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## Socioeconomic Status across the Life Course and All-Cause and Cause-Specific Mortality in Finland

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### Abstract

We used high quality register based data to study the relationship between childhood and adult socio-demographic characteristics and all-cause and cause-specific mortality at ages 35–72 in Finland among cohorts born in 1936–1950. The analyses were based on a 10% sample of households drawn from the 1950 Finnish Census of Population with the follow-up of household members in subsequent censuses and death records beginning from the end of 1970 through the end of 2007. The strengths of these data come from the fact that neither childhood nor adult characteristics are self reported and thus are not subject to recall bias, misreporting and no loss to follow-up after age 35. In addition, the study population includes several families with at least two children enabling us to control for unobserved family characteristics. We documented significant associations between early life social and family conditions on all-cause mortality and cause-specific mortality, with protective effects of higher childhood socio-demographic characteristics varying between 10% and 30%. These associations were mostly mediated through adult educational attainment and occupation, suggesting that the indirect effects of childhood conditions were more important than their direct effects. We further found that adult socioeconomic status was a significant predictor of mortality. The associations between adult characteristics and mortality were robust to controls for observed and unobserved childhood characteristics. The results imply that long-term adverse health consequences of disadvantaged early life social circumstances may be mitigated by investments in educational and employment opportunities in early adulthood.

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## Keywords

childhood socioeconomic status; adult socioeconomic status; mortality; cause-specific mortality; intergenerational; Finland

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## INTRODUCTION

Socioeconomic (SES) inequalities in mortality have been extensively documented (Cutler, Lleras-Muney, & Vogl, 2008; Elo, 2009) but factors that underlie these differentials continue to be debated. Until recently, most studies of SES inequalities in adult mortality focused on the role of adult characteristics. However, as evidence of the associations between childhood conditions and adult health has accumulated, increased attention is now being paid to the contribution of entire life course to adult health inequalities (e.g., Elo & Preston 1992; Haas, 2008; Hayward & Gorman, 2004; Næss, Hoff, Lawlor & Mortensen, 2012; Palloni, Milesi, White & Turner, 2009). This evidence suggests that childhood nutritional status, health, SES, place of residence and other household characteristics contribute to adult disparities in health and mortality (Case, Fertig, & Paxson, 2005; Galobardes, Lynch & Davey Smith, 2004, 2008; Laaksonen, Rahkonen, Martikainen, & Lahelma, 2005; Osler, Andersen, Batty, & Holstein, 2005). Among the mechanisms through which childhood environment is hypothesized to influence adult health include indirect mechanisms operating through attained adult characteristics (e.g., SES and lifestyle factors) and direct effects of childhood health (Preston, Hill, & Drevenstedt, 1998).

In this paper, we examine (1) the associations between childhood family characteristics and all-cause and cause-specific mortality in Finland, (2) whether these associations are indirect operating through attained adult SES, and (3) whether the associations between adult SES and mortality are robust to controls for observed and unobserved childhood characteristics. The data come from a unique register based data that consist of a 10% sample of households drawn from the 1950 Finnish Census of Population with follow-up of household members in subsequent censuses and death records beginning in the end of 1970 through December 31, 2007. This paper contributes to related literatures concerning the role of childhood conditions and adult SES and all-cause and cause-specific mortality in several ways. First, all information on childhood and adult characteristics are drawn from census records and thus are not subject to recall or misreporting bias or loss to follow-up after the beginning of the follow-up at age 35. Second, we are able to follow the oldest members of the study cohorts from early childhood to age 72. Third, we have sufficient sample size to study both all-cause and cause-specific mortality. Finally, we are able to analyze the impact of unobserved family characteristics on adult SES differences in mortality by comparing siblings.

## BACKGROUND

The documentation of SES inequalities in mortality dates back to the 19<sup>th</sup> century and they continue to be the subject of active investigation today (Bengtsson & van Poppel, 2011; Elo, 2009). That these inequalities have persisted and widened in many countries during the 20<sup>th</sup> century in changing disease, economic, and social environments has led some investigators

to call them “fundamental causes” of disease (Link & Phelan, 1995). Higher educational attainment and income as well as higher social position, typically measured by occupation in European studies, all predict better adult health and lower mortality (Cutler et al., 2008). Higher levels of income and wealth may protect against financial stress and give access to health-enhancing resources, including housing and health care (Smith, 2007; Herd, Goesling, & House, 2007; Martikainen, Valkonen, & Moustgaard, 2009). Occupation is related not only to social status but also working conditions that in turn can influence disease onset and accidental injuries (Brand, Warren, Crayon, & Hoonakker, 2007; Marmot, 2004). Education, which is acquired relatively early in life, is not only predictive of income and occupation later in life, but can also influence health in numerous other ways. Education may increase one’s problem solving skills and ability to acquire information and translate this information into health enhancing life styles. Education is also related to motivation and self-control and effective use of medical technologies (Mirowsky & Ross, 2003; Lleras-Muney, 2005; Goldman & Smith, 2002; Smith, 2007).

These SES inequalities in adult health may have antecedents in childhood. Childhood environment can have direct effects on adult health and mortality through exposure to physiological scarring (e.g., childhood infectious diseases, nutritional deprivation, adverse in utero environment) or acquired immunity, and indirect effects through attained adult circumstances (Preston et al., 1998). Evidence further points to differential impact of childhood conditions on various disease processes reflected in differing associations between early life characteristic and cause-specific mortality (Galobardes et al., 2004, 2008). For example, these associations appear to be stronger for heart disease than for overall cancer mortality, but may vary by cancer type (Galobardes et al., 2004; Lawlor et al., 2006; Næss et al., 2012; Strand & Kunst, 2006). Childhood conditions may also play a role in substance abuse (Melchior et al., 2007; Osler, Nordentoft & Andersen, 2006; Yang et al., 2006) and deaths from accidents and violence (Galobardes et al., 2004; Lawlor et al., 2006; Strand & Kunst, 2006; Pensola & Valkonen, 2002; Pensola & Martikainen, 2003).

Several studies have documented significant associations between father’s occupation and/or parental education, housing characteristics, or family income and adult mortality (e.g., Osler, Andersen, Batty, & Holstein, 2005; Strand & Kunst, 2006). In addition, farm residence and living with both parents in childhood or adolescence have been associated with lower mortality during the 20<sup>th</sup> century in the United States (e.g., Hayward & Gorman, 2004; Preston et al. 1998). Similarly, those born out of wedlock in early 20<sup>th</sup> century Sweden experienced higher adult mortality compared to those who were born to married couples (Modin, Koupil & Vågerö, 2009), and Finnish men living in single parent households at ages 10–14 in 1970 experienced higher mortality at ages 30–42 (Pensola & Martikainen, 2003). In most studies, however, controls for adult SES substantially attenuate the associations between childhood SES and adult mortality.

That adult characteristics substantially attenuate associations between childhood conditions and adult mortality is not surprising. Higher levels of parental socioeconomic resources predict both better childhood health and higher levels of education (Case et al., 2005; Haas, 2006). Thus children who grow up in higher SES households enter adulthood in better health and with higher levels of education and thus are themselves likely to achieve higher SES and

reap the health benefits from greater access to human and financial capital throughout their lives. In addition, childhood circumstances may predict health behaviors, such as substance abuse, and thus health-related behaviors are an additional pathway through which early life experiences may influence adult health and mortality (Hayward & Gorman, 2004; van de Mheen, Stronks, Looman, & Mackenbach, 1998; Yang et al. 2006).

In studies that have controlled for both childhood and adult SES, adult SES remains a significant predictor of adult mortality and these associations are typically robust to controls for childhood characteristics (Pensola & Martikainen, 2003). However, questions remain to what extent the estimates of adult SES on mortality remain biased by unobserved childhood characteristics such as childhood health status, parenting practices, environmental exposures and other related unobserved factors (Smith, 2007). In this paper, we not only control for observed childhood characteristics but also factors – both social and genetic - that are shared among siblings and that may bias estimates of adult SES on mortality.

## DATA AND METHODS

Our data consist of 10% sample of households from the 1950 Finnish Census of Population that has been linked to subsequent census records beginning in 1970 through 1995 and to death records through December 31, 2007 using unique personal identifiers by Statistics Finland (Statistics Finland, 1997). We utilized information on the individuals' family of origin from the 1950 census and his/her adult characteristics from the 1970, 1975, 1980 or 1985 censuses, as close to age 35 as possible, when mortality follow-up began for everyone. For 98.7% of the subjects, these characteristics were measured at ages 30–35. Death records provided information on the date and cause of death based on the 8<sup>th</sup>, 9<sup>th</sup> or 10<sup>th</sup> revision of the International Classification of Diseases and Deaths (ICD) with coding of causes made comparable across the ICD revisions.

The 1950 data contain identifiers for families and households making it possible to identify members in the same family. We included individuals aged 0–14 years at the time of the 1950 census in single mother, single father and two parent families. We used unique family identifiers, ages of the parents, and family members' relationship to the household head in linking children to their parents. Of the 116,622 children aged 0–14, we excluded those who were not in the 1970, 1975, 1980 or 1985 censuses because they were known to have died before age 35 after 1970 (n=1,001) or had died or moved out of Finland before 1970 (n=15,065). We further dropped cases for which values for mothers' age was below age 15 or above 49 and for fathers' age below age 15 and above 65 at the child's birth (n=397), and individuals for whom we could not obtain adult characteristics from the censuses between ages 25 and 35 (n=1,770). These individuals were present at some censuses but absent at these ages. Our final sample consisted of 98,389 children ages 0–14 in 1950.

### Childhood Characteristics

All childhood characteristics came from the 1950 census. They included family SES, family structure, and region of residence, factors that in prior studies have been linked to adult health and mortality. We measured the childhood SES by the father's occupational class, except in mother only families when the mother's occupation was used. The coding was

based on prior Finnish studies and distinguished among professional/administrative occupations, manual workers, and farmers, with self-employed, and other and unknown occupations grouped together (Notkola, Martelin, & Koskinen, 2002). We coded parental education to the highest level of schooling of either parent. We characterized childhood housing conditions by household crowding coded as the number of persons per heated room in the dwelling. It reflects family's housing wealth and was a stronger predictor of adult mortality than home ownership. Childhood family structure distinguished among mother only and father only families, and families where both parents were present. We differentiated among five Finnish regions: the area surrounding the capital city Helsinki, the rest of Southern Finland (rest of Uusimaa), Western Finland, Eastern Finland and Lapland, capturing the well-known regional differences in mortality between North-East Finland and South-West Finland (Blomgren, Martikainen, Mäkelä, & Valkonen, 2004; Saarela & Finnäs, 2009). We also controlled for gender and birth cohort distinguishing birth cohorts by whether or not individuals were born before November 30, 1939, between this date and September 19, 1944 or thereafter. Finland fought two wars against the Soviet Union between late November 1939 and September 1944 and thus we identified children born prior to, during, and after the war period.

### Adult Characteristics

Adult characteristics reflected attained SES and family characteristics in young adulthood when most study subjects had completed their education and begun their occupational careers. They include marital status, family formation, educational attainment, occupation, labor force attachment, and home ownership, which are known to be significant predictors of adult mortality in Finland (Martikainen, Blomgren, & Valkonen 2007; Koskinen, Joutsenniemi, Martelin, & Martikainen, 2007) and elsewhere (Elo & Preston, 1996; Smith 2007).

Marital status was coded as never married, currently married, and divorced/separated/widowed individuals. For the period under study the censuses do not provide consistent information on cohabitation, which was still relatively uncommon in Finland for those born before 1950, although it has subsequently increased substantially (Pitkänen & Jalovaara, 2007.) As a measure of family formation, we included an indicator of whether there were children in the household. Educational attainment distinguished among a wider range of educational levels compared to the educational levels of the parents: basic or primary (9 years), lower secondary (10–11 years), upper secondary (12 years) and post-secondary (13+ years) education. The 7-category occupational coding differentiated among white collar and manual occupations, farmers, self-employed and other and unknown. Home ownership differentiated among owners, renters and employer-provided housing. Labor force attachment distinguished among those in and out of the labor force and housewives.

### Causes of death

We investigated all-cause mortality and mortality from cardiovascular diseases (CVD), alcohol-related causes, accidents and violence and lung cancer. Childhood circumstances have been associated with cardiovascular disease mortality in numerous prior studies (Galobardes et al., 2004, 2008; Strand & Kunst, 2006). Alcohol-related causes, which

include among others alcoholic liver disease, alcoholic diseases of the pancreas, alcoholic cardiomyopathy, alcohol dependence syndrome and other mental and behavioral disorders due to alcohol use, are important causes of middle age male mortality in Finland (Herttua, Mäkelä, & Martikainen, 2008). In addition many accidental and violent deaths in Finland are associated with excessive alcohol use (Herttua et al., 2008). Lung cancer, a cause of death closely associated with cigarette smoking, has also been associated with childhood background and adult SES in some prior studies (Galobardes et al., 2004; Lawlor et al., 2006).

### Statistical methods

We used Cox proportional hazards regression to estimate all-cause and cause-specific mortality beginning at age 35 until the end of follow-up, which for the oldest individual was age 72. Individuals who were alive on December 31, 2007 were censored on this date. In the cause-specific mortality models, individuals who died from causes other than the one under investigation were censored at the date of death. Because censoring occurred at different ages for the study subjects, we examined the sensitivity of our results when mortality follow-up was truncated at the same age for everyone (results not shown). The results were substantively similar to those presented in this paper.

We estimated three models: Model 1 included gender and all childhood characteristics. Model 2 included gender and all adult characteristics. Model 3 included gender and all childhood and all adult characteristics. We also tested whether the associations between childhood characteristics and all-cause and cause-specific mortality varied by gender. We focus on gender differences in the associations between early life influences and mortality only because of space limitations and because previous studies have not examined this gender variation in Finland in detail. Prior Finnish studies that have examined the associations between gender and adult SES indicate that although differentials in total mortality are larger among men than women, this gender difference is largely driven by gender differences in the cause of death structure and SES differentials in mortality within specific causes of death are often relatively small (Koskinen & Martelin, 1994; Martikainen, 1995).

One of the weaknesses of this analysis is that the estimated hazard ratios for adult characteristics may be biased by unobserved genetic or social factors that influence both adult SES and adult mortality. To eliminate this bias, we employed within sibling analysis to control for all unobserved social and genetic characteristics shared among the siblings. We identified 76,001 individuals with at least one sibling in 27,379 families. Based on this sample, we estimated fixed effects models using the stratification technique suggested in Allison (2009), which allows for a separate baseline hazard for each family. Because the baseline hazards cancel out of the partial likelihood, this method reduces both the computational burden and the incidental parameter bias. All models used robust standard errors to account for clustering of individuals within families and were estimated using partial likelihood estimation in STATA 11.

## RESULTS

Table 1 provides sample characteristics and Table 2 shows distribution of causes. The hazard ratios for all-cause mortality are shown in Table 3 and for causes of death in Table 4. The results from the fixed effect models are presented in Table 5.

As seen in Table 1, just over half of the sample was male, close to half was born after September 19, 1944, and most (91%) lived with both parents in 1950. Relatively few grew up in families (15%) where a parent was employed in professional or administrative occupations, whereas over 40% grew up in families where a parent was an agricultural or manual worker, and about a third were children of farmers. Only 10% of the parents had education beyond primary school, and slightly over a third of the children grew up in relatively crowded housing conditions (3+ people per heated room).

Table 1 also reveals considerable intergenerational occupational and educational mobility. By their early 30s, over 40% of the children were employed in upper (14%) or lower white-collar occupations (29%) and only about 6% were farmers. Most others were employed as industrial (18%) or other manual workers (21%). About 50% of the children had obtained education beyond primary school compared to only 10% of their parents. Labor force participation rate was also high at 83%, although this varied between men (93%) and women (73%). Many of those not in the labor force, other than housewives, were likely to be out of the labor force for health reasons. Over 50% of the sample owned their own house or apartment.

Close to 81% had married at least once, and 7% had already separated, divorced or widowed and close to 80% had children in the household. Nineteen percent had never married.

The cause-of-death distributions varied by gender and a higher percentage of the men (18%) than women (8%) had died by the end of the follow-up (Table 2). Men were more likely to have died from CVD (33% of male deaths) compared to women (21% of female deaths). Similarly, male mortality (38% of male deaths) was much higher from alcohol-related diseases, accidents and violence, and lung cancer than female mortality (23% of female deaths).

### Childhood characteristics and adult mortality

All childhood characteristics exhibited a significant association with all-cause mortality (Model 1, Table 3). We documented significantly higher all-cause mortality among respondents who were born before the war (HR=1.092) compared to the other cohorts. Higher parental SES was associated with significantly lower mortality as was farm residence. Children who grew up on large (HR= 0.76) and small (HR= 0.85) farms or in families where mothers or fathers were employed in professional/administrative occupations (HR= 0.84) had significantly lower adult mortality than children of agricultural or other workers. Similarly higher parental education was associated with significantly lower adult mortality, whereas household crowding was predictive of higher mortality. Family structure also mattered with the excess risk ranging from 14% in mother only to 21% in father only families compared to families with both parents. Childhood residence in Eastern Finland

was associated with a significantly higher risk of adult mortality than residence in Western or Southern Finland, but not compared to the Helsinki region or Lapland.

Controls for adult characteristics attenuated these associations, in many instances by close to 50% and not all remained statistically significant (Model 3, Table 3). For example, parental education and crowded housing were no longer significant predictors of adult mortality, although the direction of these associations remained the same (Model 1 versus Model 3). We continued to find significant associations between all-cause mortality and birth cohort, region of childhood residence, parental occupation, and childhood family type. For example, children of farmers, regardless of farm size, experienced about 15–20% lower mortality than children of agricultural or other workers. The mortality of children who grew up in single parent families remained about 10–14% higher than that of children who lived with both parents. Additional analyses (not shown) indicated that most adult characteristics mediated the associations between childhood conditions and mortality with occupation and educational attainment being the most important.

The results for cause-specific mortality from the fully adjusted models (Model 3) are shown in Table 4. Most childhood characteristics remained significantly associated with CVD mortality and the direction of the associations with other cause-of-death groups examined were generally similar, but they were less robust to controls for adult characteristics. For example, farm residence in childhood was predictive of lower mortality from all cause-of-death groups analyzed, although the associations reached statistical significance only for CVD and alcohol-related causes. Higher parental education also appeared to be protective against deaths from CVD and accidents and violence. In addition, childhood residence in Western and/or Southern Finland compared to Eastern Finland was associated with significantly lower mortality from all causes of death, other than lung cancer. Childhood residence in single parent families appeared to confer excess risk, although the excess risk remained significant only for CVD.

We also documented significant birth cohort differences for CVD and alcohol-related causes. Mortality from CVD was higher for cohorts born prior to the war (HR: 1.47) and during the war (1.19) than after the war. On the other hand, the earlier birth cohorts had significantly lower mortality from alcohol-related causes than those born after the war. These cohort differences were robust to controls for unobserved heterogeneity in the fixed effects specification, except for the elevated risk of death from CVD among those born during the war years (Table 5).

We found no significant interactions between gender and childhood characteristics. This may be because the association of family conditions and adult mortality does not vary by gender, or because there were relatively few female deaths.

### **Adult characteristics**

All adult characteristics were significant predictors of all-cause mortality with and without controls for childhood characteristics. In fact, adjustment for childhood conditions had little impact on the hazard ratios of adult characteristics (Model 2 versus Model 3, Table 3). Higher SES, whether measured by educational attainment, occupation or home ownership



was associated with significantly lower adult mortality. For example, the fully adjusted hazard ratio for highest level of education compared to primary education (9 years) was 0.63, for those employed in upper white collar occupations versus manual workers, other than industrial workers, it was 0.74, and for home owners versus renters it was 0.82 (Table 3). In addition, the never married and separated/divorced/widowed individuals experienced an excess risk compared to those who were married, and living in households without children was associated with about 5% increased risk of death after age 35.

Most adult characteristics were also significant predictors of cause-specific mortality, adjusting for childhood characteristics. In particular, higher levels of schooling, home ownership and being married were associated with significantly lower mortality from all cause-of-death groups examined. Results were mixed for occupation, although in general the findings pointed to a protective effect of higher status occupations (Table 4).

Table 5 shows hazard ratios from Model 3 with a family fixed effect for all-cause and cause-specific mortality. When comparing these results with those from Model 3 in Tables 3 and 4, we see that the introduction of the family fixed effect resulted in the attenuation of the hazard ratios of many adult characteristics with some variation observed by cause of death. For example, we documented strong attenuation in the hazard ratio for separated/divorced/widowed individuals for all-cause and cause-specific mortality. The reductions for educational attainment were also sizable for lung cancer, alcohol-related mortality, and accidents and violence. At the same time, education remained a significant predictor of CVD and all-cause mortality. Occupation remained a significant predictor of all-cause mortality and CVD, lung cancer and accidents and violence, as did home ownership for all causes except lung cancer.

## DISCUSSION

We used high quality register based data to examine associations between childhood and adult socio-demographic characteristics and all-cause and cause-specific mortality in Finland in the latter half of the twentieth century. These data include prospective follow-up of children from early childhood up to age 72. Information on childhood conditions came from the 1950 Finnish Census, whereas linkage to census and death records from the end of 1970 through the end of 2007 provided information on adult characteristics and mortality.

### Childhood characteristics

We documented significant associations between childhood SES and family characteristics and all-cause mortality between ages 35 and 72 such that higher childhood family SES was associated with significantly lower mortality above age 35. Without adjustment for adult characteristics having parents with at least primary school education reduced adult mortality by about 10–18%, and having parents who were employed in professional/administrative occupations or who were farmers was associated with about 15–25% reduction. In contrast, living in crowded housing or with only one parent was associated with a significant excess risk of death. The excess risk of living with only one parent in 1950 is consistent with findings from prior studies (e.g., Hayward & Gorman, 2004; Pensola & Martikainen 2003). Given data limitations we were unable to assess whether this risk varied by the age at which

the child lost his/her parent (Reher & González-Quiñones, 2003) or whether a reconstitution of the family subsequently modified this risk. These are important questions for further research.

The attenuation of the associations between childhood characteristics and mortality above age 35 were mostly explained by adjustment for educational attainment and occupation status in adulthood. These results are consistent with the hypothesis that early life conditions influence adult health and mortality indirectly via what Preston et al. (1998) refer to as 'correlated environments.' Individuals raised in environments with greater resources obtained higher levels of schooling and better jobs in early adulthood which in turn translated into better health later in life.

Children who grow up in wealthier households are also healthier (e.g., Case et al. 2005). In the absence of information on childhood health our measures of childhood SES were likely to capture at least some of this health effect. Our finding that having grown up on a farm was predictive of lower mortality may be related to better health of children who grew up on farms. Children in our sample either lived through the years when Finland was in war against the Soviet Union or were born shortly thereafter. Farm residence during this period may have been especially protective, for example, by mitigating effect of food rationing. Alternatively or in addition, farm residence in childhood may be related to a healthier life style in adulthood. This speculation is consistent with the finding that farm residence was also a significant predictor of mortality from causes that have a large behavioral component, such as lung cancer, alcohol related causes and accidents and violence. In a related Finnish study, the protective effect of being a son of a farmer was also found for alcohol-related causes and accidental and violent deaths, but not for CVD net of adult SES among birth cohorts born between 1956 and 1960 (Pensola & Valkonen 2002). In the United States, where farm residence in childhood was also associated with lower risk of death at older ages (Hayward & Gorman, 2004; Preston et al., 1998), adult life style factors appeared to have been one pathway through which rural residence was associated with lower adult mortality (Hayward & Gorman, 2004).

Similarly our finding that mortality was higher among children who spent much of their early childhood during the period when Finland fought two wars against the Soviet Union may at least in part be related to childhood health. This association was particularly strong for mortality from CVD, causes of death that have most consistently exhibited a strong association with early life health conditions. However, this finding may also reflect improvements in the treatment of CVD that occurred from the early 1980s onwards. In other words, better treatment of CVD may have been available for the younger cohorts at the time when they reached the age of increased risk of CVD. In contrast, mortality from alcohol related causes was higher among the younger birth cohorts. This may reflect the increasing availability of alcohol from the late 1960's onwards that may have had a larger imprint on the younger cohorts approaching their most impressionable age during this period.

We found persistent regional differences in mortality with those whose childhood residence was in Eastern Finland having higher all-cause mortality and mortality from CVD, alcohol-related causes, and accidents and violence than those living in Western and/or Southern

Finland. It has been hypothesized that these regional mortality differences could be, at least partially, attributed to behavioral and social or to genetic differences (Saarela & Finnäs, 2009). We cannot evaluate the relative importance of these factors. Strong regional mortality differences were found for accidents and violence, which may depend more on behavior than genes, tentatively pointing towards the role of behavioral factors.

It is possible that associations between childhood conditions and adult mortality are either weaker for later born cohorts as adverse childhood material conditions have become less common or that they are stronger as poverty and other forms of disadvantage are concentrated in ever smaller population subgroups. In the Finnish case, a rough comparison can be made to a study based on a similar design but which draws its sample of children born in 1956–1960 from the 1970 Finnish Census (Pensola & Valkonen, 2002; Pensola & Martikainen, 2003). Although various methodological differences (e.g., younger ages at death, a shorter follow-up, and definitions of parental social class) prevent exact comparisons, the results nevertheless suggest that mortality differentials by parental social class have not disappeared in Finland, if anything they may be stronger. For men in these recent cohorts mortality differentials for accidental and violent deaths, for example, were two-fold between the top and bottom parental social class, although one's own education and class position largely explained them. Similarly, recent studies based on register-based data have documented significant associations between childhood socioeconomic position and mortality for cohorts born in 1955–1965 in Norway (Strand and Kunst, 2006), cohorts born in 1944–1960 in Sweden (Lawlor et al., 2006), and males born in 1953 in Denmark (Osler et al., 2005). The enduring influence of childhood circumstances on mortality in more recent cohorts also points to the potential importance of health habits that cluster in families and for which individuals may acquire predisposition early in life, such as smoking and alcohol consumption. In more recent cohorts, dietary habits may also play an increasing role. Among the older cohorts overweight and obesity was less common in childhood than in more recent cohorts and there is increasing evidence that duration of obesity plays an important role in mortality, especially from heart disease and diabetes (Kautiainen, Rimpelä, Vikat, & Virtanen, 2002; Preston, Mehta, & Stokes, 2013; Vartiainen et al., 2010). Further studies are needed to explore the various pathways linking childhood characteristics and adult mortality across various birth cohorts.

### Adult Characteristics

In 1950, Finland was relatively underprivileged, burdened by heavy war debts to the Soviet Union, and with a relatively large segment of its labor force employed in agriculture or manual occupations. In subsequent decades, the development of the industrial and service sectors and widening educational venues quickly opened new opportunities for the younger generations increasing social mobility and a movement away from agriculture. These patterns are also evident in our data.

At the same time, SES inequalities in mortality have persisted. Education, class position and home ownership were significant predictors of all-cause mortality and these associations were not attenuated by the inclusion of observed childhood family background and they remained significant in the fixed effects specification (see also Næss et al. 2012). However,

these results did not hold for all cause-of-death groups examined. Adult social characteristics remained strong predictors of CVD in all model specifications, but the association between educational attainment and mortality from causes of death with a substantial behavioral component, i.e., alcohol-related diseases, accidents and violence, and lung cancer, were substantially attenuated in the fixed effects specification. This finding demonstrates that family specific influences, not captured by the observed family background characteristics, may have played a role. It is possible, for example, that smoking and alcohol consumption cluster in families such that children, who grow up in households where parents engage in these behaviors, are more likely to pick up these habits as well (Osler et al., 2005, 2006). Other unobserved factors could include parenting styles that differ among families, such as the degree of parental oversight and control. Although health behaviors, such as smoking and heavy alcohol consumption, are also associated with CVD mortality other influences also play a role, such as diet and exercise, and access to latest medical treatments for heart disease. Higher levels of education have been associated with smoking cessation in Finland as elsewhere since the 1970s (Helakorpi et al., 2008) and it has been suggested that the widening of educational differences in heart disease mortality in Finland was at least in part related to access to latest treatment for heart disease (Valkonen et al., 2000). Between 1982 and 1997, when coronary heart disease mortality declined in Finland by over 60%, it has been estimated that improved medical treatment explained about 23% of this decline and changes in risk factors between 53% and 72% (Laatikainen et al. 2005).

Finally, we found robust associations for marital status and all cause and cause-specific mortality, although these associations were attenuated in the fixed effect specification.

### Methodological considerations

Our large nationally representative high quality data avoids problems caused by potential recall bias when information on childhood characteristics is retrospectively reported. All information on childhood and adult characteristics came from censuses that were conducted at the time when these characteristics were measured. Few data sets have permitted follow-up of nationally representative samples of children into late adulthood (e.g., Pensola & Martikainen, 2003; Strand & Kunst, 2006; Søndergaard et al., 2012; Næss et al. 2012), and thus most prior studies have relied on retrospectively reported data on early life circumstances (e.g., Haas, 2008; Kauhanen, Lakka, Lynch, & Kauhanen, 2006).

These data also have limitations. As noted earlier, some children could not be linked to the 1970 or subsequent Finnish censuses or death records after 1970. A small proportion of individuals had died before 1970, but most were likely young emigrants to Sweden in the late 1960s. In Appendix Table 1 we compare those lost to follow-up and the analytic sample on their childhood characteristics. Those lost to follow-up were more likely to be women, be born before 1945, come from lower SES backgrounds, be from mother only families and from Lapland. Although statistically significant these differences were not particularly large and thus they are unlikely to bias our main findings. Second, we measured adult characteristics in young adulthood prior to the beginning of the mortality follow-up at age 35. For close to 99% of the subjects these characteristics were measured at ages 30–35. The

results presented here are not sensitive to alternative measurement of these attributes at ages 35–44. Third, we can measure childhood characteristics only in the 1950 census and thus they were not measured at the same age for all subjects.

## Conclusions

We used high quality register based data to examine associations between childhood and adult social environments and all-cause and cause-specific mortality in Finland in 1970–2007. Overall, we found higher childhood SES to be a significant predictor of lower adult mortality. These associations were mediated by adult socio-demographic characteristics, suggesting that the indirect effects of childhood conditions were more important than their direct effects. We further documented that adult SES was a significant predictor of mortality, and this association was robust for inclusion of observed childhood characteristics and unobserved family characteristics shared by the siblings.

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## Appendix Table 1

Comparison of childhood characteristics between the analytic sample and those who died before 1970 or were lost to follow-up

Characteristic	Analytic sample (N=98,389)	Those who died or were lost to follow-up (N=15,065)
<i>Gender</i>		
Male	51.8	44.2
Female	48.2	55.8
<i>Birth Cohort</i>		
Prewar	20.9	23.4
War years	27.5	28.8
Postwar	51.7	47.8
<i>Region of residence</i>		
Helsinki region	7.5	7.1
Rest of Uusimaa	5.8	6.6
Western Finland	39.4	39.1
Eastern Finland	42.8	38.3
Lapland	4.5	8.9
<i>Occupation of family head</i>		
Professional/administrative	15.1	12.5
Agricultural and other workers	41.8	47.2
Farmers < 10 hectares	25.8	24.0
Farmers 10 hectares	8.1	6.0
Other <sup>b</sup>	9.3	10.3
<i>Parental Education</i>		
Less than primary school	16.4	20.1
Primary school	73.3	72.0
Past primary school	10.3	8.0
<i>Housing Conditions (people per heated room)</i>		
< 2	32.2	27.3
2-3	31.9	30.9
3-4	16.4	17.6
4+	18.3	22.7
Missing	1.1	1.6
<i>Family type</i>		
Both parents	91.0	88.2
Mother only	7.8	9.9
Father only	1.3	1.8

All Chi-square tests between the two samples were statistically significant,  $p < 0.01$ .



### Research Highlights

- We study impact of childhood and adult socio-demographic conditions on mortality
- The study is based on 1935–1950 birth cohorts in Finland
- We find childhood conditions to be significant predictors of adult mortality
- Adult educational attainment and occupation mediated these associations
- Impact of adult attributes was robust for controls of childhood conditions

TABLE 1

Sample Characteristics (%), Finland 1950 and 1970–85 (N=98,389)

Characteristics	Entire sample N=98,389	Deaths N=12,956
<i>Childhood Characteristics- 1950 Census, Ages 0–14</i>		
<i>Gender</i>		
Male	51.8	71.0
Female	48.2	29.0
<i>Number of children in the family</i>		
One	22.7	24.4
Two	30.2	28.2
Three +	47.1	47.4
<i>Birth cohort</i>		
Prewar	20.8	32.8
War years	27.5	29.8
Postwar	51.7	37.5
<i>Region of residence</i>		
Helsinki Region	7.5	8.0
Rest of Uusimaa	5.8	5.6
Western Finland	39.4	36.7
Eastern Finland	42.8	44.9
Lapland	4.5	4.8
<i>Occupation of family head<sup>a</sup></i>		
Professional/administrative	15.1	12.9
Agricultural and other workers	41.8	44.8
Farmers < 10 hectares	25.8	25.7
Farmers 10 hectares	8.1	6.9
Other <sup>b</sup>	9.3	9.7
<i>Parental Education<sup>c</sup></i>		
Less than primary school	16.4	20.6
Primary school	73.3	71.1
Past primary school	10.3	8.3
<i>Housing Conditions – people per heated room</i>		
< 2	32.2	29.3
2.0–2.99	31.9	31.3
3.00–3.99	16.4	17.1
4+	18.3	21.2
Missing	1.1	1.1
<i>Family type</i>		
Both parents	90.9	88.2
Mother only	7.8	10.1
Father only	1.3	1.7

Characteristics	Entire sample N=98, 389	Deaths N=12,956
<i>Adult Characteristics</i>		
<i>Marital Status</i>		
Never married	19.0	27.6
Married	74.4	63.0
Separated/divorced/widowed	6.6	9.3
<i>Children in the household</i>		
No	21.5	27.3
Yes	78.5	72.7
<i>Education (years)</i>		
9 years	49.4	60.8
10–11 years	25.1	21.8
12 years	13.2	9.9
13+ years	12.3	7.5
<i>Occupation</i>		
Upper white collar	13.6	8.5
Lower white collar	29.2	20.1
Manual worker – industry	17.7	22.1
Manual worker – other	20.7	24.6
Farmer	6.5	6.7
Self-employed	4.4	4.5
Unknown, including students	8.0	13.6
<i>Labor force attachment</i>		
In the labor force	83.4	81.9
Not in the labor force, excl. housewives	6.7	12.0
Housewife <sup>d</sup>	9.9	6.1
<i>Home ownership</i>		
Owner	56.9	50.0
Renter	26.1	30.2
Employer provides	12.8	12.3
Unknown	4.2	7.5

<sup>a</sup>Refers to father's occupation, except in mother only households when mother's occupation is used.

<sup>b</sup>Includes self-employed, other and unknown.

<sup>c</sup>Highest level of schooling of either the mother or the father.

<sup>d</sup>Among women 20.5% of the women were housewives.

TABLE 2

Distribution of Deaths by Cause, Ages 35–72, Finland 1970–2007

Causes of death	Men		Women		Entire Sample	
	#	%	#	%	#	%
All Causes	9,197	100.0	3,759	100.0	12,956	100.0
Cardiovascular disease	3,076	33.4	790	21.0	3,866	29.8
Lung cancer	559	6.1	165	4.4	724	5.6
Alcohol-related <sup>a</sup>	1,101	12.0	236	6.3	1,337	10.3
Accidents & violence	1,840	20.0	459	12.2	2,299	17.7
All other diseases	2,621	28.5	2,109	56.1	4,730	36.5
Mean years of follow-up (SD)						
Alive (censored)		28.3 (4.2)		28.6 (4.3)		28.5 (4.2)
Dead		19.2 (8.6)		20.2 (8.4)		19.5 (8.5)

<sup>a</sup> Alcohol-related causes refer to underlying causes of death related to excessive alcohol consumption, e.g., liver cirrhosis, or alcoholic inflammation of the heart.

TABLE 3

Hazard Ratios for All Cause Mortality at Ages 35–72, Finland 1970–2007 (N=98,389)

Characteristic	Model 1	Model 2	Model 3
<i>Childhood Characteristics</i>			
<i>Gender (Female)<sup>b</sup></i>			
Male	2.46 **	2.35 **	2.34 **
<i>Birth cohort (postwar)</i>			
Prewar	1.09 **		1.17 **
War years	1.00		1.03
<i>Region of residence (Eastern Finland)</i>			
Helsinki Region	1.05		1.06
Rest of Uusimaa	0.91 *		0.91 *
Western Finland	0.90 **		0.92 **
Lapland	0.98		0.99
<i>Occupation of family head (workers)</i>			
Professional/administrative	0.84 **		0.96
Farmers < 10 hectares	0.85 **		0.85 **
Farmers 10 hectares	0.76 **		0.79 **
Other	0.95		0.96
<i>Parental Education (&lt; primary school)</i>			
Primary school	0.89 **		0.95 *
Past primary school	0.82 **		0.96
<i>Housing Conditions (&lt; 2 per heated room)</i>			
2–3	1.04		0.98
3–4	1.06 *		0.97
4+	1.17 **		1.03
<i>Family type (both parents)</i>			
Mother only	1.14 **		1.10 **
Father only	1.21 **		1.14 *
<i>Adult Characteristics</i>			
<i>Marital Status (married)</i>			
Never married		1.57 **	1.57 **
Separated/divorced/widowed		1.77 **	1.77 **
<i>Children in the household (yes)</i>			
No		1.05 *	1.06 *
<i>Education (9 years)</i>			
10–11 years		0.81 **	0.83 **
12 years		0.77 **	0.79 **
13+ years		0.62 **	0.63 **

Characteristic	Model 1	Model 2	Model 3
<i>Occupation (other manual worker)</i>			
Upper white collar		0.74 **	0.74 **
Lower white collar		0.81 **	0.81 **
Manual worker – industry		0.98	0.98
Farmer		0.86 **	0.93
Self-employed		0.96	0.97
Unknown SES, inc. students		1.22 **	1.26 **
<i>Labor force attachment (in the labor force)</i>			
Not in the labor force, excl. housewives		1.37 **	1.35 **
Housewife		1.06	1.05
<i>Home ownership (renter)</i>			
Owner		0.80 **	0.82 **
Employer provides		0.97	0.98

\*\* p-value 0.01;

\* p-value 0.05. Standard errors are clustered at the family level.

<sup>a</sup> Model 1 controls for sex, birth cohort, and childhood conditions. Model 2 controls for sex and adult characteristics. Model 3 controls for all explanatory variables.

<sup>b</sup> Omitted category in parentheses.

TABLE 4

Hazard Ratios for Cause-specific Mortality Ages 35–72, Finland 1970–2007 (N=98,389)

Characteristic	Cardiovascular Diseases	Lung Cancer	Alcohol Related Causes	Accidents and Violence
	Model 3 <sup>a</sup>	Model 3	Model 3	Model 3
<i>Childhood Characteristics</i>				
<i>Gender (Female)<sup>b</sup></i>				
Male	3.73 **	3.37 **	4.27 **	3.64 **
<i>Birth cohort (postwar years)</i>				
Prewar	1.47 **	1.22	0.79 **	1.09
War years	1.19 **	1.17	0.78 **	1.03
<i>Region of residence (Eastern Finland)</i>				
Helsinki Region	0.90	1.32	0.97	0.99
Rest of Uusimaa	0.80 **	1.00	0.85	0.62 **
Western Finland	0.83 **	1.05	0.86 *	0.79 **
Lapland	0.88	1.08	0.87	1.10
<i>Occupation of family head (workers)</i>				
Professional/administrative	0.95	0.84	0.88	1.04
Farmers < 10 hectares	0.80 **	0.87	0.77 **	0.88 *
Farmers 10 hectares	0.81 **	0.74	0.65 **	0.89
Other	0.95	0.96	1.04	0.91
<i>Parental Education (&lt; primary school)</i>				
Primary school	0.92 *	1.05	1.01	0.83 **
Past primary school	0.91	1.03	1.11	0.88
<i>Housing Conditions (&lt; 2 per heated room)</i>				
2–3	1.03	1.07	0.97	1.07
3–4	1.05	1.23	0.82 *	1.02
4+	1.08	1.29 *	0.99	1.11
<i>Family type (both parents)</i>				
Mother only	1.22 **	1.05	1.06	1.03
Father only	1.06	1.42	1.06	1.15
<i>Adult Characteristics</i>				
<i>Marital Status (married)</i>				
Never married	1.58 **	1.52 **	1.49 **	1.67 **
Separated/divorced/widowed	1.78 **	2.23 **	2.72 **	2.10 **
<i>Children in the household (yes)</i>				
No	0.97	0.85	1.14	1.13 *
<i>Education (9 years)</i>				
10–11 years	0.85 **	0.57 **	0.86 *	0.95

Characteristic	Cardiovascular Diseases	Lung Cancer	Alcohol Related Causes	Accidents and Violence
	Model 3 <sup>a</sup>	Model 3	Model 3	Model 3
12 years	0.75 **	0.51 **	0.77 **	0.80 **
13+ years	0.54 **	0.54 **	0.57 **	0.66 **
<i>Occupation (other manual worker)</i>				
Upper white collar	0.69 **	0.64 *	0.92	0.72 **
Lower white collar	0.72 **	0.89	0.90	0.77 **
Manual worker – industry	0.94	1.06	1.14	0.87 *
Farmer	1.03	0.81	0.81	0.70 **
Self-employed	0.99	1.02	1.15	0.97
Unknown SES, inc. students	1.17	1.37	1.82 **	1.27
<i>Labor force attachment (in the labor force)</i>				
Not in the labor force, excl. housewives	1.53 **	1.06	0.68 *	1.13
Housewife	1.03	0.90	0.85	0.91
<i>Home ownership (renter)</i>				
Owner	0.80 **	0.69 **	0.71 **	0.81 **
Employer provides	0.97	0.91	0.87	0.94

\*\* p-value 0.01;

\* p-value 0.05. Standard errors are clustered at the family level.

<sup>a</sup> Model 3 controls for all explanatory variables.

<sup>b</sup> Omitted category in parentheses.



TABLE 5

Hazard Ratios for All Cause and Cause-Specific Mortality at Ages 35–72, Family Fixed Effects, Finland 1970–2007 (N=76,001)

Characteristic	All Cause	Cardiovascular Diseases	Lung Cancer	Alcohol Related Causes	Accidents & Violence
<i>Childhood Characteristics</i>					
<i>Gender (Female)<sup>a</sup></i>					
Male	2.42 **	4.23 **	3.44 **	4.77 **	4.10 **
<i>Birth cohort (Postwar years)</i>					
Prewar	1.12 *	1.38 **	1.08	0.76 *	1.07
War years	0.99	1.04	1.22	0.77 *	0.98
<i>Adult Characteristics</i>					
<i>Marital Status (married)</i>					
Never married	1.54 **	1.50 **	1.34	1.40 **	1.47 **
Separated/divorced/widowed	1.45 **	1.52 **	1.78 *	1.81 **	1.53 **
<i>Children in the household (yes)</i>					
No	1.07	0.99	1.04	1.06	1.21 *
<i>Education (9 years)</i>					
10–11 years	0.89 **	0.81 **	0.59 **	0.92	1.06
12 years	0.85 **	0.62 **	0.62	1.07	0.81
13+ years	0.76 **	0.58 **	0.93	0.60	0.78
<i>Occupation (other manual worker)</i>					
Upper white collar	0.66 **	0.71 *	0.44 *	0.91	0.63 **
Lower white collar	0.81 **	0.78 *	0.82	0.84	0.79 *
Manual worker – industry	0.97	0.95	0.94	1.04	0.92
Farmer	0.95	1.00	0.98	0.86	0.84
Self-employed	1.06	1.10	1.34	0.93	0.94
Unknown SES, inc. students	1.20	1.11	1.18	1.51	1.45
<i>Labor force attachment (in the labor force)</i>					
Not in the labor force, excl. housewives	1.48 **	1.90 **	1.39	0.93	0.95
Housewife	1.04	1.10	0.80	0.69	0.88

Characteristic	All Cause	Cardiovascular Diseases	Lung Cancer	Alcohol Related Causes	Accidents & Violence
<i>Home ownership (renter)</i>					
Owner	0.83 **	0.76 **	0.76	0.61 **	0.78 **
Employer provides	0.99	0.97	0.91	0.75	0.93

\*\* p-value 0.01;

\* p-value 0.05.

<sup>a</sup>Omitted category in parentheses.