food preference, and possibly changes in energy expenditure. The mechanisms may well alter the appraised or experienced pleasure and reward value of food, particularly of those subtypes of food that are most dense in energy. We also welcome Ambroziak et al.'s reminder that the mechanisms must also involve an individual's assessment of his or her own current bodily state, an assessment that can be inaccurate, for example, in the case of eating disorders. There may be a special role for the hypothalamic–pituitary–adrenal (HPA) axis (Blackwell). Hunger is associated with increased HPA activity (Erickson et al. 2003). Regular experience of serious hunger would thus become encoded in the individual in the form of frequent glucocorticoid stress responses, and *ex hypothesi* such a pattern would cause a shift in appetite. Indeed, there is good evidence, including experimental evidence (Tataranni et al. 1996), for the existence of this pathway (see Epel et al. 2001).

A consequence of the existence of this HPA mechanism would be that anything other than food insecurity that caused a frequent glucocorticoid stress response would also have the potential to produce weight gain. There is widespread evidence for psychosocial stress promoting weight gain, as several of our commentators (e.g., **DeJesus**; **Blackwell**; **Smith**) point out. These associations can be seen as by-products of the glucocorticoid stress response being part of the mechanism that stores extra energy in response to food insecurity. Essentially, all kinds of stressful experiences that in contemporary environments do not predict, or only very weakly predict, future food scarcity evoke mechanisms whose evolved function was to deal with food scarcity.

Chen and **Dittmann & Maner** discuss the possible role of childhood in setting adult body weight. They link the IH to a body of recent work arguing that childhood experience provides the developing person with information about the adult world into which they will mature, and hence sets them on a path towards developing the appropriate phenotypic strategy for that world. We agree with these commentators that this possibility would not be incompatible with the IH but an extension of it, and that there is a large body of correlational evidence linking childhood psychosocial adversity with high adult body weight or altered eating (see Danese & Tan [2014] and sect. 7.1 of target article). From an evolutionary point of view, the difficulty with these arguments is that, given that food availability is likely to have fluctuated over short timescales in ancestral environments, it is hard to see how it could be adaptive

to use childhood experience as information about food availability in the adult world that an individual will experience only years later. Wells (2007b) made this point forcefully and repeats it in his commentary here (**Wells;** see also Nettle et al. 2013). Retaining full plasticity into adulthood would appear to be enormously superior from an adaptive point of view over canalisation during childhood, and indeed, fat reserves can change quite dramatically in adulthood. To solve this issue is beyond the scope of this article. It may be that there are mechanistic or developmental constraints on adult plasticity that help explain why this should be the case, when using information from the adult environment would seem more beneficial.

Several commentators (**DeJesus**; **Mata, Dallacker, & Hertwig** [**Mata et al.**]) point out that eating is a social activity and, therefore, food consumption is influenced by a number of different social processes. We fully agree. Again, we would not see social determination as conceptually an alternative to the IH. It is not just that social factors such as power, wealth, and status determine food insecurity. It is that the mechanisms by which individuals extract information about their food ecology and food needs may include cues provided, passively or actively, by others. (For this reason, just as we do not agree that the IH resides in the upper part of the explanation space discussed by **Bentley & O'Brien**, we do not agree that it resides in the left side either.) Although the existence of social transmission is not an alternative to the IH at the conceptual level, it does make the epidemiological predictions more complex. For example, as Mata et al. point out, if some individuals buffer others from the effects of food insecurity, then we may fail to find associations where the theory predicts they should exist (e.g., perhaps in the case of children). If the perception of food insecurity is transmitted socially from person to person, then, even if this is basically an adaptive mechanism, there can be considerable non-adaptive cultural momentum. Eating patterns can perpetuate in particular social groups for a long time even if the initiating ecological conditions have been removed, and they can diffuse through networks not just to individuals who are actually food insecure, but to their secure associates too. These kinds of momentums and inertias arising from social transmission have been extensively discussed in the cultural evolution literature (Colleran 2016; Richerson & Boyd 2005).

R4. Constraint versus adaptationist explanations

The IH as discussed in the target article is an adaptationist hypothesis. That is, it attributes higher body weights in food-insecure social groups to the operation of evolved adaptive mechanisms for weight regulation. Note that adaptationist does not mean adaptive; if the current environment is sufficiently different from those over which the mechanism evolved, then the output of the mechanism will not maximize current fitness, even if the mechanism is functioning normally and has evolved through natural selection.

Several commentators proposed alternative explanations for the observed evidence, based instead on some kind of psychological constraint. For example, **Sacco** and **Dohle & Hofmann** invoke the idea that people with adverse lives may be too depleted or overloaded to exert the self-control needed to avoid overeating in an affluent environment, and **Wells** relatedly suggests that poverty reduces the agency required to resist commercial interests. (Wells refers to this as a reverse-causation argument to the IH, which it is not; the causality is in the same direction as in our argument, but via a different pathway to the one we proposed.) Rather than seeing these proposals as just proximate mechanisms delivering the ultimate function specified by the IH, we see them as arguments of a different kind. They characterise the consumption decisions of food-insecure people as, fundamentally, errors arising from not having the psychological capacity available to make better decisions (i.e., arising from psychological constraints). This is different from seeing them as automatic decisions that would have been beneficial, under the given environmental cues, in ancestral environments (i.e., arising from adaptations).

Constraint explanations are often invoked to explain the behaviour of the poor, though there are alternative adaptationist interpretations of the same phenomena (Nettle 2010; Pepper & Nettle 2017). We admit it can be challenging empirically to distinguish between the predictions of the two classes of explanation. We make several observations. The idea that there exists some kind of domain-general self-control or self-regulatory capacity in humans that can get depleted is contested, and it may not be well supported by evidence (Carter et al. 2015; Kurzban 2016). A number of experiments have attempted to experimentally deplete self-control to examine the impact on food consumption, and the average effect in these studies does not clearly differ from zero (Carter et al. 2015). Second, even if such a general self-control capacity did exist, it is not clear how much of the variation in people's body weights it would explain. Only a subsection of population reports using effortful restraint to control their eating, and this subsection tends to have *higher*, not lower, body weights than those who describe their eating as unrestrained (Rudermann 1986). Thus, having greater resources for effortful restraint or self-control does not seem a promising avenue for explaining why more people in the most privileged social groups remain thin.

Related to constraints explanations is the widespread argument that the growth of obesity is explained by the marketing of sugar and fast food (mentioned here by **Bentley & O'Brien** and **Mullan et al.**). But this is not in itself an explanation; one would also need to explain why people are susceptible to such marketing, and, in particular, why more insecure social groups appear to be more susceptible than others. Deeper principles are needed to explain such differential susceptibility, as **Smith** points out. The IH offers an alternative way of interpreting such differential susceptibility to ideas about constraints on self-control or agency.

R5. Dysfunction versus adaptationist explanations

Wells makes the important distinction between extra fat reserves and obesity (see also **Blackwell**). The IH as proposed provides no explanation for *obesity* (defined as extremely or pathologically high body weight). It offers an account of why food-secure individuals might carry more body fat than food-secure individuals, not of why anyone would have body mass indexes (BMIs) of 30 or 40. In a sense, we agree; we should strictly have titled our article

Fig. 6 - B/W online

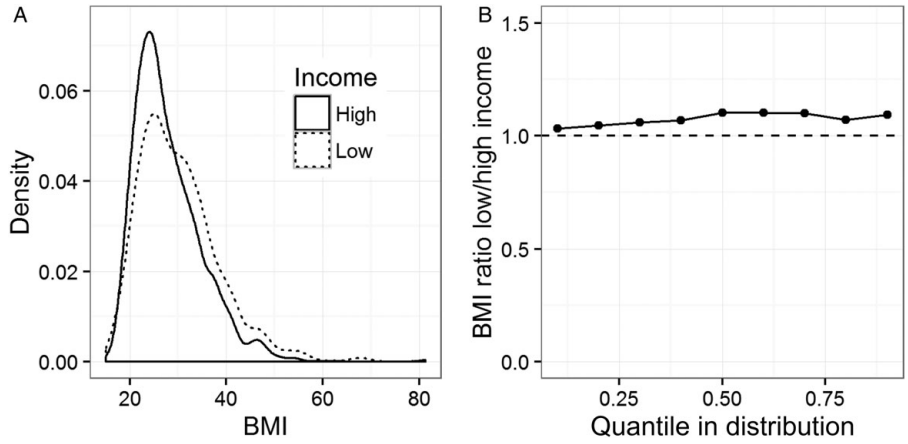

Figure R1. Distribution of BMI among adult U.S. women, from the National Health and Nutrition Examination Survey (NHANES), 1999–2004, using the NHANES R package (Pruim 2015). (A) Density plot of BMI for women in high-income households (> 200% of federal poverty level) and low-income households (≤ 200% of federal poverty level). (B) The ratio of BMI in low- and high-income households at each decile of the distribution. At every decile of the distribution, women from low-income households have higher BMI than their counterparts from high-income households.

"Food security as a driver of variation in body weight in humans." Wells stresses that obesity results from systems having gone wrong (dysfunction), not the operation of adaptive weight-regulation mechanisms. These kinds of debates often occur in the evolutionary medicine literature; how relevant is it to understand the normal function of the mood system in order to explain clinical depression, where the system has gone wrong (Nesse 2000)?

Our response would be that many extreme cases of obesity no doubt involve metabolic dysfunction, but BMI in contemporary populations follows a continuous distribution with no point of discontinuity in it. Thus, it is not obvious exactly where normal (i.e., maladaptive but not arising from system dysfunction) responses to an evolutionarily novel environment end and system dysfunction begins. In almost all studies, obesity is defined phenotypically using arbitrary BMI cutoffs. Thus, if food insecurity increases body weight over what it otherwise would be, then the proportion of individuals falling above the cutoff line will be higher under food insecurity than food security, wherever the line is set. This will be true regardless of whether metabolic pathology is present in the most obese individuals. Thus, a critical question for the relevance of the IH is whether social determinants like poverty and food insecurity are associated with a rightward shift in the whole distribution of body weights, in which case normally functioning adaptive weight-regulation mechanisms might be relevant, or the movement of a number of individuals from a healthy to a diseased mode of the distribution, in which case increased pathology is a more useful avenue of explanation.

Figure R1a plots the distributions of BMIs for adult women in the United States (from the 1999–2004 National Health and Nutrition Examination Survey [NHANES]), separated into high- and low-income households. (We are dividing by household income here rather than by food insecurity, as those were the variables immediately available to us. Given the strong association between income and food insecurity in the United States [Gundersen et al. 2011a], it is a reasonable conjecture that the pattern would be similar if we divided people into food secure and food insecure.)

As Figure R1a shows, the whole distribution of BMIs is shifted to the right in the low-income households. One simple mechanism producing this pattern would be an increase in body weight in poor women that applied multiplicatively (i.e., every low-income woman carries x% more weight than she would if rich). Figure R1b shows that such a shift is descriptively fairly accurate, by displaying the ratio of BMIs in poor as compared to rich households at each decile point in the distribution. Every decile point of the distribution is higher among the poor; that is, a woman in the thinnest 20% of poor women is heavier than a woman in the thinnest 20% of rich women. The proportionate increase is in the range of 3–10% for each decile and varies relatively little across the distribution. This is compatible with the meta-analytic evidence that food insecurity is associated with body weight regardless of whether the study uses mean BMI as the outcome variable or the probability of exceeding a cutoff. It is also compatible with our observation that associations are stronger when the higher BMI cutoff of 30 is used compared to 25. If the hypothesised weight-shift is multiplicative, then it will increase the right-skew of the distribution and produce the largest absolute (not proportional) difference at the highest body weights.

One reading of this evidence, then, is that body weight is multiply determined by factors including the food ecology, energy expenditure patterns, genetic variation, and the prevalence of metabolic or other pathologies, but cues of food insecurity also produce a proportionate increase in body weight (relative to not experiencing those cues). This would mean that problems of high and very high body weight would be exacerbated by food insecurity, even though food insecurity was not the sole shaper of the distribution. We absolutely accept that such multiple determinants must be at work, as we make clear in the target article. If food insecurity were the only driver, then, as **Lozano** points out, we should expect Western populations to have lower average adiposity than subsistence ones, which is clearly not the case. (On a related note, we find it strange that Lozano characterises us as dismissing the evolutionary mismatch hypothesis. We do not. We agree with him that the IH as we define it here is a

variant of, not an alternative to, the evolutionary mismatch hypothesis. He compares us to a tobacco company denying the smoking–cancer link because not every smoker gets cancer. On the contrary, one can accept that smoking causes cancer and still be interested in why some people are more vulnerable to the carcinogenic effects of smoking than are other people.)

A number of commentators suggest pathways by which weight regulation might become dysfunctional, through positive feedback between one component of the system and others, spiralling to the extremes of obesity (**Coppin**; **Blackwell**; **Davies, Cheke, & Clayton [Davies et al.]**) or anorexia (**Nesse**). These positive feedback loops potentially explain the "ratchet effect" mentioned by **Wells** and **Higginson et al.,** whereby individuals who are already overweight become more so over the long term. The existence of such ratchets may explain the right-skew of the distribution BMI in Western populations (Lang et al. 2016). However, the insurance principle is still relevant, because anything that causes the initial shift of body weight to the right will increase the probability of such positive feedback processes becoming established.

R6. The evidence base and alternative explanations for it

Lozano is technically incorrect when he says that only one out of six tests of the predictions of the IH using the food insecurity literature is significant. The single omnibus test at the heart of any meta-analysis – does the observed association differ significantly from zero in the whole data set? – is significant in the current case, which is one out of one. However, we go on to show that gender and country income are strong moderators of the overall association. More broadly, though, we agree with Lozano that the evidence base we review is equivocal and problematic, and it certainly does not offer emphatic support for the IH. There are two issues. First, although we offered some post hoc speculations about why associations might be weaker in men (sect. 6.2) and in low-income countries (sect. 6.3), it would be much more convincing if there were *some* evidence that the insurance principle was at work in these contexts, even if the response were more subtle.

Second, the significant correlational evidence that we found in women does not demonstrate causality; the association may indeed be explained by other processes or factors (**Boden & McLeod**). This is not unique to the food-insecurity literature. Indeed, it is a problem for all of epidemiology. It is not sufficient, though, to opine that such processes or factors could exist. We need testable proposals for what they are. As such, we welcome **Hruschka & Han**'s alternative account based on selection (lighter women end up in more food-secure households) rather than causation. This is indeed an alternative mechanism that could explain the distributions in Fig. R1. The selection account would seem to imply that (1) the association between food insecurity and body weight would disappear when socioeconomic variables such as income are controlled for, and (2) there should be no longitudinal prediction of weight gain by food insecurity, only a cross-sectional association. Our meta-analysis suggests that neither of these is the case. Nonetheless, Hruschka & Han's suggestion is a cogent one, and the predictions of selection versus causation need to be rigorously tested with data.

Two developments are needed to improve the evidence base and make a more definitive determination on the value of the IH. First, the range of evidence considered needs to be broadened. Our meta-analysis was restricted to studies using the food insecurity questionnaires as predictors and BMI as outcome. Such studies have a number of limitations, as discussed by **Nesse**, **Smith**, and others. Broadening the scope to include more general measures of economic insecurity as predictors increases the range of available high-quality evidence substantially, as Smith points out in his valuable commentary. Crucially, this evidence is often longitudinal and points to economic insecurity as a predictor of weight gain in men as well as women. Similarly, **Tapper** reviews evidence that children exposed to food restriction increase their food consumption, as the IH requires. Thus, although the evidence we meta-analysed does not find associations in children, there are other sources of evidence that the hypothesized response does exist. For non-Western societies, the food insecurity questionnaire-studies may find no average association, but there is evidence of fat reserves functioning to buffer seasonality and other forms of stochastic resource fluctuation in such societies (Wells 2012a; 2012b). Indeed, Wells (2012b), on the basis of non-Western and paleontological evidence, specifically argues that the human tendency to store fat is a risk-management strategy for dealing with environmental fluctuation, which is the essence of the IH.

Second, the research designs need to become stronger, as **Boden & McLeod** point out. The food insecurity literature is dominated with cross-sectional studies that have limited value for investigating causality. As many of our commentators point out (**Dohle & Hofmann**; **Hruschka & Han**; **Mullan et al.; Boden & McLeod**), we need more longitudinal evidence, which helps distinguish selection from causation. We also need instrumental-variable or natural-experiment studies. Examples could be policy changes affecting food insecurity that are introduced in one jurisdiction but not a neighbouring one, or exogenous events that expose some people but not others to temporary food restriction. These would allow better estimation of causal impact. Ultimately, we agree with **Cardel et al.** that randomized control trials are required to test the IH. Food stamp programmes may offer an opportunity here. In the United States, the Supplemental Nutrition Assistance Program (SNAP, commonly known as "food stamps") provides a monthly monetary amount for purchasing food. Families tend to use it up in the first three weeks of the month, producing a repeating cycle of consumption and hunger. Several studies have found that programme participation significantly predicts weight gain, including among men in some cases (reviewed by DeBono et al. 2012; Dinour et al. 2007). Randomized control trials could compare the impacts of different programme designs leading to greater or lesser temporal variation in availability. The predictions of the IH are clear. Smaller-scale experiments would also be valuable; for example, micro-comparisons of eating behaviour after short-term experimental manipulation of perceived food insecurity would help demonstrate if there really is a causal pathway of the kind the IH requires (cf. Cardel et al. 2015).

R7. Assumptions and predictions of our formal model

Several commentators questioned the applicability of a theory and formal model based on birds to mammals, such as humans. To clarify, there is nothing about the insurance principle or the formal model we present in the target article that is specific to birds, or any more applicable to birds than mammals. The model is general. The best experimental tests happen to have been in birds, though as **Nesse** points out there is supportive mammalian evidence too. **Hill, Proffitt Leyva, & DelPriore (Hill et al.)** argue that the model is too simplistic in not reflecting that one of the functions of fat stores is to fund reproduction. We agree that funding reproduction is an important function of adipose tissue, and moreover that sex differences in reproduction explain sex differences in adiposity. However, our model is not incompatible with this. All our formal model does is provide an algorithm for answering the question: If there is a given mapping between fat reserves and fitness in each time period, and a given probability of finding food in each time period, then what is the optimal amount of energy to consume and store? Although, for ease of exposition, we introduced our model by talking about the chances of surviving the time period, the model is actually agnostic about which factors determine the mapping between fat reserves and fitness. Survival, reproduction, and immunity could all contribute to setting the shape of the function shown in Fig. 1A of the target article. The model can be used to investigate the predicted effect of being a different sex simply by varying the shape of the mapping (sect. 6.2).

Hill et al. outline the need for models that explain why different taxa with different ecologies and lifecycles show different patterns of adiposity (and especially dimorphism in adiposity). We agree on the need for such models, but that is not what we were trying to do in this target article. In fact, it is complementary. Our model takes as *input* a mapping between fat reserves and fitness, and gives as *output* an optimal eating policy. Other kinds of models are needed that give a mapping between fat reserves and fitness as their *output*, using features of the organism's ecology, biology, and life history as the *input*, and thus making comparative predictions. Having said this, we agree with Hill et al. and **Chen** that another avenue for explaining the apparent sex difference in responsiveness to food insecurity is that cues of food insecurity, for women, might not only affect the reserves they need to store in order to execute a given reproductive strategy, but also affect what reproductive strategy they choose to execute. This deserves further investigation and formal modelling.

Wells suggests that the predictions of the model are falsified for humans, because the model predicts that those who are currently thinnest should gain weight, and those who are currently fattest should lose weight, whereas in human obesity, those whose body weights are highest to start with gain the most over time. But this is to confuse long-term and short-term dynamics. Over the short term, there is abundant human evidence that after a person's body weight is perturbed in either direction (e.g., due to illness or experimental intervention), it returns fairly rapidly close to a predisturbance, individual-specific set point (Speakman et al. 2011).

The short-term dynamics of human BMI distributions are dominated by regression to the centre: Those with high BMIs lose weight whereas those with low BMIs gain weight (Lang et al. 2016). This is what our formal model predicts. What we need to explain is the interindividual variation in what the set point is (and, relatedly, why the set point increases over time in some people). Here is where there is a role for chronic exposure to environmental factors, including but certainly not limited to food insecurity. Wells is right that the long-term increases in individual set points appears to be proportional to existing BMI; this is what explains the right-skew of the BMI distribution in Western populations (Lang et al. 2016). This points to the importance of ratchet effects, as discussed in section R5. Such proportionality does not arise in any obvious way from our formal model or the other behavioural-ecological models on which we based ours. To explain them will require better understanding of the proximate mechanisms involved in weight regulation, the potential for positive feedback among such mechanisms, and the mismatch between current and ancestral environments. On this point we are in agreement with **Higginson et al.,** and with Wells (2012b).

R8. Concluding remarks

We take several lessons away from this exercise. The first is that you can think you have been clear, but find that others have taken you to mean something quite different from what you intended. This is a particular peril of interdisciplinary efforts and seems to be acute whenever behavioural ecologists bring their arguments – which are based on a different level of analysis and may use the familiar terms with different meanings – to the scholarly communities of psychology, biomedicine, and public health. We are grateful to have had the chance to clarify some of these misunderstandings. Second, for a topic like obesity, there is more material to cover than can possibly be included in one article. Commentators were not shy in bringing this to light. In some cases, the additional material represents extensions and elaborations, and it is useful to be able to discuss these here. In other cases, we omitted evidence or distinctions that would have considerably improved our target article (by broadening the evidence base and thus averting some of the misunderstandings) had it been dealt with originally. We have highlighted some of these cases above.

The third lesson is that to explain a phenomenon like obesity involves not just integrating multiple factors at the same level of the explanatory web, but multiple *levels* of factors (e.g., information about the evolved function of weight-regulation mechanisms, about proximate psychological and physiological mechanisms, about pathology and dysregulation, about developmental influences, and about contemporary food ecologies and the inequalities in them). Achieving this integration is extremely challenging, as the existence of poorly connected literatures in each of these different areas demonstrates; each of the literatures takes a different set of axioms and assumptions about relevance as its starting point. The variation in the commentaries shows this very clearly. Regardless of what value the IH as set out in our target article turns out to have, if any, we feel that the attempt to integrate these different kinds of information has been a valuable one.

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