- Alzheimer's Disease / Diabetes

ALZHEIMER'S IS CALLED TYPE 3 DIABETES NOW. HERE'S WHY

Studies reveal that insulin plays a vital role in brain function, and that insulin resistance and dysfunction in the brain play a crucial role in the development of Alzheimer's

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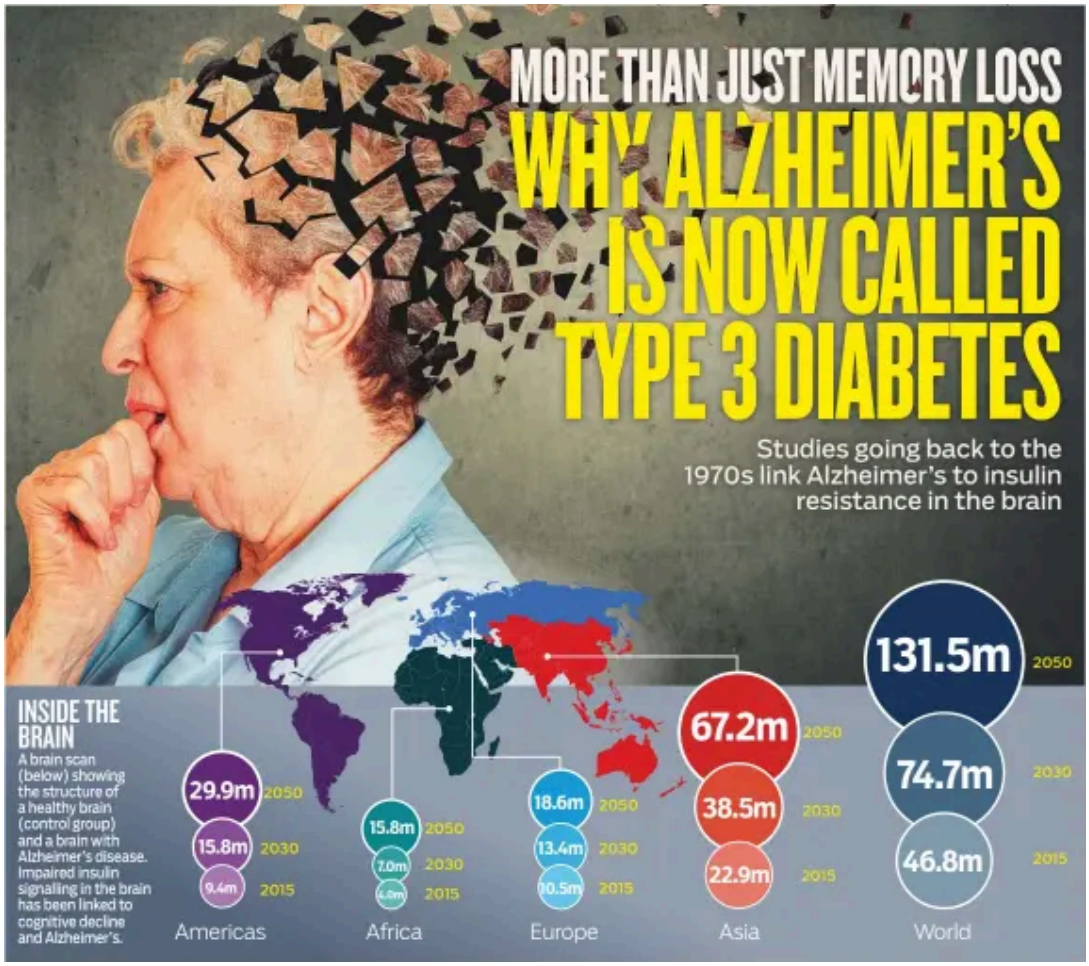
Ahidden connection between insulin resistance (IR) and Alzheimer's disease (AD) is now starting to unravel. For decades, researchers have been untangling the complex relationship between the two.



For one, studies dating back to the 1970s have revealed the presence of insulin receptors in the brain, particularly the hippocampus.

THE BRAIN HAS A SWEET TOOTH

It turns out that the brain does have a sweet tooth. Insulin, the hormone responsible for regulating blood sugar, also plays a vital role in brain function. Let's dive into it:

What is Alzheimer's disease (AD), and why is it being called 'Type 3 diabetes mellitus' (T3DM)?

AD is characterised by decline in cognition. It is often age-dependent. In its earliest stages, AD is the result of amyloid-(A)-mediated dysregulation.

This dysfunction affects the key pathway involved in learning and memory in the hippocampus.

Is Type 3 diabetes an official term? No. The Mayo Clinic, however, already refers to Alzheimer's as "Type 3 Diabetes."

Calling Alzheimer's disease T3DM has gained significant traction.

This suggests that insulin resistance and dysfunction in the brain — similar to what occurs in Type 2 diabetes mellitus (T2DM) — play a crucial role in the development of Alzheimer's.

WHY ARE AD AND IR CALLED A 'VICIOUS CYCLE'?

Alzheimer's disease is a cascade of cognitive decline, while insulin resistance is also known as the "silent killer". The two often coexist, creating what numerous researchers now call a "vicious cycle".

Studies have shown that individuals with AD often exhibit insulin resistance in their brains, similar to the insulin resistance seen in Type 2 diabetes. This suggests a potential link between the two conditions.

These two conditions mutually exacerbate each other.

For example, insulin resistance can contribute to the formation of amyloid-beta plaques and tau tangles, hallmarks of AD. Conversely, AD can impair insulin signalling in the brain, further worsening cognitive function.

Both AD and T2DM are associated with chronic inflammation in the brain and body. This suggests that shared inflammatory pathways may contribute to the development of both conditions.

HOW MANY PEOPLE ARE AFFECTED BY AD?

AD is a global issue. In 2023, more than 55 million people had dementia worldwide, over 60 per cent of whom live in low-and middle-income countries, according to the WHO.

The statistics are alarming: In the US, the number of Alzheimer's patients jumped from 4.5 million in 2010 to over 6 million today, marking a nearly 50 per cent increase in just over a decade.

WHAT TRIGGERS INSULIN RESISTANCE?

Insulin resistance is a metabolic problem, primarily caused and worsened by modern diets — high in refined sugars and processed foods.

Sharp spikes in blood sugar cause an overproduction of insulin, leading to insulin resistance. Think of insulin as a key that unlocks the cell to let glucose in.

The more resistant cells are to insulin, the harder it becomes to deliver energy to them, particularly in the brain.

Over time, this leads to significant cognitive decline, and the onset of Alzheimer's. An increasing number of experts argue that Alzheimer's, like T2DM, stems from insulin resistance.

Dr Mike Hansen highlights how the brain's ability to process insulin is compromised with constant fructose attack. Dr Hansen, who specialises in internal medicine, critical care medicine and pulmonary disease, has dedicated over 15 years of his life to understanding how to prevent, diagnose, and treat diseases that affect adults.

A leading campaigner for maintaining health and fitness, Dr Hansen explains: "Whether you consume sucrose or high fructose corn syrup, it goes into your intestines, gets absorbed, and is delivered to the liver. Fructose directly impacts the brain."

BRAIN 'CARAMELISATION'

"If you thought uric acid was bad, fructose alters brain metabolism even more. It affects astrocytes, the cells that nourish neurons, through glycation and oxidative stress. Glycation occurs when sugar molecules combine with amino acids in proteins, forming advanced glycation end products (AGEs).

"This process is also known as the Maillard reaction or 'caramelisation'. Both glucose and fructose cause glycation, but fructose does it seven times faster. Essentially, your brain becomes 'caramelised' if
you consume too much fructose.

"Fructose generates 100 times more oxygen radicals compared to glucose, increasing oxidative stress. This stress makes the mitochondria sick and eventually dysfunctional. As mitochondria fail, cells start to die."

He explained further: "To make matters worse, fructose interferes with two key trophic growth factors: leptin and brain-derived neurotrophic factor (BDNF), which help the brain develop and form new connections.

"Fructose induces insulin resistance and high insulin levels, impairing leptin's function, leading to cognitive deficits. Beta-hydroxybutyrate, a ketone produced through exercise, intermittent fasting, or a ketogenic diet, increases BDNF activity, helping to form new connections in the hippocampus.

"The hippocampus is the memory
centre of the brain and the first part to deteriorate in Alzheimer's disease. Fructose impairs BDNF activity, making it even more harmful."

ARE THERE NEW DRUGS FOR ALZHEIMER'S?

Despite the massive expenditure on AD, no new drugs have proven effective at stemming the tide of Alzheimer's.

Of the 2,000-plus drug trials conducted through 2019, none succeeded. Many experts argue that treatments aimed at managing amyloid plaques and neurofibrillary tangles are addressing symptoms rather than addressing the underlying cause — which is poor glucose metabolism in the brain.

An increasing number of experts, however, now think that the standard approach is missing the root cause of Alzheimer's disease altogether.

HOW MUCH DOES IT COST TO TREAT ALZHEIMER'S?

The financial impact of AD is staggering. The cost to care for Alzheimer's patients in the US grew from $172 billion in 2010 to $241 billion in 2020. By 2050, it's expected to surpass $1 trillion.

WHAT STUDIES SHOW THE AD-IR CONNECTION?

Experts have found that the hippocampus is especially vulnerable to insulin resistance, a key feature of ageing and AD.

In 1978, a landmark study by Havrankova J., published in Nature,

showed that insulin receptors are widespread in the central nervous system of rats. Later, in 1982, a study in Neuroscience Letters by Sara V.R. found that receptors for insulin-like growth factors (IGF-1 and IGF-2) and insulin are present throughout the adult human brain.

In 1997, Frey WH II received a US patent for a method to deliver neurologic agents to the brain noninvasively. This intranasal technique bypasses the blood-brain barrier to deliver therapeutic proteins, growth factors, and hormones, including insulin, to treat neurodegenerative diseases like AD.

In 2012, a study by Suzanne Craft, 'The Role of Insulin Resistance in the Pathogenesis of Alzheimer's Disease: Mechanisms and Therapeutic Implications', published in the journal Experimental Gerontology
connected insulin resistance to AD, suggesting that impaired insulin signalling in the brain can lead to cognitive decline and neurodegeneration.

Research published in 2016 in the Journal of Alzheimer's Disease
showed that IR exacerbates the development of amyloid plaques, further advancing the understanding of Alzheimer's.

What are the earliest signs of AD? Most neuroscientists recognise that one of the earliest detectable traits of Alzheimer's disease is when the hippocampus function and structure are compromised.