EFFECTS OF INTERLEUKIN-1β ON NOREPINEPHRINE TURNOVER IN RATS AND ITS POSSIBLE MECHANISM OF ACTION

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Interleukin-1β (IL-1β) has a wide variety of biological functions in addition to its key role in the immune response. Recently, it has been reported that IL-1 activates sympathetic nerve activity. In this study, the effects of IL-1 on norepinephrine (NE) turnover, a biochemical index of sympathetic nerve activity in both peripheral organs and the brain are examined. Furthermore, the mechanism by which IL-1 activates sympathetic nerve activity is investigated.

(1) Intraperitoneal administration of IL-1β was confirmed to increased NE turnover in the spleen, diaphragm and lung, but not in other organs such as the liver, kidney and pancreas. In the brain, the stimulatory effect of IL-1β was found only in the hypothalamus.

(2) To clarify whether or not the increased NE turnover in the hypothalamus is restricted in some specific anatomic structures, NE turnover in representative hypothalamic regions was examined using the punch out method, i. e., in the medial preoptic area, the lateral preoptic area (POL), the paraventricular nucleus, the ventromedial nucleus, the arcuate nucleus and the lateral hypothalamus. IL-1β was found to have a similar stimulatory effect in all regions except the POL.

(3) Pretreatment of rats with indomethacin, an inhibitor of prostaglandin (PG) synthesis, suppressed almost completely the increased NE turnover in the hypothalamus, while suppression was only partial in peripheral organs. These results suggest that the stimulatory effect of IL-Iβ on the sympathetic nervous system is mediated by PG at least in part.