# Alzheimer's disease-like alterations in peripheral cells from presenilin-1 transgenic mice

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Many cases of early-onset inherited Alzheimer's disease (AD) are caused by mutations in the presenilin-1 (PS1) gene. Expression of PS1 mutations in cell culture systems and in primary neurons from transgenic mice increases their vulnerability to cell death. Interestingly, enhanced vulnerability to cell death has also been demonstrated for peripheral lymphocytes from AD patients. We now report that lymphocytes from PS1 mutant transgenic mice show a similar hypersensitivity to cell death as do peripheral cells from AD patients and several cell culture systems expressing PS1 mutations. The cell death-enhancing action of mutant PS1 was associated with increased production of reactive oxygen species and altered calcium regulation, but not with changes of mitochondrial cytochrome c. Our study further emphasizes the pathogenic role of mutant PS1 and may provide the fundamental basis for new efforts to close the gap between studies using neuronal cell lines transfected with mutant PS1, neurons from transgenic animals, and peripheral cells from AD patients.

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