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Title: Association between lifestyle features and obesity phenotypes in adults from the Basque Country (Spain)

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Abstract

Lifestyle is related to the risk of obesity, but whether its association varies with different obesity phenotypes remains unclear. The aim of this study was to analyse the association between different lifestyle features (eating habits, activity, sleep patterns, and tobacco and alcohol consumption), and four obesity phenotypes (overall and abdominal obesity, distribution and percentage of fat). The sample included 521 adults aged between 18 and 70 years, residents in the Basque Country (Spain). A Multiple Logistic Regression Model was used, controlling for sex, age and SES. The duration of the main meal was inversely associated with overall obesity (p=0.010) and abdominal one (p=0.004), while the number of meals (<3 times day) was positively associated with both phenotypes (p=0.019 and p=0.031, respectively). Regular sport practice and duration (>6 years) were negatively associated with all obesity phenotypes (p-value always <0.01), while watching television was positively associated with all of them (p-values between 0.016 and <0.01). Walking was inversely related to overall and abdominal obesity (p<0.01). Sleep quality was positively associated with overall (p<0.01) and abdominal obesity (p <0.05), but not with fat distribution or its percentage. Former smokers showed a positive association with both abdominal obesity (p=0.021) and fat mass distribution (p=0.002), and the number of cigarettes were positively elated with all obesity phenotypes (p<0.01), except with fat distribution. Alcohol consumption was inversely related with excessive adiposity (p=0.030), while occasional drinking was negatively related with overall obesity and an excess of fat (p =0.050 and p=0.022, respectively). In conclusion, in the sample studied, few meals per day, a bad or regular quality of sleep, many hours spent watching television (a sedentary behaviour), and a heavy cigarette consumption increased the risk of various obesity phenotypes, while time spent at the main meal, walking and sport practice, as well as a moderate alcohol consumption were associated with a decreased in such risk.

Introduction

Obesity is defined as a complex condition characterised by an excessive increase in body fat – and consequent weight gain – due to a positive energy balance maintained over time, which increases the risk of morbidity for various chronic diseases (e.g. alterations of the metabolic profile, cardiovascular disease and some cancers) and mortality (WHO, 2000; Bray, 2004; Hruby & Hu, 2015; Bray *et al.,* 2017). Its impact on health depends not only on fat accumulation but also on its anatomical distribution (Frank *et al.,* 2019), so that central obesity increases metabolic risk related to fat storage. The definition of obesity is apparently simple but its aetiology is multifactorial in nature due to the contribution of genetic, epigenetic, physiological, behavioural, socio-cultural and

environmental factors (Bray *et al.,* 2016). Some of them, such as genetic factors, condition individual susceptibility to obesity but do not fully explain its prevalence at the population level; it is therefore important to understand the different factors involved, especially those that can be modified and their differential contribution to the development of obesity in each population. Obesity is also a heterogeneous phenotype, the determination of which varies according to the cut-off points of different measurement techniques; in the case of anthropometric variables such as body mass index (BMI, kg/m^2) or waist (W) circumference, they depend in turn on age, sex and ethnicity (Deurenberg, 2001; Cole *et al.,* 2010; Materko *et al.,* 2017).

Diet and physical activity are important "modifiable factors" related to lifestyle (Hill *et al.,* 2003; Philippou & Andreou, 2022); both are influenced by the "obesogenic environment" (Swinburn *et al.,* 1999) which encourages unhealthy dietary behaviours and increased sedentary lifestyles, and thus weight gain (Giles-Corti *et al.,* 2003). Although the energy expenditure associated with daily life has decreased since the successive industrial revolutions, the relationship between diet, physical activity and obesity is complex and remains a subject of research. As noted by Giles-Corti *et al.* (2003), food intake has decreased in some countries, such as the UK, suggesting that the increase in overweight and obesity may be due to a decrease in energy expenditure (sedentary lifestyles). While in others countries (e.g. USA), energy intake has increased in recent decades, and may be the main cause of weight gain in the population. The role of dietary habits and physical activity, but also of sleep patterns and tobacco and alcohol consumption, has been addressed with respect to their association with obesity and increased morbidity and mortality in numerous research studies (e.g. Mayer-Davies & Costacou, 2001; Bigaard *et al.,* 2003; Valdés-Badilla *et al.,* 2018; Gazdzinska *et al.,* 2022). Clinical and epidemiological studies show that systematic intervention on these factors reduces weight and cardiovascular and metabolic risk in overweight and obese individuals (Mozaffarian *et al.,* 2011; Jensen *et al.,* 2014). However, some studies indicate that tobacco and/or alcohol consumption may act on weight loss (Yeomans, 2010; Kase *et al.,* 2016; Murphy *et al.,* 2018), so their influence on the development of obesity is still controversial.

Objective

The aim of the present study was to determine the degree of association of various lifestyle features, such as eating habits, physical activity, sleep quality, and tobacco and alcohol consumption, with different obesity phenotypes (overall and abdominal obesity, fat mass distribution and percentage of fat, i.e., adiposity) in a sample of adult population of both sexes, living in the Basque Country (Spain).

Methods

Sample

A total of 626 individuals aged between 18 and 70 years, resident in the Basque Country (Spain), were studied. We obtained demographic data (sex, age, origin and place of residence), and established socioeconomic status (SES) based on educational and professional levels, using a standardised questionnaire (Ibáñez Pérez-Zamacona, 2017). Based on the indicators, the sample can be considered middle class. Persons of non-European origin, related, pregnant and with previous growth problems were excluded from the final data processing, so the final sample was 521 individuals (318 females and 203 males). Both, individuals with and without obesity were well represented (case-control study design). The study was accomplished in accordance with Helsinki Declaration; a written informed consent was obtained from all participants, and the study protocols were approved by the Ethics Committee for Human Research of the University of the Basque Country (UPV/EHU).

Anthropometric variables, obesity phenotypes and lifestyle

Height (cm), weight (kg), waist (W) and hip (H) circumferences, and four skinfolds (biceps, triceps, subscapular and suprailiac) were measured by trained personnel (M.E.I.). Four obesity phenotypes were considered: I. Overall obesity (BMI=(kg/m^2)), II. Abdominal obesity (W), III. Fat mass distribution (WHR=W/H), and IV. Adiposity (FM%). FM% was calculated by using Siri's (1961) equation based on body density (D) obtained from the general equation of Durnin & Womersley (1974), taken into account sex and age. For the first three, the cut-off points were those established by the WHO (2000, 2011), and for FM%, the criteria of the Spanish Society for the Study of Obesity (Campillo *et al.,* 2000), were taken into account. Based on these cut-off points, the phenotypes were dichotomised for statistical treatment (Box 1). Fourteen lifestyle variables were collected by means of personal interview (M.E.I.), which were grouped into four items with different number of variables each. Box 2 details the four items considered and the specific meaning of the different variables included in each one, as well as their categories and/or units.

Data analysis

Mean and standard deviation were used to describe continuous variables and percentage (%) for categorical variables, considering sex and two age groups (<45 years and ≥45

years (descriptive data can be found in Ibáñez Pérez-Zamacona, 2017). Data were tested for normality and homoscedasticity using the Kolmogorov-Smirnov test ($n>50$), or the Shapiro-Wilk test ($n\leq50$) and the Levene test. A Multiple Logistic Regression model was used to study the association between the 14 lifestyle variables (independent variables) and the different obesity phenotypes (dependent variables); sex, decimal age and SES were introduced as covariates to control for possible confounding effects. The consideration of SES is justified by its influence on the development of obesity (e.g. Dinsa *et al.,* 2012; Spinosa *et al.,* 2019; Adams, 2020); regarding sex and age its relationship with obesity is clearly established (e.g. Garawi *et al.,* 2014; Cooper *et al.,* 2021). The strength of the association between pairs of variables was expressed in terms of Odds Ratios (OR).

Two quantitative variables (sleep duration and main meal duration) were categorised for statistical analyses, as a homogeneous response of obesity could not be assumed across all the range of these variables. Bonferroni correction for multiple comparisons was calculated so that, taking into account the 14 variables analysed and the different obesity phenotypes, the critical "p" value would be $\alpha=0.05/68=0.0007$; however, the significance threshold was set at $p<0.05$ due to the sample size and the conviction that the Bonferroni correction is too conservative.

Results

Table 1 shows the number and percentage of individuals with and without obesity (BMI based) by sex and age group. The mean percentage of obesity in the whole sample studied was 42.4% (44.7% in women and 38.9% in men), of which 12.9% of women and 11.3% of men were morbidly obese ($BMI\geq40$ kg/m^2). Tables 2 to 5 show the results of the logistic regressions adjusted for sex, age and SES. Regarding eating habits (table 2), the duration of the main meal was inversely associated with both overall obesity (OR: 0.54; p-value=0.010) and abdominal obesity (OR: 0.50; p-value=0.004), but not with the FM% or fat distribution. Eating in the company canteen was inversely related to a central fat distribution pattern (OR: 0.45, p-value=0.041). People who eat less than three meals per day were more likely to have overall and abdominal obesity than those who eat more meals daily.

Concerning physical activity (table 3), regular sports practice and more than 6 years of such practice were negatively associated with all obesity phenotypes, while walking was inversely related to overall and abdominal obesity (OR=0.33 in both phenotypes and p-values <0.001 and 0.010, respectively). These phenotypes also showed negative associations with walking (as means of exercise) or sports practice since 3-6 years ago

(OR: 0.18 and 0.15, respectively, with p-values <0.001). Work intensity and daily walking were associated with the different obesity phenotypes, except with the central fat pattern, which showed a weak but significant positive association with daily walking (OR=1.01, p-value=0.016). Time spent watching TV was positively associated with all phenotypes considered.

Table 4 shows the results of multiple logistic regressions between sleep patterns (sleep duration and sleep quality) and obesity phenotypes. Only sleep quality showed associations with some obesity phenotypes, in particular with overall and abdominal obesity; bad and/or regular sleep quality increased the probability of both types of obesity between 1.78 and 2.73 times. Regarding table 5, only former smokers were more likely to develop abdominal obesity than non-smokers (OR: 1.88; p-value=0.021). Former and current smokers also showed a positive association with central fat distribution (OR: 2.46; p-value=0.002 and 1.99; p-value=0.047, respectively). The number of cigarettes was related to the studied obesity phenotypes (OR ranging between 1.04 and 1.06), with the exception of central fat distribution. Alcohol consumption was inversely associated with excessive adiposity (OR: 0.52, p-value=0.030), while occasional drinking was negatively associated with overall obesity and an excess of fat (OR: 0.51, p-value =0.050 and OR: 0.43, p-value=0.022, respectively).

Discussion

As might be expected from the study design, the analysed sample showed a high prevalence of obesity, especially in women, which exceeds the average (20%) of the general Spanish population (Pérez-Rodrigo *et al.,* 2020), and also that of the Basque Country where obesity in adults is 13.5% (Ministry of Health 2020). Other studies (e.g. Martínez-Larrad *et al.,* 2014) show somewhat higher frequencies for Spain (27.5%), with 30.2% for women and 23.7% for men; this higher frequency of obesity in women compared to men coincides with the obtained results.

Along with changes in the content and quantity of dietary intake, some dietary habits have changed over the last years, such as the time spent eating and the frequency of eating out (Bertéus-Forslund *et al.,* 2002). In this regard, the present results indicated that individuals who spent more than 20 minutes at the main meal were less likely to develop obesity and/or abdominal obesity than those who spent less time (≤20 minutes). It seems reasonable to think that the longer the time spent eating, the greater the amount of food eaten. However, energy intake is regulated by circulating factors and gut hormones which play an important role in controlling hunger and satiety (Angelopoulos *et al.,* 2014). So, according to Andrade *et al.* (2008), when meal duration is prolonged,

physiological signals have more time to express themselves, so that, regardless of the amount of food eaten, people who spend more time eating eat fewer calories, which could support the present results.

Currently, the number of people entering the labour market has increased, and eating away from home is increasingly common (Lin *et al.,* 1999): in Spain, 3 out of 4 people eat one of the three regular meals away from home every day (Blesa Jarque *et al.,* 2021). Some authors have observed a positive association between the number of meals eaten outside the home (especially in cafeterias and restaurants) and obesity, due to excessive portions and energy-dense foods that promote weight gain (Guthrie *et al.,* 2002; Bes-Rastrollo *et al.,* 2010), while others have found no relationship (Marín-Guerrero *et al.,* 2008). One possible explanation for these discrepancies is that some studies did not take into account the location or type of food consumed outside the home. In the present research, the location of the main meal did not show significant associations with the various obesity phenotypes, except when the main meal was eaten in the company canteen, this being a "protective" variable for fat distribution (less centralised). It is important to take into account that this study has only considered the place of eating the main meal on ordinary days, and not during the weekends or special days. The risk of obesity related to eating away from home was probably due to a change in leisure time activities, with an increase in large meals.

Several studies have indicated that increasing the number of meals decreases weight (Drummond *et al.,* 1998; Ma *et al.,* 2003; Vik *et al.,* 2010), as smaller but multiple meal portions can reduce hunger and serum insulin concentrations (Speechly & Buffenstein, 1999), as well as increase thermogenesis and fat utilisation. However, the results of many epidemiological studies in this regard are inconsistent, and have shown positive, negative or even no associations (see Murakami & Livingstone, 2015). Although increased meal frequency may "protect" against weight gain (as long as there is no increase in calories ingested), it is difficult to increase the number of meals without an increase in calories (Bertéus-Forslund *et al.,* 2002). Results obtained indicated that eating few meals per day (<3) increased the likelihood of both, overall and abdominal obesity, although eating more than 3 meals daily was not associated with any obesity phenotype, which is consistent with the observations of Kant *et al.* (1995).

As it is well known, an imbalance between energy intake and expenditure commonly leads to overweight and obesity. However, in some populations that have experienced an increase in the prevalence of obesity, total energy intake has not increased and has even decreased (Giles-Corti *et al.,* 2003). Thus, factors related to energy expenditure play a crucial role in the development of obesity. Non-exercise activity thermogenesis (NEAT),

i.e. the calories a person burns daily while performing daily activities has significant inter-individual variability and is crucial in weight gain (Villablanca *et al.,* 2015). According to Levine *et al.* (2008), walking is the main component of NEAT, although the intensity of the work performed can also be considered a component of interest. The present results showed no association between work intensity and obesity and, contrary to what might be expected, there was a positive (small but significant) association between daily walking minutes and central fat distribution. Both walking and working (unless the work involves high energy expenditure) are considered low-intensity daily activities and, unlike high-intensity aerobic exercise, basal metabolic rate does not increase with this type of activities (Bernstein *et al.,* 2004). This, together with the possibility that the extra energy expenditure caused by both activities may be compensated by a higher intake in more active individuals (Gutiérrez-Fisac *et al.,* 2002), could explain the results.

According to Füzéki *et al.* (2017), the results of the present study indicated that regular sports practice was a "protective" factor against different obesity phenotypes, possibly because it involves extra energy expenditure. Walking, not only during daily activities but also as a sporting activity, has a positive effect on the management of obesity and associated comorbidities, even in the absence of weight loss (Gaesser & Angadi, 2021); in the sample studied, such a relationship was observed for obesity and FM%, i.e. excess of adiposity. Although the amount of energy required to perform these activities is lower than in sports practice, lower intensity activities can be sustained for longer, so the total energy expenditure may be sufficient to influence metabolic balance and protect against weight gain (Füzéki *et al.,* 2017).

Sedentary behaviours are one of the main factors responsible for the obesity epidemic, and have been associated with various health risks. A sedentary behaviour is not only the absence of exercise (see van der Ploeg & Hillsdon, 2017), but refers to participation in activities that do not significantly increase energy expenditure (Pate *et al.,* 2008). Here, we considered TV viewing as an indicator of sedentary lifestyle or inactivity and, in agreement with other studies (e.g. Inoue *et al.,* 2012; Xie *et al.,* 2014; Rosiek *et al.,* 2015), the results confirmed its association with all obesity phenotypes. The mechanisms involved may be diverse; thus, it has been pointed out that the physical inactivity that accompanies TV viewing reduces the amount of time devoted to other physical activities (Tucker & Bagwell, 1991), although some studies (Rey-López *et al.,* 2012; Xie *et al.,* 2014) indicate that the relationship between this sedentary behaviour and obesity is independent of physical activity. In addition, snacking and consumption of high-density

foods and sugary drinks also increases while watching TV (Blass *et al.,* 2006; Pearson & Biddle, 2011), which hinders energy balance.

Sleep comprises approximately one third of a person's life span (Lauderdale *et al.,* 2006), but has not received much attention on its influence on obesity until relatively recently. According to some studies, sleep deprivation may influence obesity through changes in hunger-regulating hormones (e.g. decreased leptin and increased ghrelin), leading to increased energy intake (Spiegel *et al.,* 2004 a, b; Schmid *et al.,* 2008); furthermore, reduced sleep also means increased opportunity to eat (Sivak, 2006) and is associated with changes in dietary patterns (Gebski *et al.,* 2018). Also, sleep deprivation could lead to a reduction in daytime energy expenditure, due to feelings of tiredness, and a decrease in voluntary exercise (Patel, 2009). The results did not show association between hours of sleep and obesity, which could be due to the fact that most study participants (77.5%) slept sufficiently, between 6 and 8 hours per day, and very few slept less than 6 or more than 8 hours. Given that some disparities have been found between time spent in bed and actual time spent asleep (Lauderdale *et al.,* 2006), we also have assess sleep quality, which includes problems such as difficulty falling asleep, frequent awakenings at night or difficulty falling back to sleep. In this case, there was a clear and positive association with overall and abdominal obesity. In addition, the positive association of sleep quality with excessive adiposity was relatively significant, but not with a central fat distribution.

The relationship between smoking and obesity has been a subject of much research, but still remains unclear; smoking has shown to be associated with both low and high BMI (see Dare *et al.,* 2015). In the present study, ex-smokers showed a higher likelihood of having abdominal obesity and a centralised fat distribution. These results were consistent with those from other populations, where smoking cessation has been found to routinely lead to weight gain (Chiolero *et al.,* 2008; Liao *et al.,* 2016), although the mechanisms are not fully understood. Wack & Rodin (1982) have linked smoking cessation to changes in the perceived palatability of food, which could be responsible for increased energy intake. These authors also point to a change in the metabolism of ex-smokers due to the loss of the nicotine effect, which could lead to an increase of calorie storage, with a consequent increase in weight. A simpler explanation would be that many ex-smokers increase their energy intake, substituting food for cigarettes as a means of oral gratification (Stamford *et al.,* 1986). There are also studies showing that smokers are less obese than non-smokers (Molarius *et al.,* 1997; Dare *et al.,* 2015), although the obtained results did not coincide with these observations. Smoking (current or past) did not increased likelihood of overall obesity or excess fat in the sample analysed, although in agreement with the results of Bamia *et al.* (2004), abdominal obesity and central fat

distribution were associated with smoking. One possible explanation is that cigarette smoking stimulates the activity of the sympathetic nervous system, which is responsible for increased cortisol concentrations, and, precisely, visceral adipose tissue is strongly influenced by concentrations of this hormone (Chiolero *et al.,* 2008). For current smokers, a positive association between the number of cigarettes smoked and obesity was also confirmed as has been reported by Bamia *et al.* (2004) and Chiolero *et al.* (2008). These results may seem unusual due to the metabolic effect of smoking mentioned above; however, smoking may be associated with other unhealthy behaviours (see Murphy *et al.,* 2018), which could partly explain the results obtained.

Alcohol has a relatively high calorie content and can be considered a risk factor in the development of obesity through excessive consumption of "passive" energy (see Yeomans, 2010). However, the results obtained did not show a positive association between occasional alcohol consumption and obesity. Moreover, alcohol consumption seemed to be "protective" against an excessive adiposity (FM%). A possible explanation may lie in the thermogenic effect of alcohol and its involvement in increasing energy expenditure (Traversy & Chaput, 2015). Raben *et al.* (2003) have shown that diet-induced thermogenesis was 27% higher after a meal rich in alcohol than after meals rich in carbohydrates and fats. Epidemiological evidence suggests that moderate alcohol intake may be protective against obesity risk whereas, in the absence of alcohol dependence, a high alcohol intake may increase such risk (Yeomans, 2010). In the present research, the negative association between alcohol consumption, overall obesity and excessive adiposity was only significant when individuals drank occasionally, indicating that the protective effect was limited to moderate consumption, as noted above. Some studies show that the positive association between alcohol consumption and both obesity and abdominal obesity depends on the amount consumed (Vadstrup *et al.,* 2003), and episodes of overeating due to alcohol consumption are also frequent (Kase *et al.,* 2016).

This exploratory study had some limitations and strengths. Firstly, it should be noted that the variables used to describe lifestyle features were mostly qualitative, self-reported and, in many cases, could be interrelated. In addition, they were not always strictly comparable with those of other studies. Secondly, cases of obesity were over-represented in relation to the general population in the same geographical area; this fact could have diluted some of the effects of the variables analysed, but allowed to analyse the whole range of obesity variation, from normal weight to morbid obesity, which is not common in other research. Finally, although the results obtained were specific for this sample (otherwise quite homogeneous in terms of origin and SES), they have been consistent

with those of other research with similar objectives and methodology, confirming the association between lifestyle features and various obesity phenotypes.

In conclusion, our results confirmed the association between several modifiable lifestyle features and obesity, not only with overall obesity, but also with other obesity phenotypes, such as abdominal obesity, central fat distribution or excessive adiposity. Few meals per day, many time spent watching TV, bad/regular quality of sleep, and heavy tobacco use increased the risk of obesity, whereas, in general, more time spent at the main meal, walking and regular sport practice, and moderate alcohol consumption decreased such risk.

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Conflicts of Interest. The authors have no conflicts of interest to declare.

Ethical Approval. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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Table 1. Number and percentage (%) of individuals with and without obesity (BMI based) by sex and age group.

With obesity	Age group (years)	Females	Males
	<45	45 (31.7%)	37 (46.8%)
	≥45	97 (68.3%)	42 (53.2%)
Without obesity	**Age group (years)**	**Females**	**Males**
	<45	100 (56.8%)	74 (59.7%)
	≥45	76 (43.2%)	50 (40.3%)

Table 2. Multiple logistic regression tests for the association between eating habits and obesity phenotypes.

	Eating habits							
	Overall obesity		Abdominal obesity		Fat mass distribution		Fat mass percentage	
	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P
Main meal duration								
≤ 20 min	1.00		1.00		1.00		1.00	
>20 min	0.54 (0.3-0.9)	**0.010**	0.50 (0.3-0.8)	**0.004**	0.79 (0.5-1.3)	0.372	0.66 (0.4-1.1)	0.100
Main meal location								
Home	1.00		1.00		1.00		1.00	
Tupperware	0.85 (0.4-1.9)	0.703	0.98 (0.4-2.2)	0.955	1.04 (0.5-2.4)	0.928	1.11 (0.5-2.4)	0.795
Restaurant	1.15 (0.5-2.6)	0.726	1.11 (0.5-2.5)	0.802	1.17 (0.5-2.9)	0.739	0.84 (0.4-2.0)	0.696
Company canteen	0.53 (0.2-1.2)	0.114	0.53 (0.3-1.1)	0.098	0.45 (0.2-1.0)	**0.041**	0.71 (0.4-1.4)	0.313
Number of meals								
3 times	1.00		1.00		1.00		1.00	
4 times	0.76 (0.4-1.4)	0.372	0.69 (0.4-1.3)	0.221	1.69 (0.9-3.2)	0.107	0.86 (0.5-1.6)	0.616
5 times	1.03 (0.6-1.8)	0.910	1.18 (0.7-2.1)	0.590	1.17 (0.6-2.3)	0.634	1.08 (0.6-2.0)	0.806
≥ 6 times	1.32 (0.4-4.0)	0.620	0.88 (0.3-2.8)	0.832	0.79 (0.2-2.7)	0.714	0.58 (0.2-2.2)	0.415
< 3 times	6.16 (1.4-28.1)	**0.019**	5.38 (1.2-24.7)	**0.031**	1.67 (0.4-8.0)	0.525	1.27 (0.2-8.3)	0.804

Adjusted for age, sex, educational level and professional level (multivariable-adjusted). OR: odd ratio; CI: confidence interval; P: p-value.

Table 3. Multiple logistic regression tests for association between activity and obesity phenotypes.

	Activity							
	Overall obesity		Abdominal obesity		Fat mass distribution		Fat mass percentage	
	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P
Physical activity								
Nothing	1.00		1.00		1.00		1.00	
Walking	0.29 (0.2-0.6)	**5.1E-04**	0.40 (0.2-0.8)	**0.010**	0.95 (0.4-2.1)	0.909	1.03 (0.4-2.6)	0.947
Regular sport	0.33 (0.2-0.6)	**4.1E-05**	0.33 (0.2-0.6)	**3.9E-05**	0.42 (0.2-0.7)	**0.002**	0.45 (0.3-0.8)	**0.003**
Since how long ago								
Nothing	1.00		1.00		1.00		1.00	
<1 year	0.91 (0.3-2.5)	0.852	0.63 (0.2-1.8)	0.382	0.61 (0.2-1.9)	0.398	0.75 (0.2-2.3)	0.615
1-2 years	0.53 (0.2-1.3)	0.152	0.45 (0.2-1.1)	0.076	1.94 (0.7-5.2)	0.187	1.02 (0.4-2.5)	0.967
3-6 years	0.18 (0.1-0.5)	**4.2E-04**	0.15 (0.1-0.4)	**1.5E-04**	0.67 (0.3-1.6)	0.368	0.52 (0.2-1.3)	0.154
> 6 years	0.23 (0.1-0.4)	**3.3E-06**	0.34 (0.2-0.6)	**3.3E-04**	0.30 (0.2-0.6)	**2.8E-04**	0.39 (0.2-0.7)	**0.002**
Daily walking								
Minutes/day	1.00 (1.0-1.0)	0.502	1.00 (1.0-1.0)	0.154	1.01 (1.0-1.0)	**0.016**	1.00 (1.0-1.0)	0.999
Work intensity								
Light	1.00		1.00		1.00		1.00	
Moderate/busy	0.89 (0.6-1.4)	0.618	0.99 (0.6-1.6)	0.954	1.22 (0.7-2.0)	0.445	0.75 (0.5-1.2)	0.258
Watching television								
Hours/week	1.04 (1.0-1.1)	**2.9E-04**	1.05 (1.0-1.1)	**4.7E-05**	1.04 (1.0-1.1)	**0.002**	1.03 (1.0-1.1)	**0.016**

Adjusted for age, sex, educational level and professional level (multivariable-adjusted). OR: odd ratio; CI: confidence interval; P: p-value.

Table 4. Multiple logistic regression tests for association between sleep patterns and obesity phenotypes.

	Sleep patterns							
	Overall obesity		Abdominal obesity		Fat mass distribution		Fat mass percentage	
	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P
Sleep duration								
6-8 h	1.00		1.00		1.00		1.00	
<6 h	0.90 (0.5-1.8)	0.765	0.82 (0.4-1.7)	0.590	1.24 (0.6-2.6)	0.576	0.91 (0.4-2.1)	0.817
>8 h	1.51 (0.7-3.3)	0.290	1.46 (0.7-3.3)	0.364	1.51 (0.6-3.9)	0.397	1.15 (0.4-3.0)	0.773
Sleep quality								
Good	1.00		1.00		1.00		1.00	
Regular	2.02 (1.2-3.4)	**0.008**	1.78 (1.1-3.0)	**0.032**	1.26 (0.7-2.3)	0.431	1.68 (0.9-3.0)	0.079
Bad	2.73 (1.4-5.2)	**0.002**	2.09 (1.1-4.1)	**0.030**	0.96 (0.5-2.0)	0.913	2.08 (0.9-4.7)	0.076

Adjusted for age, sex, educational level and professional level (multivariable-adjusted). OR: odd ratio; CI: confidence interval; P: p-value.

663 **Table 5**. Multiple logistic regression tests for association between tobacco and alcohol
664 consumption and obesity phenotypes.

	Tobacco and alcohol consumption							
	Overall obesity		Abdominal obesity		Fat mass distribution		Fat mass percentage	
	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P
Smoking								
Non smoker	1.00		1.00		1.00		1.00	
Former smoker	1.56 (0.9-2.6)	0.093	1.88 (1.1-3.2)	**0.021**	2.46 (1.4-4.4)	**0.002**	1.05 (0.6-1.9)	0.862
Smoker	1.29 (0.7-2.4)	0.415	1.77 (1.0-3.3)	0.073	1.99 (1.0-3.9)	**0.047**	0.87 (0.5-1.6)	0.670
Number of cigarettes								
Cigarettes/week	1.06 (1.0-1.1)	**5.2E-05**	1.05 (1.0-1.1)	**3.7E-04**	1.03 (1.0-1.1)	0.120	1.04 (1.01-1.07)	**0.010**
Alcohol								
No/infrequently	1.00		1.00		1.00		1.00	
Yes	0.62 (0.4-1.0)	0.058	0.69 (0.4-1.2)	0.160	0.80 (0.5-1.4)	0.452	0.52 (0.3-0.9)	**0.030**
Alcohol frequency								
Never/infrequently	1.00		1.00		1.00		1.00	
Occasionally	0.51 (0.3-1.0)	**0.050**	0.66 (0.3-1.3)	0.230	0.71 (0.3-1.5)	0.361	0.43 (0.2-0.9)	**0.022**
Weekends	0.54 (0.3-1.0)	0.061	0.58 (0.3-1.1)	0.101	0.56 (0.3-1.1)	0.100	0.51 (0.3-1.0)	0.052
Some days/week	0,78 (0,3-1,9)	0.578	0.83 (0.3-2.0)	0.685	2.65 (1.0-7.2)	0.055	1.30 (0.5-3.7)	0.629
Every days	0.87 (0.4-1.8)	0.711	0.89 (0.4-1.9)	0.761	0.97 (0.4-2.3)	0.948	0.51 (0.2-1.2)	0.125

665 Adjusted for age, sex, educational level and professional level (multivariable-adjusted). OR:
666 odd ratio; CI: confidence interval; P: p-value.
667

Box legends:

Box 1. Dichotomization of the four obesity phenotypes.

Box 2. Description of the lifestyle variables grouped into four items (I to IV), as well as their categories and/or units.