Main Submission

Fat taste bud receptor CD36 identified in humans: Obesity explanation or excuse?

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Obesity is a medical condition in which an individual weighs more than 20% over their ideal weight, as characterized by their body mass index (BMI).[1] There have been a multitude of genetic and environmental factors identified that contribute to the etiology of obesity. However, regardless of the precise mechanism, the results are the same – the caloric intake of affected individuals exceeds their energy expenditure. Over time, their bodies store all unused calories as fat, resulting in a significant increase in overall body fat content. Past studies have shown that many obese people prefer, crave, and actually consume high fat foods more frequently.[2,3,4] So what is it that causes foods with a high fat content to be so attractive?

It has long been determined that the human tongue can distinguish between five distinct tastes, although fat is not one of these. The classical model of fat perception in the mouth was that it relied only on cues from food texture and smell.[5] However, there is an increasing body of evidence that has further revealed the role of the gustatory system in the oral perception of fat,[5,6] whereby the mechanism of taste detection of lipids is triggered by the hydrolysis of triacylglycerols in foods into fatty acids by oral lipases. Animal studies have been used to identify putative fat taste receptors.[7] Of these, the glycoprotein CD36 has been of particular interest owing to its involvement in a number of metabolic pathways with a particular relevance to obesity and obesity-associated complications.[5,6,7,8] For instance, CD36 is known to initiate inflammation in response to excess fat supplies, which subsequently promotes new cascades of metabolic pathologies.[6] Moreover, scientists have discovered that CD36-deficient rodents not only have decreased preference for fatty foods, but also suffer complications in digestion caused by an inhibition of the pancreatic secretions normally triggered by the exposure of their tongues to fat.[6] Importantly, CD36 expression has recently been confirmed on human taste bud cells,[8] and the relevance of the discoveries made in animal models has been further augmented by the realization that many CD36 variants found in rodents are shared by humans.[6]

A research team led by Dr. Nada A. Abumrad from the Washington University School of Medicine in St. Louis has confirmed that individuals with a particular CD36 allele are far more sensitive to the presence of fat in foods than others.[6] Such variation in the CD36 gene between individuals is a potential cause for the disparity in fat preference within a population. Adding insult to injury, Dr. Abumrad further proposed that CD36 levels in humans can be altered by the foods we eat and the amount of fat we consume. As people ingest more fat, they become less sensitive to it, thereby requiring a greater intake to achieve the same level of satisfaction.[6] Taken together, it is evident that the expression of CD36 alleles combined with a lower fat sensitivity can form a solid basis for the development of obesity. Indeed, it is estimated that as much as 20% of the world population expresses the CD36 gene variant associated with lower levels of CD36, and thus has a lower sensitivity to the taste of fat.[9] The question therefore arises – are the rising obesity trends simply a matter of us experiencing a greater predisposition towards the enjoyment of fatty foods than our previous generation?

Genetic screening for potential disease markers such as CD36 is an ever-growing field of research. Participants of such screening processes tend to stress the emotional and social consequences of potential positive test results, rather than the actual physical outcomes. However, the emphasis of the public on nonclinical burdens of a particular trait is potentially more dangerous than possession of the trait itself.[10] While it is true that carrying an 'obesity gene' will lead to greater susceptibility to developing the disease, does what we know about our genes supersede what we can do? The view that the problem 'lies in my genes and is therefore not remediable by my lifestyle changes' further

jeopardizes our health,[10] and is completely erroneous. There is always something we can do.

The pharmaceutical industry is beginning to realize the potential of gustatory perception in the treatment of obesity. Drugs such as orlistat are currently being administered to inhibit the oral lipases that initiate the pathway of fat taste receptor activation.[6] Nonetheless, simply taking anti-obesity pills while not making diet and exercise changes will not solve the problem. We should not use discoveries such as the fat taste bud receptor as an excuse to continue being unhealthy. Lifestyle changes, though difficult, are the only way in which we can combat the immutable force our genes play on our metabolism.

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