Alzheimer's and Aftins: Understanding the Role of Mitochondria in Alzheimer's Disease

Gilmore, Caleb (School: Shawnee Mission East)

Alzheimer’s Disease (AD) is a disease that causes extreme memory loss in people and can severely affect their day-to-day life. AB is a molecule that accumulates in the brains of AD patients and is a characteristic marker of AD. The Amyloid Cascade Hypothesis states that this AB is what causes AD while the Mitochondrial Cascade Hypothesis states that the mitochondria causes AD. The reason for AB being produced and the extent of the mitochondria’s role in its production is not yet fully understood. There are drugs called Aftins that induce the production of AB and can be used to interrogate the mechanism for AB production. Previous studies have shown that Aftins do affect the mitochondria by increasing membrane potential and increasing Complex IV usage, but whether or not this causes the increase in AB production is not yet known. We believe that Aftins affect the mitochondria and that this is what causes an increase in AB. Inhibiting Complex IV and observing the way this changes AB production and Aftins’ effect on the mitochondria will help us in understanding how Aftins affect the mitochondria and in turn help us better understand why AB is produced. It was found that when Complex IV was inhibited, AB production and membrane potential decreased. This shows that Aftins affecting the mitochondria is what is causing an increase in AB production. This provides evidence for the idea that the mitochondria is responsible for AB production and helps to explain why AB is produced.

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