Alzheimer’s disease is a widespread neurodegenerative pathology characterized by pronounced dementia and loss of cognitive functions and by development of extended degenerative impairments in the brain in its later stages [1]. The β-amyloid peptide (Abeta) is thought to be an essential factor in Alzheimer’s disease pathogenesis. Submicromolar concentrations of this peptide are known to impair synaptic transmission in glutamate synapses [2], whereas its micromolar concentrations cause apoptotic-type neurodegeneration [3]. Mechanisms of toxic effects of Abeta are not clear in detail. However, some data suggest that a mitochondrial toxicity of Abeta is caused in particular by stimulation of generation of reactive oxygen species (ROS) in mitochondria [4-9]. It was shown that Abeta, using the protein import system, enters the mitochondrial matrix [10] where it interacts with key mitochondrial enzymes including components controlling appearance of the nonspecific permeability of the inner mitochondrial membrane [11]. The excessive generation of mitochondrial ROS caused by Abeta with possible burst-like time course [12] could be crucial for the initiation by this peptide of synaptic dysfunction and memory impairment in Alzheimer’s disease. It was suggested that at least some impairments could be prevented by mitochondria-targeted antioxidants [13], and the present study has confirmed this suggestion using a model of synaptic changes associated with learning and memory (long-term potentiation of synaptic transmission in hippocampus).

**MATERIALS AND METHODS**

The experiments were performed using hippocampal slices from young male Wistar rats (body weight 80-
RESULTS

In our experiments, the standard HFS (100 Hz, 1 sec) of Shaffer’s collaterals caused LTP of PS in the CA1 hippocampal field resulting 30 min after tetanization in an increase in the PS amplitude to 145.9 ± 7.8% (n = 5) in the control rats (figure, panel (a)). In hippocampal slices from rats that received 1 µmol SkQR1 per kg weight 24 h before isolation, the LTP amplitude was slightly higher than in the control, i.e., 175.4 ± 17.1% (n = 6) (figure, panels (b) and (d)).

Concentrated aqueous solutions of Abeta (Sigma-Aldrich, USA) were stored as frozen aliquots. The stored Abeta solution was diluted with the perfusion medium immediately before use. SkQR1 (1 µmol/kg) was intraperitoneally injected into rats 24 h before preparing the slices.

Statistical analysis of mean values and mean errors (M ± m) were done using the nonparametric Mann–Whitney test and Student’s test.

The experimental protocols on treatment of animals were performed according to 86/609/EEC Regulations of the European Society Council for the use of animals in experiments and approved by the Moscow State University Ethics Commission.

Alzheimer’s disease is a common age-related disease that seems to be mediated by mitochondrial ROS. This disease is characterized by a progressing loss of memory and other cognitive functions. The production of Abeta from its protein precursor is a key event in the development of this disease [4]. The accumulation of Abeta in cells leads to synaptic dysfunction and memory impairments [2, 20, 21]. The Abeta-triggered pathological cascade in the initial phase may be a result of mitochondrial oxidative stress that in terminal phase gives rise to expres-