Neuroinflammation and Diabetes

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Abstract

Obesity is a chronic disease that shows the most serious global health problems. It relates to the body fat. Obesity is caused by an imbalance between energy intake and expenditure, this balance is regulated by genetic and environmental interactions. Obesity, which is a risk factor for chronic diseases, has become epidemic in the developed countries. Resistance to Leptin hormone is one of the main reasons for obesity; this hormone is released by fat cells, substantially more fat causes more releasing of Leptin hormone. In fact, the discovery of Leptin hormone has opened a new field of research in obesity studies. Leptin hormone controls metabolism, reconciliation and energy consumption. Obesity plays a key role in the development of diabetes. Effect of obesity on the development of diabetes through precipitation of Amylin. Amylin is a peptide hormone that is co-secreted with insulin from the pancreatic β-cell and in patients with diabetes, there is deficiency of that. As we know, in diabetes, insulin resistance and pancreatic β-cell hypertrophy occurs in the early stages, in this condition, Amylin is increased. Accumulation of Amylin is in the hypothalamus of the brain and it can lead to neurological disorders through inflammatory reactions. Increased insulin sensitivity in the body is related to the proper functioning of the hypothalamus. Also, hypothalamus controls Leptin, when the level of Leptin hormone is increased it is bonded to its receptors in hypothalamus. The purpose of this paper is investigation the relation between Amylin and Leptin in the hypothalamus in the process of neurological inflammation and diabetes. Leptin hormone resistance is a characteristic of obesity; studies have been shown the inflammation of the hypothalamus that is results from fat is an important mechanism that will cause to increase Leptin resistance and precipitation Amylin.

Keywords: Neuroinflammation, Diabetes, Obesity, Leptin, Amylin

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