Continuous shock to the rat spinal cord induces an antinociceptive response that is mediated by the mu and kappa opioid receptors.

Research in our laboratory has shown that just 6 minutes of intermittent, uncontrollable tail shock (80 ms shock pulses spaced 0.2-3.8 s apart) has a negative impact on spinal plasticity. Interestingly, administering continuous shock together with intermittent shock blocks the adverse effects of intermittent stimulation on spinal cord plasticity. Intermittent shock has little effect on thermal threshold and has been shown to enhance mechanical reactivity (allodynia), while continuous shock inhibits tail withdrawal from radiant heat. On the basis of these findings, we suggested that intermittent and continuous stimulation induce opponent processes that modulate spinal cord plasticity. Supporting this, we have shown that pre-exposure to intermittent shock significantly reduced the antinociceptive consequence of continuous shock. The present experiments examined which opioid systems were engaged by continuous shock (3, 25-s continuous 1.5 mA tailshocks), and investigated the effect of intermittent shock on a pharmacologically induced antinociception. Results showed that antagonizing either the mu or kappa opioid receptors with CTOP or nor-BNI, blocked the antinociceptive consequences of continuous shock. However, intermittent shock failed to attenuate the antinociception induced by the mu opioid agonist, DAMGO, and the kappa opioid agonist, Dynorphin A. These findings indicate that the antinociceptive consequence of continuous shock is mediated by the activation of the mu and kappa opioid receptors. Exposure to intermittent shock is not sufficient to block the antinociceptive effect of pharmacologically activating the mu and kappa opioid receptors.

Disclosures: D.A. Puga, None; S.N. Washburn, None; J.W. Grau, None.
Support: NS041548 to J. W. G.
NIH grant No. T32-MH65728 to D.A.P.


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