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综述

学习与记忆机制研究进展

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摘要
学习与记忆是动物最具特色的高级神经活动之一，长时程增强(LTP)被认为是与学习记忆有关的神经元可塑性的理想模型，其分子机制涉及一个信号转导级联反应——谷氨酸释放、N-甲基-D-天(门)冬氨酸(N-Methyl-D-aspartic acid,NMDA)谷氨酸受体激活、Ca2+通道和Ca2+/钙调蛋白依赖性蛋白激酶（Ca2+/calmodulin-dependent protein kinases,CaM激酶）Ⅱ、Ⅳ和丝裂原蛋白激酶（mitogen-activated protein kinase,MAPK）激活。最后，CaM激酶Ⅱ使α-氨基羟甲基恶唑丙酸(α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid,AMPA)谷氨酸受体磷酸化而激活，引起突触后神经元Ca2+ 内流增加。CaM激酶Ⅳ和MAPK通过刺激基因表达促使环磷腺苷反应元件结合蛋白(cyclic AMP response element binding protein, CREB)磷酸化水平升高，c-fos表达增加。

关键词 长时程增强；学习记忆；受体；蛋白激酶；基因表达

分类号

Progression of mechanism in learning and memory

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Abstract
Learning and memory is one of the most significant characteristic of nerve activities, which is always being paid much attention.Long-term potentiation (LTP) was thought to be a model for neuronal plasticity such as learning and memory, its molecular mechanism was found to involve a signal transduction cascade that includes release of glutamate, activation of the NMDA glutamate receptors, Ca2+entry, and activations of Ca2+/calmodulin-dependent protein kinases (CaM kinases) II and IV and mitogen-activated protein kinase (MAPK). Consequently, α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid (AMPA) glutamate receptors were activated by phosphorylation by CaM kinase II, resulting in an increase in Ca2+ entry into postsynaptic neurons. Furthermore, the activation of CaM kinase IV and MAPK increased phosphorylation of cyclic AMP response element binding protein (CREB) and the expression of c-fos by stimulation of gene expression.

Key words long-term potentiation learning and memory receptor protein kinase gene expression

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