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THE EXPRESSION AND PHOSPHORYLATION OF AMPK INDUCED BY CHRONIC RESTRAINT STRESS IN MOUSE HYPOTHALAMUS

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Abstract

Adenosine monophosphate-activated protein kinase (AMPK) mediates the energy homeostasis and neuroprotection. However, the role of AMPK in chronic stress condition has not been well demonstrated. Therefore, in the present study, we investigated the expression and phosphorylation of AMPK in the hypothalamus of ICR mice subjected to the acute restraint stress (ARS) and chronic restraint stress (CRS). We found that CRS caused an increase of AMPKα1, β2, and γ3 but not, β1 and β2 mRNA levels in hypothalamus. However, AMPKα2 mRNA expression was decreased by ARS and then RS time-dependently recovered to the normal level. In Western blot analysis, CRS increased phosphorylation of AMPKα and β1 proteins. In addition, we observed an increase of phosphorylated AMPKα2 immunoreactivity in AV, VA, VPL, VPM, and LGP regions of thalamus and in Re, Pa, Arc, and Me regions of hypothalamus following ARS and CRS. Furthermore, we investigated the possible involvement of corticosterone in the regulation of phosphorylated AMPK in adrenalectomized (ADX) mice following CRS. Phosphorylation of AMPKα was further increased in ADX mice subjected to CRS compared to the normal mice group. Although further studies are necessary to reveal the exact mechanism, our results suggest that corticosterone may play an important role for the phosphorylation of AMPK in stress model. Furthermore, AMPK may be responsible for the development of chronic stress and depression.