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Smoking status and abdominal obesity among normal- and overweight/obese adults: Population-based FINRISK study

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ABSTRACT

Several studies have reported direct associations of smoking with body mass index (BMI) and abdominal obesity. However, the interplay between them is poorly understood. Our first aim was to investigate the interaction between smoking status and BMI on abdominal obesity (waist circumference, WC). Our second aim was to examine how the association of smoking status with WC varies among normal and overweight/obese men and women. We examined 5817 participants from the National FINRISK 2007 Study. The interactions between smoking and BMI on WC were analyzed. Participants were categorized into eight groups according to BMI (normal weight vs. overweight/obese) and smoking status (never smoker, ex-smoker, occasional/light/moderate daily smoker, heavy daily smoker). The associations between each BMI/smoking status -group and WC were analyzed by multiple regressions, the normal-weight never smokers as the reference group. The smoking status by BMI-interaction on WC was significant for women, but not for men. Among the overweight/obese women, ex-smokers (β = 2.73; 1.99, 3.46) and heavy daily smokers (β = 4.90; 3.35, 6.44) had the highest estimates for WC when adjusted for age, BMI, alcohol consumption and physical activity. In comparison to never smoking overweight/obese women, the β-coefficients of ex-smokers and heavy daily smokers were significantly higher. Among men and normal weight women the β -coefficients did not significantly differ by smoking status. An interaction between smoking status and BMI on abdominal obesity was observed in women: overweight/obese heavy daily smokers were particularly vulnerable for abdominal obesity. This risk group should be targeted for cardiovascular disease prevention.

1. Background

Smoking and obesity constitute the leading causes of preventable death (Danaei et al., 2009, Stokes and Preston, 2016). Globally, tobacco use has killed 100 million people in the 20th century alone (Eriksen et al., 2015). Obese smokers live on average 13 years less than normal weight non-smokers (Peeters et al., 2003). Apart from a body mass index (BMI) 30 and above, abdominal obesity on its own is associated with higher mortality in all weight categories (Cerhan et al., 2014; Jacobs et al., 2010). Globally, approximately 30% of adult men and 6% of women are daily smokers (Ng et al., 2014), further 39% of the world's population are overweight (BMI ≥ 25 kg/m^2) and 13% are obese (BMI ≥ 30 kg/m^2) (WHO, 2015). It has been reported that 30% of daily smokers are overweight or obese (De Munter et al., 2015). Smoking alone or in combination with overweight or obesity poses the major public health burden in developed countries (Danaei et al., 2009; Eriksen et al., 2015).

Previous studies have reported on the relationship between smoking and weight. First, current daily smokers generally have lower BMIs than never smokers (Akbartabartoori et al., 2005; Audrain-Mcgovern and Benowitz, 2011; Berlin, 2008; Munafo et al., 2009; Pisinger et al., 2009; Sikorski et al., 2014; Sneve and Jorde, 2008). Second, during cessation, most of the quitters gain weight (Aubin et al., 2012; Filozof et al., 2004; Tian et al., 2015). Third, former smokers tend to be heavier than both never and current daily smokers (Akbartabartoori et al., 2005; Caks and Kos, 2009; Canoy et al., 2005).

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Fourth, some studies have reported that BMI among the smokers was positively associated with the number of cigarettes smoked per day (Bamia et al., 2004; Berlin, 2008; Chiolero et al., 2007; De Oliveira Fontes Gasperin et al., 2014; Pisinger et al., 2009) although other studies reviewed by Winslow et al. (2015) have found the opposite. However, many studies have shown that daily smokers weigh less than never smokers, but the body of knowledge about whether smokers have more abdominal obesity is controversial.

Specifically, current smokers have more abdominal obesity than never smokers (Berlin, 2008; Berlin et al., 2012; Morris et al., 2015; Pisinger and Jorgensen, 2007; Sikorski et al., 2014; Slagter et al., 2013; Yun et al., 2012) but other studies have not confirmed this association (Caks and Kos, 2009; De Oliveira Fontes Gasperin et al., 2014) or have even found the opposite (Clair et al., 2011; Lv et al., 2015; Onat et al., 2007; Onat et al., 2009; Sikorski et al., 2014). Yet other studies have reported that WC increases with increasing pack years among current daily smokers (Clair et al., 2011; Kim et al., 2012; Rom et al., 2015). The amount smoked daily by smokers have also been reported to be positively associated with abdominal obesity (Bamia et al., 2004; Barrett-Connor and Khaw, 1989; Clair et al., 2011). Moreover, a recent study that took a Mendelian randomization approach reported causal association. Morris et al. (2015) found that heavier smoking may lead to a relative increase in WC. Not only does current daily smoking increase the risk of elevated WC, former smokers also have more abdominal obesity compared with never smokers (Kwok et al., 2012; Lv et al., 2015; Sikorski et al., 2014; Yun et al., 2012), and also compared with current daily smokers (Akbartabartoori et al., 2005; Pisinger and Jorgensen, 2007). However, some studies have reported the opposite (Canoy et al., 2005; Yun et al., 2012). These contradictory findings show that the association still requires more attention.

It has been reported that smoking affects fat distribution in the abdominal area by various biological mechanisms such as the dysregulation of the hypothalamic-pituitary-adrenal axis (Audrain-Mcgovern and Benowitz, 2011; Rohleder and Kirschbaum, 2006). Another biological mechanism between smoking and increased abdominal obesity is that smoking affects the regulation of the sex hormones (Chiolero et al., 2008). For example, lower levels of androgens in male smokers and an imbalance in estrogens and androgens levels in female smokers have been found to increase abdominal obesity (Audrain-Mcgovern and Benowitz, 2011; Chiolero et al., 2008). Moreover, smoking may increase insulin resistance (Audrain-Mcgovern and Benowitz, 2011; Cena et al., 2011).

It is well established that overweight and general obesity as defined by BMI are the most important risk factors for abdominal obesity or elevated WC. However, previously published studies have reported inconsistent and even controversial findings about the association of smoking with abdominal obesity.

Considering prior knowledge about smokers' abdominal obesity, we hypothesized an interaction between smoking status and BMI on abdominal obesity (WC). We further hypothesized that normal and overweight/obese participants show different abdominal obesity according to their smoking status. The first aim of this study was to investigate the interaction between smoking and BMI on WC in a Finnish cross-sectional population-based sample. The second aim was to examine ways in which the association of smoking status with abdominal obesity varies among normal and overweight/obese men and women.

2. Methods

2.1. Data source and sample

The National FINRISK 2007 Study was a population-based health examination survey in Finland, which was used as the data source for this study. FINRISK 2007 Study has been described in detail elsewhere (Vartiainen et al., 2010). In brief, a stratified random sample was drawn from the Population Register comprising 9957 men and women aged 25 to 74 years. Members of the sample received an invitation to a health examination and also a questionnaire to complete. Of those invited, 6258 participated (62.9%). The participants had their height, weight, and WC measured by trained nurses according to an internationally accepted protocol to ensure an internationally comparable standard (Tolonen et al., 2008). Pregnant women ($n = 16$) were excluded from the present study. This study comprised 5817 participants (2738 men, 47%; 3079 women, 53%) all of whom provided complete information for any of the analyzed variables. The participants filled in a self-administered questionnaire that covered, for example, smoking habits, alcohol consumption, physical activity, and a history of non-communicable diseases. All procedures involving participants were approved by the Ethics Committee of Helsinki and Uusimaa Hospital District and in accordance with the Helsinki Declaration of 1975. The participants gave their written informed consent for health examination procedures. The study protocol followed the recommendations of the World Health Organization Multinational monitoring of trends and determinants in cardiovascular disease (Borodulin et al., 2015).

2.2. Variables

We classified the participants of this study according to their smoking status and whether they were either normal weight or overweight/obese on the basis of the BMI cut-off point of 25. Participants were classified into four categories according to their smoking status as follows: 1) never smokers, 2) ex-smokers (those who had quit smoking at least one month ago), 3) occasional smokers and light/moderate daily smokers, and 4) heavy daily smokers. Smoking classification was ascertained by asking the following questions in the questionnaire: 1) "Have you ever smoked?" 2) "Have you ever smoked at least 100 cigarettes during your lifetime?" 3) "Do you smoke currently?" 4) "Have you ever smoked regularly (in at least one year period)?" 5) "When was the last time you smoked?" In brief, those who answered "no" to questions 1 and 2 were classified as never smokers. Those who answered "yes" for 1 and 2 and "no" for question 3 were classified as ex-smokers. Those who answered "no" for question 4 were classified as occasional smokers. Finally, those who answered "yes" for questions 1, 2, 3, and 4 were classified as current daily smokers. Daily smokers were divided into light/moderate and heavy smokers according to their response to the question "How many manufactured cigarettes or hand-rolled cigarettes do you smoke on average in a day?" Those who smoked 19 or fewer cigarettes per day (CPD) were classified as light/moderate smokers and those who smoked 20 or more CPD were classified as heavy smokers. The same classification light/moderate versus heavy smokers has been used elsewhere before (Rasouli et al., 2013).

Anthropometric measures were taken and recorded by a trained study nurse using international protocols (Tolonen et al., 2008). All anthropometric measures were assessed with the subjects wearing light clothing and bare footed. The measurement of weight was rounded-off to the nearest 0.1 kg and height rounded-off to the nearest 0.1 cm. BMI was calculated as the weight in kilograms divided by the squared height in meters (kg/m^2). The participants were divided into two groups according to their BMI values, (1) normal weight (BMI < 25 kg/m^2) and (2) overweight or obese (BMI ≥ 25 kg/m^2) (Prentice and Jebb, 2001).

Abdominal obesity was defined using measures of WC in centimeters. The participant's WC was measured midway between the lower rib margin and the iliac crest (Tolonen et al., 2008). The WC was used as a continuous variable and it was rounded-off to the nearest 0.5 cm.

Based on the earlier literature, the following variables were included as the covariates in the analyses: age, BMI, alcohol consumption and physical activity and were added to the models using a stepwise procedure (Dvorak et al., 2009; Morris et al., 2015; Oh and Seo, 2001; Shimokata et al., 1989). The level of alcohol consumption was assessed as the self-reported use of alcohol products in the previous week in ethanol grams and used as a continuous logarithmically transformed

variable. The level of physical activity was assessed using self-reports on leisure-time activities, commuting and occupational physical activity that were combined to create a physical activity index and used as a continuous variable (Borodulin et al., 2016).

2.3. Statistical analyses

For the descriptive comparison of the characteristics among the study participants analyses of variance (ANOVA) were conducted by BMI (normal vs. overweight/obese) and by smoking status (never smokers, ex-smokers, occasional smokers and light/moderate daily smokers, and heavy daily smokers). The Pearson's correlations between the variables were examined. The statistical analyses were carried out using Stata version 13.1 for Windows (StataCorp., 2013).

Because of the biological and the pathophysiological differences in fat accumulation between sexes, we decided to analyze men and women separately (Incollingo Rodriguez et al., 2015, Onat et al., 2010). We used regression analysis to estimate β coefficients to illustrate the strength of the association between smoking and WC.

We categorized each sex into eight groups according to their BMI (normal weight vs. overweight/obese) and smoking habits (four classes) for statistical analyses to assess the possible differences as precisely as possible in our data. Never smoking normal weight participants were used as the reference group in all of the analyses. However, in post hoc tests examining whether there was difference in β coefficients between different smoking categories within the overweight/obese group, the overweight/obese never smokers were used as the reference group. Statistical significance was set at $p < 0.05$ values, except for the interaction testing for which statistical significance was set at $p < 0.10$.

3. Results

3.1. Sample description

The descriptive results are shown in Table 1 for men and Table 2 for women. Among the participants 2738 were men (47%) and 3079 women (53%). Approximately two thirds (71%) of men and over one half (56%) of women were overweight/obese. Over one third (36%) of the male participants were never smokers, and the corresponding proportion was 57% for women. The correlations between the continuous variables are displayed in the Supplementary material (Table S1 for men and Table S2 for women).

Among the normal weight participants, 12% of men and 25% of women were never smokers whereas 3.3% of men and 1.3% in women were heavy daily smokers. Furthermore, among the overweight/obese participants, 24% of men and 32% of women were never smokers whereas 6.7% of men and 1.7% of women were heavy daily smokers. The WC means were quite similar among normal weight smoking groups (85–88 cm for men and 77–78 cm for women). However, the differences in WC were larger in overweight/obese smoking groups, particularly between never and current daily smokers (101–105 cm for men and 96–101 cm for women). Occasional and light/moderate daily smokers were the youngest and ex-smokers the oldest among overweight/obese men. Further, occasional and light/moderate daily smokers were the youngest and never smokers the oldest for the overweight/obese women participants. The heavy daily smokers had highest mean alcohol consumption levels and were the least physically active in both men and women.

3.2. Association between smoking, BMI, and WC

We tested smoking status by BMI interactions on WC (among men LR $\chi^2 = 1.19$, $p = 0.27$, and among women LR $\chi^2 = 6.85$, $p = 0.009$). The interaction between smoking status and BMI was significant among women only, i.e. BMI modifies the association between smoking status and WC in women but not in men. For consistency, the same categorization is applied for men even though they did not have statistically significant smoking by BMI interaction. The results of all regression models for the associations of the smoking-BMI-group with the WC are shown in Table 3 for men and in Table 4 for women.

The heavy daily smoker overweight/obese males had the highest and the ex-smoker males had the second highest estimate for the WC ($\beta = 17.9$, $p = 1.6 \times 10^{-96}$ and $\beta = 17.0$, $p = 7.0 \times 10^{-157}$) in the age adjusted model when compared with the normal weight never smokers which means that daily smokers had approximately 18 cm and ex-smokers 17 cm larger waist circumferences compared with normal weight never smokers. The estimate decreased to $\beta = 2.03$ ($p = 1.1 \times 10^{-5}$) in heavy daily smokers and to $\beta = 1.52$ ($p = 1.9 \times 10^{-5}$) in ex-smokers when BMI was added to the model, and decreased further to $\beta = 1.72$ ($p = 1.6 \times 10^{-4}$) for heavy daily smokers and to $\beta = 1.51$ ($p = 1.6 \times 10^{-5}$) for ex-smokers when adjustment for alcohol consumption and physical activity was made in the final model (Table 3). However, as a post hoc test, we determined if there were differences between the smoking status groups within the overweight/obese group. These pair-wise difference tests revealed that for the male sample the respective coefficients did not differ statistically significantly. All the pair-wise difference test results for men are found in the Supplementary material Table S3.

Among the overweight/obese female participants ex-smokers and heavy daily smokers had the highest estimates for WC ($\beta = 19.7$, $p = 5.0 \times 10^{-214}$ for ex-smokers and $\beta = 24.4$, $p = 4.1 \times 10^{-66}$ for heavy daily smokers) when compared with normal weight never smokers in the age adjusted model. When BMI was added as a covariate to the model the estimates decreased for heavy daily smokers to $\beta = 4.90$ ($p = 6.1 \times 10^{-10}$) and for ex-smokers to $\beta = 2.73$ ($p = 3.9 \times 10^{-13}$) (Table 4). The pair-wise differences in all smoking status estimates within the overweight/obese participants (never smokers as the reference category) were significant or close to significant. In comparison

Table 1
Sample characteristics (means and standard deviations) by body mass index (BMI) and smoking-status in 2738 Finnish men. Based on FINRISK2007 data.

Smoking status	N (%)	Age (years)	BMI (kg/m^2)	Waist circumference (cm)	Alcohol consumption (g/week)	Physical activity (grade 1–4)
		Mean (SD)				
Normal weight (BMI < 25)						
Never smokers	340 (12)	46.3 (14)	23.1 (1.3)	85.9 (5.9)	54.3 (74)	3.38 (0.8)
Ex-smokers	208 (7.6)	52.1 (14)	23.4 (1.4)	87.6 (6.0)	64.5 (84)	3.39 (0.7)
Occasional smokers and daily light/moderate smokers	166 (6.1)	43.0 (14)	23.0 (1.7)	85.7 (6.4)	116 (170)	3.16 (0.9)
Daily heavy smokers	91 (3.3)	44.9 (10)	22.6 (1.8)	85.6 (7.1)	164 (180)	2.97 (0.9)
Overweight/obese (BMI ≥ 25)						
Never smokers	652 (24)	52.1 (13)	28.5 (3.2)	101 (9.5)	79.3 (100)	3.22 (0.8)
Ex-smokers	777 (28)	56.6 (12)	29.6 (3.8)	105 (11)	89.2 (120)	3.06 (0.9)
Occasional smokers & daily light/moderate smokers	322 (12)	47.8 (14)	29.1 (3.3)	102 (9.9)	118 (140)	3.16 (0.8)
Daily heavy smokers	182 (6.7)	49.6 (12)	29.7 (4.5)	104 (11)	141 (170)	2.88 (0.9)

Differences in characteristics between all the smoking groups were determined using one-way ANOVA. *p*-Values for all different ANOVA analyses were <0.001.

Table 2
Sample characteristics (means and standard deviations) by body mass index (BMI) and smoking-status in 3079 Finnish women. Based on FINRISK2007 data.

Smoking status	N (%)	Age (years)	BMI (kg/m^2)	Waist circumference (cm)	Alcohol consumption (g/week)	Physical activity (grade 1–4)
		Mean (SD)				
Normal weight (BMI < 25)						
Never smokers	756 (25)	46.4 (14)	22.4 (1.7)	76.6 (6.0)	30.2 (54)	3.29 (0.7)
Ex-smokers	305 (9.9)	46.7 (13)	22.6 (1.7)	77.5 (5.7)	40.4 (46)	3.30 (0.8)
Occasional smokers and daily light/moderate smokers	250 (8.1)	42.1 (13)	22.2 (1.8)	76.9 (5.7)	66.5 (110)	3.11 (0.8)
Daily heavy smokers	40 (1.3)	43.8 (10)	21.9 (2.1)	76.7 (6.9)	77.8 (120)	2.98 (0.9)
Overweight/obese (BMI ≥ 25)						
Never smokers	986 (32)	56.5 (13)	30.2 (4.6)	96.0 (11)	24.0 (42)	2.99 (0.8)
Ex-smokers	431 (14)	51.4 (12)	30.3 (5.1)	96.7 (12)	42.5 (77)	3.01 (0.8)
Occasional smokers & daily light/moderate smokers	260 (8.4)	47.1 (12)	29.9 (4.4)	95.5 (12)	53.9 (71)	2.93 (0.9)
Daily heavy smokers	51 (1.7)	50.8 (13)	31.5 (5.2)	101 (14)	101 (160)	2.47 (1.0)

Differences in characteristics between all the smoking groups were determined using one-way ANOVA. *p*-Values for all different ANOVA analyses were <0.001.

to never smoking overweight/obese women, the *p*-values of the pairwise tests were 0.02 for ex-smokers, 0.07 for occasional/light/moderate daily smokers, and 0.001 for heavy daily smokers. All test results are shown in the Supplementary Table S3.

Finally, among the normal weight participants no significant differences in WC between the smoking groups were found either in men or in women.

4. Discussion

In the present study, we found an interaction between smoking status and BMI on abdominal obesity in women. We are not aware of any studies that have reported the interaction of smoking habits and BMI on WC prior to this study.

Our sample consisted of 5817 Finnish adults drawn from the population based National FINRISK 2007 Study, where the participants were categorized into four smoking groups. Notably, among overweight/obese persons, heavy daily smoking was associated with the highest abdominal obesity, yet significantly only in women. Although not testing that interaction, some earlier studies have reported similar associations in their analyses stratified by BMI-status (Canoy et al., 2005; Lv et al., 2015; Slagter et al., 2013) but inconsistently (Kim et al., 2012). It is possible that such inconsistency is partly due to lack of taking weight status into account in a sufficient manner. In our study, we both stratified by BMI category and adjusted for linear BMI within each group.

Earlier studies have suggested that the association between heavy daily smoking and abdominal obesity is partly explained by a clustering of unhealthy lifestyle habits (Canoy et al., 2005; Chiolero et al., 2008; Kwok et al., 2012; Pisinger et al., 2009). Unhealthy habits tend to cluster among the same people (Schuit et al., 2002), which was the case also for our participants. Alcohol consumption was higher among smokers for both sexes, and for women, physical activity was lower among more frequent users of cigarettes.

Further, former smoking was associated with increased WC when compared with never smokers. Previous studies have consistently shown similar results (Kwok et al., 2012; Lv et al., 2015; Sikorski et al., 2014; Yun et al., 2012). The higher risk of abdominal obesity among ex-smokers can mostly be explained by the post-cessation weight gain (Pisinger and Jorgensen, 2007; Tian et al., 2015). However, we hypothesize that a minor part of this association may also be explained by the accumulated life-time exposure to tobacco or epigenetic changes caused by tobacco exposure (Breitling, 2013).

No significant association emerged between smoking status and abdominal obesity among normal weight participants in our study. Previous studies have reported normal weight smokers also to have a higher risk for abdominal obesity (Kim et al., 2012; Lv et al., 2015; Morris et al., 2015; Slagter et al., 2013). The fact that we could not replicate earlier

Table 3
Results of the multiple regression models for the associations of BMI-smoking-status with waist circumference in men: β coefficients with confidence intervals (95% CI) and *p*-values. Normal weight never smokers as the reference group in all the regression models. Based on FINRISK2007 data ($n = 2738$).

	1st model[a]	2nd model[b]	Final model[c]	1st model[a]	2nd model[b]	Final model[c]
	Normal weight (BMI < 25)			Overweight/obese (BMI ≥ 25)		
Never smokers	Reference group					
β				14.1	1.21	1.30
CI (95%)				(13.0, 15.3)	(0.54, 1.89)	(0.63, 1.96)
p-Value				5.0×10^{-111}	4.5×10^{-4}	1.3×10^{-4}
Ex-smokers						
β	0.82	0.42	0.47	17.0	1.52	1.51
CI (95 %)	(−0.73, 2.37)	(−0.38, 1.22)	(−0.31, 1.25)	(15.9, 18.2)	(0.83, 2.22)	(0.82, 2.19)
p-Value	0.30	0.31	0.24	7.0×10^{-157}	1.9×10^{-5}	1.6×10^{-5}
Occasional and light/moderate daily smokers						
β	0.41	0.75	0.37	16.2	1.83	1.76
CI (95%)	(−1.25, 2.07)	(−0.11, 1.61)	(−0.47, 1.21)	(14.9, 17.6)	(1.06, 2.60)	(1.00, 2.53)
p-Value	0.63	8.4×10^{-2}	0.39	6.0×10^{-110}	3.7×10^{-6}	6.3×10^{-6}
Heavy daily smokers						
β	0.01	1.19	0.55	17.9	2.03	1.72
CI (95%)	(−2.06, 2.08)	(0.13, 2.26)	(−0.51, 1.60)	(16.2, 19.5)	(1.13, 2.94)	(0.83, 2.61)
p-Value	1.0	2.8×10^{-2}	0.31	1.6×10^{-96}	1.1×10^{-5}	1.6×10^{-4}

Model 1 adjusted for age.
Model 2 adjusted for age and body mass index (BMI).
Final model adjusted for age, BMI, alcohol consumption and physical activity index.
Adjusted R-squared: a: 44.6%, b: 85.3% c: 85.9%.

Table 4
Results of the multiple regression models for the associations of BMI-smoking-status with waist circumference in women: β coefficients with confidence intervals (95% CI) and *p*-values. Normal weight never smokers as the reference group in all the regression models. Based on FINRISK2007 data ($n = 3079$).

	1st model[a]	2nd model[b]	Final model[c]	1st model[a]	2nd model[b]	Final model[c]
	Normal weight (BMI < 25)			Overweight/obese (BMI ≥ 25)		
Never smokers	Reference group					
β				18.4	1.97	2.00
CI (95%)				(17.5, 19.4)	(1.33, 2.61)	(1.36, 3.43)
p-Value				7.0×10^{-263}	1.7×10^{-9}	8.8×10^{-10}
Ex-smokers						
β	0.90	0.58	0.55	19.7	2.73	2.70
CI (95%)	(−0.37, 2.17)	(−0.11, 1.28)	(−0.14, 1.25)	(18.5, 20.8)	(1.99, 3.46)	(1.97, 3.43)
p-Value	0.16	9.9×10^{-2}	0.12	5.0×10^{-214}	3.9×10^{-13}	5.8×10^{-13}
Occasional and light/moderate daily smokers						
β	0.68	0.96	0.74	18.9	2.81	2.67
CI (95%)	(−0.69, 2.06)	(0.21, 1.71)	(−0.01, 1.50)	(17.5, 20.2)	(1.98, 3.63)	(1.85, 3.50)
p-Value	0.33	1.2×10^{-2}	0.53	8.0×10^{-149}	2.7×10^{-11}	2.2×10^{-10}
Heavy daily smokers						
β	0.38	1.21	0.93	24.4	4.90	4.48
CI (95%)	(−2.66, 3.42)	(−0.45, 2.87)	(−0.72, 2.59)	(21.7, 27.1)	(3.35, 6.44)	(2.93, 6.03)
p-Value	0.81	0.15	0.27	4.1×10^{-66}	6.1×10^{-10}	1.5×10^{-8}

Model 1 adjusted for age.
Model 2 adjusted for age and body mass index (BMI).
Final model adjusted for age, BMI, alcohol consumption and physical activity index.
Adjusted R-squared: a: 50.8%, b: 85.4% c: 85.5%.

findings about fat deposition in normal weight participants might be due to the relatively small sample size of normal weight participants in some of our smoking-BMI-groups. Moreover, there is differential hypothalamic-pituitary-adrenal (HPA) axis activity in overweight people, whereas in normal weight people the function of HPA axis is normal (Audrain-Mcgovern and Benowitz, 2011; Incollingo Rodriguez et al., 2015; Rohleder and Kirschbaum, 2006). Among the overweight people, the HPA axis has greater responsivity and moreover, there is an established upregulation of cortisol output in adipocytes whereas in hepatic tissue the cortisol is downregulated. In addition, smoking affects the regulation of the sex hormones (Audrain-Mcgovern and Benowitz, 2011; Chiolero et al., 2008). These changes are associated with higher abdominal fat.

Furthermore, many previous studies have shown that not only daily heavy smoking, but also occasional/daily light smoking elevates the WC (Clair et al., 2011; De Oliveira Fontes Gasperin et al., 2014; Kim et al., 2012, Rom et al., 2015; Slagter et al., 2013). Our results are in line with these results but only in overweight/obese female participants.

Finally, no significant association was found between smoking and abdominal obesity for male participants when stratified by BMI status. However, we may have found some associations if we had pooled men together across BMI status. Because there was no significant smoking by BMI interaction on WC, such pooling would have been justified. However, for consistency, we analyzed the data stratified by BMI also in men. We would like to acknowledge that in several previous reports (Berlin, 2008; Berlin et al., 2012; Morris et al., 2015; Pisinger and Jorgensen, 2007; Shi et al., 2013; Slagter et al., 2013; Yun et al., 2012) smoking was associated with higher WC among males, when not stratified by BMI status. Previously it has been reported that, non-smoking men in the highest sixth of the distribution of abdominal fat as measured by WC had a mean life expectancy that was three years shorter than that of men in the lowest sixth of WC (Cerhan et al., 2014). The corresponding difference in the equivalent high and low categories was five years among women. Further, obese smokers live on average 13 years less than normal weight non-smokers (Peeters et al., 2003).

4.1. Strengths and limitations

The main strength of this study is that it is based on a large population based sample, which enabled us to categorize the participants into four different groups according to their smoking status; 1) never smokers, 2) ex-smokers, 3) occasional and light/moderate daily smokers and 4) heavy daily smokers. This allowed us to take into account the association of smoking with WC in a more dose-dependent manner. This large sample allowed us also to stratify our participants by the BMI status, although we had a relative small number of participants in some of the BMI-smoking-groups. Another strength is that weight, height and WC were measured by a trained study nurse. Furthermore, the sample size was quite large and population-based, and thus the results of our study are well generalized for a Caucasian adult population.

Nonetheless, we recognize the inherent limitations of cross-sectional studies and therefore no conclusions about causality can be drawn from our results. In addition, participants voluntarily participated in the FINRISK Study, which might include bias that results from self-selection. It is well acknowledged that it is more likely for never smokers than for smokers to participate in health-related surveys (Christensen et al., 2015). This limitation may be reflected in our study because there were relatively small numbers of participants in some of the BMI-smoking-groups, even though this was a large random population-based sample. In the whole FINRISK 2007 sample, 28% of men and 24% of women were overall smokers (occasional and daily smokers combined) (Peltonen et al., 2008). The portion of overall smokers was quite similar in our sample, 28% for men and 20% for women. Furthermore, we acknowledge that our risk group covers relative small amount of our sample. Thus, among our participants, in men, 6.7% were overweight/obese daily heavy smokers and respectively, 1.7% in women. We were unable to verify the smoking status of all participants because the cotinine concentration values were measured only among a sub-sample. This is unfortunate, since cotinine has an established role as a reliable biomarker of tobacco use (Connor Gorber et al., 2009). However, among the sub-sample, the association between self-reported smoking habits and cotinine levels has been found to be at acceptable level as has been shown in earlier separate FINRISK studies (Kaprio et al., 2014; Vartiainen et al., 2002). Finally, although we adjusted for several known covariates, we cannot completely rule out all of the possibilities of some residual confounding.

5. Conclusions

We found that current and former smoking is associated with a higher WC among overweight/obese women, particularly among heavy daily smokers. Excess risk for abdominal obesity due to heavy

daily smoking among overweight/obese people should be taken into account in clinical practice and when targeting prevention for persons at high risk for diabetes and cardiovascular diseases. In community-based prevention programs co-occurring smoking and overweight/obesity should be addressed. The interventions for smoking cessation should also focus on minimizing post-cessation weight gain. The highest risk group (9% of the sample) should be noted carefully for cardiovascular disease and type 2 Diabetes prevention by regular health checks in primary health care. The complex associations between smoking and abdominal obesity need to be examined in longitudinal and biochemically verified settings in the future.

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.pmedr.2016.07.003.

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Declaration of interests

JK and TK have consulted for Pfizer Finland on nicotine dependence from 2012 to 2015. THK has consulted Pfizer (Global and Finland 2012–2014). E-L T, SES, KP, KB and SM have none to declare.

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