



Genetic Predisposition to Obesity - an Ancestral Legacy

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listed in weight loss, originally published in issue 176 - November 2010

Introduction.

Many people would like to be slimmer, have a healthier body mass index (BMI) or have less body fat deposits.

Fat deposition can be reduced by increasing exercise and/or eating less food. However, for some people, increased exercise may pose an undesirable health risk and eating less food may be a difficult challenge to many people. So, apart from drug support or surgical intervention, an alternative approach to reduction of fat deposition would be of great benefit.



The Problem

The real problem is that, to enable our ancestors to survive much better, nature altered the human genetic blueprint so that they could store bodily fat more readily. Now, where food is plentiful,

nature's genetic alteration has become a major health hazard.

We are all aware of a small number of people who can eat heartily, including foods with high caloric content, and yet maintain a healthy BMI. Why cannot we all be like those people? Part of the answer lies within our genetic makeup. We are now beginning to understand more about why most of us find it difficult to maintain a healthy BMI. Investigations have revealed that modern man has inherited a common ancestral predisposition to become overweight. So, why is this genetic predisposition to become overweight so common when a lower BMI is generally associated with optimum health. From an evolutionary standpoint, it does not seem to make sense. To understand why, we must take a closer look at the interaction between the environment and our genetic blueprint throughout evolutionary history.

The Answer

The story began to unravel about fifty years ago. At that time, a young researcher by the name of Winifred Doane was on a mission to find 'famine-survival' genes. She argued that gene types that could help survival would fulfil two criteria. Firstly, they would be common in geographical regions that were frequently subjected to times of famine and secondly, they would be found in species that had become highly efficient at storing fat.

In Nigeria, she found some unusually fat flies. On examination, she discovered that the fat flies had a defective form of a gene she called adipose (ADP). Since then, the ADP gene has been found in many different species [see Ref. 1 and references therein]. Reduced activity of the ADP gene has been shown to lead to fat accumulation in all species studied. In 2003, the same ADP gene was discovered in humans; [2] however it was not known whether the human ADP gene (now called WDTC1) was involved in any way in fat accumulation as it was in other species.

The 'Sluggish ADP gene

A few years later, scientists at Tufts University solved the mystery. They discovered that most of us have inherited a 'sluggish' ADP gene that slows down the way that fat is burnt off or turned into energy. [3] It also enables fat to be stored in our tissues more easily so that we gain weight. So, where did our sluggish ADP gene come from?

It is now thought that the poorly acting ADP gene originated many thousands of years ago and, over time, increased in frequency because it gave our ancestors an important survival advantage. The less effective fat metabolizing gene was of benefit because what little food was available was easily laid down and stored as fat deposits. Fat deposits could then be used as a source of energy and help survival in times of famine. Those who carried an efficient fat metabolizing gene, who could not store fat so well would have been more likely to perish in times of famine, resulting in loss of the efficient ADP gene within the population. That is why it is so uncommon now.

Interestingly, the sluggish ancestral ADP gene, although it predisposes to ill-health in many people, is in fact, considered normal and completely natural because it is the most common form of the ADP gene.

Health Hazard

In modern times though, in areas of the world where, for the first time in human history, food is always plentiful and refined, the sluggish ADP gene has become a health problem because it readily

stores our food as fat. Those that do not experience times of famine continue to store fat, become overweight and prone to development of diseases associated with being overweight such as diabetes and heart disease. In fact, it is suggested that the sluggish ADP gene is directly responsible for development of late onset diabetes associated with obesity.[4] Increasing the efficiency of everyone's sluggish ADP gene, would be an effective way to lose weight, reduce fat deposition and maintain a healthy BMI and to avoid development of and even reverse late onset diabetes.

Appetite Reduction

Another way to lose weight, or reduce fat deposition is to eat less food. This is very difficult for many people because, to satisfy their insatiable appetites, they have an overwhelming urge to eat lots of food. This common urge is reflected by the popularity of buffet style food outlets.

To suppress the feeling of hunger and eat less, apart from the use of appetite suppressing drugs, anorectics, many subject themselves to various surgical interventions, not without dangers, to limit their food intake. A safer approach to appetite suppression would be of great benefit.

Leptin, the Appetite Suppressant

Over a decade ago, scientists discovered a gene 'ob' now called LEP that plays an important role in appetite suppression. LEP produces the appetite controlling hormone leptin. The defective LEP gene was first found in an unusual strain of mouse that readily became obese due to its voracious appetite. Subsequently, it was discovered that the appetites of these leptin deficient obese mice could be controlled by administration of leptin. Humans with leptin deficiency also have voracious appetites and suffer profound obesity. As with the obese mice, their appetites can be controlled and weight lost by administration of leptin, confirming that leptin plays an important role in suppressing the appetite and weight control.[5] It follows that boosting LEP activity would reduce the appetite and promote weight loss.

Scientists have shown that as overweight people lose weight, leptin levels fall.[6] As a result, the urge to over-eat increases and weight is regained. Once weight is lost, increasing leptin activity plays an important role in maintaining the desired BMI.

A 'Sluggish' Ancestral LEP Gene?

Interestingly, most of us are aware of a small number of people who are slim because they have what is considered to be a 'poor' appetite and are quite satisfied on consumption of small amounts of food; they cannot do justice to the 'eat all you can' food outlets. It remains to be determined whether, in contrast to those with a poor appetite, most people have inherited a sluggish form of the LEP gene, because a poorly acting LEP gene would have helped our ancestors to eat more in times when food was accessible. If that were the case, then most people now, who had inherited a sluggish ancestral form of the LEP gene will have a greater likelihood of fracturing their bones and an increased susceptibility to development of late onset diabetes.

Why, because firstly, studies have shown that leptin plays a very important role in increasing bone density, reducing the likelihood of bone fractures, particularly in post-menopausal women. Bone is continuously being remodelled, old bone is being replaced by new bone. Leptin promotes new bone formation in a dose dependent manner, [7] so, low leptin activity is associated with reduced new bone development.

Secondly, it was previously thought that late onset diabetes, associated with obesity was relieved simply as a consequence of leptin induced weight loss. Now, scientists at Rockefeller University have made the exciting discovery that leptin can correct late onset diabetes independently of its role in weight loss. In other words, even if weight is not lost, leptin can still play a very important role in correcting diabetes.[8]

The Solution

There is now a much more focused way to lose weight, and that is to simply make everyone's sluggish ADP gene more effective. The molecular geneticist Dr Peter H Kay developed the health supplement Homeovitality Super Weight-Loss in Australia to accomplish exactly that. Homeovitality is based on the proven drug free gene targeting principles of Micro-Immunotherapy, developed by Dr Marichal in which immune response genes are targeted by highly diluted DNA molecules.

Homeovitality Super Weight-Loss has also been designed to help people lose weight more comfortably by targeting the LEP gene and maintain the desired body mass index when it has been reached. As stated, LEP reduces the feeling of hunger so it is easier to eat less food. In summary, the Homeovitality Super Weight Loss supplement is designed to help people reach and maintain a healthy BMI by promoting ADP activity, so that fat is burned off as energy more effectively and fat deposition is lessened, and by targeting the LEP gene to reduce the feeling of hunger. Everyone who has inherited the common ancestral ADP gene would benefit from this Homeovitality Super Weight Loss supplement.

References

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Further Information

Homeovitality Super Weight Loss supplement health product can be obtained by contacting your nearest Naturopath, Homeopath or from www.wellnesshp.com

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