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High Sugar Intake and Insulin Resistance in Obesity

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Abstract

High sugar intake, particularly from added sugars and sugar-sweetened beverages, is known to contribute to the increasing prevalence of obesity and insulin resistance, which are risk factors for various metabolic diseases. The Objective of the review to determine the relationship between high sugar intake and insulin resistance in obesity based on published research findings. This study used a literature review method by searching articles through the PubMed databases published between 2021 and 2026. Article selection was conducted based on the PRISMA 2020 guidelines, resulting in 12 articles that met the inclusion criteria. Literature synthesis demonstrates that high sugar consumption, particularly liquid fructose, drives insulin resistance through hepatic de novo lipogenesis (DNL), accumulation of toxic plasma ceramides, low-grade systemic inflammation (elevated CRP and IL-6), gut microbiota dysbiosis, and altered expression of lipogenic genes (e.g., MEG3 and FTO). Isocaloric fructose restriction studies showed significant improvements in insulin sensitivity and reduction of hepatic fat, independent of substantial weight loss. Furthermore, a bidirectional vicious cycle exists where higher baseline insulin resistance reinforces sensory preferences for sugar-sweetened foods. There is a strong and causal relationship between high sugar intake and insulin resistance in obesity, mediated by complex molecular and metabolic pathways. Restricting added sugar intake, especially from sugar-sweetened beverages, is highly recommended as a primary prevention and clinical management strategy for obesity.

Introduction

Obesity is a growing health problem globally and nationally (Regina & Fitriany, 2021). The WHO reports that obesity is closely linked to an increased risk of metabolic disease, one of the main mechanisms of which is insulin resistance. According to WHO data, in 2022, there were 2.5 billion cases of obesity in adults aged 18 and over, with a prevalence of 43% in men and 44% in women. By 2024, there were 35 million cases of obesity in children under 5. The global obesity prevalence in Southeast Asia and Africa was 31%, while in the Americas it was 67%. This represents a significant increase from the previous year (WHO, 2024).

Unhealthy diets, particularly high sugar intake, are a significant factor contributing to obesity. Consumption of added sugar and sugar-sweetened beverages can increase the risk of overweight or obesity and type 2 diabetes (Sitorus et al., 2020). Obesity and insulin resistance are closely related; one of the main factors causing insulin resistance in obese individuals is the accumulation of visceral fat. This fat is located around the internal organs and produces various inflammatory cytokines and hormones that can interfere with insulin function (Guerreiro et al., 2022; Wu et al., 2023; Zhao et al., 2023). Furthermore, chronic inflammation in obese individuals also plays a significant role. Fat cells in obesity produce pro-inflammatory cytokines such as TNF-alpha and IL-6. These cytokines interfere with insulin signaling and contribute to insulin resistance, as well as hyperglycemia and lipotoxicity, two conditions frequently found in obese individuals that negatively impact insulin production. Elevated blood

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sugar and lipid levels cause damage to pancreatic beta cells, reducing insulin production, and exacerbating insulin resistance (Sari et al., 2024). An unhealthy diet contributes to elevated blood glucose levels and worsens glycemic control in people with type 2 diabetes mellitus, thus supporting the importance of regulating sugar intake in preventing insulin resistance. A study by Taufik et al. showed a significant relationship between diet and fasting blood sugar levels in patients with type 2 diabetes ($p<0.001$), reinforcing the role of dietary patterns in glucose metabolism disorders (Taufik et al., 2025). Dyslipidemia and type 2 diabetes mellitus have a close pathophysiological relationship because dyslipidemia can increase insulin resistance and ultimately lead to impaired glucose metabolism. A study at Ibnu Sina Hospital in Makassar showed a significant association between dyslipidemia and type 2 diabetes mellitus ($p=0.018$), suggesting that lipid metabolism disorders should be considered as a contributing factor to insulin resistance (Naufal et al., 2022).

In clinical practice, obese individuals with insulin resistance are at higher risk of developing hyperglycemia (Huang et al., 2023; Korytkowski et al., 2022; Szablewski, 2024). Therefore, a thorough understanding of the relationship between high sugar intake and insulin resistance in obesity is crucial for prevention and early detection. Therefore, this literature review aims to examine and analyze the relationship between high sugar intake and insulin resistance in obesity.

Method

Research Design and Literature Search Strategy

This study employed a narrative literature review using a systematic literature search approach. The identification, screening, and selection of relevant articles were conducted systematically in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines to enhance transparency in the literature selection process. However, this study was not designed as a systematic review; therefore, no formal critical appraisal or risk-of-bias assessment was performed for the included studies.

The literature search was conducted using a single electronic database, PubMed. The search strategy was developed using relevant keywords combined with Boolean operators (AND and OR) to identify literature addressing the relationship between sugar intake, insulin resistance, and obesity. The search strategy included the following terms: ("high sugar intake" AND "insulin resistance") OR ("sugar consumption" AND "obesity") OR ("added sugar" AND "insulin resistance") OR ("dietary sugar" AND "obesity") OR ("sugar intake" AND "obesity"). The search was limited to articles published between 2021 and 2026.

Eligibility and Study Selection Process

Articles were considered eligible if they met the following inclusion criteria: (1) observational or experimental studies conducted in humans or animals, as well as narrative or literature reviews; (2) studies discussing the relationship between sugar intake and insulin resistance in the context of obesity; (3) articles available in full text; (4) articles published in Indonesian or English; and (5) articles published between 2021 and 2026. Articles were excluded if they were duplicates, did not specifically address sugar intake or insulin resistance, involved populations unrelated to obesity, were published before 2021 or after 2026, or were not available in full text. The study selection process followed the four stages of the PRISMA 2020 framework: identification, screening, eligibility assessment, and inclusion. The initial search in PubMed identified 1,613 articles. After removing 96 duplicate records, 1,517 articles remained for title and abstract screening. Of these, 1,451 articles were excluded because they were not relevant to the research topic. The remaining 66 articles underwent full-text assessment. Following the

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application of the eligibility criteria, 54 articles were excluded, resulting in 12 articles that met the eligibility requirements and were included in the final narrative literature review.

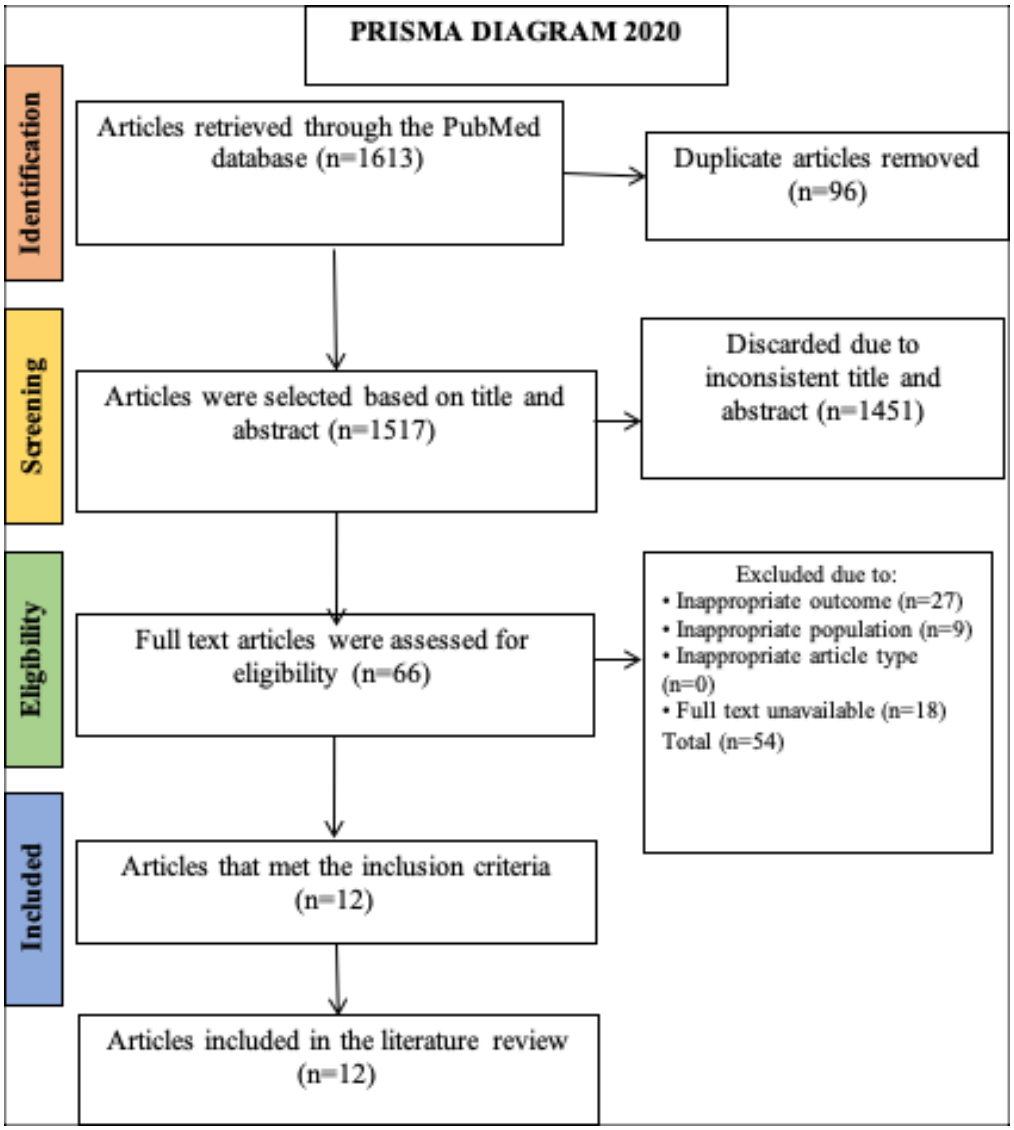

Figure 1. PRISMA 2020 Diagram

Result and Discussion

Based on the literature selection process using PRISMA 2020 guidelines, 12 articles were obtained that met the inclusion criteria and were suitable for analysis. These articles come from the PubMed database published in the period 2021-2026, with various research designs, including narrative reviews or literature reviews, animal or human research, and experimental research or observational research. Next, data extraction is carried out which includes the author's name, year of publication, design or type of research, as well as the results of the research conclusions. A summary of the characteristics and findings of the analyzed articles is presented in the following tables:

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Table 1. Narrative Review/Literature Review

Author/ Year	Research Design	Research Sample	Research Characteristics	Numerical Results	Research Conclusion
Yan, Chan, & Louie (2022)	Narrative Review	-	Review of human & animal studies	-	Administration of sucrose or fructose in supraphysiological doses, either in solid or liquid form, has been shown to cause insulin resistance, glucose intolerance, hyperglycemia and hypertriglyceridemia.
DiStefano & Gerhard (2024)	*Literature Review / Clinical Intervention Review*	-	Review 4 RCT	-	Reviewing clinical trials of sugar restriction in children with obesity and Non-Alcoholic Fatty Liver Disease (NAFLD). Fructose/sucrose restriction consistently reduced liver fat fraction significantly, which is a major driver of improvements in systemic insulin sensitivity.
Soleimani et al. (2023)	Literature Review / Narrative Review	-	Review mekanisme	-	Discusses the pathogenesis of hypertension in metabolic syndrome with a focus on the synergistic role of fructose and salt. Fructose stimulates sodium absorption in the kidneys and intestines, exacerbating circulatory burden and systemic insulin resistance.

Table 2. Animal Research

Author/ Year	Research Design	Research Sample	Research Characteristics	Numerical Results	Research Conclusion
Kawano et al. (2022)	Animal Research & Experimental Microbiota	Male C57BL/6 mice, approximately 5 weeks old; several genetic groups (WT, knockout, germ-free, and microbiota colonization). The number of animals varied between experimen	Mice were fed normal chow, a high-fat diet (HFD), a sugar-free HFD, or a HFD + sucrose. Body weight, glucose tolerance test, insulin tolerance test, Th17 cell analysis, gut microbiota, CD36 expression, and lipid absorption	Numerically, HFD was reported to cause weight gain, insulin resistance, and glucose intolerance; 10% sucrose consumption eliminated Th17-inducing bacteria; a sugar-free diet maintained Th17 cells and protected against obesity and	They found that dietary sugar intake induces an imbalance in the gut microbiota (dysbiosis) by eliminating segmented filamentous bacteria (SFB), leading to the loss of Th17 cells in the gut. This impairs gut immunological protection, triggers excess lipid absorption, and accelerates insulin resistance.

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		ts and is not reported as a total.	were measured.	metabolic syndrome. Most results are presented as graphs with significant differences ($p<0.05$).	
Osman et al. (2025)	Animal Research, Experimental (Wistar Rat Obesity Model)	Obese mice	High fat and sugar diet	-	A high-fat, high-sugar (HFHS) diet significantly induces obesity, increasing pro-atherogenic inflammatory cytokines (IL-6), increasing leptin, and decreasing adiponectin. High adiponectin levels indicate their resistance to diet-induced obesity and insulin resistance.

Table 3. Human Research

Author/ Year	**Research Design**	**Research Sample**	**Research Characteristics**	**Numerical Results**	**Research Conclusion**
Epstein et al. (2023)	Human Research, Clinical Experimental (Pilot Study)	Obesity (n=13)	BMI 39.1 kg/m²; HOMA-IR 5.2	r=0,64; p=0,019 (HOMA-IR)	Psychophysiological findings: obese individuals with higher levels of insulin resistance (HOMA-IR) and HbA1c showed significantly stronger sensory preferences and reinforcing value for high-sugar foods, thus exacerbating chronic excess sugar intake.
Olson et al. (2022)	Human Research, Experimental Clinical Trial (Isocaloric Fructose Restriction Intervention)	Obese children (n=43)	9-day fructose restriction	Weight loss 0.9±1.1 kg	*Isocaloric fructose restriction in obese children significantly reduced plasma levels of the toxic lipid ceramide. This reduction in ceramide was closely associated with a reduction in liver DNL and a direct improvement in insulin sensitivity.*
Schmidt et al. (2023)	*Randomized Controlled Trial / RCT – Secondary Analysis*	Obese Latino adolescents (n=105)	Mean age 14.8 years; BMI ≥95th percentile	11.5→7.3% energy vs 13.9→10.7 %; p=0.02	Reducing sugar intake for 16 weeks in Latino adolescents with obesity significantly reduced fasting insulin concentrations and improved the degree of insulin resistance independently of total body weight changes.

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Hernández-DíazCouder et al. (2025)	*Cross-sectional*)	Children 6–12 years old (n=71)	28 normals; 43 obese	Decreased lipogenic gene expression	*Added sugar in obese children is associated with altered expression of control genes in peripheral blood cells (PBMCs). Downregulation of the lncRNA MEG3 leads to increased expression of obesity- and lipogenic-related genes (FTO, ATF4, SREBP1, FASN, ACACA), contributing to the insulin resistance pathway. (15)*
Alves-Costa et al. (2024)	Human Research, Observational (Cross-sectional/Cohort-based)	Obese adolescents n=2,515	Age 18–19 years	99.8% high sugar; 72.3% TyG high	A study of 2,515 adolescents showed that free sugar consumption acts as a major hub in a network of non-communicable diseases. High sugar intake is closely correlated with an increase in the TyG (triglyceride-glucose) index, which is an early marker of insulin resistance.
Lin et al. (2023)	Human Research, Observational (NHANES Cross-Sectional Study)	Adults without diabetes n=5,250	Normoglycemia & prediabetes; 24-hour recall	Prediabetes risk 1.31x; CRP 1.57x; p=0.030	Found a clinically significant positive relationship between high intake of sugar-sweetened beverages (SSB) with the incidence of abdominal obesity and chronic inflammatory markers (CRP) where insulin resistance is induced by inhibition of glucose metabolism.
Della Corte et al. (2023)	Human Research, Experimental/Observational (PREVIEW Sub-study)	514 overweight/obese participants aged 25–70 years with or without prediabetes	3-year follow-up during the weight maintenance phase; evaluation of total sugars, added sugars, free sugars, GI, fasting glucose, insulin, HbA1c, C-peptide, BMI, waist circumference, and body fat.	Free sugars: fasting glucose p=0.008; HbA1c p=0.035. GI: insulin p=0.003; HbA1c p<0.001; C-peptide p=0.005. Added sugars: body fat p<0.001; BMI p=0.006.	Consumption of added sugars is closely associated with increased body fat index. However, added sugars do not show a direct relationship with fasting insulin, HbA1c, fasting glucose, or C-peptide. In contrast, the glycemic index of food, not sugar intake, has been shown to be an independent predictor of increased waist circumference and worsening insulin sensitivity.

Based on manuscript selection using the PRISMA 2020 guidelines, 12 articles met the inclusion criteria. Data extraction results indicate that high sugar consumption, particularly in the form of liquids (sugar-sweetened beverages/SSBs) and free fructose, is strongly correlated with decreased insulin sensitivity in obese populations. Clinical experimental studies have demonstrated that sugar restriction (either isocaloric or total reduction) consistently improves insulin resistance parameters, reduces ectopic lipid accumulation in the liver, and lowers toxic lipid markers such as plasma ceramides, even without significant weight loss.

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The Relationship Between High Sugar Concentration and Insulin Resistance

Obesity is a major risk factor for insulin resistance. However, various studies have shown that the quality of food intake, particularly the consumption of added sugars, fructose, and sugar-sweetened beverages (SSBs), accelerates the development of insulin resistance in obese individuals. Physiologically, high sugar consumption increases the metabolic burden, particularly on the liver, triggering impaired glucose and lipid metabolism, leading to decreased insulin sensitivity (Colosimo et al., 2023; Chandrasekaran & Weiskirchen, 2024; Cao et al., 2025).

Based on a 3-year longitudinal analysis of the PREVIEW study, there are differences in the pathways through which sugar intake affects the body. Research shows that added sugar consumption is closely associated with increased body fat index (BMI and body fat percentage). However, added sugar intake does not show a direct relationship with worsening glucose metabolism indices such as fasting insulin, HbA1c, fasting glucose, or C-peptide. Conversely, the glycemic index (GI) of food, not sugar intake, has been shown to be an independent predictor of increased waist circumference and worsening insulin sensitivity (Della et al., 2023).

Research by Alves-Costa et al. (2024) showed that added sugars are a key factor associated with the TyG index, an early marker of insulin resistance in adolescents. The study also found that nearly all respondents had high free sugar consumption, while more than two-thirds showed an elevated TyG index, illustrating a strong link between sugar consumption patterns and metabolic disorders from a young age (Alves, 2024).

Research by Soleimani et al. (2023) also showed that high sugar consumption, particularly in the form of fructose (such as high-fructose corn syrup and sucrose), has a very strong causal relationship with the development of insulin resistance and clinical manifestations of metabolic syndrome. Pathophysiologically, long-term fructose consumption not only triggers impaired glucose metabolism but also induces compensatory hyperinsulinemia, an increase in circulating insulin levels as a defense response against systemic insulin resistance (Zhao et al., 2023; Güney & Akar, 2023; Dong et al., 2024).

These findings are supported by the NHANES study, which showed that individuals with high sugar consumption from sugar-sweetened beverages/SSBs have a greater risk of developing prediabetes and elevated C-reactive protein (CRP) levels, particularly in individuals with abdominal obesity. This suggests that sugar consumption is not only associated with insulin resistance but also exacerbates the systemic inflammatory process that plays a role in the pathogenesis of type 2 diabetes mellitus (Lin et al., 2023).

Furthermore, research by Epstein et al. found that obese individuals with higher insulin resistance have a greater preference for sweet foods compared to non-caloric sweeteners. HOMA-IR and HbA1c values were positively correlated with sugar-sweetened yogurt consumption. These findings suggest a two-way relationship: sugar consumption worsens insulin resistance, while insulin resistance also increases the tendency to consume sweet foods, creating a vicious cycle (Epstein et al., 2023).

Overall, the articles reviewed consistently demonstrate that high sugar consumption, particularly in the form of added sugar and sugar-sweetened beverages, is closely associated with increased insulin resistance in obese individuals. However, the magnitude of this relationship is influenced by the amount consumed, the form of sugar consumed, the duration of exposure, physical activity, and total energy intake (Hengist et al., 2023; Stamatakis et al., 2025).

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Molecular Mechanism

The relationship between high sugar consumption and insulin resistance is mediated by various interacting molecular mechanisms.

Increased de novo lipogenesis (DNL)

Fructose is metabolized almost entirely in the liver and bypasses phosphofructokinase regulation, allowing metabolism to proceed without insulin control. This results in increased DNL, the formation of new fatty acids from excess carbohydrates. Increased DNL leads to triglyceride accumulation in the liver, increased VLDL production, and ectopic fat accumulation, leading to hepatic and systemic insulin resistance (Yan et al., 2023).

Ceramide Accumulation

Ceramide is a bioactive lipid that inhibits the insulin signal transduction pathway by inhibiting Akt phosphorylation. Research by Olson et al. showed that fructose restriction for nine days reduced levels of various ceramides and improved insulin sensitivity. The decrease in ceramides also correlated with decreased DNL activity, suggesting that ceramides are important mediators between high sugar consumption and insulin resistance (Olson et al., 2022).

Chronic low-grade inflammation

Obesity leads to increased production of pro-inflammatory cytokines such as TNF-α, IL-6, and CRP. High sugar consumption exacerbates inflammation, disrupting the phosphorylation of insulin receptor substrate (IRS-1) and decreasing the activity of the PI3K/Akt pathway. This disruption leads to reduced GLUT-4 translocation to the cell membrane, thus decreasing glucose uptake by peripheral tissues. The NHANES study showed a relationship between sugar consumption from SSBs and increased CRP, especially in prediabetic individuals with abdominal obesity (Lin et al., 2023).

Adipokine Changes

Obesity results in increased leptin levels and decreased adiponectin. This condition reduces insulin sensitivity in the liver, muscle, and adipose tissue. A mouse model of obesity induced by a high-sugar and fat diet showed increased IL-6, leptin, and decreased adiponectin, which are associated with impaired glucose metabolism (Osman et al., 2025).

Gene Expression Regulation

Recent research has shown that added sugar consumption affects the expression of various metabolic genes, such as MEG3, FTO, ATF4, SREBP1, FASN, and ACACA. These genes play a role in lipogenesis, energy regulation, and insulin sensitivity. In obese children with insulin resistance, changes in the expression of these genes were found, as well as a negative correlation between the consumption of added sugar and the expression of several lipogenic genes (Hernández et al., 2025).

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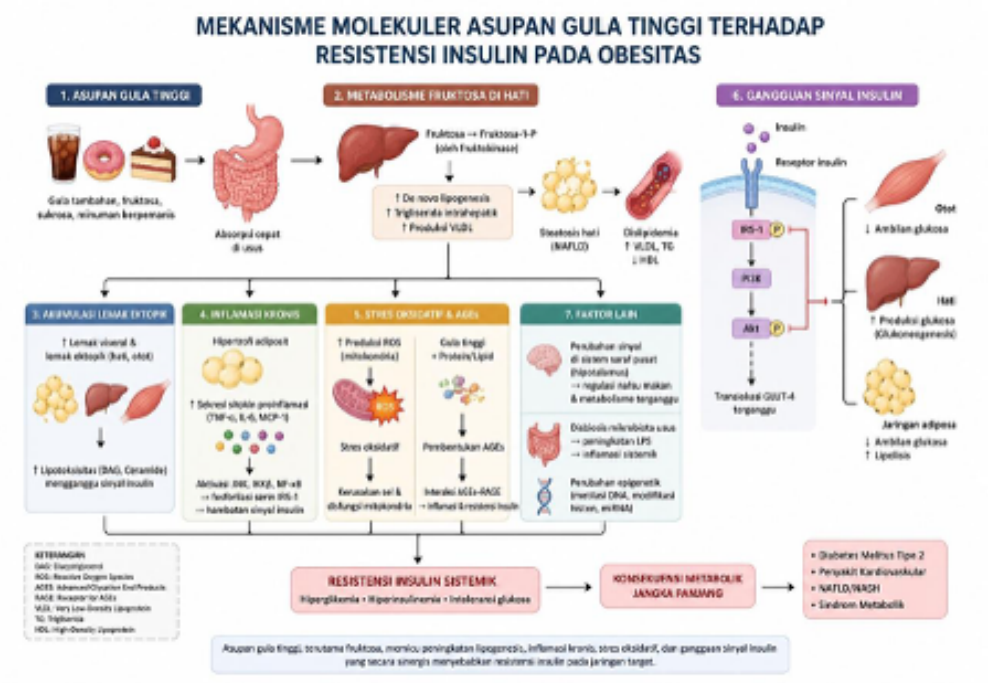

Figure 2. Graph of the Molecular Mechanism of High Sugar Intake on Insulin Resistance in Obesity

Experimental Evidence

Experimental evidence supports a causal link between high sugar consumption and insulin resistance. An interventional study by Olson et al. showed that isocaloric fructose restriction for nine days in obese children with cardiometabolic risk reduced ceramide levels, reduced DNL, and improved insulin sensitivity despite relatively small changes in body weight. This suggests that metabolic improvements can occur without significant weight loss if fructose consumption is reduced (Olson et al., 2022).

DiStefano and Gerhard's review of sugar restriction in obese children with NAFLD concluded that most clinical trials showed a decrease in liver fat after sucrose or fructose restriction. These improvements have the potential to improve insulin sensitivity, as the liver is the primary organ regulating glucose homeostasis (Distefano & Gerhard, 2024).

Animal models also provide strong biological evidence. Mice fed a high-fat, high-sugar diet experienced obesity, increased IL-6, impaired adipokine production, oxidative stress, and glucose intolerance. Intervention with calonysterone prevented weight gain and improved inflammatory biomarkers, supporting the importance of inflammation in the pathogenesis of insulin resistance induced by a high-sugar diet (Osman et al., 2025).

Animal studies also show that under normal conditions, IL-17A secreted by Th17 cells suppresses the expression of the fatty acid transporter CD36 on distal small intestinal enterocytes. When this Th17-rightarrow IL-17-rightarrow CD36 signaling axis is disrupted by high sugar consumption, CD36 repression is lost, leading to a massive increase in dietary lipid uptake by enterocytes. This excess absorbed lipid then accumulates in adipose tissue and the liver. This ectopic lipid accumulation, coupled with increased systemic inflammatory cytokines (such as IFN-gamma and TNF-alpha), ultimately disrupts the cellular insulin signaling pathway. This functional failure leads to the development of insulin resistance, glucose intolerance, and systemic obesity (Kawano et al., 2022).

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Overall, experimental evidence suggests that restricting sugar consumption can improve various metabolic biomarkers, including DNL, ceramides, liver fat, inflammation, and insulin sensitivity.

Clinical Implications

High sugar intake and insulin resistance in obese adolescents have significant clinical implications, increasing the risk of chronic metabolic diseases, such as type 2 diabetes (T2D) and cardiovascular disease (CVD). Clinical evidence from randomized controlled trials shows that interventions to reduce free sugar intake are consistently associated with improvements in biomarkers of cardiometabolic health in high-risk populations, such as obese Latino adolescents (Boxall et al., 2025). When adolescents successfully self-reduce total sugar intake, there is a significant improvement of up to 23% in the oral disposition index (oral-DI), a sensitive indicator of pancreatic beta cell function in adjusting insulin secretion to the body's sensitivity. Conversely, failure to reduce sugar intake is clinically associated with a 6.5% increase in serum triglyceride levels and an increase in the systemic inflammatory marker TNF-alpha. This accumulation of circulating lipids and ongoing subclinical inflammation accelerate endothelial dysfunction and exacerbate systemic insulin resistance, clinically placing obese adolescents on a progressive pathophysiological path toward T2D and coronary heart disease from a young age (Kosmas et al., 2023; Papaetis et al., 2025; Lonardo & Weiskirchen, 2025). Therefore, implementing clinically effective and sustainable sugar reduction strategies is crucial for preserving pancreatic beta cell function and suppressing cardiovascular risk factors in vulnerable adolescents (Schmidt et al., 2023).

Restricting the consumption of free and added sugars is a key strategy for preventing insulin resistance in obese individuals. Dietary interventions that reduce the consumption of sugar-sweetened beverages can reduce liver fat accumulation and improve insulin sensitivity. Early identification of obese individuals with high sugar consumption is necessary using dietary assessments combined with biomarkers such as HOMA-IR, TyG index, HbA1c, fasting glucose, and lipid profiles. Patient education should focus on reducing the consumption of sugar-sweetened beverages rather than simply restricting sugar in general. This is because most studies show a stronger association between sugar-sweetened beverages and insulin resistance than sugar from solid foods (Yan et al., 2022).

Limitations of Current Evidence

Although most studies demonstrate a link between high sugar consumption and insulin resistance in obesity, several limitations warrant consideration. Most studies used cross-sectional designs, so a causal relationship cannot be established. The number of randomized clinical trials evaluating sugar restriction is relatively small, with intervention durations generally short (7–12 days to several weeks), thus unable to describe long-term effects.

Most studies focus on the consumption of sugar-sweetened beverages, while research on sugar from solid foods is still limited. Furthermore, several articles indicate that the association between sugar and metabolic disorders is more consistent with the consumption of sugar-sweetened beverages than with sugar from solid foods (Yan et al., 2022).

Methods for assessing sugar consumption also vary widely, potentially introducing information bias. Measurement of insulin resistance is not uniform. Some studies use HOMA-IR, TyG index, CISI, HbA1c, or other biomarkers, making direct comparisons difficult. Most studies were conducted in developed countries, so generalization to the Indonesian population is still limited.

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There is disagreement regarding whether the adverse effects of sugar stem from the characteristics of the sugar itself or are primarily caused by excess total energy and weight gain. Yan et al.'s narrative review emphasized that the quality of the evidence still suffers from various methodological limitations, including short study duration, the use of very high fructose doses in animal studies, and the lack of direct comparisons between liquid and solid sugars (Yan et al., 2022). Overall, there is a link between high sugar consumption, especially fructose and sugar-sweetened beverages, and insulin resistance in obesity through increased de novo lipogenesis, ceramide accumulation, chronic inflammation, changes in adipokines, and metabolic gene regulation. However, prospective studies and long-term clinical trials with more uniform methods are needed, and efforts are being made to conduct research in developing countries to strengthen the causal relationship and generate better clinical recommendations.

Conclusion

A literature review of 12 articles concluded that added sugar and sugar-sweetened beverages (SSBs) intake plays a significant role in triggering and exacerbating insulin resistance in obese individuals. This relationship is mediated by complex pathophysiological pathways and molecular mechanisms, including increased de novo lipogenesis (DNL) in the liver, accumulation of bioactive ceramide lipids, stimulation of chronic low-grade inflammation (increased pro-inflammatory cytokines such as TNF-alpha, IL-6, and CRP), gut microbiota imbalance (dysbiosis), adipokine dysregulation (increased leptin and decreased adiponectin), and alterations in lipogenic gene expression (such as MEG3, FTO, ATF4, SREBP1, FASN, and ACACA). Sugar restriction has been clinically and experimentally proven to improve insulin sensitivity and cardiometabolic parameters, making dietary interventions that reduce added sugar and sugar-sweetened beverages (SSBs) a crucial strategy in the prevention and management of metabolic comorbidities in obese patients.

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