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Mechanism of Reducing Protein Aggregation Burden in Neurodegenerative Diseases

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INTRODUCTION

As environmental pollution increases, so does the incidence of asthma. Although there are no treatments available, montelukast, a cysteinyl leukotriene receptor 1 antagonist, is commonly used to treat the condition in adults and children. Recently, new molecular targets have been identified and the repurposing of montelukast for other therapeutic applications has been proposed, with several clinical trials underway. Proposed uses include control of neuroinflammation, which may be studied in some neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. However, this drug is associated with an increase in reported neuropsychiatric side effects. Moreover, although montelukast has been commercially available, its metabolism is still poorly understood and the mechanisms underlying its neuropsychiatric side effects remain unclear. The role of montelukast as a regulator of leukotriene signaling and codify current knowledge on montelukast metabolism. Focusing on Alzheimer's and Parkinson's diseases, discuss the known toxic effects of montelukast and provide a comprehensive introduction to its repurposing applications.

DESCRIPTION

In particular, neuroinflammation, mediated not only by microglial cells but also by cd8+ t cells, positively contributes to the disease pathology. Consequently, leukotriene signaling, and more specifically leukotriene receptors, has been recognized as the potential drug targets to alleviate the pathology of Alzheimer's disease. Treatment increased the number of tmem119+ microglia and downregulated genes associated with alzheimer's disease-associated microglia and the lipid droplet-accumulating microglia, suggesting that montelukast treatment targets the microglial phenotype in disease models compared to vehicle, suggested to adjust. Treatment with montelukast further reduced cd8+ t cell infiltration into the brain parenchyma. Finally, treatment with montelukast led to the improvements in cognitive function. It is noteworthy that treatment with montelukast had a more pronounced effect in women than in men, as women with Alzheimer's disease presented with more severe disease. Because the effects on neuroinflammation, i.e., on h. Microglia and cd8+ t cells, and on cognitive outcomes were dose-dependent, alzheimer's clinical trials used higher doses of montelukast compared to approve asthma doses.

CONCLUSION

Dementia with Lewy bodies accounts for up to 30% of all dementia cases and has a huge unmet medical need as there is no cure. Although the spectrum underlying the pathology is complex and presents challenges for targeted molecular therapy, here hypothesize that leukotrienes are involved in the pathology of the dementia with Lewy bodies, and leukotriene receptor antagonists. The test of hypothesis of the leukotriene blockade by montelukast, an approved anti-asthma drug. It may reduce the condition and restore cognitive function. Expression of 5-lipoxygenase, the rate-limiting enzyme for leukotriene production, was indeed increased in the brains. Treatment of the transgenic mice overexpressing cognitively dysfunctional human α-synuclein with montelukast restored memory. Treatment with the montelukast modulated Beclin-1 expression, a marker of autophagy, in transgenic mice and reduced human α-cinclein load. Reducing protein aggregation burden in the neurodegenerative diseases may represent a new mechanism of action for montelukast. Furthermore, this study indicates that the leukotriene signaling is a potential drug target for dementia with Lewy bodies, indicating that montelukast may be a promising drug candidate in the development of future treatments for dementia with the Lewy bodies.

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