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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

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ABSTRACT

Background: Dietary misreporting is the main limitation of dietary assessments and has been associated with BMI during youth. However there are no prior studies assessing misreporting and cardiometabolic risks (CMRs) in adolescence.

Objectives: To examine the associations between dietary misreporting and CMR factors in adolescents and to assess the potential bias in the association between CMR and energy intake (EI) driven by dietary misreporting.

Methods: Two 24-hour dietary recalls were obtained from 1512 European adolescents (54.8% girls) aged 12.5–17.5 years. Physical activity was measured by accelerometry. Cut-offs suggested by Huang were applied to identify misreporters. Height, waist circumference (WC), the sum of 4 skinfold thicknesses, diastolic blood pressure (DBP), systolic blood pressure (SBP), and cardiorespiratory fitness (CRF) measurements were taken and serum triglycerides and total-/high-density lipoprotein cholesterol ratio were analyzed. A sex- and age-specific clustered CMR score ($n = 364$) was computed. Associations were investigated by multilevel regression analyses adjusting for age, sex, center, socioeconomic status, and physical activity.

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

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Conclusions: CMR factors differed among misreporting groups, and both abdominal and total fat mass indicators were more strongly associated with all forms of misreporting than was BMI. Moreover, misreporting seems to bias EI and CMR associations in adolescents. Therefore, energy misreporting should be taken into account when examining diet-CMR associations. *J Nutr* 2021;151:675–684.

Keywords: diet, cardiometabolic risk, energy misreporting, adolescence, HELENA study

Introduction

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

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

Diet has been associated with CMR already in adolescence (7). However, epidemiological dietary assessment still represents a challenge, as no gold standard for the evaluation of reported dietary intakes exists (8) and many diet studies are faced with reports of implausible energy intakes (EIs), particularly underreporting (UR) (9), the main limitation of dietary assessment methods. UR is characterized by reports of habitual EIs which are implausibly low when compared with the energy requirements estimated using objective methods (10). The opposite is true for overreporting (OV).

Identification of misreporting and its characteristics is thus crucial to the appropriate interpretation of nutritional data (8). BMI in particular has been repeatedly linked to misreporting in adults (9), but also among children and adolescents (11), as inaccurate energy reporting also occurs among young populations (12). However, little is known about the association between misreporting and CMR. It is known that high levels of CMR biomarkers in blood are associated with unhealthier diets (13, 14). Hence, although individuals may not know that they have elevated glucose, insulin, triglycerides (TG), and/or high-density lipoprotein (HDL) cholesterol, they may be conscious of the lower quality of their diets and consequently underreport their dietary intakes. Therefore, we hypothesized that underreporters (URs) would have worse CMR indicators, whereas we would expect better levels of CMR indicators among overreporters (OVs).

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

Methods

Subjects and study design

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

A total of 3528 adolescents (52.3% females) aged 12.5–17.5 years were recruited from randomly selected schools in each city. Only those

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Abbreviations used: CVD, cardiovascular diseases; CMR, cardiometabolic risk; CRF, cardiorespiratory fitness; DBP, diastolic blood pressure; DIAT, Dietary Assessment Tool; EI, energy intake; HELENA, Healthy Lifestyle in Europe by Nutrition in Adolescence; HOMA, homeostasis model assessment; OV, overreporting; OVs, overreporters; PA, physical activity; PR, prevalence ratio; SBP, systolic blood pressure; TC, total cholesterol; TEE, total energy expenditure; TG, triglycerides; UR, underreporting; URs, underreporters; VIF, variance inflation ratio; WC, waist circumference; WHeR, waist-to-height ratio; 24-HDR, 24-hour dietary recalls.

adolescents who completed two 24-hour dietary recalls (24-HDR) and had objectively measured accelerometer data were included ($n = 1512$). Blood samples were drawn after an overnight fast only in one-third of the HELENA participants, who were randomly selected. Adolescents that were excluded from the analyses were older, heavier, and had higher BMIs than those included in the present study. In addition, there were higher proportions of girls, adolescents with overweight and obesity, less-affluent adolescents, and lower maternal education levels among those excluded as compared with their peers included in the analyses (**Supplemental Table 1**).

Socioeconomic status and maternal education level

Socioeconomic status was estimated using the Family Affluence Scale, an indicator of affluence based on the concept of material conditions of the household in which an adolescent lives (19). The mothers' educational levels were reported by the adolescents as lower education, lower secondary education, higher secondary education, and higher education/university degree.

Physical activity

The physical activity (PA) assessment used in the HELENA study is described elsewhere (20). Uni-axial 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 crest with an anthropometric tape (SECA 200). Waist-to-height ratio (WHeR) was computed. Skinfold 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 blood pressure (DBP) and systolic blood pressure (SBP).

Blood sampling

Blood sampling procedures have previously been described in detail (25). Serum TG, total cholesterol (TC), HDL cholesterol, and glucose were measured with an enzymatic method on the Dimension RxL clinical chemistry system (Dade Behring; coefficient of variation <1.6%). Serum insulin levels were measured with an Immulite 2000 analyzer (DPC Biermann; sensitivity 2 μIU/mL; coefficient of variation <5.5%). The homeostasis model assessment (HOMA) index was used as a measurement of insulin resistance (26). The ratio of TC to HDL was computed.

Cardiorespiratory fitness

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

Cardiometabolic risk score

A continuous score of clustered CMR factors ($n = 380$; 50.5% girls) was computed according to Andersen et al. (28) using SBP, the sum of thickness of 4 skinfolds (bicipital, tricipital, subscapular, and suprailiac), TG, TC/HDL, HOMA index, and CRF. Sex- and age-specific z-scores were calculated for each risk factor variable. All individual z-scores were summed to create the clustered CMR score. CRF was multiplied by -1 to indicate a higher CMR with an increasing value. The lower the score, the better the overall CMR factor profile. Those adolescents at +1 SD of the score were considered at CMR.

Cardiometabolic risk factors

CMR factor cut-offs were applied to identify those adolescents at higher CMR. WC, DBP, SBP, glucose, insulin, TG, and HDL were dichotomized according to the International Diabetes Federation definition of metabolic syndrome for adolescents (29). Castro-Piñero et al. (30) cut-offs were applied for CRF and those from Piña-Aguero et al. (31) were applied for the HOMA index. The 90th percentile cut-off was used for the sum of thickness of 4 skinfolds. TC/HDL was dichotomized according to the cut-off values suggested by Chu et al. (32).

Energy intake

Two nonconsecutive 24-HDRs, within a time span of 2 weeks, were completed by the adolescents (33). Each assessment was performed by a computer-based tool for self-reported 24-HDR, the HELENA-DIAT (Dietary Assessment Tool), which has been shown to provide valid measurements of dietary intake (34). The German Food Code and Nutrition Data Base (BLS, Bundeslebensmittelschlüssel, version II.3.1, 2005) (35, 36) was used to calculate EI. Energy intake was estimated in kilocalories per day (kcal/d), taking the average of the two 24-HDRs.

Energy intake misreporting: Underreporting and overreporting

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

Statistical analysis

The distribution of all variables was checked before the analysis and several transformations were conducted to improve normality (**Supplemental Table 3**). Multilevel Poisson regression analyses with robust error variance, with study center as random intercept, were performed to investigate factors associated with misreporting, considering UR and OV as outcomes. Firstly, analyses were conducted to explore the association between each CMR indicator and the CMR score (either as continuous or dichotomized variables) and UR and OV (as dichotomized variables), adjusting for age, sex, Family Affluence Scale, maternal education, PA, and study center (random intercept). Subsequently, CMR factors with $P < 0.20$ in Model 1 were entered simultaneously in the same model with UR and OV as the outcome variables. This cut-off point is widely used for the purposeful selection of variables (41, 42).

Multilevel linear regression analyses were conducted to investigate the bias of misreporting on the association between EI and the CMR indicators and score in 6 different models: *1*) a crude model, with all participants adjusting for age and sex; *2*) all participants adjusting for relevant covariates (Model 1; age, sex, Family Affluence Scale, maternal education, and PA); *3*) all participants after adjustment for UR only and covariates included in Model 1; *4*) all participants after adjustment for OV only and covariates included in Model 1; *5*) all participants after adjustment for both UR and OV and covariates included in Model 1; and *6*) only plausible reporters, excluding URs and OVs, adjusting for covariates included in Model 1. Study center was entered in all models as a random intercept. DBP, SBP, CRF, and TC/HDL were further adjusted for the sum of 4 skinfold thicknesses. Collinearity among variables entered in the same model was assessed with the variance inflation ratio (VIF). There was no collinearity (VIF <2) among the variables included

TABLE 1. Descriptive characteristics of the study participants by reporting status ($n = 1512$)

	n	All	*n*	Underreporters	*n*	Plausible reporters	*n*	Overreporters
Age, y	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/m^2	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 4 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, mg · dL^{-1}	481	55 (49–63)	116	54 (47–60.5)	246	56 (49–64)	119	54 (49–60)
TC/HDL	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 to 1.94)	81	1.50 (−0.60 to 3.75)	198	− 0.48 (−3.14 to 2.32)	101	− 1.36 (−2.28 to 0.16)

Values are means ± SDs, medians (25th–75th percentiles), or *n* (percentage). Abbreviations: CRF, cardiorespiratory fitness; DBP, diastolic blood pressure; HOMA, homeostatic model assessment; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; WC, waist circumference; WHeR, waist-to-height ratio.

in any of the multilevel models: that is, Poisson or linear regression analyses. With the Poisson regression, we obtained the prevalence ratio (PR). The statistical software package Stata version 16.0 (Stata Corp.) was used to perform the analyses, and the threshold for statistical significance was set at $P \leq 0.05$.

Results

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

Multilevel Poisson regression analyses are shown in Table 2. UR was positively associated with WC, WHeR, the sum of 4 skinfold thicknesses, and CMR score and inversely associated with DBP and CRF. In contrast, OV was significantly associated with a lower WC, WHeR, sum of 4 skinfold thicknesses, SBP, and CMR score. Some of these associations did not remain significant when the model was further adjusted for those variables with a *P* value < 0.20 in the crude model. In the adjusted model, UR was significantly associated with a greater WC (PR, 1.05; 95% CI: 1.01–1.09), WHeR (PR, 1.09; 95% CI: 1.03–1.15), and sum of 4 skinfold thicknesses (PR, 1.02; 95% CI: 1.01–1.03). OV was significantly associated with a lower WC (PR, 0.95; 95% CI: 0.93–0.97), WHeR (PR, 0.93; 95% CI: 0.88–0.99), and sum of 4 skinfold thicknesses (PR, 0.98; 95% CI: 0.97–0.99).

The associations between dichotomized CMR indicator cut-offs and energy misreporting are displayed in **Supplemental Table 5**. UR was positively associated with a higher WHeR, sum of 4 skinfold thicknesses, SBP, and CMR score and a

TABLE 2. Multilevel Poisson regression analyses on the association between dietary misreporting (underreporting, $n = 271$; overreporting, $n = 286$) and cardiometabolic risk indicators

	Crude model[1]		Model 1[2]		Model 2[3]	
	PR (95% CI)	*P* value	PR (95% CI)	*P* value	PR (95% CI)	*P* value
Plausible reporters (ref.) vs. underreporters						
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 4 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, mg · dL^{-1}	0.99 (0.97–1.00)	0.067	0.98 (0.97–1.00)	0.057	—	
TC/HDL	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. overreporters						
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 4 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, 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	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	—	

Values are PRs and 95% CIs. The statistical significance level was set at $P < 0.05$. Abbreviations: CRF, cardiorrespiratory fitness; DBP, diastolic blood pressure; HOMA, homeostatic model assessment; PR, prevalence ratio; SBP, systolic blood presure; TC, total cholesterol; TG, triglycerides; WC, waist circumference; WHeR, waist-to-height ratio.

[1] Crude model adjusted for age, gender, and center (random intercept).

[2] Model 1 was adjusted for age, gender, Family Affluence Scale, maternal education, moderate-to-vigorous physical activity (counts per minute), and center (random intercept).

[3] Model 2 was adjusted for age, gender, Family Affluence Scale, maternal education, 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.

lower CRF. OV was inversely associated with WC, WHeR, the sum of 4 skinfold thicknesses, glucose, and CMR score. When further adjustments were carried out (Model 2), these associations only remained significant for the sum of 4 skinfold thicknesses among URs. In contrast, OV was significantly and inversely associated in Model 2 with WC, WHeR, the sum of 4 skinfold thicknesses, and SBP.

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

TABLE 3. Multilevel linear regression analyses on the association between cardio-metabolic risk indicators and energy intake ($n = 1512$)

	WC, cm	WHeR	Sum 4 skinfold thicknesses, mm	SBP, mm Hg	DBP, mm Hg	CRF, mL · kg^{-1} · min^{-1}	TC/HDL	CMR score
Crude model[1]								
n	1434	1434	1427	1432	1432	1207	457	364
β (95% CI)	0.002 (0.001–0.002)	0.57 (0.43–0.71)	−0.95 (−1.14 to −0.77)	−2.35 (−3.83 to −0.88)	−1.14(−2.27 to −0.10)	2.00 (1.20–2.79)	0.02 (0.01–0.03)	−1.76 (−2.64 to −0.88)
P value	<0.001	<0.001	<0.001	0.002	0.031	<0.001	0.002	<0.001
Model 1[2]								
n	1434	1434	1427	1414	1414	1193	454	364
β (95% CI)	0.002 (0.001–0.002)	0.55 (0.42–0.69)	−0.92 (−1.11 to −0.74)	−0.24 (−1.73 to 1.24)	−0.08 (−1.14 to 0.98)	0.55 (−1.17 to 1.26)	0.02 (0.002–0.03)	−1.62 (−2.47 to −0.78)
P value	<0.001	<0.001	<0.001	0.744	0.881	0.137	0.018	<0.001
Model 2[3]								
n	1102	1102	1094	1083	1083	893	344	271
β (95% CI)	−0.004 (−0.005 to −0.003)	−0.61 (−0.88 to −0.33)	0.88 (0.51–1.26)	4.38 (1.30–7.46)	1.89 (−0.27 to 4.05)	1.27 (−1.90 to 2.74)	0.03 (0.01–0.06)	1.09 (−0.59 to 2.76)
P value	<0.001	<0.001	<0.001	0.005	0.086	0.088	0.012	0.204
Model 3[4]								
n	1079	1079	1077	1068	1068	920	347	286
β (95% CI)	−0.006 (−0.006 to −0.004)	−0.75 (−1.05 to −0.44)	1.07 (0.66–1.48)	3.24 (−0.25 to 6.73)	−1.30 (−3.82 to 1.23)	0.67 (−1.07 to 2.40)	0.02(−0.01 to 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
Model 4[5]								
n	1434	1434	1427	1414	1414	1193	454	364
β (95% CI)	−0.004 (−0.005 to −0.003)	−0.62 (−0.85 to −0.39)	0.87 (0.55–1.18)	3.44 (0.83–6.04)	0.56 (−1.30 to 2.42)	0.92 (−0.36 to 2.20)	0.02 (0.002–0.05)	1.29 (−0.07 to 0.66)
P value	<0.001	<0.001	<0.001	0.010	0.566	0.157	0.029	0.063
Model 5[6]								
n	747	747	744	737	737	620	237	193
β (95% CI)	−0.006 (−0.007 to −0.004)	−0.79 (−1.21 to −0.37)	1.27 (0.68–1.86)	5.41 (0.55–10.3)	0.67 (−2.77 to 4.11)	1.48 (−0.85 to 3.81)	0.04 (−0.10 to 0.08)	2.9
P value	<0.001	<0.001	<0.001	0.029	0.704	0.213	0.128	0.045

Values are β coefficients and 95% CIs. The statistical significance level was set at $P < 0.05$. Abbreviations: CMR, cardiometabolic risk score; CRF, cardiorespiratory fitness; DBP, diastolic blood pressure; SBP, systolic blood pressure; TC, total cholesterol; WC, waist circumference; WHeR, waist-to-height ratio.

[1]The crude model was adjusted for sex, age, and center (random intercept).

[2]Model 1 was 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 were additionally adjusted for the sum of 4 skinfold thicknesses.

[3]Model 2 was adjusted for the variables in Model 1 plus underreporting.

[4]Model 3 was adjusted for the variables in Model 1 plus overreporting.

[5]Model 4 was adjusted for the variables in Model 1 plus dietary misreporting, including underreporters and overreporters.

[6]Model 5 was adjusted for the variables in Model 1, excluding underreporters and overreporters.

Discussion

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

Misreporting and body fat mass

In our sample of adolescents, we found a prevalence of misreporting of 48.2%, with 24.8% for URs and 23.4% for OVs. The prevalence of overweight/obesity in the whole sample of our study was 20.4%. Overweight/obesity was present in 40% of the URs (10.6% for obesity), while it was 17.5% among plausible reporters (3.6% for obese), and 5.7% for the OVs (0.84% for obesity). BMI has been largely recognized as a factor associated with misreporting and, in adults, is the strongest predictor for UR (9). Among the factors suggested to explain the tendency to underreport are providing socially desirable answers due to greater weight consciousness and dieting behavior (11, 46, 47).

Although BMI has been largely used in epidemiological studies, it accounts for body mass and not actual fat. This means that high BMIs could also be found in subjects with high fat-free masses, lean masses, and/or muscle masses and could affect the association between BMI and misreporting consistently observed in the literature. Thus, the use of specific fat mass markers could give better insight into the associations with UR behavior and, in general, misreporting. For that reason, in our study, WC and WHeR were used as markers of abdominal fat, while the sum of 4 skinfold thicknesses was computed to account for overall body fat. In our sample, URs showed higher mean values for fat mass markers, sums of 4 skinfold thicknesses, WC, and WHeR, in comparison with plausible reporters and OVs. In contrast, OVs had the lowest mean values for the fat mass markers. Underreporting was significantly associated with a greater WC, WHeR, and sum of 4 skinfold thicknesses, whereas OV was significantly associated with a lower WC, WheR, and sum of 4 skinfold thicknesses in adjusted models. In line with our results, previous literature has shown that adolescents with higher body fat underestimate 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 UR their dietary intake.

A previous study based on HELENA data showed that adolescents with greater fat masses had lower EIs compared with adolescents with lower fat masses, 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 masses in our study may truly have lower EIs and, therefore, could be reliable reporters of their EIs. 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 EIs should be expected (54). For that reason, adolescents with higher fat masses in our sample seem more likely to be UR than to have low EIs. Nevertheless, it should be borne in mind that UR is likely to also include real undereating, as some adolescents may be intentionally eating lower amounts of food in an attempt to lose or not gain weight. Overall, this highlights the importance of considering misreporting when analyzing 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 was identified between the percentage of body fat and differences in EI reported by an FFQ, which was explained by misreporting (56). In this study, and in line with our findings, the probability of EI underestimation among boys was lower in those with body fat ≤10%, while it was much higher for those with a greater percentage of body fat, mainly among subjects with body fat ≥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 a higher percentage of body fat (49, 56). These results show that fat mass, like BMI, is strongly correlated with dietary misreporting. People with high BMIs and, probably, higher fat masses, may be more prone to UR for the same reasons as those previously stated. Thus, an attenuation of the association between dietary intake and risk of obesity may occur in studies in which high misreporting is present or an 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 EIs 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 ∼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 EIs can reflect real overeating rather than OV, as adolescents may consume larger amounts of food due to growth spurts.

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 than plausible reporters. Specifically, blood pressure and LDL cholesterol concentrations were higher and HDL levels were lower in URs than in plausible reporters (59). These results are similar to those from another study that reported on biochemical markers, including LDL, HDL, and TG, of URs in a small sample of South American adolescents (60). In a different study carried out among adults, associations between some biomarkers, such as HDL, LDL, or SBP, and specific food items were affected by misreporting, even changing the direction of the associations when the misreporting was taken into account

(61). In contrast, OVs showed better cardio-metabolic health when compared with URs in the present study. However, when assessing the association with metabolic profiles in the full, adjusted model, we found no associations.

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

Misreporting and the association of CMR and energy intake

Results from the present study suggest that misreporting affects the association between CMR and EI, either using CMR factors individually or using a CMR score. We found different associations when considering UR and OV individually in different models as covariates and when applying the combination of both in comparison with the model where misreporting was not considered. Moreover, when excluding the misreporters, differences in the associations between CMR and EI were also found when compared with the other approaches. These results could be explained by the prevalence of overweight/obesity being higher among URs (40%) as compared with OVs (5.7%). Alternatively, the decrease in the sample size that resulted when URs and OVs were excluded from the analyses could be another explanation for the observed results. This result highlights the major role of misreporting when assessing the association between CMR and EI, as well as highlighting 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 seems 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 to use self-reported EIs due to the limitations inherent in self-reported methods. For that reason, we did not make any attempt to report 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 not considering these results as true associations. In fact, given the impact that misreporting had on self-reported EIs in this study, these findings can be considered as an extension of Subar et al.'s (63) statement on the uselessness of EI as an 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 way to correct for misreporting, the exclusion of implausible reporters may introduce some additional concerns, such as selection bias (64), decreases in the sample size and the power to detect associations, and the exclusion of those who are truly under- or overeating (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 status, being worried about gaining weight, body image dissatisfaction, and skipping breakfast—that may need to be taken into account to improve interpretations of potentially biased findings (37). However, agreement has yet to be reached on the best correction method. Nevertheless, misreporting is a complex problem, beyond BMI differences among misreporting groups, that needs to be accounted for when assessing EI and which remains among the main limitations of dietary assessment methods.

Limitations and strengths

The present study has some limitations. Firstly, diet was assessed by self-reported 24-HDRs, which are subject to a certain degree of systematic measurement error due to the characteristics of the method and the population under study (63). The method relies on the participants' memory and on their ability to estimate quantities, and can lead to inaccurate estimates of EI (63). Hence, inferences on the specific associations between EI and CMR should be avoided. Also, only 2 nonconsecutive days were included, which may not be sufficient to characterize individuals' usual intakes and variability. However, the HELENA-DIAT provides reliable estimates of dietary intake among European adolescents (36, 34).

Another limitation that needs to be accounted for is that the applied cut-offs assumed stable body weight; that is, that the participants were not losing or gaining weight. However, we assume that this condition does not significantly affect the study outcomes, as energy costs in adolescence are small, at approximately 1% of TEE (66). Given the short period of dietary recording, distinctions could not be made between respondents who were on a diet (undereating) or those limiting their reported intake (UR), nor among those who were intentionally eating more (overeating) or those reporting a higher EI (OV). The cross-sectional nature of this study cannot be used to establish causality. Another study limitation is that analyses were not corrected for multiple testing; therefore, some of the observed significant associations may not be true associations, but due to chance. However, our study has several strengths. First is the use of European data with standardized procedures throughout the different study centers 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 errors offers a simpler and more individualized 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 masses within the UR group when compared with plausible reporters and OVs. 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, such as the 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 associations 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.

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