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FOOTSHOCK INDUCED ANALGESIA (FSIA) & CLASSICALLY CONDITIONED ANALGESIA (CCA): DIFFERENTIAL ACTIVATION OF OPIATE & NON-OPIATE SYSTEMS. L.R. Watkins*, D.A. Cobelli* & D.J. Mayer, Dept. of Physiology, MCV/VCU, Richmond, Virginia 23298 (U.S.A.)

Footshock produces reliable analgesia in rats; however, opiate mediation of this effect has remained controversial. Using 90 sec shock, we have shown that one critical factor determining the involvement of endogenous opioids is the body region shocked; front paw shock elicits opiate analgesia whereas hind paw shock elicits non-opiate analgesia.

Front paw FSIA involves opioids since it shows cross-tolerance to morphine & is attenuated by 1 mg/kg systemic or 1 μg spinal naloxone. One intriguing aspect of the efficacy of naloxone is that it can prevent but cannot reverse front paw FSIA. In contrast, hind paw FSIA does not involve opioids since it is neither cross-tolerant to morphine nor attenuated by 20 mg/kg systemic or 1 μg spinal naloxone. Spinalization & cord lesion studies have shown that front paw FSIA is mediated solely by descending pathways within the dorsolateral funiculus (DLF), whereas hind paw FSIA is effected both through descending DLF & intraspinal pathways. Hypophysectomy doesn't attenuate either front paw or hind paw FSIA; plus, front paw FSIA is potentiated by adrenalectomy or sympathetic blockade.

Upon exposure to footshock, an association is formed between environmental cues & the noxious stimulus such that the cues become capable of producing potent CCA. CCA always involves endogenous opioids, regardless of the body region shocked during conditioning trials; CCA is attenuated by systemic naloxone, spinal naloxone & morphine tolerance. Like front paw FSIA, CCA is: 1)prevented but not reversed by naloxone, 2) mediated by descending DLF pathways & 3) not affected by hypophysectomy or adrenalectomy.

These results strongly imply that FSIA & CCA are mediated by the same endogenous systems as morphine & brain stimulation. PHS Grant DA 00576 to DJM.

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ALTERATIONS IN BRAIN β-ENDORPHIN IMMUNOREACTIVITY FOLLOWING ACUTE AND CHRONIC STRESS. J.W. Lewis, J.T. Cannon, J.C. Liebeskind, and H. Akil, Department of Psychology, University of California, Los Angeles, CA 90024, and Department of Psychiatry, University of Michigan, Ann Arbor, MI 48109.

Stress may be a natural stimulus for activation of endogenous pain inhibitory systems. According to several criteria, prolonged footshock stress produces an opioid mediated analgesia whereas brief footshock stress elicits an analgesia that is nonopioid in nature. To begin identification of the neurochemical changes accompanying these stress responses, we have measured levels of β-endorphin immunoreactivity in the hypothalamus and midbrain.

To assess the effects of acute stress, rats were subjected to either prolonged, brief, or no stress immediately prior to sacrifice. Chronic stress effects were assessed in rats receiving 14 daily exposures to these same stress or control procedures. In the chronic experiments, animals were sacrificed either immediately or 24 hr following the last stress session. Radioimmunoassay for β-endorphin was carried out according to the procedures of Akil et al. (1979).

Acute brief stress significantly increased β-endorphin of the hypothalamus and midbrain, suggesting increased synthesis in preparation for release and/or transport. In the hypothalamus a similar increase was observed immediately after the 14th brief stress session, but not 24 hr later. By contrast, acute prolonged stress caused no measurable change in β-endorphin immunoreactivity. However, following chronic exposure to prolonged stress, a significant increase in hypothalamic β-endorphin was seen. This elevation persisted 24 hr later, suggesting a tonically increased production of β-endorphin in these animals. (Supported by Grants DA 02265 to H.A., NS 07628 to J.C.L., and MH 15345 to J.W.L.)