

Obesity isn't just about diet - a brief discussion on the biological theories of obesity

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Biological Basis of Behaviour
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25 March 2015

Much of the world is in the midst of an obesity epidemic. Anyone wishing to confirm this notion needn't look far. From the popular press to the multitudes of research papers on the matter, everyone is in agreement that obesity rates have risen dramatically, particularly in the west, in recent decades. It has also become increasingly clear what a chronic, insidious and dangerous disorder obesity is. If the debilitating aspects of the condition on sufferers weren't bad enough, obesity has been strongly associated with the onset of various life-threatening conditions such as heart disease, cancer, type-2 diabetes (Friedman 2000) as well as chronic mood disorders such as depression (Luppino et al 2010). Obesity itself is, for most individual sufferers, a problem that can be addressed - weight can be shed through diet and exercise and it is widely accepted that a BMI (Body Mass Index) of < 28 is optimal for life expectancy. However, as much as dieting companies would like us to believe that the 'ideal' physique is but a tofu salad away, many sufferers of obesity find the pounds difficult, if not seemingly impossible, to shed. Of those that manage to do so via intensive dieting, 90% put the weight back on again (Friedman 2000). This suggests that there's more to the obesity epidemic than poor diet. In this essay I look at some of the main contributing factors.

One of the defining factors when looking at the obesity epidemic is that, by definition, it was not always such a big problem. Widespread obesity only became a factor in the latter half of the twentieth century. Tracing human lineage back through fossil records to the palaeolithic era (some 10,000 years ago) it's clear that our ancestors were considerably slighter of figure (O'Keefe and Cordain, 2004). This notion forms the backbone of an argument that obesity is a symptom of the modern (particularly western) world, rather than any change in human physiology. Palaeolithic society was a hunter-gatherer culture. Food was scarce, and obtaining it took considerable organisation and energy. Two food groups are requisite for maintaining such a lifestyle, sugar (e.g. fruit) and fat (meat and animal fat), both of which were scarce and required considerable energy to source. In addition, the energy hungry human brain, the largest in the mammal family, required frequent refuelling to maintain the food gathering and hunting activities. Thus this virtuous cycle led to humans evolving a particular taste for these food groups (Cohen, 2008) and, as some theories suggest, gaining brain volume as a result (Aiello and Wheeler 1995). Roll on ten thousand years, and while the human body and brain has not evolved

dramatically, human society has (Chakravarthy and Booth, 2003). These days food is no longer scarce - quite to the contrary, in the West it is in abundance - but we still desire it equally to those early humans. This is a more important idea than it may at first seem. To get to grips with this we need to explore some of the physical and psychological aspects of hunger, craving and feeding.

Our appetite is regulated in the hypothalamus. Two areas of the Hypothalamus have been identified that relate to the desire to eat and satiety - the Ventromedial Hypothalamus (VMH) and the Lateral Hypothalamus (LH). Lesions to the LH cause aphagia (the cessation of eating and drinking) whereas stimulation to this area has the opposite effect. Conversely, stimulation to the VMH has the effect of damping appetite in hungry animals (Smith 1956). This suggests that the VMH regulates satiety and the LH regulates hunger. This knowledge alone doesn't help us understand the cause of the obesity epidemic. Hunger is more complex than simply being famished or sated.

Since, for our ancient ancestors, food wasn't always available, the desire to seek out food, particularly foods high in energy, would have been essential. This effect can clearly be seen in the mechanisms that lead to our desire for food. Our brain's motivation, desire and reward mechanisms are regulated by the neurotransmitter dopamine. It has been shown that the mere sight of food triggers the release of dopamine in the dorsal striatum (Cohen, 2008. Palmiter, 2009) an area of the brain which is associated with eating and decision making (Balleine et al 2007). Modern humans are constantly presented with imagery of food (via TV, print, the internet) which triggers this innate desire response much more frequently than in less technologically developed times (or, indeed, modern, but less technologically developed cultures). To make matters worse, exposure to palatable food, for example foods high in fat and sugar, have been shown to increase hunger signals generated by our digestive system while also dampening our satiety responses in the VMH (Erlanson-Albertsson, 2005). What this suggests is that the constant bombardment of alluring images of food keep modern man in a constant state of gustatory desire.

The appetite problem doesn't stop there. Another contributing factor in hunger mechanisms is leptin. Leptin is produced by adipose tissue (body fat) and acts to suppress appetite by sending signals to the arcuate nucleus of the hypothalamus. A study found that rats

fed on a high fat/sugar diet developed a leptin resistance, meaning that satiety signals did not reach their brain, or were dampened, leading to hyperphagia and morbid obesity. However, the same study found this effect predominantly in rats with a certain genetic profile, suggesting both genetic and environmental factors play a role in this effect (Friedman et al, 1998). This finding is further supported by twin studies which have noted a strong heritability factor in obesity (Stunkard et al, 1986). This problem is further compounded by the fact that leptin is produced when fat levels drop (ie. body fat levels drop below the body's homeostatic level) thus triggering hunger signals. This mechanism forms the basis of the controversial Set-Point theory which states that any given person has a body weight bracket, with high and low boundaries, within which the body tries to remain (Nisbett, 1972). This bracket differs for every person and appears to be related to the volume of fat cells a given person has which is determined by genetics, but possibly also to conditions in their mother's womb prior to birth (Robinson, 2014). The implication is seen when crash diets are partaken in - 90% of dieters return to their previous body weight, their set point (Friedman 2000). This is probably because the state of semi-starvation de-stimulates leptin production as weight decreases, while levels of insulin rise meaning that more sugar gets stored as fat.

There is another view however that contradicts, or perhaps complements, the food intake hypothesis. The abundance of food is not the only factor that is different in modern society. With the advent of farming and the automobile, it is no longer necessary to expend vast amounts of energy finding food (foraging, hunting) meaning that, on balance, significantly less exercise is partaken in, and thus less energy is expended. Since we still maintain a high energy diet, but have lowered energy expenditure, the calories are stored as fat. The implication is that we do not, in fact, ingest more calories than our ancestors, but exercise less, resulting in an imbalance in the calories consumed to expended ratio. In addition to this, our ancestors would not have had a constant supply of food, but rather cycles of feast and famine as conditions dictated. It has been theorised that our bodies are genetically configured for this boom/bust cycle. Calories could not be wasted, they would be needed for the job of finding more food, hence the body is extremely efficient at storing unused calories, yet these calories are never burned (Chakravarthy & Booth, 2003). This view, however, fails to take into account the fact that the obesity epidemic only

really became a problem in the last three to five decades, yet transport and mass farming have been around somewhat longer. So perhaps the answer lies in a combination of reduced exercise, increased food availability and genetic, epigenetic and physiological factors.

Of course, upbringing and sociological factors play a significant role in this. Obesity is socially stigmatised in Westernised cultures. This ideal figure (particularly for females) is slender and athletic, something that many (perhaps the majority of) people are unable to achieve. Failure to achieve this goal causes anxiety in many leading to mood disorders like depression.

Paradoxically, eating provides a momentary relief from these symptoms (through the release of dopamine and serotonin), leading to comfort and, in some cases, binge eating which further exacerbates the weight problem (Luppino et al 2010). In extreme cases this vicious cycle can lead to eating disorders such as anorexia nervosa and bulimia.

No single theory, regardless of how well researched, is likely to singularly explain the obesity epidemic. The physiological, sociological and psychological influencers are numerous and each incredibly complex. The palaeolithic theory, while attractive in its apparent simplicity and all encompassing nature, is hard, if not impossible to verify and is almost certainly not the whole picture as relates to the obesity epidemic. It is, after all, plausible that human physiology *has* changed in ten thousand years, even if that change is maladaptive. In addition, epigenetic heritable factors must be taken into account - perhaps the human genome hasn't changed a great deal, but what of the way that it is expressed? What's clear is that as more is understood about the contributors to the current state of affairs, the more questions arise.

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