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Treadmill exercise reduces Aβ deposition in the hippocampus of APP/PS1 mice by activating AMPK

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Objective To investigate the molecular mechanism of AMPK-mediated motion reduction of Aβ deposition in the hippocampus of APP/PS1 mice.
Methods The 3 months of C57BL/6 48 APP/PS1 mice and 48 wildtype mice were randomly divided into APP/PS1 quiet control group (ADC, n=24) and exercise group (ADE, n=24), wildtype quiet control group (the WTC, n=24) and exercise group (WTE, n=24).ADE and WTE groups performed 12 weeks of treadmill exercise, while ADC and WTC groups were placed in a quiet running platform environment. The contents of ATP, AMP in hippocampus were tested by ELISA, AMPK and BACE1 mRNA in hippocampus were tested by RT-qPCR, Protein expression of AMPK, Sirt1, PPARγ, PGC1α, BACE1, Aβ in hippocampus were tested by Western Blot, the hippocampus SPs were tested by immunohistochemical experiments.
Results 12 weeks treadmill exercise can improve the AMP、ATP content, and the AMP/ATP ratio (p<0.05) in APP/PS1 mice hippocampus, up-regulation the mRNA and protein phosphorylation of AMPK(p<0.05),the protein expression of Sirt1、PPARγ and PGC1α(p<0.05)in APP/PS1 mice hippocampus, down-regulation the BACE1mRNA and protein expression levels (p<0.05), Aβ protein and SPs level (p<0.01) in APP/PS1 mice hippocampus.
Conclusions The molecular mechanism of decrement Aβ deposition in APP/PS1 mice hippocampus mediated by 12 weeks of treadmill exercise may be related to activation of AMPK and the improvement of hippocampus energy metabolism by exercise, and then regulatiing the AMPK-BACE1 pathway.