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Monosodium Glutamate-Induced and Medicinal Plants that Fight Obesity

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Abstract

Overweight and obesity have skyrocketed worldwide over the past three decades at an alarming rate. Low-grade inflammation, oxidative stress, glucose intolerance, metabolic syndrome, and other metabolic abnormalities are all caused by obesity. Alterations in the energetic metabolism homeostasis and persistent systematic low-grade inflammation have been observed in an identical scenario as aging progresses. Poor physical performance and oxidative stress can raise metabolic disease risk. Despite the many different studies that have been done on the pathophysiological effects of obesity, not enough research has been done on its effects on gender and over time, especially as people get older. The monosodium glutamate (MSG)-induced obese model is the focus of this review, which aims to outline the pathophysiological mechanisms and metabolic changes associated with obesity. Male mice showed more signs of MSG-induced obesity, inflammation, and decreased adiponectin, while both male and female mice's glucose tolerance, insulin sensitivity, and redox balance changed with age. According to these findings, MSG-induced obesity-related metabolic alterations are gender and age related. As a result, the relationship between gender, aging, and metabolic changes in obesity can be supported by the MSG obesity model. We also looked at the medicinal plants and their active ingredients that have been used to treat obesity caused by MSG. Studies are required to investigate the beneficial effects and underlying mechanisms of medicinal plants with demonstrated anti-obesity activity due to this model's significance.

Keywords: Obesity; Medicinal plants; Monosodium glutamate

Introduction

In developed nations, obesity is regarded as the most common nutritional disorder and a health issue. Adipose tissue (AT) or body fat that is too high as a result of consuming too many nutrients or burning too few calories is considered obesity [1]. It is characterized by an abnormal, excessive increase or accumulation of energy in the form of fat in AT, which is the combined result of satiety center dysfunction at the cerebral level, an imbalance in the intake and use of energy, and genetic variations. Social norms can be profoundly influenced by environmental factors, and some studies have emphasized the role of the environment in obesity development. It has been accounted for that in 95% of the corpulence cases the hidden reason is nourishing, exogenous or essential, while it is endogenous, monoergic or seconday cause in 5% of the cases. Obesity is categorized as either central, with a predominance of fatty tissue in the intra-abdominal region, or peripheral, with fatty accumulation primarily in the femoro-gluteal region, according to the distribution of body fat. This distribution affects quality of life and varies based on gender and race. It is primarily mixed at birth. Since the 1940s, hormonal differences have been partially blamed for the gender differences in body fat mass distribution. While estrogen is linked to the accumulation of fat in the peripheral or subcutaneous areas, androgens are linked to increased visceral AT. Leptin, which causes the secretion of gonadotropin-releasing hormone (GnRH), influences changes in the distribution of body fat during puberty [2].

Literature Review

Obesity is a risk factor for a wide range of diseases, including dyslipidemia, cardiovascular disease (CVD), insulin resistance (IR), and type 2 diabetes mellitus (T2DM). In addition, obesity has been regarded as a chronic low-grade inflammation of the system that has a significant correlation with the likelihood of IR [3]. Increased inflammatory leukocyte infiltration and activation of the expression of a variety of pro-inflammatory cytokines, including TNF- and IL-6, are linked to visceral AT expansion. It is interesting to note that the distribution of AT and inflammation in obesity are significantly influenced by gender [4]. It is common knowledge that women tend to have a lot of fat under the skin, while men tend to have less total fat, mostly in the visceral area. It has been discovered that women have lower plasma concentrations of IL-6 and TNF- than men do, possibly because estrogens inhibit the expression of pro-inflammatory cytokines. Additionally, there is a positive correlation between serum leptin levels and elevated adiposity and the body mass index (BMI). During puberty, women have higher levels of leptin in their blood, but men have lower levels after initiation (stage 2 of gonadal maturation), leading to the android and gynoid pattern of fat distribution that is common in adolescents and adults [5-6].

Discussion

One of the proteinogenic amino acids is glutamic acid (Glu or E), and its codons are GAA and GAG. It has a side chain carboxylic acid functional group and is an amino acid that is not required. Glutamates are the carboxylate anions and Glu salts [7]. Long-term potentiation relies heavily on glutamate, the most abundant excitatory neurotransmitter in the central nervous system (CNS). The majority of normal brain functions, including learning, memory, and cognition, involve it. Most commonly known as "Monosodium Glutamate" (MSG), glutamate is a sodium salt of glutamic acid that is used in the food industry as a flavor enhancer. MSG imparts a flavor that cannot be replicated by other food additives and increases food's sapidity [8].

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Conclusion

Transaminase is responsible for the biosynthesis of glutamate from the TCA cycle intermediate -ketoglutarate in the mitochondria. It is transported by a high-affinity transport system, but it cannot easily cross the blood-brain barrier (BBB). Additionally, it can be converted into glutamine, which can cross the BBB, and then into glutamate through the action of phosphate-activated glutaminase in the following ways: Glutamate and NH3 vs. glutamine and water Apparently glutamate got from glutamine through this course is delivered intramitochondrially and may hence go through a transamination catalyzed by the mitochondrial isoform of aspartate aminotransferase (AST). The produced -ketoglutarate is transaminated in the cytoplasm by the cytoplasmic isoform of AST after being translocated out of the mitochondria by the dicarboxylate carrier. Alternately, a reaction sparked by alanine aminotransferase (ALT) can produce glutamate from -ketoglutarate and alanine. Vesicular glutamate transporters transport this cytoplasmic glutamate, whose release is controlled by the intracellular calcium (Ca2+) concentration. Presynaptic receptors include metabotropic, cholinergic, and gamma-aminobutyric acid (GABA) receptors, among other types. Ionotropic and metabotropic glutamate receptors are two distinct categories that can be applied to the glutamate receptors based on how they are activated. Ionotropic receptors are classified into three groups: Non-NMDA receptors such as the -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) and Kainate receptors are also known as N-methyl-d-aspartate (NMDA) receptors. The metabotropic receptors (mGluR) are dispersed throughout the central nervous system (CNS), where they regulate numerous essential metabolic and autonomic functions in the amygdala, hippocampus, and hypothalamus, respectively. Other G protein-linked metabotropic receptors share a molecular morphology with these receptors.

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Conflict of Interest

None

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