[1]张礼均,胡荣,李飞,等.瞬时受体电位通道6在大鼠大脑冲击伤后铁代谢中的作用[J].第三军医大学学报,2012,34(23):2353-2356.
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瞬时受体电位通道6在大鼠大脑冲击伤后铁代谢中的

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Title: Role of transient receptor potential channels 6 in iron metabolism in rats after traumatic brain injury

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关键词: 大脑冲击伤; 非血红蛋白铁离子; 瞬时受体电位通道6

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摘要: 目的　探讨瞬时受体电位通道6(transient receptor potential channels 6, TRPC6)在大鼠大脑冲击伤后铁代谢中的作用。　方法　健康成年雄性SD大鼠39只，分为假手术组、致伤组和DFO治疗组（*n*=13）。参照Feeney法致伤，假手术组只开颅，不打击，致伤组和DFO治疗组开颅后，打击部位和打击量完全一致，2 h后予以生理盐水及去铁胺100 mg/kg，间隔12 h给药，持续28 d。于28 d取致伤灶周围脑组织进行脑铁含量检测，采用免疫荧光和Western blot检测方法，分别定性、定量检测瞬时受体电位通道6（TRPC6）在致伤灶周围的表达情况，并采用荧光双标检测TRPC6在神经元上的定位分布情况。　结果　与假手术组比较，致伤组脑组织铁离子显著增加（*P*<0.05），但DFO治疗组并未显著降低其含量(*P*>0.05)；免疫荧光检测结果显示致伤组TRPC6显著上调，DFO治疗组显著下调其表达；Western blot检测结果显示，与假手术组比较，致伤组TRPC6显著上调（*P*<0.05），DFO治疗组显著下调其表达（*P*<0.05）；免疫荧光双标检测TRPC6与NeuN共表达情况，结果显示假手术组仅少量细胞表达TRPC6，且密度低，NeuN染色阳性的细胞胞体形态呈椭圆形，仅部分细胞共表达；致伤组致伤灶周围TRPC6表达明显增强，但却未见NeuN染色阳性的细胞；DFO治疗组可见NeuN染色阳性的细胞数量多，形态欠规则，与TRPC6共表达细胞数量增多。　结论　大鼠TBI后铁离子具有神经毒性作用，能增加致伤灶周围神经元的损

伤，而TRPC6可能在铁离子的神经毒性作用中扮演重要角色。

Abstract: Objective To determine the role of transient receptor potential channels 6 (TRPC6) in iron metabolism after traumatic brain injury (TBI) in rats and investigate the underlying mechanism. Methods Male Sprague-Dawley rats were randomly divided into 3 groups（*n*=13 for each group）, that is, sham-operation group, TBI group and deferoxamine (DFO) treatment group. Modified Feeney’s method was used to establish the rat model of experimental TBI with an impact of a weight of 30 g from 15 cm after craniotomy. The rats of DFO treatment group were treated with DFO (100 mg/kg, i.p., administered 2 h after TBI and then at 12-hour intervals for up to 28 d). Nonhemoglobin iron around the injury site was measured. Immunofluorescence staining was used to detect the expression and location of TRPC6 and NeuN, and Western blotting was also used to detect the expression of TRPC6. Results The volumes of nonhemoglobin brain iron was significantly decreased in injured group and DFO treatment group than sham-operation group ($P<0.05$), but there was no difference between TBI group and DFO treatment group. The expression of TRPC6 was up-regulated significantly after TBI compared with sham-operation group. DFO administration significantly down-regulated the expression of TRPC6. After TBI, there were more neurons positive to TRPC6, but few positive to NeuN around the injury site. However, DFO treatment resulted in more neurons positive to NeuN, which was coexpressed with TRPC6. Conclusion Over-loading of iron exerts neurotoxic effect in rats after TBI, which may deteriorate the damage of neurons around injury site. TRPC6 maybe play an important role in the neurotoxicity of the process.

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