



The preliminary investigation of Orexigenic hormone gene polymorphisms on posttraumatic stress disorder symptoms

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ABSTRACT

Post-traumatic stress disorder is a mental health condition that is triggered by a traumatic event. Symptoms can include nightmares and intense anxiety, as well as uncontrollable thoughts about the event. Examples of events that can trigger the disorder include war, crime, fire, accident, death of a loved one, or sexual abuse. According to statistics, between 7 and 8 percent of the world's population suffers from post-traumatic stress disorder (PTSD), and it affects women more than men. Although most PTSD symptoms begin within a month of exposure to the traumatic event, sometimes they do not appear for years. These symptoms cause significant problems in social or work situations and in the sufferer's relationships. They can also interfere with your ability to perform normal daily tasks. Post-traumatic stress disorder (PTSD) is a debilitating disorder that is associated with behavioral disturbances, physical problems, and some mental health disorders such as depression, with a high risk of suicide.

Introduction

Treatment of this condition is challenging, with medications such as antidepressants, anticoagulants, beta-adrenergic antagonists, narcotics, and cortisol showing mixed results [1-3]. Since the onset of this disorder is individual-dependent, prevention techniques can also be diverse and have different effects on individuals. PTSD is a public health problem with devastating effects on the affected individuals, their families, and society in general. Symptoms of posttraumatic stress disorder are associated with factors such as gender, personal and family history of anxiety, and previous traumatic experiences. Previous findings have also shown that the likelihood of inheriting symptoms of this disorder in twins is significant [3].

In fact, the age of the twins has been known to be an important factor in the development of PTSD. In addition, twins experience a common family environment while growing up, which in turn contributes to the development of common PTSD. Finally, studies on twins have shown that genetics play an important role in posttraumatic stress disorder and anxiety symptoms [4-6].

What is a traumatic event?

PTSD is the result of a traumatic event that causes intense fear, helplessness, or panic. Events such as sexual or physical assault, the unexpected death of a loved one, an accident, war, or natural disasters are some of the events that cause post-traumatic stress. The traumatic event may be life-threatening, or pose a significant threat to your physical, emotional, or spiritual health [7-9].

PTSD affects people of all ages. Most people who experience a traumatic event experience reaction such as shock [10-12], anger, aggression, fear, and even guilt. These are common reactions that go away for most people over time. However, a person with PTSD will continue to experience these feelings, and sometimes these symptoms become so strong that they interfere with the person's functioning [13-15].

Treatment for Post-Traumatic Stress Disorder

Treatment for post-traumatic stress disorder is designed to reduce the mental and physical symptoms of this disorder, improve the person's daily functioning, and help them better manage the traumatic event [14-16] Treatment for PTSD

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includes psychotherapy (talk therapy), medication, or both. Recovery from PTSD is a gradual and ongoing process. It takes time and patience to fully recover from PTSD symptoms [17-19], and most often, treatment involves teaching you how to

manage them effectively (Figure 1). Psychotherapy and medication can help reduce severe symptoms and increase your ability to manage your emotions [20-22].

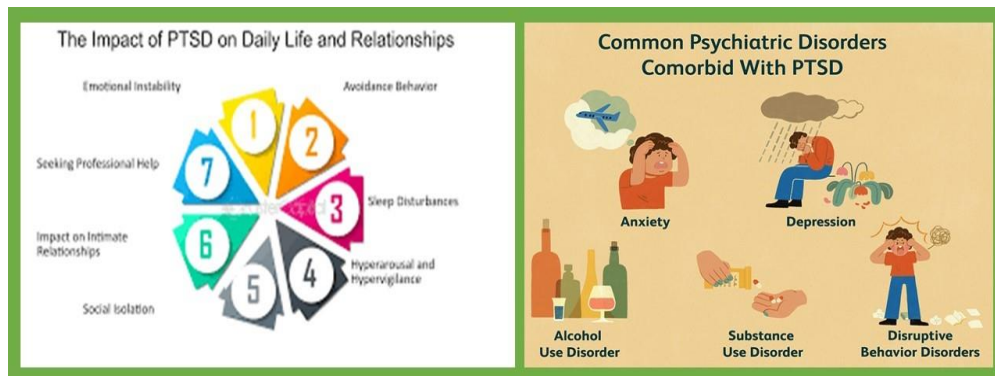


Figure 1. Treatment for Post-Traumatic Stress Disorder

Acute stress disorder (ASD) and post-traumatic stress disorder (PTSD) both occur in response to traumatic events but differ in their duration and symptoms. ASD develops within 3 days to a month after the trauma and is characterized by symptoms such as intrusive thoughts, irritability, and avoidance. If symptoms persist for more than a month, the diagnosis may change to PTSD. PTSD involves chronic symptoms and can last for years if left untreated.

Diabetes is a multifactorial metabolic endocrine disorder characterized by hyperglycemia (high blood sugar levels over a prolonged period). This high blood sugar level is caused by deficient insulin secretion, impaired insulin action, or both. Current research has shown that adipose tissue is not only a lipid storage depot but also an essential endocrine organ. Adipose tissue plays a key role in integrating endocrine, metabolic, and inflammatory signals to regulate energy homeostasis. Various functional proteins, collectively called adipocytokines or adipokines, are secreted from fat cells into the circulation. Among these adipocytokines is a hormone called adiponectin. Adiponectin has anti-inflammatory, antidiabetic, and insulin-sensitizing properties. This protein plays an important role in various metabolic processes, including glucose control. Reduced adiponectin levels are associated with type 2 diabetes mellitus (T2DM) and obesity. Adiponectin appears to be a major modulator of insulin action, and its levels are reduced in diabetes. This reduction may lead to peripheral insulin resistance.

Mechanism of Adiponectin Action in Insulin Sensitivity

Since its discovery, adiponectin has attracted much attention from scientists. Numerous studies have

demonstrated its role as an insulin sensitivity enhancer. Over the years, a large body of evidence has confirmed the multiple roles of this hormone in various tissues of the body. Adiponectin exerts numerous cellular and metabolic effects, mainly through binding to two types of adiponectin receptors, called AdipoR1 and AdipoR2. Each of these receptors has a different distribution in different tissues of the body. AdipoR1 is found mostly in skeletal muscle, and AdipoR2 is found mostly in the liver. Studies have shown that a specific type of adiponectin, called gAd, binds to the AdipoR1 receptor and mimics the effects of insulin in skeletal muscle. While another type of adiponectin, called fAd, binds more to the AdipoR2 receptor in the liver and exerts its effects there. Impaired adiponectin function in skeletal muscle of obese people with type 2 diabetes could contribute to insulin resistance. On the other hand, adiponectin in the liver can inhibit glucose production and enhance the effect of insulin in the liver.

A completely new candidate for weight loss therapy has been found, and scientists have discovered for the first time how a hormone produced by the hypothalamus during sleep has a direct effect on appetite and metabolism regulation. This is the first time that a hormone called raptin has been linked to sleep and obesity. According to New Atlas, the discovery paves the way for the development of a new class of drugs that could combat obesity, such as GLP-1 agonists. Chinese scientists noted in the study: "Sleep deprivation is associated with obesity, but the mechanisms of this association remain unclear. In this study, we identified a hypothalamic protein hormone secreted during sleep in humans and mice that suppresses obesity. This hormone is cleaved from reticulocalbin-2 (RCN2) and we named it raptin." Researchers at Xiangya Hospital of Central South University are the first to identify and

understand how this sleep-induced hypothalamic hormone signals satiety to the gut and limits food intake. In addition, poor or insufficient sleep greatly reduces levels of raptin, which blocks appetite control, and raptin levels are much lower in obese people. Raptin binds to the metabotropic glutamate receptor 3 (GRM3) in neurons in the hypothalamus and stomach to inhibit appetite and gastric emptying, respectively, the researchers say. Raptin is a truncated fragment of the protein reticulocalbin-2 (RCN2), which plays an important role in bone formation and cancer, but has never before been linked to appetite control or other metabolic functions.

The researchers identified and confirmed the role of raptin in metabolic processes through a combination of proteomics to understand the structure and biology of the RCN2 protein, as well as molecular biology, neuroscience, and human clinical data. Raptin's role was consistent in both mice and humans. Raptin is closely linked to circadian rhythm cycles, and while scientists have not stated in which specific stage of sleep the hormone is produced and secreted, its levels peak during the night and when the eyes are closed. The hormone is most likely produced and secreted during non-rapid eye movement, or NREM, sleep. Levels of the hormone gradually decline throughout the day before rising again in the next sleep cycle. However, further studies need to confirm whether sleep stages play a specific role here. Poor or insufficient sleep has been linked to weight gain associated with this hormone. One such study in 2022 found that sleep deprivation increases ghrelin production, which leads to increased appetite, and limits the secretion of the hormone leptin, which then interferes with gut satiety signals. Other research has linked poor sleep to increased levels of stress hormones and their impact on metabolic regulation. In humans, researchers assessed raptin levels, sleep quality, and other metabolic measures in 262 participants, 127 of whom were clinically obese. They then conducted a trial with 30 people, 15 in a control group and 15 who underwent sleep restriction (SRT) for three months. At the end of the trial, data was collected to assess changes in raptin levels and body weight, as well as sleep quality and energy intake. The researchers say that participants who suffered from sleep deprivation had lower levels of raptin and were more likely to develop obesity, while obese patients who underwent SRT had higher levels of raptin and reduced obesity phenotypes. These findings underscore the importance of sleep quality on metabolism. In addition, a genetic study of 2,000 obese individuals led to the discovery of a variant of RCN2 that was present in the biological code of a group of family members who all suffered from

night eating syndrome (NES). NES is currently difficult to treat and is generally considered a mental disorder and is often misdiagnosed. So these findings could have a big impact on those who suffer from these nocturnal binges. These individuals were prevented from producing raptin, and they were all obese. The team also noted the link between high-fat diets and sleep deprivation, saying that more research is needed to see how it might affect raptin production and its ability to regulate appetite. Overall, however, the findings pave the way for the development of new therapeutic approaches for weight loss and obesity treatment. The researchers concluded: "Here we show that circadian release of hypothalamic raptin reduces appetite, but does not affect other important circadian behaviors, including the sleep cycle itself or physical activity. Our study identifies raptin as a unique hypothalamic hormone that works with GRM3 to suppress appetite and obesity, thus providing a potential new avenue for treating obesity."

Adiponectin gene polymorphisms in different types of diabetes mellitus

Genetic studies on the adiponectin gene in humans indicate that this hormone plays an important role in the development of insulin resistance and diabetes. The study showed that a number of small variations in the adiponectin gene (SNPs) are directly associated with different types of diabetes, including type 1 diabetes, type 2 diabetes, and gestational diabetes in pregnant women. Type 1 diabetes is an autoimmune disease in which the insulin-producing beta cells in the pancreas are destroyed, resulting in very low or no insulin production. In type 1 diabetes, kidney complications are associated with insulin resistance and mild inflammation. Adiponectin levels are higher in the blood of type 1 diabetes patients with kidney complications. This increase in adiponectin levels may be due to increased anti-inflammatory function, improved insulin sensitivity, and reduced vascular damage. Studies have shown that adiponectin levels are increased in the blood of type 1 diabetes patients, especially those with diabetes complications (Figure 2). The adiponectin gene consists of three main parts, and small variations in this gene (SNPs) can be associated with the development of type 1 diabetes. For example, two variations in this gene, rs2241766 and rs1501299, have been associated with type 1 diabetes in different populations. The variation rs17300539 in the adiponectin gene has also been associated with kidney complications in patients with type 1 diabetes. The variation rs266729 has also been associated with kidney complications in women with type 1 diabetes, but this association has not been observed in men.

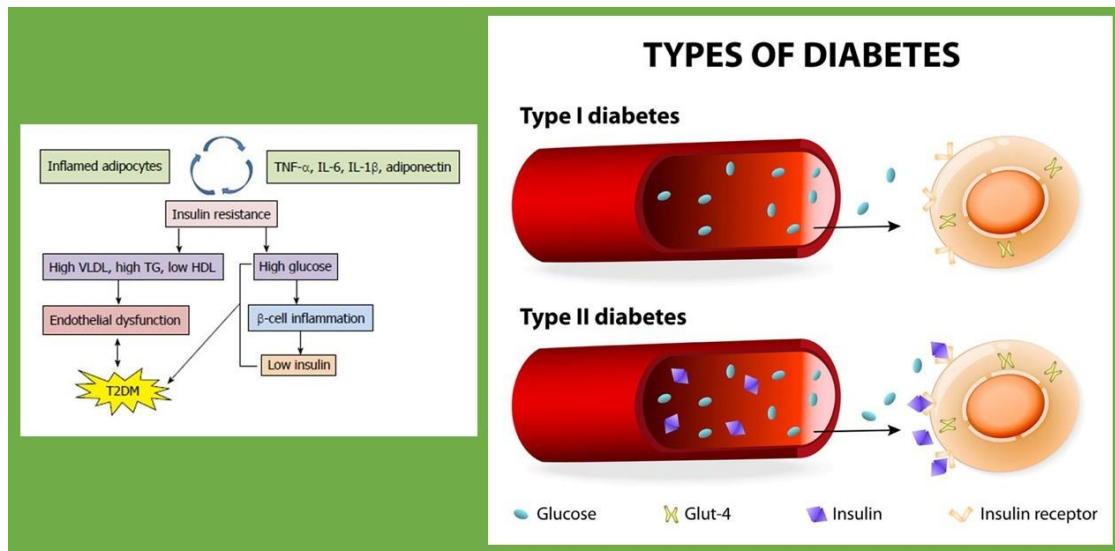


Figure 2. Understanding types of Diabetes Mellitus

In general, genetic variations in the adiponectin gene can contribute to the development of type 1 diabetes and its complications. Type 2 diabetes is a multifactorial disease that develops slowly and gradually. The disease and its complications place a heavy burden on patients and the healthcare system. Type 2 diabetes develops over time (years). During this time, insulin resistance begins, meaning that the insulin in the blood gradually loses its effectiveness. Because of this resistance, the pancreas produces more insulin to control blood sugar. Unlike type 1 diabetes, adiponectin levels are lower in the blood of type 2 diabetes patients. Studies have shown that low levels of adiponectin are associated with increased insulin resistance and the development of type 2 diabetes. Small variations in the adiponectin gene (SNPs) can also be associated with the development of type 2 diabetes.

Discussion

Ghrelin, often referred to as the hunger hormone, plays an important role in regulating appetite and energy balance. This peptide hormone is primarily secreted by the stomach and has a wide range of effects on various physiological processes in the human body. Ghrelin was first discovered in 1999 by researchers at the University of Tsukuba in Japan. The name ghrelin comes from the Proto-Indo-European root word “ger” meaning “to grow” or “to increase [38].” This nomenclature reflects its role in stimulating hunger and promoting food intake, which ultimately contributes to weight gain and maintenance [39].

Ghrelin is a 28-amino acid peptide produced primarily in the oxyntic glands of the stomach, where it is synthesized as a prohormone, but is also made in the islets, heart, lungs, kidney, immune system, and hypothalamus and pituitary glands. This precursor is then cleaved to produce the active

peptide ghrelin. Ghrelin secretion, which usually occurs in response to an empty stomach and peaks just before a meal, stimulates appetite and prepares the body for incoming nutrients. Ghrelin increases growth hormone secretion, gastric emptying, and acid secretion [40].

The primary mechanism by which ghrelin exerts its effects is binding to the growth hormone-secreting receptor (GHS-R), which is found in the hypothalamus, pituitary gland, and various other tissues. Once bound, ghrelin activates these receptors, leading to the release of growth hormone (GH) and regulating appetite:

1- Appetite stimulation: Ghrelin acts on the hypothalamus, specifically the arcuate nucleus, to stimulate the release of neuropeptide Y (NPY) and agouti-related peptide (AgRP). These neuropeptides increase appetite and food intake.

2- Energy homeostasis: Ghrelin also modulates energy expenditure. This promotes the storage of energy as fat and reduces calorie burning, contributing to weight gain.

3- Effect on reward pathways: Ghrelin has been shown to activate brain areas associated with the reward system, making high-calorie foods more appealing, potentially contributing to overeating and obesity [41].

Ghrelin's role in regulating appetite and energy balance has important implications for health and disease. Here are some key areas where ghrelin is of interest:

1- Obesity: Dysregulation of ghrelin signaling can lead to overeating and obesity. Understanding how ghrelin affects appetite can provide insight into potential interventions for weight management.

2- Eating disorders: Ghrelin levels have been found to be altered in individuals with eating disorders such as anorexia nervosa and bulimia nervosa, suggesting a role in these conditions.

3- Diabetes: In the pancreas, there is evidence that ghrelin inhibits glucose-stimulated insulin secretion and stimulates glucagon secretion [42].

4- Digestive disorders: The location of ghrelin production in the stomach makes it relevant in the context of digestive diseases. It has been studied in conditions such as gastroparesis and inflammatory bowel disease.

5- Potential therapeutic applications: Researchers are investigating ghrelin analogs and antagonists as potential treatments for obesity and other metabolic disorders.

The progesterone receptor gene plays a critical role in the female reproductive system. Therefore, polymorphisms in this gene can affect progesterone receptor function [43].

Schweikert and colleagues studied the presence of three polymorphisms in 42 patients with recurrent miscarriage and 40 healthy controls: G1031C in exon 1, G1978T in exon 4, and C2310T in exon 5. Their results showed that the presence of these polymorphisms was associated with increased miscarriage.

In India, Aruna and colleagues studied a group of 143 women with a history of recurrent miscarriage and a control group of 150 women without a history of miscarriage. In the aforementioned study, the presence of 3 polymorphisms G1031C in exon 1, G1978T in exon 4 and C2310T in exon 5 of the progesterone receptor gene was investigated. The results of this study did not show a significant association between the aforementioned polymorphisms and recurrent miscarriage [44].

In Taiwan, Su et al. examined a group of 121 women who had a history of at least 2 miscarriages and 179 controls for the presence of two polymorphisms G1978T and G1031C in the progesterone receptor gene. The results of this study showed a significant association between the G1978T polymorphism and the occurrence of recurrent miscarriage, but no significant association was found between the G1031C polymorphism and the occurrence of recurrent miscarriage.

Conclusion

Leptin is produced by adipose tissue in the human body and, upon entering the blood, reduces appetite through various processes and ultimately prevents obesity. The FTO gene is located on chromosome 16, and based on studies, it seems that different types of the FTO gene are associated with obesity in humans. The aim of this study was to investigate the relationship between FTO gene polymorphisms and serum leptin levels in human blood. This study is a systematic review that was conducted by searching in English and Persian databases, science direct Scopus, PubMed, magiran, SID, and Google Scholar. In the initial review, by searching for appropriate keywords in world articles in both English and Persian and without time limits, 48

articles were found, of which 17 articles that met the inclusion criteria were carefully reviewed. Studies show that in 10 studies, there is a positive correlation between mRNA polymorphisms of the FTO gene and serum leptin levels, and in 4 studies, no significant relationship was seen between these two components, and in 3 studies, increased FTO gene expression caused a decrease in leptin levels. The TT genotype in the rs9939609 polymorphism of the FTO gene caused a greater increase in leptin levels than other genotypes (AA+AT). FTO activity may also be useful in improving leptin sensitivity for treatment. Most studies show that there is a positive relationship between FTO gene expression and serum leptin levels, and its reduction in expression causes obesity and increased BMI. Therefore, leptin could be a possible mediator that causes the relationship between FTO polymorphisms and obesity. Given the contradictory results, further studies in this field and a comprehensive study of the relationship of all types of FTO polymorphisms, serum leptin levels, and obesity are recommended.

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Authors' Contributions

All authors contributed to data analysis, drafting, and revising of the paper and agreed to be responsible for all the aspects of this work.

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