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Current Smoking: An Independent Predictor of Elevated A1C in Persons with Type 2 Diabetes

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Abstract

Purpose: The purpose of this study is to examine the association of current smoking as one of several potential predictors of elevated A1C in adults with type 2 diabetes (T2D).

Methods: Using a cross-sectional design, baseline data ($N=282$) were analyzed from a randomized clinical trial examining treatment of obstructive sleep apnea in persons with T2D. Socio-demographic, clinical, and behavioral data were collected using questionnaires and physical examinations. Physical activity (mean daily steps walked) was measured with the BodyMedia Armband®. Participants were asked if they never smoked, had previously smoked, or currently smoke. The sample distributions of demographic and clinical characteristics were examined using descriptive statistics. Continuous variables were described using means and standard deviations; categorical variables were described as numbers and percentages. Multiple linear regression analysis with backward-selection was conducted to develop a parsimonious predictive model for the dependent variable A1C.

Results: Participants were generally middle-aged and, on average obese with sub-optimal blood glucose control; almost one out every five participants currently smoked. After controlling for age, race, education, financial difficulty, diabetes education, physical activity, and diabetes knowledge, four variables were found in the final model to be independently associated with higher A1C: (1) current smoking status, (2) younger age, (3) longer diabetes duration, and (4) higher diabetes-related distress.

Conclusions.—The study found that not only is smoking prevalent among persons with T2D with self-reported sleep problems but smoking is also an independent predictor of elevated A1C. The results highlight the vital role diabetes educators have in promoting risk reduction through education and support for smoking cessation.

The rate of smoking in the United States has declined over the last 10 years from 20.9% in 2005 to 15.5% in 2017, yet the prevalence of smoking among persons with diabetes (15.9%) remains higher than the general US population.¹ There are racial, socio-economic and geographic health disparities among smokers,^{2,3} and smokers who have type 2 diabetes (T2D) are at higher risk for debilitating complications including renal failure, retinopathy, peripheral vascular disease, peripheral neuropathy, and cardiovascular disease when compared to those who do not smoke.⁴

Nicotine, the active ingredient in cigarettes, increases insulin resistance, oxidative stress, and inflammation that can lead to microvascular and macrovascular complications.^{5–7} The mechanism responsible for the increase in blood glucose was first examined in early animal models in the 1930's.⁸ These early studies linked nicotine to increased blood glucose from nicotine-induced stimulation of adrenal catecholamines.⁸ Tsujimoto and colleagues⁹ replicated these findings in an experimental animal study that showed blood glucose levels increased significantly within five minutes of an injection of nicotine (100 µg/kg). The duration and degree of the increase in blood glucose level was dependent upon the dose of nicotine injected. Following surgical adrenalectomy, there was no hyperglycemic response to nicotine injection, prompting the investigators to conclude that nicotine-stimulated adrenaline release was responsible for the increase in blood glucose. A limitation of this study is that the potential effects of glucocorticoids (e.g. cortisol) were not addressed.

A similar effect of nicotine on blood glucose was serendipitously discovered in humans in a 1934 metabolic study that examined optimal meal intervals.⁸ These researchers observed an abrupt rise in blood glucose 15 minutes after smoking that corresponded with an increase in respiratory quotient (the ratio of the volume of carbon dioxide to that of oxygen). The overall body of research regarding glycemic effects of cigarette smoking, however, is inconclusive. A temporary increase of blood glucose occurring within 30 minutes of cigarette smoking was reported in persons with and without diabetes¹⁰ but other research reported no effect.^{11,12} There has been little further experimental inquiry in this area for almost forty years.

Although it is known that cigarette smoking increases the risk of T2D, and persons who smoke more cigarettes are at greater risk,¹³ the effect of smoking on blood glucose in persons with T2D is not well established. The purpose of this investigation was to determine the contribution of current smoking as one of several potential predictors of A1C in a sample of adults with T2D.

Methods

The primary specific aim of this study was to determine if smoking is a potential predictor of elevated A1C in adults with T2D. The study also aimed to identify covariates and other potential predictors of A1C, and to describe the prevalence and characteristics of current smokers vs. former smokers and never-smokers. Using a cross-sectional design this secondary analysis used baseline data from participants ($N=282$) in an ongoing randomized sham-controlled trial that examines the effect of the treatment of obstructive sleep apnea on glucose control and diabetes self-management.

Sample

The sample was recruited for the parent study (R01-DK096028), the Diabetes Sleep Treatment Trial, with advertisements for persons with “poor sleep quality” or who were told if they snored or had symptoms of sleep apnea. Preliminary eligibility for the baseline assessment was completed with a telephone screening script with the inclusion criteria of self-identification of a diagnosis of type 2 diabetes, ability to walk, no history of a car crash or near accident due to sleepiness, naïve to CPAP treatment (self and household members), and potential willingness to be randomized to either continuous positive airway pressure (CPAP) or a sham-CPAP device if they were found eligible to continue in the parent study.¹⁴

Measures

Smoking status (never smoked/former smoker/current smoker) was determined by questions that asked, “have you ever smoked cigarettes” (yes/no) and “are you currently smoking cigarettes” (yes/no). Another question queried if the person had “smoked even one puff of a cigarette in the last 7 days”; if positive these persons were categorized as a “current smoker”.

A1C was used to determine the 3-month average glucose level. Venipunctures were performed in the Clinical Translational Research Center (CTRC) at the University of Pittsburgh Medical Center (UPMC). Specimens were analyzed in the clinical UPMC laboratory with ion-exchange (HPLC) chromatography.

Body mass index (BMI, kg/m²) was calculated from a measured height and weight at the CTRC.

Diabetes-related distress was determined from the Problem Areas in Diabetes (PAID)¹⁵ questionnaire. The PAID includes 20 questions on a 5-point Likert scale with higher scores indicating higher emotional distress over living with a diagnosis of diabetes. It is a reliable and valid instrument with strong internal consistency (Cronbach’s alpha = .90).

Diabetes knowledge was evaluated with the Diabetes Knowledge Test (DKT).¹⁶ The DKT is scored as the percentage correct with separate scoring for persons who are prescribed insulin. The DKT was evaluated in two separate populations with participants with type 1 diabetes, T2D using insulin, and T2D without insulin. It is a reliable instrument with internal consistency (Cronbach’s alpha = .70 for the general test and the insulin-use subscale. Scores were higher in persons by educational level and in persons who received diabetes education.

Physical activity was assessed continuously with the BodyMedia SenseWare Armband® for 7 days. Days with greater than 20 hours of wear time were averaged to obtain the mean daily steps walked.

Demographic information evaluated included age, sex, race, education, perceived financial difficulty, and duration of diagnosed diabetes. Race was dichotomized as “Caucasian” or “Non-Caucasian” and educational level as either less than a 2-year associate’s degree or as having obtained a 2-year associate’s degree or higher. Participants were asked “how difficult is it to meet your basic needs (i.e., food, housing utilities, health care”) with possible responses of “not at all difficult”, “somewhat difficult”, and “extremely difficult”. Financial

difficulty was dichotomized as either “none” or “some-to-extreme”. Diabetes duration was dichotomized as less than or equal/greater than 10 years.

Statistical analysis

In this study, IBM® SPSS® Statistics version 25.0 Windows (IBM Corp., Armonk, NY) was used for data analysis. The level of statistical significance was set at 0.05, unless otherwise noted. The sample distributions of demographic and clinical characteristics were examined using standard descriptive statistics with continuous variables being summarized using means and standard deviations, and categorical variables being described using frequencies and percentages. Comparisons of variables between smoking groups (never smoked and former smoker [i.e., never/former] vs. current smoker) were analyzed using two-sample *t*-tests or Mann-Whitney *U*-tests for continuous variables or chi-square test of independence or Fisher’s exact test for categorical variables. Given the scaling of variables and whether the normality assumption was met, bivariate analyses were performed (e.g., Pearson’s versus Spearman’s correlation) to determine associations between the main outcome (A1C) and clinically important baseline factors selected *a priori*.

A multiple linear regression analysis with backward-selection was conducted to develop a parsimonious predictive model for the dependent variable A1C. Predictor variables found to have *P*-values <.20 on bivariate analyses were included in the multivariate regression model. The criterion for the removal of the candidate predictor variables from the multivariate model was set at *P*<.10. Data screening was conducted to ensure no violations of the assumptions of independence, normality, linearity, homoscedasticity, no multicollinearity, and no additivity.

Results

Table 1 presents the characteristics of the total sample and a comparison of persons who “never” smoked with persons who indicated they were “former” smokers. The current study included 282 participants with T2D (mean $\pm$ SD age 56.6 $\pm$ 10.7 years; 50.4% female; 40.1% Non-Caucasian; 44.3% < 2-year associate’s degree) who were on average obese (mean BMI of 35.1 $\pm$ 6.9 kg/m²), with poor glucose control (mean A1C of 7.9 $\pm$ 1.8 %), and almost half (42.6%) had some to extreme financial difficulty. Almost one in five participants (18.8%, *n*=53) reported they currently smoked, 50% of the participants (*n*=141) reported they never smoked and 31.2% (*n*=88) were former smokers. Participants who currently smoke had an average smoking history of 30 years. Former smokers had an average smoking history of 20 years and 82% quit >1 year ago. Both the participants who formerly smoked and those who currently smoke reported an average daily habit of one pack of cigarettes per day. Former smokers were similar to those who never smoked in terms of age, sex, race, levels of financial difficulty, diabetes duration, activity level, diabetes knowledge, diabetes-related distress, BMI and A1C (all *P*-values .05). Those that never smoked had significantly higher general education (*P*=.03). Hence, persons who never smoked and former smokers were combined to form a non-smoker group.

Table 2 presents data from a comparison of “nonsmokers” (i.e., participants who either never smoked or are now former smokers) with participants who currently smoke. Compared to

nonsmokers, current smokers were younger (52.6 ± 8.6 years vs. 57.5 ± 11.0 years) with a higher percentage of non-Caucasians (75.5% vs. 31.9%), persons with less than a 2-year associate's degree (67.9% vs. 38.9%), and financial difficulty (66.0% vs. 37.1%) (all P -values $< .05$). With regards to diabetes-related measures, the current smoker group had a higher proportion of persons with diabetes duration of < 10 years (66.0% vs. 47.6%, $P=.016$), a higher mean A1C (8.6 ± 2.1 vs. 7.8 ± 1.7 , $P=.01$), higher diabetes-related distress (31.0 ± 18.4 vs. 22.6 ± 16.6 , $P=.003$), and lower diabetes knowledge (70.0% vs. 75.0%, $P=.032$) compared to the non-smoker group.

Bivariate analyses identified age, race, education level, financial difficulty, diabetes duration, current smoker, diabetes-related distress, and mean daily number of steps as being associated with A1C at $P<.20$ and as candidate predictors for multivariate regression analysis. Table 3 depicts results from a multiple linear regression that was performed to predict A1C from all the candidate predictors identified in the bivariate analyses. The total variance in A1C explained by the final parsimonious model was 17.5% ($F(5,253) = 10.77$, $P<.001$). After controlling for physical activity, race, education, and financial difficulty, four predictor variables were identified that were independently significantly associated with higher A1C: (1) longer diabetes duration ($b = -0.26$, $P<.001$), (2) younger age ($b = -0.20$, $P=.002$), (3) higher diabetes-related distress ($b = 0.20$, $P=.001$), (4) current smoking ($b = 0.13$, $P=.03$).

Discussion

A secondary analysis of baseline data was conducted to assess potential predictors of elevated A1C including smoking among persons with T2D and sleep disturbance. The conclusion was that smoking is associated with higher A1C in persons with T2D independent of body weight or activity level. Although longer duration of diabetes was associated with higher A1C consistent with previous research¹⁷ there was no difference in A1C between participants who never-smoked and participants who were former smokers. This finding could provide an incentive to quit for smokers with T2D who struggle to reach and maintain A1C goals.

Despite clear and overwhelming evidence that smoking is associated with diabetes-related vascular complications (in addition to cancer and chronic lung disease), the question arises as to why smoking risk reduction has lost momentum in persons with diabetes.¹⁸ For example, 19% of the sample currently smoked which is higher than national rates for persons with T2D (15.9%) and the general US population (15.5%). Previous research has linked smoking to racial and socio-economic disparities.² A higher smoking rate found in the current study may be related to the demographics of the sample, since 43% (76% of the smoking group) was non-Caucasian and 43% (66% of the smoking group) reported financial difficulty, a possible indicator of lower socioeconomic status. Also, consistent with the literature, current smokers in this study had more diabetes-related distress than non-smokers.¹⁹ Smoking may serve as a means of coping²⁰ or a substitute for pleasure eating and other cravings.^{21,22} It is possible that some persons with T2D continue to smoke for these reasons, and emphasizes the importance of considering the psycho-behavioral context of smoking when assessing readiness to quit.

Post-Quit Weight Gain

Weight gain is a common concern after smoking cessation and can deter quit attempts.²³ This study found that smoking was a predictor of elevated A1C independent of body weight in persons with T2D. A cross-sectional study from Great Britain found a slight (.2%) increase in A1C during the first year after quitting in persons with T2D unrelated to weight gain.²⁴ The clinical significance of a temporary uptick in post-quit blood glucose in persons with T2D remains unclear without evidence from a randomized controlled trial, but it is notable that two meta-analyses found post-quit weight gain does not interfere with the benefits of smoking cessation (e.g. reduced cardiovascular and all-cause mortality) in persons with diabetes.^{25–27}

Implications for Diabetes Educators

Smoking risk reduction for persons with diabetes is complex. Lifestyle, behavioral, and socio-economic factors particularly in vulnerable populations and those with health disparities, deserve attention. The findings of this study can be applied to the practice of diabetes education through renewed and systematic efforts to encourage abstinence in high risk groups, identify tobacco risk behaviors, and promote quitting strategies.

The American Diabetes Association guidelines for smoking risk reduction for persons with diabetes is three-fold¹⁸:

- 1) Elicit a smoking/tobacco history at initial and follow-up diabetes visits
- 2) Discourage smoking use in youth who do not smoke, and
- 3) Encourage smoking cessation in those who do smoke.

These measures should be applied to all nicotine-containing products, since nicotine is an addictive substance that drives metabolic disregulation,^{7,28,29} and though Nicotine Replacement Therapy (NRT) is a mainstay of smoking cessation strategies, long-term use of NRT is therefore not recommended. E-cigarettes may contain nicotine and have become a popular smoking-alternative especially among teens and young adults.³⁰ One study found a 28% lower quit rate among smokers who used e-cigarettes as a quitting aide.³¹ However, there exists insufficient comparative effectiveness research with long-term data to adequately compare e-cigarettes with conventional types of NRT (patches, gum, lozenges).

There are other distinct considerations regarding smoking that are relevant to diabetes educators. Smoking rates have been found to be higher among youths who have diabetes compared to those that don't,³² highlighting a need for vigilant nicotine use-assessment and encouragement of abstinence from nicotine containing products including e-cigarettes for young persons with diabetes. Ongoing smoking assessment is also important for persons who use inhaled insulin since inhaled insulin is contraindicated in current or recent smokers.³³ Smoking assessment is also an important consideration for persons who undergo metabolic surgery because of the risk for increased substance use including nicotine in this population.³⁴

Smoking Cessation Strategies

Successful smoking cessation generally requires a combination approach rather than a single therapy. There is no evidence to confirm that reducing cigarette consumption without quitting improves health other than by possibly making it easier to eventually quit.³⁵ According to the American Lung Association, only a small percentage of people can quit “cold turkey” without any type of quitting aide.³⁶ The most practical and effective combination approach has consisted of brief counseling with telephone follow-up combined with short-term, tapered NRT.³⁷ Other effective smoking cessation therapies include pharmacotherapy such as bupropion and varenicline,³⁸ as well as use of a “buddy” accountability system,³⁹ and financial reimbursement incentives.⁴⁰ The Ottawa Model for Smoking Cessation (OMSC) is an example of a successful multicomponent approach to smoking cessation that combines counseling, a discount card toward the cost of NRT, and follow up phone calls over 6 months. A multi-site study with 313 smokers with diabetes or pre-diabetes who were randomized to either the OMSC program or a wait-control group found almost a 4 times greater abstinence at 6-months in the treatment group compared to wait-list controls.⁴¹ There is also evidence that Motivational Interviewing,⁴² a technique used in diabetes education,⁴³ and Mindfulness Training for Smoking Cessation (MTS) can effectively promote quitting,^{44,45} particularly in lower socio-economic populations.⁴⁶

Limitations and Future Directions

Secondary analyses limit the choice of instruments to those used in the parent study. For example, the Diabetes Distress Scale targets provider distress and diabetes self-management-related issues,^{47,48} and would also have been an excellent choice for the current study. However, use of the PAID was necessitated by the parent study’s protocol.¹⁴ Both the DDS and the PAID are highly valid and reliable instruments.^{15,47} Relevant to the current study, the PAID is particularly useful in measuring diabetes-related emotional issues, food-related concerns and fear of complications.⁴⁸

This study is also limited by the lack of an objective measure of smoking status with urine cotinine or breath carbon monoxide. In addition, cigarette dose effect and use of other forms of nicotine such as chewing and e-cigarettes were not assessed. Lastly, since all participants had some type of sleep disturbance, these results may not apply to other samples of persons with T2D. However, these results do provide evidence that A1C is significantly higher in persons who report current smoking, and may prompt renewed efforts to target this modifiable risk factor. Qualitative exploration of barriers to and facilitators of smoking cessation in persons with diabetes is warranted and may inform new behavioral strategies for diabetes educators.

Conclusion

Our findings suggest that smoking is prevalent and an independent predictor of elevated A1C in persons with T2D. Diabetes educators have a vital role in promoting risk reduction through education and support for smoking/tobacco/nicotine cessation. These findings may help reintroduce the conversation that smoking cessation is an important target for maintaining blood glucose goals and preventing complications. Information regarding the

link between current smoking and higher A1C may provide positive reinforcement for continued abstinence and serve as a tangible incentive for renewed quitting efforts for persons who smoke.

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Table 1.

Characteristics of the Total Sample ($N=282$) and Comparison of Participants Who Never Smoked ($n=229$) with Participants Who Formerly Smoked ($n=53$)

	Total Sample	Never-Smoked	Formerly Smoked	<i>P</i> -value
Age (years)	56.6±10.7	56.4 ± 11.2	59.2 ± 10.5	0.06
Sex				
Male	140 (49.6)	72 (51.1)	47 (53.4)	0.78
Female	142 (50.4)	69 (48.9)	41 (46.6)	
Race				
Caucasian	169 (59.9)	99 (70.2)	57 (64.8)	0.47
Non-Caucasian	113 (40.1)	42 (29.8)	31 (35.2)	
Education				
< 2-year Associate's Degree	125 (44.3)	47 (33.3)	42 (47.7)	0.04
2-year Associate's Degree	157 (55.7)	94 (66.7)	46 (52.3)	
Financial Difficulty				
None	162 (57.4)	93 (66.0)	51 (58.0)	0.26
Some-to-Extreme	120 (42.6)	48 (34.0)	37 (42.0)	
Diabetes Duration				
<10 years	144 (51.1)	70 (49.6)	39 (44.3)	0.50
10 years	138 (48.9)	71 (50.4)	49 (55.7)	
A1c (%)	7.9 ± 1.8	7.9 ± 1.8	7.6 ± 1.6	0.33
BMI (kg/m ²)	35.1 ± 6.9	35.9 ± 7.3	34.9 ± 6.5	0.52
Physical Activity (mean daily steps)	4799 ± 3158	4680 ± 2805	4411 ± 3580	0.12
Diabetes Knowledge (%)	73.7 ± 15.5	76.5 ± 14.1	71.7 ± 17.0	0.05
Diabetes Distress	24.1 ± 17.2	22.0 ± 16.1	23.4 ± 17.5	0.54

Note: Data presented as mean ± SD or *n* (%).

Table 2:

Comparison of Non-Smokers (Participants who never smoked and former smokers) with Current Smokers

Predictor		Non-Smokers 229 (81.2%)	Current Smoker 53 (18.8%)	P-value
Age (years)		57.5±11.0	52.6±8.6	.003
Sex				
	Male	119 (52.0)	21 (39.6)	.150
	Female	110 (48.0)	32 (60.4)	
Race				
	Caucasian	156 (68.1)	13 (24.5)	<.001
	Non-Caucasian	73 (31.9)	40 (75.5)	
Education				
	< 2-year Degree	89 (38.9)	36 (67.9)	<.001
	2-year Degree	140 (61.1)	17 (32.1)	
Financial Difficulty				
	None	144 (62.9)	18 (34.0)	<.001
	Some-to-Extreme	85 (37.1)	35 (66.0)	
Diabetes Duration				
	<10 years	109 (47.6)	35 (66.0)	.016
	10 years	120 (52.4)	18 (34.0)	
A1C (%)		7.8 ± 1.7	8.6 ± 2.1	.010
BMI (kg/m ²)		35.5 ± 7.0	33.5 ± 6.6	.055
Physical Activity (mean daily steps)		4578 ± 3116	5784 ± 2184	.005
Diabetes Knowledge (%)		74.6 ± 15.4	69.9 ± 15.7	.032
Diabetes Distress		22.6 ± 16.6	31.0 ± 18.4	.003

Note: Data presented as mean ± SD or *n* (%).

Table 3:

Multiple Linear Regression Analysis Summary to Predict Elevated A1C Levels Controlling for Activity Level, Race, Education, and Financial Difficulty

Final Model*	b	SE(b)	Beta	P-value
Age	−0.03	0.01	−0.20	.002
Diabetes Duration	−0.94	0.23	−0.26	<.001
PAID	0.02	0.01	0.20	.001
Steps	0.00	0.00	0.11	0.08
Smoking Status	0.64	0.28	0.13	0.03

Note: The dependent variable was A1C; $P < .001$; $F(5,253) = 10.77$; age = continuous variable; diabetes duration = dichotomous variable, 0 = 10 years or 1 = <10 years; PAID = diabetes distress, continuous variable; steps = number of steps per day, continuous variable; smoking status = dichotomous variable, 0 = never/former smoker or 1 = current smoker.

* Backward-selection method was used; variables with $P > .10$ removed from models

Model 1: age, education, race, financial difficulty, diabetes duration, PAID, steps, and smoking status.

Model 2: Model 1 (-) financial difficulty.

Model 3: Model 2 (-) education.

Final Model: Model 3 (-) race. (Final Model includes age, diabetes duration, PAID, steps, and smoking status)