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**Predictors of weight gain in a Mediterranean cohort: the SUN
4 Study**

6 (Running head: Predictors of weight gain in a Mediterranean cohort)

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(ABSTRACT AND KEY WORDS)

30 **ABSTRACT**

32 **Background:** An increased consumption of sugar-sweetened drinks has been
32 associated with weight gain and obesity in US. This trend may also be affecting
34 populations with different eating patterns who are increasingly adopting typical US
34 dietary patterns.

36 **Objective:** We assessed whether the consumption of sweetened drinks and some
36 other food items increased the likelihood of weight gain in a Mediterranean
population.

38 **Design:** Prospective cohort analysis, 7,194 men and women, mean age: 41 years,
followed-up with mailed questionnaires. Dietary exposure was assessed with a
40 previously validated semiquantitative food-frequency questionnaire.

42 **Results:** During follow-up (median= 28.5 months) we observed that 49.5% of
42 participants increased their weight (average weight gain= +0.64 Kg; 95% confidence
interval [CI]: +0.55 to +0.73 kg). Among participants who had gained ≥ 3 kg weight in
44 the five years previous to baseline, the adjusted odds ratio (OR) of subsequent
weight gain for the fifth versus the first quintile of sugar-sweetened soft drinks
46 (SSSD) consumption was 1.6 (95% CI: 1.2, 2.1; p for trend=0.02). This association
was absent among participants who had not gained weight in the previous five-year
48 period. The consumption of hamburgers, pizza and sausages (HPS) was also
independently associated with weight gain (adjusted OR for the fifth versus the first
50 quintile = 1.2 (95% CI: 1.0, 1.4; p for trend = 0.05). We also found a significant
though weaker association between red meat or sweetened fruit juice consumption
52 and weight gain.

54 **Conclusions:** In a Mediterranean cohort particularly among participants who were
54 already gaining weight, an increased consumption of SSSD and HPS was associated
with a higher risk of further subsequent weight gain.

56

Key words: obesity, overweight, body weight, body weight change, soft-drinks, fast-
58 food, fat, sugar, diet, nutrition, prospective studies, incidence, longitudinal analysis,
nutritional epidemiology.

60 (TEXT)

INTRODUCTION

62 The prevalence of obesity is dramatically increasing and represents a leading public
health problem; in fact, the millennium, it was of major concern for the clinical nutrition
64 field (1-3). A public health priority is to reduce the prevalence of obesity.

In the US population, in parallel to the obesity epidemic, the consumption of fast food
66 and of sugar-sweetened soft drinks (SSSD) has increased (+68 percent for
carbonated soft drinks during 1977-1997) (4-6). North American consumption of soft
68 drinks represents more than 44 percent of total World soft drink sales. Europe
represents 31 percent of this consumption and it is also on the rise (7). The
70 consumption of soft drinks, specially cola-drinks, has also increased in Spain
considerably (+42 percent in the period 1991-2001) (8). A higher consumption of
72 SSSD may contribute to weight gain because they contain easily absorbable
carbohydrates (9, 10) and because of the lower satiety associated with the intake of
74 liquid foods. In addition, cola-based soft drinks contain caramel rich in glycated end-
products that might increase insulin resistance (9, 11). Two small (n=41 and n=30)
76 experimental trials evidenced that increased consumption of SSSD led to weight gain
(12, 13). Additionally, there has also been an increase in the consumption of
78 sweetened fruit juices (4, 14). Sweetened fruit juices might promote weight gain if
they are drunk in large amounts but the evidence is very scarce (11, 15). On the
80 other hand, the role of meat consumption in obesity remains not clear (16).

We have previously reported a trend towards the adoption of a typical Westernized
82 dietary pattern (rich in red meat) (17, 18) in Spanish university graduates, especially
among younger males, with frequent consumption of fast foods (19). Fast foods are
84 very dense in energy, they are consumed in greater than needed portion sizes, are
highly palatable and rich in saturated fats, refined carbohydrates and added sugars,
86 but poor in dietary fiber. Several recent epidemiologic studies have reported that,

because of all the characteristics of fast foods, fast-food consumption may be a risk
88 factor for weight gain and might have contributed to the current obesity epidemic in
the United States (6, 20).

90 Observed changes in obesity have not been so dramatic in Europe as in the US, but
they are following step by step the same trend (21, 22).

92 Few studies have investigated simultaneously the effects of the consumption of
SSSD and fast food on weight gain. Moreover, no epidemiological studies have
94 assessed this issue in European countries. Recent longitudinal studies conducted in
US have reported that SSSD are associated with increased weight gain or obesity
96 among adult women (11) or children (23, 24). Similar associations have been
reported for fast food consumption (6, 20). If these findings are also confirmed in
98 European countries, the scientific evidence to support public health actions will be
strengthened. In addition, a greater opportunity for large-scale preventive
100 interventions with adequate timeliness will be available in Europe before the rates of
obesity reach the levels seen in the United States. We investigated the association
102 between the consumption of SSSD or the sum of hamburgers, pizza and sausages
(HPS) as indicator for fast food and weight change after 28.5 months of follow-up in a
104 Spanish cohort.

106 SUBJECTS AND METHODS**Study population**

108 The SUN Project (*Seguimiento Universidad de Navarra*- Follow-up University of
110 Navarra) is a prospective cohort study designed to establish the association between
112 diet and the occurrence of several diseases and chronic conditions including
114 overweight, obesity or weight change over time (25). The SUN cohort was designed
116 in collaboration with the Harvard School of Public Health using similar methodology
118 as that of large American cohorts such as the Nurses' Health Study or the Health
120 Professionals Follow-up Study (25).

Information is collected using self-administered questionnaires sent by postal mail
116 every two years. The follow-up rate exceeds 90% for the first 2-year period. The
118 recruitment of participants started in December 1999 and it is permanently on-going
120 as this is a dynamic cohort study. All participants are university graduates. The data
122 set of the SUN Project incorporated 17,170 participants up to December 2004. For
124 the present analyses we included participants who had already been followed-up for
126 at least 2 years. Those participants who reported extremely low or high values for
total energy intake (less than 800 Kcal/day in men and 600 Kcal/day in women or
more than 4,200 Kcal/day in men and 3,500 Kcal/day in women), participants with
missing values in the variables of interest in the analysis and participants with
biological implausible values for the weight or height were excluded. Finally, data
from 7,194 participants remained available for the analysis.

The study was approved by the Human Research Ethical Committee at the
128 University of Navarra. Voluntary completion of the first self-administered
questionnaire was considered to imply informed consent.

130 Assessment of dietary exposure

The dietary exposure was ascertained through a semi-quantitative food frequency
132 questionnaire (136 food items) previously validated in Spain (26). This questionnaire
was included in the general baseline assessment for each participant (Q_0,
134 hereafter).

Nutrient scores were calculated as frequency x nutrient composition of specified
136 portion size where frequencies were measured in nine categories (6+ per day/ 4-6
per day/ 2-3 per day/ 1 per day/ 5-6 per week/ 2-4 per week/ 1 per week/ 1-3 per
138 month/ never or almost never) for each food item. Data of food intake were
transformed into ml or g of food consumption. Nutrient intake scores were computed
140 using an *ad hoc* computer program specifically developed for this aim. A trained
dietitian updated the nutrient data bank using the latest available information included
142 in food composition tables for Spain (27, 28). We used separated items to assess the
consumption of SSSD and of diet (sugar-free) carbonated soda drinks. We analyzed
144 the consumption of SSSD as the main independent variable in our analyses. Milk
consumption was assessed using three different items in the questionnaire: whole
146 milk, low-fat milk and non-fat milk. We estimated total milk consumption per person
using the sum of the three items (whole milk + low-fat milk + non-fat milk). The
148 indicator for fast food consumption (HPS, g/day) was estimated as the sum of 3 items
in the food frequency questionnaire (hamburgers, pizza and sausages). All dietary
150 intakes were adjusted for total energy intake using the residuals method (29).

Assessment of non-dietary variables

152 The baseline assessment (Q_0) also included other questionnaires (totaling 46 items
for men and 54 items for women) to assess medical history, health habits, life-style
154 and socio-demographic variables. Participants were classified as never-smokers,
former smokers and current smokers. A physical activity questionnaire was also

156 completed at baseline by each participant including 17 activities. To quantify the
volume of activity during leisure time, an activity metabolic equivalent (MET) index
158 was computed by assigning a multiple of resting metabolic rate (MET score) to each
activity (30), and the time spent in each of the activities was multiplied by the MET
160 score specific to each activity, and then summed over all activities obtaining a value
of overall weekly MET-hours that has been shown to adequately correlate
162 (Spearman rho = + 0.51; p= 0.002) with objectively measured energy expenditure in
a validation subsample of our cohort (31).

164 **Assessment of the outcome**

Information on weight was collected at baseline and in the follow-up questionnaire
166 (Q_2, hereafter) which was completed by participants after at least two years from
baseline (median follow-up time = 28.5 months). The reproducibility and validity of
168 the self-reported weight was assessed in a representative subsample of the cohort.
The mean relative error in self-reported weight was 1.45 per cent, and the correlation
170 coefficient between measured and self-reported weight was 0.99 (95% CI: 0.98-
0.99).

172 The outcomes were: 1) any increase in body weight during follow-up categorized as
a dichotomous variable (cut-off point ≥ 1 kg); 2) change in body weight during follow-
174 up as a continuous variable [weight in Q_2 *minus* weight in Q_0 (Kg)].

The baseline evaluation for each participant also collected information regarding
176 his/her weight change in the five-year period prior to our baseline assessment. This
information (previous weight gain) was not considered as an outcome, but only as a
178 stratifying variable, dichotomized at the cut-off point of ≥ 3 Kg.

Statistical analysis

180 Non-conditional logistic regression models were fit to assess the relationship
between the consumption of SSSD or HPS and the risk of any weight gain during

182 follow-up. Odds ratios (OR) and their 95% confidence intervals (CI) were calculated
considering the lowest quintile of consumption as the reference category. Tests of
184 linear trend across increasing quintiles of consumption were conducted by assigning
the medians of intake for each quintile and treating the intake as a continuous
186 variable. Linear regression models were used to assess the association between the
consumption of SSSD or HPS (exposure, categorized as quintiles for both groups)
188 and change in body weight during follow-up (outcome, considered as a continuous
variable). We estimated the beta regression coefficients (and their 95% CI) for each
190 of the four upper quintiles of consumption considering the lowest quintile as the
reference category. These coefficients represent the absolute difference in weight
192 gain between each of the four upper quintiles and the lowest quintile.

For each exposure (SSSD or HPS) we fit a crude (univariate) model, an age- and
194 gender-adjusted model and a multivariate model after also adjusting for the following
variables: baseline energy intake (Kcal/day) from non-SSSD sources (or non-fast
196 food sources), energy-adjusted fiber intake (g/day), energy-adjusted alcohol intake
(g/day), energy-adjusted milk consumption (ml/day), leisure-time physical activity
198 (METs-hour/week), smoking status (never smoker, smoker and former smoker),
snacking between meals (yes/no), TV watching (h/week) and baseline weight (Kg).
200 We also conducted similar analyses to assess the association between other food
groups (24 groups in total) and weight gain.

202 We used a product-term included in the fully adjusted models to appraise the
interaction (effect modification) between the previous change in body weight (i.e.
204 during the five-year period prior to our baseline assessment) and the consumption of
SSSD (or of HPS) on subsequent weight gain (during follow-up). When the
206 coefficient for this product-term was statistically significant (or at least approached
statistical significance, $p < 0.10$) we performed separate analyses for participants who

208 had increased their weight during the previous five years (at least 3 kg) and for
those who had not.

210 The estimated basal metabolic rate was calculated according to the WHO equation
separately for gender and age groups (**32**). We repeated the main analyses after

212 excluding individuals with an energy intake to basal metabolic rate ratio < 1.2. This
low ratio is rare; therefore, such values likely reflect underreporting of dietary intakes

214 (**33**)

All p values presented are 2-tailed; $p < 0.05$ was considered statistically significant,

216 unless otherwise specified. Analyses were performed with SPSS version 12.0.1
(SPSS Inc, Chicago, IL).

218

220

RESULTS

222 During follow-up we observed that 49.5% of our study population experienced some
increase in their body weight. The mean change in body weight was +0.64 Kg (95%
224 CI: +0.55 to +0.73 Kg).

Table 1 shows the characteristics of participants according to quintiles of
226 consumption of SSSD. We stratified this table according to whether participants had
increased or not their weight (≥ 3 Kg) during the five-year period prior to our baseline
228 assessment. The consumption of SSSD was higher in men and in younger
participants. Baseline body weight increased in parallel with soft drink consumption.
230 Participants with a higher consumption of SSSD showed a lower intake of fiber, lower
milk consumption, but a higher consumption of fast food and a higher frequency of
232 between-meals snacking.

When we analyzed the association between the consumption of SSSD and weight
234 gain during follow-up, the estimates suggested an interaction (p for interaction
product-term = 0.07 in the logistic regression model and $p = 0.006$ in the linear
236 model) between the consumption of SSSD and previous weight gain. Consequently,
we split the data base according to whether participants have gained or not weight in
238 the previous five years (cut-off ≥ 3 Kg) and analyzed separately for both groups the
relationship between SSSD and subsequent weight gain (table 2). The lowest
240 proportion of participants who have gained weight was found in the lowest quintile for
the consumption of SSSD. Only among participants who reported weight gain (≥ 3 Kg)
242 during the previous 5 years we observed increasing risk of weight gain associated
with higher SSSD consumption when we adjusted for age and gender [ORs across
244 quintiles 1-5: 1.00 (reference), 1.23, 1.09, 1.23, 1.43; p for trend = 0.017 (Table 2)].
When we further adjusted for a set of potential dietary and non-dietary confounders,
246 we found a 56 percent increase in the incidence of weight gain for the fifth versus the

first quintile of SSSD consumption (multivariate-adjusted OR: 1.56; 95% CI: 1.17, 2.09). These estimates did not materially change after additional adjustment for HPS consumption and the p for trend remained statistically significant ($p=0.02$).

Among participants who did not report weight gain during the previous 5 years, the observed association between the consumption of SSSD and weight gain was of lower magnitude and suggested a threshold (Table 2). However, we did not find a significant p for trend in any model.

When we analyzed the magnitude of weight gain (outcome) as a continuous variable using linear models to assess its association with the consumption of SSSD (exposure, categorized in quintiles), we also split the sample according to previous weight gain (p for interaction product-term = 0.006). Among participants who had previously increased their body weight, a higher consumption of SSSD (4th and 5th quintiles) was associated with a significantly increased weight gain with respect to the first quintile as crude beta regression coefficients showed (fourth quintile: +0.7 Kg; fifth quintile: +0.9 Kg). These associations remained statistically significant (p for trend = 0.001) after adjusting for age and gender, and when we also adjusted for other potential confounders [beta for the fourth quintile = +1.0 Kg (95% CI: +0.4 to +1.6 kg); fifth quintile: +1.2 Kg (95% CI: +0.6 to +1.8 Kg)]. Additional adjustment for HPS consumption did not materially affect the results. On the other hand, among participants who had not experienced weight gain during the previous period to our baseline assessment, these associations were absent.

Table 3 shows the results of logistic regression analyses conducted to assess the association between HPS consumption and weight gain during follow-up. We observed the highest risk of weight gain among participants in the top quintile of HPS consumption (OR: 1.2; 95% CI: 1.1, 1.4; p for trend 0.004). This association remained statistically significant after adjusting for age and gender and also after

further adjustment for dietary and non-dietary confounders, including SSSD

274 consumption. No significant interaction was observed between the consumption of
HPS and weight gain in the previous period to our study (p interaction product-term =
276 0.41).

When we assessed the association between quintiles of HPS consumption and the
278 magnitude of weight change during follow-up, we also observed that participants in
the two upper quintiles of HPS consumption exhibited a significantly greater weight
280 gain (mean weight change during follow-up: +0.78 and +0.77 kg versus +0.47 kg in
the first quintile; p for trend: 0.014) during follow-up. When we used linear regression
282 modeling to assess the independent association between consumption of HPS and
the magnitude of weight change during follow-up these differences were attenuated
284 after multivariate adjustment (adjusted weight gain for quintiles 4 and 5 = +0.71 and
+0.64 kg respectively) and they did not remain statistically significant.

286 When we repeated the analyses excluding under-reporters (i.e. participants with a
basal metabolic rate ratio < 1.2) (**33**), we did not find any substantial difference with
288 respect to the main results shown in our tables.

When we analyzed all food items included in our food-frequency questionnaire
290 classifying them in 24 groups (legumes, pasta, cereals, biscuits, wholemeal bread,
commercial bakery, chocolate, nuts, fruits and vegetables, sweetened fruit juices,
292 SSSD, diet-sodas, dairy products, alcohol beverages, eggs, fish, lean meats, red
meats, HPS, cold/cured meats, pre-cooked meals, sauces, animal fats, and
294 vegetable oils), we found that apart from SSSD and HPS, only 2 other food groups
were significantly dose-response associated with a higher risk of weight gain (red
296 meats and sweetened fruit juices), nevertheless these associations were weaker and
even for fruit juices consumption the odds ratio did not reach statistical significance,
298 although the p for trend was 0.04 (tables 4, 5). The crude ORs for the fifth versus the

first quintile of red meat consumption was 1.16 (95% CI: 1.00, 1.36). Nevertheless,
300 the OR for the fifth quintile did not remain statistically significant after multivariate
adjustment, the p for trend was 0.004 (table 4). These two groups did not confound
302 our estimates for SSSD or HPS consumption.

DISCUSSION

304 We observed that an increase in consumption of SSSD was associated with weight
gain after 28.5-month follow-up in 7,194 university participants of a Mediterranean
306 cohort. This association was only apparent among those participants who had
reported a previous weight gain (≥ 3 kg) before our baseline assessment (during the
308 previous 5 years). This differential effect could be attributed to other lifestyle or
genetic factors occurring in participants who had not recently gained weight that
310 render them less susceptible to subsequent weight gain associated with the
consumption of SSSD. Moreover, the consumption of SSSD was higher in absolute
312 terms among participants who did gain weight in the previous 5 years, thus
supporting this interpretation. The consumption of HPS (as a proxy for fast food) was
314 also associated with higher weight gain, independently of the consumption of SSSD
and of previous weight gain.

316 Our findings represent a reason for concern regarding the global epidemic of obesity
because they suggest that the adverse association between SSSD (or fast food) and
318 weight gain reported in the US (**6**, **11**, **23**) is beginning to be evidenced also among
educated young adults in a Mediterranean country.

320 Apparently, calories that are consumed as liquid carbohydrates do not fully displace
those that are consumed from solid sources and may, in fact, lead to increased
322 consumption of other foods (**12**, **34**). A meta-analysis of studies conducted during a
25-year period demonstrated that humans compensate poorly for calories consumed
324 in liquid form (**35**). Thus, overconsumption of SSSD could be a particular problem
because it could represent an extra source of energy ingested in liquid form. Some
326 similar effect can be expected with sweetened fruit juices. On the other hand, the
similar estimates before and after multivariate adjustment that we report in our table
328 2 may suggest that a higher total energy intake from other sources is not necessarily

the single mechanism by which overconsumption of SSSD leads to overweight. In
330 fact, this similarity between crude and adjusted estimates may indicate that the main
associations we report here may be independent of the influence of the other foods
332 that we took into account in the multivariate models. Nonetheless, we also
acknowledge that our estimates of total energy intake or of the consumption of some
334 foods, as measured by the food frequency questionnaire, may present some degree
of measurement error as it seems logical to be expected in nutritional epidemiology
336 (26, 29). In any case, it is important not to forget that, independently of the additional
increase in energy intake that they represent, SSSD and many fast foods are
338 basically fiber-free foods and, therefore, they lose the advantages of a fiber-rich diet
(such as the traditional Mediterranean diet) has to prevent weight gain. A wide array
340 of evidence suggests that fiber may exert this beneficial role through several
mechanisms, including effects on satiety, glucose metabolism, energy density, and
342 rate of ingestion and gastric emptying (24, 36-42). In fact, we found in our study, a
strong inverse correlation between the consumption of SSSD and fiber intake, thus
344 supporting these mechanistic explanations.

Beyond the mechanisms related to a poorer fiber intake, the quality of sugar in SSSD
346 should be taken also into account. High fructose corn syrup has been reported to
represent > 40 percent of caloric sweeteners added to foods and beverages and it is
348 the sole caloric sweetener in SSSD (9). The digestion, absorption, and metabolism of
fructose differ from that of glucose. Hepatic metabolism of fructose favors de novo
350 lipogenesis. In addition, unlike glucose, fructose does not stimulate insulin secretion
nor enhance leptin production, whereas it may promote insulin resistance (9,10).
352 However, disagreement exists about whether fructose has an adverse effect on body
weight relative to glucose and even some authors have reported that fructose intake
354 may have the opposite effect inducing a reduction in total food intake (43, 44).

Other potential mechanism to explain the association between SSSD and weight gain is related to the inverse correlation between SSSD and dairy products. We observed this inverse trend in our study ($p < 0.001$, see table 1). This finding is consistent with the observed decrease in the intake of dairy products and an increase in the trend of soda consumption that has been repeatedly reported, specially in children and adolescents (45-47). Also an inverse association between total milk consumption and weight gain was found in our cohort (data not shown). This inverse association between dairy products (or total calcium intake) and overweight/obesity has been also reported in several other studies, which have claimed dairy products as a possible protector factor against obesity (48, 49). Although residual confounding cannot be completely ruled out, when we adjusted for milk consumption the association of SSSD with weight gain did not materially change.

When we explored all food categories in our questionnaire, classifying food items in 24 groups, we found a significantly higher risk of weight gain for only 4 groups: HPS, red meats, SSSD and sweetened fruit juices. The available scientific literature on the relation between red meat or sweetened fruit juices and weight gain is not abundant. However, our data are consistent with other studies (50, 51, 11). Newby et al. found in a longitudinal study that a diet low in red and processed meat, fast food, and soda was associated with smaller gains in body mass index and waist circumference in the ongoing Baltimore Longitudinal Study of Aging (50). In a cross-sectional study, Dennison et al reported that obesity was more common among children drinking 12 fl oz/day of juice compared with those drinking less juice (51). Schulze et al determined that an increase in fruit punch consumption was associated with greater weight gain in the Nurses' Health Study II (11). The fact that only two other food categories were associated with weight gain supports that HPS consumption and SSSD play an important role as predictors of weight gain in our cohort. Sweetened juices and red

meat are similar in their composition and other aspects to our target food-groups
382 (SSSD, HPS) respectively. Thus, their significant association with weight gain
provides additional biological support to our major findings.

384 The strengths of our study include the prospective design that avoid the possible
effect of inverse causality in the reported associations, the previous validation of the
386 methods used for the measurement of the main variables considered in the analyses,
the use of a highly educated cohort in a different culture and dietary environment that
388 had been previously studied (6, 11, 23, 24), the large sample size and the control for
an important number of potential confounders. Although we cannot rule out the
390 existence of unmeasured confounders, we have included in our analysis the main
important known risk factors for weight gain.

392 Imprecise dietary measurement could potentially have influenced our observed
associations leaving room for residual confounding and imprecise adjustment of our
394 estimates. However, random errors of classification or residual confounding in our
analyses might have accounted for a lack of association but not the reverse.

396 Our indicator for fast food consumption (HPS) was the sum of three food items
(hamburgers, pizza and sausages). We used this sum as a proxy variable for
398 categorizing exposure. We acknowledge this limitation of our assessment and it
could be possible that the hamburgers, pizza and sausages consumed at home may
400 have a healthier composition than the same food items consumed in canteen meals
or fast food restaurants. This fact may hinder our ability to find stronger associations.
402 However, in a previous study of our cohort, using factor analysis, we identified the
sum of these three food items as one of the groups that better explained the total
404 variance in the adherence to a Westernized dietary pattern (19).

We did not control for confounding due to socio economic status (SES), but we are
406 confident that SES of the participants cannot be a major confounder in our analyses

because our cohort is very homogeneous regarding SES. A recommended
408 procedure to adjust for SES is to adjust for educational level, but all our participants
have attained the same educational level (i.e. we have used restriction instead of
410 multivariate adjustment to control for confounding by SES).

Even if an absolute proof of causality cannot be firmly established when it comes
412 from an observational design, it seems clear from our results, in any case, that SSSD
or fast food consumption are at least markers of an unhealthy dietary pattern that
414 may contribute to the growing epidemic of overweight or obesity. The optimistic side
of our findings is that this dietary pattern has the potential of being used as a quick
416 screening test for increased risk of overweight/obesity (**52**).

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All authors have edited and reviewed critically the manuscript.

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TABLES

Table 1. Characteristics of the participants according to weight gain and to quintiles (Q)¹ of sugar-sweetened soft drink (SSSD) consumption.

	WEIGHT GAIN <3 KG IN THE PREVIOUS 5 YEARS²					WEIGHT GAIN ≥ 3 KG IN THE PREVIOUS 5 YEARS²					<u>p for trend³</u>
	Quintiles of SSSD consumption (1 to 5)					Quintiles of SSSD consumption (1 to 5)					
	Q1	Q2	Q3	Q4	Q5	Q1	Q2	Q3	Q4	Q5	
Participants (n)	974	975	975	975	975	464	464	464	464	464	
SSSD consumption (ml/day, limits)	<4.0	4.0 - 14.8	14.9 - 27.8	27.9 - 80.2	≥ 80.3	< 5.5	5.5 - 17.6	17.7 - 33.3	33.4 - 87.4	≥ 87.5	
Age (years)	40.6 (13)	38.1 (13)	36.6 (12)	35.0 (12)	32.3 (10)	41.0 (12)	39.2(12)	37.1 (11)	33.7 (10)	32.8 (10)	<0.001
Men (%)	37	27	36	47	40	40	35	49	61	62	<0.001
Baseline weight (Kg)	63.3 (12)	61.8 (11)	63.8 (12)	65.5 (13)	64.4 (13)	70.2 (14)	70.5 (14)	72.6 (14)	74.0 (13)	74.5 (14)	<0.001
Physical activity (METS-h/week)	22.1 (23)	19.4 (23)	17.2 (19)	19.2 (21)	18.6 (24)	18.9 (24)	16.6 (21)	18.3 (20)	16.7 (20)	18.7 (23)	<0.001
TV watching (h/week)	10.3 (6)	10.3 (6)	10.5 (6)	10.3 (6)	9.6 (6)	10.0 (6)	10.2 (6)	10.2 (6)	9.8 (6)	9.7(6)	<0.001
Current smokers (%)	20	24	24	28	32	22	21	23	25	30	<0.001

Table 1 (continued). Characteristics of the participants according to weight gain and to quintiles (Q)¹ of sugar-sweetened soft drink (SSSD) consumption.

Former smokers (%)	30	26	24	21	19	34	37	29	23	24	<0.001
Energy intake from non-SSSD sources (Kcal/day)	3,306.7 (465)	2,404.3 (571)	2,230.8 (560)	2,388.1 (700)	2,502.2 (563)	3,243.1 (495)	2,356.8 (572)	2,205.5 (543)	2,593.4 (722)	2,563.4 (647)	<0.001
Total Fat intake(%E)	36.0 (7)	36.2 (7)	37.2 (7)	37.7 (7)	38.0 (7)	36.9 (7)	37.4 (7)	37.6 (7)	38.3 (7)	37.6 (7)	<0.001
MUFA(%E)	15.6 (4)	15.7 (4)	16.0 (4)	16.1 (4)	16.2 (4)	16.0 (4)	16.4 (4)	16.2 (4)	16.3 (3)	16.0 (3)	0.002
SFA (%E)	11.9 (4)	12.3 (3)	12.7 (3)	13.1 (3)	13.3 (3)	12.4 (3)	12.7 (3)	13.2 (3)	13.4 (3)	13.4 (3)	<0.001
PUFA (%E)	5.4 (2)	5.1 (2)	5.3 (2)	5.5 (2)	5.6 (2)	5.4 (2)	5.3 (2)	5.4 (2)	5.7 (2)	5.4 (2)	<0.001
CHO intake (%E)	45.5 (8)	43.8 (8)	42.8 (7)	42.5 (7)	43.0 (7)	43.8 (8)	41.9 (8)	41.9 (8)	41.9 (7)	42.7 (7)	<0.001
Protein intake (%E)	16.8 (3)	18.3 (3)	18.4 (3)	18.0 (4)	17.1 (3)	17.4 (3)	18.4 (3)	18.3 (4)	17.4 (3)	17.2 (3)	<0.001
Fiber intake (g/day)	42.2 (12)	29.6 (13)	25.8 (11)	26.1 (13)	25.5 (12)	38.4 (12)	27.1 (10)	24.1 (10)	25.5 (13)	23.6 (11)	<0.001
Milk consumption (ml/day)	348.8 (294)	300.2 (242)	282.6 (228)	267.2 (228)	277.8 (228)	350.9 (276)	262.4 (222)	265.9 (198)	281.1 (227)	264.0 (213)	<0.001
HPS consumption (g/day)	17.9 (21)	17.0 (23)	19.1 (18)	22.5 (20)	26.1 (23)	21.7 (27)	18.0 (18)	20.2 (18)	28.4 (27)	29.9 (27)	<0.001
Snacking between meals (%)	32	30	28	31	37	38	41	42	46	50	<0.001

Footnote Table 1.

Data are expressed as means (standard deviation) unless otherwise indicated.

¹ Q1-Q5: Lowest to highest quintile for the consumption of SSSD.

² p for interaction (product-term) between consumption of SSSD and weight gain in the previous 5 years = 0.006 in the linear multivariate adjusted model (outcome: prospective weight gain) and p= 0.07 in the non conditional logistic regression adjusted model (outcome: prospective put on weight).

There was no significant interaction between consumption of SSSD and weight gain in the previous 5 years for any of the variables reported in the table.

³ Linear regression model with median values as exposure for continuous variables, linear-trend chi square for categorical variables.

% E: Percentage of energy

HPS: Hamburger, pizza and sausages; MUFA: Monounsaturated fatty acids; SFA: Saturated fatty acids; PUFA: Polyunsaturated fatty acids; CHO: Carbohydrates

Table 2. Odds Ratios¹ (ORs) for any weight gain (cut-off point ≥ 1 kg) during follow-up according to increasing quintiles (Q)² of sugar-sweetened soft drink (SSSD) consumption.

	Baseline consumption of SSSD (quintiles 1 to 5)					
	Q1	Q2	Q3	Q4	Q5	p for trend
<u>Weight gain ≥ 3 kg in the previous 5 years</u>						
Participants (n)	464	464	464	464	464	
Consumption of SSSD (ml/day, limits)	<5.5	5.5 - 17.6	17.7 - 33.3	33.4 - 87.4	≥ 87.5	
Weight gain during follow-up (%)	41	46	43	46	50	
Crude OR ⁴	1.00	1.21	1.07	1.20	1.39	0.019
(95% CI)	(ref.)	(0.94, 1.57)	(0.83, 1.39)	(0.93, 1.56)	(1.07, 1.81)	
Age and gender-adjusted OR ⁵	1.00	1.23	1.09	1.23	1.43	0.017
(95% CI)	(ref.)	(0.95, 1.59)	(0.84, 1.41)	(0.94, 1.60)	(1.09, 1.87)	
Multivariate-adjusted ³ OR ⁶	1.00	1.37	1.24	1.35	1.56	0.015
(95% CI)	(ref.)	(1.04, 1.81)	(0.93, 1.65)	(1.01, 1.79)	(1.17, 2.09)	
Additionally-adjusted for HPS consumption ⁷ (95% CI)	1.00	1.37	1.24	1.34	1.55	0.018
	(ref.)	(1.04, 1.81)	(0.93, 1.64)	(1.01, 1.78)	(1.16, 2.07)	
<u>Weight gain <3 kg in the previous 5 years</u>						
Participants (n)	974	975	975	975	975	
Consumption of SSSD (ml/day, limits)	<4.0	4.0 - 14.8	14.9 - 27.8	27.9 - 80.2	≥ 80.3	
Weight gain during follow-up (%)	48	54	53	49	53	
Crude OR ⁴	1.00	1.26	1.23	1.05	1.20	0.377
(95% CI)	(ref.)	(1.05, 1.50)	(1.03, 1.46)	(0.88, 1.26)	(1.01, 1.43)	
Age and gender-adjusted OR ⁵	1.00	1.24	1.18	0.99	1.11	0.996
(95% CI)	(ref.)	(1.04, 1.48)	(0.99, 1.42)	(0.83, 1.19)	(0.92, 1.33)	

Table 2 (continued). Odds Ratios¹ (ORs) for any weight gain (cut-off point ≥ 1 kg) during follow-up according to increasing quintiles (Q)² of sugar-sweetened soft drink (SSSD) consumption.

Multivariate-adjusted ³ OR ⁶	1.00	1.25	1.23	1.01	1.10	0.724
(95% CI)	(ref.)	(1.03, 1.51)	(1.01, 1.50)	(0.83, 1.23)	(0.91, 1.34)	
Additionally-adjusted for HPS consumption ⁷	1.00	1.24	1.21	0.99	1.08	0.611
(95% CI)	(ref.)	(1.02, 1.50)	(1.00, 1.48)	(0.82, 1.21)	(0.89, 1.32)	

95% CI: 95% Confidence Interval.

HPS: Hamburgers, pizza and sausages.

¹ Statistical test: Non-conditional Logistic Regression.

² Q1-Q5: Lowest to highest quintile for the consumption of SSSD.

³ Adjusted for age (years), gender, total energy intake from non-SSSD sources (Kcal/day), fiber intake (g/day), alcohol intake (g/day), milk consumption (ml/day), leisure-time physical activity (METs-hour/week), smoking status (never smoker, smoker and former smoker), snacking (yes/no), TV watching (h/week), baseline weight (Kg). An interaction term (age*gender, $p < 0.001$) was also added.

⁴ p for interaction (product-term) in the crude model between consumption of SSSD and weight gain in the previous 5 years = 0.14

⁵ p for interaction (product-term) in the age and gender-adjusted model between consumption of SSSD and weight gain in the previous 5 years = 0.05

⁶ p for interaction (product-term) in the multivariate-adjusted model between consumption of SSSD and weight gain in the previous 5 years = 0.07

⁷ p for interaction (product-term) in the additionally-adjusted for HPS model between consumption of SSSD and weight gain in the previous 5 years = 0.06

Table 3. Odds Ratios¹ (ORs) for any weight gain (cut-off point ≥ 1 kg) during follow-up according to increasing quintiles (Q)² of HPS (Hamburgers, pizza and sausages) consumption.

	Baseline consumption of HPS (quintiles 1 to 5)					p for trend
	Q1	Q2	Q3	Q4	Q5	
Participants (n)	1,438	1,439	1,439	1,439	1,439	
Fast food consumption (g/day, limits)	< 3.3	3.3 - 13.2	13.3 - 23.2	23.3 - 34.7	≥ 34.8	
Weight gain during follow-up (%)	47	49	51	50	52	
Crude OR	1.00	1.12	1.19	1.18	1.24	0.004
(95% CI)	(ref.)	(0.96, 1.29)	(1.03, 1.38)	(1.02, 1.37)	(1.08, 1.44)	
Age and gender-adjusted OR	1.00	1.11	1.17	1.15	1.20	0.030
(95% CI)	(ref.)	(0.96, 1.29)	(1.00, 1.35)	(0.99, 1.33)	(1.03, 1.40)	
Multivariate-adjusted ³ OR	1.00	1.13	1.19	1.17	1.21	0.040
(95% CI)	(ref.)	(0.97, 1.32)	(1.02, 1.39)	(1.00, 1.37)	(1.03, 1.42)	
Additionally adjusted for SSSD (95% CI)	1.00	1.15	1.20	1.18	1.20	0.053
(95% CI)	(ref.)	(0.98, 1.33)	(1.03, 1.40)	(1.01, 1.38)	(1.02, 1.41)	

95% CI: 95% Confidence Interval.

SSSD: Sugar-sweetened soft drinks.

¹ Statistical test used: Non-conditional Logistic Regression.

² Q1-Q5: Lowest to highest quintile of energy-adjusted baseline fast food consumption.

³ Adjusted for age (years), gender, total energy intake from non-fast food-sources (Kcal/day), fiber intake (g/day), alcohol intake (g/day), leisure-time physical activity (METs-hour/week), smoking status (never smoker, smoker and former smoker), snacking (yes/no), TV watching (h/week), baseline weight (Kg), weigh gain ≥ 3 Kg / last 5 years (yes/no). An interaction term (age*gender, $p < 0.001$) was also added.

Table 4. Odds Ratios¹ (ORs) for any weight gain (cut-off point ≥ 1 kg) during follow-up according to increasing quintiles (Q)² of red meat consumption.

	Baseline consumption of red meat (quintiles 1 to 5)					p for trend
	Q1	Q2	Q3	Q4	Q5	
Participants (n)	1,438	1,439	1,439	1,439	1,439	
Red meat consumption (g/day, limits)	< 42.9	43.0-64.3	64.4-92.4	92.3-128.6	≥ 128.7	
Weight gain during follow-up (%)	49	46	49	52	53	
Crude OR	1.00	0.88	1.01	1.12	1.16	0.002
(95% CI)	(ref.)	(0.76-1.03)	(0.87-1.18)	(0.96-1.30)	(1.00-1.36)	
Age and gender-adjusted OR	1.00	0.88	1.01	1.11	1.16	0.003
(95% CI)	(ref.)	(0.75-1.03)	(0.86-1.18)	(0.96-1.30)	(0.99-1.35)	
Multivariate-adjusted ³ OR	1.00	0.88	1.01	1.12	1.16	0.004
(95% CI)	(ref.)	(0.75-1.03)	(0.86-1.18)	(0.95-1.31)	(0.99-1.36)	

95% CI: 95% Confidence Interval

¹ Statistical test used: Non-conditional Logistic Regression

² Q1-Q5: Lowest to highest quintile of energy-adjusted baseline red meat consumption.

³ Adjusted for age (years), gender, total energy intake from non-fastn food-sources (Kcal/day), fiber intake (g/day), alcohol intake (g/day), leisure-time physical activity (METs-hour/week), smoking status (never smoker, smoker and former smoker), snacking (yes/no), TV watching (h/week), baseline weight (Kg), weigh gain ≥ 3 Kg / last 5 years (yes/no). An interaction term (age*gender, $p < 0.001$) was also added.

Table 5. Odds Ratios¹ (ORs) for any weight gain (cut-off point ≥ 1 kg) during follow-up according to increasing quintiles (Q)² of sweetened_ fruit juice consumption.

	Baseline consumption of sweetened fruit juice (quintiles 1 to 5)					p for trend
	Q1	Q2	Q3	Q4	Q5	
Participants (n)	1,438	1,439	1,439	1,439	1,439	
Fruit juice consumption (g/day, limits)	<13.3	13.3-26.7	26.8-57.1	57.2-185.7	≥ 185.8	
Weight gain during follow-up (%)	48	48	50	50	52	
Crude OR (95% CI)	1.00 (ref.)	0.98 (0.84, 1.14)	1.07 (0.92, 1.24)	1.06 (0.91, 1.24)	1.15 (0.98, 1.34)	0.042
Age and gender-adjusted OR (95% CI)	1.00 (ref.)	0.98 (0.84, 1.14)	1.06 (0.91, 1.24)	1.05 (0.90, 1.23)	1.14 (0.98, 1.33)	0.049
Multivariate-adjusted ³ OR (95% CI)	1.00 (ref.)	0.99 (0.84, 1.17)	1.08 (0.91, 1.28)	1.08 (0.92, 1.27)	1.16 (0.99, 1.36)	0.039

95% CI: 95% Confidence Interval

¹ Statistical test used: Non-conditional Logistic Regression

² Q1-Q5: Lowest to highest quintile of energy-adjusted baseline fruit juice consumption.

³ Adjusted for age (years), gender, total energy intake from non-fast food-sources (Kcal/day), fiber intake (g/day), alcohol intake (g/day), leisure-time physical activity (METs-hour/week), smoking status (never smoker, smoker and former smoker), snacking (yes/no), TV watching (h/week), baseline weight (Kg), weigh gain ≥ 3 Kg / last 5 years (yes/no). An interaction term (age*gender, $p < 0.001$) was also added.