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Review Article

Obesity and Insulin Resistance

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ABSTRACT

Insulin resistance and obesity are two health disorders associated with decreased insulin action, weight gain, and body fat accumulation. These two conditions can possess a significant effect on health and raise the chronic disease risk. There is a close relationship between obesity and insulin resistance, as people who suffer from obesity possibly develop insulin resistance. Excess body fat causes excess fatty tissue around vital organs like the pancreas and liver, hindering their insulin response. blood sugar high levels lead for type 2 diabetes risk.

When the body is unable to use insulin effectively, resistance occurs resulting in high blood sugar levels. Several factors may play a role in the development of insulin resistance, including hormones secreted by adipose tissue, like adiponectin, which is useful for enhancing insulin sensitivity. However, its levels decrease in people who suffer from overweight and obesity, and thus their resistance to insulin increases, as well the hepatonctin, which is the hormone secreted by the liver and has a role in controlling blood sugar levels and metabolism.

Additionally, obesity increases the risk of developing many other diseases that can be enhanced by the existence of insulin resistance, so it is important to monitor and manage weight properly to prevent these conditions. Regulating insulin resistance and obesity requires lifestyle changes like regular physical activity and eating healthy, balanced foods. In some cases, you may need drug or insulin therapy to control blood sugar levels and improve the body's response to insulin.

Keywords: Insulin, resistance, obesity, hormones, diabetic.

INTRODUCTION

Insulin resistance is when cells in muscles, fat and liver don't respond well to insulin and can't easily take up glucose from your blood. As a result, pancreas makes more insulin to help glucose enter your cells. The insulin hormone produced from β cells of the Langerhans islets, regulates the metabolism of fats, carbohydrates and proteins. It facilitates glucose absorption and other molecules from the blood stream for fat tissues, skeletal muscles, and the liver. However, when there is a reduction in signaling of insulin, particularly in the protein kinase B axis, insulin receptor substrate and phosphoinositide-3-kinase, resistance occurs. This condition can significantly impact the insulin metabolic actions and is associated with obesity commonly, a major pathophysiologic factor in type 2 diabetes mellitus T2DM (Huhtala *et al.*, 2023).

Obesity arises from an excessive expansion of fat tissue due to increased nutrient intake and insufficient energy expenditure. On the other hand, DM is a complex chronic condition is recognized by hyperglycemia, resulting from insulin secretion deficiencies, action or both. The interplay between insulin resistance and obesity can lead to local and chronic low grade systemic inflammation, contributing to the development of DM associated with insulin resistance. Additionally, hyperinsulinemia and insulin resistance can also contribute to the onset of obesity. As a result, various mechanisms are involved in obesity-associated insulin resistance in T2DM, such as adipocyte dysfunction, inflammation, endoplasmic reticulum stress, oxidative stress, genetic factors, aging and hypoxia. Understanding these mechanisms is crucial for devising effective pharmacological strategies not only for treating T2DM and obesity but also for their prevention (Al-Taie and Al-Jawadi, 2019; Ota, 2014).

In individuals with obesity, fatty tissue releasing higher levels of glycerol, hormones, pro-inflammatory cytokines and non-esterified fatty acids that can play a role in the development of insulin resistance. Additionally, factors like adipose tissue hypoxia, oxidative stress, endoplasmic reticulum stress, genetic predisposition and lipodystrophy also contribute to insulin resistance (Zatterale *et al.*, 2020).

This review tackled the highlighted association between obesity and insulin resistance, along with the diseases linked to these conditions. It also aims to explore the most important hormones secreted by skeletal muscles, liver, and adipose tissue and their relevance to this disease. The review will cover the causes, risk factors, and complications associated with insulin resistance, in addition to discussing its diagnosis.

Overall, understanding the intricate relationship between insulin resistance, obesity and related diseases can pave the way for developing effective approaches to manage and prevent these conditions.

The syndrome of insulin-resistance

The insulin resistance syndrome refers to a collection of abnormalities that are observed in some individuals. These abnormalities encompass dyslipidemia, glucose intolerance, hemodynamic changes, elevated procoagulant factors, endothelial dysfunction, increased inflammatory markers, disturbances in uric acid metabolism, sleep-disordered breathing and heightened secretion of ovarian testosterone. Clinical conditions linked to insulin resistance include type T2DM, essential hypertension, polycystic ovary syndrome, nonalcoholic fatty liver disease, sleep apnea, cardio-vascular disease and certain forms of cancer (Kwaifa *et al.*, 2020).

Insulin resistance mechanisms

Physiologically, the actions of insulin at the whole-body level are affected by the interaction between other hormones. While plays a predominant role in driving metabolic processes in the nutritional state, it works in conjunction with insulin-like growth factor-1 (IGF-1) and growth hormone. Growth hormone, secreted in response to various stimuli including insulin, inhibits insulin induced hypoglycemia. Other counterregulatory hormones like catecholamines, glucocorticoids and glucagon which drive metabolic processes in the fasted state. Glucagon enhances ketogenesis, gluconeogenesis and glycogenolysis while catecholamines stimulate

glycogenolysis and lipolysis. Glucocorticoids enhance lipolysis, gluconeogenesis and muscle catabolism. While hormones overproduction may contribute with resistance to insulin in certain cases, but it does not account for the majority of cases of insulin resistance (Beaupere *et al.*, 2021).

Insulin resistance, a hallmark of metabolic diseases, is recognized by impaired insulin action in peripheral tissues. This leads to increase the liver glucose production, decreased uptake of muscle glucose, and fat accumulation in insulin sensitive organs like the liver, muscles and adipose tissue (Lee *et al.*, 2022).

Recent research has distinguished liver, skeletal muscle and adipocytes as secretory organs involved in metabolic processes (Zamboni *et al.*, 2022). These organs communicate with each other to regulate insulin sensitivity and energy homeostasis. For example, improved muscle function through exercise can impact overall peripheral insulin sensitivity, glucose and lipid metabolism. Skeletal muscle communicates with other organs and regulates the energy of the entire body through the myokines secretion, which have endocrine effects as shown in Fig. (1). This study categorizes liver, muscle and fat tissue as endocrine organ closely linked to metabolic diseases. The concept of myokines, adipokines, and hepatokines is introduced as basic molecular mechanisms that facilitate communication between metabolic organs in the endocrine system (Mancilla *et al.*, 2020).

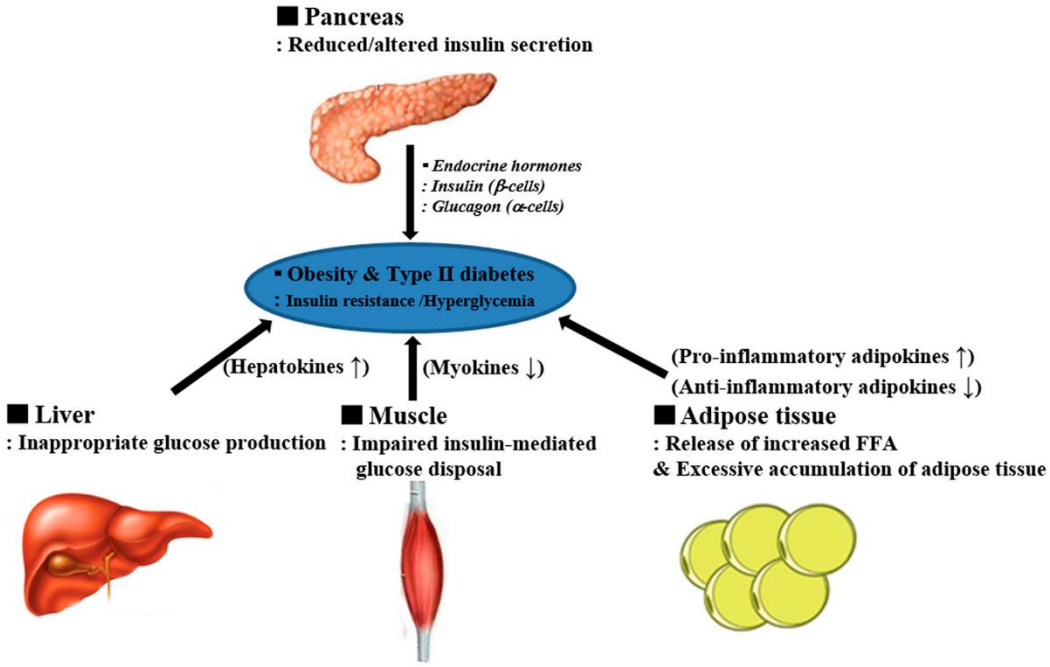

Fig. 1: Obesity and T2DM in peripheral tissues (Jin Oh *et al.*, 2017).

Myokines

A sedentary lifestyle, characterized by inactivity, excess the susceptibility to diseases like T2DM and obesity. Conversely, engaging in exercise or physical activity stimulates mitochondrial and metabolic adaptations that enhance the functioning of various organs and energy metabolism (Dan dan Li *et al.*, 2022).

Recently, (Singh *et al.*, 2021) reported that exercise training leads to a noticeable increase in energy-dissipating beige adipocytes in subcutaneous white adipose tissue (scWAT) in mice, improved whole-body mitochondrial function and glucose metabolism were observed compared to sedentary mice, suggesting that exercise induces metabolic adaptation to enhance overall energy metabolism. These results indicate that muscle-derived factors resulting from muscle contraction impact scWAT.

Skeletal muscle, comprising around 40% of body weight, has been identified as a secretory organ, releasing various cytokines and peptides collectively known as "myokines" (Iberite *et al.*, 2022). Myokines have a big role in muscle growth and regeneration (myogenesis) and also facilitate connection with other organs like liver, pancreas and adipose tissue. Because myokine production is affected by physical inactivity, muscle contraction can alter the pattern of myokine production and responses. These myokines are essential for exercise induced adaptations in skeletal muscle, including fatty acid oxidation, lipolysis and glucose disposal thus underscoring their significance in preventing and treating T2DM Fig. (2) (Balakrishnan and Thurmond, 2022).

In summary, the relationship among metabolic diseases and sedentary lifestyles like obesity and T2DM can be explained by the influence of myokines and their mechanisms, which play a pivotal role in the positive effects of exercise on whole-body energy metabolism and overall health (Chen *et al.*, 2024).

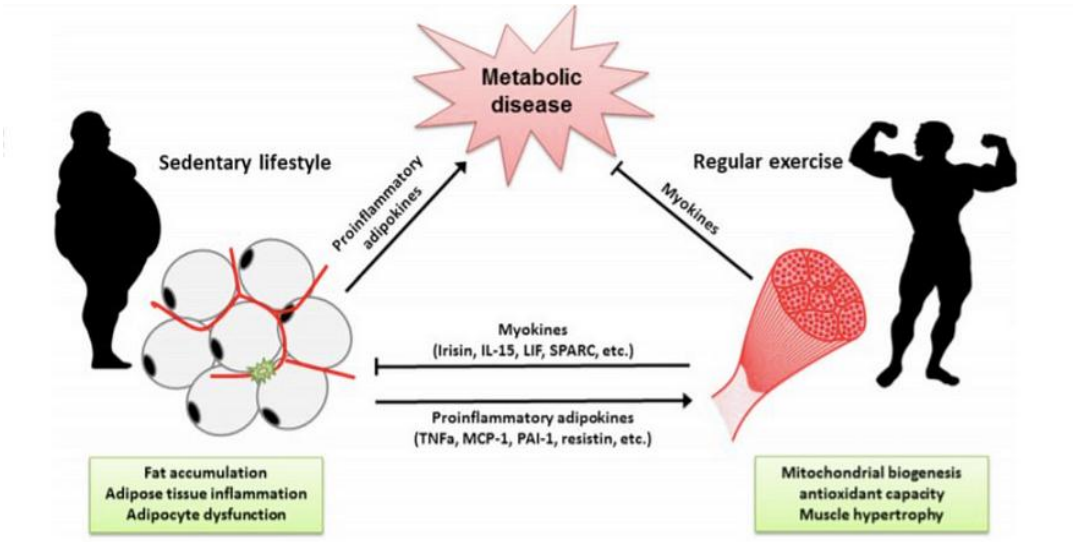

Fig. 2: Relationship between adipose tissue derived adipokines and skeletal muscle derived myokines (Huh, 2018).

Adipokines

Adipose tissue functions as an endocrine organ and plays an important role in metabolic syndrome development through the release of adipocytokines (Baygutalp, 2021). Subclinical hypothyroidism refers to a compensatory increase in thyroid-stimulating hormone (TSH) levels to maintain normal thyroid hormone levels before they drop below the normal range. It is considered one of the risk factors contributing to metabolic syndrome, as TSH receptors have been found in adipose tissue, indicating their involvement in regulating adipose tissue metabolism. Previous research has demonstrated that changes in thyroid hormones in individuals with thyroid dysfunction can alter the secretion of adipocytokines (Wu *et al.*, 2021).

Among these adipocytokines, vaspin is a member of the serine protease inhibitor family and is known for its role as an insulin-sensitizing adipocytokine. However, there are conflicting findings regarding the influence of thyroid hormones on the regulation of vaspin (Ghali *et al.*, 2024; Pilarski *et al.*, 2023).

Adiponectin

Adiponectin, predominantly found in adipocytes, plays a crucial role in exhibiting significant properties that counteract diabetes, atherosclerosis, and inflammation. In individuals with obesity, T2DM and other insulin-resistant conditions, circulating levels of adiponectin are lower when compared to lean, normal, glucose-tolerant, and insulin-sensitive individuals. Notably, it has been

observed that as insulin sensitivity improves and weight loss occurs, adiponectin expression in adipose tissue increases, leading to elevated secretion of this hormone Fig. (3). There is a suggestion
that glitazones, known for their insulin-sensitizing effects, may, in part, achieve this by influencing the rise of adiponectin serum concentrations (Khoramipour, 2021).

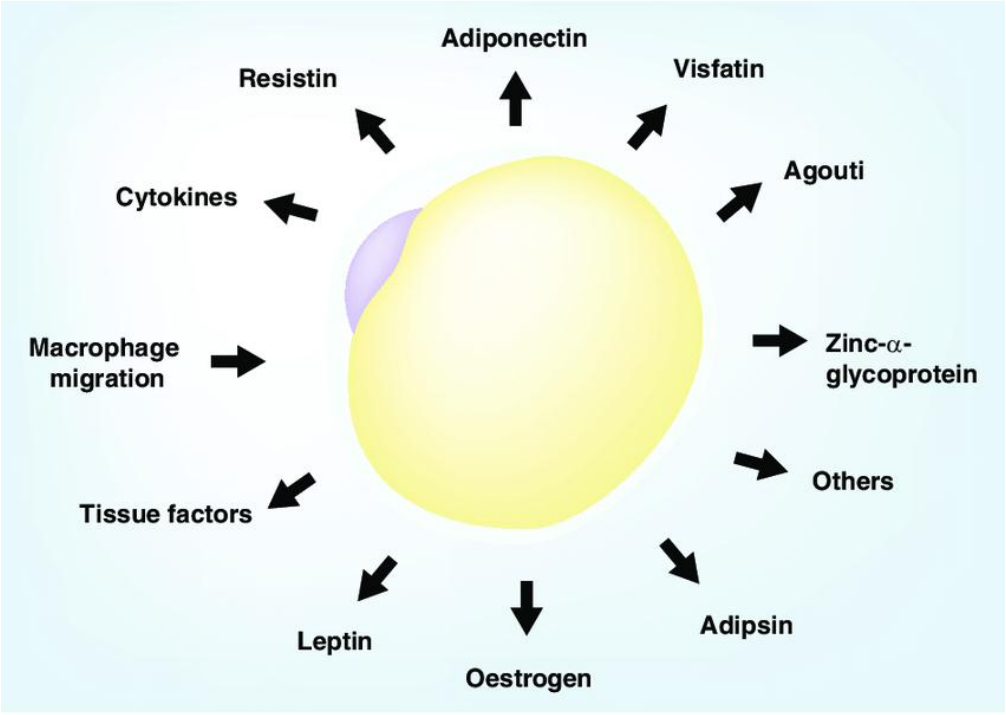

Fig. 3: An adipocyte and the factors that are known to be released from adipocytes (Russell, 2010).

Hepatokines

It is recently discovered proteins produced in the liver which function as new hormones, impacting metabolic conditions either positively or negatively (Choi, 2016). These effects are mediated through endocrine, paracrine and autocrine signaling, within the liver itself and in other tissues. However, hepatokines show a more significant influence on skeletal muscle and adipose tissue, which indicates the relationship of an endocrine dependent. They act by communicating with cytokines released through these respective tissues (Oh *et al.*, 2016).

Fetuin-A, a glycoprotein predominantly expressed by the liver, is encoded by the AHSG gene located on chromosome 3q27. This gene position is associated with conditions like obesity, insulin resistance, type 2 diabetes, nonalcoholic fatty liver disease (NAFLD), and metabolic syndrome (Chung and Choi, 2020). Fetuin-A's concentration disrupts the GLUT4 translocation and insulin signaling cascade in insulin target tissues, contributing to the metabolic patterns seen in these diseases (Iroz *et al.*, 2015). Its involvement in these metabolic disorders also includes harm to pancreatic β cells (Shen *et al.*, 2015).

On the contrary, physical activity-induced reduction in fetuin A levels may have an advantageous effect by decreaseing visceral adipose tissue through the reduction of available free fatty acids (FFA). This, in turn, facilitates the capture of triglycerides and their storage in white adipose tissue, indirectly mitigating insulin resistance. Lower fetuin-A levels might help control to buildup of visceral adipose tissue through reducing macrophage activation, which is pro inflammatory in nature (Gonzalez-Gil *et al.*, 2020).

Symptoms of insulin resistance

Some signs are (Guerra *et al.*,2021):

1. The waistline over 35 inches in women and 40 inches in men.
2. 130/80 or higher blood pressure readings.
3. Level of fasting glucose overthan 100 mg/dL.
4. level of fasting triglyceride overthan 150 mg/dL.
5. High density lipoprotein (HDL) level < 50 mg/dL in women and < 40 mg/dL in men.
6. Skin tags.
7. Acanthosis nigricans is a patch of dark, velvety skin.

Insulin Resistance (Risks and Causes)

Several things that increase the possibility of resistance (Wu *et al.,* 2014):

1. Gestational diabetes.
2. Polycystic ovary syndrome and nonalcoholic fatty liver disease.
3. Smoking.
4. Obesity, is especially related to belly fat.
5. Idleness as a lifestyle.
6. Attaining forty-five years of age or older.
7. A diet high in carbohydrates.
8. A family history of diabetes.
9. Sleep problems like sleep apnea.
10. Taking antipsychotic medications, stimulants and HIV medications.
11. Hormonal disorders like acromegaly and Cushing's syndrome.

Insulin resistance complications

Noted that if metabolic syndrome is not treated, it may lead to (Ighbariya *et al.*, 2017) as looks in Fig. (4):

1. Heart attack.
2. Severe low blood sugar.
3. Severe high blood sugar.
4. Eye problems.
5. Alzheimer's disease.
6. Stroke.
7. Cancer.
8. Kidney disease.

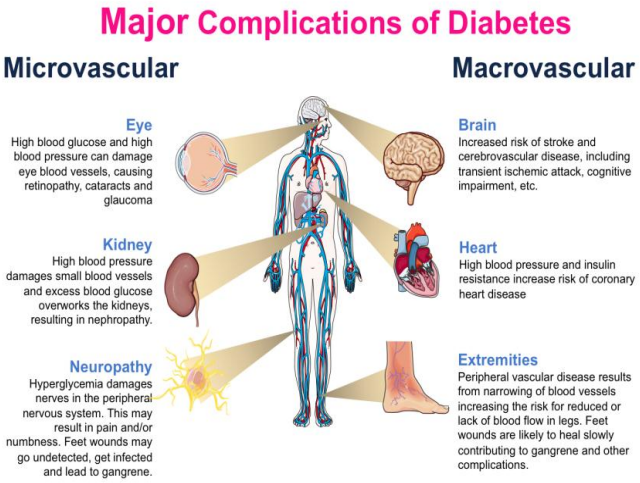

Fig. 4: Major microvascular and macrovascular complications associated with diabetesmellitus (Gazi and Rahman, 2021).

Insulin resistance (diagnosis and tests)

The doctor must take into consideration some things when diagnosing insulin resistance (Gołacki *et al*., 2022):

1. Family's medical history.
2. Physical exam such weigh and blood pressure.
3. Blood tests (FBS, RBS and Hba1c).

Treatment and prevention of insulin resistance

1. Strive for a healthy weight: You must set the goal of losing weight in mind, and if you are unable to do so, you must ask your doctor or any certified nutritionist.
2. Eat a healthy diet: Vegetables, fruits, nuts, whole grains, fish, beans, legumes, and lean protein.
3. Exercise: A person must practice brisk walking as a form of moderate activity for at least 30 minutes a day, five or more days a week.
4. Take medications: Take the medication metformin to help keep your blood sugar under control, which your doctor may prescribe for you (Wolosowicz *et al*., 2022) as shown in Fig. (5).

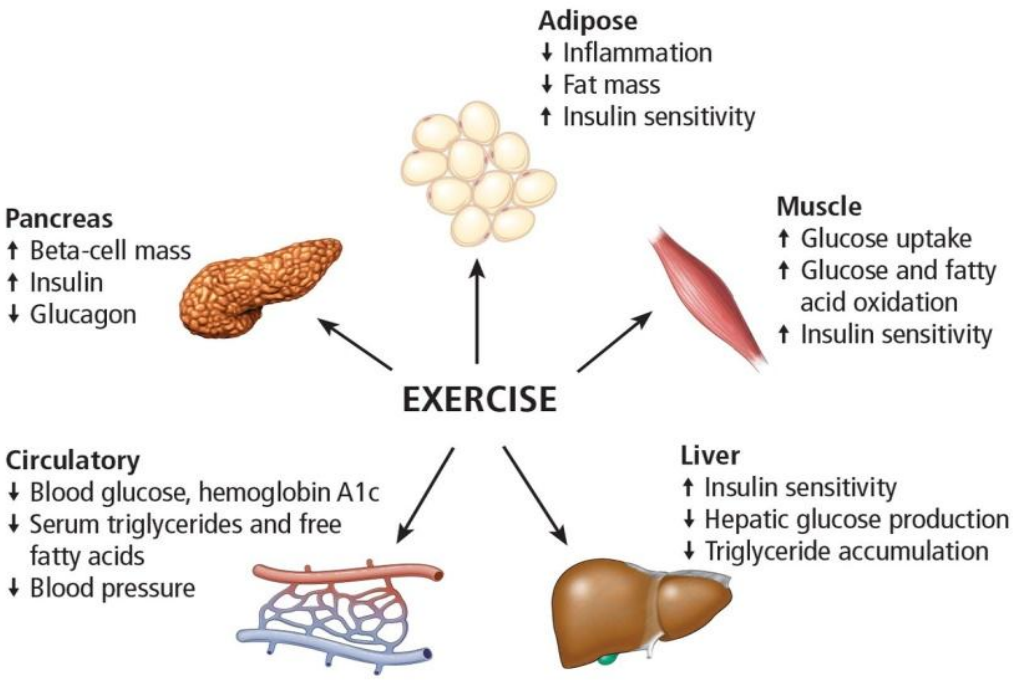

Fig. 5: Metabolic effects of exercise in patients with type 2 diabetes on some tissues (Kirwan *et al.*, 2017).

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السمنة ومقاومة الانسولين

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الملخص

تعد مقاومة الأنسولين والسمنة من الاضطرابات الصحية المرتبطة بانخفاض عمل الأنسولين وزيادة الوزن وتراكم الدهون في الجسم. يمكن أن يكون لهذين الشرطين تأثير كبير على الصحة ويزيدان من خطر الإصابة بالأمراض المزمنة. هناك علاقة وثيقة بين السمنة ومقاومة الأنسولين، فالأشخاص الذين يعانون من السمنة من المحتمل أن يصابوا بمقاومة الأنسولين. تؤدي الدهون الزائدة في الجسم إلى زيادة الأنسجة الدهنية حول الأعضاء الحيوية مثل البنكرياس والكبد، مما يعيق استجابتها للأنسولين. ارتفاع مستويات السكر في الدم يؤدي إلى خطر الإصابة بمرض السكري من النوع 2.

عندما يكون الجسم غير قادر على استخدام الأنسولين بشكل فعال، تحدث المقاومة مما يؤدي إلى ارتفاع مستويات السكر في الدم. قد تلعب عدة عوامل دورًا في تطور مقاومة الأنسولين، بما في ذلك الهرمونات التي تفرزها الأنسجة الدهنية، مثل الأديبونيكتين، وهو مفيد لتعزيز حساسية الأنسولين. إلا أن مستوياته تنخفض لدى الأشخاص الذين يعانون من الوزن الزائد والسمنة، وبالتالي تزداد مقاومتهم للأنسولين، وكذلك هرمون الهيباتونكتين وهو الهرمون الذي يفرزه الكبد وله دور في التحكم في مستويات السكر في الدم والتمثيل الغذائي.

بالإضافة إلى ذلك، تزيد السمنة من خطر الإصابة بالعديد من الأمراض الأخرى التي يمكن أن تتعزز بسبب وجود مقاومة الأنسولين، لذلك من المهم مراقبة الوزن وإدارته بشكل صحيح للوقاية من هذه الحالات. يتطلب تنظيم مقاومة الأنسولين والسمنة تغييرات في نمط الحياة مثل ممارسة النشاط البدني بانتظام وتناول الأطعمة الصحية والمتوازنة. في بعض الحالات، قد تحتاج إلى علاج بالأدوية أو بالأنسولين للتحكم في مستويات السكر في الدم وتحسين استجابة الجسم للأنسولين.

الكلمات الدالة: الأنسولين، المقاومة، السمنة، الهرمونات، السكري.