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Obesity and the Rising Burden of Type 2 Diabetes Among Adolescents

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Abstract: *The prevalence of obesity and its role in the development of Type II Diabetes Mellitus (T2DM) among adolescents has been increasing globally, contributing to a growing epidemic that was once primarily observed in adults aged 30–40 years and above. Modern lifestyle factors, including reduced physical activity, excessive consumption of high-calorie and low-fiber foods, prolonged screen time, and heightened psychological stress, have exacerbated this trend. Obesity promotes excessive fat deposition, which directly increases insulin resistance, a major pathogenic factor for T2DM. Additionally, impaired pancreatic beta-cell function further contributes to disease onset in obese adolescents. Although often asymptomatic during adolescence, obesity-induced T2DM can lead to severe complications later in life if left unaddressed. Preventive measures, such as raising awareness, promoting healthy nutrition, encouraging physical activity, and supporting weight management, are critical for mitigating the risk of diabetes and associated comorbidities in youth. Early intervention during adolescence provides an effective opportunity to reduce the long-term burden of obesity-related T2DM.*

Keywords: Obesity, Type II Diabetes Mellitus, Adolescents, Fat Deposition, Insulin Resistance

I. INTRODUCTION

Obesity is defined as an excessive accumulation of body fat that adversely affects health. This increase in body weight occurs due to abnormal growth of adipose tissue, which may result from an enlargement of existing fat cells (hypertrophy), an increase in the number of fat cells (hyperplasia), or a combination of both mechanisms [1]. One of the most commonly used indicators to assess obesity is the Body Mass Index (BMI), calculated as the ratio of a person's weight in kilograms to the square of their height in meters (kg/m²). Individuals with a BMI exceeding 30 kg/m² are classified as obese [2]. BMI provides a simple and widely applicable method to categorize individuals into underweight, normal weight, overweight, and obese, allowing for population-level monitoring and clinical assessment of obesity-related risks [1].

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by elevated blood glucose levels, resulting from defects in insulin secretion, insulin action, or both. DM is broadly categorized into two major types: Type 1 Diabetes Mellitus (T1DM) and Type 2 Diabetes Mellitus (T2DM) [3]. T1DM is primarily an autoimmune condition in which pancreatic beta-cells are destroyed, leading to an absolute insulin deficiency. In contrast, T2DM is predominantly associated with insulin resistance, where peripheral tissues fail to respond adequately to insulin, often accompanied by a relative deficiency in insulin secretion from pancreatic beta-cells [3].

Historically, T2DM was considered a disease of adulthood, typically manifesting in individuals aged 30–40 years or older. This distinction between T1DM and T2DM based on age of onset has been used extensively in both clinical and research settings. However, in recent decades, there has been a notable rise in the prevalence of T2DM among adolescents, a trend closely linked to rapid lifestyle changes and the increasing prevalence of obesity in younger populations [4]. Modern adolescents are increasingly leading sedentary lifestyles, characterized by minimal physical activity, prolonged screen time, consumption of high-calorie and low-fiber foods, and exposure to various psychosocial stressors, including peer and academic pressure. These behavioral and environmental factors contribute to excessive

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weight gain and increased fat deposition, which are strongly associated with the development of insulin resistance, a key pathogenic mechanism in T2DM.

While obesity is recognized as one of the leading risk factors for T2DM in adolescents, it is important to note that not all obese adolescents will develop diabetes. The progression from obesity to T2DM is influenced by a complex interplay of genetic predisposition, family history, ethnicity, and lifestyle factors, including diet and physical activity. Moreover, obesity-induced metabolic disturbances, such as altered fat distribution, increased visceral adiposity, and impaired beta-cell function, significantly enhance the risk of insulin resistance and subsequent hyperglycemia.

II. OBJECTIVE

This review article aims to provide a comprehensive overview of obesity as a major contributor to the development of Type II Diabetes Mellitus in adolescents. The article examines the mechanisms linking excessive fat accumulation to insulin resistance, discusses the epidemiological trends of adolescent obesity and T2DM, and highlights the clinical significance of early detection, prevention strategies, and lifestyle interventions to mitigate the growing burden of diabetes in youth populations.

METHODS AND DATA SOURCES

To review the relationship between obesity and Type II Diabetes Mellitus (T2DM) in adolescents, a comprehensive literature search was conducted using multiple databases, including PubMed, NCBI, and the American Diabetes Association resources. In addition, authoritative textbooks on preventive and social medicine as well as pathology were consulted to ensure a thorough understanding of the clinical and epidemiological aspects of T2DM. Keywords such as "Type II diabetes mellitus," "adolescents," "obesity," and related Medical Subject Headings (MeSH) were used to identify relevant studies. The evidence from these sources was analyzed and synthesized to draw conclusions regarding the incidence, risk factors, and underlying mechanisms linking obesity and T2DM in young populations.

INCIDENCE OF OBESITY-RELATED T2DM IN ADOLESCENTS

Recent decades have witnessed a significant increase in the prevalence of obesity-induced T2DM among adolescents worldwide. This trend has reached epidemic proportions in countries such as India and the United States. According to the National Health and Nutrition Examination Survey, over one-third of children in the U.S. are classified as overweight or obese, with approximately 17% meeting the criteria for obesity [5]. Adolescents with a BMI between the 85th and 95th percentile are considered at risk of becoming overweight, whereas those above the 95th percentile are classified as obese [6]. Among U.S. adolescents aged 12–19 years, roughly 15.5% are overweight, and an estimated 12.7 million children and teenagers are obese [7].

Cases of adolescent T2DM have also been reported in the United Kingdom, and epidemiological studies indicate that obesity increases the risk of developing diabetes in young adults by up to four times [8]. According to the International Diabetes Federation (IDF), approximately 1.1 million children and adolescents aged 14–19 are affected by diabetes globally. In India, the prevalence of pre-diabetes and diabetes among young adults is 12.3% and 8.4%, respectively [7]. Anthropometric measures such as BMI and subscapular skinfold thickness are critical indicators for assessing the risk of diabetes and prediabetes, with higher prevalence observed in obese adolescent girls compared to boys [7].

ETIOLOGY OF T2DM IN ADOLESCENTS

The development of T2DM is influenced by both modifiable and non-modifiable factors [9].

- **Non-modifiable factors** include intrinsic attributes that cannot be altered, such as intrauterine exposure, family history of diabetes, and ethnicity. Among these, intrauterine exposure has been identified as a particularly significant contributor to the future risk of T2DM.

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- **Modifiable factors** encompass environmental and lifestyle variables, including dietary habits, physical inactivity, and overall weight management. Among these, obesity and overweight are the most critical, as they directly increase insulin resistance and the likelihood of developing T2DM. By targeting these modifiable factors, especially during adolescence, the onset of T2DM can be delayed or prevented.

III. PATHOPHYSIOLOGY AND MECHANISMS

Obesity plays a central role in the development of Type II Diabetes Mellitus (T2DM) in adolescents, primarily through its effect on insulin resistance. Increased adiposity leads to elevated levels of free fatty acids (FFAs), which in turn promote lipolytic activity and contribute to impaired glucose metabolism. This metabolic imbalance is a key factor in the onset of T2DM.

Polycystic ovarian syndrome (PCOS) is another significant contributor to T2DM in adolescent girls. PCOS is an endocrine disorder associated with reproductive dysfunction and is frequently observed in obese individuals. Importantly, PCOS is linked to insulin resistance independent of adiposity, further increasing the risk of developing T2DM [10].

The precise pathogenesis of T2DM in adolescents is complex and multifactorial, involving a combination of genetic, environmental, and metabolic factors. These factors collectively contribute to pancreatic beta-cell dysfunction and heightened insulin resistance in hepatic and muscular tissues [11,12]. Early onset of T2DM is associated with an increased risk of complications and premature morbidity and mortality during adulthood.

Under normal physiological conditions, glucose homeostasis is maintained by a balance between insulin secretion from pancreatic beta-cells and the sensitivity of target tissues to insulin [13]. When insulin sensitivity decreases, a compensatory increase in insulin secretion is required to maintain normal glucose tolerance. In obese adolescents, reduced insulin sensitivity leads to compensatory hyperinsulinemia. Studies indicate that compared to non-obese peers, obese adolescents exhibit approximately 40% lower insulin-stimulated glucose uptake, resulting in elevated circulating insulin levels [14].

IV. KEY MECHANISMS

1. Inflammation:

Chronic low-grade inflammation associated with obesity is a major driver of T2DM. Inflammatory markers such as C-reactive protein (CRP) are frequently elevated in obese adolescents and are predictive of diabetes risk [16]. Adiponectin, another adipokine, is inversely related to insulin sensitivity and plays a role in mediating inflammation and metabolic dysfunction [16]. In a study conducted in North India among adolescents aged 14–25 years, CRP levels were elevated in 21.8% of overweight participants and 24.5% of individuals with high body fat percentage, demonstrating a strong link between obesity, inflammation, and metabolic risk [17].

2. Insulin Resistance:

Insulin resistance is closely associated with obesity and represents a critical precursor to T2DM [18]. Fasting hyperinsulinemia, a hallmark of insulin resistance, has been observed in approximately 64% of obese adolescents aged 14–19 years in India [19]. Adipose tissue in obese individuals secretes a variety of bioactive molecules, including tumor necrosis factor-alpha (TNF-α), leptin, and visfatin, which impair insulin signaling and promote insulin resistance [14].

Insulin resistance is further exacerbated by high BMI, pubertal hormonal changes, abnormal fat distribution, elevated FFAs, mitochondrial dysfunction, and systemic inflammation [20]. When pancreatic beta-cells are unable to

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compensate for increased insulin demand, glucose homeostasis is disrupted, leading to the onset of hyperglycemia and T2DM.

Here's a polished, rewritten version of your **Metabolic Syndrome, Clinical Features, Complications, and Treatment** section, structured clearly for a review article:

V. METABOLIC SYNDROME

Metabolic syndrome is a cluster of interrelated metabolic abnormalities, including glucose intolerance, hypertension, dyslipidemia, central obesity, and insulin resistance [21]. Obese children and adolescents are at a higher risk of developing metabolic syndrome, although the precise role of these abnormalities in the development of Type II Diabetes Mellitus (T2DM) in youth is still under investigation [18,22].

Body fat distribution is a critical factor in the pathogenesis of T2DM. Insulin resistance is closely associated with the accumulation of visceral fat rather than subcutaneous fat [23,24]. Ectopic fat deposition in the liver and skeletal muscles impairs insulin signaling, reduces glucose uptake in muscles, and diminishes insulin-mediated suppression of hepatic glucose production [23,25,26]. Furthermore, perivascular fat deposits may secrete endocrine factors that exacerbate insulin resistance [27]. Even in individuals without general obesity, fat accumulation in the liver can lead to insulin resistance, highlighting the complex metabolic consequences of abnormal fat distribution.

Clinical Features

The diagnosis of T2DM in adolescents is based on hallmark clinical symptoms and biochemical criteria. Typical features include polyuria (frequent urination), polydipsia (increased thirst), and unexplained weight loss. Laboratory evaluation confirms diagnosis when fasting plasma glucose is ≥126 mg/dL or when glucose concentration exceeds 200 mg/dL during an oral glucose tolerance test (OGTT) [28]. These tests should be repeated on a separate day to confirm the diagnosis in the absence of acute metabolic decompensation.

The clinical presentation of T2DM in adolescents can be acute or chronic. Acute presentations may involve mild glycosuria without ketones and minimal weight loss, while chronic cases often exhibit excessive urination, pronounced weight loss, glucosuria with ketone bodies, and persistent hyperglycemia [28]. Many adolescents remain asymptomatic in the early stages, making routine screening and laboratory testing essential for early detection. Disease severity tends to increase with age.

VI. COMPLICATIONS

Obesity-induced T2DM in adolescents can result in a wide spectrum of complications. Cardiovascular complications are among the most prominent and include alterations in cardiomyocyte metabolism, disrupted cardiac insulin signaling, impaired calcium handling, and myocardial fibrosis [29]. Additional cardiovascular pathologies include hyperlipidemia, hypertension, coronary artery disease, and diabetic cardiomyopathy [30,31].

Renal complications, such as chronic kidney disease (CKD), can arise due to hyperglycemia-induced damage to mesangial cells, leading to impaired renal function and albuminuria [32,33,34]. Peripheral neuropathy is another common complication observed in this population [33]. These complications emphasize the importance of early detection and management of obesity-related T2DM.

VII. TREATMENT

Management of T2DM in adolescents is multidisciplinary and focuses on both lifestyle modification and medical intervention [35]. Key strategies include:

1. **Weight management:** Promoting a balanced diet and regular physical activity to reduce adiposity and improve insulin sensitivity.

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2. **Integrated diabetes care:** Collaboration among healthcare providers, including physicians, diabetes-specialized nurses, nutritionists, fitness instructors, psychologists, and social workers, to provide comprehensive support [36].
3. **Family involvement:** Active participation of family members is essential to initiate and sustain lifestyle modifications, enhance adherence, and create a supportive environment for adolescents [37].

Pharmacological Management

When lifestyle interventions alone are insufficient to control hyperglycemia in adolescents with Type II Diabetes Mellitus (T2DM), pharmacological therapy is employed. Hypoglycemic agents act through various mechanisms, including enhancing peripheral glucose uptake, stimulating glycogen synthesis, improving insulin sensitivity, and reducing hepatic glucose production [35,38].

Oral hypoglycemic agents include:

- **Biguanides (e.g., metformin):** Improve peripheral insulin sensitivity, suppress hepatic gluconeogenesis, promote weight reduction, and enhance lipid profiles [38].
- **Sulfonylureas:** Stimulate pancreatic beta-cells to secrete insulin in response to blood glucose, increasing insulin availability [35,39].
- **Alpha-glucosidase inhibitors (AGIs, e.g., acarbose):** Delay carbohydrate absorption in the gut, thereby reducing postprandial glucose excursions [35,40].

Insulin therapy is reserved for adolescents who do not achieve adequate glycemic control with oral agents. It is administered via injections and is tailored to the individual's metabolic requirements [38].

Consequences and Risk Factors

Obesity in adolescents contributes to T2DM through multiple behavioral, dietary, and psychosocial mechanisms. Sedentary lifestyles, consumption of calorie-dense foods, reduced outdoor activity, and excessive screen time have all been implicated in weight gain and metabolic dysfunction [41–43].

Psychological and social factors also play a significant role. Overweight adolescents may experience teasing, bullying, and body shaming at school, leading to low self-esteem, stress, and emotional distress. These factors often promote overeating, particularly of high-calorie snacks such as cakes and chocolates, while discouraging participation in physical activity due to fear of judgment [44–46]. Academic pressure, parental expectations, and peer influence can further exacerbate emotional stress and unhealthy eating patterns, creating a vicious cycle that reinforces obesity and metabolic risk.

VIII. CONCLUSION

Evidence from multiple studies indicates that obesity is a primary contributor to the development of Type II Diabetes Mellitus in adolescents. Preventive strategies are crucial and should focus on community and family awareness, promotion of physical activity, and adoption of healthy dietary habits. Positive reinforcement, goal-setting, and supportive environments can encourage adolescents to engage in regular exercise and maintain healthy eating patterns.

In addition, health education programs should emphasize the importance of sports, balanced nutrition, and psychosocial support, while actively discouraging body shaming or peer ridicule. Early intervention is particularly important, as obesity-related T2DM often remains asymptomatic during adolescence but can lead to serious complications later in life. Given the growing prevalence of obesity-induced diabetes in youth, proactive measures are essential to prevent disease onset and reduce long-term morbidity.

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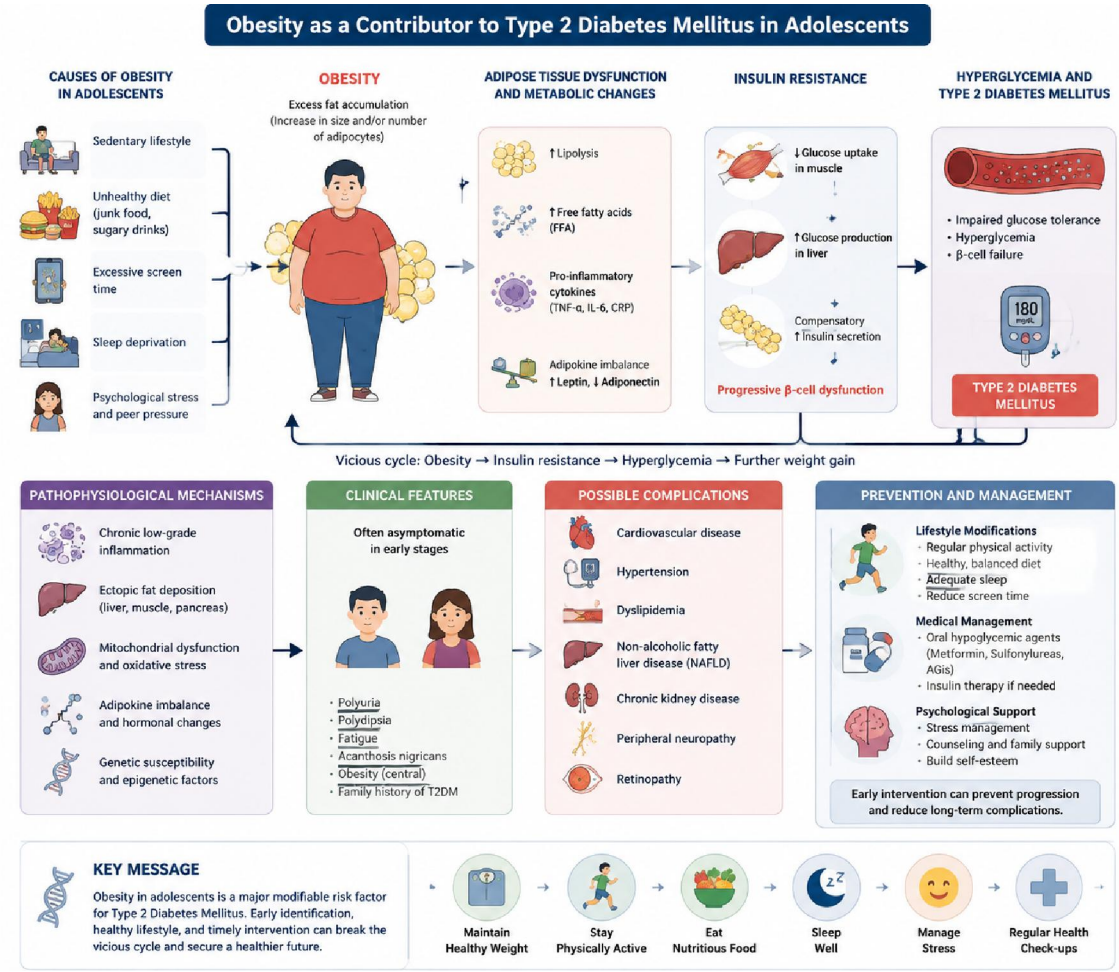

REFERENCES

1. Park, K. *Park's Textbook of Preventive and Social Medicine*. 25th ed., Banarsidas Bhanot Publishers, 1994.
2. Badhwar, R., G. Kaur, H. Popli, et al. "Pathophysiology of Obesity-Related Non-Communicable Chronic Diseases and Advancements in Preventive Strategies." *Journal Name*, vol. 25, 2020, pp. 317–340.
3. Kumar, A., Abbas AK, Jon C. *Robbins and Cotran Pathologic Basis of Disease: Professional Edition*. 2015.
4. Shaw, J. "Epidemiology of Childhood Type 2 Diabetes and Obesity." *Pediatric Diabetes*, vol. 8, 2007, p. 15.
5. Ogden, C. L., M. D. Carroll, L. R. Curtin, et al. "Prevalence of High Body Mass Index in US Children and Adolescents, 2007–2008." *JAMA*, vol. 303, 2010, pp. 242–249.
6. Fryar, C. D., M. D. Carroll, and J. Afful. "Prevalence of Overweight, Obesity, and Severe Obesity among Adults Aged 20 and Over: United States, 1960–1962 through 2017–2018." *NCHS*, vol. 10, 2020.
7. "Childhood Obesity Quadruples Risk of Developing Type 2 Diabetes." *Endocrine Society*, 2017, www.endocrine.org/news-and-advocacy/news-room/2017/childhood-obesity-quadruples-risk-of-developing-type-2-diabetes.
8. Xue, Y., M. Gao, and Y. Gao. "Childhood Type 2 Diabetes: Risks and Complications." *Experimental and Therapeutic Medicine*, vol. 12, 2016, pp. 2367–2370.

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9. Calcaterra, V., E. Verduci, H. Cena, et al. "Polycystic Ovary Syndrome in Insulin-Resistant Adolescents with Obesity: The Role of Nutrition Therapy and Food Supplements as a Strategy to Protect Fertility." *Nutrients*, vol. 13, 2021, p. 1848.
10. DeFronzo, R. A. "From the Triumvirate to the Ominous Octet: A New Paradigm for the Treatment of Type 2 Diabetes Mellitus." *Diabetes*, vol. 58, 2009, pp. 773–795.
11. D'adamo, E., and S. Caprio. "Type 2 Diabetes in Youth: Epidemiology and Pathophysiology." *Diabetes Care*, vol. 34, 2011, pp. S161–S165.
12. Pinhas-Hamiel, O., and P. Zeitler. "Acute and Chronic Complications of Type 2 Diabetes Mellitus in Children and Adolescents." *Lancet*, vol. 369, 2007, pp. 1823–1831.
13. Kumar, P., S. Srivastava, P. S. Mishra, et al. "Prevalence of Pre-Diabetes/Type 2 Diabetes among Adolescents (10–19 Years) and Its Association with Different Measures of Overweight/Obesity in India: A Gendered Perspective." *BMC Endocrine Disorders*, vol. 21, 2021, pp. 1–2.
14. Arslanian, S., F. Bacha, M. Grey, et al. "Evaluation and Management of Youth-Onset Type 2 Diabetes: A Position Statement by the American Diabetes Association." *Diabetes Care*, vol. 41, 2018, pp. 2648–2668.
15. "Pathophysiology of Diabetes." *The Ohio State University*, 2018, u.osu.edu/diabetes2018/patho/.
16. Duncan, B. B., and M. I. Schmidt. "The Epidemiology of Low-Grade Chronic Systemic Inflammation and Type 2 Diabetes." *Diabetes Technology & Therapeutics*, vol. 8, 2006, pp. 7–17.
17. Vikram, N. K., A. Misra, M. Dwivedi, et al. "Correlations of C-Reactive Protein Levels with Anthropometric Profile, Percentage of Body Fat and Lipids in Healthy Adolescents and Young Adults in Urban North India." *Atherosclerosis*, vol. 168, 2003, pp. 305–313.
18. Praveen, P. A., and N. Tandon. "Childhood Obesity and Type 2 Diabetes in India." *WHO-SEAJPH*, vol. 5, 2016, pp. 17–21.
19. Vikram, N. K., A. Misra, R. M. Pandey, et al. "Heterogeneous Phenotypes of Insulin Resistance and Its Implications for Defining Metabolic Syndrome in Asian Indian Adolescents." *Atherosclerosis*, vol. 186, 2006, pp. 193–199.
20. Cree-Green, M., T. M. Triolo, and K. J. Nadeau. "Etiology of Insulin Resistance in Youth with Type 2 Diabetes." *Current Diabetes Reports*, vol. 13, 2013, pp. 81–88.
21. Huang, P. L. "A Comprehensive Definition for Metabolic Syndrome." *Disease Models & Mechanisms*, vol. 2, 2009, pp. 231–237.
22. Jessup, A., and J. S. Harrell. "The Metabolic Syndrome: Look for It in Children and Adolescents, Too." *Journal of Diabetes Research*, vol. 23, 2005, pp. 26–32.
23. Weiss, R., and F. R. Kaufman. "Metabolic Complications of Childhood Obesity: Identifying and Mitigating the Risk." *Diabetes Care*, vol. 31, 2008, pp. S310–S316.
24. Caprio, S. O., L. D. Hyman, C. H. Limb, et al. "Central Adiposity and Its Metabolic Correlates in Obese Adolescent Girls." *American Journal of Physiology-Endocrinology and Metabolism*, vol. 269, 1995, pp. E118–E126.
25. Tiikkainen, M., M. Tamminen, A. M. Häkkinen, et al. "Liver-Fat Accumulation and Insulin Resistance in Obese Women with Previous Gestational Diabetes." *Obesity Research*, vol. 10, 2002, pp. 859–867.
26. Kelley, D. E., T. M. McKolanis, R. A. Hegazi, et al. "Fatty Liver in Type 2 Diabetes Mellitus: Relation to Regional Adiposity, Fatty Acids, and Insulin Resistance." *American Journal of Physiology-Endocrinology and Metabolism*, vol. 285, 2003, pp. E906–E916.
27. Yudkin, J. S., E. Eringa, and C. D. Stehouwer. "'Vasocrine' Signalling from Perivascular Fat: A Mechanism Linking Insulin Resistance to Vascular Disease." *Lancet*, vol. 365, 2005, pp. 1817–1820.
28. Reinehr, T. "Clinical Presentation of Type 2 Diabetes Mellitus in Children and Adolescents." *International Journal of Obesity*, vol. 29, 2005, pp. S105–S110.

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International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
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29. Mandavia, C. H., A. R. Aroor, and V. G. DeMarco, et al. "Molecular and Metabolic Mechanisms of Cardiac Dysfunction in Diabetes." *Life Sciences*, vol. 92, 2013, pp. 601–608.
30. Vinicor, F., and L. Jack Jr. "25 Years and Counting: Centers for Disease Control and Prevention Identifies Opportunities and Challenges for Diabetes Prevention and Control." *Annals of Internal Medicine*, vol. 140, 2004, pp. 943–944.
31. Jørgenrud, B., M. Jalanko, T. Heliö, et al. "The Metabolome in Finnish Carriers of the MYBPC3-Q1061X Mutation for Hypertrophic Cardiomyopathy." *PLoS ONE*, vol. 10, 2015, e0134184.
32. Min, T. Z., M. W. Stephens, and P. Kumar, et al. "Renal Complications of Diabetes." *British Medical Bulletin*, vol. 104, 2012, pp. 113–127.
33. Xue, Y., M. Gao, and Y. Gao. "Childhood Type 2 Diabetes: Risks and Complications." *Experimental and Therapeutic Medicine*, vol. 12, 2016, pp. 2367–2370.
34. Porth, C. *Essentials of Pathophysiology: Concepts of Altered Health States*. LWW, 2011.
35. Schwartz, M. S., and A. Chadha. "Type 2 Diabetes Mellitus in Childhood: Obesity and Insulin Resistance." *International Journal of Osteopathic Medicine*, vol. 108, 2008, pp. 518–524.
36. Diabetes Control and Complications Trial Research Group. "The Effect of Intensive Treatment of Diabetes on the Development and Progression of Long-Term Complications in Insulin-Dependent Diabetes Mellitus." *New England Journal of Medicine*, vol. 329, 1993, pp. 977–986.
37. Anderson, B. J. "Diabetes Self-Care: Lessons from Research on the Family and Broader Contexts." *Current Diabetes Reports*, vol. 3, 2003, pp. 134–140.
38. Krentz, A. J., and C. J. Bailey. "Oral Antidiabetic Agents." *Drugs*, vol. 65, 2005, pp. 385–411.
39. Aguilar-Bryan, L., C. G. Nichols, S. W. Wechsler, et al. "Cloning of the β Cell High-Affinity Sulfonylurea Receptor: A Regulator of Insulin Secretion." *Science*, vol. 268, 1995, pp. 423–426.
40. Van De Laar, F. A., P. L. Lucassen, R. P. Akkermans, et al. "α-Glucosidase Inhibitors for Patients with Type 2 Diabetes: Results from a Cochrane Systematic Review and Meta-Analysis." *Diabetes Care*, vol. 28, 2005, pp. 154–163.
41. Garg, M., and S. Mohale. "Prevalence of Metabolic Obesity Normal Weight (MONW) in Cardiovascular Disease Patients—A Hospital-Based Case Control Study." *Journal of Evolution of Medical and Dental Sciences*, vol. 9, 2020, pp. 2427–2432.
42. Hulkoti, V. S., S. Acharya, and S. Shukla, et al. "In Search of an Ideal Obesity Assessment Tool: Is Body Mass Index Reliable Enough." *Journal of Evolution of Medical and Dental Sciences*, vol. 9, 2020, pp. 2556–2560.
43. Rasheed, A., S. Acharya, and S. Shukla, et al. "High-Sensitivity C-Reactive Protein in Metabolic Healthy Obesity (MHO)." *Journal of Evolution of Medical and Dental Sciences*, vol. 9, 2020, pp. 443–447.
44. Inamdar, S. A., H. Agarwal, and S. Acharya, et al. "Coexistence of Hypertriglyceridemia and Hypercholesterolemia with Gestational Diabetes Mellitus in Pregnancy: A Case Report." *Medical Science*, vol. 24, 2020, pp. 594–598.
45. Jankar, J. S., K. N. Harley, K. M. Mohod, et al. "Association of Urinary Albumin with HbA1c Levels in Subjects of Type 2 Diabetes Mellitus in Central India." *Journal of Evolution of Medical and Dental Sciences*, vol. 9, 2020, pp. 3921–3925.
46. Unnikrishnan, B., A. Singh, and P. Rathi, et al. "Risk Factors of Gestational Diabetes Mellitus: A Hospital-Based Pair-Matched Case-Control Study in Coastal South India." *South African Journal of Obstetrics and Gynaecology*, vol. 26, 2020, p. 13.