Fat and genes: can they predict stroke?

The modern way of life has led to an unprecedented increase of diseases that affect the well-being of our arteries, including high blood pressure, high lipids, diabetes and obesity. These diseases promote atherosclerosis, which means the deposition of fat in the arteries, which in turn results in narrowing of the arteries culminating in myocardial infarction and stroke. Stroke is the fourth most common cause of death and the leading cause of long-term disability in high-income countries. Patients with a history of stroke are at increased risk for stroke recurrence and for myocardial infarction. Thus, there is a great need to identify patients that are at increased risk for having a stroke.

For this reason, in recent years researchers have studied some proteins called adipokines. Their name is due to the fact that these proteins are mostly produced by the adipose tissue, namely our fat. Adiponectin, leptin and resistin are the most extensively studied adipokines and play a role in a variety of pathways related to atherosclerosis, including the regulation of glucose, lipids and blood pressure. In obese patients, the circulating levels of these adipokines and their actions are perturbed contributing to the emergence of high blood pressure, high lipids, and diabetes. Given these adverse effects of adiponectin, leptin and resistin, a number of studies aimed to elucidate whether they could predict the occurrence of stroke, as well as its severity and outcome. However, these studies yielded conflicting results. Most of them suggest that elevated levels of adiponectin and resistin are associated with increased incidence of ischemic stroke in both genders whereas elevated leptin levels appear to increase the risk for ischemic stroke only in men. There are more limited data regarding the effect of these adipokines on stroke severity and outcome.

On the other hand, in the last decades, the interest of scientists has shifted towards the exploration of the genetic basis of human diseases. In this context, many investigators have tried to study the possible relation of stroke with single nucleotide polymorphisms (SNPs) of genes that encode adipokines. SNPs are changes in the structure of our DNA, similar to mutations, but much more frequent. SNPs in the genes that encode adipokines affect the levels or function of the respective adipokine. Because SNPs may be present from birth or appear during life, they can help in early identification of patients at high risk for the disease that they cause. However, very few studies have assessed the predictive role of SNPs of the adiponectin, leptin and resistin genes regarding stroke risk and the findings were controversial.

In conclusion, it seems that measurement of adiponectin, leptin and resistin and the evaluation of the genes that encode them might be helpful in identifying patients with high risk for stroke, more severe stroke and less favourable outcome. However, more studies are needed to clarify these correlations and if such associations are proven, therapeutic interventions targeting adipokine levels might represent a novel approach to reduce stroke-related mortality and disability.

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Publication

The role of adipokines in ischemic stroke risk stratification.
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Int J Stroke. 2016 Jun