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发育期铅、铝共同暴露与单独铝暴露引起的大鼠海马突触可塑性损伤的差异

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铝近年来被认为是一种神经毒，可以引起动物和人认知和行为的损伤。我们用在位电生理技术研究发现慢性0.2%氯化铝暴露（从出生到成年），结果发现铝损伤了大鼠海马齿状回PS 的LTP和EPSP的LTD，而对EPSP 的LTP和PS的LTD的影响不大。铅是人们公认的神经毒，造成中毒者智力、认知和行为的损伤。铅、铝作为常见的环境毒，在我们周围并存。本文探讨了发育期的铅、铝共同暴露和单独铝暴露所造成的突触可塑性损伤的差异。结果表明，发育期的铅、铝共同暴露明显加重了铝引起的PS 的LTP的损伤，并引起了EPSP 的LTP损伤，而与铝组的EPSP、PS的LTD的差异不明显。

Aluminum and lead postlactional exposure aggravate Aluminum-induced impairment of synaptic plasticity

Aluminum is thought as a neurotoxic agent recently, which induces neurobehavioral and cognitive toxicity in animals and human beings. The neurotoxicity of lead was known from ancient time, which causes intelligence, neurobehavioral and cognitive deficit. The present study is carried out to investigate the effects of chronic 0.2% AlCl3 exposure on synaptic plasticity (LTP and LTD) and the effects of Aluminum and Lead postlactational exposure on Aluminum-induced impairment of synaptic plasticity by in vivo eletrophysiological technique.

2.1 Both of the I/O of EPSP and PS had no significant difference ($p > 0.05$) between control and Al-exposed rats. Which means that chronic 0.2%AlCl3 exposure had no effect on baseline synaptic transmission evoked by single-shock and failed to influence synchronous firing of many dentate granule cells.

2.2 Both of the I/O of EPSP and PS had significant difference ($p < 0.05$) between Al-exposed and Al+Pb-exposed rats. Which means that chronic Al and Pb postlactational exposure affected baseline synaptic transmission evoked by single-shock and attenuated synchronous firing of many dentate granule cells.

2.3 There was no significant change of EPSP LTP between control and Al-exposed (control: 140.6 ± 15.6%, $n = 8$; 134.9 ± 7.4%, $n = 7$, $p > 0.05$). The amplitude of PS LTP was 208.6 ± 24.2% ($n = 8$), in control rats, which was depressed to 169.8 ± 9.5% ($n = 7$) by Al exposure ($p < 0.05$).

2.4 The amplitude of EPSP LTD was 87.2 ± 4.5% ($n = 8$) in control rats, which was depressed to 95.3 ± 16.5% ($n = 7$) by Al exposure ($p < 0.05$).There was no significant change of PS LTD between control and Al-exposed (control: 86.2 ± 3.9%, $n = 8$; 89.0 ± 1.31%, $n = 7$, $p > 0.05$). Chronic 0.2% AlCl3 exposure impaired PS LTP and EPSP LTD.

2.5 There were significant differences of EPSP (Al-exposed: 134.9 ± 7.4%, $n = 7$; Al+Pb-exposed: 123.3 ± 5.8%, $n = 6$, $p < 0.05$)and PS LTP ((Al-exposed: 169.8±9.5%, $n = 7$; Al+Pb-exposed: 129.6 ± 4.0% , $n = 6$, $p < 0.05$) between Al-exposed and Al+Pb-exposed rats. Which indicated that Al and Pb postlactational exposure aggravated Al-induced impairment of PS LTP and caused impairment of EPSP

LTP.

2.6 There were no significant differences of EPSP (Al-exposed: 95.3±16.5%, n = 7; Al+Pb-exposed: 97.4±8.3%, n = 6, p > 0.05)and PS LTD ((Al-exposed: 89.0±1.31%, n = 7; Al+Pb-exposed: 92.1 ± 7.2%, n = 6, p > 0.05) between Al-exposed and Al+Pb-exposed rats. Which indicated that Al and Pb postlactational exposure had no effects on Al-induced impairment of EPSP and PS LTD.

2.7 In order to measure the ability of synaptic plasticity quantitatively, the concept of the range of synaptic plasticity, which was defined as the sum of amplitudes of LTP and LTD (LTP + LTD), was used here. The range of synaptic plasticity of EPSP was 53.4, 36.9, 25.9% in control, Al-exposed and Al + Pb -exposed groups, respectively, and that of PS was 122.4, 80.0, 37.5% in control, Al-exposed and Al+Pb-exposed groups, respectively.. This result indicated that chronic Al exposure reduced the range of synaptic plasticity of both EPSP and PS; Aluminum and lead postlactational exposure aggravate Aluminum-induced impairment of synaptic plasticity.

关键词