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EFFECTS OF CENTRAL ADMINISTRATION OF NEUROTRANSMITTER BLOCKERS ON FEED INTAKE*

S. Gopalan, R. Govinda Rao** and S. Jayasundar

Department of Pharmacology and Toxicology,
Madras Veterinary College, Madras-600 007

The majority of drugs that influence feed intake apparently act centrally by influencing various components involved in the neural regulation of appetite. The hypothalamus plays an essential role in energy and water homeostasis. It is bestowed with abundant supply of putative neurotransmitters and neurohormones. There is considerable evidence that most of the drugs that suppress or stimulate appetite act by changing the activity of the central neurons containing putative neurotransmitters such as dopamine, noradrenaline and serotonin (Samanin and Garattini, 1982). Gamma aminobutyric acid, acetylcholine and enkephalins are implicated as being important in the regulation of feeding (Kelley *et al.*, 1979). This work involved the study of the effects of blockers of cholinergic, alpha-adrenergic, beta-adrenergic and dopaminergic systems on central administration.

Materials and Methods

Male wistar rats weighting between 150 G and 180 G maintained on 4 hr schedule of pellet feeding daily were used in this experiment. Water was provided *ad libitum* throughout the day. Housed in individual cages under artificial lighting (9 hr light and 15 hr darkness) and controlled temperature (25±1°C) the rats were given drugs through a chronically implanted cannula into the two selected sites in the brain *viz*, Perifornical Hypothalamus (PFH) and Posterolateral Hypothalamus (PIH), following the methods described by Meyers and Meyers (1971) using the appropriate stereotoxic co-ordinates for the two hypothalamic sites given by Konig and Klippel (1963). The drugs/NSS were administered 15 mts. prior to feeding. Feed pellets were provided in two consecutive sessions of 2hrs each. Feed consumed during each session was recorded.

Hexamethonium at 10 µg, Prazosin at 2 µg, Propranolol at 5 µg and Haloperidol at 5 µg were administered in each site through the cannula and the feed consumed in the two sessions were recorded and compared.

Since the deprived rats consumed a major portion of the feed during the first 2 hr of the 4 hr feeding schedule, the first 2 hr and total 4 hr feed intake were taken for analysis.

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** Professor and Head, Dept. of Physiology and Biochemistry

release by activation of opiate receptor. So the Met-Enk induced increase of LS activity could be also due to its action at the receptor level in stimulating prolactin release resulting in an increased level of alphalactalbumin in the mammary gland.

Summary

In vivo studies on UDPG-Py in mammary gland revealed that morphine could induce an increase in its activity. This morphine induced increase could be brought back to the normal level with naloxone. Opioid peptides failed to alter the enzyme activity in the mammary gland. Like the increased activity of UDPG-Py in the mammary gland *in vivo*, presence of morphine in the *in vitro* system also induced increased activity of the enzyme. Such increase could be counteracted by naloxone. In case of opioid peptides, presence of both Met-and Lau-Enk caused an increase in the enzyme activity *in vitro*.

A striking increase in LS activity was observed in the mammary gland of animals which were injected with morphine. Such increase could be reversed in the animals when naloxone was injected either alone or in combination with morphine. The results on *in vitro* experiment resembled the *in vivo* data to a limited extent. Unlike the *in vivo* data on opioid peptides, the enzyme activity *in vitro* was observed to decrease in the presence of Met-Enk in particular.

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