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Linking multiple pathogenic pathways in Alzheimer's disease

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#### 1 Abstract

Alzheimer's disease (AD) is a chronic neurodegenerative disorder presenting as progressive cognitive decline with dementia that does not, to this day, benefit from any disease-modifying drug. Multiple etiologic pathways have been explored and demonstrate promising solutions. For example, iron ion chelators, such as deferoxamine, are a potential therapeutic solution around which future studies are being directed. Another promising domain is related to thrombin inhibitors. In this mini-review, a common pathophysiological pathway is suggested for the pathogenesis of AD to prove that all these mechanisms converge onto the same cascade of neuroinflammatory events. This common pathway is initiated by the presence of vascular risk factors that induce brain tissue hypoxia, which leads to endothelial cell activation. However, the ensuing hypoxia stimulates the production and release of reactive oxygen species and pro-inflammatory proteins. Furthermore, the endothelial activation may become excessive and dysfunctional in predisposed individuals, leading to thrombin activation and iron ion deposition/oxidation. The evidence stress that

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