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A UNIFIED MODEL: THE FISHBONE-B PANCREATIC CELL CONVERGENCE MODEL OF BLOOD-BRAIN GLUCOSE METABOLISM

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Introduction: The brain utilizing mechanisms of glucose for neuronal functions are still poorly understood. To identify a unified model of blood-brain glucose metabolism, we developed a model that incorporates the pancreatic β-cell and the fishbone models of glucose metabolism and called it the CoBBGluM Model (or the Convergence model of Blood-Brain Glucose Metabolism) and examined the role of addictive substances (alcohol, cocaine etc) on the functions of the model.

Methods: The databases of Pubmed, Elsevier were searched for peer reviewed literatures (from 1950-2009yy) on the mechanisms and models of blood-brain glucose metabolism.

Results and conclusion: The major concept of the unified model is based on the fact that the major regulators (leptin and insulin) of blood-brain glucose metabolism work synergistically, rather than individually. Addictive substances adversely affect the blood-brain glucose transport system by their stimulating and toxic action on the control mechanisms of leptin and insulin. The metabolic byproducts of addictive substances might acquire electron-transmitter properties across mitochondrial membranes. All these processes subsequently lead to total equilibrium disorder of the CoBBGluM Model, and is the etiopathogenetic basis of most addiction-associated neurodegenerative disorders. Adequate therapies for addiction might lie on the full understanding of the CoBBGluM Model, since it serves a classical tool for explaining the role of addictive substances in the nervous system.

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