Influence of lifestyle on the course of type 1 diabetes mellitus

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Abstract

Type 1 diabetes (T1DM) is an autoimmune disease that requires insulin treatment from the time of diagnosis. Its clinical course depends on both genetic and environmental factors, and the lifestyle of a patient modulates their interaction. The evidence about the influence of lifestyle on the course of T1DM is increasing. In this paper, we present evidence on the relationship between lifestyle parameters and diabetes-related outcomes. We discuss the most commonly addressed factors associated with lifestyle, such as physical activity, nutrition and smoking, and those with sparse evidence in T1DM, such as socioeconomic status, sleep duration, psychological stress and illicit drugs intake.

Key words: physical activity, nutrition, smoking, stress, socioeconomic conditions.

Clinical course of type 1 diabetes

Since the first therapeutic use of insulin the clinical course of type 1 diabetes (T1DM) has changed dramatically from a disease with very short life expectancy to a chronic condition, the course of which is influenced largely by the development of chronic complications.

The clinical onset of T1DM is preceded by a preclinical phase of variable duration, characterized by progressive autoimmune destruction of beta cells and presence of specific autoantibodies. In genetically susceptible individuals, this autoimmune reaction seems to be initiated and modulated by exposure to various environmental triggers and regulators, which might include viral infections or introducing cow’s milk or cereals into the diet [1]. According to the accelerator hypothesis [2], an increased rate of β-cell apoptosis and insulin resistance modulate the timing of clinical onset and subsequent course of autoimmune diabetes. However, the autoimmune process is thought to be the main accelerator of β-cell destruction both before and after onset of T1DM [3].

Clinical manifestation of T1DM varies considerably in severity with greater insulin deficiency and greater risk of diabetic ketoacidosis (DKA) in children and adolescents than in adults [4]. After introduction of insulin treatment, many patients enter clinical remission of the disease with partially restored endogenous insulin secretion and near-normoglycemia on very low doses of exogenous insulin. In many patients with phenotypic
T1DM residual insulin secretion can be maintained for over 30 years, and is associated with better metabolic control, including decreased incidence of both hyper- and hypoglycemia, and decreased risk of chronic complications when compared with patients without detectable insulin secretion [5]. In the DCCT/EDIC (Diabetes Control and Complications Trial/ Epidemiology of Diabetes Interventions and Complications) cohort, good initial metabolic control had a protective effect against chronic complications even despite subsequent deterioration in glycemic control [6]. Chronic complications of T1DM typically do not occur before 5 years from the disease onset, with the highest risk of their early development in patients with diabetes diagnosed in puberty. Progression of microvascular complications leads to decreased quality of life and increased risk of macrovascular disease [7]. Cardiovascular complications are the main cause of death in patients with T1DM. In the FinnDiane study, the 7-year mortality in patients with T1DM was three times higher than in the general population, although the difference disappeared after excluding patients with nephropathy [8].

To describe quantitatively the efficacy of interventions on the course of diabetes many endpoints are used. The hard endpoints include: time of the disease onset (in prevention trials), onset and progression of microvascular complications (e.g. development of end-stage renal failure or proliferative retinopathy), acute hyperglycemic states, episodes of hypoglycemia, cardiovascular events, and mortality. The surrogate endpoints and other commonly used outcomes that characterize the course of diabetes may include: parameters of metabolic control, occurrence and duration of clinical remission, measures of residual insulin secretion, exogenous insulin dose, insulin sensitivity, and quality of life.

The clinical course of T1DM depends on a number of factors, including genetic background [9], metabolic control (control of glycaemia, lipid profile, blood pressure, body weight), treatment regimen used, physical activity, patient knowledge about diabetes, psychosocial factors and concomitant diseases. It is clear that many of these factors interact with lifestyle, and the lifestyle of a patient may in many ways influence the clinical course of T1DM.

To prepare this narrative review we searched the PubMed database using keywords describing lifestyle parameters (lifestyle, nutrition, diet, exercise, training, sport, physical activity, fitness, smoking, sleep, stress, socioeconomic, income, education) and diabetes-related outcomes (mortality, complications, cardiovascular, macroangiopathy, microangiopathy, retinopathy, nephropathy, neuropathy, glycemic control, glycated hemoglobin (HbA1c), glycated hemoglobin, lipid profile, cholesterol, triglyceride, blood pressure, remission, insulin secretion, insulin requirement, insulin dose, insulin resistance). We confined the search to papers where patients with T1DM (searched also using the keyword insulin-dependent diabetes mellitus) were the only investigated group or one of the subgroups with separately presented outcomes. Papers where the type of diabetes was not specified were excluded. The studies conducted in patients with T1DM which are discussed in this review are summarized in Table I.

**Definition of lifestyle**

Lifestyle is defined (according to the Thesaurus of Psychological Index Terms [10]) as a "typical way of life or manner of living characteristic of an individual or group" and is influenced by socioeconomic, educational, and cultural factors. The lifestyle is closely related with the health behavior, defined as behaviors expressed by individuals to protect, maintain or promote their health status. In the World Health Organization’s Health Promotion Glossary [11] the lifestyle is defined as a "way of living based on identifiable patterns of behaviour which are determined by the interplay between an individual’s personal characteristics, social interactions, and socioeconomic and environmental living conditions". The parameters most commonly analyzed in the assessment of lifestyle interventions include physical activity and nutrition. Other parameters that were taken into account include socioeconomic level, exposure to emotional stress, sleep duration, and specific unhealthy behaviors, including smoking, excessive alcohol intake and illicit drug use.

In the influence of a patient’s lifestyle on the course of T1DM there is an important role for an appropriate treatment regimen. For the majority of patients with T1DM, the treatment of choice is intensive insulin therapy with pen injections or personal pumps, with dose adjustment based on glycaemia, macronutrient (at least carbohydrate) content of the meals and planned physical activity. In the DCCT/EDIC cohort, intensive insulin treatment was effective in the prevention of chronic complications of diabetes [12]. Allowing high flexibility in dietary choices and meal planning, as well as safe practice of physical activity, the flexible intensive insulin treatment interferes little with a patient’s lifestyle and has the potential of achieving the best possible outcome.

**Physical activity**

The beneficial effects of physical activity in the general population are numerous and include, apart from enhancement of well-being, decreased...
Table I. Lifestyle parameters and diabetes-related outcomes. Summary of reviewed studies

| Ref. | First author, publication date | Intervention or exposure | Study design | N (cases/controls) | Main outcome(s) |
|------|-------------------------------|--------------------------|--------------|--------------------|-----------------|
| [15] | Moy, 1993                     | Overall physical activity level | OP           | 548                | Mortality       |
| [18] | Zinman, 1984                  | Aerobic exercise         | CT           | 13/7               | Glycemia, HbA1c |
| [19] | Durak, 1990                   | Progressive resistance training | RCT, crossover | 8          | HbA1c, lipid profile, muscle strength |
| [20] | Laaksonen, 2000               | Aerobic exercise         | RCT          | 28/28              | Lipid profile   |
| [21] | Tonoli, 2012                  | Aerobic and/or resistance training | Meta-analysis | 33 studies¹   | HbA1c, glycemia |
| [25] | Rigla, 2000                   | Aerobic and resistance training | Self-controlled | 14              | Lipid profile, BMI, VO2max |
| [26] | Mosher, 1998                  | Aerobic exercise         | CT           | 10/10              | HbA1c, fasting glycemia, lipid profile, muscle strength |
| [27] | Kaplan, 1997                  | Aerobic exercise         | Self-controlled | 20              | Lipid profile, BP, body fat, VO2max |
| [29] | LaPorte, 1986                 | Various types of exercise | Case-control | 696               | Chronic complications of diabetes, mortality |
| [31] | Chen, 2008                    | Various types of exercise | C-S          | 93/107             | Heart-rate variability |
| [33] | Yki-Jarvinen, 1984            | Aerobic exercise         | CT           | 7/6                | Glucose disposal rate, insulin requirement, HbA1c, lipid profile |
| [36] | Seeger, 2011                  | Aerobic exercise         | Self-controlled | 7                | Vascular function (flow-mediated dilation) |
| [37] | Fuchsjager-Mayrl, 2002        | Aerobic exercise         | CT           | 18/8               | Vascular function (flow-mediated dilation, fundus pulsation amplitude) |
| [40] | Zoppini, 2003                 | Various types of exercise | C-S          | 30/23              | Quality of life |
| [48] | Buyken, 2001                  | Glycemic index of diet   | C-S          | 2810               | HbA1c, lipid profile |
| [49] | Bortsov, 2011                 | Sugar-sweetened and diet beverage intake | C-S | 1806 | HbA1c, lipid profile |
| [50] | Nansel, 2012                  | Diet quality and glycemic index | C-S | 252 | HbA1c, BMI |
| [52] | Delahanty, 2009               | Macronutrient composition | RCT          | 532                | HbA1c, lipid profile, BMI |
| [53] | Snell-Bergeon, 2009           | Macronutrient composition | C-S          | 571/696            | Coronary artery calcium, coronary heart disease risk factors |
| [55] | Strychar, 2009                | Macronutrient composition | RCT          | 30 (15/15)         | BMI, BP, HbA1c, lipid profile, serum plasminogen activator inhibitor-1 |
| [60] | Matheus, 2011                 | Serum uric acid concentration | C-S | 57/53 | Microvascular endothelial function (laser Doppler perfusion monitoring) |
| [73] | Moy, 1990                     | Cigarette smoking        | OP           | 548                | Mortality       |
| [74] | Chiodera, 1997                | Cigarette smoking        | CT           | 10/10              | Growth hormone, vasopressin, and cortisol concentrations |
risk of cardiovascular and all-cause mortality, hypertension, obesity, dyslipidemia, type 2 diabetes (T2DM), osteoporosis, and cancer [13]. Some of these effects were also documented in patients with T2DM, but the body of evidence for T1DM is limited [14]. Therefore, many recommendations on physical activity for patients with T1DM are based on conclusions drawn from studies on patients with T2DM or on healthy individuals.

Patients with T1DM undertake lower than advised levels of physical activity, but these levels are similar to nondiabetic subjects [15]. The sedentary lifestyle and increased time spent watching television or using a computer was associated with poor glycemic control in young patients with T1DM [16]. However, the beneficial impact of physical activity on glycemic control of T1DM is less documented than for patients with T2DM [17]. Some studies have demonstrated the improvement in HbA1c after supervised or unsupervised physical activity [18, 19], while other studies showed no benefit [20]. The possible explanation of the lack of improvement in glycemic control may be increased energy consumption and reduced insulin dose associated with increased activity, and, very likely, lack of incorporation of exercise into a structured lifestyle modification plan, introduced along with relevant patient education [18]. The influence of physical activity on glycemic control may also depend on the form of exercise training. The results of a recent meta-analysis demonstrate that only regular aerobic exercise training programs significantly improved acute and chronic glycemic control [21]. Interestingly, addition of brief bouts of high-intensity exercise to aerobic exercise may decrease the risk of late hypoglycemic episodes occurring after training [21]. In the studies that demonstrated a positive effect of exercise on glycemic control the decrease in HbA1c was not associated with a significant increase in episodes of hypoglycemia. Decreased insulin requirement is commonly associated with increased physical activity and may be explained mainly by increased insulin-independent glucose

Table I. Continued

| Ref. | First author, publication date | Intervention or exposure | Study design | N (cases/controls) | Main outcome(s) |
|------|-------------------------------|--------------------------|--------------|-------------------|-----------------|
| [78] | Pilacinski, 2012              | Cigarette smoking        | OP           | 149               | Duration of partial remission, HbA1c |
| [79] | Haire-Joshu, 1994             | Cigarette smoking        | C-S          | 186 (83/103)      | Symptoms of depression |
| [81] | Sawicki, 1994                 | Cigarette smoking        | OP           | 93                | Progression of diabetic nephropathy |
| [82] | Muhihauzer, 1996              | Cigarette smoking        | OP           | 636               | Onset or progression of diabetic retinopathy or nephropathy |
| [83] | Mitchell, 1990                | Cigarette smoking        | Case-control | 163               | Prevalence of diabetic neuropathy |
| [86] | Lloyd, 1999                   | Stressful life events    | Case-control | 55                | HbA1c |
| [87] | Wiesli, 2005                  | Psychological stress – Trier Social Stress Test (TSST) | CT | 40 | Glycemia |
| [88] | Riazi, 2004                   | Daily stress             | OP           | 54                | Glycemia, HbA1c |
| [91] | Donga, 2010                   | Sleep restriction         | Self-controlled | 7                | Glucose disposal rate |
| [92] | Borel, 2009                   | Sleep duration            | C-S          | 20                | Blood pressure dipping status |
| [93] | van Dijk, 2011                | Sleep quality             | C-S          | 99                | HbA1c, symptoms of depression |
| [94] | Perfect, 2012                 | Sleep architecture        | CT (external controls) | 40/40 | HbA1c, glycermia, QOL |
| [95] | Secrest, 2011                 | Socioeconomic status      | OP           | 317               | Chronic complications of diabetes |
| [97] | Zgibor, 2000                  | General/specialist care, education level, income | OP | 429 | HbA1c |

Ref. – reference number, RCT – randomized controlled trial, CT – controlled trial (not randomized), OP – prospective observational study, C-S – cross-sectional study; ‘number of studies varied between analyses (studies with different types of exercises were analyzed separately)
Physical exercise increases insulin sensitivity in patients with T1DM [33], which may have an impact on the course of the disease. Insulin resistance is common in T1DM and associated with increased risk of its chronic complications [34]. In patients with diabetes physical activity may decrease oxidative stress [35] and reverse endothelial dysfunction [36, 37], and these effects may also contribute to the increased insulin sensitivity. Available evidence suggests the positive effect of physical activity on β-cell function in patients with T2DM [38] and overweight nondiabetic subjects [39], but it was not directly investigated in patients with T1DM. Physical activity also improves quality of life (QOL) and well-being in patients with T1DM, a condition generally associated with lower QOL and increased prevalence of depression [40].

Professional practice of competitive sports may have a different effect on health than leisure physical activity. Competitive sports are safe for people with T1DM if they have good metabolic control and are well educated about their disease and its interaction with physical exercise. However, aiming to improve performance and to achieve competitive success, some subjects may be involved in unhealthy practices that also have an adverse effect on metabolic control of diabetes [41]. These practices may include specific dietary patterns with excessive intake of single macronutrients, e.g. protein, use of nutritional supplements or illegal substances. Particular vigilance for early identification of disordered eating is needed in women with T1DM practicing esthetic disciplines and some endurance sports, including distance running or swimming, which are associated with increased risk of these disorders [41]. Some athletes with T1DM who practice sports with weight categories may omit insulin doses prior to weighing, which results in marked hyperglycemia, osmotic diuresis and rapid weight loss.

Nutrition

Nutrition seems to modulate the course of T1DM from its early preclinical stages. Absence or short duration of breastfeeding and early introduction of cow’s milk formulae are thought to be risk factors for the disease [42]. Also rapid weight gain in infancy, associated with improper feeding, increases the risk of developing T1DM [43]. Analyzing the influence of diet as a lifestyle component on the course of T1DM, several aspects of nutrition may be taken into account, including macro- and micronutrient content, daily meal regimen, effect of food on glycemia and other metabolic parameters, and adjustment of insulin treatment to the timing and content of meals.

According to the current treatment approach, the diet recommended to patients with T1DM in
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general does not differ from a healthy diet suggested for the general population. This enables individualized dietary advice that interferes little with a patient’s lifestyle. The restrictions on consistent day-to-day carbohydrate content of the meals might be an important consideration where premixed insulin or fixed basal-bolus treatment is used. In these treatment regimens day-to-day variations of carbohydrate content were associated with elevated HbA1c level [44]. Patients who adjust insulin doses to the carbohydrate content of the meals do not need such restrictions to achieve adequate control of glycemia [45]. The Dose Adjustment for Normal Eating (DAFNE) trial examined the value of flexible intensive insulin treatment with dietary freedom and carbohydrate counting, accompanied by a structured education program based on principles developed by a team from Dusseldorf [46]. In the intervention group of this trial, not only a significant decrease in HbA1c from Dusseldorf [46]. In the intervention group of the DAFNE trial did not increase the prevalence of hypoglycemia.

According to the above evidence, flexible meal pattern and content do not have a negative impact on the course and outcomes of T1DM in a healthy patient using the appropriate treatment regimen. However, some diet choices may have a negative effect on parameters of metabolic control of the disease and, as a consequence, may result in increased risk of complications. These improper dietary habits include especially frequent consumption of food and beverages with a high glycemic index (GI) or diet low in carbohydrates and rich in saturated fat. Despite the fact that products with a high glycemic index are not strictly forbidden for patients with T1DM, especially those using flexible intensive insulin treatment, a rapid rise of postprandial glycemia following these meals is difficult to control even using rapid acting insulin analogues. Patients with T1DM who often consume meals with a high GI have poorly controlled postprandial glycaemia, increased HbA1c value, and decreased serum HDL-cholesterol concentration [48]. Frequent intake of high-GI snacks, especially by children and adolescents, leads to obesity, dyslipidemia and poor glycemic control [49–51]. Interestingly, frequent consumption of diet beverages is associated with poor metabolic control of T1DM similarly to consumption of sweetened beverages, possibly being a marker of an unhealthy diet pattern [49]. An adverse effect of a high-GI diet on the atherogenic risk profile is a significant finding in patients with T1DM, as atherosclerotic cardiovascular diseases are a major cause of increased mortality in this group. On the other hand, patients must be advised not to increase the saturated fat intake when introducing the low-GI diet. This was one of the causes of increased fat intake in the DCCT cohort. In the intensively treated patients of this trial, saturated fat equaled nearly 13% of total caloric intake and diets higher in total and saturated fat and lower in carbohydrate were associated with worse glycemic control independent of exercise and body mass index (BMI) [52]. The other cause of increased fat intake in some patients with T1DM may be low-carbohydrate nutrition used in an effort to minimize the need for insulin injections or to lose weight. As a consequence, people with T1DM consume a diet with higher fat and saturated fat content than recommended and even higher than members of the general population [53, 54]. Meanwhile, carbohydrate intake may be partially substituted with sources of monounsaturated fat, which would reduce the glycemic index of the diet without an atherogenic effect [55].

Many young people with T1DM present a lifestyle associated with low attention to dietary choices and frequent consumption of fast food. Apart from other negative effects, this diet is usually associated with very high intake of food rich in trans-unsaturated fatty acids (from hydrogenated vegetable oils) and food additives, such as monosodium glutamate. Dietary trans fatty acids are associated with adverse cardiometabolic effects: increased low-grade inflammation, insulin resistance and accelerated atherosclerosis [56].

Consumption of sodium glutamate was associated with development of insulin resistance, T2DM and liver steatosis in animal models [57], but these effects were not investigated in humans. Consumption of a high purine diet or diet rich in fructose or sucrose contributes to hyperuricemia, which may play a pathogenic role in the metabolic syndrome and increase cardiovascular risk [58, 59]. Hyperuricemia may be associated with microvascular endothelial dysfunction in T1DM [60] and diabetic nephropathy [58].

Vitamins, including antioxidant E and C, and mineral supplements are not routinely recommended in T1DM [61] but their use among patients is very common [62]. The evidence on the supplementation of vitamin D in T1DM is also insufficient, despite its possible favorable effects on oxidative stress, insulin resistance and autoimmunity [63].

Eating disorders are more common in T1DM than in the nondiabetic population [64]. While they are not lifestyles but clinical entities, behaviors not satisfying diagnostic criteria of a particular eating disorder are also common in T1DM [65]. Disor-
Alcohol and illicit drugs intake

In patients with T1DM alcohol consumption may cause hypoglycemia mainly due to gluconeogenesis inhibition. Alcohol intake may also result in neglecting diabetes self-care – not measuring glycemia, omitting insulin doses and also involvement in risky behavior [68, 69]. These have an adverse influence on metabolic control and the course of disease, including glycemic instability and increased risk of DKA [70]. It is worth noting that moderate consumption of wine is not prohibited, and may have beneficial vascular effects in diabetes [71].

The data on illicit drug use among patients with T1DM are sparse, but the clinical observations confirm its deleterious effect on the course of the disease, severely compromising the patient’s self-care practices [72].

Smoking

Cigarette smoking adversely influences health, and the risk increases considerably when added to the risk conferred by diabetes. During 6-year observation of a T1DM cohort, Moy et al. [73] reported over 2.5 times increased risk of death in female smokers compared with nonsmokers, which was explained mainly by increased cardiovascular mortality. Hormonal responses to cigarette smoking, including growth hormone, cortisol and vasoressin, are counter-regulatory to insulin [74] and may lead to insulin resistance [75]. Nicotine may also directly inhibit insulin secretion [76]. Patients with T1DM who smoke were found to have higher HbA1c values than non-smoking subjects [77] and experience shorter partial remission [78]. Moreover, diabetic patients who smoke report less confidence in health care professionals, are more likely to report symptoms of depression, and may be less compliant to treatment [79]. Apart from accelerated atherosclerosis [80], smoking is associated with increased risk of microangiopathic complications in T1DM. This relationship is well documented for diabetic nephropathy and neuropathy, and very likely for retinopathy. Sawicki et al. [81] reported that over 1 year of observation, progression of nephropathy was less common in nonsmokers (11%) than in smokers (53%) and in past smokers (33%). In another study [82] during 6-year observation, the odds of progression of nephropathy increased by 27% for each 10 pack years. In a study by Mitchell et al. [83] patients smoking 30 pack years or more were 3.32 times more likely to have neuropathy than those smoking less than this amount.

Psychological stress

Stressful life events, such as losses within the family, were found to be associated with increased risk of development of T1DM in children, especially in the age group of 5–9 years [84], but also older ones [85]. These events occurred mainly during the second year preceding the diagnosis of T1DM and may be considered as possible triggering factors that precipitate or accelerate the autoimmune process. In patients with established T1DM, psychological stress may be associated with poor glycemic control [86]. The causality of this relationship is supported by a study in which acute psychosocial stress was associated with subsequent hyperglycemia [87]. Of note, marked individual differences occur in the blood glucose response to stress, and in many subjects the reactivity may be weak or undetectable [88].

Sleep duration

The influence of sleep duration on glycemic control of T1DM is unknown. However, partial sleep restriction decreased glucose tolerance in healthy subjects [89] and induced insulin resistance in both healthy subjects [90] and in patients with T1DM [91]. In patients with T1DM short sleep duration may also be associated with the blood pressure nondipping pattern [92]. Patients with a long history of T1DM have poor subjective sleep quality and are at increased risk for obstructive sleep apnea [93] compared with nondiabetic controls. Sleep-disordered breathing is associated with poor metabolic control and decreased quality of life [94].

Socioeconomic status

The association between socioeconomic status and the course of T1DM was analyzed in the Pittsburgh Epidemiology of Diabetes Complication Study. Lower education was associated with increased prevalence of end-stage renal disease and coronary artery disease, and lower income was associated with autonomic neuropathy and peripheral arterial disease [95]. Higher educational level was also found to be associated with lower mortality in T1DM [96]. These associations, although independent of sex and diabetes duration, may be partly mediated by poorer management of diabetes and presence of risk factors for chronic complications. Lower family income was associated with worse glycemic control in adult and adolescent patients with T1DM [97].
hand, higher socioeconomic status and higher degree of urbanization are associated with increased incidence and prevalence of T1DM [98].

Conclusions

Although lifestyle seems to modify the course of T1DM in many ways, the evidence on these relationships appears to be very incomplete. The largest body of evidence was accumulated on the influence of nutrition, physical activity and smoking on diabetes-related outcomes; however, many questions remain, including those already answered for T2DM. Other lifestyle parameters have rarely been analyzed in patients with T1DM.

This necessitates further research, especially including the assessment of the efficacy of lifestyle interventions on the course of T1DM.

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