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A Tale of Two Sites: Insulin Resistance in Peripheral Tissues and the Brain

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Abstract

Insulin resistance has been recognised since a few decades after the discovery of insulin. To paraphrase a classic Charles Dickens novel published 62 years before the discovery of insulin, this is the best of times, as the concept of insulin resistance has expanded to include the brain, with the realisation that insulin has a life beyond glucose regulation. In other words, it is the worst of times because insulin resistance is linked to deadly diseases such as diabetes, obesity, and Alzheimer's disease, all of which affect the brain. Peripheral insulin resistance affects nearly a quarter of the adult population in the United States. It has recently been linked to Alzheimer's disease, specifically the level of brain insulin.

Keywords: Insulin • Brain • Diseases • Diabetes

Introduction

By the early 1940's, clinicians had firmly established the concept of insulin resistance after observing that there were a few patients who required large doses of insulin to control blood sugar. Around the same time, the first case of endogenous hormone resistance was reported, when patients with the phenotype associated with low blood levels of parathyroid hormone were discovered to have high levels instead. Pseudohypoparathyroidism, a type of hormone resistance, was eventually discovered to be caused by low levels of parathyroid hormone receptors. The concept of hormone resistance grew in two ways over the next few decades. Other hormones were discovered to have resistance syndromes in one way. Hormone resistance is caused by inactive/underactive hormone production, antibodies that block hormone/receptor interactions, a decrease in hormone receptor number, a decrease in post receptor signalling, and downstream unresponsiveness. Many of the above-mentioned causes of hormone resistance, such as antibodies directed against the hormone or its receptor, were first described for insulin. When a hormone relies on transport across a barrier, such as the blood-brain barrier (BBB), to reach its site of action, poor transport can lead to hormone resistance. In all of these cases, elevated blood hormone levels occur in the face of inadequate hormonal activity.

Literature Review

Insulin resistance is commonly defined as a lack of or insufficient response to insulin. Insulin resistance is associated with many common diseases, including type 2 diabetes. Although not directly tested, it is hypothesised that CNS insulin resistance precedes cognitive decline because the degree of CNS insulin resistance correlates with cognitive decline. The primary component of CNS insulin resistance is the brain, as mentioned throughout this review. Spinal cord insulin resistance has not been thoroughly studied in the context of cognitive decline. Deregulated nutrient sensing, of which insulin resistance is a component, is a part of ageing and is one of the nine original Hallmarks of Aging, though this concept is likely due in part to peripheral insulin signalling.

In this review, we will briefly introduce the insulin receptor signalling pathway, the similarities and differences in the aetiology of peripheral and CNS insulin resistance, insulin resistance testing and determination, and the consequences of these two conditions, with a focus on the impact of ageing and age-related cognitive decline. Because apoE has been linked to insulin signalling and age-related cognitive decline, we will investigate the peripheral versus central action of the apoE isoforms. Finally, we highlight recent findings regarding the role of insulin resistance in the development of COVID-19 and the associated cognitive injury.

Discussion

We do not provide an overview of the past 30 years of fMRI; rather, we offer a very personal perspective on the beginnings of fMRI in Germany and our experiences in an fMRI research unit at one of Germany's smallest medical faculties, the University Medicine of Greifswald. We do so because running an fMRI group in a university radiology department is a unique challenge, and it could be a valuable strategy for combining methodological and neuroradiologic expertise with an interest in neuroscientific research. Historically, but also practically, the majority of fMRI research units are located in neurological and psychiatric institutes, despite the fact that radiology and neuroradiology have had a significant impact on the development of a number of methods.

We will look at the similarities, differences, and connections between peripheral and CNS insulin resistance, as well as the role of insulin action in each of these systems, which are made up of multiple organs or cell types. The definition of insulin resistance in peripheral tissues appears straightforward: whenever more than "normal" levels of insulin are required to maintain blood glucose levels, insulin resistance is presumed to be the cause. In a negative feedback loop, insulin and glucose levels in the blood are locked together. When blood glucose levels rise under physiologic conditions, more insulin is secreted to bring them back into the normal range, at which point the stimulus for insulin secretion is removed and insulin secretion and blood levels of insulin return to normal. In order to research protein phosphorylation and dephosphorylation, phosphotyrosine derivatives are helpful chemical tools. The first assumption that has not been held is that CNS insulin resistance occurs as an extension of or in tandem with peripheral insulin resistance; in other words, hormonal resistance occurs concurrently in all tissues, peripheral

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and CNS. As a result, if peripheral insulin resistance is linked to Alzheimer's disease, there must be a CNS insulin resistance component as well. This logic was reinforced by the discovery that many risk factors for insulin resistance, such as obesity, hyperlipidemia, and inflammation, were also risk factors for AD. Diabetes mellitus or a lack of CNS insulin action have also been linked to Alzheimer's disease. While the assumption's inferences have led to significant discoveries, the assumption itself, that insulin resistance in the periphery and CNS [1-6].

Conclusion

The effects of peripheral apoE on the CNS may be related to apoE isoforms and/or levels. Human apoE targeted replacement mice exposed to a chronic variable stress paradigm have higher plasma apoE levels than apoE3 and apoE wild-type mice. Similarly, apoE2 mice have higher cerebellar apoE levels than apoE3 and apoE4 mice. There is also a gender difference, with females having higher plasma apoE levels than males, which is likely due to apoE2 expression. The expression of one of its receptors, the low-density lipoprotein receptor, also influences mouse apoE levels. Mice expressing apoE2 in addition to human LDLR have higher plasma apoE levels than apoE3 or apoE4 mice. More importantly, the presence of the human LDLR influences the levels of apoE.

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Conflict of Interest

There are no conflicts of interest by author.

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