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Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

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THE ROLE OF LIFESTYLE MEDICINE IN TYPE 2 DIABETES: A COMPREHENSIVE REVIEW

Wojciech Gaska (Corresponding Author, Email: wgaska1@gmail.com)

Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOK in Lublin, Lublin, Poland

ORCID ID: 0009-0005-7621-3533

Julia Głowacka

Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOK in Lublin, Lublin, Poland

ORCID ID: 0009-0004-3262-5598

Mathias Spitaleri

7th Naval Hospital in Gdańsk, Gdańsk, Poland

ORCID ID: 0009-0007-0293-1764

Filip Kieloch

1st Clinical University Hospital in Lublin, Lublin, Poland

ORCID ID: 0009-0003-5116-9703

Dawid Sewruk

Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOK in Lublin, Lublin, Poland

ORCID ID: 0009-0008-4153-7126

Oskar Sienkiel

7th Naval Hospital in Gdańsk, Gdańsk, Poland

ORCID ID: 0009-0002-4524-0721

Agnieszka Fitas

4th Clinical University Hospital in Lublin, Lublin, Poland

ORCID ID: 0009-0005-9285-9174

Wiktor Gaska

University Clinical Centre in Gdańsk, Gdańsk, Poland

ORCID ID: 0009-0003-8818-988X

Karol Kanon

University Clinical Centre in Gdańsk, Gdańsk, Poland

ORCID ID: 0000-0001-6705-1302

Karolina Dębek-Kalinowska

Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOK in Lublin, Lublin, Poland

ORCID ID: 0000-0001-9931-6002

ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a progressive metabolic disorder strongly linked to modifiable lifestyle factors. Lifestyle medicine provides a multidomain, evidence-based framework for prevention and management of T2DM and its complications.

Objective: This narrative review synthesizes evidence on six lifestyle medicine domains—diet, physical activity, sleep, smoking cessation, stress management, and social support—in improving glycemic control, insulin sensitivity, and overall health in adults with T2DM.

Methods: A PubMed search was conducted using keywords including “type 2 diabetes,” “lifestyle medicine,” “diet,” “physical activity,” “sleep,” “smoking cessation,” “stress,” “mindfulness,” and “social support.” Peer-reviewed English-language studies were prioritized, emphasizing systematic reviews, meta-analyses, randomized controlled trials, and large cohort studies. Relevant guidelines and professional society reports were also reviewed.

Results: Carbohydrate-restricted, low-glycemic-index, and Mediterranean diets reduced HbA1c by 0.7–1.8% and fasting glucose by up to 1.3 mmol/L. Aerobic plus resistance training improved HbA1c (~0.7%), HOMA-IR, and VO₂ peak. Optimal sleep (7–8 h) correlated with better glycemic control, while short or long durations worsened HbA1c by 0.2–0.3%. Smoking impaired insulin sensitivity and β -cell function, whereas cessation reduced cardiovascular risk. Stress reduction and mindfulness programs lowered HbA1c by ~0.3% and improved mental health. Social interventions, including structured education and peer support, decreased HbA1c by ~0.25% and reduced isolation-related risks.

Conclusion: Lifestyle medicine offers synergistic, non-pharmacological strategies that enhance glycemic control, slow T2DM progression, and improve quality of life. Incorporating these interventions into routine practice is crucial for comprehensive diabetes care.

KEYWORDS

Type 2 Diabetes, Lifestyle Medicine, Diet, Physical Activity, Sleep, Smoking Cessation, Stress, Mindfulness, Social Support

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Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistently elevated blood glucose levels. Type 2 diabetes (T2DM) accounts for approximately 90% of all diabetes cases and results from insulin resistance combined with impaired pancreatic beta cell function. Initially, the body compensates for this condition by increasing insulin production, which allows normal glucose levels to be maintained. However, over time, beta cell function deteriorates and they are unable to maintain glucose balance, leading to hyperglycemia. [1] Environmental and metabolic factors, including visceral obesity, chronic inflammation of adipose tissue, low physical activity, and a high-calorie diet, also play an important role in the pathogenesis of the disease. In addition, genetic and epigenetic predispositions, including variants of more than 500 loci associated with β -cell function and insulin resistance, contribute to an increased risk of developing the disease. Epigenetic changes and intestinal microbiome disorders also play a role in the progression of type 2 diabetes. The pathophysiology of the disease is complemented by disturbances in glucagon secretion, impaired incretin function, oxidative stress, and the phenomenon of “metabolic memory.” [2] The diagnosis of diabetes is established based on specific glucose measurements. One criterion is a fasting plasma glucose (FPG) level of 126 mg/dL (7.0 mmol/L) or higher, where fasting is defined as no caloric intake for at least eight hours. Another diagnostic parameter is a plasma glucose concentration equal to or exceeding 200 mg/dL (11.1 mmol/L) measured two hours after administering a 75-gram oral glucose tolerance test (OGTT), which should be performed following World Health Organization (WHO) guidelines. Additionally, diabetes can be diagnosed if the glycated hemoglobin (HbA1c) level is 6.5% (48 mmol/mol) or above. Finally, in patients presenting

classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose measurement of 200 mg/dL (11.1 mmol/L) or greater is also diagnostic of diabetes. [3] The increase in the incidence of type 2 diabetes worldwide is closely related to rapid changes in lifestyle, growing populations, and longer life expectancy. This phenomenon particularly affects developing countries that are undergoing rapid urbanization and industrialization. Type 2 diabetes is associated with numerous chronic complications, such as cardiovascular disease, renal failure, neuropathy, and retinopathy. [4] Many cases of type 2 diabetes could be prevented by lifestyle modifications, including maintaining a healthy weight, eating a healthy diet, engaging in regular physical activity, avoiding tobacco use, and limiting alcohol consumption. [5]

Material and methods

This review was based on an extensive search of scientific literature in the PubMed database. The search strategy incorporated specific medical subject headings and keywords such as “type 2 diabetes,” “lifestyle medicine,” “diet,” “physical activity,” “sleep,” “smoking cessation,” “stress,” “mindfulness,” and “social support.” Only peer-reviewed articles published in English were included, with priority given to systematic reviews, meta-analyses, randomized controlled trials, and large cohort studies to ensure a high level of evidence. In addition to PubMed, authoritative sources from international scientific and regulatory bodies—such as professional society guidelines and official agency reports—were also consulted to provide comprehensive coverage of current recommendations and evidence-based practices.

Lifestyle medicine

Lifestyle medicine is a field of medicine based on scientific evidence that focuses on identifying and modifying behaviors that influence the development of chronic diseases. Its goal is to prevent, treat, and in many cases reverse diseases such as type 2 diabetes, ischemic heart disease, obesity, hypertension, and certain cancers. [6] The six pillars of lifestyle medicine include: a whole-food, plant-based diet based on unprocessed plant products, which supports metabolic health and reduces the risk of chronic diseases; regular physical activity, tailored to the patient's abilities, which improves cardiovascular fitness, insulin sensitivity, and mental well-being; adequate, restorative sleep, which is essential for the proper functioning of the endocrine, immune, and nervous systems; effective stress management using techniques such as mindfulness, meditation, or cognitive behavioral therapy; avoidance of risky substances such as tobacco, excessive alcohol, or other psychoactive substances that contribute to the development of many diseases; and building and maintaining positive social relationships, which have a documented impact on mental health and longevity. [7] Lifestyle interventions can lead to improved insulin sensitivity, lower HbA1c levels, and a reduced risk of developing type 2 diabetes. Regular physical activity improves glucose metabolism, and weight loss can help reverse insulin resistance. In addition, a healthy lifestyle reduces inflammation and improves pancreatic beta cell function, which can lead to better blood glucose control. [8]

Dietary interventions

The Nutrition and Food Council operating at the Medical Institute has determined the recommended range of macronutrients in the total energy intake: carbohydrates should provide 45–65% of energy, protein 10–35%, and fats 20–35%, while limiting the consumption of saturated and trans fats. These proportions are intended to meet the needs of most physically active people. In addition, more precise recommendations for carbohydrates and protein are often given in terms of body weight: 5 to 12 g of carbohydrates and 1.2 to 1.8 g of protein per kilogram of body weight, depending on the level of activity. [9] Low-carbohydrate diets are defined based on the proportion of carbohydrates in daily energy intake or their total amount consumed per day. There are several levels of consumption: a very low-carbohydrate diet includes less than 10% of energy from carbohydrates or 20–50 g per day. A low-carbohydrate diet is one in which the proportion is less than 26% of energy or less than 130 g per day. Moderate carbohydrate intake ranges from 26–44%, while high intake means 45% of energy or more. [10] The ketogenic diet, also known as keto, is a way of eating based on increased fat intake while limiting carbohydrates. Its goal is to promote weight loss, improve cognitive function, and increase energy levels. By significantly reducing carbohydrate intake and simultaneously increasing the proportion of fats and proteins, the body enters a state of ketosis—a metabolic process in which fat becomes the main source of energy, replacing glucose. The main goal of this diet is to reduce body fat and improve metabolic health parameters. [11] A low-fat diet is based on limiting fat in your daily menu to a maximum of 30% of your total calorie intake. A product is considered low-fat if it provides 100 calories and contains no

more than 3 grams of fat. Typical components of such a diet include vegetables, fruits, whole grains, egg whites, lean poultry (e.g., skinless chicken or turkey breast), legumes such as beans, lentils, and peas, seafood, and low-fat dairy products. [12] The Mediterranean diet (MD), described by Ancel Keys in the 1960s, is one of the most recognizable and widely studied dietary models in the world. It is a traditional way of eating characteristic of the population living in the Mediterranean region. [13] The traditional Mediterranean diet is characterized by a high proportion of plant-based products, such as fruits, vegetables, bread, and other grains, which are usually minimally processed, as well as potatoes, beans, nuts, and seeds. It is based on fresh, seasonal food from local crops, consumed in the least processed form possible. Fresh fruit is usually chosen as a dessert, while sweets with sugar or honey are consumed several times a week. The main source of fat is olive oil, especially extra virgin olive oil. Dairy products such as cheese and yogurt are consumed in moderate amounts, as are eggs – from zero to four per week. Fish and poultry are present in the diet in small or moderate portions, while red meat is rarely consumed. Wine is drunk in moderation, most often during meals. [14] The glycemic index (GI) was introduced in the early 1980s by David J. Jenkins and his team as a tool to assess how the consumption of carbohydrate products affects blood glucose levels after a meal. The GI is defined as the ratio of the area under the blood glucose concentration curve, measured 2 hours after consuming 50 g of carbohydrates from the test product, to the area under the curve obtained after consuming the reference product. Glucose or white bread is most often used as the reference product. Based on their GI values, products are divided into three groups: high GI (≥ 70), medium GI (55 to 69) and low GI (below 55). [15] Low glycemic index diets are those in which carbohydrates from low GI products predominate, such as legumes (beans, peas, lentils), pasta, pumpernickel bread, bulgur, parboiled rice, barley, and oats. [16] In 2011, the International Vegetarian Union (IVU) defined vegetarianism as a plant-based diet that may or may not include eggs, dairy products, and honey. It consists mainly of grains, vegetables, fruits, legumes, nuts, and seeds, excluding meat and certain products from live animals. There are two basic types of this diet: lacto-ovo vegetarianism, which includes eggs and dairy products but excludes meat, poultry, and animal products from saltwater and freshwater, and veganism, which eliminates all animal products, including meat, dairy products, eggs, and honey. [17] High-protein diets (providing 25–35% of energy) are often used for weight loss. In such diets, protein usually replaces carbohydrates, and the saturated fat content can be either low or high. [18] The network meta-analysis included 42 randomized controlled trials with 4809 patients with type 2 diabetes, comparing ten dietary approaches - Mediterranean, high-protein, ketogenic, vegetarian/vegan, low-fat, moderate-carbohydrate, low glycemic index, recommended, low-carbohydrate, and control diets (for example, continuing with the regular diet without making any changes).. Ketogenic, low-carbohydrate, and low-fat diets significantly reduced HbA1c by -0.73 (-1.19, -0.28), -0.69 (-1.32, -0.06), and -1.82 (-2.93, -0.71), respectively. Moderate-carbohydrate, low glycemic index, Mediterranean, high-protein, and low-fat diets significantly lowered fasting glucose by -1.30 (-1.92, -0.67), -1.26 (-2.26, -0.27), -0.95 (-1.51, -0.38), -0.89 (-1.60, -0.18), and -0.75 (-1.24, -0.27) compared to control diets. Combined outcome rankings highlighted ketogenic, Mediterranean, moderate-carbohydrate, and low glycemic index diets as effective for glycemic control. Although ketogenic diet showed the greatest effect, further high-quality, long-term studies are needed to confirm these results. [19]

Physical activity

The 2020 WHO recommendations for physical activity and reducing sedentary behavior in adults include performing at least 150–300 minutes of moderate-intensity physical activity (MPA) or 75–150 minutes of vigorous-intensity physical activity (VPA) per week, or an equivalent combination. For additional health benefits, it is recommended to increase weekly MPA to more than 300 minutes or VPA to more than 150 minutes. Physical activities are often categorized by intensity based on their MET value. MET (metabolic equivalent) is a unit used to express the rate of energy expenditure. One MET represents the energy cost of sitting quietly at rest and is conventionally defined as an oxygen uptake of 3.5 milliliters per kilogram of body weight per minute. On an absolute scale, moderate-intensity activities range from 3.0 to 5.9 METs, while vigorous-intensity activities are defined as those equal to or exceeding 6.0 METs. At least twice a week, moderate- or higher-intensity muscle-strengthening exercises involving all major muscle groups should also be performed. Resistance training is defined as repeated voluntary muscle contractions against a load greater than that encountered during daily activities. This type of training enhances strength by promoting adaptations in both the muscular and nervous systems. The WHO also recommends limiting the time spent in a seated position and replacing it with any form of physical activity. [20,21,22] The Physical Activity Guidelines for Americans recommend that individuals with type 2 diabetes engage in aerobic exercise at moderate intensity

— defined as 40 to 59 percent of heart rate reserve (HRR) or 3 to 6 METs — for 20 to 60 minutes, on 3 to 7 days per week. Vigorous aerobic exercise, at 60 to 89 percent of HRR or above 6 METs, may offer additional benefits but may not be suitable for everyone. Resistance (strength) training should be performed 2 to 3 times per week, consisting of 1 to 3 sets of 10 to 15 repetitions at 50 to 80 percent of one-repetition maximum (1-RM). Flexibility exercises, recommended 2 to 3 times weekly, help maintain joint mobility, while balance training is especially important for older adults or those at increased risk of falls. Exercise programs should be personalized based on individual health conditions, comorbidities, and preferences, with medical clearance advised before initiation — especially in those with complications. A combination of aerobic and resistance training is most effective for improving blood glucose control, cardiovascular health, and physical function in people with type 2 diabetes. [23] Exercise training recommendations by European Society of Cardiology says both aerobic and resistance training improve glycaemic control, blood pressure, weight loss, exercise capacity, and dyslipidaemia in type 2 diabetes patients. They should aim to walk over 30 minutes at least five days a week, ideally daily. A supervised exercise program should include ≥ 150 minutes of moderate-intensity (40–60% HRR) or 90 minutes of vigorous-intensity (60–80% HRR) endurance exercise weekly, ideally totaling 3–4 hours. Exercising at least five days per week with an energy expenditure of 1000–2000 kcal per week for six months or more is recommended. Resistance training should be done 2–3 times weekly, targeting major muscle groups with 2 sets of 8–12 reps at 70–80% 1RM or 25–30 reps at 40–55% 1RM. Electro muscle stimulation (EMS) twice weekly for 20 minutes can benefit patients with limited mobility by improving HbA1c. [24] The analysis comprised 27 studies with 38 intervention groups, including a total of 1372 participants—737 in exercise groups and 635 in control groups—accumulating 39,435 patient-hours of exercise training. Results demonstrated that exercise significantly improved glycosylated hemoglobin (HbA1c) with a mean difference (MD) of -0.71% (95% CI -1.11 to -0.31; $p = 0.0005$). Additionally, longer exercise duration was associated with greater HbA1c reduction, ranging from 0.009% to 0.04% per additional week ($p = 0.002$). Participants engaging in vigorous-intensity exercise showed an increase in peak oxygen consumption (peak VO₂) between 0.64 and 5.98 ml/kg/min compared to those performing low or moderate-intensity activities. Exercise also improved insulin resistance as measured by HOMA-IR (MD: -1.02; 95% CI -1.77 to -0.28; $p = 0.007$), fasting serum glucose (MD: -12.53 mmol/L; 95% CI -18.94 to -6.23; $p < 0.0001$), and serum insulin levels (MD: -10.39 IU; 95% CI -17.25 to -3.53; $p = 0.003$). [25] The impact of physical activity on HbA1c levels can be attributed to various physiological processes, such as enhanced glucose uptake by skeletal muscles, decreased production of inflammatory cytokines, improved function of fat cells, reduced autonomic nervous system activity, better endothelial and heart function, as well as increased arterial stiffness. However, individual responses to physical activity in terms of blood glucose regulation vary widely. Additionally, excessive exercise has been linked to mitochondrial dysfunction and impaired glucose tolerance, which may influence how physical activity affects changes in HbA1c. [26]

Sleep disturbances

In a previously published meta-analysis investigating the relationship between sleep disturbances and glycaemic control in individuals with type 2 diabetes, 20 studies were identified—eight assessing sleep duration and fifteen evaluating sleep quality. Of these, fifteen studies were included in the quantitative synthesis. The analysis demonstrated that both short and long sleep durations were associated with increased levels of glycated hemoglobin (HbA1c), with weighted mean differences of 0.23% [0.10–0.36] and 0.13% [0.02–0.25], respectively, when compared to normal sleep duration. These results suggest a U-shaped dose-response relationship. Additionally, poor sleep quality was associated with elevated HbA1c levels (weighted mean difference: 0.35% [0.12–0.58]). These findings highlight the relevance of both sleep quantity and quality in the metabolic regulation of patients with type 2 diabetes. [27] Another analysis demonstrated that both short (<7 hours) and long (>8 hours) nighttime sleep durations are significantly associated with an increased risk of developing type 2 diabetes mellitus (T2DM), with odds ratios of 1.18 (95% CI: 1.13–1.23) and 1.13 (95% CI: 1.09–1.18), respectively. Furthermore, poor sleep quality (OR = 1.50; 95% CI: 1.30–1.72) and an evening chronotype (OR = 1.59; 95% CI: 1.18–2.13) were also linked to a heightened risk. Daytime naps exceeding 30 minutes increased the risk of T2DM by 7–20%. The combination of poor sleep quality and nighttime sleep duration over 8 hours was associated with the highest risk of T2DM onset (OR = 2.15; 95% CI: 1.19–3.91). These findings indicate a U-shaped relationship between sleep duration and T2DM, with the lowest risk observed among individuals sleeping between 7 and 8 hours. Additionally, adverse sleep traits—including insufficient or excessive sleep duration, poor sleep quality, evening chronotype, and prolonged daytime napping—appear to interact and collectively amplify the risk of developing T2DM. [28] Due to the

negative effects of insufficient and fragmented sleep on quality of life, recovery, and diabetes management, sleep-related issues and their impact on well-being should be carefully addressed by healthcare providers treating individuals with diabetes mellitus. Diagnosing and managing sleep disturbances in patients with type 2 diabetes is essential, as it may help prevent disease progression. Therefore, incorporating sleep education into diabetes care should be considered a key component for improving health and overall quality of life. [29]

Smoking

The global average prevalence of tobacco use among people with type 2 diabetes was 20.81% (95% confidence interval: 18.93–22.76%). This rate was higher in the WHO regions of East Asia and the Pacific, as well as South Asia, compared to the Americas, the Middle East and North Africa, Europe, and Central Asia. Studies that compared tobacco use between people with type 2 diabetes and those without found that individuals with diabetes were 26% less likely to use tobacco (pooled odds ratio = 0.74; 95% CI: 0.61–0.88). [30] Smoking negatively affects type 2 diabetes through harmful changes in body composition, including increased abdominal obesity and a higher visceral-to-subcutaneous fat ratio, mainly driven by nicotine. It impairs glucose tolerance and reduces insulin sensitivity. In people with diabetes, smoking lowers total body glucose disposal and insulin action, as shown by clamp studies. Additionally, smoking dose-dependently worsens pancreatic β -cell function, leading to decreased insulin secretion. This effect remains significant after adjusting for other risk factors and is more pronounced in men than women. [31] Active tobacco smoking is associated with a significant increase in the risk of all-cause mortality and cardiovascular events among patients with diabetes, while smoking cessation is linked to a reduced risk compared to current smoking. Research findings provide strong evidence supporting the recommendation for smoking cessation in patients with diabetes. [32] Varenicline is an effective and well-tolerated pharmacological aid for smoking cessation in individuals with type 2 diabetes, significantly increasing the likelihood of sustained abstinence with minimal and mild side effects. Evidence for bupropion and nicotine replacement therapy is less certain. Behavioral support and active involvement of healthcare providers play a crucial role in enhancing the success of quitting smoking and maintaining long-term abstinence. [33]

Stress and mindfulness

Long-term stress puts a strain on the brain and the entire body, which over time can contribute to the development of serious diseases such as type 2 diabetes. Stress activates the hypothalamic-pituitary-adrenal axis, leading to increased secretion of glucocorticoids and catecholamines and activation of inflammatory mechanisms of the immune system. These processes disrupt glucose and lipid metabolism, which in turn promotes insulin resistance, damage to pancreatic beta cells, and the development of type 2 diabetes. In addition, stress can disrupt the balance of the intestinal microflora, negatively affecting the gut-brain-immune system axis, which is reflected, among other things, in stress-related depression, which often co-occurs with type 2 diabetes. [34] Research indicates that fetal exposure to maternal bereavement—occurring either before conception or during pregnancy—may be linked to an elevated risk of developing type 2 diabetes later in childhood or early adulthood. [35] Mindfulness-Based Stress Reduction (MBSR) is a structured group intervention designed to alleviate stress, enhance mental well-being, and reduce pain and suffering. The program helps individuals cultivate greater awareness and acceptance of both internal and external experiences, enabling them to respond to stressors more constructively rather than reactively. Over time, this approach fosters emotional resilience by promoting self-awareness and encouraging individuals to take responsibility for their actions, ultimately supporting more effective health-related behaviors. [36] Mindfulness-based interventions may positively influence glycemic control, as indicated by reductions in hemoglobin A1c (HbA1c) levels, and help alleviate stress, depression, and anxiety in individuals with diabetes. Four out of five systematic reviews and meta-analyses demonstrated a significant decrease in HbA1c of around 0.3%. However, concerns about the overall quality of the included studies introduce uncertainty regarding the effectiveness of mindfulness as a therapeutic approach for diabetes. Additional research is required to clarify its biological impact on physiological, neurological, and endocrine systems. [37]

Social connections

Loneliness and lack of social engagement have been linked to an increased risk of developing type 2 diabetes. Unlike social isolation, which refers to the objective absence of social interactions, loneliness reflects a subjective emotional experience that can persist despite having social contacts. This emotional distress can activate stress responses, promote depression or anxiety, and disrupt metabolic regulation. Both loneliness and isolation may trigger physiological mechanisms such as inflammation and dysregulation of the hypothalamic–pituitary–adrenal axis, which negatively affect insulin sensitivity and glucose control. Furthermore, individuals experiencing loneliness or isolation are often less likely to engage in healthy behaviors, such as regular physical activity or a balanced diet, and more likely to adopt harmful habits, further increasing their diabetes risk. [38] In a large prospective cohort study involving over 26,000 adults with type 2 diabetes in the UK, researchers investigated the relationship between social isolation, loneliness, and mortality. Based on self-reported data and national death records, the findings revealed that social isolation was significantly linked to higher risks of death from all causes, cardiovascular disease, and cancer. Loneliness, on the other hand, was associated with increased all-cause and cardiovascular mortality, but not with cancer-related deaths. These results highlight the adverse impact of limited social connection on health outcomes in individuals with type 2 diabetes. [39] A review of randomized clinical trials involving patients with type 2 diabetes showed that interventions involving social networks—although often based on education and individual behavioral changes – contributed to an increase in the level of perceived social support and a moderate improvement in glycemic control, manifested by an average decrease in glycated hemoglobin (HbA1c) of approximately 0.25 percentage points. Although no clear improvement in quality of life was observed, the results suggest that harnessing the potential of social networks can support the therapeutic process. However, these interventions have been relatively rarely studied and have rarely been based on social theories, indicating a need for further research into their effectiveness. [40]

Conclusions:

The literature review clearly shows that an interdisciplinary approach based on the principles of lifestyle medicine is a key element in both the prevention and treatment of type 2 diabetes. Changing eating habits, with particular emphasis on diets with limited carbohydrate intake (ketogenic, low glycemic index, Mediterranean), significantly reduces HbA1c levels, while regular physical activity combining aerobic and strength training improves insulin sensitivity, glycemic control, and body weight. Both sleep deprivation and excess sleep, as well as poor sleep quality, are associated with poorer metabolic control, which highlights the need to assess and optimize sleep hygiene in daily diabetes practice. Quitting smoking brings measurable benefits in terms of β -cell function and reduction of the risk of cardiovascular complications, while stress reduction techniques and mindfulness interventions have a beneficial effect on mental and metabolic parameters, although they require further validation in long-term studies. Finally, social support and counteracting isolation significantly reduce mortality and improve treatment outcomes, indicating the need to integrate social strategies into conventional therapeutic programs. Almost all of the interventions described are synergistic in nature, suggesting that comprehensive implementation of lifestyle medicine principles can significantly improve glycemic control, slow disease progression, and improve the quality of life of people with type 2 diabetes.

Disclosure:**Authors contribution:**

Conceptualization: Wojciech Gąska, Agnieszka Fitas, Karolina Dębek-Kalinowska, Mathias Spitaleri
 methodology: Julia Głowacka, Oskar Sienkiel, Wiktor Gąska
 software: Dawid Sewruk, Wiktor Gąska
 check: Wiktor Gąska, Karol Kanon, Karolina Dębek-Kalinowska, Oskar Sienkiel
 formal analysis: Agnieszka Fitas, Julia Głowacka, Filip Kieloch, Karolina Dębek-Kalinowska
 investigation: Mathias Spitaleri, Oskar Sienkiel, Wiktor Gąska
 resources: Filip Kieloch, Karol Kanon, Julia Głowacka
 data curation: Agnieszka Fitas, Mathias Spitaleri, Dawid Sewruk, Filip Kieloch, Karol Kanon
 writing - rough preparation: Karol Kanon, Mathias Spitaleri, Dawid Sewruk
 writing - review and editing: Karolina Dębek-Kalinowska, Wojciech Gąska, Dawid Sewruk, Agnieszka Fitas
 visualization: Oskar Sienkiel, Wiktor Gąska
 supervision: Wojciech Gąska, Julia Głowacka
 project administration: Wojciech Gąska, Filip Kieloch
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REFERENCES

1. Goyal, R., Singhal, M., & Jialal, I. (2025). *Type 2 diabetes*. In StatPearls. StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK513253/>
2. Galicia-Garcia, U., Benito-Vicente, A., Jebari, S., Larrea-Sebal, A., Siddiqi, H., Uribe, K. B., ... Martín, C. (2020). Pathophysiology of type 2 diabetes mellitus. *International Journal of Molecular Sciences*, 21(17), 6275. <https://doi.org/10.3390/ijms21176275>
3. ElSayed, N. A., Aleppo, G., Aroda, V. R., Bannuru, R. R., Brown, F. M., Bruemmer, D., ... Gabbay, R. A., on behalf of the American Diabetes Association. (2023). Classification and diagnosis of diabetes: Standards of care in diabetes-2023. *Diabetes Care*, 46(Suppl. 1), S19–S40. <https://doi.org/10.2337/dc23-S002>
4. He, K. J., Wang, H., Xu, J., Gong, G., Liu, X., & Guan, H. (2024). Global burden of type 2 diabetes mellitus from 1990 to 2021, with projections of prevalence to 2044: A systematic analysis across SDI levels for the global burden of disease study 2021. *Frontiers in Endocrinology*, 15, 1501690. <https://doi.org/10.3389/fendo.2024.1501690>
5. Zheng, Y., Ley, S. H., & Hu, F. B. (2018). Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. *Nature Reviews Endocrinology*, 14(2), 88–98. <https://doi.org/10.1038/nrendo.2017.151>
6. Yeh, B. I., & Kong, I. D. (2013). The advent of lifestyle medicine. *Journal of Lifestyle Medicine*, 3(1), 1–8. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4390753/>
7. Lippman, D., Stump, M., Veazey, E., Guimarães, S. T., Rosenfeld, R., Kelly, J. H., ... Katz, D. L. (2024). Foundations of lifestyle medicine and its evolution. *Mayo Clinic Proceedings: Innovations, Quality & Outcomes*, 8(1), 97–111. <https://doi.org/10.1016/j.mayocpiqo.2023.11.004>
8. John, N. A., John, J., Tarnikanti, M., Kalpana, M., Kamble, P., Singhal, A., ... Vamishidhar, I. S. (2023). Implications of lifestyle medicine in medical practice. *Journal of Family Medicine and Primary Care*, 12(2), 208–212. https://doi.org/10.4103/jfmpc.jfmpc_1587_22
9. Manore, M. M. (2005). Exercise and the Institute of Medicine recommendations for nutrition. *Current Sports Medicine Reports*, 4(4), 193–198. <https://doi.org/10.1097/01.csmr.0000306206.72186.00>
10. Oh, R., Gilani, B., & Uppaluri, K. R. (2025). *Low-carbohydrate diet*. In StatPearls. StatPearls Publishing.
11. Masood, W., Annamaraju, P., Khan Suheb, M. Z., & Uppaluri, K. R. (2025). *Ketogenic diet*. In StatPearls. StatPearls Publishing.
12. Bhandari, P., & Sapra, A. (2025). *Low fat diet*. In StatPearls. StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK553097/>

13. Kiani, A. K., Medori, M. C., Bonetti, G., Aquilanti, B., Velluti, V., Matera, G., ... Bertelli, M. (2022). Modern vision of the Mediterranean diet. *Journal of Preventive Medicine and Hygiene*, 63(2 Suppl 3), E36–E43. <https://doi.org/10.15167/2421-4248/jpmh2022.63.2S3.2745>
14. Guasch-Ferré, M., & Willett, W. C. (2021). The Mediterranean diet and health: A comprehensive overview. *Journal of Internal Medicine*, 290(3), 549–566. <https://doi.org/10.1111/joim.13333>
15. Ni, C., Jia, Q., Ding, G., Wu, X., & Yang, M. (2022). Low-glycemic index diets as an intervention in metabolic diseases: A systematic review and meta-analysis. *Nutrients*, 14(2), 307. <https://doi.org/10.3390/nu14020307>
16. Brand-Miller, J., Hayne, S., Petocz, P., & Colagiuri, S. (2003). Low-glycemic index diets in the management of diabetes: A meta-analysis of randomized controlled trials. *Diabetes Care*, 26(8), 2261–2267. <https://doi.org/10.2337/diacare.26.8.2261>
17. Baroni, L. (2015). Vegetarianism in food-based dietary guidelines. *International Journal of Nutrition*, 1(2), 48–73. <https://doi.org/10.14302/issn.2379-7835.ijn-14-588>
18. Clifton, P. M., & Keogh, J. (2007). Metabolic effects of high-protein diets. *Current Atherosclerosis Reports*, 9(6), 472–478. <https://doi.org/10.1007/s11883-007-0063-y>
19. Jing, T., Zhang, S., Bai, M., Chen, Z., Gao, S., Li, S., & Zhang, J. (2023). Effect of dietary approaches on glycemic control in patients with type 2 diabetes: A systematic review with network meta-analysis of randomized trials. *Nutrients*, 15(14), 3156. <https://doi.org/10.3390/nu15143156>
20. Okely, A. D., Kontsevaya, A., Ng, J., & Abdeta, C. (2021). 2020 WHO guidelines on physical activity and sedentary behavior. *Sports Medicine and Health Science*, 3(2), 115–118. <https://doi.org/10.1016/j.smhs.2021.05.001>
21. World Health Organization. (2010). *Global recommendations on physical activity for health* (Appendix 5, Glossary). <https://www.ncbi.nlm.nih.gov/books/NBK305048/>
22. Lee, M., & Carroll, T. J. (2007). Cross education: Possible mechanisms for the contralateral effects of unilateral resistance training. *Sports Medicine*, 37(1), 1–14. <https://doi.org/10.2165/00007256-200737010-00001>
23. Kanaley, J. A., Colberg, S. R., Corcoran, M. H., Malin, S. K., Rodriguez, N. R., Crespo, C. J., ... Zierath, J. R. (2022). Exercise/physical activity in individuals with type 2 diabetes: A consensus statement from the American College of Sports Medicine. *Medicine & Science in Sports & Exercise*, 54(2), 353–368. <https://doi.org/10.1249/MSS.0000000000002800>
24. European Society of Cardiology. (2025). Physical exercise. <https://www.escardio.org/Education/ESC-Prevention-of-CVD-Programme/Treatment-goals/Risk-factor-control/physical-exercise>
25. Grace, A., Chan, E., Giallauria, F., Graham, P. L., & Smart, N. A. (2017). Clinical outcomes and glycaemic responses to different aerobic exercise training intensities in type II diabetes: A systematic review and meta-analysis. *Cardiovascular Diabetology*, 16, 37. <https://doi.org/10.1186/s12933-017-0518-6>
26. Gallardo-Gómez, D., Salazar-Martínez, E., Alfonso-Rosa, R. M., Ramos-Munell, J., Del Pozo-Cruz, J., Del Pozo Cruz, B., & Álvarez-Barbosa, F. (2024). Optimal dose and type of physical activity to improve glycemic control in people diagnosed with type 2 diabetes: A systematic review and meta-analysis. *Diabetes Care*, 47(2), 295–303. <https://doi.org/10.2337/dc23-0800>
27. Lee, S. W. H., Ng, K. Y., & Chin, W. K. (2017). The impact of sleep amount and sleep quality on glycemic control in type 2 diabetes: A systematic review and meta-analysis. *Sleep Medicine Reviews*, 31, 91–101. <https://doi.org/10.1016/j.smrv.2016.02.001>
28. Liu, H., Zhu, H., Lu, Q., Ye, W., Huang, T., Li, Y., ... Ji, L. (2025). Sleep features and the risk of type 2 diabetes mellitus: A systematic review and meta-analysis. *Annals of Medicine*, 57(1), 2447422. <https://doi.org/10.1080/07853890.2024.2447422>
29. Darraj, A. (2023). The link between sleeping and type 2 diabetes: A systematic review. *Cureus*, 15(11), e48228. <https://doi.org/10.7759/cureus.48228>
30. Roderick, P., Turner, V., Readshaw, A., Dogar, O., & Siddiqi, K. (2019). The global prevalence of tobacco use in type 2 diabetes mellitus patients: A systematic review and meta-analysis. *Diabetes Research and Clinical Practice*, 154, 52–65. <https://doi.org/10.1016/j.diabres.2019.05.035>
31. Maddatu, J., Anderson-Baucum, E., & Evans-Molina, C. (2017). Smoking and the risk of type 2 diabetes. *Translational Research*, 184, 101–107. <https://doi.org/10.1016/j.trsl.2017.02.004>
32. Pan, A., Wang, Y., Talaei, M., & Hu, F. B. (2015). Relation of smoking with total mortality and cardiovascular events among patients with diabetes mellitus: A meta-analysis and systematic review. *Circulation*, 132(19), 1795–1804. <https://doi.org/10.1161/CIRCULATIONAHA.115.017926>
33. Martin, A., La Rosa, G. R. M., Rice, H., Bertuzzi, A., Witkowski, M., Anastasi, E., ... Polosa, R. (2025). Pharmacological interventions for smoking cessation in type 2 diabetes: A systematic review with meta-analysis and GRADE evaluation. *Diabetes Research and Clinical Practice*, 224, 112202. <https://doi.org/10.1016/j.diabres.2025.112202>
34. Lisco, G., Giagulli, V. A., De Pergola, G., Guastamacchia, E., Jirillo, E., Vitale, E., & Triggiani, V. (2024). Chronic stress as a risk factor for type 2 diabetes: Endocrine, metabolic, and immune implications. *Endocrine, Metabolic & Immune Disorders Drug Targets*, 24(3), 321–332. <https://doi.org/10.2174/1871530323666230803095118>

35. Virk, J., Li, J., Vestergaard, M., Obel, C., Kristensen, J. K., & Olsen, J. (2012). Prenatal exposure to bereavement and type-2 diabetes: A Danish longitudinal population-based study. *PLoS ONE*, 7(8), e43508. <https://doi.org/10.1371/journal.pone.0043508>
36. Nikkhah Ravari, O., Mousavi, S. Z., & Babak, A. (2020). Evaluation of the effects of 12 weeks mindfulness-based stress reduction on glycemic control and mental health indices in women with diabetes mellitus type 2. *Advances in Biomedical Research*, 9, 61. https://doi.org/10.4103/abr.abr_133_20
37. Hamasaki, H. (2023). The effects of mindfulness on glycemic control in people with diabetes: An overview of systematic reviews and meta-analyses. *Medicines*, 10(9), 53. <https://doi.org/10.3390/medicines10090053>
38. Ezzatvar, Y., Caballero, Ó., Duclos-Bastias, D., Yáñez-Sepúlveda, R., & García-Hermoso, A. (2025). Loneliness and social isolation as risk factors for type 2 diabetes onset: A systematic review and meta-analysis. *Diabetes Research and Clinical Practice*, 223, 112124. <https://doi.org/10.1016/j.diabres.2025.112124>
39. Duan, X., Wei, Y., Ge, Y., Bo, Y., Wang, X., & Zhu, Y. (2025). Association of social isolation and loneliness with all-cause and cause-specific mortality among individuals with type 2 diabetes: A prospective study in UK Biobank. *Journal of Epidemiology and Community Health*, 79(7), 498–505. <https://doi.org/10.1136/jech-2024-222775>
40. Spencer-Bonilla, G., Ponce, O. J., Rodriguez-Gutierrez, R., Alvarez-Villalobos, N., Erwin, P. J., Larrea-Mantilla, L., ... Montori, V. M. (2017). A systematic review and meta-analysis of trials of social network interventions in type 2 diabetes. *BMJ Open*, 7(8), e016506. <https://doi.org/10.1136/bmjopen-2017-016506>