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Socioeconomic status is positively associated with measures of adiposity and insulin resistance, but inversely associated with dyslipidaemia in Colombian children

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Abstract

Background—Low socioeconomic status (SES) has been associated with higher risk of cardiometabolic diseases in developed societies, but investigation of SES and cardiometabolic risk in children in less economically developed populations is sparse. We aimed to examine associations among SES and cardiometabolic risk factors in Colombian children.

Methods—We used data from a population-based study of 1282 children aged 6–10 years from Bucaramanga, Colombia. SES was classified according to household wealth, living conditions and access to public utilities. Anthropometric and biochemical parameters were measured at a clinic visit. Cardiometabolic risk factors were analysed continuously using linear regression and as binary outcomes—according to established paediatric cut points—using logistic regression to calculate OR and 95% CIs.

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Results—Mean age of the children was 8.4 (SD 1.4) and 51.1% of the sample were boys. Odds of overweight/obesity, abdominal obesity and insulin resistance were greater among higher SES. Compared with the lowest SES stratum, children in the highest SES had higher odds of overweight/obesity (OR=3.25, 95% CI 1.89 to 5.57), abdominal obesity (OR=2.74, 95% CI 1.41 to 5.31) and insulin resistance (OR=2.60, 95% CI 1.81 to 3.71). In contrast, children in the highest SES had lower odds of hypertriglyceridaemia (triglycerides 90th centile; OR=0.28, 95% CI 0.14 to 0.54) and low (10th centile) high-density lipoprotein (HDL) cholesterol (OR=0.35, 95% CI 0.15 to 0.78).

Conclusions—In Colombian children, SES is directly associated with obesity and insulin resistance, but inversely associated with dyslipidaemia (hypertriglyceridaemia and low HDL cholesterol). Our findings highlight the need to analyse cardiometabolic risk factors separately in children and to carefully consider a population's level of economic development when studying their social determinants of cardiometabolic disease.

Introduction

The worsening of socioeconomic inequalities occurring in most countries¹ is of utmost concern to public health, as disparities in wealth beget disparities in health.² Yet, the socioeconomic gradient of health and disease is not uniform across populations, with its patterns appearing to shift dynamically as a country moves through economic and nutritional transitions.³

In economically prosperous societies, overweight and obesity are more prevalent among children from lower socioeconomic status (SES).⁴ In this setting, presence of abdominal obesity and insulin resistance (IR) also appears to be more common among lower SES children.⁵ There is less evidence for a SES dyslipidaemia gradient,⁵ perhaps because lipid metabolism in children reflects a more diverse aetiology⁶ than other cardiometabolic factors that tend to cluster together.

Yet, despite the mounting evidence from studies in higher income countries, little is known about how SES impacts cardiometabolic risk factors beyond overweight and obesity in children of low and middle income countries (LMICs) who are projected to experience drastic increases in non-communicable disease-related deaths by 2030.⁷ The 'nutrition transition' theory,³ which has shown that as national income increases the burden of overweight and obesity within the population shifts from higher to lower SES groups,⁸ would suggest that the social patterning of other cardiometabolic risk factors in LMIC children might also be shifted. However, this hypothesis conflicts with the observation that cardiovascular disease mortality is higher among lower SES groups in LMICs.^{9–11} Considering this literature gap, our aim was to examine the associations between SES and cardiometabolic risk factors in Colombian children.

Methods

Study population

This cross-sectional study was embedded in SIMBA (Estudio Longitudinal para la Evaluación del Riesgo Cardiometabólico en la población Joven de Bucaramanga-Colombia),

a population-based cohort study designed to investigate the role of cardiovascular and metabolic risk factors in Colombian children.¹² A two-stage sampling process was performed to ascertain a representative sample of participants aged 6–10 years living in Bucaramanga, Colombia, in 2006/2007 (see ref 12 for complete details). In the first stage, a random selection of neighbourhoods that had at least 50 children was made. In the second stage, a random selection of a maximum of 50 houses in each neighbourhood was completed. All children who fulfilled the inclusion criteria in each of the selected houses were invited to participate in the study. In total, 1282 randomly selected children aged 6–10 years participated in the baseline examination. This included a home visit and a physical examination at the hospital research centre (Fundacion Cardiovascular de Colombia). Children with menarche history, Tanner stage 2, prior diagnosis of diabetes or other endocrine disorder, and/or previous treatments with hormones or steroids (except for sodium levothyroxine) during the month before the examination, were excluded. All procedures were approved by the Institutional Review Board of the Fundacion Cardiovascular de Colombia and the study was conducted in accordance with the guideline proposed in the Declaration of Helsinki. All parents/caregivers provided informed consent, and the children gave their informed assent to participate in the study during a formal evaluation by a trained psychologist.

Measurement of SES and other covariates

During the first home visit, parents/caregivers and children were interviewed face-to-face by six trained researchers using a questionnaire that included sociodemographic factors (child's age, sex, healthcare access and educational level of the child), health behaviour and medical history of the children. Childhood SES was defined according to the Colombian government classification DANE and SISBEN—designed to bill for public utilities and facilitate the distribution of subsidy programmes.¹³ This system classifies households into six categories (1 the lowest) by evaluating each individual home's wealth (possession of durable goods, human capital endowments and current income), housing conditions (construction type and materials), availability and quality of housing and basic public services (sewer, electricity, potable water and garbage collection). We used this system to define a child's SES as: low-low (stratum 1), low (stratum 2), middle (stratum 3), or high (strata 4–6). These definitions, consistent with previous studies^{14–16} that have used this classification system, allow for sufficient sample size (power) per category.

Child's age, sex, school attendance and healthcare access were assessed at baseline examination by questionnaire through face-to-face interview with the parent/guardian. Race/ethnicity was determined by observation of child racial phenotypic features and consultation with the parent/guardian. This variable was categorised into four groups: White, Black, indigenous and other. School attendance was determined by asking if the child is registered in school. Healthcare access, determined by examination of the child's healthcare coverage, was categorised into four groups: no healthcare coverage, low healthcare coverage (subsidised health coverage that is very restricted health coverage), medium healthcare coverage (regular health coverage) and high healthcare coverage (full elite health coverage). Medical history was derived from questions on maternal prenatal care, family history of cardiovascular diseases (CVDs), child's birth weight and breast feeding duration. We

categorised breast feeding as ≥ 6 months or <6 months. Maternal alcohol consumption and/or smoking during pregnancy was determined by asking if the mother consumed at least one of any type of alcoholic beverages and/or smoked during the pregnancy of the index child. Passive smoke exposure was determined by asking whether anyone at home smoked nearby the children. The child's alcohol consumption was defined by child's self-reported consumption of alcoholic beverages at least once. Physical activity was collected using the International Physical Activity Questionnaire,¹⁷ which included questions related to time spent in vigorous and moderate physical activity, sleep, study activities and sedentary behaviour, both at school and at home, and on weekdays as well as weekend days. These questionnaires were given to the parent/guardian and the child. Based on intensity in metabolic equivalent hours per week (METs h/week), we classified physical activity into tertiles (low <116 , moderate $116-176$ and high >176 METs h/week).¹⁷ Sedentary behaviour (h/day) was defined as time spent viewing television, playing video games or using the computer. Based on the daily time spent in sedentary behaviours, children were classified into three groups according to the recommendations from the American Academy of Pediatrics and previous literature: <2 , $2-3.9$ and ≥ 4 h/day.¹⁸ Sleep duration (h/day) was calculated and included short nap times during the day. This variable was categorised into two groups according to the National Sleep Foundation recommendations for specific age groups.¹⁹ For preschoolers (<6 years of age), a daily average sleep duration ≥ 11.1 h was considered as adequate. For school-aged children (≥ 6 years), a daily average sleep duration ≥ 10.1 h was considered as adequate. Maternal and paternal overweight or obesity was assessed by asking if the parents were diagnosed as overweight or obese by a health worker.

Measurement of cardiometabolic outcomes

During a second visit at the hospital research centre, a complete clinical and physical examination was performed by nine trained observers.¹² Height and weight were measured in the morning after urine elimination, with children wearing light clothes and no shoes, using a wall-mounted Seca 202 stadiometer (Seca, Hannover, Maryland, USA) and a Tanita HS301 electronic scale (Tanita, Arlington Heights, Illinois, USA), respectively. Body mass index (BMI; kg/m^2) age-and-sex-specific z-scores were calculated according to the 2000 Centers for Disease Control and Prevention (CDC) Growth references in the USA.^{20,21} Waist circumference and skinfolds were assessed in duplicate by two trained healthcare workers. Waist circumference was measured following standardised protocols at the midpoint between the lower ribs and the iliac crest with an inextensible measuring tape with precision to 0.1 cm. Skinfolds were measured on the right side of the body using Holtain calipers (Holtain Ltd, Crymch, UK). Age-and-sex-specific percentiles of waist circumference were calculated based on a Mexican-American reference population.²²

Systolic and diastolic blood pressure (BP) was assessed three times at 5 min intervals, after 10 min rest, using an automated device (Dinamap Pro 100; GE Medical Systems Information Technologies, Inc, Milwaukee, Wisconsin, USA). Mean arterial pressure (MAP) was calculated as: $((2 \times \text{diastolic}) + \text{systolic})/3$. Venous blood samples were collected in vacutainer tubes after >10 h of fasting. Serum was extracted and immediately processed using the colorimetric method (Biosystems BTS-303 Photometric, Barcelona, Spain) to assess plasma lipid and glucose concentrations. High-density lipoprotein cholesterol (HDL-

c) and triglyceride levels were determined from plasma (mmol/L). Very-low-density lipoprotein cholesterol (VLDL-c) was estimated by calculating triglycerides (mmol/L)/2.2. Fasting insulin concentrations were determined from the remaining samples previously stored at -80°C , in duplicate, by electrochemiluminescence immunoassay 'ECLIA' (Roche Elecsys 1010/2010 and MODULAR ANALYTICS E170 immunoassay analysers, Roche Diagnostics, Indianapolis, Indiana, USA). IR was defined according to the homeostasis model assessment of IR (HOMA-IR): fasting glucose concentration (mmol/L) $\times$ fasting insulin (IU/L)/22.5.²³²⁴ Smoothed percentiles for HOMA-IR, triglycerides and HDL-c were calculated based on our population, using the Least Mean Square (LMS) method.²⁵²⁶ Internal z-scores were calculated by taking the observed value minus the age-and-sex-specific population mean and dividing by the age-and-sex population SD.

Overweight or obesity was defined as 85th centile of CDC age-and-sex-specific BMI z-scores.²⁰ Other risk factor cut-offs were largely consistent with those described by the American Heart Association and the International Diabetes Federation for paediatric populations.²⁷²⁸ Abdominal obesity was defined as waist circumference 90th centile.²² IR was defined as HOMA-IR 90th centile.²⁴²⁹³⁰ High triglyceride level was defined as triglycerides (mmol/L) 90th centile, and low HDL-c was defined as HDL-c (mmol/L) 10th centile.²⁸ Finally, as recommended by the National High Blood Pressure Education Program (NHBPEP),³¹ we used age-sex-and-height-specific systolic and/or diastolic BP 90th centile to indicate high BP.

Statistical analysis

Differences in baseline characteristics between SES groups were assessed using one-way analysis of variance (ANOVA) or Kruskal-Wallis tests for continuous variables, and χ^2 tests for categorical variables that were not normally distributed. Non-normally distributed variables were log transformed before modelling. Multivariable logistic regression models were used to analyse the associations between SES and the odds of overweight or obesity, and MetS components (waist circumference 90th centile, HOMA-IR 90th centile, triglycerides 90th centile, HDL-c 10th centile, systolic or/and diastolic BP 90th centile). We then used multivariable linear regression to analyse SES in relation to z-scores for continuous cardiometabolic outcomes (BMI, waist circumference, HOMA-IR, MAP, triglycerides, HDL-c and VLDL-c).

Covariates were considered as potential confounders if there was evidence—from literature and internal data analysis—that the variable was associated with SES and cardiometabolic risk, and not on the causal pathway. These covariates were retained in the model if the β coefficients for main effects changed by more than 10%. We present three multivariable models. Model A was adjusted for age and sex (except for BP, HOMA-IR, triglycerides and HDL-c percentiles, which were based on age-and-sex-specific z-scores from our population). In model B we further adjusted for race/ethnicity, sleep duration, healthcare access, passive smoke exposure and paternal overweight or obesity. Finally, in model C, we additionally adjusted for BMI (except for models where BMI was the outcome). Adjusting for birth weight, breast feeding duration, physical activity and sedentary behaviour (as both continuous and categorical variables), as well as maternal smoking and alcohol consumption

during pregnancy, did not appreciably alter effect estimates and thus were not retained in the final models.

We performed multiple imputations of missing covariates (<16% missing values) based on the correlations between variables with missing values and other subject characteristics, to reduce potential bias associated with missing data.³² The Markov Chain Monte Carlo method was used to create five independent data sets, assuming no monotone missing pattern. As we observed similar effect estimates in the original data set and in the imputed data set, we present the pooled effect estimates based on imputed data sets. Analyses were performed using SAS V.9.2 (SAS Institute, Cary, North Carolina, USA), STATA V.12.0 (StataCorp LP, College Station, Texas, USA) and SPSS V.20.0 (SPSS Inc, Chicago, Illinois, USA).

Results

All of the 1282 recruited children were included in the present study, with the number of participants per outcome varying between 1278 and 1282 (due to missing values for specific outcomes). Table 1 presents the characteristics of the study population, stratified by SES group. The mean age of the children was 8.4 (SD 1.4) years and 51.1% were boys. There were 23% in the lowest SES stratum and 16.4% in the highest.

In unadjusted ANOVA (table 1), children in the lower SES stratum were more likely to be indigenous, have limited access to healthcare, have tried alcohol, have been exposed to household smoke and have missed school. Lower SES was also associated with longer sleep duration, shorter stature, lower BMI, abdominal skinfolds, waist circumference and fasting insulin. In addition, children in the lower SES had, on average, higher triglycerides and VLDL-c and lower HDL-c.

Results for analyses of SES and cardiometabolic component z-scores, after three levels (model A-C) of covariate adjustment, can be found in table 2 and figure 1. In multivariable model B, children in the highest SES had higher BMI (regression coefficient=0.53, 95% CI 0.32 to 0.74), waist circumference (regression coefficient=0.35, 95% CI 0.16 to 0.54) and HOMA-IR (regression coefficient=0.44, 95% CI 0.25 to 0.63) as compared with the lowest SES children. Furthermore, children with high SES had higher triglycerides (regression coefficient -0.53, 95% CI -0.72 to -0.34) and lower HDL-c (regression coefficient 0.18, 95% CI -0.02 to 0.37) compared with the lowest SES children.

In table 3 we present ORs and 95% CIs for SES and binary cardiometabolic risk factor outcomes. Odds of overweight or obesity, abdominal obesity and IR were greater among higher SES. As compared with the lowest SES group, children from the highest SES had greater odds of overweight or obesity (OR=3.25, 95% CI 1.89 to 5.57) and abdominal obesity (OR=2.74, 95% CI 1.41 to 5.31). Odds of IR were also higher for children in the middle (OR=2.69, 95% CI 1.45 to 5.00) or high SES (OR=2.60, 95% CI 1.81 to 3.71), as compared with the lowest SES. In contrast, odds of dyslipidaemia measures were lower among higher SES. Children in the highest SES group had lower odds of high triglycerides (OR=0.28, 95% CI 0.14 to 0.54) and low HDL-c (OR=0.35, 95% CI 0.15 to 0.78), as

compared with the lowest SES. These associations remained after adjustment for BMI (model C) and after excluding those who were <2 SDs from the BMI-for-age mean (data not shown). The results did not appear to differ by age (median split) or sex (p for interactions >0.10).

Discussion

In this population-based study of Colombian children, SES was significantly associated with cardiometabolic components, but the direction of the associations varied by the component under analysis. SES was positively associated with odds of overweight or obesity, abdominal obesity and IR, but inversely associated with odds of dyslipidaemia (high triglycerides and low HDL-c). These findings suggest that social patterning of health may vary by a population's stage in the nutrition transition and, furthermore, they stress the importance of examining cardiometabolic components in children individually and not as a composite (eg, metabolic syndrome).

A systematic review of studies in children from higher income countries found that SES (as measured by home income, occupation, housing conditions, access to public utilities, or education level) was inversely associated with overweight or obesity.⁴ There is evidence of a similar socioeconomic pattern for other cardiometabolic conditions in these settings.⁵ Our results, consistent with 'nutrition transition' theory,³ and supported by the literature from China⁸ and Latin America,^{1533–36} suggest that in contemporary LMICs such as Colombia, general and abdominal obesity is more prevalent in affluent children.

Our data suggest that this dynamic shift in the social inequalities of cardiometabolic health is not limited to measures of adiposity. We also found that IR was more common among higher SES children in Colombia. The direction of this association is in contrast with social patterning of IR in children from high income European countries,³⁷ but it aligns with observations from lower income countries.³⁷ Thus, in terms of social patterning of adiposity and IR, children who lived in Bucaramanga, Colombia in 2006/2007 are most characteristic of a population in the early stage of nutrition transition.³ In the future, as Colombia progresses in economic development, it will be interesting to observe if the nutrition transition theory³ holds and the burden of obesity and IR in children from this country shifts from the rich to the poor.

Perhaps our most interesting results are those that relate to differences in lipids across SES groups. Despite having lower rates of obesity and IR, low SES children had greater dyslipidaemia (higher triglycerides and VLDL-c, and lower HDL-c). These seemingly paradoxical findings agree with a small body of literature from LMICs showing that in absence of other cardiometabolic risk, low SES children are more likely to present with dyslipidaemia than high SES children.^{38–40} A study of preschoolers in Venezuela found higher total cholesterol, triglycerides and LDL-c among low compared with high SES.⁴⁰ Furthermore, in a series of cross-sectional studies, Mendoza *et al*³⁸³⁹ showed that (poorer) Venezuelan children from public schools not only had higher triglycerides and lower HDL-c than private school Venezuelan children, but that they also had a worse lipid profile (with less fatness) than age-matched US children.³⁸³⁹ If childhood dyslipidaemia tracks into

adulthood,⁴¹ our findings suggest that low SES children in LMICs may be at future atherogenic risk despite having normal body weight and insulin homeostasis. This could explain why CVD mortality rates in LMICs are greater among lower SES groups^{9–11} despite them having lower BMI and IR.⁴²

Many LMICs, including Colombia, have experienced a rapid epidemiological and nutrition transition, which has modified health structures and social behaviour in their populations.³ In societies going through nutrition transition, subpopulations with higher SES may have the highest exposure to (global and local) environmental and cultural forces that reduce physical activity, and promote a Western dietary pattern high in refined carbohydrates,⁴³ thereby increasing certain cardiometabolic risk factors. On the other hand, the low prevalence of excess weight in lower SES groups may be due to a lack of access to foods, especially those of high nutrient density, along with relatively high physical activity levels due to lower television and automobile use.⁴⁴ Yet these postulates, and why low SES children in these settings have adverse lipid profiles despite low levels of obesity and IR, remain to be determined. Causal factors to be explored in future studies include exposure to stress, air pollution, and dietary patterns that prominently feature fried street food, and foods high in carbohydrates and sugars, but low in protein and other nutrients.

A strength of the SIMBA study is the population-based sampling of Colombian youth, and the careful face-to-face measurement of demographic, environmental, lifestyle and clinical factors by trained staff. Careful measurement of cardiometabolic variables should have reduced the potential for outcome misclassification and, thus, for biased estimates. Another strength is the analytic approach taken, which, by modelling individual metabolic factors as continuous and binary outcomes, heeds the literature questioning the relevance of the metabolic syndrome as composite measure and the cut-offs used to define it in paediatric populations.²⁸ Yet, because cardiometabolic outcomes and SES were measured in a cross-sectional manner, the associations observed in our study can only provide evidence of the social patterning of cardiometabolic risk factors, and should not be interpreted causally.

A limitation of our study is the lack of multiple measures of SES, which precluded us from disaggregating household-level and community-level factors. The previously validated⁴⁵ SISBEN and DANE classification system aggregates domicile wealth, housing conditions, and access to public services and utilities to classify household SES. It does not, however, discern the heterogeneous contribution of these factors (eg, household wealth vs public utilities), or educational status and income within households, on our study outcomes. As such, future research of these factors and neighbourhood-level factors, such as walkability and access to recreation and food outlets, is needed for a more nuanced understanding of how SES works across multiple levels to affect health. Another limitation of our study was that we did not have detailed information on all potential confounding variables. For example, we did not have information on exclusivity of breast feeding, which may better reflect breast feeding patterns than duration of breast feeding only. Furthermore, no information was available on levels of smoking and alcohol consumption. Finally, although we were able to control for multiple potential confounders, residual confounding due to unmeasured or poorly measured variables cannot be ruled out.

In sum, SES was positively associated with general and abdominal adiposity, and IR, but was inversely associated with indicators of dyslipidaemia, in this population-based study of Colombian children. These results demonstrate that socioeconomic inequalities in cardiometabolic health of children are dynamic—varying by societal context (ie, place and time) and risk factor. As such, they highlight the potential peril of not being able to detect individual atherogenic risk indicators when composite measures of risk, such as metabolic syndrome, are used in children. Moreover, our findings draw attention towards the need to better understand cultural and lifestyle factors that might differentiate cardiometabolic risk factors across SES groups in children from LMICs as compared with developed countries. Future prospective studies in children from LMICs, which include measures of nutritional and lifestyle factors, may shed light on the drivers of the observed social patterning of health, and thereby clarify candidates for cardiometabolic health intervention strategies in these children and their families as they move through economic and nutritional transitions.

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What is already known on this subject?

- The socioeconomic gradient of cardiometabolic health appears to shift dynamically as a country goes through economic and nutritional transitions.
- In high income countries, children from higher socioeconomic groups have better cardiometabolic health than groups below them.
- In low and middle income countries, children from higher socioeconomic groups have higher rates of obesity, suggesting that, in contrast to children from high-income societies, they may have worse cardiometabolic health.

What this study adds

- Socioeconomic status was positively associated with overweight/obesity, abdominal obesity and insulin resistance, but inversely associated with measures of dyslipidaemia, in Colombian children.
- The socioeconomic gradient of cardiometabolic health appears to be dynamic in children from low and middle income countries, highlighting the importance of analysing cardiovascular disease risk factors individually and not as a composite.
- Further research is needed to understand the sociodemographic, cultural and lifestyle factors that might differentiate cardiovascular risk factors across socioeconomic groups in children from low and middle income countries.

Figure 1.

Internal z-scores (mean $\pm$ SE) for cardiometabolic risk factors across socioeconomic status in Colombian children aged 6–10 years from the SIMBA study (HDL, high-density lipoprotein; HOMA-IR, homeostasis model assessment of insulin resistance; VLDL, very-low-density lipoprotein).

Table 1
Characteristics of prepubertal children aged 6–10 years participating in the SIMBA study of Bucaramanga, Colombia

	Total	Low-low SES (n=295)	Low SES (n=384)	Middle SES (n=393)	High SES (n=210)	p Value*
Male sex, n (%; total n=1282)	655 (51.1)	158 (53.6)	186 (48.4)	212 (53.9)	99 (47.1)	0.222
Low healthcare access, n (%; total n=1276)	616 (48.2)	215 (73.1)	228 (59.8)	142 (36.2)	31 (14.8)	<0.001
Race/ethnicity (total n=1277)						
White, n (%)	167 (13.1)	17 (5.8)	31 (8.1)	75 (19.1)	44 (21.1)	<0.001
Black, n (%)	76 (5.9)	8 (2.7)	21 (5.5)	29 (7.4)	18 (8.6)	
Indigenous, n (%)	963 (75.1)	262 (88.8)	305 (80.1)	261 (66.6)	135 (64.6)	
Other, n (%)	71 (5.5)	8 (2.7)	24 (6.3)	27 (6.9)	12 (5.7)	
School attendance, n (%; total n=1282)	1263 (98.5)	290 (98.3)	371 (96.6)	392 (99.7)	210 (100)	0.001
Physical activity (h/day; total n=1277)	4.6 (1.9)	4.6 (1.9)	4.5 (1.9)	4.6 (2.0)	4.6 (1.7)	0.885
Sedentary behaviour (h/day; total n=1277)	3.3 (1.7)	3.2 (1.7)	3.4 (1.7)	3.4 (1.7)	3.1 (1.8)	0.176
Sleep duration (h/day; total n=1277)	10.1 (1.2)	10.3 (1.2)	10.1 (1.1)	10.0 (1.3)	9.9 (1.1)	0.002
Passive smoking, n (%; total n=1282)	343 (26.8)	100 (33.9)	118 (30.0)	79 (20.1)	46 (21.9)	<0.001
Alcohol at least once, n (%; total n=1279)	631 (49.3)	160 (54.2)	188 (49.1)	203 (51.8)	80 (38.3)	0.003
Breast fed 6 months, n (%; total n=1190)	979 (82.3)	220 (82.7)	288 (80.9)	313 (84.6)	158 (79.8)	0.441
Birth weight (g; total n=1069)	3332.9 (543.0)	3350.2 (588.7)	3326.3 (531.8)	3325.0 (563.0)	3337.8 (464.3)	0.948
Birth weight (<2500 g), n (%; total n=1069)	40 (3.7)	10 (4.4)	12 (3.8)	14 (4.1)	4 (2.2)	0.641
Smoking in pregnancy, n (%; total n=1245)	41 (3.3)	12 (4.2)	16 (4.3)	12 (3.1)	1 (0.5)	0.071
Maternal obesity, n (%; total n=1274)	89 (7.0)	12 (4.1)	34 (8.9)	26 (6.7)	17 (8.2)	0.092
Paternal obesity, n (%; total n=1271)	74 (5.8)	13 (4.4)	20 (5.2)	27 (7.0)	14 (6.7)	0.467
Childhood clinical measurements						
BMI (kg/m ² ; total n=1278)	16.0 (13.2, 23.1)	15.3 (13.0, 21.4)	16.0 (13.1, 23.2)	16.0 (13.4, 23.4)	16.4 (13.5, 24.9)	<0.001 [†]
BMI (z-score<-2), n (%; total n=1278)	47 (3.7)	18 (6.1)	19 (5.0)	10 (2.6)	0 (0.0)	<0.001
Overweight, [‡] n (%; total n=1278)	155 (12.1)	25 (8.5)	42 (11.0)	51 (13.0)	37 (17.6)	0.016
Height (cm; total n=1280)	127.6 (9.6)	126.6 (9.4)	127.3 (9.1)	127.9 (9.8)	129.0 (9.9)	0.037
Height (z-score <-2), n (%; total n=1280)	76 (5.9)	30 (10.2)	22 (5.8)	20 (5.1)	4 (1.9)	<0.001
Waist circumference (cm; total n=1282)	56.5 (7.2)	55.2 (6.2)	56.2 (6.9)	57.1 (7.5)	58.0 (8.2)	<0.001
Abdominal skinfolds (mm; total n=1282)	8.5 (4.0, 32.0)	7.5 (3.9, 30.0)	8.3 (4.1, 30.6)	9.1 (4.2, 33.8)	9.9 (4.1, 34.1)	<0.001 [†]
Hip circumference (cm; total n=1282)	65.2 (7.5)	63.3 (6.9)	64.9 (7.0)	65.8 (7.9)	67.1 (8.0)	<0.001

	Total	Low-low SES (n=295)	Low SES (n=384)	Middle SES (n=393)	High SES (n=210)	p Value*
Mean arterial blood pressure (mm Hg; total n=1282)	72.3 (6.5)	71.8 (7.0)	72.3 (6.6)	72.8 (6.1)	72.2 (6.2)	0.290
Triglyceride (mmol/L; total n=1282)	0.6 (0.2, 1.5)	0.9 (0.3, 1.6)	0.7 (0.2, 1.6)	0.6 (0.2, 1.5)	0.6 (0.2, 1.5)	<0.001 [†]
HDL cholesterol (mmol/L; total n=1282)	1.4 (0.3)	1.3 (0.3)	1.4 (0.3)	1.4 (0.3)	1.5 (0.3)	0.002
VLDL cholesterol (mmol/L; total n=1282)	0.3 (0.1, 0.8)	0.4 (0.2, 0.8)	0.3 (0.1, 0.9)	0.3 (0.1, 0.7)	0.3 (0.1, 1.8)	<0.001 [†]
Fasting glucose (mmol/L; total n=1278)	4.5 (0.5)	4.5 (0.5)	4.4 (0.4)	4.6 (0.5)	4.4 (0.5)	<0.001
Fasting insulin (µU/mL; total n= 1278)	5.0 (1.2, 17.0)	3.9 (0.9, 14.5)	4.9 (1.3, 13.4)	5.5 (0.8, 18.4)	6.2 (1.8, 18.6)	<0.001 [†]

Values are means (SD), numbers (percentages) or medians (95% range).

* Statistical significance was evaluated using one-way analysis of variance or Kruskal-Wallis test † for continuous variables, and χ^2 tests for categorical variables.

[†] Defined according to Centers for Disease Control and Prevention classification.

BMI, body mass index; HDL, high-density lipoprotein; SES, socioeconomic status; VLDL, very-low-density lipoprotein.

Table 2
Associations between SES and cardiometabolic risk factors in prepubertal children aged 6–10 years: the SIMBA study of Bucaramanga, Colombia

	Low-low SES (N=295) β (95% CI)	Low SES (N=384) β (95% CI)	Middle SES (N=393) β (95% CI)	High SES (N=210) β (95% CI)
BMI (z-score)*				
Model A	Reference	0.37 (0.21 to 0.54)	0.51 (0.38 to 0.63)	0.70 (0.50 to 0.89)
Model B	Reference	0.31 (0.14 to 0.47)	0.38 (0.21 to 0.56)	0.53 (0.32 to 0.74)
WC (z-score)				
Model A	Reference	0.20 (0.06 to 0.35)	0.33 (0.18 to 0.48)	0.52 (0.34 to 0.69)
Model B	Reference	0.15 (0.01 to 0.30)	0.21 (0.05 to 0.36)	0.35 (0.16 to 0.54)
HOMA-IR (z-score)*				
Model A	Reference	0.15 (0.01 to 0.30)	0.47 (0.32 to 0.61)	0.53 (0.36 to 0.70)
Model B	Reference	0.13 (−0.02 to 0.28)	0.41 (0.25 to 0.56)	0.44 (0.25 to 0.63)
Model C	Reference	0.03 (−0.10 to 0.17)	0.27 (0.13 to 0.41)	0.23 (0.06 to 0.41)
Blood pressure (z-score)				
Model A	Reference	0.09 (−0.06 to 0.24)	0.16 (0.01 to 0.32)	0.10 (−0.08 to 0.28)
Model B	Reference	0.06 (−0.09 to 0.21)	0.08 (−0.09 to 0.24)	−0.01 (−0.21 to 0.18)
Model C	Reference	0.00 (−0.15 to 0.15)	0.03 (−0.16 to 0.15)	−0.14 (−0.33 to 0.05)
Triglycerides (z-score)				
Model A	Reference	−0.33 (−0.48 to −0.18)	−0.40 (−0.54 to −0.25)	−0.47 (−0.64 to −0.30)
Model B	Reference	−0.36 (−0.51 to −0.21)	−0.45 (−0.61 to −0.29)	−0.53 (−0.72 to −0.34)
Model C	Reference	−0.39 (−0.54 to −0.24)	−0.50 (−0.66 to −0.34)	−0.60 (−0.79 to −0.41)
HDL-c (z-score)				
Model A	Reference	0.14 (−0.01 to 0.29)	0.21 (0.06 to 0.36)	0.31 (0.13 to 0.49)
Model B	Reference	0.08 (−0.07 to 0.24)	0.11 (−0.05 to 0.27)	0.18 (−0.02 to 0.37)
Model C	Reference	0.13 (−0.02 to 0.28)	0.17 (0.01 to 0.33)	0.27 (0.08 to 0.46)
VLDL-c (z-score)				
Model A	Reference	−0.17 (−0.32 to −0.02)	−0.19 (−0.34 to −0.04)	−0.12 (−0.30 to 0.06)
Model B	Reference	−0.19 (−0.34 to −0.04)	−0.22 (−0.38 to −0.06)	−0.14 (−0.34 to 0.06)
Model C	Reference	−0.21 (−0.37 to −0.06)	−0.25 (−0.41 to −0.09)	−0.19 (−0.39 to 0.01)

Values are regression coefficients and 95% CIs and reflect the difference in cardiometabolic risk factors by groups of SES, as compared with the reference group. Model A is adjusted for age and sex. Model B is adjusted for model A and additionally adjusted for sleep duration, healthcare access, race/ethnicity, passive smoking and paternal overweight/obesity. Model C is adjusted for model B and additionally adjusted for BMI.

*
n=1278.

BMI, body mass index; HDL-c, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; SES, socioeconomic status; VLDL-c, very-low-density lipoprotein cholesterol; WC, waist circumference.

Table 3
Associations between SES and cardiometabolic components in prepubertal children aged 6–10 years: the SIMBA study of Bucaramanga, Colombia

	Low-low SES (N=295) OR (95% CI)	Low SES (N=384) OR (95% CI)	Middle SES (N=393) OR (95% CI)	High SES (N=210) OR (95% CI)
Overweight or obesity, cases (%) [*]	29 (9.8)	67 (17.6)	77 (19.6)	64 (30.5)
Model A [†]	Reference	1.97 (1.24 to 3.15)	2.23 (1.41 to 3.53)	4.12 (2.54 to 6.69)
Model B [‡]	Reference	1.81 (1.13 to 2.92)	1.86 (1.14 to 3.05)	3.25 (1.89 to 5.57)
WC 90th centile, cases (%)	18 (6.13)	33 (8.7)	41 (10.5)	39 (18.8)
Model A [†]	Reference	1.50 (0.82 to 2.73)	1.83 (1.03 to 3.27)	3.77 (2.08 to 6.84)
Model B [‡]	Reference	1.36 (0.74 to 2.50)	1.42 (0.77 to 2.65)	2.74 (1.41 to 5.31)
HOMA-IR 90th centile, cases (%) [*]	16 (5.4)	29 (7.7)	59 (15.1)	31 (14.9)
Model A [†]	Reference	1.33 (0.70 to 2.51)	2.98 (1.67 to 5.30)	3.02 (1.61 to 5.68)
Model B [‡]	Reference	1.30 (0.68 to 2.48)	2.69 (1.45 to 5.00)	2.60 (1.81 to 3.71)
Model C [‡]	Reference	0.99 (0.50 to 1.97)	2.22 (1.15 to 4.29)	1.52 (0.70 to 3.27)
SBP or DBP 90th centile, cases (%)	7 (2.4)	13 (3.5)	12 (3.1)	9 (4.3)
Model A	Reference	1.48 (0.58 to 3.76)	1.29 (0.50 to 3.32)	1.89 (0.69 to 5.19)
Model B	Reference	1.51 (0.58 to 3.91)	1.22 (0.44 to 3.38)	1.92 (0.61 to 6.07)
Model C	Reference	1.25 (0.47 to 3.30)	0.96 (0.34 to 2.71)	1.14 (0.34 to 3.87)
Triglycerides 90th centile, cases (%)	44 (14.9)	38 (10.1)	33 (8.4)	16 (7.7)
Model A [‡]	Reference	0.63 (0.39 to 0.99)	0.52 (0.32 to 0.84)	0.47 (0.26 to 0.86)
Model B [‡]	Reference	0.53 (0.33 to 0.85)	0.34 (0.20 to 0.58)	0.28 (0.14 to 0.54)
Model C [‡]	Reference	0.47 (0.29 to 0.77)	0.29 (0.17 to 0.50)	0.21 (0.10 to 0.42)
HDL-c 10th centile, cases (%)	39 (13.2)	42 (11.1)	36 (9.2)	9 (4.3)
Model A [‡]	Reference	0.81 (0.51 to 1.28)	0.66 (0.41 to 1.07)	0.29 (0.14 to 0.62)
Model B	Reference	0.88 (0.55 to 1.42)	0.72 (0.43 to 1.23)	0.35 (0.15 to 0.78)
Model C [‡]	Reference	0.80 (0.49 to 1.29)	0.43 (0.37 to 1.09)	0.27 (0.12 to 0.63)

Values are ORs and 95% CIs and reflect the risk of overweight/obesity by groups of SES, as compared with the reference group. Model A is adjusted for age and sex. Model B is adjusted for covariates in model A and sleep duration, healthcare access, race/ethnicity, passive smoking and paternal overweight/obesity. Model C is adjusted for covariates in model B and BMI (continuous).

^{*} N=1278.

[†] F test for trend p<0.01.

[‡] F test for trend p<0.05.

BMI, body mass index; DBP, diastolic blood pressure; HDL-c, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; SBP, systolic blood pressure; SES, socioeconomic status; WC, waist circumference.