Singlet oxygen is protective against β-amyloid-induced neurotoxicity

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The main component of the senile plaques in Alzheimer's Disease is shown to be neurotoxic in oligomeric form. The mechanism of β-amyloid toxicity includes abnormal calcium signaling, induction of oxidative stress and mitochondrial dysfunction induced by oxidation of DNA and activation of the DNA repair enzyme poly(ADP-ribose)-polymerase (PARP), which consumes nicotinamide adenine dinucleotide that reduces substrate availability [1,2]. A 1267 nm laser shown to be able to generate singlet oxygen in cells and tissues and activate ATP production. We studied the effect of laser-induced singlet oxygen on β-amyloid toxicity in primary co-culture neurons and astrocytes.

We have found that laser-induced singlet oxygen reduced the effect of β-amyloid on NADH depletion, decreasedmitochondrial membrane potential and protected cell agains cell death induced by full peptide βA-1-42 or short βA25-25. This protective effect could be due to the reduction of the oligomerization of βA by singlet oxygen which we found on the full peptide 1-42.

Thus, laser-induced singlet oxygen is protective against βA-induced toxicity.

This work was supported by Grant No. 075-15-2024-621.of the Government of the Russian Federation (cell study) and by RSF grant No. 22-75-10088 (1267 nm system development).

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