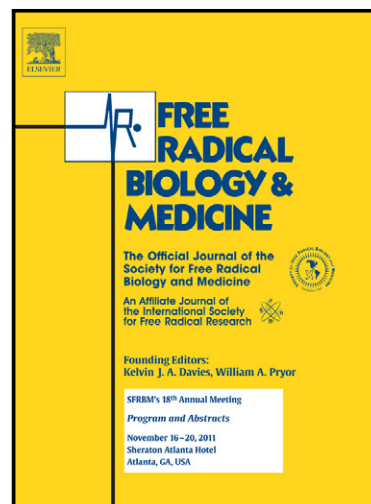


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Antisense directed against *PS-1* gene decreases brain oxidative markers in aged senescence accelerated mice (SAMP8) and reverses learning and memory impairment: a proteomics study

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Abstract

Amyloid β -peptide ($A\beta$) plays a central role in pathophysiology of Alzheimer's disease (AD) through the induction of oxidative stress. This peptide is produced by proteolytic cleavage of amyloid precursor protein (APP) by the action of β - and γ -secretases. Previous studies demonstrated that reduction of $A\beta$, using an antisense oligonucleotide (AO) directed against the $A\beta$ region of APP, reduced oxidative stress-mediated damage and prevented or reverted cognitive deficits in senescence-accelerated prone mice (SAMP8), a useful animal model to investigate the events related to $A\beta$ pathology and possibly to the early phase of AD.

In the current study, aged SAMP8 were treated by AO directed against PS-1, a component of the γ -secretase complex, and tested for learning and memory in T-maze foot shock avoidance and novel

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