Nutrition Research 111 (2023) 14–23

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/NTR

Original Research

# A lower energy intake contributes to a better cardiometabolic profile in adolescence: Data from the EPITeen cohort

*Joana Pinto Costa*[a,b,*], *Vânia Magalhães*[a,b], *Joana Araújo*[a,b,c], *Elisabete Ramos*[a,b,c]

[a] *EPIUnit, Instituto de Saúde Pública, Universidade do Porto, Rua das Taipas n° 135, 4050-600 Porto, Portugal*
[b] *Laboratório para a Investigação Integrativa e Translacional em Saúde Populacional (ITR), Instituto de Saúde Pública da Universidade do Porto, Porto, Portugal, Rua das Taipas, n° 135, 4050-600, Porto, Portugal*
[c] *Departamento de Ciências da Saúde Pública e Forenses e Educação Médica, Faculdade de Medicina da Universidade do Porto, 4200-319, Porto, Portugal*

ARTICLE INFO

*Article history:*
Received 21 July 2022
Revised 12 January 2023
Accepted 19 January 2023

*Keywords:*
Adolescents
Caloric restriction
Cardiometabolic risk factors
Cohort
Energy intake
Young adults

ABSTRACT

Caloric restriction has been associated with improved cardiometabolic health. Available data in humans are commonly based on short follow-up periods, specific diets, or population groups. We hypothesized that participants of a population-based cohort (Epidemiological Health Investigation of Teenagers in Porto) with a dietary pattern characterized by a lower energy intake during adolescence have a better cardiometabolic profile in adolescence and young adulthood than other dietary patterns. At aged 13 and 21 year evaluations, diet, anthropometric, and cardiometabolic measures were assessed. Diet was assessed through a food frequency questionnaire and, at 13 years, summarized in dietary patterns identified by cluster analysis. The lower intake dietary pattern included 40% of the participants. The energy intake misreport was estimated using the Goldberg method. Analysis of variance and analysis of covariance were used to compare cardiometabolic risk factors according to dietary patterns. The mean energy intake was 2394 and 2242 Kcal/d for the total sample at aged 13 years (n = 962) and 21 years (n = 862), respectively. Those belonging to the lower intake dietary pattern showed a 25% and 5% lower energy intake, respectively. In the cross-sectional analysis at aged 13, adolescents belonging to the lower intake dietary pattern presented lower glucose, insulin, triglycerides, and blood pressure values after adjusting for body mass index and parents' education level. Among the plausible reporters, differences were only statistically significant for glucose and systolic blood pressure. Our data support

*Abbreviations:* BMI, body mass index; CALERIE, Comprehensive Assessment of Long-term Effects of Reducing Intake of Energy; CR, calorie restriction; EPITeen, Epidemiological Health Investigation of Teenagers in Porto; estBMR, estimated basal metabolic rate; FFQ, Food Frequency Questionnaire; HDL-C, high-density lipoprotein-cholesterol; NCD, noncommunicable disease; PAL, physical activity level; repEI, reported energy intake; WHO, World Health Organization.

* Corresponding author at: EPIUnit – Institute of Public Health of the University of Porto, Rua das Taipas, 135, 4050-600 Porto, Portugal.
*E-mail address:* joanapintodacosta@ispup.up.pt (J.P. Costa).

https://doi.org/10.1016/j.nutres.2023.01.002

that a dietary pattern characterized by a lower energy intake may contribute to a better cardiometabolic profile in adolescents. However, no significant effect was found in young adulthood.



## 1. Introduction

The prevalence of overweight/obesity in children and adolescents has been increasing over past decades [1]. Childhood obesity is associated with cardiometabolic risk factors in the short term and with an increased risk of noncommunicable diseases (NCDs) in the long term [2–5]. NCDs are the leading death cause worldwide [6].

Several approaches to address NCDs have been discussed, such as caloric restriction (CR), studied since 1935 when McCay found that it increased lifespan in mice [7]. CR has been described as a reduction in energy intake without causing malnutrition [8–10]. Studies using rodents described several health benefits [11]. However, these studies were frequently based on short-term interventions [12,13]. Two prospective studies in nonhuman primates demonstrated improved cardiometabolic risk factors, consistent with the effects found in rodents [14–16].

Studies on humans are scarce because of ethical and methodological barriers. One of the first reports relating low energy intake to human lifespan was in the Okinawan population [17]. Okinawans consumed approximately 16.4% below the average energy intake in Japan, presenting 30% to 40% lower death rates from NCDs than the rest of Japan [17]. Another study comparing Caloric Restriction Society members with healthy age-matched individuals eating typical American diets found that the members of this society had lower adiposity, total serum cholesterol, low-density lipoprotein-cholesterol, fasting plasma insulin, glucose, and blood pressure, and higher high-density lipoprotein-cholesterol (HDL-C) [18]. These observational studies were conducted in specific populations, so it is impossible to exclude that health benefits result from other factors that characterize these groups. The Comprehensive Assessment of Long-term Effects of Reducing Intake of Energy (CALERIE) trial was the first controlled clinical trial of CR with adequate nutrient provision over 2 years in healthy, nonobese, young and middle-aged adults [19]. At 12 months and 24 months, the CR group presented reduced body weight, body fat, systolic and diastolic blood pressure, metabolic syndrome score, glucose values, total cholesterol, low-density lipoprotein-cholesterol, and insulin resistance measures, whereas HDL-C increased [20].

The study of the dietary patterns of 13-year-old adolescents from the Epidemiological Health Investigation of Teenagers in Porto (EPITeen) cohort [21] identified 4 dietary patterns, with 1 being characterized by a lower energy intake (i.e., lower consumption of the majority of food groups), integrating 40% of subjects. Although the healthier pattern (16.1%) was characterized by the highest consumption of seafood, soup, vegetables/legumes, fruit, and added fats, the highest consumption of dairy marked the dairy products pattern (29.7%) and the fast food and sweets pattern (14.2%) was characterized by the highest intake of fast food, sweets, and pastry, soft drinks, and coffee or tea, as described in detail elsewhere [22]. Later analysis showed that the food group consumption at aged 13 years seems to track into young adulthood [23]. This observational study is a unique opportunity to study the effect of a lower intake energy diet from adolescence to young adulthood in real life, addressing the ethical limitations that an experimental study would pose. To the best of our knowledge, no other studies have focused on the effect of lower energy intake during adolescence and no observational studies have been based on the general population. We hypothesized that participants with a dietary pattern characterized by a lower energy intake during adolescence have a better cardiometabolic profile in adolescence and young adulthood than participants with other dietary patterns. Thus, using data from this population-based cohort, we aimed to evaluate it.

## 2. Methods and materials

### 2.1. *Participant selection*

Data were collected as part of EPITeen, a population based-cohort that recruited 13-year-old adolescents born in 1990 and enrolled in public or private schools in Porto, Portugal [21]. Participants were evaluated at 13 (baseline), 17, 21, 24, and 27 years. The evaluations comprised a standardized questionnaire and a physical examination, and dietary evaluation was performed at 13 and 21 years; thus, this study includes data from these later evaluations.

The study complies with the Declaration of Helsinki, and the Ethics Committee of Hospital São João and the Institute of Public Health from the University of Porto approved the research protocol (193-12). Written informed consent was obtained from parents or legal tutors and adolescents in the waves performed under those aged 18 years and from participants who were aged 21 years and older.

For this analysis, all the EPITeen participants were eligible. Those who did not provide dietary information at baseline, were outliers regarding food intake, or did not provide blood samples at 13 years and 21 years of age were excluded. Therefore, at baseline, 1489 participants (68.9%) provided dietary information used to identify the dietary patterns as described elsewhere [22]; from those, 962 (64.6%) participants provided blood samples and were included in the cross-sectional analysis. There were no significant differences between included (n = 962) and excluded (n = 527) participants (Supplemental Table S1). Of the 1489 participants with a dietary pattern identified at 13 years of age, 989 (66.4%) were evaluated at 21 years;

88 were outliers regarding food intake, and 30 did not provide a blood sample. Thus, 862 (57.9%) participants were included in the longitudinal analysis. They were generally similar to those excluded (n = 627), except for dietary patterns and parents' education (Supplemental Table S2).

### 2.2. Covariates

Parents' education was the number of completed school years, and participants were classified into 1 of the categories (≤6, 7-9, 10-12, or >12 years) according to parental highest educational level, based on data from those aged 21 years. Time spent in sedentary activities (watching television, playing computer and/or videogames, reading, and/or doing homework) was self-reported for weekdays (minutes/d) and weekends (minutes/weekend), and the mean time spent in sedentary activities was calculated (minutes/d). Leisure-time physical activity was self-reported through a validated question [24], and 3 categories were considered: most of the time sitting, most of the time standing and/or walking, or most of the time active/very active.

### 2.3. Anthropometrics

Anthropometric measures were obtained with the subject standing in light indoor clothes and no shoes, according to international guidelines, at both waves [25]. Weight was measured (kg), and body fat (%) by foot-to-foot bioelectrical impedance was estimated using a digital scale (Tanita TBF-300, Tanita Corporation of America, Inc, Arlington Heights, IL, USA), and height (cm) was measured with the head of the participant in the Frankfurt plane. Waist circumference (cm) was measured with a flexible and nondistensible tape at the midpoint between the lower limit of the rib cage and the iliac crest at the end of expiration.

At aged 13 years, adolescents were classified into 4 categories according to the age- and sex-specific body mass index (BMI) reference z scores from the World Health Organization (WHO) [26]: thinness ($z < -2$ SD), normal weight ($-2$ SD $\leq z \leq +1$ SD), overweight ($+1$ SD $< z \leq +2$ SD), and obesity ($z > +2$ SD). WHO classification for adults aged 21 years was used [27]: underweight, BMI <18.5 kg/m$^2$; normal weight, 18.5 ≤ BMI <25.0 kg/m$^2$; overweight, 25.0 ≤ BMI <30.0 kg/m$^2$; and obesity, BMI ≥30.0 kg/m$^2$.

### 2.4. Dietary information and dietary patterns

A semiquantitative food frequency questionnaire (FFQ) [28] reporting the previous 12 months, validated for the Portuguese adult population [29], was applied. In the aged 13 years group, the FFQ comprised 91 food or beverage items adapted for adolescents, including foods more frequently eaten by this age group [30], and was completed at home by the adolescent with the help of the parents or legal tutor. For the aged 21 years group, an interviewer applied the questionnaire through a face-to-face interview at the university department. Both versions comprised a frequency section with 9 possible responses ranging from never to 6 times or more a day and an open-ended section for foods not listed in the questionnaire when eaten at least once a week.

Considering data from the FFQ at aged 13 years, food and beverages were combined into 14 groups according to nutritional similarities, as previously described [30]. Food intake data were obtained by multiplying the frequency of consumption of each food item by the nutrient content of the specified portion size. Seasonal variation in food consumption was also considered according to participants' replies. To estimate nutrient intake from the evaluated food intake, we used the software Food Processor Plus (version 7.2, 1997, ESHA Research, Salem, OR, USA) based on US Department of Agriculture values. Based on the intake of the food groups and considering the standardized energy contribution of each food group, 4 dietary patterns were identified by cluster analysis, at aged 13 years, with the following frequencies and mean (SD) energy intake: healthier (n = 239; 16.1%; 2724.4 [487.5] Kcal/d), dairy products (n = 442; 29.7%; 2621.3 [362.3] Kcal/d), fast food and sweets (n = 212; 14.2%; 3443.2 [482.1] Kcal/d), and lower intake (n = 596; 40.0%; 1811.9 [378.3] Kcal/d) [22].

#### 2.4.1. Energy intake misreport

Individual reported energy intake (repEI) (Kcal/d) was based on the FFQ, and the estimated basal metabolic rate (estBMR) (Kcal/d) was calculated according to age- and sex-specific Schofield equations [31]. Individual energy intake misreport was evaluated by applying the Goldberg method [32], later corrected by Black [33]. Briefly, the ratio between the repEI and estBMR (repEI:estBMR) was computed. The physical activity level (PAL) was estimated based on self-reported leisure-time activity through a validated question [24]. The question allowed us to assign participants to 3 PAL groups: low, moderate, and vigorous. The age-specific PAL values were 1.6, 1.8, and 2.0 at 13 years of age, and 1.4, 1.6, and 1.8 at 21 years of age, according to the European Food Safety Authority Panel on Nutrition, Novel Foods and Food Allergens [34]. The corresponding PAL value was multiplied by a factor considering the usual intake variability, the BMR, and physical activity. Assuming a normal distribution, we used the 2 SDs to define the limits for PAL. Participants were classified as underreporters or overreporters if the repEI:estBMR ratio was below the lower limit or above the upper limit of the expected PAL, respectively.

### 2.5. Cardiometabolic risk factors

In the 13-year-old group, blood pressure was measured with a mercury sphygmomanometer using the auscultatory method, following the recommendations of the American Academy of Pediatrics [35]. In the 21-year-old group, blood pressure was measured using an oscillometric method (OMRON Blood Pressure Monitor, M6 Comfort) according to standardized procedures. After 10 minutes of rest, 2 blood pressure measurements were taken, separated by at least 5 minutes, at both ages. A third measure was taken when the difference between the first 2 was higher than 5 mm Hg. The average of the 2 closest measurements was used in this analysis. An intravenous blood sample after an overnight fast was taken from an antecubital vein at both ages. Serum glucose, cholesterol, triglycerides, and HDL-C were determined using an Olympus AU5400 automated clinical chemistry analyzer (Beckman-Coulter, Brea, CA, USA). Serum insulin was measured by electrochemiluminescent immunoassay using

**Table 1 – Anthropometric and cardiometabolic characteristics according to dietary patterns, cross-sectional analysis at 13 years old, considering all the participants (n = 962) and those classified as plausible reporters (n = 616).**

| 13 y old | Dietary patterns identified at aged 13 y | | | | | | | | P value |
|---|---|---|---|---|---|---|---|---|---|
| | Lower intake | | Healthier | | Dairy products | | Fast food and sweets | | |
| All participants (n = 962) | Mean (SD) | | | | | | | | |
| **n (%)** | 401 | (41.7) | 143 | (14.9) | 290 | (31.1) | 128 | (13.3) | |
| **Energy intake, Kcal/d** | 1807[a] | (380) | 2730[b] | (495) | 2587[c] | (338) | 3426[d] | (474) | <.001 |
| **Anthropometric measures** | | | | | | | | | |
| Waist circumference, cm | 73.3 [a] | (9.1) | 72.8 [a,b] | (9.4) | 72.1 [a,b] | (8.1) | 70.6[b] | (7.1) | .016 |
| Fat mass, % | 22.4 [a] | (9.6) | 20.8 [a,b] | (9.4) | 20.3 [b] | (9.2) | 20.2 [a,b] | (9.2) | .012 |
| BMI z scores[1] | 0.59[a] | (1.13) | 0.52[a,b] | (1.07) | 0.45[a,b] | (1.05) | 0.29[b] | (0.93) | .036 |
| BMI, kg/m$^2$ | 21.4[a] | (3.8) | 21.1[a,b] | (3.8) | 20.8[a,b] | (3.4) | 20.2[b] | (2.8) | .005 |
| **Glucose metabolism parameters** | | | | | | | | | |
| Glucose, mg/dL | 85 | (10) | 84 | (8) | 86 | (9) | 86 | (9) | .191 |
| Insulin, μUI/mL | 7.96 | (5.87) | 8.17 | (5.19) | 7.70 | (6.64) | 8.19 | (5.47) | .821 |
| HOMA-IR [2] | 1.68 | (1.29) | 1.72 | (1.13) | 1.66 | (1.48) | 1.75 | (1.24) | .912 |
| **Serum lipid levels, mg/dL** | | | | | | | | | |
| Total cholesterol | 166 | (31) | 165 | (32) | 170 | (32) | 164 | (29) | .229 |
| Triglycerides | 64 | (26) | 67 | (35) | 64 | (27) | 66 | (27) | .727 |
| HDL-cholesterol | 49 | (11) | 49 | (12) | 50 | (11) | 49 | (10) | .309 |
| LDL-cholesterol | 105 | (26) | 102 | (27) | 107 | (25) | 103 | (22) | .269 |
| **Blood pressure, mm Hg** | | | | | | | | | |
| Systolic | 113 | (11) | 113 | (11) | 114 | (11) | 112 | (11) | .365 |
| Diastolic | 68 | (8) | 68 | (8) | 68 | (9) | 70 | (7) | .114 |
| Plausible reporters (n = 616) | Mean (SD) | | | | | | | | |
| **n (%)** | 186 | (30.2) | 107 | (17.4) | 248 | (40.3) | 75 | (12.2) | |
| **Energy intake, Kcal/d** | 2042[a] | (272) | 2726[b] | (441) | 2585[c] | (312) | 3263[d] | (482) | <.001 |
| **Anthropometric measures** | | | | | | | | | |
| Waist circumference, cm | 70.1[a] | (7.0) | 72.5[b] | (9.0) | 71.4[a,b] | (7.5) | 71.4[a,b] | (7.2) | .054 |
| Fat mass, % | 22.3[a] | (9.1) | 21.1[a,b] | (9.1) | 20.4[a,b] | (9.4) | 18.6[b] | (9.2) | .024 |
| BMI z scores[1] | 0.23 | (1.01) | 0.49 | (1.06) | 0.37 | (1.01) | 0.37 | (0.99) | .182 |
| BMI, kg/m$^2$ | 20.3 | (3.2) | 21.0 | (3.77) | 20.5 | (3.2) | 20.4 | (2.9) | .310 |
| **Glucose metabolism parameters** | | | | | | | | | |
| Glucose, mg/dL | 84[a,b] | (10) | 84[a] | (8) | 86[a,b] | (8) | 87[b] | (9) | .012 |
| Insulin, μUI/mL | 7.68 | (5.88) | 7.84 | (4.73) | 7.65 | (6.71) | 7.88 | (5.31) | .987 |
| HOMA-IR [2] | 1.61 | (1.28) | 1.62 | (0.99) | 1.65 | (1.50) | 1.72 | (1.24) | .942 |
| **Serum lipid levels, mg/dL** | | | | | | | | | |
| Total cholesterol | 171 | (32) | 166 | (32) | 169 | (32) | 164 | (26) | .290 |
| Triglycerides | 64 | (24) | 65 | (33) | 65 | (28) | 67 | (28) | .880 |
| HDL-cholesterol | 51 | (11) | 50 | (12) | 51 | (11) | 48 | (9) | .340 |
| LDL-cholesterol | 107 | (27) | 103 | (26) | 106 | (25) | 102 | (21) | .346 |
| **Blood pressure, mm Hg** | | | | | | | | | |
| Systolic | 110[a] | (10) | 112[a,b] | (12) | 113[b] | (11) | 114[b,c] | (10) | .039 |
| Diastolic | 67 | (8) | 67 | (8) | 68 | (9) | 69 | (8) | .188 |

Abbreviations: BMI, body mass index; HDL, high-density lipoprotein; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; LDL, low-density lipoprotein.
Different superscript letters indicate significant differences among dietary patterns at $p < 0.05$ in the Tukey comparison.
[1] According to World Health Organization criteria for z scores [26].
[2] HOMA-IR = insulin (μU/mL) × glucose (mg/dL)/405 [36].

a Cobas e411 automated analyzer (Roche, Roche Diagnostics GmbH, Mannheim, Germany). All determinations took place in the Clinical Pathology Department of the São João Hospital Centre, Porto. The Homeostatic Model Assessment for Insulin Resistance [36] was used as a marker of insulin resistance.

### 2.6. *Statistical analyses*

Data are presented as counts (percentages) or means (SD). Proportions were compared using $\chi^2$ or Fisher tests when appropriate. The analysis of variance and Tukey tests were used to compare the anthropometric measures and cardiometabolic risk factors according to dietary patterns. The analysis of covariance and Tukey tests were used to estimate adjusted means and respective 95% CIs according to dietary patterns. In the 13-year-old group, the means were adjusted for participants' BMI and parents' education level; in the 21-year-old group, the means were adjusted for sex, BMI, and participants' education level. Statistical analysis was performed using IBM SPSS 24, considering a bilateral significance level of 0.05.

**Table 2 – Anthropometric and cardiometabolic characteristics according to dietary patterns, cross-sectional analysis at 13 years old adjusted for BMI of participants and education level of the parents, considering all the participants (n = 962) and those classified as plausible reporters (n = 616).**

| 13 y old | Dietary patterns identified at aged 13 y | | | | | | | | P value |
|---|---|---|---|---|---|---|---|---|---|
| | Lower intake | | Healthier | | Dairy products | | Fast food and sweets | | |
| All participants (n = 962) | Estimated mean (95% CI) | | | | | | | | |
| **n (%)** | 401 | (41.7) | 143 | (14.9) | 290 | (31.1) | 128 | (13.3) | |
| **Anthropometric measures**[1] | | | | | | | | | |
| Waist circumference, cm | 73.4[a] | (72.5–74.2) | 72.9[a] | (71.5–74.3) | 72.2[z,b] | (71.2–73.2) | 70.6[b] | (69.0–72.1) | .013 |
| Fat mass, % | 22.4[a] | (21.5–23.3) | 21.2[a,b] | (19.6–22.7) | 20.5[b] | (19.4–21.6) | 20.1[b] | (18.5–21.8) | .023 |
| BMI z scores [2] | 0.59[a] | (0.48–0.69) | 0.51[a,b] | (0.33–0.69) | 0.45[a,b] | (0.32–0.57) | 0.28[b] | (0.10–0.47) | .036 |
| BMI, kg/m$^2$) | 21.4[a] | (21.1–21.8) | 21.2[a,,c] | (20.6–21.7) | 20.8[b.,c] | (20.4–21.3) | 20.2[b] | (19.6–20.8) | 005 |
| **Glucose metabolism parameters** | | | | | | | | | |
| Glucose, mg/dL | 84[a] | (83–85) | 84[a,b] | (83–86) | 86[b] | (85; –87) | 85[a,b] | (84–87) | 0.159 |
| Insulin, µUI/mL | 7.60 | (7.04–8.15) | 8.48 | (7.55–9.42) | 8.05 | (7.40–8.70) | 8.42 | (7.44–9.40) | 282 |
| HOMA-IR[3] | 1.61 | (1.48–1.73) | 1.79 | (1.58–2.00) | 1.74 | (1.59–1.89) | 1.79 | (1.58–2.01) | 0.271 |
| **Serum lipid levels, mg/dL** | | | | | | | | | |
| Total cholesterol | 166 | (163–169) | 165 | (160–170) | 170 | (166–173) | 165 | (159–170) | .322 |
| Triglycerides | 63 | (61–66) | 67 | (62–72) | 65 | (62–68) | 66 | (62–71) | .483 |
| HDL-cholesterol | 49 | (48–50) | 49 | (48–51) | 50 | (49–51) | 48 | (46–50) | .333 |
| LDL-cholesterol | 104 | (102–107) | 102 | (98–106) | 107 | (104–110) | 103 | (99–108) | .350 |
| **Blood pressure, mm Hg** | | | | | | | | | |
| Systolic | 112 | (111–113) | 113 | (111–114) | 114 | (113–115) | 113 | (111–115) | .113 |
| Diastolic | 67[a] | (66–68) | 68[a,b] | (67–69) | 68[b,c] | (68–69) | 70[c] | (69–71) | .005 |
| **Plausible reporters (n = 616)** | Estimated mean (95% CI) | | | | | | | | |
| **n (%)** | 186 | (30.2) | 107 | (17.4) | 248 | (40.3) | 75 | (12.2) | |
| **Glucose metabolism parameters** | | | | | | | | | |
| Glucose, mg/dL | 84[a] | (83–85) | 84[a,c] | (82–86) | 86 [b] | (85–87) | 87[b,c] | (85–89) | .017 |
| Insulin, µUI/mL | 7.71 | (6.88–8.55) | 7.98 | (6.86–9.09) | 7.86 | (7.13–8.59) | 7.72 | (6.40–9.04) | .983 |
| HOMA-IR[3] | 1.61 | (1.43–1.79) | 1.67 | (1.42–1.92) | 1.70 | (1.54–1.87) | 1.67 | (1.38–1.96) | .914 |
| **Serum lipid levels, mg/dL** | | | | | | | | | |
| Total cholesterol | 171 | (167–176) | 166 | (160–172) | 170 | (166–173) | 164 | (157–171) | .317 |
| Triglycerides | 64 | (60–68) | 65 | (60–70) | 65 | (61–68) | 66 | (60–73) | .939 |
| HDL-cholesterol | 51 | (50–53) | 51 | (49–53) | 51 | (50–52) | 48 | (46–51) | .236 |
| LDL-cholesterol | 107 | (104–111) | 102 | (97–107) | 106 | (102–109) | 103 | (97–109) | .304 |
| **Blood pressure, mm Hg** | | | | | | | | | |
| Systolic | 111[a] | (109–112) | 112[a,b] | (110–114) | 113[b] | (112–114) | 114[b] | (112–117) | .030 |
| Diastolic | 67[a] | (66–68) | 68[a,b] | (66–69) | 68[a,b] | (67–69) | 69[b] | (67–71) | .159 |

Abbreviations: BMI, body mass index; HDL, high-density lipoprotein; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; LDL, low-density protein.
Different superscript letters indicate significant differences among dietary patterns at $P < .05$ in the Tukey comparison.
[1] Adjusted for parents' education level.
[2] According to World Health Organization criteria for z scores [26].
[3] HOMA-IR = insulin (µU/mL) × glucose (mg/dL)/405 [36].

## 3. Results

The mean energy intake among those belonging to the lower intake dietary pattern, at 13 years of age, was 25% lower compared with the total sample (1807 Kcal/d vs. 2394 Kcal/d, respectively) whereas, at age 21 years, this difference was 5% (2129 Kcal/d vs. 2242 Kcal/d, respectively). Regarding the energy intake misreport, the proportion of underreporting among those belonging to the lower intake dietary pattern was 49.1% at 13 years and 28.8% at 21 years (Supplemental Table S3). When considering only the plausible reporters, the mean energy intake was 19% and 2% lower among those belonging to the lower intake dietary pattern than among all the plausible reporters, at age 13 and 21 years, respectively.

In the 13-year-old group, no significant differences were found between dietary patterns regarding glucose metabolism, serum lipid levels, and blood pressure; regarding anthropometric measures, those belonging to the lower intake pattern presented the highest anthropometric measures (Table 1). Considering only those identified as plausible reporters (n = 616), those in the lower intake pattern showed the lowest values for waist circumference and BMI, although not statistically significant, and the highest fat mass percentage. They also presented the best cardiometabolic profile, although the differences were statistically significant only for glucose levels and systolic blood pressure (Table 1). After adjusting for

**Table 3 – Longitudinal approach for comparing anthropometric and cardiometabolic characteristics of the participants according to the dietary patterns identified at baseline, considering all the participants (n = 862) and only those with a plausible report (n = 598).**

| 21 y old | Dietary patterns identified at aged 13 y | | | | | | | | P value |
|---|---|---|---|---|---|---|---|---|---|
| | Lower intake | | Healthier | | Dairy products | | Fast food and sweets | | |
| All participants (n = 862) | Mean (SD) | | | | | | | | |
| **n (%)** | 347 | (40.3) | 135 | (15.7) | 280 | (32.5) | 100 | (11.6) | |
| **Energy intake (Kcal/d)** | 2129[a] | (589) | 2328[b] | (873) | 2318[b] | (703) | 2302[a,b] | (753) | .002 |
| **Anthropometric measures** | | | | | | | | | |
| Waist circumference, cm | 78.3[a] | (10.5) | 76.9[a,b] | (9.1) | 77.3[a,b] | (8.8) | 75.3[b] | (9.8) | .049 |
| Fat mass, % | 20.7 | (9.29) | 19.7 | (8.3) | 19.3 | (8.4) | 20.0 | (8.4) | .225 |
| BMI, kg/m$^2$ | 23.4[a] | (4.2) | 22.7[a,b] | (3.5) | 22.7[a,b] | (3.4) | 22.3[b] | (3.6) | .018 |
| **Glucose metabolism parameters** | | | | | | | | | |
| Glucose, mg/dL | 84 | (11) | 82 | (7) | 83 | (6) | 83 | (7) | .539 |
| Insulin, µUI/mL | 9.36 | (5.45) | 8.29 | (4.35) | 8.54 | (4.57) | 9.01 | (4.37) | .082 |
| HOMA-IR[1] | 1.95 | (1.29) | 1.70 | (0.91) | 1.76 | (0.98) | 1.86 | (0.94) | .064 |
| **Serum lipid levels, mg/dL** | | | | | | | | | |
| Total cholesterol | 177[a,b] | (33) | 170[a] | (33) | 181[b] | (35) | 176[a,b] | (33) | .026 |
| Triglycerides | 86 | (38) | 82 | (35) | 87 | (41) | 84 | (40) | .623 |
| HDL-cholesterol | 57 | (13) | 55 | (13) | 58 | (12) | 57 | (12) | .393 |
| LDL-cholesterol | 102[a,b] | (26) | 98[a] | (27) | 106[b] | (29) | 102[a,b] | (26) | .062 |
| **Blood pressure, mm Hg** | | | | | | | | | |
| Systolic | 108 | (11) | 108 | (12) | 109 | (11) | 108 | (13) | .309 |
| Diastolic | 69 | (8) | 67 | (7) | 69 | (7) | 68 | (8) | .270 |
| **Plausible reporters (n = 598)** | Mean (SD) | | | | | | | | |
| **n (%)** | 228 | (38.1) | 96 | (16.1) | 205 | (34.3) | 69 | (11.5) | |
| **Energy intake (Kcal/d)** | 2250 | (490) | 2312 | (524) | 2333 | (544) | 2275 | (509) | .389 |
| **Anthropometric measures** | | | | | | | | | |
| Waist circumference, cm | 75.7 | (7.7) | 75.2 | (8.5) | 76.6 | (8.5) | 74.2 | (9.0) | .189 |
| Fat mass, % | 19.7 | (8.5) | 19.3 | (8.0) | 19.0 | (8.3) | 19.6 | (7.4) | .843 |
| BMI, kg/m$^2$ | 22.3 | (3.0) | 22.2 | (3.3) | 22.5 | (3.2) | 22.1 | (2.9) | .735 |
| **Glucose metabolism parameters** | | | | | | | | | |
| Glucose, mg/dL | 83 | (8) | 82 | (7) | 83 | (6) | 83 | (6) | .393 |
| Insulin, µUI/mL | 8.58 | (4.64) | 8.16 | (4.26) | 8.52 | (4.73) | 8.68 | (3.93) | .867 |
| HOMA-IR [1] | 1.78 | (1.17) | 1.65 | (0.89) | 1.75 | (1.01) | 1.78 | (0.87) | .787 |
| **Serum lipid levels, mg/dL** | | | | | | | | | |
| Total cholesterol | 178 | (32) | 171 | (34) | 180 | (35) | 178 | (33) | .178 |
| Triglycerides | 86 | (38) | 83 | (34) | 88 | (43) | 85 | (43) | .735 |
| HDL-cholesterol | 59 | (12) | 57 | (14) | 58 | (12) | 58 | (12) | .697 |
| LDL-cholesterol | 102 | (24) | 97 | (28) | 104 | (28) | 103 | (26) | .231 |
| **Blood pressure, mm Hg** | | | | | | | | | |
| Systolic | 107 | (11) | 106 | (11) | 109 | (11) | 107 | (12) | .107 |
| Diastolic | 68 | (7) | 67 | (7) | 69 | (7) | 68 | (8) | .199 |

Abbreviations: BMI, body mass index; HDL, high-density lipoprotein; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; LDL, low-density lipoprotein.

[1] HOMA-IR = insulin (µU/mL) × glucose (mg/dL) /405 [36].

the participant's BMI and parents' education level, those in the lower intake dietary pattern presented the highest values of anthropometric measures and the lowest parameters of glucose metabolism, triglycerides, and blood pressure, with only anthropometric measures and systolic blood pressure being statistically significant. Considering only the plausible reporters, participants in the lower intake pattern presented the lowest parameters of glucose metabolism, triglycerides, and blood pressure, with only glucose metabolism parameters and blood pressure levels being statistically significant (Table 2).

On the longitudinal analyses, considering dietary patterns at aged 13 years and metabolic parameters at aged 21 years, no significant differences were found, both in the crude analysis (Table 3) and after adjustment (Table 4), considering the total sample or the plausible reporters.

An additional model with physical leisure time activity was tested. Because the results were similar, we opted for the simplest model.

## 4. Discussion

Our results support the hypothesis that a lower energy intake may contribute to a better cardiometabolic profile during adolescence and showed that glucose metabolism and blood

**Table 4 – Anthropometric and cardiometabolic characteristics at 21 years of age, adjusted for sex, BMI, and participants' education level, considering all the participants included in the 21-year-old analysis (n = 862) and only those identified as plausible reporters (n = 598).**

| 21 y old | Dietary patterns identified at aged 13 y | | | | | | | | *p* value |
|---|---|---|---|---|---|---|---|---|---|
| | Lower intake | | Healthier | | Dairy products | | Fast food and sweets | | |
| All participants (n = 862) | Estimated mean (95% CI) | | | | | | | | |
| **n (%)** | 347 | (40.3) | 135 | (15.7) | 280 | (32.5) | 100 | 11.6 | |
| **Anthropometric measures** [1] | | | | | | | | | |
| Waist circumference, cm | 79.3 | (78.1–80.4) | 78.2 | (76.5–79.9) | 78.3 | (77.0–79.6) | 76.9 | (75.0–78.7) | .108 |
| Fat mass, % | 20.8[a] | (19.9–21.7) | 20.1[a,b] | (18.8–21.4) | 19.8[a,b] | (18.8–20.8) | 18.8[b] | (17.4–20.2) | .054 |
| BMI, kg/m$^2$ | 23.7[a] | (233–24.2) | 23.1[a,b] | (22.4–23.8) | 23.1[b] | (22.6–23.6) | 22.6[b] | (21.9–23.4) | .029 |
| **Glucose metabolism parameters** | | | | | | | | | |
| Glucose, mg/dL | 84 | (83–85) | 83 | (82–85) | 83 | (82–85) | 84 | (82–86) | .734 |
| Insulin, µUI/mL | 8.94 | (8.37–9.50) | 8.29 | (7.44–9.13) | 8.61 | (7.99–9.24) | 9.04 | (8.14–9.94) | .436 |
| HOMA-IR[2] | 1.87 | (1.74–2.00) | 1.71 | (1.52–1.91) | 1.78 | (1.64–1.92) | 1.89 | (1.68–2.09) | .388 |
| **Serum lipid levels, mg/dL** | | | | | | | | | |
| Total cholesterol | 173[a] | (169–177) | 166[b] | (160–172) | 179[c] | (174–183) | 172[a,b] | (166–178) | .002 |
| Triglycerides | 81 | (76–86) | 76 | (69–84) | 83 | (77–88) | 79 | (71–86) | .415 |
| HDL-cholesterol | 55 | (54–57) | 53 | (51–55) | 56 | (54–57) | 54 | (52–57) | .113 |
| LDL-cholesterol | 101[a] | (98–105) | 98[a] | (93–103) | 106[b] | (102–110) | 102[a,b] | (97–107) | .015 |
| **Blood pressure, mm Hg** | | | | | | | | | |
| Systolic | 107[a] | (106–109) | 108[a,b] | (106–109) | 109[b] | (108–112) | 110[b] | (108–112) | .027 |
| Diastolic | 68 | (67–69) | 67 | (66–68) | 68 | (67–69) | 68 | (67–70) | .370 |
| **Plausible reporters (n = 598)** | Estimated mean (95% CI) | | | | | | | | |
| **n (%)** | 228 | (38.1) | 96 | (16.1) | 205 | (34.3) | 69 | (11.5) | |
| **Glucose metabolism parameters** | | | | | | | | | |
| Glucose, mg/dL | 84 | (83–85) | 83 | (81–84) | 83 | (82–85) | 84 | (82–85) | .623 |
| Insulin, µUI/mL | 8.12 | (7.42–8.81) | 7.59 | (6.61–8.58) | 8.17 | (7.42–8.92) | 8.24 | (7.17–9.31) | .699 |
| HOMA-IR[2] | 1.70 | (1.54–1.86) | 1.56 | (1.33–1.79) | 1.69 | (1.51–1.87) | 1.71 | (1.46–1.96) | .693 |
| **Serum lipid levels, mg/dL** | | | | | | | | | |
| Total cholesterol | 173[a] | (168–178) | 165[b] | (158–173) | 177[a] | (171–182) | 173[a,b] | (165–181) | .034 |
| Triglycerides | 78 | (72–84) | 74 | (65–82) | 81 | (75–88) | 76 | (67–86) | .397 |
| HDL-cholesterol | 56 | (54–58) | 54 | (51–56) | 56 | (54–58) | 55 | (52–58) | .287 |
| LDL-cholesterol | 102 | (97–106) | 97 | (91–103) | 104 | (100–109) | 102 | (96–109) | .147 |
| **Blood pressure, mm Hg** | | | | | | | | | |
| Systolic | 107 | (106–108) | 107 | (105–108) | 108 | (107–110) | 109 | (107–111) | .178 |
| Diastolic | 67 | (66–68) | 66 | (64–68) | 68 | (67–69) | 68 | (66–69) | .238 |

Abbreviations: BMI, body mass index; HDL, high-density lipoprotein; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; LDL, low-density lipoprotein.
Different superscript letters indicate significant differences among dietary patterns at $P < .05$ in the Tukey comparison.
[1] Adjusted for sex and participant's education level.
[2] HOMA-IR = insulin (µU/mL) × glucose (mg/dL)/405 [36].

pressure were the most helped parameters. However, in the longitudinal analysis, the hypothesis was not confirmed because the results did not reach statistical significance for parameters in early adulthood. In addition to the health benefits described, a generalized lower energy intake may also benefit planetary health [37].

To our knowledge, there is no widely accepted cutoff to define CR. Although no intervention was applied in the EPITeen cohort, the difference in participants' energy intake in to the lower intake pattern identified as plausible, approximately 19% less Kcal/day than the mean energy intake of all the plausible reporters, was even higher than the CR achieved in the CALERIE trial [20]. Results from that trial showed that even a mild CR (approximately 12%) seems to have beneficial effects, with improved cardiometabolic risk factors in adults with average risk at baseline [20].

In this study, participants in the lower intake dietary pattern had the lowest mean energy intake and simultaneously the highest anthropometric measures. The evaluation of energy intake identified almost half (49.1%) of those participants as underreporters. Repeating the analysis excluding those identified as misreporters, the differences in the energy intake were attenuated to around 19% less Kcal/day than the mean energy intake of all the plausible reporters. Following the literature, those who are overweight/obese are more likely to underreport energy intake [38,39]. Thus, when considering only those identified as plausible reporters, those participants belonging to the lower intake dietary pattern, in general, presented the lowest anthropometric measures but a higher fat mass percentage. The highest fat mass percentage might be explained by the higher proportion of females (56.9%) in this dietary pattern, who also have among the highest

proportions of less-educated parents (29.0%) (Supplemental Table S4).

Although there is a dietary pattern designated "healthier," it is worth reinforcing that the lower intake dietary pattern is not considered unhealthy. It is characterized by a general lower consumption of all the food groups but with a varied and relatively healthy diet [22], which may also explain some similarity of results with the healthier pattern.

Although not statistically significant, at 13 years of age, participants belonging to the lower intake dietary pattern tended to present lower values of Homeostatic Model Assessment for Insulin Resistance, triglycerides, and blood pressure. Although the difference is tiny, because the cluster of these factors is associated with a higher cardiometabolic risk in the short and long term [40,41], even a slight difference at this age may have relevant gains from a population viewpoint [42], despite the lack of differences in the longitudinal analysis.

Our results seem to be consistent regarding the glucose metabolism parameters and the triglycerides values, despite this not being valid for all serum lipid parameters evaluated. Studies have suggested an association between hypertriglyceridemia and insulin resistance; a decrease in triglyceride levels seems to be associated with improved insulin sensitivity [43–45].

Although the patterns of food group consumption seem to track from adolescence into young adulthood [23], the differences in the energy intake between dietary patterns were attenuated. This may explain the lack of differences in the longitudinal approach in the cardiometabolic parameters evaluated, which may reinforce the relevance of a lower energy intake (i.e., our results suggest that despite the potential benefit of a lower energy intake dietary pattern, the benefit does not persist when the energy intake differences are attenuated, even when maintaining a similar rank of food consumption). The potential selection bias because of follow-up losses and participant exclusion may reduce the generalizability of our results. However, because this bias may result in a more homogenous sample, we expected it would contribute to reduced differences between groups. Losses to follow-up occurred mainly among adolescents from lower socioeconomic levels, with less-educated parents and belonging to the fast food and sweets dietary pattern, leading to a potentially more homogeneous group at 21 years of age. The proportion of participants belonging to the lower intake dietary pattern remained similar, but there was a sample size reduction.

Assessing diet through an FFQ has some limitations; we minimized it using an FFQ validated for the Portuguese population [29]. Additionally, for those in the 13-year-old age group, the FFQ was self-administered, which may increase the bias, mainly because of a low ability to estimate the amount of food eaten and minimized by considering only the plausible reporters. However, an FFQ has been described as a valuable technique for evaluating diet in population-based studies and a valid method to rank adolescents regarding energy and nutrient intakes [46,47]. To identify the plausible reporters, we used the Goldberg method, later corrected by Black, which is valid compared with the gold standard (Doubly Labelled Water) [48]. Besides, we used some methods, such as using individual PAL instead of assuming a sedentary lifestyle for all, which increased the detection sensitivity [49].

The main strength of our study is the longitudinal approach and the design using an observational population-based approach that allowed us to study the effect of a diet with a lower energy intake from adolescence to young adulthood in real life, addressing the ethical limitations that an experimental study would pose. Using cohort data also allowed us to use cardiometabolic measures obtained, by trained health professionals, from standardized evaluations over the years, reducing the chances of bias compared with data from routine appointments.

To our best knowledge, no other studies have focused on the effect of lower energy intake during adolescence or even an observational study based on the general population rather than a specific group that changes their habits according to the study protocol.

In conclusion, our study seems to bear out that, in adolescents, a dietary pattern characterized by a lower energy intake may contribute to a better cardiometabolic profile by promoting better glucose metabolism and lower blood pressure. However, the results do not achieve statistical significance. Further studies with larger samples across different age ranges, accounting for misreporting and addressing the implications of a lower energy intake during a maturation period and in the long term, are needed. The long-term analysis showed no effect on the cardiometabolic profile in young adulthood, which may result from changes in dietary patterns because the differences in the energy intake between patterns were attenuated from adolescence to adulthood.

## CRediT authorship contribution statement

**Joana Pinto Costa:** Validation, Formal analysis, Writing – original draft. **Vânia Magalhães:** Methodology, Formal analysis, Writing – review & editing. **Joana Araújo:** Conceptualization, Writing – review & editing. **Elisabete Ramos:** Conceptualization, Methodology, Validation, Writing – review & editing, Supervision, Project administration, Funding acquisition.

## Sources of support

This study was based on the EPITeen cohort, funded by a project supported by national funding from the Portuguese Foundation for Science and Technology – FCT (Portuguese Ministry of Education and Science) FCOMP-01-0124-FEDER-015750 (PTDC/DTP-EPI/6506/2014) and by the funding for the Epidemiology Research Unit – Institute of Public Health, University of Porto (UIDB/04750/2020). V. M. received an individual grant (SFRH/BD/143747/2019) from the Portuguese Foundation for Science and Technology; FCT is gratefully acknowledged. The funding sources had no role in the study.

## Data availability statement

Data used in this study came from the EPITeen Cohort. The dataset analyzed during the current study is available from

the Cohort Coordination (ER, eliramos@med.up.pt) on reasonable request.

## Author Declarations

None.

## Acknowledgment

The authors have no acknowledgments to declare.

## Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.nutres.2023.01.002.

## REFERENCES

[1] Abarca-Gómez L, Abdeen ZA, Hamid ZA, Abu-Rmeileh NM, Acosta-Cazares B, Acuin C, et al. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. Lancet. 2017;390:2627–42. doi:10.1016/S0140-6736(17)32129-3.
[2] Bendor CD, Bardugo A, Pinhas-Hamiel O, Afek A, Twig G. Cardiovascular morbidity, diabetes and cancer risk among children and adolescents with severe obesity. Cardiovasc Diabetol. 2020;19:79. doi:10.1186/s12933-020-01052-1.
[3] World Health Organization. Noncommunicable diseases: childhood overweight and obesity. Accessed March 20, 2020. https://www.who.int/news-room/questions-and-answers/item/noncommunicable-diseases-childhood-overweight-and-obesity
[4] Park MH, Falconer C, Viner RM, Kinra S. The impact of childhood obesity on morbidity and mortality in adulthood: a systematic review. Obes Rev. 2012;13:985–1000. doi:10.1111/j.1467-789X.2012.01015.x.
[5] Weihrauch-Blüher S, Wiegand S. Risk factors and implications of childhood obesity. Curr Obes Rep. 2018;7:254–9. doi:10.1007/s13679-018-0320-0.
[6] World Health Organization. Global action plan for the prevention and control of noncommunicable diseases 2013-2020. Geneva: World Health Organization 2013:5.
[7] McCay CM, Crowell MF, Maynard LA. The effect of retarded growth upon the length of life span and upon the ultimate body size. J Nutr Biochem. 1935;10:63–79. doi:10.1093/jn/10.1.63.
[8] Soare A, Weiss EP, Pozzilli P. Benefits of caloric restriction for cardiometabolic health, including type 2 diabetes mellitus risk. Diabetes Metab Res Rev. 2014;30(suppl 1)):41–7. doi:10.1002/dmrr.2517.
[9] Weiss EP, Fontana L. Caloric restriction: powerful protection for the aging heart and vasculature. Am J Physiol Heart Circ Physiol. 2011;301:H1205–19. doi:10.1152/ajpheart.00685.2011.
[10] Weindruch R, Walford RL, Fligiel S, Guthrie D. The retardation of aging in mice by dietary restriction: longevity, cancer, immunity and lifetime energy intake. J Nutr Biochem. 1986;116:641–54. doi:10.1093/jn/116.4.641.
[11] Speakman JR, Mitchell SE. Caloric restriction. Mol Asp Med. 2011;32:159–221. doi:10.1016/j.mam.2011.07.001.
[12] Ding Q, Ash C, Mracek T, Merry B, Bing C. Caloric restriction increases adiponectin expression by adipose tissue and prevents the inhibitory effect of insulin on circulating adiponectin in rats. J Nutr Biochem. 2012;23:867–74. doi:10.1016/j.jnutbio.2011.04.011.
[13] Cardoso A, Silva D, Magano S, Pereira PA, Andrade JP. Old-onset caloric restriction effects on neuropeptide Y- and somatostatin-containing neurons and on cholinergic varicosities in the rat hippocampal formation. Age (Dordr). 2014;36:9737. doi:10.1007/s11357-014-9737-x.
[14] Mattison JA, Roth GS, Lane MA, Ingram DK. Dietary restriction in aging nonhuman primates. Interdiscip Top Gerontol. 2007;35:137–58. doi:10.1159/000096560.
[15] Colman RJ, Anderson RM. Nonhuman primate calorie restriction. Antioxid Redox Signal. 2011;14:229–39. doi:10.1089/ars.2010.3224.
[16] Kemnitz JW. Calorie restriction and aging in nonhuman primates. ILAR J. 2011;52:66–77. doi:10.1093/ilar.52.1.66.
[17] Kagawa Y. Impact of westernization on the nutrition of Japanese: changes in physique, cancer, longevity and centenarians. Prev Med. 1978;7:205–17. doi:10.1016/0091-7435(78)90246-3.
[18] Holloszy JO, Fontana L. Caloric restriction in humans. Exp Gerontol. 2007;42:709–12. doi:10.1016/j.exger.2007.03.009.
[19] Rochon J, Bales CW, Ravussin E, Redman LM, Holloszy JO, Racette SB, et al. Design and conduct of the CALERIE Study: comprehensive assessment of the long-term effects of reducing intake of energy. J Gerontol - Biol Sci Med Sci. 2011;66A:97–108. doi:10.1093/gerona/glq168.
[20] Kraus WE, Bhapkar M, Huffman KM, Pieper CF, Krupa Das S, Redman LM, et al. 2 years of calorie restriction and cardiometabolic risk (CALERIE): exploratory outcomes of a multicentre, phase 2, randomised controlled trial. Lancet Diabetes Endocrinol. 2019;7:673–83. doi:10.1016/S2213-8587(19)30151-2.
[21] Ramos E, Barros H. Family and school determinants of overweight in 13-year-old Portuguese adolescents. Acta Paediatr. 2007;96:281–6. doi:10.1111/j.1651-2227.2007.00107.x.
[22] Araújo J, Teixeira J, Gaio AR, Lopes C, Ramos E. Dietary patterns among 13-y-old Portuguese adolescents. Nutrition. 2015;31:148–54. doi:10.1016/j.nut.2014.06.007.
[23] Cruz F, Ramos E, Lopes C, Araujo J. Tracking of food and nutrient intake from adolescence into early adulthood. Nutrition. 2018;55-56:84–90. doi:10.1016/j.nut.2018.02.015.
[24] Magalhães A, Severo M, Autran R, Araújo J, Santos P, Pina MF, et al. Validation of a single question for the evaluation of physical activity in adolescents. Int J Sport Nutr Exerc Metab. 2017;27:361–9. doi:10.1123/ijsnem.2016-0276.
[25] World Health Organization Measuring obesity: classification and description of anthropometric data: report on a WHO consultation on the epidemiology of obesity, Warsaw, 21-23 October 1987. Copenhagen: World Health Organization, Regional Office for Europe; 1989.
[26] de Onis M, Onyango AW, Borghi E, Siyam A, Nishida C, Siekmann J. Development of a WHO growth reference for school-aged children and adolescents. Bull World Health Organ. 2007;85:660–7. doi:10.2471/blt.07.043497.
[27] Health Organization World. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: executive summary. Expert Panel on the Identification, Evaluation, and Treatment of Overweight in Adults. Am J Clin Nutr. 1998;68:899–917. doi:10.1093/ajcn/68.4.899.
[28] Willett WC, Sampson L, Stampfer MJ, Rosner B, Bain C, Witschi J, et al. Reproducibility and validity of a semi-quantitative food frequency questionnaire. Am J Epidemiol. 1985;122:51–65. doi:10.1093/oxfordjournals.aje.a114086.

[29] Lopes C. Diet and myocardial infarction: a community based case–control study [PhD Thesis]. Porto, Portugal: University of Porto; 2000.
[30] Araújo J, Severo M, Lopes C, Ramos E. Food sources of nutrients among 13-year-old Portuguese adolescents. Public Health Nutr. 2011;14:1970–8. doi:10.1017/S1368980011001224.
[31] Schofield WN. Predicting basal metabolic rate, new standards and review of previous work. Hum Nutr Clin Nutr. 1985;39:5–41 PMID:4044297 .
[32] Goldberg G, Black A, Jebb S, Cole T, Murgatroyd P, Coward W, et al. Critical evaluation of energy intake data using fundamental principles of energy physiology: 1. Derivation of cut-off limits to identify under-recording. Eur J Clin Nutr. 1991;45:569–81 PMID:1810719 .
[33] Black A. Critical evaluation of energy intake using the Goldberg cut-off for energy intake:basal metabolic rate. A practical guide to its calculation, use and limitations. Int J Obes Relat Metab Disord. 2000;24:1119–30. doi:10.1038/sj.ijo.0801376.
[34] EFSAScientific opinion on dietary reference values for energy. EFSA J. 2013;11:3005. doi:10.2903/j.efsa.2013.3005.
[35] de Ferranti SD, Gauvreau K, Ludwig DS, Neufeld EJ, Newburger JW, Rifai N. Prevalence of the metabolic syndrome in American adolescents: findings from the Third National Health and Nutrition Examination Survey. Circulation. 2004;110:2494–7. doi:10.1161/01.CIR.0000145117.40114.C7.
[36] Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985;28:412–19. doi:10.1007/BF00280883.
[37] Demaio AR, Rockström J. Human and planetary health: towards a common language. Lancet. 2015;386:e36–7. doi:10.1016/S0140-6736(15)61044-3.
[38] Forrestal SG. Energy intake misreporting among children and adolescents: a literature review. Matern Child Nutr. 2011;7:112–27. doi:10.1111/j.1740-8709.2010.00270.x.
[39] Livingstone MBE, Robson PJ, Wallace JMW. Issues in dietary intake assessment of children and adolescents. Br J Nutr. 2004;92:S213–SS22. doi:10.1079/bjn20041169.
[40] Morrison JA, Friedman LA, Wang P, Glueck CJ. Metabolic syndrome in childhood predicts adult metabolic syndrome and type 2 diabetes mellitus 25 to 30 years later. J Pediatr. 2008;152:201–6. doi:10.1016/j.jpeds.2007.09.010.
[41] Juhola J, Magnussen CG, Viikari JS, Kahonen M, Hutri-Kahonen N, Jula A, et al. Tracking of serum lipid levels, blood pressure, and body mass index from childhood to adulthood: the Cardiovascular Risk in Young Finns Study. J Pediatr. 2011;159:584–90. doi:10.1016/j.jpeds.2011.03.021.
[42] He FJ, Pombo-Rodrigues S, MacGregor GA. Salt reduction in England from 2003 to 2011: its relationship to blood pressure, stroke and ischaemic heart disease mortality. BMJ Open. 2014;4:e004549. doi:10.1136/bmjopen-2013-004549.
[43] Simental-Mendía LE, Rodríguez-Morán M, Guerrero-Romero F. The product of fasting glucose and triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. Metab Syndr Relat Disord. 2008;6:299–304. doi:10.1089/met.2008.0034.
[44] Navarro-González D, Sánchez-Íñigo L, Pastrana-Delgado J, Fernández-Montero A, Martinez JA. Triglyceride–glucose index (TyG index) in comparison with fasting plasma glucose improved diabetes prediction in patients with normal fasting glucose: the Vascular-Metabolic CUN cohort. Prev Med. 2016;86:99–105. doi:10.1016/j.ypmed.2016.01.022.
[45] Kelley DE, Goodpaster BH. Skeletal muscle triglyceride. Diabetes Care. 2001;24:933. doi:10.2337/diacare.24.5.933.
[46] Tabacchi G, Filippi AR, Amodio E, Jemni M, Bianco A, Firenze A, et al. A meta-analysis of the validity of FFQ targeted to adolescents. Public Health Nutr. 2016;19:1168–83. doi:10.1017/S1368980015002505.
[47] Araujo MC, Yokoo EM, Pereira RA. Validation and calibration of a semiquantitative food frequency questionnaire designed for adolescents. J Am Diet Assoc. 2010;110:1170–7. doi:10.1016/j.jada.2010.05.008.
[48] Tooze JA, Krebs-Smith SM, Troiano RP, Subar AF. The accuracy of the Goldberg method for classifying misreporters of energy intake on a food frequency questionnaire and 24-h recalls: comparison with doubly labeled water. Eur J Clin Nutr. 2012;66:569–76. doi:10.1038/ejcn.2011.198.
[49] Black A. The sensitivity and specificity of the Goldberg cut-off for EI:BMR for identifying diet reports of poor validity. Eur J Clin Nutr. 2000;54:395–404. doi:10.1038/sj.ejcn.1600971.