TIINA PENSOLA

FROM PAST TO PRESENT:
EFFECT OF LIFECOURSE ON MORTALITY,
AND
SOCIAL CLASS DIFFERENCES IN MORTALITY
IN MIDDLE ADULTHOOD

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Abstract

Social class differences in mortality are larger in middle adulthood than at any other time of life. Circumstances over the lifecourse may contribute to these adult social class differences. However, it is only rarely that the lifecourse approach has been applied to mortality studies among persons in their middle adulthood. The aim of this thesis is to disentangle the effects of the living conditions in the parental home and major transition in youth on social class differences in mortality from various causes of death among women and men aged 31-42 at death, and to evaluate whether the effect of the past circumstances on mortality is through latency, accumulation or pathway mechanisms. This thesis (papers II-V) is based on the 1990 census data for all Finnish persons born in 1956-60 linked with death records (4,369 deaths) for 1991-98 and with information on lifecourse circumstances from the 1970, 1975, 1980 and 1985 censuses. These aggregated cross-tables are analysed by means of Poisson regression.

Parental home had an association with disease mortality from age 20 onwards, indicating a latency effect. However, the direct effect of the parental home on mortality was minor, and therefore the contribution of latency model to differential mortality remained small. An indication for the accumulative effect of disadvantageous social class was found for cardiovascular diseases and alcohol-related causes. The living conditions in the parental home, i.e. the manual class and one-parent family, had an effect on the transitions a person experienced in youth, and thus contributed to the effect youth paths exerted on adult social class differences in mortality from various causes of death. Youth paths had a substantial effect (about 60-90%) over and above the preceding effect of living conditions in the parental home on mortality. The higher mortality in the lower social classes was mainly attributable to disadvantageous educational path. Moreover, both family formation, particularly early marriage in women and staying single in men, and experience of unemployment in youth, had independent effects on class differences in mortality. These results strongly suggest that youth is a 'sensitive period' affecting social class differences in mortality in middle adulthood.

Contents

The list of original publications

Paper I: Pensola T, Valkonen T. Mortality differences by parental social class from childhood to adulthood. *Journal of Epidemiology and Community Health* 2000;54:525-9.

Paper II: Pensola T, Martikainen P. Cumulative social class and mortality from various causes in young men. *Journal of Epidemiology and Community Health* 2003;57:745-751.

Paper III: Pensola T, Valkonen T. Effect of parental social class, own education and social class on mortality among young men. *European Journal of Public Health* 2002;12:29-36 .

Paper IV: Pensola T, Martikainen P. Effect of the living conditions in the parental home and youth paths on social class differences in mortality among women. *Scandinavian Journal of Public Health,* in print.

Paper V: Pensola T, Martikainen P. Life course experiences and mortality by adult social class among young men. *Social Science & Medicine,* in print.

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1 Introduction

Social class differences in mortality have been found in all periods (Townsend & Davidson 1992, Marmot 1994) and in all countries in which they have been studied (Machenbach et al. 1999), and in both sexes and all age groups (Vågerö 1992, Valkonen et al. 1993). It is only for the age group 10-14 that the results have been somewhat inconsistent, showing clear differences in some countries (Östberg1992, Östberg & Vågerö 1991) and minor or no differences in others (West 1991, Rimpelä 1993, Blane et al. 1994). Young and middle adulthood are times of great social inequality in this respect, the relative mortality differences being larger in one's 30s and 40s than at any other time of life (Townsend & Davidson 1992:123, Valkonen et al. 1993, Vågerö 1992). Although mortality rates in young and middle adulthood are relatively low, they strongly contribute to social class differences in life expectancy or potential years of life lost (Blane et al. 1990). Furthermore, in many cases these deaths could be regarded as avoidable: in 1991-97, of all deaths in Finland of men and women in the age 30-39-year age group, approximately 60% and 40% respectively were attributed to external causes (Statistics Finland, causes of death 1991-97).

There are several explanations for these differences based on theories of social causation and selection, or variants of them. Many risk factors for premature death, contemporaneous or from years before, as well as complex paths from early experiences to subsequent disease, have been suggested and found. However, the reasons for the social class differences are not fully understood. There is evidence that circumstances over the lifecourse may contribute to social class differences in mortality during adulthood. In addition, early living conditions affecting health directly may be a relevant factor in that they determine adult social and material circumstances. Particularly important life stages in this respect are youth and early adulthood when major transitions, i.e. gaining educational qualifications, leaving the parental home, forming a family, and entering the labour market, take place (Kuh & Ben-Shlomo 1997, 7). However, it is only rarely that the lifecourse approach has been applied to mortality studies among persons in middle adulthood, particularly women – and there is a lack of knowledge of the extent to which experiences over the lifecourse explain social class differences.

The consistency and persistence of social class differences in mortality is a very strong indication of inequality among population groups in society. Narrowing the gap has been, and is, a central target in many national and international programmes (WHO 1985, Townsend & Davidson 1992, Valtioneuvosto 2001). The lifecourse approach may contribute to policy formation by helping us to identify the factors and life phases

that are most affected, and the population groups most in need of interventions to reduce these social class differences in mortality in young and middle adulthood and to achieve more equality between population groups in the whole life span (Wadsworth 1997a, Ben-Shlomo & Kuh 1997). Moreover, when we know how factors in childhood and youth affect adult social class and health, we may be able to better estimate what kind of health and health inequalities the children of this day are likely to face when they are adults (Wadsworth & Kuh 1997).

This study aims to reveal the effect of socio-demographic circumstances and events along the lifecourse of persons in early middle adulthood on social class differences in mortality. The focus is on population groups and not on individuals. The study question and context, therefore, belong to the domain of sociology, although the approach is not traditionally sociological.

2 The definition of the central concepts in this study

2.1 Middle adulthood

This thesis concerns the effect of socio-demographic lifecourse experiences on mortality in middle adulthood. Although we instinctively know where childhood, youth, and old age should be placed in the lifecourse, the exact ages referred to vary by country and time according to different cultures and related life circumstances. In this study, childhood covers years before the age of ten, early youth is from 10 to 14, late youth from 15 to 20, and young adulthood from 21 to 30. Early middle adulthood, which is called middle adulthood here, covers the years from 31 to 42. Middle adulthood is a rather serene time of life. Most people have finished their studies, have moved from their parental home to their own home, have found a partner, and have started their family and their own career. Many major transitions that belong to youth and young adulthood have taken place, and physical decline related to late middle adulthood has not as yet set in (see e.g. Ward 2002). Nevertheless, causes of death at this age are changing from accident-dominant to disease-dominant, and deaths from chronic diseases are gradually increasing.

2.2 Health and mortality in middle adulthood

Studying social class differences in mortality in middle adulthood is complicated due to the relatively small number of deaths at these ages. In general, middle adulthood is one of the healthiest times of life in spite of the fact that approximately one third of the Finnish population aged 30-44 have at least one long-term illness; the proportion of people in this age group perceiving their health as at least relatively good was 82% among women and 79% among men in 2000 (Koskinen & Aromaa 2002). At this age, diseases may limit life to a different extent than chronic diseases in older age.

Difficulties related to the concept of health will not be abolished by the substitution of 'health' with measures such as health potential, health capital, and positive health capital (measured by height, for instance), or even health constitution (see e.g., Mheen 1998). These measures are widely used, but are problematic as they are difficult, or even impossible, to define accurately and in a way that the whole research community would accept. For instance, Koivusilta (2000: 39) defines *health potential* as one form of coping resource. Kuh and colleagues define health capital as "*the accumulation of biological resources, inherited and acquired during earlier stages of life which*

determine current health and future health potential, including resilience to future environmental insults" (Kuh et al. 1997, 173). According to this definition, health potential is something that is determined by health capital. Capital connotes the idea of assets, which may be invested and which accrue (used by Mheen1998:6), dwindle or are retained. In this context, health potential may be considered the byword for health capital. If anything, it is a philosophical question whether or not they are different.

As far as middle adulthood is concerned, it might be more relevant to study conditions predisposing to poor health in later life rather than poor health at that age (Davey Smith et al. 1994). However, the interest here is to study the effects of past circumstances on mortality.

Mortality cannot be understood as a direct health indicator. It may be stating the obvious, but death means clear exclusion from physical and mental well-being and from all social relations. Furthermore, death due to disease follows a shorter or longer period of poor health. Moreover, an external cause of death, such as suicide, alcohol or drug poisoning, or even some traffic accidents and assaults, may end a process involving mental or physical disorders, or social marginalization, and therefore be closely connected to the concept of health. However, most disabilities or chronic diseases, although they might limit daily social life, do not lead to death. Death may also strike a person who has not experienced any remarkable deterioration in health.

However, mortality in young and middle adulthood may, in some respects, describe social inequalities even better than health measures. First, mortality leaves very little room for speculation on the possible effects of different tendencies of people in different social classes or with different social backgrounds to react to and report health problems. Second, health measures might be difficult to associate with external causes of death. Such causes in young and middle adulthood affect life expectancy and thus they contribute to social inequalities across the whole life span. Therefore, in order to diminish the effect of social inequalities in mortality, we need to disentangle the factors that are related to diseases and also those that are related to external causes. In this respect, little is known of the effect of sociodemographic factors across the lifecourse on social class differences in mortality from external causes. In middle adulthood, i.e. in the age span from 31 to 43, mortality from external causes is predominant, but disease mortality is on the increase. Therefore, it is possible to study the effect of the lifecourse on both external causes and diseases (see 4.6).

2.3 Social class

2.3.1 Social class and mortality

Health, morbidity and mortality have been systematically related to social class at different times, for example in the Registrar General classification from the 1920s to this day (Townsend & Davidson 1992, Marmot 1989), in different countries (advanced, industrialized countries: there is not much data for developing countries) (Marmot 1994, Mackenbach et al.1999), in both sexes (Valkonen et al. 1993), in different causes of death (Marmot et al. 1984, Marmot 1989, Valkonen et al. 1993), and with different occupational-class classifications (Elstad 2000:37). Occupation-based social class (together with some other indicators of socio-economic position such as education, income and home ownership) has established its position as a workable indicator of social inequalities in health and mortality.

2.3.2 The concept of social class

Social class has assumed various roles in health-related studies: confounder, risk factor or a variable descriptive of the study sample (Liberatos et al. 1988, Marmot et al. 1995). What is actually meant by social class is not always well defined – a problem perhaps even more intensified at the present time (Marmot 1989, Macdonald 2001). In the context of health-related sciences, discussion on 'class conflict' and the 'theory of class action' is less salient. The following discussion thus focuses on the characteristics and components of class (the theory of class formation) that are related to mortality. In this respect, what is more relevant than the possession of wealth and the means of production in definitions of social class (Dahrendorf 1959, Giddens 2001) are occupational circumstances, social relations, living conditions, life styles, and access to 'life chances' (see Dahrendorf 1959:219, 249-50,317, Weber 1968:964, Hasan 1988, Scott 2002: 24,25). Social class is the structural context of women's and men's lives rather than their individual property (see Sørensen 1991, Annandale & Hunt 2000;22). Belonging to the particular social class is likely to affect its members' life circumstances. However, people may possess many characteristics that are common to the class when they enter into it (Graham & Graham 1985), for instance their life styles are likely to be affected by their education and upbringing (Weber 1968).

Denny Vågerö (2000) remarked: '*How to measure social position is a different issue than how to understand the causal pathway between social position and health*.' The main theme of this thesis is to study how social class is related to death through socio-economic and demographic factors preceding social class. When we understand how social classes are formed, where and how people are recruited to them and what constitutes them, we are in a better position to understand how and through what pathways and components the social environment affects health and mortality.

Mobility

Social mobility is one mode of social class formation, but it is also a measure of ine-quality of opportunity in society (see Sørensen 1991). In open societies, high levels of social mobility may have a salient influence on the characteristics and coherence of social classes. Mobility is more limited during the occupational career, although it may encompass changes in prestige, while in inter-generational mobility crossing class boundaries is common at the beginning of the employment career (see Goldthorpe 1980). However, there may be differences how permanent some moves to some social classes are. For instance, the intermediate non-manual class is characterized as a 'waiting room': some mobility into it may be temporary or at least is experienced as such pend-ing moving even higher (Goldthorpe 1980, Spruit 1989).

Due to the changing occupational structure, inter-generational social mobility has been and still is mainly directed upwards between the manual and non-manual class bound-aries. Thus, the non-manual class is increasing in absolute and relative size and is becoming more heterogeneous as it recruits educated people from the manual class. The manual class has become more homogeneous from the intergenerational perspec-tive because of low downward (both inter and intra-generational) mobility and the shrinkage of the farming class, which was an earlier source of labour (Pöntinen 1983). If people make normative compromises between the lifestyles in their destination and origin class, it affects social distinctiveness of the ways of life in the non-manual class (Goldthorpe 1980:264). In addition, those who remain in the manual class may adopt some habits from their friends and relatives who have ascended to the non-manual class. (Goldthorpe 1980:212,264). This has been thought to substantially reduce the differences in lifestyle between the social classes. Moreover, as unemployment has become established as a permanent phenomenon and temporary employment relations have increased, it seems that class no longer reflects the dynamics of the modern world – and that it has therefore decreased in significance. However, social mobility is not likely to eliminate class as a source of prestige, social identity and reward, and also of minor privileges, and lifestyles such as leisure-time activities are also related to occu-pation. Class relations are restructuring, but they still exist and have an effect on life chances and conditions of living. (see Dahrendorf 1959:60, 70, 220-2, Scott 2002).

The non-manual class and the manual class

There are several different classification schemes that have been used to stratify the population according to occupational title (Giddens 2001, Erikson & Goldthorpe 1992). Occupational class is frequently used in health-related sciences not only because the schemes are available and traditionally used, but also because the multifaceted entity of social class operates through work to a large extent. Work is a central factor in life: it provides a core social identity outside of family life, takes up most of one's active time and also influences one's leisure time (Giddens 2001:62, 287, Marmot et al. 1999).

Classifications of occupational social class draw a dividing line between non-manual and manual classes. People in 'a non-manual class' are in very miscellaneous occupations and, strictly speaking, they do not form a uniform group, class or stratum (Dahrendorf 1959:52). In the Marxian sense, even the upper levels of the non-manual class do not necessarily belong to the 'property class' or the 'upper class'. They are dependent on their salaries, thus in that regard are not so different from the manual class (Weber 1968:994). In addition, the lower strata of the non-manual class are close to the manual class in terms of levels of autonomy and authority, while the upper strata have high levels of autonomy, authority and prospects of promotion close to those in the *upper class*. (Scott 2002, Giddens 2001, Dahrendorf 1959, Weber 1968:963.)

Automated factories have brought about the division of the manual class into skilled and unskilled workers. At first it was assumed that unskilled workers would totally replace skilled workers. (Dahrendorf 1959.) Nevertheless, this division of the manual class according to skills has remained, and further automation has lead to the demand for highly specialized workers. Jobs for unskilled workers have become less common and are concentrated in a few branches of the construction and service industries. There are differences between skilled and unskilled workers in responsibility, authority, prestige, and wages - in terms of quantity and form of payment -, and therefore they may be considered to form two separate classes.

Occupational social class and gender

Assigning women to an occupation-based social class is even more problematic than social class classification in general (Kitagawa & Hauser 1973, Moser et al. 1990, Martikainen 1995c, Mackenbach et al. 1999, Krieger et al. 1999). First, the basis for many occupational classifications is male oriented, and as a consequence the distribution of women is biased, most of them being clustered in the lower non-manuals (Annandale & Hunt 2000). In addition, men and women may be in very different occupations within the same social class. The segregation of the labour market is very pronounced in the Nordic countries (Sweden, Finland and Norway), where public employment is common and dominated by women (see Kosonen 1998, 174), and where women's participation in employment is high.

Second, women may experience status incongruity between potential (measured by their educational achievement and previous working experience, for instance) and current social class due to domestic commitments, which may further be related to fragmented employment. However, status incongruity is not confined to women: there are also men who would be assigned to different socio-economic groups depending on whether the assignment is based on their education, social class or income (see 2.3.4) (Liberators et al. 1988, Geyer & Peter 2000, see also Macintyre 1986:399, 400).

Third, women's occupations at all stages of their lives do not correspond with their social circumstances at home, which are also influenced by their partner's occupational social class. (see Annandale & Hunt 2000). Accordingly, some mortality (Vågerö & Lundberg 1995, Sacker et al. 2000) and health (Krieger et al. 1999) studies have revealed greater socio-economic differences in household-based measures of social class than in individual-based measures.

Fourth, because of problems in assigning non-working women to a social class, many analyses are restricted to the economically active population. However, the exclusion of economically inactive persons leads to a serious underestimation of social class differences in mortality, especially among women (Martikainen & Valkonen 1999a).

For all of these reasons, it has been suggested that household-based measures (taking account of both spouses' occupational positions) or educational attainment should be used with women. As family roles are a salient part of women's lives, and consequently in some phases of their lives attachment to the labour force may be weaker, a wider range of characteristics reflecting their social and economic standing, such as their marital status, hours worked and access to a car, should also be taken into account (see Haavio-Mannila et al. 1985, Moser et al. 1990, Arber & Khat 2002). This is highly recommendable in cases in which marital status and employment status are highly correlated (see Goldblatt 1990). In Finland, where the participation of women in full-time work is high (Statistics Finland 2001) regardless of their marital status and number of children, occupation-based social classification may be as feasible for them as it has been found to be for men (see Goldblatt 1990, Sacker et al. 2000). In fact, studies on Finland have shown that the association between occupation-based class and mortality is similar regardless of whether the class is based on the woman's own or her husband's occupation (Martikainen 1995a).

2.3.3 Other socio-economic dimensions and mortality

In health-related studies it has not always been possible to define occupational class in a uniform way for different sex, age and employment groups. This is one reason why other indicators of socio-economic status have also been used. Essentially, indicators, or attributes, of socio-economic status such as education, income, savings, property, housing, residential status and prestige, and also access to them, are likely to exert an active influence on health and the risk of death. Moreover, these attributes should be differently distributed in the population as a consequence of class structure. (Townsend & Davidson 1992, 109, Bartley et al. 1999c, Siegrist 2000).

Occupational class, education and income are the most-often used indicators of socio-economic status (Spruit 1989, Liberators et al. 1988). However, rather than being parallel indicators, they reflect different dimensions of socio-economic status or social

stratification in concordance with Weber's status and class domains (see Liberators et al. 1988). The advantage or disadvantage of being in a particular occupational class may be conditioned by other socio-economic characteristics (Sørensen 1991). In other words, the effect of being in the manual class, for instance, may be different for those with higher education than for those with basic education (see also Backlund 1999, Macintyre 1997).

Many studies have found that education, occupational class and income have independent effects on various measures of health, health behaviors, and mortality, while the mutual correlations have remained relatively small (Kessler 1982, Liberators et al. 1988, Sorlie et al. 1995, Geyer & Peter 2000). This would strengthen the suggestion that occupational class, education and income form different dimensions of social class, which in turn affect health through different intermediaries. For instance, Bartley and her colleagues (1999b) showed that the mechanism that mediated the effect of socio-economic status on cardiovascular risk factors depended on the type of measure; the measure related to social advantage and lifestyle (Cambridge scale) was associated with health-related behavior, and that related to employment relations and conditions (Erikson-Goldthorpe) was associated with work control. Health-related behaviors have frequently been suggested to be more strongly related to education than to occupational class (Winkleby et al. 1992, Power & Hertzman 1997), although there are discrepancies between different studies on this issue (Davey Smith et al. 1998a). Further, educational attainment has been said to reflect personal traits and cognitive abilities (Elo & Preston 1996). On the other hand, occupational class may also reflect the effect of these characteristics. Moreover, whereas occupational class has been thought to reflect the effects of actual income, education has been thought to reflect the effect of potential income. In practice, the effect of education has been shown to be mediated to a large extent through actual income and material circumstances (Elo & Preston 1996, Schrijvers et al. 1999).

Educational attainment and social class reflect exposure to different phases of life, although such exposure is not always easy to determine unambiguously. For instance, parental social class exerts an influence on children's communication skills and level of socialization. Due to differences in educational readiness children with different backgrounds show differences in their ability to utilize the knowledge they gain at school, and this affects their likelihood of achieving higher social class. (Townsend & Davidson 1992:113.) Therefore, education reinforces class structure, although its independent effect is very difficult to distinguish from the effect of the parental class. Furthermore, this example showed that these different measures of social structure, parental class, education and social class, are hierarchically or intersectionally ordered: parental class is associated with education, education is an important determinant of occupation, and occupation constitutes a continuous opportunity for income.

Occupational social class does not encompass all of the features included in the socio-
logical concept of class, and perhaps it is merely a status group or an indication of
stratification. According to Wilkinson (1986), if we knew what social class was, we
would use that concept instead of occupational class. However, occupation-based so-
cial class has been associated with mortality among the previously or currently em-
ployed, and predicts the mortality of their children (Östberg 1986, Rimpelä 1993) and
partners (Martikainen 1995a, Marmot et al. 1995). These associations show that occu-
pational social class reflects circumstances of life other than those directly related to
occupational conditions. Thus it serves as meaningful and grounded way to distin-
guish social groups that have decisively common features in life circumstances and
opportunities (see Sørensen 1991). Therefore, (occupational) social class facilitates
description of the social inequality prevailing in society, and identification of the struc-
tural determinants of differential mortality.

3 Explanations of social class differences in mortality

3.1 Introduction

Social class has been found to be inversely related to most causes of death in women and men of different ages (see above). This gradient has endured, although the distribution patterns have changed in the whole population and within each social class, as new diseases have become prevalent and former killers have become extinct (see Marmot 1994, Hertzman 1999). This perseverance of the inequality in changing social circumstances and disease incidence has led to the need to find an explanation for these differences. The Black Report in 1980 (Townsend & Davidson 1992 – but mentioned earlier by Brotherston in 1974 (Davey Smith et al. 1994)) gave four types of explanation, artefact, social selection, behavioral/cultural and materialist. This framework has since been used as the basis of many studies and models of social class differences. Nevertheless, there is variation in emphasis on the importance of different explanations, and new mechanisms have been introduced (see e.g., Lundberg & Vågerö 1988, Williams 1990, Feinstein 1993, Davey Smith et al. 1994, Vågerö & Illsley 1995, Macintyre 1997, Elstad 2000, Koivusilta 2000).

Since artefactual theories have been dominant in advocating caution in the measurement and conceptualisation of health and class variables, but not in explaining their existence, they are not discussed further here (Fox et al. 1986, Vågerö & Illsley 1995, Davey Smith et al. 1994). What is common to different causal explanations is the idea that social circumstances and conditions exert an effect on health (Figure 3.1). In order to account for social class differences in mortality, exposure to the factors that decrease or exacerbate mortality risk ought to vary by social class, or then their effect should be differently conditioned in various social classes.

According to explanations based on direct or indirect selection, health differences by social class are, at least partly, a consequence of the allocation of people into different social classes according to health-related characteristics (see e.g., Graham & Graham 1985, West 1991, Blane et al. 1993). These different explanations are not mutually exclusive, and there are features that are relevant to several sections below: for instance, psychosocial pathway could be discussed in relation to relative deprivation and behavior. Furthermore, these explanations are introduced in terms of the lifecourse, which directs the focus of the whole chapter.

Figure 3.1 The association between adult social class (SES) and mortality accounted for by causation and selection explanations. Simplified models.

Time in lifecourse

Childhood

Adulthood

a) Causation

Bi

SES

M

Death

b) Direct selection

Health

SES

Death

Childhood

Adulthood

c) Indirect selection

Ci

Yi

Bi

SES

Death

d) Causation & indirect selection

Ci

Yi

Bi

M

SES

Death
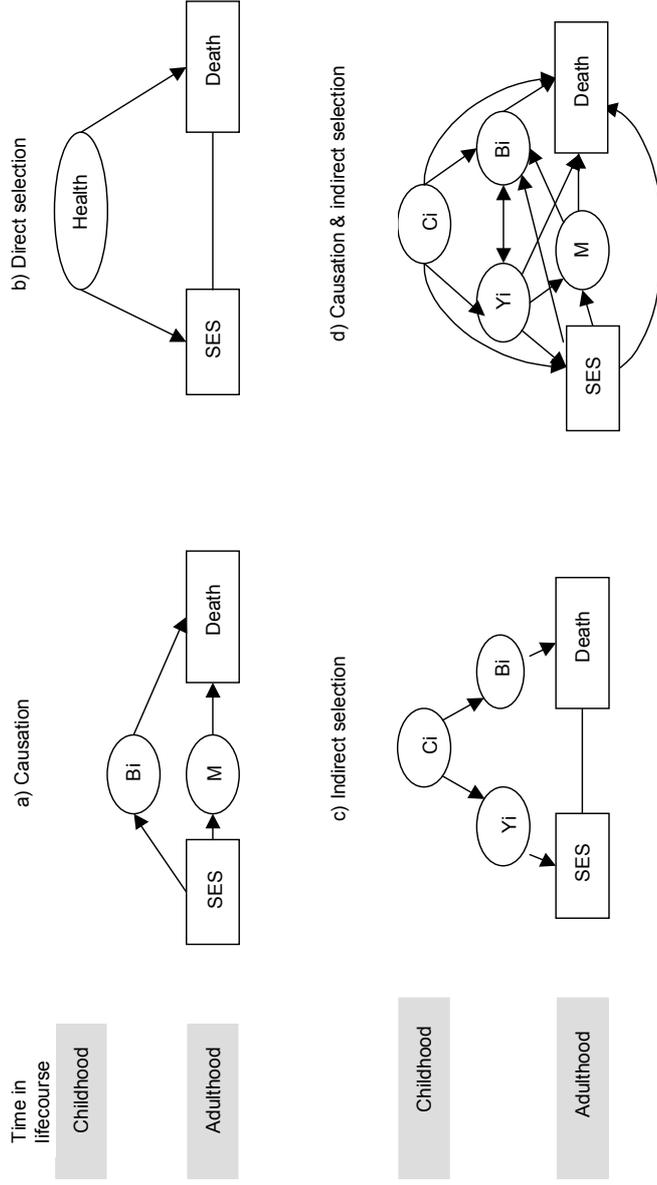
Abbreviations: **SES**= social class, **C**= social and material living conditions in childhood but also personal characteristics, **Y**= educational attainment, family formation, employment path **B**= health-related behaviors (smoking, intoxication, diet, sleeping patterns, exercise) **M**= material conditions related to social class (e.g., income, housing conditions)

3.2 Explanations based on causation

3.2.1 Materialist and structuralist explanations

In the most reduced form of the material explanation, social circumstances affect the determinants of health mainly regardless of people's characteristics or actions. People are exposed to conditions connected to their social position (see Blane et al. 1996a). In the Black report, material deprivation was divided into absolute and relative versions of deprivation (Townsend & Davidson 1992). Absolute deprivation means a lack of economic resources (Shaw et al. 1999) to fulfil the needs in the basic domains of life, and includes poor housing, malnutrition (meaning not only a lack of food, but also insufficient in terms of necessary nutrients), unsafe and polluted living and work environments, and shortcomings in hygiene and health care. In other words, poverty accounts for social inequalities in health. Absolute deprivation was a more relevant explanation in times when the major fatal diseases were more directly related to poverty than now, when the most common causes of death are cardiovascular diseases, cancer and accidents – which is not to say that these causes of death are unrelated to economic circumstances (Townsend & Davidson 1992:106).

The mere existence of a social gradient in health, for which there are no obvious biological reasons (Marmot et al. 1995), and its persistence in changed circumstances (in societies, which at least in theory, have reached a certain threshold level in the living and working environment, and in which all population groups have adequate material resources to live healthy lives and gain access to health care) have been interpreted as an indication of relative deprivation (Brunner & Marmot 1999).[1] One idea of relative deprivation is captured by Mr Townsend's well-known definition of poverty (1979:31): "*Individuals, families and groups in the population can be said to be in poverty when they lack the resources to obtain the types of diet, participate in the activities and have the living conditions and amenities which are customary, or at least widely encouraged or approved, in the societies in which they belong. Their resources are so seriously below those commanded by the average individual or family that they are, in effect, excluded from ordinary living patterns, customs and activities.*" According to this definition, poverty is related not only to the lack of necessities, but also to the lack of items *perceived as necessities in the prevailing society*, or to the *relative lack of access* to the fruits of a wealthy society (Marmot et al. 1995).

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[1] However, the existence of the social gradient – systematic differences between affluent groups (see Whitehall studies) and not only between the bottom and top groups - may not give sufficient proof of ˈrelative deprivation'. A linear association has also been found between social conditions and outcomes, such as growth retardation in the foetus, for which relative deprivation is not perhaps the most likely explanation (Vågerö and Illsley 1995).

Therefore, although the basic needs may be fulfilled in all social classes, there may still be structural differences in the possession of goods and resources, and in the chances of gaining possession (see Orwell 1945:87). Income inequality is a salient manifestation of relative deprivation (Wilkinson 1986, 1999, Marmot & Wilkinson 2000). However, there may be differences between population groups in other factors, partly independent of incomes, which account for social class differences in health. For instance, social class differences in mortality exist in all Nordic countries (Kunst 1997) where income inequalities are very small (Kosonen 1998, see Martikainen & Valkonen 1999b). In this context, influential factors might include differences in housing conditions, access to amenities (Stronks et al. 1998a), educational possibilities (Pöntinen 1988, Valkonen et al. 1996, see 5.2.2), living environments (Haan et al. 1987, Carstairs & Morris 1989), working conditions (Lundberg 1991b, Siegrist 1996, Vågerö & Lundberg 1995, Karasek et al. 1998, Bosma et al. 1998a, Jonge et al. 2000), health care (including variation in the latent time of disease and quality of treatment) (see e.g., Hetemaa et al. 2003), and in the resources to cope with stress (see psychosocial explanations below). In addition, people in lower classes have to make greater efforts to achieve the living conditions and amenities that are common in the upper classes, such as a decent home in a good location and access to hobbies for children. This pursuit may expose them to prolonged stress (see Goldthorpe 1980:238-52, Hasan 1988). In this respect, relative deprivation is an objective concept; health inequalities are not necessarily based on people's subjective perceptions of their position in the current social hierarchy.

According to the 'neo-material interpretation', health inequalities are accounted for by *'the differential accumulation of exposures and experiences that have their sources in the material world'* (Lynch et al. 2000). Differences in material circumstances, and also in other living conditions, are an essential part of the social structure (see Macintyre 1986, Valkonen 1995:317, Elstad 2000:25). Attention should also be directed to the ways in which the social structure leads to the *clustering* of advantage or disadvantage, in numbers, length, and level (Davey Smith et al. 1994), *over the lifecourse*. The neo-material explanation is not a pure material explanation as it incorporates other pathways such as psychosocial explanations.

3.2.2 Psycho-social explanations

Psychosocial factors form one plausible mechanism for determining how external circumstances affect psychological well-being, health and risk of death among individuals and population groups – in other words, social inequalities in mortality.

The term 'psychosocial' is used very commonly in various connections including causes, risk factors, mediating factors, contexts and outcomes (Martikainen et al. 2002). The

salient idea behind it is that social and psychological phenomena are combined, i.e. the identity of the psychological and social entities and processes is to be found in their relations and representations (Spicer & Chamberlain 1996). According to this pathway, stress experienced in the *social* environment has an effect on the *psychological* processes, which in turn influences the neuro-endocrine pathways, i.e. the sympatho-adrenaline pathway (SA-axis) and the hypothalamus-pituitary-adrenaline axis (HPA-axis). This induces pathophysiological changes and thus finally leads to disease. (Brunner & Marmot 1999, Hertzman 1999, Ursin & Eriksen 2000, 2001, Steptoe & Marmot 2002, Sluiter et al. 2001, Karasek et al. 1998, McEwen 1998.)

People in different social classes are confronted by stressful and strenuous situations and mundane stressful events with different frequency (Steptoe & Marmot 2002, see also Hasan 1988 and Macintyre 1986). Prolonged adversities may lead to allostatic load, i.e. an imbalance in the systems (e.g., in the cortisol level) that promote adaptation to internal (such as infection and glucose challenge) or external challenges. This, in consequence, leads to higher susceptibility to disease, or it may have an exacerbating effect on illness (McEwen 1998, Brunner & Marmot 1999, Kristenson et al. 2001).

The experience of stress stimuli and responses to it are not determined only by the current circumstances. Exposure to the social environment and to experiences in early life modifies a person's experience and appreciation of the stress stimuli. The resources to respond to the stimuli at an adequate level, and the ability to revert when activation of the neuroendocrine pathways is no longer needed, are also related to early experiences (McEwen 1998, Ursin & Eriksen 2000, Kristenson et al. 2001, see also Stronks et al. 1997). For instance, studies on animals have found that maternal care, or possibly long periods of maternal separation in infancy, have long-term effects on stress-mediating pathways, expressed in behavioral and endocrinal responses to stress (Francis & Meaney 1999, Caldji et al. 2000).

An individual's commitment to and interaction with his or her immediate surroundings (primary group relations), and society in general, affects health or disease defences and 'general susceptibility', which in turn affect morbidity and mortality risk through psychological processes (see Lundberg & Vågerö 1988:55, Syme & Berkman 1976). Social support (or a negative form of it, which may actually be more influential) has been found in many studies to be related to mortality through its reciprocal behavioral, cognitive and affective component (Stansfeld 1999); however there is, as yet, no convincing evidence of its contribution to social class differences in mortality.

Social interaction in adulthood is likely to be based on experiences earlier in life, and other factors in adult life may similarly have their basis in social interaction in early life: the ability to develop positive social relationships is likely to be dependent on

satisfactory early relationships with one's mother, father or other caretaker. Disturbed attachment may exacerbate the development and maintenance of future relationships, and also lead to the substitution of relationships with health-deteriorating behaviors and increased stress (Stansfeld 1999, Suomi 1997).

3.2.3 Behavioral explanations

Cigarette smoking, physical activity, alcohol consumption, diet and sleeping patterns have been shown to have strong separate and joint associations with mortality risk (e.g., Macintyre 1986, Feinstein 1993, Davey Smith et al. 1994). These behaviors are differently distributed between population groups in that there is more health-damaging and less health-promoting behavior in the lower social classes (e.g. Macintyre 1986, Marmot et al. 1995).

Lynch and his colleagues asked: "*Why do poor people behave badly*?" (1997), and Rutter (1989) answered: "*People act in ways that serve to shape their experiences and there are equally important links between different types of environment*." In other words, the differences in health behaviors are related to differences in other elements between social classes; behaviors are to large extent embedded in social structures and cultures, which are often transmitted from one generation to the next, and constrained by material factors such as incomes, the environment, and opportunities to practise and accessibility to certain behaviors (Townsend & Davidson 1992:110, Macintyre 1986, Marmot 1988, Vågerö & Illsley 1995, Link & Phelan 1995, see also Mare 1990, Lundberg 1991b, Whitehead 1992, Lynch et al. 1997). Material conditions may affect a particular behavior by promoting it, such as smoking, alcohol use and a sedentary lifestyle, which may be resorted to in order to alleviate the stress caused by non-satisfied needs (Graham 1994). Material conditions may also, in some cases, affect behavior by limiting or hindering the practise of it, such as purchasing safe cars, using effective health services, getting exercise and following a healthy diet (Stronks et al. 1997, see also Williams 1990, Carroll et al. 1993). Approximately half of the effect of health-related behaviors on health inequality is assumed to relate to material circumstances mediated by these behaviors (Stronks et al. 1996). Moreover, psychosocial pathways may also have behaviorally mediated effects on health, and behaviors such as drinking and following an unhealthy diet may also affect psychosocial pathways (Syme & Berkman 1976, Eriksen & Ursin 2000, McEwen 1998 and 2002, Gorwood 2001).

However, health-related behaviors are not fully determined by material circumstances. The "hard form" (see Macintyre 1997) of behavioral explanations underscores the decision of people to choose the (collective) lifestyles of their particular group (albeit they might have chosen the group); membership of the group is reinforced by a mutual lifestyle, distinguishable from others (see 2.3.2). In this respect, youth culture may

have a particular importance through peer groups because youth health-related behaviors, such as substance use, involve social activities (West 1997, Friedman 1989, Glendinning 1995, Koivusilta 2000). There are also behaviors that are not based on a person's own choice, such as whether to take part into a vaccination programme as a child. Smoking and alcohol use are also examples of behaviors that may affect the health of other people. For instance, approximately one-third of the association between social class and birth weight, and one-quarter of that between social class and perinatal mortality, is attributable to smoking (Davey Smith et al. 1994:138).

Smoking and the excess use of alcohol are among the most risky behaviors with respect to mortality. Although smoking has not accounted for social class differences in mortality (Marmot et al. 1984, Davey Smith et al. 1994), it has made a clear contribution. For instance, in Finland, approximately one fifth of the 5.1 year difference in life expectancy at age 35 between men with higher and basic education, and one tenth of the 3.2 year difference in women, are attributable to smoking (these preliminary figures are based on Martelin's presentation in 1998). Deaths related to alcohol use accounted for 24% of the difference in life expectancy at age 20 in men, and 9% in women. The contribution of alcohol-related causes to differential mortality was greater for external causes, in younger age groups, and in men. (Mäkelä et al. 1997a.)

The relation of health-related behaviors to adult class seems to be stronger than to childhood class (Blane et al. 1996b, Wannamethee et al. 1996). However, childhood and youth are likely to be important times in respect of the development of a person's behavior or lifestyle, because health-related behaviors are mainly established in adolescence (Lenthe et al. 2001, Lynch et al. 1997). Social conditions in the parental home, such as parental smoking and alcohol use, 'neglectful' or authoritarian parenting, family type, and social integration into the parental home, together with peer groups and the school context, have been shown to have an effect on a person's own health-related behaviors in youth (Glendinning et al. 1997, 1995, Jackson et al. 1997, Shucksmith et al. 1997). Experiments accompanied by *"exuberance, a lack of experience, a sense of invulnerability, and the need to demonstrate independence and courage"* belong to youth (Friedman 1989). The parental home and environment may modify the "experiments" that are often related to youth culture, or their consequences, by affecting how often and how much alcohol is consumed, and how permanent the behavior will be, for instance (Glendinning et al. 1995, Koivusilta 2000, Karvonen & Rimpelä 1997). Health-related behaviors in youth are important in terms of later social inequalities in that they have been found to be associated with subsequent health and social class (see West 1991, Pietilä et al. 1994, Pietilä & Järvelin 1995).

Any estimation of the effect of health-related behaviors on social class differences in health should take into account the development of behaviors across the *lifecourse,* and their association with other mechanisms affecting mortality and social class.

3.3 Explanations based on selection

3.3.1 Direct selection

According to theories of direct health-related selection, or 'reverse causation', social class differences are based on the rise of healthy, robust, vigorous and agile persons to the top of the social hierarchy, while weak, unhealthy and frail persons drift to the bottom (Townsend & Davidson 1992:105, Power et al. 1991:62). A key factor in this explanation is social mobility between or within generations (see e.g., Stronks 1997, Goldman 2001). The effect of selection on social class differences is likely to be most salient when people move from their parental class to their own class via education and early employment experiences. Health status is an influential factor in this process (Illsley 1986, Stern 1983, West 1988,1991, Mackenbach & Maas 1989:50). The clear increase in health inequality in early adulthood could be due to health-related inter-generational social mobility (West et al. 1990). In general, there is evidence that child-hood health status has an effect on mobility (although there is also a contradictory evidence that health had no direct effect (Lundberg 1991a)) (Wadsworth 1986). Approximately 5 to 10% of the increased risk among the lower educational groups of having less-than-good perceived general health is attributable to childhood health, and this effect cannot be accounted for by parental class (Mheen et al. 1998b).

Health-related selection has also been shown to have an effect on social class differences in mental illness (Antunes et al. 1974, Turner & Gartrell 1978, Rodgers & Mann 1993), but this effect is likely to be small (Lundberg 1991b). Moreover, if there is an adequate disability-pension system as in Sweden, health-related intra-generational mobility is not likely to affect adult social class differences because ill health is likely to result in leaving the labour force rather than in downward mobility (Lundberg 1991b). In general, the contribution of direct selection to social class differences in mortality, and in health in general, is likely to be small (Fox et al. 1986:47, Wilkinson 1986, Whitehead 1992:315, Power et al. 1991, Blane et al. 1993, Carroll et al. 1993, Rah-konen et al. 1997a).

What is interesting in theories of health-related selection is the notion that health and social position are in mutual interaction: health exerts an effect on social position or type of employment (full-time or part-time), and correspondingly, health-enhancing or health-deteriorating social circumstances exert an effect on health status (Vågerö &

Illsley 1995, Elstad 1995, Mheen 1998:3-4, Elstad 2000:116). This idea applies life-course approach as it has a longitudinal perspective on health status and adult social class.

3.3.2 Indirect selection

The common feature of theories of indirect and direct selection is that current social differences in health are attributed to some factors in the past (Figures 3b and 3c). According to the direct selection approach, previous health status affects social status and thus social inequalities in health, while under indirect selection, health inequalities are considered a consequence of some factor that has exerted its influence on both subsequent health and social position (see Wilkinson 1986, West 1991). Such factors include health-related behavior, personal characteristics and attitudes such as 'orientation towards the future', body height, educational attainment, coping skills, and sometimes also childhood conditions (Blane et al. 1993, Schwartz 1995, Vågerö & Illsley 1995, Mheen 1998:17, Goldman 2001).

A good example of indirect selection is given in a study by Nyström (1992), which showed that shorter persons were more likely to be downwardly mobile and taller persons upwardly mobile. Moreover, a larger proportion of downwardly mobile and non-mobile men and women had longstanding illnesses than the upwardly mobile. According to the same study, however, mortality was not associated with mobility – possibly due to the small numbers of deaths.

Illsley's (1986) observations of a positive association between the mother's height and educational achievement with their baby's birth-weight and perinatal mortality are indicative of indirect selection, although often interpreted as some of the most influential empirical evidence of the effects of health-related selection. The difference between direct and indirect selection is not always unambiguous. For instance, Dahl and Kjærsgaard (1993) regarded selection based on 'health potential', which they further defined as "*the factors that may influence both social mobility and manifest health, e.g. mortality*", as health selection and not indirect selection (see also Stern 1983).

Furthermore, whether they be 'health potential', social circumstances in early life or educational attainment, the factors generating the process in which health and social position are mutually interrelated leading to social inequalities in health are often the outcome of social processes (see e.g., West 1991, Blane et al. 1993, Davey Smith et al. 1994, Fox et al. 1986). Thus indirect selection is linked very closely to social causation. Whether they differ from each other is a matter of perspective and definition (Mheen 1998:4). For instance, Lundberg considered the effect of childhood living conditions to be part of 'indirect selection' (1991a), but also a causative factor (Lundberg 1991b).

Moreover, it is not clear that the effect of 'indirect selection' on social class differences in mortality are confined to the time of entry into the labour market (see Davey Smith et al. 1994), because the processes that lead to adult circumstances may continue to exert an influence on health in interaction with current exposures (West et al. 1990). In fact, the term 'selection' in this context is dubious (West 1991, Blane et al. 1993). Lundberg (1991a) suggests 'health-and-social-mobility potential' as a more adequate term (West's suggestion (1991) was 'health-related-occupational-mobility'), although he admitted it was 'clumsy'.

The notion of mobility needs further specifications. Whether it is health or some other factor that exerts an influence on a person's subsequent position, there is always the possibility that this position is the same as that person's initial social position, and therefore no social mobility occurs intergenerationally or intragenerationally. Although mobility from one's parental social class to one's own class that is based on indirect mechanisms is likely to be more significant in terms of social class differences in health (West et al. 1990), intra-generational mobility may also contribute to these differences. For instance, alcohol or drug abuse may hinder promotion in one's occupational career (see e.g., Dahl & Kjærsgaard 1993 on persons failing to move up).

The effect of social mobility on social class differences in health may depend on its nature. Direct selection is likely to slightly increase health inequalities if those moving upwards are healthier than those already in the upper echelons, and those who are moving downwards have poorer health than those in the lower class that they have joined (see Stern 1983, Dahl & Kjærsgaard 1993, Figure 3b). However, there is more evidence of constraining rather than increasing social class differences in studies on social mobility (Bartley & Plewis 1997, Blane et al. 1999, Elstad 2001). When 'indirect selection' is seen as a causative explanation, people may be assumed to carry with them health-promoting favourable factors that affect their mobility, and also their childhood exposures and further disadvantages accumulated in youth (Blane et al. 1993). Therefore, mortality risk among people who have moved up to the non-manual class may be higher than that of people who started out there because of the effects of their more adverse childhood. Correspondingly, people moving down to the lower classes may have a lower mortality risk than the descendants of the manual class due to their more favourable early exposures.

3.4 Summary

Lower social classes have higher morbidity and mortality from various causes with different aetiologies. There are different explanations based on causation and selection, which describe how health determinants and social processes interact in society

and thereby produce health inequalities (see Mheen 1998, Elstad 2000). However, none of these different models of explanations have fully accounted for health inequalities (see Davey Smith et al. 1994, North et al. 1993, Marmot et al. 1995). Back in 1986, Macintyre remarked that, instead of confining ourselves to specific explanation models, we should explore *the processes* linking social position and health (Macintyre 1986). Materialist, psychosocial and behavioral explanations related to indirect selection could be seen as such a process, or mechanism, that generates social class differences in health. These explanations are not mutually exclusive, but they affect health simultaneously through complex social and biological processes (Figure 3d and e.g., Lichtenstein et al. 1993, review by Macintyre 1997, Goldman 2001).

Valkonen (1995) divided the line from social class to disease into three levels. First, a disease has to have specific physiological risk factors that affect it in a similar manner in all societies and in all subjects of the same age and sex, and whose genes are not aberrant. Second, the level of physiological risk factors is determined by health-related behaviors and other social risk factors, which again exert a similar effect on people under the conditions stated above. Finally, these social risk factors are determined by social class, which is affected by the conditions of the specific society at the specific time.

If our aim is to disentangle the processes leading up to social inequalities in mortality, we need information from all levels. First, we have to know the physiological risk factors for specific diseases. Second, we have to know how these risk factors are distributed across social classes. Third, we have to find out why these risk factors are distributed differently between social classes (Marmot et al. 1995), and to do this we have to take into account the effect of macro-level factors on gender, age and ethnic group (Macintyre 1986 and 1997, Graham 2002). Furthermore, the relative effect of a health determinant may depend on the social context (see Kuh et al. 1997:182, Bartley et al. 2000)

Information on current material circumstances, behaviors and psychosocial factors is not sufficient to explain health inequalities. Abuse of health accumulates during the whole life course (Marmot et al. 1995), and previous exposure modifies people's physiology, while response mechanisms to different stressors in the current environments differs in various social classes. Furthermore, attitudes, personality and health-related behavior, the factors that are also related to the indirect-selection mechanism, are affected by circumstances in the parental home, which give this explanation a clear lifecourse connotation. The next chapter reviews how all of the explanations introduced above may be combined with the lifecourse approach, and suggests how this approach could be related to social class differences in mortality.

4 The lifecourse approach

4.1 Paths from the past to the present

From early to current studies

Earlier research has convincingly indicated that the risk factors prevailing in adulthood are important with respect to the progress of diseases, but that these factors do not solely account for morbidity or social inequalities in morbidity or mortality (see Kuh & Davey Smith 1993). According to Mare (1990:365), "*The risks at any moment are a weighted function of all past activities*". In the other words, the origin of disease and premature death may lie in earlier phases of life.

The authors of the Black Report drew attention to the lifecourse by remarking that the explanation of social class differences in health were likely to be found in different mechanisms at different stages of life (Townsend & Davidson 1992:14). However, this approach did not sufficiently consider the possibility that the onset of disease may lie in the circumstances that prevailed decades previously, or the mechanisms by which different exposures at various stages of life accumulate or have a direct effect on subsequent health status or risk factors.

No discussion on the effect of different life phases on health and mortality is complete without acknowledging Kermack, McKendrick and McKinley (republished in 2001), Forsdahl (1973, 1977, 1978), and Notkola (1985). Kermack and his colleagues drew attention to the effect of the early years by showing systematically that ten-year birth cohorts had different mortality rates through their lives, probably due to the gradually improving conditions in childhood from generation to generation. This was also seen in the decreasing rate of infant mortality through improved maternal health. The importance of the year of birth in relation to the year of death in predicting mortality risk diminished after 1925 (Kuh & Davey Smith 1993). However, these early cohort studies revealed the possible pervasive effect of early living conditions on subsequent mortality.

Forsdahl analysed district-based, age-standardized mortality rates and found that people with Norwegian and Finnish backgrounds in Finnmark, Northern Norway, had elevated levels in comparison with the whole of Norway. He suggested that the higher mortality in these groups, in which those with Finnish ancestors fared worst, was probably due to permanent damage in the cardiovascular metabolism caused by nutritional deficiency in childhood. Although his suggestion of an interactive effect of childhood poverty and adult prosperity on cardiovascular risk (Forsdahl 1977, 1978) has not been proved

correct (Notkola 1985), the idea of interactive effects of childhood and adult conditions on the development of coronary heart disease has received support (Frankel et al. 1996).

Notkola (1985) showed in a population-based follow-up study that morbidity and mortality from coronary heart disease were more strongly related to social class in childhood than in adulthood. This result suggested that coronary heart disease was "*the result of long personal history*" (p. 96). Similar relations were not observed in other causes of death. Notkola also found that coronary heart disease was related to small body size in Eastern Finland but not in Western Finland. Adult risk factors such as smoking, high blood pressure and high cholesterol only slightly accounted for the higher risk among short men from eastern Finland and those from poor childhood homes. This difference is possibly accounted for by nutritional differences between Eastern and Western Finland at the beginning of the 20th century when the cohort members were children, and/or heredity (see also Koskinen 1994).

Pathways from early circumstances to adult disease and death

The origins of adult disease may lie in a specific critical or sensitive period, most likely in very early life, or in the accumulation of detrimental exposure across life, or even in the preceding generation (Kuh & Ben-Shlomo 1997, Ben-Shlomo & Kuh 2002). For example, bronchitis has been shown to be associated with poor parental home circumstances, and with a parental history of bronchitis, which may in turn be related to circumstances in their childhood (Wadsworth & Kuh 1997).

Cardiovascular disease and its risk factors, cancers such as stomach and breast cancer, and respiratory disease such as chronic bronchitis, allergies and diabetes, are degenerative diseases that have been found to have their origins in early life (Kuh & Ben-Shlomo 1997). However, the progression of these and other diseases is not likely to be only dependent on exposure in early and/or the current life. Between gestation and adult disease are many possible pathways with potential mediating and confounding factors (Ben-Shlomo & Kuh 2002). In general, the search for an explanation of premature death and its social variation should use information on different phases of life and focus on the different mechanisms mediating the effects of material, social and psychological circumstances on increased mortality risk (see Chapter 3). Theoretically, it is possible to distinguish two broad lifecourse approaches: biological and social pathways or programming (Power & Hertzman 1997). Biological programming describes the development of disease from exposure to onset, while social programming charts the socially determined paths along which exposure may accumulate in deprived social circumstances and lead to disease onset in adulthood. Although the mechanism on these paths may be biological or social in nature, the interrelationships are

very complex. Initial biological exposure may have social consequences and vice versa (Ben-Shlomo & Kuh 2002, Vågerö & Illsley 1995). This chapter trawls through lifecourse models by dividing them according to biological and social pathways, even if this is not always a good solution because of their reciprocal interactions. The focus in this chapter is also on in the development of disease and premature death risk across life. The effect of early factors on social class differences in mortality is only in the background, and is considered in more detail in Chapter 5.

Critical and sensitive periods

Central concepts in the lifecourse approach are the critical and sensitive periods that are specified times in human life at which exposure has an influence on the starting of any mechanism leading to the subsequent onset of disease or death. Ben-Shlomo and Kuh (2002:288) defined a critical period as '*a limited time window in which an exposure can have adverse or protective effects on development and subsequent disease outcome',* and a sensitive period as a time period when an exposure has a stronger effect on development and hence disease risk than it would have at other times. Critical periods may be more evident in chronic disease risk associated with developmental biological subsystems (Ben-Shlomo & Kuh 2002), while sensitive periods are particularly related to child development. As Sylva (1997:88) points out, '*A sensitive period may last for months or even years, and denotes the time in which the developing child is particularly responsive to certain forms of experience or particularly hindered by their absence'*. In addition, these sensitive periods are not necessarily confined to specific development periods, albeit their effects would be weaker at other times. They may be more applicable than critical periods in terms of social pathways (Hertzman & Wiens 1996).

4.2 Biological pathways

According to "biological programming theories", the factors triggering subsequent disease may be either genetic (e.g., there are two types of genetic diathesis for alcoholism (Cadoret et al. 1995)) or biological in nature (Barker & Osmond 1986, Kuh & Ben-Shlomo 1997). They may affect later morbidity after a latent period independently of later experiences (Wilkinson 1986:6, Frankel et al. 1999, Leon & Davey Smith 2000), or in interaction with subsequent risks (Frankel et al.1996). For instance, birth weight, independently of social background, has been shown to be associated with cognitive ability in childhood and early adulthood, probably through foetal and postnatal programming associated with the HPA-axis (Richards et al. 2002).

Furthermore, the origins of biological pathways may lie in the previous generation. For instance, a low birth-weight may be a consequence of nutritional deprivation in

the mother at the time of her own birth (Townsend & Davidson 1992:115-6, Wadsworth 1997a, Ben-Shlomo & Kuh 2002). Explanations based on biological pathways fall into two main areas: latency models depending on a critical period, and biological programming emphasising the development of risk in different phases of life and not necessarily associating a specific critical period with disease initiation.

The latency model

According to the latency model, a strong, discrete event or exposure in early life, before or after birth, initiates or programmes the subsequent development of disease without the interventions of later events (see Power & Hertzman 1997, Hertzman et al. 2001). A very salient example is biological programming in utero (Barker 1995,1997).

There are well-defined critical periods for specific tissues, i.e. the times during foetal life in which different body cells are divided. As a consequence of disturbances in growth due to under-nutrition, which has an effect only when it occurs during a brief, but critical period, there may be changes in the distribution of cell types, in patterns of hormonal secretion, in metabolic activity, and in organ structure (see Barker 1997, Eriksson et al. 2000). As a consequence of this programming, a person is diseased in middle adulthood regardless of exposure in later life; these later exposures, such as smoking and obesity, can only exacerbate the effect of the biological programming, which is unchangeable (see Barker 1997). For instance, small abdominal circumference at birth, possibly due to under-nutrition in late gestation, is associated with raised low-density lipoprotein cholesterol and plasma fibrinogen concentration in adult life, and further with mortality from coronary heart disease (Barker 1995). Another feature of the latent model is the independent effect of childhood circumstances (possibly helicobacter pylori) on stomach cancer and stroke (Davey Smith et al. 1998a, Davey Smith 2001). This evidence indicates that programming may take place after birth (see Lucas et al. 1998).

According to the model, the origin of disease is in early life. Subsequent factors during childhood and adulthood may only modify the effects of biological programming (Eriksson et al. 2000, Barker et al. 2001), or they may even change it. One such example is the case in which reduced foetal growth through insulin resistance does not increase the risk of diabetes if a person is not obese as an adult (Lithell et al. 1996). An equivalent trigger effect of adult obesity has been found for the association between low birth weight in babies and the increased risk of coronary heart disease (Frankel et al. 1996). It is likely that the latent model does not play a major role in generating social class differences in mortality in adulthood, because for this to take place the same childhood exposure that exerted an influence on adult disease in very early life would also have determined adult social class. Moreover, it assumes that youth and adult circumstances only have a minor effect on mortality.

The extended biological pathway

According to the extended form of the biological-pathway model, the development of disease may have its origins other than in a strictly-defined critical period, i.e. during sensitive periods. In terms of developing the immune system, childhood and adolescence may be sensitive periods, if it is not developed until adolescence (Hall et al. 2002). For instance, it may be possible that continuous stress in the parental home due to social or economic difficulties (Lundberg 1993, Rahkonen et al. 1997a) has long-term effects on the immune system, thus leading to adult disease.

The biological pathway is likely to affect social class mortality differences in interaction with the social pathway; both of these processes may originate in the same circumstances, such as in adverse living conditions in the parental home. It may be possible to identify the latent effect of the parental home on adult disease within this framework. For instance, shortness at age 7, measuring slow growth possibly as a consequence of disadvantage in early years over and above the chronic disease, had a stronger association with limiting illness at age 33 than at 23 (Power et al. 2000, Marmot et al. 2001). In addition, Power and her colleagues (2000) found that the effect of disadvantage in the parental home (i.e. a single-parent home, a large family, free school meals and a lack of hot water) on chronic disease and disability strengthened by age and thus indicated the latency effect of childhood circumstances, possibly operating with later assaults, on adult disease. An extended example of the effect of growth is given in Chapter 4.3, which also describes the social-pathway model.

Genetic programming

Biological programming may be based initially on genetic factors. On the individual level, this may be a major reason why a person is vulnerable to a specific disease. However, as Marmot and his colleagues (1995:174) remarked, "*The causes of social inequalities in health are likely to be different from the causes of individual differences, in which genetic factors will play a bigger role*". There is evidence to suggest that the effect of genetic programming on social class differences in mortality is minor. First, studies on immigrants have shown that disease patterns change in the new environment to resemble that of the indigenous population, indicating the effect of the social environment on morbidity (Marmot 1999:3). Second, a social class gradient has been found for most causes of death, and in all Western countries with different mortality patterns. This points to explanations other than genes, because different genes affect different outcomes. In addition, the fact that changes in mortality patterns have not lead to changes in patterning of mortality by social class also points to other explanations as it is unlikely that the gene structure of populations changes so quickly. Moreover, the increasing mortality differentials by social class indicate explanations other than genes. In fact, there is no evidence of a differential distribution of genetic

characteristics among socio-economic groups (Mheen 1998:18). Thus, the extent of genetic programming in the social distribution of disease is likely to be small (see also Lynch et al. 1998). This discussion is not continued further in this thesis.

4.3 Social pathways

Social programming, according to the definition put forward by Vågerö and Illsley (1995), means '*those social influences in early life which directly or indirectly determine adult health or which interact with adult experience to determine adult health*.' Because the links between different circumstances are considered probabilistic rather than deterministic in this model (see Ben-Shlomo & Kuh 2002, Rutter 1989), the term 'social-pathway' is preferred here. According to this model, early circumstances affect the paths, such as education, that lead to the adult position. These paths mediate the effects of early circumstances on health, but simultaneously modify them. In addition, the risks related to different phases of life may accumulate along the paths. Adult circumstances have an effect on health, which is likely to depend on antecedent exposures and experiences. Biological and social factors interact across this life-long process. (Blane et al. 1993, Power & Herzman 1997, Wadsworth 1997a, Hart et al. 1998, Power et al. 1999a). Vågerö and Illsley (1995) end up to rejecting 'social programming' as a one-sided concept, and instead suggest the 'co-evolution of health and social achievement' to depict this dynamic process.

In the following, the social-pathway model is divided into two elements, the pathway model and the accumulation model. This division is made to highlight differences in the accumulation of risks. The pathway model emphasizes the social process that has affected a person's social achievement and establishment of one's adult social class. Premature death may be a consequence of exposures along these paths, which run parallel to the accumulation model. However, premature death may also be a consequence of adverse concurrent circumstances due to which a person has ended up along paths influenced by unfavourable living conditions in early life. According to the accumulation model, disease and premature death in adulthood are consequences of long-term exposure to adverse social and economic circumstances, which have affected the accumulation of biological risks.

The pathway model

Lundberg's (1993) '*The unhealthy life career*' describes the pathway idea well. According to his hypothesis, poor social and economic conditions in childhood may affect educational performance, the choice of one's first job and, through these factors, one's later occupational career and health status (1991a). In other words, the effect of economic or social hardship on subsequent health is partly mediated through youth

paths, education and employment, and through adult living conditions, as he later showed (1997). It is possible to distinguish several paths, hence the term 'pathways', along which experiences and exposures may cluster cross-sectionally and accumulate longitudinally (see Blane 1999).

In human development, critical periods affecting social and psychological development and subsequent morbidity are the early years and different transitions (see 3.2.2, Sylva 1997, Hertzman 1999). Important transitions, i.e. socially critical periods, include that from primary school to secondary school, school examinations, entry to and exit from the labor market, leaving the parental home and establishing one's own, parenthood, job insecurity (change or loss of work), and the onset of chronic illness (Bartley et al. 1997). These times are 'critical' because, apart from the fact that, during them, exposure may have a stronger effect on subsequent development, it is also possible to change the course of one's social path and therefore the accumulation of risks.

There are several points worth noting in the pathway model. First, factors affecting health are socially patterned (see causal explanations above). Second, their effect may be long lasting. Thus, in the next stage of life, a person is exposed to socially-patterned new health-enhancing opportunities (a term used by Kuh et al. 1997) or to health-damaging material and psychosocial factors in their social circumstances at both micro and meso levels, i.e. the home, the neighbourhood, the school, the work place, and the leisure environment (Hertzman 1999). However, this person is still influenced by earlier factors. At each stage, the effect of new exposures may be added to earlier exposures (as with blood clotting), or older exposures may modify the effects of new ones. Therefore, this part of the pathway model incorporates the accumulation model as it describes the accumulation of health risks along a social path. However, it is important to take into account the time order of different causative factors affecting health inequalities, i.e. whether they mediate or modify the effects of factors preceding them (see Valkonen & Martelin 1988).

Third, people do not move from circumstance A to circumstance B by chance, and old experiences affect the likelihood of new ones. There is a 'chain of risks', a term that was perhaps first advocated by Michael Rutter (1989). Briefly, social events or conditions may affect subsequent health through biological or social chains of risks, which lead to the adult state including health hazards (Kuh et al. 1997). Fourth, stressful events may confront a person at any stage regardless of past and, to a certain extent, also of current circumstances. However, the ability to respond to stress stimuli depends on earlier circumstances and events that have affected response mechanisms and also probably intrinsic systems such as the HPA axis, all of which affect the stimulus level (see 3.2.2).

Sequels on social paths need not be negative. Favourable conditions in the parental home have been found to be associated with better subsequent social achievement and health (Blane et al. 1999). In addition, favourable conditions in later life may suppress the effects of unfavorable early experiences (see Power & Hertzman 1997), or modify the effect of current disadvantages. Blane (1999) calls this mechanism 'social protection'. For instance, after adverse experiences in childhood, including that of being taken into care, positive experiences in school may start a new path that leads to (steady) employment and a good marriage (Rutter 1989), and therefore may decrease the effect of early circumstances, although would not nullify them.

The accumulation model

According to the accumulation model, advantages and disadvantages accumulate over time and have an effect on health depending on their duration and intensity of exposure to the factors of interest (see Hertzman 1999, Hertzman et al. 2001). The following sentence in Wunsch and his colleagues' study (1996) describe the accumulative process across the lifecourse: "*Life or death is assumed to be dependent upon the type of states one has experienced during one's lifetime, the amount of time one has spent in each state, and finally the order of the states*." Therefore, it is not only the length of the exposure (accumulation), but also the order of the states that is important in terms of death risk. However, according to the 'pure' accumulation model, childhood conditions form the basis of the further accumulation of social and material disadvantages or health-enhancing factors, but the timing of these exposures to any specific phase of life is not crucial (see Carroll et al. 1993, Power et al. 1999a). In other words, the critical or sensitive periods may be less significant in a cumulative effect. However, even if this were true for some health outcomes (Hart et al. 1998, Power et al. 1999a), it is unlikely that it could be generalized across all health conditions and causes of death.

The stronger association of mortality with the longest-held occupation rather than with the current or last one has been interpreted as evidence for the effect of accumulation (Power & Hertzman 1997, Wunsch et al. 1996, Hart et al. 1998). (However, this may also be an indication of the stronger health risks related to the class circumstances in adulthood than in childhood.) There was also an indication of the accumulative effect of social circumstances on mortality in Fox and colleagues' study (1986), in which social class differences in mortality were found to increase with the length of follow-up. More recent indications of accumulative effects are to be found in a study by Hart et al. (1998), according to which lifetime accumulation of social position was an important factor behind social class differences in mortality. A study by Power et al. (1999a) showed that a cumulative indicator for social class was at least as important a factor with regard to self-rated health at the age of 33 than education reflecting the pathway effect (Power et al. 1999a, see Hertzman 1999). Therefore, the length of ex-

posure to adverse social circumstances might affect social class differences in morbidity and mortality. In that case, there should be interactions between social class positions across life such that those who have been longest in the most adverse social position have higher morbidity and mortality than those who are in the same position but have been there for only a short time.

4.4 Biological and social pathways in the same framework

Differentiating social and biological programming from each other is not quite unambiguous, although in principle it is possible to differentiate between paths of a social and biological origin. Ben-Shlomo and Kuh (2002) make a distinction between the socio-biological path and the bio-social path. On the former, adverse childhood socio-economic position affects the physiological function and the likelihood of disease in early years, leading to deteriorated health in adulthood. For instance, adverse experiences in childhood may increase vulnerability to later stressors (Maughan & McCarthy 1997), such as through changes in the HPA axis. These effects may also be latent. For instance, women who were exposed to parental divorce when they were aged 0-15 have been found to suffer from psychological symptoms when they were aged 43 only if they had themselves divorced or were single (the trigger effect), but not if they were in their first marriage (Rodgers1994). On the bio-social path, early disease affects social achievement by exerting an influence on poor educational achievement and lower social position in adulthood, for instance. In general, biological and social factors interact with each other: there is a "*continuous interplay over time between intrinsic and extrinsic influences*" (Rutter 1989:24-5). Social disadvantage may exacerbate the disease outcome, regardless of its origin, and this process may lead to deteriorating social circumstances (Vågerö & Illsley 1995, Bartley et al. 1997); differentiation between intertwined biological and social mechanisms may be rather arbitrary (Ben-Shlomo & Kuh 2002).

Both the origin of chronic disease and the social trajectory are embedded in the same social context. Biological exposures are related to social circumstances, and over the lifecourse biological risk is modified by the social environment, which individuals do not confront by chance or unprepared (see Hertzman & Wiens 1996, Wadsworth 1997a, 1999, Gillman 2002). For instance, physiological changes in the foetus due to the mother's smoking may be regarded as part of biological programming, while the mother's smoking during gestation is a behavioral component related to socio-economic circumstances.

The example given above of the latent effect of growth retardation on adult limiting illness may comprise the interaction of social and biological pathways. Growth retardation may have been influenced by social conditions: family conflict has been shown

to be associated with short stature at age seven (Montgomery et al. 1997). Biological processes may, in turn, have social consequences, which may be related to new social circumstances and biological exposures, and so on. Accordingly, smaller than average size in childhood may exacerbate psychological development and affect a person's self esteem. Alternatively it is possible that the same mechanism that has affected growth retardation, which affects biological processes determining disease development, also exerts an influence on the capacity and skills to learn and memorize, and therefore also on social achievement (Montgomery et al. 1997). In any case, short height at age seven has been shown to be associated with low educational attainment in youth, and to both unemployment and exposure to occupational hazards such as fumes and dust and a low level of autonomy in young adulthood (Montgomery et al. 1996, 1997, Holland et al. 2000). Further, childhood short size has been associated with the prevalence of long-standing illness and cardiovascular disease such as stroke in adult life (Kuh & Wadsworth 1993, Power et al. 2000, Eriksson et al. 2000). Furthermore, the social context may modify the effect of early biological processes differently for men and women. For instance, it has been shown that, for those who were short or chronically ill as a child, a manual class background further exacerbated men's but not women's life chances on the employment path (Pless et al. 1989, Holland et al. 2000). This example describes how early circumstances may affect adult social class differences in health via the social and biological pathways that affect the development of adult disease and social achievement.

However, one salient difference between the accumulative-risk model and the 'biological-programming' theory is that in the former, a single exposure leading to a subsequent disease is not necessary (see Hertzman 1994, Power & Hertzman 1997, Kuh & Ben-Shlomo 1997). In connection with this, while biological programming (in its reduced form) does not require a later trigger other than aging, social programming evolves all the time and is possible to break at any time point, even if not easily. Therefore, according to the biological-programming model, exposures in later life may only influence the risk in simple additive way, but this risk is based on permanent changes in the anatomical structures and immunological and metabolic systems resulting from early exposures (see Ben-Shlomo & Kuh 2002). However, it may be possible to alter biological programming by genetic or environmental means.

In sum, in studying the influence of the lifecourse on health, biological processes need to be examined in relation to living standards and stressful life conditions, which vary across the lifecourse and according to one's position in the social hierarchy (Kuh et al. 1997, 184). On the other hand, social effects, in the case of diseases, should somehow "get under the skin" in order to be able to influence a person's health status. Systematic differences in social-lifecourse factors in early environments may influence health by affecting cognitive, social, and behavioral development in terms of the neurochemistry

of the central nervous system and interactions of this system with hormone, immune, and clotting systems. (see Hertzman 1999.) It is through these processes that the early social environment may have an effect on a person's coping styles, and on how he or she considers and responds to stress in later events (see 3.2.2). Early social environments may have an effect on a large range of diseases (general susceptibility) through long-term effects on the function of organ systems. This type of 'programming' does not necessarily have to be specific in that it leads to a certain outcome, such as coronary heart disease or stomach cancer, but it affects the function of neuroendocrine pathways such as the HPA axis, and through that pathway exacerbates the effects of disease developed in later life.

4.5 The lifecourse in a wider macro-social and historical context

Many life events are related to and modified by external macro-social circumstances such as the level of medical care. These wider social contexts in which people live are related to specific historical times and affect the implications of belonging to a specific social class, e.g., in terms of prestige and life chances (Goldthorpe 1980, Graham 2002). They also affect latent, pathways and cumulative models, and modify the effects of these lifecourse mechanisms (see e.g., Hertzman 1999). For instance, in modern society, the scope and effectiveness of social income transfers and services, i.e. how social rights are actually materialized and how extensively they cover different sectors of life (see e.g., Kosonen 1998), affect the life chances of people directly and indirectly by influencing inequality between classes, and therefore they modify the effects of different life paths on social and health processes (Dahrendorf 1959:62-3).

On the national level, unemployment rates, the composition of the labour force and households, and income differences and changes in these factors, are likely to affect health (Wadsworth 1997b). In addition, community-level factors such as polarization, social capital and cohesion may affect health and social class differences in it (Davey Smith & Morris 1994, see Stansfeld 1999, Woodward & Kawachi 2000). The effects of these macro-level factors (see Hertzman 1999) may differ in women and men if there are gender differences in norms, labour-market involvement, and educational possibilities, for instance (see Moss 2002).

The macrosocial context is important for all age groups. For instance, in early years the key immediate environment, i.e. the parental home, is influenced by culture, beliefs, social policy, and the availability and quality of medical care (see Wadsworth 1997a). In adulthood, the reasons for being made redundant and the experience of unemployment and its influence on mortality may differ greatly depending on the economic situation and the unemployment rates (Iversen 1987, Bartley 1988, Martikainen

& Valkonen 1996, Julkunen 2001). For instance, in Finland, the relative change in mortality among men aged 15-24 was greater (14% versus 2%) in the municipalities in which the increase in the unemployment rate was greater than in those in which it was smaller (Valkonen & Martikainen 1995).

The significance of different phases of life on the development of disease or premature mortality, and social class differences in these outcomes, is likely to depend on the level of living conditions in general, and on variations in these conditions in childhood and adulthood. In particular, if poverty in childhood has been severe, and hygiene poor and infectious diseases common, it is likely that the contribution of childhood living conditions to adult mortality is greater. Correspondingly, relatively good conditions in childhood may produce a smaller effect (see Notkola 1985). In particular, if the effects of the parental home on adult social class differences are compared cohort-wise, both the changed conditions in childhood and possible changes in mortality patterns in adulthood should be taken into account (Morris & Montgomery 2000). For instance, mortality from stomach cancer and stroke has been declining, possibly due to better living conditions in childhood among younger cohorts. Simultaneously, the importance of early factors in relation to later factors in explaining geographical variation in stomach-cancer mortality in adulthood has been shown to be smaller in a British cohort born in the 1930s (Maheswaran et al. 2002) than in the cohorts born in the first decades of the 20th century (Davey Smith et al. 1998a, Leon & Davey Smith 2000). As one possible explanation for this discrepancy, the authors of the latter study (Maheswaran et al. 2002) suggested that, among younger cohorts, the effect of earlier factors might be smaller in relation to later life factors such as diet.

The effect of these early circumstances on adult social class differences in mortality also depends on the variation in living conditions between households. For instance, if there has not been a lot of variation in these living conditions in terms of health, as perhaps in wartime Finland (Lynch et al. 1994), their effect on social variation in mortality in adulthood is likely to be small.

4.6 The lifecourse and mortality in young and middle adulthood

The lifecourse approach is widely used in studies on mortality (Kuh et al. 1997, Leon & Ben Shlomo 1997), and the need to apply it to inequality in health research is also acknowledged (Davey Smith et al. 2001). Indeed, the (accumulation of) factors preceding adult social class transition have been shown to be important in generating social class differences in mortality in later adulthood in men, but there is less evidence on women (Mare 1990, Wunsch et al. 1996, Hart et al. 1998). The lifecourse approach is mainly used in studies of morbidity in younger people, such as those focusing on self-assessed health or limiting longstanding illness (Rahkonen et al.1997a, Mheen et al.1997, Power et al. 1997, Power et al. 2000). However, middle adulthood is an interesting age phase in terms of studying the effect of the lifecourse on mortality. By this time, most people have established their own adult social position, which has its own direct risks and health-enhancing effects. People are no longer under the immediate influence of the parental home, but if the parental home has affected the development of disease or increased the mortality risk, this may be detected in increased mortality rates at this age. In addition, the major transitions associated with youth and early adulthood are also behind for most persons. These, too, might affect mortality. Moreover, both the parental home and different transitions in youth are likely to affect the formation of the adult social class. Thus middle adulthood provides an interesting age phase in which to study the effects of the parental home and factors related to youth transitions on mortality and social class differences in it. It may also be a suitable point at which to study different lifecourse influences, whether the effect of earlier circumstances on adult social class differences are mediated through latency, pathways or accumulation effects. These models are not mutually exclusive, and are likely to show simultaneous influence, even though their importance may vary according to the cause of death.

The lack of research on lifecourse effects on differential mortality in early middle adulthood is possibly attributable to two reasons. First, mortality is low at young ages, and data with sufficiently large numbers of participants and long follow-up are not readily available. Second, most deaths in young adulthood are due to external causes, while the focus in lifecourse theories is usually on diseases. The applicability of lifecourse theories to mortality from external causes is not necessarily obvious. However, some life trajectories may predispose a person to external causes. I will turn next to the possible influence of early circumstances on cardiovascular disease, suicide, and alcohol-related mortality in middle adulthood.

Cardiovascular disease

Social class differences in early middle adulthood are observable in cardiovascular disease measured by coronary artery calcification (Colhoun et al. 2000). Even earlier, in 20- and 30-year-old men and women, there is a high prevalence of coronary atherosclerosis indicating the early induction of cardiovascular diseases (Tuzcu et al. 2001, see also Strong et al. 1999). In fact, the relevance of past circumstances on social class differences in mortality is perhaps most apparent precisely in cardiovascular diseases (Vågerö & Leon 1994, Davey Smith et al. 1997, Frankel et al. 1999, Wannamethee et al. 1996, Östberg 1996, Hart et al. 1998). However, the prevailing knowledge of the mechanisms that may account for this association is as yet deficient. The parental home has been found to be associated both with the foetal growth rate (Vågerö et al.1999), which is likely to be aetiologically important in the development of cardiovascular disease (Leon et al. 1998, Koupilova et al. 1997), and in adult physiological risk factors such as body mass (Blane et al. 1996b, Wannamethee et al. 1996, Brunner et al. 1996, Wright et al. 2001); babies with a low birth weight have been found to have a higher risk of coronary heart disease only if they are obese as adults (Frankel et al. 1996).

However, it is likely that the possible contribution of the parental home to the development of cardiovascular disease is not confined to the time before birth. Bartley and her colleagues (1994) showed that birth weight was related to socio-economic disadvantage across childhood, and that this relationship was graded – possibly indicating that higher mortality among low-birth-weight babies could be attributable to exposures at different phases of development. It is possible that, for cohorts born in the latter part of the 20th century, the childhood environment is more important than pre-natal circumstances in respect of cardiovascular disease risk (Whincup et al. 1996, Gunnell et al. 1998a). In this context, a study by Barker et al. (2001) indicated that thinness at birth (measured by the Ponderal Index) seemed to be associated with an increased risk of cardiovascular disease in lower adult social classes, but there was no such association among those whose parents were in the upper middle class. The authors suggested that altered early growth might increase vulnerability to later poor circumstances. Furthermore, other studies provide evidence of the effect of the parental home on growing patterns (Montgomery et al. 1997, Gunnell et al. 1998b, Wright et al. 2001, Teranishi et al. 2001), and of the effect of these growing patterns on physiological changes and the development of disease (Wright et al. 2001, see Eriksson et al. 2000, also Gunnell et al. 1998a).

There are likely to be several other mechanisms that may account for the contribution of parental class to cardiovascular disease risk, such as nutrition during early years (Notkola 1985, Gliksman et al. 1995), psychological attributes (Bosma et al. 1998b:127, Mheen 1998:212), and health-related behaviors related to the parental home (Lenthe et al. 2001, Lynch et al. 1997, Power & Hertzman 1997, Brunner et al. 1999).

Thus it is likely that cardiovascular disease develops earlier in people with poor socio-economic conditions in the parental home (Kaplan & Salonen 1990). In addition, smoking in adolescence, which is related to the parental home, substantially increases mortality from cardiovascular disease even at a relatively young age (McCarron et al. 2001). Therefore, although adult behaviors and psychosocial circumstances may be more important in determining the development of cardiovascular disease risk (Wannamethee et al. 1996, Blane et al. 1996b, Brunner et al. 1996, Whincup & Cook 1997), living conditions in childhood may contribute considerably to mortality and related social class differences in middle adulthood when exposure to adult risks is not long lived.

Suicide and alcohol-related causes

Failures or inadequate support at early or vulnerable times of life (Rutter 1989) may make people more open to substance abuse or taking unnecessary risks, and thus increase the risk of accidental and alcohol-related death (see Kalland et al. 2001). Furthermore, suicide risk in adolescence and adulthood has been shown to be related to various factors in the parental home and youth, including family type, low social class, relationship with parents, and being out of education and employment (Gould et al. 1996, Neeleman et al. 1998, Neeleman 2001, Sauvola et al. 2001a). Very early factors, such as low weight gain in infancy, have also been shown to be related to subsequent mortality from suicide (Barker et al. 1995).

Possible links between the parental home and adult suicide include early trauma and depression, which are significant determinants of suicide (Montgomery 1997, Maris 2002). The association of parental social class and subsequent depression is controversial: one study found that adult depression was related to a non-manual background (Marmot et al. 2001), another found it to be related to low parental class (Gilman et al. 2002), and others have found nonexistent or very weak associations (Rodgers 1994, Lynch et al. 1997, Harper et al. 2002). Instead, there are indications that later suicidal and other self-destructive behavior is related to disruptions in early attachment, significant loss or other forms of psychological trauma (Malone et al. 1995, see Kalland 2002).

Moreover, there is an indication that parental social class is related to feelings of hopelessness in adulthood (Lynch et al. 1997). Hopelessness is associated with lower serotonergic function (Heeringen et al. 2002). In studies of non-human primates[2] early experiences such as maternal neglect have been found to affect in interaction with

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[2] The effects of genes and environments on physiology and complex behaviors have mainly been studied in nonhuman primates (Bennett et al. 2002, Chaouloff et al. 1999).

genetic regulatory region variation central serotonin functioning and concentration. These are related to suicide risk in humans. (Linnoila et al. 1995, Kamali et al. 2001, Bennett et al. 2002). It is therefore possible that early experiences related to the parental home are linked to adult suicide through their impact on feelings of hopelessness and the serotonin function. Furthermore, middle adulthood may bring experiences of depression or other major psychiatric disorders and severe stress, which may trigger the effect of early events and be manifest as suicide (see Roy 2001, Chaouloff et al. 1999).

Alcohol abuse is also predictive of suicide, which may be at least partly related to the association of ethanol with depleted serotonin (Gorwood 2001, Maris 2002). Moreover, alcohol abuse is also related to other suicide risk factors, such as reduced impulse control, and diminished social support and less social involvement (Maris 2002). Furthermore, alcohol intoxication may lead to unintentional accidents, and abuse has been found to be related to the parental home (Glendinning et al. 1997, Jackson et al. 1997). Problems in the parental family increase the likelihood of drinking problems and an unstable employment career in men (Rönkä 1999).

Disadvantages in the parental home and youth increase the likelihood of alcohol abuse. Alcohol has fewer preventive effects in youth and young adulthood, and its health consequences may be observed at a young age (McElduff & Dobson 1997, Mäkelä et al. 1997b, Rahkonen et al. 1993, Upmark et al. 2001). In Finland, alcohol-related diseases account for approximately 15% of all deaths in men and more than 5% of all deaths in women in middle adulthood. In all, more than 40% of all deaths in men and approximately 20% of all deaths in women in middle adulthood are related to alcohol. (Mäkelä 1999:53.) It is at this age that early conditions may have a stronger effect on social class differences in alcohol-related causes, and through them on differences in total mortality.

5 The lifecourse approach and mortality in young and middle adulthood

5.1 Living conditions in the parental home

5.1.1 Factors related to the parental home

Living conditions in the parental home, such as the father's occupation, the mother's and father's education, material deprivation, parental unemployment, social deprivation, family type, and family size or overcrowding, may influence both adult social class and health directly or by influencing factors in youth that affect social class differences in mortality in adulthood (Lundberg 1991b, Mheen et al. 1997, Wadsworth & Kuh 1997). Thus, living conditions in the parental home may offer an explanation for adult social class differences in mortality (Fuhrer et al. 2002).

Associations of parental social class, family type, number of siblings, and broad cultural factors related to region and language, with adult social class and mortality or health are discussed separately below. In register-based studies on mortality, these belong to the most commonly used measures of living conditions in the parental home, and therefore warrant close examination in the context of this thesis.

5.1.2 Parental social class

There is a lot of evidence of the effect of parental social class on youth paths and adult social class (Goldthorpe 1980, Pöntinen 1983, Blane et al. 1999, Mheen et al. 1997, Power & Hertzman 1997). The effect of parental social class on subsequent mortality has also been shown in many studies (Davey Smith et al. 1998a, Vågerö & Leon 1994, Mare 1990, Nyström 1994). In particular, the effect of parental class has been found in mortality from cardiovascular disease (Forsdahl 1977, Notkola 1985, Vågerö & Leon 1994, Whincup et al. 1996; Frankel et al. 1999, Davey Smith et al. 1998a, Davey Smith, et al. 2001, Barker et al. 2001, see 4.6). Parental social class has also been found to be related to self-rated health and cognitive function (in men aged 58-64), and to some specific causes such as major depression in adulthood, regardless of one's own education (Kaplan et al. 2001, Gilman et al. 2002).

One important path from parental social class to adult disease is through parental health-related behaviors, including factors such as an unhealthy diet, the duration of breast feeding, maternal smoking during pregnancy, and parental smoking during adoles-

cence, all of which are factors that are likely to directly affect health, and physical and mental functioning, and also to have an indirect effect through the impact of parental behavior on the formation of health-related behaviors (Lynch et al. 1997, Power & Matthews 1997, Wadsworth & Kuh 1997, Mann et al. 1992, Wadsworth 1997a). For instance, maternal smoking has been found to have a direct effect on psychological and somatic distress at age 26 (Cheung 2002). According to a study by Mheen et al. (1998a), behavioral factors explained approximately 10% of the association between parental social class and perceived general health in adulthood. There was more unhealthy behavior in the form of increased body-mass index, lower physical activity and heavier drinking among descendants of unskilled manual workers, regardless of the effect of the current social class (see also Lynch et al. 1997).

Parental social class was also associated with 'educational stimulation' indicated with the frequency with which parents read to their child when she or he was seven years old (Power & Matthews 1997). This kind of parental involvement and aspiration affects the child's ability at age seven, and further his or her social and emotional development. Therefore, positive factors in childhood are likely to be conducive to good health and even lower mortality in early middle adulthood (Romelsjö et al. 1992).(Hertzman et al. 2001, Power et al. 2002).

A study by Bosma et al. (1999) showed that an external locus of control, neuroticism, and active problem-focused coping mediated approximately 34% of the effect of parental class on adult "less-than-good" perceived general health, adjusted for the subjects' own class (calculated from tables 1 and 2 in Bosma et al. 1999). The authors suggested that the associations between the parental home and negative personality profiles and adverse coping might be attributable to differences in rearing styles between social classes. Rearing styles, i.e. neglectful, supportive or authoritarian parenting, have an effect on a person's subsequent health-related behaviors, and (self-rated) health and psychological well-being (see Sweeting & West 1995, Maughan & McCarthy 1997, Bosma et al. 1999, Rönkä 1999). However, little is known of the effects of parenting on social class differences in mortality.

5.1.3 Parental family type

Family type in childhood is associated with many different dimensions of growth circumstances. Families with children are categorised as one-parent or two parent. One-parent families are mainly divided into those in which the other parent is not living in the same household due to divorce or separation, and those in which the other parent has died. The most common types of two-parent families are those with biological parents (intact) and those in which one is biological (reconstituted families). In many but not all studies (an exception being Mheen et al. 1997), a single-parent background

has been shown to predict lower educational achievement (see Aro 1988, Krein & Beller 1988, Wadsworth et al. 1990, Koivusilta et al. 1995, Ely et al. 1999). Parental family type may also be related to other youth paths, because a single-parent background has been found to be associated with early and premarital parenthood, and further with marital breakdown (Wadsworth et al. 1990, Michael & Tuma 1985, Rodgers 1994). Parental divorce has also been associated with frequent job changes (Rodgers 1994), and an increased risk of unemployment in men (Wadsworth et al. 1990). In adulthood, possibly partly as a consequence of its association with youth paths, parental divorce is associated with lower social class and lower earnings (Wadsworth et al. 1990, Pietilä & Järvelin 1995).

Family type in childhood also affects subsequent health. Parental separation or a single-parent background has been shown to be associated with many behaviors related to health such as heavy smoking, excess drinking and spontaneous and induced abortions and pregnancy complications in women, and also with physical-condition and conduct problems related to ill health, suicide in adolescence and young adulthood, and all-cause mortality at a later age (Wadsworth 1990, Cole & Cole 1992, Maughan & McCarthy 1997, Romelsjö et al. 1992, Schwarz et al. 1995, Gould et al. 1996, Mäkikyrö et al. 1998, Sauvola et al. 2001a, 2001b, Lundberg 1993, Buchanan et al. 2000, Wadsworth et al. 2002, Kuh & MacLean 1990). Even at age 52, the pervasive effect of parental divorce, which was not accounted for by adult interpersonal difficulties or circumstances, has been associated with psychological distress (Kuh et al. 2002a).

The effect of growing up in a single-parent family may be different for men and women, although there are differences between countries (see e.g., Aro 1988, Rodgers 1994, Tulisalo & Aro 2000, Kemppainen 2001, Dworkin & Larson 2001). In Finland, the excess risk of depression related to parental divorce was decreased in women who had a close relationship with their father, but not in men (Palosaari et al. 1996). In the United Kingdom, parental divorce has been related to higher depression in women, but not in men, at age 43 (Rodgers 1994).

Family type and health may be associated through three key mechanisms, which are partly related to each other: (1) psychological, (2) behavioral and (3) material. A significant link between family type and subsequent health may be through the relationship between parents and children, and through chronic parental discord and its effect on children (Aro 1988, Rodgers 1994, Wadsworth et al. 2002, Maughan & McCarthy 1997). However, these factors are not confined to single-parent homes (Lundberg 1993, Wadsworth et al. 1990). In general, the lack of time for children (Östberg 1996), inadequate parenting (Rodgers 1994, Maughan & McCarthy 1997), and a combination of low care and high control (Wadsworth & Kuh 1997) have been shown to have a significant effect on subsequent health-related behavior and health. In

fact, parental divorce has been found to carry no risk of adult depression if it did not lead to poor care (Rutter 1989). With respect to functional parenting, the number of parents may be significant in cases which one of them has problems (McCord 1990). Inadequacy in parenting may be a consequence of economic difficulties or a lack of resources to confront daily needs, possibly alone (Conger et al. 1984, Scannapieco & Connell-Carrick 2002, Krein & Beller 1988). Emotional disturbances may also arise from an irregular or distant relationship with a non-custodial parent (Nieminen 1990, Palosaari et al. 1996). Indeed, frequent contact with a child is not common to all divorced parents: in Finland approximately 32% (Nieminen 1990, Kaarela, personal information), and in the US 17%, of non-custodial parents have been reported to be in frequent contact with their children (Furstenberg et al. 1983); this infrequency is likely to increase uncertainty in a child's life (Nieminen 1990).

Children in single-parent families have been found to engage in some risky behaviors such as smoking, drinking, sexual intercourse and more frequent abortions at a young age, and thus they are exposed to these effects longer than children in two-parent families (Aro 1988, Aro & Palosaari 1992, Blum et al. 2000, Isohanni et al. 1994). A greater prevalence of alcohol abuse and smoking in middle adulthood has been found in women with divorced parents (Kuh & MacLean 1990). One British study found an association between excess drinking among adolescents who were from broken homes and parental support and control (Shucksmith et al. 1997). The genetic component of some behavior may also play a role. Parental alcoholism may be a reason for separation, and increase the susceptibility of offspring to later alcoholism (Kendler et al. 1996).

The material conditions in single-parent homes may also impose constraints on everyday life that may affect life chances on social paths. On average, the disposable income per consumption unit (OECD) in one-parent families is less than in other households (Valtioneuvoston kanslia 2001, Östberg 1996). For example, according to results of a study conducted in Finland in 1986, while 27% of all families with children belonged to the three lowest income deciles (based on per consumption unit), the figure was 44% for one-parent families (Sauli et al. 1989). In the United Kingdom, separated one-parent families have been found generally to have lower incomes, and to be dependent on supplementary benefits to a much greater extent than two-parent families (Townsend 1979). Graham (2002) highlights the position of single-parent families thus: "*Lone motherhood in the UK is synonymous with poverty*". The relation of family type and economic situation may account for various associations with family type, social position and health in adulthood. For instance, in Sweden, the effect of a disunited family on adult height has been accounted for by the economic situation in the family (Nyström & Lundberg 1995). In Britain, too, family income has accounted for lower educational attainment among those born in 1970 and living in single-parent households (Ely et al.

1999). Family type in childhood may negatively affect future health and life paths through the accumulation of difficulties regarding material and social conditions.

The effect of parental family type on subsequent health may be through latency and pathway mechanisms, as suggested in the introduction. According to many studies, the effects of parental divorce on psychiatric problems and health behavior are clearer in adulthood than in childhood and adolescence after a latent period (Rodgers 1994, Wadsworth & Kuh 1997, Buchanan et al. 2000). In terms of pathway mechanism, family type may be associated with youth paths, which exert an effect on health. For instance, the main impact of parental divorce on depressive symptoms has been found to be through its negative effect on education (Maughan & McCarthy 1997).

5.1.4 Number of siblings

In the lifecourse perspective, the number of siblings may reflect childhood circumstances, which may be connected directly to later health or indirectly through other factors associated with health (Power et al. 1991, Kemppainen et al. 2000, Montgomery et al. 1997, Mheen et al. 1997, Cassidy 2000). The number of siblings has been shown to be inversely associated with adult social class (Blane et al. 1999), early marriage and parenthood (Michael & Tuma 1985, Gohel et al. 1997).

Growing up in a large family and growing up as an only child may both affect subsequent health through different mechanisms. There is less evidence of the effect of having been an only child. However, for the Northern-Finnish 1966 birth cohort, an association between growing up as an only child and committing violent crimes was observed (Kemppainen et al. 2001). A larger family size has been shown to be associated with poorer self-rated health and psychological well-being, and with depression and alcoholism among young adults (Cassidy 2000, Kemppainen et al. 2000). Having many children in the family may be associated with crowding, which in turn may be associated with less available parental time and, as a consequence, less parental encouragement, scarce material resources, more infectious diseases, and disruptive sleeping patterns, all of which may have an impact on subsequent health (Kemppainen et al. 2000, Montgomery et al. 1997). One pathway from crowding to adult disease may be via its observed association with short stature (Montgomery et al. 1997), and subsequent unemployment (Montgomery et al. 1996). However, the effect of crowding on mortality in adulthood seems to be minor (Dedman et al. 2001).

5.1.5 Cultural and macro-social factors

Cultural factors in the parental home have also been found to have a significant effect on child's behavior, personality and attitudes, and thus possibly to have consequences in terms of adult social class and health (Mheen 1998). Childhood and adolescent behaviors related to culture may have an independent effect on health, and they may be hard to change later (Koskinen 1994; 157). Broad cultural aspects in the parental home may be reflected in the region of residence, religion, ethnicity, and minority status. In Finland, the region of residence and language have been found to be related to social class, and also to mediating variables such as education and mortality (Sauli 1979, Finnäs 1986, Valkonen 1987, Koskinen 1994, Koskinen & Martelin 1994a, Valkonen et al. 1996). For example, the educational opportunities in different regions of the country have differed during the childhood of this cohort. Language and region of residence illustrate socio-cultural characteristics of childhood, which may direct young people to different youth paths, but which may also have a direct effect on health.

5.2 Youth paths

5.2.1 What are youth paths?

Important turning points in people's lives are the major transitions taking place in youth and early adulthood, such as leaving the parental home, gaining educational qualifications, forming a family and entering into the labour market (Rutter 1989). In this 'socially critical period' (Bartley et al. 1997, Kuh et al. 1997), failure to embark on adult roles may generate a drift towards socially adverse life trajectories. It may also extend its influence on adult social class and mortality by exerting an effect on somatic alterations, and also on intellectual and behavioral development (Rutter 1989) in subsequent years. Life trajectories in youth (youth paths) closely reflect the idea of social programming; they are influenced by living conditions in the parental home and further determine the constraints on social chances in adult life. In addition, they may modify and mediate the effects of the parental home, but they are also likely to have their own independent effect on adult social class differences in mortality. At least three important trajectories are identifiable: educational, family-formation and employment path.

There are other mechanisms apart from major transitions that are underway in youth, and that have an effect on both adult social class and health. In this context, one key mechanism is the formation of one's own lifestyle, particularly with regard to the establishment of social networks and health-related behaviors, which may have an effect on youth paths (see Glendinning et al. 1995, Koivusilta 2000). The development

of health-related behaviors and self-esteem is likely to exert an effect on youth paths. For instance, positive experiences in a hobby may motivate the setting of higher educational goals. An effect may also be mediated through social networks gained or lost, or social, cognitive and coping skills learned along one of the youth paths or during activities practised in youth. The following section introduces each of these three paths and their effect on the formation of adult social class and subsequent mortality separately, and highlights findings from studies that have examined the effects of youth paths on social class differences in health.

5.2.2 Educational path

Educational attainment could be seen a pathway from the parental home to the adult social class. The parental home, i.e. the social class and education of the parents, family type and parental control, largely determines a person's further education after compulsory school, the type of education and their educational performance (Sandford et al. 1994, Koivusilta 2000, Pöntinen 1988, Isohanni et al. 2001). Low educational achievement restricts many life chances, increases the likelihood of fragmental employment and unemployment, and is a strong determinant of adult class (Pöntinen 1982, Wadsworth et al. 1990, Pietilä & Järvelin 1995, Power & Hertzman 1997, Lynch et al. 1997, Blane et al. 1999, see Rönkä 1999, Chapter 2.3.4). Educational achievement has also been shown to be associated with a variety of health indicators such as psychological functioning, less than good perceived health, chronic conditions, long-term disabilities and illness, and mortality (although there is some variation by cause of death and sex) (Valkonen 1989, Elo & Preston 1996, Cavelaars et al. 1998, Rönkä 1999, Mackenbach et al. 1999). Therefore, given the strong associations between education and adult social class and mortality, adult social class differences in health and mortality may be partly accounted for by educational attainment (see e.g., Power et al. 2002, Valkonen 1990, Martelin 1990).

Presumably, the effect of education on health is stronger in young than in late adulthood. First, it is likely that factors related to education are more salient in the lives of young adults (see 2.3.4), and that the effect of material factors strengthens in later middle adulthood. Second, any exposure to occupational risk factors cannot have been very long-lived. However, there is evidence that the effect of education is not confined to young adulthood, but is pervasive, and that it continues to contribute to adult social class differences among people at older ages (Martelin 1990, Wadsworth & Kuh 1997, Elo & Preston 1996, Lundberg 1991b).

There are several mechanisms related to youth and young adulthood that could account for the effect of education on social class differences in mortality. First, educational attainment may simply reflect psycho-social characteristics such as self-esteem

and coping strategies, which are developed in late adolescence and early adulthood and are partly, directly or indirectly, attributable to education (Ross & Mirowsky 1999, Lynch et al. 1997, Koivusilta et al.1999, West 1997, Hendry et al. 1996, Rutter 1989, see also Pietilä et al. 1994, Rutter et al. 1990, Lundberg 1997).

Second, the years spent at school in youth are likely to enhance a person's knowledge about health-related risks, and adherence to instructions both before and after the diagnosis of disease (Goldman & Smith 2002, see also Valkonen 1995). A shorter period of education in itself may reduce the ability to care for one's health, to use health care and follow health and safety instructions (Lundberg 1991b). Less-educated people who have been given more intensive patient monitoring have been shown to achieve better treatment results and to have been more compliant with the treatment regime (see Goldman & Smith 2002, also Ursin & Eriksen 2001).

In addition, many studies show that a health-related lifestyle that includes exercise, non-smoking, moderate drinking and the avoidance of overweight is related to higher education (Ross & Mirowsky 1999, Winkleby et al. 1992, Choinière et al. 2000). In general, the association between education and health-related behaviors is partly attributable to health knowledge, for instance in terms of smoking. However, health knowledge does not automatically lead to changes in behavior. (Kenkel 1991.)

Health-enhancing and damaging behaviors may have been adopted in adolescence before decisions concerning further education have been made (Koivusilta et al. 1998). Indeed, there may be a common background factor for both educational attainment and health-related behaviors (Isohanni et al. 2001). For example, the time of beginning school could be a sensitive period, since if the child drops out from the educational track at the start, for instance due to weak school maturity, it is very difficult to catch up later.

The association of education with mortality may also be attributable to exposures in subsequent circumstances in which educational level has had an effect. A study by Schrijvers and her colleagues (1999) found that approximately half of the association between education and mortality was explained by material factors acting through health-related behaviors, i.e. alcohol consumption, smoking, body-mass index and physical activity.

5.2.3 Family formation: partnership and parenthood

Many studies have shown that married people have lower mortality rates than those who have never been married, or who have previously been married (Hu & Goldman 1990, Ben-Shlomo et al. 1993, Koskinen & Martelin 1994b, Ebrahim et al. 1995, Joung et al. 1996, Johnson et al. 2000). In general, the protective effect of marriage has been related both to marriage selection and to the causative effects of being married

(Mastekaasa 1992, Goldman 1993, Joung et al. 1996). Johnson et al. (2000) suggest that marriage has a positive effect on mortality because it defines a social role and a sense of purpose, and provides a social network. These effects may vary according to the age of transition to marriage, cohabitation or parenthood.

However, early marriage may be a consequence of the processes connected to a disadvantageous parental home and problems with school and behavior (Michael & Tuma 1985, Rutter 1989, Kiernan 1986, Michael & Tuma 1985, Dearden et al. 1995, Gohel et al. 1997). Early marriage may interfere with other transitions in youth, and lead to disruptions in educational and employment careers. Disruptions in transitions and subsequent experiences may have negative health consequences and complicate the transition to one's own adult social class. (see Rutter 1989:25, Rönkä 1999, Hobcraft & Kiernan 2001, Mirowsky & Ross 2002). However, an intimate relationship in young adulthood may also act as a buffer against low self-esteem (Palosaari & Aro 1995).

Moreover, remaining single may be associated with increased risks. In older age groups, the elevated mortality of those who have never married may be related partly to selection and partly to risk factors such as smoking, which may be related to the living circumstances of single persons. For instance, a study by Ben-Shlomo and his colleagues (1993) showed that men who had never married were shorter than those who were currently or previously married. However, people who marry at a very young age have been found to have poorer self-rated health and psychological well-being than those who are single (Cassidy 2000).

The transition to parenthood changes one's life in many ways, including one's personal goals, and affects the lives of women more than those of men (Salmela-Aro et al. 2000). However, early parenthood has also been associated with the accumulation of problems such as law breaking and aggression in men (Dearden et al. 1995, Gohel et al.1997). There are also indications that early parenthood may be associated with a stable employment career and better social functioning in men, but not in women (Rönkä 1999). The negative effects of early parenthood may be stronger the younger the age at first birth, and in general, first birth before the age of 23 has been found to be associated with higher depression than birth at older ages (Mirowsky and Ross 2002). However, although early motherhood (in this study before age 22) has been related to risk factors in childhood and youth, it was not associated with lowered psychological well-being unless combined with low education and work involvement (Rönkä 1999).

It is through these pathways and psychosocial processes connected to transition effects that marital path and early parenthood may be associated with adult social class and mortality, and therefore to social class differences in health and mortality. There is evidence that early marriage and parenthood have a pervasive effect on health among

women and men aged 55-69, although the effect of early parenthood is more salient among older women than among men (Grundy and Holt 2000). The social gradient in distress has been shown to be partly related to early parenthood at age 33 in the UK (Power et al. 2002). However, the effect of family formation on social class differences in mortality has received less research attention.

5.2.4 Employment path

The experience of unemployment at the beginning of one's career may have a long-lasting effect on socio-economic achievement in terms of income, occupation and home ownership, for example (Wadsworth et al. 1999). Unemployment has also been shown to be associated with social isolation, alienation, cynicism, loss of self-esteem, criminal behaviors, problem drinking, smoking and illicit drug use (Bartley 1994, Montgomery et al. 1998, Bartley et al. 1999c, Wadsworth et al. 1999, Fergusson et al. 2001), and directly with psychological well-being involving physiological changes such as raised cholesterol levels and lowered immunity, morbidity, and suicide and other causes of mortality (Bartley 1994, Montgomery et al. 1999, Fergusson et al. 2001, Johnson & Sundquist 1997, Hammarström 1994, Stefansson 1991, Hall et al. 2002, Jin et al. 1995).

One unemployment spell may mark the beginning of a new chain in stigmatising the person concerned and leading to further unemployment (Hammarström 1994, Bartley & Plewis 2002). In general, repeated unemployment experiences may be an indication of being in the 'secondary' sector, characterized by job insecurity, lower wages and poorer working conditions. It is along this path that health disadvantages accumulate over the lifecourse partly due to the increased psychological burden of insecurity (Bartley 1988, Bartley et al. 1999c, see also Karasek et al. 1998, Power et al. 1998, Matthews & Power 2002). For people in middle and later adulthood, there is evidence that mortality is related to the length of unemployment (Jin et al. 1995, Martikainen & Valkonen 1996), but poor self-rated health and limiting long-term illness have also been shown to be associated with long-term unemployment even years afterwards (Grundy & Holt 2000, see also Bartley & Plewis 2002). The duration of the unemployment may intensify its effect on health and subsequent career. If it leads to deteriorating health, the likelihood of re-employment decreases (see Jin et al. 1995, Bartley & Owen 1996).

Most studies in this area have shown that the association between unemployment and mortality is strongest in young and middle adulthood (Stefansson 1991, Hammarström 1994). The experience of unemployment may be different for younger and older people, and thus lead to different health consequences. In young adulthood, unemployment interferes with the transition to adulthood by limiting expectations of autonomy and opportunities to make life choices (Hendry et al. 1996), and it has been shown in many

studies to have a clear effect on self-esteem and psychological ill-health in terms of depression, for example (Hammarström 1994, see also Montgomery et al. 1999). It is possible that dissatisfaction with life generated by an unemployment experience at a socially sensitive time could be connected to mortality even years later (see Koivumaa-Honkanen 2000). A brief unemployment experience at the beginning of one's career may already produce long-term effects on health, but it is likely that these effects are intensified according to the duration of the unemployment. At this age the duration of unemployment is likely to be in proportion to the difficulties in establishing adult class membership.

Unemployment experience is not necessarily a single independent event, but may be part of a chain of other events and factors that influence social class differences in health. The chain leading to unemployment may possibly start with poor living conditions in the parental home, which are associated with slow growth in childhood, under-achievement at school and conduct problems in adolescence (Fergusson & Horwood 1998, Fergusson et al. 2001, Montgomery et al. 1996, Bartley 1988, 1996, Montgomery et al. 1998, Martikainen & Valkonen 1996).

In sum, the employment path, which differentiates steady employment from short and long or repeated unemployment experiences, and periods of exclusion from the labour force, is likely to influence social-class membership and mortality risk in young and middle adulthood.

5.3 The effect of living conditions in the parental home and youth paths on social class differences in mortality

In short, there is a lot of evidence that living conditions in the parental home are associated with morbidity, mortality and social class in adulthood. For example, analyses of the 1958 British Birth Cohort have shown that factors from birth to age 23, such as social class at birth and educational qualifications, explained 51% of social class differences in self-rated health at age 33 among men, and 74% among women (Power et al. 1998). These effects are likely to be attributable to the independent effects of the parental home during childhood, the cumulative effect of social class and educational attainment, and factors such as life control developing in the long term (Hertzman et al. 2001). Factors related to the parental home have also been shown to be associated with youth paths, which are likely to exert an effect on subsequent mortality and social class. However, there is a lack of evidence of the simultaneous effects of these factors on adult social class, mortality, and the contribution to social class differences in mortality in middle adulthood.

6 The aim of the thesis

The aim of this thesis is to disentangle the effect of the lifecourse from childhood to young adulthood on mortality and social class differences in mortality in middle adulthood. My interest is particularly in the effects of socio-demographic living conditions in the parental home and youth paths on social class differences in mortality by cause of death.

Figure 6.1 gives a simplified illustration of the causal relationships between the parental home, youth paths, adult social class and mortality. The focus of this thesis is on the contribution of living conditions in the parental home and youth paths, i.e. factors preceding social class, to the relationship between social class and mortality. As has been shown, the parental home may have a direct effect on mortality differences by affecting mortality and social class, but it may also affect these differences via youth paths. Youth paths may have an independent effect on both social-class membership and mortality, but they are likely to both mediate and possibly modify the effect of the parental home.

Within this framework my aim is to disentangle the effects of the parental home and youth paths on social class differences in mortality in middle adulthood. A further objective is to find out whether there are differences in these effects by cause of death. Ultimately, the intention is to evaluate whether the effects of the lifecourse on adult social class differences in mortality are due to latency, accumulation or to pathway models.

The questions are considered in detail in the five separate papers (listed below), which have been published (or accepted for publication) in international refereed journals. These papers are also attached to the end of this thesis. The results section therefore only gives a summary.

Figure 6.1 The effect of living conditions in the parental home and youth paths on adult social class differences in mortality
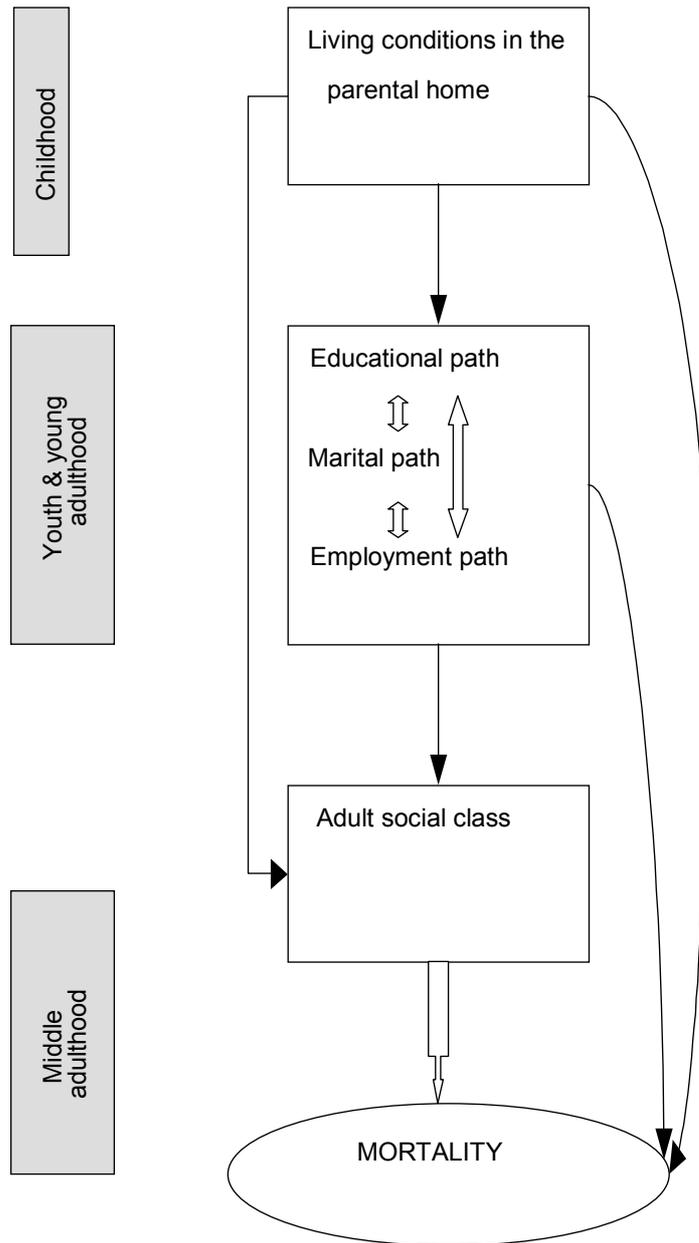
More specifically, the study questions are the following (the numbers in brackets refer to the papers in which the question is addressed):

1) What are the effects of the living conditions in the parental home on adult social class differences in mortality?

- Are the living conditions in the parental home associated with mortality in middle adulthood?
  - Does the association between parental social class and mortality change with age? (PI)
  - Are the living conditions in the parental home associated with mortality from various causes of death in middle adulthood? (PIII-PV)
- What is the effect of the living conditions in the parental home on adult social class differences in mortality by cause of death (PII-PV)?

2) What is the effect of youth paths on adult social class differences in mortality?

- Are youth paths associated with mortality in middle adulthood? (PIII-PV) What are the effects of the educational, family-formation, and employment paths on mortality from different causes of death? (PIII-PV)
  - What are the effects of different youth paths on adult social class differences in mortality from various causes of death? (PIII-PV)

3) Are the effects of the lifecourse on adult social class differences in mortality due to latency, accumulation or pathway models? (PI-PV)

The list of original papers:

Paper I : Pensola T, Valkonen T. Mortality differences by parental social class from childhood to adulthood. *Journal of Epidemiology and Community Health* 2000;54:525-9.

Paper II: Pensola T, Martikainen P. Cumulative social class and mortality from various causes in young men. *Journal of Epidemiology and Community Health* 2003;57:745-751.

Paper III: Pensola T, Valkonen T. Effect of parental social class, own education and social class on mortality among young men. *European Journal of Public Health* 2002;12:29-36.

Paper IV: Pensola T, Martikainen P. Effect of the living conditions in the parental home and youth paths on social class differences in mortality among women. *Scandinavian Journal of Public Health*, in print.

Paper V: Pensola T, Martikainen P. Life course experiences and mortality by adult social class among young men. *Social Science & Medicine,* in print.

7 Data, variables and methods

7.1 Data

The data are based on the 1990 census records for persons who were born in 1956-60 and who lived in Finland at the time of the 1970, 1975, 1980, 1985 and 1990 censuses. Those whose household status was not 'a child' at the time of the 1970 census, approximately 1% of the cohort, were excluded from the data to make sure that the 1970 census information described the parental home. The study cohort was restricted to those who were born in 1956-60 because information on the parental home was not available for 15% of those born in 1955, and for less than 10% of those born before 1955. Persons born after 1960 were too young to have been established in their adult social class by 1990.

The final study population consisted of about 177,000 women and 183,000 men aged 30-34 at the time of the 1990 census. The death records for 1991-98 and the information concerning life events from the 1970 to the 1985 censuses were linked to the 1990 census records by means of personal identification numbers. The proportion of unlinked deaths was less than one percent. The design varies slightly between the studies, and therefore the actual number of person-years or length of the follow-up also vary. Accurate descriptions of the study samples are given in the papers.

7.2 Variables

7.2.1 Adult social class

Adult social class in 1990 was determined on the basis of the subject's own occupation and occupational status. In 1990, 84% of the women in this cohort were in the labour force. Most of these women were working full time. Only 10% of women in Finland were in part-time employment in 1990 (Statistics Finland 2001). If an individual had no occupation for reasons other than being a student, information was sought from the 1985 census. For the persons who were taking care of the home the partner's social class was used (approximately 8% of women were housewives). If occupational information was still missing, the person was classified in the group 'others' (3% of all women and 4% of all men), which also included students. This classification was based on the Statistics Finland schema, which in turn is based on the United Nations recommendation for the 1980 population census (Statistics Finland 1983): 1) upper non-manual employees, 2) lower non-manual employees, which in women is divided

into intermediate and lower non-manual employees, the latter having less authority and more monotous work tasks than those in the intermediate non-manual class, 3) manual workers, 4) employers and farmers, 5) others. I have additionally divided manual workers into skilled and unskilled groups, a division that is used in schemes such as the Registrar General, the Erikson-Goldthorpe-Portocarero, the Swedish social class classification and the Nord-SEI classification (Erikson & Goldthorpe 1992, Statistics Sweden 1983, Nordisk Statistik Sekretariat 1990). The division of the manual class into skilled and unskilled workers has not been in use in Finnish censuses since1975. For reasons of coherence between the 1970s and later censuses, and because this differentiation also appears to have been a meaningful social class indicator in the1990s, I have reformulated the division of the manual class into unskilled and skilled manual workers (see Pensola 2002 for a detailed explanation).

7.2.2 Variables related to living conditions in the parental home

Parental social class is based on the occupation of the head of household, usually the father, when the cohort members were aged 10-14 in 1970. The coding is based on Statistics Finland's classification (Official Statistics of Finland 1974), and is consistent with the classification of adult social class used in this study.

Family type distinguishes families with two parents from single-parent families. *The Number of siblings* is classified as zero, one, and two or more.

Social and cultural background is described by two factors, viz. language, categorized as 1) Finnish speaking and 2) others (of which 80% are Swedish speaking), and region of residence in 1970, categorized as 1) western Finland, 2) the capital (Helsinki) area, and 3) eastern Finland.

7.2.3 Variables related to youth paths

Educational path is based on educational attainment reported in the 1980 and 1990 censuses, reflecting qualifications obtained roughly at age 20-24. The classification is based on the International Standard Classification of Education (Statistics Finland 1991), and separates the following categories: higher education (at least 13 years of education), higher-intermediate education (12 years), lower-intermediate education (10-11 years), middle school (basic education with an academic orientation, 9-10 years), and basic education (9 years or less).

The family-formation process is described with two variables, *marital path* and *early parenthood*. *Marital path* is classified in the following groups: 1)'partner in 1990' consists of those who were not married at age 20-24, but who were married or cohabiting at age

30-34 (in the 1990 census); 2) 'Partner in 1980' consists of those who were married at age 20-24 (in the 1980 census), 3) 'Partner in 1975' consists of those who were married at age 15-19 (for women only) and 4) 'no partner' consists of those who were single in 1980 and who lived without a partner in 1990. Unfortunately, the data on cohabitation are not consistent across the censuses. Cohabitation was not as common in 1980 as in the 1990s, and information on those who were cohabiting but did not have common biological children was not given in the 1980 census. It was separately indicated in the 1990 census, however, and accounted for approximately 27% of all cases in the group 'partner in 1990'.

Early parenthood distinguishes 1) 'no child in 1980' from those 2) who had a 'child in 1980' 3) a 'child in 1975' (for women only).

Employment path identifies different experiences of the working career: 1) 'steady' employment means continuous employment after compulsory or further education up to the end of 1990, with the exception of national service; 2) 'short unemployment' means less than 6 months unemployment during 1986-90, or only one spell of unemployment at the time of the 1975, 1980, 1985, or 1990 census; 3) 'long unemployment' means unemployment lasting at least 6 months during 1986-90, or repeated unemployment experiences at the time of at least two censuses; 4) a 'fragmental' employment path means occasional exclusion from the labour force for reasons other than unemployment, retirement or study. We had no information on the reasons for this, but it was safe to assume this is a heterogeneous group that includes those taking care of the home, and also socially marginalized persons as well as those having a gap year; 5) 'retired' consists of persons on disability pension on a temporary or permanent basis. This is a very different group from the rest because its members were chronically seriously ill or injured so that they were at the time unable to work gainfully.

7.3 Mortality - causes of death

The mortality follow-up varies between papers: it covers the years 1987-95 in PI, 1991-98 in P II, IV and V, and 1991-95 in PIII. The causes of death in 1987-95 were coded according to the 1987 Finnish Classification of Diseases, which is based on the Ninth Revision of the International Classification of Diseases (ICD9) (National Bord of Health 1986). The codes for 1996-98 are based on the Tenth Revision of this Classification (ICD10) (Stakes 1999). This change of classification had no effect on the results. Definitions of cause of death in those aged 31-42 are highly reliable in Finland, as cause-of-death certificates are based on autopsies in approximately 90% of all cases among men and 80% among women[3]. The following broad groups are used:

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[3] These figures are calculated from these data.

- all causes (PI-PV)
- all diseases (including cardiovascular diseases) (PII-PV)
- cardiovascular diseases (ICD9:390-459, ICD10:i00-i99) (PII-PV)
- neoplasms (ICD9:140-239, ICD10:c00-d48) (PII)
- external causes (including suicide) (PII-PV)
- suicide (ICD9:E95, ICD10:x60-x84,Y870) (PIII-PV)

We also distinguished alcohol-related mortality as a separate group because of its importance in this age group (PI-PIV). Contributory causes of death are also used in the definition of alcohol-related mortality. Those included in this category are those in which alcoholic disease such as alcoholic psychoses (ICD9: 291, ICD10: F10.5), alcoholic diseases of the liver (ICD9: 571.0-571.3, ICD10: K70.0-K70.9), and alcoholic diseases of the pancreas (ICD9: 577.0D-F,577.1C-D, ICD10: K86.00-K86.08) or alcohol poisoning (ICD9: E851, ICD10:X45) are mentioned as underlying or contributory causes of death, or alcohol intoxication (ICD9: 305, ICD10: F10.0) is mentioned as a contributory cause of death. The decision to use contributory causes here was based on the fact that a large proportion of alcohol-related mortality at a young age is due to external causes in which alcohol has an important role. Alcohol-related causes overlap with all diseases and all external causes. The classification of alcohol-related causes has been discussed in closer detail elsewhere (Mäkelä et al. 1997).

7.4 Statistical methods

Person-years and deaths by cause of death were tabulated by period or age and variables related to the study context in each paper. The cross-tables were analysed by means of Poisson regression analysis, with the cell in the cross-tabulation taken as the unit of analysis. The GLIM statistical package was used in fitting the models (Francis et al. 1993). The results are presented in terms of mortality rates and relative mortality rates (and their 95% confidence intervals). The modelling strategy is based on the time order of the explanatory variables. Thus the models were first adjusted only for age. In PIV and V the adjustment was then done for variables preceding individual adult social class, first for the living conditions in the parental home and then for the youth paths one by one. Information on the living conditions in the parental home was all from the 1970 census, but that on youth paths took into account the approximate time order of these factors on the basis of the1975, 1980, and 1985 censuses. The design used in P I-III are explained in them.

In Poisson modelling, multicollinearity of variables, particularly between youth paths, may be a cause for concern. However, the mutual relationships between these paths were modest: the contingency coefficient between the educational and employment paths was 0.24 for both sexes, while it was 0.16 for men and 0.30 for women between the educational and the marital paths, and 0.25 for men and 0.20 for women between the marital and the employment paths. The effects of these paths in the full models were approximately the same as in the models in which they were adjusted for separately.

We used *average deviation* (AD) and the *index of relative dissimilarity* (IRD) as a summary measure of mortality differences between the categories of a specific variable. AD measures how much the mortality rates of the categories deviate on average from the mortality rate for the whole cohort.

$$AD = (\Sigma n_i \cdot |MR_i - MR|) \, / \, \Sigma n_{i,}$$

where n_i is the number of person-years in category i, MR_i is the mortality rate for a category, and MR is the mortality rate for the whole cohort. The average deviation for total mortality is the sum of the average deviations in different causes of death. In addition, the average relative deviation (ARD) was used to measure how much the mortality of the different social classes deviated on average from that of the upper non-manual class. The *average change* was then calculated from the ARD. This is the percentage difference in the ARDs obtained from the model in which the explanatory variable under study has been adjusted for the ARD in the uncontrolled model.

The IRD gives the average deviation in the group-specific relative rates from their mean in percentage terms. It was computed to allow and facilitate a valid comparison of the effects of the different variables on mortality. All of these indicators take into account the sizes of the groups, and thus adjust for the differences in the distributions of the social class variables.

$$IRD = 100 \cdot (\Sigma n_i \cdot |RR_i - RR| \, / \, RR) \, / \, \Sigma n_{i,}$$

where n_i is the number of person-years in category i, RR_i is the relative mortality rate for a category, and $RR = \Sigma n_i \cdot RR_i$ is the mortality rate for the whole cohort.

8 Results

8.1 Social class differences in mortality in middle adulthood

Social class differences in mortality in adulthood were considerable in both women and men, albeit there were differences in the patterning between the sexes (see Table 8.1, PII-V). In women, the mortality difference between the upper and lower non-manual classes was small, but those in the intermediate non-manual class had higher mortality than those in the upper or lower non-manual class for all causes of death.

The following assesses the contribution of the living conditions in the parental home and youth paths to these adult social class differences according to the empirical results reported in the papers. The likely mechanisms through which the parental home and youth are connected to social class differences in mortality in adulthood are considered in Chapter 10.

8.2 Living conditions in the parental home

8.2.1 The association of living conditions in the parental home with adult social class

Living conditions in the parental home should affect both social-class membership and mortality in adulthood in order to be able to exert an effect on social class differences in mortality. Parental social class was associated with adult social class in both sexes (PIII, Table 8.2). People were most likely to end up in the same class as their parents, or in one close to it. Moving from the manual to the lower non-manual class was not uncommon, but only 7% of the women and men from the unskilled manual class ended up in the upper non-manual class. The associations between parental family type and number of siblings and adult class were weaker. However, growing up in a single-parent family or with many siblings was more often associated with membership of the manual class in adulthood and less often with the upper non-manual class.

Adult social class compositions by parental social class depend not only on mobility opportunities, but also on changes in the class structure. For instance, a shrinking class is likely to be more homogenous than a growing class. If factors related to the parental home affect mortality in adulthood, these compositions (shown in P IV-V) are relevant in terms of mortality differentials. In these data, the upper non-manual class was twice as big measured by adult class membership than by parental class; 20% of people in that class originated from the upper non-manual class, another 20% were from the

Table 8.1 Age-adjusted relative mortality rates by adult social class, average mortality deviation, mortality rates, and number of deaths for various causes of death in 1991-98. Women and men aged 30-34 in 1990.

Adult social class	All causes		Diseases		Cardiovascular disease		External causes		Suicide		Alcohol-related causes	
Women												
Upper non-manual	1.00		1.00		1.00		1.00		1.00		1.00	
Intermediate non-manual	1.44	1.16-1.79	1.38	1.06-1.80	1.57	1.07-2.30	2.47	1.40-4.38	2.47	1.40-4.38	2.27	1.28-4.05
Lower non-manual	1.18	0.94-1.47	1.08	0.82-1.41	1.40	0.95-2.04	2.15	1.21-3.81	2.15	1.21-3.81	1.89	1.06-3.37
Manual	1.96	1.59-2.41	1.65	1.28-2.13	2.63	1.84-3.75	2.96	1.69-5.18	2.96	1.69-5.18	4.69	2.73-8.03
Employer/Farmer	1.14	0.85-1.52	0.87	0.59-1.27	1.72	1.09-2.73	2.00	0.98-4.06	2.00	0.98-4.06	2.80	1.45-5.39
Other	7.42	5.88-9.36	7.45	5.63-9.86	7.36	4.87-11.1	9.04	4.79-17.1	9.04	4.79-17.1	10.1	5.44-18.9
Average deviation (%*)	28.6 (100)		17.0 (59)		3.9 (14)		11.6 (41)		4.2 (15)		8.2 (29)	
Mortality rates	83.8		52.4		10.9		31.4		15.1		17.5	
Number of deaths	1185		741		154		444		213		247	
Men												
Upper non-manual	1.00		1.00		1.00		1.00		1.00		1.00	
Lower non-manual	1.51	1.28-1.79	1.35	1.06-1.71	1.35	0.87-2.09	1.71	1.34-2.19	1.94	1.38-2.73	1.92	1.44-2.56
Skilled manual	2.94	2.54-3.40	2.12	1.73-2.60	2.73	1.88-3.96	3.95	3.19-4.89	3.88	2.87-5.23	4.62	3.60-5.94
Unskilled manual	4.08	3.51-4.73	2.86	2.32-3.53	3.37	2.29-4.96	5.56	4.48-6.91	5.24	3.86-7.12	6.82	5.30-8.79
Employer/Farmer	1.92	1.61-2.28	1.36	1.05-1.76	2.08	1.35-3.22	2.59	2.02-3.32	2.92	2.07-4.11	2.46	1.83-3.30
Other	7.72	6.54-9.11	7.20	5.73-9.04	6.32	4.09-9.79	8.37	6.55-10.7	8.73	6.19-12.3	8.64	6.48-11.5
Average deviation (%*)	95.4 (100)		35.7 (37)		11.9 (13)		59.7 (62)		25.3 (27)		51.9 (54)	
Mortality rates	218.9		93.5		30.9		125.4		58.8		99.1	
Number of deaths	3184		1360		449		1824		856		1441	

* A proportion of the variation in each cause of death from the variation in total mortality.

Table 8.2 The distribution of person-years (%) by adult social class according to parental-home characteristics and youth paths. Women and men aged 30-34 in 1990.

Parental-home Characteristics Youth paths	Women: Adult class								Men: Adult class							
	Upper non-manual	Middle non-manual	Lower non-manual	Manual	Employer	Other	All	N in 000s	Upper non-manual	Lower non-manual	Skilled manual	Unskilled manual	Employer	Other	All	N in 000s
Parental class																
Upper non-manual	42	20	21	8	6	4	100	116	50	22	9	7	6	6	100	125
Lower non-manual	22	26	28	15	6	3	100	209	27	26	22	13	7	4	100	220
Skilled manual	12	25	28	26	7	3	100	505	13	19	38	19	7	4	100	516
Unskilled manual	7	22	24	36	8	4	100	152	7	14	40	26	7	5	100	150
Employer	14	21	25	23	14	3	100	410	12	15	27	17	26	3	100	425
Other	8	18	23	34	8	9	100	22	9	14	37	22	11	7	100	18
Family type																
Intact	17	23	26	22	9	3	100	1248	18	19	30	17	13	4	100	1306
Single	11	23	25	29	8	4	100	166	13	17	34	21	9	6	100	149
Number of siblings																
None	18	26	27	19	8	3	100	129	19	20	28	17	11	5	100	138
One	20	25	27	18	8	3	100	367	22	21	26	15	12	4	100	394
Two or more	14	22	26	26	9	3	100	918	15	18	32	18	13	4	100	922
Education																
Higher	62	11	17	2	4	3	100	253	78	11	1	1	6	3	100	226
Higher intermediate	11	35	33	11	8	2	100	440	19	49	10	7	10	5	100	302
Lower intermediate	3	18	28	38	11	2	100	450	2	10	51	21	14	2	100	587
Middle school	4	30	24	30	9	4	100	121	5	15	30	29	17	4	100	128
Basic	2	20	16	44	8	9	100	151	1	9	33	33	15	8	100	211
Marital path																
Partner in 1990	20	24	27	19	8	2	100	675	21	21	29	15	13	2	100	832
Partner in 1980	10	22	26	28	11	3	100	407	13	17	35	17	16	2	100	214
Partner in 1975	4	22	23	38	11	3	100	90	*	*	*	*	*	*	*	*
No partner	19	24	25	20	3	8	100	241	12	15	30	23	11	9	100	408

lower non-manual class, and approximately 30% were from the manual class. Almost 60% of those in the manual class had a manual background, and only 3% an upper non-manual background. In addition, women and men with a two-parent and two-children family background were over-represented in the upper non-manual class, while the opposite held for the manual class.

8.2.2 The association of the living conditions in the parental home with various causes of death

8.2.2.1 The age pattern of mortality by parental social class

The age pattern of mortality by parental social class was studied among persons aged 5-34 for main causes of death (PI). Women aged 10-19 and men aged 5-14 whose parents were in the manual and lower non-manual classes had lower mortality from diseases than those whose parents were in the upper non-manual class, although the rate increased among children of manual-class parents after age 25 in women and after age 15 in men. There was a statistically significant gradient (p<0.05) in disease mortality in the oldest age group studied, 30-34.

In women, an association between parental social class and external causes excluding alcohol-related causes was observed at age 10-19, but this vanished at age 20-24. On the other hand, men with a manual background had excess mortality in all age groups. After age 15 the excess was about 50%. For alcohol-related causes there was a clear excess among males over 15, but less so among women, who showed no clear mortality pattern in alcohol-related causes possibly due to the low incidence. The association between parental class and total mortality at ages 25-34 in women was mainly attributable to mortality from diseases, while alcohol-related causes explained a large proportion of the excess total mortality among men.

The increasing association between parental social class and mortality by age indicates that the parental home has an effect on differential mortality in middle adulthood. This may be attributable to the direct effects of parental class on mortality, and also to the association between parental class and subsequent circumstances that affect mortality. The association between parental class and external and alcohol-related causes in men in young and middle adulthood may indicate that the effect of parental class on mortality may be mediated through behavior.

8.2.2.2 The accumulative effect of social class on mortality in middle adulthood - social mobility from the parental class to the adult class

The effect of accumulative disadvantage was studied by using information on social class from childhood and adulthood (PII). Paper II also gave the results concerning the effect of social class on mortality at three measurement points, i.e. at ages 10-14, 20-24, and 30-34 (Table 8.3). The mortality rates for cumulative social class measured a number of times at censuses in the manual class, from zero to three, indicate a clear cumulative effect. For instance, the total mortality rates were 81, 117, 175, and 235 according to a cumulative social class measure. However, when time was taken into account, it was revealed that mortality was related more closely to the latest social class than to the sum of the ordinal number of social classes (Figures 2 and 3 in PII, Table 8.3). There was an accumulative effect in mortality from cardiovascular diseases and alcohol-related causes in that the effect of the parental class was observable in both in the non-manual and the manual classes. Such an effect was not distinguishable in other causes of death. Social mobility constrained the mortality difference between the non-manual and manual classes by 29% in the case of cardiovascular diseases, and by 24% in alcohol-related causes. This effect was 2% in total mortality excluding alcohol-related causes.

We were also interested in finding out whether restricting the data to men who were employed would affect the results (PII). Therefore all men who did not belong to the labour force or who were unemployed in 1990 were excluded, in other words 4% in the non-manual class and 14% in the manual class. Adult social class differences in mortality were smaller in the analyses restricted to employed men than in the whole data, and as a consequence the results changed, albeit not uniformly, for different causes of death. As far as mortality from cardiovascular diseases was concerned, the difference between upwardly and downwardly mobile men was abolished, and thus the cumulative effect of social class was overestimated. This difference remained for alcohol-related causes but the difference between men with different social backgrounds was abolished within the manual class.

Table 8.3 Age-standardized mortality rates per 100,000 person-years for all causes, all diseases and external causes according to adult (in 1990), first (in 1980 at age 20-24), and parental (in 1970) social class. Men aged 30-34 in 1990.

Social position In adulthood	Non-manual				Manual			
At entry	Non-manual		Manual		Non-manual		Manual	
In the parental home	Non-manual	Manual	Non-manual	Manual	Non-manual	Manual	Non-manual	Manual
Person-years	125190	102526	41310	67939	8032	15420	75856	338004
%	16	13	5	9	1	2	10	44
Number of times in manual class	0	1	1	2	1	2	2	3
Number of deaths	102	105	49	74	23	34	171	795
All causes*	81	117	117	175	117	175	175	235
All causes	81	102	119	109	286	221	225	235
All diseases*	45	59	59	70	59	70	70	93
All diseases	45	55	58	46	100	(52)	(96)	93
External causes*	36	58	58	105	58	105	105	142
External causes	36	47	61	63	187	169	129	142

*The mortality rate is calculated on the basis of the number of times a person has been in the manual class.

8.2.2.3 The association between living conditions in the parental home and mortality in middle adulthood

From the variables describing the living conditions in the parental home, mortality variation in middle adulthood was greatest by parental social class, but also varied according to family type, region of residence in both men and women, and number of siblings in men (PIV-PV). In general, higher mortality was observed for those in poorer circumstances.

Variables related to the parental home were clearly associated with all diseases in women, and with alcohol-related causes of death in men. For instance, of the total deviation in mortality by parental social class, 82% was attributable to diseases in women, while 76% was attributable to alcohol-related causes in men.

However, it is possible that these associations reflect the effects of circumstances in youth and adulthood. In PIII, the children's own education and social class were adjusted for in the mortality analyses by parental social class. It was revealed that the effect of parental social class was mainly indirect, and was mediated through its influence on education and adult social class. It was only for cardiovascular diseases and alcohol-related causes that these later circumstances did not fully explain the higher mortality of men with a manual background.

Furthermore, it was reported in PV that the excess mortality of men from one-parent families and from the capital region, as well as the lower mortality in Swedish-speaking families, remained after controlling for variables related to youth and adult social class. The excess mortality among women who had grown up in the capital area also remained, as did the lower mortality among Swedish-speaking women. On the other hand, the higher mortality of women from single-parent families was largely attributable to other living conditions in the parental home (46%) and educational attainment (further reduction by 67%).

8.2.3 The effect of the living conditions in the parental home on social class differences in mortality

Adjusting for living conditions in the parental home, i.e. social class, family type, number of siblings, language and region of residence, had only a moderate effect on social class differences in mortality in middle adulthood, reducing the total mortality differences by 4% in women and 10% in men. In women, class differences reduced the figure by 13% for diseases, but there was no effect for external causes. Adjusting for living conditions in the parental home had an effect on mortality differences in cardio-vascular diseases in both sexes (27%), and also in alcohol-related causes in men (15%). The total effect of the parental home was to a large extent (more than 70%) attributable to the contribution of the parental social class. Family type accounted for approximately half of the remaining effect. (PIV-PV)

8.3 Youth paths

8.3.1 The association of youth paths with adult social class

Youth paths were highly associated with adult social class in both women and men. The adult class was largely determined by education (see Table 8.2). Early marriage (having a partner in 1975 or 1980) was more common for those in the manual class, while late partnership (having a partner in 1990) was more common in the upper non-manual class. Among women, the tendency not to have a partner at age 30-34 decreased from the upper non-manual to the manual class, while for men, not having a partner was clearly more common in the manual class. The highest proportion of those who had experienced unemployment was in the manual class.

8.3.2 The association of youth paths with various causes of death in middle adulthood

Youth paths, the educational path, the marital path and the employment path were all associated with mortality from various causes of death in women and men (PIII-V). Only the average deviation in mortality by early parenthood was small due to the low frequency of young parents. Even for this variable, however, mortality differences were consistent showing higher mortality for those having their first child at a young age.

Mortality was higher for those who had experienced some disadvantage on any of these paths from all diseases, and also from external causes. Approximately half of the total deviation in mortality by youth path was attributable to diseases in women, approximately 40% in men. Alcohol-related causes also played a significant role in the variation in total mortality for all of these paths, particularly among men.

There were no statistically significant interactions between youth paths in the model in which age, the living conditions in the parental home, and adult social class were adjusted for. Disadvantage on any of these paths meant higher mortality in comparison to those who were in this respect in a better position, and even higher mortality if the person had some further disadvantage on any other path. In addition, because there were no significant interactions between youth paths and adult social class, the effects of youth paths on mortality were similar throughout.

8.3.3 The effect of youth paths on social class differences in mortality

The effects of adjusting for the educational path, the marital path and the employment path on relative mortality rates by adult social class in the model in which the parental home was adjusted for are shown in Table 8.4. The effect of educational path on differential mortality was crucial. Following adjustment for the effect of the living conditions in the parental home, further adjustment for the educational path abolished the excess mortality in the lower non-manual class, and reduced that in the manual class by approximately 60-80% for various causes of death. The only exception was in suicides among women, where the reduction was 8% (PIV).

The effects of family formation and employment paths on social class differences in mortality were smaller than that of the educational path. However, both of these paths had an independent effect on differential mortality. Adjustments for these paths reduced the excess mortality of those in the manual class, particularly among men in the unskilled manual class. In addition, the employment path was the most important factor in respect of the excess mortality in the 'others' class because this path adjusts for the effect of being on disability pension.

When all youth paths were adjusted for, adult social class differences in cause-specific mortality were reduced by about 80-90%. The only exception was in suicide for women where the reduction was 25%. In women, the social class patterning of suicides was different than in other causes of death: those in the upper non-manual class had clearly lower mortality than those in all other social classes, between which there were no differences.

Table 8.4a Relative mortality rates (and their 95% confidence intervals) for adult social class (upper non-manuals as the reference group for which the rate is 1.00) adjusted for age, living conditions in the parental home*, educational, marital and employment path, and differences (%) in relative rates after these adjustments, women.

Adult social class	Model 1: age+ parental home	Diff. **	Model 2: Model 1 + educational path	Diff. M2/ M1	Model 3: Model 1 + marital path	Diff. M3/ M1	Model 4: Model 1 + employment path	Diff. M4/ M1
All causes								
Intermediate non-manual	1.39 (1.12-1.73)	11.4	1.09 (0.85-1.40)	76.9	1.35 (1.08-1.68)	10.3	1.30 (1.04-1.62)	23.1
Lower non-manual	1.16 (0.93-1.45)	11.1	0.92 (0.72-1.19)	***	1.13 (0.90-1.42)	18.8	1.09 (0.87-1.36)	43.7
Manual	1.94 (1.56-2.40)	2.1	1.27 (0.99-1.64)	71.3	1.84 (1.48-2.28)	10.6	1.47 (1.18-1.84)	50.0
Farmer/Employer	1.18 (0.88-1.58)	-28.6	0.86 (0.63-1.19)	***	1.20 (0.89-1.61)	-11.1	1.14 (0.85-1.53)	22.2
Others	7.17 (5.67-9.07)	3.9	4.55 (3.48-5.96)	42.5	5.88 (4.63-7.46)	20.9	1.32 (0.98-1.76)	94.8
Diseases								
Intermediate non-manual	1.30 (1.00-1.70)	21.1	1.09 (0.80-1.48)	70.0	1.29 (0.99-1.69)	3.3	1.24 (0.95-1.62)	20.0
Lower non-manual	1.03 (0.78-1.35)	62.5	0.87 (0.64-1.19)	***	1.03 (0.78-1.35)	0.0	0.97 (0.74-1.28)	***
Manual	1.57 (1.21-2.05)	12.3	1.12 (0.82-1.54)	78.9	1.56 (1.19-2.03)	1.8	1.24 (0.94-1.62)	57.9
Farmer/Employer	0.87 (0.60-1.29)	***	0.69 (0.45-1.04)	***	0.91 (0.62-1.34)	***	0.85 (0.58-1.25)	***
Others	7.09 (5.34-9.42)	5.6	4.86 (3.51-6.72)	36.6	6.15 (4.61-8.21)	15.4	1.16 (0.81-1.66)	97.4
Cardiovascular diseases								
Intermediate non-manual	1.18 (0.63-2.22)	47.1	0.93 (0.45-1.91)	***	1.12 (0.59-2.10)	33.3	1.09 (0.58-2.04)	50.0
Lower non-manual	1.07 (0.57-2.00)	63.2	0.83 (0.41-1.71)	***	1.01 (0.54-1.90)	85.7	0.98 (0.52-1.84)	***
Manual	1.97 (1.09-3.57)	26.0	1.20 (0.59-2.47)	79.4	1.78 (0.98-3.24)	19.6	1.39 (0.76-2.55)	59.8
Farmer/Employer	1.57 (0.74-3.33)	8.1	1.09 (0.47-2.53)	84.2	1.50 (0.71-3.21)	12.3	1.48 (0.70-3.15)	15.8
Others	5.32 (2.66-10.6)	15.5	3.29 (1.49-7.27)	47.0	4.23 (2.10-8.54)	25.2	0.65 (0.28-1.48)	***
External causes								
Intermediate non-manual	1.58 (1.07-2.32)	- 1.8	1.09 (0.70-1.69)	84.5	1.48 (1.00-2.18)	17.2	1.43 (0.97-2.11)	25.9
Lower non-manual	1.44 (0.98-2.12)	-10.0	1.03 (0.67-1.59)	93.2	1.37 (0.93-2.01)	15.9	1.35 (0.91-1.99)	20.5
Manual	2.77 (1.92-4.01)	-8.6	1.57 (1.01-2.42)	67.8	2.47 (1.70-3.58)	16.9	1.99 (1.37-2.91)	44.1
Farmer/Employer	1.86 (1.17-2.96)	-19.4	1.20 (0.72-2.01)	76.7	1.83 (1.14-2.92)	3.5	1.79 (1.13-2.86)	8.1
Others	7.30 (4.81-11.1)	0.9	4.03 (2.52-6.46)	51.9	5.46 (3.58-8.34)	29.2	1.65 (1.00-2.72)	89.7
Suicides								
Intermediate non-manual	2.60 (1.45-4.65)	-8.8	2.42 (1.27-4.63)	11.3	2.43 (1.35-4.37)	10.6	2.36 (1.31-4.25)	15.0
Lower non-manual	2.32 (1.29-4.16)	-14.8	2.19 (1.15-4.16)	9.8	2.21 (1.22-3.97)	8.3	2.13 (1.18-3.85)	14.4
Manual	3.34 (1.87-5.96)	-19.4	2.78 (1.43-5.41)	23.9	2.96 (1.65-5.33)	16.2	2.35 (1.30-4.25)	42.3
Farmer/Employer	2.23 (1.08-4.58)	-23.0	1.99 (0.92-4.33)	19.5	2.21 (1.07-4.58)	1.6	2.14 (1.03-4.41)	7.3
Others	9.36 (4.92-17.8)	-4.0	7.62 (3.77-15.4)	20.8	6.95 (3.61-13.4)	28.8	1.51 (0.71-3.21)	93.9
Alcohol-related causes								
Intermediate non-manual	2.22 (1.24-4.00)	3.9	1.20 (0.61-2.36)	83.6	2.07 (1.15-3.74)	12.3	1.94 (1.06-3.56)	23.0
Lower non-manual	1.92 (1.07-3.47)	-3.3	1.07 (0.54-2.10)	92.4	1.80 (0.99-3.27)	13.0	1.78 (0.97-3.28)	15.2
Manual	4.86 (2.79-8.48)	-4.6	1.86 (0.96-3.63)	77.7	4.26 (2.42-7.49)	15.5	3.24 (1.81-5.81)	42.0
Farmer/Employer	3.07 (1.57-5.98)	-15.0	1.45 (0.68-3.07)	78.3	2.78 (1.41-5.45)	14.0	2.93 (1.47-5.81)	6.8
Others	9.77 (5.20-18.3)	4.0	3.68 (1.78-7.61)	69.4	8.62 (4.54-16.4)	13.1	2.65 (1.26-5.55)	81.2

Table 8.4b Relative mortality rates (and their 95% confidence intervals) for adult social class (upper non-manuals as the reference group for which the rate is 1.00) adjusted for age, living conditions in the parental home*, educational, marital and employment path, and differences (%) in relative rates after these adjustments, men.

Adult social class	Model 1: age+ parental home	Diff. **	Model 2: Model 1 + educational path	Diff. M2/ M1	Model 3: Model 1 + marital path	Diff. M3/ M1	Model 4: Model 1 + employment path	Diff. M4/ M1
All causes								
Intermediate non-manual	1.46 (1.23-1.73)	10.2	0.97 (0.80-1.18)	***	1.42 (1.20-1.68)	8.7	1.40 (1.18-1.66)	13.5
Skilled manual	2.73 (2.35-3.18)	10.7	1.46 (1.21-1.77)	73.4	2.50 (2.15-2.90)	13.6	2.28 (1.96-2.66)	26.1
Unskilled manual	3.76 (3.23-4.38)	10.4	1.93 (1.59-2.34)	66.4	3.19 (2.74-3.73)	20.5	2.71 (2.32-3.17)	38.1
Farmer/Employer	1.88 (1.57-2.25)	3.3	1.05 (0.85-1.30)	94.4	1.78 (1.49-2.14)	11.0	1.89 (1.58-2.26)	-1.0
Others	7.18 (6.07-8.49)	8.0	4.07 (3.34-4.96)	50.3	5.07 (4.28-6.01)	34.2	2.46 (2.03-2.97)	76.4
Diseases								
Lower non-manual	1.32 (1.04-1.68)	8.3	0.93 (0.71-1.22)	***	1.29 (1.02-1.63)	10.3	1.27 (1.00-1.61)	16.8
Skilled manual	2.04 (1.65-2.52)	7.4	1.18 (0.90-1.55)	82.4	1.87 (1.51-2.32)	15.8	1.71 (1.38-2.12)	31.4
Unskilled manual	2.72 (2.19-3.38)	7.6	1.49 (1.13-1.96)	71.7	2.33 (1.87-2.90)	22.8	1.98 (1.58-2.47)	43.0
Farmer/Employer	1.36 (1.04-1.77)	1.4	0.81 (0.59-1.10)	***	1.29 (0.99-1.69)	17.5	1.36 (1.04-1.77)	-0.6
Others	6.84 (5.43-8.62)	5.8	4.08 (3.11-5.37)	47.2	4.87 (3.84-6.16)	33.8	2.11 (1.61-2.77)	81.0
Cardiovascular diseases								
Lower non-manual	1.22 (0.78-1.89)	38.3	0.83 (0.50-1.39)	***	1.19 (0.76-1.85)	12.5	1.18 (0.75-1.83)	19.0
Skilled manual	2.24 (1.53-3.29)	28.3	1.23 (0.75-2.01)	81.9	2.08 (1.42-3.05)	13.0	1.95 (1.32-2.87)	23.8
Unskilled manual	2.73 (1.84-4.06)	26.9	1.46 (0.88-2.41)	73.5	2.40 (1.61-3.57)	19.4	2.13 (1.42-3.19)	34.7
Farmer/Employer	1.77 (1.13-2.78)	28.4	1.02 (0.60-1.74)	97.8	1.69 (1.08-2.65)	11.0	1.76 (1.12-2.77)	1.3
Others	5.36 (3.45-8.33)	18.1	3.17 (1.88-5.34)	50.2	4.05 (2.59-6.35)	30.0	2.16 (1.29-3.61)	73.5
External causes								
Lower non-manual	1.63 (1.27-2.09)	11.5	1.02 (0.77-1.36)	96.3	1.58 (1.23-2.03)	7.5	1.56 (1.21-2.00)	11.1
Skilled manual	3.57 (2.86-4.44)	13.1	1.76 (1.34-2.32)	70.3	3.24 (2.60-4.04)	12.6	2.97 (2.38-3.70)	23.4
Unskilled manual	5.00 (4.00-6.25)	12.3	2.40 (1.82-3.16)	65.0	4.23 (3.38-5.29)	19.3	3.58 (2.86-4.49)	35.5
Farmer/Employer	2.51 (1.95-3.24)	4.9	1.31 (0.98-1.77)	79.3	2.37 (1.84-3.06)	9.3	2.53 (1.96-3.26)	-1.3
Others	7.65 (5.97-9.79)	9.8	4.10 (3.08-5.46)	53.4	5.37 (4.18-6.90)	34.3	2.85 (2.17-3.74)	72.2
Suicides								
Lower non-manual	1.90 (1.32-2.72)	4.4	1.18 (0.79-1.77)	80.1	1.85 (1.29-2.65)	5.7	1.82 (1.28-2.58)	8.8
Skilled manual	3.69 (2.67-5.10)	6.7	1.87 (1.26-2.78)	67.6	3.38 (2.44-4.68)	11.3	3.15 (2.30-4.32)	19.8
Unskilled manual	4.92 (3.54-6.84)	7.5	2.49 (1.67-3.72)	62.0	4.25 (3.05-5.92)	17.0	3.72 (2.70-5.14)	30.6
Farmer/Employer	2.93 (2.03-4.22)	-0.4	1.59 (1.04-2.42)	69.6	2.76 (1.91-3.99)	8.5	2.93 (2.05-4.18)	0.1
Others	8.20 (5.71-11.8)	6.9	4.66 (3.08-7.04)	49.2	6.08 (4.21-8.76)	29.5	3.19 (2.16-4.72)	69.5
Alcohol-related causes								
Lower non-manual	1.79 (1.34-2.39)	13.8	1.10 (0.79-1.53)	87.3	1.73 (1.29-2.31)	7.8	1.70 (1.27-2.27)	11.9
Skilled manual	4.01 (3.10-5.18)	16.9	1.90 (1.38-2.62)	70.0	3.58 (2.77-4.63)	14.2	3.19 (2.46-4.14)	27.2
Unskilled manual	5.90 (4.56-7.65)	15.8	2.68 (1.94-3.70)	65.7	4.85 (3.73-6.29)	21.6	3.96 (3.04-5.15)	39.7
Farmer/Employer	2.36 (1.74-3.20)	6.4	1.18 (0.83-1.68)	86.9	2.21 (1.63-2.99)	11.5	2.39 (1.76-3.24)	-1.8
Others	7.69 (5.75-10.3)	12.4	3.90 (2.78-5.47)	56.7	5.11 (3.81-6.87)	38.5	3.20 (2.33-4.40)	67.1

* Parental social class, family type, number of siblings, language and regions of residence in 1970.
** Difference from the model in which only age is adjusted, presented in Table 8.1.

Table 8.5 The age-adjusted average excess mortality in all social classes in comparison to mortality in the upper non-manual class, and the contribution of parental-home living conditions and youth paths to the excess mortality.

| | All causes | Cause of death | | | | |
		All diseases	Cardio-vascular disease	All external causes	Suicide	Alcohol-related causes
Women :						
Average excess (%)	58.5	47.4	64.6	87.5	143.1	182.1
		Effect of living conditions in the parental home				
Total*	-4.4	-12.9	-27.3	6.3	13.4	2.6
Direct**	0.3	-0.1	2.4	6.9	7.7	5.8
Indirect***	-4.7	-12.9	-29.6	-0.6	5.7	-3.2
		Effect of youth paths				
Total	-85.1	-77.2	-89.5	-88.7	-25.2	-92.3
Independent**	-80.4	-64.3	-59.9	-88.1	-31.0	-89.1
		Joint effect of the parental home and youth paths				
Total effect	-84.8	-77.3	-87.2	-81.7	-17.5	-86.5
Parental home/youth paths*	5.5	16.7	33.1	0.7	-22.8	3.4
Men:						
Average excess (%)	164.9	105.5	139.0	237.9	240.2	284.4
		Effect of living conditions in the parental home				
Total	-9.6	-6.8	-26.7	-11.6	-6.1	-15.1
Direct	1.2	0.6	-2.2	0.6	1.5	0.2
Indirect	-10.8	-7.4	-24.6	-12.2	-7.6	-15.3
		Effect of youth paths				
Total	-85.6	-88.8	-85.4	-82.8	-76.9	-85.0
Independent	-74.8	-81.4	-60.9	-70.6	-69.3	-69.7
		Joint effect of the parental home and youth paths				
Total effect	-84.4	-88.2	-87.6	-82.2	-75.4	-84.6
Parental home/youth paths*	12.6	8.3	28.7	14.7	9.8	18.0

Parental-home variables include parental social class, family type, number of siblings, language and region of residence in 1970. The youth paths are educational, marital and employment.
Total effect= the percentage change in the excess mortality achieved by adjustment of the variables (%).
Direct effect= the effect of the parental home that is not mediated by youth paths
Indirect effect =the effect of the parental home that is mediated by the youth paths.
Independent effect= the effect of the youth paths that is not attributable to living conditions in the parental home

*The proportion of the effect of youth paths, that is attributable to the effect of the living conditions in the parental home.

8.4 Summary of the empirical results

Table 8.5 summarizes the effects of the living conditions in the parental home and youth paths on the average excess mortality of those in the lower non-manual and manual classes, employers and farmers, and 'others' for various causes of death.

Overall, the parental home and youth paths explained 85% of the social class differences in all-causes, 77% in all diseases, 87% in cardiovascular diseases, 82% in all external causes, 18% in suicide, and 87% in alcohol-related causes among women. The equivalent figures for men were 84%, 88%, 88%, 82%, 75%, and 85%, respectively.

The total effect of youth paths on social class differences was crucial for most causes of death. However, these effects were not fully independent, but were attributable to the preceding effects of the living conditions in the parental home, and were different for men and women. In women, of the effect of youth paths, approximately 17% for diseases but only 1% for external causes and 3% for alcohol-related causes was attributable to the living conditions in the parental home. In men, the corresponding figures were 8%, 15% and 18%.

9 Discussion and conclusions

9.1 The reliability of the variables and the limitations and strengths of the study design

9.1.1 The study design in register-based data

Cohort studies are essential in investigating the importance of different factors across the life course (Ben-Shlomo & Kuh 1997). Ideally, a study on lifecourse effects on mortality would be a prospective cohort study, beginning before birth with information on pregnancy, and continuing through birth, infancy, childhood, youth and adulthood. This study was carried out on register-based longitudinal census data. The nature of this kind of data gives rise to both the strengths and the shortcomings of the study.

These data allowed the retrospective collecting of information on childhood and youth circumstances, and prospective information on mortality, but it was not possible to cover the whole lifecourse from birth on. The first census year available was 1970 and the last was 1990. Therefore, the mortality follow-up did not begin at birth: information on deaths before the year 1991 was not available. Estimations based on the information in the Official Annual Vital Statistics suggest that approximately 5% of those born in 1956-60 died before the 1990 census. Of these deaths, about half were infant deaths, which have been shown to be related to conditions in the parental home (Notkola & Valkonen 1989, Leon et al. 1992, Arntzèn et al. 1995). There was mortality variation in external causes at ages 5-29, but mortality variation in diseases by parental social class was very small as shown in PI. Therefore, it is likely that restricting the follow-up in adulthood underestimates the total effect of the parental home on mortality over the whole lifecourse due to higher mortality among the offspring of the manual class, particularly in infancy. However, because the focus here is on the extent to which adjusting for the living conditions in the parental home affects adult social class differences in mortality in later life, selective mortality before the follow-up is unlikely to essentially bias the main results reported in this thesis.

9.1.2 Shortcomings in the variables

This study found that the living conditions in the parental home had a minor effect on social class differences in mortality in middle adulthood. This may to some extent be affected by the nature of our variables, the outcome variable itself, and the age of the cohort.

Family type

The measure for family type was very conservative and not adequate to cover the whole period of upbringing. There was information on whether a person had only one parent living in the same household when aged 10-14, but it was not known how long this situation had endured. Neither was it possible to differentiate single parents according to the reason for their single custody. Earlier studies have shown that, while the death of a parent only has a very small effect, the divorce of parents has a clear effect on subsequent mortality (Friedman et al. 1995) and on other health consequences (Wadsworth et al. 2002). It was not possible to distinguish these two groups in these data, but according to official statistics, approximately 70% of the children in single-parent families in these age groups in 1970 were not living with both parents for reasons other than the death of one parent (Central Statistical Office 1973). Given the impossibility of categorising divorced parents, it is possible that the association of mortality and family type is somewhat underestimated in these data.

It is more difficult to estimate how the lack of information on the age at which one parent was lost is associated with mortality. The effect of the timing of parental divorce may differ by sex. There is an indication that parental divorce when boys were aged 7-12 increases vulnerability to depression in young adulthood more than at a younger or older age (Palosaari & Aro 1994), while age has not been found important in terms of subsequent depression in women (ibid., Rodgers 1994). A single-parent background at preschool age has also been found to be more influential on education than at other times in childhood and adolescence (Krein & Beller 1988). There is no evidence on the effect of the timing of divorce on later mortality. However, because the subjects from divorced family backgrounds accounted for only 7%, maximum, of the total cohort in this study, it is unlikely that this minor underestimation would significantly alter the observation that adjusting for family type has a limited effect on adult social class differences in mortality.

Parental education, material conditions and social relationships in the parental home

Family type and parental social class may not have been able accurately to capture the living conditions in the parental home. There are factors concerning which we did not have any information. On the evidence of earlier studies, what might have been important was parental education (Kuh & Wadsworth 1993, Pietilä & Järvelin 1995, Harper et al. 2002), and particularly that of the mother, which has been shown to be related to both education and IQ (independent of parental social class) in women and men (Kuh & MacLean 1990, Kaplan et al. 2001, Sylva 1997). In these data, parental social class had a very strong effect on the education of their offspring, and thus it could be as-

sumed that the effect of the parental home on the social path is reliably captured. Although it is likely that parental education partly reflects socio-economic dimensions other than occupational class, and has an independent effect on mortality, equally it is unlikely that this effect would essentially change our results on the effect of the living conditions in the parental home on social class differences in mortality. This is supported by the fact that in the British 1946 Birth Cohort, parental education did not have an additional effect on mortality between ages 26 and 54 once the father's social class was taken into account (Kuh et al. 2002b).

Information on the affluence or economic hardship in the parental home might have been important variables to include (see Lundberg 1991b, Rahkonen et al. 1997). For instance, Kuh et al. (2002a) found that living in a council house in childhood had a direct effect on adult psychological distress, while the father's social class did not. The authors suggested that this might be because living in a council house was a more sensitive measure of childhood circumstances than social class in their study. Lundberg found that economic hardship in the parental home (social class not controlled) accounted for more (21%) of the social class variation in mental illness than physical illness (6%). In the Boyd Orr cohort, poor housing conditions in Britain in the 1930s, i.e. lack of facilities, poor cleanliness, and crowding, had only a minor effect over and above parental social class (Dedman et al. 2001). Very adverse, poor living conditions were not very common in the 1960s and early 1970s in Finland. Therefore it is unlikely that the more precise measurement of mere material conditions in the parental home would have provided more information on mortality in our cohort, and this effect is likely to be small on social class differences in mortality in middle adulthood.

What is, perhaps a more serious omission in respect of the effect of the parental home on subsequent inequalities than the lack of information on education and wealth is the lack of information on specific social relations in the parental home (Lundberg 1993, Bosma et al. 1999, Sweeting & West 1995, Cassidy 2000). Although parental social class and family type are related to family culture (Sweeting & West 1995), at best these variables only partly reflect the psychological conditions and social relations in the parental home. Parental care has been shown to be associated with subsequent mental well-being (Kalimo & Vuori 1990), and it may be the most influential factor in terms of subsequent social status and mortality (Francis & Meaney 1999). Care for the child (at age 4) and the house is the only socio-economic indicator from childhood that has been found to have an independent effect over the father's social class on mortality between ages 26-54 in the British study (Kuh et al. 2002b). Moreover, it is not only adverse circumstances, but also favourable circumstances in childhood such as positive events that are likely to have an effect on subsequent health (Carroll et al. 1993). These kinds of elaborations were beyond the scope of our data.

Different characteristics of the parental home may affect health and health differences at different stages of life. For example, it is possible that childhood social relations affect health at younger ages, and that childhood material conditions affect health in later middle adulthood and at older ages (see Sweeting & West 1995). Because social class reflects more material circumstances than social relations in the parental home, it may be an insufficient measure of parental-home conditions in respect of mortality risk in middle adulthood. It is therefore likely that, although the lack of information on parental education and economic circumstances may not have had a strong influence, the lack of information on psychological conditions and social relations such as parental care may underestimate the explanatory power of the parental home on adult social class differences in mortality. In particular, what may be significant is the lack of information on social relationships in the family at the critical period of 0-3 years, and in the whole pre-school period in general. Conditions and experiences in early years may have long-lasting effects on development and social processes, and it may have consequences on health that are visible years later (Scannapieco & Connell-Carrick 2002, Siltala 2002, Montgomery et al. 2000, Maughan & McCarthy 1997, Caspi & Silva 1995, see Wadsworth 1999).

Health in childhood

Information on health status and physiological measures across childhood was not available. Findings on the effect of chronic diseases in childhood on social class differences in adulthood are somewhat controversial. In general, poor childhood health is associated with downward social mobility, not marrying, and unemployment if it continues into adulthood - but this concerns only small numbers of people (see Pless et al. 1989). Furthermore, the effect of deteriorated health status in adolescence may even have a reverse effect on social status, as chronic disease may increase the willingness to study and thus compensate the difficulties caused by the disease (Koivusilta 2000). In fact, the effect of youth paths on mortality is not likely to be accounted for by poor childhood health (see Mare 1990, see 3.3.1). According to the study by Pless et al. (1989) on the British 1946 birth cohort, it seems that chronic illness in childhood increases mortality independent of social class background from childhood up to at least 35 years of age. Of the mortality difference by parental background, chronic illness explained approximately 5.6% in men and 13.6% in women. If the same holds true in our data, it is not likely that the lack of health information essentially affected the associations we found between the parental home and subsequent mortality. Most authors suggest that early health has no substantial effect on social class differences in health and mortality (Mare 1990, Lundberg 1991a, Mheen 1998).

Because we used census data, information on physiological factors such as body length and weight at different ages, and on behaviors such as smoking and nutrition in child-

hood, was not available. Overall material conditions may have been rather good in the 1960's, but it is likely that poor living conditions in the lower social classes may have affected subsequent health in children. For instance, disposable equivalent income in Finnish households measured by the Gini-coefficient was about 30 in the 1960s and early 1970s, while it was about 20 in 1975-95 (Gustafsson et al. 1999). The Finnish cohorts born in the 1950's and 1960's show variation in body height according to social background, which is an indication of the relative differences in standard of living. It may also reflect differences in material aspects, in maternal care, and in smoking, stress and diseases in childhood and adolescence (Silventoinen at al. 2001). The effects of these unmeasured factors on adult social class differences in health may be assumed to be captured to some extent by the variables included in this study, such as parental social class. However, if these variables are not related to the study variables, and if there were significant differences between parental homes in terms of unbalanced diet, for instance (Wadsworth 1999), then our data indicated a too small effect of the parental home.

Mortality and health in adulthood

Death is a rare event in young and middle adulthood: during the follow-up, only 1.7% of our cohort died. However, different health problems are likely to affect a much larger proportion of persons of the same age as our cohort (Rahkonen et al. 1997a, Power & Mathews 1997, Kuh & Wadsworth 1993). Therefore morbidity, and perhaps emotional health in particular (Stewart-Brown and Layte 1997) in early and middle adulthood, might have been a more sensitive measure in respect of the effects of the living conditions in the parental home. On the other hand, a Dutch study (Mheen et al. 1997) found that parental-home characteristics explained only a relatively small proportion of the variation in educational differences in self-assessed health.

Although Mheen's results are in line with the findings in this thesis, generalization of our results should not be extended to apply to morbidity. In our data there were no indicators reflecting health, disabilities, mental or physical symptoms or disorders, well-being, or health potential or capital. The focus was only on *mortality* and social class differences in mortality.

9.1.3 Strengths

Although there are some problems related to register-based data in this study, their overall effect on the effect of the parental home and youth paths measured by socio-demographic variables on mortality is not likely to be considerable. There are also some clear advantages and strengths related to the study design. Our population comprised all persons born in 1956-60 and who were living in Finland during the 1970 and 1990 censuses. The data therefore provided sufficient statistics on deaths to allow us to study mortality in middle adulthood in both men and women. Moreover, there were

no problems related to selection. Another major strength of the data is that all of the information is covered through registers. Given the good registries in Finland, the information covered over the years is not marred by attrition, which is a problem in many longitudinal follow-ups. Furthermore, the linkage of deaths in the census data succeeded well: the percentage of non-linkages was less than one percent. Besides, because information on each year is based on contemporary registries and was never requested retrospectively, it is not weakened by recall bias.

Social class variables were measured with the same exactness in both childhood and adulthood in this study. Socio-economic and sociodemographic variables are hardly ever able to capture all the variation between population groups given the more or less crude measurement, and residual confounding therefore remains (Ben-Shlomo & Davey Smith1991, see 2.2.4). Differences in the measurement precision of the explanatory variables are likely to be reflected in the prediction values so that the most precisely measured variable is the most likely be the most strongly predictive (Marmot et al. 1995). Different levels of accuracy in measurement make comparisons more difficult. For instance, if parental social class is measured more roughly or according to different principles than adult social class, it may be more weakly associated with mortality purely for measurement reasons. Thus, in our data, it is unlikely that differences in measurement at different stages of life had a major effect on the size of the relative effects of these variables. It is therefore likely that the associations found are real, and when no associations were found, if they nevertheless exist, it is likely that they are weak (see Vågerö & Illsley 1995).

Methodologically, the exact measurement of social class and the large number of study subjects provided advantages particularly in the analyses of accumulation. If study subjects are excluded according to their employment status at the beginning of the follow-up, results on the cumulative effect of social class on mortality may be affected. In these data, it was possible to find information on the previous occupational class of those who were not employed in 1990. This enabled us to assess the extent of the exclusion bias. Exclusion of the economically inactive and unemployed men would have lead to underestimation of the social class differences in mortality. As a consequence of this the cumulative effect of social class on mortality from cardiovascular disease would possibly have been overestimated in the analysis. In general, the contribution of the exclusion of economically-inactive persons from the accumulative effect of social class on mortality is likely to differ according to the prevailing mortality pattern.

In addition, in assessing the accumulative effect we were not under duress to sum up the social class scores from different stages of life because the large data made it possible to divide the exposures into several phases of life. If the lifetime exposure to

any social class is constructed by simply summing up the ordinal number of social classes over the lifecourse, the result of the cumulative exposure is likely to be reached as a consequence of the steep distribution of this indicator. For instance, in these data, a clear accumulative effect of social class on mortality was found when a cumulative indicator of exposure to the manual class was used. These results on the association with the cumulative social class indicator and mortality were in line with those in other studies (Davey Smith et al. 1997). However, if the cumulative indicator was split among different lifetime phases, it was revealed that, in these data, mortality was related more closely to adult than to earlier social class.

9.2 The effect of the living conditions in the parental home

9.2.1 The patterning of mortality from childhood to adulthood

In this study, there were social class differences in mortality among those aged 5-9, but they were non-existent or minor among those aged 10-19. This 'disappearance of the mortality gradient' in early adolescence (West 1991) was attributable to small social class differences in external causes and the lack of differences in disease mortality at these ages. Mortality differences by parental social class increased in early adulthood. This increase was not due to social mobility, as the social class information on all participants dated from the time they were aged 10-14. These results were consistent with those of Blane et al. (1994) for male mortality in England and Wales. Rahkonen and his colleagues (1995) also found clearer differences in self-assed health by parental social class after age 25 than in younger age groups in Britain and Finland.

In terms of health differences, adolescence and youth may be regarded as a time of equality in life more than any other (West 1997). Youth is a time of major transitions, which may lower the predictable power of parental class; according to West (1997), youth culture is an equalising factor accounting for small social class differences in health in these age groups (see also Hendry et al. 1996, Starfield et al. 2002). In this thesis, the 're-appearance' of mortality differences by parental social class among men was attributable to alcohol-related causes at age 15-19.

9.2.2 Social mobility

In this thesis, social mobility in relation to mortality was more extensively studied among men than among women. In general, mortality in mobile groups was more closely related to that in the class of destination than in the class of origin.

In most causes of death studied, the men who have moved up from the manual to the non-manual class had slightly higher mortality than the stable non-manual men. This may be attributed, at least to some extent, to environmental effects: those moving may have carried with them the effects of their earlier environment (accumulation). Such factors are more complex than the pure material circumstances that affect a person's subsequent life trajectories (Power & Hertzman 1997). One's own class, which may be the same as the class of origin, is a part of these trajectories. In our study, all of the men in the non-manual class, regardless their class of origin, had lower mortality than the men in the manual class, again regardless their class of origin. However, there was no means of controlling whether the mobile men had adopted the health behaviours, such as drinking habits, and risk-taking behaviours of the receiving class, or if they had them already. According to earlier studies, health behaviors are associated more strongly with achieved social class or position than with the class of origin (Karvonen et al. 1999, Wannamethee et al. 1996).

In this thesis indications of health-related selection (see 3.3, Nyström 1992, Rahkonen et al. 1997b) were weak and mainly based on a slightly elevated disease mortality among those who had descended to the manual class. In all, social mobility constrained adult social class differences in mortality from cardiovascular disease (contraction attributable to mobility was 29%) and alcohol-related causes (24%). In other causes of death, and in total mortality excluding alcohol-related causes, social mobility constrained the mortality difference between the non-manual and the manual classes very little, which is in line with the results of earlier studies (Bartley & Plewis 1997, Hart et al. 1998, Blane et al. 1999, Power et al. 2002, on health Power et al. 1996). The present results thus indicate that social mobility has both selective (Mackenbach & Maas 1989, Bartley & Plewis 1997) and accumulative effects (upwardly-mobile persons carry some exposures from their past), but they have only a very limited effect on social class differences in total mortality (see 3.3.2).

In general, the effect of social mobility on social class differences in mortality apparently depends on the mobility opportunities and mortality patterns in society. As extensive movement is rare (Pöntinen 1983, Blane et al. 1996b), the effect of mobility may remain minor. Prevailing patterns of mortality may have an influence on whether mobility constrains or enlarges mortality differentials. In Finland, where cardiovascular disease is the major cause of death, social mobility is likely to constrain mortality differences, because living conditions in the parental home are likely to have a prolonged effect on cardiovascular disease, and social mobility is directed more upwards than downwards (see also Blane 1999).

It could be assumed that, given health-related social mobility and the constant effects of social circumstances, social class differences in health should increase with age, as

Ford et al. (1994) suggested. However, this has not usually been found in longitudinal studies (Bartley & Plewis 1997, Power et al. 1996). It has been shown that health-related social mobility is more likely to constrain health differences than to widen them. The tendency of relative social class differences not to widen with increasing age in late adulthood, however, is not necessarily an indication of the non-accumulative effects of disadvantage or advantage on health and mortality. It may be due to the effects of increasing morbidity and mortality with increasing age. The accumulative effect of disadvantage may be less salient at higher levels of morbidity and mortality.

9.3 The effect of living conditions in the parental home on mortality and on social class differences in mortality

9.3.1 The association of the living conditions in the parental home with mortality in middle adulthood

Parental social class

Parental social class was associated with mortality from all causes of death in middle adulthood in men, but only with total mortality and diseases in women.

In general, and particularly with regards to all-cause mortality and cardiovascular diseases, the results concerning men and women were consistent with those of many earlier studies, in spite of many differences in study design (Notkola et al. 1985, Hart et al. 1998, Vågerö & Leon 1994, Mare 1990, Kuh et al. 2002b) in terms of size (from small cohorts to census-based studies), composition (general populations or recruited from work places), and age distribution. This means that, apart from showing different patterns of mortality, the different study cohorts are likely to have spent their childhood in very different circumstances ranging from a poor agrarian and urban environment in the 1910s to a more affluent one in the 1950s.

Parental social class was not associated with external causes of death in women after age 15-19. It has been found to have no association with mortality from external causes in late adulthood in other studies on men (Davey Smith et al. 1998a), but in our study, it continued to exert an effect on excess accidental and violent mortality in men originating from the manual class. It also had a clear effect on mortality from alcohol-related causes. About half of the excess mortality due to external causes of men with a manual-class background could be attributed to alcohol poisoning and accidents, and to violence in which alcohol was involved as a contributory cause.

Parental class was found to be associated with adult social class in both women and men, which was consistent with the results of earlier studies. Due to the different

occupational distribution by sex, the daughters of manual-class parents more often moved to the lower non-manual class, whereas the sons of manual-class parents remained there. Parental class had a small effect on adult class over and above the person's educational achievement. For instance, in this study, among women who had a higher education, the probability of being in the upper non-manual class was greater if the parents were in the same class than if they were in the lower non-manual (OR 1.4, 1.2-1.3), the skilled manual (OR 1.6, 1.5-1.7) or the unskilled manual class (1.8, 1.6-2.1). The odds ratios for men were 1.1 (1.0-1.2), 1.2 (1.1-1.3), 1.6 (1.3-1.8), respectively.

The association between the parental and adult social classes was mainly attributable to the contribution of parental class to the educational, marital and employment paths. The associations between the parental class and the youth paths were similar in women and men, only the association with the marital path being slightly stronger in women. In general, the manual parental social class was associated more often with lower educational attainment and unemployment, and less often with late marriage. Consistent with our study, in the 1958 British Birth Cohort, parental social class was found to be related to educational qualification and unemployment in young adulthood, and also to work characteristics such as job insecurity (Power and Matthews 1997). The effect of parental social class on youth paths highlights the importance of the pathway model in explaining mortality and social class differences in it (see also Marmot et al. 2001, Power and Hertzman 1997, chapter 10.1.3).

Family type

In our data, coming from a single-parent household meant increased mortality at age 31 to 43 in men from all specific causes and in women from all-causes and diseases. In women, the youth path and the adult social class accounted for the excess mortality attributed to a single-parent home background, while in men, the excess remained statistically significant after all our controls; i.e. those with a single-parent background had higher mortality than those from two-parent families in all adult social classes.

It was not possible in these data to disentangle the effect of family type from the economic, behavioural or parenting components. We only had information on parental social class, which may reflect, at least to some extent the economic situation in the parental home. Single parenting was clearly more common in the lower non-manual class (the most common female occupational class) and in the unskilled manual class. However, controlling for parental social class and other parental home factors did not explain the higher mortality of the offspring from single-parent homes, although it reduced the excess mortality in women. Similarly, it has been found that adjustment for parental class, the educational level of parents, and the home conditions in pre-school years does not alter the relationship between the childhood family type and

adult mental symptoms (Rodgers 1994). It is possible that part of the effect of family type found here reflects psychological factors such as dissension in the family, which have been shown to have a direct effect on adult health (Lundberg 1997). However, direct evidence of the mechanism between family type and mortality is lacking.

Some of the association of family type and subsequent mortality occurred through its effect on adult social class attainment. In this study, the likelihood of a person from a single-parent family joining the upper non-manual class was lower than for those with two parents. The odds ratio for men to achieve upper non-manual status was 0.75 (95% CI 0.72-0.79), and for women 0.70 (0.67-0.73), adjusted for parental social class. This disadvantage was accounted for by educational attainment, particularly in women. The excess mortality of men from single-parent families has also been observed in other studies. In the cohort of intelligent Californian men and women born in 1905-14 and followed from 1922 to 1991, parental divorce before the age of 21 shortened life expectancy by more than 4 years (Schwartz et al. 1995). Accordingly, in the North-Finnish 1966 birth cohort, suicide was more common among men from single-parent families (Sauvola et al. 2001a). In this study, too, men originating from single-parent families had a higher suicide rate than men from intact families. However, the higher mortality of men from single-parent families was not accounted for by their higher suicide risk.

The subjects of our data spent their childhood during a time when single-parent families were not as common as they are today. Single-parent families consisted of 11% of all families with children of 17 years old and under in 1960 and 1970, while the equivalent figure in 1999 was 19% (Statistics Finland 2000:12). In this respect, one could assume that growing up in a single-parent family today would not be as stigmatising, and not as materially disadvantageous due to better social benefits. Health consequences might therefore be expected to be smaller in younger cohorts. However, Ely et al. (1999) found that the effect of parental divorce on children's educational attainment did not change among children born in 1946, 1958 and 1970, when the prevalence of divorce had increased from 6% to 18% (see also Joshi et al. 1999, Elstad 1996). These results may also indicate that the effects of divorce on health are due to reasons other than stigma. Alternatively, other causes such as income problems may exceed the positive effects of changed social attitudes towards divorce (see Ely et al. 1999). The economic situation of single-parent families is generally weaker than average in most countries, although the material conditions differ between countries due to different social-security benefits and taxation systems (Graham 2002), as well as selectivity into single parenthood. This complicates the generalisation of the results of this study to other countries.

Moreover, it is likely that not all children from single-parent homes have increased premature mortality risk; 2.7% (0.9%) of men (women) died during follow-up in contrast to 1.7% (0.6%) from intact families. Low self-esteem has been found to mediate the effect of parental divorce on depression in young adulthood (Palosaari et al. 1996). It could be that persons with low self-esteem comprised the group with an elevated risk of death.

Number of siblings

In these data, the women and men with one sibling had lower total mortality than those without any or with several. Likewise, men with many siblings had 27% higher mortality from alcohol-related causes than men with one sibling, which was in accordance with earlier findings reported by Kemppainen et al. (2000). The men without siblings at age 10-14 showed elevated mortality from external causes – and the women without siblings had small excess mortality.

In our cohort, both men and women with at least two siblings were less likely to move upwards (odds ratio 0.80) and more likely to move downwards (1.10 for men and 1.36 for women) on the social scale than the subjects with one sibling or without any. This result (not shown in the papers) is congruent with the findings of Blane and his colleagues (1999) for Great Britain. If growing up in a large family has an effect on subsequent mortality, the same kind of risks exist among those without siblings, although the factors behind these associations may be different. In any case, the number of siblings had only a small association with adult social class and mortality.

9.3.2 The effect of living conditions in the parental home on social class differences in mortality in adulthood

In this cohort, living conditions in the parental home, particularly family type and parental social class, were associated with mortality in adulthood in men, although their direct effect on adult social class differences in mortality proved to be minor. There are several reasons for these small effects over and above the data problems. The most obvious reason for the small effect of living conditions in the parental home on mortality inequality was that the associations between the two were substantially weaker than those between the adult social class and mortality, and therefore they cannot explain the whole difference (see also Davey Smith et al. 1990).

Furthermore, some of the adverse conditions in the parental home were not common enough to have any substantial effect on mortality patterns. For example, family type was found to have a small direct effect on mortality in men, which was comparable to the effect of dissension in the parental home in Lundberg's study (1997). However, in this study only 10% of the men came from single-parent families. Even if all these men

had died during the follow-up, it would not have been enough to fully explain the mortality difference between the non-manual and manual classes. In addition, although characteristics of the parental home such as family type impinged on the adult social class, these associations were not strong enough to have a substantial effect on social class differences in mortality.

However, if the hazards of adult life were weaker, the potential effect of past circumstances on social class differences in mortality could be stronger. For instance, in the Swedish cohort of men aged 25-40 years, the effect of the parental social class was stronger, explaining approximately 17% of the excess all-cause mortality (in this cohort 8%) and 32% of that from ischaemic heart disease (in this cohort 24% in cardiovascular diseases – ischaemic diseases were not separated here) (Vågerö & Leon 1994). Mortality is lower and adult social class differences are smaller in Sweden than in Finland. In addition, 12% of the men in the Swedish cohort were classified as 'others' due to non-employment or other reasons. These 'others' had more than sixfold mortality in comparison to the men in the non-manual class. Of the excess mortality of the 'others' parental class explained clearly less than among the classified men: 5.6% from all causes and 13.3% from ischaemic diseases (versus 17% and 32%). If we had excluded unemployed men from our cohort, which would have made adult circumstances more homogenised, the effect of parental class on adult social class differences in mortality would have increased.

Bearing all these facts in mind, it is understandable that living conditions in the parental home had only a weak direct effect on social class differences in mortality in this relatively young cohort (see Wagstaff et al. 2001). It is possible that the material circumstances in the 1960's were so good that the effect of the parental home in terms of material factors will not essentially increase when the cohort ages and chronic diseases that may have their origin in the material circumstances in early life become more common (see Chapter 4.5). On the other hand, if the psychological mechanisms related to the parental home become more important with increasing age it may strengthen its effect on social class differences in mortality (see Bosma et al. 1999).

The living conditions in the parental home, which were in focus in my thesis, had only a small direct effect on adult mortality differences. However, they were clearly related to adult social class. Power and Hertzman (1997) concluded: *"Childhood environment can be regarded as contributing to adult disease through its strong influence on adult socio-economic circumstances".* The results reported in this thesis confirmed this, and showed further that the living conditions in the parental home had an effect on the transitions a person went through in youth, i.e. youth paths, and contributed to the effect these paths exerted on adult social class differences in mortality from various causes of death. This is consistent with Mare's study (1990), in which the gross effect

of the father's social class on adult mortality was, to a large extent, mediated through its effect on educational achievement (see also Lynch et al. 1997).

It is possible that the effect of the parental home on adult social class differences in mortality could increase indirectly when the effects of youth paths on later advantages and disadvantages accumulate (see also Blane 1999). In this study, from 3% to 33%, depending on sex and cause of death, of the effect of youth paths on social class differences in mortality were attributable to the living conditions in the parental home. A discussion on these effects follows in sections 9.6.2 and 9.6.4. The conclusions regarding the lifecourse mechanism in terms of the parental home and youth paths are summarized in Chapter 10.1.

9.4 The effect of youth paths on mortality and social class differences in mortality

9.4.1 Educational path

In our cohort, educational attainment was related to parental social class, which was consistent with earlier findings (Pöntinen 1982, Sandford et al. 1994, Koivusilta 2000, Isohanni et al. 2001, Graham 2002). The effect of the parental home on educational attainment seems to be very pervasive. For instance, for Finland, this effect has also been observed in cohorts born after 1956-60 who have gone to school after the school reform, which was planned to attenuate socio-economic inequalities in education, among other things (Valkonen et al. 1996). Further, in our cohort, educational attainment was strongly associated with adult social class in a similar way in women and men. Thus, as regards educational qualifications, the requirements for men and women to be in the upper social levels seemed to be similar. Meanwhile, men with higher education were more likely to be in the upper non-manual class as opposed to any other class than women (odds ratio: 2.14, 95% CI 2.06-2.22). However, the effect of education on adult social class, particularly in terms of differences between the sexes, depends on the distribution of adult social class by sex - which is affected by the employment possibilities for both sexes - and also on ways other than through education of reaching the upper non-manual class (see Chapter 2.3.2).

Consistent with earlier findings, educational path was strongly associated with mortality from all causes in men and women (Valkonen et al. 1993, Mackenbach et al. 1999, Kitagawa & Hauser 1973, Valkonen 1989, Sorlie et al. 1995, Backlund et al. 1999). Among young and middle-aged adults, different health indicators such as self-rated health have shown larger differences by education than by adult or parental social class in Finland and Britain (Rahkonen et al. 1995, Manor et al. 1997). In this thesis,

adjusting for educational path abolished mortality differences between the upper non-manual and lower non-manual classes, the exception being suicide in women. The excess mortality in the manual class was reduced by 65-80% in all specific causes of death after adjusting for educational path.

The strong effect of educational path on social class differences in mortality is likely to be through its strong effect on social class in Finland; this association is likely to explain, to a large extent, why pathway influence is important in shaping lifecourse relationships between social class and mortality (Graham 2002:2008). Educational path is likely to partly mediate the effects of earlier lifecourse phases. However, in this thesis, the variables concerning parental home were adjusted first, and therefore it is likely that the effect of education was largely based on factors directly related to it, or on factors that are related to youth but reflected by education (see Chapter 5.2.2). These factors include psychosocial mechanisms and health-related behaviors. Health-related behaviors might be more strongly related to youth and education in our cohort born in 1956-60 than in older cohorts (see West 1997). It is possible that, in older cohorts, health-related behaviors may have been more clearly adopted in the parental home and strengthened by the adult social class, on which the parental class had a stronger direct effect than it does at present. In line with the above hypothesis, among older men, smoking and mortality were more strongly associated with adult social class than with education (Davey Smith et al. 1998b). The stronger effect of education in younger age groups may also be attributed to the fact that any exposure to occupational or other adult risk factors in early middle adulthood cannot have been very long-lived. Whether the strong effect of education in explaining social class differences in mortality is a cohort or an age effect will be seen in the future.

In sum, educational path was an essential part of the social-programming process from the parental home to the adult class. It also mediated the effect of the parental home, but it had a strong independent effect on social class differences in mortality in middle adulthood.

9.4.2 Family formation

In this thesis, parental social class and family type were associated with family formation. Those who married early or had had a child before the age of 20-24 more often came from the lower social classes and single-parent homes. For instance, 32% of women with an unskilled manual background were early parents, versus 10% from the upper non-manual class. This is in accordance with the findings for the 1946 British Birth Cohort (Kiernan 1986, Kuh & Maclean 1990). Furthermore, early family formation is also an instigator of future processes, such as the formation of one's social class, which in this study was more likely to be a lower one in both women and men (see also Hobcraft & Kiernan 2001).

Women and men who had children or married at an early age had higher mortality than those who established their family after the age of 20-24. These results confirm earlier findings on the association of early family formation with subsequent poor health (Power et al. 1998, Hobcraft & Kiernan 2001, Mirowsky & Ross 2002, Grundy & Holt 2000). Judging by the variables available in this study, the group of people who had not married early but had a partner at the age of 30-34 had more favourable characteristics, such as higher social background and education, than those in other categories on the marital path (see also Nikander 1992). However, variables related to social background did not account for the effect of marital path on mortality in this study.

On the basis of these results, it seems that the effect of marriage (see Mastekaasa 1992, Goldman 1993) on mortality in middle adulthood is related to age at marriage or cohabitation. It is possible that part of the excess mortality related to early marriage was attributable to subsequent factors such as divorce and health behaviors (see Power et al. 1999b). For instance, in our data, women and men[4] who were married at age 15-19 had clearly higher mortality from alcohol-related causes. On the other hand, it is possible that some background factors, such as disadvantaged parental home and problems with school and behavior, affected early marriage, possible divorce, and premature mortality (see Chapter 5.2.3). These factors are likely to be related to the prevailing culture and marital patterns, and therefore to the association between early marriage and later mortality.

Having no partner at age 30-34 was also associated with excess mortality in both women and men. This is consistent with earlier results on elevated mortality among those who have never married (Ben-Shlomo et al. 1993, Koskinen & Martelin 1994b, Ebrahim et al. 1995, Cheung 2000). However, the group without a partner was different in women and men, a higher social background and higher adult social class being more common for the women.

In women, the effect of early family formation on social class differences in mortality was approximately 10% in various causes of death, i.e. about the same size as that of the parental home. This effect was greater in men, varying from 16% in external causes to 27% in all diseases. Early parenthood made only a minor contribution to this effect, because only a few percent of subjects had their first child young.

The effect of marital path was partly attributable to different factors in women and men. All of those who married very early or who had no partner in 1990 had higher

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[4] Only 1.4% of the men were married at that age but their age-standardized mortality rate from alcohol-related causes was 331 per 100,000 men versus 99 per 100,000 in the whole cohort.

mortality, but the proportion of early married was too small in men to contribute to differential mortality, and in women the proportion of those without a partner was actually higher in the upper non-manual class. Therefore, in women, the effect of family formation could be attributed to the higher mortality of those who marry young and their larger prevalence in the lower non-manual and manual classes. In men, a considerable proportion of the excess mortality among the manual classes could be attributed to the high proportion of men without a partner.

9.4.3 Employment path

In this study, the living conditions in the parental home were associated with the employment path. Women and men experiencing unemployment were more likely to have an adverse background than average: they included 19% more women from a single-parent home and 44% more women from the unskilled manual class, than might have been expected on the basis of the population proportions in these groups. The equivalent figures for men were 35% and 50%. This finding is in accordance with the findings from the British 1946 and 1958 Birth Cohort studies (Wadsworth et al. 1990, Power & Mathews 1997). Employment path was further associated with adult social class in both sexes. In women, the prevalence of steady employment was clearly lower in the manual class than in the non-manual classes, within which there were no differences, while in men it decreased with decreasing social class.

Further, employment path was associated with mortality. Those who were in steady employment had lower mortality from all causes of death than any other group. Part of the explanation for this lower mortality lies in their more advantaged background. It is possible that those in this group were also already career- and health-oriented when they entered the labour market. Some of the lower mortality among those in steady employment is likely to be attributable to its beneficial effects, such as security in income and social networks.

In our data, women and men who had experienced short- or long-term unemployment in their youth had clear excess mortality from all specific causes of death. However, mortality was approximately twice as high among those who were unemployed long term than among the short-term unemployed. The association of unemployment with mortality has been shown earlier in men and women (Stefansson 1991, Hammarström 1994, Martikainen & Valkonen 1996, Nylén et al. 2001, Kposowa 2001). The length of unemployment has also been shown to be significant in terms of mortality (Martikainen & Valkonen 1996, Jin et al. 1995). These data further show that the effect of unemployment experienced in youth or early adulthood on mortality is very persistent: excess mortality was observed even 18 years after the unemployment experience. An earlier study by Kposowa (2001) showed that excess mortality from suicide among

those who had experienced unemployment vanished in men after the fourth year of the follow-up, but was not affected by the length of the follow-up in women.

Serious antecedent illness may account for the effect of unemployment on mortality (see Pless et al. 1989, Bartley & Plewis 1996). However, this effect is likely to be small (Jin et al. 1995). Furthermore, unemployment rates among people of the same age as this cohort were low in Finland, varying between 7.2% in 1980 to 2.8% in 1990. Thus, the characteristics of unemployed people as well as the experience of unemployment could be different for this cohort than for the young persons who were unemployed during the period of the high youth unemployment (Iversen 1987, Bartley 1988, Martikainen & Valkonen 1996, Julkunen 2001). Nevertheless, it is possible that young adulthood is a socially sensitive period in terms of the effects of unemployment on the formation of adult identity and material life circumstances, and thus has a long-term effect on mortality risk (see Chapter 5.2.4). However, it was not possible to adjust for psychosocial processes or to observe this mechanism more closely in these data.

In this thesis, the categories of employment path were differently distributed between adult social classes, and they substantially contributed to social class mortality differences. Both short-and long-term experiences of unemployment had the strongest effect on subsequent mortality. The higher frequency of long-term-unemployment spells in the unskilled manual class contributed to the excess mortality in that class. The effect of unemployment on social class differences in mortality is not likely to be fully attributed to the consequences of unemployment; according to this and earlier studies (Bartley 1988, Hammarström 1994, Valkonen & Martikainen 1995, Fergusson et al. 2001), some of the effect of unemployment on mortality is a reflection of earlier exposures that affected both the formation of the employment path and, through that, adult social class and mortality risk.

9.4.4 The combined effects of youth paths on social class differences in mortality

The previous section discussed the effects of youth paths on social class differences in mortality one by one. The total effect of youth path on adult social class differences in mortality was substantial. A major proportion of this effect was attributable to factors in youth independent of the effect of living conditions in the parental home. Among women and men, excess mortality in the manual class over the upper non-manual class in particular was attributable to the higher proportion of those with basic education and unemployment experiences. There was also a higher proportion of single men and early-married women with elevated mortality in the manual class. Furthermore, having no disadvantageous youth-path experiences was characteristic of the upper non-manual class, whereas the number of such experiences increased from the low non-manual to the unskilled manual class. The clustering of disadvantages on the educa-

tional, family-formation and employment paths was associated with an ever-increasing mortality risk. However, this increase was not over and above what would have been expected on the basis of the effect of the single risks related to these disadvantages.

In these analyses, the interactions between adult social class and living conditions in the parental home or youth paths did not prove to be significant. This could be interpreted to mean that the effects of life events along these paths on mortality were similar in all social classes. These findings support those of a study by Stronks et al. (1998b), who showed that poorer self-rated health in lower educational groups was associated with a greater prevalence of stressful events and conditions rather than with a stronger association of these factors with health. However, although in this thesis, the effect of different life events on youth paths was basically similar in different social classes, this may have happened through a different mechanism, which we cannot assess.

Unemployment experiences in youth, disrupted education, and early marriage or staying single up to age 30-34, could be regarded as exclusion from core social roles. According to Siegrist (2000), this kind of exclusion, which means disturbances and breaks in social exchange, leads to impaired personal self-regulation and 'social reward deficiency' (or effort-reward imbalance). 'Social reward deficiency' may lead to prolonged stressful experience followed by adverse health consequences through damaging health behavior and neuroendocrine processes: this is beyond the scope of this thesis. However, the framework offered by Siegrist (2000) provides an intriguing mechanism that may help us to understand how youth paths may affect health and mortality risk.

9.5 The effect of the lifecourse on mortality from specific causes

9.5.1 The effect of the parental home on social class differences from specific causes

The above discussion mainly focuses on the effect of the parental home and youth paths on adult social class differences in total mortality. There are also differences in the effects of these factors on different causes of death. Furthermore, lifecourse models that explain the development of social class differences in mortality are likely to differ by cause of death. In the following, cardiovascular diseases, suicide and alcohol-related causes are considered separately. The parental home was found to have a substantial effect on social class differences in middle-adulthood in mortality from cardiovascular diseases in both women and men, and from alcohol-related causes in men. Furthermore,

there was some indication of an accumulative effect of social class from childhood to adulthood in both causes. The parental home had no effect on adult social class differences in suicide among women, and the effect of the youth paths was smaller than in other causes of death in both women and men.

9.5.2 Cardiovascular disease

Earlier results have shown a consistent effect of early living conditions on cardiovascular diseases mainly in men born before the Second World War and aged 60 and above at death (Notkola 1985, Davey Smith et al. 1997, Frankel et al.1999, Wannamethee et al. 1996, Hart et al. 1998). Our cohort was born in 1956-60, and thus probably lived in more prosperous circumstances in their childhood. Even then, the parental home had a clear effect (18-37%) on adult social class differences in mortality from cardiovascular diseases in both women and men. Two other studies concerning younger age groups, that is those born in the latter part of the last century and studied in early or middle adulthood, showed parental social class to be associated with cardiovascular (Östberg & Vågerö 1991) and coronary heart disease in men (Vågerö & Leon 1994).

Although the parental home had a clear effect on adult social class differences in cardiovascular-disease mortality, approximately 60% of the difference was attributable to factors subsequent to living conditions in the parental home, i.e. youth paths. On the basis of these results, it seems that the risk of cardiovascular disease develops throughout life, although having a manual background seems to have a persisting influence that was apparent in adulthood in both the non-manual and the manual social classes.

These findings point to the importance of a cumulative effect on mortality from cardiovascular disease in accordance with the results of Hart et al. (1998, see Chapter 10.1.2). The accumulative effect describing a pathway from adverse circumstances in childhood and youth to increased risk of cardiovascular disease in a lower social class in adulthood may partly be accounted for by faster atherosclerotic progression for those with higher cardiovascular reactivity (Lynch et al. 1998). Heightened cardiovascular reactivity may be a consequence of "*a cascade of undesirable social circumstances*" (Lynch et al. 1998). There are indications that greater cardiovascular stress reactivity, chronic life stress, and mundane stressful events are more prevalent in lower classes (Steptoe & Marmot 2002). Therefore, a higher prevalence of cardiovascular disease, and possibly higher cardiovascular disease mortality, in the lower classes may be partly accounted for by synergetic interaction with the current stressful circumstances, clustered disadvantages across youth paths, and a biological disposition determined by early circumstances. Our data thus confirm the statement by Davey Smith and colleagues (2001) that coronary heart disease is '*a cause of death which illustrates the life-course perspective par excellence*'.

Generalization of these results to older age groups should be done cautiously. First, albeit cardiovascular disease is the most common cause of death from diseases after cancers at ages 31-42, the level of mortality is as yet rather small. Therefore it was not possible to separate cardiovascular diseases into specific causes in the mortality analyses, and particularly not into different cerebrovascular diseases and coronary heart disease. These cause the majority of deaths from cardiovascular disease, which was the case in this cohort and also applies to persons aged 50 and over. However, the distribution of specific cerebrovascular diseases differs by age. In this cohort, cerebral haemorrhage was the most typical cause of death in this respect, while ischaemic stroke become more common with increasing age. Earlier studies have revealed an association between strokes and coronary heart disease and the parental home (Frankel et al. 1999, Davey Smith et al. 1998b, Notkola 1985, Kaplan & Salonen 1990, Vågerö & Leon 1994, Frankel et al. 1999). Thus, because the parental home has been associated with mortality from cardiovascular disease in different age groups and in both sexes, and because ischaemic causes, which might be more strongly related to longer development, become more common at older ages, it appears that extrapolating these results to older ages may be possible.

Second, the effect of the parental home on social class differences in cardiovascular mortality was weaker than the effect of youth paths. It is difficult to project whether the effect of the parental home will change when this cohort grows older. Its future cardiovascular mortality will depend not only on exposures early in life, but also on subsequent exposures across the lifecourse, many of which have not yet occurred and which may even affect mortality in different directions.

In sum, to the extent that mortality from cardiovascular diseases reflects morbidity, these results provide support to suggestions that cardiovascular disease is likely to develop earlier in people with a poor socio-economic background (Kaplan & Salonen 1990). The accumulation of manual-class circumstances from the parental home to the adult class increased mortality risk from cardiovascular diseases in middle adulthood and contributed to our understanding of the social class differences. These effects were mainly mediated through youth paths.

9.5.3 Suicide

Suicide was the most common single cause of death in women and men during our follow-up, constituting 18% of all deaths and 48% of external deaths in women, and 27% and 47% in men, respectively. There was a clear social class gradient in both sexes. Adjustment for the parental home slightly decreased these differences in men, but increased them in women. A single-parent background was associated with an elevated suicide risk in both men (55%) and women (28%), thus supporting earlier

findings (Sauvola et al. 2001a). Adjusting for other parental-home factors and youth paths abolished the elevated mortality of persons with a single-parent background in women, and reduced it to 23% in men.

Parental social class was inversely associated with suicide in men, while women coming from the upper non-manual class did not exhibit a lower risk. Adjusting for parental social class increased adult social class differences in suicide in women, because women with an upper non-manual background had a higher suicide risk in the lower non-manual and manual classes. It is possible that suicide risk in middle adulthood is associated with the size of the difference between early aspirations (related to conditions in the parental home) and later reality (the current lower social class) (Kuh & Maclean 1990:134, see also Davey Smith et al. 2001). However, in the results reported here, the effect of parental class was small and may have been attributable to chance variation. It is also possible that the slightly higher suicide mortality of women with an upper non-manual background was real, but specific to this cohort. When the women in this cohort were children and adolescents in the 1970s, women in the upper non-manual class had a higher suicide rate in Finland (Valkonen 1990), which may have had a small effect on the daughter generation.

It is possible that the parental home had an association with suicide attempts, although no association was found with successful suicide. There are approximately ten to 25 times more people who have "come close to taking their own life", or who have made attempts to kill themselves, than people who have actually committed suicide; these estimations are even higher in young people and in women (Malone et al. 1995, Eckersley and Dear 2002, Maris 2002).

The associations between youth paths and suicide were similar for men and women, and did not differ from the associations with other causes of death (see Neeleman et al. 1998). Lower education, early marriage or staying single, and having spells of unemployment, were related to higher suicide rates. Adjusting for youth paths contributed to social class differences in suicide less than in the other causes of death. In men, the social-class gradient for suicide was reduced but still remained after adjustments, while the pattern was different in women. In the fully adjusted model, suicide mortality for women in the manual, intermediate and lower non-manual classes was at the same level, but approximately 150% higher than the level in the upper non-manual class.

In our cohort, 49% of the women and 62% of the men who committed suicide were intoxicated. The frequency in men was higher than in other studies showing a 50% incidence of intoxication at the time of death (Maris 2002). The excess in our study could be attributable to the young age of the cohort. We had no information on the history of alcohol consumption among the deceased. Therefore we were not able to

distinguish those who only drank to get the courage to bring their intention to a conclusion from those for whom alcohol was part of the suicide process. However, as the parental home may provide a model for alcohol use (see the next section), it may also be connected to suicide through alcohol consumption as well.

It is apparent that there may be differences between men and women in terms of how the living conditions in the parental home and factors related to youth paths affect suicide risk. First, according to these results, a single-parent home background had a more permanent effect on men than on women. Being from an upper non-manual background slightly increased the suicide risk for the women in the lower classes, but it is not clear whether this was an association related to this cohort or to the psychosocial circumstances in childhood and adulthood. Second, although the youth paths were related to suicide in both women and men, they accounted much less for social class differences in women. This may be attributable to the different pattern of social class differences in suicide between the sexes. There is some uncertainty here due to the low level of suicide among women, but it is not unlikely that the social factors affecting the probability of suicide in women are the same as in men. Suicide was more related to contemporaneous than earlier factors in both sexes, although more pronouncedly in women.

9.5.4 Alcohol-related causes

In these data, alcohol-related causes accounted for 21% of all deaths in women, and 45% of those in men. Characteristics of the parental home were clearly associated with alcohol-related mortality in men, and the excess mortality of those with a manual class background remained even if it was substantially reduced after adjusting for education and adult social class. Moreover, in men, mortality variation in all causes of death by parental home was attributable to a great extent to alcohol-related causes, which accounted for a stronger effect of the parental home on adult differential mortality than in women. The association of parental home with alcohol-related mortality was modest in women.

This association in men, but not in women, may be related to sex differences in alcohol consumption in the 1960s and 1970s when the cohort members were children and adolescents. In pre-1969 Finland, only state-owned retail monopoly stores and 940 licensed restaurants, located mainly in cities, were allowed to sell alcohol that was stronger than 2.8%. Alcohol consumption was low particularly in rural areas, and more than 30% of the population were abstainers, and an even higher proportion of women. (Mäkelä 2002, Mustonen 2003.) Generally, women drank very infrequently (2-3 times a year) and even then very little. The approximate annual consumption was 40 cl; which was 12% of the consumption of men (Mustonen 2003). Furthermore, while

about 10% of higher-educated women were abstainers, the equivalent figure for lower-educated women was 50% (Mäkelä 2002, and Pia Mäkelä's personal information based on unpublished data used for the above paper).

These large sex differences in alcohol consumption in the parental home, and the possibility that parental drinking habits may influence their children's drinking so that the daughters of heavy-drinking mothers become excess drinkers themselves and boys adopt their fathers' drinking styles (see Kuh & Maclean 1990), may partially explain the weak association between the parental home and alcohol-related mortality in women. In addition, men's drinking habits could be more influenced by the drinking patterns in the parental home than by the use of alcohol as such. Social classes consume roughly equivalent amounts of absolute alcohol, but drinking is more aimed at intoxication in the lower classes (Mäkelä 1999).

Of the excess all-cause mortality of lower non-manual and manual class men over upper non-manual class men, approximately 40% was attributable to alcohol-related causes, while in women, 25% of the excess mortality of those in the intermediate non-manual class, 56% of those in the lower non-manual class and 38% of those in the manual class was related to alcohol-related causes. The parental home accounted for 15% of adult social class variation in alcohol-related causes in men. However, the main effect of the factors preceding adult social class on differences in alcohol-related mortality was accounted for by youth paths. Adjusting for education and employment paths substantially decreased the differences between the social classes, while the marital path had a minor effect (10%), in both women and men. Therefore it seems that, in this cohort, youth was more important in the adoption of risky drinking patterns.

10 Conclusions

10.1 The lifecourse: latency, accumulation, or pathway?

Lifecourse experiences before transition to the adult social class, i.e. living conditions in the parental home and youth paths, may be related to adult disease and social inequalities in mortality through latency, accumulation or the pathway model. According to the latency model, the origin of disease is in early childhood, although the initiation of disease leading to death might be years later. The critical period for disease has been childhood, and subsequent exposures may at best modify the progress of disease. A latent effect of living conditions in the parental home on adult mortality and mortality differences is most likely to develop simultaneously or in co-evolution with social pathways. The accumulation model sets out no specific critical period in early childhood for the onset of disease, although exposure in childhood is likely to increase the risk of disease, in combination with subsequent exposure in youth and later life. According to the pathways model, living conditions in the parental home affect the formation of youth paths that lead to adult social class and health hazards related to adult circumstances. In pathways model, the development of disease and the transition to the adult class may also be related to biological and social processes on youth paths.

10.1.1 The latency model

In these data, one indication of the latent effect was the continuous and slightly increasing effect of parental class on disease mortality from age 20 onwards in women and men. However, the effect of the parental home on disease mortality that was independent of subsequent exposures was small. Although it is unlikely that economic hardship or social problems in the parental home increases general vulnerability to disease years later (Lundberg 1991b, 1993), it is possible that the contribution of the latency model to social class differences in mortality will increase modestly in some specific causes of death. This increase will depend on the prevalence of diseases that initiate at older ages (as yet rare in our cohort), but which have their onset in the living conditions in early life, such as ischaemic cardiovascular diseases. The material living conditions in childhood were relatively good here, but there were more people with a more disadvantaged background in the manual class than in the non-manual class. Thus it is likely that there are more persons exposed to disease in the manual class. Furthermore, if the progress of disease is modified by adult circumstances in a way that exacerbates its development, it is likely that the latency effect on social class differences in mortality may intensify with increasing age.

It seems very apparent from this thesis that early circumstances contribute to adult social class mortality differences through a latent effect, but it is likely that this effect will not prove to be very substantial.

10.1.2 Accumulation

The length of exposure clearly contributes to disease outcome. For example, the health risks are different depending on whether a person is exposed to tobacco smoke for a month or for 30 years, or to mouldy housing for several days or for several years. Therefore it is important to assess the accumulative effect of the particular condition on mortality and social class differences in mortality. It was also shown in this thesis that in studying the accumulative effects, childhood and adulthood should be differentiated to make it possible to assess the contribution of social circumstances at different stages of life on mortality and social class differences in mortality. This is meaningful, as the risk factors for specific causes of death are likely to differ or to have a different impact in childhood and in adulthood (see Davey Smith et al. 1997:551, Vågerö & Illsley 1995). The parental home was shown to have an effect on social class differences in mortality from diseases in women and men, and in external causes in men. Similarly, differential independent effects of childhood, adolescence and adulthood on physical health among adults have been detected in the British 1946 and 1958 National Birth Cohorts (Kuh & Wadsworth 1993, Power et al. 1999a). Furthermore, without differentiation of different life stages, it is not possible to assess whether it is a question of the effect of accumulation or the effects from different phases of life (see Mheen et al. 1998a, Mheen 1998:229).

A small accumulative effect of social class was found in mortality from cardiovascular diseases and alcohol-related causes. These causes of death were associated with both the parental and the adult social class: the longer a person had been in the manual class, the higher the mortality risk. No such effect was identified in other causes of death. There is a lot of evidence that cardiovascular disease develops gradually during life, and may have its origins in the circumstances of early life (see 9.5.2 and Davey Smith et al. 1997, Kaplan & Salonen 1990, Vågerö & Leon 1994, Koskinen 1994, Notkola 1985). The effect of accumulative disadvantage may be different for other causes of death and through other mechanisms or exposures.

The accumulative effect found in studies by Power et al. (1999a) on the 1958 British birth cohort, and by Hart et al. (1998) on the Scottish male cohort employed at age of 35-55 at the beginning of the follow up, was stronger than in this study. This is probably because the differences in the variation of mortality and health by social class were small in the former studies, regardless of whether social class was measured in childhood, or in early or later adulthood. Thus the impact of the accumulative effect of

social class may prove to be stronger than in the data in which the mortality was more strongly associated with the adult than with the parental social class. The small amount of evidence of an accumulative effect found in this thesis may also be attributable to the roughness of the social class distinction in capturing the effect of a number of small disadvantages across life. Social class may not be adequate for capturing the continuity of disadvantageous environmental circumstances that intensify the risk of poorer health and premature death (see Blane 1999, Rutter et al. 1990). Moreover, this cohort may be far too young to be lethally affected by adverse circumstances even if exposed to them for a long time.

10.1.3 Pathway effects

The results of this thesis mainly confirmed Lundberg's (1993) notion of 'the unhealthy life career', i.e. the pathways model. Poorer conditions in the parental home, particularly in respect of social class and family type, were associated with poorer conditions on youth paths. Lower education, unemployment experiences in young adulthood, early family formation in women, and staying single in men were further associated with low adult social class. Disadvantages on youth paths increased the risk of premature death.

With respect to explaining social class differences in mortality, the effect of the parental home was mainly based on its effect on youth paths, which was in accordance with some earlier findings (see Marmot et al. 2001, Power & Hertzman 1997). This further supported the pathways model. In addition, youth paths mediated the effect of living conditions in the parental home on social class differences in mortality. A moderate proportion of the effect of youth paths on mortality from cardiovascular diseases in both sexes, alcohol-related causes in men, and all diseases in women, was attributable to the living conditions in the parental home (see Lundberg 1991a). Nonetheless, youth paths exerted a strong independent effect over and above the effect of the parental home on social class differences in adult mortality.

The stronger effect of youth paths than the parental home could indicate that factors related to these paths are important in respect of development of one's own social standing and mortality risk (see West 1997). The likely factors include the adoption of health-related behaviors in youth. Alcohol use is a significant factor in mortality at this age, while the effects of smoking, poor nutrition and sedentary life styles might be detectable gradually. In addition, part of the effect of youth paths may be attributable to the effect of disadvantages in young adulthood on self-esteem and psychological well-being.

Youth paths largely explained the mortality differences between social classes in middle adulthood, and played a salient role in the adoption of the adult class. At a later age, the accumulative effect of the poorer social circumstances, to which youth paths may lead, may increase when the persons are exposed to poorer material circumstances, behaviors and psychosocial factors for a longer time. The dynamic process through which youth paths exerted their effect on both mortality and adult social class proved to be important in middle adulthood. Therefore, the results reported in this thesis underscore the pathway influence of the lifecourse in generating social class differences in middle adulthood.

10.2 Future research and possible policy implications

The knowledge we have on the processes of lifecourse effects on health and mortality has shown the importance of social factors in different phases of life in the development of subsequent mortality risk. The process leading to adult social-class membership seems to be important. In particular, the critical transitions in youth have a salient role. Sociodemographic variables are sufficient to identify the most sensitive age phases and vulnerable population groups. Co-operation with other scientific fields, particularly with biology, medicine, and psychology, is needed when the focus is on revealing the mechanisms that mediate the effects of social exposure at different phases of life on subsequent mortality risk.

A strong effect of youth paths on adult social class differences in mortality was found. The continuation of the follow-up of this cohort to older ages will reveal whether the effects found here will remain, diminish, or even increase. Furthermore, this study consisted of a narrow birth cohort born in 1956-60. Some of the findings of a study including only those born within five years may be related to the specific lifecourse experiences of this cohort. Therefore there is a need for a study including several cohorts. The resulting information would lead to increased knowledge about the emergence and future trends of social class differences in mortality, and of the lifecourse model that is likely to account for these differences.

More knowledge is needed on the effects of the pathways distinguished here over a longer period of life. Educational, family formation, and employment paths were called youth paths here, because the information concerning them was from youth and young adulthood. In this phase of life they were important factors in terms of the adult social class, although transitions along the 'family formation' and employment paths may continue to an older age. These later changes, new unemployment experiences and retirement, divorces and remarriages, may all have different effects on health risks depending on the experiences on these paths in young adulthood. Therefore studies

following these paths from youth to later adulthood would be of interest in terms of their effects on health, mortality, and differential mortality.

This study identified elevated mortality among those who had only basic education and those who had been unemployed in young adulthood. On the basis of these results, it seems that youth and young adulthood are 'socially sensitive periods' in which time disadvantages may have long-term effects on mortality risk. Data with additional variables is needed to specify the findings of the effects of the youth paths on mortality and social class differences in mortality. We need information on the actual factors that affect people's transitions to different educational, marital and employment paths. Furthermore, we need to identify the specific factors on these paths that affect premature mortality. The results of such studies may help us to identify a modifiable risk factor that would enable us to better design and execute health-promoting interventions. For example, these studies may help us to develop programmes that help people not to drop out of education, and that may also have positive health consequences. Such programmes may also decrease the effects of youth unemployment.

Furthermore, the analyses reported in this thesis already show that men from single-parent families had elevated mortality in adulthood. With data on more specific information on childhood circumstances it may be possible to identify the group of sons of single-parent families that have the highest risk of premature mortality, and to find meaningful means to give support to such groups and possibly lower their risk of early death. Younger cohorts could also be used to observe whether there are differences between men and women in the adoption of drinking habits and the effect of social background on mortality from alcohol-related causes. A large proportion of deaths in middle adulthood are related to alcohol, and alcohol-related causes contributed to a large extent to social class differences in mortality. It is imperative to intervene as early as possible in alcohol use in order to reduce such social class differences in mortality.

10.3 Conclusions

The main interest in this thesis was to study the effect of the living conditions in the parental home, i.e. parental social class, family type, number of siblings, language and region of residence, and youth paths, i.e. educational, family formation and employment paths, on adult social class differences in mortality. The analyses showed that, in middle adulthood, factors related to the parental home had a minor effect on social class differences in mortality, with the exception of cardiovascular diseases in both sexes and alcohol-related causes in men.

However, youth paths had a substantial effect over and above the preceding effect of living conditions in the parental home. For most causes of death, about 60-80% of social class differences in mortality could be accounted for by a disadvantageous educational path. Moreover, both family formation and employment path had independent effects, reducing the social class differences by about 10% and 20-60%, respectively.

The effects of the living conditions in the parental home and of youth paths on social class differences in mortality are attributable to different lifecourse models. The results reported in this thesis indicate that the parental home has a latent effect on mortality differences, but that the contribution of this effect is minor. Indications of the accumulative effect of disadvantageous social class circumstances were found for cardiovascular diseases in women and men, and also for alcohol-related causes in men. However, the most influential lifecourse mechanism in these data was the pathway model. The social pathway from the parental home via education, family formation and employment path to adult social class was significant. Youth paths mediated the effect of the parental home on mortality, but they had an extensive independent effect on the emergence of social class differences in mortality from diseases and external causes.

Youth and young adulthood are times of significant social transitions from education to work life, from one's childhood home to one's own home, from the position of a child to the position of a parent and partner. On the basis of the results reported in this thesis, it seems that this is also a 'sensitive period' with regard to mortality risk. Women and men in middle adulthood who had disadvantaged youth paths had much greater mortality than those who succeeded better. It is not clear whether these effects will decrease or increase when the cohort ages. A decreasing effect is possible because when more time since youth has elapsed, the disadvantages experienced in youth may become less important, nevertheless the disadvantages in youth paths may lead to later hazards. The effect of the parental home behind social class differences in mortality may also increase when the cohort grows older and ischeamic heart and cerebrovascular diseases become more prevalent.

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Mortality differences by parental social class from childhood to adulthood

Tiina H Pensola, Tapani Valkonen

Abstract

Study objective—To examine mortality differences by parental social class and cause of death from age 5 to age 34.

Design—Register-based follow up study based on census records for 1985 and 1990 linked with death records for the period 1987–95.

Setting and subjects—The study covers all males and females in non-manual and manual classes in Finland aged 5–34 years in 1987–95 (8135 deaths). Parental social class is defined on the basis of the occupation of the head of household at the time the child was 0–14 years.

Main outcome measures—All cause mortality, mortality from diseases, mortality from accidents and violence, and alcohol related mortality during the period 1987–95.

Main results—At ages 5–14 there is no systematic gradient in mortality by parental social class. Both absolute and relative differences increase with age. The relative rate of male all cause mortality among manual class descendants at ages 25–29 compared with that of upper non-manual class descendants is 1.60 (95% CI 1.37, 1.86). At ages 30–34 the relative rate among males is 1.95 (95 % CI 1.58, 2.42) and among females 1.47 (95% CI 1.03, 2.10). Among males alcohol related causes of death account for 70% of the excess mortality of sons of manual class parents compared with sons of upper non-manual class parents at ages 25–34. At ages 25–34, both among females and males, the contribution of diseases to the mortality difference increases.

Conclusions—Parental social class has an impact on mortality after childhood mainly through health related behaviours and lifestyles up to age 34.

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Results from studies on socioeconomic mortality differences among adults have consistently shown a clear rising gradient of mortality from high to low socioeconomic positions.[1–5] On the other hand, the results from the few studies[6–11] concerning social variation in mortality from childhood to adulthood are inconsistent. Rimpelä[6] found no differences in total mortality among females aged 5–24 in Finland and only minor differences among males under age 20. Östberg[10] and Östberg and Vågerö[11] concluded that there were wide relative differences in total mortality between social classes at ages 1–19 in Sweden, whereas West[7 8] concluded that the differences were very narrow in the age group 10–14 in England and Wales. Blane and colleagues[9] found no class differences in mortality from diseases in this age group in England and Wales.

Some of the discrepancies discovered in earlier studies in the association between childhood class and mortality may be attributable to the limited number of deaths in some of these studies, leading to very wide confidence limits for death rates.[9 12 13] Earlier research has also been marred by the unsystematic measurement of social class. Many young persons cannot yet be assigned to a social class on the basis of their occupation because they are either studying or unemployed, and for many of those who have an occupation, occupational status is transient.[14] Because of these difficulties children under 16 have been classified on the basis of parental class, while those over 16 have usually been assigned to their own class. Those who as yet have no occupation are either left in the category of unknown[8] or classified on the basis of parental class.[6] However, it is important to have a uniform definition of social class within and between age groups to avoid apparent changes in class patterning of mortality attributable to different classifications. The system of personal identification numbers that is in use in Finland means it is possible to follow the mortality of the whole population by social class. Although death rates are very low at young ages, the large number of persons and the long follow up period allow us to study mortality even by certain main categories of causes of death. Our data also allow for uniform measurement of socioeconomic position for all young persons up to age 34. The following questions are investigated in this paper:

(1) To what extent do there occur systematic social class differences in all cause mortality by parental class in different age groups at ages from 5 to 34?
(2) What is the class patterning of mortality by age and cause of death?
(3) What causes of death account for the class difference in mortality between non-manual and manual classes in different age groups?

Methods

The data used in this study were extracted from two data files compiled by Statistics Finland. The first one is based on the 1985 census records for persons born in 1956–85. The death records for 1986–90 and socioeconomic information from the censuses of 1970, 1975 and 1980 were linked to the records by means of personal identification numbers. The second data file consists of the 1990 census records,

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Table 1   Distribution of person years (%) covered by the study by age and parental social class

	Parental social class							
Age	Upper non-manual	Lower non-manual	Manual	Farmer	Entrepreneur	Other	All	Person years (1000s)
5–9	20	24	38	6	7	5	100	2874
10–14	20	26	38	7	8	2	100	2904
15–19	19	25	40	7	7	2	100	2829
20–24	15	22	45	9	6	2	100	3025
25–29	12	18	45	15	7	3	100	3329
30–34	10	16	44	18	7	5	100	1888

which were linked to the death records for 1991–95 and the census records for 1970, 1975, 1980 and 1985 for persons born in 1956–90. The analyses cover person years and deaths in ages from 5 to 34 during 1987–95 in the two data files.

In Finnish censuses economically non-active children living at home are assigned to socioeconomic classes on the basis of the occupation of the head of household. In this study parental social class refers to the class of the head of household in the census where the person was 10–14 years of age. In the birth cohorts younger than 10 years of age in the 1985 or 1990 census, parental social class is based on the information in that census. For approximately 7% of the study population parental class was unknown in the most relevant census; for these people the information was obtained, if possible, from the preceding census.

The classification of parental class used in this study is based on Statistics Finland's classification of socioeconomic classes.[3][15] Table 1 shows the distribution of the person years covered by the dataset by parental social class. There are some differences between the age groups that are attributable to changes in the occupational structure of the population. The following categories are used in the study:
(1) Upper non-manual class (for example, managers and higher administrative or clerical workers).
(2) Lower non-manual class (for example, lower administrative or clerical workers).
(3) Manual class.

Farmers, entrepreneurs and others have been excluded from the analyses because they form small but heterogeneous groups that are

impossible unequivocally to slot in the socioeconomic hierarchy.

Causes of death were coded according to the Finnish Classification of Diseases 1987,[16] which is based on the Ninth Revision of the International Classification of Diseases but which uses five digit instead of four digit codes. For this study deaths were first classified by cause into two groups: diseases (1–799) and accidental and violent causes (E800–999), and then within these groups into alcohol related and non-alcohol related deaths. The definition of alcohol related death is the same as that used by Mäkelä et al.[17]

Alcohol related deaths are those in which either the underlying cause of death explicitly mentions alcohol or in which the underlying cause does not mention alcohol, but at least one of three possible contributory causes is either alcoholic intoxication or one of the causes of death explicitly mentioning alcohol, such as alcoholic psychoses, alcoholic diseases of the liver, alcoholic diseases of the pancreas, accidental poisoning by alcohol and accidental poisoning by medicinal agents in combination with alcohol (see table 2).

Person years and deaths by cause of death were tabulated by parental social class and period (1987–90 and 1991–95). Statistical analyses were based on the Poisson regression model, with the cell in cross tabulation taken as the unit of analysis. The GLIM statistical package[18] was used in fitting the models. The results are presented as relative mortality rates (and their 95% confidence intervals). The reference group is children in the upper non-manual class. Tests for gradients of period adjusted rates were obtained by the Poisson regression model in GLIM with period and social class (coded from one to three and used as continuous variable) as covariates.

As the summary measure that adjusts for the differences among age groups in the occupational structure, we have used the relative index of inequality (RII).[19] Poisson regression was used to estimate the slopes.

Results

Table 2 gives the numbers of deaths by age, sex and cause of death. The number of alcohol related deaths from diseases is low, and therefore in the remaining analyses all alcohol related deaths are analysed as one group.

Figure 1 presents the relative death rates in all cause mortality and for three groups of causes of death by parental social class and age for females and males. In all cause mortality

Table 2   Number of deaths in five year age groups from diseases, and accidental and violent deaths by relation to alcohol. Females and males in non-manual and manual classes aged 5–34 in 1987–1995

	Females				Males				
	Cause of death								
	Diseases		Accidents and violence		Diseases		Accidents and violence		
	Alcohol related underlying or contributing cause of death*								
	no	yes	no	yes	no	yes	no	yes	All
Age group									
5–9	115	0	52	0	112	0	125	0	404
10–14	101	0	67	3	112	1	135	8	427
15–19	116	2	194	55	171	2	561	332	1433
20–24	166	6	205	61	246	18	738	624	2064
25–29	238	14	200	75	313	51	704	714	2309
30–34	197	13	98	43	283	90	355	419	1498
All	933	35	816	237	1237	162	2618	2097	8135

*The following are defined as alcoholic causes: 265.0A, 291, 303, 305, 375.5, 425.5, 535.3, 571.0–571.3, 577.0D-F, 577.1C-D, E849 and E851.
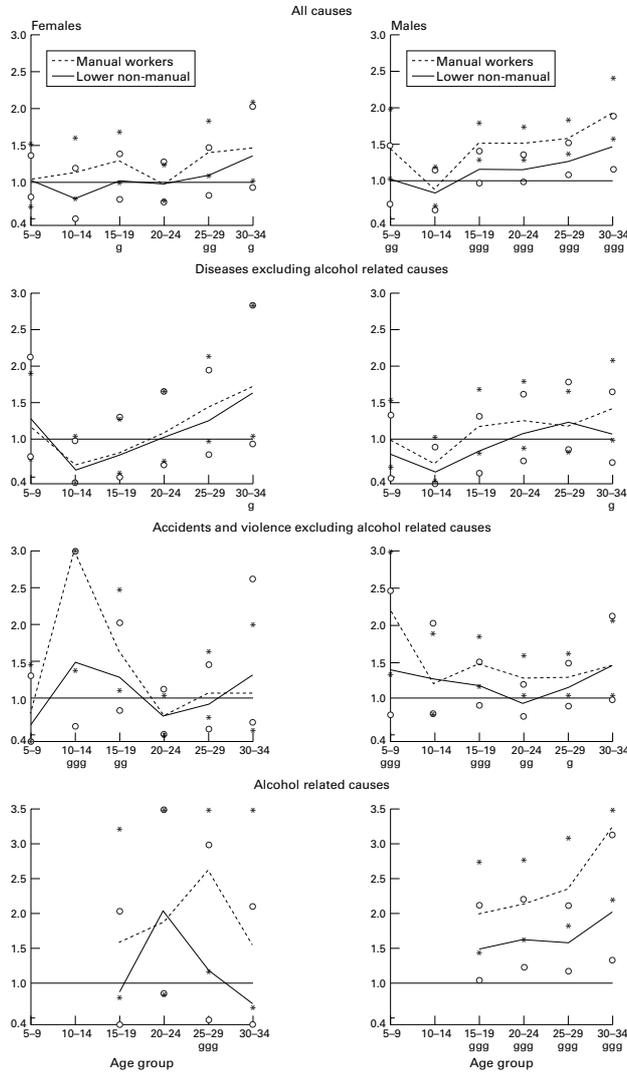
Figure 1   *Relative death rates in all cause mortality, and in three causes of death by age (five year age groups) and parental social class 1987–95, females and males (children of upper non-manual class=1). o=95% CI for lower non-manual employees. *=95% CI for manual workers. Statistically significant gradients: g=p<0.05, gg=p<0.01, ggg=p<0.001.*

KEY POINTS
- At ages 5–14 among females and ages at ages 10–14 among males there are no systematic gradients in all cause mortality by parental social class.
- There is a statistically significant social class gradient among females at ages 15–19 and 25–34, and among males at ages 5–9 and 15–34.
- In different age and gender groups the differences in all cause mortality are attributable to different causes of death.
- Among males at ages 15–34, alcohol related causes account for a crucial part of the mortality differences by parental class.

there is a statistically significant mortality gradient among women at ages 15–19 and 25–34 and among males in all age groups except 10–14.

Disease mortality among children of the upper non-manual class is higher than among children of lower non-manual and manual classes at ages 10–19 among females, and at ages 5–14 among males, but the differences are statistically insignificant. After age 20 descendants of manual and lower non-manual classes show a higher mortality than the reference group, but the mortality gradient is statistically significant only at ages 30–34.

In mortality from accidental and violent causes excluding alcohol related deaths, daughters of manual class parents show a statistically significant excess mortality only at ages 10–19. Sons of manual class parents have statistically significant excess mortality compared with sons of upper non-manual class in all age groups except 10–14.

Among males the clearest differences in mortality by parental social class are found in alcohol related deaths. These statistically significant differences increase with age.

Table 3 shows the absolute mortality differences and the contribution of selected causes of death to the total absolute difference between manual and upper non-manual, and between lower and upper non-manual classes. Among females the differences between upper non-manual and other classes are small. For them the differences in disease mortality have the greatest impact on the total difference at ages 30–34. Among males the contribution of accidents and violence without alcohol related causes to the mortality difference between manual and upper non-manual class declines with increasing age. The impact of diseases is quite minor, while alcohol related causes are predominant in all age groups in both comparisons. At ages 25–34 approximately 70% of the difference between the manual and upper non-manual class and 60% of the difference between the upper and lower non-manual class can be attributed to alcohol related causes.

According to table 1 the distribution of parental class is different in young and old age groups. Table 4 shows an estimate of the extent to which this difference affects the results. The table first presents the strength of the association between parental class and mortality for five year age groups by using the adjusted RII, which eliminates the effect of the differences between the age groups in the distribution of parental class. The "non-adjusted" RII* is calculated on the basis of the assumption that the parental class distribution is the same in all age groups as in the age group 20–24. The comparison shows only minor differences between the two columns (RII and RII*), indicating that the differences in the distribution of parental class have no more than a minimal effect on the results.

Table 3   Absolute difference in all cause mortality between descendants of (1) manual and upper non-manual classes, and (2) lower and upper non-manual classes, and per cent of *the difference accounted for by three groups of causes of death by age, females and males

	Absolute difference between descendants of									
	(1) manual and upper non-manual classes					(2) lower and upper non-manual classes				
		% of difference accounted for by					% of difference accounted for by			
Age	Total diff†	Diseases‡	Accidents and violence‡	Alcohol related	Total	Total diff†	Diseases‡	Accidents and violence‡	Alcohol related	Total
Females										
5–9	0.2	—	—	—	—	0.1	—	—	—	—
10–14	1.6	—	—	—	—	−3.3	—	—	—	—
15–19	8.7	−23	95	28	100	0.7	—	—	—	—
20–24	−1.3	—	—	—	—	−1.4	—	—	—	—
25–29	13.6	47	11	42	100	2.8	—	—	—	—
30–34	18.5	74	6	20	100	14.3	83	31	−13	100
Males										
5–9	7.4	−2	102	0	100	0.3	—	—	—	—
10–14	−2.4	—	—	—	—	−3.7	—	—	—	—
15–19	36.7	6	48	46	100	12.5	−17	50	67	100
20–24	51.2	9	28	63	100	16.3	8	−21	113	100
25–29	58.4	7	22	72	100	28.5	17	22	61	100
30–34	95.0	15	18	67	100	48.1	4	34	62	100

*Per cent of the difference accounted for by the cause of death category:$((mr_i^c − mr_i^r)/(MR^c − MR^r))*100$, where mr_i = mortality rate in cause i, MR = mortality rate in all causes, c = comparison group, r = upper non-manual class. †Deaths per 100 000 person years. ‡Excluding alcohol related deaths. —Total difference is too small to be divided meaningfully into different causes of death.

Table 4   Relative index of inequality (RII) and "non-adjusted" relative index of inequality (RII*) for all cause mortality among descendants of upper and lower non-manual classes and manual class, females and males

	Females		Males	
Age	RII	RII*	RII	RII*
5–9	1.0184	1.0181	1.9544	1.9666
10–14	1.3562	1.3970	0.8854	0.8971
15–19	1.6152	1.6253	1.9666	1.9672
20–24	0.9620	0.9620	1.9480	1.9480
25–29	1.7657	1.7618	1.9192	1.9270
30–34	1.5274	1.5488	2.4006	2.4344

The RII was calculated on the assumption that the distribution of parental class in all age groups was the same as that in the age group 20–24 years.

Discussion

DISEASE MORTALITY

Our findings on the non-existence of differences in disease mortality for females under age 20 are consistent with the results for Britain[9] and for Sweden in 1961–79,[11] but in 1981–86 Swedish females aged 1–19 with a manual class background showed statistically significant excess mortality.[10] Our results concerning males under age 20 differ from the results for both Sweden and Britain. In Sweden in 1961–79[11] sons of manual workers and in 1981–86,[10] sons of unskilled workers showed higher mortality than sons of non-manual workers. In Britain there was a gradient in mortality at ages 0–9 but not at ages 10–14.[9]

In older age groups our results are consistent with the results for Sweden[11] and the results for Britain for males,[9] but in Britain females aged 15–29 showed higher disease mortality in the non-manual than the manual class. The differences observed between our results and those from Britain and Sweden may reflect real differences between the countries, but it is also possible that the differences between Finland and Britain are attributable to chance variation. The number of deaths covered by the British study was small (304 deaths at ages 0–29) and none of the gradients reported were statistically significant.

ACCIDENTS AND VIOLENCE

In accidental and violent deaths daughters of manual class parents show excess mortality at ages 10–19 and sons at ages 5–9 and 15–34, when alcohol related causes are excluded, compared with children from an upper non-manual class background. In the Swedish studies mentioned above[10][11] the results were essentially the same. In England and Wales in 1985–92, children of manual workers aged 0–14 showed excess mortality in every accidental and violent cause category.[20]

ALCOHOL RELATED DEATHS

Earlier Finnish studies[17][21] have highlighted the role of alcohol in the mortality difference between both social classes and genders in adulthood. In this study the exclusion of alcohol related causes at ages 15–34 would reduce the mortality differences by parental social class; in some cases the differences would disappear almost completely.

In this study the information on alcohol related deaths comes mainly from contributory causes of death; in the age groups concerned alcohol is mentioned as an underlying cause of death in only 22% of females and 14% of males with an alcohol related cause of death. It is not clear how far these results can be generalised to other countries because earlier studies have not made use of data on contributory causes of death. Some insight is gained by comparing mortality attributable to accidental and violent deaths in Finland in 1989–91 to mortality in nine West European countries where data were available for about the same period.[22–24] For males aged 15–24, Switzerland was the only country where mortality attributable to accidents and violent causes was as high as in Finland; in all other countries, and in all countries at ages 25–34, it was much lower (for example, in France 25%, in Sweden 44% and in the United Kingdom 60% lower than in Finland; for females the corresponding figures were 1%, 12% and 47%). It seems probable that both the

high male mortality from accidents and violence and the observed major contribution of alcohol related deaths to the class difference in mortality among young men is attributable to the high level of alcohol related accidents in Finland.

The predominant role of alcohol related mortality underscores the importance of health behaviours and life styles in explaining mortality differences in young adulthood in Finland. To reduce class differences in mortality and in general to reduce mortality among young persons, it is imperative that action is taken to encourage teenagers in different groups to change their drinking habits.

ALL CAUSE MORTALITY

In the youngest age groups studied here mortality is low and the absolute differences are small. Earlier studies in Sweden[10] and Britain[25] have reported excess mortality in children from a manual class background compared with children of non-manual class parents.

An issue of concern, as Judge and Benzeval[26] have shown for Britain, is the group of "others". In our data "others" is so small a group that their inclusion in the manual class would have only a minor impact on the results. Thus, at least in Finland, small mortality differences at ages 5–14 are not a result of incomplete data. They may, however, be attributable to the low mortality in this age group. During the first year of life, when the probability of death among males is eight times and among females six times greater than the probability of death at ages 5–14,[3] mortality rates between manual and non-manual classes do differ.[6 27–30]

West[7] has argued that if the size of mortality differences changed with age among young persons, the conclusions we would need to draw about the causes of socioeconomic mortality differences would be different than if the gradient were found to be the same in all age groups. The latter possibility suggests causal[31] and the former both causative and selective explanations.[8] Our results lend support to West's[7 8] conclusion that youth is characterised by relative equality of health, but not to his suggestion that mortality differences diminish in adolescence after greater inequalities in mortality in childhood. Nevertheless a class gradient in all cause mortality is found after age group 20–24 among females and among males 15 on, as West[7 8] has suggested.

In this study the class differences at ages 15–34 were smaller than in an earlier Finnish study[3] for the period 1986–90, in which social class was based principally on own occupation. The greater mortality differences based on own occupation might partly be attributable to social mobility, involving direct and indirect health selection.[7]

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RESEARCH REPORT

Cumulative social class and mortality from various causes of adult men

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Study objective: It is possible that circumstances over the lifecourse contribute to social inequalities in mortality in adulthood. The aim of this study is to assess the cumulative effect of social class at childhood and adulthood on mortality from various causes of death in young adult men.
Design: The data consist of census records for all Finnish men born in 1956–60 (112 735 persons and 895 001 person years), and death records (1834 deaths) by cause of death for 1991–98.
Main results: Mortality from each cause of death increased from the stable non-manual group to mobile groups, and further to the stable manual group. However, mortality in the downwardly mobile group was 150% higher than in the upwardly mobile group. Furthermore, analyses show that mortality was mainly related to current adult social class, though, within each adult social class men with a manual parental background showed slightly increased mortality from cardiovascular disease and from alcohol related causes.
Conclusions: In these data the effects of adult social class were stronger than childhood class for all causes of death. It is more useful to differentiate between childhood and adulthood effects than to use a combined measure of social class to assess the contribution of social class at different stages of life on mortality.

The association between adult social class and mortality is well established.[1–3] It is quite obvious that if at any given moment a person's risk to die is associated with factors related to low (high) social class, longer exposure to these factors will increase (decrease) the risk of death. Accordingly, this kind of lifetime accumulation of social position may be an important factor behind social class mortality[4] as well as health differences in adulthood.[5]

A typical approach to the accumulation of social class or "lifetime social class" is simply to sum up social class scores at different stages of the lifecourse. In principle, this combined measure expresses "the cumulative exposure to poor socioeconomic conditions"[6] more accurately than any single measure from some part of the life cycle, and it is also likely to produce larger social class differences in health. However, as analyses based on combined measures do not take into account upward and downward social mobility, it is difficult to assess the relative contribution of social class at different stages of life. Nevertheless, in the study on men employed at the beginning of the follow up, the cumulative effect of social class on mortality was observed when the direction of mobility was taken into account.[7]

Research on the effects of cumulative social class on health needs first of all to have a reliable measure of class at different stages of life. For this reason there has been a tendency to focus on the economically active (employed) population, a subgroup of the total population for whom social class is readily available. However, if unemployed and economically inactive persons are excluded the social class differences in mortality tend to be smaller than when information for the whole population is available.[8,9] This underestimation of social class differences in mortality is explained by the poor health of the economically inactive population, and the greater probability that they come from lower occupational categories. It is probable that the exclusion of economically inactive men bias the results of studies with a lifecourse perspective.

Most research on the effects of accumulation of social class on health and mortality have analysed older adult populations. In studies concerning young adults, the accumulation perspective has been earlier used with self rated health as outcome variable[5] but not with mortality. However, research results on self rated health cannot be assumed to apply to mortality, even though the first does predict the second.[10]

We have access to census based data on Finnish men born in 1956–60. Information on their social mobility from childhood social class to adult class covers the period from 1970 to 1990. This is somewhat later than in earlier studies[4,7] and therefore reflects different economic conditions and occupational opportunities. In addition, the adverse early socioeconomic conditions faced by this younger cohort have been very different to those observed in older cohorts.[11,12] Given these disparities in mortality and mobility chances between countries and birth cohorts, it is interesting to see whether social class has a cumulative effect in the context of our study as well. Furthermore, we will be able to see whether inter-generational mobility has any effect on social class differences in mortality, and whether this effect constrains[7,13] or increases[14,15] the social gradient in mortality.

The specific objective of the study is to assess the cumulative effect of social class at childhood and adulthood on mortality from various causes of death in the period 1991–98. We are mainly interested in the effects of the direction of inter-generational mobility on mortality.

METHODS
Data
Compiled by Statistics Finland, the data are based on 1990 census records for men who were born in 1956–60 and who were living in Finland at the time of the 1970 and 1990 censuses. Death records for 1991–98 and information concerning social class from the 1970, 1975, 1980, and 1985 censuses were linked to the 1990 census records by means of personal identification numbers. The share of deaths that cannot be linked to the census records is less than 1%.

The cohort born in 1956–60 was chosen for these analyses, because at the time of the 1970 census—when the cohort members were aged 10–14—childhood social class was

Table 1 Number of deaths and age standardised mortality rates (with 95% confidence intervals) per 100,000 person years for various causes of death in 1991–98 according to adult and childhood social class, men aged 30–34 in 1990

Cause of death	Number of deaths	Adult social class				
		Non-manual		Manual		
		Childhood social class				
		Non-manual	Manual	Non-manual	Manual	All
All causes	1834	96 (83 to 110)	115 (101 to 131)	288 (255 to 324)	286 (269 to 303)	205
Diseases	772	50 (41 to 61)	55 (46 to 67)	123 (102 to 148)	111 (101 to 122)	86
Cardiovascular	243	12 (7 to 17)	15 (10 to 21)	27 (17 to 39)	41 (35 to 48)	27
Neoplasm	158	18 (13 to 25)	14 (9 to 21)	22 (13 to 33)	18 (14 to 23)	18
Other	371	20 (14 to 27)	26 (20 to 35)	75 (59 to 94)	52 (45 to 59)	41
External causes	1062	46 (37 to 56)	60 (49 to 71)	165 (140 to 192)	175 (162 to 188)	119
Suicide	492	26 (20 to 34)	29 (22 to 38)	77 (61 to 97)	77 (69 to 87)	55
Other	570	19 (14 to 27)	31 (23 to 39)	87 (70 to 108)	97 (88 to 108)	64
Alcohol related*	874	31 (24 to 39)	47 (38 to 57)	134 (112 to 159)	149 (137 to 161)	98
as underlying cause	354	15 (10 to 21)	16 (11 to 23)	65 (50 to 83)	58 (51 to 66)	40
as contributory cause	520	16 (11 to 23)	31 (23 to 39)	69 (53 to 88)	91 (82 to 101)	58

*Alcohol related causes overlap with diseases and external causes.

available on the basis of the occupation of the head of the household (usually the father). Accurate information on parents' occupation was not available for persons aged 15–19 because almost half of them were not living with their parents or they were working.

We excluded those born in 1956–60 whom we were not able to assign to an adult social class (4%). In addition, we excluded those who were farmers or entrepreneurs by adult or childhood social class. This exclusion was based on the recognition that it would be extremely difficult to establish how entrepreneurs and farmers compare hierarchically with non-manual and manual classes in the social class structure and therefore to say whether shifts between non-manual/manual and farmer/entrepreneur positions are ascending or descending. After these exclusions the actual study cohort consisted of 62% of all those born in 1956–60 and numerated in the 1990 census.

For the actual study cohort adult non-manual and manual social class was determined on the basis of own occupation in the 1990 census for those who were employed (91% of the cohort) or unemployed but had information on their last held occupation (6%). If a person had no occupation (2%) or occupational information was lacking (1%), information on previous occupation was searched from the 1985 census. Childhood social class was determined by occupational information on the head of household in the same manner. The social class coding at childhood and adulthood is based on Statistics Finland's classification.[16]

Mortality follow up covers the period from 1991 to 1998. Altogether 1834 men died at ages 30–42. Causes of death for 1991–95 are coded according to the 1987 Finnish Classification of Diseases, which is based on the Ninth Revision of the International Classification of Diseases (ICD9).[17] The codes for 1996–98 are based on the Tenth Revision of the International Classification of Diseases (ICD10).[18] This change of classification has no effect on the results. The following broad groups of death are used (number of deaths in table 1):

- all causes
- all diseases (that is, all causes excluding external causes)
- cardiovascular diseases (ICD9: 390–459, ICD10:i00-i99)
- neoplasm (ICD9:140–239, ICD10:c00-d48)
- external causes (that is, all causes excluding diseases)
- suicides (ICD9:E95,ICD10:x60-x84,Y870) (the single most common cause of death)

In addition to these causes of death we have distinguished alcohol related mortality as a separate cause of death because of its importance in young adults (see table 1). For the same reason we have used contributory causes of death to distinguish all deaths related to alcohol. Of the 874 deaths attributable to alcohol, alcohol is the underlying cause for 185 deaths due to disease and for 159 deaths caused by alcohol poisonings. It is a contributory cause in 71 deaths in the disease category and in 459 deaths in the external cause category. Causes included in this category are those in which alcoholic disease, such as alcoholic psychoses (ICD9:291, ICD10:F10.5), alcoholic diseases of the liver (ICD9:571.0–571.3, ICD10:K70.0-K70.9), and alcoholic diseases of pancreas (ICD9:577.0D-F,577.1C-D, ICD10:K86.00-K86.08) or poisoning (ICD9:E851, ICD10:X45) are mentioned as underlying or contributory causes of death. The classification of alcoholic causes is discussed in closer detail elsewhere.[19]

Study design

Person years and deaths by cause of death were tabulated by age, period, and childhood and adult social class. From these tables we have indirectly calculated age standardised mortality rates (with 95% confidence intervals),[20] with mortality in the whole cohort as the standard. Approximate p values for the difference between two mortality rates are based on the Gaussian log likelihood approximation.[21]

We also calculated relative mortality rates (and their 95% confidence intervals) by means of Poisson regression analyses, with the cell in cross tabulation taken as the unit of analysis. The relation between mortality and the childhood and adult social classes is based on the following model:

$$\text{Log}(E(d_i)) = \log(V_i) + a + b_1 x_{i1} + b_2 x_{i2}$$

Social class

	Childhood		Adulthood
Non-manual	38 (303)	26 (206) →	50 (402)
		25 (196) →	
		12 (97) →	
Manual	75 (592)	50 (396) →	62 (493)
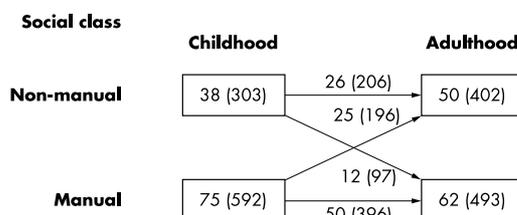
Figure 1 Number of person and person years (in thousands) in 1991–98 according to childhood and adulthood social class, men aged 30–34 in 1990.
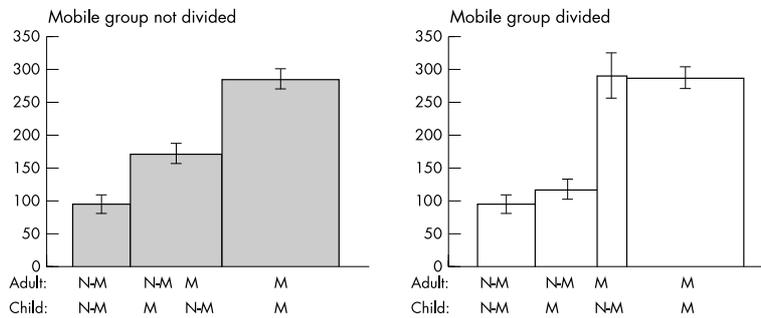
Figure 2 Age standardised mortality rates (with 95% confidence intervals) per 100 000 person years for all cause mortality by adult and childhood social class in period 1991–98, men aged 30–34 in 1990. N-M, non-manual; M, manual. The width of the bar is directly proportional to the size of the group it refers to.
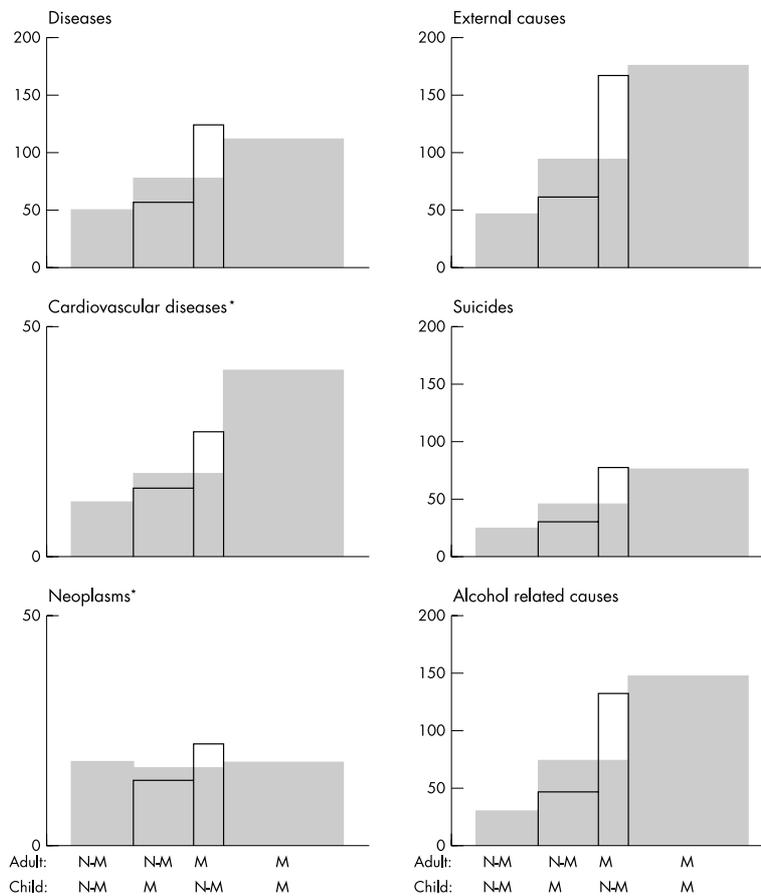
Figure 3 Age standardised mortality rates per 100 000 person years for broad causes of death by adult and childhood social class in 1991–98, men aged 30–34 in 1990. Grey bars show mortality rates when mobile groups are not divided, bars with solid line show mortality rates separately for upwardly and downwardly mobile men (see fig 2). The width of the bar is directly proportional to the size of the group it refers to. The confidence intervals are given in table 1. *Notice the different scaling.

Where $E(d_i)$ is the expected number of deaths and V_i is the number of person years lived in the ith cell , x_1 is age, x_2 is the variable describing adult and childhood social class, and a, b_1, b_2 are the parameters to be estimated. The Glim statistical package was used in fitting the models.[22]

RESULTS

Mortality according to adult and childhood social class

In our cohort, 23% of all men had a non-manual background and belonged themselves to the non-manual class. We call this group stable non-manual. Correspondingly, 44% of all men belonged to the manual class in childhood and adulthood—

that is, to the stable manual group. Movement between social classes from childhood to adulthood was recorded for one in three men: 22% of all men moved upwards and 11% downwards. The proportion of men in the non-manual class is considerably higher for adult social class (45%) than for childhood social class (33%). In the non-manual class, 49% of the men had a manual background, while 20% of manual men had a non-manual background (fig 1).

In figure 2 all cause mortality rates are first presented for the stable non-manual class, for mobile groups combined, and for the stable manual class. This highlights the accumulative effects of social class on mortality. The first bar illustrates the

Table 2 Relative age standardised mortality rates for selected causes of death according to adult and childhood social class for employed men and for the whole cohort in 1991–98

	Social class			
	Own class			
	Non-manual		Manual	
	Childhood class			
	Non-manual	Manual	Non-manual	Manual
All causes				
Employed men	1.00	1.12 (0.91 to 1.37)	2.47 (2.01 to 3.04)	2.27 (1.93 to 2.68)
Whole cohort	1.00	1.20 (1.00 to 1.45)	3.01 (2.51 to 3.60)	2.98 (2.57 to 3.47)
Cardiovascular disease				
Employed men	1.00	1.40 (0.80 to 2.45)	1.45 (0.73 to 2.88)	3.04 (1.92 to 4.81)
Whole cohort	1.00	1.27 (0.74 to 2.16)	2.29 (1.32 to 3.97)	3.55 (2.33 to 5.42)
Alcohol related causes				
Employed men	1.00	1.50 (1.06 to 2.14)	3.63 (2.57 to 5.14)	3.59 (2.68 to 4.80)
Whole cohort	1.00	1.53 (1.11 to 2.10)	4.37 (3.24 to 5.88)	4.86 (3.76 to 6.28)
Person years (number of persons in 1990) in thousands				
Employed men	198 (25)	188 (24)	84 (11)	341 (43)
Whole cohort	206 (26)	196 (25)	97 (12)	396 (50)

mortality of men who have not experienced low social class, the second bar represents the mortality of men who have experienced low social class to a lesser extent than men whose mortality is expressed in the third bar. The all cause mortality pattern alludes to a cumulative effect of social classes: there is a statistically significant trend for these three groups. However, when the socially mobile group is divided into the upwardly and downwardly mobile groups (on the right hand side in fig 2), men who have descended from the non-manual to manual class show significantly higher mortality than men moving in the opposite direction. Figure 2 shows that total mortality is clearly related to adult class. Within the adult non-manual class there is only a minor difference (20%) and within the adult manual class there is very little difference in all cause mortality between the stable and mobile groups (table 1).

For all other specific causes of death except neoplasms, mortality increases with number of times in the manual class (shaded bars in fig 3). For neoplasms mortality differences according to childhood and adult social class are very small. For other causes of death, as above in the case of all cause mortality, the accumulative effect of social class either disappears or is attenuated when the mobility group is divided. For cardiovascular diseases and alcohol related causes the accumulative effect of social class was decreased, but we still observe a small mortality difference according to childhood class within both adult social classes. Men with a manual background show excess mortality in comparison with men with a non-manual background for cardiovascular diseases in the adult manual class (p=0.017 for the difference between the rates), and for alcohol related causes in the adult non-manual class (p=0.009) (table 1).

We carried out further analyses in which we measured social class at three different time points: in childhood (in 1970, as above), at age 20–24 (in 1980), and at age 30–34 (in 1990). The number of times in the manual class (0, 1, 2, or 3) showed a clearly increasing trend for age standardised mortality rates (all cause mortality rates were: 81, 117, 175, and 235, respectively). However, when the mobility groups were separated as in the previous analyses, mortality was observed to be much more strongly associated with adult social class than childhood class. This is consistent in the results presented in the figure 2. For instance, in the non-manual social class at age 30–34, the age standardised mortality rate was only somewhat higher for those who had

been twice or once in the manual class as compared with those who had never been in the manual class (mortality rates were 102, 119 (once),102 (twice) 81 (never)).

Bias related to missing occupational information among economically inactive people

The exclusion of economically inactive persons may underestimate social class inequalities in mortality. To estimate the effects of this bias on the accumulation of social class on mortality, we analysed separately all men belonging to the actual study cohort and the subgroup of employed men in 1990. In the study cohort the proportion who were employed stood at 91%, but the figure varied from 96% in the non-manual class to 86% in the manual class (table 2). The distribution of childhood social class is similar in the both groups. The results for mortality are shown for all causes and for causes for which a cumulative effect of social class was found.

The pattern of association between mortality and social class is different in these two groups, albeit we do not observe uniform differences for different causes of death. The effect of childhood class is greater for cardiovascular diseases than for alcohol related causes. In employed men, there is no difference between the socially mobile groups in cardiovascular disease mortality. For alcohol related causes mortality is greater in the downwardly than upwardly mobile group, but among manual men parental class is not associated with mortality.

For all specific causes of death, mortality differences are greater in the whole cohort than in employed men. These results indicate that the effect of childhood class in comparison with the effect of adult class may be greater in employed men than in the whole cohort, and therefore analyses of employed men may overestimate the effects of cumulative social class on mortality from cardiovascular diseases.

DISCUSSION

Our results showed a clear association between the number of times in low social class and increased mortality among Finnish men aged 30–42 at death. However, when the socially mobile group was further divided into the downwardly and upwardly mobile, mortality appeared to be almost entirely related to current adult class. In all causes of death, the relative mortality ratio between non-manual and manual men was about 3.0 irrespective of childhood social class, while within the non-manual class mortality for men with a manual background was 1.2 times higher than for men with a

non-manual background. However, for alcohol related causes and particularly for cardiovascular diseases, a somewhat greater effect was observed for childhood class. It also became clear that analyses, which exclude economically inactive persons, may overestimate the cumulative effect of social class on mortality.

Reliability of the results and comparisons with other studies

In some respect the results of this register based study are more reliable than those obtained earlier. Firstly, loss to follow up and non-linkage of deaths is very small (less than 1%). Secondly, social class is determined on the basis of the same coding scheme in different phases of life. In addition, this information is always based on a contemporary register and never inquired from a person retrospectively. Thirdly, information on social class from both adulthood and childhood is available for 90% of the birth cohort.

Comparisons with our and earlier studies have to be made with caution. Firstly, studies differ with regard to the age groups they cover. For example, the Scottish cohort in the study by Hart *et al*[7] consisted of men in the latter part of their working careers being aged 35–64 at the baseline of a 21 year follow up. Because of this age difference, compared with our study, the distribution of causes of death is not the same. Whereas in the Scottish cohort 83% of deaths were attributable to cardiovascular disease and cancer, the corresponding figure in our study was 22%. The association between all cause mortality and cumulative social class is therefore probably different in these studies as well.

Secondly, studies differ with regard to their coverage of economically inactive people. In our study, which comprised both unemployed and other economically inactive men, the most recent social circumstances had the strongest impact on mortality and the effect of accumulation was weaker. By contrast studies of employed men in Sweden[23] and Scotland[4 24] showed a weaker association between the most recent adult class and mortality. Our results indicate that studies confined to the economically active population may partly overestimate the effects of childhood class within the manual adult class.

Finally, social structure and social mobility also differ between countries and study periods. There were no important differences in the social structure between our study and the Scottish study mentioned above (with the non-manual class accounting for 45% of the population in our study and for 50% in the Scottish study), but even so, there were clear differences in social mobility. The figure for upward mobility in our study was 20% and in Scotland 30%, for downward mobility they were 11% and 4%, respectively. Comparison of inter-generational mobility is complicated by the use of different selection criteria of participants at baseline. The Scottish cohort was in the active labour force and in the latter part of its working career and was therefore a more selected group than ours.

Malignant neoplasms and cardiovascular disease

Our finding of no association between adult and childhood social class and mortality from neoplasms was in accordance with another Finnish study on men aged 35–49.[25] However, at older ages mortality from neoplasms is more related to adult social class,[4 25] partly because of an increasing incidence of lung cancer. There are some cancers such as liver, testis, prostate, and stomach cancer that may have their origin in very early life,[26–28] but these cancers are rare in our cohort.

There was some indication in our data that lifetime social class could have an accumulative effect on cardiovascular disease. In these causes men with a manual background had excess mortality in both the non-manual and the manual adult social class. Only 13% of all deaths were attributable to cardiovascular disease, but by age 65 this proportion will increase to almost 50%. Therefore, if the mortality pattern found for cardiovascular disease holds with age, it will probably also be established for mortality from all causes of death combined.

Social circumstances in different stages of the lifecourse probably have different effects on different specific cardiovascular diseases.[29–31] However, the age groups we are studying are too young for us to study various cardiovascular diseases such as stroke and coronary heat disease separately.[32] In several earlier studies cardiovascular diseases have been analysed as one category, and their results have confirmed that living conditions in childhood and youth have an effect on mortality from this cause of death.[4 23 24] Our results further showed that adult social class had a much greater effect on total cardiovascular disease mortality than childhood class. This is consistent with the findings showing that adult socioeconomic position and lifestyle are more important characteristics of cardiovascular disease and cardiovascular risk factors than childhood socioeconomic position or other childhood circumstances.[11 33 34]

In a Scottish cohort there was less divergence in the mortality risks associated with childhood and adulthood social class.[7] However, when we excluded economically inactive and unemployed men from our analyses the results were in line with the Scottish findings,[7] indicating that the different mortality patterns in these two studies may be partly attributable to differences in data compositions. In older cohorts where larger numbers tend to drift out of the labour force, there may be more intense bias related to the exclusion of the economically inactive population than in our study.

Alcohol related and external causes

In this Finnish male cohort we found that childhood social class was associated with mortality from alcohol related causes within the non-manual and manual class in adulthood. Furthermore, if alcohol related causes were excluded from all causes of death, the excess mortality of those with a manual background in the non-manual class disappeared.

Among young Finnish men alcohol related causes account for a large proportion of all deaths, and they are an important factor behind socioeconomic differences in mortality.[35 36] Furthermore, social background has an independent effect on alcohol related mortality.[32]

Studies of cumulative lifetime exposure to adverse environments have usually been concerned with their effects on adult chronic disease.[12 37 38] However, not only biological but also social, behavioural, and psychosocial chains of risk operate during the life span affecting besides biological embedding,[39] but purportedly also external risks.[40 41] Cumulative or pathway effects may thus be seen not only for mortality from diseases, but for mortality from external and alcohol related causes, especially at young ages when people may take more risks, for example on the road.[42 43]

Social mobility and its association with social class differences in mortality

In our study men entering the non-manual class had substantially lower mortality than men in the manual class, and slightly higher mortality than stable non-manual men. The mortality of manual class men originating from the non-manual class was substantially higher than the mortality of men in the non-manual class (with the exception of neoplasms) but their mortality was clearly lower than mortality of stable manual men in cardiovascular diseases, slightly lower in external and alcohol related causes, and slightly higher in all diseases combined. In all diseases the mortality differences between the mobile groups point in the direction of health related or selective social mobility.

In general, however, social mobility marginally constrained the mortality differences by social class in adulthood. Particularly for cardiovascular diseases and alcohol related causes, the excess mortality of the adult manual class including a downwardly mobile group compared with the non-manual class including an upwardly mobile group, is substantially smaller than the excess mortality of the stable manual class compared with the stable non-manual class (by 24% and 28%, respectively). In earlier studies it has been shown that social mobility either constrains[7][13] or enlarges,[44] albeit moderately, social class differences in mortality. Very small effects have also been found in studies using other outcomes than mortality, for instance self reported health in young adulthood.[45] These results thus indicate that social mobility has both selective (persons with, for example, poorer health or some other unfavourable characteristic[46] are somewhat more likely to move downwards in the social hierarchy) and accumulative effects (upwardly mobile persons are carrying some exposures from their past), but they have only very limited impact on social class differences in mortality.

In summary, the research evidence on the accumulative effects of social disadvantage on health and mortality is inconclusive. It is likely that the extent of accumulation differs for different health outcomes and age groups. Furthermore, national differences and differences in the experiences of successive birth cohorts, for example in the degree of social mobility, may modify the accumulative effects of social disadvantage on health.

The attraction of a composite lifetime indicator of social class is that it yields larger socioeconomic differences in mortality than any single measure of social class of at any particular time point. On the other hand, this kind of combined measure does cause difficulties in assessing the contribution of social class at different stages of life. In our study the conclusions changed essentially when the direction of social mobility was taken into account. These analyses showed that it is more useful to differentiate between childhood and adulthood effects than to use a combined measure of social class.

In conclusion, these data on 30–34 year old Finnish men at the beginning of an eight year follow up show that adverse social circumstances as measured by social class in childhood and early adulthood, have only a weak cumulative effect on total mortality. A modest cumulative association was observed in mortality from alcohol related causes and cardiovascular diseases. However, even in these causes of death the effects of adult class were much stronger than those of childhood class.

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Effect of parental social class, own education and social class on mortality among young men

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Background: The aim of the study is to examine the effects of parental class, own education and social class on mortality by cause of death among young men. Methods: The study is based on 1990 census records for 186,408 Finnish men, aged 30–34 in 1990, linked with death records for 1991–95 (1530 deaths). Results: Parental class was associated with all-cause (Index of Relative Dissimilarity (IRD) =12%) and cause-specific mortality before adjustment for one's own social class and education. Adjustment reduced the IRD by 18–58%, depending on the cause of death, and caused mortality differences to disappear for causes other than cardiovascular diseases and those related to alcohol. The unadjusted variation in all-cause mortality by educational attainment (IRD=33%) and by own class (IRD=38%) was greater than by parental class. Adjustment for parental class had only a minor effect on the variation by education and own class. Adjustment for own class reduced the IRD for education in all-cause mortality by 31%, in mortality from disease by 17%, and in mortality from external causes by 33%. For own class the reductions in the IRD obtained by adjustment for education were 36%, 46%, and 33%, respectively. Conclusions: The effect of parental class on the mortality of young men is indirect and mainly mediated through its influence on education and social class. The effect of education on mortality is as strong as that of occupation-based social class. These variables are not interchangeable measures of socioeconomic status; they both should be taken into account in studies on inequalities in mortality.

Keywords: mortality, socioeconomic differentials, social background, young adulthood

Aₙ inverse association between adult social class and mortality is well established,[1,2] but the causes of this association are not fully understood. Several researchers have pointed out that a meaningful explanation of health and mortality differences between classes needs to consider circumstances both in childhood and adulthood.[3–7] It has been shown that mortality from some causes of death, such as stomach cancer, ischaemic heart disease, stroke, and respiratory diseases, is influenced by socio-economic circumstances in early life,[8–12] while mortality from other causes, such as lung cancer and accidental and violent deaths, is said to be related mainly to later socio-economic circumstances.[13]

Parental class, educational attainment and own social class are linked with different phases of the life course (figure 1), and their effect on health and mortality is probably mediated through different mechanisms. Some studies have simultaneously studied the effects of education and own class on mortality in adulthood,[14–16] and others have taken into account both parental and own class.[13,17–21] To the best of our knowledge only one study[22] has used all three factors in studying mortality among adult men. Several studies on the 1958 British birth cohort have analysed the effects of occupational class measured at different ages and educational attainment on various health indicators.[3,23–25]

The purpose of this paper is to study the effects of parental social class, own education and adult social class on mortality by cause of death among young men in Finland. We also estimate to what extent the social class differentials observed in mortality in adulthood can be attributed to socioeconomic factors preceding social class measured by parental class and own education. The analysis draws on register-based census data on all men aged 30–34 in 1990 in Finland. These data allow us to study the effects of social background and current social class on mortality from main groups of causes of death in 1991–95 in young adulthood, the phase of life in which the relative variation in mortality by social class is the greatest.[26,27]

DATA AND METHODS

The data were compiled by Statistics Finland. They are based on the 1990 census records for males who were born in the period 1956–60 and lived in Finland during the censuses of 1970 and 1990. The death records for 1991–95 and the information from the 1970 and 1985 censuses were linked to the 1990 census records by means of personal identification numbers. The share of unlinked deaths is less than 1%[27] and the numbers of deaths are consistent with the figures in the official statistics.[28]

Social class is determined on the basis of occupation. If an individual had no occupation for other reasons than

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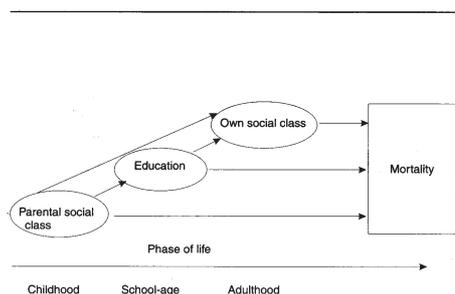
Figure 1 Direct and indirect effects of parental class, education and own class on mortality in adulthood

being a student in the 1990 census, information on this person's occupation was searched for in the 1985 census. If this information was not found, the person was classified into the group of 'others', which also included students. This group was excluded from all mortality analyses. The classification (table 1) is based on Statistics Finland's classification,[27] but manual workers are divided into skilled and unskilled groups on the basis of a combination of three classification schemes used in earlier studies.[29–31] Men were classified as unskilled if they belonged to this category according to all these schemes.

Parental social class is based on the occupation of the head of household (usually father) when the cohort members were aged 10–14 in 1970. The coding is based on Statistics Finland's classification,[32] but farmers are divided into two groups on the basis of farm size: farmers (at least 10 hectares of arable land) and small farmers (less than 10 hectares of arable land). The classifications for social classes in 1970 and 1990 are basically the same except for the above mentioned differences in divisions of manual workers and farmers. The changes in occupational structure in Finland between 1970 and 1990 have an effect on the relative sizes of the non-manual class and farmers in this study. The proportion of the population working in agriculture has decreased from 20% to 9%, while the proportion of people working in finance, insurance etc., and services has increased.[33]

Educational attainment is classified on the basis of the International Standard Classification of Education[34] using the following categories: higher education (at least 13 years of education), higher secondary education (12 years), lower secondary education (10–11 years), middle school (basic education with academic orientation, 9–10 years), and basic education (9 years or less).

All analyses are adjusted for age. Mother tongue (Finnish speaking, Swedish speaking and others), type of residence (urban, rural), and region of residence (Western Finland, capital area, Eastern Finland) in 1970 are also adjusted for. These social background factors are associated with later socioeconomic status and mortality.

During the study period 1530 men from the cohort died (174 men per 100,000 person-years). Causes of death are coded according to the Finnish Classification of Diseases 1987, which is based on the Ninth Revision of the International Classification of Diseases.[35] The causes of death used in the analyses are as follows (ICD codes and number of deaths given in brackets):

- All causes (1–799,E800–999, 1530 deaths).
- Diseases (1–799, 544 deaths), including cardiovascular diseases.
- Cardiovascular diseases (390–459, 187 deaths).
- External causes (E800–990, 986 deaths), including suicides.
- Suicides (E950–959, 485 deaths).

Our study is restricted to two specific causes of death, suicides and cardiovascular diseases, which are the two most common causes of death in this male age group. However, alcohol-related mortality was distinguished as a separate cause of death because earlier studies have shown its importance in Finland.[36,37] The definition of alcohol-related death is the same as that used by Mäkelä et al.[36] This category includes deaths in which the underlying cause of death explicitly mentions alcohol (Finnish classification: alcoholic thiamine deficiency (265.0A), alcoholic psychoses (291), alcohol dependence syndrome (303), alcoholic polyneuropathy (375.5), alcoholic cardiomyopathy (425.5), alcoholic gastritis (535.3), alcoholic diseases of the liver (571.0–571.3), alcoholic diseases of the pancreas (577.0D–F, 577.1C–D), accidental poisoning by alcohol (E851), and accidental poisoning by medical agents in combination with alcohol (E849)). In addition deaths in which at least one contributory cause is alcoholic intoxication (305), or one of the causes listed above, are classified as alcohol-related.

Person-years and deaths by cause of death were tabulated by the control variables, and by own social class, parental class and education. The cross-tables were analysed by means of Poisson regression analysis, with the cell in cross-tabulation taken as the unit of analysis. The GLIM statistical package was used in fitting the models.[38] The results are presented as relative mortality rates (and their 95% confidence intervals).

Our summary measure for the magnitude of variation in mortality rates among groups was a variant of the Index of Dissimilarity,[39] which we call the Index of Relative Dissimilarity (IRD). The following formula was used:

$$IRD = 100 \times \sum p_i \times \frac{|RR_i - RR|}{RR}$$

where p_i is the proportion of group i of all men, and RR_i is the relative mortality rate for group i obtained from the Poisson regression analysis, and $RR = \Sigma\ p_i \times RR_i$.

The IRD gives the average deviation of the group-specific relative rates from their mean in percentage of the mean. It takes into account the sizes of the groups and thus adjusts for the differences in the distributions of the socioeconomic variables. The IRD was computed to allow and facilitate a valid comparison of the effects of the three socioeconomic indicators on mortality.

RESULTS

Approximately 30% of the men in this cohort share the same class position as their parents *(table 1a)*. *Table 1b* shows that own class is largely determined by education. For example, 90% of the men in the highest educational category as opposed to 10% of the men with basic education belong to the non-manual class.

The differences in mortality by cause of death between socioeconomic categories without and with adjustments for the other two socioeconomic variables are shown in *tables 2–4* for parental class, own educational attainment and one's own social class.

There are clear differences by parental class in all-cause mortality and cause-specific mortality when adjustment is made for the confounders only *(table 2)*. Adjustment for education and one's own class reduces the differences substantially, and causes them to disappear in other causes than cardiovascular diseases and alcohol-related causes. In cardiovascular diseases descendants of unskilled manual workers, farmers, self-employed, and unclassified have elevated mortality even after the adjustment. In alcohol-related causes sons of manual workers and unclassified show higher mortality than sons of upper non-manual class, although these differences are not statistically significant. Parental class thus has a minor

direct effect on mortality, but its effects on mortality are mediated through education and one's own class.

Educational differences in all-cause mortality, as well as in mortality from each of the causes of death studied here, are substantial. Mortality increases from higher levels of education to lower ones, but the differences between educational groups vary by cause of death. The greatest differences are found in alcohol-related mortality. Adjustment for parental class reduces mortality variation only marginally. Own social class accounts for 31% of the variation in all-cause mortality by education. The reduction obtained by adjusting for own class in diseases is smaller, 17%. In external causes and in suicides it is 33%. The direct effect of education on mortality is clear: all educational groups show statistically significant excess mortality in comparison to the higher educated when all adjustments are made in each of the studied causes of death.

Mortality is associated with own social class in all groups of causes of death studied *(table 4)*. Adjustment for parental class has a minor effect on mortality differences between the upper non-manual class and manual classes in cardiovascular diseases and in alcohol-related causes of death, where the reduction in relative mortality rates is 21 and 11–12%, respectively. For other causes, adjustment

Table 1a Own class in 1991 by parental class in 1970 (%), Finnish men aged 30–34 on 1 January 1991

| Parental class | Own social class | | | | | | | | | |
	Upper non-manual	Lower non-manual	Skilled manual	Unskilled manual	Farmer	Self-employed	Other	All	P-years in 1991	% of all
Upper non-manual	50	22	9	6	1	6	7	100	15763	8
Lower non-manual	27	26	23	12	1	7	5	100	27777	15
Skilled worker	13	19	39	18	1	7	5	100	65452	35
Unskilled worker	7	14	41	24	2	5	7	100	19182	10
Farmer (≥10 ha)	13	14	22	12	30	6	3	100	16965	9
Small farmer	9	14	33	19	14	7	5	100	23856	13
Self-employed	16	20	23	15	1	20	4	100	13292	7
Other	9	14	35	20	3	7	13	100	3919	2
All	17	19	30	16	5	7	5	100	186206	100
p-years in 1991	31801	34590	56723	29869	9679	13596	9948	186206		

Table 1b Own class in 1991 by education (%), Finnish men aged 30–34 on 1 January 1991

| Education | Own class | | | | | | | | | |
	Upper non-manual	Lower non-manual	Skilled manual	Unskilled manual	Farmer	Self-employed	Other	All	P-years in 1991	% of all
Higher	78	11	1	1	2	4	4	100	28466	15
Higher secondary	20	50	9	6	2	8	6	100	34926	19
Lower secondary	2	11	50	19	7	7	4	100	78813	42
Middle school	5	15	31	27	6	10	6	100	16432	9
Basic	2	9	33	31	6	9	11	100	27570	15
All	17	19	30	16	5	7	5	100	186206	100
p-years in 1991	31801	34590	56723	29869	9679	13596	9948	186206		

Table 2 Number of deaths (n), and relative rates of mortality (with 95% CI) according to parental class, and values of Index of Relative Dissimilarity (IRD) by cause of death without and with adjustment for own education and own class (age, mother tongue, and type of residence and region of residence in 1970, adjusted for in all models)

Cause of death Model	Upper non-manual	Lower non-manual	Skilled manual	Unskilled manual	Farmer	Small farmer	Self-employed	Other	IRD %
				Parental class					
All causes (n)	86	196	544	211	125	221	102	45	
1.Parental class	1	1.26 (1.0–1.6)	1.47 (1.2–1.8)	1.98 (1.5–2.6)	1.31 (1.0–1.7)	1.65 (1.3–2.1)	1.36 (1.0–1.8)	2.21 (1.5–3.2)	12
2.1+own class	1	0.99 (0.8–1.3)	0.96 (0.8–1.2)	1.18 (0.9–1.5)	0.88 (0.7–1.2)	1.03 (0.8–1.4)	0.99 (0.7–1.3)	1.36 (0.9–2.0)	5
3.1+education	1	0.93 (0.7–1.2)	0.90 (0.7–1.1)	1.12 (0.9–1.5)	0.83 (0.6–1.1)	0.97 (0.7–1.3)	0.88 (0.7–1.2)	1.28 (0.9–1.9)	6
4.2+education	1	0.91 (0.7–1.2)	0.86 (0.7–1.1)	1.03 (0.8–1.4)	0.81 (0.6–1.1)	0.92 (0.7–1.2)	0.89 (0.7–1.2)	1.19 (0.8–1.7)	6
Diseases[a] (n)	35	78	186	67	50	70	44	14	
1.Parental class	1	1.25 (0.8–1.9)	1.26 (0.9–1.8)	1.62 (1.1–2.4)	1.37 (0.9–2.1)	1.39 (0.9–2.1)	1.50 (1.0–2.3)	1.72 (0.9–3.2)	9
2.1+own class	1	1.10 (0.7–1.6)	0.99 (0.7–1.4)	1.19 (0.8–1.8)	1.05 (0.7–1.7)	1.04 (0.7–1.6)	1.29 (0.8–2.0)	1.30 (0.7–2.4)	8
3.1+education	1	1.03 (0.7–1.5)	0.93 (0.6–1.3)	1.13 (0.7–1.7)	1.04 (0.7–1.6)	1.00 (0.6–1.5)	1.13 (0.7–1.8)	1.22 (0.7–2.3)	6
4.2+education	1	1.04 (0.7–1.5)	0.92 (0.6–1.3)	1.09 (0.7–1.7)	0.99 (0.6–1.6)	1.00 (0.6–1.5)	1.19 (0.8–1.9)	1.19 (0.6–2.2)	7
Cardiovascular (n)	9	18	58	29	20	24	21	8	
1.Parental class	1	1.11 (0.5–2.4)	1.50 (0.8–3.0)	2.76 (1.3–5.8)	2.26 (1.0–5.0)	1.97 (0.9–4.3)	2.81 (1.3–6.0)	3.93 (1.5–10)	31
2.1+own class	1	0.97 (0.4–2.1)	1.16 (0.6–2.3)	1.99 (0.9–4.3)	1.68 (0.7–3.9)	1.44 (0.6–3.2)	2.32 (1.1–5.1)	2.89 (1.1–7.5)	26
3.1+education	1	0.87 (0.4–1.9)	1.02 (0.5–2.1)	1.77 (0.8–3.8)	1.57 (0.7–3.5)	1.30 (0.6–2.9)	2.01 (0.9–4.4)	2.55 (1.0–6.7)	26
4.2+education	1	0.90 (0.4–2.0)	1.04 (0.5–2.1)	1.75 (0.8–3.8)	1.54 (0.7–3.5)	1.28 (0.6–2.9)	2.09 (1.0–4.6)	2.55 (1.0–6.7)	25
External causes[a] (n)	51	118	358	144	75	151	58	31	
1.Parental class	1	1.27 (0.9–1.8)	1.61 (1.2–2.2)	2.23 (1.6–3.1)	1.29 (0.9–1.9)	1.82 (1.3–2.5)	1.28 (0.9–1.9)	2.54 (1.6–4.0)	17
2.1+own class	1	0.92 (0.7–1.3)	0.93 (0.7–1.3)	1.15 (0.8–1.6)	0.79 (0.5–1.2)	1.01 (0.7–1.4)	0.87 (0.6–1.2)	1.38 (0.9–2.2)	17
3.1+education	1	0.86 (0.6–1.2)	0.88 (0.7–1.2)	1.10 (0.8–1.5)	0.73 (0.5–1.1)	0.95 (0.7–1.3)	0.75 (0.5–1.1)	1.29 (0.8–2.0)	9
4.2+education	1	0.84 (0.6–1.2)	0.81 (0.6–1.1)	0.98 (0.7–1.4)	0.70 (0.5–1.0)	0.87 (0.6–1.2)	0.73 (0.5–1.1)	1.17 (0.7–1.8)	8
Suicide (n)	28	63	159	73	39	79	31	13	
1.Parental class	1	1.24 (0.8–1.9)	1.31 (0.9–2.0)	2.05 (1.3–3.2)	1.23 (0.7–2.0)	1.73 (1.1–2.7)	1.26 (0.8–2.1)	1.93 (1.0–3.7)	17
2.1+own class	1	0.90 (0.6–1.4)	0.76 (0.5–1.2)	1.08 (0.7–1.7)	0.74 (0.4–1.2)	0.96 (0.6–1.5)	0.81 (0.5–1.4)	1.05 (0.5–2.1)	12
3.1+education	1	0.84 (0.5–1.3)	0.72 (0.5–1.1)	1.03 (0.7–1.6)	0.70 (0.4–1.2)	0.91 (0.6–1.4)	0.74 (0.4–1.2)	0.99 (0.5–1.9)	13
4.2+education	1	0.82 (0.5–1.3)	0.67 (0.4–1.0)	0.93 (0.6–1.5)	0.66 (0.4–1.1)	0.84 (0.5–1.3)	0.71 (0.4–1.2)	0.91 (0.5–1.8)	13
Alcohol-related[b] (n)	28	83	277	118	52	101	38	25	
1.Parental class	1	1.63 (1.1–2.5)	2.29 (1.6–3.3)	3.32 (2.2–5.0)	1.61 (1.0–2.6)	2.19 (1.4–3.3)	1.53 (0.9–2.5)	3.68 (2.2–6.3)	23
2.1+own class	1	1.17 (0.8–1.8)	1.30 (0.9–1.9)	1.67 (1.1–2.5)	1.01 (0.6–1.6)	1.21 (0.8–1.9)	0.98 (0.6–1.6)	1.95 (1.1–3.3)	12
3.1+education	1	1.09 (0.7–1.7)	1.21 (0.8–1.8)	1.59 (1.0–2.4)	0.89 (0.6–1.4)	1.10 (0.7–1.7)	0.86 (0.5–1.4)	1.80 (1.0–3.1)	13
4.2+education	1	1.05 (0.7–1.6)	1.12 (0.7–1.7)	1.40 (0.9–2.2)	0.90 (0.6–1.5)	1.03 (0.7–1.6)	0.84 (0.5–1.4)	1.63 (0.9–2.8)	10

a: Diseases include cardiovascular diseases. External causes include suicide.
b: Alcohol-related causes are also included in the other categories of causes of death.

for parental class has no effect. Other than manual classes show no statistically significant excess mortality compared to the upper non-manual class in any of the causes of death studied after adjustment for education. Manual classes have statistically significant excess mortality from external causes, suicide, and alcohol-related causes of death, although the adjustment reduces their excess by 70%.

Adjustment for education leads to a greater reduction in the variation by one's own class than was the case when education was adjusted for social class in mortality from all diseases and cardiovascular diseases. In external causes, suicide, and alcohol-related causes of death the reduction in the IRD obtained by adjusting for one another is almost as large, approximately 30%, as educational attainment and own class.

We also studied interactions between the three socio-economic variables to see whether any of them have a different effect in different classes of another variable.

However, these interactions proved to be statistically insignificant.

DISCUSSION
The effect of parental class

Parental class was found to have only a minor direct effect on all-cause mortality and mortality from both diseases and external causes after controlling for education and current social class. However, descendants of unskilled manual workers, farmers, and self-employed showed a small excess mortality from cardiovascular diseases. In alcohol-related causes men with a manual class background had elevated mortality. There are some earlier reports which suggest that parental class has at least some direct effect on all-cause mortality[21] and on mortality from such causes of death as coronary heart disease,[13,17] stroke, stomach cancer, and respiratory disease,[13] but these studies have covered older men than our study. Among men under age 40 mortality from these diseases is

Table 3 Number of deaths (n), and relative rates of mortality (with 95% CI)according to education, and values of Index of Relative Dissimilarity (IRD) by cause of death without and with adjustment for parental class and own class (age, mother tongue, and type of residence and region of residence in 1970, adjusted for in all models)

Cause of death Model	Higher	Higher secondary	Education Lower secondary	Middle school	Education	IRD %
All causes (n)	85	189	771	163	322	
1.Education	1	1.85 (1.43–2.38)	3.26 (2.61–4.08)	3.43 (2.64–4.45)	4.25 (3.35–5.39)	33
2.1+parental class	1	1.85 (1.43–2.40)	3.25 (2.58–4.10)	3.43 (2.63–4.48)	4.19 (3.27–5.36)	33
3.1+own class	1	1.78 (1.32–2.39)	2.35 (1.73–3.18)	2.47 (1.78–3.44)	2.90 (2.11–3.98)	22
4.2+own class	1	1.79 (1.33–2.40)	2.36 (1.74–3.22)	2.49 (1.80–3.47)	2.90 (2.10–3.99)	23
Diseases[a] (n)	46	84	251	61	102	
1.Education	1	1.53 (1.07–2.17)	2.01 (1.48–2.73)	2.39 (1.64–3.47)	2.45 (1.74–3.45)	21
2.1+parental class	1	1.53 (1.07–2.17)	2.02 (1.46–2.77)	2.38 (1.62–3.50)	2.44 (1.71–3.48)	21
3.1+own class	1	1.62 (1.07–2.44)	1.88 (1.22–2.89)	2.20 (1.37–3.54)	2.18 (1.38–3.46)	17
4.2+own class	1	1.62 (1.07–2.44)	1.88 (1.22–2.91)	2.20 (1.36–3.54)	2.18 (1.37–3.46)	17
Cardiovascular diseases (n)	11	28	95	17	36	
1.Education	1	2.09 (1.09–4.04)	3.11 (1.73–5.57)	2.74 (1.33–5.64)	3.63 (1.92–6.86)	27
2.1+parental class	1	2.02 (1.05–3.92)	2.84 (1.55–5.18)	2.52 (1.21–5.24)	3.18 (1.65–6.14)	24
3.1+own class	1	2.65 (1.26–5.58)	3.20 (1.46–7.03)	2.81 (1.17–6.79)	3.59 (1.56–8.23)	22
4.2+own class	1	2.61 (1.24–5.50)	3.05 (1.38–6.72)	2.69 (1.11–6.52)	3.30 (1.43–7.63)	20
External causes[a] (n)	39	105	520	102	220	
1.Education	1	2.22 (1.55–3.16)	4.70 (3.44–6.43)	4.63 (3.24–6.62)	6.35 (4.58–8.82)	39
2.1+parental class	1	2.23 (1.56–3.19)	4.69 (3.40–6.46)	4.66 (3.24–6.70)	6.25 (4.45–8.76)	39
3.1+own class	1	1.96 (1.30–2.96)	2.81 (1.85–4.26)	2.79 (1.78–4.37)	3.58 (2.33–5.51)	26
4.2+own class	1	1.98 (1.31–2.99)	2.84 (1.87–4.33)	2.83 (1.81–4.44)	3.59 (2.33–5.55)	26
Suicide (n)	16	60	266	47	96	
1.Education	1	3.08 (1.80–5.27)	5.85 (3.59–9.53)	5.22 (3.00–9.06)	6.69 (4.00–11.18)	37
2.1+parental class	1	3.16 (1.84–5.41)	6.08 (3.68–10.03)	5.43 (3.10–9.51)	6.88 (4.06–11.65)	37
3.1+own class	1	2.79 (1.52–5.13)	3.67 (1.97–6.83)	3.31 (1.70–6.45)	4.01 (2.11–7.62)	25
4.2+own class	1	2.84 (1.54–5.21)	3.80 (2.04–7.11)	3.43 (1.76–6.70)	4.13 (2.16–7.89)	26
Alcohol-related causes[b] (n)	24	71	363	85	179	
1.Education	1	2.45 (1.55–3.88)	5.38 (3.57–8.10)	6.33 (4.04–9.92)	8.32 (5.46–12.96)	42
2.1+parental class	1	2.38 (1.50–3.78)	4.98 (3.28–7.56)	5.98 (3.79–9.43)	7.54 (4.90–11.62)	41
3.1+own class	1	2.05 (1.21–3.47)	3.05 (1.79–5.18)	3.60 (2.06–6.28)	4.40 (2.56–7.57)	30
4.2+own class	1	2.04 (1.20–3.45)	2.95 (1.73–5.03)	3.52 (2.01–6.16)	4.19 (2.42–7.23)	29

a: Diseases include cardiovascular diseases. External causes include suicide.
b: Alcohol-related causes are also included in the other categories of causes of death.

rare, and our data do not allow for an analysis of specific diseases.

Parental class was found to have almost no impact on social class differences in mortality. A similar result has been reported earlier in studies concerning older men in Finland[17,19] and the United States.[22] In Sweden, on the other hand, adjusting for parental class reduced the all-cause mortality difference between non-manual and manual classes among males aged 25–40 by 17%[18] (here 3%). Part of the difference in the effects of parental class may be due to larger relative mortality differences in Finland than in Sweden,[40] which is partly caused by the higher proportion of economically inactive men in Sweden[41] (12%) than in Finland (5%).

In this age group the indirect effect of parental class via education is more obvious than the direct effect. It is possible that when our cohort grows older and disease mortality becomes more common, the direct effect[42–44] increases. It may be the stronger the lower the mortality and the smaller the socioeconomic mortality differences in adulthood are, as the comparison between this study and the Swedish study indicated. In our study the effect of parental class was detected not only in chronic diseases, but also in alcohol-related causes of death. Presumably parental class has an impact on some health-related habits transmitted to adulthood,[45,46] such as alcohol use. However, this may also be a reflection of an accumulation of detrimental conditions[47] leading to misuse of alcohol rather than an adaptation of a single habit.

The effect of education and own class

In this study own education and social class had almost equally large effects on mortality when examined separately. The difference between the reference group

Table 4 Number of deaths (n), and relative rates of mortality (with 95% CI) according to own class, and values of Index of Relative Dissimilarity (IRD) by cause of death without and with adjustment for parental class and own education (age, mother tongue, type of residence, and region of residence in 1970 adjusted for in all models)

Cause of death Model		Own class					
	Upper non-manual	Lower non-manual	Skilled manual	Unskilled manual	Farmer	Self-employed	IRD %
All causes (n)	127	191	600	436	87	89	
1.Own class	1	1.38 (1.10–1.73)	2.64 (2.18–3.20)	3.66 (3.01–4.47)	2.30 (1.75–3.04)	1.64 (1.25–2.15)	38
2.1+parental class	1	1.37 (1.09–1.72)	2.60 (2.13–3.18)	3.58 (2.92–4.40)	2.40 (1.79–3.23)	1.63 (1.24–2.15)	37
3.1+education	1	0.90 (0.69–1.17)	1.42 (1.09–1.85)	1.91 (1.46–2.50)	1.26 (0.91–1.75)	0.94 (0.69–1.30)	23
4.2+education	1	0.91 (0.69–1.18)	1.43 (1.09–1.87)	1.92 (1.46–2.52)	1.34 (0.95–1.88)	0.96 (0.70–1.32)	24
Diseases[a] (n)	67	86	190	139	34	28	
1.Own class	1	1.19 (0.87–1.63)	1.61 (1.22–2.13)	2.26 (1.69–3.01)	1.78 (1.17–2.71)	0.99 (0.64–1.52)	25
2.1+parental class	1	1.18 (0.86–1.63)	1.61 (1.20–2.15)	2.22 (1.65–3.01)	1.80 (1.15–2.82)	0.95 (0.61–1.48)	25
3.1+education	1	0.85 (0.58–1.24)	1.01 (0.68–1.50)	1.38 (0.93–2.06)	1.13 (0.69–1.86)	0.65 (0.40–1.06)	13
4.2+education	1	0.85 (0.58–1.24)	1.03 (0.69–1.53)	1.39 (0.93–2.08)	1.15 (0.69–1.94)	0.64 (0.39–1.05)	13
Cardiovascular diseases (n)	21	22	71	46	13	14	
1.Own class	1	0.97 (0.54–1.73)	1.90 (1.18–3.08)	2.36 (1.42–3.93)	2.20 (1.09–4.42)	1.57 (0.81–3.06)	30
2.1+parental class	1	0.91 (0.51–1.64)	1.71 (1.04–2.83)	2.07 (1.22–3.51)	1.84 (0.87–3.89)	1.32 (0.67–2.61)	28
3.1+education	1	0.54 (0.28–1.06)	0.92 (0.47–1.77)	1.13 (0.57–2.23)	1.09 (0.48–2.48)	0.82 (0.38–1.78)	17
4.2+education	1	0.53 (0.27–1.04)	0.88 (0.46–1.72)	1.06 (0.54–2.11)	0.97 (0.41–2.29)	0.73 (0.34–1.59)	17
External causes[a] (n)	60	105	410	297	53	61	
1.Own class	1	1.60 (1.17–2.18)	3.78 (2.90–4.94)	5.22 (3.97–6.87)	2.90 (2.00–4.20)	2.37 (1.66–3.37)	45
2.1+parental class	1	1.59 (1.16–2.18)	3.71 (2.81–4.89)	5.09 (3.84–6.76)	3.09 (2.08–4.57)	2.39 (1.67–3.43)	44
3.1+education	1	0.96 (0.66–1.39)	1.80 (1.25–2.58)	2.42 (1.68–3.48)	1.41 (0.91–2.19)	1.23 (0.81–1.86)	31
4.2+education	1	0.97 (0.67–1.41)	1.82 (1.26–2.62)	2.43 (1.68–3.51)	1.54 (0.98–2.42)	1.28 (0.84–1.94)	30
Suicide (n)	29	55	203	134	29	35	
1.Own class	1	1.73 (1.10–2.70)	3.87 (2.63–5.70)	4.85 (3.26–7.24)	3.26 (1.94–5.48)	2.81 (1.72–4.59)	41
2.1+parental class	1	1.77 (1.12–2.77)	3.98 (2.66–5.94)	4.92 (3.26–7.44)	3.50 (2.02–6.07)	2.91 (1.77–4.79)	42
3.1+education	1	0.93 (0.56–1.55)	1.72 (1.04–2.84)	2.15 (1.29–3.58)	1.50 (0.82–2.74)	1.37 (0.78–2.42)	27
4.2+education	1	0.96 (0.57–1.60)	1.79 (1.08–2.96)	2.21 (1.32–3.69)	1.62 (0.87–3.04)	1.44 (0.81–2.54)	28
Alcohol-related causes[b] (n)	38	78	296	237	53	61	
1.Own class	1	1.87 (1.29–2.71)	4.30 (3.12–5.94)	6.56 (4.73–9.10)	2.73 (1.72–4.34)	2.49 (1.62–3.82)	49
2.1+parental class	1	1.78 (1.22–2.59)	3.89 (2.79–5.42)	5.93 (4.24–8.31)	2.83 (1.74–4.60)	2.42 (1.57–3.73)	46
3.1+education	1	1.07 (0.68–1.68)	1.90 (1.22–2.96)	2.74 (1.75–4.29)	1.22 (0.70–2.12)	1.19 (0.71–1.99)	34
4.2+education	1	1.06 (0.67–1.67)	1.83 (1.17–2.87)	2.65 (1.69–4.16)	1.33 (0.75–2.35)	1.22 (0.73–2.04)	32

a: Diseases include cardiovascular diseases. External causes include suicide.
b: Alcohol-related causes are also included in the other categories of causes of death.

and the lowest group was greater according to educational level than own class in all causes of death studied, but the overall variation did not differ greatly when the distribution of the variables was taken into account.

Educational attainment proved to be an important factor behind social class differences in mortality. This is consistent with findings from earlier Finnish studies among males aged 35–64[15] and over 60.[18] In the United States, too, education has been shown to account for a substantial part of class differences.[22] On the other hand, class differences in educational attainment were found to contribute very little to mortality differences according to own social class in Scotland.[20] The Scottish male cohort was born several decades earlier than in our study and was 35–85 years old during the mortality follow-up. The difference between the results for Scotland and Finland might be due to the different time of birth and age of the cohorts, and to other differences between the countries. In the Scottish cohort father's social class determined one's own class more strongly than was the case in our study. However, it is possible that country-specific differences are changing: at ages 15–39 education seemed to have a stronger effect on self-rated health than parental or own class both in Britain and Finland.[7] Although it is evident that both education and social class have independent effects on health and mortality, it is not clear which of the two is the more important factor. In a study which used a cumulative measure of social class, the effect of education was claimed to be smaller than that of lifetime social class on self-rated health at age 33.[25]

The large effect of education observed in this study may partly be due to the young age of our cohort. At these ages any exposure to occupational risk factors cannot have been very long-lived. In this study mortality from diseases was not associated with own class after controlling for education, while accidental and violent deaths were related to own class, as has been found earlier.[13] The effect of education on health behaviours and lifestyles may be stronger than the effect of current social class.[48] Furthermore, higher education provides a person with health-related knowledge, values and abilities. However, it is possible that those with more cultural capital as measured in terms of education, have been more likely to get and use (health) knowledge and to avoid risks even before completing any formal education. Health-related behaviours have been shown to be associated with educational track in adolescence and predict educational attainment in adulthood.[49,50] According to this Finnish study[49] a key factor affecting association between health behaviours and educational success[51] was social background.

Age of the cohort and other possible difficulties

Our data do not as yet allow us to study the effects of parental class for cohorts older than those used here, aged 31–39 years during the study period. This restriction should be taken into account when interpreting the results. For example, at this age one's own social class is still in transition. The low mortality in the young cohort restricted the choice of causes of death, and the relatively

small number of deaths might have affected statistical significance, particularly the non-significance of interactions. The distribution of deaths in this age is skewed to external causes of death. The prevalence of diseases increases with age, for example at ages over 40 half of male deaths are due to cardiovascular diseases. One might also assume that greater integration into occupational class with increasing age has an increasing effect on a person's health and risk of death.

Although the multicollinearity of socioeconomic variables might be a problem in the analyses, we found no inconsistencies in the results, and the confidence intervals were rather small. It seems likely that the large effect of education on social class differences in mortality at these ages among males is real, and not a statistical artefact.

One of the advantages of register-based census data is that the information about the past is not based on retrospective surveys. Its main disadvantage is the lack of information on such factors as lifestyles and personal traits, which might be confounders in association with education, social class and later mortality. On the other hand, in studies that have collected these kinds of data, the number of persons has often been too small to study mortality in reliable ways.

CONCLUSIONS

Except for cardiovascular diseases and alcohol-related causes of death parental social class has virtually no direct effect on the mortality of young men in Finland. It does, however, have an indirect effect which is mediated through its influence on education and one's own class. The effect of educational attainment on mortality is at least as crucial as that of the conventionally used occupation-based social class. A substantial proportion of social class differences in mortality, especially from diseases, can be attributed to educational attainment. In external causes of death occupational class has a substantial effect. Parental class, own education and own class are not interchangeable measures of socioeconomic status. Their effect may vary in different age groups. They all should be taken into account in studies of inequalities in health and mortality.

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ORIGINAL ARTICLE

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Effect of living conditions in the parental home and youth paths on the social class differences in mortality among women

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Aims: A longitudinal study was undertaken to assess the effects of parental home and youth paths on the adult social class differences in mortality among women. *Methods:* The study used population registration data on all Finnish women aged 30–34 in 1990 for whom information on their childhood characteristics and youth paths were available from the 1970, 1975, 1980, 1985, and 1990 censuses. Cause of death follow-up was for the period 1991–98 (1,185 deaths). *Results:* Adjusting for parental social class, family type, and number of siblings attenuated the effects of adult social class on cardiovascular disease mortality by 27%; for all external causes this attenuation was negligible. Educational, marital, and employment paths accounted for a substantial part (30–85%) of the social class differences in mortality for all specific causes of death. *Conclusions:* Although living conditions in the parental home were associated with mediating life trajectories in youth their effect on adult social class differences in mortality was moderate. Youth paths have a pervasive influence on mortality risks and social class differences in mortality in middle adulthood.

Key words: childhood, early marriage, life-course, mortality, socioeconomic mortality differences, unemployment, women, youth.

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INTRODUCTION

Social class differences in mortality are a very strong indication of inequity in society. When these differences exist at young age, they strongly contribute to social class differences in life expectancy. The reduction of these differences has been, and is, a central target in many national and international health promotion initiatives (1). Studying how these differences are generated may help to find the age and population groups for whom interventions are most needed in order to achieve greater equity between social classes. Among men a clear inverse association is found between current social class and mortality but among women associations are more ambiguous (2–8).

In many cases the very task of assigning women to a certain occupation-based social class is problematic (3, 6, 9). Women's occupations do not in all stages of their lives and in all countries always correspond to their formal education, abilities, or their socioeconomic standing at home (10). Furthermore, in some mortality (7) and health (9) studies household-based measures of social class have revealed greater

socioeconomic differences than individual-based measures. For these reasons, it has been suggested that the household-based measure (taking account of both spouses' occupational positions) should be used with women. On the other hand, results from Finland, where labour force participation of women in full-time work is high, have shown that the association between occupation-based class and mortality is similar regardless of whether the class is based on the woman's own or her husband's occupation (5). In addition, because of problems in assigning non-working women to a social class many analyses are restricted to the economically active population. However, especially among women, the exclusion of inactive persons leads to a serious under estimation of socioeconomic differences in mortality (11).

Almost equally well established as the idea of an inverse association between social class and mortality is the view that mortality in adulthood is not only related to prevailing circumstances but evolves over a lifetime (12), or has its origins very early in life (13, 14). Several living conditions in the parental home, such as social class, family type, and number of siblings,

may influence both adult social class and health directly or by exerting an effect on youth transitions, which affect social class differences in mortality in adulthood (15, 16). Thus, living conditions in the parental home may form the basis for the explanation of adult social class differences in mortality. Major transitions in youth and early adulthood, i.e. gaining educational qualifications, leaving the parental home, forming a family and entering the labour market, are important turning points in people's lives (17). In these "socially critical periods" (18) failure to embark on adult roles may generate a drift to socially adverse life trajectories and also extend their effects on the formation of own adult social class and mortality. Life trajectories in youth – youth paths – may modify the effects of parental home but are also likely to have their own independent effect on adult social class differences in mortality. Besides major life transitions, other mechanisms exerting an effect on both adult social class and health are also in progress in youth. An important mechanism is the formation of one's own lifestyle (19), which is likely to be related to educational, family formation, and employment path.

According to earlier findings, which are not adjusted for the effects of own education and social class, there is a clear association with lower parental class and higher all-cause and disease mortality at ages 25–34 in Finland (21) and with higher disease mortality at ages 19–33 in Sweden (22). There are several studies that provide information on the effects of living conditions in the parental home, or youth paths, on current social class differences in mortality but these studies were only concerned with men (12, 22–26).

The aim of this study is to examine the effects of living conditions in the parental home and youth paths – i.e. factors preceding adult social class – on mortality and social class differences in mortality from several causes of death among women.

We use Finnish census-based data sets that contain information on education, occupation, marital and employment status, and also on a few parental home factors for persons aged 34 or under in 1990. Because all our information from childhood to adulthood is based on concurrent censuses, we only have a small number of missing observations, the social class variables are always collected in a similar manner, and the quality of data is not affected by individual recall bias.

DATA AND METHODS

Study cohort
The data were compiled by Statistics Finland on the basis of the 1990 census records for women born in 1956–60 and who were living in Finland at the time of the 1970 and 1990 censuses. The death records for 1991–98 and the information from the 1970, 1975, 1980, and 1985 censuses were linked to the 1990 census records by means of personal identification numbers. The percentage of unlinked deaths was less than 1%.

Adult social class
Women's adult social class is mainly determined on the basis of their own occupation: in 1990 84% of the women in this cohort were in the labour force. We used the husband's social class for housewives (approximately 8% of the cohort). For those whom we were not able to assign to any social class in 1990 (8% of the cohort), occupational information was searched for from the 1985 census. If a woman was a student or was not in the labour force even in 1985, she was classified in the group of "others". Social class codes are based on Statistics Finland's classification (4) (see Table III).

Parental home
Parental social class is based on the occupation of the head of household (usually the father) when the cohort members were aged 10–14 in 1970. Parental social class classification follows the same schema as that for adult social class but combines the intermediate and lower non-manual classes and separates skilled and unskilled manual classes. *Family type* distinguishes families with two parents from single-parent families. *Number of siblings* is classified as zero, one, and two or more.

Broad cultural aspects in the parental home may be reflected by region of residence and minority status (as measured for example by mother tongue), which have both been found to be related to social class but also to mediating variables such as education, and also to mortality (27). The region of residence in 1970 (western Finland, capital area, eastern Finland) and language (Finnish-speaking and others, of whom 80% are Swedish speakers) are included as confounders.

Youth paths
Educational path is based on educational attainment, which is classified into five categories on the basis of the International Standard Classification of Education.

The family formation process is measured with *marital path* and *early motherhood*. *Marital path* is categorized into the following groups: (1) "partner in 1990" consists of those who were single at age 20–24, but who were married or cohabiting at age 30–34 (in the 1990 census); (2) "partner in 1980" consists of those who were married at age 20–24, (3) "partner in 1975" consists of those who were married at age

15–19 and (4) "no partner" consists of those who were single in 1980 and who lived without a partner in 1990. This variable is not fully consistent since information on cohabitation in 1975 or 1980 was not available, though we have equated cohabitation with marriage in 1990. This has been done because in 1990 14% of the cohort was cohabiting (in 1975 approximately 2%).

Early motherhood is categorized into the following groups: (1) "no children" consists of those women who had no children at the 1980 census, (2) "child in 1980" consists of those women who had a child at the 1980 census but not at the 1975 census, and (3) "child in 1975" consists of those women who were mothers at the 1975 census.

The *employment path* identifies different experiences during the employment career: (1) "steady" employment means continuous employment up to the end of 1990 after compulsory or further education; (2) "short unemployment" means less than six months' unemployment during 1986–90 or experience of unemployment at the time of one census; (3) "long unemployment" means unemployment lasting at least six months during 1986–90, or repeated unemployment experiences at the time of at least two censuses; (4) a "fragmental" employment path means occasional exclusion from the labour force for reasons other than unemployment, retirement, or education, (5) "retired" consists of women on disability pension.

Mortality in 1991–98

Causes of death for 1991–95 are coded according to the Ninth Revision of the International Classification of Diseases (ICD9), and for 1996–98 according to the Tenth Revision of the International Classification of Diseases (ICD10). This change of classification has no effect on the results. Information on cause of death is based on autopsies in approximately 78% of deaths in our data.

Causes of death used here are *all diseases* (63% of all deaths in the disease category were due to cardio-vascular diseases and neoplasms; neoplasms were not related to adult social class and are thus not separately analysed here), *cardiovascular diseases* (ICD9:390–459, ICD10:i00–i99), *all external causes* (mainly due to suicides, traffic accidents and poisonings), and *suicides* (ICD9:E95, ICD10:x60–x84, Y870).

Methods

Person-years and deaths by cause of death were tabulated by period, own adult social class, and variables related to parental home and youth. The cross-tables were analysed by means of Poisson regression analysis, with the cell in cross-tabulation taken as the unit of analysis. The GLIM statistical package was used to fit the models (28). All analyses are adjusted for age, language, and region. The results are presented as relative mortality rates (and their 95% confidence intervals).

We have also used the index of relative dissimilarity (IRD) (23) as a summary measure of variation in mortality rates between groups of each explanatory variable. The following formula wasused: $IRD = 100 \cdot \Sigma p_i \cdot |RR_i - RR|/RR$ where p_i is the proportion of women in group i of all study participants, and RR_i is the relative mortality rate for those in group i obtained from the Poisson regression analysis, and $RR = \Sigma p_i \cdot RR_i$. The IRD adjusts for the differences in the distribution of the population.

RESULTS

Path from parental home to one's own adult social class

Table I shows how the living conditions in the parental home, described by parental social class and family type, are associated with youth paths. The proportion of women with basic education, early marriage, and long-term unemployment experiences increases with descending parental social class. These proportions tend to be larger for women from a single-parent family background within non-manual and skilled manual classes.

Youth paths exert an influence on the formation of own class. As a consequence, social background and distributions of youth paths vary between social classes (Table II). For instance, in the manual class those with a manual and single-parent background, low education, early marriage and motherhood, and unemployment experiences are over-represented. The variation in the background of women in intermediate and lower non-manual classes is more subtle.

Variation of mortality by adult social class

In all causes of death studied, there is a sizeable variation in mortality by adult social class (see the IRDs in Table III). Manual class and "others" show statistically significant excess mortality in comparison with the mortality of the upper non-manual class from all causes of death studied (Table IV). Also the intermediate non-manual class has an excess mortality, while mortality of the lower non-manual class is lower than average, and it deviates statistically significantly from that of the upper non-manual class only for suicides. Mortality variation in suicides can be attributed to the low mortality of the upper non-manual class, and the high mortality of "others".

Table I. *Educational path, marital path, and employment path by parental social class and family type (%): women aged 30–34 in 1990*

	Parental social class										Trend test for parental social class[b]	
	Upper non-manual		Lower non-manual		Skilled manual		Unskilled manual		All		Pr>|t|	
	Parental family type											
	Two parents[a]	Single parent	Two parents[a]	Single parent	Two parents[a]	Single parent	Two parents[a]	Single parent	Two parents[a]	Single parent	Two parents	Single parent
Educational path:												
Higher	49	43	27***	18	13***	8	8	6	19***	11	<0.0001	<0.0001
Higher intermediate	38	36	39***	33	30***	23	21	20	32***	25	<0.0001	<0.0001
Lower intermediate	7***	13	21***	24	33***	36	44***	40	31***	35	<0.0001	<0.0001
Middle school	4	5	8***	13	11	12	10	11	8***	11	<0.0001	0.4083
Basic	2	4	6***	12	13***	21	18***	23	10***	18	<0.0001	<0.0001
Marital path:												
Partner in 1990	57***	47	52***	47	47***	40	42***	38	49***	41	<0.0001	<0.0001
Partner in 1980	19	22	25	26	30	32	33	33	29	30	<0.0001	<0.0001
Partner in 1975	1	3	4***	7	7***	12	9***	13	6***	10	<0.0001	<0.0001
No partner in 1990	23	28	19	20	16	17	15	17	17	18	<0.0001	<0.0001
Employment path:												
Steady	69	63	68**	65	65***	61	58	60	65***	61	<0.0001	<0.0001
Short unemployment	3	3	3	4	4	5	5	5	4	5	<0.0001	0.0028
Long unemployment	6	9	8	9	12**	14	17	17	11***	14	<0.0001	<0.0001
Fragmental	22	22	19	19	18	17	16	16	18	17	<0.0001	<0.0001
Retired	1	3	1	2	2*	3	3	3	2***	3	<0.0001	0.0545
All	100	100	100	100	100	100	100	100	100	100		
Numbers in 1990	13835	722	21690	4561	58056	5259	15457	3636	109038	14178		

Notes: [a]P-values ($p<0.05$ (*), $p<0.01$ (**), $p<0.001$ (***)), for the difference of the proportions of women originating from two- and one-parent families within each social class. These tests are adjusted for Bonferroni (Moore DS, McCabe GP. Introduction to the practice of Statistics. New York: W.H. Freeman, 1999). For instance, in upper non-manual class there is no significant difference in the proportion of women having higher education according to family type (49% versus 43%), while there is a significant difference in lower non-manual class (27%versus 18%).
[b]Trend tests are regressing the row variable (coded 0 and 1) on the continuous parental social class variable, which are treated as continuous, separately by parental family type. For instance, there is a statistically significant trend for the proportions of women who have higher education from the upper non-manual class to unskilled manual class in women originating from both two-parent families (49%, 27%, 13%, 8%) and one-parent families (43%, 18%, 8%, 6%).

Association of variables preceding adult social class with mortality and their effect on social class differences in mortality

Variables related to parental home show only a small variation in mortality for all causes of death studied, as measured by the IRD. In general, women from single-parent families or without siblings have higher mortality. Women originating from an upper non-manual background have lower mortality rates in all causes of death combined and in all diseases.

Table IV shows the reductions in the excess mortality of other social classes in comparison with mortality of the upper non-manual class after controlling for the living conditions in the parental home and youth paths. Percentage changes in excess mortality are shown for each social class. In addition, the "average change" is shown. This index compares the average deviation of mortality (for all social classes) in the model in which a variable under study has been controlled for to the average deviation in the uncontrolled model. The "average change" takes into account the size of the excess mortality and the number of person-years in each class; therefore large reductions in small excess mortality make only a minor contribution to this measure.

For all diseases, controlling for variables related to parental home leads to a moderate reduction in the excess mortality of the other classes. For cardiovascular diseases, controlling for parental home leads to a 27% reduction in the excess mortality by adult social class. For external causes there are no mortality differences between parental social classes with the exception of the higher mortality of "others". As a consequence, control for parental home has no effect on adult social class differences in external causes.

Table III shows that there are considerable differences

Table II. *Variables related to parental home and youth by adult social class (%):women aged 30–34 in 1990*

	All	Upper non-manual (a)*	Intermediate non-manual (b)*	Lower non-manual (c)*	Manual (d)*	Employer/ farmer (e)*	Other (f)*
All Parental home:	100	100	100	100	100	100	100
Parental class							
- upper non-manual	8.2	21.6[bcdef]	7.0[adef]	6.5[adef]	2.7[abcef]	5.5[abcde]	11.5[abcde]
- lower non-manual	14.8	20.6[bcdef]	16.5[ade]	16.0[ade]	9.5[abcf]	10.3[abcf]	14.3[ade]
- skilled manual	35.7	26.7[bcdf]	38.8[aef]	37.9[adef]	40.2[acef]	27.3[bcd]	30.2[abcd]
- unskilled manual	10.8	4.8[bcdef]	10.1[ad]	10.1[adf]	16.5[abcf]	9.7[adf]	12.2[acde]
- farmer/employer	29.0	25.4[cde]	26.5[cde]	28.2[abe]	28.8[abe]	45.7[abcdf]	27.5[e]
- other	1.5	0.8[bcdef]	1.2[adf]	1.3[adf]	2.3[abcef]	1.4[adf]	4.3[abcde]
Family type							
- two parents	88.2	91.7[bcdef]	88.3[adf]	88.7[adf]	85.4[abce]	89.5[adf]	83.8[abce]
- single parent	11.8	8.3[bcdef]	11.7[adf]	11.3[adf]	14.6[abce]	10.5[adf]	16.2[abce]
Number of siblings							
- zero	9.1	10.1[de]	10.1[de]	9.4[d]	7.4[abce]	8.6[abd]	9.0
- one	26.0	32.4[bcdef]	27.6[adef]	26.5[ade]	20.5[abcef]	23.1[abcd]	24.6[abd]
- two or more	64.9	57.5[bcdef]	62.3[acdef]	64.1[abde]	72.2[abcef]	68.3[abcd]	66.4[abd]
Youth:							
Educational path							
- higher	17.9	69.6[bcdef]	8.4[acdf]	11.8[abdef]	1.8[abcef]	9.5[acdf]	15.0[abcde]
- higher intermediate	31.1	21.7[bcde]	46.6[acdef]	39.6[abdef]	14.7[abcef]	28.6[abcdf]	23.2[bcde]
- lower intermediate	31.8	5.4[bcdef]	24.6[acdef]	34.4[abdef]	52.0[abcef]	42.4[abcd]	20.4[abcde]
- middle school	8.6	2.2[bcdef]	11.1[ace]	7.8[abdef]	11.0[ace]	9.0[acdf]	9.8[ac]
- basic	10.6	1.2[bcdef]	9.3[acdf]	6.5[abdef]	20.4[abcef]	10.6[acdf]	31.6[abcde]
Marital path							
- partner in 1990	47.8	60.4[bcdef]	48.7[adf]	48.9[adf]	39.7[abcef]	47.5[adf]	25.8[abcde]
- partner in 1980	28.8	17.7[bcdef]	27.3[acdef]	29.1[abdef]	34.9[abcef]	38.2[abcdf]	24.2[abcde]
- partner in 1975	6.4	1.6[bcdef]	6.1[ade]	5.5[ade]	10.4[abcef]	7.9[abcd]	5.4[ade]
- no partner before 1990	17.1	20.3[bcdef]	17.9[acdef]	16.5[abdef]	14.9[abcef]	6.4[abcdf]	44.6[abcde]
Early parenthood							
- no child before 1985	76.6	92.7[bcdef]	78.4[ade]	77.7[ade]	65.2[abcef]	68.8[abcdf]	77.6[ade]
- child in 1980	20.8	6.8[bcdef]	19.4[ade]	20.1[ade]	30.2[abcef]	28.1[abcdf]	20.0[ade]
- child in 1975	2.6	0.4[bcdef]	2.3[ade]	2.1[ade]	4.7[abcef]	3.1[ad]	2.5[ad]
Employment path							
- steady	64.7	72.5[def]	72.1[def]	72.0[def]	50.6[abcef]	68.2[abcdf]	4.2[abcde]
- short unemployment	4.2	2.1[bcdef]	4.2[acd]	3.5[abd]	6.5[abcef]	4.0[ad]	4.4[acd]
- long unemployment	11.4	5.8[bcdf]	10.4[acdef]	8.6[abdef]	19.5[abce]	7.9[abdf]	19.3[abce]
- fragmental	17.8	19.4[bcdf]	12.9[acdef]	15.3[abdf]	21.8[abcef]	19.5[bcdf]	33.1[abcde]
- retired	1.9	0.1[bcdef]	0.4[adf]	0.6[adf]	1.6[abcef]	0.5[adf]	39.0[abcde]

Notes: *The superscript letters show whether the proportion for a given social class differs from an equivalent proportion for an indicated social class at the 5% significance level. For instance, in the case of number of siblings, category zero, "10.1[de]" shows that the proportion of women without siblings (10.1) in the upper non-manual class is significantly different from the proportion of women without siblings in the manual social class (d) and in employers and farmers (e), where the proportions were 7.4 and 8.6, respectively. In addition, the proportion of women in the upper non-manual class with zero siblings does not differ from the corresponding proportions for intermediate non-manual class (b), lower non-manual class (c) and others (f).

in mortality by educational attainment, particularly for all causes combined and external causes. In these causes, mortality increases for every educational group from higher to basic education. Controlling for education has a crucial effect on mortality differences by adult social class in all other causes except for suicides (Table IV).

There is a small variation in mortality according to marital path and early parenthood as indicated by IRDs (Table III). Living with a "partner in 1990" is associated with lower than average mortality, while having a partner or a child in 1975 or not living with a partner in 1990 is associated with elevated mortality

rates. After educational attainment is added to the model the further effect of controlling for marital path is small. However, adjusting for family formation leads to an average change in mortality deviation of 19% in external causes.

Long-term unemployment and early retirement are associated with elevated mortality (Table III). A further control for employment path has an effect on the excess mortality of the manual class and the "others". Both these classes have a large share of long-term unemployed (19%), and among "others" 39% of women were retired.

The excess mortality of intermediate non-manual

Table III. *Number of person-years and all deaths, mortality rates and index of relative dissimilarity (IRD), controlled for age, language, andregion of residence: various causes of death in 1991–98 among women aged 30–34 in 1990*

Variable			Mortality rates				
	Number of person-years	Number of all deaths	All causes	Diseases	Cardiovascular disease	External causes	Suicide
Social class (IRD)			(34.6)	(32.8)	(35.6)	(37.6)	(27.6)
- upper non-manual	226933	120	52.5[L]	35.9[L]	6.7	16.6[L]	6.1[L]
- middle non-manual	326389	249	75.1	49.3	8.8	25.8	15.0
- lower non-manual	367745	229	62.4[L]	39.0[L]	7.9	23.5[L]	13.4
- manual	327305	339	104.7[H]	60.1	15.2[H]	44.6[H]	18.5
- farmer/employer	121195	73	61.5[L]	31.9[L]	10.9	29.7	12.8
- other	44654	175	389.8[H]	268.0[H]	40.0[H]	122.0[H]	55.7[H]
Parental home:							
Parental class (IRD)			(10.8)	(14.6)	(21.1)	(5.9)	(6.7)
- upper non-manual	116171	78	64.5[L]	34.1[L]	5.2	30.3	15.7
- lower non-manual	209306	199	93.2	63.5[H]	11.4	29.8	16.3
- skilled manual	504939	437	86.2	54.1	12.7	32.1	14.6
- unskilled manual	152229	139	91.6	59.4	11.7	32.2	12.6
- farmer/employer	409898	294	73.6[L]	44.4[L]	8.5	29.3	14.8
- other	21678	38	173.4[H]	105.3[H]	32.0[H]	68.3[H]	32.1
Family type (IRD)			(7.4)	(8.1)	(12.5)	(6.1)	(5.1)
- two parents	1247918	1000	80.3	50.0	10.1	30.3	14.6
- single parent	166303	185	109.6[H]	70.1[H]	16.6[H]	39.5	18.2
Number of siblings (IRD)			(2.8)	(2.6)	(8.8)	(3.4)	(6.6)
- zero	128986	125	96.5	59.4	10.9	37.2	17.5
- one	367147	296	80.1	49.8	9.0	30.3	16.1
- two or more	918087	764	83.5	52.5	11.6	31.0	14.3
Youth:							
Education (IRD)			(31.8)	(27.3)	(45.9)	(39.8)	(24.1)
- high	252850	132	52.3[L]	36.4[L]	6.4[L]	15.9[L]	9.9[L]
- higher intermediate	439991	262	59.2[L]	38.9[L]	5.4[L]	20.3[L]	12.2
- lower intermediate	449594	372	84.3	52.1	13.0	32.1	15.3
- middle school	121239	129	104.3[H]	53.5	16.4	50.6[H]	20.8
- basic	150547	290	188.3[H]	117.5[H]	23.3[H]	70.8[H]	26.2[H]
Marital path (IRD)			(29.6)	(23.9)	(36.9)	(42.4)	(42.9)
- partner in 1990	675330	329	58.0[L]	40.5[L]	6.7[L]	17.5[L]	8.4[L]
- partner in 1980	407380	338	83.5	50.3	13.0	33.2	14.9
- partner in 1975	90040	110	123.0[H]	59.2	17.6	63.8[H]	32.4[H]
- no partner before 1990	241471	345	141.4[H]	86.3[H]	16.5[H]	55.1[H]	27.4[H]
Early parenthood			(6.5)	(2.4)	(19.6)	(15.4)	(13.9)
- no child in 1980	1083383	872	80.3	52.0	9.5	28.3	13.7
- 1 child in 1980	294384	266	91.2	50.7	14.3	40.5[H]	17.9
- 1 child in 1975	36453	47	130.0[H]	77.3	24.6[H]	52.6[H]	33.2[H]
Employment career (IRD)			(54.9)	(50.4)	(57.7)	(63.7)	(61.5)
- steady	914762	499	54.4[L]	35.7[L]	6.8[L]	18.7[L]	9.4[L]
- short unemployment	59298	58	98.7	47.6	10.1	51.1[H]	20.6
- long unemployment	160537	240	151.9[H]	75.9[H]	19.8	76.1[H]	32.6[H]
- fragmental	252298	156	61.7[L]	40.3[L]	8.4	21.4[L]	9.9[L]
- retired	27326	232	847.3[H]	601.0[H]	120.5[H]	246.9[H]	142.5[H]
All	–	–	83.8	52.4	10.9	31.4	15.1
Number	1414221	1185	1185	741	154	444	213

Notes: *Uppercase letters show whether the mortality rate for a specific group is lower (L) or higher (H) than the average mortality rate for the whole cohort at the 5% significance level.

and manual classes is substantially reduced after living conditions in the parental home and youth paths are controlled for. Only for suicides does their excess mortality remain statistically significant, at the 95% level.

There are no statistically significant interactions between own adult social class and the other explanatory variables. Therefore, the associations between parental home, own education, family formation, and employment path with mortality are similar within each adult social class.

Table IV. *Change (%) in relative mortality after controlling for factors related to parental home and youth on social class differences in various causes of death in 1991–98, women aged 30–34 in 1990*

Social class (%)	Controlled for age, language, region of residence	+Parental class +family type +number of siblings	+Educational path	+Marital path +early motherhood	+Employment path	
All causes						
Intermediate non-manual (23)	1.43 (1.15,1.78)	-9.3	-79.1	-79.1	-60.5	1.17 (0.90,1.51)
Lower non-manual (26)	1.19 (0.95,1.49)	-15.8				0.99 (0.76,1.28)
Manual (23)	2.00 (1.62,2.46)	-6.0	-73.0	-73.0	-80.0	1.20 (0.92,1.56)
Employer/farmer (9)	1.17 (0.88,1.57)	+5.9	-44.9	55.7		1.00 (0.72,1.38)
Other (3)	7.44 (5.90,9.38)	-4.2	-61.8	-66.7	-85.2	0.98 (0.71,1.36)
Average change[a]		-6.5				
All diseases						
Intermediate non-manual (23)	1.37 (1.06,1.79)	-18.9	-75.7	-73.0	-43.2	1.21 (0.88,1.65)
Lower non-manual (26)	1.09 (0.83,1.42)	-66.7				0.94 (0.69,1.29)
Manual (23)	1.68 (1.30,2.17)	-16.2	-82.4	-80.9	-83.8	1.11 (0.80,1.54)
Employer/farmer (9)	0.89 (0.61,1.30)	-5.9	-40.3	-49.9		0.80 (0.53,1.22)
Other (3)	7.47 (5.65,9.89)	-14.1	-51.9	-56.3	-77.6	0.97 (0.65,1.45)
Average change[a]						
Cardiovascular diseases						
Intermediate non-manual (23)	1.32 (0.71,2.46)	-43.8	-84.6	-86.9	-98.5	0.97 (0.46,2.04)
Lower non-manual (26)	1.19 (0.64,2.22)	-63.2				0.85 (0.41,1.78)
Manual (23)	2.30 (1.29,4.09)	-25.4	-86.4	-83.3	-72.7	1.02 (0.48,2.16)
Employer/farmer (9)	1.66 (0.79,3.48)	-13.6	-54.5	-65.0		1.18 (0.50,2.78)
Other (3)	6.03 (3.04,12.0)	-14.1	-70.8	-73.1	-87.1	0.47 (0.19,1.21)
Average change[a]		-26.6				
All external causes						
Intermediate non-manual (23)	1.55 (1.06,2.27)	+5.5	-83.6	-85.5	-80.0	1.11 (0.71,1.74)
Lower non-manual (26)	1.42 (0.97,2.08)	+4.8	-92.9	-95.2	-83.3	1.07 (0.69,1.67)
Manual (23)	2.71 (1.89,3.87)	+3.5	-66.7	-68.4	-78.9	1.36 (0.87,2.13)
Employer/farmer (9)	1.80 (1.14,2.86)	+7.5	-75.0	-66.3	-52.5	1.38 (0.82,2.31)
Other (3)	7.36 (4.87,11.1)	-0.9	-52.4	-64.9	-99.8	1.01 (0.58,1.77)
Average change[a]		+3.2	-69.7	-73.1	-82.3	
Suicides						
Intermediate non-manual (26)	2.44 (1.38,4.33)	+11.1	-1.4	-3.5	+9.0	2.57 (1.33,4.99)
Lower non-manual (23)	2.18 (1.23,3.87)	+11.9	+0.8	0	+13.6	2.34 (1.21,4.50)
Manual (23)	3.02 (1.72,5.30)	+15.8	-11.9	-14.9	-27.7	2.46 (1.24,4.89)
Employer/farmer (9)	2.08 (1.02,4.24)	+13.9	-8.3	+2.8	+26.9	2.37 (1.08,5.20)
Other (3)	9.07 (4.80,17.1)	+3.6	-18.0	-36.1	-94.4	1.45 (0.64,3.31)
Average change[a]		+11.7	-7.6	-11.7	-18.8	

Notes: The reference group is upper non-manual class, for whom the relative mortality rate is 1.00.

[a] Average change is the percentage change in the average relative deviation achieved by controlling for a particular variable or variables in comparison with the first model. Average relative deviation $= 100 \cdot \Sigma (n_i|RR_i - 1|)/\Sigma n_i$, where n_i = person-years lived by social class i during the follow-up, RR_i = relative mortality rate of social class i.

[b] Excess mortality in comparison with upper non-manual class is fully explained (or no excess mortality was observed in the first model).

DISCUSSION

Controlling for living conditions in the parental home had a moderate effect on adult social class differences in mortality from diseases but no effect at all on adult social class differences for mortality from external causes. Life paths in youth explained a substantial proportion of social class differences in mortality; the average attenuation in the excess mortality of other classes in comparison with mortality of the upper non-manual class was 78% for diseases and 82% for external causes. A predominant proportion of the excess mortality of other social classes can be attributed to differences in educational attainment. Also early family formation and long-term unemployment (in the manual class) had pervasive effects on mortality and explained part of the excess mortality of the other classes.

Effects of living conditions in the parental home on mortality andsocial class differences in mortality

Our analyses showed higher mortality for those women who came from one-parent families, who had no siblings, and who came from lower social classes. Strongest association with mortality and the factors related to parental home was found in cardiovascular diseases. Also earlier studies have found equivalent associations with different variables related to parental home and elevated mortality in adulthood (20, 21). Among middle-aged women in the USA, it has been shown that childhood class had an independent effect on the risk of cardiovascular disease, which was not explained by birth weight, adult social class, or behavioural and biological risk factors (29).

However, though in our study women originating from lower non-manual, skilled, and unskilled manual class had higher mortality than women from the upper non-manual class, the mutual differences between these three classes with elevated mortality were minor. The descendants of upper non-manual class comprised 8% of the cohort and the descendants of the other three classes 61% of the cohort. Therefore, the variation in mortality by parental social class remained rather small.

In our analysis parental class was determined on the basis of the occupation of the male head of household. The classification of social class was based on the mother's occupation only when she was a single parent or the father was not employed. However, earlier studies (30) have shown that the class of both parents has an impact on the child's class, and the mother's economic activity has an impact on the class and economic activity of the daughter. Furthermore, the education of the parents, and especially the education of the mother, is important in many ways in determining the child's success at school (30). Besides the educational qualification of the parents, their support and encouragement to their children as well as family centredness and low rate of rows in the family have been shown to be associated with both social achievement and the health of the descendants (31–33). Unfortunately we did not have access to information on these variables. It is possible that in Finland, where the participation of women in the labour force is very high, the additional effects of a mother's economic activity and occupational class would be smaller on a daughter's decisions concerning her educational and employment career than in societies where the full-time participation of women in the labour force is not as common. A lack of information on the parents' educational attainment and rearing styles may to some extent underestimate the explanatory power of parental home on adult social class differences in mortality.

In general, it is possible that material conditions during childhood have been so good for this cohort born in 1956–60 that they cannot have had any major impact on mortality. Women from the upper non-manual class have possibly differed from other classes with regard to such factors as parental reading and encouragement, which have, additionally with material prosperity, a pervasive effect on good health (31, 34, 35). Furthermore, child health clinics and school healthcare may also have served as an intervention that reduced the effects of parental social class.

As a consequence of the weak associations between parental home and mortality, and the small proportion of the cohort with adverse parental home characteristics, parental home does not have a great impact on adult social class differences in total or external mortality among women in their middle adulthood. It is unclear, owing to the facts mentioned above, whether we would have been able to observe a stronger effect of the parental home on adult social class differences in mortality if we had had more extensive information on the living conditions in the parental home. However, variables related to parental home did have a moderate effect on social class differences for mortality from diseases, particularly from cardiovascular diseases. Consistent with findings in the Whitehall II study with regards to morbidity (36), the main path through which the parental home makes a contribution to mortality in adulthood and social class differences in mortality was by affecting life paths in youth.

Distribution of different specific cardiovascular causes of death is different in our study of women aged 31–42 at death from that in older women, and may thus limit the generalizability of our results to older age groups. Statistics Finland's cause of death

statistics show that in the age group 31–44 approximately 19% of deaths due to cardiovascular disease were classified as ischaemic heart disease and 50% were cerebrovascular diseases; in the age group 50 and over these figures were 53% and 28%, respectively. However, our results are consistent with earlier findings among older women (29, 32, 37) and among Finnish men of the same age as women in our study cohort (38). Among these Finnish men the distribution of cardiovascular disease deaths was different from that among women (48% and 19%).

Effects of educational, family formation, and employment paths in youth on mortality and social class differences in mortality

In this study, controlling for education substantially attenuated mortality differences by adult social class. This attenuation is attributable to the whole educational distribution, not to any particular group. The strong inverse association between educational attainment and mortality is consistent with many earlier studies (3, 4).

Education may affect social class differences in mortality through health behaviours and lifestyles. In an American study (39) it was shown that part of the association between health and years of schooling was due to healthy lifestyles, including moderate alcohol consumption, a sense of control over one's life, and the availability of supportive relationships. Also in our cohort, it is likely that educational attainment is associated with lifestyles and health behaviours, and part of the effect of education on social class differences in mortality can be attributed to these factors. In our data, deaths related to excessive alcohol use are of particular importance. It can be estimated that from the 1,185 observed deaths 442 can be attributed to social class, of which educational distribution explained 318 deaths. From these 318 deaths 115 can be attributed to alcohol-related causes (i.e. either underlying or contributory cause of death is alcohol-related disease or alcohol poisoning (40)), which is to say that from the effect of education on social class differences in mortality 36% (115/318) was attributable to alcohol-related causes (calculations not presented here).

The effect of early family formation on social class differences in mortality was approximately 10% in various causes of death, i.e. about the same size as that of the parental home. Early motherhood and early marriage, as well as not living with a partner in 1990, were associated with higher mortality in each adult social class. The association of early motherhood and poor health has been shown previously (41, 42). However, the effects of these factors on adult social

class differences in mortality varied. Not living with a partner in 1990 did not explain adult social class differences in mortality, because having no partner was more common in the upper manuals than in other classes. Furthermore, the effects of early motherhood on social class differences in mortality remained small, because only a small percentage of women had their first child young. Therefore the effect of family formation can be attributed to the higher mortality of those who marry young and their larger prevalence in lower non-manual and manual classes.

In many studies married persons have been shown to have lower mortality rates than never-married (43), widowed or divorced people (8, 44). This protective effect of marriage has been related both to selection into marriage and the causative effects of being married (45). However, our study indicated that the effect of marriage is not always protective (see also Grundy and Holt (46)) but depends on the timing of marriage on life course, early marriage and motherhood being associated with increased mortality. In Rutter's (17) terminology: early marriage is the end product of past processes, such as a disrupted transition into adulthood connected to poor circumstances in the parental home (47). In our study those who married early originated more often from lower social classes. Furthermore, early marriage is also an instigator of future processes, such as the formation of one's social class, which in this study was more likely to be a lower one (see also Hobcraft and Kiernan (42)). To conclude, we showed that approximately 10% of the mortality difference by adult social class is attributable to a higher prevalence of early-married women with elevated mortality in the lower social classes. The elevated mortality among early-married women may be due to past exposures, adverse circumstances within early marriage, and the effects of subsequent experiences such as divorce. With our data, it is not possible to determine the contributions of these different components.

Employment path also had a clear effect on social class differences in mortality, particularly on the excess mortality of the manual class and "others". This effect was attributable to the large proportion of unemployed persons in both of these groups and to the large proportion of persons on disability pension amongst the "others". Information on unemployment concerned a period from one to 18 years before the mortality follow-up. Similar observations indicating the persisting effect of unemployment on women's health have been found earlier (48, 49). The strong effect of unemployment experience in youth on social class differences in mortality does not reflect only the direct effect of unemployment but also other aspects of employment career, such as job insecurity and low

incomes (34, 50). Therefore, unemployment experience in youth may reflect the continuum of lifetime adverse circumstances and experiences affecting adult social class and health.

Controlling for youth paths contributed to social class differences for suicide less than for the other causes of death. After all the controls the excess mortality for the intermediate and lower non-manual classes, and the manual class in comparison with the mortality of the upper non-manual class, was approximately 150%. Among men the corresponding association between suicide mortality and social class does not follow the same pattern, as suicide differences in the same age male cohort is substantially reduced after controlling for youth paths (38).

CONCLUSIONS

Among women aged 31–42 at death, intermediate and lower non-manual, and manual classes had elevated mortality from various causes of death. Althoug had verse living conditions in the parental home were associated with possible mediating life trajectories in youth, the associations between parental home and mortality in middle adulthood were usually modest. Thus, controlling for the living conditions in the parental home was not very significant in accounting for adult social class differences in mortality for most causes of death. However, for mortality from cardiovascular diseases controlling for parental home accounted for about 27% of the adult class differences.

Elevated mortality was found for women who had low education, who were married at age 20–24, or had never married and lived without a partner at age 30–34, or who had experienced unemployment in youth. These life paths in youth accounted for a substantial part (30–85%) of the social class differences in mortality for all specific causes of death. However, for suicides a large excess mortality of all other social classes in comparison with mortality of the upper non-manual class remained after all our controls. Overall, our results suggest that life paths in youth have a pervasive influence on mortality risks and should be taken into account in mortality studies aiming to unravel the mechanisms behind social class differences in mortality.

ACKNOWLEDGEMENTS

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Life-course experiences and mortality by adult social class among young men

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Abstract

Circumstances over the life-course may contribute to adult social class differences in mortality. However, it is only rarely that the life-course approach has been applied to mortality studies among young adults. The aim of this study is to determine to what extent social class differences in mortality among young Finnish men are explained by living conditions in the parental home and life paths related to transitions in youth. The data for males born in 1956–60 based on the 1990 census records are linked with death records (3184 deaths) by cause of death for 1991–98, and with information on life-course circumstances from the 1970, 1975, 1980, and 1985 censuses. Controlling for living conditions in the parental home—social class, family type, number of siblings, language and region of residence—reduced the high excess mortality of the lower non-manual (RR 1.51, 95% CI: 1.28–1.79), skilled manual (RR 2.94, 2.54–3.40), and unskilled manual class (RR 4.08, 3.51–4.73) by 10% in all-cause mortality. The equivalent reduction for cardiovascular disease was 28% and for alcohol-related causes 16%. The effect of parental home on mortality differences was mainly mediated through its effect on youth paths (pathway model). Educational, marital, and employment paths had a substantial effect—independent of parental home—on social class differences from various causes of death. When all these variables were controlled for adult social class differences in cause specific mortality were reduced by 75–86%. Most of this reduction in mortality differences can be attributed to educational path. However, marital and employment paths had their independent effects, particularly on the excess mortality of unskilled manual workers with disproportionately common exposure to long-term unemployment and living without a partner. In summary, social class differences in total mortality among men in their middle adulthood were only partly determined by parental home but they were mainly attributable to educational, marital, and employment paths in youth.

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Introduction

Early life and social class differences in mortality in early middle age

There is strong evidence that concurrent circumstances affect the adult social class differences in mortality. There is also evidence that the origins of adult disease may lie in circumstances preceding adult social class, either in a specific critical or sensitive period probably in very early life, or in the accumulation of detrimental exposures over the life-course (Kuh & Ben-Shlomo, 1997; Ben-Shlomo & Kuh, 2002). According to 'biological programming theories', the factors triggering subsequent disease may be either genetic (e.g. some forms of alcoholism (Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995)) or biological in nature (Barker, Bull, Osmond, & Simmonds, 1990; Barker, 1995; Kuh & Ben-Shlomo, 1997; Barker, Forsén, Uutela, Osmond, & Eriksson, 2001), affecting morbidity after a latent period independently of later experiences (Wilkinson, 1986, p. 6; Frankel, Davey Smith, & Gunnell, 1999; Leon & Davey Smith, 2000), or in

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interaction with subsequent risks (Frankel, Elwood, Sweetnam, Yarnell, & Davey Smith, 1996). According to the 'life-course' or 'pathways' approach to disease epidemiology (Blane, Davey Smith, & Bartley, 1993; Herztman, 1994; Power & Hertzman, 1997; Ben-Shlomo & Kuh, 2002), disease or premature death in adulthood is either a consequence of long-term exposure to physical risks or adverse social and economic circumstances (Wadsworth, 1997; Hart, Davey Smith, & Blane, 1998; Power, Manor, & Matthews, 1999), i.e. accumulation of risk, or a consequence of adverse concurrent circumstances unfolding as a result of unfavourable living conditions earlier in life. These life-course theories recognize that socially patterned exposures are not mutually exclusive with the 'biological programming' theories, particularly in the case where the effect of an early exposure is a cause of adult disease in connection with the effects of subsequent events and circumstances, for instance where the low birthweight babies have an increased risk of coronary heart disease or diabetes if they are obese as adults (Frankel et al., 1996; Lithell et al., 1996). However, one important difference between 'the accumulative risk model' and the 'biological programming' theory is that the former does not regard a single exposure leading to a subsequent disease as necessary.

The life-course approach is widely used in mortality studies (Kuh, Power, Blane, & Bartley, 1997; Leon & Ben-Shlomo, 1997), and there is an acknowledged need to apply it in health inequalities research (Davey Smith, Gunnell, & Ben-Shlomo, 2001). Indeed, it has been shown that factors preceding own social class are important in generating social class differences in mortality in late middle and old age males (Mare, 1990; Wunsch, Duchêne, Thiltgès, & Salhi, 1996; Hart et al., 1998). The impact of past circumstances on social class differences in mortality is perhaps most apparent in cardiovascular diseases (Vågerö & Leon, 1994; Davey Smith, Hart, Blane, & Hole, 1998a; Frankel et al., 1999), but among younger people the life-course approach is mainly used in studies of morbidity, such as those concerned with self-assessed health or limiting long-standing illness (Rahkonen, Lahelma, & Huuhka, 1997; Mheen van de, Stronks, Van den Bos & Mackenbach, 1997; Power, Matthews, & Manor, 1996; Power, Li, & Manor, 2000). The lack of research on young or middle-aged adults is probably due to two reasons. First, at young ages mortality is still low, and data with sufficiently large numbers of participants and long follow-up times are not readily available. Second, most deaths in young adulthood are due to external causes, whereas life-course theories are usually focussed on diseases. The applicability of life-course theories to studying mortality from external causes is not entirely clear. However, certain life trajectories may predispose a person to external causes. Failures or inadequate support in early life or at vulnerable periods (Rutter, 1989) may give rise to an increased tendency to substance abuse or unnecessary risk taking, and thus to an increased risk of accidental death (Kalland, Pensola, Meriläinen, & Sinkkonen, 2001). Furthermore, there is evidence that problems in childhood and adolescence as well as low parental social class are associated with premature mortality from suicides and accidental causes between ages 16 and 50 in Britain (Neeleman, Wessely, & Wadsworth, 1998; Neeleman, 2001). It has also been shown that very early factors, such as low weight gain in infancy, are related to subsequent mortality from suicide (Barker, Osmond, Rodin, Fall, & Winter, 1995).

Young adulthood is period of great social inequality with respect to mortality. From age 30 through to age 50, relative mortality differences by social class are greater than in other times of life (Valkonen, Martelin, Rimpelää, Notkola, & Savela, 1993; Vågerö, 1992). Mortality in young adulthood contributes significantly to social class differences in life expectancy. Furthermore, many of these deaths may be regarded as avoidable: more than one half of all deaths among males are due to external causes. The life-course approach provides important clues for policy-making by helping us to identify the factors and life phases that are most effective in reducing social class differences in mortality in young adulthood (Wadsworth, 1997).

Living conditions in the parental home

Several living conditions in the parental home may influence both adult social class and health through intermediate social factors in childhood and adulthood, and thus form the basis of explanation for adult social class differences in mortality. Crucial childhood living conditions include parental social class and family type. There is abundant evidence on the effects of parental social class on youth paths and adult social class (Goldthorpe, 1980; Pöntinen, 1983; Blane, Davey Smith, & Hart, 1999). The impact of parental social class on subsequent mortality has been shown in numerous studies (Davey Smith et al., 1998a; Vågerö & Leon, 1994; Mare, 1990; Pensola & Valkonen, 2000): the evidence is most convincing for mortality from cardiovascular disease (Forsdahl, 1977; Barker et al., 2001; Notkola, 1985; Whincup, Cook, & Adshead, 1996; Frankel et al., 1999; Davey Smith et al., 1998a). This effect has varied not only by cause of death, but also between areas (Notkola, 1985; Frankel et al., 1999; Davey Smith et al., 2001). Furthermore, there is also some evidence that parental class has no effect on adult mortality (Lynch, Kaplan, & Cohen, 1994).

Broken homes in childhood may negatively affect future health and life paths through the accumulation of difficulties in respect to material and social conditions. For instance one-parent households have on average less

disposable income (Valtioneuvoston kanslia 2001; Sauli, Ahola, Lahelma, & Savolainen, 1989; Östberg, 1996). Another mechanism that ties family type to subsequent health is lack of time for children (Östberg, 1996) and inadequate parenting (Maughan & McCarthy, 1997). Parental separation or a single-parent background has been shown to be associated with conduct problems, physical growth and suicide in adolescence and young adulthood, ill-health and all-cause mortality at later age, and adult alcohol-related problems (Cole & Cole, 1992; Maughan & McCarthy, 1997; Romelsjö, Kaplan, Cohen, Allebeck & Andreasson, 1992; Schwarz et al., 1995; Gould, Fisher, Parides, Flory, & Shaffer, 1996; Sauvola et al., 2001a; Sauvola, Miettunen, Jarvelin, & Rasanen, 2001b; Lundberg, 1993; Buchanan, Brinke, & Flouri, 2000; Wadsworth, Hardy, Paul, Marshall, & Cole, 2002). A single-parent background, and a low parental social class are associated with lower education (see Krein & Beller, 1988; Koivusilta, Rimpelä, & Rimpelä, 1995; Ely, Richards, Wadsworth, & Elliot, 1999), early parenthood (Michael & Tuma, 1985), and lower socio-economic position (Pietilä & Järvelin, 1995).

Number of siblings and crowding also reflect childhood circumstances that may be directly or indirectly connected to health (Power, Manor, & Fox, 1991; Kemppainen et al., 2000; Montgomery, Bartley, & Wilkinson, 1997; Mheen et al., 1997; Cassidy, 2000). In Finland there is evidence that large family size in the parental home is associated with later alcoholism and depression (Kemppainen et al., 2000), and that growing up without siblings is associated with violent crime among males (Kemppainen, Jokelainen, Järvelin, Isohanni, & Räsänen, 2001). There is also an inverse association between the number of siblings and adult social class (Blane et al., 1999), early marriage and parenthood (Michael & Tuma, 1985; Gohel, Diamond, & Chambers, 1997).

Besides social and economic factors, many cultural aspects related to parental home, such as region, ethnicity, and religion, may affect behaviours, personality, and attitudes in childhood as well as opportunities to "self advancement" and thus possibly influence adult social class and health (Mheen van de, 1998). In Finland region of residence and language have been found to be related to social class and mortality, but also to mediating variables such as education (Sauli, 1979; Finnäs, 1986; Koskinen & Martelin, 1994a; Valkonen, 1987). For example, during the childhood of our study cohort educational possibilities in the southern and coastal regions, where the Swedish speaking population is concentrated, have been better than in other parts of the country.

Life trajectories in youth

Youth and early adulthood is a period of major transitions and upheavals in life during which individuals leave their parental home, gain a professional education, set up a family, and enter the labour market. During this 'socially critical period' (Bartley, Blane, & Montgomery, 1997) failures to embark on adult roles may generate a drift to socially adverse life trajectories which extend their effect on own adult social class and mortality in subsequent years. Life trajectories in youth may modify the effects of parental home, but they are also likely to have their own independent effect on adult social class differences in mortality. At least three important trajectories can be identified: educational, family formation and employment path. First, knowledge about health risks, health-related behaviours, and psycho-social characteristics, such as self-esteem and coping strategies, are developed in late adolescence and early adulthood, and are partly, directly or indirectly, attributable to education (Lenthe et al., 2001; Lynch, Kaplan, & Salonen, 1997; Koivusilta et al., 1999; West, 1997; Hendry, Glendinning, & Shucksmith, 1996; Rutter, 1989).

Second, early family formation, both early marriage and early parenthood, may interfere with typical transitions in youth. This may have negative health consequences and complicate the formation of own adult social class. It has been shown in a number of studies that mortality of married men is lower than that of unmarried men (Hu & Goldman, 1990; Ben-Shlomo, Davey Smith, Shipley, & Marmot, 1993; Koskinen & Martelin, 1994b). However, it is possible that early marriage and parenthood may have a detrimental effect both on the formation of own social class and on health status (Mirowsky & Ross, 2002). Part of this effect may be attributable to earlier problems with home, school, behaviour, or family history of those men who have become husbands or fathers in their teens or early 20s (Michael & Tuma, 1985; Dearden, Hale, & Woolley, 1995; Gohel et al., 1997). However, the independent effects of early marriage and parenthood may also be related to material circumstances and health behaviours (Joung et al., 1996; Joung, Stronks, Mheen, & Mackenbach, 1995) that differ from those of men who marry later.

Third, employment trajectories in youth are crucially involved in the formation of own social class, health status and mortality risk. In young adulthood, while individuals are building up their sense of self-esteem, digression from a steady employment career can have a long-lasting effect on socio-economic achievement (Wadsworth, Montgomery, & Bartley, 1999), social isolation (Bartley, 1994), health behaviour (Wadsworth et al., 1999; Fergusson, Horwood, & Woodward, 2001), and directly on psychological well-being, morbidity, and suicides and other mortality (Bartley, 1994; Montgomery, Cook, Bartley, & Wadsworth, 1999; Fergusson et al., 2001; Johansson & Sundquist, 1997; Hammarström, 1994; Stefansson, 1991).

In particular, experiences of unemployment during the early phases of the employment career may be crucial (Hammarström, 1994; Stefansson, 1991). One short spell of unemployment is likely to have different effects on subsequent career and health than long-term or repeated unemployment. However, the unemployment experience is not necessarily a single independent event, but rather one part of chain of other events and factors, possibly starting from poor socio-economic and sociodemographic family background, which are correlated with underachievement at school and adverse adolescent behavioural patterns (Fergusson & Horwood, 1998, Montgomery, Bartley, Cook, & Wadsworth, 1996; Bartley, 1996).

Aim of the study

The aim of this study is to quantify the contribution of living conditions in the parental home and life-events and trajectories in youth to adult social class differences in mortality from various causes of death. Our data are based on the 1990 census records of all men living in Finland, which are linked with the previous censuses from 1970 onwards. Because all our information from childhood to adulthood is based on concurrent censuses, we have only a small number of missing observations, and the data quality is not affected by individual recall bias. The analyses presented in this paper are for men born in 1956–60: this was the cohort for which the 1970 census information describes living conditions in the parental home. Data from subsequent censuses are used to obtain information on educational attainment, family formation and employment careers in youth.

Data and methods

Study population and mortality follow-up

The data are based on the 1990 census records for males who were born in 1956–60 and who lived in Finland at the time of the 1970, 1975, 1980, 1985, and 1990 censuses. Those whose household status was not 'a child' at the time of the 1970 census, approximately 1% of the cohort, were excluded from the data to make sure that the 1970 census information described parental home. The final study population consists of 186,408 men aged 30–34 at the time of the 1990 census. The death records for 1991–98 and the information concerning life events from the 1970 to 1985 censuses were linked to the 1990 census records by means of personal identification numbers. The share of unlinked deaths is less than 1%. The structure of the data is shown with a Lexis diagram (Fig. 1), which describes the information obtained from each census, the age of the study cohort at each census and the subsequent mortality follow-up.
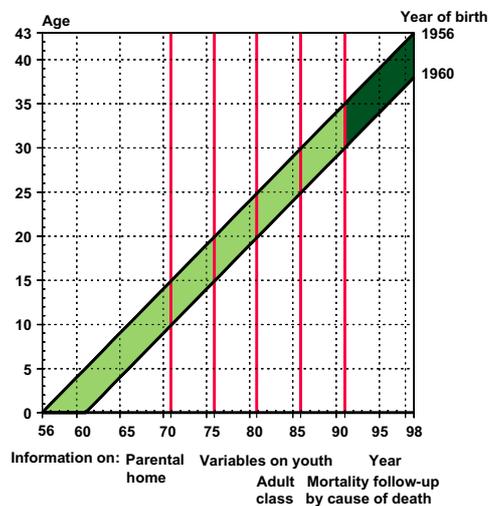
Fig. 1. The study cohort at the time of the 1970, 1975, 1980, 1985, and 1990 censuses (exact time of the census in solid lines), and morality follow-up in 1991–98 shown in dark grey.

Definition of adult social class

Adult social class in 1990 is determined on the basis of own occupation and occupational status. If an individual had no occupation for other reasons than being a student in the 1990 census, information on this person's occupation was searched from the 1985 census. If occupational information was still not found, the person was classified into the group of 'others' (4% of all subjects), which also includes students. The classification is based on Statistics Finland's schema, which in turn is based on the United Nation recommendation for the 1980 population census (Statistics Finland, 1983), distinguishing upper and lower non-manual employees, manual workers, employers, and farmers. We additionally divided manual workers into skilled and unskilled groups (see Pensola, 2002) and combined farmers and employers into one group as these were small groups consisting 13% of the cohort.

Variables related to parental home

Parental social class is based on the occupation of the head of household, usually the father, when the cohort members were aged 10–14 in 1970. The coding is based on Statistics Finland's classification (Official Statistics of Finland, 1974) is consistent with the classification of adult social class used in this study.

Family type distinguishes families with two parents from single-parent families. *Number of siblings* is classified as zero, one, and two or more.

Social and cultural background is described by two factors, viz. *language*, categorized as (1) Finnish speaking and (2) others (of which 80% are Swedish speaking), and *region* of residence in 1970, categorized as (1) western Finland, (2) capital (Helsinki) area, and (3) eastern Finland.

Variables related to youth

Educational path is based on educational attainment in the 1980 and 1990 censuses reflecting qualifications obtained at age 20–24. We use the International Standard Classification of Education (Statistics Finland, 1991) and separate the following categories: higher education (at least 13 years of education), higher intermediate education (12 years), lower intermediate education (10–11 years), middle school (basic education with academic orientation, 9–10 years), and basic education (9 years or less).

The family formation process is described with two variables, *marital path* and *early parenthood*. *Marital path* is categorized into the following groups: (1) 'partner in 1990' consists of those who were not married at age 20–24, but who were married or cohabiting at age 30–34 (in the 1990 census); (2) 'partner in 1980' consists of those who were married at age 20–24 (in the 1980 census), and (3) 'no partner' consists of those who were not married in 1980 and who lived without a partner in 1990. Unfortunately the data for cohabitation are not consistent across the censuses. In 1980 cohabitation was not as common as in the 1990s, and information on cohabitation for those who did not have common biological children was not given in the 1980 census. In the 1990 census, however, cohabitation was separately indicated. Cohabitation accounts for approximately 27% of all cases in the group 'partner in 1990'. *Early parenthood* distinguishes (1) 'no child in 1980' from those (2) who had their first 'child in 1980' or before.

Employment path identifies different experiences of employment career: (1) 'steady' employment means continuous employment up to end of 1990, with the exception of national service, after compulsory or further education; (2) 'short unemployment' means less than 6 months unemployment during 1986–90 or only one spell of unemployment at the time of the 1975, 1980, 1985, or 1990 census; (3) 'long unemployment' means unemployment lasting at least 6 months during 1986–90, or repeated unemployment experiences at the time of at least two censuses; (4) a 'fragmental' employment path means occasional exclusion from the labour force for reasons other than unemployment, retirement, or education. We had no information on these reasons, but it is safe to assume this is a heterogeneous group that includes socially marginalized persons as well as those

having a gap year; (5) 'retired' consists of men on disability pension.

Mortality

The mortality follow-up covers the period from 1991 to 1998, during which 3184 men died. Causes of death for 1991–95 were coded according to the 1987 Finnish Classification of Diseases, which is based on the Ninth Revision of the International Classification of Diseases (ICD9) (National Board of Health, 1986). The codes for 1996–98 are based on the Tenth Revision of the International Classification of Diseases (ICD10) (STAKES, 1999). This change of classification had no effect on the results. In these male age groups definitions of cause of death are highly reliable in Finland, as cause of death certificates are based on autopsies in approximately 86% of all deaths. The following broad groups of causes of death are used:

- all causes,
- all diseases (including cardiovascular diseases),
- cardiovascular diseases (ICD9:390-459, ICD10:i00-i99),
- external causes (including suicides),
- suicides (ICD9:E95, ICD10: ×60–×84,Y870).

In addition to these causes of death we have distinguished alcohol-related mortality as a separate group of cause of death because of its importance in this age group (see Table 1).

Contributory causes of death are also used in the definition of alcohol-related causes. Causes included in this category are those in which alcohol intoxication (ICD9: 305, ICD10: F10.0) is mentioned as a contributory cause of death, or alcoholic disease, such as alcoholic psychoses (ICD9: 291, ICD10: F10.5), alcoholic diseases of the liver (ICD9: 571.0-571.3, ICD10: K70.0-K70.9), and alcoholic diseases of pancreas (ICD9: 577.0D-F,577.1C-D, ICD10: K86.00-K86.08) or alcohol poisoning (ICD9: E851, ICD10:X45) are mentioned as underlying or contributory causes of death. The decision to use contributory causes here was based on the fact that at a young age, a large proportion of alcohol-related causes are due to external causes in which alcohol has a vitally important role. In our data which comprised 1441 deaths due to alcohol, alcohol was the underlying cause for 40% of deaths, of which 315 deaths were due to disease and 261 to alcohol poisoning. It was a contributory cause of death in 128 deaths in the disease category and in 737 deaths in the external-cause category. Alcohol-related causes overlap with all diseases and all external causes. The classification of alcoholic causes has been discussed in closer detail elsewhere (Mäkelä, Valkonen, & Martelin, 1997).

Table 1
Person-years, number of deaths, age-standardized mortality rates (per 100,000) and average deviations of mortality (AD) for various causes of death in 1991–98. Men aged 30–34 in 1990

Variable	Number of person-years	Number of deaths	Age-standardized mortality rates					
			All causes	Diseases	Cardio-vascular disease	External causes	Suicide	Alcohol-related causes
Adult social class (AD)			(95.4)	(35.7)	(11.9)	(59.7)	(25.3)	(51.9)
Upper non-manual	251,063	211	84.0*	46.2*	13.1*	37.8*	17.5*	26.3*
Lower non-manual	271,579	345	126.9*	62.2*	17.7*	64.8*	34.3*	50.4*
Skilled manual	438,356	1084	247.1*	97.8	35.8	149.3*	68.5*	121.7*
Unskilled manual	250,717	859	342.5*	132.1*	44.3*	210.5*	92.5*	179.5*
Farmer/employer	182,636	294	161.0*	62.9*	27.3	98.0*	51.5	64.6*
Other	60,292	391	649.0*	332.2*	83.1*	316.7*	154.2*	227.4*
Parental home								
Parental class (AD)			(28.2)	(8.0)	(5.5)	(20.2)	(7.3)	(21.3)
Upper non-manual	125,087	181	144.7*	74.3*	14.4*	70.4*	37.6*	50.3*
Lower non-manual	219,682	412	187.6*	83.7	21.8*	108.8*	53.7	75.1*
Skilled manual	516,392	1194	231.2	98.3	30.9	132.9	57.3	110.5*
Unskilled manual	149,884	443	295.6*	113.4*	44.7*	182.1*	90.7*	155.4*
Farmer/employer	320,120	657	208.7	90.4	33.6	118.3	57.1	89.3*
Other	18,221	66	362.3*	109.8*	71.3*	252.4*	87.8	159.2*
Family type (AD)			(25.5)	(11.8)	(5.4)	(13.7)	(5.7)	(12.0)
Two parents	1,305,591	2673	204.6*	86.9*	27.8*	117.7*	55.7	92.3*
Single parent	149,052	511	343.0*	150.9*	57.0*	191.9*	86.5*	157.0*
Number of siblings (AD)			(11.2)	(3.2)	(2.1)	(9.2)	(3.4)	(9.1)
Zero	138,313	330	238.6	109.8	27.4	128.7	61.5	94.7
One	393,919	781	198.2*	89.8	28.1	108.4*	52.6	83.7*
Two or more	922,411	2073	224.7	92.5	32.5	132.2	61.1	106.1*
Language (AD)			(8.7)	(3.4)	(1.7)	(5.3)	(3.3)	(5.1)
Finnish	1,385,172	3096	223.4	95.2	31.7	128.1	60.5	101.7
Other	69,470	88	126.7*	57.5*	12.9*	69.1*	24.5*	44.6*

	Population	Numbers						
Region of residence (AD)								
Western	438,508	868	197.9* (12.6)	84.6 (5.3)	26.9 (2.4)	113.4* (8.2)	50.0* (5.6)	86.6* (7.9)
Capital	215,138	496	230.6	108.2*	31.1	122.2	58.1	97.6
Eastern	800,996	1820	227.2	94.3	32.9	132.9	63.9	106.1*
Youth								
Education (AD)								
High	225,700	164	72.7* (78.2)	40.7* (27.4)	11.1* (11.1)	31.9* (50.7)	15.1* (21.4)	21.7* (43.3)
Higher intermediate	302,225	423	140.0*	66.8*	18.8*	73.1*	40.0*	52.6*
Lower intermediate	587,009	1428	243.2*	94.5	36.1*	148.7*	72.4*	116.6*
Middle school	128,329	377	293.8*	129.3*	40.5	164.4*	71.7	134.8*
Basic	211,379	792	374.9*	163.2*	48.7*	211.4*	87.0*	177.4*
Marital path (AD)								
Partner in 1990	832,032	1125	135.2* (98.3)	59.4* (42.3)	20.8* (11.5)	75.7* (56.8)	37.4* (24.6)	54.7* (50.7)
Partner in 1980	214,366	451	210.4	82.0	32.2	128.3	65.8	101.7
No partner	408,245	1608	393.9*	168.8*	50.7*	225.2*	99.0*	187.9*
Early parenthood (AD)								
No child in 1980	1,335,525	2900	217.0 (3.3)	93.2 (0.4)	30.4 (0.8)	123.8 (2.9)	57.5 (2.4)	97.4 (3.0)
Child in 1980	122,117	284	238.4	95.6	35.2	142.7	73.0	116.7
Employment path (AD)								
Steady	1,108,919	1493	134.6* (130.5)	59.0* (54.7)	21.8* (15.1)	75.7* (75.8)	38.3* (31.3)	53.9* (69.2)
Short unemployment	60,180	165	274.2*	106.3	44.8	167.8*	93.4*	134.6*
Long unemployment	176,085	961	546.2*	205.1*	62.5*	340.7*	132.8*	331.2*
Fragmental	71,699	143	199.4	73.9	18.1	125.5	58.6	96.2
Retired	37,760	422	1119.0*	605.7*	151.3*	513.6*	256.7*	291.7*
All	1,454,643	—	218.9	93.5	30.9	125.4	58.8	99.1
Numbers		3184	3184	1360	449	1824	856	1441

*Mortality rate differs from the average mortality rate for the whole cohort at 5% significance level.

Methods

Person-years and deaths by cause of death were tabulated by period, own adult social class, variables related to parental home and youth. The cross-tables were analysed by means of Poisson regression analysis, with the cell in cross-tabulation taken as the unit of analysis. The GLIM statistical package was used in fitting the models (Francis, Green, & Payne, 1993). The results are presented in terms of mortality rates and relative mortality rates (and their 95% confidence intervals). Our modelling strategy is based on the time order of the explanatory variables. Thus we have first adjusted only for age. We have then adjusted for variables preceding own adult social class, first for the living conditions in the parental home and then for youth paths one by one. Information on living conditions in the parental home were all from the 1970 census, but with youth paths we have taken into account the approximate time order of these factors on the basis of 1975, 1980, and 1985 censuses.

In Poisson modelling multicollinearity of the variables, particularly between youth paths, may be a cause of concern. However, mutual relationships between these paths were modest: contingency coefficient was 0.24 between educational and employment path, 0.16 between educational and marital path, and 0.25 between marital and employment path. Therefore, the effect of employment path, which is associated with both education and marital path, on adult social class differences in mortality is only slightly smaller in Table 4 than what it would be in the model where marital path and education are not adjusted for.

We have used *average deviation* (AD) as a summary measure of mortality differences between categories of a specific variable (Moore & McCabe, 1999). AD measures how much the mortality rates of the categories deviate on average from the mortality rate for the whole cohort. AD is weighted for the number of person-years in the categories in order to reduce the weight of small categories with very low or very high mortality.

$$AD = \left(\sum n_i |MR_i - MR| \right) / \sum n_i,$$

where n_i is the number of person-years in category i, MR_i is the mortality rate for a category, and MR is the mortality rate for the whole cohort. The average deviation for total mortality is the sum of average deviations in different causes of death.

In addition, we have calculated the average relative deviation (ARD), which differs from the above equation in that instead of differences between mortality rates we have used differences between the relative mortality rates for category i (RR_i) in relation to mortality in the upper non-manual class (reference group with $RR = 1$). This measure shows how much the mortality of the

different social classes deviates on average from mortality of the upper non-manual class. From ARDs, we further calculated the *average change*. Average change is the percent difference in the ARDs obtained from the model in which an explanatory variable under study has been adjusted for to the ARD in the uncontrolled model. In the context of this study average change quantifies the extent to which an adjusted variable explains adult social class differences in mortality.

Results

According to own adult social class, mortality increased clearly from the upper non-manual class to the unskilled manual class in all causes of death studied (Table 1). For farmers and employers mortality was lower than average. Social class 'Others' comprised only 4% of the cohort, but due to their very high mortality (their all-cause mortality was sevenfold in comparison to mortality of the upper non-manual class (Table 3)), they contribute significantly to the average deviation (AD). When these men are excluded, AD for total mortality decreases from 95.7 to 80.8 but the relative contribution of different causes of death to AD for total mortality is not changed.

The only cause of death where social class differences were not observed was from neoplasms (254 men died of neoplasms); therefore we have not included neoplasms as a separate cause in this study.

Variables related to living conditions in parental home were associated with all causes of death. Mortality increased from higher to lower parental social class consistently for all causes of death. Men from single-parent families had higher than average mortality, which was at least 55% higher than amongst men from two-parent families. Men who had one sibling had lower mortality than average in all causes combined, as well as in external and alcohol-related causes. Men speaking other languages than Finnish had lower than average mortality: their mortality ranged from 40% to 60% of that of Finnish speaking men for various causes of death. However, as this group speaking other languages was small (5%), average mortality deviations—our summary measure of relative mortality differences—were also minor. Men who lived in western Finland in 1970 had lower than average mortality in external and alcohol-related causes. Men in the capital area had elevated mortality from diseases, and men in eastern Finland from alcohol-related causes. However, although there were marked mortality differences by variables related to parental home as described here, these differences were much smaller than those by own adult social class. For example, for total mortality average deviation was 95 by own adult social class and 28 by

parental class. Alcohol-related causes accounted for a relatively large part of the average deviation in total mortality by variables related to parental home; the proportion varied from 47% to 81%. The equivalent proportion accounted for by cardiovascular diseases was about 20%.

Among the variables related to youth, educational, marital, and employment path were associated with mortality. Mortality increased from the basic educated to the higher educated at every level of education in all specific causes of death, with one exception: there were no differences between those men with middle school and lower intermediate education for suicide mortality. External causes accounted for 65% from the average deviation in total mortality by education. Those who had a steady employment path had lower than average mortality for all causes of death. Men who had experienced long-term unemployment or who were retired had elevated mortality. In addition, men who had a partner in 1990 had lower, and men who did not have a partner higher mortality. Those men who had a partner in 1980 showed elevated mortality in comparison to those men who had a partner in 1990. The association with early parenthood and mortality was weak. Mortality for men with children in 1980 was consistently, albeit only slightly, higher than for men who did not have children at that time. Alcohol-related causes accounted for 91% of the average deviation in total mortality.

Variables related to parental home or youth paths have to be related not only to mortality, but also to adult social class in order to be able to explain social class differences in mortality in adulthood. Table 2 shows that there were clear differences in the background of adult social classes. Non-manual background was more than twice as common in non-manual classes than in manual classes, while there was only a minor difference between the lower non-manual class and 'others'. In manual classes, more than half of the men had manual class parents. For men in upper non-manual and farmer/employer classes, a manual background was less common. In particular, the proportion of men from an unskilled manual class background was low, 4% in the upper non-manual class and 6% among farmers and employers. Approximately 10% of the cohort had a single-parent home, this being a more common background for manual classes and 'others' and less common for upper non-manuals and farmers and employers. Two-children families and Swedish or other languages were more prevalent in non-manual classes and farmers and employers. Parental home in the capital area was most prevalent in the non-manual class. The differences within the non-manual class, that is between upper non-manuals and lower non-manuals, and within the manual class, between skilled and unskilled manuals, were relatively small.

There were huge differences between the social classes according to variables related to youth. The proportion of men with a higher education was substantially larger in the upper non-manual class (70%) than in manual classes (1%), the rate being approximately 10% for all other classes. Higher intermediate education was typical of men in the lower non-manual class, and lower intermediate education for men in the skilled manual class. Men in the unskilled manual class had more commonly only basic education than men in the skilled manual class.

There were also small differences in family formation. Having a partner or a child in 1980 was somewhat more infrequent for men in the non-manual classes than for men in any other class. Among unskilled manuals and 'others' there were clearly more men who had no partner in 1990 than in any other class.

A steady employment path was most common in the non-manual classes, while manual classes had more experience of unemployment. The proportion of the long-term unemployed was particularly high (22%) in the unskilled manual class.

The effect of controlling for factors related to parental home and youth paths on social class differences in total mortality

Table 3 describes the effect of controlling for factors related to parental home and youth on social class differences in all-cause mortality in adulthood. Although the variables related to living conditions in the parental home, i.e. parental class, family type, number of siblings, language and region of residence in 1970 were associated with mortality (Table 1 and first column in Table 3) and adult social class (Table 2), they contributed very little to social class differences in all-cause mortality. When all these variables were controlled for (model 5 in Table 3), the excess mortality of lower non-manual and both manual classes is reduced by 10%.

The four last columns in Table 3 show that unlike variables related to parental home, controlling for variables related to youth considerably attenuated the mortality differences by own social class. However, controlling for early parenthood had no effect on social class differences in mortality. Early parenthood is therefore excluded from the following analyses. By contrast, having only a basic education, having a partner in 1980 or not living with a partner in 1990, or drifting from a steady employment path due to shorter or longer unemployment or retirement, all contributed to the excess mortality of lower social classes (Table 3). Although educational path had a very strong effect, both marital and employment path had independent effects on social class differences in mortality by significantly reducing the deviance of the model.

Table 2
Distribution (%) of explanatory variables related to living conditions in the parental home and youth paths by adult social class. Men aged 30–34 in 1990

	All	Adult social class					
		Upper non-manual*	Lower non-manual*	Skilled manual*	Unskilled manual*	Farmer/ Employer*	Other*
Parental home							
Parental social class							
Upper non-manual	8.6	24.9	10.0	2.6	3.3	4.4	13.3
Lower non-manual	15.1	23.3	21.2	11.1	11.5	8.8	16.2
Skilled manual	35.5	26.8	35.8	45.0	39.4	20.2	31.8
Unskilled manual	10.3	4.1	7.9	13.8	15.7	5.7	12.9
Farmer/employer	29.2	20.1	24.2	25.9	28.6	59.9	23.7
Other	1.3	0.7	0.9	1.5	1.6	1.1	2.1
Family type							
Two parents	89.8	92.1	90.9	88.4	87.3	92.6	85.9
Single parent	10.2	7.9	9.1	11.6	12.7	7.4	14.1
Number of siblings							
Zero	9.5	10.7	10.2	8.7	9.4	8.7	10.4
One	27.1	34.2	30.0	23.8	23.9	25.5	26.7
Two or more	63.4	55.1	59.8	67.6	66.8	65.8	62.9
Language							
Finnish	95.2	93.4	94.8	96.8	95.9	93.5	96.1
Swedish/other	4.8	6.7	5.2	3.2	4.2	6.5	3.9
Region							
Western	30.1	30.8	29.6	31.9	28.3	29.2	27.2
Capital	14.8	21.1	17.5	11.9	14.8	8.3	16.9
Eastern	55.1	48.1	52.8	56.2	56.9	62.5	55.9
Youth							
Education							
Higher	15.5	70.2	9.0	0.7	0.7	6.8	12.6
Higher intermediate	20.8	22.7	54.7	6.7	8.3	17.0	25.5
Lower intermediate	40.4	3.6	22.2	67.9	48.1	46.6	24.1
Middle school	8.8	2.3	7.3	8.9	14.9	11.7	8.2
Basic	14.5	1.2	6.9	15.8	27.9	17.8	29.6
Marital path							
partner in 1990	57.2	69.0	64.1	54.8	48.4	57.4	30.4
Partner in 1980	14.7	11.1	13.6	17.1	14.6	18.4	6.6
No partner	28.1	19.9	22.2	28.0	37.0	24.2	63.0
Early parenthood							
No child in 1980	91.8	96.6	93.2	89.2	90.6	89.4	91.8
Child in 1980	8.2	3.3	6.8	10.8	9.4	10.6	8.2
Employment path							
Steady	76.2	86.7	83.8	76.1	67.0	87.2	4.6
Short unemployment	4.1	2.3	3.5	5.4	5.5	2.9	3.8
Long unemployment	12.1	5.0	7.4	14.8	21.9	6.0	20.7
Fragmental	4.9	5.8	4.1	2.3	2.9	3.2	35.3
Retired	2.6	0.2	0.6	1.4	2.6	0.7	35.5
All	100	100	100	100	100	100	100
Number of persons	183,354	31,483	34,110	55,308	31,747	22,974	7732

*Because of a large study population all row-wise differences of more than one percentage points are statistically significant at the 1% level.

Table 3
Relative all-cause mortality rates in 1991–98 for adult social class, living conditions in the parental home and youth paths obtained from various regression models. Men aged 30–34 in 1990

Model	(1) Age adjusted	(2) Age + soc + pc[a]	(3) 2 + ft	(4) 3 + nc	(5) 4 + la + reg	(6) 5 + edu	(7) 6 + mar	(8) 7 + ep	(9) 8 + ec
Social class (soc)									
Upper non-manual	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Lower non-manual	1.51*	1.47*	1.46*	1.46*	1.46*	0.97	0.97	0.97	1.01
Skilled manual	2.94*	2.78*	2.75*	2.74*	2.73*	1.46*	1.39*	1.39*	1.38*
Unskilled manual	4.08*	3.86*	3.79*	3.78*	3.76*	1.93*	1.72*	1.72*	1.54*
Farmer/employer	1.92*	1.87*	1.87*	1.87*	1.88*	1.05	1.03	1.03	1.19
Other	7.72*	7.43*	7.26*	7.25*	7.18*	4.07*	2.96*	2.95*	1.41*
Parental home									
Parental class (pc)									
Upper non-manual	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Lower non-manual	1.30*	1.11	1.05	1.05	1.05	0.92	0.93	0.93	0.92
Skilled manual	1.60*	1.17	1.16	1.16	1.16	0.96	0.97	0.97	0.97
Unskilled manual	2.04*	1.34*	1.26*	1.26*	1.26*	1.01	1.00	0.99	0.95
Farmer/employer	1.44*	1.12	1.11	1.10	1.11	0.92	0.91	0.91	0.92
Other	2.50*	1.65*	1.23	1.22	1.24	1.02	1.00	1.00	0.96
Family type (ft)									
Two parents	1.00		1.00	1.00	1.00	1.00	1.00	1.00	1.00
Single parent	1.68*		1.49*	1.48*	1.47*	1.42*	1.39*	1.39*	1.31*
Number of siblings (nc)									
Zero	1.20*			1.09	1.10	1.09	1.07	1.07	1.05
One	1.00			1.00	1.00	1.00	1.00	1.00	1.00
Two or more	1.13*			1.05	1.05	1.02	1.02	1.02	0.98
Language (la)									
Finnish	1.00				1.00	1.00	1.00	1.00	1.00
Swedish/other	0.57*				0.65*	0.64*	0.65*	0.65*	0.71*
Region (reg)									
Western	1.00				1.00	1.00	1.00	1.00	1.00
Capital	1.17*				1.22*	1.17*	1.18*	1.18*	1.26*
Eastern	1.15*				1.11*	1.12*	1.12*	1.12*	1.03
Youth									
Education (edu)									
High	1.00					1.00	1.00	1.00	1.00
Higher intermediate	1.93*					1.69*	1.63*	1.63*	1.54*
Lower intermediate	3.35*					2.42*	2.31*	2.30*	1.94*
Middle school	4.04*					2.90*	2.73*	2.73*	2.30*
Basic	5.16*					3.19*	2.88*	2.87*	2.21*
Marital path (mar)									
Partner in 1990	1.00						1.00	1.00	1.00
Partner in 1980	1.56*						1.37*	1.26*	1.28*
No partner	2.91*						2.29*	2.29*	1.81*
Early parenthood (ep)									
No child in 1980	1.00							1.00	1.00
Child in 1980	1.10							1.15	1.11
Employment path (ec)									
Steady	1.00								1.00
Short unemployment	1.80*								1.69*
Long unemployment	3.59*								2.97*
Fragmental	1.31*								1.47*
Retired	7.37*								4.72*

*95% confidence interval does not include 1.00.
[a] Abbreviations for variables are given in the first column.

The effect of factors related to living conditions in the parental home and youth on adult social class differences in mortality from various causes

Controlling for living conditions in the parental home may have an effect on adult social class differences in some specific causes of death, although that effect was limited for total mortality. Table 4 shows that this effect was small on mortality differences by adult social class in all diseases and external causes. However, the excess mortality of lower non-manual and manual classes was clearly reduced, by 28% in cardiovascular diseases and by 16% in alcohol-related causes. In all, approximately half of the total effect of parental home on adult social class differences in mortality can be attributed to alcohol-related causes; the reduction in the average relative deviation was 56% smaller when alcohol-related causes were excluded from all causes of death.

Controlling for educational path explained more than half of the social class differences in total mortality. The total reduction in social class differences ranged from 69% (in suicides) to 82% (in cardiovascular diseases). In diseases the excess mortality of lower non-manual class was abolished after controlling for educational path.

Controlling for marital path had an independent effect over and above the effect of educational path on adult social class differences in mortality. In all diseases controlling for marital path led to a further reduction of 7% in the average relative deviation. The effect of marital path on the overall social class differences in mortality from external and alcohol-related causes was smaller. However, in these causes of death the excess mortality of the unskilled manual class was reduced by approximately 21% after controlling for marital path (for example, for alcohol-related causes: 100((100–71.1)–(100–77.3))/(100–71.1) = 21%).

The further control for employment path had an extremely small effect on the excess mortality of manual classes in diseases. However, for mortality from external and alcohol-related causes controlling for employment path further reduced the excess mortality of manual classes. For example, in alcohol-related causes the excess mortality of skilled manual workers was reduced by 18% and that of unskilled manual workers by 34% in comparison to the model where parental home factors, educational and marital paths were controlled for.

The factors preceding own social class explained the mortality difference between the two non-manual classes in all specific causes of death studied. Although not statistically significant, the lower non-manual class had 20% excess mortality from suicides, after all controls in suicides. Suicide was the cause of death in which a smaller proportion of social class differences was explained by the variables preceding own class than in other causes of death. The excess suicide mortality of the

skilled manual class remained at 64% and that of the unskilled manual class at 86%.

After all controls the excess mortality of manual classes in comparison to the upper non-manual class was reduced by at least 80%, but remained statistically significant in external and alcohol-related causes. Controlling for all variables substantially diminished the mutual differences between the manual classes.

Discussion

In our cohort of men in middle adulthood, variables related to parental home, i.e. social class, family type, number of siblings, language, and region of residence, were associated with mortality. However, with the exception of cardiovascular diseases and alcohol-related causes, the effect of these variables on social class differences in mortality remained modest. Youth trajectories—educational, marital, and employment path—had substantial effects on social class differences in mortality from various causes of death in adulthood. After controlling for these variables, the mortality difference was abolished between upper and lower non-manual classes in all causes of death studied. The mortality difference between the upper non-manual and manual classes was substantially reduced in external and alcohol-related causes and abolished in diseases.

Shortcomings and strengths of the data

Our mortality follow-up did not begin at birth: information on deaths before the year 1991 was not available in these data. Estimating on the basis of the information in the Official Annual Vital Statistics, approximately 6% of the men born in 1956–60 died before the 1990 census. Of these deaths, 45% were infant deaths, which have been shown to be related to conditions in the parental home (Notkola & Valkonen, 1989; Leon, Vågerö, & Olausson, 1992; Arntzten et al., 1995). At ages 5–29 there is mortality variation in external causes but mortality variation in diseases is very small by parental social class (Pensola & Valkonen, 2000). Therefore, it is likely that confining to mortality follow-up in adulthood underestimates the total effect of parental home on mortality over the whole life-course due to higher mortality of men originating from manual class, particularly in infancy. However, because our focus is to study the contribution of adjusting for living conditions in the parental home on adult social class differences in mortality in later life, selective mortality before the follow-up is unlikely to essentially bias our main results.

Another possible source of difficulty has to do with the coverage of the information collected in censuses and with the information being restricted to 1 year only. For

Table 4
Change (%) in relative mortality rates for various causes of death after successively controlling for factors related to parental home and youth in 1991–98. Men aged 30–34 in 1990. The reference group is upper non-manual class, for whom the relative mortality rate is 1.00

	Age + adult class	Change (%) in the relative rates in comparison to the first model						Full model[b]
		+ Parental class	+ Family type	+ No of siblings + region + language	+ Educational path	+ Marital path[a]	+ Employment path	
All diseases								
Lower non-manual	1.35 (1.06–1.70)	−5.8	−6.9	−7.5	−120.2	−120.2	−103.7	0.99 (0.75–1.30)
Skilled manual	2.12 (1.73–2.60)	5.3	−8.4	−7.4	−83.7	−88.3	−83.8	1.18 (0.89–1.56)
Unskilled manual	2.86 (2.32–3.53)	−4.3	−7.3	−7.6	−73.9	−82.0	−85.5	1.27 (0.96–1.69)
Average change*		(−4.9)	(−7.8)	(−7.5)	(−79.0)	(−84.7)	(−85.7)	
Cardiovascular diseases								
Lower non-manual	1.35 (0.87–2.09)	−33.0	−34.7	−37.3	−148.2	−149.0	−137.1	0.87 (0.52–1.46)
Skilled manual	2.73 (1.88–3.95)	−23.6	−26.4	−28.2	−87.0	−89.7	−87.8	1.21 (0.73–2.01)
Unskilled manual	3.37 (2.29–4.96)	−22.1	−25.1	−26.9	−80.6	−85.9	−88.1	1.28 (0.77–2.15)
Average change*		(−23.6)	(−26.4)	(−28.2)	(−82.1)	(−85.7)	(−86.3)	
External causes								
Lower non-manual	1.71 (1.28–2.11)	−9.6	−10.1	−11.8	−96.8	−97.3	−93.7	1.05 (0.79–1.39)
Skilled manual	3.95 (2.93–4.55)	−10.0	−11.3	−13.0	−74.1	−77.3	−79.4	1.61 (1.22–2.12)
Unskilled manual	5.56 (4.12–6.42)	−9.3	−10.8	−12.3	−69.3	−75.3	−81.7	1.84 (1.39–2.43)
Average change*		(−9.7)	(−11.0)	(−12.6)	(−73.7)	(−77.9)	(−81.5)	
Suicides								
Lower non-manual	1.94 (1.38–2.73)	−3.9	−4.2	−4.4	−81.0	−81.4	−78.0	1.21 (0.80–1.82)
Skilled manual	3.88 (2.87–5.23)	−4.7	−5.9	−6.6	−69.7	−72.7	−73.5	1.76 (1.18–2.63)
Unskilled manual	5.24 (3.86–7.12)	−5.3	−6.6	−7.5	−64.8	−70.3	−75.4	2.04 (1.36–3.07)
Average change*		(−4.8)	(−6.0)	(−6.8)	(−68.8)	(−72.6)	(−74.7)	
Alcohol-related causes								
Lower non-manual	1.92 (1.44–2.56)	−11.2	−11.8	−13.8	−89.1	−90.1	−88.0	1.11 (0.80–1.55)
Skilled manual	4.62 (3.60–5.93)	−13.1	−14.6	−16.9	−75.1	−78.6	−82.5	1.64 (1.19–2.25)
Unskilled manual	6.82 (5.30–8.79)	−11.9	−13.6	−15.8	−71.1	−77.3	−85.2	1.86 (1.35–2.57)
Average change*		(−12.5)	(−14.0)	(−16.2)	(−74.4)	(−78.9)	(−84.1)	
All causes								
Lower non-manual	1.51 (1.28–1.79)	−8.4	−9.2	−10.4	−105.7	−106.3	−98.6	1.01 (0.83–1.23)
Skilled manual	2.94 (2.54–3.40)	−8.2	−10.0	−10.7	−76.3	−80.0	−80.3	1.38 (1.14–1.68)
Unskilled manual	4.08 (3.51–4.73)	−7.2	−9.2	−10.3	−69.9	−76.6	−82.3	1.54 (1.27–1.88)
Average change*		(−7.8)	(−9.6)	(−10.5)	(−74.9)	(−79.6)	(−82.6)	
All causes excluding alcohol-related causes								
Lower non-manual	1.33 (1.07–1.64)	−4.0	−5.2	−4.9	−128.2	−127.9	−110.7	0.97 (0.75–1.24)
Skilled manual	2.18 (1.81–2.61)	−2.9	−5.3	−4.1	−81.6	−85.4	−78.6	1.25 (0.98–1.60)
Unskilled manual	2.83 (2.34–3.41)	−2.7	−5.2	−5.1	−72.7	−79.8	−80.4	1.36 (1.06–1.75)
Average change*		(−2.9)	(−5.2)	(−4.6)	(−176.9)	(−81.9)	(−80.3)	

[a] Early parenthood has not been controlled for due to its minor effect.
[b] Full model = Age + adult class + parental class + family type + number of siblings + region in 1970 + language + educational path + marital path + employment path.

example, although we were able to identify one-parent families, we do not know how long this situation has continued, nor do we know the cause of single parenthood in the childhood home. Earlier studies have shown that while the death of a parent had only a very small effect, the divorce of parents had a clear effect on subsequent mortality (Friedman, Tucker, & Schwartz, 1995) or other health consequences (Wadsworth et al., 2002). We were unable in our data to distinguish these two groups, but according to official statistics approximately 70% of the children in single-parent families in these age groups in 1970 were not living with two parents for some other reason than death of a parent (Central Statistical Office, 1973). Therefore it is possible that the association of mortality and family type is somewhat underestimated in our data. However, because men from divorced family backgrounds account for only 7% of the total cohort, it is unlikely that this minor underestimation significantly alters our observation that adjusting for family type has a limited effect on adult social class differences in mortality.

There are also some relevant variables on which we have no data at all. Parent's wealth and both the mother and father's education have been shown to be important factors with regard to the descendant's type of education and achievement in high school (Mheen et al., 1997; Koivusilta et al., 1995). Furthermore, it has been shown that school performance at age 14 predicts both adult social class and health in another Finnish cohort study (Pietilä & Järvelin, 1995). It seems that besides socioeconomic circumstances other characteristics in the parental home, such as family centredness, low number of rows, relationship with parents, time spent with children, raring styles, and parents' orientation to their children's education (Sweeting & West, 1995; Hertzman, Power, Matthews, & Manor, 2001; Maughan & McCarthy, 1997; Bosma, Mheen van de, & Mackenbach, 1999) may also exert an influence on trajectories in youth with later afflictions on the risk of premature death from external causes (e.g. Lundberg, 1993; Davey Smith et al., 2001). With respect to these variables, particularly important periods are around the time when a person starts school and during the critical developmental period around the age 0–3 (Montgomery, Berney, & Blane, 2000; Siltala, 2002; Scannapieco & Connell-Carrick, 2002). In this study we measured parental class at age 10–14. This may provide a more accurate reflection of the living environment in childhood than parental class at the time of birth, when parents' occupations may have been transient due to their young age (Goldthorpe, 1980). However, it is likely to be too late to provide information on the social and psychological conditions during critical periods in early childhood.

In addition, transitions in youth could be measured differently. For example, for early parenthood, our age limit of 20–24 may slightly underestimate the association of early parenthood and mortality. However, only 0.3% of the participants in the cohort were fathers at a younger age. Furthermore, in the US the pivotal age of parenthood for reporting negative health consequences for males was 23 (Mirowsky & Ross, 2002). It would also have been interesting to take into account the possible changes of marital or partnership status. In our data 10% of those men who had married young, had a new spouse and a further 10% were without a partner in 1990. These groups were not separated in our analyses because of their small size, and it is unlikely that the subsequent changes in their marital status would have had a major influence on the results presented here.

The major strength of our data is that all information is always based on a contemporary register and never inquired from a person retrospectively. Further, social class and other explanatory variables are determined on the basis of the same coding scheme in different phases of life. Thirdly, data quality is not affected by loss to follow-up and non-linkage of deaths. And fourthly, we did not have to restrict our analyses to those employed at the beginning of the mortality follow-up.

Living conditions in the parental home

All the parental home variables we were able to control for were consistently related to mortality in adulthood. Earlier they have been shown to be associated with a wide range of other health outcomes (see Introduction). The effects of parental class and number of siblings on all-cause mortality were mediated by subsequent factors (see also Pensola & Valkonen, 2002; Mare, 1990). However the elevated mortality of men from single-parent homes remained, albeit at a reduced level, when own adult social class and factors related to youth were controlled for. Similar results have been found for a cohort of Finnish women of the same age (Pensola & Martikainen, 2003).

These associations indicate that the variables chosen to represent childhood circumstances at ages 10–14 in this study are reliable. However, the effect of parental home on social class differences was minor. It is likely that only a small part of this minor contribution is attributable to the nature of the data discussed above, and that the relative strength of the associations with the parental home variables and mortality is more relevant. These associations are substantially weaker than the association between own adult social class and mortality, and therefore they can certainly not explain the whole difference. For example, although conditions in the parental home were clearly associated with mortality from cardiovascular diseases, they showed only a weak association with other diseases. Therefore the effect of parental home on adult social class differences in mortality from all diseases is minor. Furthermore, some

of the adverse exposures in the parental home have not been common enough to have any substantial effect on mortality patterns even if they were associated with both adult social class and mortality. For example, only 10% of the men came from single-parent families. Even if all these men had died during the follow-up that would not have fully explained the mortality difference between the non-manual and manual classes. (Incidentally, a single parent background was associated with elevated mortality in all the social classes.) Bearing all these facts in mind, it is evident that parental class background has only a moderate effect on social class differences in mortality (see Wagstaff, Paci, & Joshi, 2001).

The exception to this general finding is that living conditions in the parental home explained 28% of adult social class differences in cardiovascular mortality. Similar findings have been obtained for Finnish women (Pensola & Martikainen, 2003). The evidence from earlier studies suggests that cardiovascular disease is likely to develop earlier in people with poor socio-economic conditions in the parental home (Kaplan & Salonen, 1990), and these conditions have a persisting effect on morbidity (Wannamethee, Whincup, Shaper, & Walker, 1996) and mortality (Östberg, 1996; Frankel et al., 1999; Pensola & Valkonen, 2002; Hart et al., 1998) from cardiovascular disease. However, the mechanism between parental home and adult social class and cardiovascular disease is not yet fully understood. Parental class has been shown to be related to certain risky behaviours (Lynch et al., 1997; Power & Hertzman, 1997) and psychological attributes (Bosma et al., 1999) that are associated with cardiovascular disease. Although even well established health habits, such as heavy smoking and fatty diet, have not had much time to contribute to cardiovascular disease before the age of 43, there is clear evidence that coronary atherosclerosis begins at a very early age (Tuzcu, Kapadia, & Tutar, 2001; Strong, Malcom, & McMahan, 1999), and that smoking in adolescence substantially increases mortality from cardiovascular disease even at a relatively young age (McCarron, Davey Smith, Okasha, & McEwen, 2001). These habits are also associated with own class, so it is likely that parental class has an effect on these adult class mortality differences. Another intriguing possibility is that low birthweight babies have a higher risk of coronary heart disease only if they are obese as adults (Frankel et al., 1996). Parental class has been shown to be associated with birthweight (Vågerö, Koupilova, Leon, & Lithell, 1999) as well as with growth patterns in early childhood (Barker et al., 2001) and body mass index in adulthood (Blane, Hart, Davey Smith, Gillis, & Hole, 1996; Wright, Parker, Lamont, & Craft, 2001). Our data thus confirm the statement by Davey Smith and colleagues (2001) that coronary heart disease is 'a cause of death which illustrates the life-course perspective par excellence'. However, even though parental home had a clear effect on adult social class differences in cardiovascular disease, 70% of the difference could be attributed to factors subsequent to living conditions in the parental home.

To the extent that living conditions in the parental home explain adult social class differences in mortality in our cohort this effect if mediated by other factors in later life (pathways model). Living conditions in the parental home were shown to be associated with youth paths, which in turn exert an effect on adult class and mortality (Pensola & Valkonen, 2002; Pensola & Martikainen, 2003). To summarize so far, in men in their middle adulthood the effect of parental home on adult social class differences in mortality was modest with the exception of cardiovascular diseases. This effect was mainly mediated through its effect on the formation of youth paths.

Life paths in youth

Educational path

In the models we present on the effects of youth paths on adult social class differences in mortality, all characteristics of the parental home were controlled for. The effects of the youth paths we discuss in the following are thus all independent of the effects of living conditions in the parental home.

Continuous information on a person's registration in different types of educational institutions was not available for us. Therefore we used information on educational attainment. However, this information provides a good proxy for educational careers. Having only a basic education indicates that a person has not been at school after age 15 or 16, and different levels of further educational degrees encompass information on the length of educational careers. Educational qualifications obtained in youth and early adulthood were clearly associated with all causes of death studied. Mortality among people with a basic education was five times higher than among those with a higher education, with the mortality of the other educational groups falling between these groups. In our data it is not possible that the strong association observed reflects socio-economic circumstances in the parental home, as was possibly the case in a Scottish sample of older men, in which parental class and education were not simultaneously controlled for (Davey Smith, Hart, & Hole, 1998b). Completed education probably reflects other factors involved, for instance, health-related behaviours and psychosocial characteristics such as self-esteem and coping strategies, which develop in late adolescence and early adulthood and which are directly or indirectly attributable to education (Lenthe et al., 2001; Lynch et al., 1997; Koivusilta et al., 1999; West, 1997; Hendry et al., 1996). Furthermore, in a Dutch study, a considerable extent (25–31%) of educational differences in mortality were

mediated by material factors in later life (Schrijvers, Stronks, van de Mheen, & Mackenbach, 1999). Material factors such as incomes and financial problems no doubt comes into play in our study as well, though their role cannot be unambiguously estimated. For example, in our data the average income in 1990 for men with a basic education (16,800€) did not differ very much from the income of men in the next two educational categories (19,963€ and 18,631€, respectively). In our cohort education was the main path to own social class, and it had a strong effect on social class mortality differences in adult life. The mortality difference between the upper non-manual class and the lower non-manual class was mainly explained by educational path, and the differences between the upper non-manual class and manual classes were substantially reduced.

Family formation, marital path

In our data marital path was associated with mortality in adulthood. Men who had married relatively young or who did not have a partner by the age of 30 had higher mortality than men who had moved to live with a partner only in their late 20s or early 30s. Early age at marriage (less than 25) has also been shown to be associated with poor health amongst men at retirement age in Great Britain (Grundy & Holt, 2000). Demonstrating the differences in mortality between the two married groups by marital age and the elevated mortality of those without a partner, our study underscores the importance of taking into account the whole life course in analysing marital status differences in mortality and the contribution of selection and social causation to these differences (Ben-Shlomo et al., 1993).

Marital path was associated with own adult class. The proportion of men who had not married young but had a partner at age 30–34 was highest in the upper non-manual class (69%), declining steadily to the unskilled manual class (48%). Marital path explained a considerable part of the excess mortality of manual classes in various causes even after the effects of social background and educational path had been taken into account. It is possible that this effect is attributable to social causation effects, but further longitudinal evidence is needed on the psychosocial and material factors preceding and concurrent to marital transitions, and on their effects on social class and mortality.

Employment path

Mortality differences between different groups on the employment path were clear. Mortality among men on a steady employment path was low, among men whose employment path was disrupted by unemployment or other reasons it was considerably elevated. There was also a clear difference between the two unemployed groups, with mortality twice as high among the long-term unemployed as among the short-term unemployed. These findings are in line with the study where unemployment 20 years earlier had an independent effect, and number of unemployment experiences an increasing effect, on limiting long-term illness (Bartley & Plewis, 2002). In a number of studies economic activity has been related to lower mortality, and unemployment to excess mortality and other health risks (Martikainen & Valkonen, 1996; Stefansson, 1991). However, the exact magnitude and causes of these differences are still a matter of debate.

The clear pattern of mortality by employment experiences may partly be attributable to the young age of the cohort. At the beginning of the employment career success or failure may have greater effects on self-esteem, behaviours and social networks (Stefansson, 1991; Bartley, 1994). Unemployment experiences may also trigger a new chain of experiences leading to stigma and an increased likelihood of new and extended unemployment spells (Hammarström, 1994). Prolonged unemployment at a young age has been shown to be associated with health and health behaviours and social achievement (Wadsworth et al. (1999)), possibly through the accumulation of the direct risks of unemployment (Moser, Goldblatt, Fox, & Jones, 1987) and by keeping a person in insecure jobs and incomes (Bartley, 1988), thus further increasing the psychological burden of this employment path (Karasek et al., 1998). However, a considerable part of the association of employment path and mortality may be attributable to characteristics antecedent to employment experiences (e.g. Martikainen & Valkonen, 1996).

Employment path was found to have a clear effect on adult social class variation in mortality. This effect can be attributed to the distribution of different categories of employment path between adult social classes. Employment path was only slightly more disadvantageous amongst men in the lower non-manual class than amongst men in the upper non-manual class, and thus its effect on mortality difference between these classes was minor. Much of the elevated mortality in the unskilled manual class is due to the over-representation of the long-term unemployed in that class. In our study experiences of unemployment were recorded at a time of low jobless rates (between 2.8% in 1990 and 7.2% in 1980), when structural reasons for unemployment were weaker. Therefore, the unemployed may be a more select group in these data, and the effect of employment path on social class differences in mortality may be much stronger than in the social context of a higher jobless rate.

Mortality and causes of death in young and middle adulthood

Death is a rare event among young adults: during the follow-up only 1.7% of our cohort died. However,

different health problems are likely to affect a much larger proportion of persons of the same age as our cohort (Rahkonen et al., 1997; Power & Matthews, 1997; Kuh & Wadsworth, 1993). Therefore in early and middle adulthood morbidity, and perhaps emotional health in particular (Stewart-Brown & Layte, 1997), may be more clearly affected by living conditions in the parental home. On the other hand, in line with our findings, Mheen et al. (1997) found that parental home characteristics explained only a relatively small proportion of the variation in educational differences for self-assessed health in a Dutch sample of persons aged 25–74.

Distribution of causes of death is different in our study of men aged 31–42 at death from that in older men, and may thus limit the generalizability of our results to older age groups. Most saliently, the proportion of external causes from all deaths decreases with increasing age: in our age group it was 57% and in men above 70 years approximately 4%.

An effect of living conditions in the parental home on social class differences in cardiovascular diseases was found in this study. However, the distribution of specific causes of death within cardiovascular diseases also changes with age. Statistics Finland's cause of death statistics (Statistics Finland, 1992–99) show that among men aged 31–44 at death approximately 48% of deaths due to cardiovascular disease were classified as ischaemic heart disease and 19% were cerebrovascular diseases. In the age group 50 and over these figures were fairly similar, 65% and 20% respectively. Furthermore, our results are consistent with earlier findings among older men (Hart et al., 1998; Frankel et al., 1999) and among Finnish women of the same age as men in our study cohort (Pensola & Martikainen, 2003). In addition, it is possible that with increasing age diseases, which have their origin in early life but which are not common as yet, become more prevalent. However, the contribution of early circumstances on these diseases may vary with time and place. For instance, stomach cancer has shown to be related to early circumstances in older cohorts but not in younger cohorts, possibly due to more favourable living conditions in later times (Davey Smith et al., 1998a; Maheswaran, Strachan, Dodgeon, & Best, 2002). This may indicate, taking into account that our cohort has spent its childhood in relatively good circumstances with respect to housing, nutrition, and health care, that the effect of the parental home on social class differences in mortality will not increase considerably with increasing age.

Conclusions

Our data showed that large social class differences in all-cause mortality among men aged 31–42 at death were only moderately attributable to living conditions in the parental home. However, in mortality from cardiovascular diseases and alcohol-related causes parental home accounted for about 28% and 16%, respectively, of social class differences. The effect of the living conditions in the parental home on adult social class differences in mortality was mainly mediated through its effect on the formation of youth paths (pathways model). A large proportion (about 67–77%) of adult social class differences in mortality can be accounted for by a disadvantageous educational path. Marital path had a further effect on the excess mortality of manual classes, and employment path had an effect on the elevated mortality from external and alcohol-related causes in the unskilled manual class. This latter effect is attributable to the large number of unskilled manual men who had been unemployed for at least 6 months.

In this study life experiences in youth proved to be very important in explaining social class differences in mortality in early adulthood independently of living conditions in the parental home. It is likely that these life paths in youth reflect the effects of long chains of detrimental events on social class differences in mortality. Our study has identified social processes in youth that may contribute to the development of social inequalities in mortality and that may provide important clues for interventions aimed at reducing these social inequalities.

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