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Original Research

Impact of education, income and chronic disease risk factors on mortality of adults: does 'a pauper-rich paradox' exist in Latin American societies?

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SUMMARY

Objectives: To test the hypothesis that an inverse association exists between socioeconomic position and all-cause mortality in a developing country in Latin America.

Study design: Prospective cohort study carried out in Chile using data from a simple random sample of 920 apparently healthy subjects (weighted population 11,600 aged 30–89 years) followed for 8 years.

Methods: Education level (0–8 years, 9–12 years and \geq 13 years) and income quartiles were established at the outset of the study, along with behavioural and biological risk factors for chronic diseases: smoking, alcohol use, obesity, diabetes, hypertension, lipids and family history of death by cardiovascular disease. Relative risks of all-cause mortality were estimated using age-adjusted Cox regression models.

Results: During the follow-up period, 46 deaths were observed. Adjusting for age, gender, and behavioural and biological risk factors, the mortality risk for increasing categories of education after controlling for income was 1.0, 0.76 and 0.33 (P for trend < 0.01). In contrast, the relative risk for increasing levels of income after controlling for education was 1.0, 0.98, 1.33 and 1.17 (P for trend = 0.07).

Conclusion: While education level had a protective effect on mortality risk of Chilean adults, income had a slightly unfavourable effect on survival. This finding is described as suggestive of a 'pauper-rich paradox', since the higher income quantiles in this study correspond with the lower income levels in most developed countries. Nevertheless, due to the small number of deaths, additional research is required to assess the validity of these findings.

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Introduction

Education and income are important measures of socioeconomic position (SEP)¹ that have demonstrated a significant impact on health and mortality through multiple studies in recent decades.²⁻⁵ The mechanisms involved in this relationship are not well understood, and the adjustment for traditional biological and behavioural risk factors does not completely explain the relationship between SEP and mortality.^{2,6–10} The influence of the SEP determinants has focused the attention of investigators on issues of inequality and inequity affecting outcomes in health care worldwide.^{11–14} In addition, the level of education, considered as an early exposure measure, has been identified as a robust predictor for adult health and survival in several cohort studies.^{1–3,5,6} However, the general applicability of previous research on this subject has not been established, since most studies have included specific gender or ethnic groups in developed countries characterized by wealthier and more egalitarian societies.4,15,16 In addition, very few of these studies have been conducted prospectively in less affluent and more unequal societies, limiting their applicability to developing countries, especially in Latin America.

The present investigation (San Francisco Project, SFP) was conducted in Chile, a country in an advanced stage of economic, demographic and epidemiologic transition in Latin America.^{17–19} In the last three decades, the average per-capita income has increased up to US\$5000, the fertility rate has dropped dramatically (from 4.4 to 1.8 average births per childbearing woman), the average life expectancy has reached 79 years, and cardiovascular diseases and cancer are the main causes of death in the adult population. However, despite sustained economic growth, Chile presents significant inequalities in the distribution of income, with a Gini coefficient of 0.57.20 The Gini score can range from 0 to 1, with 0 corresponding to perfect equality and 1 corresponding to perfect inequality. While most developed European nations and Canada tend to have Gini indices between 0.24 and 0.36, the Gini coefficients of the USA and Mexico are above 0.40. South America is the most unequal region worldwide, with Gini indices above 0.50. Moreover, although the Chilean literacy rate has reached 96%, the average length of school attendance (8.5 years at present) is still lower than in developed countries.

The aim of this study was to test the hypothesis that an inverse association exists between SEP and all-cause mortality in a developing country after controlling for behavioural and biological chronic disease risk factors. Preliminary findings in the SFP cohort indicated that a low level of education was a strong predictor of all-cause mortality.²¹ However, in that study, education was only assessed utilizing a dichotomous variable; consequently, it was not possible to establish a dose-response gradient. In addition, income was never evaluated independently, but instead combined in a socio-economic rating scale with years of approved schooling. Therefore, the need to evaluate education and income separately became apparent. Finally, the higher income quartiles in Chile correspond to the lower income levels in most developed countries. These higher income groups constitute what is described in this study as the 'pauper rich'.

Methods

Population

The study was carried out in San Francisco de Mostazal, a town in the central region of Chile, 60 km south of Santiago. With a population of 21,896, 97.8% are Chilean-Hispanics (Spanish heritage with a variable indigenous component), with similar demographic features to the national average.²² Furthermore, this population presents a similar health risk profile with the participants of the National Health Survey²³ (Table S1, available at www.sciencedirect.com). San Francisco de Mostazal's main economic activity is the agrarian industry. The health centre (Consultorio Municipal de Mostazal) provides primary medical care, including laboratory tests, to 90% of the population. This centre has access to a complete registry of the social and demographic data of the population, along with patients' medical records. Medical care is provided by physicians, nurses, psychologists, dieticians, physical therapists and laboratory technicians. In addition, specialized health services are provided by two hospitals in nearby cities ('Regional Hospital', Rancagua, located 22 km to the south of San Francisco de Mostazal, and 'Barros Luco-Trudeau' Hospital, Santiago, 60 km to the north).

Sampling scheme

Using a geographic information system, an urban perimeter of 1995 neighbourhoods, distributed in eight geographic polygons, was identified in the centre of San Francisco de Mostazal. Excluding the population living in rural areas, 17,903 people were identified as stable residents in the urban sector of the town. A simple random sample of 518 neighbourhoods was selected and a population census was carried out from August to December 1996, selecting a simple random list of 1980 potentially eligible individuals for a general health survey in the region. A medical examination, laboratory test and population survey were performed between January 1997 and December 1999 to evaluate the prevalence of chronic disease risk factors, with an overall response rate of 73%.²⁴ Individuals with documented cardiovascular diseases (congestive heart failure, coronary heart disease and valvular heart disease), cancer, chronic renal failure, chronic obstructive lung disease, and physical or mental disabilities were excluded at baseline. After these exclusions and only considering subjects with complete data over the total period of follow-up, 920 subjects aged 30-89 years were included in the present study. In subsequent statistical analyses, mixed sample weights (integer and proportional) were used for each individual based on geographic distribution and census data.¹⁹ After correcting for exclusions, non-response, geographic location, gender and age composition, the weighted population corresponded to 11,600 apparently healthy individuals.

Education and income measures

Demographic and socio-economic information was obtained through a questionnaire at the outset of the study during a visit

to each participant's home. In Chile, education comprises three levels: (1) primary or elementary school education (1–8 years), (2) secondary education or high school (9–12 years) and (3) tertiary or college education (\geq 13 years). The education level of each participant was ascertained by self-report of the years of approved schooling, and coded in one of the aforementioned categories. Annual household income in Chilean currency (pesos), corrected for the number of dependent members, was assessed by self-report. In a subsequent analysis, income was converted to US dollars and categorized in the following population quartiles (Q): Q1, <US\$ 4200; Q2, US\$ 4200–6000; Q3, US\$ 6000–10,200; and Q4, > US\$10,200.

Biological and behavioural risk factors

Health risk factors were evaluated through medical examination at the local health centre. Weight and height were measured using a calibrated physician scale to the nearest 0.1 kg and a height rod to the nearest 0.2 cm, respectively. All the measurements were taken twice and their averages were used. Body mass index (BMI) was calculated by dividing the weight in kilograms by the square of the height in metres. Obesity was defined as $BMI > 29.9 \text{ kg/m}^2$. Three serial measurements of systolic and diastolic blood pressure (SBP and DBP; mmHg) were performed to diagnose arterial hypertension according to the criteria proposed by the Seventh Joint National Committee.²⁵ Fasting blood samples were obtained to determine blood glucose and lipid profile in the health centre laboratory, and processed using standard techniques. Dyslipidaemia was defined according to the cut-off values proposed by the National Cholesterol Education Program for total cholesterol, high-density lipoprotein cholesterol (HDL), low-density lipoprotein (LDL) and triglycerides.²⁶ Type 2 diabetes mellitus was diagnosed using a glucose tolerance test in subjects with a plasma glucose level ≥110 mg/dl.²⁷ Current smokers and the number of cigarettes smoked per day were assessed. In addition, the status of those who had stopped smoking or who had never smoked was considered. Alcohol consumption was assessed with the 'Escala breve de beber anormal' questionnaire (or 'Guidelines to assess the excessive drinker'), which has been validated in Chile to identify heavy drinkers.²⁸ Information about family history of death due to cardiovascular disease was self-reported.

Ascertainment of mortality outcomes

The dependent variable was all-cause mortality during the study period, from enrolment until January 2006. The participants were assessed by questionnaires, telephone calls, home visits, reports from family members and review of the medical records. Follow-up time for incident mortality events was determined by the number of days between the baseline survey and either death, last contact or 31 January 2006, whichever came first. The average follow-up time was 8 years. All deaths were confirmed by death certificate obtained from the National Office of Vital Records and National Health Service in Chile. This governmental organization uses International Classification of Diseases, Version 10 to classify the causes of mortality, with a national scope of 99% for death certification done by physicians.²⁹

Statistical analyses

Baseline risk factors are reported as means with standard deviations or proportions, and differences were tested by ageadjusted analysis of variance (ANOVA) or the Chi-squared statistic. Correlations were assessed by Pearson coefficient. The relative risks of mortality events were computed in increasing categories of education (reference group, basic education <9 years) and income (reference group, quartile Q1 < US\$4200) using Cox proportional hazards regression models to assess different pathways: Model 1 was adjusted by age (linear, quadratic and >65 years) and gender; Model 2 was adjusted by income and education (joint-effect model); Model 3 was adjusted by behavioural and biological risk factors; and Model 4 was obtained by adjusting Model 3 by income and education. All risk estimates are expressed as hazard ratios with 95% confidence intervals (CI).

Pathway modelling was employed to explore a possible causal model. Therefore, trends across categories of education and income were tested by use of a single ordinal term (1, 2, 3 for education; 1, 2, 3, 4 for income) for the category in the Cox regression model. The impact of the traditional chronic disease risk factors on the linear terms (changes in the β -coefficients with two-tailed P-values) for education or income using Cox proportional hazards models were evaluated.

Results

Baseline characteristics

The mean age of participants was 50.5 ± 14.9 years. The median duration of education and annual household income were 8 years (interquartile range 5–12) and US\$ 6000 (interquartile range US\$4200–10,800), respectively. From the total sample, 52.5% had a primary education, 34.5% had a secondary education and 13% had a college or tertiary education (P < 0.001; χ^2 for multiple proportions). A moderate inverse correlation between education and age was observed (r=-0.41, P < 0.001), and a modest correlation between income level and age was also noted (r=-0.15 P < 0.001). The age-adjusted partial correlation between years of education and income was r = 0.33 (P < 0.001). A primary education was a common finding in all groups, including the higher income group (over 33%, Fig. 1).

Table 1 shows that glucose levels, total cholesterol, LDL, triglycerides, BMI, SBP and DBP decreased with increasing education (P < 0.01, age-adjusted ANOVA). With respect to the annual household income, the highest quartile exhibited a higher blood glucose level, and lower total cholesterol, LDL cholesterol and HDL cholesterol compared with the lowest quartile.

Education, income and mortality

Table 2 shows the mortality risk with respect to education and income according to the Cox regression analysis. Over an average follow-up of 8 years, 46 cases of death were observed (aged 33–97 years). Since no difference was found in the association of income and education with mortality according to gender (interaction test P values of 0.25 and 0.31, respectively),





mortality risk estimates for men and women were combined. In Model 4, the relative mortality risks for increasing levels of education were 1.0, 0.76 and 0.33, and for increasing quartiles of income were 1.0, 0.99, 1.33 and 1.17. Fig. 2 shows the changes in risk expressed as percentages for different levels of education and income using Model 1 and Model 4; while education showed a protective effect, a higher income appeared to increase the mortality risk. Finally, in gender- and age-adjusted regression models, a significant impact on incident cases of allcause mortality for smoking, alcohol use, hypertension, diabetes, elevated cholesterol and family history of cardiovascular diseases was observed (Table 3).

Pathway modelling

The impact of pathway variables on the linear relationships of education and income with all-cause mortality was analysed through changes in β -coefficients (Table 4). In the age- and

| Table 1 – Means of fasting blood glucose, lipid factors, body mass index (BMI), systolic blood pressure (SBP) and diastolic blood pressure (DBP) based on education and income categories. | | | | | | | | | |
|---|----------------------|-----------------------------------|------------------------------------|----------------|----------------|------------------------------------|----------------------------------|------------------------------------|-----------------------------------|
| | n (N _w)ª | Blood glucose (mg/dl) | Total cholesterol (mg/dl) | LDL (mg/dl) | HDL (mg/dl) | Triglycerides (mg/dl) | BMI (kg/m²) | SBP (mmHg) | DBP (mmHg) |
| Education | | | | | | | | | |
| Basic (<9 years) | 483 (6092) | 95.3 ± 13.6 | 204.0 ± 27.3 | 136.4 ± 23.4 | 42.7 ± 6.3 | 133.9 ± 60.2 | $\textbf{27.7} \pm \textbf{4.6}$ | 131.7 ± 20.8 | $\textbf{83.0} \pm \textbf{11.5}$ |
| Secondary (9–12 years) | 317 (4003) | 95.1 ± 10.7 | 191.5 ± 25.6 | 126.6 ± 22.2 | 41.4 ± 5.2 | 122.5 ± 40.5 | 26.8 ± 4.8 | 125.8 ± 17.6 | $\textbf{79.3} \pm \textbf{12.1}$ |
| Tertiary (≥13 years) | 120 (1505) | 92.8 ± 10.3 | 189.2 ± 33.2 | 125.9 ± 25.6 | 41.1 ± 6.4 | 119.7 ± 39.0 | $\textbf{26.2} \pm \textbf{4.3}$ | 123.5 ± 16.2 | $\textbf{78.9} \pm \textbf{11.0}$ |
| Annual income o | uartiles US | \$ | | | | | | | |
| Q1: < 4,200 | 198 (2498) | $\textbf{94.0} \pm \textbf{13.9}$ | $\textbf{202.7} \pm \textbf{34.4}$ | 136.2 ± 28.1 | 43.7 ± 6.8 | $\textbf{129.0} \pm \textbf{59.3}$ | $\textbf{26.9} \pm \textbf{4.8}$ | 131.5 ± 23.0 | $\textbf{80.5} \pm \textbf{11.8}$ |
| Q2: 4200–6000 | 267 (3368) | $\textbf{93.3} \pm \textbf{12.8}$ | 196.0 ± 24.6 | 129.6 ± 21.6 | 42.8 ± 5.5 | 126.8 ± 59.6 | $\textbf{26.9} \pm \textbf{4.9}$ | 126.4 ± 17.3 | $\textbf{80.0} \pm \textbf{11.4}$ |
| Q3: 6000–10,200 | 223 (2810) | $\textbf{95.2} \pm \textbf{12.2}$ | $\textbf{196.3} \pm \textbf{26.4}$ | 131.5 ± 22.7 | 40.8 ± 5.7 | 127.7 ± 39.0 | $\textbf{27.1} \pm \textbf{4.3}$ | $\textbf{128.8} \pm \textbf{18.9}$ | $\textbf{82.2} \pm \textbf{12.7}$ |
| Q4: >10,200 | 232 (2924) | $\textbf{97.1} \pm \textbf{10.5}$ | 196.9 ± 28.0 | 130.3 ± 23.9 | 40.9 ± 5.5 | 129.3 ± 56.4 | $\textbf{27.7} \pm \textbf{4.5}$ | 128.6 ± 18.7 | $\textbf{81.9} \pm \textbf{11.2}$ |
| LDL/HDL, low-/high-density lipoprotein; Q1 to Q4, quartile 1 to quartile 4.Values refer to means \pm standard deviations. | | | | | | | | | |

a Weighted sample size by gender- and age-specific weights (integer and proportional) based on geographic distribution and census data form 2002.

| Table 2 – Relative risk with 95% confidence intervals for mortality based on education and income categories. | | | | | | |
|---|---------------------------------------|------------------|------------------|-------------------|------------------|--|
| Category | Deaths (N _w) ^a | Model 1 | Model 2 | Model 3 | Model 4 | |
| <9 years (basic) | 30 (516) | 1.0 | 1.0 | 1.0 | 1.0 | |
| 9–12 years (secondary) | 10 (105) | 0.69 (0.54–0.86) | 0.67 (0.53–0.85) | 0.78 (0.61–0.99) | 0.76 (0.60–0.96) | |
| \geq 13 years (tertiary) | 6 (73) | 0.27 (0.16–0.48) | 0.26 (0.15–0.27) | 0.36 (0.20–0.63) | 0.33 (0.19–0.59) | |
| P for trend | | 0.01 | 0.001 | 0.002 | 0.006 | |
| Annual income quartiles (L | JS\$) | | | | | |
| Q1: <4200 | 21 (292) | 1.0 | 1.0 | 1.0 | 1.0 | |
| Q2: 4200–6000 | 10 (152) | 0.91 (0.74–1.12) | 0.95 (0.77–1.17) | 0.93. (0.75–1.15) | 0.99 (0.78–1.21) | |
| Q3: 6000–10,200 | 7 (115) | 0.99 (0.79–1.25) | 1.08 (0.85–1.36) | 1.23 (0.96–1.57) | 1.33 (1.04–1.71) | |
| Q4: >10,200 | 8 (135) | 0.97 (0.78–1.19) | 1.11 (0.89–1.37) | 1.08 (0.86–1.34) | 1.17 (0.93–1.46) | |
| P for trend | | 0.863 | 0.298 | 0.282 | 0.06 | |
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Relative risks (RRs) refer to hazard ratios obtained from Cox proportional regression hazard models.

Model 1: adjusted for age (linear, quadratic and >65 years) and gender.

Model 2: adjusted for age (linear, quadratic and >65 years), gender, income (education model) or education (income model).

Model 3: adjusted for age (linear, quadratic and >65 years), gender, smoking status, alcohol use, body mass index, hypertension, diabetes, total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides and family history of cardiovascular disease. Model 4: Model 3 additionally adjusted for income level (for education model) or education (for income model).

a Weighted cases using gender- and age-specific mixed weights (integer and proportional) based on geographic distribution and census data.

gender-adjusted regression model, the β-coefficient related to education was -0.50 (P < 0.01). No change was observed after adjustment for income. In the model adjusted by behavioural and biological risk factors, the β -coefficient decreased to -0.36 (P < 0.01), increasing to -0.38 (P < 0.01) in the fully adjusted model. This corresponds to a 33% decrease in mortality events with each higher education category (relative risk 0.67; 95% CI 0.56-0.81). Only 24% of the inverse relationship between education and mortality was accounted for by combined effects of chronic disease risk factors and income. In the age- and gender-adjusted regression model, the β coefficient related to income was -0.01 (P = 0.863), changing to +0.04 (P = 0.298) and +0.03 (P = 0.282) after adjustment for education or risk factors, respectively. In the fully adjusted model, the β -coefficient related to income increased to +0.07 (P = 0.06). This corresponds to a 7% increase in mortality events with each higher income category (relative risk 1.07; 95% CI 0.99-1.15).

Discussion

In this adult cohort followed for 8 years in a developing country in Latin America, it was found that increasing levels of education had a protective effect against all-cause mortality. After the adjustment by behavioural and biological risk factors, there was a 33% reduction in the mortality risk for each increment in the education level. In addition, as in developed societies, this prospective study corroborates that traditional risk factors for chronic diseases have a major impact on adult mortality.⁴ In contrast with other studies,^{4,7,9,30–33} higher income levels had a neutral effect or even a slight increase in risk for all-cause mortality when adjusting for chronic diseaser risk factors and education. Nevertheless, due to the small absolute number of deaths, a longer follow-up period with higher rates of mortality is required to assess the validity of these findings.

The inverse association between SEP indicators and mortality is a reproducible epidemiological finding among many different cohort studies.^{2–7,9,15,30–37} Increasing attention is now being paid to understanding the possible pathways through which socio-economic inequalities exert their deleterious effects on health.³⁸ Education and income measures are not simply interchangeable, but emphasize different aspects of social stratification. In life-course perspectives, ^{1,3,9} education represents acquired knowledge and reflects the experiences of early life. Income, on the other hand, represents material resources and mirrors experiences in adult life. However, the proper point of placement of behavioural and biological risk factors in the causal pathway for SEP measures and mortality remains unclear. In this analysis, after controlling for age differences, the inverse relationship between education and mortality was not completely explained by traditional chronic disease risk factors. Moreover, the protective effect of education against all-cause mortality was not related to income level. Therefore, these data provisionally support the hypothesis that education may be an antecedent variable to income, chronic disease risk factors and mortality, and not a mediator of their relationship (Fig. 3).

Early findings in the SFP cohort indicated that a low level of education was a strong predictor of all-cause mortality.²¹ However, in previous analyses, the reference group for education was those who had completed primary school (i.e. 8 years or more of approved schooling). Therefore, without analysing education in three categories using the low educational level as the reference group, the existence of a protective doseresponse gradient on mortality risk - an important causal criterion in epidemiologic research - could not have been established. In addition, because income was never evaluated separately but by using a socio-economic rating scale (SRS) that combined years of education and income,^{21,22,24} the authors' previous study could not capture a paradoxical relationship as the current analysis has done. Moreover, the previous analyses were limited to crude and multivariate risk calculations based on Cox regression models adjusted for age, gender and chronic disease risk factors without a pre-established conceptual causal modelling. The relative risk of mortality associated with low education in that study was 1.54 and for the SRS was 1.24. In



Fig. 2 – Changes in mortality risk with increasing education (A) and income (B) in a cohort of Chilean adults followed for 8 years. Bars refer to the percentage of all-cause mortality risk with respect to the reference category for education (basic education) and income (lower quartile, Q1).

an unpublished re-analysis of the data, the relative risk of the SRS decreased to the null value after adjusting directly for education, suggesting that education completely explained the association of the SRS with mortality. This was an important limitation of the previous publication, which motivated the authors to undertake the current approach using a modelling pathway to capture possible differential effects of education and income on mortality risk.

It needs to be noted that the categories of income in this cohort were very modest and limited when compared with those obtained from studies carried out in developed countries.^{4,16,31–34,39} Albert et al.¹⁶ conducted a study among US

Table 3 – Relative risks for all-cause mortality associated with behavioural and biological risk factors for chronic diseases in the San Francisco Project Cohort Study.

| | Relative risk (95% confidence intervals) | | | | | |
|--|--|-----------------------------------|--|--|--|--|
| | Unadjusted risk | Age- and gender- adjusted risk | | | | |
| Smoking | | | | | | |
| No | 1.0 | | | | | |
| Yes | 1.66 (0.89–1.27) | 1.97 (1.63–2.39) | | | | |
| Alcohol use | | | | | | |
| No | 1.0 | | | | | |
| Yes | 0.88 (0.74–1.05) | 1.51 (1.24–1.84) | | | | |
| Obesity (body | mass index \geq 30 kg/m ²) | | | | | |
| No | 1.0 | | | | | |
| Yes | 1.30 (1.11–1.54) | 1.16 (0.98–1.38) | | | | |
| Hypertension | | | | | | |
| No | 1.0 | | | | | |
| Yes | 5.21 (4.43–6.13) | 1.74 (1.45–2.07) | | | | |
| Diabetes | | | | | | |
| No | 1.0 | | | | | |
| Yes | 6.27 (5.25–7.52) | 2.75 (2.29–3.30) | | | | |
| Total choleste | erol >240 mg/dl | | | | | |
| No | 1.0 | | | | | |
| Yes | 2.54 (2.03–3.16) | 1.37 (1.10–1.71) | | | | |
| LDL cholester | ol >160 mg/dl | | | | | |
| No | 1.0 | | | | | |
| Yes | 3.16 (2.65–3.76) | 1.22 (0.94–1.34) | | | | |
| HDL cholester | rol <40 mg/dl | | | | | |
| No | 1.0 | | | | | |
| Yes | 0.94 (0.80–1.10) | 0.89 (0.76–1.05) | | | | |
| Triglycerides >150 mg/dl | | | | | | |
| No | 1.0 | | | | | |
| Yes | 2.22 (1.89–2.61) | 1.14 (0.96–1.35) | | | | |
| Family history of death by cardiovascular disease | | | | | | |
| No | 1.0 | 4 40 (4 07 4 70) | | | | |
| Yes | 1.49 (1.29–1.74) | 1.48 (1.27–1.73) | | | | |
| LDL/HDL, low-/high-density lipoprotein. | | | | | | |
| Relative risks refer to hazard ratios obtained from Cox proportional | | | | | | |
| regression ha | zard models. | | | | | |

female health workers and considered US\$ <19,999 as their lowest level of annual household income: in the present study, the highest quartile for this parameter was US\$10,200. The higher income groups in the present cohort correspond with the lowest income level of most cohorts from developed countries; therefore, these highest income groups in a low absolute income milieu constitute what have been referred to as the 'pauper rich'. After controlling for traditional chronic disease risk factors, Albert et al. found no independent effect of income on cardiovascular events, including death. In contrast, education had a protective effect after controlling for traditional and novel risk factors. The present results corroborate and extend these findings to all-cause mortality in a population with a much lower annual household income and education level. Moreover, when controlling for education, the higher income groups in the present cohort showed an increase in the mortality risk. Thus, these results suggest the existence of a 'pauper-rich paradox'; the effects of education and income on all-cause mortality risk in this cohort of relatively impoverished Chilean adults seem to operate in opposite directions.

These findings are in direct contrast with previous studies conducted in developed societies. For example, Backlund et al., in a longitudinal study carried out in a US population,³⁴ Table 4 – Changes in β -coefficients in single linear terms related to education and income in their association with mortality.

| | Edu | Education | | come | |
|--|-------|-----------|-------|---------|--|
| | β | P-value | β | P-value | |
| Unadjusted | -1.36 | 0.001 | -0.36 | 0.001 | |
| Age and gender | -0.50 | 0.001 | -0.01 | 0.863 | |
| Education and/or income ^a | -0.49 | 0.001 | 0.04 | 0.298 | |
| Behavioural and biological | -0.36 | 0.002 | 0.03 | 0.282 | |
| risk factors ^a | | | | | |
| All factors ^a | -0.38 | 0.006 | 0.07 | 0.06 | |
| P-values refer to education and income β -coefficients | | | | | |

Risk factors: smoking status, alcohol use (only heavy drinkers), arterial hypertension, diabetes mellitus, body mass index, total cholesterol, low-density lipoprotein, high-density lipoprotein, triglycerides, and family history of death by cardiovascular disease. a All models controlled for age (linear, quadratic and >65 years) and gender.

concluded that the influence of income on mortality was more important at the lower income levels. There may be several possible explanations why the 'pauper-rich groups' in a middle-income developing country such as Chile fare slightly worse in all-cause mortality outcomes. One such factor could be the Chilean socio-economic structure. In spite of its sustained development over the past two decades, Chile has one of the most unequal income distributions in the world.²⁰ In addition, the correlation between education and income is much weaker in the present study than the correlation found in developed countries. Therefore, individuals with a better income do not necessarily have a good education, and reciprocally, individuals with a better education do not necessarily have a higher income; these individuals are found mainly in the 'middle class' Chilean social stratum.²¹ It is hypothesized that the unfulfilled lifestyle expectations of those at the higher end of a still low absolute income (i.e. the 'pauper-rich') have a detrimental effect on the adult individual, manifested in psychological and emotional factors such as stress, frustration or depression. Higher levels of inflammatory markers (e.g. C-reactive protein, interleukin-6, monocyte chemo-attractant protein-1, intercellular adhesion molecule, etc.) and neuroendocrine-related abnormalities (e.g. cortisol) have been suggested to play a mediating role in their association with psychosocial factors, which provides a biological plausibility to this hypothesis.^{40–44}

Wilkinson and Pickett,⁴⁵ in their review of the relation of income distribution with health and other social outcomes in 24 countries that included societies as diverse as Denmark, Japan, Portugal, Singapore and the USA, suggested that '....problems linked to relative deprivation are also associated with income inequality but are not associated with absolute levels of income as such'. Thus, in the generally low absolute income of the present cohort, relative deprivation may have a higher impact on the higher income groups ('the pauper rich'). Furthermore, in developing countries where income is expectedly lower than in developed societies, education may play a more decisive role in determining better health outcomes and survival. However, the notion that income inequality is a robust factor for determining health and life expectancy has been questioned by other researchers.⁴⁶ In



Fig. 3 – Diagram illustrating possible causal pathways to explain the relationship between education, income, chronic disease risk factors and mortality risk.

a systematic review of 40 studies assessing the relationship between childhood SEP and adult cardiovascular diseases, it has been found that childhood life circumstances may be an important factor predicting cardiovascular disease risk in adults.47 Thus, education inequalities in Chile may be reflecting early exposure to adverse life circumstances (unhealthy nutrition, infectious diseases, biological stressors and psychosocial factors) that act partially as 'programming' the future risk of morbidity and mortality. Finally, when the authors carried out their previous analyses,²¹ the 'pauperrich' concept had not been developed, principally because education and income had not been evaluated separately. The concept began to gestate in 2007 after the authors' previous publication had been written; the present article is the first to suggest the possibility of a 'pauper-rich' paradox in a Latin American country such as Chile.

Limitations

Several limitations in this study should be taken into account. Although San Francisco de Mostazal is similar in demographic composition to the rest of the country, this study was conducted in a small Chilean community that does not necessarily represent the country as a whole. For example, the very rich Chilean social group (a very small group indeed) was obviously absent from the SFP cohort. In order to address the representativeness of the sample, mixed sample weights were used based on census data to compensate for differences between the sample and the population, allowing for scaling of risk estimators to the population.⁴⁸ Another issue is the wide age range used in the SFP cohort and the strong correlation with education years. It is conceivable that cohorts of varying age could relate differently to socio-economic indicators. For example, older individuals in the SFP cohort had a lower education level; a plausible explanation for why age showed a stronger impact on this study. Education has improved in Chile in the last four decades because of legislation enacted in 1965 providing children with free and obligatory schooling equivalent to 8 years, recently extended to 12 years. Therefore, the authors previously suggested the importance of making more robust adjustments for age in order to provide more solid evidence for the protective role of education.²¹ The current analyses were adjusted for age, incorporating it as a continuous variable, squaring it, and accounting for age >65 years. Consequently, keeping the effect of age and other variables constant in the Cox regression models, the protective effect of education was found to persist. Moreover, the effects of education and other chronic disease risk factors on all-cause mortality in this Chilean cohort are consistent with findings of most cohorts worldwide.^{2,6–10,15,16,30} Another limitation is that the chronic disease risk factors were only measured at baseline, and thus do not represent exposure over a lifetime. In addition, psychological risk factors and emotional stress, the impact of which on all-cause and cardiovascular mortality has been reported previously,⁴ were not measured in this phase of the study. Finally, because of the small number of deaths, only all-cause mortality was considered as the outcome variable in this study. Nevertheless, cardiovascular mortality and cancer were by far the predominant causes of death (nearly 60%) in this adult cohort.

Conclusion

In this prospective cohort study conducted in a middleincome Latin American country, the protective effect of education was not related to income, and it was not completely explained by the effects of adverse biological and behavioural chronic disease risk factors detected in adult life and consistently associated with all-cause mortality. Nevertheless, it is important to remember that no epidemiological study logically contributes more than what is contained in its design.⁴⁹ Thus, the unique finding that income may have a different impact on survival in a developing country, such as Chile, compared with wealthier and more egalitarian societies opens the field for further research on the causal pathways and global significance of SEP measures.

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Ethical approval

The San Francisco Project Cohort Study was approved by an institutional board of the Faculty of Medicine, University of Chile.

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Competing interests

None declared.

Appendix. Supplementary data

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.puhe.2009.11.008

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