Is VEGF a key target of cotinine and other potential therapies against Alzheimer disease?

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Background: The vascular endothelial growth factor (VEGF) is a neuroprotective cytokine that promotes neurogenesis and angiogenesis in the brain. In animal models, it has been shown that environmental enrichment and exercise, two non-pharmacological interventions that are beneficial decreasing the progression of Alzheimer disease (AD) and depressive-like behavior, enhance hippocampal VEGF expression and neurogenesis. Furthermore, the stimulation of VEGF expression promotes neurotransmission and synaptic plasticity processes such as neurogenesis. It is thought that these VEGF actions in the brain, may underly its beneficial therapeutic effects against psychiatric and other neurological conditions. Conclusion: In this review, evidence linking VEGF deficit with the development of AD as well as the potential role of VEGF signaling as a therapeutic target for cotinine and other interventions in neurodegenerative conditions are discussed. © 2017 Bentham Science Publishers.

Alzheimer disease

Amyotrophic lateral sclerosis

Angiogenesis

Cotinine

Dementia

Depression

Neurodegeneration

Nicotinic receptors

Tobacco

VEGF

cotinine

neuroprotective agent

vasculotropin A

Alzheimer disease

animal

human

metabolism

Alzheimer Disease

Animals

Cotinine

Humans

Neuroprotective Agents

Vascular Endothelial Growth Factor A