

COMMENTARY ON: SYSTEMIC ADMINISTRATION OF GHRELIN INDUCES FOS AND EGR-1 PROTEINS IN THE HYPOTHALAMIC ARCUATE NUCLEUS OF FASTED AND FED RATS. HEWSON AK, DICKSON SL. *J NEUROENDOCRINOL.* 2000; 12: 1047-1049.

Alfonso Abizaid and Melissa J Chee¹

¹Department of Neuroscience, Carleton University, Ottawa, ON, Canada, K1S 5B6

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Ghrelin is a hormone secreted by the gut in response to hunger and acts to stimulate appetite and fat deposition (1). It is the endogenous ligand for the growth hormone secretagogue receptor (GHSR) found in the pituitary and the brain (2). The discovery of ghrelin was preceded by the cloning and characterization of the growth hormone secretagogue receptor (GHSR), the only known receptor for ghrelin (2). The central expression of GHSR suggested that ghrelin release from the gut could act in the brain to drive feeding. This pioneering article by Hewson and Dickson (3) confirmed that ghrelin targets GHSR in the hypothalamic arcuate nucleus to drive feeding. They demonstrated that peripheral injections of ghrelin stimulated neurons in the hypothalamic arcuate nucleus. This increased neuronal activation, shown by the increase in the immunocytochemical expression of the immediate early gene markers Fos and Egr-1. Moreover, fasting enhanced the effects of ghrelin-stimulated arcuate neurons. These results confirmed previous data showing that growth hormone secretagogues similarly stimulated cells in the arcuate to promote feeding and adiposity (4). This article was one of the earliest accounts of the central mechanisms mediating the orexigenic effects of ghrelin.

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