

Metabolic Restoration via NAD⁺ Augmentation Reverses Synaptic Dysfunction and Cognitive Decline in an Experimental Model of Alzheimer's Disease

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TAKE HOME MESSAGE

NAD⁺ augmentation restores neuronal metabolism, improves cognition, preserves synaptic integrity, and reduces Alzheimer's pathology in an experimental mouse model.

BACKGROUND

Amyloid- β buildup, tau pathology, and synaptic dysfunction are the primary contributors of the gradual cognitive deterioration associated with Alzheimer's disease (AD). There is growing indication that poor neuronal energy utilization plays a major role in the development of the disease. It is unknown if existing AD pathology may be reversed by restoring metabolic balance.

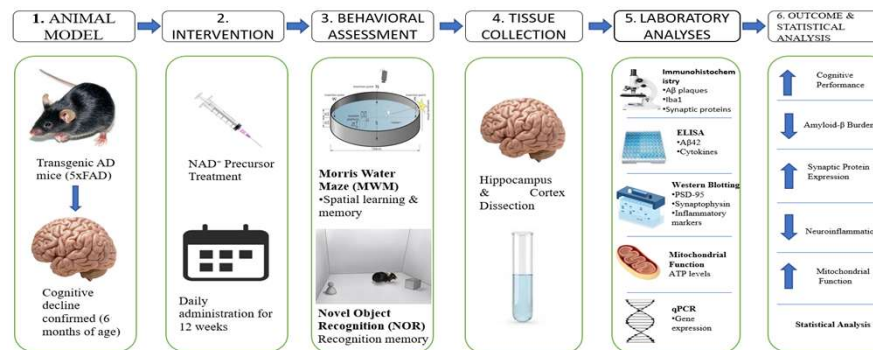
CONCLUSION

These results show that in an animal model, focusing on neuronal energy utilization may restore important clinical and cognitive aspects of Alzheimer's disease. In addition to amyloid-centric techniques, metabolic recovery through NAD⁺ amplification is a potentially therapeutic strategy that facilitates additional scientific investigation utilizing clinical investigations guided by biomarkers.

METHOD

The effects of NAD⁺ augmentation were assessed using a well-established transgenic mouse model of AD. For twelve weeks after the first sign of cognitive decline, animals were given a pharmaceutical NAD precursor. The Morris water maze and tests of novel item recognition were used to evaluate cognitive capacity. Immunohistochemistry, ELISA, and molecular tests were used to examine neural integrity, amyloid load, mitochondrial activity, and neuroinflammation.

METHODOLOGY OVERVIEW



RESULT

When compared to uncontrolled AD subjects, NAD⁺ augmentation dramatically enhanced spatial learning and memory ($p < 0.01$). Treatment decreased the amount of amyloid- β plaque for around 35%, decreased activity of microglial cells, and restored the expression of proteins found in synaptic cells (PSD-95, synaptophysin). Revival of metabolic and synaptic indicators was more significantly associated with cognitive rehabilitation than amyloid decrease alone.

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