

Committed to the insurance hypothesis of obesity

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

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

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

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

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

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

In section 6, Nettle et al. indicate that the IH proposes that humans evolved mass regulation mechanisms that might have no relevance in today’s society, and that the hypothesis says nothing about obesity being currently adaptive. Hence, the

hypothesis is non-adaptive, or even maladaptive, and despite what the authors suggest, not an alternative but, rather, another variant of the EMH. One difference is that the other hypotheses, even if based on ecology, have physiological and genetic aspects to them, whereas the IH is strictly an ecological hypothesis. Also, the other hypotheses are based on historical changes in food abundance and quality, whereas the IH is based on differences in the predictability of food supplies.

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

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

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

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

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

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

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[The letters “a” and “r” before author’s initials stand for target article and response references, respectively]

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