Journal of Experimental and Basic Medical Sciences 2023;4(3):175-179

Review

The Influence of Socioeconomic Status on Pregnancy Characteristics and Type 2 Diabetes

Hilal Özgül[1], Orhan Sadık Kızılyalın[1], Oytun Erbaş[1]

ABSTRACT

Although Type 2 diabetes mellitus (T2DM) is typically a disease seen in individuals over the age of 40, in today's world, it is frequently observed in younger individuals due to improper nutrition leading to obesity, lack of physical activity, and genetic predisposition. As a result of the examined articles, it has been found that children from families with a higher socioeconomic level have a lower incidence of T2DM. It is concluded that to prevent the progression of T2DM, increased physical activity in daily life and the intake of nutrients that assist in regulating glucose metabolism should be sufficient. The purpose of this review is to examine the impact of socioeconomic factors on T2DM, a non-communicable chronic disease. Additionally, the influence of these factors on a pregnant individual diagnosed with the disease and on the offspring to be born has also been investigated.

Keywords: Fetal period, insulin, obesity, pregnancy, socioeconomic factors, type 2 diabetes mellitus

Type 2 diabetes mellitus (T2DM), typically observed in individuals aged 40 and above, is increasingly prevalent in younger people in today's world due to poor dietary habits, rising obesity resulting from physical inactivity, and genetic predisposition. The regulation of blood sugar in the body is achieved through the complex interaction of numerous chemical substances and hormones.[1,2]

One of the most crucial hormones involved in regulating sugar metabolism is insulin, secreted by the beta cells of the pancreas. Diabetes mellitus is a common term used to describe several diseases resulting from either insufficient insulin secretion, impairment in insulin action, or a defect in the insulin response, leading to elevated blood sugar levels. Type 2 diabetes mellitus is generally a hereditary condition.[1]

Type 2 diabetes mellitus can arise from errors in maternally inherited mitochondrial deoxyribonucleic acid (DNA). However, mitochondrial mutations cannot fully explain the impact on the transmission of T2DM. In addition to genetic factors, environmental influences also play a role in the onset of T2DM. In an experiment conducted in China, consecutive generations exposed to famine were examined. The study revealed an increase in hyperglycemia among those exposed to prenatal famine. The rise in hyperglycemia supports the idea that the increase in T2DM in these consecutive generations may be influenced by both genetics and nutrition.[2]

Mitochondria, the organelle responsible for transmitting T2DM from mother to baby, possesses its own DNA that is independent of the nucleus and has a self-replicating system. Mitochondrial DNA has a shorter base sequence than nuclear DNA, making it prone to rapid mutations. As an organelle transmitted from the mother, all mutations present in the maternal lineage accumulate and are transferred to the baby. Due to these mutations or DNA coding errors, mitochondrial dysfunction occurs, leading to evidence supporting its role in T2DM and insulin resistance. Additionally, inadequate and unhealthy nutrition in early life can alter mitochondria, causing dysfunction, and is hypothesized to contribute to the development of T2DM in adulthood.[1,2]

[1]ERBAS Institute of Experimental Medicine, Illinois, USA & Gebze, Türkiye

Correspondence: Hilal Özgül. Institute of Experimental Medicine, 41470 Gebze-Kocaeli, Türkiye

E-mail: hilalozgll@gmail.com

Cite this article as: Özgül H, Kızılyalın OS, Erbaş O. The Influence of Socioeconomic Status on Pregnancy Characteristics and Type 2 Diabetes. JEB Med Sci 2023;4(3):175-179.

doi: 10.5606/jebms.2023.1062

Received : September 23, 2023
Accepted : November 5, 2023
Published online : December 25, 2023

Some individuals with T2DM are observed to have defects in mitochondrial DNA, such as point mutations.[3] These identified mutations adversely affect the insulin secretion system by impacting the pancreatic beta (β) cells.[4] As a consequence of the disrupted system, diabetes mellitus and insulin resistance ensue.

According to another finding, mitochondrial mutations have been suggested as a potential cause of T2DM. Subsequently, a reverse correlation with components of metabolic syndrome has been observed in mitochondrial DNA.[5] The abnormalities in DNA have strengthened the evidence that they may be related to insulin resistance. The density of this mitochondrial DNA supports the possibility of insulin sensitivity in the children of diabetic patients.[6]

In an experiment conducted in China, consecutive generations exposed to famine were examined. The study revealed an increase in hyperglycemia among those exposed to prenatal famine. The rise in hyperglycemia suggests that the increase in T2DM in these consecutive generations may be attributed to both genetic inheritance and nutritional factors.[7]

DIABETES IN PREGNANCY

The risks of planned pregnancy in women with T2DM are higher compared to healthy individuals. Type 2 diabetes mellitus increases the risks of maternal mortality, ischemic stroke, and heart attack. The effects of hyperglycemia can be exacerbated by factors such as obesity, smoking, and inadequate nutrition. In individuals with poorer glucose control, the risk of stillbirth may be higher. In addition to the increased risk of stillbirth in women with T2DM, early delivery and birth trauma are 30-60% more likely compared to healthy women. Moreover, babies of diabetic mothers may experience conditions such as respiratory distress, hypoglycemia, and hypocalcemia more frequently than babies of healthy mothers. The advanced maternal age in diabetic mothers can also contribute to adverse outcomes.[8]

Poor Nutrition and Fetal Development

Type 2 diabetes mellitus is a chronic disease that commonly emerges after the age of 40. One of its fundamental causes is the inadequate or imbalanced nutrition of the mother. Inadequate or imbalanced nutrition can lead to intrauterine growth restriction, which may disrupt fetal growth. Intrauterine growth restriction can affect the development of adipose tissue and β cells in the pancreas. As a consequence, insulin secretion may decrease, and the capacity for fat storage may increase. These alterations in secretion and capacity can ultimately lead to the development of obesity and T2DM.[9]

In addition to genetic factors, improper nutrition is a significant contributor to the occurrence of T2DM. While incorrect and inadequate nutrition can impact adult life, research has also provided evidence that nutritional inadequacy during the fetal period may contribute to the development of T2DM. One example demonstrating the potential contribution of non-genetic factors to the development of the disease is evident in studies conducted among core families of the Pima Indians.[10] In this research, it was found that siblings born after the mother was diagnosed with diabetes had a 3.7 times higher risk of developing diabetes, despite having similar living conditions to siblings born before the diabetes diagnosis. Numerous examples like this have demonstrated that adverse conditions experienced in the early stages of life increase the risk of T2DM.[11]

Adverse conditions encountered during the fetal period can lead to structural and functional abnormalities in the development of the fetus.[12] This perspective has given rise to the emergence of the 'predictive adaptive response' hypothesis, which involves the organism's ability to maximize adaptation to the environment.[13] However, it has also been suggested that individuals who can easily access food as a result of such adaptations may be predisposed to the development of T2DM. These ideas form the basis of the thrifty phenotype hypothesis.[14] According to this hypothesis, conditions such as inadequate nutrition and stress explain the development of adaptations that maximize nutrient storage to the highest level.[15,16]

Another study related to inadequate nutrition is the examination of the Dutch famine (1944-1945). In a study conducted by Lumey et al.[17], a food restriction of 900 calories per day was implemented during pregnancy for 3307 individuals born between 1945 and 1946. As a result, there was an observed higher likelihood of becoming a T2DM patient in adult offspring at the age of 59.[16,18]

TYPE 2 DIABETES IN CHILDREN AND ADOLESCENCE

According to research findings, early diagnosis of diseases or the identification of multiple risk factors in advance can positively influence the course of potential illnesses. The likelihood of insulin resistance developing into T2DM is high in later stages if detected

early in life. Detecting glucose metabolism in early life is a crucial step in determining the progression of the disease.[19,20]

Glucose metabolism is a regulatory mechanism that emerges as a result of the functioning of a series of enzymes involved in the regulation of blood glucose levels. In the liver, insulin enhances the interaction between protein phosphatase-1 and glycogen, facilitating the conversion and storage of excess glucose into glycogen. If blood glucose levels are low, it regulates the blood values by breaking down the stored form. All of these processes occur in response to insulin signals, controlling enzyme activity and ensuring antagonistic functioning.[21,22]

Insulin resistance can arise from varying responses of tissues in the body to insulin. As the same effect and severity may not be exhibited in every patient, clinical manifestations may not necessarily reflect a one-to-one correlation.[23]

In adolescents, the most common indicators of insulin resistance include dysfunctional adipose tissue, medication-induced hormonal disorders, insufficient physical activity, and nutritional imbalances.[24]

Ethnicity is one of the factors influencing insulin resistance. Afro-American, Pima-Indian, Asian, and Hispanic children exhibit lower insulin sensitivity compared to Caucasian children.[25] This condition affects the risk of T2DM. In Afro-American adolescents, the increased risk of T2DM is attributed to reduced insulin secretion during puberty.[26-28]

Many of these pathological findings are prevalent in children who become obese at a young age. Insulin resistance is unequivocally a cause of obesity. Therefore, obesity is considered a direct contributing factor to insulin resistance and T2DM.[29] One of the causes of obesity is adipose tissue. Depletion of fat stores leads to obesity, as adipose tissue is recognized as a complex endocrine tissue that influences body metabolism.[30,31] Childhood obesity is more common in industrialized countries.[32-35] The causes of obesity include socio-economic context, genetic predisposition, and environmental factors. Obesity is directly linked to changes in the environment. As environmental conditions change, children's eating habits change, and they tend to consume more unhealthy foods. Additionally, as physical activity decreases in children, the risk of obesity increases.[36,37] Childhood obesity should not be underestimated, as it can lead to infectious diseases and have long-lasting health implications.[38-40]

In recent years, T2DM has shown a rapid increase among all children. According to data from the search database, it has been found that there will be a fourfold increase in the incidence of the disease in individuals under the age of 20 over a 40-year period.[41]

In conclusion, through all the examined articles and experimental studies, it has been proven that inadequate nutrition and insufficient physical activities during critical development periods increase the risk of T2DM in adults. Recent studies on gene expression have provided data suggesting that the regulation of epigenetic mechanisms reduces the risk of T2DM. However, in recent years, only a few mechanistic pathways related to these epigenetic mechanisms have been identified. To better understand the underlying cause-and-effect relationship in this process, more unresolved aspects need to be addressed. Additionally, it is still unclear how much early-life issues contribute to T2DM in adulthood and how consistently they are transmitted. While some data has been obtained on this matter, certainty is still lacking. Ongoing research for a solution remains promising. Apart from genetic transmission, preventive measures should be taken against obesity, one of the major causes of this disease. Obesity is increasing day by day and can lead to significant chronic illnesses even without T2DM. Awareness campaigns against childhood obesity should be conducted, and equal opportunities for physical activity should be provided in schools, regardless of socioeconomic status. Families and children should be simultaneously educated about nutrition, and schools should offer healthy food options.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Rother KI. Diabetes treatment--bridging the divide. N Engl J Med. 2007 Apr 12;356:1499-501.
2. Lee YY, Park KS, Pak YK, Lee HK. The role of mitochondrial DNA in the development of type 2 diabetes caused by fetal malnutrition. J Nutr Biochem. 2005 Apr;16:195-204.
3. Maechler P, Wollheim CB. Mitochondrial function in normal and diabetic beta-cells. Nature. 2001 Dec 13;414:807-12.
4. Poulton J, Brown MS, Cooper A, Marchington DR, Phillips

DI. A common mitochondrial DNA variant is associated with insulin resistance in adult life. Diabetologia. 1998 Jan;41:54-8.

5. Lee HK, Song JH, Shin CS, Park DJ, Park KS, Lee KU, et al. Decreased mitochondrial DNA content in peripheral blood precedes the development of non-insulin-dependent diabetes mellitus. Diabetes Res Clin Pract. 1998 Dec;42:161-7.
6. Song J, Oh JY, Sung YA, Pak YK, Park KS, Lee HK. Peripheral blood mitochondrial DNA content is related to insulin sensitivity in offspring of type 2 diabetic patients. Diabetes Care. 2001 May;24:865-9.
7. Li J, Liu S, Li S, Feng R, Na L, Chu X, et al Prenatal exposure to famine and the development of hyperglycemia and type 2 diabetes in adulthood across consecutive generations: a population-based cohort study of families in Suihua, China. Am J Clin Nutr. 2017 Jan;105:221-7.
8. Cowie CC, Casagrande SS, Menke A, Cissell MA, Eberhardt MS, Meigs JB, et al. Diabetes in America. 3rd ed. Bethesda (MD): National Institute of Diabetes and Digestive and Kidney Diseases (US); 2018 Aug.
9. Vaiserman A, Lushchak O. Prenatal Malnutrition-Induced Epigenetic Dysregulation as a Risk Factor for Type 2 Diabetes. Int J Genomics. 2019 Feb 28;2019:3821409.
10. Bianco-Miotto T, Craig JM, Gasser YP, van Dijk SJ, Ozanne SE. Epigenetics and DOHaD: from basics to birth and beyond. J Dev Orig Health Dis. 2017 Oct;8:513-9.
11. Dabelea D, Hanson RL, Lindsay RS, Pettitt DJ, Imperatore G, Gabir MM, et al. Intrauterine exposure to diabetes conveys risks for type 2 diabetes and obesity: a study of discordant sibships. Diabetes. 2000 Dec;49:2208-11.
12. Vaiserman A. M. Early-life nutritional programming of longevity. Journal of Developmental Origins of Health and Disease. 2014;5:325-38.
13. Bateson P, Gluckman P, Hanson M. The biology of developmental plasticity and the Predictive Adaptive Response hypothesis. J Physiol. 2014 Jun 1;592:2357-68.
14. Hales C. N., Barker D. J. P. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. International Journal of Epidemiology. 2013;42:1215-22.
15. Stöger R. The thrifty epigenotype: an acquired and heritable predisposition for obesity and diabetes? BioEssays. 2008;30:156-66.
16. Zhou LY, Deng MQ, Zhang Q, Xiao XH. Early-life nutrition and metabolic disorders in later life: a new perspective on energy metabolism. Chin Med J (Engl). 2020 Aug 20;133:1961-70.
17. Lumey LH, Stein AD, Kahn HS, Romijn JA. Lipid profiles in middle-aged men and women after famine exposure during gestation: the Dutch Hunger Winter Families Study. Am J Clin Nutr 2009; 89:1737-43.
18. Ravelli AC, van der Meulen JH, Michels RP, Osmond C, Barker DJ, Hales CN, et al. Glucose tolerance in adults after prenatal exposure to famine. Lancet 1998; 351:173-7.
19. Polidori N, Mainieri F, Chiarelli F, Mohn A, Giannini C. Early Insulin Resistance, Type 2 Diabetes, and Treatment Options in Childhood. Horm Res Paediatr. 2022;95:149-66.
20. Senkal E, Bagcioglu E, Eryigit U, Erbas O, Solmaz V. Exposure to hypertonic solutions during pregnancy induces autism-like behaviors via the NFAT-5 pathway in offspring in a rat model. Physiol Behav. 2021 Oct 15;240:113545.
21. Bansal P, Wang Q. Insulin as a physiological modulator of glucagon secretion. Am J Physiol Endocrinol Metab. 2008;29:E751-61.
22. Lomberk G, Urrutia R. Primers on molecular pathways: the insulin pathway. Pancreatology. 2009;9:203-5.
23. Mainieri F, Tagi VM, Chiarelli F. Insulin resistance in children. Curr Opin Pediatr. 2022 Aug 1;34:400-6.
24. Tagi VM, Chiarelli F. Obesity and insulin resistance in children. Curr Opin Pediatr. 2020 Aug;32:582-8.
25. Goran MI, Bergman RN, Cruz ML, Watanabe R. Insulin resistance and associated compensatory responses in African-American and hispanic children. Diabetes Care. 2002;25:2184-90.
26. Goran MI, Shaibi GQ, Weigensberg MJ, Davis JN, Cruz ML. Deterioration of insulin sensitivity and beta-cell function in overweight hispanic children during pubertal transition: a longitudinal assessment. Int J Pediatr Obes. 2006;1:139-45.
27. Saad RJ, Danadian K, Lewy V, Arslanian SA. Insulin resistance of puberty in African-American children: lack of a compensatory increase in insulin secretion. Pediatr Diabetes. 2002;1:4-9.
28. Ayhan İ, Altuntaş İ, Üzümcü İ, Erbaş O. Premenstrual syndrome mechanism in the brain. D J Med Sci 2021;7:213-24.
29. Levy-Marchal C, Arslanian S, Cutfield W, Sinaiko A, Druet C, Marcovecchio ML, et al. Insulin resistance in children: consensus, perspective, and future directions. J Clin Endocrinol Metab. 2010;95:5189-98.
30. Giannini C, de Giorgis T, Scarinci A, Cataldo I, Marcovecchio ML, Chiarelli F, et al. Increased carotid intima-media thickness in pre-pubertal children with constitutional leanness and severe obesity: the speculative role of insulin sensitivity, oxidant status, and chronic inflammation. Eur J Endocrinol. 2009;161:73-80.
31. Elmas O, Erbas O, Yigitturk G. The efficacy of Aesculus hippocastanum seeds on diabetic nephropathy in a streptozotocin-induced diabetic rat model. Biomed Pharmacother. 2016 Oct;83:392-6.
32. Kershaw EE, Flier JS. Adipose tissue as an endocrine organ. J Clin Endocrinol Metab. 2004;89:2548-56.
33. Bentham J, Di Cesare M, Bilano V, Bixby H, Zhou B, Stevens GA, et al. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. Lancet. 2017;390:2627-42.
34. Gölboyu BE, Erdoğan MA, Erbaş O. Trimetazidine provides protection against diabetic polyneuropathy in rats via modulation of soluble HMGB1. Eur Rev Med Pharmacol Sci. 2023 Jun;27:5854-61.
35. Yilmaz M, Aktug H, Oltulu F, Erbas O. Neuroprotective effects of folic acid on experimental diabetic peripheral neuropathy. Toxicol Ind Health. 2016 May;32:832-40.

36. Koplan JP, Dietz WH. Caloric imbalance and public health policy. J Am Med Assoc. 1999;282:1579-81.
37. Gluckman P, Nishtar S, Armstrong T. Ending childhood obesity: a multidimensional challenge. Lancet. 2015;385:1048-50.
38. Gurnani M, Birken C, Hamilton J. Childhood Obesity: Causes, Consequences, and Management. Pediatr Clin North Am. 2015 Aug;62:821-40.
39. Pulgarón ER. Childhood obesity: a review of increased risk for physical and psychological comorbidities. Clin Ther. 2013;35:A18-32.
40. Yalçın MB, Bora ES, Çakır A, Akbulut S, Erbaş O. Autophagy and anti-inflammation ameliorate diabetic neuropathy with Rilmenidine. Acta Cir Bras. 2023 Dec 1;38:e387823.
41. Imperatore G, Boyle JP, Thompson TJ, Case D, Dabelea D, Hamman RF, et al. Projections of type 1 and type 2 diabetes burden in the US population aged <20 years through 2050. Diabetes Care. 2012;35:2515-20.