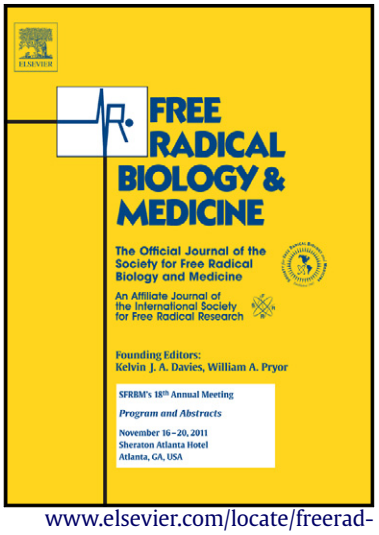


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# Antisense directed against *PS-1* gene decreases brain oxidative markers in aged senescence accelerated mice (SAMP8) and reverses learning and memory impairment: a proteomics study

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## Abstract

Amyloid β-peptide (Aβ) 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 Aβ, using an antisense oligonucleotide (AO) directed against the Aβ 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 Aβ 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