



Anthropometric and body composition changes in smokers vs abstainers following an exercise-aided pharmacotherapy smoking cessation trial for women



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HIGHLIGHTS

- Weight gain associated with quitting was minimal for women completing the “Getting Physical on Cigarettes” trial.
- The minimal weight gain for abstainers was related to an increase in lean mass and not to an increase in fat.
- Exercise-aided smoking cessation programs are recommended to manage weight and body composition indices.

ARTICLE INFO

Keywords:

Body composition
Smoking cessation
DXA
Weight gain

ABSTRACT

Introduction: Post-cessation weight gain contributes to smoking relapse, especially for women. Furthermore, excess weight in the form of android or visceral fat is associated with metabolic health problems. For this study, a secondary analysis was conducted in 2015 to determine whether quitting status, achieved through a 14 week supervised exercise-aided nicotine replacement therapy (NRT) cessation program [Getting Physical on Cigarette Trial-2009 to 2013; Prapavessis, et al., 2016], affects anthropometric and body composition parameters in female smokers ($N = 413$, M age = 42.39 years).

Methods: Anthropometric (weight and BMI) and body composition (% total body fat, % android fat, lean mass and visceral fat) indices were assessed at baseline and end of treatment. Smoking status was confirmed weekly from expired breath carbon monoxide. Adherence to exercise and NRT patch was calculated from the number of exercise sessions attended and patches worn to the number of exercise sessions offered and patches supplied, respectively.

Results: Factorial (smoking status) ANCOVAs controlling for baseline anthropometric and body composition parameters as well as adherence to exercise and NRT revealed significant differences in weight ($p = .033$; $\eta_p^2 = 0.017$) and BMI ($p = .020$; $\eta_p^2 = 0.020$) at week 14. This equated to abstainers weighing 1.26 kg more and having a 0.52 higher BMI than smokers. No significant differences were found for any of the body composition parameters at week 14 (η_p^2 range from 0.001–0.007).

Conclusions: Abstainers gain modest weight compared to smokers. This weight gain is related to increases in lean mass and not total, android, or visceral fat.

1. Introduction

Tobacco smoking is the leading cause of preventable death, killing approximately six million individuals each year worldwide (WHO, 2013). Altered metabolic functioning and DNA damage, which promote carcinogenicity, can be avoided by sustained smoking cessation at any age (International Agency for Research on Cancer, 2007; USDHHS. U.S. Department of Health and Human Services, 2010). Regardless of

numerous compromises to health, most smokers find it difficult to quit smoking and failure rates are consistently high (Hughes, Keely, & Naud, 2004). In specific, one in ten Canadians who made a quit attempt were successfully abstinent from smoking at time of survey (one year later) (CTUMS. Canadian Tobacco Use Monitoring Survey, 2010). The inability to cope with cravings and tobacco withdrawal symptoms during a quit attempt are cited as pitfalls in becoming tobacco-free (Allen, Bade, Hatsukami, & Center, 2008).

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Differential risks exist for continued tobacco use in women, compared to men. Namely, women are at increased risk for smoking-related deaths and developing cancer than men (Gandini, Botteri, Iodice, et al., 2008; McCartney, Mahmood, Leyland, Batty, & Hunt, 2011). For women, smoking is perceived as a method of weight control and hence, post-cessation weight gain is a common culprit for both postponing and unsuccessful quit attempts, but also smoking relapses (Klesges, Brown, Pascale, et al., 1988). Women also have a tendency to gain somewhat more weight than their male counterparts (Swan & Carmelli, 1995). Without treatment to assist cessation, a previous meta-analysis (included both men and women) showed a mean weight gain of 1.12 kg, 2.26 kg, 2.85 kg, 4.23 kg and 4.67 kg at one, two, three, six and twelve months after quitting, respectively (Aubin, Farley, Lycett, Lahmek, & Aveyard, 2012).

Multiple and multifaceted mechanisms have surfaced to explain post-cessation weight gain. Nicotine (or the absence thereof) has been shown to affect the concentration and expression of multiple hormones in the body, caloric intake, metabolic rate during rest, fat oxidation, the regulation of energy balance, and abdominal adipose tissue metabolism (Ferrara, Kumar, Nicklas, McCrone, & Goldberg, 2001; Li, Parker, & Kane, 2000; Miyata, Meguid, Varma, Fetissov, & Kin, 2001; Moffart & Owens, 1991; Schutz, Tremblay, Weisier, & Nelson, 1992; Stamford, Matter, Fell, & Papanek, 1986). A Cochrane review conducted to evaluate interventions designed to achieve tobacco abstinence and minimize weight gain concluded that some pharmacological treatments decreased post cessation weight gain during use (Farely, Hajeck, Lycett, & Aveyard, 2012). In specific, nicotine replacement therapy (NRT) attenuated weight gain at end of treatment by -0.45 kg [95% CI -0.66 to -0.027 , $N = 18$] after quitting and significant differences between different forms of NRT were not found. No evidence of an effect of weight gain was found at 12 months [Mean difference = 0.45 , 95% CI -0.92 to 0.08 , $N = 15$]. Bupropion, fluoxetine and varenicline mitigated post cessation weight gain at the end of treatment [bupropion Mean difference = -1.12 kg, 95% CI -1.47 to -0.77 , $N = 7$; fluoxetine Mean difference = -0.99 kg, 95% CI -1.36 to -0.61 , $N = 2$; varenicline Mean difference = -0.41 kg, 95% CI -0.63 to -0.19 , $N = 11$], but not at 6 or 12 months. Finally, exercise had limited impact on diminishing weight at the end of treatment [Mean difference = -0.25 kg, 95% CI -0.78 to -0.29 , $N = 4$], but there was a significant weight reduction 12 months post-treatment [Mean difference = -2.07 kg, 95% CI -3.78 to -0.36 , $N = 3$].

These end of treatment non-significant exercise findings can be explained by a number of limitations. First, an objective appraisal of exercise adherence during treatment was absent in all four of the randomized controlled trials included in the Cochrane review. In order to gauge the impact of an exercise-aided smoking cessation intervention on attenuating post-cessation weight gain, exercise adherence must be measured and controlled for. Second, the limited but nevertheless encouraging evidence that combining exercise and NRT has a positive effect on end of treatment smoking status and weight gain was not included in the review. Prapavessis and colleagues, for instance, found when abstainers were on the patch there was no differences in treatment weight gain; however for abstainers not on the patch those that received cognitive behavior therapy put on significantly more weight than those who received exercise (Prapavessis, Cameron, Baldi, et al., 2007). Third and finally weight and BMI is an inexpensive measure which does not discriminate between fat and lean mass (Zanovec, Lakkakula, & Johnson, 2009). Excess weight, particularly in the form of android or visceral fat, is associated with cardiovascular and metabolic disease (Direk, Cecelja, Astle, et al., 2013). Thus, examining high quality body composition parameters in smokers engaged in cessation treatment programs is important in determining disease risk. To our knowledge, only two studies have examined this issue. Kleppinger, Litt, Kenny, & Oncken, (2010) showed that postmenopausal women who quit smoking after 16 weeks of NRT patch plus group behavioral counseling demonstrated significantly higher absolute change in fat

tissue (i.e., 3.5 kg) and lean tissue (i.e., 0.6 kg) compared to their smoking counterparts. In a pilot study, Ciccolo et al. reported end of treatment (i.e., 12 weeks) 7-day point prevalence rates of 46% for participants that received NRT/counseling plus resistance training compared to 17% for those that received NRT/counseling plus non-specific contact. Resistance training participants showed a mean reduction in body weight (0.6 kg; SD = 1.7) and fat (0.5%; SD = 1.8) while their non-specific counterparts showed a mean increase in weight (0.6 kg; SD = 2.8) and fat (0.6%; SD = 0.7). These differences correspond to 0.7 and 0.8 Cohen's d effect sizes, respectively (Ciccolo, Dunsiger, Williams, et al., 2011).

With the abovementioned limitations in mind, the current study was a secondary analysis of women smokers who had participated in an exercise-aided pharmacotherapy smoking cessation trial for women (Getting Physical on Cigarettes - NCT01305447; for primary outcome analysis see Prapavessis, De Jesus, Fitzgeorge, et al., (2016)). The objective was to examine the influence of smoking status on anthropometric (i.e., weight and BMI) and high quality body composition (e.g., % android fat, lean mass and visceral fat) changes over 14 weeks of treatment.

2. Methods

2.1. Sample

Participants included 411 healthy, inactive female smokers. They were recruited from local businesses, hospitals, academic institutions and organizations, and through advertisements placed in newspapers, radio stations and city buses in London, Ontario. Individuals between 18 and 65 years of age, engaged in two or less 30-min sessions of moderate or vigorous intensity exercise in the previous six months, smoked > 10 cigarettes per day for the past two years, and wished to achieve smoking abstinence were eligible to participate in the trial. Individuals were excluded if they had contraindications to regular exercise or using nicotine replacement therapy, were prescribed medication for physical and/or mental health reasons that would impair compliance with study protocol, had other substance dependency problems and/or were pregnant.

2.2. Measures

2.2.1. Demographics and smoking history

Participants completed a demographic and smoking history questionnaire, which collected information such as age, level of education, annual household income, number of years smoking, cigarette consumption (e.g. number of cigarettes smoked per day) and weight concerns (Levine, Perkins, & Marcus, 2001).

2.2.2. Smoking status

Self-report cigarette consumption was assessed weekly throughout the program from baseline through week 14. Continuous abstinence (i.e., smoking status) was verified at the same intervals from expired breath carbon monoxide using the piCO+™ Smokerlyzer® (Bedfont Scientific Ltd., Kent, England). Carbon monoxide (CO) readings less than six parts per million (ppm) denoted smoking abstinence (Middleton & Morice, 2000). To be considered smoke-free for analyses participants had to show CO levels < 6 ppm for the full 10 weeks of the treatment program (i.e., weeks 4–14). Those who provided CO levels ≥ 6 ppm or failed to provide CO level at any time over the treatment program were considered a smoker.

2.2.3. Anthropometrics

Height and weight were collected (Health-o-meter professional, Pelstar 500KL) after asking participants to remove their shoes and heavy clothing (e.g., sweater). Height and weight were recorded to the nearest 10th of a centimetre or kilogram, respectively. Weight served as

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