Foundational Concepts for Understanding Body Weight and Obesity

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1. Introduction

There has been much research on food consumption and obesity yet, its causes and solutions remain poorly understood. I believe the study of obesity and the discovery of cures has been hindered due to a focus on identifying proximate rather than ultimate level explanations. In this paper I will provide an overview of foundational concepts that will enable a deeper understanding of human body weight, the biological function of fat and factors that may be drivers of the obesity epidemic.

2. How big is the problem of obesity?

The World Health Organization has identified obesity as a worldwide health epidemic. In 2014 it reported 1.9 billion adults were overweight or obese. Child obesity is also on the rise. In 2013 the World Health Organization reported 42 million children under the age of five years of age were overweight. Obesity is associated with many health problems including increased risk of diabetes, fatty liver disease, hypertension, heart disease, sleep apnea, cancer, endocrine and orthopaedic disorders as well as psychiatric disorders (Lobstein & Dibb, 2005).

Many biological and environmental factors have been linked with obesity. The challenge has been to understand how these factors are related to obesity and whether these factors are causal, contributing factors, risk factors, antecedent factors or, spurious associations.

3. The obesity epidemic is new, obesity itself is an ancient problem

Obesity is a result of a prolonged positive energy imbalance where energy intake exceeds energy expenditure (Moore & Cunningham, 2012). One is considered to be overweight if they have a BMI from 25 to 29.9 kg/m^2 and obesity is defined as having a body mass index of 30 kg/m^2 (Mitchell, Catenacci, Wyatt & Hill, 2011). While the current obesity epidemic is a relatively new phenomenon, obesity itself is not. Historically, excessive thinness and malnutrition have been the overriding societal problem. While malnutrition has been the norm, obesity existed among the privileged social classes. Throughout history obesity has been common in the upper classes of society whom lived in

an obesogenic environment. An obesogenic environment is one which enables excessive food consumption coupled with low energy expenditure.

Wells (2010) reinforces this point and states, obesity and its associated health problems have been documented since ancient times. The Egyptian papyrus from approximately 1550 B.C. lists magical formulas and remedies to cure conditions caused by obesity. Greek philosophers such as Hippocrates and Pythagoras also wrote about the problems associated with obesity and made recommendations for its cure.

4. Why is it important to differentiate between obesity and the obesity epidemic?

It is commonly claimed the obesity epidemic is caused by environmental factors (Carlos, Poston & Foreyt, 1999). However, environmental factors are most likely proximate variables that enable rather than cause obesity. Ultimate level explanations for obesity and the drive to consume beyond the point of satiation will be explained by discovering how genes interact with the environment and why the drive to over consume was an adaptive trait that helped our early ancestors survive (Wells, 2010).

The answer to why it is important to differentiate between obesity and the obesity epidemic is best described through the following metaphor. Obesity and the obesity epidemic are like a dandelion. Dandelions are notoriously difficult to eradicate. If you cut off the foliage that grows above the ground the dandelion will grow back when the environmental conditions are favorable. To eradicate the dandelion you have to deal with the roots of the plant (or the root problem). Obesity and its genetic basis are the roots of the dandelion and the foliage is the obesity epidemic. If you only address environmental conditions that enable or contribute to obesity, it will return when the environmental conditions are favorable. Therefore, the discovery of a solution to the obesity epidemic will require an understanding of how genes interact with environmental variables.

5. The benefits of body fat

The human brain and body have evolved in a manner that supports survival and reproduction. As previously noted, humans evolved in a world where food was often scarce. Fruits and vegetables are seasonal and only available during short periods throughout the year. Capturing wild game would be challenging and would involve high levels of physical activity and energy expenditure. Due to these challenges it is reasoned that historically, food shortages and famine may have been frequent (Pinel, Assanand & Lehman, 2000). Those who would survive a famine would likely be those who had the genetic predisposition to continue consuming beyond the state of satiation. From an evolutionary perspective, body fat plays a vital survival role by acting as an energy reservoir that can be drawn upon when food is short in supply (Kardum, Gracanin & Hudek-Knezevic, 2008., Korte, Koolhaas, Wingfield & McEwen, 2005., Zafon & Simo, 2011).

In addition to being a storage reservoir for future energy needs, fat stores are needed to support reproduction. The initiation of puberty in both males and females is dependent upon sufficient levels of body fat. Studies have shown a relationship between the onset of puberty and fat composition. Prepubescent girls who have higher fat content begin to ovulate at an earlier age. Whereas girls with low fat content ovulate later that the norm (Cheng et al., 2012). Prolonged low body fat can also terminate ovulation in the short-term and lead to a permanent loss of fertility (Kardum, Gracanin & Hudek-Knezevic, 2008).

Breast milk plays a vital role in the survival of infants. Human breast milk contains 87% water, 1.6% protein, 6.8% carbohydrates, 2% ash and 4% fatty acids (Robson, 2004). The fatty acids come from both fats the mother consumes and from the mothers own fat stores. These fatty acids provide a source of calories, nutrients and promote the development of the infant's immune system. (Bellisari,2007., Hoppu, Isolauri, Laakso, Matomaki & Laitinen, 2012).

While body fat provides immunities to new born infants it continues to support the maintenance of a healthy immune system throughout one's life. When fat stores are depleted the body's immune system weakens and becomes more susceptible to disease. Wells (2010) reinforces this point and states, the majority of famine related deaths are due to infectious diseases is due to immunocompetence caused by malnutrition rather than starvation.

6. The influence of genetic factors on body fat composition

The level of body fat one contains is an outcome of the interaction between genetic and environmental factors. More than 250 genes have been identified that relate to body fat composition (Lopes, 2009). Some genes influence how the body uses fat stores for physiological functions such as breast feeding, immune system development and maintenance. Genes have also been linked to the body's stress response system and food preference (Foss & Dyrstad, 2011). Other genes direct the location of fat deposition, metabolism (Pervanidou & Chrousos, 2012), appetite and energy expenditure (Bell, Walley & Froguel, 2005).

Studies have estimated the strength of genetic factors by conducting twin studies. Estimates of heritability vary based on whether the twins in the study were dizygotic or monozygotic, whether the dependent variable was based on BMI or percentage of body fat and whether the twins were raised together or separated at birth.

Based on an analysis of the Danish Twin Registry, Schousboe et al. (2004) found the average heritability of monozygotic twins reared together was .58 for women and .63 for men. It was also determined that heritability rates increase with age. Other studies have duplicated this finding. In a longitudinal study of twins reared apart, Stunkard et al. found the initial heritability rates were .66 for women and .70 for males. However, in a follow-up study with the same twins 25 years later, it was concluded the average heritability of body weight for both genders increased to .81 (Bell et al., 2005).

It has been argued that genetic factors cannot be the reason for the rapid rise of the obesity epidemic. If the obesity epidemic is a result of genetic factors, then there must have been rapid changes in the population's genetic profile. Genetic changes normally spread through a population slowly over numerous generations. Therefore, it is argued genetics cannot be the root of the current obesity epidemic because genetic changes could not have moved through the world's population as quickly as obesity has risen (Hebebrand & Hinney, 2008., Hebebrand, Wulftange, Georg, Ziegler, Hinney, Barth, Mayer & Remschmidth, 2000).

Hebebrand et al. (2012) disagree and argue genetic traits can move through a population quickly through the process of assortative mating. With regards to obesity, the theory of Assortative Mating

suggests when two people who carry the genes for obesity reproduce; the offspring are more likely to be carriers of a genetic profile that supports the development of higher levels of body fat. Therefore, theoretically it is possible to have a rapid genetic change in a population in a short period of time. The Assortative Mating hypothesis is supported by the findings that 80% of obese children have one parent who is obese and 25%-30% of obese children have two parents who are obese (Poskitt, 1993).

A second argument put forth by the author of this paper is that genetics can be the ultimate causal factor in obesity without requiring changes in genetic make-up of a population and without the practice of assortative mating.

It is possible that one's genetic endowment may not be fully expressed in the absence of supportive environmental factors. For example, under optimal environmental conditions a stalk of wheat may have the genetic code that enables the grass to grow to a height of 3 feet. The hours of sun per day, level of soil moisture and sufficient nutrients are all necessary for the wheat to reach a height of 3 feet. The genetic growth potential of a stalk of wheat may be stunted or delayed until optimal environmental conditions occur. The same logic may be applied to understanding the obesity epidemic. The genetic predisposition to over consume and gain fat may be prevented or delayed in the absence of supportive environmental conditions. These conditions will be discussed in the following section. It is interesting to note as world poverty has decreased (World Bank, 2013) world obesity has increased. Decreasing poverty is a significant environmental change that enables food acquisition and consumption.

7. The influence of environmental factors on body fat composition

When studying food consumption and obesity it is sometimes said, "genetics is the gun and the environment is the trigger" (Hebebrand & Hinney, 2008). This metaphor reinforces the concept that obesity is a product of a gene-environment interaction. However, the term "trigger" in this metaphor does not accurately represent the total category of environmental factors. Not all environmental factors act as a trigger. Some environmental factors are more accurately classified as enabling factors rather than triggering factors.

For the purpose of this paper, enabling environmental factors are considered to be factors that enable the acquisition and consumption of food. For example, decreasing food prices enables consumers to purchase greater quantities of food (Epstein, Jankowiak, Nederkoorn, Raynor, French & Finkelstein, 2012). However, it would be illogical to assume decreasing prices would trigger an increase the physical consumption of foods that are less desirable.

Other variables that may enable food consumption include income, as income rises people are able to buy greater quantities of food. However, buying food is not the same as consuming food and it would be incorrect to assume that there is a one-to-one relationship between buying and eating.

There are many proximate variables that enable food consumption. The purpose of mentioning enabling factors is to develop a deeper understanding of environmental factors by contrasting enabling factors with triggering factors. For the purpose of this paper triggering factors include any variables that stimulate appetite or the desire to eat.

Advertising is often implicated as a trigger that increases food consumption (Hoek, 2006., Thomson, Spence, Raine, & Laing, 2008,. Palmer, 2010). While the claim is often made there is a lack of clear evidence to support the claim of causality (Young & Hetherington, 1996., Lobstein and Dibb, 2005). Other researchers conclude advertising cannot be a causal factor because children's food preferences are well-established before advertising is understood and because many other sociocultural variables, such as parents, peers and schools can influence consumption decisions (Ambler, 2006., Young, 2003,. Young & Hetherington, 1996,. Harker, Harker & Burns, 2007).

Another environmental factor that has been implicated as contributing to childhood and adult obesity is prenatal and early childhood under nutrition. Under nutrition contributes to obesity in a number of ways. First, under nutrition tends to lower one's metabolism and promotes the development of metabolic disorders such as insulin resistance which leads to excessive weight gain. Second, people who have experienced under nutrition early in life report higher levels of hunger and increased appetite.

8. The relationship between stress and food consumption

Humans evolved to be in a state of physical and psychological homeostatic balance. For example,

humans have an ideal body temperature, ideal level of oxygen and an ideal level of acidity. When the body is knocked out of balance, the stress response system turns on and attempts to reestablish a homeostatic state (Sapolsky, 2004). A stress response can be triggered when one senses a threat, real or implied to their psychological or physiological well-being. The stressor can be sensed in the present moment or at some time in the future (Greenberg, Carr & Summers, 2002).

Humans are not always conscious of threats to their well-being. Sometimes the subconscious mind feels the threat and responds in a protective manner. For example, threats to ones self-esteem (Adam & Epel, 2007) and threats to one's social status (Tamashiro, Hegeman & Sakai, 2006) are felt at a subconscious level resulting in a state of psychological tension.

There are two types of stress responses; acute and chronic. The acute stress response system acts like a fire alarm. It signals to the individual that their safety and survival are in immediate danger. When the body senses acute stress the adrenal glands produce cortisol which shunts blood to the large muscles to prepare it for a fight or flight response. Once the threat has been removed the stress response system shuts off. When an acute stressor has been removed the body goes back to a homeostatic state. Acute stress triggers the suppression of appetite and hunger (Torres & Nowson, 2007., Sinha & Jastreboff, 2013). A possible evolutionary explanation for appetite suppression during acute stress is that it would be counter-productive for an organism to engage in a search for food while at the same time trying to remove an immediate environmental threat (Dagher, 2010).

A second type of stress is chronic stress. Like acute stress, chronic stress triggers cortisol production. However, unlike acute stress chronic stress is enduring and lasts for long periods of time. Chronic stress is less intense than acute stress and tends to not be a threat to one's immediate survival. For example, if one is continually worrying about where their next meal will come from or, if they will experience a violent encounter in the near future they will likely have chronic stress. What's more is that an individual may experience multiple chronic stressors simultaneously. It is well documented that chronic stress increases appetite and hunger (Born, Lemmens, Rutters, Nieuwenhuizen, Formisano, Goebel & Westerterp-Plantenga, 2010). Chronic stress may act as a moderator in that it sends a signal to the brain to increase appetite while at the same time acting as a mediator in directing food selection towards high motivation or energy dense food. (Torres & Nowson, 2007., Dagher, 2010).

There are two theories as to why energy dense foods are preferred during a time of chronic stress. First, energy dense foods are often high in fat, sugar or salt. These foods stimulate dopamine production in the brains reward center (Carter, 2009). Simultaneously, there is a reduction in cortisol production. The increase in dopamine and decrease in cortisol work-together to turn off the stress response system (Adam & Epel, 2007). This process helps repair ones mood and brings the body back to a homeostatic state (Sinha & Jastreboff, 2013). A third theory proposed by the author of this paper is that if stress prepares one's body for a fight or flight response, it would make sense that the body would want to 'fuel-up' to provide energy for when the response is needed.

9. Environmental harshness and food consumption

There are many factors that trigger acute and chronic stress. One stressor is environmental harness. Harsh environments are rife with uncertainty and may include exposure to pathogens, inhospitable climates and weather, exposure to threats from predators and, a scarcity of resources such as food (Brumbach, Figueredo & Ellis, 2009). Laran & Salerno (2012), state humans may subconsciously perceive cues of environmental harshness and interpret these cues as signals that resources needed for survival are scarce. In response to harshness cues people may seek and consume high density foods in order to build fat reserves that protect them from future food shortages (Dallman, Pecoraro, Akana, la Fleur, Gomez & Houshyar et al. (2013) , Griskevicius, Ackerman, Cantu, Delton, & Robertson, 2013).

A study by Laran & Salerno (2012) tested this hypothesis and found that when exposed to the words "survival, withstand, persistence, shortfall, struggle and adversity," the consumption of M&M candies increased. Whereas, when exposed to neutral words the consumption of candies did not increase.

One limitation of this study was that while it did demonstrate that harsh words can stimulate the desire to eat, it did not show how food preference changes as a result of perceived harshness. Specifically, there was not an option to choose between low and high motivation foods.

To develop a deeper understanding of the relationship between perceived harshness and food preference an experiment was conducted by Jim Swaffield under the supervision of Dr. Craig Roberts. This experiment added to this body of knowledge and demonstrated that perceived

environmental harshness can mediate food choice. In sum, as environmental harshness increases the desire for high density food also increases. This study supports the evolutionary perspective that the desire for energy dense food is an adaptive response that prepares one for potential future food shortages.

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