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Endocrinology. 1985 Oct;117(4):1389-96.

Evidence that beta-endorphin binds to specific receptors in rat peripheral tissues and stimulates the adenylate cyclase-adenosine 3',5'-monophosphate system.

Dave JR, Rubinstein N, Eskay RL.

Every organ has bliss receptors

Abstract

With the use of [125I]acetyl human beta-endorphin (Ac-hBE), specific binding sites for beta-endorphin (BE) were identified in the liver, kidney, adrenal, spleen, and testis of adult male rats, whereas specific BE-binding sites were not present in the ventral prostate or pancreas. In those tissues containing specific BE-binding sites, microsomal membranes (15,000-100,000 X g pellet) exhibited higher BE-binding capacity than the crude homogenate (125-100,000 X g pellet). The binding of BE was saturable, and maximal, specific binding was achieved with a 60-min incubation at 22 C. Furthermore, optimal BE binding was dependent on the presence of magnesium chloride. Scatchard analysis of BE binding to hepatic membranes revealed the existence of two classes of binding sites. One class had an apparent Ka of 0.019 X 10(9) M-1 and a lower number of binding sites (9.1 pmol BE/mg protein), whereas the other class had a lower affinity (apparent Ka of 0.0006 X 10(9) M-1) and a higher number of binding sites (159 pmol/mg protein). Specific BE binding to hepatic membranes was inhibited (80-100%) by rat AcBE-(1-27) and -(1-31), nonacetylated rat BE-(1-31), and human beta-lipotropin. At substantially higher peptide concentrations (greater than 10(-5) M), gamma-endorphin, met-enkephalin, or leu-enkephalin inhibited BE binding by 20-40%. In addition, opiate receptor-binding drugs, such as morphine and naloxone, at 10(-5) M did not alter BE binding to hepatic membranes. Incubation of hepatic membranes with BE induced a dose-related increase in membrane adenylate cyclase activity, and 0.5 X 10(-10) M BE resulted in a maximal enhancement of adenylate cyclase activity to 148% above control values. Water-deprived or salt-loaded male rats with chronically lowered immunoreactive plasma BE exhibited substantially increased BE binding to adrenal and kidney tissue. Specific binding sites for BE occur in a variety of peripheral tissues, and alterations of circulating BE result in changes in the capacity of certain peripheral tissues to bind BE. Finally, occupancy of specific BE-binding sites in peripheral tissue stimulates the adenylate cyclase-cAMP system, which suggests that the peripheral actions of circulating BE may be mediated via this system.

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