Addictive Behaviors 39 (2014) 239–245

Contents lists available at ScienceDirect

Addictive Behaviors

Factors associated with weight changes in successful quitters participating in a smoking cessation program

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HIGHLIGHTS

- Smokers gain an average of 5 kg post 1 year of quitting cigarettes.
- Smokers with heavier dependence to cigarettes gain more weight.
- Heavier smokers should address weight management during smoking cessation.

ARTICLE INFO

Keywords:
Post-cessation
Smoking
Weight gain

ABSTRACT

Objective: To identify possible predictors of post-cessation weight gain in smoking abstainers.
Patients and methods: A sample of 607 successful abstainers seen at the Centre for Tobacco-Dependent in Prague, Czech Republic, between 2005 and 2010, was included in this analysis. This sample was followed up for 1 year and included 47.9% women (N = 291) with the mean age of 48 years (18–85).
Findings: Post-cessation weight gain occurred in 88.6% of the 607 abstainers. The mean weight gain after one year post-quit was 5.1 kg (95% confidence interval 4.7–5.5 kg). Baseline characteristics associated with increased weight gain included a higher baseline smoking rate ($p < 0.001$), more severe cigarette dependence ($p = 0.003$), less physical activity ($p = 0.008$), and a report of increased appetite on the baseline assessment of withdrawal symptoms ($p < 0.001$).
Conclusions: Smokers who are more dependent and have minimal physical activity are at increased risk for post-cessation weight gain. For these smokers, incorporating interventions targeting the weight issue into tobacco dependence treatment is recommended. Further research should be done to identify reasons for this important quitting complication.

1. Introduction

Although post-smoking cessation weight gain is well documented (Caan et al., 1996; Froom, Melamed, & Benbassat, 1998; John et al., 2005; Perkins, 1993) and occurs in about 84% of quitters (Aubin, Farley, Lycett, Lahmek, & Aveyard, 2012; Cairella et al., 2007; Klein, Corwin, & Ceballos, 2004), the exact mechanisms underlying this important health issue are not clearly understood. Several factors contribute to this increase in body fat, including insulin resistance (Chiolero, Faeh, Paccaud, & Cornuz, 2008); however, some of the increase may be related to changes in the reinforcing properties of foods due to their association with the nicotine in cigarettes (Grimm, Ratliff, North, Barnes, & Collins, 2012).

The average weight gain is 3 to 6 kg at one year post-quit (Aubin et al., 2012; Chatkin & Chatkin, 2007), develops most rapidly initially, and then the rate of gain slows (Aubin et al., 2012; O'Hara et al., 1998) with the tendency to stabilize over time (Reas, Nygard, & Sorensen, 2009), not increasing. Former smokers revert to a mean BMI roughly equivalent to that of never-smokers when they achieve long-term

Abbreviations: CO, carbon monoxide; FTCD, Fagerström Test of Cigarette Dependence; MNWS, Minnesota Withdrawal Scale; NRT, nicotine replacement therapy.

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http://dx.doi.org/10.1016/j.addbeh.2013.10.010

abstinence. This difference in BMI persists even after adjustment for energy and alcohol consumption, suggesting that the effects of smoking on BMI are not caused only by changes in dietary or alcohol consumption (Munafo, Tilling, & Ben-Shlomo, 2009).

Several factors associated with weight gain, including the absence of nicotine or other tobacco smoke substances, can increase energy intake (Chiolero, Jacot-Sadowski, Faeh, Paccaud, & Cornuz, 2007). Evidence also suggests that hunger increases urges to smoke (Cheskin, Hess, Henningfield, & Gorelick, 2005; Leeman, O'Malley, White, & McKee, 2010). Self-administration of palatable foods, especially sweets, increases after nicotine deprivation (Hughes, Gust, Skoog, Keenan, & Fenwick, 1991).

Although nicotine is the only one addictive substance in tobacco (Le Houezec, 2003), the effects of nicotine and its metabolites are based on metabolic, central, and gastrointestinal influence. Nicotine does not acutely reduce hunger and eating and does not have an anorectic action (Henningfield, London, & Pogun, 2009). On the other hand, nicotine reduces food consumption and increases metabolic rate (Henningfield et al., 2009).

Metabolic impact of nicotine includes a thermogenic effect via increased lipid oxidation (Yoshida, Yoshioka, Hiraoka, & Kondo, 1990) and sympathetic stimulation (Hofstetter, Schutz, Jequier, & Wahren, 1986) and thus stimulates the basal metabolism (Dallosso & James, 1984) with an energy expenditure increase of 5%–10% (Hofstetter et al., 1986). It has been described that smoking of one cigarette increases the energy expenditure by 3% in 30 min (Dallosso & James, 1984). This effect on basal metabolism seems to be lower in obese smokers (Audrain, Klesges, & Klesges, 1995), as the change in the resting energy expenditure of obese smokers was described to be on average 300 kJ (71 calories) lower than in non-obese ones (Audrain et al., 1995). In contrast, people who gain the most weight are more likely to succeed in quitting smoking (Hall, Tunstall, Vila, & Duffy, 1992).

All forms of nicotine are linked to dopamine and serotonin release which in turn leads to a decrease in appetite (Chatkin & Chatkin, 2007). Nicotine contributes to appetite decrease by binding to the β4 subunit of nicotinic acetylcholine receptors in hypothalamus nicotine which stimulates the MC-4 subunit of pro-opiomelanocortin neurons, resulting in decreased food intake (Mineur et al., 2011). This knowledge has contributed to a better understanding of nicotine's central influence leading to appetite suppression in smokers. Several studies have reported delayed gastric emptying of solids in smokers (Gritz et al., 1988; Miller, Palmer, Smith, Ferrington, & Merrick, 1989). Recent data show that gastric motility is accelerated after stopping smoking, and this acceleration may be involved in the temporary appetite increase and weight gain (Kadota et al., 2010).

There is evidence from a twin study that weight gain on cessation is mediated genetically (Aveyard, Lycett, & Farley, 2012). The role of gender differences however remains unclear in the current literature. While some studies have found greater weight gain in women when they stop smoking (Swan & Carmelli, 1995), others have not (Dale et al., 1998). Thus, there is contradictory data on whether women gain more weight. Even if weight gain is similar, this represents a greater percentage of weight gain in women (Froom et al., 1998).

An increase in appetite is an independent withdrawal symptom (Hughes & Hatsukami, 1986), and smokers with an increased appetite have been found to gain more weight when they stop smoking (John, Meyer, Rumpf, Hapke, & Schumann, 2006). In addition, as physical activity leads to energy expenditure and is one of the most important mechanisms to reduce weight, we assume that the baseline level of physical activity may be a possible predictor of post-cessation weight gain.

According to current knowledge, all first-line medications for smoking cessation seem to prevent weight gain while being used with differing degrees of effect (Farley, Hajek, Lycett, & Aveyard, 2012). Whereas no differences have been observed in the long term (Kawada, 2004), these findings are supported also by the *Cochrane Database of Systematic Reviews* paper published in 2012 (Farley et al., 2012). On the other hand, prolonged use of transdermal nicotine therapy may help to reduce weight gain in the short term. Extended treatment increased nicotine patch adherence which, in turn, reduced weight gain (Schnoll, Wileyto, & Lerman, 2012). Several options were tested regarding weight gain prevention. Some of these may be effective in the short term, e.g. the use of nicotine replacement therapy (NRT) or bupropion (Allen, Hatsukami, Brintnell, & Bade, 2005; Hurt et al., 1997; Levine et al., 2010) or inclusion of a behavioral weight control component within a tobacco treatment program (Spring et al., 2009). The only one strategy which seems to be effective in the long-term is physical activity (Farley et al., 2012).

Our aim is to identify possible predictors of post-cessation weight gain utilizing a sample of 607 biochemically confirmed smoking abstainers being treated, and followed up for 1 year, in the Czech Republic, which makes this sample unique as no other similar Czech data are available.

2. Methods

2.1. Treatment program

This study included patients treated at the Center for Tobacco-Dependent in Prague, Czech Republic, between 2005 and 2010. This Center used evidence-based procedures including pharmacotherapy and psychobehavioral interventions according to Czech and international treatment recommendations and guidelines (Fiore et al., 2008; Králíková et al., 2005) described on our web page at http://www.slzt.cz/intervention-structure. The treatment team consisted of nurses and medical doctors who were certified as Tobacco Treatment Specialists by the Mayo Clinic accredited program (accredited by the Council on Tobacco Treatment Training Programs) and its equivalent in the Czech Republic under the Society for Treatment of Tobacco Dependence (accredited by the Czech Medical Association). Because this treatment targeted the individual smoker, the treatment might differ in the type of medication used (varenicline; bupropion; NRT as patch, lozenge, inhaler and gum and/or their combination if needed) and the number of visits depending on the history, preferences, and compliance of patients, and according to patient comorbidities. The first-line medications included varenicline, bupropion, and nicotine. Whereas varenicline and bupropion were used in standard or reduced dosing (according to Fiore et al., 2008), NRT was recommended usually either as patch plus method (i.e. patch in combination with oral form of NRT — gum, inhaler, or lozenge) or in oral form only. Dosing of NRT was adjusted individually according to the presence of withdrawal symptoms during treatment. The type and dosage of medication selected were based on the therapist's recommendation (after a thorough assessment of the patient's history) and patient's choice. The initial visit, which included baseline assessments, was the same for all patients, regardless of the counselor assigned. The degree of tobacco dependence (Fagerström Test of Cigarette Dependence [FTCD] and Minnesota Withdrawal Scale [MNWS]) was evaluated, medical history was collected, and a basic physical exam was performed. Physical activity was assessed based on the following 4 categories: (1) Regular, at least 2–3× weekly 46–60 min or 3× weekly 20–30 min or more; (2) regular, weekly, but less frequent; (3) irregular, 1–2× monthly; and (4) no physical activity.

At the second visit, physical dependence and psycho-social tobacco dependence were discussed during the 2-hour intervention. Habits and rituals associated with tobacco use, alternative or surrogate means of resolving the problem or means of avoiding such situations were analyzed and pharmacotherapy was introduced. At the end of the second visit, the patient and therapist decided the subsequent course of treatment including pharmacotherapy type and dose. They also planned the target quit date and the date of the first follow-up visit. The second visit also contained brief weight management recommendation, as described by Fiore et al. (2008).