[1]李扬,罗勇,秦文熠,等.IKK-NBD多肽通过调节c-rel对抗局灶脑缺血再灌注大鼠大脑皮质炎症反应[J].第三军医大学学报,2013,35(16):1721-1725.

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IKK-NBD多肽通过调节c-rel对抗局灶脑缺血皮质炎症反应(PDF)

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Title: IKK-NBD peptides exert anti-inflammation effect in rats after focal cerebral cortex ischemia/reperfusion *via* regulating c-rel

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关键词: 局灶脑缺血再灌注; 炎症因子; IKK-NBD多肽; c-rel; IκBα

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摘要: 目的 探讨局灶脑缺血再灌注大鼠不同时间点大脑皮质炎症反应及IKK-NBD多肽干预对抗炎症反应的机制。 方法 采用线栓法制备Spragne-Dawley（SD）大鼠局灶脑缺血再灌注模型。将大鼠分为假手术组、模型组及IKK-NBD组，再下设再灌注1 d和7 d 2个时相点。IKK-NBD组通过侧脑室定位定量注入4 μL IKK-NBD。对各组进行Zea-Longa神经功能评分，HE染色观察病理变化，Western blot与荧光定量PT-PCR分别检测缺血皮质区c-rel蛋白和IκappaBα（IκBα） mRNA表达，ELISA检测缺血区炎症因子IL-1β和IL-10的含量。 结果 ①再灌注1、7 d时，IKK-NBD对模型大鼠行为学改善明显；②再灌注1、7 d时，模型组病理改变较IKK-NBD组明显；③胞核c-rel蛋白：再灌注1、7 d时，模型组均高于假手术组（$P<0.05$）；再灌注1 d时，IKK-NBD组高于假手术组（$P<0.05$）；再灌注1、7 d时，IKK-NBD组低于模型组（$P<0.05$，$P<0.01$）；随着时间延长模型组和IKK-NBD组均降低

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（P<0.01）；④IκBα mRNA：再灌注1、7 d时，模型组和IKK-NBD组均高于假手术组（P<0.05），7 d较1 d回落；再灌注1 d时，IKK-NBD组高于模型组（P<0.01）；⑤IL-1β：再灌注1、7 d时，模型组与IKK-NBD组均明显高于假手术组（P<0.01），浓度随时间降低；再灌注1 d时，IKK-NBD 组较模型组减少（P<0.01）；IL-10：再灌注1、7 d时，模型组与IKK-NBD组均明显高于假手术组（P<0.01），浓度随时间降低；再灌注1、7 d时，IKK-NBD组均高于模型组（P<0.01）。 结论 IKK-NBD在局灶脑缺血再灌注损伤后早期可以通过上调IκBα限制c-rel入核，下调IL-1β，上调IL-10而减轻再灌注后炎症损伤；而脑缺血再灌注恢复期，少量c-rel可上调IL-10起到抗炎作用。

Abstract: Objective To investigate the cerebral cortex inflammation of SD rats model of focal cerebral ischemia/reperfusion and the underlying mechanism of IKK-NBD peptides' anti-inflammation effect. Methods The MCAO/R SD rats were randomly assigned to 3 groups, that is, sham operation group, model group and IKK-NBD group, and each group was further randomly divided into 2 subgroups with reperfusion of 1 and 7 d after 2 hours' ischemia. IKK-NBD of 4 μL was injected into the right lateral ventricle in 2 h before model infliction. Neurobehavior was evaluated by Zea-Longa score. HE staining was used for pathological observation, Western blotting and quantitative RT-PCR for mRNA and protein expression levels of c-rel and IκappaBα (IκBα), and ELISA for the contents of IL-1β and IL-10 in the ischemic cortex. Results IKK-NBD improved the behavioral recovery and reduced edema and necrosis after 1 and 7 d of reperfusion. The protein expression of c-rel in the cell nucleus was significantly higher in the model group than in the sham operation group (P<0.05), but significantly lower in IKK-NBD group than model group in 1 and 7 d after reperfusion (P<0.05, P<0.01), while, in 1 d after reperfusion, it was significantly higher in IKK-NBD group than the sham operation group (P<0.05). It was continuously decreased in the IKK-NBD group and model group with the elapse of time (P<0.05). Expression level of IκBα mRNA was significantly higher in the model and IKK-NBD groups than the sham operation group in 1 and 7 d after reperfusion (P<0.05), with that in day 7 lower than in day 1, and it was higher in IKK-NBD group than in model group in 1 d after reperfusion (P<0.01). IL-1β and IL-10 were significantly higher in the model and IKK-NBD groups than the sham operation group in 1 and 7 d after reperfusion (P<0.01), and continued to reduce with the time, but in the day 1, IL-1β was lower in the IKK-NBD group than in the model group (P<0.01), and IL-10 was higher in the IKK-NBD group than in the model group for the days 1 and 7 (P<0.01). Conclusion IKK-NBD prevents c-rel against nuclear translocation by up-regulating IκBα in early stage after focal cerebral I/R, and thereby attenuates the

inflammatory damages by down-regulating IL-1β and up-regulating IL-10. However, in later period after focal cerebral I/R, mild expression of c-rel exerts its anti-inflammatory effect by up-regulating IL-10.

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