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Impact of Stopping Smoking on Metabolic Parameters in Diabetes Mellitus: A Scoping Review

Stopping Smoking and Metabolic Parameters

Magdalena Walicka, Cristina Russo, Michael Baxter, Isaac John, Grazia Caci, Riccardo Polosa

Abstract

The purpose of this scoping review is to create a single narrative that describes the impact of smoking cessation on metabolic parameters in people with diabetes. It is generally well accepted that smoking enhances the harmful effects of elevated blood glucose levels accelerating the vascular damage seen in patients with diabetes. Smoking cessation has clear benefits in terms of reducing cardiovascular morbidity and mortality. However, there is less evidence for the impact of smoking cessation on other diabetes-related complications. Studies in people with diabetes have shown improvement as well as temporary deterioration in glycemic control after ceasing smoking. Only a few studies have described the effect of quitting smoking on insulin resistance and lipid parameters, however, their results have been inconclusive. In this situation, healthcare professionals should not assume that cessation of smoking will improve metabolic parameters in patients with diabetes. It seems they should, first of all, emphasize on the prevention of weight gain that may be associated with quitting smoking. The lack of data regarding the metabolic effects of smoking and smoking cessation in diabetes is very disappointing and this area needs to be addressed.

Key Words: Smoking; Smoking cessation; Diabetes; Insulin resistance; Glucose; Lipids

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Core Tip: Results of the studies regarding the impact of smoking cessation on metabolic parameters in patients with diabetes are inconsistent. Healthcare professionals should not assume that metabolic parameters in patients with diabetes who stop smoking will improve. It seems that the top priority after smoking cessation should be the prevention of weight gain. Further studies of the effects of quitting smoking on metabolic parameters among people with diabetes are required to provide an evidence base for

healthcare advice to managed patients and to assist healthcare providers to implement the most effective interventions.

INTRODUCTION

Approximately 1.3 billion people worldwide use tobacco, most commonly in the form of tobacco smoking, and more than 7 million people die every year as a result of smoking related conditions^[1,2]. Smoking is the main cause of lung cancer, chronic obstructive pulmonary disease, and cardiovascular disease^[3,4].

Exposure to cigarette smoke is associated with vascular damage, endothelial dysfunction, and activation of oxidative stress, inflammatory pathways, coagulation, and fibrinolysis^[5,6]. A similar mechanism of endothelial dysfunction is described for people with diabetes. It is therefore not surprising that smoking enhances the combined harmful effects of elevated blood glucose levels, accelerating vascular damage in diabetic patients who smoke^[7,8].

Smokers with diabetes (both type 1 and type 2) may be at a higher risk due to the direct effect of vascular damage as well as the indirect adverse effect that smoking has on glycemic control and lipid levels^[9].

The risk of cardiovascular events in diabetic patients is reduced with smoking cessation^[10]. In the ADVANCE study (Action in Diabetes and Vascular Disease: Preterax and Diamicron MR Controlled Evaluation), smoking cessation in those with diabetes was associated with a 30% reduction in all-cause mortality^[11]. A comprehensive evaluation of predicted coronary heart disease (CHD) among current and ex-smokers who had type 2 diabetes in Spain found that the ex-smokers had approximately 20% lower CHD risk at 10 years compared to current smokers^[12].

Although there is evidence that patients with diabetes can reduce the risk of macrovascular complications by giving up smoking, there is no conclusive evidence for the impact on the risk of microvascular complications^[9,13,14]. The impact of quitting smoking on microvascular complications of diabetes and its metabolic indices is

unclear. Furthermore, stopping smoking is known to cause weight gain which in turn may have unpredictable metabolic effect ¹ in patients with diabetes.

To the best of our knowledge, there have been no published systematic reviews to quantify the health benefits of smoking cessation in the diabetes population to date. The purpose of this scoping review is to create a single narrative describing the impact of smoking cessation in people with diabetes on glycemic control, insulin resistance and insulin secretion, and lipid abnormalities as well as biochemical parameters of nephropathy.

METHODS

The published literature on the impact of stopping smoking on metabolic indices, including glycemic control, insulin resistance, and lipid abnormalities was systematically reviewed in September and October 2021. The studies on biochemical parameters of nephropathy were also included. The literature search was conducted using the following databases: PubMed, Embase, ScienceDirect library, Database of Abstracts of Reviews of Effects (DARE), Scopus, and Google Scholar, using medical subject headings. Search queries were developed by a trained librarian experienced in developing search strategies for reviews and were based on diabetes, smoking cessation, fasting plasma glucose levels, HbA1C, insulin resistance, insulin secretion, lipids (total cholesterol, HDL, LDL, VLDL), microalbuminuria, creatinine. More specifically, search terms included ("smoking cessation" OR "former smokers" OR "ex-smokers" OR "stop smoking" OR "quitting") AND ("diabetes") AND ("glucose" OR "glycemi*" OR "HbA1C" OR "insulin resistance" OR "HOMA" OR "insulin secretion" OR "total cholesterol" OR "HDL" OR "LDL" OR "VLDL" OR "microalbuminuria" OR "albuminuria" OR "creatinine" OR "GFR"). Search results were filtered to include only human studies and published from 1980. The titles, abstracts, and full texts of the search results were sequentially and independently screened by MW and GC for inclusion. A few studies were identified, including cross-sectional, case-control, and cohort studies, randomized clinical trials, and observational

clinical studies, as well as systematic reviews and meta-analyses. The references of relevant studies were also manually reviewed for additional eligible citations.

RESULTS

Smoking cessation and incidence of diabetes

According to a meta-analysis conducted by Pan and colleagues, recent quitters are at higher risk for developing diabetes, although this risk progressively declines with time^[15].

It is often found that giving up smoking leads to a significant increase in weight^[16,17]. According to a large prospective UK study, smoking abstinence was associated with a gain of 8.79 kg on average at eight years, while continuing smokers gained only 2.24 kg^[17]. This has been confirmed in a meta-analysis showing that quitting smoking is associated with a bodyweight gain of 4-5 kg after 12 mo of abstinence, with most of the weight gain occurring between the third and the sixth month after quitting^[18]. As nicotine (in tobacco cigarettes) suppresses appetite and increases resting metabolic rate^[19], people who stop smoking gain weight because they have diminished resting energy expenditure and increased appetite. Moreover, quitters often substitute smoking by excessive eating/snacking, as shown in several studies of eating behaviors^[20].

It is likely that the weight gain associated with stopping smoking is responsible for the initial increase in risk. The increase in the risk of type 2 diabetes after quitting was directly proportional to weight gain, but not increased among quitters without weight gain^[21].

Smoking cessation and glycaemic control

Patients with diabetes can become more insulin resistant with worse glycemic control when they gain weight. Pani *et al*^[22], examining the predictors of diabetes progression (defined as HbA1C \geq 7% or the initiation of hypoglycemic therapy), found

that weight gain was an independent predictor: each extra pound of weight that is gained increases the risk of developing diabetes by 2%.

In patients with diabetes, quitting smoking may cause increased appetite, caloric intake, and weight gain, which would predictably lead to the worsening of glycemic control. In contrast, stopping smoking appears to have a beneficial effect on carbohydrate metabolism in the long run which may potentially mitigate the initial adverse metabolic effects of smoking cessation^[23].

Considering the complex interplay among factors that affect glycemic control, some uncertainty in the findings of studies that investigate the impact of stopping smoking on glycemic control might be anticipated. Studies comparing smokers to ex-smokers, both with type 1 and type 2 diabetes, demonstrated that active smoking is associated with worse glycemic control. In the study of Dinero *et al*^[24] current smokers (with an average smoking history of 30 years, an average daily habit of one pack of cigarettes per day) had higher mean HbA1c in comparison with former smokers. In the multiple linear regression analysis, current smoking was independently and significantly associated with higher HbA1c. Braffett *et al*^[14], using the data of a well-characterized cohort group with type 1 diabetes from the Diabetes Control and Complications Trial (DCCT 1983-1993), showed that in comparison to former smokers (subjects who previously smoked but quit > 3 mo prior to baseline), current smokers (subjects who currently smoked or quit < 3 mo prior to baseline) had higher mean HbA1c levels (average difference of 0.31%) over an average of 6.5 years of follow-up. The mean HbA1c levels for former smokers were similar to those of whom have never smoked. In relation to not only the current smoking status but also to its lifetime intensity and duration, the mean HbA1c levels were higher (average difference 0.22%) for current smokers with more than 10 pack-years in comparison to former smokers with less than 10 pack-years.

In an observational study of 10,692 adult smokers with type 2 diabetes, 29% of patients who had quit smoking and remained abstinent for at least 1 year revealed an increase in HbA1c of 0.21% with the need to intensify glucose-lowering treatment^[25]. In

further observation, HbA1c level decreased as abstinence continued, and became comparable to this in people who continued to smoke after a 3-year follow-up. Patients who stopped smoking gained weight (4.68 kg on average), but the results suggested that the change in weight was not directly related to the increase in HbA1c.

In Asiatic patients quitting smoking is generally associated with an improvement in glycemic control. In a study of 2,490 Japanese male patients with type 2 diabetes, HbA1c decreased linearly with the years after stopping smoking; however, there was no correlation with fasting plasma glucose (FPG)^[23]. Similarly, in a study of 7,763 Chinese men with type 2 diabetes, the HbA1c level decreased progressively with each year that the patients had stopped smoking; in this study, FPG levels decreased^[26]. In a smaller retrospective cohort study comprising 241 Taiwanese patients with type 2 diabetes, the group completing the smoking cessation program showed a significant decrease in FPG and HbA1c levels at 3-month follow-up compared to baseline. Due to the fact that the analyses of cardiometabolic factors were carried out before and after participation in the smoking cessation program in the whole group (regardless of the outcome of the smoking cessation program), it is difficult to interpret these results^[27].

In contrast, there are a number of studies on Asian patients failing to show improvement in glycemic control after stopping smoking. In a randomized controlled trial conducted in China, results of quitting smoking did not affect HbA1c levels at 1-year follow-up. The study included 557 smokers with type 2 diabetes^[28].

In the meta-analysis published by Kar *et al*^[29] there was no statistically significant difference in HbA1c between smokers and quitters. However, when the meta-analysis was reanalyzed by including studies comparing nonsmokers and active smokers, a statistically significant difference was demonstrated and this was positively associated with smoking duration; increasing as the years of smoking increased.

A summary of the studies evaluating smoking cessation's effect on HbA1c is shown in Table 1. The table also includes HbA1c data from studies of smokers with diabetic nephropathy.

Smoking cessation and insulin resistance and insulin secretion

The pathogenic mechanisms underlying type 2 diabetes are a balance between insulin resistance and beta-cell dysfunction. Smoking has been shown to influence both insulin resistance and insulin secretion. Studies on animals have shown that cigarette smoke can impair insulin production and secretion in addition to reducing beta-cell viability and proliferation^[30].

There has also been speculation that nicotine in tobacco smoke could play a significant role in promoting insulin resistance. Although chronic exposure to nicotine may be necessary to impact insulin sensitivity in nicotine naive subjects, acute exposure to nicotine can cause negative effects on insulin sensitivity in individuals with pre-existing insulin resistance^[31-33].

However, the direct effect of nicotine on insulin resistance is not supported in studies looking at the use of snus. Snus is an oral tobacco product that delivers significant levels of nicotine without producing any toxic combustion byproducts^[34]. Since the 1980s, snus consumption has been growing in popularity in Sweden, gradually replacing cigarette smoking^[35,36].

With the exception of one study, which has methodological issues including a flawed cross-sectional design and the lack of adjustment for smoking history in snus users^[37], there is clear evidence that snus use does not produce a significant rise in diabetes risk^[38-41]. Moreover, there was no association between snus use and insulin levels or glucose tolerance in a large study involving 1,266 subjects and primarily focused on cardiovascular risk factors^[42]. Insignificant relative risks for type 2 diabetes were reported in a meta-analysis for never-smoking current, former and ever-snus users^[43]. In addition, impaired glucose tolerance and related endpoints were not associated in any significant way.

It has been demonstrated that smokers have greater waist-to-hip circumference ratios^[44,45]. Waist-to-hip circumference ratio is one of the most pragmatic clinical measures of central obesity. One of the major contributing factors in obesity-related metabolic complications is fat distribution. The visceral abdominal depot (abdominal

obesity) is linked to metabolic dysfunction (cardiovascular disease, insulin resistance, type 2 diabetes). Conversely, lower body adiposity (gluteofemoral obesity) is associated with improved cardiovascular and metabolic profiles^[46]. The abdomen adipose tissue is characterized by the rapid uptake of diet-derived fat and a high lipid turnover that is easily stimulated by stress hormones^[46]. Increased release of free fatty acids and abnormalities in adipokine secretion observed in people with abdominal obesity promote insulin resistance^[47].

Compared with nonsmokers, smokers are characterized by greater insulin resistance and hyperinsulinemia^[48]. However, little research has been conducted on the impact of smoking cessation on insulin resistance and insulin secretion.

Smoking cessation may be associated with worsening fat distribution. In a population-based study (Inter99 Study) performed in Copenhagen, the mean increase in waist circumference after quitting at the one-year follow-up was 3.88 cm (42% of the quitters had increased their waist circumference by ≥ 5 cm). Quitters with high baseline tobacco consumption were more likely to have substantially increased waist circumference. In this study, abstinence from smoking was the most important predictor of substantial weight gain and a substantial increase in waist circumference^[49]. Likewise, a study with the use of computed tomography showed that both current and former smoking is associated with increased visceral adipose tissue^[50]. In a population-based study performed in Norway, former smokers compared with current smokers had a lower waist to hip ratio (additionally among women, waist circumference was lower)^[51].

However, studies assessing insulin resistance indicators in quitters do not have consistent results. There was a statistically significant negative correlation between HOMA-IR (the homeostasis model assessment-estimated insulin resistance) values among asymptomatic, Korean male ex-smokers without diabetes^[52]. In contrast, other studies (also in the groups without diabetes) showed that quitting smoking was associated with greater insulin resistance as measured by Quicki or HOMA-IR^[53,54].

The observed discordance amongst the insulin sensitivity findings is likely to be due to a change in body weight. After stopping smoking, insulin sensitivity is likely to change because of weight fluctuations. It was shown that the HOMA-IR index after quitting significantly increases in weight gainers, but not in weight maintainers^[55]. In the study by Heggen *et al*^[56], no differences were found in HOMA-IR between quitters and smokers but the findings must be interpreted within the context of similar modest body weight changes in quitters and smokers at 3-month follow-up.

These studies have most commonly included people without diabetes. The only study investigating the relationship between insulin resistance and smoking cessation among patients with diabetes is that of Ohkuma T *et al*^[23]. The authors found that smoking cessation has a time-dependent link with insulin resistance in Japanese patients with type 2 diabetes; HOMA-IR levels decreased in ex-smokers over time relative to current smokers. HOMA-IR was also assessed in the prospective study, evaluating the effect of smoking on the progression of microalbuminuria in type 2 diabetes. Smoking cessation was associated with the amelioration of insulin resistance parameters in spite of the small but significant increase in BMI. This observation may be explained by the fact that many quitters increased their physical activity^[57].

A summary of the studies evaluating smoking cessation's effect on HOMA-IR is shown in Table 2.

The search for publications on quitting and insulin secretion in patients with diabetes was unproductive. In the population without diabetes, Morimoto *et al*^[58] found that the risk of impaired insulin secretion in an ex-smoker is similar to that in never-smokers, where the risk is almost twice as high in current smokers when compared with never smokers, with the magnitude of this increase being dose-dependent (i.e. increasing with a number of pack-years). Stadler *et al*^[54] showed a 31% increase in beta-cell secretion (as measured by insulinogenic index 140) after > 3 mo of not smoking.

Smoking cessation and lipids abnormalities

Patients with type 2 diabetes characteristically have abnormal plasma lipids profiles which are marked by hypertriglyceridemia, reduced high-density lipoprotein (HDL) cholesterol levels, and increased concentration of small dense low-density lipoprotein (LDL). These abnormalities are a result of a multifactorial process, including abdominal obesity, insulin resistance, increased free fatty acid flux, and inflammation^[59]. Cigarette smoke has been shown to increase the atherogenic nature of the lipid profile^[60]. Smoking is associated with increased triglycerides (TG), total cholesterol, and LDL, as well as reduced levels of cardioprotective HDL^[61]. In a prospective study of 808 young Asian adults, smokers were three times more likely to have low HDL cholesterol and were 2.6 times more likely to develop hypertriglyceridemia^[62]. There is a clear assumption in healthcare messaging that stopping smoking may correct dyslipidemia, which is especially relevant in smokers with diabetes. Studies, conducted on patients without diabetes, indicate that quitting smoking increases HDL levels^[63,64]. The increase in HDL has frequently been observed in spite of weight gain experienced after cessation of smoking^[63]. Evidence also indicates that smokers may have improved HDL function (increased cholesterol efflux capacity and decreased HDL inflammatory index) after quitting smoking^[65].

Data on TG levels are conflicting. Some studies performed in the group without diabetes showed that smoking cessation is associated with a reduction of this lipid fraction^[66], however, others studies have failed to confirm this^[64].

Data on LDL is also limited, but evidence seems to suggest that smoking cessation does not affect LDL levels or LDL size^[63,67].

A few studies have tested diabetic patients' lipid profiles after quitting smoking. Results are inconsistent. In Reynolds *et al*^[68], 3466 youth who had type 1 diabetes ($n = 2887$) or type 2 diabetes ($n = 579$) and were smokers were examined for prevalence of tobacco use and the coexistence of cardiovascular risk factors. Compared to patients who were non-smokers, past smokers with type 1 diabetes had significantly higher odds of having high LDL cholesterol levels, and those who were current smokers had significantly higher chances of having high TG levels. Patients with type 2 diabetes did

not exhibit these relationships, but the smaller numbers of patients included in the study could have influenced the statistical significance of the results.

In the study of Luque-Ramirez *et al*^[12] patients with type 2 diabetes who smoke had lower HDL and higher TG levels compared to their nonsmoking counterparts.

Lipid parameters were examined in two studies in patients with diabetic nephropathy. After stopping smoking for at least 1 year, patients had significantly lower total cholesterol, low-density lipoprotein (LDL), and high-density lipoprotein (HDL) levels than those who continued to smoke^[57]. In a similar study of patients with type 1 diabetes, total cholesterol, TG, and LDL levels of current and former smokers were higher than those of non-smokers, whereas lower HDL levels were observed in current smokers^[69].

A summary of the studies evaluating smoking cessation's effect on lipid parameters is shown in Table 3.

Smoking cessation and biochemical parameters of nephropathy

9 It is well known that chronic kidney disease (CKD) and end-stage renal disease (ESRD) can complicate diabetes mellitus. Diabetic nephropathy is characterized by proteinuria and/or the decline of renal function (e.g. reduced glomerular filtration rate - GFR)^[70]. Aside from high blood sugar levels, other risk factors that contribute to the development and progression of diabetic kidney disease include high blood pressure, dyslipidemia, and genetic predisposition^[71]. Smoking may also be a factor in the development and progression of kidney failure possibly through a mechanism of progressive arteriolar damage, increased renovascular resistance, and increased intraglomerular capillary pressure^[72-75]. While many studies have examined the relationship between cigarette smoking and kidney disease with conflicting results, a meta-analysis of 15 prospective cohort studies with 65,064 incident cases of CKD suggests that smoking is as an independent risk factor in the general population^[76].

There is no clear impact of stopping smoking on characteristics associated with diabetic nephropathy. The effect of smoking cessation on microalbuminuria was

investigated by Voulgari *et al*^[57] in subjects with newly diagnosed type 2 diabetes mellitus. Within a year of stopping smoking, the prevalence of those with microalbuminuria markedly declined compared to those who continued smoking. However, eGFR was comparable between the two study groups. Smokers who stopped smoking had a lower microalbuminuria rate irrespective of the effect of drug therapy (antihypertensive, hypolipidemic, and antidiabetic).

In a study of 2,770 patients with type 2 diabetes, Ohkuma *et al*^[77] investigated the association of smoking and its abstinence with parameters of CKD. In comparison to non-smokers, former and current smokers had higher urinary albumin-creatinine ratios (UACR). In the former smokers, this ratio decreases linearly with increasing years after quitting smoking. Furthermore, current smokers' risk is related to how many cigarettes per day they smoke. After quitting smoking, age-adjusted creatinine-based GFR declined compared to the never-smokers but increased in parallel with increasing cigarette consumption. The increased eGFR of smokers may be related to glomerular hyperfiltration which is implicated as a mechanism for the progression of diabetic nephropathy^[78].

Progressive kidney damage can result from glomerular hyperfiltration over time. According to Ohkuma's study, the proportion of smokers with CKD increased with the number of cigarettes they smoked per day (compared with never-smokers). However, as the years passed since quitting, the proportion of patients with CKD decreased. There was a significant increased HbA1c level for current smokers and a greater proportion of hypertension for ex-smokers compared with never smokers and current smokers with respect to the other risk factors for nephropathy in this study^[77].

Using data from the Finnish Diabetic Nephropathy Study, which included 3613 type 1 diabetes patients, the 12-year cumulative risk of microalbuminuria, macroalbuminuria, and end-stage renal disease by smoking status was calculated. Current and former smokers were more likely to have micro- and macro-albuminuria (ESRD for current smokers only) than non-smokers. There were no statistically significant differences in the 12-year cumulative risk of microalbuminuria and

macroalbuminuria between former smokers and never smokers. There were significantly poorer glycemic control and lipid parameters for smokers compared to nonsmokers. Adjusting for HbA1c and lipid variables, the increased risk of diabetic nephropathy progression among current and former smokers was attenuated. Smoking-related changes in lipids and glucose control may account for the majority of nephropathic changes due to diabetes^[69]. This observation suggests that poor glucose control and lipid alterations in smokers are the main drivers of nephropathic changes in diabetes.

A summary of the studies evaluating smoking cessation's effect on characteristics associated with diabetic nephropathy is shown in Table 4

CONCLUSION

In addition to reducing overall and cardiovascular mortality, stopping smoking may provide significant additional health benefits to people with diabetes. It is important to note, however, that weight gain experienced after stopping smoking may attenuate some of these health benefits^[79].

When considering the potential impact of stopping smoking on metabolic parameters in patients with diabetes, the benefits of cessation of smoking are less clear because the expected outcomes have not been consistently demonstrated. Studies have shown both improvements and temporary deterioration in glycemic control after quitting smoking. Only a few available studies have investigated the effect of quitting smoking on insulin resistance and lipid parameters in diabetic patients. These studies also report inconsistent results. Smoking cessation appears to have a clear beneficial effect on markers of nephropathy, particularly after longer periods of smoking abstinence.

The review of the published literature found only a few studies, many of which had design and methodological shortcomings, and - as such - need to be interpreted with caution. A major issue for this area of study is the lack of randomized controlled trials that have been carried out to date.

In an era of evidence-based medicine, the lack of data regarding the metabolic effects of smoking and smoking cessation in diabetes is very disappointing and needs to be addressed. Diabetes is one of the major population health issues, the consequence of which appears to be amplified by smoking. The lack of good quality research on the impact of smoking cessation on metabolic parameters in this population hampers clinicians' ability to give informed advice on the effectiveness and management of stopping smoking. This is a complex medical and sociological issue that demands a greater research focus to better inform people with diabetes and assist healthcare providers to implement the most effective interventions.

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The authors' thoughts and prayers are with all the victims of the Russian - Ukrainian conflict

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