González-Gil, E., Huybrechts, I., Nutrición, CIBER. F. D. L. O., Béghin, L., Breidenassel, C., Gesteiro, E., González-Gross, M., De Henauw, S., Kersting, M., Le Donne, C., Manios, Y., Marcos, A., Meirhaeghe, A., De Miguel-Etayo, P., Molina-Hidalgo, C., Molnár, D., Papadaki, A., Widhalm, K., Moreno, L. A., & Bel-Serrat, S. (2021). Cardiometabolic Risk is Positively Associated with Underreporting and Inversely Associated with Overreporting of Energy Intake Among European Adolescents: The Healthy Lifestyle in Europe by Nutrition in Adolescence (HELENA) Study. *Journal of Nutrition*, 151(3), 675-684. <https://doi.org/10.1093/jn/nxaa389>

Peer reviewed version

Link to published version (if available):
[10.1093/jn/nxaa389](https://doi.org/10.1093/jn/nxaa389)

[Link to publication record in Explore Bristol Research](#)
PDF-document

This is the author accepted manuscript (AAM). The final published version (version of record) is available online via Oxford University Press at <https://doi.org/10.1093/jn/nxaa389>. Please refer to any applicable terms of use of the publisher.

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Cardiometabolic risk is positively associated with under-reporting and inversely associated with over-reporting of energy intake among European adolescents: The HELENA study

Esther M González-Gil^{1,2,3}, Inge Huybrechts⁴, Concepción M. Aguilera^{1,3}, Laurent Béghin⁵, Christina Breidenassel^{6,7}, Eva Gesteiro⁶, Marcela González-Gross^{3,6,7}, Stefaan de Henauw⁸, Mathilde Kersting⁹, Cinzia Le Donne¹⁰, Yannis Manios¹¹, Ascensión Marcos¹², Aline Meirhaeghe¹³, Pilar De Miguel-Etayo^{2,3,14}, Cristina Molina-Hidalgo¹⁵, Dénes Molnár¹⁶, Angeliki Papadaki^{17,18}, Kurt Widhalm¹⁹, Luis A. Moreno^{2,3,14*}, Silvia Bel-Serrat^{2,20}

¹Department of Biochemistry and Molecular Biology II, Instituto de Nutrición y Tecnología de los Alimentos, Center of Biomedical Research (CIBM), Universidad de Granada.

²Growth, Exercise, Nutrition and Development (GENUD) Research Group, University of Zaragoza, Pedro Cerbuna 12, 50009 Zaragoza, Spain.

³CIBER Fisiopatología de la Obesidad y Nutrición, Instituto de Salud Carlos III, Madrid, Spain.

⁴Section of Nutrition and Metabolism, International Agency for Research on Cancer, World Health Organization, Lyon, France.

⁵University Lille, Inserm, CHU Lille, CIC 1403 – Clinical Investigation Center and U1286 - INFINITE - Institute for Translational Research in Inflammation, F-59000 Lille, France.

⁶ImFine Research Group, Department of Health and Human Performance, Universidad Politécnica de Madrid, Madrid, Spain.

⁷Institut of Nutritional and Food Sciences, University of Bonn, Bonn, Germany.

⁸Department of Public Health and Primary Care, Ghent University, Ghent, Belgium.

⁹Research department of Child Nutrition, Pediatric University Clinic, Ruhr-University Bochum, Bochum, Germany.

¹⁰Council for Agricultural Research and Economics, Research Centre for Food and Nutrition, Rome, Italy.

¹¹Department of Nutrition, Harokopio University of Athens, Athens, Greece.

¹²Institute of Food Science, Technology and Nutrition (ICTAN), Spanish National Research Council (CSIC), Madrid, Spain.

¹³Université de Lille, Inserm, CHU Lille, Institut Pasteur de Lille, U1167 - RID-AGE - Facteurs de risque et déterminants moléculaires des maladies liées au vieillissement, F-59000 Lille, France

¹⁴Instituto Agroalimentario de Aragón (IA2), Spain; Instituto de Investigación Sanitaria Aragón (IIS Aragón), Zaragoza, Spain.

¹⁵EFFECTS 262 Department of Physiology, Faculty of Medicine, University of Granada, 18016, Granada, Spain.

¹⁶Department of Pediatrics, Medical School, University of Pécs, Pécs, Hungary.

¹⁷Department of Social Medicine, Faculty of Medicine, University of Crete, Heraklion, Greece.

¹⁸Centre for Exercise, Nutrition & Health Sciences, School for Policy Studies, University of Bristol, Bristol, United Kingdom.

¹⁹Medicine University Vienna, Department of Pediatrics, Division of Gastroenterology and Hepatology and Austrian Academic Institute for Clinical Nutrition, Vienna, Austria.

²⁰National Nutrition Surveillance Centre, School of Public Health, Physiotherapy and Sports Science, University College Dublin, Dublin, Ireland.

***Corresponding author:**

Luis A. Moreno Aznar

Growth, Exercise, Nutrition and Development (GENUD) Research Group, University of Zaragoza

C/ Pedro Cerbuna, 12

50009 Zaragoza, Spain

Tel.: 0034 876554457

e-mail: lmoreno@unizar.es

Funding

The HELENA study was carried out with the financial support of the European Community Sixth RTD Framework Programme (contract FOODCT-2005-007034). The writing group takes sole responsibility for the content of this article. The European Community is not liable for any use that may be made of the information contained therein. EMGG holds a Juan de la Cierva-Formación grant from the Spanish Government.

Conflicts of interest

The authors declare that they have no conflicts of interest.

IARC disclaimer

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Word count: 4,994

Number of tables: 3

Supplementary tables: 5

Additional supplementary data: Helena Study group, Supplementary Tables 1-5.

Running title: Adolescents' misreporting and cardiometabolic risk.

List of abbreviations:

- **BMI:** Body mass index
- **CVD:** Cardiovascular diseases
- **CMR:** Cardiometabolic risk
- **CRF:** Cardiorespiratory fitness

- **EI:** Energy intake
- **UR:** Under-reporting
- **OV:** Over-reporting
- **PA:** Physical activity
- **TEE:** Total energy expenditure
- **WC:** Waist circumference
- **WHeR:** Waist-to-height ratio
- **DBP:** Diastolic blood pressure
- **SBP:** Systolic blood pressure
- **TG:** Triglycerides
- **TC:** Total cholesterol
- **HDL-c:** High-density lipoprotein cholesterol
- **HOMA:** Homeostasis model assessment
- **URs:** Under-reporters
- **OVs:** Over-reporters

1 Abstract

2 **Background:** Dietary misreporting is the main limitation of dietary assessment and has been
3 associated with body mass index in youth. However there are no prior studies assessing
4 missreporting and cardiometabolic risk (CMR) in adolescence.

5 **Objectives:** The aim was to examine the associations between dietary misreporting and CMR
6 factors in adolescents and to assess the potential bias on the association between CMR and energy
7 intake (EI) driven by dietary misreporting.

8 **Methods:** Two 24-hour dietary recalls were obtained from 1,512 European adolescents (54.8%
9 girls) aged 12.5-17.5 years. Physical activity was measured by accelerometry. Cut-offs suggested by
10 Huang were applied to identify misreporters. Height, waist circumference (WC), sum of four
11 skinfold thicknesses, diastolic (DBP) and systolic blood pressure (SBP), and cardiorespiratory
12 fitness (CRF) were taken and serum triglycerides and total-/high-density lipoprotein cholesterol
13 ratio (TC/HDL-c) were analyzed. A sex- and age-specific clustered CMR score (n=364) was
14 computed. Associations were investigated by multilevel regression analyses adjusting for age, sex,
15 center, socioeconomic status and physical activity.

16 **Results:** Under-reporting (24.8% adolescents) was significantly ($p<0.05$) associated with higher
17 WC, waist-to-height ratio (WHeR) and sum of skinfold thickness whereas overreporting (23.4%
18 adolescents) was significantly associated with lower WC, WHeR, sum of skinfold thickness and
19 SBP. Associations between CMR factors and EI were significantly affected by misreporting
20 considering various approaches. Significant positive associations became inverse after adjusting for
21 misreporting for WC and WHeR. The opposite was true for sum of skinfold thickness, SBP and
22 CMR score. Associations between EI and DBP and CRF did not remain significant after adjusting
23 for misreporting.

24 **Conclusions:** CMR factors differed among misreporting groups and both abdominal and total fat
25 mass indicators were more strongly associated with all forms of misreporting than BMI. Moreover,

26 misreporting seems to bias EI and CMR associations in adolescents. Therefore, energy misreporting
27 should be taken into account when examining diet-CMR associations.

28 **Keywords:** Diet, cardiometabolic risk, energy misreporting, adolescence, HELENA study

29

30 **Introduction**

31 Childhood obesity is considered a worldwide concern as in 2015, obesity affected 107.7
32 million children (1). Globally, prevalence of combined overweight and obesity has risen by 47.1%
33 for children between 1980 and 2013 (2). For adults, more than two-thirds of deaths related to high
34 body mass index (BMI) are due to cardiovascular diseases (CVD) (1). Obesity is associated with a
35 cluster of cardiometabolic risk (CMR) features, including dyslipidemia, elevated glucose and
36 insulin levels, and blood pressure, among others (1). These CMR markers related to obesity have an
37 early onset and may predict type 2 diabetes, metabolic syndrome and CVD later in life (3).

38 Traditionally, this combination of CMR factors has been identified as metabolic syndrome:
39 metabolic abnormalities associated mainly with insulin resistance and CVD (4). The use of scores
40 versus dichotomical definitions to assess CMR seems a better approach as it gives an accurate
41 insight of the metabolic profile (5). Recently, cardiorespiratory fitness (CRF) has emerged as an
42 important marker of cardiometabolic health and has been independently associated with metabolic
43 risk in European adolescents (6). Therefore, these new scores for CMR identification should also
44 consider new risk markers.

45 Diet has been associated with CMR already in adolescence (7). However, epidemiological
46 dietary assessment still represents a challenge as no gold standard for evaluation of reported
47 dietary intake exists (8) and many diet studies are faced with the reporting of implausible energy
48 intake (EI), particularly under-reporting (UR) (9), the main limitation of dietary assessment
49 methods. Under-reporting (UR) is characterized by reports of habitual EI which are implausibly low
50 when compared with the energy requirements estimated using objective methods (10). The opposite
51 is true for over-reporting (OV).

52 Identification of misreporting and its characteristics is thus crucial to the appropriate
53 interpretation of nutritional data (8). BMI in particular has been repeatedly linked to misreporting in
54 adults (9), but also among children and adolescents (11) as inaccurate energy reporting also occurs
55 among young populations (12). However, little is known about the association between

56 misreporting and CMR. It is known that high levels of CMR biomarkers in blood are associated
57 with unhealthier diets (13, 14). Hence, although individuals may not know that they have elevated
58 glucose, insulin, TG and/or HDL-c, they may be conscious of the lower quality of their diets and
59 consequently under-report their dietary intake. Therefore, we hypothesized that under-reporters
60 (URs) would have worse CMR indicators whereas we would expect better levels of CMR indicators
61 among over-reporters (OVs).

62 Adolescence is characterized by increasingly greater food requirements, unstructured eating
63 patterns, rapidly-changing food habits and more frequent out-of-home eating (15). These factors,
64 along with a possibly reduced level of interest to recall their own intake, might lead to less
65 motivation, forgetfulness and lack of compliance and thus to a reduced reporting accuracy (15). To
66 the best of our knowledge, there are no prior studies assessing misreporting and CMR in
67 adolescents. Thus, the aim of the present study is to assess the relationship between energy
68 misreporting and a set of CMR factors, and the bias potentially introduced by misreporting on the
69 association between EI and CMR factors among European adolescents. We hypothesized that
70 adolescents with a worse CMR profile would be more likely to UR whereas OV would have a
71 healthier CMR profile.

72

73

74 **Materials and methods**75 *Subjects and study design*

76 Data for this study were obtained from a random sample of European adolescents
 77 participating in the Healthy Lifestyle in Europe by Nutrition in Adolescence (HELENA) Study (16,
 78 17). Data collection was carried out between September 2006 and December 2007 in 10 European
 79 cities: Athens (inland city) and Heraklion in Greece, Dortmund in Germany, Ghent in Belgium,
 80 Lille in France, Pécs in Hungary, Rome in Italy, Stockholm in Sweden, Vienna in Austria, and
 81 Zaragoza in Spain. A detailed description of general procedures has been published elsewhere (17,
 82 18). Written informed consent was obtained from all adolescents and their parents or guardians. The
 83 study was conducted according to the guidelines laid down in the Declaration of Helsinki and all
 84 procedures involving human subjects/patients were approved by the Human Ethics Committees of
 85 the centers involved (18).

86 A total of 3,528 adolescents (52.3% females) aged 12.5-17.5 years were recruited from
 87 randomly selected schools in each city. Only those adolescents who completed two 24-hour dietary
 88 recalls (24-HDR) and had objectively measured accelerometer data were included (n=1,512). Blood
 89 samples were drawn after an overnight fast only in one-third of the HELENA participants randomly
 90 selected. Adolescents that were excluded from the analyses were older, heavier and had higher BMI
 91 than those included in the present study. In addition, there was a higher proportion of girls,
 92 adolescents with overweight and obesity, less affluent adolescents and lower maternal education
 93 levels among those excluded as compared with their peers included in the analyses
 94 (**Supplementary Table 1**).

95 *Socioeconomic status & maternal education level*

96 Socioeconomic status (SES) was estimated using the Family Affluence Scale, an indicator of
 97 affluence based on the concept of material conditions of the household in which adolescents lived

(19). The mothers' educational level was reported by the adolescents as lower education, lower secondary education, higher secondary education and higher education/university degree.

Physical activity

Physical activity (PA) assessment in the HELENA study is described elsewhere (20). Uniaxial accelerometers Actigraph MTI (model GT1M) were used to objectively measure PA. Total PA was expressed as total counts recorded, divided by total daily registered time (counts/minute). Total energy expenditure (TEE, kcal/day) was estimated from activity counts using the equation of Ekelund et al. (21), which has been validated in youth. The equation also considers the individual's sex and weight.

Physical examination

Weight and height were measured in underwear and barefoot with an electronic scale (Type SECA 861) and a stadiometer (Type SECA 225), respectively. BMI was calculated and was additionally categorized according to Cole et al. (22, 23). Waist circumference (WC) was taken at the midpoint between the lowest rib and the iliac creast with an anthropometric tape (SECA 200). Waist-to-height ratio (WHeR) was computed. Skin-folds thickness was measured with a Holtain Calliper in triplicate on the left side at biceps, triceps, subscapular and suprailiac sites. All anthropometric measures were taken following a standardized protocol (24). Blood pressure was measured twice with an automatic oscillometric device (Omron M6, HEM-7001-E). The lowest value was retained for both diastolic (DBP) and systolic blood pressure (SBP).

Blood sampling

Blood sampling procedures have previously been described in detail (25). Serum triglycerides (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-c) and glucose were measured with an enzymatic method on the Dimension RxL clinical chemistry system (Dade Behring, Schwalbach, Germany, coefficient of variation <1.6%). Serum insulin levels were measured with an Immulite 2000 analyzer (DPC Biermann, Bad Nauheim, Germany, sensitivity 2

123 $\mu\text{IU/mL}$, coefficient of variation $<5.5\%$). The homeostasis model assessment (HOMA) index was
 124 used as a measurement of insulin resistance (26). The ratio TC/HDL-c was computed.

125 ***Cardiorespiratory fitness***

126 CRF was predicted using the maximum speed that an individual reached during the 20-m
 127 shuttle run test. The maximum oxygen uptake ($\text{VO}_{2\text{max}}$) ($\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) was calculated
 128 according to the Léger et al. (27) formula.

129 ***Cardiometabolic risk score***

130 A continuous score of clustered CMR factors ($n=380$, 50.5% girls) was computed according
 131 to Andersen et al. (28) using: SBP, sum of thickness of four skinfolds (bicipital, tricipital,
 132 subscapular and supra-iliac), TG, TC/HDL-c, HOMA index and CRF. Sex- and age-specific z-
 133 scores were calculated for each risk factor variable. All individual z-scores were summed to create
 134 the clustered CMR score. CRF was multiplied by -1 to indicate higher CMR with increasing value.
 135 The lower the score the better the overall CMR factor profile. Those adolescents at + 1SD of the
 136 score were considered at CMR.

137 ***Cardiometabolic risk factors***

138 CMR factors cut-offs were applied to identify those adolescents at higher CMR. WC, DBP,
 139 SBP, glucose, insulin, TG, and HDL-c were dichotomised according to the International Diabetes
 140 Federation definition of metabolic syndrome for adolescents (29). Castro-Piñeiro et al. (30) cut-offs
 141 were applied for CRF and those from Piña-Aguero et al. (31) for the HOMA index. The 90th
 142 percentile cut-off was used for the sum of thickness of four skinfolds. TC/HDL-c was dichotomised
 143 according to the cut-off values suggested by Chu et al. (32).

144 ***Energy intake***

145 Two non-consecutive 24-HDR, within a time span of two weeks, were completed by the
 146 adolescents (33). Assessment was performed by a computer-based tool for self-reported 24-HDR,
 147 HELENA-DIAT (Dietary Assessment Tool), which has been shown to provide valid measurements
 148 of dietary intake (34). The German Food Code and Nutrition Data Base (BLS,

149 (Bundeslebensmittelschlüssel), version II.3.1, 2005) (35, 36) was used to calculate EI. Energy
 150 intake was estimated in kilocalories per day (kcal/d) taking the average of the two 24-HDRs.

151 *Energy intake misreporting: under-reporting & over-reporting*

152 More detailed information on the identification of UR and OV in this sample can be found
 153 elsewhere (37). Misreporters were identified according to the approach proposed by Huang et al.
 154 (38). The method relies on the direct comparison of reported energy intake (EI) and predicted TEE
 155 (%EI/TEE). The approach uses ± 1 SD cut-offs to statistically compare reported EI with predicted
 156 TEE. A report is considered implausible if %EI/TEE is too low (< -1 SD, under-reporter) or too
 157 high ($> +1$ SD, over-reporter) to represent the habitual intake. Number of days were $d=2$. The
 158 CV_{rEI} and the CV_{pTEE} were calculated separately by sex based on the HELENA data (39). The
 159 CV_{mTEE} was set to 8.2%, as estimated from doubly labeled water measurements (40). Adolescents
 160 were classified as URs, plausible reporters or OVs according to these cut-offs values
 161 (**Supplementary Table 2**).

162 *Statistical Analysis*

163 The distribution of all variables was checked before the analysis and several transformations
 164 were conducted to improve normality (**Supplementary Table 3**). Multilevel Poisson regression
 165 analyses with robust error variance, with study center as random intercept, were performed to
 166 investigate factors associated with misreporting, considering UR and OV as outcomes. Firstly,
 167 analyses were conducted to explore the association between each CMR indicator and the CMR
 168 score (either as continuous and dichotomised variables) and UR and OV (as dichotomised
 169 variables), adjusting for age, sex, family affluence scale, maternal education, PA and study center
 170 (random intercept). Subsequently, CMR factors with $p<0.20$ in Model 1 were entered
 171 simultaneously in the same model with UR and OV as the outcome variables. This cut-off point is
 172 widely used for purposeful selection of variables (41, 42).

173 Multilevel linear regression analyses were conducted to investigate the bias of misreporting
 174 on the association between EI and the CMR indicators and score in six different models: 1) crude

175 model: all participants adjusting for age and sex; 2) all participants adjusting for relevant covariates
176 (age, sex, family affluence scale, maternal education and PA); 3) all participants after adjustment
177 for UR only and covariates included in model 2; 4) all participants after adjustment for OV only
178 and covariates included in model 2; 5) all participants after adjustment for both UR and OV and
179 covariates included in model 2; and 6) only plausible reporters, excluding URs and OVs, adjusting
180 for covariates included in model 2. Study center was entered in all models as random intercept.
181 DBP, SBP, CRF and TC/HDL-c were further adjusted for sum of four skinfold thicknesses.
182 Collinearity among variables entered in the same model was assessed with the variance inflation
183 ratio (VIF). There was no collinearity ($VIF < 2$) among the variables included in any of the
184 multilevel models, i.e., Poisson or linear regression analyses. With the Poisson regression we
185 obtained the prevalence ratio (PR). The statistical software package Stata version 16.0 (Stata Corp.,
186 College Station, TX, USA) was used to perform the analyses, and the threshold for statistical
187 significance was set at $p \leq 0.05$.

188

189 **Results**

190 We identified 375 (24.8%) URs and 353 (23.4%) OV. Baseline characteristics of the study
 191 participants by reporting group are displayed in **Table 1**. Overall, URs showed a worse
 192 cardiometabolic profile, with greater WC, WHeR, sum of four skinfold thicknesses, SBP, DBP,
 193 fasting insulin, HOMA index, TG, TC, TC/HDL-c and CMR score and lower CRF in comparison
 194 with plausible reporters and OVs. In contrast, OVs had better cardiometabolic health as they had the
 195 lowest values for all CMR indicators as compared with plausible reporters. The dichotomized CMR
 196 indicators are presented in **Supplementary table 4**.

197 Multilevel Poisson regression analyses are shown in **Table 2**. UR was positively associated
 198 with WC, WHeR, sum of four skinfold thicknesses and CMR score and inversely associated with
 199 DBP and CRF. On the other hand, OVs were significantly associated with lower WC, WHeR, sum
 200 of four skinfold thicknesses, SBP and CMR score. Some of these associations did not remain
 201 significant when the model was further adjusted for those variables with $p < 0.20$ in the crude model.
 202 In the adjusted model, UR was significantly associated with greater WC (PR: 1.05; 95% CI: 1.01,
 203 1.09), WHeR (PR: 1.09; 95% CI: 1.03, 1.15) and sum of four skinfold thicknesses (PR: 1.02; 95%
 204 CI: 1.01, 1.03). Over-reporting was significantly associated with lower WC (PR: 0.95; 95% CI:
 205 0.93, 0.97), WHeR (PR: 0.93; 95% CI: 0.88, 0.99) and sum of four skinfold thicknesses (PR: 0.98;
 206 95% CI: 0.97, 0.99).

207 The associations between dichotomized CMR indicators cut-offs and energy misreporting
 208 are displayed in **Supplementary Table 5**. Under-reporting was positively associated with higher
 209 WHeR, sum of four skinfold thicknesses, SBP and CMR score, and lower CRF. OV was inversely
 210 associated with WC, WHeR, sum of four skinfold thicknesses, glucose and CMR score. When
 211 further adjustments were carried out (model 2), these associations only remained significant for sum
 212 of four skinfold thicknesses among URs. On the other hand, over-reporting was significantly and
 213 inversely associated in the model 2 with WC, WHeR, sum of four skinfold thicknesses and SBP.

214 Multilevel linear regression analyses showed the bias introduced by energy misreporting in
215 the association between EI and CMR indicators (**Table 3**). WC, WHeR, sum of four skinfold
216 thicknesses and SBP were significantly associated with EI; these associations remained significant
217 when analyses were further adjusted for either UR, OV (not significant for SBP) or both. However,
218 the direction of the associations changed when adjustment for misreporting was applied. In more
219 detail, the significant positive associations between WC and WHeR and EI prior to misreporting
220 adjustment changed to inverse following adjustment for misreporting. In contrast, sum of four
221 skinfold thicknesses and SBP were inversely associated with EI and these associations became
222 positive once misreporting adjustment was applied. The inverse association observed between DBP
223 and EI did not remain significant when further covariates were entered into the model and when
224 analyses were adjusted for misreporting. Cardiorespiratory fitness and TC/HDL-c were positively
225 associated with EI. For CRF, these associations, however, were no longer significant when analyses
226 were adjusted for any form of misreporting or misreporters were excluded from the analyses. The
227 direct association between TC/HDL-c and EI remained significant in Models 1, 2 and 4 when UR
228 and other covariates were entered into the analyses. No change in the direction of the association
229 was observed though. The CMR score was inversely associated with EI but this association became
230 positive when analyses were adjusted for OV (Model 3) or when URs and OVs were excluded from
231 the analyses (Model 5). No associations were observed when analyses were adjusted for UR (Model
232 2) and for both UR and OV (Model 4). Results remained quite similar when analyses were limited
233 to those adolescents with available data for all the CMR indicators (data not shown), except for SBP
234 as not significant associations were observed across the models. These discrepancies would be
235 mainly explained due to differences in sample size.

236

237 **Discussion**

238 In the present study, differences in CMR factors were found by misreporting group in a
 239 relatively large sample of European adolescents. When assessing the role of misreporting in the
 240 association between EI and CMR features, we observed that associations took different directions
 241 depending on the adjustment approach applied, that is, inversed associations became direct and vice
 242 versa, which suggests that misreporting could affect this relationship. Although several studies have
 243 examined characteristics of URs among youth from various countries with different dietary
 244 behaviours and cultures (11, 43-45), there is a lack of studies assessing OV and the influence of
 245 misreporting in CMR and EI in adolescents.

246 247 ***Misreporting and body fat mass***

248 In our sample of adolescents, we found a prevalence of misreporting of 48.2%, being 24.8%
 249 for URs and 23.4% for OVs. The prevalence of overweight/obesity in the whole sample of our
 250 study was 20.4%. Overweight/obesity was present in 40% of the URs, (10.6% for obesity), while
 251 among plausible reporters it was 17.5% (3.6% for obese) and 5.7% (0.84% for obesity) for the OVs.
 252 Finally, 5.7% of the OV reporters had overweight/obesity (0.84% obesity). BMI has been largely
 253 recognized as a factor associated with misreporting and, in adults, is the strongest predictor for UR
 254 (9). Among the factors suggested to explain the tendency to under-report, providing socially
 255 desirable answers due to greater weight consciousness and dieting behaviour is included, among
 256 others (11, 46, 47).

257 Although BMI has been largely used in epidemiological studies, it accounts for body mass
 258 and not actual fat. This means that high BMI could also be found in subjects with high fat-free
 259 mass, lean mass and/or muscle mass and could affect the association between BMI and
 260 misreporting consistently observed in the literature. Thus, the use of specific fat mass markers could
 261 give a better insight of the associations with UR behaviour and, in general, with misreporting. For
 262 that reason, in our study, WC and WHeR were used as markers of abdominal fat while the sum of
 263 four skinfold thicknesses was computed to account for overall body fat. In our sample, URs showed

higher mean values for fat mass markers, sum of four skinfold thicknesses, WC and WHeR in comparison to plausible reporters and OV's. On the other hand, OV's had the lowest mean values for the fat mass markers. Under-reporting was significantly associated with greater WC, WHeR and sum of four skinfold thicknesses whereas OV was significantly associated with lower WC, WHeR and the sum of four skinfold thicknesses in adjusted models. In line with our results, previous literature has shown that adolescents with higher body fat under-estimate their EI the most (48-50). Therefore, fat mass indicators need to be taken into consideration when assessing diet-disease associations to counteract the higher likelihood of those having higher body fat to under-report their dietary intake.

A previous study based on HELENA data, showed that adolescents with greater fat mass had a lower EI compared with adolescents with less fat mass, regardless of their PA level (51). In that HELENA evaluation, Cuenca-García et al. (51) claimed that their results remained similar when the analyses were adjusted for energy misreporting. This could suggest that teenagers with greater fat mass in our study may be truly having lower EI and, therefore, being reliable reporters of their EI. However, other studies have shown that fat free mass, and not fat mass, is positively associated with portion size and total EI in adults (52) and adolescents (53) having overweight/obesity. Among those subjects with overweight and obesity, fat mass increases alongside fat free mass so higher EI should be expected (54). For that reason, adolescents with higher fat mass in our sample seem more likely to be UR than having low EI. Nevertheless, it should be borne in mind that UR is likely to also include real under-eating as some adolescents may be intentionally eating lower amounts of food as an attempt to lose or not gain weight. Overall, this highlights the importance of considering misreporting when analysing dietary data to take into account any discrepancies between the reported information and the physiologically plausible expected intake.

Energy misreporting influences the association between eating patterns and adiposity (55). In youth, a correlation between percentage of body fat and differences in EI reported by a food

frequency questionnaire was identified, which was explained by misreporting (56). In this study, and in line with our findings, the probability of EI under-estimation among boys was lower in those with body fat $\leq 10\%$ while it was much higher for those with a greater percentage of body fat, mainly among subjects with body fat $\geq 25\%$ (56). Additionally, studies evaluating the validity of dietary assessments using the doubly labelled water method found that the likelihood of UR in adolescents was most strongly predicted by higher percentage of body fat (49, 56). These results show that fat mass, as BMI, is strongly correlated with dietary misreporting. People with high BMI and, probably, higher fat mass, may be more prone to UR for the same reasons as those with high BMI, as previously stated. Thus, attenuation of the association between dietary intake and risk of obesity may occur in studies in which high misreporting is present or adjustment for misreporting is not considered (57). Therefore, studies aiming to investigate potential obesity dietary risk factors need to consider some evaluation of misreporting.

In contrast, more physically active and leaner adolescents have been shown to have higher EI than less active adolescents with larger amounts of fat mass, which could lead to OV (51). The positive association of higher fat free mass and EI is expected, given the high metabolic activity of organs and tissues that constitute the fat free mass which accounts for $\sim 70\%$ of the variance in resting energy expenditure (58). We have not been able to find any studies available on the association between fat-free mass and OV in adolescents. This could be because OV identification among adolescents is hindered by the fact that they are in a phase of growth and development. Therefore, high EI can reflect real over-eating rather than OV as adolescents may consume larger amounts of food due to growth spurts.

311 ***Misreporting and cardio metabolic risk***

In addition to having higher body fat, URs showed a worse cardio-metabolic profile in our sample of European adolescents. Overall, there is a lack of studies assessing the association of misreporting and CMR in adolescents. In agreement with some of our findings, a previous study from Suissa et al. (59) among children at risk of obesity showed that URs had worse CMR factors

316 than plausible reporters. Specifically, blood pressure and low density lipoprotein cholesterol (LDL-
 317 c) concentrations were higher and HDL-c levels were lower in URs than in plausible reporters (59).
 318 These results are similar to those from another study that reported on biochemical markers,
 319 including LDL-c, HDL-c and TG, of URs in a small sample of South American adolescents (60). In
 320 a different study carried out among adults, associations between some biomarkers such as HDL-c,
 321 LDL-c or SBP and specific food items were affected by misreporting, even changing the direction
 322 of the associations when it was taken into account (61). In contrast, OV's showed better cardio
 323 metabolic health when compared with URs in the present study. However, when assessing the
 324 association with metabolic profile in the full adjusted model, we found no associations.

325 We hypothesised that these associations found with CMR in the crude model could be
 326 partially explained by the differences in body fat between both groups. Obesity increases the
 327 adipose tissue and that could lead to an impaired endocrine function and, consequently, to cardio
 328 metabolic implications that track into adulthood (62). Thus, being at higher CMR could be a
 329 consequence of body fat and therefore observing worse CMR factors in URs, who had higher BMI
 330 and fat mass in comparison with the rest of the adolescents, could be expected. However, this could
 331 only explain crude model. In fact, associations were no longer significant in the fully adjusted
 332 model when body fat indicators were entered into the analyses.

333 *Misreporting and the association of CMR and energy intake*

334 Results from the present study suggest that misreporting affects the association of CMR and
 335 EI, either using CMR factors individually or a CMR score. We found different associations when
 336 considering UR and OV individually in different models as covariates and when applying the
 337 combination of both in comparison with the model where misreporting was not considered.
 338 Moreover, when excluding the misreports, differences in the association between CMR and EI were
 339 also found when compared with the other approaches. These results could be explained by the
 340 prevalence of overweight/obesity being higher among URs (40%) as compared with OV's (5.7%).
 341 On the other hand, the decrease in the sample size that resulted when URs and OV's were excluded

from the analyses could be another explanation to the observed results. This result highlights the major role of misreporting when assessing the association between CMR and EI and the fact that a method to appropriately take into account misreporting is still missing. Finally, statistical significance is not a sufficient condition for an effect to be meaningful. However, after careful interpretation of the results from an epidemiological practice point of view, we conclude that misreporting seem to affect the association between EI and CMR already in adolescence.

Nevertheless, these findings need to be interpreted with caution. As highlighted by Subar et al. (63), it is not appropriate the use of self-reported EI due to the limitations inherent to self-reported methods. For that reason, we did not make any attempts in reporting the associations between EI and CMR and focused our findings on the changes of these associations when misreporting was taken into account. We used EI as an overall measure of adolescents' diet, but we would recommend to not consider these associations as true associations. In fact, given the impact that misreporting had on self-reported EI in this study, these findings can be considered as an extension of Subar et al. (63) statement on the uselessness of EI as exposure variable.

Our findings showed that either adjusting the analyses for both forms of misreporting, that is, UR and OV, or excluding misreporters from the analyses yielded similar results despite the decrease in sample size when only plausible reporters were included in the analyses. Although both approaches seem to be appropriate to correct for misreporting, the exclusion of implausible reporters may introduce some additional concerns such as selection bias (64) together with a decrease in the sample size and the power to detect associations, and the exclusion of those being truly under- or over-eating (59). An alternative to this option could be to conduct the analyses using both approaches and compare the obtained results before making a decision. Thus, accounting for misreporting reduces the bias in the association measured. Other methods include stratification of results by reporting status or the use of a propensity score to adjust the analyses for all predictors of misreporting (65). A previous study based on HELENA data that assessed misreporting and its correlates in European adolescents, suggested that there are several factors, specifically weight

368 status, being worried about gaining weight, body image dissatisfaction and skipping breakfast, that
369 may need to be taken into account to improve interpretation of potentially biased findings (37).
370 However, agreement has yet to be reached on the best correction method. Nevertheless,
371 misreporting is a complex problem, beyond BMI differences among misreporting groups, that needs
372 to be accounted for when assessing EI and which remains among the main limitations of dietary
373 assessment methods.

374 *Limitations and strengths*

375 The present study has some limitations. Firstly, diet was assessed by self-reported 24-HDRs
376 which is subject to certain degree of systematic measurement error due to the characteristics of the
377 method and the population under study (63). The method relies on the participants' memory and on
378 their ability in estimating quantities and can lead to inaccurate estimates of EI (63). Hence,
379 inferences on the specific association between EI and CMR should be avoided. Also, only two-non-
380 consecutive days were included which may not be sufficient to characterise individual's usual
381 intakes and variability. However, the HELENA-DIAT provides reliable estimates of dietary intake
382 among European adolescents (36, 66).

383 Another limitation that needs to be accounted for is that the applied cut-offs assumed stable
384 body weight, that is, that they are not losing or gaining weight. However, we assume that this
385 condition does not significantly affect the study outcomes as energy costs in adolescence are small,
386 approximately 1% of TEE (67). Given the short period of dietary recording, a distinction between
387 respondents who are on a diet (under-eating) or those limiting their reported intake (UR) and among
388 those who are intentionally eating more (over-eating) or reporting higher EI (OV) could not be
389 made. The cross-sectional nature of this study cannot be used to establish causality. Another study
390 limitation is that analyses were not corrected for multiple testing; therefore, some of the observed
391 significant associations may not be true associations but due to chance. On the other hand, our study
392 has several strengths. First, the use of European data with standardised procedures throughout the
393 different study center with a geographical spread. Cut-off values to identify misreporting were

calculated for each individual based on their own PA levels, which resulted in a more accurate classification of reported EI. The method suggested by Huang et al. (38) to deal with reporting error, offers a simpler and more individualised alternative in comparison with other existing methods. Lastly, we found differences in sociodemographic variables among adolescents included in this study and those excluded from the analyses. This may limit the generalizability of our findings to the entire HELENA study sample.

Conclusion

This study showed that 48.2% of European adolescents tend to misreport their EI. Additionally, and in line with previous literature, our results confirm that there is a higher percentage of subjects with higher fat mass within the UR group when compared with plausible reporters and OV. Overall, misreporting was associated with body fat mass and CMR markers, which could be partially explained by specific lifestyle behaviors within each reporting group. Finally, misreporting affected the association between CMR factors and EI, independently of the approach followed: exclusion of misreporters or adjustment for misreporting. Therefore, it is crucial to assess misreporting when evaluating CMR and EI in youth to avoid potentially biased findings that could lead to misleading conclusions. Further research should examine the underlying factors that explain the association between misreporting and CMR. Likewise, future research is needed to identify more accurate techniques to take into account misreporting in studies investigating diet-disease associations.

Acknowledgements

The authors gratefully acknowledge all participating children and adolescents and their parents and teachers for their collaboration. We wish to thank Petra Pickert, Birgit Stoffel-Wagner and Anke Ernst for their contribution to laboratory work. HELENA Study Group (**Supplementary data 6**).

Authors' contributions

E.M.G.G, I.H., S.B.S and L.A.M designed research; I.H., M.G.G, S.H., M.K., Y.M., A.M., D.M., K.W. and L.A.M conducted research; E.M.G.G and S.B.S. analyzed data and performed statistical analysis; and E.M.G.G. and S.B.S. wrote the paper. I.H., C.M.A., L.B., C.B, E.G., M.G.G., S.H., M.K., C.L.D., Y.M., A.M., A.M., P.D.M-E., C.M.H., D.M., A.P., K.W. and L.A.M. critically revised the manuscript several times. E.M.G.G, L.A.M. and S.B.S had primary responsibility for final content. All authors read and approved the final manuscript.

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TABLE 1. Descriptive characteristics of the study participants by reporting status (n=1,512).¹

	n	All	n	Under-reporters	n	Plausible reporters	n	Over-reporters
Age (years)	1512	14.7 ± 1.21	375	14.7 ± 1.2	784	14.6 ± 1.2	353	14.6 1.2
Sex								
Boys	684	45.2	157	41.9	359	45.8	168	47.6
Girls	828	54.8	218	58.1	425	54.2	185	52.4
Family Affluence Scale								
Low	147	9.8	37	10	74	9.5	36	10.2
Medium	824	54.7	203	54.7	427	54.7	194	55
High	534	35.5	131	35.3	280	35.8	123	34.8
Maternal education								
Lower education	98	6.8	26	7.3	50	6.6	22	6.5
Lower secondary education	353	24.4	107	30.1	166	22.1	80	23.7
Higher secondary education	427	29.5	92	25.8	228	30.3	107	31.6
Higher education/university degree	569	39.3	131	36.8	309	41	129	38.2
Energy intake (kcal/d)	1512	2087 (1596-2745)	375	1301 (1067-1552)	1552.1	2102.4 (1793-2490)	353	3244 (2777-3895)
Physical activity (cpm)	1512	403.1 (322-516)	375	426 (337-553)	784	399 (323-510)	353	387.1 (312-487)
Weight (kg)	1512	56.4 (49.9-63.9)	375	61.5 (54.5-70.1)	784	56.1 (50-63.1)	353	52.3 (45.8-58.9)
Height (cm)	1512	165.5 ± 9.1	375	165.6 ± 8.5	784	165.5 ± 9	353	165.3 ± 9.9
BMI (kg/m²)	1512	20.4 (18.6-22.8)	375	22.5 (20.3-25.2)	784	20.4 (18.7-22.6)	353	18.9 (17.5-20.5)
BMI category								
Underweight (<18.5 in adults)	108	7.1	7	1.9	43	5.5	58	16.4
Normal weight (18.5-24.9 in adults)	1097	72.6	218	58.1	604	77	275	77.9
Overweight (25-30 in adults)	236	15.6	110	29.3	109	13.9	17	4.8
Obesity (>30 in adults)	71	4.7	40	10.7	28	3.6	3	0.9
WC (cm)	1497	70.2 (66-75.4)	372	74.4 (69.1-80.6)	778	70.1 (66.-74.9)	347	67.2 (63.9-71.5)
WHeR	1497	0.42 (0.4-0.46)	372	0.45 (0.42-0.49)	778	0.42 (0.4-0.45)	347	0.41 (0.39-0.43)
Sum of four skinfold thicknesses (mm)	1490	46.1 (33.1-65.3)	367	62.2 (44.9-81.8)	775	46 (33.4-63.3)	348	35.9 (27.4-46.8)
SBP (mm Hg)	1496	115.3 ± 12.7	370	118.1 ± 13.3	776	115.2 ± 12.8	350	112.4 ± 11.4
DBP (mm Hg)	1496	64.2 ± 8.3	370	65.3 ± 8.5	776	64.1 ± 8.4	350	63.3 ± 7.9
CRF (mL · kg⁻¹ · min⁻¹)	1258	42.2 ± 7.3	289	40.2 ± 7	651	42.6 ± 7.2	318	43.4 ± 7.5
Serum glucose (mg · dL⁻¹)	481	90.3 ± 6.8	116	90.1 ± 6.2	246	90.3 ± 7.4	119	90.5 ± 6.3

Serum insulin (mg· dL⁻¹)	474	8.7 (6.2-12)	111	9.5 (6.8-14)	245	8.5 (5.9-11.5)	118	8.2 (6.2-11.5)
HOMA index	473	2 (1.35-2.7)	111	2.2 (1.6-3)	244	1.9 (1.3-2.7)	118	1.9 (1.4-2.6)
Serum TG (mg · dL⁻¹)	481	60 (46-80)	116	65 (49.5-89.5)	246	58.5 (45-80)	119	58 (46-80)
Serum TC (mg· dL⁻¹)	481	160 (143-178)	116	165 (150.5-184)	246	158.5 (142-178)	119	157 (140-175)
HDL-c (mg· dL⁻¹)	481	55 (49-63)	116	54 (47-60.5)	246	56 (49-64)	119	54 (49-60)
TC/HDL-c	481	2.9 (2.5-3.3)	116	3 (2.7-3.4)	246	2.8 (2.5-3.2)	119	2.9 (2.5-3.3)
Cardio metabolic risk score	380	-0.29 (-2.38-1.94)	81	1.50 (-0.60-3.75)	198	-0.48 (-3.14-2.32)	101	-1.36 (-2.28-0.16)

¹Values are means $\pm$ standard deviations (SDs), medians (25th-75th percentile) or n (percentage).

BMI, body mass index; CRF, cardiorespiratory fitness; DBP, diastolic blood pressure; HDL-c, high density lipoprotein cholesterol; HOMA, homeostatic model assessment; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; WC, waist circumference; WHeR, waist-to-height ratio.

TABLE 2. Multilevel Poisson regression analyses on the association between dietary misreporting (under-reporting n=271, over-reporting n=286) and cardiometabolic risk indicators.¹

Plausible reporters (ref.) vs under-reporters	Crude model ²		Model 1 ³		Model 2 ⁴	
	PR (95% CI)	p-value	PR (95% CI)	p-value	PR (95% CI)	p-value
WC (cm)	1.04 (1.01,1.07)	0.019	1.05 (1.01,1.08)	0.004	1.05 (1.01,1.09)	0.017
WHeR	1.07 (1.02,1.12)	0.003	1.09 (1.03,1.15)	0.002	1.09 (1.03,1.15)	0.004
Sum of four skinfold thicknesses (mm)	1.02 (1.01,1.02)	<0.001	1.02 (1.01,1.03)	<0.001	1.02 (1.01,1.03)	<0.001
SBP (mm Hg)	1.01 (0.99,1.03)	0.283	1.01 (0.99,1.03)	0.053	1.00 (0.99,1.02)	0.838
DBP (mm Hg)	0.99 (0.98,0.99)	0.049	0.99 (0.98,1.01)	0.276	-	-
CRF (mL · kg ⁻¹ · min ⁻¹)	0.96 (0.92,0.99)	0.016	0.94 (0.90,0.98)	0.006	0.98 (0.95,1.01)	0.207
Glucose (mg·dL ⁻¹)	1.00 (0.98,1.02)	0.821	1.01 (0.99,1.03)	0.615	-	-
Insulin (μIU·dL ⁻¹)	1.01 (0.97,1.05)	0.670	1.02 (0.98,1.06)	0.326	-	-
HOMA index	1.02 (0.96,1.09)	0.562	1.05 (1.01,1.10)	0.031	0.95 (0.86,1.05)	0.331
TG (mg·dL ⁻¹)	1.00 (0.99,1.01)	0.064	1.01 (1.00,1.01)	0.020	1.00 (1.00,1.01)	0.292
TC (mg·dL ⁻¹)	1.00 (0.99,1.01)	0.900	1.00 (0.99,1.01)	0.933	-	-
HDL-c (mg·dL ⁻¹)	0.99 (0.97,1.00)	0.067	0.98 (0.97,1.00)	0.057	-	-
TC/HDL-c	1.17 (0.89,0.54)	0.251	1.20 (0.94,1.54)	0.141	0.94 (0.71,1.26)	0.696
Cardiometabolic risk score	1.08 (1.03,1.014)	0.003	1.11 (1.06,1.17)	<0.001	-	-
Plausible reporters (ref.) vs over-reporters	Crude model ²		Model 1 ³		Model 2 ⁴	
	PR (95% CI)	p-value	PR (95% CI)	p-value	PR (95% CI)	p-value
WC (cm)	0.95 (0.93,0.98)	<0.001	0.95 (0.93,0.97)	<0.001	0.95 (0.93,0.97)	<0.001
WHeR	0.92 (0.87,0.98)	0.006	0.92 (0.88,0.97)	0.001	0.93 (0.88,0.99)	0.015
Sum of four skinfold thicknesses (mm)	0.98 (0.97,0.99)	<0.001	0.98 (0.97,0.99)	<0.001	0.98 (0.97,0.99)	<0.001
SBP (mm Hg)	0.98 (0.97,0.99)	0.001	0.98 (0.97,0.99)	<0.001	0.99 (0.98,1.00)	0.055
DBP (mm Hg)	0.99 (0.97,1.00)	0.125	0.98 (0.96,1.00)	0.084	1.00 (0.98,1.02)	0.782
CRF (mL · kg ⁻¹ · min ⁻¹)	1.01 (0.97,1.04)	0.708	1.01 (0.96,1.06)	0.758	-	-

Glucose (mg·dL⁻¹)	0.99 (0.98,1.01)	0.625	0.99 (0.98,1.01)	0.853	-	-
Insulin (μIU·dL⁻¹)	0.98 (0.95,1.02)	0.311	0.99 (0.95,1.02)	0.398	-	-
HOMA index	0.92 (0.83,1.02)	0.099	0.92 (0.84,1.02)	0.108	1.03 (0.94,1.11)	0.564
TG (mg·dL⁻¹)	0.99 (0.99,1.00)	0.284	0.99 (0.99,1.00)	0.167	1.00 (0.99,1.00)	0.730
TC (mg·dL⁻¹)	0.99 (0.98,0.99)	0.040	0.99 (0.98,0.99)	0.022	0.99 (0.99,1.00)	0.081
HDL-c (mg·dL⁻¹)	0.99 (0.98,1.99)	0.026	0.99 (0.98,1.00)	0.169	0.99 (0.98,1.01)	0.179
TC/HDL-c	0.95 (0.77,1.18)	0.653	0.89 (0.72,1.12)	0.326	-	-
Cardiometabolic risk score	0.94 (0.91,0.98)	0.004	0.93 (0.90,0.96)	<0.001	-	-

¹Values are prevalence ratio (PR) and 95% confidence intervals (95% CI).

²Crude model adjusted for age, gender and center (random intercept)

³Model 1 adjusted for age, gender, family affluence scale, maternal education and moderate-to-vigorous physical activity (counts per minute) and center (random intercept).

⁴Model 2 adjusted for age, gender, family affluence scale, maternal education and moderate-to-vigorous physical activity (counts per minute) and center (random intercept) plus those variables with $p > 0.200$ in Model 1. Only those variables that are part of the cardiometabolic risk score were entered as covariates.

Statistical significance level at $p < 0.05$.

CRF, cardiorrespiratory fitness; DBP, diastolic blood pressure; HDL-c, high density lipoprotein cholesterol; HOMA, homeostatic model assessment; PR, poisson regression; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; WC, waist circumference; WHeR, waist-to-height ratio.

TABLE 3. Multilevel linear regression analyses on the association between cardio metabolic risk indicators and energy intake (n=1,512).¹

	WC (cm)	WHeR	Sum 4 skinfold thicknesses (mm)	SBP (mm Hg)	DBP (mm Hg)	CRF (mL · kg⁻¹ · min⁻¹)	TC/HDL-c	CMR score
<i>Crude model²</i>								
n	1,434	1,434	1,427	1,432	1,432	1,207	457	364
β (95% CI)	0.002 (0.001,0.002)	0.57 (0.43,0.71)	-0.95 (-1.14,-0.77)	-2.35 (-3.83,-0.88)	-1.14(-2.27,-0.10)	2.00 (1.20,2.79)	0.02 (0.01,0.03)	-1.76 (-2.64,-0.88)
p-value	<0.001	<0.001	<0.001	0.002	0.031	<0.001	0.002	<0.001
<i>Model 1³</i>								
n	1,434	1,434	1,427	1,414	1,414	1,193	454	364
β (95% CI)	0.002 (0.001,0.002)	0.55 (0.42,0.69)	-0.92 (-1.11,-0.74)	-0.24 (-1.73,1.24)	-0.08 (-1.14,0.98)	0.55 (-1.17,1.26)	0.02 (0.002,0.03)	-1.62 (-2.47,-0.78)
p-value	<0.001	<0.001	<0.001	0.744	0.881	0.137	0.018	<0.001
<i>Model 2⁴</i>								
n	1,102	1,102	1,094	1,083	1,083	893	344	271
β (95% CI)	-0.004 (-0.005,-0.003)	-0.61 (-0.88,-0.33)	0.88 (0.51,1.26)	4.38 (1.30,7.46)	1.89 (-0.27,4.05)	1.27 (-1.90,2.74)	0.03 (0.01,0.06)	1.09 (-0.59,2.76)
p-value	<0.001	<0.001	<0.001	0.005	0.086	0.088	0.012	0.204
<i>Model 3⁵</i>								
n	1,079	1,079	1,077	1,068	1,068	920	347	286
β (95% CI)	-0.006 (-0.006,-0.004)	-0.75 (-1.05,-0.44)	1.07 (0.66,1.48)	3.24 (-0.25,6.73)	-1.30 (-3.82,1.23)	0.67 (-1.07,2.40)	0.02(-0.01,0.05)	2.70 (0.84,4.56)
p-value	<0.001	<0.001	<0.001	0.069	0.314	0.452	0.268	0.004
<i>Model 4⁶</i>								
n	1,434	1,434	1,427	1,414	1,414	1,193	454	364

β (95% CI)	-0.004 (-0.005,-0.003)	-0.62 (-0.85,-0.39)	0.87 (0.55,1.18)	3.44 (0.83,6.04)	0.56 (-1.30,2.42)	0.92 (-0.36,2.20)	0.02 (0.002,0.05)	1.29 (-0.07,0.66)
p-value	<0.001	<0.001	<0.001	0.010	0.566	0.157	0.029	0.063
Model 5⁷								
n	747	747	744	737	737	620	237	193
β (95% CI)	-0.006 (-0.007,-0.004)	-0.79 (-1.21,-0.37)	1.27 (0.68,1.86)	5.41 (0.55,10.3)	0.67 (-2.77,4.11)	1.48 (-0.85,3.81)	0.04 (-0.10,0.08)	2.9
p-value	<0.001	<0.001	<0.001	0.029	0.704	0.213	0.128	0.045

¹Values are β coefficients and 95% confidence intervals (95% CI).

²**Crude model** adjusted for sex, age and center (random intercept).

³**Model 1** adjusted for sex, age, center (random intercept), family affluence scale, maternal education and moderate-to-vigorous physical activity (counts per minute). SBP, DBP, CRF and TC/HDL-c were additionally adjusted for sum of four skinfold thicknesses.

⁴**Model 2** adjusted for variables in model 1 plus under-reporting.

⁵**Model 3** adjusted for variables in model 1 plus over-reporting.

⁶**Model 4** adjusted for variables in model 1 plus dietary misreporting including under-reporters and over-reporters.

⁷**Model 5** adjusted for variables in model 1 excluding under-reporters and over-reporters.

Statistical significance level at $p < 0.05$.

CMR, cardiometabolic risk score; CRF, cardiorespiratory fitness; DBP, diastolic blood pressure; HDL-c, high density lipoprotein cholesterol; SBP, systolic blood pressure; TC, total cholesterol; WC, waist circumference; WHeR, waist-to-height ratio.

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Supplementary Table 1. Sociodemographic data for those included in the present analyses vs those excluded.¹

	Included	Excluded	p-value*
n	1512	2016	
Age (years)	14.7 ±1.2	14.8 ±1.2	0.002
Sex			
Boys	684 (45.2)	999 (49.6)	0.011
Girls	828 (54.8)	1017 (50.4)	
Family Affluence Scale			
Low	147 (9.8)	314 (15.9)	<0.001
Medium	824 (54.8)	1150 (58.4)	
High	534 (35.4)	506 (25.7)	
Maternal education			
Lower education	98 (6.8)	183 (9.8)	<0.001
Lower secondary education	353 (24.4)	525 (28.1)	
Higher secondary education	427 (29.5)	612 (32.8)	
Higher education/university degree	569 (39.3)	548 (29.3)	
Weight (kg)	56.4 (49.9-63.9)	58.2 (51.0-66.4)	<0.001
Height (cm)	165.5 ±9.1	166.1 ±9.2	0.051
BMI (kg/m²)	20.4 (18.6-22.8)	21.0 (19.1-23.4)	<0.001
BMI category			
Underweight (<18.5 in adults)	108 (7.1)	106 (5.3)	0.001
Normal weight (18.5-24.9 in adults)	1097 (72.6)	1400 (69.4)	
Overweight (25-30 in adults)	236 (15.6)	383 (19.0)	
Obesity (>30 in adults)	71 (4.7)	127 (6.3)	

¹Values are means ± standard deviations (SDs), medians (25th-75th percentile) or n (percentage).

**p*<0.005. Paired *t*-test for age and height, Kruskal-Wallis test for weight and BMI, and chi-squared test for sex, Family Affluence Scale, maternal education and BMI category.

BMI: body mass index.

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Supplementary Table 2. Reference and cut-off values used to identify misreporting among adolescents.¹

Sex	n	CV _{rel} ² (%)	CV _{per} ³ (%)	CV _{mTEE} ⁴ (%)	Cut-offs values (%) ⁵
Boys	684	32.1	12.9	8.2	±27.4
Girls	828	32	13.4	8.2	±27.6

¹Values are percentages of the Coefficient of Variation (CV).

²Intraindividual variation of energy intake.

³Error in predicted energy expenditure requirements.

⁴Day-to-day variation and measurement error for total energy expenditure based on the doubly labelled water ⁽³⁸⁾.

⁵Cut-offs values calculated according to Huang et al. ⁽³⁶⁾.

CV, coefficient of variation; EI, energy intake; TEE, total energy expenditure.

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Supplementary Table 3. Transformations made to continuous variables to reach normality.

Variable	Transformation	
WC	1/square	$1/WC^2$
WHeR	1/square	$1/WHeR^2$
Sum four skinfold thicknesses	square root	sum four skinfold thicknesses ^{1/2}
SBP	no transformation needed	-
DBP	no transformation needed	-
CRF	no transformation needed	-
Glucose	no transformation needed	-
Insulin	logarithm	log insulin
HOMA	logarithm	log HOMA
TG	logarithm	log TG
HDL-c	logarithm	log HDL-c
TC/HDL-c	1/(square root)	$1/(TC-HDL-c)^{1/2}$
Cardiometabolic risk score	no transformation needed	-
EI	logarithm	log EI

CRF, cardiorrespiratory fitness; DBP, diastolic blood pressure; EI, energy intake; HDL-c, high density lipoprotein cholesterol; HOMA, homeostatic model assessment; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; WC, waist circumference; WHeR, waist-to-height ratio.

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Supplementary Table 4. Descriptive characteristics of the study participants by reporting status (n=1,512).¹

	n	All	n	Under-reporters	n	Plausible reporters	n	Over-reporters
WC								
<90th percentile	1343	90	300	80.7	707	90.9	340	98
≥90th percentile	150	10	72	19.3	71	9.1	7	2
WHeR								
<0.5	1339	89.5	296	79.6	705	90.6	338	97.4
≥0.5	158	10.5	76	20.4	73	9.4	9	2.6
Sum 4 skinfold thickness								
<90th percentile	1340	89.9	296	80.7	704	90.8	340	97.7
≥90th percentile	150	10.1	71	19.3	71	9.2	8	2.3
SBP								
<130 mm Hg	1309	87.5	307	83	680	87.6	322	92
≥130 mm Hg	187	12.5	63	17	96	12.4	28	8
DBP								
<85 mm Hg	1479	98.9	364	98.4	769	99.1	346	98.9
≥85 mm Hg	17	1.1	6	1.6	7	0.9	4	1.1
CRF (mL · kg⁻¹ · min⁻¹)								
≤39 mL · kg⁻¹ · min⁻¹ (boys)/ ≤37.5 mL · kg⁻¹ · min⁻¹ (girls)	842	66.9	164	56.8	449	69	229	72
>39 mL · kg⁻¹ · min⁻¹ (boys)/ >37.5 mL · kg⁻¹ · min⁻¹ (girls)	416	33.1	125	43.2	202	31	89	28
Glucose								
<100 mg· dL⁻¹	440	91.5	110	94.8	219	89	111	93.3
≥100 mg· dL⁻¹	41	8.5	6	5.2	27	11	8	6.7
Insulin								
<14 μIU· dL⁻¹	390	82.3	83	74.8	205	83.7	102	86.4
≥14 μIU· dL⁻¹	84	17.7	28	25.2	40	16.3	16	13.6
HOMA index								

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	<3	385	81.4	83	74.8	202	82.8	100	84.8
	≥3	88	18.6	28	25.2	42	17.2	18	15.2
TG									
	<150 mg· dL⁻¹	465	96.7	109	94	240	97.6	116	97.5
	≥150 mg· dL⁻¹	16	3.3	7	6	6	2.4	3	2.5
HDL									
	≤40 mg· dL⁻¹	453	94.2	109	94	232	94.3	112	94.1
	>40 mg· dL⁻¹	28	5.8	7	6	14	5.7	7	5.9
TC/HDL-c									
	<3.8	439	91.3	100	86.2	228	92.7	111	93.3
	≥3.8	42	8.7	16	13.8	18	7.3	8	6.7
Cardiometabolic risk score									
	<1 SD	329	86.6	61	75.3	170	85.9	98	97
	≥1 SD	51	13.4	20	24.7	28	14.1	3	3

¹Values are n (percentage).

CRF, cardiorespiratory fitness; DBP, diastolic blood pressure; HDL-c, high density lipoprotein cholesterol; HOMA, homeostatic model assessment; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; WC, waist circumference; WHeR, waist-to-height ratio.

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Supplementary Table 5. Multilevel Poisson regression analyses on the association between dietary misreporting (under-reporting n=271, over-reporting n=286) and dichotomized cardiometabolic risk indicators¹

Plausible reporters (ref.) vs under-reporters	Crude model ²		Model 1 ³		Model 2 ⁴	
	PR (95% CI)	p-value	PR (95% CI)	p-value	PR (95% CI)	p-value
WC (cm)						
<90th percentile	Ref.		Ref.		Ref.	
≥90th percentile	1.54 (0.85,2.79)	0.160	1.76 (0.98,3.16)	0.058	1.33 (0.55,3.20)	0.525
WHeR						
<0.5	Ref.		Ref.		Ref.	
≥0.5	1.92 (1.15,3.20)	0.012	2.15 (1.15,4.02)	0.016	1.71 (0.75,3.89)	0.204
Sum 4 skinfold thicknesses (mm)						
<90th percentile	Ref.		Ref.		Ref.	
≥90th percentile	1.94 (1.34,2.82)	<0.001	2.12 (1.40,3.22)	<0.001	1.69 (1.05,2.73)	0.031
SBP (mm Hg)						
<130	Ref.		Ref.		Ref.	
≥130	1.55 (1.09,2.21)	0.016	1.68 (1.27,2.23)	<0.001	1.35 (0.83,2.21)	0.230
DBP (mm Hg)						
<85	Ref.		Ref.			
≥85	0.99 (0.27,3.66)	0.992	1.30 (0.43,3.92)	0.646	,	,
CRF (ml·kg ⁻¹ ·min ⁻¹)						
≤39 (boys)/≤37.5 (girls)	1.67 (1.02,2.74)	0.041	1.91 (1.14,3.19)	0.014	1.56 (0.92,2.65)	0.098
>39 (boys)/>37.5 (girls)	Ref.		Ref.		Ref.	
Glucose (mg·dL ⁻¹)						
<100	Ref.		Ref.			
≥100	0.55 (0.59,1.06)	0.075	0.63 (0.32,1.25)	0.188	,	,

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Insulin ($\mu\text{IU}\cdot\text{dL}^{-1}$)						
<14	Ref.		Ref.			
≥ 14	1.36 (0.92,1.99)	0.124	1.46 (1.00,2.14)	0.052	,	,
HOMA index						
<3	Ref.		Ref.		Ref.	
≥ 3	1.29 (0.87,1.90)	0.206	1.43 (1.00,2.07)	0.057	1.10 (0.64,1.87)	0.738
TG ($\text{mg}\cdot\text{dL}^{-1}$)						
<150	Ref.		Ref.			
≥ 150	1.34 (0.55,3.25)	0.519	1.46 (0.58,3.64)	0.423	,	,
HDL ($\text{mg}\cdot\text{dL}^{-1}$)						
≤ 40	0.96 (0.40,2.35)	0.935	0.95 (0.40,2.28)	0.909	,	,
>40	Ref.		Ref.			
TC/HDL,c						
<3.8	Ref.		Ref.			
≥ 3.8	1.19 (0.72,1.99)	0.499	1.22 (0.72,2.04)	0.460	,	,
Cardiometabolic risk score						
<1 SD	Ref.		Ref.			
≥ 1 SD	1.50 (1.04,2.15)	0.030	1.80 (1.14,2.84)	0.011	,	,
Plausible reporters (ref.) vs over-reporters	Crude model ²		Model 1 ²		Model 2 ³	
	PR (95% CI)	p-value	PR (95% CI)	p-value	PR (95% CI)	p-value
WC (cm)						
<90th percentile	Ref.		Ref.		Ref.	
≥ 90 th percentile	0.12 (0.04,0.36)	<0.001	0.12 (0.03,0.45)	0.001	0.14 (0.04,0.52)	0.004
WHeR						
<0.5	Ref.		Ref.		Ref.	

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≥ 0.5	0.26 (0.12,0.57)	0.001	0.27 (0.13,0.58)	0.001	0.31 (0.14,0.69)	0.004
Sum 4 skinfold thicknesses (mm)						
<90th percentile	Ref.		Ref.		Ref.	
≥ 90 th percentile	0.21 (0.06,0.78)	0.020	0.20 (0.06,0.64)	0.007	0.21 (0.07,0.65)	0.007
SBP (mm Hg)						
<130	Ref.		Ref.		Ref.	
≥ 130	0.59 (0.35,1.01)	0.054	0.54 (0.32,0.89)	0.016	0.61 (0.37,0.99)	0.048
DBP (mm Hg)						
<85	Ref.		Ref.			
≥ 85	1.03 (0.38,2.78)	0.948	0.99 (0.31,3.11)	0.983	,	,
CRF ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)						
≤ 39 (boys)/ ≤ 37.5 (girls)	1.02 (0.72,1.44)	0.932	1.12 (0.68,1.83)	0.668	,	,
>39 (boys)/>37.5 (girls)	Ref.		Ref.			
Glucose ($\text{mg}\cdot\text{dL}^{-1}$)						
<100	Ref.		Ref.			
≥ 100	0.69 (0.49,0.97)	0.032	0.67 (0.45,1.01)	0.057	,	,
Insulin ($\mu\text{IU}\cdot\text{dL}^{-1}$)						
<14	Ref.		Ref.			
≥ 14	0.76 (0.56,1.04)	0.085	0.77 (0.53,1.11)	0.162	,	,
HOMA index						
<3	Ref.		Ref.		Ref.	
≥ 3	0.77 (0.57,1.04)	0.087	0.76 (0.54,1.07)	0.120	0.93 (0.73,1.21)	0.600
TG ($\text{mg}\cdot\text{dL}^{-1}$)						
<150	Ref.		Ref.			
≥ 150	0.91 (0.37,2.23)	0.833	0.97 (0.50,1.89)	0.927	,	,

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HDL (mg·dL ⁻¹)						
≤40	1.04 (0.56,1.91)	0.910	0.83 (0.46,1.51)	0.544	,	,
>40	Ref.		Ref.			
TC/HDL,c						
<3.8	Ref.		Ref.			
≥3.8	0.85 (0.48,1.49)	0.569	0.82 (0.50,1.33)	0.411	,	,
Cardiometabolic risk score						
<1 SD	Ref.		Ref.			
≥1 SD	0.26 (0.09,0.75)	0.013	0.25 (0.08,0.80)	0.020	,	,

¹Values are prevalence ratio (PR) and 95% confidence intervals (95% CI.)

²Crude model adjusted for age, gender and center (random intercept)

³Model 1adjusted for age, gender, family affluence scale, maternal education and moderate,to,vigorous physical activity (counts per minute) and center (random intercept).

⁴Model 2adjusted for age, gender, family affluence scale, maternal education and moderate,to,vigorous physical activity (counts per minute) and center (random intercept) plus those variables with $p>0.200$ in Model 1. Only those variables that are part of the cardiometabolic risk score were entered as covariates.

Statistical significance level at $p<0.05$.

CRF, cardiorrespiratory fitness; DBP, diastolic blood pressure; HDL-c, high density lipoprotein cholesterol; HOMA, homeostatic model assessment; SBP; systolic blood presure; TC, total cholesterol; TG, triglycerides; WC, waist circumference; WHeR, wais-to-height ratio.