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# UNIVERSITÀ DEGLI STUDI DI TORINO

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25 **Contributors to the obesity and hyperglycemia epidemics. A prospective study in a**  
26 **population-based cohort**

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28 **Running title:** Contributors to obesity and hyperglycemia epidemics

29

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**Abstract**

*Objective:* Relatively unexplored contributors to the obesity and diabetes epidemics may include: sleep restriction, increased house temperature (HT), television watching (TW), consumption of restaurant meals (RM), use of air conditioning (AC) and use of antidepressant/antipsychotic drugs (AD). *Design and Subjects:* In a population based-cohort ( $n=1597$ ), we investigated the possible association among these conditions and obesity or hyperglycemia incidence at six-year-follow-up. Subjects with obesity ( $n=315$ ) or hyperglycemia ( $n=618$ ) at baseline were excluded: respectively 1282 and 979 individuals were therefore analyzed. *Results:* At follow-up, 103/1282 became obese; these subjects showed significantly higher BMI, waist circumference, saturated fat intake, RM frequency, TW hours, HT, AC and AD use and lower fiber intake, metabolic equivalent of activity in hours/week (METS) and sleep hours at baseline. In a multiple logistic regression model, METS (OR=0.94;95%CI 0.91-0.98), RM (OR=1.47 per meal/week;1.21-1.79), being in the third tertile of HT (OR=2.06; 1.02-4.16) and hours of sleep (OR=0.70 per hour; 0.57-0.86) were associated with incident obesity. Subjects who developed hyperglycemia ( $n=174/979$ ; 17.8%) had higher saturated fat intake, RM frequency, TW hours, HT, AC and AD use at baseline and lower METS and fiber intake. In a multiple logistic regression model, fiber intake (OR=0.97 for each g/day; 0.95-0.99), RM (1.49 per meal/week; 1.26-1.75) and being in the third tertile of HT (OR=1.95; 1.17-3.26) were independently associated with incident hyperglycemia.

*Conclusions:* Lifestyle contributors to the obesity and hyperglycemia epidemics may be regular consumption of RM, sleep restriction and higher HT, suggesting potential adjunctive nonpharmacologic preventive strategies for the obesity and hyperglycemia epidemics.

**Key words:** obesity, hyperglycemia, restaurant meals, sleep restriction, home temperature

## 76 **Introduction**

77 Obesity and diabetes have reached epidemic proportions in the United States and Europe.  
78 Changes in dietary habits and reductions in physical activity are the two most common  
79 explanations for the increasing burden of these diseases. Recently, attention has been directed  
80 to other contributors, that may influence the balance between energy expenditure and intake  
81 [1-8]. Furthermore, dietary and exercise habits are not easily modifiable, and the search for  
82 risk factors that are more amenable to change might be potentially interesting and relatively  
83 unexplored in European cohorts.

84 The average amount of sleep per night has been reported to be declining slightly [9] and an  
85 increased incidence of obesity and hyperglycemia has been noted along with this decrease in  
86 sleep, particularly in younger cohorts [10]. This finding is probably due to the profound  
87 metabolic hormonal changes exerted by sleep debt and increased fatigue, leading to increased  
88 caloric intake and reduced energy expenditure, respectively [2-3, 10]. Other features of  
89 modern societies include psychosocial stress, indoor heating during cold seasons and air  
90 conditioning during warm seasons.

91 The use of antidepressants and atypical antipsychotics that are associated with weight gain  
92 has increased substantially in the last decade [1]. These conditions, together with increasing  
93 television watching and consuming foods that have been prepared outside the home, might  
94 promote a decline in physical activity and overeating [4, 6].

95  
96 In a population based-cohort we investigated possible associations among sleep restriction,  
97 house temperature, hours of television watching, consumption of restaurant foods, use of air  
98 conditioning, use of antidepressant/antipsychotic drugs and the risk of obesity and  
99 hyperglycemia, after an average follow-up period of six-years.

100

## 101 **Subjects and Methods**

102 All 1,877 Caucasian patients aged 45-64 years of six family physicians were invited to  
103 participate in a metabolic screening between 2001 and 2003. These subjects were  
104 representative of the Local Health Units of the province of Asti (North-western Italy) as  
105 reported previously [11]. In total, 1658 patients (88.3%) agreed to participate by written  
106 informed consent, whereas 219 declined. Both participants and non-participants showed the  
107 same gender distribution, level of education, prevalence of known diabetes and subjects living  
108 in rural areas as the resident population of the corresponding age-group, in the same area [11].  
109 Clinics were held in the morning after fasting overnight; for each patient, weight, height,  
110 waist circumference (measured by a plastic tape meter at the level of the umbilicus) and blood  
111 pressure were measured, and a fasting blood sample was drawn. Systolic and diastolic blood  
112 pressures were measured twice with a standard mercury sphygmomanometer with the patient  
113 in a sitting position after at least 10 min of rest. The reported values are the means of two  
114 measurements.

115 All patients answered a questionnaire at the health screening. The following data were  
116 collected for each subject: smoking habits, alcohol consumption, education level, health  
117 conditions, drugs used, sleep duration, mean house temperature during autumn/winter, mean  
118 daily number of hours of television watching, mean weekly number of meals consumed in  
119 restaurants (also considering fast-food restaurants and pizzerias) and regular use (>2  
120 days/week) of air conditioning during the summer season. Sleep duration was defined as self-  
121 reported time in bed (calculated from bedtime to rise time) minus sleep latency.

122 All subjects completed the validated, semi-quantitative food-frequency questionnaire used in  
123 EPIC (European Prospective Investigation into Cancer and Nutrition) studies [12] and the  
124 Minnesota-Leisure-Time-Physical-Activity questionnaire [13]. A dietician who was blinded to  
125 the study details checked all questionnaires for completeness, internal coherence and

126 plausibility. Each nutrient was adjusted for total energy using the residual method [14]. The  
127 leisure physical activity level was calculated as the product of the duration and frequency of  
128 each activity (in hours/week), weighted by an estimate of the metabolic equivalent of the  
129 activity (MET), and summed for all activities performed.

130

131 From January to November 2008, patients were contacted for follow-up visits. Deaths  
132 occurred in 61/1658 (3.7%) subjects during the follow-up period. All the remaining 1597  
133 patients had weight, waist circumference and blood pressure measurements taken, and a blood  
134 sample was drawn for the determination of fasting metabolic parameters.

135 All procedures were in accordance with the Declaration of Helsinki. The study was approved  
136 by the local Ethics Committee.

137 Laboratory methods have been described previously [11]. Diabetes and impaired fasting  
138 glucose (IFG) were defined in accordance with guidelines [15].

### 139 *Statistical analyses*

140 When analyzing the association between the incidence of obesity and baseline variables,  
141 subjects with obesity ( $\text{BMI} \geq 30 \text{ kg/m}^2$ ) at baseline ( $n=315/1597$ ; 19.7%) were excluded. When  
142 analyzing the association between the incidence of hyperglycemia and baseline variables,  
143 subjects with hyperglycemia (fasting glucose  $\geq 5.6 \text{ mmol/L}$ ) at baseline ( $n=618/1597$ ; 38.7%)  
144 were excluded. Therefore, analyses were performed in 1282 and 979 subjects, respectively.

145 Due to the low number of expected incident cases of type 2 diabetes, our study did not have  
146 sufficient statistical power to detect differences in baseline variables between patients with  
147 and without incident diabetes. Therefore, incident IFG and diabetes were combined into one  
148 category, termed incident hyperglycemia.

149 A Student's t test (normal distribution) or Mann-Whitney test (skewed distribution) and a  $\chi^2$ -  
150 test were performed to assess raw differences in baseline continuous and categorical variables,

151 respectively. A logistic regression analysis was performed to estimate adjusted odds ratios  
152 among baseline fiber intake, saturated fat intake, METS, degrees centigrade (°C) of house  
153 temperature, hours of sleep, hours of television watching, mean number of restaurant meals  
154 per week, antidepressant/antipsychotic drug use, air conditioning use and incident obesity and  
155 incident hyperglycemia, after controlling for sex, baseline BMI, education level and (in cases  
156 of incident hyperglycemia) baseline glucose values and alcohol intake. A multiple linear  
157 regression model was conducted to evaluate the association among these variables and  
158 continuous values of BMI and fasting glucose at follow-up.  
159 Due to the suspect of a non-linear relationship between incremental house temperature and  
160 BMI and fasting glucose at follow-up, tertiles of house temperature were used as dummy  
161 variables, using the lowest tertile as a reference.

162

## 163 **Results**

### 164 *Incident obesity*

165 Baseline characteristics according to obesity development are reported in Table 1. At follow-  
166 up, 103/1282 (8.0%; 95%CI 6.5-9.5) subjects had become obese; those subjects showed  
167 significantly higher values of BMI, waist circumference, saturated fat intake, frequency of  
168 restaurant meals, hours of television watching, house temperature, use of air conditioning and  
169 antidepressant/antipsychotic drugs at baseline. They also had significantly lower fiber intake,  
170 METS and hours of sleep.

171 BMI values at follow-up showed an overall linear increase with increasing numbers of  
172 restaurant meals, hours of television and reduced hours of sleep, fiber intake and METS  
173 (Figure 1). The mean BMI at follow-up was highest in the small group of subjects consuming  
174  $\geq 4$ /restaurant meals/week:  $1.72\text{kg/m}^2$  (corresponding to a 3kg increase in weight and a 4cm  
175 increase in waist circumference).

176 In a multiple logistic regression model, after adjusting for sex, education level, baseline BMI  
177 and all the variables listed in Table 3, the following variables were independently associated  
178 with incident obesity: reduced level of exercise, increased number of restaurant meals (for  
179 each additional meal per week), being in the highest tertile of house temperature and reduced  
180 hours of sleep (Table 3).

181 When using BMI at follow-up as a continuous variable, both a reduced level of exercise ( $\beta$ =  
182 -0.02; 95%CI -0.03 to -0.01,  $p<0.001$ ) and an increased number of restaurant meals ( $\beta$ =0.27;  
183 95%CI 0.19 0.35,  $p<0.001$  for each additional meal per week) remained significantly  
184 associated with BMI at follow-up, after carrying out a multiple linear regression model that  
185 was adjusted for all the variables listed in Table 3.

#### 186 *Incident hyperglycemia*

187 Baseline characteristics according to hyperglycemia development are reported in Table 2. At  
188 follow-up, 174/979 (17.8%; 95%CI 15.4-20.2) subjects had developed hyperglycemia; those  
189 subjects were more frequently male and had significantly higher values of waist  
190 circumference, fasting glucose, saturated fat intake, alcohol intake, mean number of restaurant  
191 meals per week, hours of television watching, house temperature and use of air conditioning  
192 and antidepressant/antipsychotic drugs at baseline. They also showed significantly lower fiber  
193 intake and exercise levels.

194 Fasting glucose values at follow-up increased with increasing numbers of restaurant meals per  
195 week and hours of television watching as well as reduced fiber intake (Figure 2). The greatest  
196 increase in fasting glucose values during follow-up was 1.2 mmol/L in the group eating  $\geq 4$   
197 restaurant meals/week (incident hyperglycemia=61%; 95%CI 43-79), whereas the lowest (  
198 -0.02 mmol/L) was in the group with the highest fiber intake (incident hyperglycemia=14.5%;  
199 95%CI 10.6-18.4).

200 In a multiple logistic regression analysis, after adjusting for sex, education level, alcohol  
201 intake, baseline BMI and glucose and all the variable listed in Table 4, the following variables  
202 were independently associated with incident hyperglycemia: reduced fiber intake, increased  
203 number of restaurant meals (for each additional meal per week) and being in the highest tertile  
204 of house temperature (Table 4). When using glucose level at follow-up as a continuous  
205 variable, both reduced fiber intake ( $\beta = -0.008$ ; 95%CI  $-0.012$  to  $-0.004$ ,  $p=0.001$ ) and  
206 increased number of restaurant meals per week ( $\beta=0.22$ ; 95%CI  $0.18$   $0.26$ ,  $p<0.001$  for each  
207 additional meal per week) remained significantly associated with glucose values at follow-up  
208 after performing a multiple linear regression model that was adjusted for all the variables  
209 reported in Table 4.

210

211 A sensitivity analysis was performed in all patients including the 61 subjects who had died  
212 during the follow-up period, applying two extreme scenarios: assuming that either none of  
213 them or all of them had developed obesity or hyperglycemia at the follow-up. These results  
214 were consistent with those obtained when analyzing the living individuals only.

215

## 216 **Discussion**

217 Alternative independent contributors to the obesity and hyperglycemia epidemic, other than  
218 physical inactivity and increased energy intake, may be the regular consumption of restaurant  
219 meals, sleep restriction and higher home temperature. Other factors, such as television  
220 watching and the use of antidepressant/antipsychotic drugs and air conditioning, may have a  
221 lesser impact.

### 222 *Sleep restriction*

223 Sleep duration has declined from 8-9 hours per night to 7 hours or less per night in the last 50  
224 years, largely as a consequence of voluntary sleep restriction (watching television, using the

225 Internet and getting more work done, etc.) [1]. Sleep debt is associated with decreased rates of  
226 glucose clearance, insulin response and glucose effectiveness, increased sympathetic nervous  
227 system activity and impaired glucose regulation by reduced lipolytic effects [16]. During  
228 sleep restriction, plasma leptin levels are decreased, whereas ghrelin, cortisol and orexin  
229 secretion is increased [1, 16]. Thus, the link between sleep debt and hormones implicated in  
230 feeding regulation explains the observed increase in appetite and food intake [17], particularly  
231 for energy-dense, high-carbohydrate foods [16]. This finding and the observed decrease in  
232 daytime physical activity after sleep loss [18] may contribute to the documented increased  
233 risk of obesity [2, 19]. However, these associations have been criticized because the  
234 epidemiological evidence is weak and the risk seems very small and develops over many  
235 years in very short sleepers (around 5 hours) [20-21]. The relationships between sleep  
236 duration and incident diabetes are contrasting, since large US epidemiological studies have  
237 found associations between both short and long sleep duration and diabetes [3, 22-23].  
238 Associations have been found to be significant only in a subset of diabetic patients with  
239 severe symptoms, and not for short sleepers after adjusting for BMI [22], in men but not in  
240 women [24], and other studies failed to find any association [25]. On the other hand, a  
241 growing number of epidemiological studies and meta-analyses have provided evidence of an  
242 association between short-duration sleep and the risk of obesity, as reviewed [1, 16]. In  
243 accordance with the literature, in our cohort, sleep restriction was associated with obesity at  
244 follow-up, but not with incident hyperglycemia at follow-up.

#### 245 *Indoor temperature*

246 The ability of brown adipose tissue (BAT) to burn rather than store calories depends on its  
247 mitochondrial uncoupling proteins [26]. Cold temperature can activate BAT in adult humans,  
248 irrespective of age and gender [27]. Over thirty years ago, research suggested that obesity  
249 could be treated by exercise in the cold [28]. On the other hand, in a hot environment, the

250 propensity for feeding is diminished [5], and air conditioning may contribute to rising obesity  
251 because the body expends less energy in temperature ranges associated with climate-  
252 controlled settings (via postural adjustments and evaporative cooling ) [1, 29]. Furthermore,  
253 the rising trend in central air conditioning could provide an incentive for people to remain  
254 indoors and exercise less [7]. We found a non-linear relationship between mean house  
255 temperature and BMI and fasting glucose levels at follow-up; a 2-fold increased risk for both  
256 incident obesity and hyperglycemia was estimated in subjects living at an indoor temperature  
257 greater than 20°C. It might be hypothesized that metabolic processes are favorably affected by  
258 an ambient temperature within the thermal neutral zone, i.e., not requiring energy expenditure  
259 to be allocated to maintaining a constant body temperature [1]. However, no evidence exists  
260 to support this and socio-economic factors might confound these associations.

261 In our cohort, air conditioning use, although associated with more than three-fold higher  
262 incidence of both obesity and hyperglycemia, showed a lower impact on these conditions  
263 compared to other risk factors.

#### 264 *Diet and exercise*

265 Several dietary factors that increase the risk for obesity and diabetes have been identified;  
266 among them, a reduced fiber intake has shown to play a strong predictive role for the  
267 incidence of type 2 diabetes [30], in line with our results. Meal consumption in restaurants  
268 was unusual in this middle-aged cohort, as less than 10% of the study subjects regularly  
269 consumed food away from home. Those who did consume food away from home did so with  
270 the following distribution: 47% pizzerias, 35% full-service restaurants and 18% fast-food  
271 restaurants. Compared to food prepared at home, restaurant food has high energy density,  
272 more fat and high glycemic load, and portion sizes are usually larger [6]. It has been shown  
273 that a higher ratio of fast-food to full-service restaurant density was associated with higher  
274 BMI and risk of obesity [31]. Furthermore, people find it difficult to estimate the caloric

275 contents of food items at restaurants and tend to underestimate their energy content [32]. Due  
276 to the low number of subjects regularly consuming meals in restaurants, our study did not  
277 have sufficient statistical power to detect risk differences by restaurant type. Nevertheless, the  
278 associations between the incremental number of meals consumed away from home and  
279 incident obesity and hyperglycemia were strong and largely independent of other potentially  
280 confounding lifestyle factors. The incremental rises in BMI and glucose levels at follow-up  
281 were greatest in patients eating  $\geq 4$  restaurant meals/week and the values were comparable to  
282 the data obtained for fast-food consumption [33]. These results are of potential interest for  
283 public programs aimed at reducing the diabetes and obesity epidemics.

284 Physical activity plays a central role in diabetes and obesity prevention [34], and reduced  
285 exercise at baseline predicted the incidence of obesity in our cohort. Television watching was  
286 positively associated with both incident obesity and hyperglycemia, but this relationship was  
287 not significant in the multivariate model, probably due to the predominant roles of reduced  
288 exercise and un-healthy dietary factors in obesity and hyperglycemia respectively; both of  
289 these conditions are strongly associated with increased amounts of time watching television  
290 [35].

#### 291 *Other possible contributors*

292 Even if incident obesity and hyperglycemia were 5- and 2-fold higher, respectively, in users  
293 of antidepressant/antipsychotic drugs, the associations were smaller and no longer statistically  
294 significant in the multivariate model, suggesting that the crude association was confounded by  
295 other risk factors. However, due to the low prevalence of antidepressant/antipsychotic drug  
296 users in our cohort (95/1597; 5.9%), the study lacked sufficient statistical power to detect  
297 small risks.

298 *Limitations and strengths*

299 A potential limitation of this study was the reliance on self-reported diet and other lifestyle  
300 factors. In addition, because this was an observational study, the possibility of confounding by  
301 unmeasured variables cannot be excluded. Socioeconomic status, which was not analyzed,  
302 might be a potential confounder. Nevertheless, we introduced education level into the  
303 multivariate models; this variable is a reliable indicator of socioeconomic status because it is  
304 stable, established in early adulthood and not modified by chronic disease [36]. Random  
305 misclassification and measurement errors in our prospective study would result in attenuated  
306 estimates of the strengths of the association with the outcome variables.

307 Finally, our results were limited to middle aged individuals, many of whom lived in rural  
308 areas and had low levels of education. However, the population-based cohort and the  
309 biological plausibility of our results, which are in accordance with previous studies each  
310 analyzing a single contributor, lend support to our conclusions.

311 The strengths of this study were the fact that a large proportion of subjects were enrolled from  
312 a defined community and its focus on multiple novel explanations for incident obesity and  
313 hyperglycemia at once.

314 *Conclusions*

315 Sleep restriction, higher home temperature and regular consumption of restaurant meals might  
316 represent lifestyle contributors to the obesity and hyperglycemia epidemics. Avoiding these  
317 behaviors could be a potential adjunctive nonpharmacologic strategy for preventing the  
318 obesity and hyperglycemia epidemics.

319

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321 **Conflicts of interest:** none

322

## References

- 1) McAllister EJ, Dhurandhar NV, Keith SW, Aronne LJ, Barger J, Baskin M, *et al.* Ten putative contributors to the obesity epidemic. *Critic Rev Food Sci Nutr* 2009; **49**: 868-913.
- 2) Bjorvatan B, Sagen IM, Oyane N, Waage S, Fetveit A, Pallesen S, *et al.* The association between sleep duration, body mass index and metabolic measures in the Hordaland Health Study. *J Sleep Res* 2007; **16**: 66-76.
- 3) Gottlieb DJ, Punjabi NM, Newman AB, Resnick HE, Redline S, Baldwin CM, *et al.* Association of sleep time with diabetes mellitus and impaired glucose tolerance. *Arch Intern Med* 2005; **165**: 863-867.
- 4) Krishnan S, Rosenberg L, Palmer JR. Physical activity and television watching in relation to risk of type 2 diabetes. *Am J Epidemiol* 2009; **169**: 428-434.
- 5) Siervo M, Wells JCK, Cizza G. The contribution of psychosocial stress to the obesity epidemic. *Horm Metab Res* 2009; **41**: 261-270.
- 6) Krishnan S, Coogan PF, Boggs DA, Rosenberg L, Palmer JR. Consumption of restaurant foods and incidence of type 2 diabetes in African American women. *Am J Clin Nutr* 2010; **91**: 465-471.
- 7) Jacobs DE, Wilson J, Dixon SL, Smith J, Evens A. The relationship of housing and population health: a 30-year retrospective analysis. *Environ Health Perspectives* 2009; **117**: 597-604.
- 8) Landsberg L, Young JB, Leonard WR, Linsenmeier RA, Turek FW. Is obesity associated with lower body temperatures? Core temperature: a forgotten variable in energy balance. *Metabolism* 2009; **58**: 871-876.
- 9) Knutson KL, Van Cauter E, Rathouz PJ, DeLeire T, Lauderdale DS. Trends in the prevalence of short sleepers in the USA: 1975-2006. *Sleep* 2010; **33**: 37-45.




- 10) Patel SR, Hu FB. Short sleep duration and weight gain: a systematic review. *Obesity* 2008; **16**: 643-653.
- 11) Bo S, Gentile L, Ciccone G, Baldi C, Benini L, Dusio F *et al*. The metabolic syndrome and high C-reactive protein: prevalence and difference by sex in a southern-European population-based cohort. *Diabetes Metab Research Rev* 2005; **21**: 515-524.
- 12) Kroke A, Klipstein-Grobusch K, Voss S, Möseneder J, Thielecke F, Noack R, Boeing H. Validation of a self-administered food-frequency questionnaire administered in the European Prospective Investigation into Cancer and Nutrition (EPIC) study: comparison of energy, protein, and macronutrient intakes estimated with the doubly labeled water, urinary nitrogen, and repeated 24-h dietary recall methods. *Am J Clin Nutr* 1999; **70**: 439-447.
- 13) Taylor HL, Jacobs DR Jr, Schucker B, Knudsen J, Leon AS & Debacker G. Questionnaire for the assessment of leisure time physical activities. *Journal of Chronic Diseases* 1978; **31**: 741-755.
- 14) Willett W, Stampfer MJ. Total energy intake: implications for epidemiologic analyses. *Am J Epidemiol* 1986; **124**: 17-27.
- 15) The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care* 2003; **26**: S5-S20.
- 16) Spiegel K, Tasali E, Leproult R, Van Cauter E. Effects of poor and short sleep on glucose metabolism and obesity risk. *Nat Rev Endocrinol* 2009; **5**: 253-261.
- 17) Brondel L, Romer MA, Nougues PM, Touyarou P, Davenne D. Acute partial sleep deprivation increases food intake in healthy men. *Am J Clin Nutr* 2010; **91**: 1550-1559.

- 18) Schmid SM, Hallschmid M, Jauch-Chara K, Wilms B, Benedict C, Lehnert H, *et al.*  
Short-term sleep loss decreases physical activity under free-living conditions but does not increase food intake under time-deprived laboratory conditions in healthy men. *Am J Clin Nutr* 2009; **90**: 1476-1482.
- 19) Patel SR, Blackwell T, Redline S, Ancoli-Israel S, Cauley JA, Hillier TA, *et al.* The association between sleep duration and obesity in older adults. *Int J Obes* 2008; **32**: 1825-1834.
- 20) Marshall NS, Glozier N, Grunstein RR. Is sleep duration related to obesity? A critical review of the epidemiological evidence. *Sleep Med Rev* 2008, **12**: 289-298
- 21) Horne J. Short sleep is a questionable risk factor for obesity and related disorders: statistical versus clinical significance. *Biol Psychol* 2008; **77**: 266-276.
- 22) Ayas NT, White DP, Al-Delaimy WK, Manson JA, Stampfer MJ, Speizer FE, *et al.* A prospective study of self-reported sleep duration and incident diabetes in women. *Diabetes Care* 2003; **26**: 380-384.
- 23) Yaggi HK, Araujo AB, McKinlay JB. Sleep duration as a risk factor for the development of type 2 diabetes. *Diabetes Care* 2006; **29**: 657-661.
- 24) Mallon L, Broman JE, Hetta J. High incidence of diabetes in men with sleep complaints or short sleep duration. *Diabetes Care* 2005; **28**: 2762-2767.
- 25) Björkelund C, Bondyr-Carlsson D, Lapidus L, Lissner L, Månsson J, Skoog I, *et al.*  
Sleep disturbance in midlife unrelated to 32-year diabetes incidence. *Diabetes Care* 2005; **28**: 2739-2744.
- 26) Farmer SR. Be cool, lose weight. *Nature* 2009; **485**: 839-840.
- 27) Celi FS. Brown adipose tissue –when it pays to be inefficient. *New Engl J Med* 2009; **360**: 1553-1556.

- 28) O'Hara WJ, Allen C, Shephard RJ. Treatment of obesity by exercise in the cold. *Can Med Assoc J* 1977; **117**: 773-778.
- 29) Keith SW, Redden DT, Katzmarzyk PT, Boggiano M H, Hanlon EC, Benca RM, *et al.* Putative contributors to the secular increase in obesity: exploring the roads less traveled. *Int J Obes* 2006; **30**: 1585-1594.
- 30) Montonen J, Knekt P, Jarvinen R, Aromaa A, Reunanen A. Whole-grain and fiber intake and the incidence of type 2 diabetes. *Am J Clin Nutr* 2003; **77**: 622-629.
- 31) Mehta NK, Chang VW. Weight status and restaurant availability. A multilevel analysis. *Am J Prev Med* 2008; **34**: 127-133.
- 32) Mc Call K. The fattening truth about restaurant food. *BMJ* 2008; **337**: a2229.
- 33) Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR Jr, *et al.* Fast-food habits, weight gain, and insulin resistance (the Cardia study): 15-year prospective analysis. *Lancet* 2005; **365**:36-42.
- 34) The Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 2002; **346**: 393-403.
- 35) Vioque J, Torres A, Quiles J. Time spent watching television, sleep duration and obesity in adults living in Valencia, Spain. *Int J Obes Relat Metab Disord* 2000; **24**: 1683-1688.
- 36) Bobák M, Hertzman C, Skodová Z, Marmot M. Own education, current conditions, parental material circumstances, and risk of myocardial infarction in a former communist country. *J Epidemiol Community Health* 2000; **54**: 91-96.

Figure 1 Baseline environmental characteristics and BMI at follow-up, by group of increment of the variables

Legend to Figure 1:

-  Upper -lower values
-  75th-25th percentile
-  median

Restaurant foods: group 1: no consumption of restaurant foods,  $n=1165$ ; group 2: 1-3/week,  $n=78$ ; group 3:  $\geq 4$ /week,  $n=39$

House temperature: first tertile  $\leq 18^\circ$ ,  $n=354$ ; second tertile  $\Rightarrow 18^\circ < 20^\circ$ ,  $n=401$ ; third tertile  $\geq 20^\circ$ ,  $n=527$

Television hours: group 1: 0-1 h/day,  $n=533$ ; group 2: 2-3 h/day,  $n=546$ ; group 3:  $> 3$  h/day,  $n=203$

Sleep hours: group 1:  $\leq 6.5$  h/day,  $n=309$ ; group 2:  $= 7$ h/day,  $n=456$ ; group 3:  $> 7$ h/day,  $n=517$




METS: group 1:  $< 18$ h/week,  $n=410$ ; group 2:  $\geq 18$ h/week  $< 24$ h/week,  $n=411$ ; group 3:  $\geq 24$ h/week,  $n=461$

Fiber intake (g/day): group 1: first tertile  $< 16.5$ g/day,  $n=447$ ; group 2: second tertile  $\geq 16.5$ g/day  $< 23.5$ g/day,  $n=413$ ; group 3: third tertile  $\geq 23.5$ g/day,  $n=422$

Saturated fat intake (% energy): group 1: first tertile  $< 10.5\%$ ,  $n=428$ ; group 2: second tertile  $\geq 10.5\% < 12.7\%$ ,  $n=426$ ; group 3: third tertile  $\geq 12.7\%$ ,  $n=428$

Figure 2. Baseline environmental characteristics and fasting glucose values at follow-up, by group of increment of the variables

Legend to Figure 2:

-  Upper -lower values
-  75th-25th percentile
-  median

Restaurant foods: group 1: no consumption of restaurant foods,  $n=893$ ; group 2: 1-3/week,  $n=55$ ; group 3:  $\geq 4$ /week,  $n=31$

House temperature: first tertile  $\leq 18^\circ$ ,  $n=250$ ; second tertile  $\Rightarrow 18^\circ < 20^\circ$ ,  $n=325$ ; third tertile  $\geq 20^\circ$ ,  $n=404$

Television hours: group 1: 0-1 h/day,  $n=413$ ; group 2: 2-3 h/day,  $n=427$ ; group 3:  $> 3$  h/day,  $n=139$

Sleep hours: group 1:  $\leq 6.5$  h/day,  $n=252$ ; group 2:  $= 7$ h/day,  $n=320$ ; group 3:  $> 7$ h/day,  $n=407$

METS: group 1:  $< 18$ h/week,  $n=312$ ; group 2:  $\geq 18$ h/week  $< 24$ h/week,  $n=318$ ; group 3:  $\geq 24$ h/week,  $n=349$

Fiber intake (g/day): group 1: first tertile  $< 16.5$ g/day,  $n=334$ ; group 2: second tertile  $\geq 16.5$ g/day  $< 23.5$ g/day,  $n=335$ ; group 3: third tertile  $\geq 23.5$ g/day,  $n=310$

Saturated fat intake (% energy): group 1: first tertile  $< 10.5\%$ ,  $n=316$ ; group 2: second tertile  $\geq 10.5\% < 12.7\%$ ,  $n=321$ ; group 3: third tertile  $\geq 12.7\%$ ,  $n=342$

Table 1. Baseline characteristics according to obesity development at follow-up

	Obesity yes	Obesity no	P
Number	103	1179	
Age (years)	54.8±6.1	54.3±5.6	0.37
Males (%)	52.4	46.1	0.22 <sup>1</sup>
BMI (kg/m <sup>2</sup> )	28.4±2.0	24.5±2.7	<0.001
Waist circumference (cm)	95.0±8.9	86.9±10.5	<0.001
Fasting glucose (mmol/L)	5.8±1.3	5.7±1.6	0.35
Energy intake (kcal/day)	2173.9±773.7	2072.8±656.7	0.14
Fat (% energy)	35.6±6.0	34.9±5.9	0.23
Saturated fat (% energy)	13.6±5.0	11.8±2.9	<0.001
Carbohydrates (%energy)	47.7±7.3	48.7±7.1	0.16
Fiber (g/day)	18.1±7.9	21.4±9.6	0.005
Actual smoking (%)	23.3	24.6	0.77 <sup>1</sup>
Alcohol (g/day)	17.9±28.0	17.3±26.4	0.72 <sup>2</sup>
Number of restaurant food intake/week	1.2±1.8	0.2±0.9	<0.001 <sup>2</sup>
METS (h/week)	17.3±7.0	22.1±9.4	<0.001
Television watching (h/day)	2.8±2.0	1.9±1.6	<0.001 <sup>2</sup>
Use of antidepressant/ antipsychotic drugs (%)	12.6	2.5	<0.001 <sup>1</sup>
Air conditioning use (%)	16.5	5.0	<0.001 <sup>1</sup>
Hours of sleep/day	6.3±1.4	7.2±1.1	<0.001
Mean house temperature (°C)	20.8±2.1	19.4±1.6	<0.001

## Education level (%):

Primary school	78.6	72.3	
Secondary school	13.6	19.1	
University	7.8	8.6	0.34 <sup>1</sup>
Living in a rural area (%)	35.9	39.8	0.44 <sup>1</sup>

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P-values calculated by *t*-Student test

<sup>1</sup> p-values calculated by Chi-square test

<sup>2</sup> p-values calculated by Mann-Whitney test

Table 2. Baseline characteristics according to hyperglycemia development at follow-up

	Hyperglycemia yes	Hyperglycemia no	P
Number	174	805	
Age (years)	53.6±5.3	53.9±5.7	0.53
Males (%)	47.1	36.5	0.009 <sup>1</sup>
BMI (kg/m <sup>2</sup> )	26.2±4.6	25.4±4.2	0.02
Waist circumference (cm)	90.8±13.0	87.2±12.1	<0.001
Fasting glucose (mmol/L)	5.3±0.3	5.0±0.4	<0.001
Energy intake (kcal/day)	2130.9±706.5	2055.9±655.0	0.18
Fat (% energy)	34.5±5.6	35.4±6.0	0.06
Saturated fat (% energy)	12.6±4.3	11.9±2.7	0.008
Carbohydrates (%energy)	49.6±6.7	48.2±7.2	0.02
Fiber (g/day)	18.5±9.2	21.7±9.2	<0.001
Actual smoking (%)	19.0	24.3	0.13 <sup>1</sup>
Alcohol (g/day)	18.5±24.4	13.4±24.3	<0.001 <sup>2</sup>
Number of restaurant food intake/week	0.9±1.7	0.1±0.7	<0.001 <sup>2</sup>
METS (h/week)	19.8±8.7	21.9±9.4	0.008
Television watching (h/day)	2.5±1.8	1.8±1.6	<0.001 <sup>2</sup>
Use of antidepressant/ antipsychotic drugs (%)	6.3	3.2	0.05 <sup>1</sup>
Air conditioning use (%)	10.9	4.6	0.001 <sup>1</sup>
Hours of sleep/day	7.0±1.2	7.2±1.2	0.07
Mean house temperature (°C)	20.6±2.4	19.4±1.3	<0.001

## Education level (%):

Primary school	75.3	72.5	
Secondary school	17.8	18.4	
University	6.9	9.1	0.62 <sup>1</sup>
Living in a rural area (%)	44.8	38.8	0.14 <sup>1</sup>

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P-values calculated by *t*-Student test

<sup>1</sup> p-values calculated by Chi-square test

<sup>2</sup> p-values calculated by Mann-Whitney test

Table 3. Association between baseline variables and obesity at follow-up in a logistic regression model: crude (left) and adjusted (right)

	OR	95%CI	P	OR <sup>1</sup>	95%CI	P
Male sex	1.29	0.86-1.93	0.22	1.02	0.57-1.82	0.95
BMI (kg/m <sup>2</sup> )	2.75	2.51-3.03	<0.001	2.50	2.03-3.05	<0.001
Saturated fat (% energy)	1.14	1.08-1.20	<0.001	1.07	0.99-1.15	0.09
Fiber (g/day)	0.96	0.93-0.98	<0.001	0.99	0.96-1.02	0.60
METS (h/week)	0.93	0.91-0.96	<0.001	0.94	0.91-0.98	<0.001
Number of restaurant food intake/week	1.65	1.45-1.86	<0.001	1.47	1.21-1.79	<0.001
Television watching (h/day)	1.32	1.18-1.46	<0.001	1.07	0.91-1.27	0.42
Antidepressant/antipsychotic drug use (%)	5.61	2.82-11.2	<0.001	1.56	0.52-4.63	0.43
Air conditioning use (%)	3.81	2.12-6.85	<0.001	2.21	0.93-5.27	0.07
Hours of sleep/day	0.53	0.45-0.63	<0.001	0.70	0.57-0.86	<0.001
First tertile house temperature (°C) <sup>2</sup>	1			1		
Second tertile house temperature (°C)	0.65	0.31-1.35	0.24	1.05	0.44-2.48	0.92
Third tertile house temperature (°C)	3.14	1.82-5.43	<0.001	2.06	1.02-4.16	0.04
Primary school (%)	1.50	0.91-2.46	0.11	1.41	0.73-2.72	0.30

<sup>1</sup> Odd ratios adjusted for all the variables listed in the table

<sup>2</sup> Tertiles of house temperature were: first tertile  $\leq 18^\circ$ ,  $n=354$ ; second tertile  $\Rightarrow 18^\circ < 20^\circ$ ,  $n=401$ ; third tertile  $\geq 20^\circ$ ,  $n=527$

Table 4. Association between baseline variables and hyperglycemia at follow-up in a logistic regression model: crude (left) and adjusted (right)

	OR	95%CI	P	OR <sup>1</sup>	95%CI	P
Male sex	1.55	1.11-2.16	0.009	1.12	0.73-1.70	0.61
Glucose (mmol/L)	23.7	11.6-48.4	<0.001	20.3	9.37-44.1	<0.001
Saturated fat (% energy)	1.07	1.02-1.12	0.009	1.00	0.93-1.07	0.99
Fiber (g/day)	0.96	0.94-0.98	<0.001	0.97	0.95-0.99	0.01
METS (h/week)	0.98	0.96-0.99	0.008	0.99	0.97-1.01	0.21
Number of restaurant food intake/week	1.66	1.44-1.91	<0.001	1.49	1.26-1.75	<0.001
Television watching (h/day)	1.27	1.16-1.39	<0.001	1.10	0.98-1.23	0.11
Antidepressant/ antipsychotic drug use (%)	2.02	0.98-4.18	0.06	0.93	0.36-2.34	0.87
Air conditioning use (%)	2.54	1.42-4.55	0.002	1.47	0.73-2.96	0.28
Hours of sleep/day	0.88	0.77-1.01	0.07	1.06	0.91-1.24	0.43
First tertile house temperature (°C) <sup>2</sup>	1			1		
Second tertile house temperature (°C)	0.72	0.42-1.22	0.22	0.76	0.43-1.35	0.35
Third tertile house temperature (°C)	2.82	1.81-4.37	<0.001	1.95	1.17-3.26	0.01
Primary school (%)	1.15	0.79-1.68	0.46	1.07	0.69-1.66	0.76

<sup>1</sup> Odd ratios adjusted for all the variables listed in the table, plus BMI and alcohol intake at baseline

<sup>2</sup> Tertiles of house temperature were: first tertile  $\leq 18^\circ$ ,  $n=250$ ; second tertile  $\Rightarrow 18^\circ < 20^\circ$ ,  $n=325$ ; third tertile  $\geq 20^\circ$ ,  $n=404$

Figure 1. Baseline environmental characteristics and BMI at follow-up, by group of increment of the variables

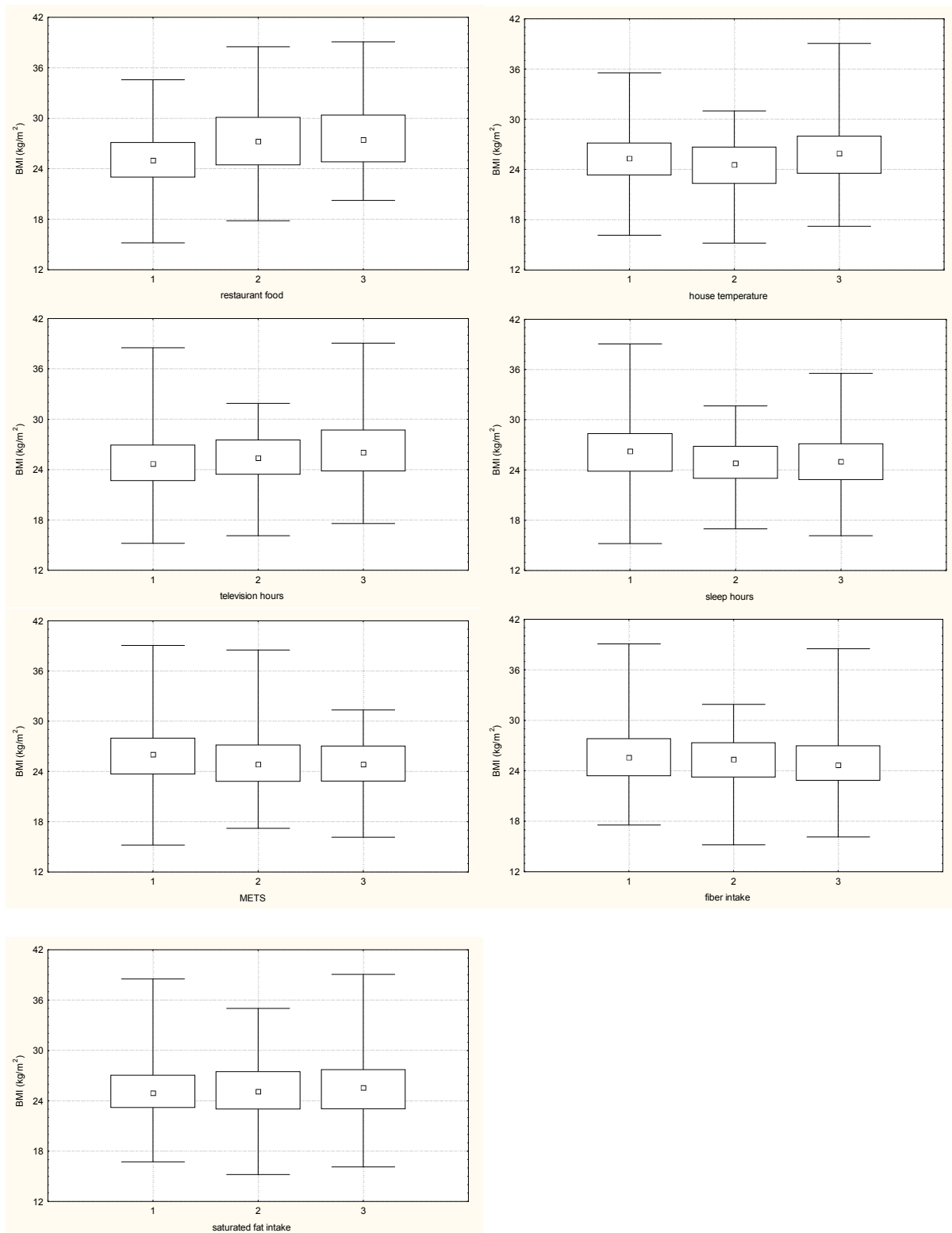


Figure 2. Baseline environmental characteristics and fasting glucose at follow-up, by group of increment of the variables

