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Rapid Autonomic Nervous System Responses Contribute to the Homeostasis of Plasma Glucose and Insulin Levels During Food Intake

Steffens, A.B.; Strubbe, J.H.; Balkan, B.

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SESSION V: NEURAL INTEGRATION

Moderator: L. Arthur Campfield, Hoffmann-La Roche, Inc.

Neuroanatomical Bases of Cephalic Phase Reflexes. T. L. POWLEY and H.-R. BERTHOUD. *Purdue University, Lafayette, IN, U.S.A.*

We recently delineated the neural substrates of the cephalic phase insulin response (CPIR) of the rat by employing simultaneously the techniques of selective vagal branch cuts, monitoring of the initial phase of insulin release to a meal stimulus, and retrograde axonal tracing. The results indicate that three of the five abdominal vagal branches (the anterior gastric, hepatic and posterior gastric) mediate virtually all of the CPIR, whereas the celiac and anterior celiac branches have little effect. These observations contrast with suggestions found in the literature, namely that insulin release is controlled predominantly by one vagal trunk (the posterior abdominal trunk) and mediated by one abdominal branch (the celiac) of the vagus. Centrally, the CPIR is represented bilaterally within the medial longitudinal cell columns or subnuclei of the dorsal motor nucleus of the vagus, i.e. by the preganglionic motor neurons projecting through the gastric (and hepatic) branches of the abdominal vagus. Additionally, correlations of the number of preganglionics projecting through each of the individual vagal branches with the relative amplitudes of the CPIRs elicited from the respective branches suggest that even a very few vagal preganglionic neurons are able to mediate a significant cephalic response. When taken together with recent mapping studies of afferent inputs to the dorsal vagal complex of the medulla, the present observations on the motor pathways of the CPIR offer a relatively complete description of the medullary organization of the cephalic phase reflex of insulin secretion.

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A. B. STEFFENS, J. H. STRUBBE and B. BALKAN. *University of Groningen, The Netherlands.*

Food intake in rats leads to increases of blood glucose and insulin during the after a meal. Blood glucose levels and presumably also those of other nutrients start to rise in the 3rd min after meal onset whereas plasma insulin and glucagon levels are enhanced in the 1st min. The preabsorptive insulin response (PIR) is absent by elimination of both afferent signals from the oral cavity to the central nervous system (CNS) and efferent signals from the CNS to the isle of Langerhans. Efferent signals to the islets are of a para-sympathetic as well as a sympathetic origin and both systems are activated during food intake. Elimination of muscarinic receptor activity by atropine and elimination of alpha-adrenoceptor activity by phentolamine on the B cell leads to a suppression and an increase of PIR, respectively.

The second phase of insulin release is also abnormal. Noradrenergic mechanisms in the hypothalamus are involved in the reflexive mediation of PIR. Norepinephrine infusion into the LHA causes an exaggerated insulin release which indicates an involvement of the LHA in establishing the setpoint of the B cell regarding insulin release after a certain glucose challenge. Small VMH lesions in rats subjected to a strictly pair feeding situation with controls lead to obesity. In these VMH lesioned animals intravenous as well as oral glucose tolerance tests are deteriorating in the weeks after lesioning. The hypothalamus is involved in the stabilization of glucose and insulin profiles during and after food intake by controlling the sensitivity at which the B cell releases insulin. Loss of control leads to deviations in nutrient flow to different compartments in the body eventually causing obesity.