

for Met-enkephalin is found in β -lipo-tropin. The physiological significance of the different distribution of these two types of opiate-like peptides is also unclear. Chronic morphine treatment alone or together with naloxone does not appear to affect regional or whole brain levels of enkephalin or total opioid activity or pituitary opioid activity. Stress is reported to increase (Santagostino *et al.*) or to decrease (Cox *et al.*) pituitary opioid activity.

It is of interest that neuroanatomical, neurophysiological, and neuropharmacological studies provide evidence that opioid peptides interact with other neural systems; that is, endorphin interacts with dopamine and enkephalin interacts with dopamine, gamma-aminobutyric acid, substance P, neurotensin, and acetylcholine. Some of these interactions may be involved in mediating the behavioral and neuroendocrine effects noted for endorphin and enkephalin. Guidotti and Grandison report evidence that the effect of endorphin in eliciting prolactin release is mediated by an inhibitory effect on the arcuate-nucleus-median-eminence dopaminergic tract; Schwartz *et al.* present evidence that inhibitory effects of opiates on dopamine release are mediated by a presynaptic contact of enkephalinergic neurons on dopaminergic nerve terminals, leading initially to decreased dopamine release.

The discovery of endorphins, their behavioral effects, and their relationship to exogenous opiates has led to much speculation concerning their role in normal mental function, response to pain, psychiatric disease, and addictive states. Research in this area has in part been hampered because of the lack of valid assays for measuring endorphin-like material (in peripheral tissue, blood, urine, cerebrospinal fluid) and determining the exact nature of the substance being measured (for example, a substance active in radioreceptor assays is not necessarily endorphin or enkephalin; additionally, at present there is no antibody that reacts with endorphin that does not cross react with lipotropin, necessitating some form of separation prior to final assay). This has made it difficult to correlate the presence of certain forms of behavioral symptomatology with levels of a specific opiate-like compound (assuming that such a compound circulates normally, which has by no means been proven). Moreover, level is a very imprecise measure of turnover, correlation with which would be far more significant. Several papers comment on the difficulty of assigning endorphins a role in normal behavior, for naloxone itself has no behav-

ioral effect in animals or humans. Naloxone, however, has been reported to be effective to a greater or lesser degree in reversing acupuncture analgesia (Pomeranz), in acupuncture treatment of alcohol- and tobacco-dependent states (Malizia *et al.*), and in treatment of psychotic hallucinations (Herz *et al.*). The last finding has not been confirmed by several other studies, which are not reported in this volume. Terenius reports very limited correlation of efficacy of acupuncture treatment in human chronic pain with increases in the content of a nonendorphin opioidlike material in cerebrospinal fluid.

It is apparent that there are many questions, both basic and clinical, that require further methodological advances before they can be answered. It is also apparent that great strides have been made in the short period of time that active investigation has proceeded in this field, so that a compilation of research reports a year hence may clarify some of the questions noted in this review.

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(Continued on page 446)

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