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Review

The food-insecurity obesity paradox: A resource scarcity hypothesis

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HIGHLIGHTS

- Mechanistic hypotheses explaining the food insecurity-obesity paradox do not exist.
- A resource scarcity hypothesis is proposed to explain this paradox.
- Implications of this theory for research and intervention are discussed.

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ABSTRACT

Food insecurity is paradoxically associated with obesity in the United States. Current hypotheses to explain this phenomenon are descriptive regarding the low food security population's dietary and physical activity habits, but are not mechanistic. Herein it is proposed that a resource scarcity hypothesis may explain this paradox, such that fattening is a physiologically regulated response to threatened food supply that occurs specifically in low social status individuals. Evidence that this may be occurring, the implications for addressing the food insecurity-obesity paradox, and future areas of research, are reviewed and discussed.

Contents

1. Introduction

Low food security is associated with obesity in some circumstances (reviewed in [1,2]). Low food security, defined as, "reports of reduced quality, variety, or desirability of diet. Little or no indication of reduced food intake," by the USDA, does not involve hunger, whereas very low food security, defined as, "Reports of multiple indications of disrupted eating patterns and reduced food intake," is accompanied by hunger [3]. The prevalence of low food security has risen in the United States in the last 15 years, and was 10.7% of households in 2001, and peaked at 14.9% in 2011 following a spike during the Great Recession [4]. It is not well understood if low food security plays a causative role in the development of obesity, and if it does, what the mechanisms may be.

There are two predominant, related hypotheses that have been proposed to explain this link in the literature:

1. Low food security is associated with obesity because of the high calorie, palatable food consumed by low food secure populations [5,6].

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2. Low food security is associated with obesity because of the limited knowledge, time, and resources that low food-secure populations experience to engage in healthful eating and exercise.

Both hypotheses are descriptive, but not probative or mechanistic in nature, as an explanation for the relationship between low food security and obesity. For substantial weight gain to occur, energy intake must be greater than energy expenditure on a long-term, chronic basis. There is a physiologically regulated, adaptable system that is designed to resist weight change.

Therefore, although documenting increased intake of high-energy, palatable foods or reduced physical activity in low food secure populations may document crucial *parts* of a mechanism, neither is a probative mechanistic explanation. Concluding that the intake of high calorie foods is sufficient to explain weight gain in low food secure populations is similar to concluding that individuals with Prader-Willi Syndrome gain weight because of the food they eat.

Though it is true that Prader-Willi Syndrome patients do consume more food than they require, this does not explain *how* this occurs on a chronic basis. The presence of food and the intake of food is a permissive, but not causative factor in their weight gain. The neurobiological mechanisms that *cause* increased food intake and weight gain are becoming well understood [7] so that therapeutics can be developed. Similarly, although an abundance of high calorie, palatable food may be a crucial permissive factor in the development of obesity in low food secure populations, its presence alone does not explain *how* low food security may drive the development of chronic positive energy balance. It is crucial to understand *why* and *how* low food secure populations gain weight, and what about low food security may be a fundamental driver of a net, chronic shift in the homeostatic regulation of energy balance. Such a mechanistic explanation may lead to more effective, cause-specific interventions.

The need for a more probative mechanism to explain the link between low food secure populations and obesity is clear from the lack of results from interventions that focus on food, resources, and knowledge to reduce weight gain. For example, when exercise facilities are made available to low SES populations, they are often not utilized [8]. Similarly, providing monetary resources or food caused weight *gain* in a low SES population in rural Mexico [9]. In another study, increasing food stamp funds to $2000/year had no effect on social BMI disparities [10]. It is plausible that such interventions are ineffective because they are not addressing the root mechanisms behind low food security's association with weight gain and obesity.

A probative, mechanistic explanation for the relationship between low food security and obesity can be proposed from intersections in findings from the fields of evolutionary biology, ecology, and obesity (Fig. 1). This "Resource Scarcity Hypothesis" suggests that perceived food insecurity, in a permissive environment where there is access to high calorie foods, may cause positive energy balance specifically in low social status individuals, but not in high social status individuals. Evidence suggesting this may be the case is reviewed in the following sections.

2. Social status and metabolic efficiency

Social status may be associated with low energy expenditure and metabolic efficiency. Since low food security tends to be associated with low social status, the role of social status in determining metabolic efficiency may contribute to the development of obesity in this population. Both animal and human studies suggest that low social status organisms may be more metabolically efficient.

For example, dominant mice have higher energy expenditure compared to subordinate mice, and are more obesity resistant on a high fat diet as a result [11]. Therefore, even when all social ranks are exposed to the same palatable, high energy diet and consume the same amount of it, only the subordinate animals gain fat stores due to their higher metabolic efficiency.

Evidence suggests that human minority populations may also be more metabolically efficient. Resting metabolic rate (RMR) is an established marker of high metabolic efficiency, and is a risk factor for weight gain. RMR is 5% higher in white young adults compared to black young adults. In addition, fat oxidation, as measured by 24 h RQ, is also higher in whites compared to blacks [12]. There may be mitochondrial genetic differences that make blacks more metabolically efficient compared to whites [13], therefore, future research is warranted to determine if any of these effects are explained by social status, rather than genetic differences, or possibly because of transgenerational interactions between both. However, since low food security is more

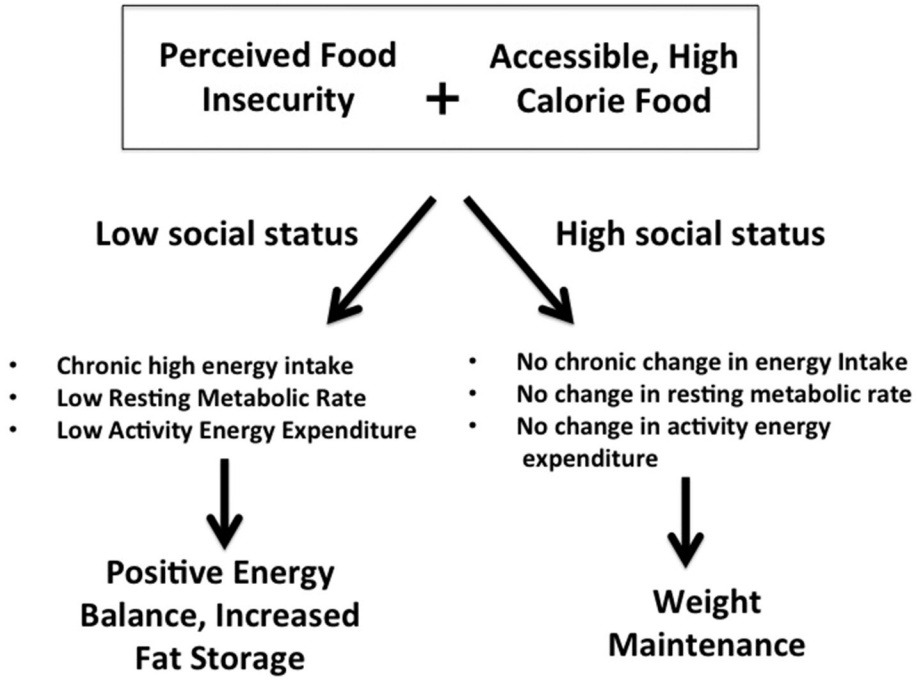

Fig. 1. Resource scarcity hypothesis. An overview of the proposed mechanisms by which low food security may lead to weight gain and obesity.

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prevalent in low social status individuals and minorities, it is plausible that these populations are also metabolically efficient, predisposing to weight gain.

3. Social status and excess food intake

Some evidence suggests that social rank may not only influence energy expenditure and metabolic efficiency, but energy intake as well. In animals, several experiments suggest social hierarchy has a causative influence on energy intake. Subordinate primates consistently consume more energy than dominant primates, regardless of diet type available (high or low-fat) [14]. This effect seems to be enhanced in the presence of a palatable, high fat diet. While both dominant and subordinate primates prefer a high fat diet when given a choice, subordinate animals consume more of a high fat diet, whereas dominant animals do not consume it in excess of their energy requirements [15]. Finally, subordinate rats display rapid fat gain and consume more food per gram of body weight compared to dominant animals, when allowed to recover in the presence of food from social housing stress [16].

Social status may also influence diet preference and dietary intake in humans. Education, employment grade, and income are associated with poor diet quality that is high in energy density [17–19]. Various kinds of stress, including social stress, have been associated with increased food intake, particularly in women [20–22]. Defeat, reflecting a change in social status, may influence energy intake in humans. Increased calorie consumption is observed in the cities of losing football teams the day following the game, compared to cities of winning teams or cities that did not play a game. Cities of losing teams eat more fatty, unhealthful foods than cities of winning teams [23]. These findings are particularly true for cities most devoted to their teams [23]. Similarly, randomly priming individuals to cues that imply resource scarcity and harshness causes them to be more likely to choose and consume high-calorie food items [24]. Together, these findings suggest that adversity due to low social status may be a key determinant of food choice and energy intake.

4. Evidence that low food security influences body fat stores selectively in low social status individuals

In animals, an unpredictable or threatened food supply, in comparison to a predictable and secure food supply, has an influence on body weight and body fat stores, and the effect may be pronounced in low social status organisms in particular. For example, when female rats undergo just 5% energy restriction, they respond paradoxically by increasing fat stores [25]. In animals, experimental manipulation or natural circumstances that threaten the perception of food security increase body fat in subordinate, but not dominant animals [26–28], suggesting that the response of an animal to a threatened food supply may depend on their social status. This is logical since if there is a shortage of food, the subordinate animals are least likely receive adequate food, and most likely to need excess adipose tissue stores to survive and reproduce.

In humans, there is evidence that low social status individuals may be more susceptible to the perception of low food security. The desire for money and caloric resources are highly intertwined, such that the desire for money increases the desire for caloric resources, and vice-versa [29]. Therefore, low social status, and poverty in particular, may lead to an increased desire for food, and an increased *perception* that the food supply is inadequate or may be in the near future, even when high calorie, energy dense foods are available. In contrast, higher social status individuals may be resistant to the *perception* of reduced food availability, if monetary resources are not a limiting factor on food purchasing power. However, there are no studies that clearly demonstrate the effect of low food security on food intake or energy expenditure is specific to only low social status individuals, and this would be an important area of future research.

There is also evidence that low food security is associated with a weight gain response in the presence of high calorie, energy dense foods in humans. Longitudinally, being marginally food insecure or food insecure without hunger is associated with greater weight gain compared to being fully food secure [30]. Low food security is particularly associated with obesity when Food Assistance programs are in place, and the relationship between Food Assistance programs and obesity is greatest in those who are most food insecure [31]. Thus, it appears that improved access to healthful food through assistance programs is not sufficient to reduce energy intake and obesity, and in fact, greater access to food may make the issue worse. Together, these findings suggest that increased energy intake may be a fundamental response to threats to food security, that is persistent independent of the actual food supply, in low social status humans.

In addition, two inconsistencies in the relationship between socioeconomic status and obesity suggest that weight gain may be a strategic response to perceived low food security to ensure survival. First, there is a negative association between obesity and SES in developed countries, but a positive association in developing countries, such that higher class individuals are more obese in developing countries [32]. It is possible that higher social classes in developing countries, because of their education and resources, are aware that although their social class is high relative to their countrymen, it is not high on a global scale. The stressful environment in developing countries is hard to escape, even for the wealthy. The underlying stress of living in developing countries may place higher class individuals of those countries in a similar place as lower social status individuals in developed countries, on a global scale. The "desire for money = desire for calories" effect may create an underlying perception of low food security, even in the presence of a plentiful food supply. Because higher classes in developing countries would plausibly have access to an abundance of high calorie foods, this combination would make them very similar to lower class individuals in developed countries, and may drive weight gain.

Another inconsistency about the relationship between socioeconomic status and obesity is that it is more consistent in women than it is in men [32]. Adequate levels of body fatness play a crucial role in successful reproduction and offspring survival for women [33], but may not be as important in men. Therefore, it is plausible that any physiologically regulated fattening response to low food security would be specific to women to ensure successful survival and reproduction. Future studies on the role of sex hormones in the mechanistic link between low food security and weight gain may be warranted.

5. Potential physiological mechanisms linking low food security and social status with weight gain

Cortisol metabolism may mediate weight gain in response to low food security in low social status individuals. Low social status is associated higher basal cortisol levels, lower cortisol reactivity to acute stress, and a lack of cortisol habituation [34–36]. Impaired cortisol habituation is hypothesized to be a robust indicator of cumulative exposure to elevated cortisol that may accompany repeated exposure to stress [37]. Thus, higher basal cortisol, and lower cortisol habituation levels in low social status individuals may be indicative of higher allostatic load in these individuals and the resulting dysregulation of the stress response [38]. Chronically elevated cortisol may influence both food intake and fat metabolism.

Cortisol metabolism is involved in regulating food choice and food intake. In humans, food intake is increased in response to stress in high cortisol reactors [22], and glucocorticoid administration causes higher food intake in men [39]. The regulation of food intake behavior by chronically high cortisol levels may occur through a hypothalamic-pituitary-adrenal axis mechanism, whereby inhibition of a corticotrophin-releasing hormone by cortisol results in subsequent increases in orexigenic neuropeptide Y (NPY) [40].

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Interestingly, NPY is not only known to increase food intake, but it is also a crucial negative feedback mechanism that reduces the physiological effects of certain stressors, and it has a reported soothing effect that negates the physiological impact of stressors [41]. And finally, chronically high cortisol is associated with leptin resistance [42,43], which is well known to suppress energy expenditure, increase appetite, and contribute to weight gain.

In addition to hypothalamic effects, cortisol acts specifically on fat deposition in the visceral region. Its receptors are higher in density in this region, and high cortisol reactivity is associated with abdominal obesity [44,45]. Chronically high cortisol levels can cause insulin resistance [46], and high levels of insulin interact with cortisol to promote fat deposition and reduce lipolysis [43,47]. However, it is unclear if this cortisol-induced response is involved in the effects of social hierarchy on food intake and body fatness in humans.

6. Implications of the resource scarcity hypothesis in addressing the food insecurity-obesity paradox

If fat gain is a physiologically regulated, strategic response to low food security to ensure survival and reproduction, this would have several implications. First, interventions that seek to educate low food security populations about reducing energy intake and increasing energy expenditure may be insufficient to address the underlying problem, and therefore be unproductive. A lack of effect of such interventions is reflected in the literature [8–10].

On the other hand, the "Moving to Opportunity Study" suggests that interventions focused on improving perception of social hierarchy, with no focus on nutrition or physical activity, are sufficient to improve rates of obesity and diabetes. This study demonstrated that randomizing families to move to a higher-class neighborhood, without changing income, education, or occupation, was sufficient to reduce average body mass index [48]. Thus, social position, independent of access to material resources, influences susceptibility of low SES individuals to weight gain. This is also reflected in comparisons across 35 countries where income inequality better explains inequalities in overweight than absolute wealth in adolescents [49]. These data underscore the potential of social interventions in reducing obesity.

In addition, a future area of research that may be productive in addressing the food insecurity-obesity paradox, if the resource scarcity hypothesis is playing a role, would be interventions that focus on enabling individuals to gain control over access to their own food supply (for example: gardening, financial planning, dietary interventions focused on strategic buying). Reducing perceived threat of low food security may be crucial in changing energy intake behaviors. Another area of research that would be crucial is determining the physiological mechanisms for both sensing a threat to low food security, and responding to it by up regulating appetite and reducing energy expenditure. It would be important to understand the environmental and social factors that trigger this perception, as well, as potential points of intervention to address the food insecurity-obesity paradox.

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