Published by Oxford University Press on behalf of the International Epidemiological Association
© The Author 2005; all rights reserved. Advance Access publication 3 March 2005

International Journal of Epidemiology 2005;**34**:630–637
doi:10.1093/ije/dyi043

Explaining socioeconomic inequality in mortality among South Koreans: an examination of multiple pathways in a nationally representative longitudinal study

Young-Ho Khang[1]* and Hye Ryun Kim[2]

Accepted 19 January 2005

Background South Korea has a different cause-specific structure of mortality compared with North America and northern European countries where studies on pathways to socioeconomic mortality inequalities have been performed. We examined the ability of multiple pathways to explain socioeconomic differentials in all-cause mortality in South Korea.

Methods The 1998 National Health and Nutrition Survey data of South Korea were linked to data on mortality. The socioeconomic position (SEP) indicator was household income. Twelve variables represented biological risk factors (body mass index, systolic blood pressure, cholesterol, and glucose), health behaviours (smoking, alcohol consumption, and regular exercise), psychosocial factors (feelings of sadness and depression, perceived level of stress, and marital status), and early life exposures (education and adulthood height).

Results Mortality differentials by income level did not decrease after exclusion of subjects with severe chronic illness or functional limitation. Biological risk factors, health behaviours, and psychosocial factors caused minor reductions in relative risk for income levels. The ability of early life exposures to explain socioeconomic differentials in mortality was greater than that of biological risk factors, health behaviours, and psychosocial factors.

Conclusions The contribution of multiple pathways to socioeconomic differentials in all-cause mortality may vary in place with the different cause-specific structure of mortality. Future studies with specific pathway variables and specific disease outcomes would provide better understanding of causal mechanisms between SEP and health.

Keywords Socioeconomic position, South Korea, mortality

Since the publication of the Black Report,[1] research efforts to explain the inverse relationship between socioeconomic position (SEP) and all-cause mortality have been common in the West. Studies have explored the role of various pathways: health behaviours,[2–6] material conditions,[5] psychosocial attributes,[3,6] and early life exposures.[7,8] Recently, investigations into socioeconomic inequality in mortality have increased in South Korea. Several longitudinal studies confirmed that socioeconomic differences in mortality were not due to artefact (numerator/ denominator bias).[9,10] In addition, the contributions of biological risk factors and health behaviours,[9] area level material deprivation,[11] and childhood socioeconomic status were examined.[10] However, most prior studies exhibited some limitations. For example, in some studies populations were confined to a limited occupation or urban area.[9,10] In other studies longitudinal data were not used.[11] Due to a paucity of information these prior studies could not examine the role of a wide range of variables.[9–11]

The extent to which pathways explain the association between SEP and mortality has important implications for health and social policy. South Korea has demonstrated a different cause-specific

[1] Department of Preventive Medicine, University of Ulsan College of Medicine, Seoul, Korea.

[2] Korea Institute for Health and Social Affairs, Seoul, Korea.

* Corresponding author. Department of Preventive Medicine, University of Ulsan College of Medicine, 388-1 Pungnap-Dong Songpa-Gu, Seoul, 138-736 Korea. E-mail: youngk@amc.seoul.kr

structure of mortality than populations from North American and northern European countries where studies on pathways to socioeconomic mortality inequalities have been performed. Therefore, exploring the role of various pathways may be salient for the South Korean population. Unlike western countries, mortality from coronary heart disease (CHD) accounts for only 4–6% of all-cause mortality in South Korea.[12] Meanwhile, stroke, stomach cancer, and liver disease, which share early life exposures as important elements of their etiology,[8,13–15] account for about 30% of all deaths.[12] This pattern remains true among males, females, adults, and the elderly.[12] Therefore, in South Korea, it is expected that early life exposure measures would demonstrate a greater ability to explain socioeconomic inequalities in all-cause mortality than risk factors closely related to CHD. However, the relative contribution of each of these pathway variables remains to be examined. The aim of this study was to examine the ability of different pathway variables to explain socioeconomic inequalities in mortality using representative longitudinal data of South Korea.

Methods

Study subjects

This study was approved by the Institutional Review Board of the Asan Medical Center, Seoul, South Korea. Data were obtained from the 1998 National Health and Nutrition Examination Survey (NHANES) conducted by the Korea Institute for Health and Social Affairs. Information was collected from a stratified multistage probability sample of South Korean households representing the civilian, non-institutionalized population. Additional details regarding study design and methods are provided elsewhere.[16] The survey was divided into four parts: (i) health interview survey, (ii) health examination survey, (iii) health behaviour survey, and (iv) nutrition survey. The health examination surveys contained unique 13-digit personal identification numbers (PINs) which were linked to data on mortality from the National Statistical Office (NSO) of Korea. The response rate of the health examination survey for adults aged 30+ was 86.5% (n = 6468). A total of 1.4% (n = 93) did not have a 13-digit PIN and 3.0% (193) did not participate in the health interview or health behaviour survey. The validity of PINs was checked with a validation programme, by which 11.5% (745) were ascertained to be invalid. Of the remaining 5437 respondents 242 died through December 2003.

SEP and outcome variables

The SEP indicator for this study was annual household income. Household income was measured as combined income from all sources of the respondent and his or her family members and divided into three categories: ⩾$20 000; $10 000–$19 999; $0–$9999 (1 US$ = 1200 Korean won). The highest income group was the reference. More refined categories of income or equivalent income [i.e. household income ÷ (household size)$^{0.5}$] showed similar results. The outcome variable for this study was all-cause mortality. Date of death was obtained from NSO death certificate data.

Pathway variables

The following variables were chosen because of their potential or known association with mortality risk.

Biological risk factors

Body mass index (BMI) was calculated by dividing body weight by height squared (kg/m^2) and grouped into four categories: <18.5, 18.5–24.9, 25.0–29.9, and ⩾30.0. Blood pressure (mm Hg) was measured according to American Heart Association guidelines[17] and calculated as the mean of two successive readings. Blood pressure measurements were also grouped into three categories: <140, 140–159, and ⩾160. Serum total cholesterol and glucose levels were measured after an overnight fast, using an autoanalyser (Hitachi 747; Daiichi, Tokyo, Japan). Levels of total cholesterol (mg/dl) were grouped into categories of <200, 200–239, and ⩾240. Serum glucose (mg/dl) was grouped into categories of <110, 110–124, and ⩾125.

Health behaviours

The health behaviour variables were based on self-reported information from respondents. For this analysis, cigarette smoking was classified as 'never smoked', 'former smoker', 'irregular smoker', 'daily smoker smoking <20 cigarettes per day', and 'daily smoker smoking ⩾20 cigarettes per day'. Alcohol consumption was measured by questions on the current status of drinking, the number of days during the past month that the respondent drank, the average amount consumed per day. Quartiles of alcohol consumption were determined for drinkers and classified into six categories: 'non-drinkers', 'former drinker', and quartiles of alcoholic consumption. The lowest quartile was the reference. Exercise behaviour was measured by asking whether respondents exercised regularly in the past month. A 'no' response was the reference.

Psychosocial factors

The psychosocial factors measured were also based on self-reported information from respondents. Subjects were asked to report the frequency of feelings of sadness and depression during the past year. Responses were grouped into four categories: 'none', 'rare', 'often', and 'always'. Perceived level of stress was measured with a single question and response choice of 'nearly none', 'low', 'high', and 'very high'. Because it has been used as an important measure for social support and networks in prior research,[18] marital status was considered a psychosocial factor for this study. Marital status was categorized as married, never married, and divorced or separated.

Early life exposure

As in prior studies[19–23] education and adulthood height were used as proxies for early life exposure. Levels of education, determined by the highest level attained, were categorized as no formal education, elementary, middle, high school, and college. Adulthood height, measured as a component of the health examination survey, was categorized separately for males and females and grouped into three levels at the nearest tertile points.

Baseline health status

Baseline health status should be taken into account when pathways to socioeconomic health inequality are examined.[4,5,21] Subjects with severe illness may have changed their behaviours and probably affect the role of behavioural factors on socioeconomic health inequality.[5] The health selection hypothesis states when people become unhealthy they tend to move into a lower socioeconomic status.[24] This explanation of socioeconomic health inequalities can be partly examined by

excluding subjects with identifiable disease at the beginning of the study[21] or adjusting for baseline health status. In this study, two variables were used to measure baseline health status; (i) the number of severe chronic illnesses reported, and (ii) level of functional limitation. Using a checklist of 37 chronic conditions, respondents were asked whether they had any chronic illness during the past year. Respondents with severe chronic diseases (cancer, diabetes, stroke, heart disease, chronic obstructive pulmonary disease, chronic liver disease including liver cirrhosis, and chronic renal disease) were identified and the number of illnesses was recorded. The level of functional limitation was reported as no limitation, slight limitation, limitation in some major activities, and limitation in all major activities.

Analysis

All variables were coded as dummy variables. Cox proportional hazard models were used to estimate relative risks and their 95% confidence intervals (CIs) of mortality by income level and by single pathway variables, adjusted for confounders. Age- and gender-standardized percentages of respondents in each of the 12 pathway variables by income level were calculated with the direct method. The total number of subjects in this study ($n = 5437$) was the reference. Confidence intervals of age- and gender-standardized percentages were estimated. Three models were created to assess the contribution of each pathway variable on income-related mortality differentials: (i) model I adjusted for age (age and age square), gender, degree of urbanization (metropolis, small- and medium-sized city, and rural county), and number of family members ($n = 5437$); (ii) model II adjusted for the covariates in model I and two baseline health status variables ($n = 5437$); (iii) model III adjusted for the covariates in model I, in a sample without severe chronic illness or limitation in activities ($n = 4173$). The number of deaths in Model III was 98. The contribution of pathway variables was determined by the percentage reduction of excess risk for income level due to the inclusion of specific pathway variables to each of these three models. All statistical analyses were performed with SAS statistical software,[25] with a P-value of 0.05 considered statistically significant.

Results

Of 242 deaths, cancer and cardiovascular deaths accounted for 27.7% ($n = 67$) and 23.6% ($n = 57$), respectively. Liver cancer ($n = 15$), lung cancer ($n = 14$) and stomach cancer ($n = 11$) were the 3 leading causes of cancer deaths. Deaths from stroke ($n = 33$) accounted for 58% of cardiovascular deaths while CHD deaths were only 8. External causes (e.g. transport accidents, suicide) and respiratory diseases accounted for 10.3% ($n = 25$) and 9.3% ($n = 23$) of all deaths, respectively. Deaths from liver disease (e.g. liver cirrhosis) were 15 (6.2% of all deaths) while each of infectious diseases, diabetes, and mental disorders accounted for $<4\%$ of all deaths. Ill defined causes of death (senility, R54) were also found ($n = 18$, 7.4%).

Results in Table 1 show a graded increase in mortality risk according to household income level after adjustment for confounders (age, gender, degree of urbanization, and number of family members). As there was no evidence of a difference in the association between income and mortality when comparing the sexes (interaction test $P = 0.13$), data on men and women were combined. With the exception of serum total cholesterol, biological risk factors were shown to be significantly associated with mortality risk when adjusted for age, gender, and degree of urbanization. Increasing patterns in mortality were observed as systolic blood pressure and serum glucose levels increased. All three health behaviours (cigarette smoking, alcohol intake, and regular exercise) were significantly associated with mortality risk. Higher relative risks of mortality were detected in the highest levels of sadness/depression and stress as compared with the lowest levels. Subjects who never married or divorced/separated had greater mortality risks than those currently married. Differentials in mortality based on education level were observed with adjustment for age, gender, and degree of urbanization. Short stature was associated with higher mortality risk.

Table 2 presents age- and gender-standardized percentage of pathway variables according to income level. Biological risk factors generally showed no differences in prevalence by income. The only exception was a BMI <18.5 kg/m^2, which was more common in the lowest income level. Smoking and regular exercise were patterned by income level. The percentage of daily smokers who smoked $\geqslant 20$ cigarettes per day was significantly higher in the lowest income group. The percentage of respondents who participated in regular exercise showed a graded pattern by income level, with those in the highest income group demonstrating higher levels of exercise. However, alcohol consumption showed no differences in standardized percentage by income. When examining psychosocial factors, respondents in the low income category reported a poor level of psychosocial status. The age- and gender-standardized percentage of respondents who reported feelings of sadness and depression as 'always' was over twice as high in the lowest income group than in the highest (15.4 vs 7.4%). Levels of stress rated at 'very high' were more common in the lowest income group. In addition, subjects in the lowest income group also showed a greater percentage of 'never married' and 'divorced or separated' as compared with the highest income group. Education attainment and adulthood height were also positively associated with income. Lower educational groups and lower height tertiles were more common in lower income groups and vice versa.

Table 3 presents the relative risks of mortality by household income level in three models. Mortality differentials by income level did not decrease after exclusion of subjects with severe chronic illness or functional limitation. Four categories of pathway variables were added to the base models separately. In all three models, biological risk factors, health behaviours, psychosocial factors caused a minor reduction in relative risk for income level. The largest reduction in relative risk for income level was made by measures for early life exposures in all three models. When education and adulthood height were added to the three models, the resulting average percentage changes in relative risk were 13.7% (model I), 14.6% (model II), and 12.2% (model III).

Discussion

Results of this study show that biological risk factors and health behaviours, which include major established CHD risk factors, made small contributions to the reduction of excess mortality risks for subjects in the low income groups. The result was anticipated because age- and gender-standardized percentages

Table 1 Number and percentage of study subjects, number and percentage of deaths, and relative risk of all-cause mortality according to income and 12 pathway variables: The 1998 National Health and Nutrition Survey of South Korea, 1998–2003 (n = 5437)

	No. of subjects (%)	No. of deaths	Relative risk[a] (95% CI)
SEP			
Annual household income (USD)			
⩾20 000	1367 (25.1)	25	1.00 (reference)
10 000–19 999	2066 (38.0)	63	1.81 (1.13–2.89)
<10 000	2004 (36.9)	154	2.33 (1.45–3.75)
Biological risk factors			
BMI (kg/m^2)			
<18.5	229 (4.2)	36	2.46 (1.50–4.03)
18.5–24.9	3638 (66.9)	170	1.73 (1.19–2.52)
25.0–29.9	1433 (26.4)	33	1.00 (reference)
⩾30	137 (2.5)	3	1.39 (0.43–4.55)
Systolic blood pressure (mm Hg)			
<140	4224 (77.7)	123	1.00 (reference)
140–159	787 (14.5)	66	1.56 (1.14–2.12)
⩾160	426 (7.8)	53	1.74 (1.24–2.44)
Total cholesterol (mg/dl)			
<200	3388 (62.3)	150	1.00 (reference)
200–239	1517 (27.9)	60	0.78 (0.57–1.05)
⩾240	532 (9.8)	32	1.22 (0.82–1.80)
Glucose (mg/dl)			
<110	4241 (78.0)	146	1.00 (reference)
110–124	620 (11.4)	46	1.52 (1.09–2.12)
⩾125	576 (10.6)	50	1.92 (1.39–2.65)
Health behaviours			
Smoking			
Never smoker	3050 (56.1)	74	1.00 (reference)
Former smoker	557 (10.2)	50	1.82 (1.19–2.79)
Irregular smoker	104 (1.9)	6	2.15 (0.92–5.04)
Daily smoker, <20 cigarettes	735 (13.5)	52	1.89 (1.25–2.84)
Daily smoker, ⩾20 cigarettes	991 (18.2)	60	2.28 (1.50–3.45)
Alcohol consumption			
Non-drinker	1737 (31.9)	83	1.89 (1.06–3.37)
Stop drinking	312 (5.7)	56	4.30 (2.37–7.82)
First (lowest)	940 (17.3)	14	1.00 (reference)
Second	734 (13.5)	10	0.87 (0.39–1.97)
Third	852 (15.7)	32	1.84 (0.98–3.49)
Fourth (highest)	862 (15.9)	47	2.53 (1.37–4.67)
Regular exercise			
No	4409 (81.1)	213	1.00 (reference)
Yes	1028 (18.9)	29	0.61 (0.41–0.90)
Psychosocial factors			
Feelings of sadness and depression			
None	695 (12.8)	29	1.00 (reference)
Rare	1323 (24.3)	44	1.05 (0.66–1.68)
Often	2796 (51.4)	111	1.34 (0.89–2.02)
Always	623 (11.5)	58	2.39 (1.52–3.74)
Perceived level of stress			
Nearly none	992 (18.2)	80	1.00 (reference)
Low	2450 (45.1)	70	0.87 (0.62–1.22)
High	1620 (29.8)	62	1.11 (0.78–1.57)
Very high	375 (6.9)	30	2.24 (1.45–3.46)
Marital status			
Married	4575 (84.1)	150	1.00 (reference)
Never married	153 (2.8)	10	6.31 (3.13–12.72)
Divorced or separated	709 (13.0)	82	1.77 (1.23–2.53)
Early life exposure measures			
Education			
College	830 (15.3)	12	1.00 (reference)
High	1709 (31.4)	34	1.40 (0.72–2.71)
Middle	911 (16.8)	29	1.60 (0.80–3.19)
Elementary	1268 (23.3)	76	1.89 (0.97–3.67)
No formal education	719 (13.2)	91	2.15 (1.06–4.37)
Height tertile			
High	1816 (33.4)	31	1.00 (reference)
Middle	1800 (33.1)	75	1.55 (1.01–2.38)
Low	1821 (33.5)	136	1.40 (0.92–2.14)

[a] Relative risks of income level were estimated after adjustment for age, gender, degree of urbanization, and number of members in the family. Relative risks of pathway variables were estimated after adjustment for age, gender, and degree of urbanization.

of respondents in each category were not patterned by income level (Table 2). Only the worst categories of smoking behaviour and regular exercise showed significant differences according to income. In western developed countries low SEP was generally associated with poor cardiovascular risk factor profiles.[26] However, the relationship between SEP and risk factors changes in countries with different stages of economic development.[27–30] High serum cholesterol level and obesity were associated with high SEP or urban residence in less-developed countries.[27,31–33] Higher employment grades were associated with higher BMI in male Japanese workers.[34] The relationship between SEP and health behaviours such as smoking also varied with countries.[35] However, it should not be concluded that SEP had a neutral or no effect on biological risk factors in South Korea. Considering the rapid economic development that has occurred in South Korea since the 1960s, age-specific analyses would be more informative because different age cohorts can be assumed to have experienced different socioeconomic conditions over their lifetimes.[36] For instance, in this study the level of BMI among

Table 2 Age- and gender-standardized percentage of pathway variables according to income level: The 1998 National Health and Nutrition Survey of South Korea (n = 5437)

	Annual household income level (US$)		
	⩾20 000 (95% CI)	10 000–19 999 (95% CI)	<10 000 (95% CI)
Biological risk factors			
BMI (kg/m^2)			
<18.5	2.7 (1.7–3.7)	4.1 (3.0–5.2)	5.2 (4.1–6.2)
18.5–24.9	65.0 (60.4–69.7)	65.5 (61.8–69.2)	67.8 (63.7–71.9)
25.0–29.9	30.0 (26.7–33.2)	27.3 (24.9–29.7)	24.7 (22.2–27.2)
⩾30	2.1 (1.3–3.0)	3.1 (2.2–3.9)	2.3 (1.5–3.0)
Systolic blood pressure (mm Hg)			
<140	78.6 (73.7–83.5)	77.5 (73.6–81.4)	77.0 (72.5–81.5)
140–159	14.7 (12.1–17.4)	14.0 (12.1–15.8)	15.0 (13.3–16.8)
⩾160	6.5 (4.8–8.2)	8.5 (6.7–10.2)	7.9 (6.8–9.0)
Total cholesterol (mg/dl)			
<200	62.2 (57.6–66.7)	62.2 (58.6–65.8)	62.9 (58.8–66.9)
200–239	26.8 (23.8–29.8)	28.9 (26.3–31.5)	27.3 (24.8–29.8)
⩾240	10.8 (8.8–12.8)	8.9 (7.5–10.3)	9.7 (8.3–11.2)
Glucose (mg/dl)			
<110	76.9 (71.9–81.9)	78.5 (74.4–82.6)	76.3 (72.0–80.7)
110–124	11.3 (9.2–13.4)	11.0 (9.4–12.6)	12.6 (10.9–14.3)
⩾125	11.6 (9.5–13.7)	10.5 (8.9–12.1)	11.0 (9.4–12.6)
Health behaviours			
Smoking			
Never smoker	58.0 (53.6–62.4)	56.6 (53.1–60.2)	54.2 (50.6–57.8)
Former smoker	11.7 (9.5–13.9)	10.1 (8.6–11.7)	10.0 (8.4–11.5)
Irregular smoker	1.4 (0.9–2.0)	2.2 (1.6–2.9)	1.2 (0.7–1.7)
Daily smoker, <20 cigarettes	13.6 (11.5–15.8)	13.4 (11.6–15.1)	13.8 (11.9–15.6)
Daily smoker, ⩾20 cigarettes	15.1 (13.0–17.2)	17.6 (15.8–19.4)	20.8 (18.5–23.2)
Alcohol consumption			
Non-drinker	31.9 (28.4–35.4)	33.0 (30.1–35.9)	31.8 (29.2–34.4)
Stop drinking	4.7 (3.1–6.3)	4.7 (3.6–5.7)	7.4 (6.1–8.6)
First (lowest)	17.2 (15.0–19.4)	19.0 (17.1–21.0)	14.8 (12.8–16.8)
Second	14.4 (12.4–16.5)	14.1 (12.5–15.8)	11.9 (10.1–13.8)
Third	16.7 (14.4–18.9)	14.0 (12.4–15.6)	15.5 (13.5–17.5)
Fourth (highest)	14.8 (12.6–17.1)	15.1 (13.4–16.8)	18.6 (16.3–20.9)
Regular exercise			
Yes	24.6 (21.7–27.6)	19.1 (17.1–21.0)	14.6 (12.7–16.5)
Psychosocial factors			
Feelings of sadness and depression			
None	12.0 (9.8–14.1)	14.0 (12.2–15.8)	12.2 (10.5–13.9)
Rare	27.3 (24.3–30.2)	24.9 (22.6–27.3)	20.7 (18.5–23.0)
Often	53.2 (49.0–57.4)	52.0 (48.7–55.3)	51.6 (48.0–55.3)
Always	7.4 (5.7–9.1)	9.0 (7.6–10.5)	15.4 (13.6–17.1)
Perceived level of stress			
Nearly none	16.8 (14.3–19.4)	18.5 (16.3–20.8)	18.6 (16.6–20.5)
Low	48.3 (44.3–52.3)	48.0 (44.9–51.1)	39.9 (36.7–43.2)
High	29.0 (25.9–32.1)	27.7 (25.3–30.1)	32.9 (30.0–35.7)
Very high	5.6 (4.3–6.9)	5.8 (4.7–6.9)	8.6 (7.2–10.0)

Table 2 continued

	Annual household income level (US$)		
	⩾20 000 (95% CI)	10 000–19 999 (95% CI)	<10 000 (95% CI)
Marital status			
Married	86.7 (81.5–91.9)	84.6 (80.5–88.7)	77.8 (73.3–82.2)
Never married	1.9 (1.3–2.6)	2.1 (1.5–2.7)	5.6 (4.2–7.0)
Divorced or separated	11.2 (8.7–13.7)	13.3 (11.1–15.4)	16.6 (14.8–18.3)
Early life exposure measures			
Education			
College	28.6 (25.8–31.3)	12.7 (11.2–14.2)	4.5 (3.3–5.7)
High	33.1 (30.0–36.2)	35.5 (33.0–38.0)	27.5 (24.5–30.5)
Middle	13.2 (11.1–15.2)	19.1 (17.1–21.0)	20.4 (18.0–22.7)
Elementary	16.0 (13.2–18.8)	22.8 (20.4–25.1)	31.1 (28.5–33.6)
No formal education	9.0 (6.8–11.3)	9.9 (7.9–11.8)	16.5 (15.1–18.0)
Height tertile			
High	39.1 (35.8–42.5)	32.2 (29.8–34.6)	30.0 (27.0–33.0)
Middle	30.8 (27.5–34.0)	34.8 (32.1–37.5)	33.9 (31.0–36.8)
Low	29.9 (26.4–33.5)	33.0 (30.0–35.9)	36.0 (33.4–38.7)

Table 3 Effect of adjustment for pathway variables on the relationship between income and all-cause mortality: relative risks (RR) and their 95% confidence intervals (CIs) for income levels and percentage change in relative risk. The 1998 National Health and Nutrition Survey of South Korea, 1998–2003

	RR (95% CI) for annual house hold income level (US$)			Percentage change in relative risk[a]		
	⩾20 000	10 000–19 000	<10 000	10 000–19 999 (A)	<10 000 (B)	(A + B)/2
Model I[b]	1.00	1.81 (1.13–2.89)	2.33 (1.45–3.75)			
Model I + biological risk factors	1.00	1.88 (1.17–3.01)	2.24 (1.40–3.60)	−8.6	6.8	−0.9
Model I + health behaviours	1.00	1.82 (1.14–2.92)	2.08 (1.29–3.35)	−1.2	18.8	8.8
Model I + psychosocial factors	1.00	1.76 (1.10–2.82)	2.11 (1.30–3.40)	6.2	16.5	11.4
Model I + early life exposures	1.00	1.71 (1.06–2.74)	2.13 (1.31–3.47)	12.3	15.0	13.7
Model II[c]	1.00	1.78 (1.11–2.85)	1.98 (1.21–3.22)			
Model II + biological risk factors	1.00	1.79 (1.11–2.87)	1.83 (1.12–2.98)	−1.3	15.3	7.0
Model II + health behaviours	1.00	1.76 (1.09–2.83)	1.84 (1.13–3.00)	2.6	14.3	8.4
Model II + psychosocial factors	1.00	1.74 (1.08–2.78)	1.87 (1.15–3.06)	5.1	11.2	8.2
Model II + early life exposures	1.00	1.68 (1.04–2.71)	1.82 (1.11–3.00)	12.8	16.3	14.6
Model III[d]	1.00	1.98 (0.98–3.98)	3.19 (1.57–6.47)			
Model III + biological risk factors	1.00	2.01 (1.00–4.07)	3.07 (1.52–6.22)	−3.1	5.5	1.2
Model III + health behaviours	1.00	2.04 (1.01–4.14)	3.05 (1.49–6.22)	−6.1	6.4	0.1
Model III + psychosocial factors	1.00	1.92 (0.95–3.86)	3.07 (1.51–6.26)	6.1	5.5	5.8
Model III + early life exposures	1.00	1.87 (0.92–3.80)	2.90 (1.39–6.03)	11.2	13.2	12.2

[a] Percentage change in relative risk was calculated by [(RR in baseline model) − (RR in model adjusted for pathway variables)]/[(RR in baseline model) − 1]. Baseline model refers to model I, II, and III.

[b] Adjusted for age (5-year age groups), gender, degree of urbanization (metropolis, small-sized and medium-sized city, and rural county), and number of family member (n = 5437).

[c] Adjusted for the covariates in model I and two baseline health status variables (n = 5437).

[d] Adjusted for the covariates in model I, in a sample without severe chronic illness or functional limitation (n = 4173).

participants aged 30–39 was negatively associated with income level (P for trend = 0.03), while participants aged 60+ showed a positive association between income and BMI (P for trend = 0.002). In addition, higher income was associated with lower systolic blood pressure among the 30–39 age group (P for trend < 0.001) while no association was found among those aged 60+ (P for trend = 0.57). These changing or even reversing patterns in cardiovascular risk factors among age cohorts are probably attributable to a westernization of lifestyle which has accompanied South Korea's rapid economic

development. Therefore, additional age-specific analyses on the role of pathway variables in this study may be warranted. These analyses were not possible, however, due to the small numbers of deaths in this study. Future larger longitudinal studies should include age-specific analysis.

Direct comparison of these results with prior reports in western countries is problematic because of differences in age distribution, SEP indicators used, and adjustment for confounders. In this study, there was no meaningful reduction of excess risk in all three models by inclusion of these pathway variables. Nevertheless, we may conclude that this study showed less explanatory power for biological risk factors and health behaviours on socioeconomic mortality differentials than western studies. Among Finnish men, a sizeable part of excess all-cause mortality risk among unskilled blue collar workers was associated with their unfavourable health behaviour.[2] The excess risk of all-cause mortality for the lowest income quintile was reduced 58 and 35% by adjustment for 14 biological risk factors and 3 behavioural risk factors (smoking, alcohol drinking, and physical activity) in Kuopio, Finland.[3] In the US, 12–13% of excess mortality risk for lower income groups was explained by 4 health behaviours: smoking, alcohol drinking, physical activities, and BMI.[4] These four variables caused about 50% reduction of excess all-cause mortality risk for lower educational groups in a Dutch longitudinal study.[5] One-third of the excess all-cause mortality was explained by smoking, blood pressure, cholesterol and glucose measured at the initial examination of the first Whitehall study in England.[37] Among Scottish men and women, smoking explained around 40% of the excess all-cause mortality.[6]

Interest in psychosocial mechanisms in socioeconomic health inequality has increased during the past two decades,[22,38,39] but has also received criticism.[40,41] In this study, any meaningful reduction of excess mortality risk was not achieved by adjustment for psychosocial factors in the three different models.

In life course perspectives, education can be seen as a measure of childhood socioeconomic circumstances while occupation and income are used as adulthood markers.[19,20,42,43] Adulthood height, which is suggested to reflect childhood socioeconomic conditions as well as genetic factors,[21] was inversely associated with all-cause mortality and cause-specific mortality such as stroke and stomach cancer in South Korea.[23] Of multiple pathway variables, these indicators had the most important effect on the reduction of excess mortality risk although the size of the effect was not very large. Use of more direct measures for early life exposure,[15,19,20] which were not available in the 1998 NHANES data of South Korea, might have resulted in greater percentage changes in relative risk for lower income groups.

The reason why the ability of early life exposures to explain socioeconomic differentials in all-cause mortality was greater than that of biological risk factors, health behaviours, and psychosocial factors may lie in the cause-specific structure of mortality in the South Korean population. Galobardes *et al*. suggested that the relative contribution of early life socioeconomic disadvantage will depend on cause structure of deaths, which differs across countries and time periods.[15] South Korea, like several southern European countries, has a low rate of CHD. According to our analysis on causes of death for 242 decedents, only 8 (3.3%) died from CHD while 73 (30.2%) died from stroke, stomach cancer, liver cancer, and liver disease. This cause structure of deaths may have limited the role of well established CHD risk factors to explain socioeconomic mortality inequality while measures for early life exposure demonstrated a relatively greater explanatory ability of the pathway variables. Future studies with specific pathway variables and specific disease outcome would provide better understanding of causal mechanisms between SEP and health.

In this study, mortality differentials did not decrease significantly when those with severe chronic illness or functional limitation at baseline were excluded or the baseline health status was adjusted. This suggests that health selection associated with illness was not a major determinant.

This study has limitations regarding measurement of pathway and outcome variables. Although the precision of biological risk factors in this study would be high because measurements were made with well-designed quality control,[16] these factors were measured at a single point and thus do not represent exposure over a lifetime. Health behaviours and psychosocial factors were based on self-reported information from respondents. Psychosocial factors were rather simple and imprecise compared with prior studies.[3,22,44] In addition, only all-cause mortality was considered as the outcome variable due to small number of deaths.

Despite these limitations, this study contributes to the growing literature on socioeconomic health inequalities in Asian countries where mechanisms to these inequalities have been rarely explored. This study showed that the role of pathways to socioeconomic inequality in all-cause mortality may vary in place with different cause-specific structure of mortality. Future studies need to examine the contribution of specific pathway variables to specific disease outcomes.

KEY MESSAGES

- South Korea has demonstrated a different cause-specific structure of mortality compared with populations from North American and northern European countries where studies on pathways to socioeconomic mortality inequalities have been performed.
- This study showed that biological risk factors, health behaviours, and psychosocial factors made minor contributions to the reduction of excess mortality risks for low income groups. The ability of early life exposures to explain socioeconomic differentials in mortality was greater than that of biological risk factors, health behaviours, and psychosocial factors.
- The relative contribution of a pathway to explain socioeconomic health inequality may well depend on cause structure of deaths, which differs across countries.

References

[1] Townsend P, Davidson N. *Inequalities in Health. The Black Report*. Harmondsworth, Middlesex, England: Penguin Books, 1982.

[2] Pekkanen J, Tuomilehto J, Uutela A, Vartiainen E, Nissinen A. Social class, health behaviour, and mortality among men and women in eastern Finland. *BMJ* 1995;**311:**589–93.

[3] Lynch JW, Kaplan GA, Cohen RD, Tuomilehto J, Salonen JT. Do cardiovascular risk factors explain the relation between socioeconomic status, risk of all-cause mortality, cardiovascular mortality, and acute myocardial infarction? *Am J Epidemiol* 1996;**144:**934–42.

[4] Lantz PM, House JS, Lepkowski JM, Williams DR, Mero RP, Chen J. Socioeconomic factors, health behaviors, and mortality: results from a nationally representative prospective study of US adults. *JAMA* 1998;**279:**1703–08.

[5] Schrijvers CTM, Stronks K, van de Mheen HD, Mackenbach JP. Explaining educational differences in mortality: the role of behavioral and material factors. *Am J Public Health* 1999;**89:**535–40.

[6] Woodward M, Oliphant J, Lowe G, Tunstall-Pedoe H. Contribution of contemporaneous risk factors to social inequality in coronary heart disease and all causes mortality. *Prev Med* 2003;**36:**561–68.

[7] Davey Smith G, Hart C, Blane D, Gillis C, Hawthorne V. Lifetime socioeconomic position and mortality: prospective observational study. *BMJ* 1997;**314:**547–52.

[8] Davey Smith G, Hart C, Blane D, Hole D. Adverse socioeconomic conditions in childhood and cause specific adult mortality: prospective observational study. *BMJ* 1998;**316:**1631–35.

[9] Song YM, Byeon JJ. Excess mortality from avoidable and non-avoidable causes in men of low socioeconomic status: a prospective study in Korea. *J Epidemiol Community Health* 2000;**54:**166–72.

[10] Khang YH. *Socioeconomic Mortality Inequalities in Korea: Mortality Follow-up of Korea Labor and Income Panel Study*. Seoul: Korea Labor Institute, 2003, pp. 1–32.

[11] Son M. The relationships of occupational class, educational level and deprivation with mortality in Korea. *Korean J Prev Med* 2002;**35:**76–82.

[12] National Statistical Office of Korea. *Annual Report on the Cause of Death Statistics*. Daejun: National Statistical Office of Korea, 2001.

[13] Leon DA, Davey Smith G. Infant mortality, stomach cancer, stroke, and coronary heart disease: ecological analysis. *BMJ* 2000;**320:**1705–06.

[14] Leon D. Common threads: underlying components of inequalities in mortality between and within countries. In: Leon D, Walt G (eds). *Poverty, Inequality and Health: An International Perspective*. New York: Oxford University Press, 2001, pp. 58–87.

[15] Galobardes B, Lynch JW, Davey Smith G. Childhood socioeconomic circumstances and cause-specific mortality in adulthood: Systematic review and interpretation. *Epidemiol Rev* 2004;**26:**7–21.

[16] Ministry of Health and Welfare of Korea. *Interim Report on the 1999 National Health and Nutrition Survey*. Kwachon: Ministry of Health and Welfare of Korea, 1999.

[17] Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. The sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med* 1997;**157:**2413–46.

[18] Berkman LF, Glass T. Social integration, social networks, social support, and health. In: Berkman LF, Kawachi I (eds). *Social Epidemiology*. New York: Oxford University Press, 2000, pp. 137–73.

[19] Lynch JW, Kaplan GA, Salonen JT. Why do poor people behave poorly? Variation in adult health behaviours and psychosocial characteristics by stages of the socioeconomic lifecourse. *Soc Sci Med* 1997;**44:**809–19.

[20] Davey Smith G, Hart C, Hole D *et al*. Education and occupational social class: which is the more important indicator of mortality risk? *J Epidemiol Community Health* 1998;**52:**153–60.

[21] Davey Smith G, Shipley MJ, Rose G. The magnitude and causes of socio-economic differentials in mortality: further evidence from the Whitehall study. *J Epidemiol Community Health* 1990;**44:**265–70.

[22] Marmot M, Bosma H, Hemingway H, Brunner E, Stansfeld S. Contribution of job control and other risk factors to social variations in coronary heart disease incidence. *Lancet* 1997;**350:**235–39.

[23] Song YM, Davey Smith G, Sung J. Adult height and cause-specific mortality: a large prospective study of South Korean men. *Am J Epidemiol* 2003;**158:**479–85.

[24] Illsley R. Occupational class selection, and the production of inequalities in health. *Q J Soc Aff* 1986;**2:**151–65.

[25] Allison PD. *Survival Analysis Using the SAS System: A Practical Guide*. Cary, NC: SAS Institute Inc., 2000.

[26] Kaplan GA, Keil J. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation* 1993;**88:**1973–98.

[27] INCLEN Multicentre Collaborative Group. Socio-economic status and risk factors for cardiovascular disease: a multicentre collaborative study in the International Clinical Epidemiology Network (INCLEN). *J Clin Epidemiol* 1994;**47:**1401–09.

[28] Chang CL, Marmot MG, Farley TMM, Poulter NR. The influence of economic development on the association between education and the risk of acute myocardial infarction and stroke. *J Clin Epidemiol* 2002;**55:**741–47.

[29] Aboderin I, Kalache A, Ben-Shlomo Y *et al*. *Life Course Perspectives on Coronary Heart Disease, Stroke and Diabetes: Key Issues and Implications for Policy and Research*. Geneva: World Health Organization, 2002.

[30] Kim S, Symons M, Popkin BM. Contrasting socioeconomic profiles related to healthier lifestyles in China and the United States. *Am J Epidemiol* 2004;**159:**184–91.

[31] Forrester TE, Wilks RJ, Bennett FI *et al*. Fetal growth and cardiovascular risk factors in Jamaican schoolchildren. *BMJ* 1996;**312:**156–60.

[32] Perova NV, Davis CE, Tao S *et al*. Multi-country comparison of plasma lipid relationship to years of schooling in men and women. *Int J Epidemiol* 2001;**30:**371–79.

[33] Bavdekar A, Yajnik CS, Fall CHD *et al*. Insulin resistance syndrome in 8-year-old Indian children. *Diabetes* 1999;**48:**2422–29.

[34] Martikainen P, Ishizaki M, Marmot MG, Nakagawa H, Kagamimori S. Socioeconomic differences in behavioural and biological risk factors: a comparison of a Japanese and an English cohort of employed men. *Int J Epidemiol* 2001;**30:**833–38.

[35] Cavelaars AEJM, Kunst AE, Geurts JJM *et al*. Educational differences in smoking: international comparison. *BMJ* 2000;**320:**1102–07.

[36] Khang YH, Lynch JW, Kaplan GA. Health inequalities in Korea: age- and sex-specific educational differences in the 10 leading causes of death. *Int J Epidemiol* 2004;**33:**299–308.

[37] van Rossum CTM, Shipley MJ, van de Mheen H, Grobbee DE, Marmot MG. Employment grade differences in cause specific mortality. A 25 year follow up of civil servants from the first Whitehall study. *J Epidemiol Community Health* 2000;**54:**178–84.

[38] Adler NE, Boyce T, Chesney MA *et al*. Socioeconomic status and health: the challenge of the gradient. *Am Psychol* 1994;**49:**15–24.

[39] Macintyre S. The Black Report and beyond: what are the issues? *Soc Sci Med* 1997;**44:**723–45.

[40] Davey Smith G, Harding S. Is control at work the key to socioeconomic gradients in mortality? *Lancet* 1997;**350:**1369–70.

[41] Macleod J, Davey Smith G, Heslop P, Metcalfe C, Carroll D, Hart C. Psychological stress and cardiovascular disease: empirical demonstration of bias in a prospective observational study on Scottish men. *BMJ* 2002;**324:**1247–53.

[42] Davey Smith G. Socioeconomic factors and determinants of mortality [Letter]. *JAMA* 1998;**280:**1744.

[43] Lynch JW, Kaplan GA. Socioeconomic position. In: Berkman LF, Kawachi I (eds). *Social Epidemiology*. New York: Oxford University Press, 2000, pp. 13–35.

[44] Lynch JW, Krause N, Kaplan GA, Tuomilehto J, Salonen JT. Workplace conditions, socioeconomic status, and the risk of mortality and acute myocardial infarction: The Kuopio Ischemic Heart Disease Risk Factor Study. *Am J Public Health* 1997;**87:**617–22.