

论著

超低分子肝素对原代培养大鼠大脑皮质神经元化学诱导损伤的保护作用

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摘要 目的 探讨超低分子肝素 (ULMWH) 对不同化学诱导损伤大鼠皮质神经元的保护作用。方法 谷氨酸、叠氮钠、KCl 和咖啡因诱导损伤原代培养的大鼠大脑皮质神经元, 观察神经元存活率、培养液中乳酸脱氢酶 (LDH) 漏出量及细胞内游离钙离子浓度 ($[Ca^{2+}]_i$)。结果 ULMWH (0.01~1.0 mg·L⁻¹) 预处理 24 h 可显著提高谷氨酸损伤神经元的存活率, 降低细胞 LDH 的漏出量和 $[Ca^{2+}]_i$, 高浓度 (1.0 mg·L⁻¹) 时对叠氮钠引起的神经元损伤也有一定的保护作用, 但对咖啡因和 KCl 所致的神经元损伤无影响。结论 ULMWH 对谷氨酸和叠氮钠所致大鼠大脑皮质神经元损伤有一定的保护作用, 可能与其抑制 $[Ca^{2+}]_i$ 升高有关; 但不能对抗 KCl 和咖啡因所致的皮质神经元损伤。

关键词 [超低分子肝素](#) [神经保护药](#) [大脑皮质](#) [神经元](#) [钙, 细胞内](#) [谷氨酸](#) [叠氮钠](#)

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Protective effect of ultra low molecular weight heparin on chemically-induced injury in primary cultured rat cortical neurons

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Abstract

AIM To investigate the effect of ultra low molecular weight heparin (ULMWH) on chemically-induced injury to primary cultured rat cortical neurons and related mechanisms. **METHODS** Cortical neurons of fetal rats cultured *in vitro* were treated with glutamate (Glu), sodium azide, KCl and caffeine, respectively. The viability of cortical neurons, the efflux of lactate dehydrogenase (LDH), and the concentration of intracellular free Ca²⁺ ($[Ca^{2+}]_i$) was measured, respectively.

RESULTS Pretreatment with ULMWH (0.01-1.0 mg·L⁻¹) increased the cell viability, decreased the efflux of LDH and $[Ca^{2+}]_i$ induced by Glu, and so for sodium azide-induced injury in higher concentration (1.0 mg·L⁻¹), while had no effect on these changes induced by KCl or caffeine. **CONCLUSION** ULMWH has a certain protective effect on cortical neurons damaged by Glu or sodium azide, which maybe related to its inhibitory effect on increment of $[Ca^{2+}]_i$. However, it has no protective effect on cortical neurons damaged by KCl or caffeine.

Key words

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