INVOLVEMENT OF K^+- CHANNELS IN THE ANTINOCICEPTION CAUSED BY DIPHENYL DISELENIDE IN THE FORMALIN TEST

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Diphenyl diselenide, a simple organochalcogenide, possesses antinociceptive and anti-inflammatory activities in mice and rats. The present study investigated the antinociceptive effect caused by diphenyl diselenide in formalin test and also the possible involvement of k^+ channels in its the antinociceptive activity. Diphenyl diselenide injected orally (p.o.) in mice caused antinociception against the first and second phase of formalin test, with mean ID_{50} values of 25.55 (9.52 - 68.58) and 6.45 (1.75 - 23.8) mg/Kg, respectively. This compound also significantly inhibited (43 ± 4%) the mice paw oedema induced by intraplantar injection (i.pl.) of formalin. Moreover, $(PhSe)_2$ (10 mg/Kg), given 5 min after the formalin injection, revealed an significant inhibition (71 ± 6%) in the second phase of the formalin-induced pain, whereas the prophylactic treatment caused more intense inhibition (89 ± 3%). The antinociceptive effect caused by $(PhSe)_2$ (10 mg/Kg, p.o.) was reversed by intratechal (i.t.) injection of several K^+ channels blockers such as apamin and charybdotoxin (large- and small-conductance Ca^{2+}-activated K^+ channel inhibitors, respectively), tetraethylammonium (TEA, non-selective voltage-dependent K^+ channel inhibitor), but not glibenclamide (ATP-sensitive K^+ channel inhibitor). These results suggested the participation K^+ channels on the antinociceptive effect caused by diphenyl diselenide.