INTRODUCTION

There is convincing evidence that exposures acting across the life course influence adult health outcomes (1–3). Life-course epidemiology examines a range of potential processes through which exposures acting at different stages of life can, singly or in combination, influence disease risk (table 1) (4). In the critical period model, an exposure acting at a specific time has long-lasting effects on the structure or function of the body. The fetal origins hypothesis, in its original formulation, took this approach (5). Other examples of processes where outcomes appear to depend upon the time window during which an exposure acts are limb development (in relation to maternal thalidomide use); infection with hepatitis B and risk of adulthood liver cancer (with very early postnatal infection being most implicated); and environmental lead exposure, which results in serious neurodevelopmental deficits only if occurring in infancy and childhood (3). However, the influence of exposures acting during critical periods of susceptibility may be modified by later life exposures. This is the case for the associations of birth weight with coronary heart disease, high blood pressure, and insulin resistance, where associations are stronger (or only evident) among those who become obese during adolescence or adulthood (6–8).

In contrast, the accumulation of risk model suggests that effects accumulate over the life course, although it also allows for developmental periods during which susceptibility may be greater (4). Health damage may increase with the duration and/or number of detrimental exposures. This has been shown in relation to exposure to poor socioeconomic conditions, where additive effects of experiencing low socioeconomic position across different stages of the life course influence the risk of several diseases (9, 10). Accumulation of risk can also be due to clustering of exposures. For example, children from a lower socioeconomic background are more likely to be of low birth weight, to have poorer diets, to be more exposed to passive smoking and some infectious agents, and to have fewer educational opportunities (4). Exposures may also form chains of risk and thus tend to occur together. For example, low educational attainment will increase the probability that a person works in an occupation with a high risk of toxic exposures and lower income (3).

Coronary heart disease is a good example of a disease that develops throughout the life course. Although coronary heart disease manifests itself in adulthood, atherosclerosis, an important underlying process leading to the disease, begins at a much earlier age. Fatty streaks in the artery walls are found in children (11, 12), and arterial lesions are evident in young men suffering violent deaths (13–15). Moreover, the presence of risk factors during childhood or adolescence is associated with an increased risk of developing coronary heart disease (16). For example, serum cholesterol in early adult life was strongly associated with coronary heart disease in the Johns Hopkins Precursors Study (17). The magnitude of this association was greater than that reported with respect to adulthood cholesterol measures. Recent studies have suggested that unfavorable preadulthood measures of cholesterol, blood pressure, and adiposity are associated with increased intimal-medial thickness, a presymptomatic indicator of coronary heart disease (18, 19).

Socioeconomic circumstances at different stages of the life course influence specific adulthood health outcomes through the different pathways outlined in table 1. While associations of adulthood socioeconomic position with a
wide range of specific health outcomes have been described extensively (20–25), relatively little investigation of how early life social circumstances influence adult health has been conducted (26). This paper therefore presents a systematic review of epidemiologic evidence on the association between preadulthood socioeconomic position and cause-specific mortality.

MATERIALS AND METHODS

We performed a systematic review of individual-level studies examining childhood socioeconomic circumstances and adult overall and cause-specific mortality. For the electronic search, childhood socioeconomic circumstances were defined in general and via specific indicators such as father/mother/parental education or occupation, housing conditions, overcrowding, number of siblings, maternal marital status, illegitimacy, and residence in an orphanage or similar facility. Full details on the search strategy are available from the authors. The electronic search was supplemented by the authors’ files. We excluded those publications that reported exclusively on infant/childhood mortality or adolescent suicide. However, studies that presented all-age mortality were included. In these populations, most deaths are likely to have occurred in adulthood, except for those due to external causes. We also excluded studies that grouped nonfatal and fatal events to ensure comparability between the outcomes.

We have not performed formal meta-analysis as the necessary conditions of comparability of exposures and outcomes, together with homogeneity of association direction and strength (27), are not—and perhaps could not be expected to be—met.

RESULTS

We located 29 studies of childhood socioeconomic position and all-cause or cause-specific mortality, two based on different subgroups of the Longitudinal Study in the United Kingdom and two based on different groups of the Harvard Alumni Study. A full description of these studies can be found in Web table 1. (This information is described in the first of five supplementary tables; each is referred to as “Web table” in the text and is posted on the Journal’s website (http://epirev.oupjournals.org/).) All were prospective or cohort designs with the exception of two case-control studies (28, 29) and one cross-sectional survey of reported sibling mortality (30). The populations included in these studies were from the United Kingdom, Sweden, Finland, Norway, Denmark, the Netherlands, the United States, and Russia. The oldest cohorts were born at the beginning of the last century, with only three studies including people born before that (28, 31, 32). The majority included people born during or before the 1940s and 1950s, and the youngest birth cohorts dated from the late-1950s to the 1960s in the United Kingdom (33), Sweden (34), Finland (35, 36), the Netherlands (37), and the United States (38). Thus, with the exception of the youngest cohorts, most participants lived their childhood, or were born, at the beginning of the century and lived through the Second World War at some point in their lives. Sixteen studies included samples representative of the general population. Nineteen of the 29 studies measured the participants’ socioeconomic circumstances during childhood or young adulthood. The remainder obtained data on childhood circumstances by participant recall during adulthood. The father’s occupation was the most common indicator of childhood socioeconomic position. Other indicators used were crowding, housing characteristics (either as a score of several items or evaluating each item separately), home ownership, parental education, number of siblings, farm size, indicators of the mother’s marital status, and presence of both natural parents during childhood. Most studies reported participants’ own occupation (current or longest) and deprivation indices as measures of adult socioeconomic position. Education was also assessed in some studies, but this was treated mainly as a measure of adult socioeconomic position despite that fact that, for most people, education is completed early in life and will also capture aspects of childhood circumstances (39).

Overall mortality

Eighteen of 22 studies found a higher risk of all-cause mortality associated with worse childhood socioeconomic circumstances (Web table 2) (29–31, 33, 37, 38, 40–51). For example, in the Collaborative Study and in the Boyd Orr cohort, men whose fathers had manual occupations or were unemployed had higher risk of death (40, 43). A similar effect was also found in the Boyd Orr cohort with other childhood circumstances, such as crowding (trend \( p = 0.02 \)), lack of tap water (trend \( p = 0.04 \)), and poor ventilation (trend \( p < 0.01 \)) (52). In a younger population, the 1946 birth cohort, men and women whose fathers had manual occupations had, respectively, 50 percent and 150 percent elevated risk of death (45). In northern European cohorts, similar magnitudes of effects were generally reported. Having had a father with a manual occupation resulted in 36 percent higher risk in Sweden (47), 42 percent higher risk in the Netherlands (37), 41 percent higher risk in Denmark (49), and 46 percent higher risk in a younger cohort of men in Finland (41) (T. H. Pensola, Department of Sociology, Population Research Unit, University of Helsinki, personal communication, 2003). Women in the Alameda County Study in the United States who experienced low socioeconomic position during childhood had an increased mortality risk (hazard ratio = 1.20, 95 percent confidence interval (CI):

<table>
<thead>
<tr>
<th>TABLE 1. Conceptual life-course models according to Ben-Shlomo and Kuh (4)</th>
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<tbody>
<tr>
<td>Critical period model</td>
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<tr>
<td>With or without later life risk factors</td>
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<tr>
<td>With later life effect modifiers</td>
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<tr>
<td>Accumulation of risk</td>
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<tr>
<td>With independent and uncorrelated insults</td>
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<td>With correlated insults</td>
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<tr>
<td>“Risk clustering”</td>
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<td>“Chains of risk” with additive or trigger effects</td>
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...
found a greater risk of cardiovascular disease mortality with less favorable childhood social circumstances (31, 36, 48). Living in a family with a higher number of siblings was not associated with increased risk of cardiovascular disease mortality in two studies (36, 54).

Generally, childhood circumstances remained associated with cardiovascular disease mortality after accounting for adult socioeconomic circumstances and/or adult risk factors. However, in the cohort of young Finnish men, the effect of father’s social class on cardiovascular disease mortality was explained largely by adult occupation (35) (T. H. Pensola, Department of Sociology, Population Research Unit, University of Helsinki, personal communication, 2003).

Coronary heart disease mortality

Coronary heart disease (myocardial infarction or ischemic heart disease) was related to childhood circumstances in seven (40, 43, 47, 50, 52, 56–60) of 10 studies (table 3). Children with parents working in manual occupations had a higher risk of coronary heart disease into adult life, independent of their adult socioeconomic characteristics. Adjustment for adult socioeconomic position decreased the strength of the associations in some (40, 57) but not all (43, 58) studies. Men born to unmarried mothers had a higher risk of coronary heart disease, but only among those who themselves never married, in a Swedish study (59).

Most studies indexed childhood circumstances with father’s social class. The housing conditions experienced during childhood in Norway were related to adult coronary heart disease (56). Having more siblings was associated with higher risk of coronary heart disease in the Collaborative Study, although this association was explained by adult behaviors and risk factors (57). On the other hand, in the Harvard and Pennsylvania Alumni Cohort, cases of myocardial infarction were more likely to have had no siblings (61). However, the selection of controls was based on the alumni records of those presumed to be alive, so this could reflect death from other causes being associated with number of siblings. Moreover, the spectrum of father’s social class was limited in this sample of advantaged university students. In the Helsinki Hospital cohort, increased crowding was not related to mortality from coronary heart disease (62).

In summary, the results of these studies suggest that socioeconomic circumstances during both childhood and adulthood contribute to adult coronary heart disease mortality. Mother’s marital status may be a marker for an additional dimension of deprivation than that determined by low socioeconomic position.

Stroke

Four of six studies reported a higher risk of overall stroke among those who experienced less favorable childhood socioeconomic circumstances (table 4). In the Boyd Orr and Oslo cohorts, socioeconomic circumstances were indexed during childhood (not through adulthood recall), and the effect of childhood circumstances on overall stroke remained unchanged after accounting for adult socioeco-
### TABLE 2. Results of the studies relating childhood socioeconomic circumstances and cardiovascular disease mortality

<table>
<thead>
<tr>
<th>Study, author(s), and reference no.</th>
<th>Unadjusted or age adjusted</th>
<th>Plus adjusted for adult socioeconomic position</th>
<th>Plus adjusted for risk factors</th>
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<tbody>
<tr>
<td>Collaborative Study, Heslop et al. (10)</td>
<td>Women—hazard ratio (cardiovascular disease) of father's social class: manual vs. nonmanual = 1.56 (95% CI: 0.76, 3.20)</td>
<td></td>
<td>Women—adjusted for diastolic blood pressure, cholesterol, body mass index, forced expiratory volume in 1 second, smoking, cigarette consumption, exercise, alcohol, age; hazard ratio (cardiovascular disease) of father's social class: manual vs. nonmanual = 1.57 (95% CI: 0.76, 3.23)</td>
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<td>Glasgow Alumni Cohort, Davey Smith et al. (42)</td>
<td>Men—relative risk (cardiovascular disease) of father's social class: I (referent), II = 1.51 (95% CI: 1.08, 2.11), III = 1.63 (95% CI: 1.17, 2.27), IV = 1.85 (95% CI: 1.12, 3.07), V = 2.36 (95% CI: 1.11, 4.99); trend p = 0.002</td>
<td>Men—adjusted for systolic blood pressure, smoking; relative risk (cardiovascular disease) of father's social class: I (referent), II = 1.46 (95% CI: 1.05, 2.05), III = 1.66 (95% CI: 1.19, 2.32), IV = 1.91 (95% CI: 1.15, 3.17), V = 2.31 (95% CI: 1.09, 4.89); trend p = 0.001</td>
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<td>Kuopio Study, Lynch et al. (53)</td>
<td>Relative risk (cardiovascular disease) of adulthood-childhood socioeconomic position: high-high (referent), high-med = 0.78 (95% CI: 0.32, 1.92), high-low = 0.99 (95% CI: 0.39, 2.51), low-high = 1.59 (95% CI: 0.52, 4.88), low-med = 2.26 (95% CI: 1.02, 4.99), low-low = 2.02 (95% CI: 0.90, 4.54); trend p = 0.001</td>
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<td>Cohort from Finland; Pensola and Martikainen (35, 36), (T. H. Pensola, University of Helsinki, personal communication, 2003)</td>
<td>Men—relative risk (cardiovascular disease) of parental class: manual vs. nonmanual = 1.98 (95% CI: 1.45, 2.69)</td>
<td>Men—adjusted for own social class; relative risk (cardiovascular disease) of parental class: manual vs. nonmanual = 1.44 (95% CI: 1.04, 1.99)</td>
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<td>Oslo Mortality Study, Claussen et al. (48)</td>
<td>Women—mortality rate (cardiovascular disease)</td>
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<td>Of parental class: upper nonmanual = 5.2, lower nonmanual = 11.4, skilled manual = 12.7, unskilled manual = 11.7, farmer/employer = 8.5, other = 32.0; p &lt; 0.05; IRD* = 21.1%</td>
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<td>Of family type: two parents = 10.1, single parent = 16.0; p &lt; 0.05; IRD = 12.5%</td>
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<td></td>
<td>Of no. of siblings: none = 10.9, 1 = 9.0, ≥2 = 11.6; IRD = 8.8%</td>
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<td>Men—relative index of inequality (cardiovascular disease) of childhood housing score = 2.79 (95% CI: 1.71, 4.55)</td>
<td>Men—adjusted for household income in adult life; relative index of inequality (cardiovascular disease) of childhood housing score = 2.68 (95% CI: 1.64, 4.38)</td>
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<td></td>
<td>Women—relative index of inequality (cardiovascular disease) of childhood housing score = 3.96 (95% CI: 1.52, 10.3)</td>
<td>Women—adjusted for household income in adult life; relative index of inequality (cardiovascular disease) of childhood housing score = 3.80 (95% CI: 1.45, 9.96)</td>
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<tr>
<td>Project Metropolit, Osler et al. (49)</td>
<td>Adjusted for birth weight, intelligence quotient in childhood; hazard ratio (cardiovascular disease) of father's occupation; high/ middle class (referent), working class = 1.54 (95% CI: 0.91, 2.64), unknown = 3.64 (95% CI: 1.75, 7.53)</td>
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</table>
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This suggests that exposures related to poor socioeconomic circumstances during childhood might be stronger determinants of stroke than of coronary heart disease.

Likewise, mortality risk from all stroke was higher among men whose fathers had manual occupations in the Collaborative Study, but not among those who had more siblings (40, 57). However, the age-adjusted relative risk of hospital admission and death from hemorrhagic stroke was 2.84 (95 percent CI: 1.12, 7.20) for men whose fathers had manual occupations, increasing to 3.22 (95 percent CI: 1.15, 9.03) after adjusting for adult socioeconomic position and risk factors (57). For ischemic stroke, the respective relative risks were 1.25 (95 percent CI: 0.77, 2.03) and 0.92 (95 percent CI: 0.53, 1.61) after adjustment for adult circumstances and risk factors (57). The hazard ratio for stroke, combining deaths and hospital admissions, in the Helsinki study was significantly higher with increased crowding in the home ($p = 0.02$) (63). Further adjustment for height did not change the association of stroke with crowding (63). The Nurses’ Health Study (55) did not find an association of adulthood recalls of childhood social circumstances with stroke mortality.

Rheumatic heart disease

Crowding and the number of persons per bedroom were not associated with higher mortality risk due to rheumatic heart disease in one study, although the authors acknowledged the lack of power this study had to find an association (44).

Overall cancer mortality

There was little evidence that children whose fathers were from lower social classes (10, 35, 42) (T. H. Pensola, Department of Sociology, Population Research Unit, University of Helsinki, personal communication, 2003) or who had more siblings (54) had higher risk of overall cancer mortality (Web table 3). The exception was the Collaborative Study, although this effect was removed by adjustment for adult socioeconomic circumstances (9). Given that total cancer mortality includes an amalgam of diseases resulting from very different etiologic processes, this result is perhaps not surprising.

Lung and smoking-related cancer mortality

In a study from Norway, there was an increased risk of death due to lung cancer among men who were exposed to worse housing conditions during childhood, independent of adult socioeconomic circumstances (56). Similar results were found in the Collaborative Study (40, 57) and the Boyd Orr cohort (52), although in these cases the association was explained largely by adult socioeconomic position. There was no association of childhood circumstances with a group of other smoking-related cancers in the Collaborative Study (40).
### TABLE 3. Results of the studies relating childhood socioeconomic circumstances and coronary heart disease mortality

<table>
<thead>
<tr>
<th>Study, author(s), and reference no.</th>
<th>Unadjusted or age adjusted</th>
<th>Plus adjusted for adult socioeconomic position</th>
<th>Plus adjusted for risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Collaborative Study, Davey Smith et al. (40)</strong></td>
<td>Men—relative risk (coronary heart disease) of father's social class: manual vs. nonmanual = 1.52 (95% CI: 1.24, 1.87); trend p = 0.0003</td>
<td>Men—adjusted for adult social class, deprivation, car, age; relative risk (coronary heart disease) of father's social class: manual vs. nonmanual = 1.28 (95% CI: 1.03, 1.61); trend p = 0.11</td>
<td>Men—adjusted for adult social class, deprivation, car, smoking, diastolic blood pressure, cholesterol, body mass index, forced expiratory volume in 1 second, age; relative risk (coronary heart disease) of father's social class: manual vs. nonmanual = 1.26 (95% CI: 1.01, 1.58); trend p = 0.12</td>
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<tr>
<td><strong>Collaborative Study, Hart and Davey Smith (57)</strong></td>
<td>Men—relative risk (coronary heart disease) of no. of siblings: none = 1.18 (95% CI: 0.91, 1.55), 1–2 (referent), 3–4 = 1.22 (95% CI: 1.03, 1.48), 5–6 = 1.52 (95% CI: 1.06, 2.16), 7+ = 1.31 (95% CI: 0.91, 1.86); relative risk per sibling = 1.04 (95% CI: 1.01, 1.06); trend p = 0.0055</td>
<td>Men—adjusted for social class, father's social class, car user, deprivation, education; relative risk (coronary heart disease) of no. of siblings: none = 1.21 (95% CI: 0.93, 1.58), 1–2 (referent), 3–4 = 1.11 (95% CI: 0.92, 1.33), 5–6 = 1.29 (95% CI: 1.06, 1.57), 7+ = 1.08 (95% CI: 0.86, 1.34); relative risk per sibling = 1.01 (95% CI: 0.98, 1.03); trend p = 0.66</td>
<td>Men—adjusted for social class, father's social class, car user, deprivation, education, bronchitis, height, adjusted forced expiratory volume in 1 second, systolic blood pressure, cholesterol, alcohol; relative risk (coronary heart disease) of no. of siblings: none = 1.20 (95% CI: 0.92, 1.57), 1–2 (referent), 3–4 = 1.11 (95% CI: 0.92, 1.32), 5–6 = 1.29 (95% CI: 1.05, 1.56), 7+ = 1.08 (95% CI: 0.87, 1.35); relative risk per sibling = 1.01 (95% CI: 0.98, 1.03); trend p = 0.58</td>
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<tr>
<td><strong>Boyd Orr cohort, Frankel et al. (43)</strong></td>
<td>Hazard ratio (coronary heart disease) of father's social class: I–II = 0.41, III (referent), IV = 0.88, V = 1.14, unemployed = 1.04; trend p = 0.12</td>
<td>Adjusted for Townsend deprivation score, age; hazard ratio (coronary heart disease) of father's social class: I–II = 0.43 (95% CI: 0.19, 0.98), III (referent), IV = 0.89 (95% CI: 0.56, 1.42), V = 1.12 (95% CI: 0.71, 1.76); unemployed = 1.04 (95% CI: 0.68, 1.59); trend p = 0.15</td>
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<tr>
<td><strong>Boyd Orr cohort, Dedman et al. (52)</strong></td>
<td>Hazard ratio (coronary heart disease) of the following childhood household conditions</td>
<td>Adjusted for income, food expenditure, adult Townsend deprivation score, childhood social class; hazard ratio (coronary heart disease) of the following childhood household conditions</td>
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<tr>
<td><strong>Uppsala Birth Cohort Study, Modin (59)</strong></td>
<td>Never married men—adjusted for mother's age, birth year, mother's marital status, social class at birth; relative risk (ischemic heart disease) of the following conditions</td>
<td>Never married men—adjusted for mother's age, birth year, mother's marital status, social class at birth, own social class, income, education; relative risk (ischemic heart disease) of the following conditions</td>
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</table>
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* CI, confidence interval.

Birth social class: higher and mediate nonmanual (referent),
entrepreneurs and farmers = 0.97 (95% CI: 0.65, 1.45),
lower nonmanual = 1.31 (95% CI: 0.84, 2.06), skilled
manual = 1.14 (95% CI: 0.76, 1.70), unskilled manual =
1.17 (95% CI: 0.82, 1.67), housedaughters = 0.76 (95% CI:
0.41, 1.40), not classified = 1.49 (95% CI: 0.90, 2.46); p = 0.233

Birth social class: higher and mediate nonmanual (referent),
entrepreneurs and farmers = 0.73 (95% CI: 0.48, 1.12),
lower nonmanual = 1.06 (95% CI: 0.67, 1.68), skilled
manual = 0.83 (95% CI: 0.54, 1.28), unskilled manual =
0.90 (95% CI: 0.61, 1.32), housedaughters = 0.59 (95% CI:
0.31, 1.11), not classified = 1.29 (95% CI: 0.77, 2.16); p = 0.101

Uppsala Birth Cohort Study,
Vagero and Leon (47)

Odds ratio (ischemic heart disease) of head of household
during childhood: nonmanual (referent), manual = 2.29
(95% CI: 1.51, 3.46), nonemployed = 2.23 (95% CI: 1.08,
4.59)

Adjusted for own social class; odds ratio (ischemic heart
disease) of head of household during childhood: nonmanual (referent), manual = 1.99 (95% CI: 1.30, 3.05),
nonemployed = 1.82 (95% CI: 0.88, 3.77)

Partly based on the East-West
Study, Notkola et al. (58)

Relative risk (coronary death) of parental social class, East
region: farmers (referent), small farmers = 1.44, land-
less = 1.88, craftsman = 0.69, others and missing = 1.17

No differences in the West region

Helsinki Hospital cohort,
Eriksson et al. (62, 63)

Hazard ratio (coronary heart disease) of crowding in
childhood: ≤1.5 (referent), 1.6–2.5 = 1.19, 2.6–3.5 = 1.11,
3.6–4.5 = 1.06, >4.5 = 0.87; trend p = 0.11

Oslo Mortality Study, Naess et
al. (56)

Men—relative index of inequality (coronary heart disease) of
childhood housing score: 2.73 (95% CI: 1.87, 4.00)

Harvard Alumni Cohort,
Gillum and Paffenbarger (60)

Men—relative risk (coronary heart disease) of father with blue
collar occupation = 1.67; p > 0.05

Harvard (and Pennsylvania)
Alumni Cohort, Paffenbarger et al. (61)

Men—proportion of cases vs. controls (coronary heart
disease) for no. of siblings: none = 21.2 vs. 14.8 (p =
0.01), 1 = 22.5 vs. 24.3 (p = 0.55), 2–4 = 48.2 vs. 51.0
(p = 0.42), ≥5 = 8.0 vs. 10.0 (p = 0.33)

Women—adjusted for body mass index, smoking, parental
myocardial infarction before age 60 years, history of
hypertension, cholesterol, diabetes, aspirin, hormone
replacement therapy, past oral contraceptive use, alcohol,
exercise, trans-fatty acids, vitamin E; relative
risk (coronary heart disease) of father's occupation:
white collar (referent), blue collar = 0.94 (95% CI: 0.72,
1.25), farmer = 0.69 (95% CI: 0.41, 1.16)

Nurses' Health Study,
Gliksman et al. (55)

Women—relative risk (coronary heart disease) of father's
occupation: white collar (referent), blue collar = 1.07
(95% CI: 0.82, 1.39), farmer = 0.65 (95% CI: 0.40, 1.04)

Women—adjusted for body mass index, smoking, parental
myocardial infarction before age 60 years, history of
hypertension, cholesterol, diabetes, aspirin, hormone
replacement therapy, past oral contraceptive use, alcohol,
exercise, trans-fatty acids, vitamin E; relative
risk (coronary heart disease) of father's occupation:
white collar (referent), blue collar = 0.94 (95% CI: 0.72,
1.25), farmer = 0.69 (95% CI: 0.41, 1.16)
### TABLE 4. Results of the studies relating childhood socioeconomic circumstances and stroke mortality

<table>
<thead>
<tr>
<th>Study, author(s), and reference no.</th>
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<td><strong>Oslo Mortality Study, Naess et al. (56)</strong></td>
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<tr>
<td><strong>Alameda County Study, Beebe-Dimmer et al. (31)</strong></td>
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<tr>
<td><strong>Nurses’ Health Study, Gilsman et al. (55)</strong></td>
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</tbody>
</table>

- **Men—relative risk (stroke) of father's social class:**
  - **Collaborative Study, Davey Smith et al. (40):**
    - Men—relative risk (stroke) of father's social class: manual vs. nonmanual = 1.83 (95% CI: 1.13, 2.94); trend p = 0.031
  - Men—adjusted for adult socioeconomic position, deprivation, car, age; relative risk (stroke) of father's social class: manual vs. nonmanual = 1.87 (95% CI: 1.12, 3.12); trend p = 0.049
  - Men—adjusted for adult socioeconomic position, deprivation, car, age, smoking, diastolic blood pressure, cholesterol, body mass index, forced expiratory volume in 1 second, age; relative risk (stroke) of father's social class: manual vs. nonmanual = 1.74 (95% CI: 1.05, 2.90); trend p = 0.079

- **Men—relative risk (stroke) for no. of siblings:**
  - **Collaborative Study, Hart and Davey Smith (57):**
    - Men—relative risk (stroke) for no. of siblings: 0 = 0.50 (95% CI: 0.23, 1.08), 1–2 (referent), 3–4 = 1.03 (95% CI: 0.98, 1.09); trend p = 0.20
    - Men—adjusted for adult social class, deprivation, education; relative risk (stroke) for no. of siblings: 0 = 0.50 (95% CI: 0.23, 1.09), 1–2 (referent), 3–4 = 0.93 (95% CI: 0.64, 1.35), 5–6 = 1.01 (95% CI: 0.67, 1.53); trend p = 0.049
    - Men—adjusted for adult social class, deprivation, education, bronchitis, height, adjusted forced expiratory volume in 1 second, systolic blood pressure, cholesterol, alcohol; relative risk (stroke) for no. of siblings: 0 = 0.45 (95% CI: 0.2, 0.98), 1–2 (referent), 3–4 = 0.90 (95% CI: 0.57, 1.42); trend p = 0.079

- **Hazard ratio (stroke) of father's social class:**
  - **Boyd Orr cohort, Frankel et al. (43):**
    - Hazard ratio (stroke): I–II = 0.80, III (referent), IV = 1.53, V = 1.43, unemployed = 2.89; trend p = 0.01
    - Adjusted for Townsend deprivation score, age; hazard ratio (stroke): I–II = 0.83 (95% CI: 0.16, 4.37), III (referent), IV = 1.55 (95% CI: 0.50, 4.77), V = 1.40 (95% CI: 0.46, 4.24), unemployed = 3.0 (95% CI: 1.17, 7.75); trend p = 0.01
  - **Boyd Orr cohort, Dedman et al. (52):**
    - Hazard ratio (stroke): Crowding (persons/room): <1.5 = 0.40 (95% CI: 0.16, 0.99, 2.5) <2.5 (referent), 2.5–<3.5 = 0.59 (95% CI: 0.29, 1.18), ≥3.5 = 0.99 (95% CI: 0.44, 2.25); trend p = 0.53
    - Adjusted for income, food expenditure, adult Townsend deprivation score, childhood social class; hazard ratio (stroke): Crowding (persons/room): <1.5 = 0.38 (95% CI: 0.14, 0.99, 2.5) <2.5 (referent), 2.5–<3.5 = 0.63 (95% CI: 0.31, 1.28), ≥3.5 = 1.07 (95% CI: 0.47, 2.43); trend p = 0.50
  - **Ventilation:**
    - Very good (referent), fair = 1.72 (95% CI: 0.99, 2.99), poor = 1.71 (95% CI: 0.66, 4.40); trend p = 0.08
    - Very good (referent), fair = 1.72 (95% CI: 0.99, 2.98), poor = 1.85 (95% CI: 0.70, 4.86); trend p = 0.08
  - **Cleanliness:**
    - Very good = 1.05 (95% CI: 0.49, 2.23), fair (referent), poor = 0.52 (95% CI: 0.28, 0.95); trend p = 0.07
    - Very good = 1.18 (95% CI: 0.53, 2.62), fair (referent), poor = 0.56 (95% CI: 0.30, 1.05); trend p = 0.08

- **Men—relative index of inequality (stroke) in childhood housing score:**
  - Trend p for crowding = 0.7 (effect size not reported)

- **Women—relative index of inequality (stroke) in childhood housing score:**
  - Trend p for crowding = 0.7 (effect size not reported)

- **Women—cerebrovascular findings were similar to coronary heart disease (effect size not reported separately)**

* CI, confidence interval.
Other cancer mortality

In the Collaborative Study, men whose fathers had manual occupations or those who had more siblings had higher mortality from stomach cancer, independent of adult circumstances (40, 57). The Oslo Mortality Study found higher risk of large-bowel and rectal cancer among those who had the poorest housing conditions during childhood (56), but this was imprecisely estimated. There was no association between childhood socioeconomic characteristics and later death from non-smoking-related cancers in three studies (40, 48, 52), from prostate cancer mortality in Sweden (28), or from malignant melanoma in Norway (56).

In summary, childhood socioeconomic circumstances have a strong influence on stomach cancer and are likely to contribute, along with adult circumstances, to lung cancer through exposure to smoking. No consistent effect was found for other or overall cancers.

Respiratory disease mortality

Two studies from the United Kingdom investigated the associations between respiratory deaths and childhood circumstances (Web table 4). In the Collaborative Study, men whose fathers had manual occupations had higher respiratory-related mortality (relative risk = 2.01, 95 percent CI: 1.17, 3.48) (40). Adult socioeconomic position and risk factors (smoking, body mass index, and score for forced expiratory volume in 1 second) explained part of the association (40). Those with a higher number of siblings suffered more respiratory mortality, but this was explained entirely by adult circumstances (57). Coggon et al. (44) did not find higher mortality with greater crowding or with more persons per room. There is little evidence on respiratory mortality, but it appears that poor childhood circumstances are related to respiratory deaths, at least partly through adult circumstances. Nevertheless, childhood exposures, such as poor intrauterine development, childhood infections, poor childhood growth, indoor and outdoor pollution, housing dampness and mold, passive smoke exposure, and poor nutrition, are socially patterned and may contribute to poor respiratory health in adult life (64).

External causes of death

Accidents and violence. Three of five studies found higher risk of accidental and violent death with worse childhood socioeconomic circumstances (Web table 5) (35, 48, 49) (T. H. Pensola, Department of Sociology, Population Research Unit, University of Helsinki, personal communication, 2003). In the Finnish cohort, men whose fathers had manual occupations during childhood had a higher risk of death from external causes, but this was explained entirely by adult socioeconomic position (35). Lower father’s social class was not associated with risk in women (36). Likewise, the Oslo Mortality Study found that housing conditions during childhood were strongly predictive of future death from external causes among men but not among women (48, 56). The Collaborative Study in the United Kingdom did not find a higher risk of death due to accidents and violence associated with childhood circumstances (40, 57), but the 1946 birth cohort from the United Kingdom reported 70 percent higher risk among those with fathers of low social class during childhood (odds ratio (OR) = 1.7, 95 percent CI: 0.8, 3.6) (65).

Suicide. Only two studies reported separately on associations of childhood socioeconomic position with risk of suicide in adulthood (Web table 5). Although only 11 deaths were due to suicide in the 1946 birth cohort from the United Kingdom, suicide was more likely to occur among those of lower childhood socioeconomic background (OR = 2.8, 95 percent CI: 0.6, 13.1) (65). In the Finnish cohort, men whose fathers worked in manual occupations had a higher risk of suicide death; this was accounted for by adult socioeconomic position (35) (T. H. Pensola, Department of Sociology, Population Research Unit, University of Helsinki, personal communication, 2003). Among women, parental social class was not associated with suicide (36).

Alcohol- and illegal drug-related mortality

There were more alcohol-related deaths among men with fathers from a lower social class in the Finnish cohort (OR = 1.81, 95 percent CI: 1.54, 2.12), but this risk decreased substantially after accounting for adult socioeconomic position (OR = 1.21, 95 percent CI: 1.03, 1.43) (Web table 5) (35) (T. H. Pensola, Department of Sociology, Population Research Unit, University of Helsinki, personal communication, 2003). In the Oslo Mortality Study, housing conditions during childhood were associated with psychiatric deaths (largely due to conditions related to alcohol or drug dependence; International Classification of Diseases, Ninth Revision, codes 290–319) after adjustment for adult household income (48). Similar results were seen for alcohol-related and other psychiatric deaths in this study (56).

Thus, socioeconomic circumstances during childhood played some role in external causes of death in northern European countries, but results in the United Kingdom were inconclusive. Differences in the distribution of particular causes of death and/or proximal risk processes in these birth cohorts may partly explain these different patterns.

DISCUSSION

The evidence from individual-level studies suggests that childhood socioeconomic circumstances contribute to a variety of different causes of death. In particular, childhood conditions appear strongly related to mortality from stomach cancer and hemorrhagic stroke. Childhood circumstances, together with adulthood socioeconomic position, contribute to mortality from coronary heart disease, lung cancer, and respiratory-related diseases. Poorer childhood conditions are not generally associated with mortality from non-smoking-related cancers and prostate cancer, but they may contribute to external and alcohol-related causes of death, especially in northern European countries (table 5).

The findings relating childhood social circumstances to cause-specific mortality are congruent with data from studies relating height and mortality. Adult height is determined early in life, so shorter stature is partly a marker for an
TABLE 5. Key messages

- Childhood socioeconomic circumstances are particularly important for mortality from stomach cancer and hemorrhagic stroke.
- Childhood circumstances contribute, together with socioeconomic conditions in adult life, in determining mortality from coronary heart disease, lung cancer, and respiratory-related deaths. The relative contribution of child-versus-adult circumstances may vary in different contexts.
- Childhood circumstances may contribute to external and alcohol-related causes of death, especially in northern European countries.
- There is no evidence for an association with overall non-smoking-related cancers or prostate cancer.

Unfavorable profile of socially patterned exposures acting during the growing period (from the intrauterine period until final height is achieved). Height is negatively associated with risk of hemorrhagic stroke (66, 67), stomach cancer, coronary heart disease, and chronic obstructive pulmonary disease (67–69), and it is either not associated or positively related to mortality from site-specific cancers, such as breast cancer (70).

The association between childhood social circumstances and mortality probably comes about through a variety of processes, such as those illustrated in the models of life-course influences on adulthood disease outlined by Ben-Shlomo and Kuh (4) and presented in table 1. Infection with Helicobacter pylori during infancy and childhood offers a plausible mechanism to explain the association between poor childhood circumstances and stomach cancer (71) and possibly illustrates a critical period model during early life when individuals are most susceptible to acquiring this infection. Moreover, the similarity of associations of stomach cancer and hemorrhagic stroke with poor childhood circumstances, together with similarities in their association with height (66, 67, 72), and the cross-national relation between hemorrhagic stroke and stomach cancer risk raise, speculatively, the possibility that an early life infection—or a factor related to early life infection such as dehydration due to childhood diarrhea—is associated with hemorrhagic stroke risk.

On the other hand, coronary heart disease, ischemic stroke, lung cancer, and chronic obstructive pulmonary disease appear to be influenced by factors acting across the entire life course and therefore may conform more to a cumulative risk model. For example, poor childhood and adult social circumstances could independently influence lung cancer risk via increased probability of initiation, earlier age of initiation, lower probability of quitting, and higher age of quitting smoking (73, 74). Similarly, an accumulation of adverse exposures over the life course may result in higher risk of coronary heart disease (9).

Evidence for early life socioeconomic effects from ecologic, migrant, and disease trend studies, and nonfatal outcomes

In an effort to “triangulate” the evidence on the potential for early life socioeconomic conditions to influence adult health, it is informative to examine other types of epidemiologic studies in addition to the individual-level studies described above.

Ecologic studies. In these studies, infant and maternal mortalities at some earlier period have been used as markers of socioeconomic conditions and standards of hygiene during childhood. Overall, these report strong effects of childhood circumstances on stomach cancer, stroke, and mortality from respiratory infections including tuberculosis, but associations with coronary heart disease are weaker and inconsistent across studies. In an early example, Forsdahl (75, 76) demonstrated that socioeconomic conditions during childhood might be an important determinant of adult cardiovascular disease. He used infant mortality around the time of birth as an indicator of the prevalent socioeconomic conditions, an approach later applied in other studies (77–80). Leon and Davey Smith (81) correlated cause-specific mortality in 1991–1993 with infant mortality in 1921–1923 across 27 countries, adjusting for current infant mortality as a measure of current socioeconomic conditions. Higher stomach cancer mortality in 1991–1993 was strongly correlated with higher infant mortality in 1921–1923 ($r = 0.83$ for men, $r = 0.82$ for women), and this correlation remained unchanged after adjusting for infant mortality in 1991–1993. Similar results were found for stroke and mortality due to respiratory tuberculosis. The stronger correlation for stroke than for coronary heart disease may reflect the greater influence of childhood deprivation on hemorrhagic stroke than ischemic stroke, with ischemic stroke and coronary heart disease being influenced by similar etiologic factors, and hemorrhagic stroke having some separate determinants. This model is supported also by data on secular trends in stroke subtypes and coronary heart disease, where there have been continuous declines for hemorrhagic stroke since the beginning of the century, whereas ischemic stroke trends mimic those of coronary heart disease (82).

Migration and place of birth studies. Migration studies may suggest susceptibility periods during childhood for some diseases (83–86), because migrants may carry with them the disease risk conferred by their childhoods in the country of origin. For instance, a Swedish register study (85) showed that immigrants retained the higher rates of stomach cancer experienced in their country of origin. Interestingly, women preserved lower breast cancer rates from their country of origin, suggesting that their risk was not entirely reset by the new environment.

These results contrast with those of second-generation immigrants born in Sweden. Among men, their risk of stomach and lung cancers was similar to that of native Swedes, and the second generation of women no longer benefited from a lower risk of breast cancer (86). The role of H. pylori infection during infancy and childhood with respect to stomach cancer in later adulthood is concordant with the findings of migrant studies (including a study demonstrating high rates of stomach cancer among Viet-
namely to the United Kingdom (87)). Results for lung cancer were not consistent, perhaps reflecting that factors acting in both childhood (related to initiation of smoking) and in adult life (related to quitting) are important.

Elford and Ben-Shlomo (83) reviewed migrant studies assessing mortality due to cardiovascular disease. These studies were not consistent, with both childhood and adulthood place of residence apparently contributing to rates of coronary heart disease and stroke for migration within Britain (88), while others reported adult place of residence as the more important risk indicator for coronary heart disease (89). Differences in cardiovascular disease mortality associated with place of birth between Blacks and Whites in the United States have also been reported (90). Indeed, the variation of cardiovascular disease mortality between Blacks born in the North and Blacks born in the South exceeded the interracial differences between Blacks and Whites living in New York City (90). For coronary heart disease in both men and women, Whites born in the Northeast had similar overall coronary heart disease rates as Blacks but, compared with Whites born in the Northeast, Caribbean-born Blacks had lower coronary heart disease rates, while Blacks born in the South had higher rates. In contrast, Blacks had uniformly higher rates of stroke and hypertensive heart disease mortality compared with Northeastern-born Whites, but again the highest rates were generally observed among Blacks born in the South. Both the disadvantage of Blacks born in the South, relative to Blacks born in the Northeast of the United States, and the relative protection against coronary heart disease experienced by Blacks born in the Caribbean may be suggestive of early life influences conditioning disease risk in adulthood (90).

Evidence from long-term disease trends. Studying long-term disease trends can provide evidence for childhood determinants of later adult mortality (3, 64). Evidence of birth cohort effects has been used as an indicator of the importance of conditions in childhood on later mortality risk. In a classic 1934 cohort analysis, Kermack et al. concluded that, “Each generation after the age of 5 years seems to carry along with it the same relative mortality throughout adult life,...” (91, p. 679) and that “...these results are consistent with the hypothesis that the important factor from the point of view of health of the individual during his whole life is his environment up to the age of say 15 years...” (91, p. 683).

The limitations of this type of evidence have been well described and relate to the difficulty in establishing true changes in trends from artifacts and in the inherent ambiguity of differentiating period from cohort effects (92, 93). Nevertheless, long-term trends can make a distinction between diseases for which childhood socioeconomic circumstances can plausibly have played a determining role and those where this is unlikely. Following the massive improvements in socioeconomic circumstances and hygienic conditions occurring throughout the last century in most industrialized countries (94), we would expect these to translate into trends for those diseases where these conditions played a central role. This may be the case for stomach cancer, hemorrhagic stroke, and hypertension. Cohort effects in stomach cancer have been described (95, 96), and these could follow a falling prevalence of H. pylori infection and improvement of hygienic conditions (97). Similarly, secular trends suggest that there was a continuous fall in mortality from hemorrhagic stroke throughout the 20th century (82). Moreover, the ratio of ischemic stroke to hemorrhagic stroke increased fourfold between the 1930s and the 1990s, with most of the increase taking place between the 1930s and the 1970s (82). Decreasing secular trends in systolic and diastolic blood pressure between the end of the 1940s and the 1970s observed in two studies from the United Kingdom (98, 99) and consistent cohort effects observed across birth years from 1890 to 1960 in an analysis of US data (100, 101) suggest preadult influences (102).

Overall adult mortality in the early part of the 20th century included major contributions from tuberculosis, stomach cancer, valvular heart disease (including the sequel of rheumatic heart disease), and hemorrhagic stroke—conditions for which there is strong evidence of early life influences. At the end of the 20th century, mortality was dominated by chronic diseases, such as coronary heart disease, ischemic stroke, and lung, breast, and prostate cancers, for which the contribution of deprivation in childhood is much less clear. Thus, the relative contribution of early life socioeconomic disadvantage will depend on the extant disease environment, which will differ across countries and time periods. There is, thus, no single answer to the question rhetorically posed in the title of an early paper on this issue: “Deprivation in infancy or in adult life: which is more important for mortality risk?” (103).

Specificity of effects of childhood circumstances

It has been noted that socioeconomic factors in adulthood are related to the risk of many different diseases (25). Such a perspective highlights a supposed general association between socioeconomic circumstances and poor health and implicitly or explicitly invokes theories of increased “general susceptibility” of the poor (104). However, this view regarding processes of general susceptibility is challenged by evidence showing that health among the disadvantaged has not always been worse (3, 105), that different patterns of socioeconomic health inequalities can be found in different countries (106), and that the magnitude and direction of the social patterning of specific causes of death are heterogeneous (3).

The early ecologic studies of Barker and Robinson (5) and Barker and Osmond (107), who related maternal, neonatal, and later postneonatal mortality, together with cause-specific infant mortality, to adulthood mortality, take into account specific early life risk processes. The particular exposure indicator was taken to index particular influences, and different relations with adulthood mortality were observed (5, 107). For instance, deaths from stroke were associated with neonatal mortality, and this was interpreted as indicating that exposure to adverse maternal factors that occurred before and during gestation was key. In contrast, deaths from bronchitis, stomach cancer, and rheumatic disease were more closely related to postneonatal mortality, suggesting that exposure to worse living conditions, including risk of infections, during the first years of life was important (5, 107).

This review adds evidence that the contribution of childhood and adult socioeconomic circumstances varies across...
different diseases. Accounting for and interpreting these differing contributions should provide richer insights into the specific mechanisms that link life-course socioeconomic circumstances to different health outcomes than does a “general susceptibility” approach. In addition, the specificity of effects of childhood circumstances argues against residual confounding by adult socioeconomic position as explaining the observed associations, since in this case an increased risk consequent on less favorable childhood socioeconomic circumstances should be observed for all outcomes with strong socioeconomic patterning in adulthood. Our review shows that this is not the case.

Examining different indicators of childhood socioeconomic position sheds light on the mechanisms that may explain their link with health in adulthood. Different markers of early life socioeconomic position may have both general and specific implications. Thus, measures of crowding and number of siblings may specifically reflect poorer hygiene conditions and act as a marker for infectious disease risk, while for other outcomes such as coronary heart disease they may act as a general marker of social disadvantage (57, 63, 108). Poor ventilation associated with overall and lung cancer mortality may be a marker of more deprived households, and/or it may reflect increased exposure to household smoke and fumes (52). Other childhood markers of deprivation, such as having a single mother, suggest that different dimensions of deprivation exist than those captured with more conventional markers of child socioeconomic position and that these can also influence mortality (38, 59).

**Measurement and other issues**

Some studies measured childhood socioeconomic conditions through recall in adulthood. Two studies report a reasonably high agreement in recalling father’s occupation. There was 66 percent exact agreement (and 83 percent exact ± 1 unit of difference in agreement) in father’s social class between the historical records and the participant’s recall 50 years later (109). In the Kaiser Permanente Women Twins Study, 81 percent of the twins agreed in their recall of their father’s occupation (110). Nevertheless, with later life recall, early life measures will generally be more poorly indexed in comparison with measures of adult socioeconomic circumstances, resulting in greater measurement error for the childhood measures. Therefore, statistical adjustment for the two indicators will favor the adulthood indicators. Moreover, part of the association between early life socioeconomic circumstances and adult mortality will act through how childhood factors condition later life social trajectory (4). Even for diseases where adulthood factors entirely determine mortality risk, if these adulthood socioeconomic circumstances are at least in part an outcome of childhood experiences, the latter should also be considered as contributing to the disease outcome.

One approach to the relative influence of childhood and adulthood social circumstances is to examine populations with reasonably homogenous adulthood social circumstances. This is the case with the Glasgow alumni (42), the Harvard alumni (60), and the managers that participated in the study by Christenson and Hinkle (50). In these populations, father’s social class was related to cardiovascular disease risk in later life.

**Conclusion**

This review presents evidence of both direct and indirect contributions of childhood socioeconomic circumstances to mortality risk. Evidence from individual-level, migrant, ecologic, and disease trend studies provides a reasonably consistent picture of the contribution of childhood socioeconomic circumstances in shaping adult disease risk. These effects are particularly strong for stomach cancer and hemorrhagic stroke. Poorer socioeconomic conditions during early and adult life accumulate throughout the life course in their influence on the risk of coronary heart disease and chronic obstructive pulmonary disease. The relative contribution of child and adulthood socioeconomic circumstances in determining the risk of smoking-related conditions and external causes of death varies in different countries, perhaps reflecting context specificities in how early life factors condition later processes that influence risk.

The results presented here can be interpreted as evidence for health impacts of early and later life deprivation. These may reflect historically contingent biologic, behavioral, and developmental processes operating over the life course of individuals born during the 20th century and expressed in outcomes such as stomach cancer, hemorrhagic stroke, and cardiovascular disease. Childhood deprivation in 1950 may not be associated with the same health consequences as childhood deprivation in 2000 in industrialized/rich countries, where most research of life course influences on adult health is currently conducted. Nevertheless, it is disturbing that, in countries like the United States and the United Kingdom, child poverty has increased markedly since the 1960s, so that about 15–20 percent of children in those countries are now in poverty (111). International comparisons show there are social policy choices made about how many children are exposed to deprivation in early life. For instance, market-generated child poverty in Sweden, the United States, and the United Kingdom is 23.4 percent, 26.7 percent, and 36.1 percent, respectively (112). Tax and transfer policies in Sweden reduce this child poverty burden to below 3 percent, while in the United Kingdom it is only reduced to about 19.8 percent and in the United States to 22.4 percent. Rainwater and Smeeding (111, p. 48) showed that children in the United States are worse off, in terms of material goods, than equivalently poor children in 10 of 14 rich countries (111). If early and later life socioeconomic conditions are important for health, then it is worth considering what the population health burden of current policies that expose large numbers of children to poverty will be in the future.

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