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Obesity

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Synonyms

Adiposity; Body mass index; Diet; Nutrition; Overweight

Definition

Clinicians define the state of being overweight or obese by body mass index (BMI), which is calculated by body mass in kilograms divided by the square of the person's height in meters. BMI from 18.5 to 25 is considered within a healthy range, or less likely to be associated with increased risk from certain weight-related diseases (González-Muniesa et al. 2017). The categories of underweight (BMI <18.5), overweight (BMI 25–30) to obese (BMI >30) are associated with increased risks of certain diseases, costs to reproductive performance, and life expectancy (GBD 2015 Obesity Collaborators et al. 2017; Global BMI Mortality Collaboration et al. 2016; Hammiche et al. 2012; van der Steeg et al. 2008; Zain and Norman 2008).

Introduction

Conservative estimates suggest that over one billion people worldwide are either overweight or obese (Wang and Speakman 2016). Obesity levels have more than doubled since the 1980s, with the estimate of overweight and obese adults climbing to ~38% of the global population (NCD-RisC 2016; Ng et al. 2014). Obesity is responsible for approximately three to four million deaths per year worldwide, predisposing suffers to a plethora of related diseases such as type 2 diabetes, cardiovascular diseases, metabolic syndrome, chronic inflammatory diseases, and 13 types of cancer (Buescher et al. 2013; González-Muniesa et al. 2017; Ng et al. 2014; Pearson-Stuttard et al. 2018; Whiteman et al. 2015; Wilson et al. 2018). Despite the costly implementation of public health policies, obesity levels continue to rise around the globe due to the complicated epidemiology and etiology of obesity (González-Muniesa et al. 2017). This entry concisely considers some of the possible evolutionary reasons why obesity has arisen, beginning with the evolution of our diet. Next, it will cover the evidence for the longstanding thrifty genotype hypothesis as a possible origin of obesity, and finally, consideration will be given to the role protein targets may play in the rise of obesity in humans.

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The Evolution of Our Diet

The diet of an individual consists of many nutrients that are required in various proportions relative to other nutrients. These nutrients interact with each other and with the individual's life stage, genome, microbiota and condition. It is generally true that a single nutrient needs to be consumed at balance, and that over- or underconsumption is harmful to an individual's health (Raubenheimer et al. 2005). Therefore, in order to maximize fitness, an individual must balance ingestion and absorption of nutrients in appropriate proportions. Precisely which foods an organism ingests to achieve this balance is determined by how each nutrient effects appetite and satiety – which is specific to the evolutionary history of each organism (Simpson et al. 2004). The three major energy-contributing nutrients in any diet are carbohydrate, fat, and protein, also termed macronutrients. These macronutrients can explain a great deal of the physiological and behavioral variation between organisms and thus play an important role in determining evolutionary fitness (Raubenheimer and Simpson 2016).

Over the past ~40,000 years since the Paleolithic period, human diet has changed drastically, this change in diet has accelerated even further over the past ~50 years. Fossil studies of past humans show us they were limited for energy because sources of simple fats, starches, and sugars were much less abundant than they are today (Eaton et al. 1996), whereas complex sources of carbohydrate such as roots or tubers and sources of protein from fish or lean game meat were the main staples of a hunter-gatherer diet. Hunter-gatherer populations today, such as the Hadza, have very low levels of obesity (<5%) and rarely experience metabolic or cardiovascular diseases (Pontzer et al. 2018). Importantly, the Hadza people spend a lot longer engaging in physical activity than most of their Western counterparts, accumulating over 120 min each day (Pontzer et al. 2018). When many societies transitioned from a hunter-gather to agricultural lifestyle, diets increased in carbohydrate – mainly starchy grains. Although the shift to agriculture occurred at differing times across societies, evidence from multiple regions indicate this rise in starch coincided with a reduction in protein. The agricultural revolution also produced an increased disease burden, due to closer living quarters, and increased the risks of famine due to reliance on crops (Prentice 2005; Wells 2012). Due to the availability of mass refining and improved transport of sugar and grains, the carbohydrate content in human diet increased further during the industrial revolution, although still at this time, obesity prevalence was low and considered a luxury of the wealthy (Simpson and Raubenheimer 2012).

In approximately the last 50 years, the macronutrient composition of our diet has changed even more, with most in developed nations having access to an abundance of food. We now have access to an unprecedented amount of simple sugars and fats, and highly processed foods are cheap and plentiful. In tandem with this nutritional shift, we are required to expend much less energy on subsistence activities than did our forebears (Simpson and Raubenheimer 2012). Despite this change in lifestyle, our physiology remains very similar to that of our ancestors. This led many researchers to hypothesize about evolutionary discordance, which suggests significant departures from our ancestral diet but not our ancestral physiology, have greatly contributed to noncommunicable diseases such as obesity (Konner and Eaton 2010). However, research investigating the interaction between our culture, genes, and environment (termed gene-culture coevolution) has shown recent positive selection for mutations such as lactose tolerance, that has arisen in societies that moved toward dairy farming for subsistence ~5000–10,000 years ago (Swallow 2003; Tishkoff et al. 2007). Moreover, the genes that confer some resistance to diabetes may well be under positive selection (Helgason et al. 2007) indicating that an adaptive lag between our genes and diet is not present for all nutritional changes in recent evolutionary history. Such evidence from gene-culture co-evolution studies may lend credence to another possible explanation for obesity: the thrifty gene hypothesis, because these selection signatures are what

one would expect to find under this hypothesis, and this is considered in the next section.

Thrifty Genes?

The thrifty gene hypothesis postulates that obesity may be the result of positive selection for genes that promote fat storage – these so-called thrifty genes may have come under selection during times of food scarcity in our evolutionary history (Gibson 2007; Wang and Speakman 2016). Now that most people in developed, and even many developing nations, have access to an abundance of food – these once thrifty genes – may now be maladaptive and thus lead to obesity. There are several reasons why this hypothesis is alluring to researchers. First, there is variation in obesity and related disease susceptibility in humans, and these traits are both genetically correlated and heritable (Gibson 2007). Second, when we look into our own evolutionary past, we can see shifts in diet, beginning with an energy limited environment changing to an energy abundant one (Simpson and Raubenheimer 2012). Third, when we put humans in context with other extant primates, we see many have an annual body mass cycle where they consume in caloric excess when fruit is abundant and experience the lean season when fruit is more scarce (Irwin et al. 2015). In fact, this type of endogenous cycle where animals eat to a positive energy balance in preparation for energy scarcity is found in other taxa such as barnacle geese, ground squirrels, alpine marmots, bears, and hairy-nosed wombats (Atkinson et al. 1996; Finlayson et al. 2010; John 2005; Körtner and Heldmaier 1995; Portugal et al. 2007). It may appear plausible then that humans are perhaps accumulating weight for a winter that will never come.

However, studies that have investigated positive selection in alleles that may confer a fitness advantage to efficient fat storage have failed to demonstrate any selection signature that would be congruent with the thrifty genotype hypothesis (Ayub et al. 2014; Wang and Speakman 2016). Additionally, there are several other limitations to this hypothesis; they all relate to famine as a selective pressure. It seems that while Paleolithic people were in an energy scarce environment (comparative to now), famines were more likely during the agricultural and industrial revolutions. To confer a great enough selective pressure, famines would likely need to be quite frequent and quite devastating – the mortality rate from the famine would need to be high enough to exert sufficient selective pressure (Gibson 2007; Speakman 2006). There is much debate in the literature about the frequency and severity of famines throughout the past ~50,000 years; some researchers define famines occurring as frequently as every decade, others as only once every century, all with differing mortality rates. This makes determining whether famine is a sufficiently strong and regular selective pressure difficult (Prentice 2005; Speakman 2006). It is also more likely during these times of scarcity that people died from infectious diseases, rather than starvation itself, although interestingly a BMI >30 is associated with an increased chance of survival from some infections such as community-acquired bacterial pneumonia (Corrales-Medina et al. 2011). It is possible therefore that the value of the thrifty gene confers different advantages to those previously investigated, although a BMI >30 is also associated with an increased risk of mortality from other acute infections (Dhurandhar et al. 2015). Nevertheless, little direct evidence exists for the thrifty genotype hypothesis.

Are We Eating to a Protein Target?

Rather than thrifty genotypes, perhaps the more recent changes in our diet have led to obesity, because many organisms, including humans, are eating to specific nutrient targets (Raubenheimer and Simpson 2016). Organisms possess nutrient-specific appetites that maintain homeostasis and alter the consumption of different nutrients (Corrales-Carvajal et al. 2016). If the correct proportion of nutrients required for maintaining health is not available however, organisms attempt to remedy this by ingesting a range of foods with differing but complementary nutrients

(Raubenheimer and Simpson 1997; Waldbauer and Friedman 1991).

Much data across taxa, (including primates, flies, and murids) indicates that protein is the primary determinant of appetite and satiety (Felton et al. 2009; Gosby et al. 2011, 2014; Lee et al. 2008; Simpson et al. 2004; Solon-Biet et al. 2014; Sørensen et al. 2008). Therefore, generally food is ingested in a manner that ensures protein consumption is within a strict range of values, but a wider range of carbohydrate and lipid intake can be tolerated. This may be because protein overconsumption can carry physiological and metabolic costs associated with excretion, whereas underconsumption leads to a decrease or cessation in reproductive output (Piper et al. 2005; Solon-Biet et al. 2015). If excess carbohydrate or fat is consumed however, energy stored as body fat can serve as an important buffer against seasonal variations in energy supply (Prentice 2005; Walker et al. 2017).

Because protein consumption is so closely linked to satiety, it has the capacity to shape feeding behavior, and can be exploited to facilitate the loss of body fat by curtailing appetite through consumption of high protein diets. By contrast, consuming low protein foods to meet a protein intake target can result in overconsumption of sugar and fat, which in the long term can lead to obesity (Gosby et al. 2014; Raubenheimer et al. 2005). Together, this has led to the hypothesis that the current obesity epidemic is fuelled (at least in part) by the unprecedented abundance of low protein, energy dense foods that are highly palatable, yet have little satiety value (Martínez Steele et al. 2018; Raubenheimer et al. 2005).

Direct evidence suggesting that humans are eating to reach a protein target comes from an experiment where participants volunteered to stay in a chalet in the Swiss Alps. They stayed in the chalet for 6 days in total, during the first 2 days they were free to select their own food for each meal from a buffet, which varied in macronutrient proportions. The experimenters were interested only in protein consumption relative to fat plus carbohydrate consumption. For the next 2 days, participants were split into groups, one group received higher protein food (but lower fat and carbohydrate) than they had self-selected in days 1 and 2, and the other group lower protein (but higher in fat and carbohydrate), food than they had self-selected in days 1 and 2. The high and low protein food was designed to seem identical to the participants. For the final 2 days of the experiment, all participants ate as they chose – the same as on days 1 and 2 of the experiment. The results showed that participants ate to a protein target at the expense of carbohydrate and fat, thus, those put on a higher protein diet (than they self-selected in days 1 and 2) on days 3 and 4 underconsumed carbohydrate and fat, whereas the group on the lower protein overconsumed carbohydrate and fat in an attempt to consume more protein. The researchers concluded that when humans are forced to make choices about food, protein intake is prioritized over carbohydrate and fat (Simpson et al. 2003). This is consistent with many other species studied, including other primates, murids, fruit flies and Mormon crickets, suggesting that leveraging protein intake over carbohydrate and fat is evolutionarily conserved (Gosby et al. 2014; Simpson et al. 2003; Simpson and Raubenheimer 2012).

Conclusion

The origins and increased prevalence of obesity are multifaceted, and evolutionary explanations are paramount to understanding the obesity epidemic. The rapid change in our diet in recent years, as well as a shift to a more sedentary lifestyle, interacts with our nutritional balancing act to attempt to eat optimal quantities of protein, which perhaps leads to overconsumption of carbohydrate and fats. Although an overabundance of highly processed sugar and fat dense foods undoubtedly plays a role, the rise in the prevalence of obesity is unlikely to be caused by positive selection for a thriftier genotype.

Cross-References

- Body Fat Percent and Distribution
- Diet
- Food Preferences

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