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Changes in socio-economic status and lipoproteins in Chilean adolescents: a 16-year longitudinal study

Zachary J Madewell[1,*], Estela Blanco[2,3], Raquel Burrows[4], Betsy Lozoff[5,6] and Sheila Gahagan[2,5]

[1]University of California, San Diego/San Diego State University, PhD Program in Public Health (Epidemiology), 9500 Gilman Drive, La Jolla, CA 92093, CA, USA: [2]University of California, San Diego, Department of Pediatrics, La Jolla, CA, USA: [3]Universidad de Chile, Doctoral Program in Public Health, School of Public Health, Santiago, Chile: [4]Universidad de Chile, Instituto de Nutrición y Tecnología de los Alimentos, Santiago, Chile: [5]University of Michigan, Center for Human Growth & Development, Ann Arbor, MI, USA: [6]University of Michigan, Department of Pediatrics and Communicable Diseases, Ann Arbor, MI, USA

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Abstract

Objective: The present longitudinal study assessed whether changes in socio-economic status (SES) from infancy to adolescence were associated with plasma lipoprotein concentrations in adolescence, of which low HDL-cholesterol (HDL-C) and high LDL-cholesterol (LDL-C), TAG and total cholesterol (TC) concentrations are associated with higher cardiovascular risk.

Design: SES, assessed using the modified Graffar Index, was calculated at 1, 5, 10 and 16 years. Principal components factor analysis with varimax rotation extracted two orthogonal SES factors, termed 'environmental capital' and 'social capital'. Generalized linear models were used to analyse associations between environmental and social capital at 1 and 16 years and outcomes (HDL-C, LDL-C, TAG, TC) at 16 years, as well as changes in environmental and social capital from 1–5, 5–10, 10–16 and 1–16 years, and outcomes at 16 years.

Setting: Santiago, Chile.

Participants: We evaluated 665 participants from the Santiago Longitudinal Study enrolled at infancy in Fe-deficiency anaemia studies and examined every 5 years to age 16 years.

Results: Social capital in infancy was associated with higher HDL-C in adolescence. Environmental capital in adolescence was associated with higher LDL-C and TC during adolescence. Changing environmental capital from 1–16 years was associated with higher LDL-C. Changing environmental capital from 1–5 and 1–16 years was associated with higher TC.

Conclusions: Improvements in environmental capital throughout childhood were associated with less healthy LDL-C and TC concentrations in adolescence. We found no evidence of associations between changing environmental capital and HDL-C or TAG, or changing social capital and HDL-C, LDL-C, TAG or TC.

Keywords
Socio-economic status
Lipoproteins
Chile
Child health

Social inequalities in adult cardiovascular risk factors are well described in many contexts worldwide[1–4]. Furthermore, populations that have undergone very rapid economic transitions, from very low income and subsistence conditions to environments conducive to high energy intake, have experienced dramatic increases in CVD and diabetes[1,2]. Less is known about the effect of change in socio-economic status (SES) during childhood on the development of cardiovascular risk[5,6]. The role of changing SES related to the worldwide rise in childhood obesity and development of adolescent hyperlipidaemias has not been adequately studied. Over the last several decades, such changes in the socio-economic landscape were experienced in Chile following major advancements in infrastructure, quality of education, access to potable water, sanitation and health care[7–11]. Chile also experienced a concomitant rise in adverse health risk factors, including poor diet, sedentary behaviour, smoking and alcohol consumption[7,8,10]. Pervasive undernutrition in Chile in the 1960s–1980s was supplanted by overnutrition. Beginning in the late 1980s, the diet resembled a Western diet replete with high energy and hydrogenated

*Corresponding author: Email zmadewel@ucsd.edu

fats[7,10,11]. Consequently, overweight and obesity rates increased in Chilean adults and children. By 2015, the prevalence of obesity was 24–25 % in pre-school, kindergarten and first-grade students and 12–13 % for high-school freshmen[12].

In Chile, obesity, hypertension and metabolic syndrome are more prevalent in low-SES children than high-SES children[7,9,10,13]. Low-SES households consume half of the fruits and vegetables recommended by the WHO, instead consuming more processed foods, which are inexpensive and accessible[9,14]. In addition, there are disparities in physical activity. Children of high-SES households have more physical activity than children of low-SES households, possibly because of better access to secure recreational facilities[9,15]. There has been scant research on how changing SES influences these health behaviours. The few longitudinal studies reporting changes in SES and their influences on outcomes have focused on obesity, but results have been inconsistent. Some previous cohort studies found that upward SES mobility was protective against physical health problems and obesity for children and adolescents[16–18]. In contrast, other cohort studies reported that upward SES mobility in infancy and childhood resulted in higher obesity in childhood and adulthood, respectively[6,19]. None of these studies were based in Chile.

Plasma HDL-cholesterol (HDL-C), LDL-cholesterol (LDL-C), TAG and total cholesterol (TC) concentrations are surrogate markers for cardiovascular risk in children and adolescents[20,21]. Low HDL-C, high LDL-C, high TAG and high TC concentrations are associated with higher cardiovascular risk[22]. Results of cross-sectional studies are mixed, but many report lower concentrations of HDL-C and higher concentrations of LDL-C, TAG and TC among low-SES children, independent of other cardiometabolic risk factors[18,23–25]. The purpose of the present study was to determine whether changes in SES over time from infancy to adolescence are associated with plasma HDL-C, LDL-C, TAG and TC concentrations in adolescence in the Santiago Longitudinal Study (SLS). A secondary objective was to assess associations between SES in infancy and adolescence with HDL-C, LDL-C, TAG and TC in adolescence.

Methods

Study design and population

The present study includes 665 adolescents (51·6 % male) who were part of infancy Fe-deficiency anaemia projects and follow-up study in Santiago, Chile[26]. Between 1991 and 1996, 4- to 6-month-old infants were recruited from community clinics in four low- to middle-income, working class communities near the research centre, the Institute for Nutrition and Food Technology (INTA), University of Chile. Inclusion criteria were uncomplicated, singleton, term, vaginal birth with birth weight of 3 kg or more, no major congenital abnormalities and no prior Fe therapy. As part of a preventive trial, infants without Fe-deficiency anaemia were randomly assigned to high- or low-Fe supplementation, or usual nutrition. Primarily breast-fed infants were randomized to vitamins with or without Fe. The conditions lasted 6 months when infants were 6–12 months of age. Details about the enrolment and trial are described elsewhere[26]. Those who had Fe-deficiency anaemia at 6 months and the next non-anaemic control entered a neuromaturation study where infants received medicinal Fe (n 135; Fig. 1)[27].

Follow-up studies occurred at 5, 10, 16 and 21 years. At 5 years, 888 participants had comprehensive assessments of health and development. At 16 years, 679 participants from the 5-year follow-up participated in a study of adolescent obesity and cardiovascular risk[28], of whom 673 had complete laboratory data. Of these, 511 (76·8 %) had SES information at 1 year, 665 (100·0 %) at 5 years, 585 (88·0 %) at 10 years and 597 (89·8 %) at 16 years. The final sample for the present study included 665 participants with complete SES data for at least two time points and HDL-C, LDL-C, TAG and TC at 16 years. The study was approved by institutional review boards at INTA, University of Chile, University of California, San Diego and the University of Michigan. Parents or primary caregivers provided informed written consent and children provided informed written assent, according to the norms for Human Experimentation, Code of Ethics of the World Medical Association (Declaration of Helsinki, 1995).

Variables

The exposure of interest was change in SES, which was assessed using the modified Graffar Index at 1, 5, 10 and 16 years[29,30]. This index is based on thirteen items scored from 0 to 5 with higher scores indicating lower SES: (i) number of people in a household 'eating from one pot' (in the same house); (ii) father's presence in household; (iii) head of household's highest educational level; (iv) head of household's current occupation; (v) retirement and health insurance; (vi) property ownership; (vii) type of house construction; (viii) characteristics of kitchen; (ix) sewage/plumbing; (x) water infrastructure; (xi) number of garbage collections per week; (xii) count of household possessions (car/automobile, refrigerator, stereo, washing machine, black and white television, colour television); and (xiii) necessity of two people sharing a bed or overcrowding. Graffar variables were reverse coded for the present study for ease of interpretation. Thus, higher index scores indicate higher SES and could range from 0 to 65. The change in SES was calculated as the difference in SES indices between 1 and 5, 5 and 10, 10 and 16, and 1 and 16 years.

To represent SES, a principal components factor analysis with varimax rotation was used to extract orthogonal factors from the modified Graffar Index variables at 1 year

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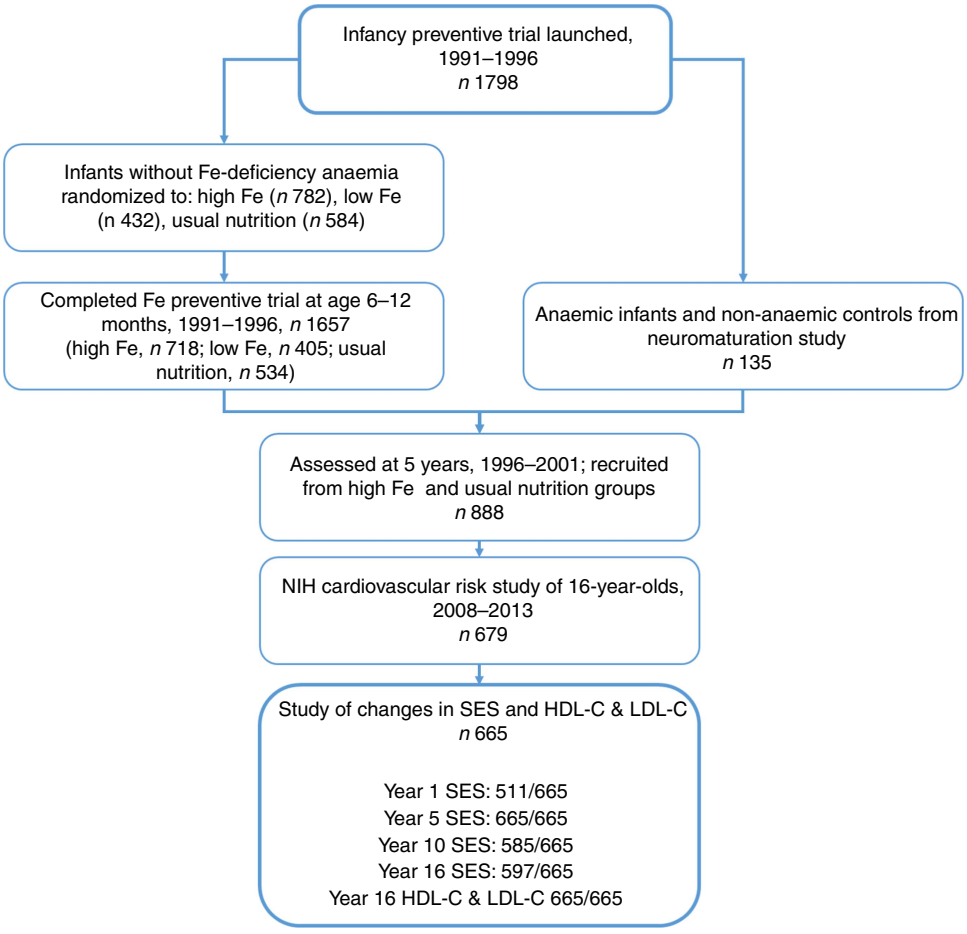

Fig. 1 (colour online) Flowchart of selected sample from the Santiago Longitudinal Study (NIH, National Institutes of Health; SES, socio-economic status; HDL-C, HDL-cholesterol; LDL-C, LDL-cholesterol)

(see online supplementary material, Supplemental Table 1). The eigenvalues of the correlation matrix from principal components analysis demonstrated that the first factor explained 19·9 % of the variability in the data and the second explained 12·4 % of the variability. Subsequent factors explained little variability. Therefore, only the first two factors were retained. Factor 1 included number of people eating from one pot or in the same house, property ownership, type of housing construction, characteristics of kitchen, water infrastructure and count of household possessions, which we term 'environmental capital'. Factor 2 included head of household education, head of household occupation, retirement and health insurance, and necessity of people sharing a bed or overcrowding, which we term 'social capital'. Variables highly correlated with each factor were weighted against their eigenvectors. The same two factors representing SES at 1 year (environmental and social capital) were used to represent SES at every other time point. Environmental and social capital ranged from 0 to 16 and 0 to 12, respectively.

The outcome variables for the present study were plasma concentrations of HDL-C (mg/dl), LDL-C (mg/dl), TAG (mg/dl) and TC (mg/dl) at 16 years, which were determined by dry analytical methodology (Vitros®; Ortho Clinical Diagnostics Inc., Raritan, NJ, USA) after a 12 h overnight fast.

Covariates at 16 years included BMI *Z*-score (WHO norms); age (years); parent-reported family history of diabetes, high cholesterol, hypertension or heart attack before age 60 years (yes, no); and infancy Fe group (high Fe, low Fe, no added Fe, neuromaturation study).

Statistical analysis

Means and standard deviations were calculated for continuous variables in adolescence (age, BMI, HDL-C, LDL-C, TAG, TC); environmental and social capital at 1, 5, 10 and 16 years; and change in environmental and social capital from 1 to 5, 5 to 10, 10 to 16, and 1 to 16 years (Table 1). Frequency distributions were calculated for categorical variables in adolescence (sex; parental history of diabetes, high cholesterol, hypertension, heart attack; supplementation group). Frequencies of low HDL-C, high LDL-C, high TAG and high TC were also reported using cut-off points defined by the American Academy of Pediatrics Committee on Nutrition[31]. HDL-C

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concentrations ≤38 mg/dl for girls and ≤34 mg/dl for boys were considered 'low', LDL-C concentrations ≥129 mg/dl for girls and ≥123 mg/dl for boys were considered 'high', TAG concentrations ≥112 mg/dl for girls and ≥125 mg/dl for boys were considered 'high', and TC concentrations ≥198 mg/dl for girls and ≥183 mg/dl for boys were considered 'high'.

As a previous study of this cohort found sex differences in the relationship between various cardiovascular risk factors and meeting criteria for the metabolic syndrome[28], we used two-way ANOVA to test interactions between sex and changes in environmental and social capital on the outcomes (HDL-C, LDL-C, TAG, TC).

Generalized linear models were used to analyse unadjusted (Model 1) and adjusted (Model 2) associations between environmental and social capital at 1 year and 16 years and outcomes (HDL-C, LDL-C, TAG, TC) at 16 years. Model 2 adjusted for confounders identified *a priori* from the literature: sex[28]; age[32]; and parental history of high cholesterol[33], diabetes[34], hypertension[35] and heart attack.

Three separate generalized linear models were constructed to analyse associations between the change in environmental and social capital between 1 and 5, 5 and 10, 10 and 16, and 1 and 16 years, and continuous outcomes (HDL-C, LDL-C, TAG, TC) at 16 years. Model 1 did not adjust for any variables. Model 2 adjusted for confounders (stated above). The final model (Model 3) additionally included baseline environmental or social capital. The baseline value considered for each association was the first value involved in the SES change. For example, environmental capital at 5 years was considered baseline for the environmental capital change between 5 and 10 years. Pearson correlation coefficients were examined to assess potential collinearity between baseline SES and changes in SES. Significance was determined using the Wald *F* test. Parameter coefficients, standard errors and *P* values were reported. All analyses were calculated using the statistical software package SAS version 9.4.

Table 1 Descriptive statistics of adolescents aged 16–17 years, Santiago, Chile (*N* 665)

Characteristic	Mean or *n*	SD or %
Continuous variables (mean and SD)		
Age (years)	16·8	0·3
BMI (*Z*-score)	0·7	1·2
HDL-C (mg/dl)	40·3	10·6
LDL-C (mg/dl)	94·1	24·4
TAG (mg/dl)	85·7	41·9
TC (mg/dl)	151·6	25·9
Categorical variables (*n* and %)		
Sex		
Female	318	47·8
Male	347	52·2
Parental history of		
Diabetes	86	14·0
High cholesterol	204	32·7
Hypertension	253	40·8
Heart attack	28	4·6
Infancy Fe group		
High Fe	312	46·9
Low Fe	18	2·7
No added Fe	284	42·7
Neuromaturation study*	51	7·7
HDL-C†		
Low	235	35·3
Normal	430	64·7
LDL-C†		
High	62	9·3
Normal	603	90·7
TAG†		
High	101	15·2
Normal	564	84·8
TC†		
High	44	6·6
Normal	621	93·4

HDL-C, HDL-cholesterol; LDL-C, LDL-cholesterol; TC, total cholesterol.
*Participants in the neuromaturation study were infants found to have Fe-deficiency anaemia at age 6 months and the next non-anaemic infant (control) whose neurodevelopment was evaluated with neurophysiological and electrophysiological techniques.
†By age and sex according to Daniels and Greer[31]: low HDL-C, ≤38 mg/dl for girls and ≤34 mg/dl for boys; normal HDL-C, >38 mg/dl for girls and >34 mg/dl for boys; high LDL-C, ≥129 mg/dl for girls and ≥123 mg/dl for boys; normal LDL-C, <129 mg/dl for girls and <123 mg/dl for boys; high TAG, ≥112 mg/dl for girls and ≥125 mg/dl for boys; normal TAG, <112 mg/dl for girls and <125 mg/dl for boys; high TC, ≥198 mg/dl for girls and ≥183 mg/dl for boys; normal TC, <198 mg/dl for girls and <183 mg/dl for boys.

Results

Sample characteristics

The mean age of participants in adolescence was 16·8 years and 52·2 % were male (Table 1). Overall, 35·3 % of

Table 2 Environmental* and social† capital‡ derived from principal components analysis§ of the modified Graffar Index‖ at 1, 5, 10 and 16 years, and change from years 1 to 5, 5 to 10, 10 to 16, and 1 to 16, Santiago, Chile

	Environmental capital		Social capital	
	Mean	SD	Mean	SD
Year				
1¶	8·2	4·6	5·0	2·3
5	9·5	3·8	5·5	2·2
10	10·4	3·0	5·8	2·2
16	10·5	2·9	6·0	2·1
Change				
Years 1 to 5	1·3	4·8	0·5	2·7
Years 5 to 10	0·9	3·5	0·4	2·1
Years 10 to 16	0·1	2·7	0·3	2·0
Years 1 to 16	2·4	4·7	1·1	2·7

*Environmental capital included: water infrastructure, type of housing construction, characteristics of kitchen, count of household possessions, property ownership and number of people eating from one pot or in the same house. Score range: 0 to 16.
†Social capital included: head of household education, head of household occupation, necessity of people sharing a bed or overcrowding, and retirement and health insurance. Score range: 0 to 12.
‡Higher capital = higher socio-economic status.
§Principal components factor analysis included an orthogonal varimax rotation.
‖According to Méndez Castellano and Méndez[30].
¶Missing data: year 1, *n* 154; year 10, *n* 80; year 16, *n* 68; years 1 to 5, *n* 154; years 5 to 10, *n* 80; years 10 to 16, *n* 124; years 1 to 16, *n* 201.

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adolescents had low HDL-C, 9·3 % had high LDL-C, 15·2 % had high TAG and 6·6 % had high TC. The mean environmental and social capital increased between each time point, with the largest changes occurring from 1 to 5 years and from 1 to 16 years (Table 2). From 1 to 16 years, 66·0 % of families increased in environmental capital, 32·1 % decreased and 1·9 % did not change. From 1 to 16 years, 61·8 % of families increased in social capital, 28·9 % decreased and 9·3 % did not change. There was no evidence of collinearity between baseline SES and changes in SES.

Environmental and social capital and HDL-cholesterol

There was an association between increasing environmental capital from 1 to 16 years with lower HDL-C ($P = 0{\cdot}03$) which was no longer significant after adjusting for age, sex and parental history of diabetes, high cholesterol, hypertension and heart attack (Table 3).

There was an association between social capital at 1 year and higher HDL-C during adolescence in the unadjusted and adjusted models ($P < 0{\cdot}01$). Increasing social capital from 1 to 16 years was negatively associated with HDL-C at 16 years in the unadjusted and adjusted models ($P \leq 0{\cdot}01$). This association was no longer significant after adjusting for baseline social capital in infancy (Table 3).

Environmental and social capital and LDL-cholesterol

Environmental capital at 16 years was associated with higher LDL-C during adolescence in Models 1 and 2 ($P = 0{\cdot}04$ and 0·03, respectively; Table 4). Increasing environmental capital from 1 to 16 years was associated with higher LDL-C in adolescence, after adjusting for environmental capital in infancy ($P = 0{\cdot}01$). We did not find associations of social capital or change in social capital with LDL-C ($P > 0{\cdot}10$).

Table 3 Unadjusted and adjusted associations between environmental* and social† capital‡ in infancy and adolescence and change in environmental and social capital from years 1 to 5, 5 to 10, 10 to 16, and 1 to 16, derived from principal components analysis§ of the modified Graffar Index‖, and HDL-cholesterol (HDL-C) levels in adolescence, Santiago, Chile

	HDL-C (mg/dl)					
	Environmental capital			Social capital		
Variable	Estimate	SE	*P* value	Estimate	SE	*P* value
Year 1 (*n* 511)¶						
Model 1**	0·15	0·11	0·16	0·66	0·21	<0·01
Model 2††	0·14	0·10	0·18	0·61	0·20	<0·01
Year 16 (*n* 597)¶						
Model 1**	−0·17	0·15	0·25	0·17	0·20	0·41
Model 2††	−0·12	0·15	0·40	0·12	0·20	0·56
Years 1 to 5 (*n* 511)¶						
Model 1**	−0·05	0·10	0·59	−0·39	0·18	0·03
Model 2††	−0·03	0·10	0·79	−0·31	0·18	0·08
Model 3‡‡	0·12	0·13	0·37	0·01	0·22	0·98
Years 5 to 10 (*n* 585)¶						
Model 1**	−0·14	0·13	0·27	−0·25	0·21	0·22
Model 2††	−0·19	0·12	0·26	−0·32	0·20	0·11
Model 3‡‡	−0·13	0·16	0·41	−0·23	0·23	0·31
Years 10 to 16 (*n* 541)¶						
Model 1**	−0·13	0·17	0·42	0·17	0·22	0·44
Model 2††	−0·06	0·16	0·70	0·07	0·22	0·76
Model 3‡‡	−0·13	0·19	0·51	0·07	0·25	0·79
Years 1 to 16 (*n* 464)¶						
Model 1**	−0·24	0·11	0·03	−0·47	0·19	0·01
Model 2††	−0·19	0·11	0·08	−0·48	0·18	<0·01
Model 3‡‡	−0·26	0·18	0·15	−0·20	0·24	0·41

*Environmental capital included: water infrastructure, type of housing construction, characteristics of kitchen, count of household possessions, property ownership and number of people eating from one pot or in the same house.
†Social capital included: head of household education, head of household occupation, necessity of people sharing a bed or overcrowding, and retirement and health insurance.
‡Higher capital = higher socio-economic status.
§Principal components factor analysis included an orthogonal varimax rotation.
‖According to Méndez Castellano and Méndez[30].
¶Missing data: year 1, *n* 154; year 10, *n* 80; year 16, *n* 68; years 1 to 5, *n* 154; years 5 to 10, *n* 80; years 10 to 16, *n* 124; years 1 to 16, *n* 201.
**Model 1 is unadjusted.
††Model 2 adjusted for sex; age; family history of high cholesterol, diabetes, hypertension and heart attack; and supplementation group.
‡‡Model 3 adjusted for sex; age; family history of high cholesterol, diabetes, hypertension and heart attack; supplementation group; and baseline capital (the first value involved in the capital change; i.e. environmental capital at year 1 was considered baseline for the environmental capital change between years 1 and 5).

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Table 4 Unadjusted and adjusted associations between environmental* and social† capital‡ in infancy and adolescence and change in environmental and social capital from years 1 to 5, 5 to 10, 10 to 16, and 1 to 16, derived from principal components analysis§ of the modified Graffar Index‖, and LDL-cholesterol (LDL-C) levels in adolescence, Santiago, Chile

	LDL-C (mg/dl)					
	Environmental capital			Social capital		
Variable	Estimate	SE	*P* value	Estimate	SE	*P* value
Year 1 (*n* 511)¶						
Model 1**	0·17	0·25	0·49	−0·50	0·48	0·30
Model 2††	0·19	0·25	0·45	−0·30	0·49	0·54
Year 16 (*n* 597)¶						
Model 1**	0·72	0·35	0·04	−0·28	0·47	0·55
Model 2††	0·73	0·35	0·03	−0·28	0·47	0·55
Years 1 to 5 (*n* 511)¶						
Model 1**	0·19	0·23	0·40	0·39	0·42	0·35
Model 2††	0·17	0·23	0·46	0·37	0·42	0·37
Model 3‡‡	0·55	0·32	0·08	0·34	0·52	0·52
Years 5 to 10 (*n* 585)¶						
Model 1**	−0·19	0·30	0·53	0·43	0·48	0·37
Model 2††	−0·23	0·30	0·45	0·38	0·48	0·44
Model 3‡‡	0·09	0·39	0·81	0·58	0·55	0·30
Years 10 to 16 (*n* 541)¶						
Model 1**	0·25	0·39	0·53	−0·74	0·53	0·16
Model 2††	0·22	0·40	0·58	−0·87	0·53	0·10
Model 3‡‡	0·63	0·46	0·17	−0·81	0·6	0·18
Years 1 to 16 (*n* 464)¶						
Model 1**	0·18	0·25	0·47	0·40	0·44	0·36
Model 2††	0·15	0·25	0·55	0·26	0·44	0·56
Model 3‡‡	1·06	0·43	0·01	0·13	0·58	0·82

*Environmental capital included: water infrastructure, type of housing construction, characteristics of kitchen, count of household possessions, property ownership and number of people eating from one pot or in the same house.
†Social capital included: head of household education, head of household occupation, necessity of people sharing a bed or overcrowding, and retirement and health insurance.
‡Higher capital = higher socio-economic status.
§Principal components factor analysis included an orthogonal varimax rotation.
‖According to Méndez Castellano and Méndez[30].
¶Missing data: year 1, *n* 154; year 10, *n* 80; year 16, *n* 68; years 1 to 5, *n* 154; years 5 to 10, *n* 80; years 10 to 16, *n* 124; years 1 to 16, n 201.
**Model 1 is unadjusted.
††Model 2 adjusted for sex; age; family history of high cholesterol, diabetes, hypertension and heart attack; and supplementation group.
‡‡Model 3 adjusted for sex; age; family history of high cholesterol, diabetes, hypertension and heart attack; supplementation group; and baseline capital (the first value involved in the capital change; i.e. environmental capital at year 1 was considered baseline for the environmental capital change between years 1 and 5).

Environmental and social capital and total cholesterol

Environmental capital at 16 years was associated with higher TC during adolescence in Model 2 ($P = 0{\cdot}04$; Table 5). Increasing environmental capital from 1 to 5 years ($P = 0{\cdot}03$) and from 1 to 16 years ($P = 0{\cdot}02$) were associated with higher TC in adolescence, after adjusting for environmental capital in infancy. We did not find associations of social capital or change in social capital with TC ($P > 0{\cdot}30$).

These findings did not vary by sex ($P > 0{\cdot}24$). Additionally, we did not find associations between environmental or social capital or change in environmental or social capital and TAG ($P \geq 0{\cdot}08$; Table 6).

Discussion

Over one-third of Chilean adolescents in our study had low HDL-C at age 16 years, 9·3 % had high LDL-C, 15·2 % had high TAG and 6·6 % had high TC. Higher social capital in infancy, assessed by head of household education, occupation, retirement and health insurance, and lower necessity of sharing a bed, was associated with higher HDL-C during adolescence. Environmental capital in adolescence, which included lower number of people eating from one pot, property ownership, house construction, kitchen characteristics, water infrastructure and household possessions, was associated with higher LDL-C and TC during adolescence. Increasing environmental capital from 1 to 16 years was associated with higher LDL-C in adolescence. Additionally, increasing environmental capital from 1 to 5 years and from 1 to 16 years were associated with higher TC. Finally, increasing social capital from 1 to 16 years was associated with lower HDL-C, but this association was no longer significant after holding baseline social capital constant.

Our finding that 35·3 % of adolescents had low HDL-C is similar to that in a study by Barja *et al.* who found low concentrations of HDL-C in 31·4 % of a sample of

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Table 5 Unadjusted and adjusted associations between environmental* and social† capital‡ in infancy and adolescence and change in environmental and social capital from years 1 to 5, 5 to 10, 10 to 16, and 1 to 16, derived from principal components analysis§ of the modified Graffar Index‖, and total cholesterol (TC) levels in adolescence, Santiago, Chile

	TC (mg/dl)					
	Environmental capital			Social capital		
Variable	Estimate	SE	*P* value	Estimate	SE	*P* value
Year 1 (*n* 511)¶						
Model 1**	0·26	0·26	0·31	0·12	0·51	0·82
Model 2††	0·25	0·26	0·32	0·22	0·50	0·67
Year 16 (*n* 597)¶						
Model 1**	0·68	0·37	0·07	−0·14	0·50	0·77
Model 2††	0·74	0·37	0·04	−0·19	0·50	0·70
Years 1 to 5 (*n* 511)¶						
Model 1**	0·21	0·24	0·40	−0·11	0·44	0·80
Model 2††	0·22	0·24	0·36	0·01	0·44	0·99
Model 3‡‡	0·72	0·33	0·03	0·18	0·54	0·74
Years 5 to 10 (*n* 585)¶						
Model 1**	−0·33	0·31	0·30	0·15	0·51	0·76
Model 2††	−0·42	0·31	0·18	0·02	0·51	0·97
Model 3‡‡	0·43	0·38	0·26	0·23	0·59	0·69
Years 10 to 16 (*n* 541)¶						
Model 1**	0·24	0·42	0·57	−0·34	0·56	0·54
Model 2††	0·32	0·42	0·45	−0·58	0·56	0·30
Model 3‡‡	0·69	0·49	0·16	−0·59	0·64	0·36
Years 1 to 16 (*n* 464)¶						
Model 1**	0·06	0·26	0·82	−0·05	0·46	0·91
Model 2††	0·11	0·27	0·67	−0·15	0·46	0·75
Model 3‡‡	1·07	0·45	0·02	−0·09	0·61	0·88

*Environmental capital included: water infrastructure, type of housing construction, characteristics of kitchen, count of household possessions, property ownership and number of people eating from one pot or in the same house.
†Social capital included: head of household education, head of household occupation, necessity of people sharing a bed or overcrowding, and retirement and health insurance.
‡Higher capital = higher socio-economic status.
§Principal components factor analysis included an orthogonal varimax rotation.
‖According to Méndez Castellano and Méndez[30].
¶Missing data: year 1, *n* 154; year 10, *n* 80; year 16, *n* 68; years 1 to 5, *n* 154; years 5 to 10, *n* 80; years 10 to 16, *n* 124; years 1 to 16, *n* 201.
**Model 1 is unadjusted.
††Model 2 adjusted for sex; age; family history of high cholesterol, diabetes, hypertension and heart attack; and supplementation group.
‡‡Model 3 adjusted for sex; age; family history of high cholesterol, diabetes, hypertension and heart attack; supplementation group; and baseline capital (the first value involved in the capital change; i.e. environmental capital at year 1 was considered baseline for the environmental capital change between years 1 and 5).

Chilean schoolchildren aged 10–14 years[36]. These findings are consistent with the interpretation by Barja *et al.* that the Chilean diet follows a more atherogenic pattern than the international norm[36]. High consumption of ultra-processed foods, carbohydrates, snacks, cereals and sweets are associated with low HDL-C[37] and are more common among children from low-SES neighbourhoods[7,9].

Our finding that social capital in infancy was associated with greater HDL-C in adolescence is in accord with other studies[23,24,38]. At least one other study has shown that children of higher SES are less likely to have dyslipidaemia and lower HDL-C than children of low-SES families, which may in part be attributed to better access to nutritious foods[24]. Our study also demonstrated that positive change in social capital from 1 to 16 years was associated with lower HDL-C. To assess associations between the change in social capital (e.g. head of household's education, occupation, retirement and health insurance, and lower necessity of sharing a bed) and lipoproteins, we held social capital in infancy constant. The finding that the association was no longer significant after controlling for social capital at infancy suggests that social capital in infancy may have a greater influence on later HDL-C levels than subsequent changes in social capital. These results are also consistent with a large US multisite longitudinal study which showed that SES in infancy had a larger impact on adiposity in the first 10 years of life compared with changes in SES in childhood[6].

Many studies have demonstrated associations between low SES and increases in LDL-C and TC[18,24,25]. Our study suggests that certain aspects of SES are especially relevant. We found that higher environmental capital (e.g. lower number of people eating from one pot, property ownership, house construction, kitchen characteristics, water infrastructure and household possessions) in adolescence was associated with higher LDL-C and TC in adolescence. In countries that have undergone a rapid social and

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Table 6 Unadjusted and adjusted associations between environmental* and social† capital‡ in infancy and adolescence and change in environmental and social capital from years 1 to 5, 5 to 10, 10 to 16, and 1 to 16, derived from principal components analysis§ of the modified Graffar Index‖, and TAG levels in adolescence, Santiago, Chile

	TAG (mg/dl)					
	Environmental capital			Social capital		
Variable	Estimate	SE	*P* value	Estimate	SE	*P* value
Year 1 (*n* 511)¶						
Model 1**	−0·29	0·41	0·49	−0·19	0·82	0·82
Model 2††	−0·36	0·41	0·39	−0·46	0·82	0·57
Year 16 (*n* 597)¶						
Model 1**	0·61	0·60	0·31	−0·19	0·81	0·81
Model 2††	0·67	0·60	0·27	−0·16	0·81	0·85
Years 1 to 5 (*n* 511)¶						
Model 1**	0·33	0·39	0·41	−0·56	0·71	0·43
Model 2††	0·36	0·39	0·36	−0·30	0·71	0·67
Model 3‡‡	0·25	0·54	0·65	−0·83	0·88	0·34
Yeasr 5 to 10 (*n* 585)¶						
Model 1**	−0·01	0·51	0·98	−0·12	0·83	0·88
Model 2††	−0·02	0·51	0·98	−0·16	0·83	0·85
Model 3‡‡	−0·35	0·67	0·60	−0·77	0·90	0·39
Years 10 to 16 (*n* 541)¶						
Model 1**	0·62	0·68	0·36	1·14	0·91	0·21
Model 2††	0·80	0·68	0·24	1·09	0·91	0·23
Model 3‡‡	0·92	0·80	0·25	0·75	1·04	0·47
Years 1 to 16 (*n* 464)¶						
Model 1**	0·58	0·43	0·18	0·05	0·75	0·95
Model 2††	0·73	0·43	0·09	0·36	0·76	0·63
Model 3‡‡	1·29	0·73	0·08	−0·14	0·99	0·89

*Environmental capital included: water infrastructure, type of housing construction, characteristics of kitchen, count of household possessions, property ownership and number of people eating from one pot or in the same house.
†Social capital included: head of household education, head of household occupation, necessity of people sharing a bed or overcrowding, and retirement and health insurance.
‡Higher capital = higher socio-economic status.
§Principal components factor analysis included an orthogonal varimax rotation.
‖According to Méndez Castellano and Méndez[30].
¶Missing data: year 1, *n* 154; year 10, *n* 80; year 16, *n* 68; years 1 to 5, *n* 154; years 5 to 10, *n* 80; years 10 to 16, *n* 124; years 1 to 16, *n* 201.
**Model 1 is unadjusted.
††Model 2 adjusted for sex; age; family history of high cholesterol, diabetes, hypertension and heart attack; and supplementation group.
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nutritional transition, higher SES may promote sedentary behaviour and increase Western dietary patterns high in carbohydrates and simple sugars, thereby increasing LDL-C and TC[24,38].

The apparent contradictory finding that social capital in infancy and environmental capital in adolescence were associated with greater HDL-C and LDL-C in adolescence, respectively, may be a reflection of measurements of social and environmental capital at different time points. It is conceivable that high social capital during infancy has long-term benefits for HDL-C into adolescence, whereas environmental capital in adolescence has a greater short-term influence on LDL-C in adolescence. Greater head of household education, occupation and health insurance early in life, represented by social capital, may indicate a better understanding of paediatric nutrition and better access to healthy foods over time. Greater property ownership, house construction and household possessions, represented by environmental capital, may relate to increased sedentary behaviour in the short term.

Positive changes in environmental capital from infancy to adolescence were associated with greater LDL-C and TC during adolescence. Additionally, positive changes in environmental capital from 1 to 5 years were associated with greater TC during adolescence. We are unaware of previous studies examining associations between changing SES and HDL-C, LDL-C, TAG or TC in a paediatric cohort. Previous studies of associations between SES change in childhood and cardiometabolic risk have been inconsistent. Some report benefits of upward SES mobility on cardiometabolic risk[16–18], whereas others do not[6,19]. In contrast to our study, these studies considered only occupation, education and income as measures of SES. In our study, environmental capital included measures of household infrastructure. Studies of adults in Jerusalem and Great Britain (aged 30–35 years and 44–45 years) have shown that upward SES mobility in childhood was positively associated with HDL-C and negatively associated with LDL-C[18,39]. Those studies of adults are in contrast to our findings among adolescents in a country with a rapidly

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expanding economy. One might hope that the positive effects of increasing environmental capital on SES will manifest for this cohort in adulthood. However, the contexts of these two studies are quite different from our study in Chile. The two reference studies focus on upward mobility in more stable economies, whereas ours takes place during a rapid economic transition with dramatic changes in the food and physical activity environments. It is conceivable that increasing environmental capital throughout childhood may influence behaviours, such as dietary preferences, that increase LDL-C. These behaviours during the first 5 years of life may be particularly influential on TC in adolescence. These results suggest that the rapid increase in environmental capital may pose health risks if not accompanied by an increase in educational and social capital. The divergent findings from the three studies suggest that experiencing dramatic society-wide economic and environmental changes is not comparable to and does not have the same health effects as upward mobility.

Environmental and social capital and the change in environmental and social capital were not found to be associated with TAG in adolescence. Since TAG are strongly affected by recent diet[40], it is possible that our one-time measurement of TAG is insufficient to adequately assess associations with environmental and social capital.

There are a few notable limitations of the present study. First, the sample of adolescents was recruited from neighbourhoods of low to middle SES and thus is not representative of the larger population of Chilean youth[41]. Second, despite the temporal precedence of change in SES measured since infancy related to HDL-C, LDL-C, TAG and TC measured in adolescence, we cannot infer causality. Third, the modified Graffar Index was designed to differentiate SES within low-income populations[29]. As SES improved for this cohort over the 16 years of study, some of the variables became irrelevant, such as access to sewers. Therefore, the measure may have become less useful in differentiating between levels of lower SES.

Notwithstanding these limitations, the longitudinal nature of the study design allowed us to assess the change in specific dimensions of SES over time. These results demonstrate that there may be early-life sensitive periods related to the role of SES in relation to lipoprotein concentrations later in life.

Conclusion

In a Chilean cohort, we found evidence to show that improvements in environmental capital throughout childhood, over a period of rapid economic transition, were associated with higher LDL-C and TC concentrations. However, social capital in infancy was associated with higher HDL-C in adolescence, while environmental capital in adolescence was associated with higher LDL-C and TC in adolescence.

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Supplementary material

To view supplementary material for this article, please visit https://doi.org/10.1017/S1368980018003087

References

1. Loria A, Arroyo P, Fernandez V *et al.* (2018) Prevalence of obesity and diabetes in the socioeconomic transition of rural Mayas of Yucatan from 1962 to 2000. *Ethn Health*. Published online: 20 February 2018. doi: 10.1080/13557858.2018.1442560.
2. Veazie M, Ayala C, Schieb L *et al.* (2014) Trends and disparities in heart disease mortality among American Indians/Alaska Natives, 1990–2009. *Am J Public Health* **104**, Suppl. 3, S359–S367.
3. Adjaye-Gbewonyo K, Kawachi I, Subramanian S *et al.* (2018) Income inequality and cardiovascular disease risk factors in a highly unequal country: a fixed-effects analysis from South Africa. *Int J Equity Health* **17**, 31.
4. Kim YJ, Lee JS, Park J *et al.* (2017) Trends in socioeconomic inequalities in five major risk factors for cardiovascular disease in the Korean population: a cross-sectional study using data from the Korea National Health and Nutrition Examination Survey, 2001–2014. *BMJ Open* **7**, e014070.
5. Kagura J, Adair LS, Pisa PT *et al.* (2016) Association of socioeconomic status change between infancy and adolescence, and blood pressure, in South African young adults: Birth to Twenty Cohort. *BMJ Open* **6**, e008805.
6. Starkey L & Revenson TA (2015) Early changes in socioeconomic status do not predict changes in body mass in the first decade of life. *Ann Behav Med* **49**, 212–220.

7. Albala C, Vio F, Kain J *et al.* (2002) Nutrition transition in Chile: determinants and consequences. *Public Health Nutr* **5**, 123–128.
8. Kain J, Uauy R, Vio F *et al.* (2002) Trends in overweight and obesity prevalence in Chilean children: comparison of three definitions. *Eur J Clin Nutr* **56**, 200–204.
9. Liberona Y, Castillo O, Engler V *et al.* (2011) Nutritional profile of schoolchildren from different socio-economic levels in Santiago, Chile. *Public Health Nutr* **14**, 142–149.
10. Vio F & Albala C (2000) Nutrition policy in the Chilean transition. *Public Health Nutr* **3**, 49–55.
11. Muzzo S, Burrows R, Cordero J *et al.* (2004) Trends in nutritional status and stature among school-age children in Chile. *Nutrition* **20**, 867–872.
12. Lira M & Vio A (2016) *Informe Mapa Nutricional 2015*. Santiago: JUNAEB, Ministerio de Educación-Gobierno de Chile.
13. Olivares C, Bustos Z, Lera M *et al.* (2007) Estado nutricional, consumo de alimentos y actividad física en escolares mujeres de diferente nivel socioeconómico de Santiago de Chile. *Rev Chil Nutr* **135**, 71–78.
14. Olivares S & Bustos N (2006) Consumo de verduras y frutas en grupos específicos de consumidores chilenos: elementos a considerar en su promoción. *Rev Chil Nutr* **33**, 260–264.
15. Burrows R, Díaz E, Sciaraffia V *et al.* (2008) Hábitos de ingesta y actividad física en escolares, según tipo de establecimiento al que asisten. *Rev Med Chil* **136**, 53–63.
16. Boylan SM, Gill TP, Hare-Bruun H *et al.* (2014) Associations between adolescent and adult socioeconomic status and risk of obesity and overweight in Danish adults. *Obes Res Clin Pract* **8**, e163–e171.
17. Cundiff JM, Boylan JM, Pardini DA *et al.* (2017) Moving up matters: socioeconomic mobility prospectively predicts better physical health. *Health Psychol* **36**, 609–617.
18. Savitsky B, Manor O, Friedlander Y *et al.* (2017) Associations of socioeconomic position in childhood and young adulthood with cardiometabolic risk factors: the Jerusalem Perinatal Family Follow-Up Study. *J Epidemiol Community Health* **71**, 43–51.
19. Aitsi-Selmi A, Batty G, Barbieri M *et al.* (2013) Childhood socioeconomic position, adult socioeconomic position and social mobility in relation to markers of adiposity in early adulthood: evidence of differential effects by gender in the 1978/79 Ribeirao Preto cohort study. *Int J Obes (Lond)* **37**, 439–447.
20. Amutha A, Pradeepa R, Chella S *et al.* (2017) Lipid profile in childhood-and youth-onset type 2 diabetes and their association with microvascular complications. *J Assoc Physicians India* **65**, 42–47.
21. Sultan S, Dowling M, Kirton A *et al.* (2018) Dyslipidemia in children with arterial ischemic stroke: prevalence and risk factors. *Pediatr Neurology* **78**, 46–54.
22. Centers for Disease Control and Prevention (2017) LDL and HDL cholesterol: 'bad' and 'good' cholesterol. https://www.cdc.gov/cholesterol/ldl_hdl.htm (accessed October 2017).
23. Velásquez E, Barón MA, Solano L *et al.* (2006) Perfil lipídico en preescolares Venezolanos según nivel socioeconómico. *Arch Latinoam Nutr* **56**, 22–28.
24. Buitrago-Lopez A, van den Hooven EH, Rueda-Clausen CF *et al.* (2015) Socioeconomic status is positively associated with measures of adiposity and insulin resistance, but inversely associated with dyslipidaemia in Colombian children. *J Epidemiol Community Health* **16**, 580–587.
25. Manios Y, Dimitriou M, Moschonis G *et al.* (2004) Cardiovascular disease risk factors among children of different socioeconomic status in Istanbul, Turkey: directions for public health and nutrition policy. *Lipids Health Dis* **3**, 11.
26. Lozoff B, De Andraca I, Castillo M *et al.* (2003) Behavioral and developmental effects of preventing iron-deficiency anemia in healthy full-term infants. *Pediatrics* **112**, 846–854.
27. Roncagliolo M, Garrido M, Walter T *et al.* (1998) Evidence of altered central nervous system development in infants with iron deficiency anemia at 6 mo: delayed maturation of auditory brainstem responses. *Am J Clin Nutr* **68**, 683–690.
28. Burrows R, Correa-Burrows P, Reyes M *et al.* (2016) High cardiometabolic risk in healthy Chilean adolescents: associations with anthropometric, biological and lifestyle factors. *Public Health Nutr* **19**, 486–493.
29. Graffar M (1956) Une méthode de classification sociale d'échantillons de population. *Courrier* **6**, 455–459.
30. Méndez Castellano H & Méndez MC (1986) Estratificación social y biología humana: método Graffar modificado. *Arch Venez Pueric Pediatr* **49**, 93–104.
31. Daniels SR & Greer FR (2008) Lipid screening and cardiovascular health in childhood. *Pediatrics* **122**, 198–208.
32. Mahley RW, Arslan P, Pekcan G *et al.* (2001) Plasma lipids in Turkish children: impact of puberty, socioeconomic status, and nutrition on plasma cholesterol and HDL. *J Lipid Res* **42**, 1996–2006.
33. Lauer RM, Lee J & Clarke WR (1988) Factors affecting the relationship between childhood and adult cholesterol levels: the Muscatine Study. *Pediatrics* **82**, 309–318.
34. Purnell JQ, Dev RK, Steffes MW *et al.* (2003) Relationship of family history of type 2 diabetes, hypoglycemia, and autoantibodies to weight gain and lipids with intensive and conventional therapy in the Diabetes Control and Complications Trial. *Diabetes* **52**, 2623–2629.
35. Facchini F, Ida Chen Y-D, Clinkingbeard C *et al.* (1992) Insulin resistance, hyperinsulinemia, and dyslipidemia in nonobese individuals with a family history of hypertension. *Am J Hypertens* **5**, 694–699.
36. Barja S, Barrios X, Arnaiz P *et al.* (2013) Niveles de lípidos sanguíneos en escolares Chilenos de 10 a 14 años de edad. *Nutr Hosp* **28**, 719–725.
37. Tavares LF, Fonseca SC, Rosa MLG *et al.* (2012) Relationship between ultra-processed foods and metabolic syndrome in adolescents from a Brazilian Family Doctor Program. *Public Health Nutr* **15**, 82–87.
38. Ruiz N, Bosch V, Rodriguez V *et al.* (2012) Estratificación socioeconómica, estado nutricional y lípidos plasmáticos en escolares venezolanos. *Rev Venez Endocrinol Metab* **10**, 28–37.
39. Power C, Atherton K, Strachan DP *et al.* (2007) Life-course influences on health in British adults: effects of socioeconomic position in childhood and adulthood. *Int J Epidemiol* **36**, 532–539.
40. Sadrzadeh H & Kline G (2017) *Biomarkers: Clinicians and Clinical Chemists in Partnership*. Amsterdam/London, Cambridge, MA: Elsevier.
41. Delva J, Lee W, Sanchez N *et al.* (2014) Ecological factors and adolescent marijuana use: results of a prospective study in Santiago, Chile. *Int J Environ Res Public Health* **11**, 3443–3452.