



AperTO - Archivio Istituzionale Open Access dell'Università di Torino

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

This is the author's mar	nuscript				
Original Citation:					
Availability:					
This version is available	http://hdl.handle.net/2318/93295	since			
Published version:					
DOI:10.1038/ijo.2011.5					
Terms of use:					
Open Access					
Anyone can freely access the full text of works made available as "Open Access". Works made available under a Creative Commons license can be used according to the terms and conditions of said license. Use of all other works requires consent of the right holder (author or publisher) if not exempted from copyright protection by the applicable law.					

(Article begins on next page)



UNIVERSITÀ DEGLI STUDI DI TORINO

2 3	
4	
5	This is an author version of the contribution published on:
6	Questa è la versione dell'autore dell'opera:
7	Int J Obes (Lond). 2011 Nov;35(11):1442-9. doi: 10.1038/ijo.2011.5.
8	Epub 2011 Feb 1
9	
10	The definitive version is available at:
11	La versione definitiva è disponibile alla URL:
12	
13	http://www.nature.com.offcampus.dam.unito.it/ijo/journal/v35/n11/ful
14	1/ijo20115a.html
15	,
16	http://creativecommons.org/licenses/by-nc-nd/4.0/
17	
18	
19	
20	
21	
22	
23	
24	

Contributors to the obesity and hyperglycemia epidemics. A prospective study in a population-based cohort Running title: Contributors to obesity and hyperglycemia epidemics Simona Bo¹, MD, Giovannino Ciccone², MD, Marilena Durazzo¹, MD, Lucia Ghinamo¹, Paola Villois¹, Stefania Canil¹, Roberto Gambino¹, PhD, Maurizio Cassader¹, MD, Luigi Gentile³, MD, Cavallo-Perin P¹, MD ¹Department of Internal Medicine, University of Turin ²Unit of Cancer Epidemiology, University of Turin, Italy ³Diabetic Clinic, Hospital of Asti, Italy Corresponding author: Simona Bo, Department of Internal Medicine, University of Turin, Corso Dogliotti 14, 10126 Turin, Italy Telephone +(39)(011)6967864 Fax+(39)(011)6634751 E-mail: sbo@molinette.piemonte.it **Grant:** This study was supported by a grant from: Regione Piemonte, 2008. Word count: abstract 250, text 2777, 4 tables, 2 figures

50	Abstract
51	Objective: Relatively unexplored contributors to the obesity and diabetes epidemics may include:
52	sleep restriction, increased house temperature (HT), television watching (TW), consumption of
53	restaurant meals (RM), use of air conditioning (AC) and use of antidepressant/antipsychotic drugs
54	(AD). Design and Subjects: In a population based-cohort (n=1597), we investigated the possible
55	association among these conditions and obesity or hyperglycemia incidence at six-year-follow-up.
56	Subjects with obesity (n =315) or hyperglycemia (n =618) at baseline were excluded: respectively 1282
57	and 979 individuals were therefore analyzed. Results: At follow-up, 103/1282 became obese; these
58	subjects showed significantly higher BMI, waist circumference, saturated fat intake, RM frequency,
59	TW hours, HT, AC and AD use and lower fiber intake, metabolic equivalent of activity in
60	hours/week (METS) and sleep hours at baseline. In a multiple logistic regression model, METS
61	(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
62	(OR=2.06; 1.02-4.16) and hours of sleep (OR=0.70 per hour; 0.57-0.86) were associated with incident
63	obesity. Subjects who developed hyperglycemia (n=174/979; 17.8%) had higher saturated fat intake,
64	RM frequency, TW hours, HT, AC and AD use at baseline and lower METS and fiber intake. In a
65	multiple logistic regression model, fiber intake (OR=0.97 for each g/day; 0.95-0.99), RM (1.49 per
66	meal/week; 1.26-1.75) and being in the third tertile of HT (OR=1.95; 1.17-3.26) were independently
67	associated with incident hyperglycemia.
68	Conclusions: Lifestyle contributors to the obesity and hyperglycemia epidemics may be regular
69	consumption of RM, sleep restriction and higher HT, suggesting potential adjunctive

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

nonpharmacologic preventive strategies for the obesity and hyperglycemia epidemics.

Introduction

76

77

78

79

80

81

82

83

84

85

86

87

88

89

90

91

92

93

94

95

96

97

98

99

Obesity and diabetes have reached epidemic proportions in the United States and Europe. Changes in dietary habits and reductions in physical activity are the two most common explanations for the increasing burden of these diseases. Recently, attention has been directed to other contributors, that may influence the balance between energy expenditure and intake [1-8]. Furthermore, dietary and exercise habits are not easily modifiable, and the search for risk factors that are more amenable to change might be potentially interesting and relatively unexplored in European cohorts. The average amount of sleep per night has been reported to be declining slightly [9] and an increased incidence of obesity and hyperglycemia has been noted along with this decrease in sleep, particularly in younger cohorts [10]. This finding is probably due to the profound metabolic hormonal changes exerted by sleep debt and increased fatigue, leading to increased caloric intake and reduced energy expenditure, respectively [2-3, 10]. Other features of modern societies include psychosocial stress, indoor heating during cold seasons and air conditioning during warm seasons. The use of antidepressants and atypical antipsychotics that are associated with weight gain has increased substantially in the last decade [1]. These conditions, together with increasing television watching and consuming foods that have been prepared outside the home, might promote a decline in physical activity and overeating [4, 6]. In a population based-cohort we investigated possible associations among sleep restriction, house temperature, hours of television watching, consumption of restaurant foods, use of air conditioning, use of antidepressant/antipsychotic drugs and the risk of obesity and hyperglycemia, after an average follow-up period of six-years.

Subjects and Methods

101

102

103

104

105

106

107

108

109

110

111

112

113

114

115

116

117

118

119

120

121

122

123

124

125

All 1,877 Caucasian patients aged 45-64 years of six family physicians were invited to participate in a metabolic screening between 2001 and 2003. These subjects were representative of the Local Health Units of the province of Asti (North-western Italy) as reported previously [11]. In total, 1658 patients (88.3%) agreed to participate by written informed consent, whereas 219 declined. Both participants and non-participants showed the same gender distribution, level of education, prevalence of known diabetes and subjects living in rural areas as the resident population of the corresponding age-group, in the same area [11]. Clinics were held in the morning after fasting overnight; for each patient, weight, height, waist circumference (measured by a plastic tape meter at the level of the umbilicus) and blood pressure were measured, and a fasting blood sample was drawn. Systolic and diastolic blood pressures were measured twice with a standard mercury sphygmomanometer with the patient in a sitting position after at least 10 min of rest. The reported values are the means of two measurements. All patients answered a questionnaire at the health screening. The following data were collected for each subject: smoking habits, alcohol consumption, education level, health conditions, drugs used, sleep duration, mean house temperature during autumn/winter, mean daily number of hours of television watching, mean weekly number of meals consumed in restaurants (also considering fast-food restaurants and pizzerias) and regular use (>2 days/week) of air conditioning during the summer season. Sleep duration was defined as selfreported time in bed (calculated from bedtime to rise time) minus sleep latency. All subjects completed the validated, semi-quantitative food-frequency questionnaire used in EPIC (European Prospective Investigation into Cancer and Nutrition) studies [12] and the Minnesota-Leisure-Time-Physical-Activity questionnaire [13]. A dietician who was blinded to the study details checked all questionnaires for completeness, internal coherence and

plausibility. Each nutrient was adjusted for total energy using the residual method [14]. The leisure physical activity level was calculated as the product of the duration and frequency of each activity (in hours/week), weighted by an estimate of the metabolic equivalent of the activity (MET), and summed for all activities performed. From January to November 2008, patients were contacted for follow-up visits. Deaths occurred in 61/1658 (3.7%) subjects during the follow-up period. All the remaining 1597 patients had weight, waist circumference and blood pressure measurements taken, and a blood sample was drawn for the determination of fasting metabolic parameters. All procedures were in accordance with the Declaration of Helsinki. The study was approved by the local Ethics Committee. Laboratory methods have been described previously [11]. Diabetes and impaired fasting glucose (IFG) were defined in accordance with guidelines [15]. Statistical analyses When analyzing the association between the incidence of obesity and baseline variables, subjects with obesity (BMI \geq 30 kg/m²) at baseline (n=315/1597; 19.7%) were excluded. When analyzing the association between the incidence of hyperglycemia and baseline variables, subjects with hyperglycemia (fasting glucose ≥ 5.6 mmol/L) at baseline (n=618/1597; 38.7%) were excluded. Therefore, analyses were performed in 1282 and 979 subjects, respectively. Due to the low number of expected incident cases of type 2 diabetes, our study did not have sufficient statistical power to detect differences in baseline variables between patients with and without incident diabetes. Therefore, incident IFG and diabetes were combined into one category, termed incident hyperglycemia. A Student's t test (normal distribution) or Mann-Whitney test (skewed distribution) and a χ^2 test were performed to assess raw differences in baseline continuous and categorical variables,

126

127

128

129

130

131

132

133

134

135

136

137

138

139

140

141

142

143

144

145

146

147

148

149

respectively. A logistic regression analysis was performed to estimate adjusted odds ratios among baseline fiber intake, saturated fat intake, METS, degrees centigrade (°C) of house temperature, hours of sleep, hours of television watching, mean number of restaurant meals per week, antidepressant/antipsychotic drug use, air conditioning use and incident obesity and incident hyperglycemia, after controlling for sex, baseline BMI, education level and (in cases of incident hyperglycemia) baseline glucose values and alcohol intake. A multiple linear regression model was conducted to evaluate the association among these variables and continuous values of BMI and fasting glucose at follow-up.

Due to the suspect of a non-linear relationship between incremental house temperature and BMI and fasting glucose at follow-up, tertiles of house temperature were used as dummy variables, using the lowest tertile as a reference.

increase in waist circumference).

Results

164 Incident obesity

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

BMI values at follow-up showed an overall linear increase with increasing numbers of restaurant meals, hours of television and reduced hours of sleep, fiber intake and METS (Figure 1). The mean BMI at follow-up was highest in the small group of subjects consuming ≥4/restaurant meals/week: 1.72kg/m² (corresponding to a 3kg increase in weight and a 4cm

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 (β = 182 -0.02; 95%CI -0.03 to -0.01, p<0.001) and an increased number of restaurant meals (β =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 ≥4 restaurant meals/week (incident hyperglycemia=61%; 95%CI 43-79), whereas the lowest (197 198 -0.02 mmol/L) was in the group with the highest fiber intake (incident hyperglycemia=14.5%; 199 95%CI 10.6-18.4).

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

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

Discussion

- Alternative independent contributors to the obesity and hyperglycemia epidemic, other than physical inactivity and increased energy intake, may be the regular consumption of restaurant meals, sleep restriction and higher home temperature. Other factors, such as television watching and the use of antidepressant/antipsychotic drugs and air conditioning, may have a lesser impact.
- 222 Sleep restriction
- Sleep duration has declined from 8-9 hours per night to 7 hours or less per night in the last 50 years, largely as a consequence of voluntary sleep restriction (watching television, using the

Internet and getting more work done, etc.) [1]. Sleep debt is associated with decreased rates of glucose clearance, insulin response and glucose effectiveness, increased sympathetic nervous system activity and impaired glucose regulation by reduced lipolytic effects [16]. During sleep restriction, plasma leptin levels are decreased, whereas ghrelin, cortisol and orexin secretion is increased [1, 16]. Thus, the link between sleep debt and hormones implicated in feeding regulation explains the observed increase in appetite and food intake [17], particularly for energy-dense, high-carbohydrate foods [16]. This finding and the observed decrease in daytime physical activity after sleep loss [18] may contribute to the documented increased risk of obesity [2, 19]. However, these associations have been criticized because the epidemiological evidence is weak and the risk seems very small and develops over many years in very short sleepers (around 5 hours) [20-21]. The relationships between sleep duration and incident diabetes are contrasting, since large US epidemiological studies have found associations between both short and long sleep duration and diabetes [3, 22-23]. Associations have been found to be significant only in a subset of diabetic patients with severe symptoms, and not for short sleepers after adjusting for BMI [22], in men but not in women [24], and other studies failed to find any association [25]. On the other hand, a growing number of epidemiological studies and meta-analyses have provided evidence of an association between short-duration sleep and the risk of obesity, as reviewed [1, 16]. In accordance with the literature, in our cohort, sleep restriction was associated with obesity at follow-up, but not with incident hyperglycemia at follow-up. Indoor temperature The ability of brown adipose tissue (BAT) to burn rather than store calories depends on its mitochondrial uncoupling proteins [26]. Cold temperature can activate BAT in adult humans, irrespective of age and gender [27]. Over thirty years ago, research suggested that obesity could be treated by exercise in the cold [28]. On the other hand, in a hot environment, the

225

226

227

228

229

230

231

232

233

234

235

236

237

238

239

240

241

242

243

244

245

246

247

248

propensity for feeding is diminished [5], and air conditioning may contribute to rising obesity because the body expends less energy in temperature ranges associated with climatecontrolled settings (via postural adjustments and evaporative cooling) [1, 29]. Furthermore, the rising trend in central air conditioning could provide an incentive for people to remain indoors and exercise less [7]. We found a non-linear relationship between mean house temperature and BMI and fasting glucose levels at follow-up; a 2-fold increased risk for both incident obesity and hyperglycemia was estimated in subjects living at an indoor temperature greater than 20°C. It might be hypothesized that metabolic processes are favorably affected by an ambient temperature within the thermal neutral zone, i.e., not requiring energy expenditure to be allocated to maintaining a constant body temperature [1]. However, no evidence exists to support this and socio-economic factors might confound these associations. In our cohort, air conditioning use, although associated with more than three-fold higher incidence of both obesity and hyperglycemia, showed a lower impact on these conditions compared to other risk factors. Diet and exercise Several dietary factors that increase the risk for obesity and diabetes have been identified; among them, a reduced fiber intake has shown to play a strong predictive role for the incidence of type 2 diabetes [30], in line with our results. Meal consumption in restaurants was unusual in this middle-aged cohort, as less than 10% of the study subjects regularly consumed food away from home. Those who did consume food away from home did so with the following distribution: 47% pizzerias, 35% full-service restaurants and 18% fast-food restaurants. Compared to food prepared at home, restaurant food has high energy density, more fat and high glycemic load, and portion sizes are usually larger [6]. It has been shown that a higher ratio of fast-food to full-service restaurant density was associated with higher BMI and risk of obesity [31]. Furthermore, people find it difficult to estimate the caloric

250

251

252

253

254

255

256

257

258

259

260

261

262

263

264

265

266

267

268

269

270

271

272

273

contents of food items at restaurants and tend to underestimate their energy content [32]. Due to the low number of subjects regularly consuming meals in restaurants, our study did not have sufficient statistical power to detect risk differences by restaurant type. Nevertheless, the associations between the incremental number of meals consumed away from home and incident obesity and hyperglycemia were strong and largely independent of other potentially confounding lifestyle factors. The incremental rises in BMI and glucose levels at follow-up were greatest in patients eating ≥4 restaurant meals/week and the values were comparable to the data obtained for fast-food consumption [33]. These results are of potential interest for public programs aimed at reducing the diabetes and obesity epidemics. Physical activity plays a central role in diabetes and obesity prevention [34], and reduced exercise at baseline predicted the incidence of obesity in our cohort. Television watching was positively associated with both incident obesity and hyperglycemia, but this relationship was not significant in the multivariate model, probably due to the predominant roles of reduced exercise and un-healthy dietary factors in obesity and hyperglycemia respectively; both of these conditions are strongly associated with increased amounts of time watching television [35]. Other possible contributors Even if incident obesity and hyperglycemia were 5- and 2-fold higher, respectively, in users of antidepressant/antipsychotic drugs, the associations were smaller and no longer statistically significant in the multivariate model, suggesting that the crude association was confounded by other risk factors. However, due to the low prevalence of antidepressant/antipsychotic drug users in our cohort (95/1597; 5.9%), the study lacked sufficient statistical power to detect

275

276

277

278

279

280

281

282

283

284

285

286

287

288

289

290

291

292

293

294

295

296

297

small risks.

Limitations and strengths

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

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

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

Conclusions

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

Acknowledgments: This study was supported by a grant from: Regione Piemonte, 2008.

Conflicts of interest: none

References

- 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 Psycol* 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 fattering 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, <i>n</i> =1165; group 2: 1-3/week,
$n=78$; group 3: ≥ 4 /week, $n=39$
House temperature: first tertile $\leq 18^{\circ}$, $n=354$; second tertile=>18°<20°, $n=401$; third tertile
≥20°, <i>n</i> =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: ≤6.5 h/day, <i>n</i> =309; group 2: =7h/day, <i>n</i> =456; group 3: >7h/day, <i>n</i> =517
METS: group 1: <18h/week, <i>n</i> =410; group 2: ≥18h/week <24h/week, <i>n</i> =411; group
3:≥24h/week, <i>n</i> =461
Fiber intake (g/day): group 1: first tertile <16.5g/day, n=447; group 2: second tertile
\geq 16.5g/day <23.5g/day, n =413; group 3: third tertile \geq 23.5g/day, n =422
Saturated fat intake (% energy): group 1: first tertile <10.5%, <i>n</i> =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, <i>n</i> =893; group 2: 1-3/week,
$n=55$; group 3: $\geq 4/\text{week}$, $n=31$
House temperature: first tertile $\leq 18^{\circ}$, $n=250$; second tertile= $>18^{\circ}<20^{\circ}$, $n=325$; third tertile
≥20°, <i>n</i> =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: $\leq 6.5 \text{ h/day}$, $n=252$; group 2: $=7\text{h/day}$, $n=320$; group 3: $>7\text{h/day}$, $n=407$
METS: group 1: <18h/week, <i>n</i> =312; group 2: ≥18h/week <24h/week, <i>n</i> =318; group
3:≥24h/week, <i>n</i> =349
Fiber intake (g/day): group 1: first tertile <16.5g/day, n=334; group 2: second tertile
\geq 16.5g/day <23.5g/day, n =335; group 3: third tertile \geq 23.5g/day, n =310
Saturated fat intake (% energy): group 1: first tertile <10.5%, <i>n</i> =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^{1}
BMI (kg/m^2)	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^{1}
Alcohol (g/day)	17.9±28.0	17.3±26.4	0.72^{2}
Number of restaurant food	1.2±1.8	0.2±0.9	< 0.001 ²
intake/week			
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 ²
Use of antidepressant/ antipsychotic	12.6 2.5		< 0.001
drugs (%)			
Air conditioning use (%)	16.5	5.0	< 0.001
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^{1}
Living in a rural area (%)	35.9	39.8	0.44^{1}

P-values calculated by *t*-Student test

¹ p-values calculated by Chi-square test

²p-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^{1}
BMI (kg/m^2)	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^{1}
Alcohol (g/day)	18.5±24.4	13.4±24.3	< 0.001 ²
Number of restaurant food	0.9±1.7	0.1±0.7	< 0.001 ²
intake/week			
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 ²
Use of antidepressant/ antipsychotic	6.3	3.2	0.05^{1}
drugs (%)			
Air conditioning use (%)	10.9	4.6	0.001^{1}
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^{1}
Living in a rural area (%)	44.8	38.8	0.14 ¹

P-values calculated by *t*-Student test

¹ p-values calculated by Chi-square test

²p-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 ¹	95%CI	P
Male sex	1.29	0.86-1.93	0.22	1.02	0.57-1.82	0.95
BMI (kg/m^2)	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) ²	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
				1		

Odd ratios adjusted for all the variables listed in the table

² Tertiles of house temperature were: first tertile $\leq 18^{\circ}$, n=354; second tertile=>18°<20°, 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 ¹	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) ²	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

Odd ratios adjusted for all the variables listed in the table, plus BMI and alcohol intake at baseline

² Tertiles of house temperature were: first tertile ≤18°, n=250; second tertile=>18°<20°, n=325; third tertile ≥20°, n=404

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

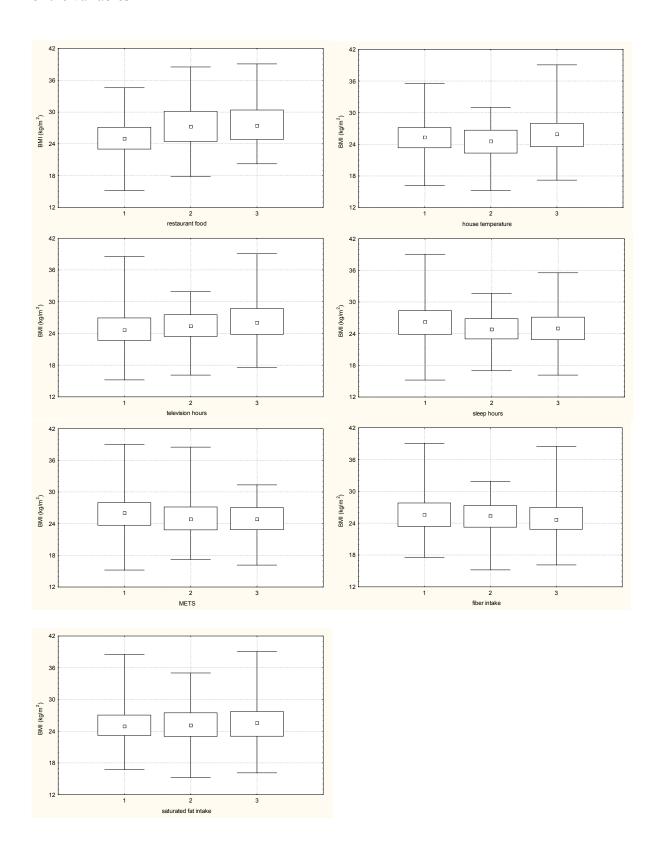


Figure 2. Baseline environmental characteristics and fasting glucose at follow-up, by group of increment of the variables

