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EFFECTS OF SUPPLEMENTATION, DIET AND FASTING ON INSULIN RESISTANCE

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ABSTRACT

Insulin resistance is a condition in which the decreased tissue sensitivity to insulin, which consequently leads to impaired glucose metabolism. This condition is the starting point for many chronic diseases, primarily type 2 diabetes, metabolic syndrome, PCOS, NAFLD and cardiovascular disease. This paper discusses the current state of knowledge on the effects of supplementation, diet and intermittent fasting on improving insulin sensitivity. The pathophysiological mechanisms of insulin resistance, diagnostic methods (including HOMA-IR, OGTT, metabolic clamp method), and its impact on the development and course of chronic diseases are presented.

Special attention was given to supplements such as vitamin D, magnesium, zinc, Myo-inositol and omega-3 fatty acids, which through a variety of mechanisms can support glucose metabolism and improve insulin action. Also discussed was the role of caloric restriction and intermittent fasting, which have positive effects on body weight, metabolic profile and, most importantly, insulin sensitivity.

The aim of the study was to highlight the importance of lifestyle modification and dietary interventions in the prevention and treatment of insulin resistance and its consequences. Due to the increasing prevalence of these disease entities in the general population, including among increasingly younger patients, it is necessary to implement appropriate preventive and therapeutic strategies in daily clinical practice.

KEYWORDS

Insulin, Insulin Sensitivity, Lifestyle Modification, Glucose Metabolism, Nutritional Interventions, Insulin Resistance, Supplementation, Diet, Intermittent Fasting

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Introduction

The modern world, despite technological advances and increasing public awareness of one's own health, presents people with new challenges that increasingly lead to metabolic disorders. The lifestyle of most people is based on constant haste and stress, sedentary lifestyles and unlimited access to high-calorie, processed and nutrient-poor foods which is becoming a major factor in the development of insulin resistance and, consequently, obesity and many other metabolic diseases.^{1,2}

More and more people are neglecting regular physical activity, healthy eating and sleep hygiene which is the cause of disturbed homeostasis of the body. Obesity, especially abdominal obesity, is one of the main factors causing insulin resistance and its formation strongly correlates with excessive caloric supply, lack of exercise and chronic stress. Excessive fatigue and persistent stress put the body in a constant state of readiness which further causes hormonal and inflammatory disorders consequently exacerbating insulin resistance. In turn, a sedentary lifestyle impairs the ability of muscles to uptake glucose which translates into permanently elevated blood glucose levels and causes excessive accumulation of visceral fat, which has a pro-inflammatory effect.^{32,39,40}

In an era of increasing incidence of disease and growing public awareness of their health, preventive measures are becoming increasingly important. Understanding the impact of lifestyle and, in particular, proper nutrition with simultaneous appropriately selected supplementation is important not only in the context of diseases of civilization, but also in the development of effective therapeutic strategies.^{38,40}

This paper is a review, based on an analysis of clinical and observational studies found in PubMed, and indicates possible directions for intervention in daily medical practice.

Methods

This study analyzed articles and studies found in PubMed using keywords such as “insulin resistance,” “insulin sensitivity,” “glucose metabolism,” “prediabetic state,” “type 2 diabetes,” “hyperinsulinemia,” “insulin resistance,” ‘metabolic syndrome’, ‘intermittent fasting’, ‘vitamin D in insulin resistance’, ‘myo-inositol and PCOS’, ‘supplementation in insulin resistance’, ‘dietary window’, ‘glycemic index’.

The work is based on randomized trials, meta-analyses and systematic reviews with publication from 1995 to the present with special emphasis on papers published after 2000.

Insulin sensitivity and insulin resistance

Insulin sensitivity is the ability of cells to respond to insulin, the hormone responsible for regulating blood glucose levels. A high level of insulin sensitivity is key to maintaining normal metabolism and overall health, ensuring efficient glucose utilization and preventing excessive insulin production as a compensatory mechanism. Insulin sensitivity is multifactorial determined, influenced by aspects such as lifestyle, body weight and also the amount of sleep and stress.³¹

Insulin resistance is called a condition in which there is reduced sensitivity of target tissues in particular skeletal muscle, liver and adipose tissue to insulin despite normal or elevated serum levels. This results in impaired glucose metabolism consequently causing hyperglycemia and other metabolic disorders.²⁹

Clinical implications of insulin resistance

Insulin resistance is the starting point of many chronic diseases such as type 2 diabetes, polycystic ovarian syndrome, cardiovascular disease, metabolic syndrome or non-alcoholic fatty liver disease (NAFLD). There is growing evidence of the impact of insulin resistance on cognitive impairment through disorganization of glucose metabolism in the brain consequently leading to dementia or even Alzheimer's disease. Therefore, its early recognition and treatment is so important to prevent the development and progression of these conditions,^{43,44}.

Understanding the mechanisms that affect insulin sensitivity is essential for the diagnosis, treatment and, above all, prevention of such disease entities as type 2 diabetes, metabolic syndrome, atherosclerosis, non-alcoholic fatty liver disease (NAFLD), chronic cardiovascular disease or polycystic ovary syndrome (PCOS) as well as dementia or Alzheimer's disease.^{30,32,41}

Pathogenesis of insulin resistance

Insulin resistance is defined as the inability of tissues to respond to normal insulin concentrations, i.e. higher concentrations of insulin are required to maintain adequate insulin action, that is, for cellular glucose uptake from plasma and glycogen synthesis^{1,39}.

There are several theories about the mechanisms of insulin resistance. After food intake, plasma glucose levels rise to a threshold value that stimulates pancreatic beta cells to secrete insulin. Under physiological conditions, insulin promotes glucose uptake in skeletal muscle and adipose tissue, it also affects fat metabolism by increasing triglyceride storage and decreases lipolysis. Insulin is also involved in the metabolism of ketone bodies, reducing their concentration and affecting protein metabolism. However, the prevailing sedentary lifestyle and excess food intake these days causes an unphysiological increase in plasma glucose levels. Chronic hyperglycemia disrupts the physiological state and causes a continuous buildup of insulin concentrations and eventually leads to chronic hyperinsulinemia, followed by pancreatic beta-cell failure and eventually diabetes.^{2,40}.

Diagnosis

Early signs of insulin resistance that may indicate severe insulin resistance syndromes include characteristic phenotypic changes, which include skin lesions such as growths, dark acanthosis in the groin, abdomen or axilla, hyperpigmentation and epidermal thickening as well as abnormal adipose tissue topography, abnormal musculature, acromegaly features and growth abnormalities³⁸.

Depending on the type of insulin resistance, the distribution of subcutaneous adipose tissue sometimes varies; excessive fat accumulation can be observed on the face and neck, on the trunk or abdomen, and a “buffalo hump” is also typical.²⁸

The gold standard - the metabolic clamp method

A test involving the intravenous infusion of insulin, maintaining its serum concentration at about 100 Miu/L, and the intravenous infusion of glucose keeping the glycemia within the normal range, or about 100mg/dl. This procedure results in complete blockage of insulin production by the pancreas and glucose production by the liver and so the amount of glucose administered reflects its tissue consumption i.e. indirectly the tissue sensitivity to insulin. The lower the glucose dose needed to maintain euglycemia, the greater the insulin resistance.^{19,37}

HOMA-IR method

The ratio of insulinemia to fasting glucose, which is the quotient of insulin concentration in uIU/ml and glucose concentration in mg/dl. A value > 0.3 indicates insulin resistance³⁷.

The HOMA-IR insulin resistance index is calculated according to the formula

Insulinemia [Uj./ml] x glycemia [mmol/l]/22.5³⁷

HOMA-IR under physiological conditions chooses 1.0⁴⁶

Diagnostic values are assumed¹⁹:

in adults a value >2.5

in children and adolescents:

a) At puberty 2.67 in boys, 2.22 in girls

b) Before puberty 5.22 in boys 3.82 in girls¹⁹

OGTT test

The oral glucose load test, the current gold standard for diagnosing diabetes, which involves administering 75g of glucose orally on an empty stomach and then assessing fasting venous blood glucose levels and 2 hours after the load.⁴⁵

Measurement of insulin and glucose during a glucose load test where insulin resistance may be indicated by¹⁹:

(i) fasting insulinemia value >15 (17) mIU/l

(ii) insulinemia value at 120 minutes of the Test >75 mIU/l

(iii) insulinemia value at any point in the test >150mIU/l¹⁹

Supplementation

Vitamin D

In skeletal muscle, adipose tissue and liver, vitamin D increases insulin receptor expression thereby improving insulin sensitivity^{3,4}.

A correlation between vitamin D deficiency and insulin resistance assessment has been proven in the HOMA-IR model, a measure of insulin resistance understood as the increase in insulin secretion required to maintain glycemia at normal levels, the inverse correlation becoming stronger in proportion to an increase in BMI5. It follows that vitamin D supplementation reduces the risk of insulin resistance. Vitamin D has also been shown to improve insulin receptor sensitivity and intracellular glucose transport^{6,7,8, 33,34,35}.

Magnesium

Magnesium is a major cofactor of enzymatic reactions. It regulates insulin signaling through phosphorylation of insulin receptor kinase and postreceptor insulin action and cellular glucose uptake^{9,10}.

Postreceptor insulin resistance is a clinical consequence of chronic magnesium deficiency¹¹.

There are hypotheses that magnesium supplementation in deficient individuals may improve fasting and postprandial glycemia and increase insulin sensitivity. Unfortunately, there is no large randomized clinical trial to confirm these speculations.¹²

Zinc

This is an essential micronutrient responsible for the processing, storage and secretion and action of insulin in pancreatic cells. Deficiency of this element is associated with insulin resistance and diabetes¹³

Based on a study on fifty-six women aged 25-45 years, BMI around 36 kg/m2 with documented insulin resistance, who supplemented with 30mg of zinc daily for 4 weeks, it was observed that there was a significant decrease in insulin and HOMA index in the zinc supplement group compared to placebo. Based on this, it is reasonable to believe that zinc supplementation has a beneficial effect on improving insulin sensitivity.¹⁴

Inositol

Myo-inositol inhibits duodenal glucose uptake and lowers blood glucose levels by competitive affinity for the same transporter and also improves muscle glucose uptake.³² Insulin resistance causes increased urinary loss of Myo-inositol by inhibiting glucose-mediated renal reabsorption of Mio-inositol³³.

Decreased concentrations of Myo-inositol decrease concentrations of D-chiro-inositol and deficiency of both forms causes insulin resistance in skeletal muscle, adipose tissue and liver³⁵.

Abnormal concentrations of Myo-inositol and D-chiro-inositol in urine and plasma may be an early indicator of insulin resistance³⁶.

In a study involving 80 postmenopausal women with metabolic syndrome, supplementation with 2g of Myo-inositol daily resulted in lower blood glucose levels, lower insulin levels and normalization of the HOMA-IR index, and improvements in BMI and a reduction in waist circumference were observed compared to baseline values after 12 months of supplementation. 20% of the women no longer had metabolic syndrome³⁴.

Supplementation with 2g of myo-inositol daily for 8 weeks has been shown to regulate insulin levels and lower fasting glucose.¹⁵

Polyunsaturated fatty acids

Omega-3 fatty acids are essential nutrients, as they can only be supplied exogenously. They act as metabolic precursors necessary to control inflammation in the body.¹⁶

Polyunsaturated omega-3 fatty acids include alpha-linolenic acid (ALA) and the longer-chain acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), which are obtained from seafood. Fatty acids are involved in the formation of cell membranes by maintaining their elasticity which aids intercellular communication and homeostasis of the body. Their main benefit is reducing inflammation, which is an important component of insulin resistance¹⁷

Studies have shown that obese mice fed a high-fat diet rich in omega-3 fatty acids where 60% of calories came from fat for 12 weeks increased their glucose tolerance and insulin sensitivity¹⁸

IR vs. diet

Insulin resistance is in most cases closely related to obesity so calorie restriction is essential to reduce body weight and improve insulin sensitivity. Obesity treatment guidelines recommend a deficit of 500-750 kcal/day in relation to total energy requirements, that is, the sum of basal metabolism and exercise-related energy expenditure. An average weight loss of 0.5-0.75 kg per week is recommended. US guidelines state that about 20-35% of daily calories should come from fat with a maximum of 10% from saturated fat, 15-20% from protein and the remainder from carbohydrates. For carbohydrates, it is emphasized to eat mainly complex carbohydrates and plenty of fresh fruits and vegetables.^{20,21}

When choosing a diet, the patient's dietary preferences should be taken into account mainly because in long-term follow-up (>1 year) it has been noted that weight loss is similar in both low-carbohydrate, Mediterranean, high-protein and low-glycemic index diets. A maintained caloric deficit plays a key role.²⁵

CALERIE 2 is a multicenter, randomized study to evaluate the effects of caloric restriction of 11.9% on cardiometabolic risk factors for 2 years on 220 young and middle-aged (21-50 years) subjects. Participants were both men and women without obesity. During the 2-year period, the calorie-restricted study group achieved a 10% reduction in body weight, with 71% loss of body fat. A significant and sustained reduction in all measured cardiometabolic risk factors including glucose tolerance and insulin sensitivity index was observed in this group. Insulin sensitivity increased significantly and fasting glucose and glucose-stimulated insulin levels decreased.^{22,23}

The CRON observational study, whose participants self-administered a caloric restriction of 1800 kcal/d with optimal nutrition for about 15 years. They noted extremely low fasting glucose and insulin levels and significant improvements in insulin sensitivity.²⁴

Intermittent fasting

Intermittent fasting means eating only within a certain time frame, outside of which a fasting period is in effect.²⁸

Intermittent fasting is able to reduce insulin resistance by reducing body weight due to restricted caloric intake. There is also a hypothesis that the restricted energy intake caused by intermittent fasting causes a prolonged decrease in insulin production which results in improved insulin sensitivity and glucose metabolism.^{25, 26}

A study was conducted on eight healthy men who were subjected to 20-hour intermittent fasting every other day for 15 days. Their average BMI was about 26 and daily physical activity was constant. It was observed that insulin-dependent total glucose absorption increased after the 20-hour fast, and there were higher plasma levels of adiponectin, a hormone that improves insulin sensitivity. This study showed that intermittent fasting can accelerate insulin-dependent glucose uptake.²⁷

Potential for future research

Some of the findings are inconclusive, not all of the studies mentioned in this review consider the dosage of the indicated supplements, and studies examining the simultaneous use of several supplements and the sum effect of supplementation and diet simultaneously were not included. It is worth considering new treatments with modern techniques for treating obesity and diabetes and also consider combining different treatments simultaneously. Establish unequivocally the impact of intestinal dysbiosis on insulin resistance and the role of probiotic and prebiotic therapy in its treatment. Clarify the impact of gut microbiota transplantation. Elaborate on the potential of gene therapy or immunotherapy in the treatment of IR. The mechanisms of insulin resistance should also be further investigated, taking into account genetic and molecular predispositions. Focus on the development of non-invasive tests for early detection of IR or the use of artificial intelligence that could estimate the risk of developing IR based on genetic or environmental factors.

Conclusions and clinical implications

This review underscores that insulin resistance is an increasingly commonly diagnosed metabolic disorder involving reduced tissue sensitivity to insulin action. Contemporary research clearly emphasizes the role of modifiable factors, primarily diet, supplementation and physical activity on tissue insulin sensitivity.

The review presents evidence of the beneficial effects of supplementation with vitamin D, magnesium, zinc, myo-inositol and omega-3 fatty acids, which promote glucose metabolism and improve insulin sensitivity. Of equal importance are dietary interventions, both caloric deficit and the quality of food consumed and strategies such as intermittent fasting, which has been shown to have positive effects on metabolic parameters and insulin levels.

Given the increasing prevalence of insulin resistance in the general population, especially among increasingly younger people, our priority should be to implement effective preventive and therapeutic strategies in clinical practice. Patient education, early diagnosis and individually tailored nutritional interventions and supplementation can effectively improve quality of life and reduce the risk of metabolic complications.

Author's contribution

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REFERENCES

1. Lee, S., Park, S., & Choi, C. S. (2021d). Insulin resistance: From mechanisms to therapeutic strategies. *Diabetes & Metabolism Journal*, 46(1), 15–37. <https://doi.org/10.4093/dmj.2021.0280>
2. Gastaldelli, A. (2022b). Measuring and estimating insulin resistance in clinical and research settings. *Obesity*, 30(8), 1549–1563. <https://doi.org/10.1002/oby.23503>
3. Szablewski, L. (2024). Changes in Cells Associated with Insulin Resistance. *International Journal of Molecular Sciences*, 25(4), 2397. <https://doi.org/10.3390/ijms25042397>
4. Calle, C., Maestro, B., & García-Arencibia, M. (2008). Genomic actions of 1,25-dihydroxyvitamin D3 on insulin receptor gene expression, insulin receptor number and insulin activity in the kidney, liver and adipose tissue of streptozotocin-induced diabetic rats. *BMC Molecular Biology*, 9(1), 65. <https://doi.org/10.1186/1471-2199-9-65>
5. Argano, C., Mirarchi, L., Amodeo, S., Orlando, V., Torres, A., & Corrao, S. (2023b). The role of vitamin D and its molecular bases in insulin resistance, diabetes, metabolic syndrome, and cardiovascular disease: state of the art. *International Journal of Molecular Sciences*, 24(20), 15485. <https://doi.org/10.3390/ijms242015485>
6. Rafiq, S., & Jeppesen, P. B. (2021). Vitamin D Deficiency Is Inversely Associated with Homeostatic Model Assessment of Insulin Resistance. *Nutrients*, 13(12), 4358. <https://doi.org/10.3390/nu13124358>
7. Mo, M., Shao, B., Xin, X., Luo, W., Si, S., Jiang, W., Wang, S., Shen, Y., Wu, J., & Yu, Y. (2021). The Association of Gene Variants in the Vitamin D Metabolic Pathway and Its Interaction with Vitamin D on Gestational Diabetes Mellitus: A Prospective Cohort Study. *Nutrients*, 13(12), 4220. <https://doi.org/10.3390/nu13124220>
8. Argano, C., Mirarchi, L., Amodeo, S., Orlando, V., Torres, A., & Corrao, S. (2023). The role of vitamin D and its molecular bases in insulin resistance, diabetes, metabolic syndrome, and cardiovascular disease: state of the art. *International Journal of Molecular Sciences*, 24(20), 15485. <https://doi.org/10.3390/ijms242015485>
9. Wang M., Chen Z., Hu Y., Wang Y., Wu Y., Lian F., Li H., Yang J., Xu X. The Effects of Vitamin D Supplementation on Glycemic Control and Maternal-Neonatal Outcomes in Women with Established Gestational Diabetes Mellitus: A Systematic Review and Meta-Analysis. *Clin. Nutr.* 2021;40:3148–3157. doi: 10.1016/j.clnu.2020.12.016
10. Huang S., Fu J., Zhao R., Wang B., Zhang M., Li L., Shi C. The Effect of Combined Supplementation with Vitamin D and Omega-3 Fatty Acids on Blood Glucose and Blood Lipid Levels in Patients with Gestational Diabetes. *Ann. Palliat. Med.* 2021;10:5652–5658. doi: 10.21037/apm-21-1018.
11. Christiano Argano, Mirarchi, L., Amodeo, S., Orlando, V., Torres, A., & Corrao, S. (2023). The Role of Vitamin D and Its Molecular Bases in Insulin Resistance, Diabetes, Metabolic Syndrome, and
12. Cardiovascular Disease: State of the Art. *International Journal of Molecular Sciences*, 24(20), 15485–15485. <https://doi.org/10.3390/ijms242015485>
13. Günther, T. (2011). Magnesium in bone and the magnesium load test. *Magnesium Research*, 24(4), 223–224. <https://doi.org/10.1684/mrh.2011.0297>
14. Saris, N.-E. L., Mervaala, E., Karppanen, H., Khawaja, J. A., & Lewenstam, A. (2000). Magnesium. *Clinica Chimica Acta*, 294(1-2), 1–26. [https://doi.org/10.1016/s0009-8981\(99\)00258-2](https://doi.org/10.1016/s0009-8981(99)00258-2)
15. Barbagallo, M. (2015). Magnesium and type 2 diabetes. *World Journal of Diabetes*, 6(10), 1152. <https://doi.org/10.4239/wjd.v6.i10.115>
16. McCarty, M. F. (2005). Nutraceutical resources for diabetes prevention – an update. *Medical Hypotheses*, 64(1), 151–158. <https://doi.org/10.1016/j.mehy.2004.03.036>
17. Dubey, P., Thakur, V., & Chattopadhyay, M. (2020). Role of minerals and trace elements in diabetes and insulin resistance. *Nutrients*, 12(6), 1864. <https://doi.org/10.3390/nu12061864>
18. Marreiro, D. D. N., Geloneze, B., Tambascia, M. A., Lerário, A. C., Halpern, A., & Cozzolino, S. M. F. (2006). Effect of zinc supplementation on serum leptin levels and insulin resistance of obese women. *Biological Trace Element Research*, 112(2), 109–118. <https://doi.org/10.1385/bter:112:2:109>
19. DiNicolantonio, J. J., & O’Keefe, J. H. (2022). Myo-inositol for insulin resistance, metabolic syndrome, polycystic ovary syndrome and gestational diabetes. *Open Heart*, 9(1), e001989. <https://doi.org/10.1136/openhrt-2022-001989>
20. González-Rodríguez, L. G., Aparicio, A., López-Sobaler, A. M., & Ortega, R. M. (2013). Omega 3 and omega 6 fatty acids intake and dietary sources in a representative sample of Spanish adults. *International Journal for Vitamin and Nutrition Research*, 83(1), 36–47. <https://doi.org/10.1024/0300-9831/a000143>
21. Gammone, M. A., Riccioni, G., Parrinello, G., & D’Orazio, N. (2018). Omega-3 polyunsaturated fatty acids: benefits and endpoints in sport. *Nutrients*, 11(1), 46. <https://doi.org/10.3390/nu11010046>
22. Leonardi, B. F., Gosmann, G., & Zimmer, A. R. (2020). Modeling Diet-Induced Metabolic Syndrome in rodents. *Molecular Nutrition & Food Research*, 64(22). <https://doi.org/10.1002/mnfr.202000249>
23. Przemysława Jarosz-Chobot, B. G.-O. Ocena wrażliwości na insulinę. *medycyna praktyczna dla lekarzy*.
24. Wadden, T. A., Tronieri, J. S., & Butryn, M. L. (2020). Lifestyle modification approaches for the treatment of obesity in adults. *American Psychologist*, 75(2), 235–251. <https://doi.org/10.1037/amp0000517>

25. *Handbook of Obesity Treatment, second edition.* (n.d.). Google Books. https://books.google.pl/books?hl=pl&lr=&id=P2s8DwAAQBAJ&oi=fnd&pg=PP1&ots=LjyvslAFXq&sig=r09hiCD40V6pqQAdUAYDZklyChs&redir_esc=y#v=onepage&q&f=false
26. Napoleão, A., Fernandes, L., Miranda, C., & Marum, A. P. (2021). Effects of Calorie Restriction on Health Span and Insulin Resistance: Classic Calorie Restriction Diet vs. Ketosis-Inducing Diet. *Nutrients*, 13(4), 1302. <https://doi.org/10.3390/nu13041302>
27. Kraus, W. E., Bhapkar, M., Huffman, K. M., Pieper, C. F., Das, S. K., Redman, L. M., Villareal, D. T., Rochon, J., Roberts, S. B., Ravussin, E., Holloszy, J. O., & Fontana, L. (2019). 2 years of calorie restriction and cardiometabolic risk (CALERIE): exploratory outcomes of a multicentre, phase 2, randomised controlled trial. *The Lancet Diabetes & Endocrinology*, 7(9), 673–683. [https://doi.org/10.1016/s2213-8587\(19\)30151-2](https://doi.org/10.1016/s2213-8587(19)30151-2)
28. Most, J., Tosti, V., Redman, L. M., & Fontana, L. (2016). Calorie restriction in humans: An update. *Ageing Research Reviews*, 39, 36–45. <https://doi.org/10.1016/j.arr.2016.08.005>
29. Makris A, & Foster GD (2011). Dietary approaches to the treatment of obesity. *Psychiatric Clinics of North America*, 34(4), 813–827.
30. Antoni, R., Johnston, K. L., Collins, A. L., & Robertson, M. D. (2017). Effects of intermittent fasting on glucose and lipid metabolism. *Proceedings of the Nutrition Society*, 76(3), 361–368. <https://doi.org/10.1017/s0029665116002986>
31. Vasim, I., Majeed, C. N., & DeBoer, M. D. (2022). Intermittent fasting and metabolic health. *Nutrients*, 14(3), 631. <https://doi.org/10.3390/nu14030631>
32. Albosta, M., & Bakke, J. (2021). Intermittent fasting: is there a role in the treatment of diabetes? A review of the literature and guide for primary care physicians. *Clinical Diabetes and Endocrinology*, 7(1). <https://doi.org/10.1186/s40842-020-00116-1>
33. Halberg, N., Henriksen, M., Söderhamn, N., Stallknecht, B., Ploug, T., Schjerling, P., & Dela, F. (2005). Effect of intermittent fasting and refeeding on insulin action in healthy men. *Journal of Applied Physiology*, 99(6), 2128–2136. <https://doi.org/10.1152/japplphysiol.00683.2005>
34. Angelidi, A. M., Filippaios, A., & Mantzoros, C. S. (2021). Severe insulin resistance syndromes. *Journal of Clinical Investigation*, 131(4). <https://doi.org/10.1172/jci142245>
35. Lee, S., Park, S., & Choi, C. S. (2021). Insulin resistance: From mechanisms to therapeutic strategies. *Diabetes & Metabolism Journal*, 46(1), 15–37. <https://doi.org/10.4093/dmj.2021.0280>
36. Alagiakrishnan, K., & Halverson, T. (2024). Role of peripheral and central insulin resistance in neuropsychiatric disorders. *Journal of Clinical Medicine*, 13(21), 6607. <https://doi.org/10.3390/jcm13216607>
37. Park, S. S., & Seo, Y. (2020). Excess accumulation of lipid impairs insulin sensitivity in skeletal muscle. *International Journal of Molecular Sciences*, 21(6), 1949. <https://doi.org/10.3390/ijms21061949>
38. Bevilacqua, A., & Bizzarri, M. (2018). Inositols in insulin signaling and glucose metabolism. *International Journal of Endocrinology*, 2018, 1–8. <https://doi.org/10.1155/2018/1968450>
39. Croze, M. L., Gélou, A., & Soulage, C. O. (2015). Abnormalities in myo-inositol metabolism associated with type 2 diabetes in mice fed a high-fat diet: benefits of a dietary myo-inositol supplementation. *British Journal of Nutrition*, 113(12), 1862–1875. <https://doi.org/10.1017/s000711451500121x>
40. Clements, R. S., & Darnell, B. (1980). Myo-inositol content of common foods: development of a high-myo-inositol diet. *American Journal of Clinical Nutrition*, 33(9), 1954–1967. <https://doi.org/10.1093/ajcn/33.9.1954>
41. Greene, D. A., & Lattimer, S. A. (1982). Sodium- and energy-dependent uptake of myo-inositol by rabbit peripheral nerve. Competitive inhibition by glucose and lack of an insulin effect. *Journal of Clinical Investigation*, 70(5), 1009–1018. <https://doi.org/10.1172/jci110688>
42. Dinicola, S., Minini, M., Unfer, V., Verna, R., Cucina, A., & Bizzarri, M. (2017). Nutritional and Acquired Deficiencies in Inositol Bioavailability. Correlations with Metabolic Disorders. *International Journal of Molecular Sciences*, 18(10), 2187. <https://doi.org/10.3390/ijms18102187>
43. Zeng, P., Cai, X., Yu, X., Huang, L., & Chen, X. (2023). HOMA-IR is an effective biomarker of non-alcoholic fatty liver disease in non-diabetic population. *Journal of International Medical Research*, 51(10). <https://doi.org/10.1177/03000605231204462>
44. Garaulet, M., Ordovás, J. M., & Madrid, J. A. (2010). The chronobiology, etiology and pathophysiology of obesity. *International Journal of Obesity*, 34(12), 1667–1683. <https://doi.org/10.1038/ijo.2010.118>
45. Saklayen, M. G. (2018). The global epidemic of the metabolic syndrome. *Current Hypertension Reports*, 20(2). <https://doi.org/10.1007/s11906-018-0812-z>
46. Barazzoni, R., Cappellari, G. G., Ragni, M., & Nisoli, E. (2018). Insulin resistance in obesity: an overview of fundamental alterations. *Eating and Weight Disorders - Studies on Anorexia Bulimia and Obesity*, 23(2), 149–157. <https://doi.org/10.1007/s40519-018-0481-6>
47. Xu, Y., & Qiao, J. (2022). Association of Insulin Resistance and Elevated Androgen Levels with Polycystic Ovarian Syndrome (PCOS): A Review of Literature. *Journal of Healthcare Engineering*, 2022, 1–13. <https://doi.org/10.1155/2022/9240569>

48. Kosmas, C. E., Bousvarou, M. D., Kostara, C. E., Papakonstantinou, E. J., Salamou, E., & Guzman, E. (2023). Insulin resistance and cardiovascular disease. *Journal of International Medical Research*, 51(3). <https://doi.org/10.1177/03000605231164548>
49. Da Silva, A. A., Carmo, J. M. D., Li, X., Wang, Z., Mouton, A. J., & Hall, J. E. (2020). Role of hyperinsulinemia and insulin resistance in hypertension: Metabolic Syndrome revisited. *Canadian Journal of Cardiology*, 36(5), 671–682. <https://doi.org/10.1016/j.cjca.2020.02.066>
50. Yoon, J. H., Hwang, J., Son, S. U., Choi, J., You, S., Park, H., Cha, S., & Maeng, S. (2023). How can insulin resistance cause Alzheimer's disease? *International Journal of Molecular Sciences*, 24(4), 3506. <https://doi.org/10.3390/ijms24043506>
51. *Oral glucose tolerance testing*. (2012, June 1). PubMed. <https://pubmed.ncbi.nlm.nih.gov/22675678/>
52. Tang, Q., Li, X., Song, P., & Xu, L. (2015). Optimal cut-off values for the homeostasis model assessment of insulin resistance (HOMA-IR) and pre-diabetes screening: Developments in research and prospects for the future. *Drug Discoveries & Therapeutics*, 9(6), 380–385. <https://doi.org/10.5582/ddt.2015.01207>