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Comparison of pathogenesis and pathophysiology between inter-ventricular (I-VMH) and ventromedial hypothalamic (VMH) obesity in rats

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We have been investigating the pathogenesis and pathophysiology of ventromedial hypothalamic (VMH) obese animals, a representative model of hypothalamic obesity, for nearly 40 years and recently we discovered a novel hypothalamic obesity induced by bilateral destructions of the area between arcuate nuclei (ARC) and paraventricular nuclei (PVH) in the hypothalamus (referred to as inter-VMH; I-VMH) in rats. My talk deal with comparison of pathogenesis and pathophysiology between VMH obesity and I-VMH obesity.

VMH lesioned rats show more hyperphagia and more obesity, and more higher levels of serum insulin and leptin than I-VMH lesioned rats. Disconnection of α-MSH pathway by determined histochemical neural fiber staining in ARC-PVH axis for food intake regulatory system contributes to hyperphagia and obesity in I-VMH lesioned rats. The origin of hyperphagia in VMH lesioned rats is, in part, due to disturbance of α-MSH pathway but more complex. Hyperinsulinemia induced by autonomic derangement (vagal hyperactivity with sympathetic hypoactivity) is the primary cause of VMH lesion-induced obesity. Cell proliferation in visceral organs (stomach, small intestine, liver and pancreas) by histochemical stainings and electron microscopic examinations are observed in VMH lesioned rats, which is due to vagal hyperactivity in autonomic derangements by VMH lesions, in contrast, no cell proliferation in the visceral organs are observed in I-VMH lesioned rats, due to a lack of autonomic derangements.

Biography

Shuji Inoue has completed his M.D. at the age of 26 years old and got Ph.D from University of Tokyo, School of Medicine. He is the vice-president and dean of Faculty of Health Care. He has published more than 200 English papers in reputed journals and serving as Editor-in-Chief of Obesity Research & Clinical Practice.

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