Archives of Surgery and Clinical Case Reports
Alshararni A. Arch Surg Clin Case Rep 5: 169.
www.doi.org/10.29011/2689-0526.100169.
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Research Article

The Psychological Effects of Smoking Cessation in Patients with Type II Diabetes

Ali Alshararni[1*] and Ahmed M Ashour[2]

[1]Department of Clinical Pharmacy, College of Pharmacy, Taif University, Saudi Arabia

[2]Department of Pharmacology and Toxicology, College of Pharmacy, Umm Al Qura University, P.O. Box 13578, Makkah 21955

[*]**Corresponding author:** Ali Alshararni, Department of Clinical Pharmacy, College of Pharmacy, Taif University, P.O. BOX: 888, Haweiah 21974, Saudi Arabia

Citation: Alshararni A and Ashour AM (2022) The Psychological Effects of Smoking Cessation in Patients with Type II Diabetes. Arch Surg Clin Case Rep 5: 169. DOI: 10.29011/2689-0526.100169

Received Date: 11 March 2022**; Accepted Date:** 15 March 2022**; Published Date:** 16 March 2022

Abstract

The increasing evidence of an association between active and even passive smoking and risk of diabetes in a dose-dependent pattern, the association between smoking and glycaemia-related parameters in diabetic patients, including HbA1c levels, has been variably demonstrated. Several factors may be implicated in smoking-related insulin resistance. These include nicotine-associated elevation of cortisol and growth hormone, elevated oxidative stress and inflammatory biomarkers, and enhanced sympathetic activation. The population targeted by this study consisted of adult patients with comorbid association of T2DM for a duration of 5 years or longer, who smoked and lived in Jeddah, Saudi Arabia. The 21-item version of the Depression, Anxiety and Stress Scale (DASS), was used to assess the psychological impact of smoking cessation. The stress factor element showed that patients with extremely severe stress had the greatest decrease in HbA1c (p=0.747), Q-Risk (p=0.088), TG level (p=0.290), and systolic BP (p=0.011). Patients with extremely severe anxiety achieved the highest increase in diastolic BP (mean change = +10.0 mmHg; p=0.025). Patients with severe depression had the smallest decrease in systolic BP (p=0.004) and they achieved the greatest decrease in daily number of cigarettes smoked (p=0.004). These findings are in favour of the multi-dimension positive impact of education interventions among diabetic patients, which may explain part of the findings in this study.

Keywords: Diabetes mellitus; Primary care; Cessation; Smoking; Body mass index

Introduction

High stress levels have been linked to difficulties in the management of diabetes across several studies. Notably, a variety of parameters of stress can be an important starting point in understanding stress levels and hence possibilities of difficulties in the management of diabetes in patients. Hormones from stress can increase the blood pressure or even increase the heart rate of a diabetic patient, consequently impacting efforts to manage diabetes in a negative way. Top parameters of stress that can be an important starting point towards the understanding of why stress can be a negative factor in the management of diabetes include the heart rate, body temperature, blood pressure, and the galvanic skin temperature. Activities like cigarette smoking can influence these stress parameters significantly, making it necessary for research on the impact of the effects of smoking on patients with diabetes [1]. With increasing evidence of an association between active and even passive smoking and risk of diabetes in a dose-dependent pattern [1], the association between smoking and glycaemia-related parameters in diabetic patients, including HbA1c levels, has been variably demonstrated. An Italian study has shown that smoking was associated with increased levels of HbA1c in T2DM and this association was also accompanied with deteriorative effects on nephropathic parameters [2]. The same results were obtained by Nilsson et al. [3] indicating a significant association between smoking habits and HbA1c levels (Nilsson et al, 2009). In a large epidemiological study involving Japanese male patients with T2DM, HbA1c levels increased markedly in smokers with a mean increase of 0.2% in active smokers relative to non-smokers, and remained elevated even after controlling the confounding factors, such as patients' age, duration of diabetic evidences, and BMI [4]. In addition, HbA1c levels were elevated with the increase in smoking intensity, represented as the number of cigarettes per day and pack-years of smoking when compared to non-smokers [4]. Moreover, in former smokers, HbA1c levels decreased remarkably

with longer smoking cessation periods. It is imperative to note that such downward trends were similarly reported for insulin resistance in smokers [5-7]. Several factors may be implicated in smoking-related insulin resistance. These include nicotine-associated elevation of cortisol and growth hormone [8], elevated oxidative stress and inflammatory biomarkers [9], and enhanced sympathetic activation [10]. It may be presumed that the resultant increase in waist circumference (despite BMI reduction) in smoking individuals, as mediated by the anti-estrogenic effects of cigarette smoking might contribute to increased insulin resistance [11,12]. Furthermore, smoking may lead to chronic pancreatitis and subsequent reduction of β-cell function [13,14].

On the other hand, the correlation between smoking and FBG was much weaker than that with HbA1c [4]. Baggio et al. [2] found no relationship between FBG and smoking amongst 96 patients with T2DM. In non-diabetic smokers, although the levels of HbA1c were significantly increased, 2-h plasma glucose levels were significantly reduced and FBG values remained unchanged when all values were compared to non-smokers. These effects may be attributable to rapid gastric emptying in smokers which would eventually lead to elevation of glucose levels early after food intake with subsequent increases in HbA1c levels. Other possible explanations are accelerated glucose uptake through red blood cell membranes into the cell along with the overall increase of their lifespan by carbon monoxide of cigarettes and the accelerated glycation rate of deoxyhaemoglobin. Despite the harmful effects of smoking, most smokers fail to quit smoking on their own during the first months of abstinence, indicating a robust evidence of tobacco addiction. The pharmacological impacts of nicotine were identified as the main inducers of addiction. The risk of becoming addicted to tobacco (nicotine) may be a result of a complex interaction of psychological, pharmacological, genetic and socioeconomic factors such as marketing and design. However, studies showed that two genes play a role in tobacco addiction, CYP2A6 and CYP2D6. These genes are involved in the metabolism of nicotine to cotinine (major metabolite) and individuals with low activity CYP2A6 and CYP2D6 alleles are less likely to be nicotine dependent [15].

Nicotine is a volatile alkaloid that binds to nicotinic cholinergic receptors in the brain after its diffusion from the lungs. Because of such binding, the ionic channel opens, facilitating the entry of sodium and calcium ions. The most prevalent subtypes of cholinergic receptors in human brain are those of the α4β2 subtype, which is thought to be responsible for nicotine addiction. For confirmation, it has been shown that the knockout of β2 subunit gene in mice leads to the lack of nicotine effects, while nicotine-stimulated dopamine release in the ventral striatum in normal mice produced the traditional responses to nicotine [18]. Furthermore, re-injecting the β2 subunit gene into the ventral tegmental area in mice with deleted β2 subunits produced considerable re-expression of the response [18]. On the other hand, the α4 subunit plays an important role in the nicotine dependence, including tolerance and sensitisation by repeated chronic administration of nicotine [19]. Therefore, nicotine is thought to stimulate the release of dopamine in the brain. In addition, other neurotransmitters are produced, such as serotonin, acetylcholine, and gamma amino butyric acid, leading to development of nicotine-related behaviours [20]. The reinforcing effects of nicotine are attributed to the resultant pleasurable impacts of dopamine and activation of reward circuit. Between cigarettes, nicotine levels in the brain are reduced leading to induction of several processes which contribute to cigarette craving and the need to smoke. Acute administration of nicotine leads to an increase in brain reward function, while its withdrawal produces diverse effects, which are considered key elements of nicotine addiction and strong barriers to abstinence [21]. Chronic repeated exposure to nicotine is associated with an increase in the binding sites of nicotinic receptors. Neuroadaptation, or tolerance, occurs with chronic exposure due to desensitization.

Methods

Study population

The study population targeted by the current study consisted of adult patients with comorbid association of T2DM for a duration of 5 years or longer, who smoked and lived in Jeddah, Saudi Arabia.

Eligibility criteria

To be eligible, participants complied with the following characteristics:

- Female and male individuals in the age range of between 25 and 75 years (World Health Organization, 2017);
- English or Arabic speaking;
- Absence of mental disorders or intellectual disability;
- T2DM diagnosed ≥ 5 years ago;
- To have been smoking cigarettes within in at least 5 consecutive years, with ≥ 10 cigarettes per day [22];
- Not to have significantly involved in tobacco abstinence during the last 1 year; which is defined as ≥ 3 consecutive months or at least 6 months of cumulative abstinence over the last 3 years. Recent abstinence from tobacco might bias the observed effect of smoking cessation during the trial.
- Lack of class III or extreme obesity which is defined as body mass index (BMI) ≥ 40 Kg/m², because such patients are more likely to develop severe obesity after a smoking cessation [15];
- Absence of any history of stroke or myocardial infarction (MI),

because of a reported, although controversial, vasoconstrictor action of nicotine [23].

- A lack of any skin sensitivity history [23].
- Not breastfeeding or pregnant; because there is insufficient assessment of the safety of NRT during pregnancy and lactation [24].

The patient information sheet (PIS) and eligibility questionnaire were administered by a trained investigator for the pre-selection of the participants based on the previous criteria (Appendix 1 & 2). Three licensed general practitioners were involved in the study to ensure participants' follow up.

Study groups

Patients eligible to participate in the study based on the above eligibility criteria, having consented to participate in the study, were randomly allocated to one of the two groups:

Group 1 (intervention group): T2DM patients benefited from an 8-week smoking cessation program according to the UK NHS guidelines (Pharmacy Department Medicines Management Services, 2015), including daily application of a 24 hour nicotine patch (Nicotinell®, Roche, Swaziland) combined with behavioural intervention consisting of telephone counselling.

Group 2 (control group): smoking T2DM patients.

Psychological tests

The 21-item version of the Depression, Anxiety and Stress Scale (DASS), was used to assess the psychological impact of smoking cessation. It is a validated tool, which was developed by the Psychology Foundation of Australia to measure the presence and intensity of symptoms related to depressive, anxious or stress disorders. It is widely used in clinical studies as a screening tool for anxiodepressive disorders. It has shown to have good psychometric characteristics and was translated into different languages (http://www2.psy.unsw.edu.au/dass/over.htm, last accessed March 2018). A copy of the questionnaire is presented in Appendix 3.

Outcomes

Interim assessment of glycaemic control

Glycaemic control including HbA1c level was compared between the two groups at 3, 6 and 9 months. As baseline values may probably vary considerably between subjects and between treatment groups, the change in HbA1c (ΔHbA1c) by reference to baseline was considered to be a better indicator and was used for these analyses.

Other metabolic profile indicators

- Fasting glucose level: (FBG) and ΔFBG (Δ FBG = outcome FBG -baseline FBG);
- Lipid profile: TC and ΔTC (Δ TC = outcome TC -baseline TC); TG and ΔTG (Δ TG = outcome TG -baseline TG);
- Body weight and weight change;
- BMI and ΔBMI (Δ BMI = outcome BMI -baseline BMI);
- Systolic and Diastolic BP as well as ΔBP (Δ BP = outcome BP -baseline BP) [25];
- Cardiac risk score (QRISK) and ΔQRISK.

Recording of Data

On enrolment, the following information was recorded: The name of the research site, the name of the participant, telephone number, the participant's address, the participant's date of birth and GP name, and the telephone number. Other study variable, including clinical and biochemical factors and outcomes, and smoking-related parameters, were also recorded. The telephone counselling calls were recorded.

Results

To analyse the impact of psychological factors on outcomes, subgroup analyses were carried out by respect of the stress, anxiety and depression levels among intervention group. Most remarkably, the stress factor element showed that patients with extremely severe stress had the greatest decrease in HbA1c (p=0.747), Q-Risk (p=0.088), TG level (p=0.290), and systolic BP (p=0.011). In parallel, although not statistically significant, this subgroup achieved the highest % reduction in smoking amount (p=0.746) and the longest cumulative abstinence duration (p=0.163) and was the only subgroup with increase in expired CO (p=0.454) compared to the other subgroups. On the other hand, patients with no stress (normal DASS stress score) exhibited the second smallest reduction in both HbA1c, Q-Risk and weight, the smallest reduction in systolic BP and achieved the least reduction in number of daily cigarettes smoked (p=0.033) and the second least percentage reduction in smoking amount (Figure 1).

As to the anxiety factor, patients with extremely severe anxiety achieved the highest increase in diastolic BP (mean change = +10.0 mmHg; p=0.025); along with other statistically non-significant negative outcomes including HbA1c (mean change = +1.4%; p=0.141), weight (mean change = +0.9 Kg; p=0.512), total cholesterol (mean change= +15.7 mg/dL; p=0.771), and expired CO (only -3.0 mean change; p=0.652), although they achieved the greatest reduction in the number of cigarettes (mean change = -43.3 cigarettes per day; p=0.001) (Figure 2). These observations suggest that heavy smokers may typically be more exposed to anxiety or to develop anxiety during smoking cessation (Piper et al, 2011) [18]. Depression factor showed that patients with severe depression had the smallest decrease in systolic BP (p=0.004) and they achieved the greatest decrease in daily number of cigarettes

smoked (p=0.004). On the other hand, these patients were likely to achieve the greatest decrease in Q-Risk (p=0.069), smallest decrease in HbA1c (p=0.808), increase in weight (p=0.094); and longest cumulative abstinence duration (p=0.809) (Figure 3).

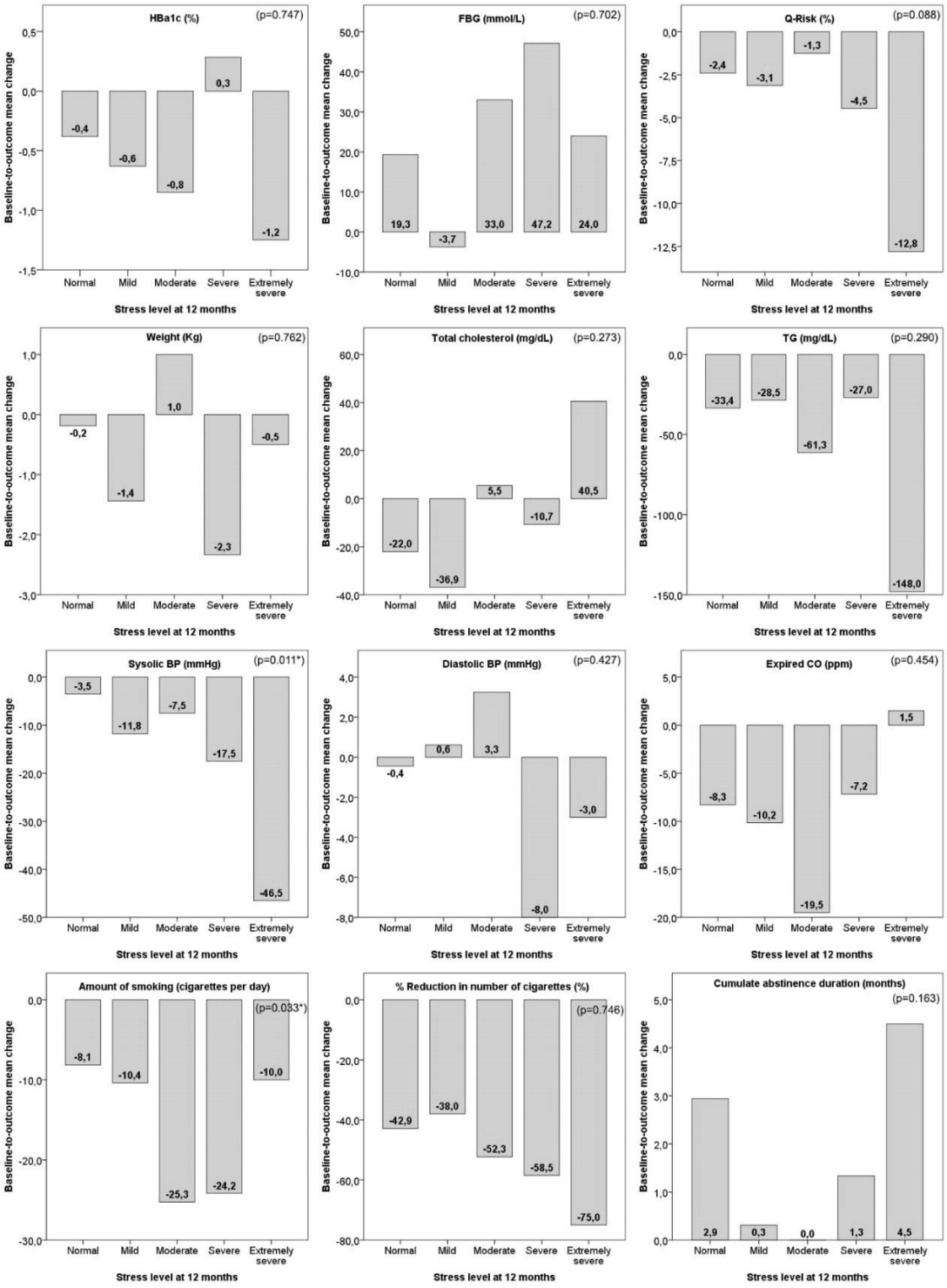

Figure 1: Correlation of outcome stress level with main study variables.

Bars represent the mean baseline-to-outcome changes in different study parameters by the level of stress at 12 months.

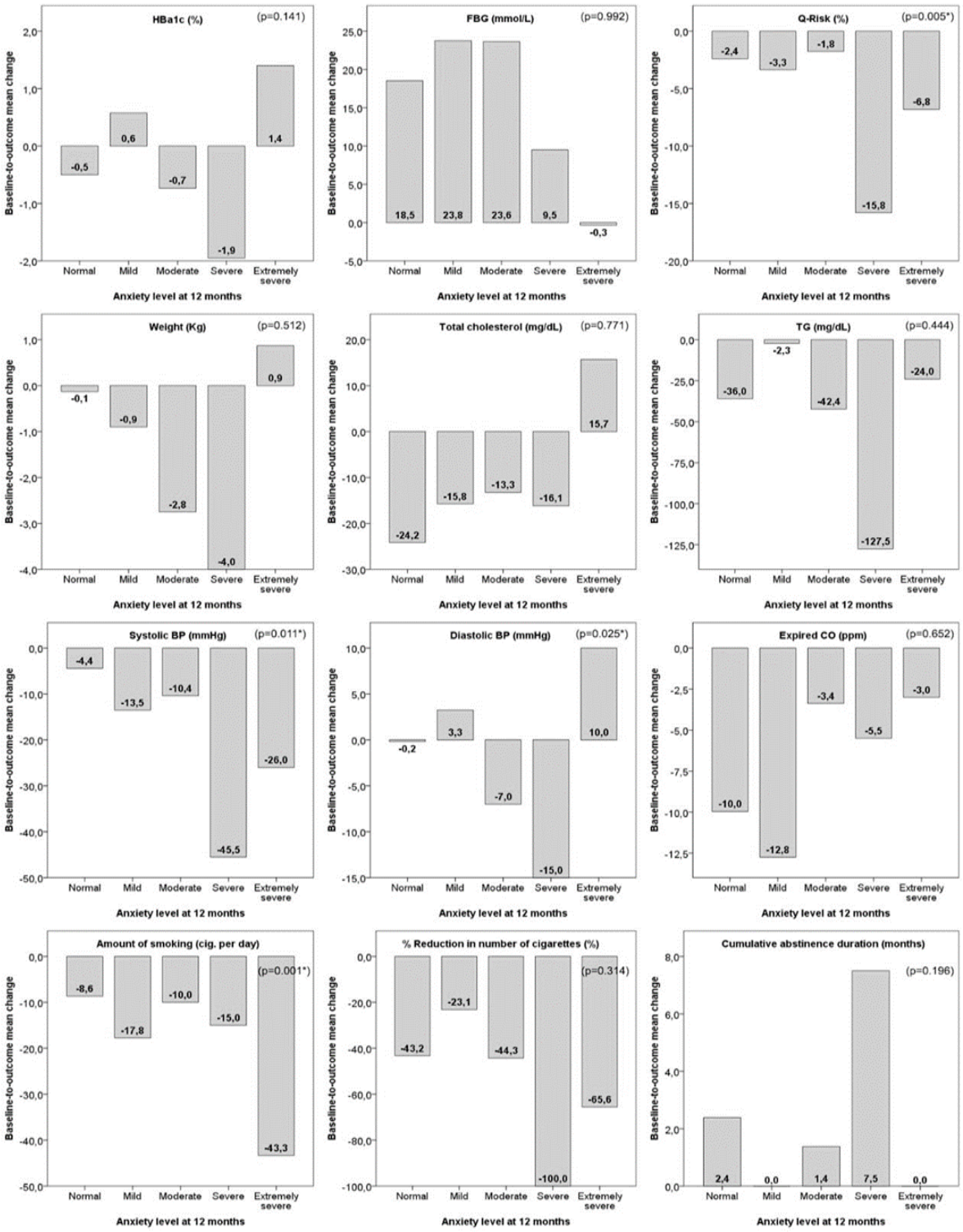

Figure 2: Correlation of outcome anxiety level with main study variables.

Bars represent the mean baseline-to-outcome changes in different study parameters by the level of anxiety at 12 months.

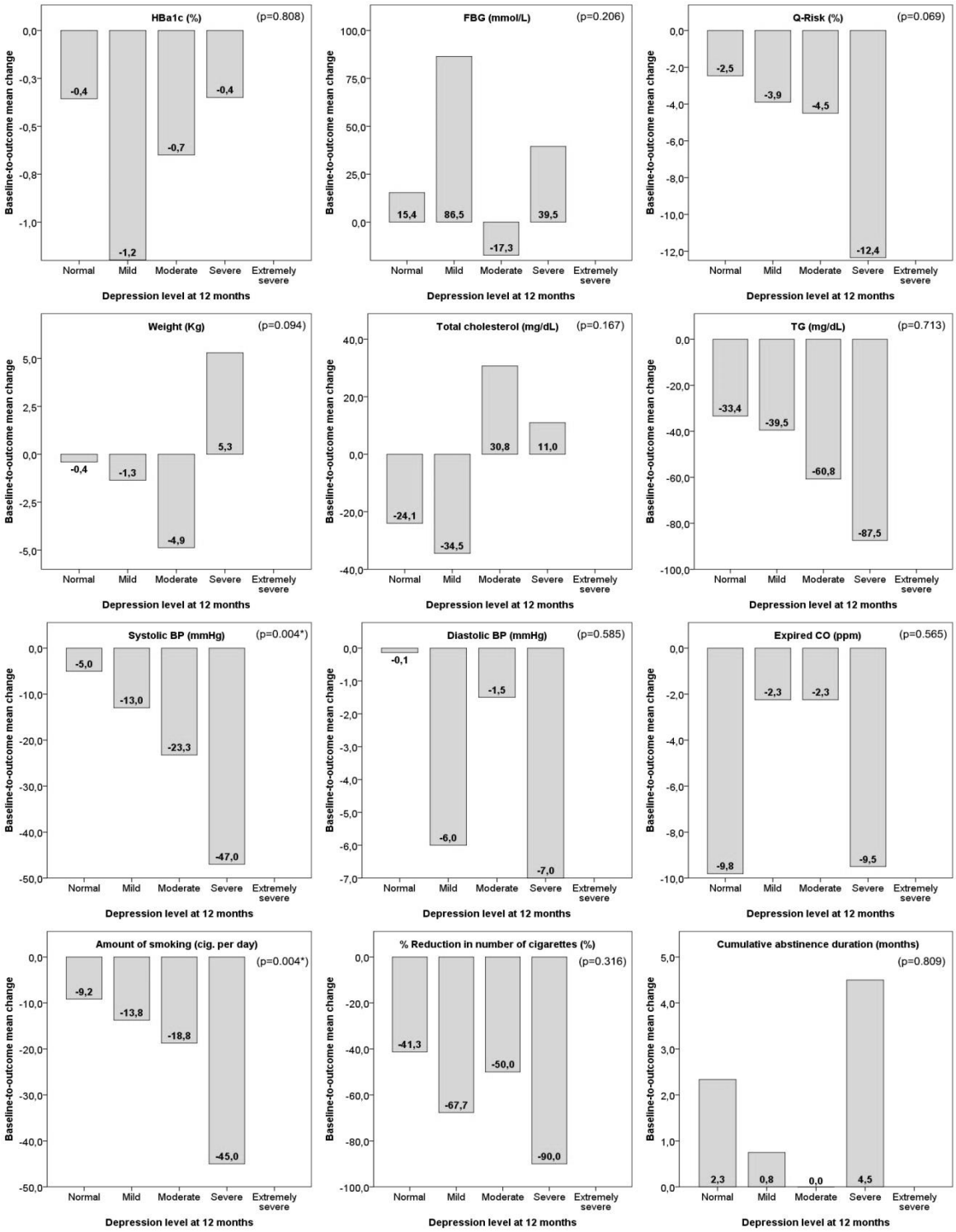

Figure 3: Correlation of outcome depression level with main study variables

Bars represent the mean baseline-to-outcome changes in different study parameters by the level of depression at 12 months.

Discussion

Remarkably, the per-protocol analysis showed a significant weight loss (mean -1.38 kg) among patients who followed the smoking cessation programme in comparison to control (-0.48 kg), at 12-month follow-up. This weight loss was preceded by a transient weight gain (+2.6 kg) from the third to sixth month of follow-up. On the other hand, ITT analysis showed no significant difference between the two groups at all study phases. This indicates that weight reduction was associated with cigarette abstinence and may be related to proactive changes in lifestyle and dietary habits, in parallel with the cigarette abstinence. These findings are in contrast with data generally reported in the literature, where weight gain is acknowledged to be one of the main adverse effects of smoking cessation with variable amount predicted by several sociodemographic and genetic factors, in addition to the amount of smoking [26]. Weight gain is considered one of the major barriers to smoking cessation among patients and the obstacle may be further accentuated among people who are overweight or obese [26]. On the other hand, weight gain is mostly observed in the early period following smoking cessation and the weight curve levels off afterwards. A meta-analysis by Aubin et al. demonstrated that 12-months of cigarette abstinence is associated with an average 4-5 kg weight gain, regardless of the method used to support smoking cessation (untreated, NRT, bupropion or varenicline), with the weight curve being more accentuated in the first months. However, a proportion of individuals (16%) do lose weight following smoking cessation [14]. Comparably, a randomised trial one-year smoking cessation induced +4.6 kg of mean weight gain, along with +2.8 cm mean increase in waist circumference and +1.6 kg/m^2 mean increase in BMI [27,28]. In the study by Voulgari et al. [17], one-year smoking cessation among patients newly diagnosed with T2DM was associated with increase in BMI (+0.8 versus -1.7 kg/m^2) and relative increase in waist circumference (-7.4 versus -8.3 cm), versus continuing smokers. Clair et al. [15] observed that the median post-cessation weight gain among diabetic patients was 3.6 kg among recent quitters, which was very high by comparison to 4-year weight gain among long-term quitters (0.9 kg). Parallel findings were observed among nondiabetic smokers, indicating no major difference between diabetic and nondiabetic quitters in weight variability. Interestingly, the same study highlighted a significant reduction in the cardiovascular risk following smoking cessation, equally among recent and long-term quitters and regardless of the amount of weight gain [15]. The latter observations are in line with my cardiac risk estimation, which strengthens the evidence that the cardiovascular benefit outweighs the post-cessation weight gain.

Mechanisms of weight gain after smoking cessation are not completely elucidated; however, increased energy intake, decreased resting metabolic rate and increased lipoprotein lipase activity appear to be among the main mechanisms. It is well established that nicotine consumption is associated with reduced appetite, impaired glucose tolerance and insulin resistance. Following quitting smoking, body insulin sensitivity was gradually improved leading to enhanced glucose cellular intake, which combined to low physical activity and high intake leads to high fat production. Therefore, it was observed that NRT may reduce post-cessation weight gain, in addition to physical activity and dietary intervention; all being part of the necessary advice to quitters [24]. Among eventual measures regarding weight gain issue, it was observed that bupropion, with or without NRT, reduced post cessation weight gain. However, such results were not verified among diabetic patients (Filozof et al, 2004) [19]. Interestingly, a study by Davies et al. (2008a) [16] assessed the efficacy of a structured group education programme for patients newly diagnosed with T2DM and evidenced a mean reduction in weight of approximately 3 kg, at 12-month follow up, along with higher rate of smoking cessation. Further, the study highlighted significant positive changes in patients' perception and beliefs regarding the disease and improvement of anxiodepressive symptoms. These findings are in favour of the multi-dimension positive impact of education interventions among diabetic patients, which may explain part of the findings in the current study.

Conclusion

The study shows that more stressed individuals are less likely to cease their smoking habit while those experiencing anxiety tend to smoke more. For severely depressed individuals, the risk of continued smoking after NRT intervention is minimal. The study's conclusion would be that it is important to address the stress and anxiety factors before applying intervention protocols if any meaningful results are to be achieved.

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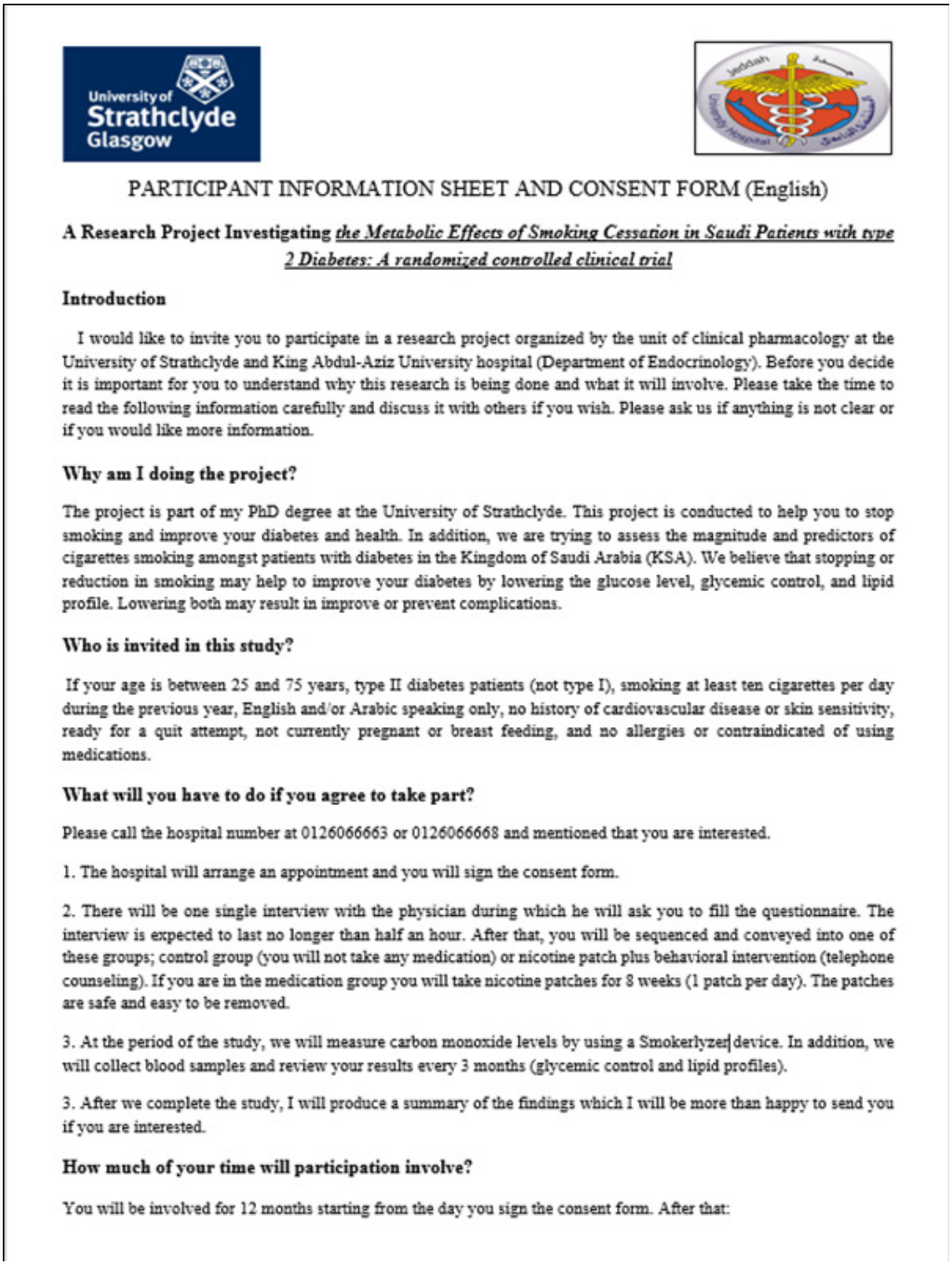
Appendix 1

Participant Information Sheet and Consent Form (English versions)



PARTICIPANT INFORMATION SHEET AND CONSENT FORM (English)

A Research Project Investigating *the Metabolic Effects of Smoking Cessation in Saudi Patients with type 2 Diabetes: A randomized controlled clinical trial*

Introduction

I would like to invite you to participate in a research project organized by the unit of clinical pharmacology at the University of Strathclyde and King Abdul-Aziz University hospital (Department of Endocrinology). Before you decide it is important for you to understand why this research is being done and what it will involve. Please take the time to read the following information carefully and discuss it with others if you wish. Please ask us if anything is not clear or if you would like more information.

Why am I doing the project?

The project is part of my PhD degree at the University of Strathclyde. This project is conducted to help you to stop smoking and improve your diabetes and health. In addition, we are trying to assess the magnitude and predictors of cigarettes smoking amongst patients with diabetes in the Kingdom of Saudi Arabia (KSA). We believe that stopping or reduction in smoking may help to improve your diabetes by lowering the glucose level, glycemic control, and lipid profile. Lowering both may result in improve or prevent complications.

Who is invited in this study?

If your age is between 25 and 75 years, type II diabetes patients (not type I), smoking at least ten cigarettes per day during the previous year, English and/or Arabic speaking only, no history of cardiovascular disease or skin sensitivity, ready for a quit attempt, not currently pregnant or breast feeding, and no allergies or contraindicated of using medications.

What will you have to do if you agree to take part?

Please call the hospital number at 0126066663 or 0126066668 and mentioned that you are interested.

1. The hospital will arrange an appointment and you will sign the consent form.

2. There will be one single interview with the physician during which he will ask you to fill the questionnaire. The interview is expected to last no longer than half an hour. After that, you will be sequenced and conveyed into one of these groups; control group (you will not take any medication) or nicotine patch plus behavioral intervention (telephone counseling). If you are in the medication group you will take nicotine patches for 8 weeks (1 patch per day). The patches are safe and easy to be removed.

3. At the period of the study, we will measure carbon monoxide levels by using a Smokerlyzer device. In addition, we will collect blood samples and review your results every 3 months (glycemic control and lipid profiles).

3. After we complete the study, I will produce a summary of the findings which I will be more than happy to send you if you are interested.

How much of your time will participation involve?

You will be involved for 12 months starting from the day you sign the consent form. After that:

Eligibility Questionnaire

Patient's Name	……	……..	……..
Date of birth		Serial number	

You are consulting for type II diabetes in our clinic and we pray Allah to give you quick recovery. Please answer the following questions:

Criterion	Inclusion	Exclusion
1. Are you more than 25 years old?	□ Yes	□ No
2. Are you less than 75 years old?	□ Yes	□ No
3. When were you diagnosed as diabetic?	□ More than 5 years ago	□ Less than 5 years ago
4. Do you smoke cigarettes?	□ Yes	□ No
5. When did you start smoking?	□ More than 5 years ago	□ Less than 5 years ago
6. In average, how many cigarettes do you smoke per day?	□ 10 or more	□ Less than 10
7. During the last year, have you quitted smoking for more than 3 consecutive months or more than 6 cumulative months?	□ No	□ Yes
8. Do you have a history of stroke or myocardial infarction?	□ No	□ Yes
9. Do you have a history of sensitivity skin reaction?	□ No	□ Yes
10. Are you currently using any other psychoactive substances?	□ No	□ Yes
11. Are you pregnant or breast-feeding?	□ No	□ Yes
12. What is your weight? ……..Kg	□ BMI <40 kg/m²	□ BMI ≥40 Kg/m²
13. What is your height? ……..m		
Investigator's decision:		
□ Eligible	□ Not eligible	
Researcher Name:	Signature:	
Date:		

Citation: Alshararni A and Ashour AM (2022) The Psychological Effects of Smoking Cessation in Patients with Type II Diabetes. Arch Surg Clin Case Rep 5: 169. DOI: 10.29011/2689-0526.100169

Appendix 2

King Abdul-Aziz University Ethical Approval

KINGDOM OF SAUDI ARABIA
Ministry of Education
KING ABDULAZIZ UNIVERSITY
Faculty of Medicine

المملكة العربية السعودية
وزارة التعليم
جامعة الملك عبد العزيز
كلية الطب

Ref.: .... | الرقم : [illegible]
Date: / / | التاريخ ٢٦/ ٥ / ١٤٣٧هـ
Encl.: .... | المرفقات: ....

UNIT OF
BIOMEDICAL ETHICS
Research Committee

Initial Approval

TO: Principal Investigator: **Dr. Ahmed M. A. Ashour**
(Clinical Pharmacology at Umm ALQura University)
Co-investigator: Prof .AbdulRahman ALShaikh
Date: Sunday, March 06, 2016

From: Professor. Hasan Alzahrani

RE:"Smoking Among Patients with Diabetes in The Kingdom of Saudi Arabia : Cohort Case- Control Study On Nicotine Patch and Behavioral Interventions Used For Smoking Cessation ."
(Reference No 77-16) Case Control -Cohort

The above titled research/study proposal has been examined with the following enclosures:

- The Study Protocol.
- Informed consent Form

The REC recommended granting permission of approval to conduct the project along the following terms:

1. The PI and Investigators are responsible to get Academic Affairs, hospital and departmental approval.
2. The Investigators will conduct the study under the direct supervision by the consultant **Prof .AbdulRahman ALShaikh.**
3. Provide to committee" Continuing Review Progress Report "each 3 months.
4. Any amendments to the approved protocol or any element of the submitted documents should NOT be undertaken without prior re-submission to, and approval of the REC for prior approval.
5. Monitoring: the project may be subject to an audit or any other form of monitoring by the REC.
6. The PI is responsible for the storage and retention of original data of the study for a minimum period of five years.
7. The PI is expected to submit a final report at the end of the study.
8. The PI must provide to REC a conclusion abstract and the manuscript before publication.
9. To follow all regulations issued by the National Committee of Bio & Med ethics - King Abdul Aziz City for Science and Technology.

The Organization & operating procedure of the KAU. Faculty of Medicine - Research Ethics Committee(REC) are based on the Good Clinical Practice (GCP) Guidelines.
PLEASE NOTE THAT THIS APPROVAL IS VALID FOR ONE YEAR COMMENCING FROM THE DATE OF THIS LETTER.

Professor Hasan Alzahrani

Chairman of the Research Ethics Committee

(HA-02-J-008) No of Registration At National Committee of Bio.& Med. Ethics.
Mohammed ALsearee (Reference No 77-16)

ص.ب : ٨٠٢٠٥ جـدة : ٢١٥٨٩
P.O.Box: 80205 Jeddah: 21589
فاكس : ٦٤٠٠٥٩٢ / ٦٤٠٨٤٥١
Fax.: 6408451 /6400592
☎ : ٦٩٥٢٠٦٣ / ٦٩٥٢٤٤٦
☎ : 6952446 / 6952063