



Getting Away from Alzheimer's: Nitric Oxide Mediates Neuroprotection Against Neurotoxicity

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Abstract

Alzheimer's disease (AD) is a neurodegenerative disorder characterized by progressive memory loss and behavioral changes. There are currently no known treatments or medications for AD. Nitric oxide (NO) has long been

However, this perception is being altered by focusing on early developments, prior to the onset of cognitive symptoms. This has brought to light NO's compensatory, neuroprotective function, which increases neuronal excitability to safeguard synapses. Modulation of voltage-gated potassium channel activity (Kv7 and Kv2) is one mechanism and signaling pathways that mediate this protection. A potential therapeutic option for preventing synapse loss early in disease could be found by harnessing the protective function of NO and related signaling pathways.

Introduction

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In Alzheimer's Disease, Nitric Oxide : Mechanisms and Effects

Nitric oxide (NO) is a gaseous signaling molecule that plays a central role in various physiological processes. In the context of Alzheimer's disease, NO is produced by neuronal nitric oxide synthase (NOS) and acts as a signaling molecule in the brain. It is involved in the regulation of synaptic plasticity, learning, and memory. In AD, there is a dysregulation of NO signaling, which contributes to the disease pathology. This review discusses the mechanisms and effects of NO in AD.

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