『CXBK mice deficient in opiate receptors show poor electroacupuncture analgesia』J. M. PEETS & B. POMERANZ ;　Nature volume 273, pages675–676(1978)

Abstract

RECENT research has led to the hypothesis that acupuncture produces analgesia through the release of endorphins. In neurophysiological studies on single neurones in lamina of cat spinal cord, electroacupuncture reduced responses to noxious stimuli, and the delays of this effect suggested a hormonal mediator. Analgesia due to electroacupuncture on mice and acupuncture on humans can be prevented by the opiate receptor antagonist naloxone. These studies strongly suggest that endorphins may mediate the observed analgesia. Endorphins, specifically β-endorphin and the enkephalins, are peptides whose abilities to bind to opiate receptors and produce analgesia have been well characterised. The implication of endorphins in electroacupuncture analgesia has rested heavily on the ability of naloxone to block the acupuncture effect. Reliance on this single line of evidence has been criticised, since naloxone may possess unknown activities unrelated to its opiate antagonist role. We show here that mice of the CXBK strain which are deficient in opiate receptors show poor electroacupuncture analgesia. This constitutes a naloxone-independent test of the hypothesis.