SPINAL MECHANISMS OF ANTINOCICEPTIVE ACTION CAUSED BY DIPHENYL DISELENIDE

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Nociception or the nociceptive sensation results from the activation of specific primary sensory neuron subpopulations that transmit the nociceptive information to the spinal cord from where it is relayed to supra spinal levels. Diphenyl diselenide, a simple diaryl diselenide, exerts biological actions, including antioxidant, hepatoprotective, anti-ulcer and anti-inflammatory and antinociceptive effects. The present study was designed to investigate further the mechanisms involved in the antinociception caused by diphenyl diselenide in behavioral model of pain in mice. Diphenyl diselenide (1-100mg/Kg), given orally, produced significant inhibition of the biting behavior induced by intrathecal (i.t.) injection of glutamate (175 nmol/site) and N-methyl-D-aspartate (NMDA; 450 pmol/site), with mean ID_{50} values of 45.92 (39.74- 60.4) and 55.77 (36.52-77.5) mg/Kg respectively. This compound also reduced the nociceptive response induced by substance P (SP) (135 ng/site, i.t.), interleukin 1β (IL-1β; 1 pg/site), tumor necrosis factor-α (TNF-α; 0.1 pg/site), bradykinin (BK; 0.1 µg/site) and capsaicin (30 ng/site) with mean ID_{50} values of 16.22, 7.06, 6.06, 4.18 and 7.90 mg/Kg, respectively. Together, these results indicate that diphenyl diselenide produces antinociception at spinal sites, with a possible interaction with glutamatergic pathways, more specifically via interaction with NMDA receptors, peptidergic or vanilloid systems.