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Effects of Obesity in Various Cardio diseases

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Obesity'type 2 Diabetes; Qsymia; Liraglutide; Contrive; Hfdbs (High Frequency Deep Brain Stimulation; VNS (Vagus Nerve Stimulation); VBLOC (Vagal Blocking)

Abstract

Beneficial effects of FGF21 are obtained in obesity and metabolic consequences like myocardial infarction. The role of saturated fatty acids using a ceramide pathway to affect insulin resistance vs unsaturated fatty acids like linoleate using Diacylglycerol pathway is further discussed. We reviewed the literature relating to hypothalamic inflammation (HI); gliosis in relation to high-fat diet (HFD) and that how this could be reversed with various types of therapies. We searched PubMed articles with the MeSH terms "hypothalamic inflammation", "gliosis", "HFD", "obesity", and "treatments" used. During HFD intake, we found that the ventromedial hypothalamus (VMH) astrocytes uses fatty acids (FA's) to generate ketone bodies which are then exported to neurons where they produce excess adenosine triphosphate (ATP) and reactive oxygen species (ROS), which overrides CD36 mediated FA sensing and role of astrocyte derived ketone bodies in reducing calorie intake in diet resistant but not diet-induced obese strains was emphasized. The further role of HAM-RS2-a special starch, resolvins abscisic acid KBH1 unsaturated fatty acid receptor targeting GPR120/GPR40. Hepatic clock genes were effective in tackling obesity. We found that in rodent's hypothalamic inflammation and gliosis were found to occur immediately with HFD consumption before any significant weight gain. Sensitivity or resistance to diet-induced obesity in rodents also correlates with the presence or absence of hypothalamic inflammation and reactive gliosis. Further functional interventions with the increase or decrease inflammation in neurons and glia alter diet associated weight gain. Various human magnetic resonance imaging (MRI) studies have found gliosis and disrupted connectivity in obese humans. Various factors which can be used to tackle obesity like HAMRS2- a special starch, resolvins, abscisic acid, KBH1, unsaturated fatty acid receptors, GPR120 and GPR40 are some of the explored routes by which these pathways may be explored to prevent the further extension of the HFD and one may get newer answers for arresting obesity development.

Human obesity differs from that of rodents and animals which are usually utilized for studying both aetioathogenesis along with treatment of obesity. This is in view of hypothalamus generally considered to be the organ for homeostatic control is under control by various supra homeostatic control besides peripheral regulation. Hence the needs to understand how the higher centres regulate hyothalamus for a given response. Various functional neuroimaging studies have proved to be helpful in depicting these roles. Further it has helped in understanding how the most effective treatment like bariatric surgery (BS) alters these supra hypothalamic control in altering hippocampus, areas involved in executive function along with reducing ghrelin besides changing the cortical thickness with alterations of ratios of gray and white matter. These changes need to be translate to drugs utilized for anti-obesity therapy as has been done by lorcaserin, liraglutude and other natural food products like walnuts and other such natural products to help better get some safe options for anti-obesity therapies that prove to be clinically effective.

In our previous reviews regarding obesity we have concentrated on different aspects of aetiopathogenesis and treatment on BAT physiology and metabolism and how micro RNA's affect metabolism in obesity and its consequences. In this review we have tried to look into the dietary aspects of obesity along with fructose, fatty acid metabolism with special emphasis on ceramide signaling, protein and positive effects of green tea components. Further the interaction of Fibroblast growth factor 21 with ceramidase and adinopectin has been highlighted