Delta-opioid receptor (DOR) activation prolongs respiratory motor output during oxygen-glucose deprivation (OGD) in neonatal rat spinal cord *in vitro*

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ABSTRACT:

Severe reduction of blood flow to the brain results in oxygen-glucose deprivation (OGD) and neuronal damage. DOR activation is neuroprotective during OGD in the cerebellum and cortex, but it is not known whether DOR activation protects spinal motor circuits that contribute to breathing. To address this question, a split-bath in vitro neonatal rat brainstem/spinal cord preparation was used to record spontaneous respiratory motor output from cervical (C4-C5) and thoracic (T4-T7) ventral spinal roots. A plastic barrier at spinal segment C1 allowed oxygenated solution to continuously bathe the brainstem while the solution on the spinal cord was switched from oxygenated to OGD (0 mM glucose, bubbled with 95% nitrogen/5% carbon dioxide) with or without DADLE (1.0 uM, DOR agonist). After OGD solution was applied to the spinal cord (n=7), cervical and thoracic motor output was abolished at 25.8 ± 2.0 and 26.4 ± 1.6 min, respectively. However, when DADLE was applied 10 min prior to and during spinal OGD solution application (n=8), cervical and thoracic motor output was abolished at 41.5 ± 4.9 min ($p = 0.014$) and 57.0 ± 4.2 min ($p = <0.001$), respectively. These data suggest that spinal DOR activation protects both cervical and thoracic respiratory motor output during OGD.

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