Nettle D, Andrews C, Bateson M. Food insecurity as a driver of obesity in humans: The insurance hypothesis. *Behavioral and Brain Sciences* 2016

DOI: http://dx.doi.org/10.1017/S0140525X16000947

Date deposited:

15/08/2016

Embargo release date:

28 January 2017

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

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Short abstract

Common sense says that obesity is the consequence of too much food. Adaptive reasoning says something rather different: individuals should store fat when access to food is insecure, to buffer themselves against future shortfall. Applied to humans, this principle suggests that food insecurity should be a risk factor for overweight and obesity. We provide a meta-analysis of the extensive epidemiological literature, finding that food insecurity robustly predicts high body weight, but only amongst women in high-income countries. We discuss the relevance of food insecurity to understanding the global obesity problem.

Long 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 whilst 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.

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

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

1. Introduction

The prevalence of obesity and overweight is increasing across almost all countries of the world (NCD Risk Factor Collaboration, 2016; Wang, McPherson, Marsh, Gortmaker, & Brown, 2011). This is considered to constitute one of the major current global public health challenges. 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 to eat, and of which foods— 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 paper, but it is worth stating its main constituent claims up front. These are the following:

- 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 amongst 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, 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 the paper, 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 over two decades ago (see section 3), and have been most thoroughly tested empirically in birds (see section 4). There is already an extensive human social science literature on the relationship between obesity and food insecurity (see section 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 to the empirical evidence from non-human animals. Thus, our goal in this paper 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.

2. Existing approaches to the psychology of human obesity

The IH is fundamentally a psychological hypothesis, since 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 over-consumption, 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 (see McNamara, Houston, & Higginson, 2015 for a recent version of this argument). A variant of the evolutionary mismatch hypothesis states that it is energy expenditure, rather than food supply, in modern environments that falls outside the ancestral range (Prentice & Jebb, 1995). Since ancestral energy expenditure was always high, we do not downregulate 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 lacked mechanisms to appropriately limit their intake of energy-dense foods when they 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 cut-off for classification as overweight (Wang et al., 2011). Even in the USA, which has very high rates of obesity, around one third of adults are neither overweight nor obese (Wang, Beydoun, Liang, Caballero, & Kumanyika, 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, Kelly, Brunner, Lobstein, & Wilkinson, 2005), or (relatedly) where large numbers of individuals face economic insecurity (Offer, Pechey, & Ulijaszek, 2010). For example, whereas the 2014 rate of adult obesity (BMI ≥ 30) is 33.7% in the unequal USA, it is only 3.3% for Japan and 19.4% for Switzerland (World Health Organization, 2015). Yet it would be hard to argue 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 fails to predict the within-country variation too. Within high-income countries, obesity has been consistently linked to 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 who carry the most body fat. Thus, whilst 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, Stanczyk, Nederkoorn, & Jansen, 2012; Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006; Weller, Cook, Avsar, & Cox, 2008). The central finding of this literature is that obese individuals are relatively impulsive (present-oriented, unable to delay gratification, sensitive to reward, etc.). Since 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, Houben, & Jansen, 2015; Houben, Nederkoorn, & Jansen, 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. While 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, McNamara, & Houston, 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 covary within a species, including within humans (Garby et al., 1988; Johnstone, Murison, Duncan, Rance, & Speakman, 2005; Leibel, Rosenbaum, & Hirsch, 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 their ability to buffer periodic shortfalls, but do so at the cost of requiring greater energy intake to maintain their 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, Fransson, & Jakobsson, 1996; O'Hagan, Andrews, Bedford, Bateson, & Nettle, 2015; Witter, Cuthill, & Bonser, 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 3,000m and further, 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 400m 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 (for example, osteoarthritis, Bray, 2004; Felson, 1988).

In view of the consequences of increased body weight, behavioural ecologists have long accepted that increased fat storage has both benefits, in terms of enhanced ability to buffer shortfalls, and 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 showed, 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; Higginson, McNamara, & Houston, 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 paper.

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, whilst Online Appendix A provides the details. Our model uses a similar approach 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 appears 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 steeply with increasing body weight. It is reasonable to assume that it should not do so, since 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 (figure 1A): there is a cliff-edge at the critically-low threshold, and a gentler slope away 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 section 6.2 below 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. Since 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 maximises 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, and the maximand the probability of successful reproduction. The computations and predictions would be the same under this interpretation. This is important since 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 (figure 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, it should take in 7 units per period. When *p* = 0.8, eating only kicks in when reserves drop to 4 units, and the most it 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 (figure 1C). As the figure 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*. Since 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 individual's 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.
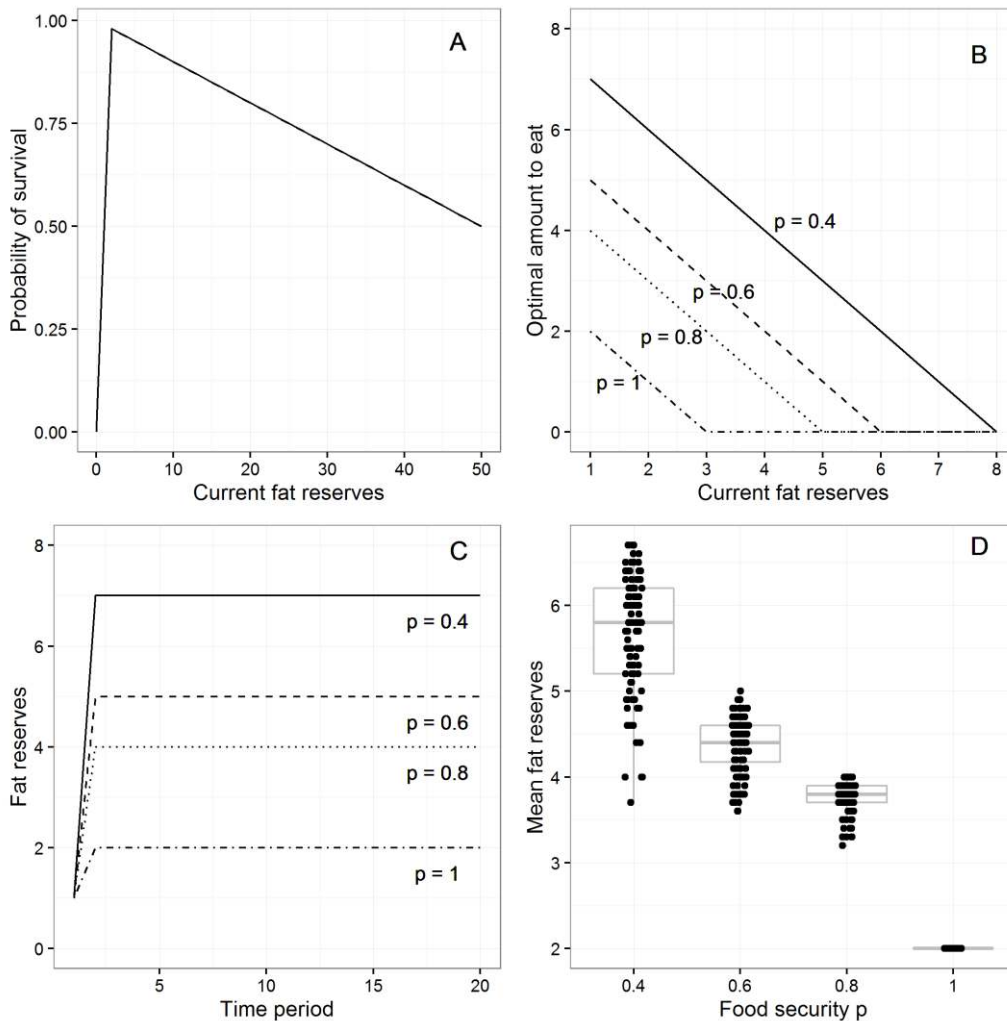
Figure 1. Output from the model described in section 3 (for details, see Online Appendix A). A. The assumed probability of survival against current level of fat reserves. B. The optimal amount to eat for different levels of fat reserves and four values of the food security parameter *p*. C. Fat reserves 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.

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 amongst those whose access to food is assured, but exactly amongst 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 paper) 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. Since 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 (see Witter & Cuthill, 1993 for review).

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. Ten of eleven birds significantly increased their weight in response to the unpredictable regime; the lightest and leanest individuals showed the strongest response. Witter, Swaddle and Cuthill (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, whilst 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 (see Clark & Ekman, 1995 for a related theoretical model). 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 amongst 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, Cope, Johnson, Smith, & Nagy, 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' 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) ask, "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' 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, Bergen, & Yeh, 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, Olson, & Frongillo, 1995; Radimer, Olson, Greene, Campbell, & Habicht, 1992) and its derivative, the US Department of Agriculture's core

household food security module (Nord, Andrews, & Carlson, 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, though in practice this is often reduced to a food secure/ food insecure dichotomy, or a three-way or 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, Gundersen, Lohman, Garasky, & Stewart, 2011; Franklin et al., 2012; Laraia, 2012; Larson & Story, 2011; Morais, Dutra, Franceschini, & Priore, 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 USA) from which most of the evidence comes.

5.2 Meta-analysis methods

Whilst 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. Since 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 it 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 ≥ 30] versus normal weight, for others overweight [BMI ≥ 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 (for example, 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 multi-level 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 or not the authors controlled for covariates 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 summarise 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 figure 2). The central LOR estimate of 0.19 corresponds to an OR of 1.21 (95% CI 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 figure 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, Kreider, & Pepper, 2011). If this was the mechanism producing the association, we would expect estimated associations from analyses that control for socioeconomic and demographic factors to be substantially weaker than those from unadjusted analyses. This was not the case: the adjusted LORs in the dataset 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). Since 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 ≥ 30) than when it is

the less extreme outcome overweight (BMI ≥ 25; lines 7, 8 and 9 of figure 2). We found no significant differences in association strength according to exactly which predictor was used (lines 10-14 in figure 2). This is of note since in one of the most influential studies (Townsend, Peerson, Love, Achterberg, & Murphy, 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.

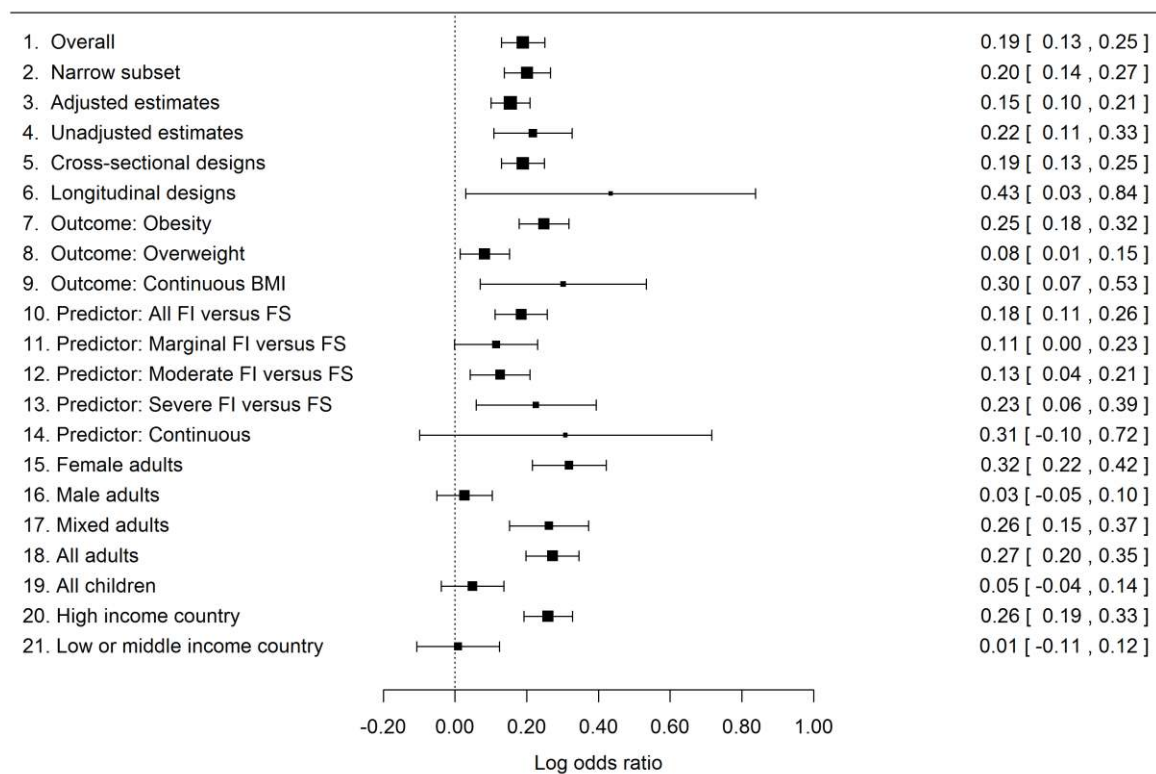|	Log odds ratio
1. Overall	0.19 [0.13 , 0.25]
2. Narrow subset	0.20 [0.14 , 0.27]
3. Adjusted estimates	0.15 [0.10 , 0.21]
4. Unadjusted estimates	0.22 [0.11 , 0.33]
5. Cross-sectional designs	0.19 [0.13 , 0.25]
6. Longitudinal designs	0.43 [0.03 , 0.84]
7. Outcome: Obesity	0.25 [0.18 , 0.32]
8. Outcome: Overweight	0.08 [0.01 , 0.15]
9. Outcome: Continuous BMI	0.30 [0.07 , 0.53]
10. Predictor: All FI versus FS	0.18 [0.11 , 0.26]
11. Predictor: Marginal FI versus FS	0.11 [0.00 , 0.23]
12. Predictor: Moderate FI versus FS	0.13 [0.04 , 0.21]
13. Predictor: Severe FI versus FS	0.23 [0.06 , 0.39]
14. Predictor: Continuous	0.31 [-0.10 , 0.72]
15. Female adults	0.32 [0.22 , 0.42]
16. Male adults	0.03 [-0.05 , 0.10]
17. Mixed adults	0.26 [0.15 , 0.37]
18. All adults	0.27 [0.20 , 0.35]
19. All children	0.05 [-0.04 , 0.14]
20. High income country	0.26 [0.19 , 0.33]
21. Low or middle income country	0.01 [-0.11 , 0.12]

Figure 2. Estimated log odds ratios for high vs. normal body weight in food insecure vs. 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 text and Online Appendix B.

All-male adult samples showed significantly weaker associations than all-female or mixed-sex ones (which are often female-biased; lines 15-17 of figure 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. 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. 41 papers still constitutes 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 Online Appendix B, section B3.2).

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 find significant positive associations in line with the high-income country evidence (e.g. Chaput, Gilbert, & Tremblay, 2007 in urban Kampala), whilst there are several associations in the opposite direction (i.e. food insecurity reduces odds of overweight: Dubois et al., 2011; Isanaka, Mora-Plazas, Lopez-Arana, Baylin, & Villamor, 2007) in children. The geographical coverage of the dataset is very uneven: 209 of the 301 associations came from high-income countries, and 178 of these from the USA. More evidence is thus needed from different kinds of samples in the developing world, and also from non-US 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 dataset assembled through a search of the literature will over-estimate 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, Garasky, & Lohman, 2009; Townsend et al., 2001). These both featured large, nationally-representative samples (from the US *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 US women, and an LOR not significantly different from 0 (exact value and CI unstated) for US men. The aggregated studies from high income countries in our dataset give LORs of 0.42 (95% CI 0.29 – 0.55) for women and 0.03 (95% CI -0.05 - 0.10) for men. The individual LOR from Gundersen, Garasky and Lohman (2009) for US children (0.13, 95% CI -0.17 – 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, Davey Smith, Schnieder, & Minder, 1997; see Online Appendix B section 3.3 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 Online Appendix B section B3.3).

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 USA. For example, the mapping between cues of food insecurity and evolutionary fitness might be quite different in contemporary environments than in historical ones.

Whilst 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, though 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 section 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 (figure 1A) different for men and women. The model allows three ways of doing this (see Online Appendix A sections 3.2-3.4). First, we can move the location of the fitness cliff-edge further to the right for women (figure 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 (figure 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 (figure 3C). This is another way of capturing the intuition that for women there are costs of low reserves 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 figure 3C). However, it does not lead to greater responsiveness to changes in the level 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 (figure

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 figure 3E) produce individuals who maintain lower average reserves, and are also less responsive to the current level of food insecurity p (figure 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 intra-specific violent conflict, thought to be an important selection pressure in ancestral human societies (McDonald, Navarrete, & Van Vugt, 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, since 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.
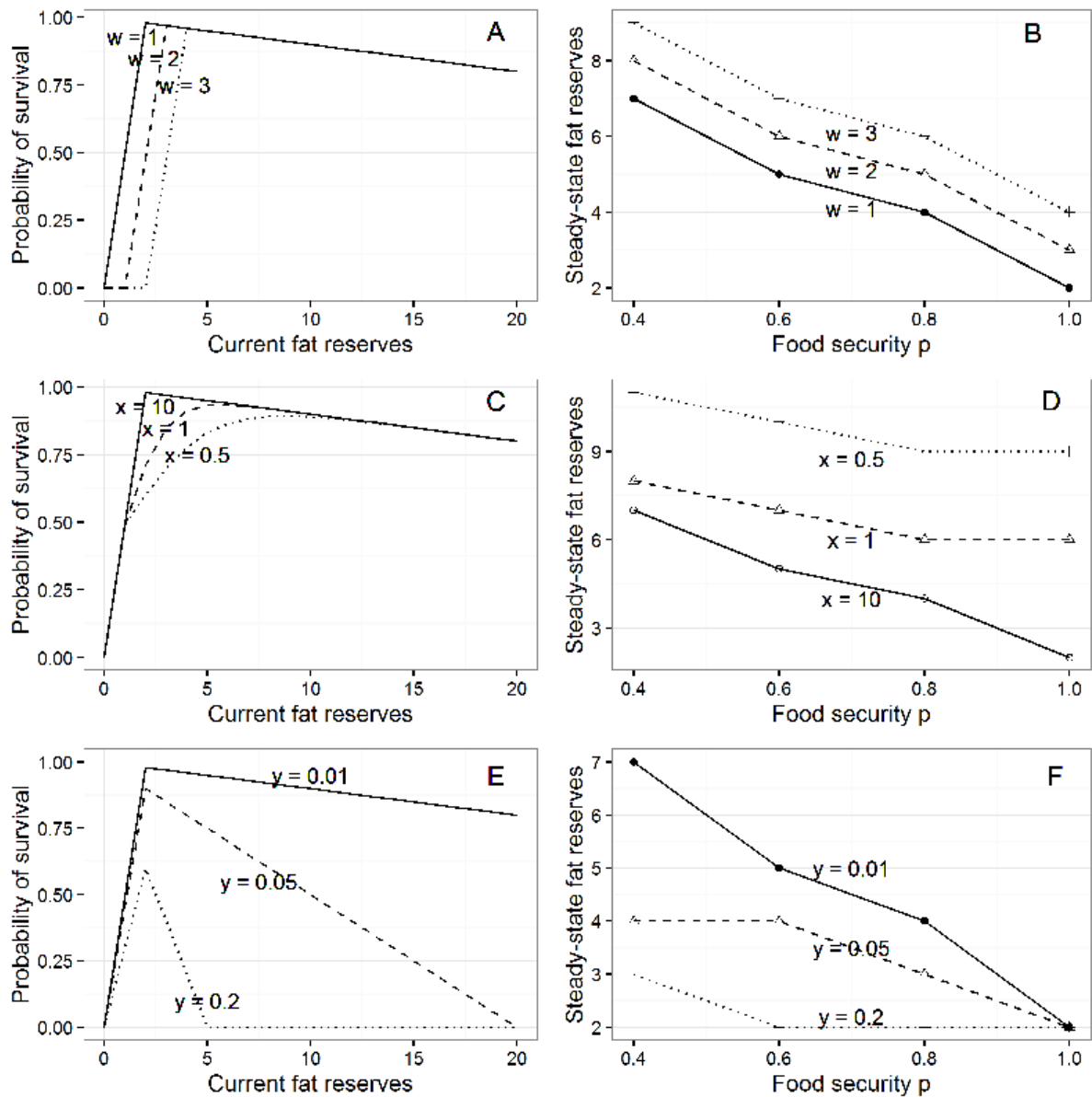
Figure 3. Modifications to the model from section 3 to explore potential explanations for sex differences. See Online Appendix A, sections 3.2-3.4 for full details. A. Three different locations for the cliff-edge below which starvation becomes likely (controlled by parameter *w*). B. Steady-state target 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-hand 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-hand 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 when food is available, it may not be energy-dense enough to allow the build-up of fat reserves before the next period of scarcity strikes.

In Online Appendix A, section A3.5, 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 (figure 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 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.
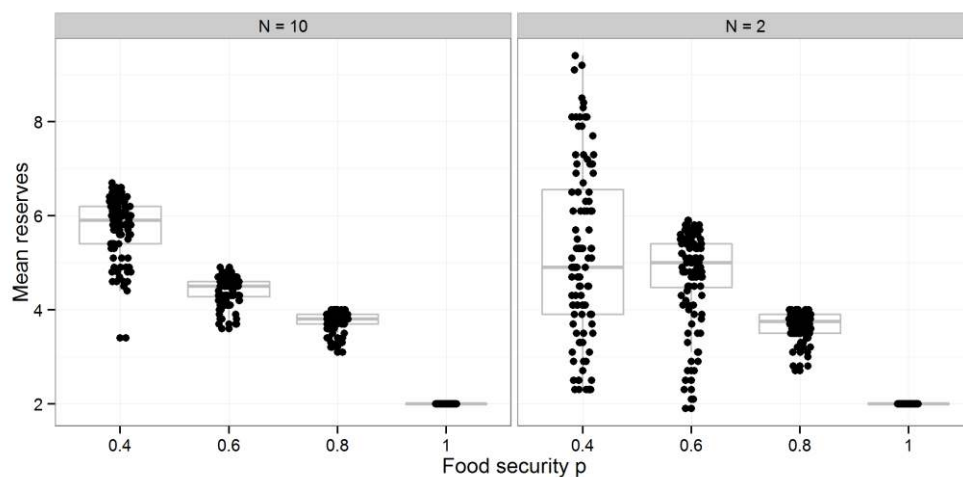

Figure 4. Mean level of reserves over 40 days for simulated individuals experiencing different levels of food security p, for two different values of the maximum energy available from food per period, N. When N is small, p becomes a poor predictor of body weight as the variability between individuals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

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 should 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, Crawford, Nadeau, & Lindberg, 1992; Tovee, Swami, Furnham, & Mangalparsad, 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 subjects 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 characterised 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 industrialised 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 one [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, Neale, & Eaves, 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 paper not only allows, but 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, Mangel, Metcalfe, & Huntingford, 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). *Contra* 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, and 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 section 2 and models like it, due to the asymmetry of the survival function shown in figure 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, though potentially explicable, undermines any claim that the IH by itself 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, Barker, Osmond, Fall, & Simmonds, 1992; though see Rogers, 2003), childhood exposure to food scarcity (Olson, Bove, & Miller, 2007), or psychosocial stress more generally (D'Argenio et al., 2009; Greenfield & Marks, 2009; Gundersen, Mahatmya, Garasky, & Lohman, 2011; Gunstad et al., 2006). 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, Bateson, Bedford, Brilot, & Nettle, 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, Frankenhuis, & Rickard, 2013; Stamps & Krishnan, 2014). It depends, amongst 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 counterproductive in the majority of individuals (Mann et al., 2007; Pietiläinen, Saarni, Kaprio, & Rissanen, 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

Whilst 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, Reba, Siega-Riz, & Reichborn-Kjennerud, 2005). Though a full discussion is beyond the scope of this paper, 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, Heshmati, & Koupil, 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). Since 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, since 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, Amianto, Delsedime, De-Bacco, & Fassino, 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, Omeara, Melville, Sheldon, & Wilson, 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 over-ride them. Certain interventions, such as restrictive dieting, in fact look potentially counterproductive. Indeed, the IH suggests the interventions most

likely to work are the very antithesis of restrictive dieting: in the words of Dietz' original paper, the IH suggests "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.

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Online Appendix A: Theoretical model

This appendix presents in detail the theoretical model outlined in section 3 of the main paper. Section A1 gives details of the modelling methods and assumptions. Section A2 demonstrates that the model converges to give an optimal eating policy. Section A3 examines the consequences of varying each of the principal parameters of the model. Section A4 gives details of implementation, code availability and instructions for running the model.

A1. Modelling framework

As described in the main paper (section 3), we consider individuals who carry some level of stored fat reserves r ($0 \leq r \leq 50$) with an energy requirement $e(r)$ in each time period as follows:

$$e(r) = a + br$$

Here, a is a fixed lean-mass metabolic requirement, and b is a parameter controlling how strongly the energy requirement increases with increasing body weight. All results unless otherwise stated use the simplest possible scenario of $a = 1$ and $b = 0$. That is, the energy requirement is assumed to be a fixed 1 unit of energy in each time period. Sections 3.1 and 3.2 of this document explore the consequences of varying a and b.

The individual is deemed to have starved to death if energy reserves fall to zero. Where reserves are greater than zero, survival each period is given by:

$$s(r) = \frac{1}{1 + e^{-x(r-w)}} - \mathrm{yr}$$

As we can see, this function has two additive components. First, there is a logistic function increasing in reserves. This models the probability of avoiding death by starvation, and the logistic function is used to capture the intuition that death by starvation in a time period, which is certain at $r = 0$, rapidly becomes very unlikely as long as the individual has a threshold level of reserves $r = w$. We use $w = 1$ throughout unless specifically stated otherwise. The logistic function is controlled by a steepness parameter x. Where x is large (e.g. $x = 10$), the probability of avoiding starvation in a time period approximately follows a step function: 0 at reserves of 0, 0.5 at reserves of 1, 1 at reserves of 2. All results use $x = 10$ unless otherwise stated. The second component of the survival function is a linear decrease in survival with increasing resources. This models the increase in morbidity and decrease in mobility as the individual becomes heavier. It is controlled by another steepness parameter y, which represents the survival cost of each additional unit of weight. We use $y = 0.01$ throughout except where otherwise stated. We explore the consequences of varying w, x and y for optimal eating and weight regulation in sections 3.3 and 3.4 of this document.

Each time period, the individual finds food with probability p. If food is available, the individual consumes n units ($0 \leq n \leq N$; $N = 10$ unless otherwise stated); its reserves will thus change by $n - e(r)$ units. If it does not find food, its reserves will change by $-e(r)$. We consider the consequences of varying N, the maximum amount of energy that can be consumed in a time period, in in section 3.5 of this document.

To compute the optimal policy, we consider a run of *T* time periods (the value of *T = 100* is used for all results presented; see section 2). We need to determine the survival function *F(r, t)*, which gives the maximum probability of survival to time *T* for an individual with fat reserves *r* at time *t.* First, we note that if the individual is still alive at the final time period, then they have survived. Hence:

$$F(r, T) = \begin{cases} 1 & r > 0 \\ 0 & otherwise \end{cases}$$

For each earlier time step, we can write down the dynamic programming equation:

$$F(r, t) = \begin{cases} \max_n(s(r)[p(F(r + n - e(r), t + 1) + (1 - p)(F(r - e(r), t + 1)]) & r > 0 \\ 0 & otherwise \end{cases}$$

Note that the metabolic requirement in each time period is rounded to the nearest full unit, and the amount eaten also has to be an integer number of units. Given that we now have a survival function for the final time period, and for each time period given the next step, we can work backwards to time 1 by repeated application of the dynamic programming equation. This allows us to determine *n**, which is the amount to eat that produces the maximum survival probability for each possible value of *r* when *t = 1*. This is what is referred to throughout as the optimal policy; the amount to eat that maximizes survival into the distant future for every possible level of current fat reserves.

A2. Model convergence

Figure A1 shows the optimal amount to eat, as determined by application of the dynamic programming equation, for each possible level of current reserves at each time period, with *T* = 100. In the final few time periods, individuals maximise survival by stopping eating in order to reach the terminal time point with no excess. However, as long as the terminal time point is moderately distant, an optimal eating policy emerges that depends on current reserves only and not on time. This convergence occurs for all values of *p* within fewer than 20 time periods. Thus, using *n** at *t = 1* with *T* = 100 as the eating policy that maximizes survival into the distant future appears justified.
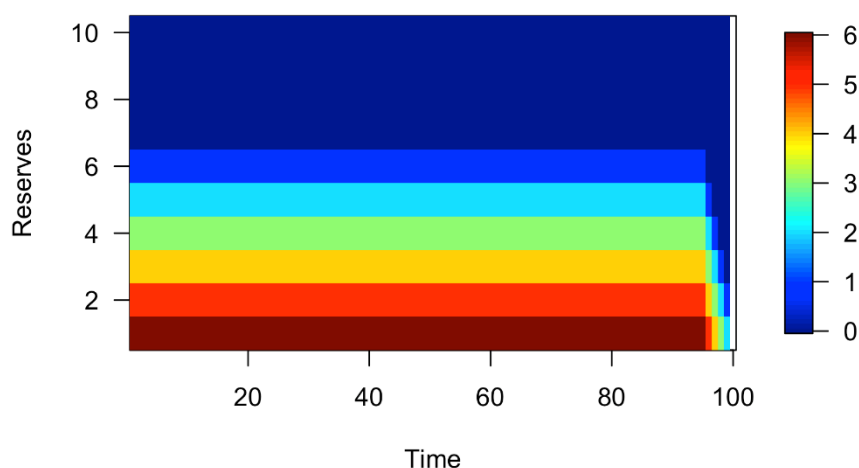

Figure A1. Amount to eat that maximises survival to time period 100 (colours), in relation to current time period (horizontal axis) and current reserves (vertical axis), for *p* = 0.5. All other parameters have their default values.

A3. Varying model parameters

This section investigates the effects of changing the values of each of the principal parameters of the model on the relationship between food security and fat reserves.

A3.1 Varying the fixed metabolic requirement

In section 3 of the main paper, the fixed component a of the metabolic requirement per time period is set to 1. Here we investigate the consequences of varying it. Repeating figure 1B of the main paper but with a set successively to 1, 2 and 3 produces the results shown in figure A2. Increasing a leads, unsurprisingly, to optimal policies that eat more per time period, but decreasing food security p still produces the same qualitative effect: it leads to eating more at a given level of reserves. In fact, the impact of p on amount eaten and hence reserves maintained becomes more marked when a is larger.
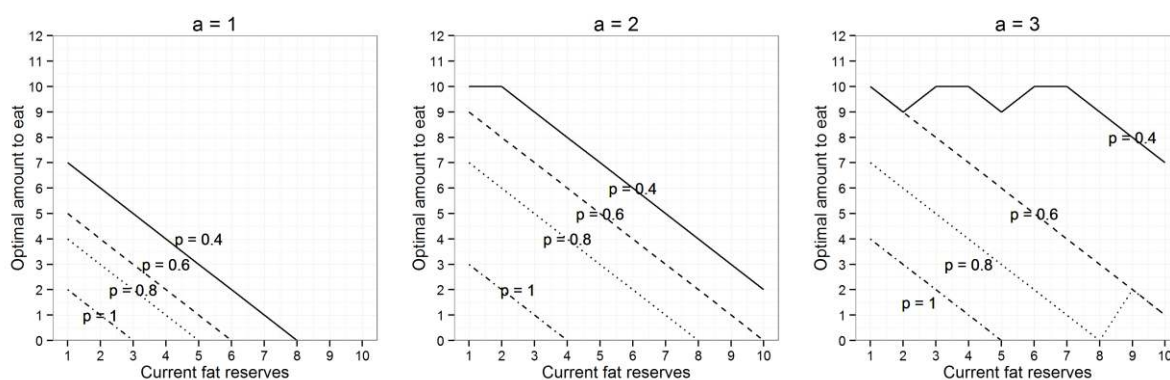

Figure A2. Figure 1B of the main paper repeated with different values of a, the fixed component of the metabolic requirement per time period. All other parameters have their default values.

A3.2 Varying the mass-dependent metabolic requirement

In section 3 of the main paper, b (the mass-dependent component of metabolic requirement) is set to zero in order to illustrate the simplest possible scenario. Here we investigate the consequences of giving this parameter a non-zero value, though we only consider cases where $b < a$, since lean mass has a larger influence on metabolic rate than fat mass (Garby et al., 1988; Johnstone, Murison, Duncan, Rance, & Speakman, 2005). Holding a at 1, we consider three values of b, 0, 0.2, and 0.5. For each level of b, we plot the optimal policy for four values of p (0.4, 0.6, 0.8 and 1), as in figure 1B of the main paper. Figure A3 shows the results.

The central result—that the optimal point to start eating and the optimal amount to eat increase as p decreases—is not altered by increasing the value of b up to 0.5. In fact, the increase in food consumption as p decreases becomes more marked with increasing b. This is because the eating policies for lower p have to eat to fund the additional metabolic requirements of the buffer they will need to build up, whereas the policies for higher p will build up little buffer and hence experience little increased metabolic cost.
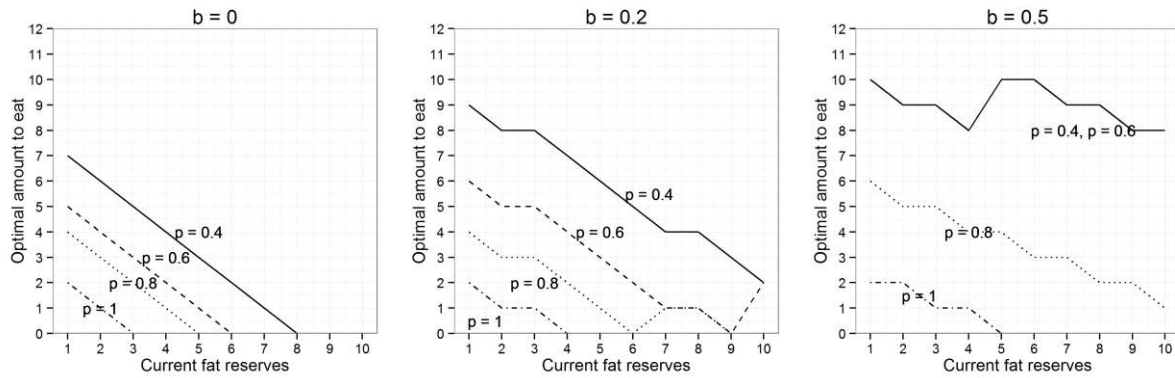
Figure A3. Figure 1B of the main paper repeated with different values of *b*, the parameter controlling how strongly metabolic requirements per time period increase with increasing weight. All other parameters have their default values.

A3.3 Varying the parameters of the logistic component of the survival function

In results presented thus far, the survival function in each time period steps up abruptly around $r = 1$, so that starvation, which is certain at $r = 0$, is essentially impossible at $r = 2$. We can vary this assumption in two ways. First, we can increase the value of the location parameter *w* in the logistic component of the survival function. This has the effect of moving the fitness cliff-edge to the right without changing its steepness (figure A4, panel A). Figure A4 panel B shows the consequences of increasing *w* for the relationship between food security and steady-state target reserves (i.e. the level at which the individual will stabilise under the optimal policy if it finds enough food to do so). As the figure shows, and unsurprisingly, increasing *w* increases the steady-state target level of fat reserves at any value of *p*. It does not however, change the gradient of the relationship between *p* and steady-state fat reserves. Thus, increasing *w* should be expected to produce individuals who are fatter at all levels of *p*, but no more or less responsive to their level of food insecurity.
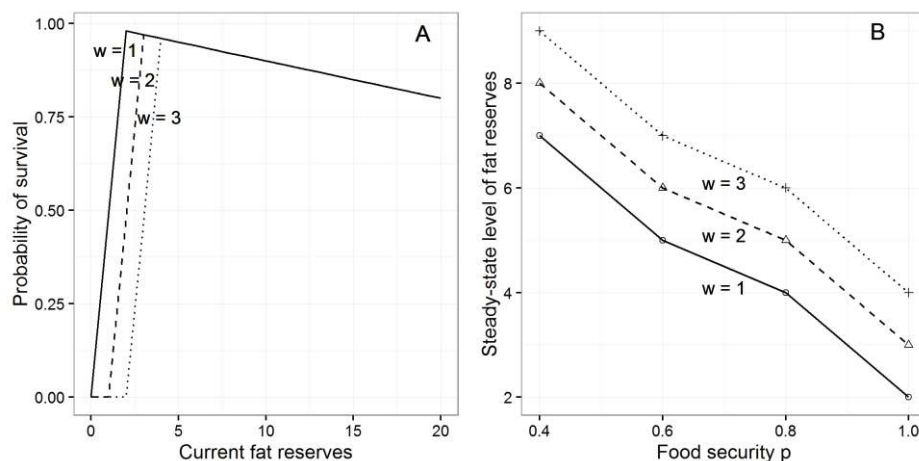

Figure A4. A. The probability of survival against current fat reserves for three values of the logistic location parameter *w.* B. The steady-state level of fat reserves that optimally-behaving individuals reach if they find food in every period against *p*, the level of food security, for three values of *w.* All parameters other than *x* have their default values. This figure is reproduced as part of figure 3 of the main paper.

Second, we can change the steepness parameter x, and hence make the increase in survival more gradual with increasing reserves (figure A5, panel A). Figure A5, panel B, shows the effect of food security on steady-state target reserves (i.e. the level at which the individual will stabilise under the optimal policy if it finds enough food to do so), for different values of the steepness parameter x. As the figure shows, decreasing x leads to individuals optimally carrying more fat. This is unsurprising since the effect of decreasing x is to move the point of maximal survival to the right (see figure A5, panel A). However, a secondary effect of reducing x is that individuals become somewhat less responsive to food insecurity; the difference in optimal reserves between $p = 0.4$ and $p = 1$ is 5 units when $x = 10$, but only 2 units when $x = 0.5$. Thus, a survival function that is less step-like at the lower end leads to individuals carrying more fat, but also being somewhat less responsive to their food security.
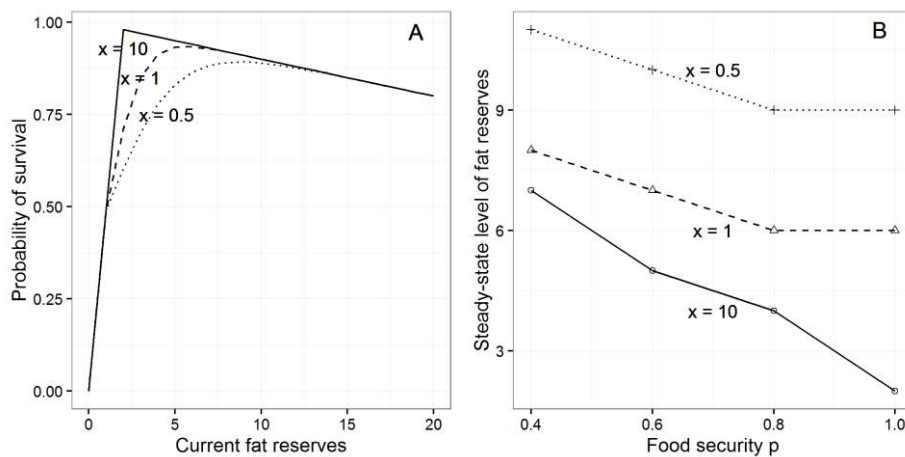

Figure A5. A. The probability of survival against current fat reserves for three values of the logistic parameter x. B. The steady-state level of fat reserves that optimally-behaving individuals reach if they find food in every period against p, the level of food security, for three values of x. All parameters other than x have their default values. This figure is reproduced as part of figure 3 of the main paper.

A3.4 Varying the steepness of the linear component of the survival function

The slope of the right-hand arm of the survival function is controlled by the parameter y in the model. In this section we investigate the consequences of making y larger, and hence the survival cost of every extra unit of weight greater. We do this by considering the consequences of using y values of 0.05 and 0.2 as well as the 0.01 used until this point (whilst keeping all other parameters, including x, at their usual values). This produces the three survival functions shown in figure A6, panel A.

We now compute the steady-state target reserves (i.e. the level at which the individual will stabilise under the optimal policy if it finds enough food to do so), for different values of y (figure A6, panel B). As the figure shows, a higher y leads to individuals carrying less fat at all levels of p except $p = 1.$ Moreover, increasing y also makes individuals less responsive to food insecurity. The difference in steady-state reserves between $p = 1$ and $p = 0.4$ is 5 units for $y = 0.01$. It is only 1 units for $y = 0.2$. This is a logical result: making the carrying of a buffer more costly reduces the size of buffer individuals should carry.
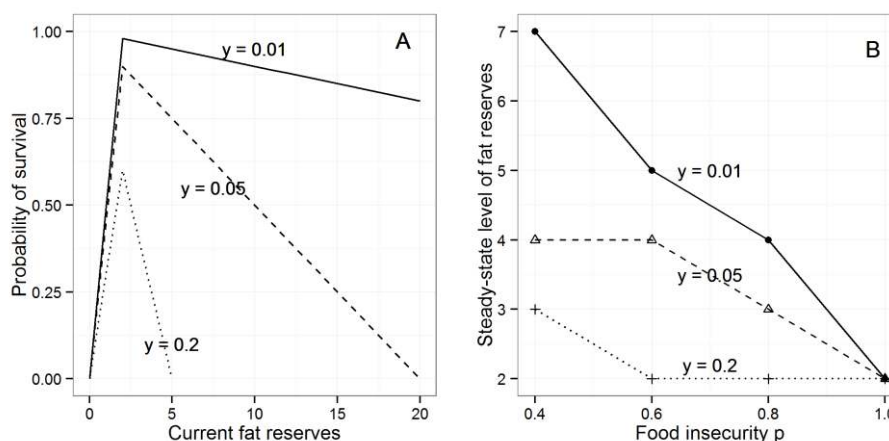
Figure A6. A. The probability of survival against current fat reserves for three values of the parameter *y*. B. The steady-state level of fat reserves that optimally-behaving individuals reach if they find food in every period against *p*, the level of food security, for three values of *y*. All parameters other than *y* have their default values. This figure is reproduced as part of figure 3 of the main paper.

A3.5 Varying the energy density of food

As a final variation of the parameters of the model, we investigate limiting *N* (the maximum energy available from food per time period assuming food can be found). The previously used value of *N = 10* effectively meant that individuals could build up a large reserve in a single time period in which food is available. We now explore setting *N* at the lower values of 3 and 2; this captures a situation where even when food is available, its energy-density is not sufficient to be able to consume many more calories than needed for metabolism in a given period. First we show the consequences of following the optimal eating policy and finding food in every period, for high food insecurity (*p* = 0.4), for the different values of *N* (figure A7). As the figure shows, the lower *N* is, the higher the individual's steady-state target weight (this is only true for high levels of food insecurity, *p* < 0.6). However, the lower *N* is, the more slowly the individual is able to put on weight to attain that target.
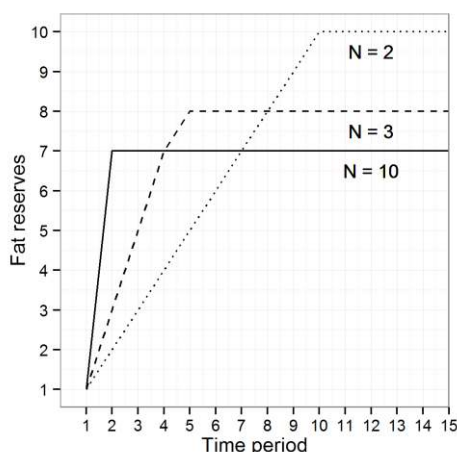

Figure A7. Level of fat reserves over time for individuals experiencing *p* = 0.4 who start with reserves of 1, follow the optimal eating policy, and find food every period for three values of the maximum energy available from food per period (*N*). All other parameters have their default values.

6

Next, we consider the consequences of decreasing *N* for the relationship between food security and average reserves/weight. We do this by repeating the simulations underlying figure 1D of the main paper, but with *N* = 2 as well as *N* = 10*.* The results are shown in figure A8. As the figure shows, when *N* = 2, the variance in individual weight within a value of *p* becomes substantial, particularly when *p* is low. This is because although individuals have a high steady-state target weight, in practice they are often operating well below it, because stochastic runs of foodless periods reduce their reserves, and it takes them a long time to build their reserves back up again because of the limited energy available in the periods when they do find food. The effect of this increased variability *within* groups of individuals at the same level of *p* is to attenuate the statistical relationship between *p* and mean weight. In the data underlying the left (*N* = 10) panel of figure A8 (and excluding individuals for whom *p* = 1), the value of *p* explains 77% of the variance in mean body weight. In the data underlying the right (*N* = 2) panel, the value of *p* explains only 20% of the variance in mean body weight, even though the relationship between steady-state target weight and food insecurity is actually steeper for the *N* = 2 world than the *N* = 10 world. We consider how variation in the energy-density of available food might explain differences between high- and low-income countries in terms of the relationship between food insecurity and obesity in section 6.3 of the main paper.
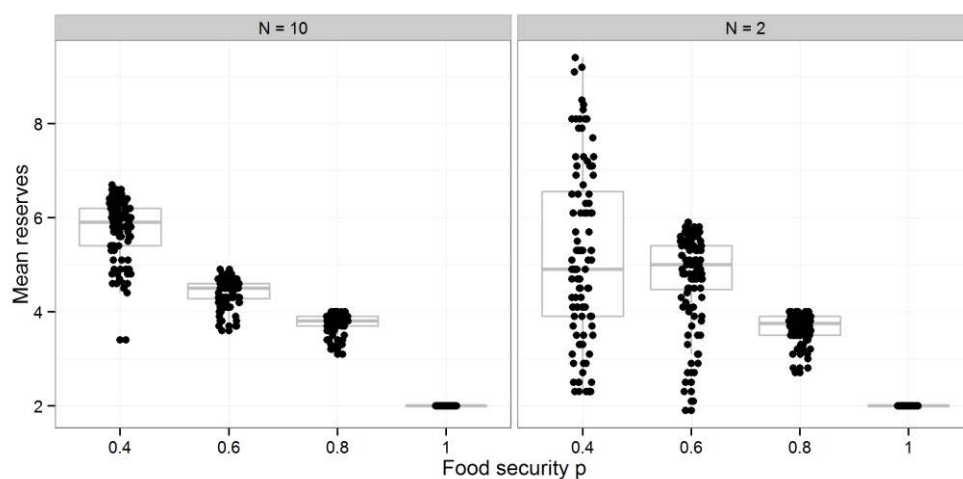

Figure A8. Mean body weight over 40 periods for simulated individuals at different levels of *p*, for two different values of the maximum energy available from food per period, *N.* Points have been jittered in the horizontal dimension to make individual data points more visible. All other parameters have their default values. This figure is reproduced as figure 4 in the main paper.

A4. Implementation and code availability

The model is programmed as a series of functions in R (R Core Development Team, 2015) and available via the Open Science Framework at https://osf.io/zarn6/. There are two R scripts. Sourcing the script 'obesity model functions.r' makes all the functions underlying the model available and allows for the user's own exploration of the model. The second script, 'obesity model for replication.r' produces all the figures and output from this document and the main paper, and relies on 'obesity model functions.r' having previously been run.

The main functions are as follows. The *policy* function gives the optimal eating policy for a given value of *p*, in the form of a vector corresponding to increasing levels of current reserves. Thus *policy(p=0.5)* will produce the a vector of 50 optimal amounts to eat corresponding to reserves of 1-50, for the specified value of *p*. In this and other functions, the parameters *w, x, y, a, b* and *N* are given their

default values unless otherwise specified (i.e. *w*=1, *x*=10, *y*=0.01, *a*=1, *b*=0, *N*=10; you can vary them by specifying the required value in the function call). The *simulate.food* function gives the weight trajectory of an individual who follows the optimal eating policy for a given value of *p* and finds food every time period. Its output is a vector indexed by time period. For example, *simulate.food(current.p=0.8, reps=20)* gives a vector of reserve/weight levels corresponding to 20 successive time periods for an individual following the optimal policy for *p=0.8*.

The *simulate* function is the same as *simulate.food* except that food is found with probability *p* in each time period. Thus, every run is unique. The call *simulate(current.p=0.8, reps=20)* gives a weight history for an individual facing *p*=0.8 and following the optimal policy. The first ten time periods are removed to avoid initialization artefacts. *NA* means the individual has died. Finally, *run.of.simulations* automates a run of many simulations for varying values of *p* and saves the output (see script for details).

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Nettle, Andrews & Bateson: Food insecurity as a driver of obesity in humans: The insurance hypothesis

Online Appendix B: Meta-analysis methods and descriptive statistics

This appendix presents more details of the methods of the meta-analysis reported in section 5 of the main paper. Section B1 gives more details of the aims and scope of the meta-analysis. Section B2 gives the details of the methods used. Section B3 presents the results, including analyses to check for the accuracy of the data coding (section B3.1), the main meta-analytic models (section B3.2), and the analysis of potential publication bias (section B3.3).

B1. Aims and scope

We sought to review the published empirical evidence relating food insecurity to overweight or obesity. The scope of our dataset was thus papers that reported quantitative data on an association between, as the predictor, a measure of food insecurity, and, as outcome, a variable relating to body weight, in a sample of adults or children. We did not seek to include studies where the predictor was participation in a food stamps programme, unless the study also included a direct measure of food insecurity. A number of studies have found positive associations between food-stamp programme participation and high body weight, and suggested that this might be because programmes where food stamps are issued periodically lead to cycles of food insecurity, but we left this question outside the scope of our review (see DeBono, Ross, & Berrang-Ford, 2012; Dinour, Bergen, & Yeh, 2007). We also did not seek to include studies where the outcome variable was not directly weight-based but indirectly related to weight (for example, diet quality or diabetes).

B2. Methods

B2.1 Search strategy

Our search strategy was as follows. We first searched the databases Scopus and PubMed (July 2015) for papers with either "obesity" or "overweight", and either "food security" or "food insecurity" in title, abstract, or keywords (Scopus) or title and abstract (Pubmed). We read the abstracts of all these papers to identify those likely to meet the inclusion criteria (see below). This produced 156 candidate articles (114 identified through both Scopus and Pubmed, 35 through Scopus alone, and 7 through Pubmed alone). We then enriched this sample by adding in any papers not already identified that had been discussed in key previous empirical reviews (Dinour et al., 2007; Franklin et al., 2012; Larson & Story, 2011; Morais, Dutra, Franceschini, & Priore, 2014), and also by reading abstracts of all the papers that had cited those reviews on Scopus. This enrichment produced 17 more candidates, giving a final candidate set of 173. We obtained and read the full text for all articles in the candidate set.

B2.2 Inclusion of papers from the candidate set

Our criteria for inclusion in the final sample from the candidate set were as follows. First, the study had to present empirical data not already published in an earlier paper in the candidate set. Second, food insecurity (FI) must have been measured. This was usually via one of the validated questionnaires (possibly abbreviated or translated), but on occasion a single question or interview theme was used instead. Third, data had to be presented on body weight or some categorisation derived from it. This was generally BMI-based (BMI itself, overweight = BMI≥25, obese = BMI≥30 for adults; weight percentile for age or some categorization derived from this for children). Fourth, data associating FI with body weight had to be presented in a format that could be made comparable to other studies (see next section). Given these criteria, we were able to include 125 of the 173 candidate papers in the final data set.

B2.3 Estimates of association strength

The analytic methods used in this literature are varied, and consequently representation of associations in a common format was not straightforward. The most frequently presented result is an odds ratio for a weight outcome (e.g. obese versus non-obese) by a FI comparison (e.g. insecure versus secure), accompanied by its standard error or 95% confidence interval. We converted these to log odds ratios (LORs, zero denotes no association). Where studies did not present odds ratios but did present: 1. numbers of participants falling into the different combinations of weight category and FI category; 2. means and standard deviations of body mass index by category of FI; or 3. correlations/standardized regression coefficients between a continuous FI measure and BMI, we were able to convert these to LORs using the methods described in Borenstein, Hedges, Higgins and Rothstein (2009) and Peterson and Brown (2005). These methods involve assumptions about how categorical distinctions map onto underlying continua, and moreover, in some cases, such as where sampling weights had been applied, conversion involved numerical computations and approximations on our part. We recorded whether the LORs were directly provided in the original paper, or had been converted by us.

Where studies reported prevalence ratios, we took these as if they were odds ratios. Close to the point of null association, prevalence ratios and odds ratios are identical (both equal to 1); as the association becomes stronger, the odds ratio exceeds the prevalence ratio by an amount determined by the prevalence of the predictor and outcome (Zocchetti, Consonni, & Bertazzi, 1997). Thus, taking prevalence ratios as odds ratios is conservative, leading to the underestimation of strong associations. Associations are in any case likely to be weak or moderate in this domain.

B2.4 Statistical adjustment

Studies varied in the degree of statistical adjustment made for covariates such as income, education and ethnicity. Where both adjusted and unadjusted estimates were available, we recorded both the unadjusted and (maximally) adjusted ones in the dataset. The main models reported in the paper use only the adjusted associations in cases where both were available. Where only unadjusted estimates were available, we included these but recorded their unadjusted status to include as a moderator in the meta-analysis.

B2.5 Multiple comparisons

The final data set included 301 associations from the 125 studies; hence, many studies provided multiple associations. In some cases this was because there were multiple subgroups, such as men and women, or different ethnic groups. Since there was prior evidence of sex differences in association, we preferred separate-by-sex associations to whole sample associations where both of these were available. Since we had no *a priori* interest in differences by ethnicity, we preferred whole sample associations to separate-by-ethnicity associations where both of these were available. However, where only separate-by-ethnicity associations were reported in the original papers, these were all used. Multiple associations from the same study also arose because of multiple categories of predictor (e.g. three levels of FI, with the most secure taken as the reference category) or of outcome (e.g. three levels of body weight, with the lightest taken as the reference category). In general, *j* levels of predictor and *k* levels of outcome produces *j-1 x k-1* associations to report. To account for the non-independence arising from the inclusion of multiple comparisons from the same study in the dataset, we analysed the data using multilevel meta-analytic models (Van den Noortgate, López-López, Marín-Martínez, & Sánchez-Meca, 2012), implemented in R (R Core Development Team, 2015) using the 'metafor' package (Viechtbauer, 2010). These models include two levels of random effect. First, there is a random effect at the level of the association, reflecting the fact that the associations measured within a study are heterogeneous and are a subset of all the possible associations that could have been measured. This is nested within a random effect at the level of the study, reflecting the fact that

associations from within each study are non-independent, and each study is drawn from a theoretical population of many other potential studies that could have been done, and about which we wish to generalise.

B2.6 Moderating variables

We also recorded, for each association in the dataset, a set of variables describing the methodology, sample and exact nature of the comparison made, to analyse as potential moderators of association strength. These are described below.

Design. This variable had two levels, cross-sectional and longitudinal. For associations to count as longitudinal, they not only had to have multiple time points, but had to report statistics in which the predictor was change in FI status and/or the outcome was change in body weight. Note that several longitudinal studies provided cross-sectional associations at baseline as well as any longitudinal associations.

Association type. We recorded whether the association was originally given as a (log) odds ratio (or prevalence ratio, see above), or whether the odds ratio had been converted from other kinds of association or descriptive statistics by us. The 'narrow subset' of studies mentioned in the main paper is the set restricted to those where the odds ratio was provided in the original paper.

Adjustment. Associations were recorded as adjusted or unadjusted, although the exact set of covariates adjusted for varied from study to study.

Classification of predictor variable. Most studies used a dichotomy of food insecure versus food secure. We assigned these predictor type 'All FI versus FS'. Some studies used a three-way classification of FI. We classified the associations from these studies as either 'Moderate FI versus FS' or 'Severe FI versus FS'. (Individual studies differed in their terminology for the more and less severe type of insecurity, but we mapped all of these onto 'moderate' and 'severe' respectively). For four-way classifications where there was a clear hierarchy of severity, we equated the most severe category to the severe FI category from papers using a three-way classification; the next most severe category to the moderate FI category; and assigned the remaining category a new status of 'Marginal FI'. This reflected the papers' own terminology in most cases. For some cases of four-way classification, it was not clear that the three insecure classes formed a hierarchy of severity (specifically, the four-way classification of food security/household insecurity/individual insecurity/child hunger); for these cases, all three insecure categories were combined to make a single dichotomous comparison of 'All FI versus FS'. For studies that used food insecurity score as a continuous quantitative variable, we classified the predictor as continuous.

Classification of outcome variable. We recorded whether the outcome in the association was obesity, overweight, or a continuous measure of body mass. Almost all papers in this literature use the same standard definitions of overweight (BMI ≥ 25) and obesity (BMI ≥ 30) for adults. Definitions for children are somewhat more variable but usually involve the 85th and 95th percentiles of weight for age, or weight for height). We did not distinguish associations where the outcome was obesity versus normal weight from those where it was obesity versus non-obesity, nor those where the outcome was 25 ≤ BMI ≤ 30 versus BMI < 25 from those where it was BMI ≥ 25 versus BMI < 25. Any differences between these slightly different classifications are likely to be slight.

Sex and age group. We recorded sex of sample (male, female, or mixed), and whether the sample were adults or children. A child sample was defined for these purposes as one in which most participants were under 16 years old. For child samples, we also recorded the mean age or, where this was not given, the central point of the age range.

Country. To establish whether there were differences in association between samples from developed and developing countries, we recorded whether the country that was the source of the sample was classified by the World Bank as high-income or middle/low-income (World Bank, 2015).

B2.7 Analysis strategy

We fitted a first meta-analytic statistical model with no moderators to examine whether there was an association between food insecurity and high body weight overall (statistical model 1). We subsequently explored moderator variables to attempt to explain the variation in association strength. As the number of moderators was relatively large, we were not able to enter them all simultaneously in one model. We hence fitted a series of statistical models, including non-overlapping sets of moderators respectively to do with the design and statistics (statistical model 2), the outcome and predictor variables (statistical model 3), and the participants included in the study (statistical model 4).

B3. Results

B3.1 Dataset and coding checking

The final set of papers included is listed in Online Appendix C, whilst the raw data are provided as Online Appendix D. The R script for the analysis is also available via the Open Science Framework at https://osf.io/zarn6/. Table B1 gives descriptive statistics for the associations included in the dataset.

To verify that our coding of the data was accurate and that our conversion of different types of associations to the common format of LORs had not led to any anomalies, we classified associations according to whether the authors of the original paper had stated the association to be positive and statistically significant, or not. For 58 associations, no explicit statement was made either way in the original papers. For the remainder, we calculated Cohen's Kappa measure of inter-rater agreement between statistical significance as stated by the original authors, and the lower 95% confidence limit for the LOR being greater than 0 in our dataset. The Kappa was 0.96, indicating a very high level of agreement. Figure B1 plots the lower 95% confidence limit in our dataset by the authors' original claim of significance, separated our by each of the major types of pre-conversion association measure: odds ratios, prevalence ratios, correlations or standardized beta coefficients, frequencies of individuals in each combination of FI and obesity, or means and standard deviations of BMI for different FI groups. As the figure shows, for every association type, the significant associations as claimed by the original authors have lower 95% confidence limits greater than 0, whilst the non-significant associations have 95% confidence limits less than 0. We thus infer that our encoding and conversion of the literature has faithfully preserved the key features of the original analyses.

Table B1. Descriptive statistics for the final meta-analysis data set.

| Variable	Level	Number of associations	Number of distinct studies
Design	Cross-sectional	287	123
	Longitudinal	14	7
Association type	Given as odds ratio	185	64
	Converted	116	62
Adjustment	Adjusted	172	65
	Unadjusted	129	64
Predictor variable	All FI versus FS	137	83
	Marginal FI versus FS	23	13
	Moderate FI versus FS	66	36
	Severe FI versus FS	67	37
	Continuous	8	7
Outcome variable	Obesity	121	60
	Overweight	129	64
	Continuous BMI	42	25
	Other for longitudinal designs	9	3
Sex	Female	117	53
	Male	41	20
	Mixed	143	74
Age group	Adults	189	79
	Children	112	52
Country	High income	209 [178 from USA]	91 [77 from USA]
	Low or middle income	92	36
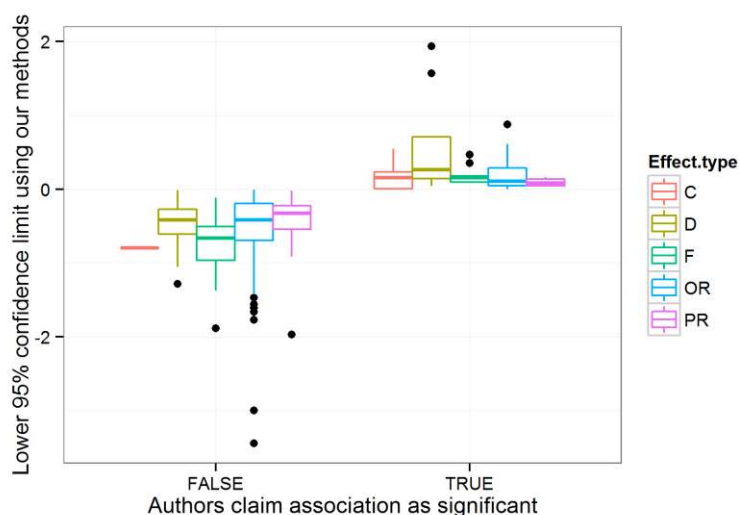
Figure B1. Lower 95% confidence limit of the log odds ratio for each association in our data set, by whether the authors stated the association to be positive and statistically significant in the original paper, and by the original format of the association. C: Correlation coefficient or standardized beta; D: Group means and standard deviations; F: Frequencies of individuals in each FI/Obesity combination; OR: Odds ratio or log odds ratio; PR: Prevalence ratio.

B3.2 Main statistical analyses

Table B2 gives the full summaries of the statistical models. Statistical model 1 tested whether there was a significantly positive association between food insecurity and high body weight overall. There was (LOR = 0.19, 95% CI 0.13 – 0.25, z = 6.30, p < 0.01). There was however significant heterogeneity in the associations above and beyond that expected due to sampling variation (Q_{300} = 1261.63, p < 0.01). Of the observed variability, 70.39% was between studies, whilst the remaining 29.61% was among associations reported within the same study. To satisfy ourselves that the pattern was not an artefact of our conversion of non-odds ratio associations into odds ratios, we reran statistical model 1 using the narrower subset of 154 comparisons that were stated in the form of odds ratios in the original papers. The results were extremely similar to those from the full dataset (LOR = 0.20, 95% CI 0.14 – 0.27, z = 6.11, p < 0.01). In light of this, all subsequent results use the full dataset.

Statistical model 2 examined the effects of methodological and statistical factors, by including the design of the study (longitudinal versus cross-sectional), and whether or not the results were adjusted for possibly confounding factors like socioeconomic position and ethnicity. The moderators did not explain a significant portion of the heterogeneity in association (Q_2 = 0.86, p = 0.65). Although unadjusted estimates tended to be slightly higher than adjusted ones, this difference was not significant (B = 0.05, 95% CI -0.07 – 0.16, z = 0.81, p = 0.42). This conclusion was supported by a paired t-test directly comparing the adjusted and unadjusted LORs for the 49 cases where the original authors provided both for exactly the same comparison in their data (mean unadjusted 0.28; mean adjusted 0.23; t_{48} = -0.82, p = 0.42). There was no significant difference in association strength between longitudinal and cross-sectional study designs (B = 0.04, 95% CI -0.15 – 0.23, z = 0.41, p = 0.68).

Table B2. Full model output for the four main meta-analysis statistical models.

Model	Fixed effects	Parameter (95% CI)	Residual variances		Q-statistics	
Model 1	Intercept	0.19 (0.13 – 0.25)*	Study	0.067	Residual	$Q_{300} = 1261.63$, p < 0.01
			Association	0.028		
Model 2	Intercept	0.17 (0.09 – 0.25)*	Study	0.067	Moderators	$Q_2 = 0.86$, p = 0.65
	Design cross-sectional	Reference category	Association	0.029	Residual	$Q_{298} = 1246.66$, p < 0.01
	Design longitudinal	0.04 (-0.15 – 0.23)				
	Adjusted	Reference category				
	Unadjusted	0.05 (-0.07 – 0.16)				
Model 3	Intercept	0.27 (0.17 – 0.36)*	Study	0.059	Moderators	$Q_6 = 19.41$, p < 0.01
	Outcome obesity	Reference category	Association	0.027	Residual	$Q_{280} = 1087.25$, p < 0.01
	Outcome overweight	-0.16 (-0.25 – - 0.07)*				
	Outcome continuous	0.02 (-0.17 – 0.20)				
	Predictor all FI versus FS	Reference category				
	Predictor marginal FI versus FS	0.01 (-0.14 – 0.16)				
	Predictor moderate FI versus FS	-0.07 (-0.20 – 0.05)				
	Predictor severe FI versus FS	-0.01 (-0.14 – 0.12)				
	Predictor continuous	0.13 (-0.19 – 0.46)				
Model 4	Intercept	0.37 (0.29 – 0.46)*	Study	0.048	Moderators	$Q_4 = 57.60$, p < 0.01
	Sex female	Reference category	Association	0.019	Residual	$Q_{296} = 965.48$, p < 0.01
	Sex male	-0.26 (-0.35 – 0.16)*				
	Sex mixed	-0.06 (-0.18 – 0.05)				
	Adults	Reference category				
	Children	-0.14 (-0.25 –0.03)*				
	High income country	Reference category				
	Low or middle income country	-0.27 (-0.39 – -0.15)*				

* 95% confidence interval does not include zero.

Statistical model 3 included moderators to do with the predictor and outcome variables (this model used the data from just the cross-sectional studies, since outcome variables are different in longitudinal designs). The possible outcomes were obesity (usually defined as BMI ≥ 30 or weight for age above 95th percentile, versus its absence), overweight (BMI ≥ 25 or weight for age above 85th percentile, again versus its absence), or a continuous BMI variable. The possible predictors were: dichotomous comparison of food insecurity versus food security; marginal food insecurity versus food security; moderate food insecurity versus food security; severe food insecurity versus food security; and a continuous food security variable. In model 3, the moderators did explain a significant proportion of the heterogeneity in association (Q_6 = 19.41, p < 0.01), though substantial unexplained heterogeneity remained (Q_{280} = 1087.25, p < 0.01). Comparisons using overweight as the outcome found weaker associations than those using obesity (B = -0.16, 95% CI -0.25 – 0.07, z = -3.58, p < 0.01). There was no significant difference between associations using obesity and those using continuous BMI as the outcome. Thus it appears that stronger associations may be detected by using the more extreme category of high body weight, or else BMI as a continuous variable, than by using the less extreme overweight category. There were no significant differences between the different types of predictor.

Statistical model 4 examined the types of participants of the study as a source of heterogeneity in association. The participants variables were: composition of the sample by sex (male, female, mixed); by age group (adults or children); and whether the study was performed in a World Bank-defined high-income country or not. These moderators accounted for significant heterogeneity (Q_4 = 57.60, p < 0.01), although again substantial unexplained heterogeneity remained (Q_{296} = 965.48, p < 0.01). There was a significant effect of participant sex, with male samples showing significantly weaker associations than female ones (B = -0.26, 95% CI -0.35 – -0.16, z = -5.25, p < 0.01). Indeed, there was no evidence of a non-zero association in the male samples considered as a subset. Mixed-sex samples did not differ significantly from female ones (B = -0.06, 95% CI -0.18 – 0.05, z = -1.07, p = 0.26). There was also an effect of age group, with children showing significantly weaker associations than adults (B = -0.14, 95% CI -0.25 – -0.03, z = -2.54, p < 0.01). We also found an effect of being in a high income country, with studies from low- and middle-income countries finding weaker associations than those from high income countries (B = -0.27, 95% CI -0.39 – -0.15, z = -4.51, p < 0.01). Associations from low- and middle-income countries considered separately did not differ significantly from zero, whereas those from high-income countries had a central LOR estimate of 0.26, equivalent to an odds ratio of 1.29.

To investigate the pattern within the child samples, we ran additional statistical models using just the child samples, both in an intercept-only model and including age and sex of the children as moderators (table B3). The association in the intercept-only model did not differ from zero (LOR = 0.06, 95% CI -0.03 – 0.14, z = 1.29, p = 0.20). The only significant moderator in the model with moderators was an interaction between age and sex. Figure B2 illustrates this interaction by dividing the child samples up into those where the modal age was ten years or older, and those where it was younger, and according to whether the sample was male, female, or mixed. In the younger age group, all types of samples have similar estimates of association. In the older age group, the female samples show a significantly stronger association than the male ones, albeit not significantly different from zero. Thus, the sex difference that is so clear in adults seems to be beginning to emerge in adolescents, whereas there is no evidence of it in younger children.

Table B3. Full model output for the two meta-analysis models of the child samples alone.

Model	Fixed effects	Parameter (95% CI)	Residual variances		Q-statistics	
Model C1	Intercept	0.05 (-0.03 – 0.14)*	Study	0.048	Residual	Q_{111} = 487.61, p < 0.01
			Association	0.033		
Model C2	Intercept	-0.16 (-0.61 – 0.29)*	Study	0.050	Moderators	Q_5 = 10.71, p = 0.06
	Sex female	Reference category	Association	0.024	Residual	Q_{106} = 347.51, p < 0.01
	Sex male	0.29 (-0.13 – 0.70)				
	Sex mixed	0.10 (-0.40 – 0.59)				
	Age	0.03 (-0.01 – 0.08)				
	Sex male * Age	-0.06 (-0.10 – 0.01)*				
	Sex mixed * Age	-0.0 (-0.06 – 0.03)				

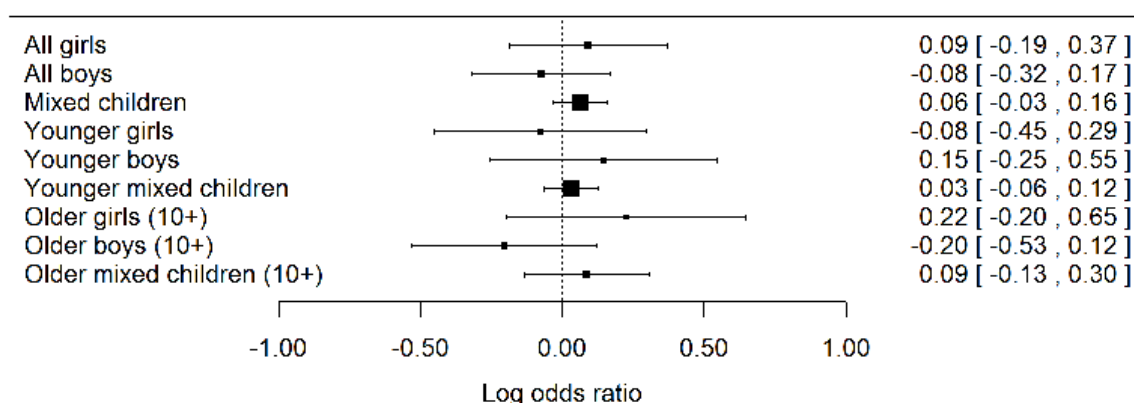* 95% confidence interval does not include zero.

Figure B2. Central estimates (log odds ratios and their 95% confidence intervals) for associations between food insecurity and high body weight in subgroups of the child samples.

B3.3 Publication bias

Methods for testing publication bias are not implemented in the 'metafor' package for multilevel models. We thus repeated the main model 1 using a simple random-effects model. This yielded very similar results to the multilevel version (central estimate: LOR = 0.18, 95% CI 0.14 – 0.22, z = 8.17, p < 0.01). The funnel plot for this model (figure B3, left panel) fails to show clearly the predicted greater variability of association estimates where the precision is low (i.e. the funnel shape). This may reflect the fact that the associations come from studies that in most instances have high precision, but are heterogeneous in a number of substantive ways.

9

We performed the random-effects version of the Egger test (Egger, Davey Smith, Schnieder, & Minder, 1997) on this model: the test was significant, suggesting that publication bias might be operative ($z = 2.60$, $p < 0.01$). We then used the trim and fill method (Duval & Tweedie, 2000) to estimate how many unreported null associations might be missing (figure B3, right panel). The estimated number was 26 (standard error 10.98), or just under 10% of the total number of associations. Repeating the equivalent of model 1 with the missing associations imputed via the trim and fill method produced a central LOR estimate of 0.12 (95% CI $0.07 - 0.17$, $z = 4.89$, $p < 0.01$). Thus, this analysis suggests that there may be publication bias in operation, but that even imputing 26 null associations to make the funnel plot symmetrical does not abolish the significant association overall between FI and high body weight; the association is attenuated by around one third, but remains significant.
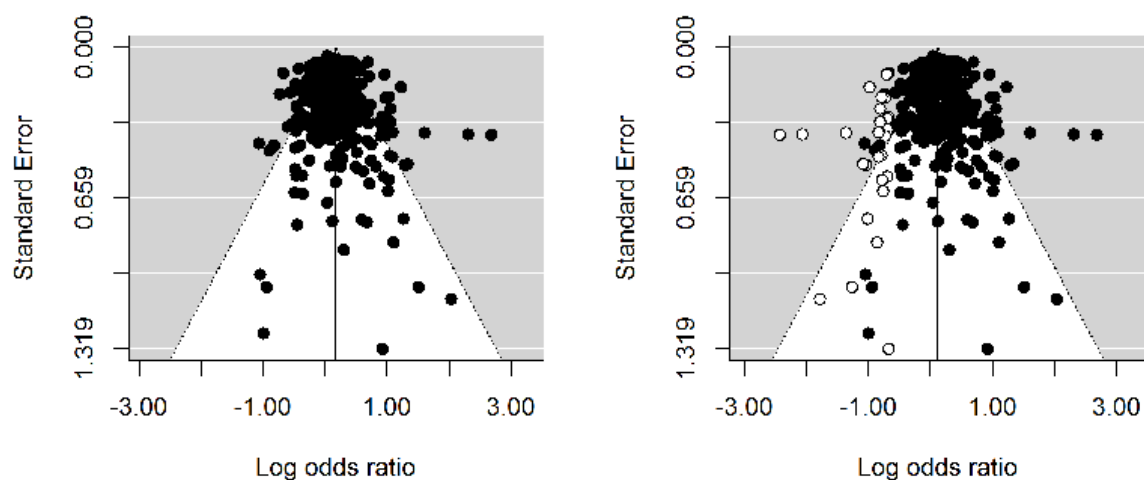

Figure B3. Funnel plots for the data (left panel); and the data (filled points) with 26 associations imputed by the trim and fill method (open points) to make the funnel plot symmetrical (right panel).

It is not possible to retest models 1-4 from table B2 using the dataset with the extra associations imputed via the trim and fill method, since there is no method for imputing the moderator variables for the missing associations. Instead, to examine whether the effects of age, sex and high-income country survived adjustment for publication bias, we divided the data up into a series of relevant subsets, and performed a trim and fill analysis on each subset in turn. We then extracted the central estimate of association strength, and its 95% confidence interval, for each of these trimmed and filled subsets. The results are shown in figure B4. As for the analysis in the main paper, the association is clearly present in high-income countries, but not in low and middle income countries; is present in adult women but not adult men; and is weak (though in this specification, just significantly different from 0) for children. Thus, it seems unlikely that any of the main conclusions of our meta-analysis is a consequence of publication bias.
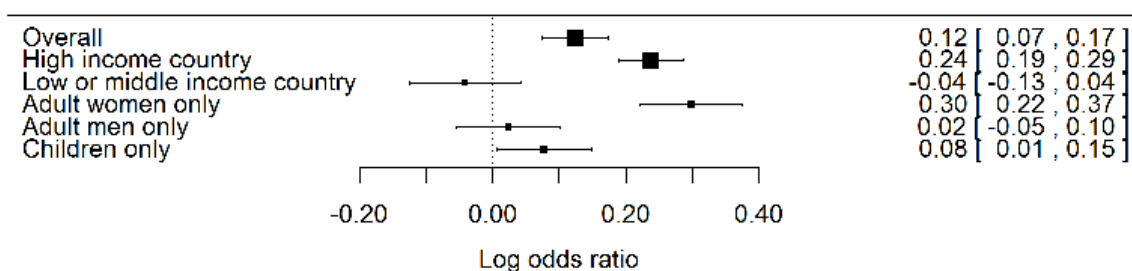
Figure B4. Central estimates of association (log odds ratios and their 95% confidence intervals) overall, and various subsets of the data, with missing associations imputed via the trim and fill method. Note that these estimates employ a standard random-effects model, not a multilevel model.

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Online Appendix C
List of references used or considered for the meta-analysis

Entries marked * were in the initial candidate set but not included in the final dataset

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