Nutrition, Metabolism & Cardiovascular Diseases (2017) 27, 335–341

Available online at www.sciencedirect.com

Nutrition, Metabolism & Cardiovascular Diseases

journal homepage: www.elsevier.com/locate/nmcd

Smoking cessation improves cardiometabolic risk in overweight and obese subjects treated with varenicline and dietary counseling

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Received 9 September 2016; received in revised form 18 December 2016; accepted 29 December 2016
Available online 4 January 2017

KEYWORDS
Smoking cessation;
Metabolic syndrome;
Cardiometabolic risk;
Body weight

Abstract *Background and Aim:* Weight gain after stopping smoking potentially counteracts improvements in cardiometabolic risks. We investigated changes in metabolic syndrome (MetS) components and homeostasis assessment model insulin resistance (HOMA-IR) in smokers given dietary counseling during their quit attempt.
Methods and results: Smokers (≥10 cigarettes/day) with BMI 25–40 kg/m^2 were randomized to a low-carbohydrate or low-fat diet and treated with a standard course of varenicline for 12 weeks. Quitters were assessed according to the Russell standard (≤5 cigarettes after the quit date) validated with expired breath carbon monoxide (CO) < 10 ppm. Of 122 randomized participants, 108 (89%) completed clinical and laboratory assessments at 12 weeks. As changes in metabolic risk factors did not differ between dietary groups, we combined the groups to compare quitters to continuing smokers. We found similar weight change among 78 validated quitters as 30 continuing smokers (−0.1 ± 3.0 kg vs 0.3 ± 3.1 kg; p = 0.7) and change in waist circumference (−2.0 ± 3.8 cm vs −0.9 ± 3.9 cm; p = 0.2). Changes in triglyceride concentrations (−0.16 ± 0.52 mmol/l vs 0.21 ± 0.95 mmol/l; p = 0.015) and diastolic blood pressure (−0.9 ± 6 mmHg vs 1.9 ± 8 mmHg; p = 0.039) were more favorable in quitters. Changes in other cardiometabolic risks and HOMA-IR did not differ between quitters and continuous smokers, nor did energy intake or resting metabolic rate.
Conclusion: Dyslipidemia and blood pressure improved and no early weight gain was seen in quitters, suggesting that dietary intervention can mitigate some of the effects of stopping smoking on cardiometabolic risk factors in overweight and obese smokers.
Clinical trials registration: NCT01069458.
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Introduction

Smoking cigarettes is the key reversible risk factor for cardiovascular disease (CVD), while stopping smoking rapidly lowers risk [1]. The mechanisms behind the risk of smoking include promotion of dyslipidemia, inflammation, endothelial dysfunction, coagulation, platelet adherence and insulin resistance [2]. Both active and passive smoking increase the risk of type 2 diabetes, as shown in recent meta-analysis [3]. Likewise, smoking increases risk of metabolic syndrome (MetS), a cluster of cardiometabolic risk factors associated with central obesity and insulin resistance. A meta-analysis of prospective cohort studies found a dose–response relationship between smoking and risk of MetS [4]. Furthermore, the population-based LifeLines Cohort study demonstrated

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http://dx.doi.org/10.1016/j.numecd.2016.12.011
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that smokers experienced a greater incidence of MetS independent of BMI [5]. Additionally, cross-sectional studies have indicated that waist or waist-to-hip ratio is higher in smokers than non-smokers [5,6].

Conversely, recent smoking cessation is associated with a temporary risk of type 2 diabetes, mostly driven by weight gain following cessation. This risk decreases substantially as the time since quitting lengthens [3,7]. Weight gain is an almost inevitable consequence of stopping smoking, particularly in persons at risk because of overweight or obesity. In a recent meta-analysis the average weight gain among quitters was 4–5 kg after 1 year [8]. Furthermore, obese smokers gain more weight than their normal weight counterparts with some reports indicating substantial weight gain in this group [9].

Together with an increased risk of diabetes, stopping smoking may worsen MetS and some of its components. There is evidence pointing to an increased risk of central obesity following stopping smoking. In a cross-sectional analysis of 7462 women, past smokers had significantly higher odds for MetS than current smokers mostly due to greater waist circumference [10]. Stadler et al. [11] reported increased weight, fat mass, fasting insulin and a deterioration in fasting insulin sensitivity 3 months after smoking cessation. In a recent clinical trial smokers treated with cognitive behavioral therapy alone or combined with medication, showed increased triglyceride concentrations in all treatment groups and increased homeostasis assessment model insulin resistance (HOMA-IR) in the behavioral therapy plus varenicline group [12].

Obese smokers have been pinpointed as the group with the greatest need for interventions to ameliorate weight gain [9]. Thus, we conducted a trial among 122 overweight or obese smokers of whom 64 participants were randomized to follow a low-carbohydrate diet while 58 were randomized to a low-fat diet, in conjunction with treatment with varenicline as an aid for smoking cessation [13]. We found minimal weight gain after 12 weeks in quitters with no difference between the dietary groups. In the current analysis we combined both dietary groups, and compared changes in body weight, MetS components and HOMA-IR between participants who showed sustained abstinence from smoking versus non-quitters. Our study aim was to better understand cardiometabolic changes in overweight or obese quitters given dietary counseling in the immediate post-quit period.

Methods

Study design and subjects

The study had a randomized, controlled, parallel group design as described previously [13]. Participants were recruited among referrals to the Section for Preventive Cardiology at Oslo University Hospital and mostly by newspaper advertisement. In brief, men and women aged 20–65 years who were overweight or obese (BMI 25–40 kg/m^2) and smoked ≥10 cigarettes daily, were motivated to quit and willing to be treated with varenicline to aid cessation were included. Exclusion criteria were the occurrence of a cardiovascular event within 2 months prior to screening, diabetes mellitus type 1 or 2 treated with insulin, serious psychiatric disorder, alcohol or drug abuse, pregnancy and lactation, bariatric surgery, medication for weight loss or recent change in weight (>4 kg during the last 3 months), vegetarian diet, and disorder impairing compliance with dietary recommendations. The Regional Committees for Medical and Health Research Ethics in Norway evaluated the study and the work was conducted in accordance with the Declaration of Helsinki. All subjects signed a written informed consent before screening procedures.

Following screening subjects returned 1 week later for randomization to 1 of 2 diets (baseline visit). Treatment with a 12-week course of varenicline was started 4 days thereafter. The target quit date (TQD) was 10 days after the initiation of varenicline. Follow-up visits were scheduled weekly after baseline to the 4-week post-TQD visit and thereafter biweekly to the 12-week post-TQD visit. At each study visit, the study physician or trained nurses provided up to 10 min of motivational counseling for cessation.

Randomization diets were either a low-carbohydrate diet planned to provide ≤20 percentage energy (E%) from carbohydrates and ≥25 E% from protein with the remaining from fat or a moderately fat-reduced diet planned to provide ≤30 E% from fat, ≤20 E% from protein with the remaining from carbohydrates. Both diets were equally reduced in energy by 500 kcal/day. Dietary advice and support were given by trained dietitians at each study visit.

Subjects reported the number of cigarettes smoked since the last visit at each visit. Exhaled carbon monoxide (CO) concentrations were tested using a Bedfont piCO + Smokerlyzer at each visit. Quitters were counted according to the Russell standard for sustained quitting that allows ≤5 cigarettes in the entire follow-up period confirmed by CO levels <10 ppm [14].

Body weight was measured on a digital and calibrated scale (Seca 770) in light indoor clothing without shoes. Waist circumference was measured midway between the lowest rib and iliac crest. Blood pressure was measured three times at 2-minute intervals using an appropriate cuff and an automatic device (Omron M10-IT, Omron Healtcare Co. Ltd) after the participant rested quietly in a sitting position for at least 5 min.

Resting metabolic rate (RMR) was measured at the baseline and 4-week post-TQD visits using the ventilated-hood system Vmax Spectra 229 indirect calorimeter (SensorMedics). The subjects fasted overnight and refrained from smoking and heavy physical activity in the morning before the test was performed according to standardized procedures.

Participants completed a weighed dietary record during 7 days before randomization and before the 4-week post-TQD visit. Energy intake was calculated using software “Mat på data” 5.0 based on the Norwegian food composition table. Physical activity was measured for 7 consecutive days as the participant wore an Actigraph GT3X + accelerometer

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