BRADYKININ INTO AMYGDALA INDUCES THERMAL HYPERALGESIA IN RATS

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Introduction and objectives: The peptide bradykinin is one of the most potent algogenic substances, but its action in pain transmission in central nervous system remains unclear. The aim of this study, we studied the action of bradykinin into amygdala, a limbic structure highly involved on pain modulation. **Results and conclusions:** Administration of bradykinin (0.025-0.5 nmol/site) into right amygdala of rats promoted a thermal hyperalgesia, verified by a reduction in paw withdrawal latency produced by noxious heat, only in the ipsilateral paw. The hyperalgesic effect of bradykinin (0.25 nmol/site) was not due to an unspecific effect on locomotor activity, visualized on open-field test. The hyperalgesia induced by intra-amygdala injection of bradykinin was abolished by co-administration with a B_2, but not with a B_1 receptor antagonist. This hyperalgesic effect was also inhibited by co-administration of bradykinin (0.25 nmol/site) with the glutamatergic NMDA antagonist MK801 (5 nmol/site), with the cyclooxygenase inhibitor indomethacin (10 nmol/site) or with the glial metabolic inhibitor fluorocitrate (1 nmol/site) into amygdala of the rats. The results showed that intra-amygdalar administration of bradykinin induces pain sensitization through the release of cyclooxygenase products and the activation of NMDA and B_2 receptors present in amygdala's neurones and glia. These findings provide evidence that bradykinin participates of the central pain-modulating circuit. **Acknowledgements:** CAPES, FAPERGS, CNPq. **Key words:** kinin, glutamate, peptide.