

NEONATAL MORPHINE TREATMENT ALTERS GLUTAMATE UPTAKE IN SPINAL CORD OF RATS

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Introduction: infants that receive opioids in ICU develop symptoms of opiate withdrawal, such as antinociceptive tolerance. We previously showed that morphine exposure in early life promotes a hyperalgesic response to noxious events in adult life of rats that was reverted by NMDA antagonist receptor. Thus, it suggests that early morphine exposure can be lead to changes in the glutamatergic system. The aim of this study was to determine if morphine administration in early life, once a day for 7 days, alters glutamate uptake in spinal cord synaptosomes. Materials and Methods: each animal received saline (C) or morphine (5 µg s.c. in the mid-scapular area; M) starting at postnatal day 8 (P8) until P14, once a day. At P30 and P60 animals were killed by decapitation and spinal cords were removed to determination of Na⁺-dependent high-affinity glutamate uptake, as described by Leal et al (2001) (n=3-4/group). The results were expressed as mean + SEM of pmol/min/mg protein. Statistical analysis was performed by Student's t test, and was considered significant if P<0.05. This work was approved by Ethical Committee of HCPA (GPPG 08345). Results: morphine group showed a decreased glutamate uptake in synaptosomes of spinal cord compared to control group at P30 (C=0.3509±0.1244; M=0.0477±0.01347; F_(1,6)=4.746; Student's t test, P<0.05) and at P60 (C=0.04289±0.009; M=0.01345±0.003; F_(1,5)=5.771; Student's t test, P<0.05). Conclusion: this study showed that morphine exposure in early life alters glutamate uptake in spinal cord at medium and long-term after end of treatment. In this way, we can suggest that this glutamatergic changes can contribute to hyperalgesia induced by morphine treatment in neonatal period.