Associations of dietary energy density with body composition and cardiometabolic risk in children with overweight and obesity: role of energy density calculations, under-reporting energy intake and physical activity

Running title: Dietary energy density and body composition in children with overweight/obesity

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Abstract

This study examined 1) the association of dietary energy density from solid (ED_S) and solid plus liquids (ED_{SL}) with adiposity and cardiometabolic risk factors (CRF) in children with overweight and obesity, 2) the effect of under-reporting on the mentioned associations, and 3) whether the association between ED, and body composition and CRF is influenced by levels of physical activity. In a cross-sectional design, 208 children with overweight and obesity (8 to 12-year-old;111 boys) completed two non-consecutive 24-hour recalls. ED was calculated using two different approaches: ED_S and ED_{SL}. Under-reporters were determined with the Goldberg method. Body composition, anthropometry and fasting blood sample measurements were performed. Moderate-to-vigorous physical activity (MVPA) was registered with accelerometers (7-day-register). Linear regressions were performed to evaluate the association of ED with the previously mentioned variables. Neither ED_S nor ED_{SL} were associated with body composition nor CRF. However, when under-reporters were excluded, ED_S was positively associated with BMI (p=.019), body fat percentage (p=.005), abdominal fat (p=.008) and fat mass index (p=.018), while ED_{SL} was positively associated with body fat percentage (p=.008) and fat mass index (p=.026). When stratifying the group according to physical activity recommendations, the aforementioned associations were only maintained for non-compliers. Cluster analysis showed that the low-ED and high-MVPA group presented the healthiest profile for all adiposity and CRF. These findings could partly explain inconsistencies in literature, as we found that different ED calculations entail distinct results. Physical activity levels and excluding under-reporters greatly influence the associations between ED and adiposity in children with overweight and obesity.

Keywords: Nutrition, diet, dual energy x-ray, adiposity, childhood, moderate to vigorous physical activity

Background

Childhood overweight and obesity pose a major public health concern worldwide, as they have shown to be responsible for increases in cardiovascular disease risk factors[1], psychological problems[2] and orthopaedic problems[3], among many other complications. Moreover, an extensive body of research indicates that children with obesity are at a higher risk of becoming adults with obesity than their counterparts without obesity[4].

Although the etiology of obesity is multifactorial and complex, it is clear that physical activity and dietary habits play important roles in the development of this disease as they can induce an energy imbalance and promote excessive fat deposition. Focusing on nutrition, a range of specific nutrients and foods have been suggested as important dietary determinants of obesity in childhood and adolescence, including fruit and vegetables[5], fibre[6] and sugar-sweetened beverages[7]. Although the individual effect of the previously mentioned dietary components is important, it might not reflect the overall effect of diet. For this purpose, dietary energy density (ED), has been suggested as an appropriate measure of overall diet, specifically when evaluating the association between diet and obesity/adiposity[8].

A recent systematic review aiming to evaluate the association between ED and obesity[8] including 14 studies with children and adolescents found inconsistent results in this population with some studies showing an association between ED and body composition[9–11] and others reporting no significant associations between both variables[12–14]. Surprisingly, few studies[15] including children assessed the association between ED and cardiometabolic risk factors (i.e. blood pressure and triglyceride levels), which have shown to be increased in children with obesity[16]. Although most studies presented body composition as an outcome variable, some studies used body mass index (BMI), while others used body fat or anthropometric variables (skinfold thickness or waist circumference) as main outcomes, which could partly explain the mentioned inconsistent results among studies.

Another factor that could explain these inconsistencies is the definition of ED, as some researchers use solids to compute it (ED_S), while others use a combination of solids and liquids (ED_{SL}). McCaffrey et al.[17] used 5 different classifications of ED and found that the definitions that did not include beverages showed the best association with changes in fat mass. Besides the definition of ED, it is obvious that other external factors could regulate the association between ED and body composition, such as physical activity, that was not registered in several previous studies[9,17,18], or was registered through questionnaires[19–21], which have shown to have a limited validity and reliability in previous studies.[22]

Two studies evaluated the association between ED and body composition in children with obesity. The first developed by Butte et al.[12] found that ED did not predict weight gain during one year follow-up, while a recent study developed by Aburto et al.[23] found a positive association between ED and overweight and obesity. Interestingly, the association was stronger when only plausible reporters (i.e., excluding from the analyses both under- and over-reporters) were considered. Taking into account that previous studies have found energy intake under-reporting to be more prevalent and severe among children and adolescents with a higher BMI[24,25], identifying under-reporters when measuring the associations between ED and body composition in children with overweight and obesity is of great importance.

It therefore seems timely to evaluate the association between ED and different body composition variables (i.e., BMI, adiposity) and cardiometabolic markers, taking into account levels of objectively measured physical activity, to ascertain if ED is critical to body composition and if physical activity modulates this association in children with overweight and obesity, a population in which energy intake under-reporting has been shown to be more prevalent and severe[24,25]. Consequently, the aims of the present study were, 1) to evaluate the association of ED_S and ED_{SL} with adiposity and cardiometabolic risk in children with overweight and obesity, 2) to estimate the effect of under-reporting on the mentioned associations, and 3) to assess if the association between ED and body composition/cardiometabolic risk is influenced by levels of physical activity.

Material and methods

Participants

Participants were recruited from two different cities of Spain (Granada and Vitoria-Gasteiz), as part of two projects that are briefly explained below:

a. The ActiveBrains project (NCT02295072) that is described in detail elsewhere[26], was developed in the city of Granada (south Spain) and aimed to examine the effects of a physical exercise program on brain, cognition, academic achievement as well as physical and mental health in 110 participants aged 8 to 11 that presented overweight or obesity. Although the

ActiveBrains is a randomized controlled trial, data from the first cross-sectional evaluation were used for the present study (data collected from November 2014 to February 2016 in three different waves). The study protocol was approved by the Review Committee for Research Involving Human Subjects at the University of Granada (Ref: 848).

b. The EFIGRO project (NCT02258126), developed in Vitoria-Gasteiz (north Spain), aimed to measure 160 children with overweight or obesity aged 8 to 12 and determine the effects of a multidisciplinary intervention on hepatic fat fraction and cardiometabolic risk factors. Data for the first cross-sectional evaluation were used for the present study (data collected from October 2014 to January 2017 in five different waves). The study protocol was approved by the Ethic Committee of Clinical Investigation of Euskadi (PI2014045). More details of the EFIGRO project can be found in the methodological manuscript[27].

Following the inclusion criteria of the previously mentioned projects, the main inclusion criteria for the present study were: 1) children between 8 and 12 years-old, 2) classified as presenting overweight or obesity based on the sex- and age-specific international BMI standards[28], and 3) that were not taking medications that influenced the central nervous system function.

From all the measured participants of the ActiveBrains and the EFIGRO projects, 208 (101 from ActiveBrains and 107 from the EFIGRO project) presented complete data for body composition and accelerometry and were, therefore, included in the study. From the 208 included participants, six did not report their pubertal status, seven did not have blood samples, and 18 did not have data for DXA abdominal regions (not collected due to technical reasons). These participants were included in the analyses and consequently number of participants for each analysis varied slightly and is specified in the results section.

Body composition

Body composition assessments were performed in the morning in a non-fasted state.

Anthropometric variables: Height and weight were measured in children wearing minimal clothing and no shoes using a wall stadiometer and electronic scale respectively (SECA model, Hamburg, Germany). BMI was calculated as weight in kilograms divided by the squared height in meters, and participants were classified into BMI categories according to the World Obesity Federation cut-offs [28]. Waist circumference was measured at the narrowest point by standard procedures with an anthropometric non-elastic tape (SECA 200, Hamburg, Germany).

Body fat: Dual energy X-ray absorptiometry (DXA, Hologic QDR 4500W) was used to evaluate body composition. A whole-body scan was performed from which, whole body fat percentage (BF%) and fat mass index (FMI), expressed as body fat divided by squared height expressed in meters were obtained. Additionally a regional analysis was performed to assess abdominal adiposity following the protocol of previous studies[29]. One region was calculated as a rectangle drawn on the digital scan image with the lower border of the rectangle at the lower horizontal border on the top of iliac crest and the upper border established parallel with the end of the lowest rib (this regions was defined as R1 in previous studies[29]). The lateral side of the defined region was adjusted to include all the body tissue. As this region might be larger (and therefore include more fat) in taller participants, it was adjusted by height and was called abdominal FMI.

Dietary intake assessment

Total energy intake (EI): following the European Food Safety Authority guidelines[30] total EI was obtained from two non-consecutive 24-hour recalls referring to weekdays which were collected by trained nutritionists, conducted in presence of the child´s parents or legal guardians. A photographic manual of food portion size was used to improve the estimated amount of dietary intake. All the data was registered by the Easydiet software (Biocentury©, S.L.U. 2016), which is the software supported by the Spanish Association of Dietetics and Nutritionists.

Dietary energy density (ED): was calculated following two different approaches.

a. Only solids ED (ED_S): Total EI from solid foods relative to total grams of solid foods consumed, expressed as kcal/g. This calculation was done based on solid foods only, excluding all energy-containing and non-energy-containing beverages (tea, coffee, water, soft drinks, fruit juice and milk).

b. Solid and liquids ED (ED_{SL}): The ED of food and energy-containing beverages.

Miss-reporters: The Goldberg[31] and Black´s[32] equations (Table 1), were used to estimate possible under- and over-reporters (children who reported non-plausible energy intakes). This method is used to predict total energy expenditure using physical activity levels (PAL) and basal metabolic rate which was estimated with three different equations[33,34]: the Schofield equation[34] , and two different equations proposed by Lazzer[33] both taking Tanner stage into account with one using body weight ($Lazzer_{weight}$) and the other using fat mass and fat free mass from DXA ($Lazzer_{DXA}$). The three equations are defined in Table 1. Previous validation studies have demonstrated that the Goldberg equation presents a high predictive value for detecting under-reporters when compared to doubly labeled water[35]. Specific details of the formula and used coefficients of variation are presented in Table 1.

Cardiometabolic variables

Blood samples were collected after an overnight fast. Glucose was analysed using the glucose oxidase method with automatic analysers (Roche-Hitachi Modular P and D Autoanalyser, Roche Laboratory Systems, Mannheim, Germany), and plasma insulin was analysed by radioimmunoassay using automatic microparticle analysers (Axsym, Abbott Laboratories, Chicago, Ill., USA). Triglycerides, total cholesterol LDL and HDL were measured using an automatic analyser (Roche-Hitachi Modular P and D Autoanalyser, Roche Laboratory Systems). Blood pressure was assessed with the automatic OMRON®M6 device. Mean arterial blood pressure (MAP), and two different cardiometabolic risk scores (CM Risk) (following the indications of Nystrom et al.[36]), were calculated as described in Table 1.

Physical activity

Physical activity was assessed with tri-axial accelerometers (GT3x+ and wGT3X-BT, Pensacola, FL, USA.) attached to the non-dominant wrist during 7 consecutive days (24 hours). ActiGraph.csv files were analyzed with R-package GGIR version 1.2 (http://cran.r-project.org)[37]. Data of participants were included in the analyses if they recorded at least 1 weekend day and 3 week valid days (≥ 16 wearing hours) as previously recommended[38]. Identification of waking and sleeping hours was done using an automatized algorithm guided by the times reported by the participants as explained by Van Hees et al.[39]. The Hildebrand et al.[40] cut-off points were used to classify moderate to vigorous physical activity (MVPA).

Sociodemographic status

Parental educational level was registered for its known influence of nutritional status[41]. Mothers educational highest degree was registered, and mothers were classified as low education (compulsory secondary school), medium education (high school), and higher education (University). Pubertal development was registered through direct examination by trained pediatricians following the Tanner scale[42].

Statistical analysis

Normality of the variables was checked with histograms. For non-normal variables data were transformed to obtain normalized variables with mean 0 and standard deviation 1. Mean and standard deviations are presented for the whole sample and stratified by sex.

Power calculation and sample size estimations were computed based on the primary outcome in each of the studies which are reported in the corresponding methodological articles published elsewhere[26,27]. The present study is based on a secondary analysis using baseline data from both studies, and therefore a specific power calculation was not developed for the present study.

Linear regression analyses were performed to explore the influence of ED on body composition and CM Risk after adjustment for total EI, age, study center, sex and mothers' educational level. Further models were explored taking MVPA into account. MVPA by ED interactions were tested in order to determine if MVPA influenced the association between ED and the outcome variables. All the previously mentioned regressions were also performed stratifying participants according to compliance or not with physical activity recommendations (60 daily minutes or more of MVPA[43]).

A sensitivity analysis was conducted excluding under-reporters who were detected by the Goldberg method[31] using three different equations[33,34] as shown in Table 1. Dietary differences between under- and plausible-reporters were evaluated with independent *t*-tests (using under-reporters estimated with the Schofield equation[34] as it was the most used equation in the literature).

Cluster analysis was performed with ED_S and minutes of MVPA. To be consistent with clustering methods reported in previous studies[44,45], two types of cluster analyses were used: hierarchical clustering (Ward´s method) and k-means clustering. To reduce the sensitivity of the Ward´s method to outliers, individual outliers and multivariate outliers (those with high Mahalanobis values distance) for any variable were investigated. Hierarchical cluster analysis was initially used, as the numbers of clusters in the data were unknown beforehand. Number of clusters was determined by examining dendrograms, that suggested a solution of 3 cluster groups. K-means cluster analysis was therefore performed with 3 possible solutions. This approach minimizes the within-cluster variance and maximizes the between-cluster distance so that resulting clusters are as homogeneous as possible. K-means cluster analysis is considered superior to hierarchical methods because it is less sensitive to outliers and has been found to result in greater within-cluster homogeneity and between-cluster heterogeneity.[46]

One-way analysis of variance (ANOVA) with ED and MVPA variables were performed to classify and name the 3 cluster groups that emerged, that were described as HEALTHY (low ED and high MVPA), INACTIVE-LOW-ED (low ED and low MVPA), and AVERAGE-MVPA-HIGH-ED (high ED and medium MVPA). Finally, age, sex, study center, total energy intake and mothers' education adjusted analyses of covariance (ANCOVA) were performed to evaluate adiposity and cardiometabolic risk differences among cluster groups.

Results

Descriptive characteristics

Participant characteristics, dietary variables, body composition values, cardiometabolic risk markers and physical activity levels are detailed in Table 2. Briefly, the sample was 10.4 years old, with a mean BMI of 26.1kg/m^2, waist circumference of 84 cm and 41 BF%. Regarding dietary habits, participants reported an EI of 1741 daily kilocalories with an average ED_S of 1.81 kcal/g and ED_{SL} of 2.28 kcal/g. Basal metabolic rates varied slightly depending on the used formula ranging from 1433 kilocalories with the $Lazzer_{DXA}$ to 1483 kilocalories with the Schofield equation.

Data for the comparison between under-reporters and plausible-reporters is presented in Supplementary Table 1. Under-reporters presented lower values of total energy intake (1348 vs. 1876 kcal; p<.001), DE_S (1.60 vs. 1.87 kcal/g; p<.001) and DE_{SL} (2.06 vs. 2.36 kcal/g; p<.001) when compared to plausible-reporters. Under-reporters, presented higher values than plausible-reporters for all the body composition variables (Supplementary Table 1).

Associations between dietary energetic density (ED), body composition and cardiovascular risk

Neither ED_S nor ED_{SL} demonstrated significant associations with any of the body composition or cardiovascular risk variables when the whole sample was analyzed (Tables 3 and 4).

When under-reporters were excluded (Table 3), ED_S was significantly associated with BF% ($Lazzer_{DXA}$ p=.013, $Lazzer_{weight}$ p=.008 Schofield p=.005), FMI ($Lazzer_{DXA}$ p=.025, $Lazzer_{weight}$ p=.014 Schofield p=.008) and abdominal FMI ($Lazzer_{DXA}$ p=.040, $Lazzer_{weight}$ p=.033 Schofield p=.018). These results remained significant after adjusting by MVPA (Table 3, model 2). EDs was also significantly associated with BMI when the Schofield (p=.019) and $Lazzer_{weight}$ (p=.036) equations were used to detect under-reporters, and remained significant after adjusting by MVPA (Table 3, model 2). A tendency was also found for the $Lazzer_{DXA}$ equation when predicting BMI (p=.053 and p=.067 without and with adjustment of MVPA respectively).

Regarding the associations between ED_{SL} and body composition when under-reporters were excluded (Table 4), ED_{SL} was significantly associated with BF% $Lazzer_{DXA}$ p=.031, $Lazzer_{weight}$ p=.017 Schofield p=.008) even after adjusting by MVPA (Table 4, model 2). ED_{SL} was also associated with FMI when under-reporters were detected with the Schofield (p=.026) and the $Lazzer_{weight}$ (p=.040) equations, but only results for the Schofield equation remained significant after adjusting by MVPA (p=.042 for the Schofield equation and p=.055 for the $Lazzer_{weight}$ equation, Table 3, model 2).

No significant interactions were found between MVPA and ED in the performed linear regression models (p>.10) for the whole sample. Nonetheless when under-reporters were excluded the interaction terms for BF% (Schofield p=.049), became significant. When the whole sample was stratified according to compliance with physical activity recommendations, no association was found between ED and any of the outcome variables for neither compliers (n=73) or non-compliers (n=135). When under-reporters were excluded, different results were found for compliers (n=63, 62 and 62 for the $Lazzer_{DXA}$, $Lazzer_{weight}$ and Schofield equations respectively) and non-compliers (n=99, 95 and 95 for the $Lazzer_{DXA}$, $Lazzer_{weight}$ and Schofield equations respectively). For the complier group, no significant associations were found between ED and any of the outcome variables, while for the non-complier group positive associations were found between ED, BF% ($Lazzer_{DXA}$ p=.016, $Lazzer_{weight}$ p=.011 Schofield p=.004) and FMI ($Lazzer_{DXA}$ p=.080, $Lazzer_{weight}$ p=.060 and Schofield p=.024). Additional adjustment by MVPA did not modify results of the stratified aforementioned regressions.

Combined effect of ED and MVPA: Cluster analysis

Finally, when analyzing the cluster analysis results (Figure 1), we observed that the HEALTHY group (low ED and high MVPA levels) presented lower values of BMI (both p<.001), waist circumference (both p<.001), FMI (p<.001 vs. INACTIVE-LOW-ED and p=.006 vs. AVERAGE-MVPA-HIGH-ED) and abdominal FMI (p=.003 vs. INACTIVE-LOW-ED and p=.015 vs. AVERAGE-MVPA-HIGH-ED) when compared to the other two groups. The healthy group also presented lower values than the INACTIVE-LOW-ED group for both estimated cardiovascular risk indexes (cardiorisk-1 p=.005; cardiorisk-2 p=.002) and BF% (p=.003). No differences were found between INACTIVE-LOW-ED and AVERAGE-MVPA-HIGH-ED for any of the measured variables (all p>.05; Figure 1).

Discussion

The main findings of the present study are: 1) dietary ED is associated with higher total and abdominal adiposity in plausible reporters of dietary energy intake, with ED_S showing stronger associations than ED_{SL}, 2) the aforementioned associations persist even after adjusting by MVPA. Nonetheless, when stratifying plausible reporters according to complying or not with physical activity recommendations, the inverse associations between ED and adiposity disappear for the complier group and are maintained for the non-complier group. Those children with overweight/obesity with a combination of high MVPA levels and low dietary ED present a healthier body composition and cardiometabolic profile, and 3) the effect of excluding under-reporters from the analysis is critical in the study of children with overweight and obesity. Although previous literature is inconsistent, the influence of dietary ED on body composition when under-reporters were excluded in our sample was clear, and we believe that inconsistencies found among previous studies[8] could partly be explained by some of the findings discussed below.

On the one hand, it becomes clear that ED_S and ED_{SL}, do not show the same association with adiposity in children with overweight and obesity, as only ED_S significantly predicted BMI and abdominal FMI. This is in line with previous studies performed with participants without obesity that also found that ED calculated with only solids demonstrated better associations with body composition in children[17,47] and adults[48] than ED calculated with solids and liquids. We thought it was important to evaluate if differences between ED calculations also emerged when evaluating children with overweight and obesity, as their intake of sugar-sweetened drinks might be higher, which, in turn, could modify associations between ED_{SL} and body composition. Differences between the association of ED_S and ED_{SL} with body composition found in the present and in previous studies could be due to the different effects that drinks and solid foods have on satiety[49] and to the differences in ED between solids and liquids, as beverages tend to have a high-water content and low ED. Consequently, the inclusion of beverages in ED calculations will lower the ED of the entire meal and bias the association between ED and body composition towards the null as seen in our and previous studies[17,48].

On the other hand, we found that the association between ED in plausible reporters and adiposity was only significant for some variables; likewise, ED_S was associated with BF%, FMI and abdominal FMI, all measured with DXA, but did not show an association with less accurate proxies of body fat such as waist circumference or BMI (when excluding under-reporters with the $Lazzer_{DXA}$ equation). Regarding the associations of ED and these proxies, literature is inconsistent, as some studies show positive associations[21,50] while others do not[14,51]. Nonetheless, these are just proxies, with the measurement of body fat being more important. To the best of our knowledge, the only previous study that evaluated ED and body composition of children with obesity with DXA was the Viva la familia study[12]. This study used total body mass gain (during one year follow-up) as an outcome variable, finding that ED did not significantly predict weight gain. DXA variables (fat mass and fat free mass) were not used as outcome variables in the aforementioned study and, therefore, comparisons with our findings in children with overweight/obesity are not possible. Regarding children without obesity, three previous studies evaluated the influence of ED on body fat measured with DXA, with two of them reporting positive associations between both variables[11,52], and one developed by Kral et al.[18] not finding significant associations. Kral et al. suggested that sample size (n=49) might have been too small to detect significant associations between the two variables. Moreover, although 3-day weighed food records were used in the previous study, there was no control for under-reporters that have shown to affect results in our and previous studies[15,23,53].

The effect of excluding the under-reporters from the analysis was large in the present sample. They presented higher adiposity values, 27% lower energy intake, 18% lower levels of MVPA and more importantly a significantly lower energy density when compared to plausible reporters, as published previously[53]. This lower ED found in under-reporters could bias the association between ED and body composition variables towards the null if not accounted for in the analysis, explaining why no associations were found between ED and body composition variables when the whole sample was analyzed. The findings of MVPA not modifying the associations between ED and body composition (only FMI for one equation passed from p .040 to p.055) are of great importance, as it demonstrates that ED is crucial for children with overweight and obesity even when taking into account their levels of physical activity.

Nonetheless, this does not mean that MVPA is not determinant to adiposity in this population, as when the sample was stratified according to compliance with physical activity recommendations, no significant associations were found between ED and body composition or cardiovascular risk variables in the complier group. Moreover, from the cluster analysis it became clear that those who performed the highest levels of MVPA and showed low levels of ED presented the healthier profile for all the measured variables.

In agreement with a previous study[15], ED was not directly associated with cardiometabolic risk in children. This lack of associations could be partly explained by the characteristics of the measured sample, a homogeneous group of children with overweight and obesity, who showed high cardiometabolic marker values when compared to previous studies[36] using the same methodology in normal-weight children (Systolic blood pressure 106 vs. 99, diastolic blood pressure 66 vs. 61, triglycerides 89 vs. 61, HDL 50 vs. 62, LDL 103 vs. 95 in our study vs. Nystrom et al.[36] study respectively). It is possible that if a more heterogeneous sample of both children with normal weight and overweight had been included, significant associations between ED and cardiometabolic risk would have been found, as seen in previous studies that included both children with normal weight and overweight[54]. Although the associations between ED and cardiometabolic risk were non-significant, in the cluster analysis, the HEALTHY group presented significantly lower cardiometabolic risk than the INACTIVE-LOW-ED, and no differences with the AVERAGE-MVPA-HIGH-ED. These findings suggest that MVPA levels in children with obesity might be determinant for cardiometabolic risk, which is in line with previous studies that have underlined the importance of physical activity on cardiometabolic risk factors in children[55].

Although the current study presents several strengths such as the measurement of objective physical activity, body fat with DXA and collection of cardiometabolic risk markers, it also presents several limitations. Firstly, 24 hour-recalls were only collected for weekdays and the photographic manual used to help participants select portion sizes is still in the process of being validated. It is therefore important to notice that results could be biased by the previously mentioned tool and by the use of only weekdays, as previous research has demonstrated important differences between week- and weekend-days[56]. Secondly, the cross-sectional nature of the study does not allow for a cause effect conclusion, and although we found associations, these do not imply that a higher ED intake will necessarily mean an increase in the measured body composition variables. Moreover, body composition assessments were performed during the morning in a non-fasted state, which could affect the quantification of lean and fat mass[57]. Thirdly, non-energy-containing beverages, such as water, tea or diet-soda were not included in the ED calculations and therefore the findings of the present study could have been different if the whole diet of each participant had been taken into account. Finally, although the present study included a homogeneous sample of 8 to 12-year-old children with overweight-obesity the sample size was smaller than in previous similar studies[14,21] that included heterogeneous samples. This sample size could entail a low power and a type 2 error, and therefore it is possible that if a larger sample size had been analyzed further associations would have been found between ED and cardiometabolic markers.

Summarizing, findings from the present study suggest that that dietary ED is associated with total and abdominal adiposity in children with overweight and obesity when under-reporters are excluded from the analysis. The large inconsistencies among previous studies could be due to the use of different ED calculations, the use of different methodologies to estimate body fat mass and under-reporting concerns (as for the present study most results were modified when excluding under-reporters). Those overweight and obese participants who performed high levels of MVPA and presented a low ED intake demonstrated the healthier profile for both body composition and cardiometabolic risk variables. In conclusion, findings from the present study suggest that children with overweight or obesity with a low ED intake and high levels of MVPA present healthier body composition and cardiometabolic profiles when compared to children with overweight or obesity who perform lower amounts of MVPA and have a higher ED intake. Consequently, nutritional programs aiming to prevent or treat childhood obesity should try to avoid high ED_S foods, and promote physical activity. These findings might have important public health implications, yet they need to be confirmed by randomized controlled trials that target both diet density and MVPA.

Conflict of interest: On behalf of all authors, Alejandro Gómez-Bruton states that there is no conflict of interest

Author Contributions.

Collected the data: VMH, JMG, EMR, MO, JMH, LA, MM, WDMA, JM, IT, IL, FBO

Performed the statistical analyses and drafted the manuscript: AGB

Reviewed the manuscript: LA, MM, JMG, CCS, JMH, VMH, EMR, WDMA, JM, MO, IT, LGM, GVR, FBO, IL

Conceived and planned the experiments: FBO, IL,

Coordinated the project: FBO, IL, LA and GVR

Approved the final version: AGB, LA, MM, JMG, CCS, JMH, VMH, EMR, WDMA, JM, MO, IT, LGM, GVR, FBO, IL

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Figure 1. Cluster analysis results. Body composition comparisons adjusted by mothers' education, study center, sex, age, and total energy intake.

$= Differences between the Average MVPA high ED and the other two groups.
&= Differences among the 3 groups (p<.05).
**=Differences between the healthy group (low ED and high MVPA) and the other two groups.*
#=Differences between the healthy group (low ED and high MVPA) and the inactive and low ED group.

ABD_FMI=Abdominal fat mass index; BMI=Body mass index; CM Risk 2=Cardiometabolic risk score created by Martinez Vizcaino et al.[58]; CM Risk1 =Cardiometabolic risk score described by Alberti et al.[59]; FAT%=Body fat percentage; FMI=Fat mass index; MVPA= Moderate and vigorous physical activity

Table 1. Calculations of cardiometabolic risk, resting metabolic rate, and number of under-reporters

Cardiometabolic risk	
CM Risk 1	(N_waist + N_mean blood pressure + N_triglicerides + N_glucose + N_inverted HDL) / 5
CM Risk 2	(N_TG/HDL + N_waist + N_insulin + N_MAP) / 4
MAP	Diastolic blood pressure + (0.333 x [systolic blood pressure - diastolic blood pressure])
Resting metabolic rate (kcal)	
Lazzer 1	((FFM (kg) × 0.082) + (FM (kg) × 0.037) − (Tanner stage × 0.125) + (sex × 0.706) + 2.528) * 239.006
Lazzer 2	((BW (kg) × 0.044) + (Height (m) × 2.836) − (Tanner stage × 0.148) + (sex × 0.781) − 0.551) * 239.006
Schofield boys <10y	19.6 x BW (kg) + 1.033 x Height (m) + 414.9
Schofield girls <10y	16.97 x BW (kg) + 1.618 x Height (m) + 371.2
Schofield boys >10y	16.25 x BW (kg) + 1.372 x Height (m) + 515.5
Schofield girls >10y	8.365 x BW (kg) + 465 x Height (m) + 200

Goldberg equation EIrep / BMR > $PAL * \exp\left[\pm 2 * \frac{(S/100)}{\sqrt{n}}\right]$ where $S=\sqrt{\frac{CVei^2}{d} + CVwb^2 + CVtp^2}$

Imputed values	CVei	d	CVwb	CVtp	PAL
	23	2	8.5	15	1.55
Cut-off points					
	Under-reporters	0.96	*Over-reporters*	2.49	
Total number of under-reporters					
Under-reporters Lazzer1			40 out of 202 (19%)		
Under-reporters Lazzer2			45 out of 202 (22%)		
Under-reporters Schofield			48 out of 208 (23%)		

BMR=Basal metabolic rate; BW=Body weight; CM Risk 1=Cardiometabolic risk score described by Alberti et al.[59] CM Risk 2=Cardiometabolic risk score created by Martinez Vizcaino et al.[58]; CVei=Within-subject coefficient of variation in energy intake; CVwb=Coefficient of variation of repeated BMR measurements or the precision of estimated compared with measured BMR; CVtp=Total variation in PAL; d=number of days of diet assessment; EIrep=Reported energy intake; FFM=Fat free mass; FM=Fat mass; HDL=High density lipoprotein; Kg=kilograms; Lazzer 1=Lazzer equation taking into account body composition; Lazzer 2=Lazzer equation taking into account weight; MAP=Mean arterial blood pressure; N_=Normalized value with Bloom technique (similar to z-scores but all values go from -1 to 1). N_inverted HDL=values of normalized HDL multiplied by -1; PAL=Physical activity levels; TG/HDL=Triglycerides to HDL ratio.

Table 2. Characteristics of the whole sample and stratified according to sex and study

Descriptive characteristics	Whole sample (n=208)	Boys (n=111)	Girls (n=97)	EFIGRO (n=107)	Activebrains (n=101)
Age (y)	10.4±1.2	10.5±1.2	10.2±1.1	10.6±1.1	10.0±1.1
Weight (kg)	55.5±10.7	56.4±10.3	54.5±11.0	54.8±10.6	56.1±10.7
Height (cm)	145.2±8.1	145.9±8.1	144.5±8.1	146.3±8.0	144.2±8.2
Tanner stage (I/II/III/IV/V)*	78/74/35/13/2	50/43/10/3/0	28/31/25/10/2	41/28/20/10/2	37/46/15/3/0
Mothers education (Low/medium/high)	29/101/78	14/56/40	14/45/38	3/52/52	26/49/26
Dietary variables					
Energy intake (kcal/day)	1741±396	1806±400	1678±369	1820±409	1669±355
Total CHO (g/day)	182±49	188±46	176±51	192±52	172±43
Total Fat (g/day)	77±24	80±25	75±23	82±26	74±21
Total Protein (g/day)	76±19	81±20	71±17	77±20	76±18
Solids ED (kcal/g)	1.80±0.42	1.80±0.41	1.81±0.43	1.93±0.44	1.68±0.35
Solids and liquids ED (kcal/g)	1.14±0.21	1.15±0.21	1.14±0.21	1.20±0.22	1.09±0.19
Body composition					
Body Fat %	41.7±5.5	41.0±5.2	42.6±5.7	39.5±4.6	44.1±5.3
Fat mass index (kg/m^2)	10.9±2.7	10.8±2.7	11.1±2.7	10.1±2.4	11.8±2.8
Abdominal FMI (kg/m^2)[‡]	0.81±0.30	0.79±0.29	0.81±0.31	0.72±0.27	0.91±0.31
Waist circumference (cm)	84.43±10.19	86.65±9.55	81.89±10.34	79.0±7.6	90.2±9.4
BMI (kg/m^2)	26.1±3.5	26.3±3.4	25.9±3.5	25.5±3.3	26.8±3.5
Cardiometabolic markers					
Systolic BP (mm Hg)[&]	106.4±14.8	107.7±14.6	105.0±14.9	96.7±9.9	117.5±11.1
Diastolic BP (mm Hg)[&]	66.2±9.9	66.9±10.4	65.3±9.2	61.9±8.1	70.9±9.4
MAP (mm Hg)[&]	79±10	80±10	78±10	73±7	86±8
Triglycerides (mg/dL)[$]	89±43	85±41	94±44	84±40	94±45
TG/HDL ratio[$]	2.0±2.1	1.8±1.2	2.3±2.8	1.8±1.1	2.3±2.8
Total cholesterol (mg/dL)[$]	169.9±27.5	169.7±27.9	170.1±27.1	171.5±27.4	168.1±27.6
HDL (mg/dL)[$]	50.3±11.4	51.9±11.8	48.5±10.7	50.3±11.0	50.4±11.8
Glucose (mg/dL)[$]	85.9±6.2	87.1±5.7	84.5±6.4	85.5±5.5	86.3±6.8
LDL(mg/dL)[#]	102.5±24.6	101.6±24.2	103.5±25.2	104.4±23.6	100.1±25.6
Insulin(IU/dL)[&]	13±7	12±6	14±8	12±5	13±8
Physical activity levels					
MVPA (min/day)	54.3±21.2	59.7±21.8	48.2±18.7	56.8±21.9	51.7±20.2
Resting metabolic rate (Kcal)					
BMR Lazzer1*	1462±188	1573±148	1340±147	1441±189	1483±190
BMR Lazzer 2*	1433±171	1535±130	1319±136	1422±171	1423±171
BMR Schoffied	1483±234	1641±183	1303±152	1457±232	1510±244

*n=202 (96 girls and 106 boys)
[&]n=201 (94 girls and 107 boys)
[$]n=204 (94 girls and 110 boys)
[#]n=195 (88 girls and 197 boys)
[‡]n=190 (85 girls and 105 boys)

BMI=Body mass index; BMR=Basal metabolic rate; BP=Blood pressure; CHO=Carbohydrates; cm=centimeters; ED=Dietary energy density; ENMO=Euclidean norm minus one; FMI=Fat mass index; g=grams; HDL=High density lipoprotein; Kcal=kilocalories; MVPA=Moderate and vigorous physical activity; Lazzer1=Lazzer equation calculated with tanner stage and height; Lazzer2=Lazzer equation calculated with fat percentage from DXA; LDL=Low density lipoprotein; m=meters; MAP=Mean arterial pressure; TG=Triglycerides

Table 3. Linear regressions testing the influence of energetic density (only solids) on body composition variables and cardiometabolic risk scores.

Sample	*BMR equation*	*Independent var.*			*BMI*	*Waist*	*BF%*	*FMI*	*ABD_FMI*	*CM Risk1*	*CM Risk2*
Whole sample	**Not used**	**Energetic density**	M1	**n**	208	208	208	208	190	196	191
				B standard.	.064	.057	.107	.081	.095	-.041	.014
				p	.389	.364	.163	.298	.251	.604	.861
			M2	**B standard.**	.044	.043	.087	.060	.081	-.063	-.004
				p	.536	.482	.238	.418	.306	.408	.953
Under-reporters excluded	**Lazzer$_{DXA}$**	**Energetic density**	M1	**n**	**162**	162	**162**	**162**	**149**	154	151
				B standard.	**.162**	.092	**.208**	**.193**	**.188**	-.038	.037
				p	**.053**	.196	**.013**	**.025**	**.040**	.672	.666
			M2	**B standard.**	**.148**	.084	**.194**	**.179**	**.183**	-.047	.029
				p	**.067**	.232	**.017**	**.033**	**.041**	.591	.726
	Lazzer$_{weight}$	**Energetic density**	M1	**n**	**157**	157	**157**	**157**	**146**	149	147
				B standard.	**.175**	.094	**.223**	**.211**	**.195**	-.042	.030
				p	**.036**	.183	**.008**	**.014**	**.033**	.638	.728
			M2	**B standard.**	**.158**	.083	**.204**	**.193**	**.186**	-.055	.019
				p	**.050**	.233	**.012**	**.020**	**.036**	.528	.822
	Schofield	**Energetic density**	M1	**n**	**157**	157	**157**	**157**	**145**	149	149
				B standard.	**.198**	.121	**.237**	**.228**	**.216**	-.014	.062
				p	**.019**	.098	**.005**	**.008**	**.018**	.874	.474
			M2	**B standard.**	**.181**	.111	**.218**	**.209**	**.206**	-.029	.051
				p	**.028**	.125	**.007**	**.012**	**.021**	.743	.551

Grey and bold p≤.050; Bold p<.07.

M1=Model 1, adjusted by sex, age, mother's education and total energy intake.

M2=Model 1 + Moderate and vigorous physical activity adjustment

BMR=Resting metabolic rate; BMI=Body mass index; BF%=Body fat percentage; FMI=Fat mass index; ABD_FMI=Abdominal fat mass index; CM Risk1 =Cardiometabolic risk score described by Alberti et al.[59]; CM Risk 2=Cardiometabolic risk score created by Martinez Vizcaino et al.[58]; Waist=Waist circumference

Table 4. Linear regressions testing the influence of energetic density (solids and liquids) on body composition variables and cardiometabolic risk scores.

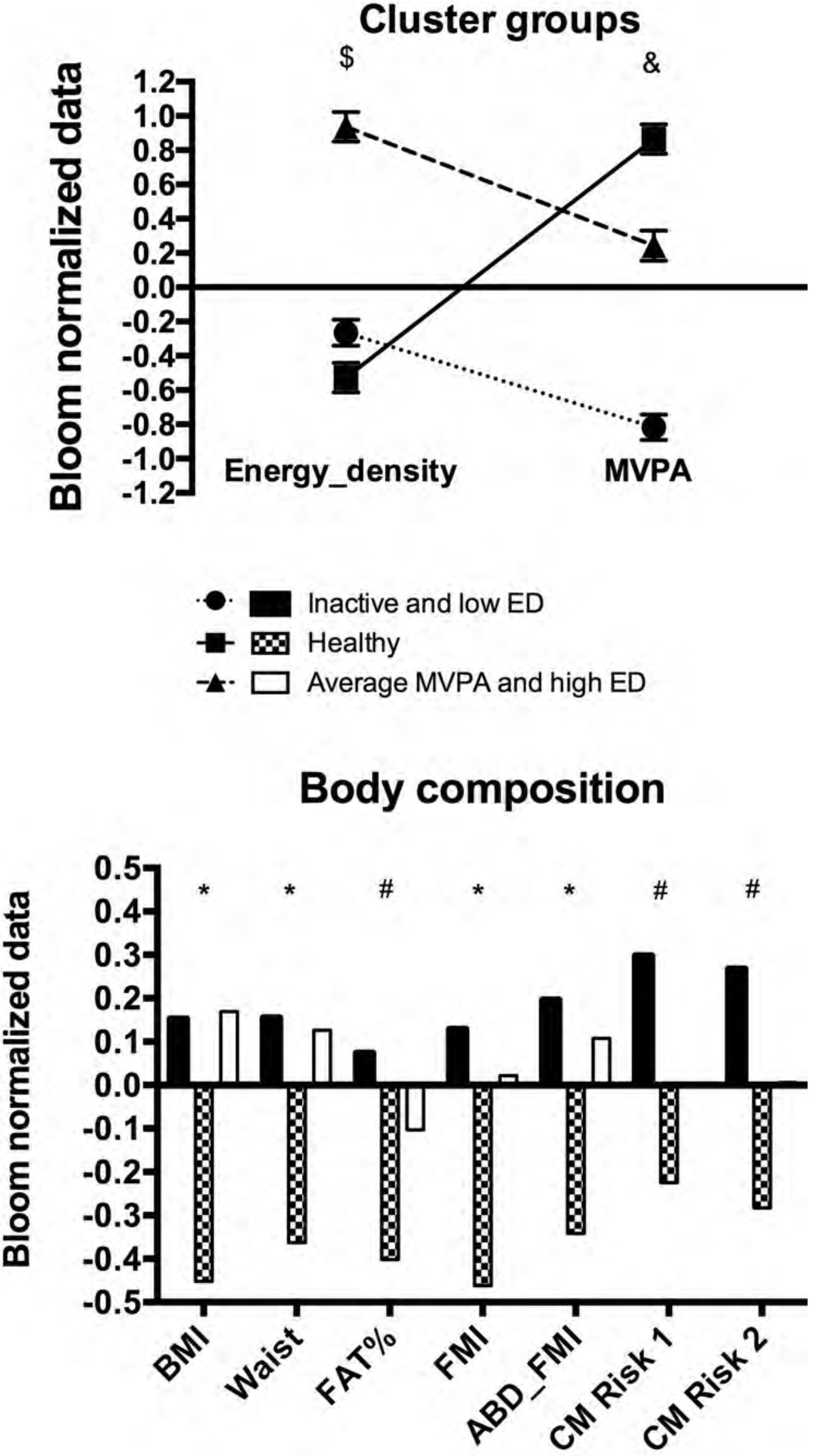
Sample	*BMR equation*	*Independent var.*			*BMI*	*Waist*	*BF%*	*FMI*	*ABD_FMI*	*CM Risk1*	*CM Risk2*
Whole sample	**Not used**	**Energetic density**	M1	**n**	208	208	208	208	190	196	191
				B standard.	.004	.002	.067	.026	.013	-.097	-.033
				p	.957	.979	.387	.736	.871	.213	.673
			M2	**B standard.**	-.017	-.013	.045	.004	-.003	-.122	-.053
				p	.808	.827	.541	.952	.974	.107	.471
Under-reporters excluded	**Lazzer$_{DXA}$**	**Energetic density**	M1	**n**	162	162	**162**	162	149	154	151
				B standard.	.120	.040	**.182**	.152	.122	-.131	-.006
				p	.149	.569	**.031**	.077	.179	.274	.940
			M2	**B standard.**	.105	.031	**.165**	.136	.298	-.108	-.017
				p	.195	.656	**.042**	.103	.199	.214	.841
	Lazzer$_{weight}$	**Energetic density**	M1	**n**	157	157	**157**	**157**	146	149	147
				B standard.	138	.050	**.202**	**.178**	.133	-.092	-.006
				p	.098	.483	**.017**	**.040**	.145	.299	.943
			M2	**B standard.**	.121	.038	**.184**	**.159**	.122	-.106	-.018
				p	.133	.580	**.023**	**.055**	.167	.222	.825
	Schofield	**Energetic density**	M1	**n**	157	157	**157**	**157**	145	149	149
				B standard.	.153	.072	**.222**	**.191**	.150	-.069	.019
				p	.071	.329	**.008**	**.026**	.099	.434	.823
			M2	**B standard.**	.133	.060	**.201**	**.170**	.136	-.087	.005
				p	.108	.409	**.013**	**.042**	.126	.316	.956

Grey and bold p≤.050; Bold p<.07. All models adjusted by sex, age, mother's education and total energy intake.

M1=Model 1, adjusted by sex, age, mother's education and total energy intake.

M2=Model 1 + Moderate and vigorous physical activity adjustment

BMR=Resting metabolic rate; BMI=Body mass index; BF%=Fat percentage; FMI=Fat mass index; ABD_FMI=Abdominal fat mass index; CM Risk1 =Cardiometabolic risk score described by Alberti et al.[59]; CM Risk 2=Cardiometabolic risk score created by Martinez Vizcaino et al.[58]. Waist=Waist circumference


Cluster groups
$
&
Bloom normalized data
1.2
1.0
0.8
0.6
0.4
0.2
0.0
-0.2
-0.4
-0.6
-0.8
-1.0
-1.2
Energy_density
MVPA
Inactive and low ED
Healthy
Average MVPA and high ED
Body composition
Bloom normalized data
0.5
0.4
0.3
0.2
0.1
0.0
-0.1
-0.2
-0.3
-0.4
-0.5
*
*
#
*
*
#
#
BMI
Waist
FAT%
FMI
ABD_FMI
CM Risk 1
CM Risk 2