Vitamin C deficiency and mitochondrial dysfunction in Alzheimer's disease

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Early clinical features of sporadic Alzheimer's disease include alterations in mitochondrial function that appear prior to classical features. Mitochondrial dysfunction increases the production of reactive oxygen species (ROS), which then drive the production of Amyloid beta, creating a vicious cycle accelerating Alzheimer's pathology. Vitamin C is an effective antioxidant and thus, could be protective against disease pathology.

Fiona Harrison, Ph.D., and colleagues used mouse models to examine the effects of Alzheimer's-linked mutations and vitamin C deficiency on mitochondrial function.

Vitamin C deficiency led to diminished mitochondrial respiration and increased ROS, while mitochondria from the mouse model of Alzheimer's displayed increased respiration compared to wild type controls. The results suggested that both vitamin C deficiency and the presence of amyloid contribute to mitochondrial dysfunction but via differing pathways.

These findings, published in *Free Radical Biology and Medicine*, suggest that vitamin C deficiency could contribute to the pathogenesis of Alzheimer's through altered mitochondrial function and that avoiding deficiency through diet and supplementation could protect against disease onset.

More information: Shilpy Dixit et al. Mitochondrial dysfunction in the APP/PSEN1 mouse model of Alzheimer's disease and a novel protective role for ascorbate, *Free Radical Biology and Medicine* (2017). DOI: 10.1016/j.freeradbiomed.2017.08.021

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