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There may be up too one hundred different types of dementias, some that we know the causes and some that we do not. To date, we still do not know the exact cause of Alzheimer's disease and this has been one of the main hurdles to treatment and prevention. I am asked all of the time by patients “but why is this happening” and I am often left feeling defeated as a provider when all I can say is “I dont know”.

Below is an article explaining the latest findings as well as why it has been so hard to develop medications to address Alzheimer's disease based dementias as well as Lewy Body and Frontal Temporal Dementias. They are now thought to work similar to Multiple Sclerosis (MS) and Lou Gehrig’s disease (ALS) in that the body’s own autoimmune response may be causing them.

We do know that the vast majority of cases of dementia are related to blood flow issues in the brain. Collectively these are call Vascular Dementia (VD). We know the risk factors for VD are; high blood pressure, high blood sugar, smoking, sleep apnea, high cholesterol and cardiac arrhythmias. Many if not all of these risk facts were life style choices not genetic factors (cardiac arrhythmia and an high cholesterol may be the exception)

The Cell That Might Trigger Alzheimer's Disease

Bret Stetka, MD

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It all started with genetic data.

A gene here, a gene there.

Eventually the story became clearer: If scientists are to one day find a cure for Alzheimer's disease, they should look to the immune system.
Over the past couple decades, researchers have identified numerous genes involved in various immune system functions that may also contribute to Alzheimer's.

Some of the prime suspects are genes that control immune cells called microglia, now the focus of intense research in developing new Alzheimer's drugs.

Microglia are amoeba-like cells that scour the brain for injuries and invaders. They help clear dead or impaired brain cells and literally gobble up invading microbes. Without them, we'd be in trouble. In a normal brain, a protein called beta-amyloid is cleared away through our lymphatic system by microglia as molecular junk.
But sometimes it builds up. Certain gene mutations are one culprit in this toxic accumulation. Traumatic brain injury is another, and, perhaps, impaired microglial function.

One thing everyone agrees on is that in people with Alzheimer's, too much amyloid accumulates between their brain cells and in the vessels that supply the brain with blood. Once amyloid begins to clog networks of neurons, it triggers the accumulation of another protein, called tau, inside of these brain cells. The presence of tau sends microglia and other immune mechanisms into overdrive, resulting in the inflammatory immune response that many experts believe ultimately saps brain vitality in Alzheimer's.

To date, nearly a dozen genes involved in immune and microglial function have been tied to Alzheimer’s. The first was CD33, identified in 2008.

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