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生命科学

# aCaMKII在前脑过 量表达损伤小鼠灵活 性学习能力

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摘要 将3月龄实验小鼠分为 aCaMKII F89G转基因组和同 窝野生对照组,进行疲劳转棒实 验和Morris水迷宫实验测试.结 果显示,转基因组小鼠的体力和 运动协调能力与对照组相比无显 著的差异; 在Morris水迷宫实验 的可视平台测试中, 转基因鼠的 视觉和求生的动机表现正常; 在 定位航行训练和第一次空间探索 测试中, 两组鼠在训练时逃避潜 伏期及测试中在目标象限探索时 间无统计学差异; 但是在反向定 位空间学习阶段, 转基因组在第 1、三天逃避潜伏期和距离明显 长于同窝对照组(P<0.05). 由此认为, aCaMKII在前脑过量 表达对小鼠的灵活性学习有损伤

关键词 钙/钙调蛋白依赖性蛋

作用,推测这种损伤有可能由前脑

自激酶Ⅱ;疲劳转棒实验; Morris水迷宫实验; LTD; 空间再学习

分类号 **Q6** 

LTD的缺陷造成的.

# Forebrain overexpression of aCaMKII disrupts behavioral flexibility

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### Abstract

The aCaMKII F89G transgenic mice and their littermate controls were subjected to the rotarod test and Morris water maze test. There was no significant difference in motor coordination and ability between these two groups in the rotarod test. In the visible platform test, transgenic mice showed the normal perception, motivation and motor ability. In addition, transgenic mice performed normally in learning and memory in both place navigation training and the 1st spatial probe test. However, compared with wild type mice, transgenic mice spent significantly more time and swam longer distance to reach hidden platform in the 2nd and 3rd day of spatial reversal learning. These results indicate that aCaMKII overexpression in the forebrain can impair spatial reversal learning. The mechanism of the performance deficit may be relevant to disrupted or abolished LTD in the forebrain.

Key words CaMKII
rotarod test Morris
water maze LTD
spatial reversal learning

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