Abstract

The relationship between women's reproductive histories and later all-cause mortality has been investigated in several studies, with mixed results. Some studies have also considered cause-specific mortality and some have included men, but none has done both. We analyse associations between parity and age of first birth for women and men across 11 cause-of-death groupings using Norwegian register data for complete cohorts born 1935-1968 whose mortality was observed 1980-2003 (i.e. at ages 45-68). Age, period, educational level, marital status, region of residence and population size of municipality were included as co-variates. In total, there were 63,000 deaths. Results showed that relative to parents of two children, childless men and women and those with one child had higher mortality risks for nearly all cause of death groupings. High parity (4+ children) was associated with raised male mortality from accidents and violence and higher mortality from cancer of the cervix among women. For other cause and gender groupings there was either little difference between those with two children and those of higher parities or an overall negative association between parity and mortality. Among men with the lowest level of education, however, high parity was positively associated with mortality from circulatory diseases. For all causes except female breast cancer, there was an inverse association between age at first birth and mortality risk. Similarities observed across cause groups and for women and men suggest that much of the fertility-mortality relationship is a result of selection or effects of reproductive behaviour on lifestyle. The latter may include both beneficial effects and harmful stress responses. However, physiological mechanisms are most probably important for some causes of death for women. Research on associations between parenting histories, health related behaviours, social support exchanges and reported or measured stress is needed to clarify mechanisms underlying the associations reported here.

Introduction

Family life has long been recognised as an important influence on health. Over a century ago, William Farr, the father of British social statistics, drew attention to the 'healthy estate' of marriage and Emile Durkheim, the father of French academic sociology, proposed that marriage and parenthood were bulwarks against suicide (Farr, 1858; Durkheim, 1951). Subsequent studies of links between parenthood and health are sparse in comparison with the thousands of investigations into differentials by marital status. Nevertheless, a growing body of evidence points to associations between this aspect of the life course and health and mortality in the post reproductive period, although there are inconsistencies in results from different studies (Beral, 1985; Doblhammer, 2000; Grundy & Tomassini, 2005; Hurt, Ronsmans, & Thomas, 2006; Kendig, Dykstra, van Gaalen, & Melkas, 2007; Grundy & Kravdal, 2008; Spence, 2008).

Relationships between parity or age at first birth and health or mortality may partly reflect selection effects (factors affecting both fertility and mortality). Education, for example, is associated with timing of childbearing and overall parity (Kravdal & Rindfuss, 2008) as well as with mortality (Kunst & Mackenbach, 1996). Similarly marriage and cohabitation bring large health benefits, especially for men, and are strongly associated with fertility (Murphy, Grundy, & Glaser, 1997; Koskinen, Joutsenniemi, Martelin, & Martikainen, 2007). Childhood circumstances and health status may also be relevant. A recent Norwegian study found that children born prematurely had lower fertility than their full term peers (Swamy, Østbye, & Skjærven, 2008) and other studies have shown associations between poor childhood health and lower rates of reproduction (Kiernan, 1989), but earlier childbearing among those women who do have families (Henretta et al., 2008). Causal mechanisms linking fertility histories to later health include physiological effects (among women) and more generalised biosocial consequences of childrearing for women and men. These latter include beneficial influences, such as associations between parenthood and increased social control of unhealthy behaviours, greater community participation and the availability of social support from children (Weitoft, Burström, & Rosén, 2004; Knoester & Eggebeen, 2006; Kendig et al., 2007) but also potentially harmful

effects of stress associated with parenting (Evenson & Simon, 2005). For example, the demands of raising children may lead to economic strain, pressures to increase working hours and less wealth accumulation, all of which have been associated with later life mortality (OECD, 2002; Aassve, Mazzuco, & Mencarini, 2006). Young parents, lone parents and parents of large families may be under particular pressure (D'Elio et al. 1997; Harrison, Barrow, Gask, & Creed, 1999).

The relative importance of these pathways is poorly understood, partly because most previous studies have considered all-cause mortality or only a few selected diseases or causes of death and have often been restricted to women. Analysis of associations between fertility histories and mortality from specific causes, or groups of causes, and comparisons of associations found for women and men may provide more insights. Lawlor et al.'s (2002) analysis of associations between parity and cardiovascular disease in women and men, for example, enabled them to distinguish probable lifestyle influences from biological factors related to pregnancy. Similarly Kravdal's (1996) analyses of parity and cancer incidence in Norway allowed him to distinguish cancers which were associated with parity among women but not men, suggesting physiological effects, from those in which patterns were similar for both sexes, suggesting associations with health related behaviours and other biosocial influences. The innovatory contribution of this paper is that we analyse associations between fertility (parity and age of first birth) and mortality from eleven groups of causes, for both women and men, using data on all Norwegians at ages 45-68.

Previous studies

Parity

Most research on associations between women's reproductive careers and their later health in contemporary low fertility populations indicates a U shaped relationship with higher risks of death or poor health for childless and high parity women compared with mothers of two children (Kvåle, Heuch, & Nilssen, 1994; Doblhammer, 2000). However, as pointed out in a systematic review of studies up to 2005, some have lacked adequate control for marital and socio-economic status (Hurt, et al., 2006). Subsequent studies include several which have controlled for these factors. A record linkage study of 1% of the population of England and Wales, for

example, showed raised mortality at ages over 50 among nulliparous women and mothers of five or more children (Grundy & Tomassini, 2005). However, a study based on Norwegian register data found raised mortality at ages 45-68 for women (and men) who were childless or had only one child, but no excess mortality for women with five or more children (Grundy & Kravdal, 2008). Similarly, a Finnish study of high parity mothers found no all-cause mortality disadvantage for mothers of five or more children, or even for mothers of ten or more, as higher risks from some diseases, such as ischaemic heart disease, were offset by lower mortality from others (Hinkula, Kauppila, Nayha & Pukkala, 2006). Recent studies based on US survey data also found no adverse effects of high parity once extensive controls for early and mid life circumstances were included (Henretta, 2007; Spence, 2008).

There have been fewer studies of links between paternity history and later mortality, but the Norwegian study referred to above found that childless men had higher mortality in late mid life than fathers, although differences were smaller than for women (Grundy & Kravdal, 2008). Hyponnen, Smith, Shepherd, & Power (2005) also reported below average mortality among the fathers (and mothers) of members of a British birth cohort study. Other studies have suggested that number of children, or receipt of social support from children, may be more important for the health of men, particularly those of lower education, than for women (Antonucci, Arjouch, & Janevic, 2003; Buber & Englehardt, 2008). However, a large study of men in long-term first marriages in England and Wales found no mortality or health disadvantage for childless older men, although fathers of four or more children had higher mortality and worse health than fathers of two (Grundy & Tomassini, 2006); another British study also reported higher rates of disability among fathers of large families (Grundy & Holt, 2000).

Age at childbearing

Analyses of associations between age at childbearing and later health consistently show disadvantages for women, and in a few studies men, who embark upon parenthood at an early age (Doblhammer, 2000; Grundy & Holt, 2000; Mirowsky & Ross, 2002; Grundy & Tomassini, 2005; Grundy & Kravdal, 2008; Henretta, Grundy, Okell, & Wadsworth, 2008; Spence, 2008). These may reflect consequences of early parenthood, such as disruption of educational and occupational attainment (Sigle-

Rushton, 2005) and adverse childhood circumstances, propensities for risk taking and other factors associated both with early entry to parenthood and poor health (Hills, Anda, Dube, Felitti, Marchbanks, & Marks, 2004; Sigle-Rushton, 2005; Henretta et al., 2008; Schmidt, 2008).

Associations between late parenthood and subsequent health or all-cause mortality are less clear. Some studies suggest advantages for those having children relatively late in life (Perls, Alpert, & Fretts, 1997; Yi & Vaupel, 2004; Grundy & Tomassini, 2005; Grundy & Kravdal, 2008), but others the reverse (Cooper, Baird, Weinberg, Ephross, & Sandler, 2000; Alonso, 2002; Spence, 2008).

Cause-specific analyses

Cause-specific analyses have predominantly been motivated by an interest in physiological consequences of pregnancy, childbirth and lactation among women although some studies have examined effects of social support from children or interpreted parity effects as reflecting such support. Numerous studies have shown inverse associations between women's parity and the incidence of cancers of the breast, ovary and uterus (Rieck & Fiander, 2006; Russo & Russo 2007; Salehi, Dunfield, Philips, Krewski, & Vanderhyden, 2008) and a positive association between late age at first birth and breast cancer (Harvard Report, 1996). These associations are assumed to largely reflect hormonal and other physiological changes triggered by pregnancy or lactation, which may also affect risks of developing other cancers (Harvard Reports 1996; Kabat, Miller, & Rohan, 2007).

Pregnancy is a state of relative insulin resistance and it has been suggested that repeated pregnancies may therefore result in permanent deficiencies in lipid and glucose metabolism and degenerative changes in arterial walls (Fletcher, Gulanick, & Lamendola, 2002). Consistent with this, studies have reported associations between higher parity and either prevalence of or mortality from obesity, diabetes or cardiovascular disease (Hinkula et al., 2006; Henretta, 2007). Lawlor et al.'s (2002) study of British women aged 60-79, for example, found a positive relationship between number of children and BMI, waist-hip ratio, adverse lipids, and diabetes, and a J shaped relationship between parity and cardiovascular disease with lowest risks among those with 2 children. Associations between higher parity and obesity

were also found for men suggesting a role for lifestyle factors. However, in a recent US study the investigators found associations between parity and adiposity, glucose levels, Framingham risk score, and carotid atherosclerosis for women but not men and concluded that childbearing, rather than childrearing, increased risks (Skilton, Sérusclat, Begg, Franzcog, Moulin, & Bonnet, 2009).

Some research suggests benefits of availability of social support from children on incidence of or survival from cancers and circulatory diseases. Results from the Copenhagen Heart Study, for example, showed that parents, particularly fathers, with less than monthly contact with a child had higher rates of mortality and a higher incidence of heart disease than other parents (Barefoot, Grønbæk, Jensen, Schnohr, & Prescott, 2005). Social support from children has also been hypothesised to contribute to the longer survival from certain cancers among parents found in several studies (Salvesen 1998; Egan 1999; Skuladottir & Olsson, 1996; Kravdal 2003), but not all (Jacobsen, Vollset, & Kvåle, 1995; Nagle, Bain, Green, & Webb, 2007).

In summary, mechanisms underlying fertility-mortality associations may encompass both selection effects and causal influences, possibly operating differentially for women and men and for those of different socio-economic groups. In some cases mechanisms may be offsetting. For example, high parity among women is associated with higher risks of obesity and possibly with specific cardiovascular related effects but also with potentially more social support and social control of health related behaviours. This complexity may explain the divergent results from previous studies and emphasises the need to consider a range of cause groups and both women and men as we do here.

Aims and hypotheses

In this paper we analyse associations between parity and age at first birth and mortality from 11 cause-specific groupings. These include causes which may be related to physiological changes associated with pregnancy and lactation, such as cancers of the breast, ovary and uterus among women; causes for which health related behaviours such as smoking and alcohol consumption play a particularly important role (lung cancer, respiratory diseases, alcohol related deaths) and causes associated both with health related behaviours and with exposures to stress and the availability of

social support, such as deaths from circulatory diseases and accidents and violence. Although we are unable to explicitly identify causal pathways, the aim of the analysis is to elucidate the likely relative importance of particular mechanisms through the first detailed comparisons of effects across a large number of cause groups for women and men. On the basis of the previous literature and taking into account the fact that some effects may be offsetting, we formulated the following hypotheses about associations between cause-specific mortality and completed fertility (given age at first birth and other control variables mentioned below) and age at first birth (given completed fertility and other control variables):

1) *Nulliparity and low parity* (one child) would be associated in both women and men with excess mortality from causes of death related to early poor health and health-related behaviours (i.e. a selection effect) and causes related to a lack of social control of health behaviours and a lack of social support. These include all cause groups we consider in this study but particularly deaths from alcohol related diseases; lung cancer; accidents and violence; and circulatory and respiratory diseases. For physiological reasons, nulliparity and low parity would also be associated with higher risks of female mortality from cancers of the breast, ovary and uterus.

2) *High parity (4+ children)* might have adverse effects arising from stress, socio-economic disadvantages and lifestyles, perhaps especially among those with low education, offsetting or even outweighing social benefits of parenthood. If so we would expect to see raised mortality risks especially from circulatory diseases and accidents and violence, for both women and men. For women there may be additional physiological reasons further increasing risks of circulatory disease mortality but reducing risks of breast, ovarian and uterine cancer.

3) *Early age at first birth* would be associated in both women and men with excess mortality from causes of death related to childhood disadvantage and propensities for risk taking (selection effect). This would be evident in raised mortality from all causes, particularly deaths from alcohol related diseases; lung cancer; accidents and violence; circulatory and respiratory diseases and, among women, cancer of the cervix. Early parenthood may also lead to additional stress and disruption of educational and occupational careers; effects we would expect to be relevant

especially for circulatory diseases. Among women, however, risks of breast cancer would be reduced.

Data and methods

Data

We investigate these hypotheses using data on all Norwegian women and men born 1935-1958 whose mortality was observed from age 45 (1980 or later) to 2003 (age 68 or lower). The study is based on data from the Norwegian Central Population Register which was established drawing on the 1960 Census and subsequently has been continuously updated. All Norwegian residents are assigned a personal identification number used in dealings with all official and many commercial agencies. Other registers based on the same identification number include registers of level of education, and of cause of death (Longva, Thomsen, & Severeide, 1998). Data compiled through linkages between these registers have been widely used in epidemiological and demographic research (Kvåle at al., 1994; Kravdal & Rindfuss, 2008).

For cohorts born 1935-1958 almost complete maternity and paternity histories can be assembled as parents' identification numbers have been recorded at registration of births since 1965, when those in this study were aged 7-30, and earlier births to the oldest members can be captured through linkage of parent-child information from the 1970 and 1960 censuses undertaken by Statistics Norway; further details have been reported elsewhere (Grundy & Kravdal, 2008).

The analysis was restricted to ages above 45 when women had largely completed their childbearing, and below 68, the age of the oldest cohort at the end of follow-up in 2003. In the period considered, fewer than 5% of men and 3% of women died before age 45 so these survivors constitute the vast majority of their respective birth cohorts (Statistics Norway, 2009a). 785,317 men contributed 40,068 deaths during the 7.36 million person-years of follow-up, and 744,784 women contributed 23,241 deaths and 7.20 million person-years of follow-up.

Completed fertility declined across these cohorts, from about 2.4 to 2.0, and average age at first birth increased (Statistics Norway 2009b). Period life expectancy at birth increased steadily over the study period (Statistics Norway 2009c). We therefore control for calendar year in all models. Better-educated women in Norway, as elsewhere, have fewer children than the less well educated although the pattern

among men is less clear (Kravdal & Rindfuss, 2008); the better educated also have lower mortality. Further, marital status is associated with differentials in both fertility and mortality, as previously discussed. Additionally in Norway there is some geographic variation in fertility and mortality (Statistics Norway, 2009 c;d). We therefore include controls for current education, marital status, region of residence and the logarithm of the population size of the municipality (as a proxy for degree of urbanization). Even with all these variables, there are remaining confounders related to attitudes, lifestyle and possibly economic situation on which we lack information and we are unable to identify specific pathways from reproductive history to later mortality.

Variables and modelling strategy

Outcome variable: cause of death groupings

We wanted to identify causes of death related to lifestyle factors, including alcohol consumption, and those, such as female cancers, for which there is substantial prior evidence on associations with fertility patterns, but restrict the number of cause groupings investigated to a manageable number. We therefore used a Finnish classification developed to allow identification of alcohol related deaths (Statistics Finland, 2005) and considered 11 cause of death groupings. These were (number of deaths shown in parentheses): breast cancer (2956); ovarian cancer (825); cervical cancer (558); cancer of the uterus (282); lung and respiratory cancers (2113 and 2964 among women and men respectively); other cancers (6416 and 9422); circulatory diseases (3605 and 12640); deaths from accidents and violence (1513 and 4558); other respiratory diseases (1064 and 1230); alcohol-related deaths (742 and 2572); and other causes (2860 and 5435). Those with missing cause of death (307 and 1247) are not shown separately but are included in totals.

Co-variates

Calendar year and age were included as continuous control variables in all models. A five fold classification of educational level (in the year of observation) distinguished those with compulsory (10 years of schooling); lower secondary (11-12 years); higher secondary (13 years); higher (14-17 years); and postgraduate education. Current marital status distinguished four groups; the never-married, married, divorced, and

widowed. The data do not allow identification of those in non-marital cohabiting unions but in these cohorts and age groups rates of cohabitation were low (Statistics Norway, 2009e). Categorisation of age group at first birth was based on the distribution of these ages for women and men respectively. Additional variables were region of residence (standard grouping of the 19 counties into 5 regions) and log of population size of municipality.

Analysis and models presented

Discrete time hazard models were estimated using standard procedures (Allison, 1984). After excluding periods relating to temporary absences abroad, sex-specific logistic models were estimated using the Proc Logistic procedure in the SAS software suite. To illustrate the importance of controlling for education (our best indicator of childhood and early life influences) we present some models including only age (in single years) and year (Model 1). We also show results from models including education but not the marital status or geographic variables (Model 2) since the direction of causality is particularly ambiguous in the case of the latter. For example, having a child may lead couples to marry or act as a deterrent to divorce and people may move to or from areas considered particularly suitable or unsuitable for childrearing. In models restricted to parous men and women, we also controlled for age at first birth (Model 4). For simplicity, only two types of models were estimated in the analysis of relationships between age at first birth and mortality: Model 1a, which included age, year and parity and Model 4 (controlling for all co-variates). As we hypothesised that high parity might have more negative consequences for those in less advantaged circumstances, we undertook further analyses stratified by level of education, but only refer briefly to these results here.

Table 1 shows the distribution of the study population by parity and age at first birth and co-variates included in the analysis. Women with lower levels of education were over-represented in high parity groups and those with an early age at first birth. Among men patterns were slightly different and those with the lowest level of education were most strongly represented among the childless.

Results

Parity and mortality from cancers of the breast, ovary and uterus

Associations between parity and female cancers, shown in Table 2, were consistent with results from many previous studies. Breast cancer mortality was negatively associated with parity being lowest among those with 4+ children and highest among the childless. Adding educational status to models of mortality from breast cancer (and the other female cancers considered in Table 2) (Model 2) made essentially no difference to results. However, when marital status and (less importantly) the geographic variables were included (Model 3) the odds ratio for nulliparous women, although raised, ceased to be statistically significant. Among the parous, the excess mortality of women of parity one was no longer significant at the 5% level when age at first birth was controlled (Model 4), although mortality risks for mothers of three or more children were lower than for mothers of one. Mortality from cancer of the uterus was raised among nulliparous women and although this effect was reduced in Model 3, it remained significant. Mortality from ovarian cancer was raised among nulliparous and low parity women. Control for age at first birth did not change this pattern (the association between mortality from cancer of the uterus and parity one was no longer significant at the 5% level, but very close)

Parity and deaths from other causes

Tables 3 and 4 present for women and men respectively mortality by parity for cause groups other than the female cancers already considered. Nulliparous men and women, and those of parity one, had raised mortality from all the cause groups examined. Generally, controls for education had little effects on estimates, especially for women; control for marital status and the two geographic variables reduced the effects of nulliparity (more strongly for some causes than others and more strongly for men than for women), but did not have a strong impact, except perhaps in the case of deaths from respiratory and circulatory diseases among nulliparous men. When these variables were included (Model 3), the largest positive associations with low parity were seen for alcohol related and 'other causes' for both men and women; for mortality from cervical cancer, accidents and violence and circulatory diseases for women; and for respiratory disease mortality in men. In models excluding the nulliparous, additional control for age at first birth (Model 4) had little effect on the excess mortality of parity-one parents.

Including controls for education had more noticeable effects on estimated associations between high parity and mortality, particularly for lung cancer and respiratory

diseases, while additional control for marital status and the geographic variables had less effects on estimates. When these factors were controlled (Model 3), high parity in women was positively associated with mortality from cancer of the cervix and negatively with mortality from alcohol related diseases and 'other diseases'. Results from Model 4, additionally controlling for age at first birth among the parous, also showed a negative association between high parity and female mortality from accidents and violence. High parity among men, however, was positively associated with mortality from accidents and violence and circulatory diseases (Model 3). When age at first birth was controlled, a negative association with alcohol-related mortality was found and the positive association between high parity and circulatory disease mortality was no longer significant. However, in analyses stratified by level of education (not shown but available on request) we found a significant positive association between high parity and circulatory disease mortality for men with only compulsory education (OR 1.19; 95% CI 1.08-1.30 P<0.001), but not the better educated, with indications of a similar association for women in this educational group. Similarly, the association between high parity and mortality from accidents and violence among men was positive only among those with lower levels of education, while the association between high parity and alcohol-related deaths was negative only for men at the highest educational levels.

Age at first birth

Tables 5 and 6 present results from analyses for parous women and men by age at first birth. In the fully adjusted model (Model 4) there was excess mortality among those who became parents at a young age (<20 for women, <23 for men) for all causes except the female cancers. This was particularly marked for mortality related to smoking and alcohol (lung cancer, alcohol related diseases, respiratory diseases). Among women later ages at first birth were positively associated with breast cancer mortality but mothers who were aged 30 or more at their first birth had reduced risks of death from all other cause groups except cervical cancer (effect only significant at the 10% level); 'other cancers'; and deaths from accidents and violence. Men who first became fathers at ages 35 or over had lower mortality than those becoming fathers at ages 23-28 for four of the seven cause groupings; the reduced risk was particularly sharp for alcohol related mortality. Comparing results from this model with Model 1a showed that controlling for the socio-demographic variables generally

weakened associations between age at first birth and mortality, most noticeably for cervical cancer mortality. However, inclusion of the socio-demographic variables had no noticeable effect on estimates for mortality from cancers of the breast, ovary and uterus.

Discussion

Results from this analysis of associations between the fertility histories of Norwegian women and men and their late mid-life mortality from 11 cause groups showed the expected negative association between parity and mortality from cancers of the breast, uterus and ovary and positive associations between nulliparity and low parity for all other cause groups. Excess mortality among the childless and those of low parity was particularly marked for alcohol related mortality (also inversely associated with *high* parity), accidents and violence among women (also inversely associated with *high* parity), respiratory diseases among men, and circulatory diseases and cancer of the cervix among women.

High parity (four or more children) was associated in women with excess mortality from cancer of the cervix and among men with higher odds of death from accidents and violence and circulatory disease mortality. When age at first birth (generally earlier among those of high parities) was controlled in models for the parous this latter effect was no longer statistically significant.

Mortality from all cause groups except the female cancers was positively associated with early childbearing and excess mortality from diseases associated with health related behaviours, notably lung cancer, alcohol related diseases and respiratory diseases, was particularly marked. Late age at first parenthood was associated with reduced mortality risks for most causes except breast cancer.

These results lend support to our first hypothesis about a positive association between nulliparity and low parity and causes of death related to poor health behaviours (reflecting both selection and lack of social control) and lower levels of social support. Relationships between reproductive factors and mortality from uterine and ovarian cancer were also as expected and accord with ideas about pregnancy-induced changes in sex hormones affecting incidence and generally poorer cancer survival among the childless for social support reasons. Controlling for education made little difference to these estimates or to any of the estimates for mortality from cancers of the breast, ovary and uterus. However the modification of results when marital status

was controlled suggests that social support from spouses may play a role in survival from breast cancer (Kravdal 2003), some other causes for women and most non-cancer causes among men and may to some extent compensate for lack of potential social support from children (though there are also other benefits of having a spouse and selection may be involved as well). Such compensation was further suggested by investigation of the surprising absence of excess breast cancer mortality among the nulliparous, which showed that risks of death from this cause were significantly raised among unmarried, but not married, childless women. More detailed examination of interactions by marital status is beyond the scope of this paper but merits further attention.

Including education had quite large effects on estimates of associations between high parity and mortality, which illustrates the importance of socio-economic selection. Controlling for age at first birth also had a large impact. With these and the three other control variables included in the models, we found a positive association between high parity and cervical cancer mortality among women, which may reflect an association between higher parity and multiple partnerships. There was limited support for the hypothesis that stress and adverse physiological effects of high parity might outbalance protection effects. Stratified analyses, however, provided some support for the idea that stresses associated with high parity might have adverse effects on the socio-economically disadvantaged. This also requires further investigation. The association between high parity and mortality from accidents and violence was positive for men but negative for women. Possibly high-parity men may have been less likely than equivalent women to have lived with their children for significant periods, thus weakening the social control and social support effects of parenthood. This interpretation is consistent with results from a Swedish study of younger men aged 29-54 which showed that men living with a partner and children had the lowest mortality and that non custodial fathers without a partner had higher mortality than either lone fathers or partnered childless men (Weitoft et al., 2004).

The relationships between age at first birth and cause-specific mortality accord well with ideas about physiological mechanisms, selection with respect to childhood conditions and personality traits, and socioeconomic stresses (beyond disadvantages related to lower educational achievements, which are controlled for

This is the first study of associations between fertility and mortality that considers all main causes of death as well as both sexes. We used a whole population data source

with low levels of missing or misreported data. However, the study has some limitations. Only women and men younger than 68 could be included and possibly there are longer term implications of fertility histories for health that are only apparent at ages older than these when, for example, social support from children may be more important. Moreover, although we controlled for education, marital status and characteristics of location, we lacked explicit information on early life circumstances, on history of co-residence with children, and on attitudes and values that may be associated with both fertility and health. We also had no information on health related behaviours or social support which we hypothesised to be important intermediate variables.

Our results show that there are strong relationships between reproductive factors and relative odds of death from a number of different causes. For most causes relevant to both women and men other than accidents and violence, there is great similarity across sexes. Much is also common across causes. This pattern is consistent with the idea that number of children and age at first birth are linked with lifestyle factors (health behaviours), with causality running both ways. In addition, physiological effects may be involved for some causes of death for women. The fact that control for education has so little impact on estimates for cancers of the breast, ovary and uterus may suggest that, for these causes, the physiological factors are relatively strongly involved. A higher level of stress among those in high-parity families may contribute for some causes for both sexes, particularly for those of disadvantaged socio-economic status.

Previous research has shown some differences between Norway and other contemporary developed countries in the pattern of associations between fertility and all-cause mortality (Grundy & Kravdal, 2008) with negative rather than positive associations between high parity and mortality among Norwegian women, in contrast to results for other countries. It has been suggested that this may be because high levels of state support for parents in Norway may offset stresses attendant on larger family sizes to a greater extent than in other countries. Different selection into higher parities, possibly differing cultural valuation of children and the generally greater involvement of men in childrearing in the Nordic countries may also be relevant. Analyses of data from other populations would help to show the extent to which the patterns we have seen are common or particular to Norway. More research on associations between parenting histories, health related behaviours, social support

exchanges and reported or measured stress is also needed to further clarify mechanisms underlying the associations reported here.

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Table 1. Distributions and means (across one-year observations) of variables used in the analysis, by sex, parity and (for the parous) age at first birth.

	Parity						***Age at first birth***				
Women	*0*	*1*	*2*	*3*	*4+*	*All*	*<20*	*20-24*	*25-29*	*30+*	*All parous*
Education (years):											
Missing	5.2	2.1	1.2	1.1	1.9	1.8	1.0	1.0	1.8	2.8	1.4
10	30.2	29.3	25.7	30.5	42.8	30.3	52.0	32.8	19.0	17.8	30.3
11-12	37.0	41.3	43.9	43.0	39.6	42.0	38.3	46.4	39.8	34.6	42.7
13	7.7	8.6	8.5	7.0	4.6	7.5	5.2	7.3	8.5	8.7	7.5
14-17	21.2	17.9	19.6	17.7	12.0	18.0	4.4	12.2	29.1	32.7	17.6
18+	3.8	2.9	2.3	1.7	1.1	2.2	0.2	0.8	3.5	6.2	2.0
Marital status											
Never-married	40.6	10.1	1.2	0.7	0.7	6.5	1.6	1.4	2.0	6.5	2.1
Married	43.3	61.4	77.2	79.4	78.4	72.3	68.2	76.1	79.6	76.4	76.0
Widowed	4.1	5.5	4.6	5.2	7.1	5.2	6.7	5.7	4.4	4.0	5.3
Divorced	12.0	22.9	17.0	14.7	13.8	16.1	23.4	16.8	14.0	13.0	16.6
Region											
East	55.3	61.7	57.1	44.7	34.5	51.0	48.3	47.4	54.9	57.1	50.5
South	5.5	4.4	4.9	6.5	7.3	5.7	5.1	5.8	5.9	5.3	5.7
West	22.5	17.1	20.8	28.3	32.8	24.2	22.6	25.7	23.6	22.5	24.4
Central	7.3	7.6	8.5	9.6	9.9	8.7	10.2	9.4	7.8	7.4	8.9
North	9.5	9.1	8.7	11.0	15.6	10.4	13.8	11.6	7.8	7.6	10.5
Age (Mean)	52.0	51.7	51.7	52.2	53.0	52.0	51.7	52.2	52.1	51.7	52.0
Year-1900 *(Mean)*	95.8	96.1	96.2	95.6	94.8	95.8	96.1	95.7	95.8	96.2	95.8
Log of population size (Mean)	10.1	10.0	9.9	9.6	9.2	9.7	9.5	9.6	9.9	10.0	9.7
Men	*0*	*1*	*2*	*3*	*4+*	*All*	*<23*	*23-28*	*29-34*	*35+*	*All parous*
Years of Education											
Missing	5.4	2.2	1.2	1.1	2.1	2.1	0.9	1.1	1.9	3.2	1.4
10	36.3	27.1	21.2	23.6	31.6	26.1	32.5	23.2	19.8	21.9	24.2
11-12	28.9	29.3	29.7	30.1	31.6	29.8	34.3	30.7	26.2	25.3	30.0
13	14.6	18.6	20.1	18.4	15.5	18.1	19.9	19.2	17.6	16.2	18.7
14-17	14.3	18.0	20.1	18.6	14.3	17.8	11.0	18.7	23.1	23.2	18.5
18+	5.9	7.1	9.0	9.2	7.0	8.1	2.2	8.2	13.3	13.4	8.5
Marital status											
Never-married	57.2	9.7	1.7	0.8	0.5	11.1	0.8	1.1	3.3	11.5	2.3
Married	31.9	66.1	81.7	83.9	82.5	72.6	76.6	82.1	81.7	75.0	80.4

Widowed	0.9	1.7	1.3	1.3	1.3	1.3	1.6	1.4	1.2	0.9	1.4
Divorced	10.1	22.5	15.3	14.0	15.7	15.0	20.9	15.5	13.8	12.5	15.9
Region											
East	49.8	59.0	55.5	43.8	36.8	49.8	49.0	48.4	52.9	52.9	49.8
South	5.3	4.5	5.0	6.5	7.2	5.6	4.9	6.0	5.8	5.7	5.7
West	24.1	17.6	21.5	29.4	32.5	24.8	24.8	25.7	23.7	23.2	24.9
Central	8.0	8.0	8.7	9.4	9.3	8.8	9.7	9.1	8.1	7.8	8.9
North	12.8	10.8	9.3	10.9	14.2	11.0	11.6	10.9	9.5	10.4	10.7
Age (Mean)	51.7	51.5	51.7	52.1	52.7	51.9	51.6	52.1	52.0	51.7	52.0
Year-1900 (Mean)	95.8	96.1	96.0	95.6	95.1	95.8	96.1	95.6	95.8	96.4	95.8
Log of population size (Mean)	9.8	9.9	9.8	9.5	9.4	9.7	9.6	9.6	9.8	9.8	9.7

Table 2. Associations between parity and mortality from female cancers 1980-2003 (Odds Ratios, 95% confidence intervals), Norwegian women aged 45-68.

Cause of death	Model	Parity				
		0	1	2	3	4+
Breast cancer	**1**	1.23***(1.10-1.37)	1.18**(1.06-1.32)	1.00	0.80***(0.73-0.89)	0.72***(0.63-0.81)
	2	1.22***(1.09-1.37)	1.18** (1.06-1.33)	1.00	0.81***(0.73-0.89)	0.72***(0.64-0.82)
	3	1.09 (0.96-1.24)	1.16* (1.03-1.30)	1.00	0.81***(0.74-0.90)	0.74***(0.65-0.84)
	4		1.11 (0.98-1.25)	1.00	0.85** (0.77-0.94)	0.79***(0.70-0.90)
Cancer of the uterus	**1**	2.27***(1.63-3.17)	1.36 (0.92-2.02)	1.00	1.07 (0.77-1.47)	0.91 (0.61-1.36)
	2	2.31***(1.66-3.23)	1.35 (0.91-2.01)	1.00	1.06 (0.77-1.46)	0.88 (0.59-1.31)
	3	1.90** (1.28-2.80)	1.30 (0.87-1.93)	1.00	1.07 (0.77-1.48)	0.89 (0.59-1.33)
	4		1.50 (0.99-2.27)	1.00	0.99 (0.72-1.37)	0.79 (0.52-1.20)
Ovarian cancer	**1**	1.67***(1.36-2.05)	1.42***(1.15-1.76)	1.00	1.02 (0.85-1.23)	0.84 (0.66-1.06)
	2	1.69***(1.38-2.08)	1.42** (1.14-1.76)	1.00	1.02 (0.85-1.22)	0.82 (0.64-1.04)
	3	1.56***(1.23-1.97)	1.37** (1.10-1.71)	1.00	1.03 (0.86-1.24)	0.84 (0.66-1.08)
	4		1.48***(1.18-1.85)	1.00	1.00 (0.83-1.21)	0.81 (0.63-1.04)

1: Controlling for age and year; **2**: Controlling for age, year, and level of education; **3**: Controlling for age, year, level of education, region of residence, logarithm of population size of municipality and marital status; **4**: Parous only; controlling for age, year, level of education, region of residence, logarithm of population size of municipality, marital status and age at first birth

* p<0.05; ** p< 0.01; *** p< 0.001

Table 3. Associations between parity and mortality from selected cause groups (Odds Ratios, 95% confidence intervals), Norwegian women aged 45-68.

Cause of death	Model	Parity							
		0	1	2	3		4+		
Cervical cancer	**1**	1.86***(1.43-2.42)	1.95***(1.51-2.53)	1.00	1.23	(0.98-1.55)	1.51**	(1.16-1.96)	
	2	1.84***(1.41-2.40)	1.89***(1.46-2.45)	1.00	1.20	(0.95-1.51)	1.35*	(1.04-1.76)	
	3	1.56***(1.15-2.11)	1.67***(1.29-2.17)	1.00	1.27*	(1.01-1.61)	1.50**	(1.15-1.96)	
	4		1.71***(1.30-2.24)	1.00	1.22	(0.96-1.55)	1.40**	(1.06-1.84)	
Lung cancer	**1**	1.24**(1.07-1.43)	1.40***(1.22-1.60)	1.00	1.10	(0.98-1.23)	1.18*	(1.031.34)	
	2	1.28***(1.11-1.47)	1.36***(1.18-1.56)	1.00	1.06	(0.95-1.19)	1.04	(0.91-1.19)	
	3	1.25** (1.07-1.47)	1.29***(1.12-1.48)	1.00	1.10	(0.98-1.23)	1.10	(0.96-1.26)	
	4		1.37***(1.18-1.58)	1.00	1.01	(0.90-1.14)	0.95	(0.82-1.09)	
Other cancers	**1**	1.31***(1.21-1.41)	1.23***(1.13-1.32)	1.00	1.01	(0.94-1.07)	1.05	(0.97-1.13)	
	2	1.32***(1.22-1.43)	1.22***(1.12-1.32)	1.00	0.99	(0.94-1.07)	1.02	(0.94-1.10)	
	3	1.29***(1.18-1.41)	1.19***(1.10-1.29)	1.00	1.01	(0.94-1.08)	1.03	(0.96-1.12)	
	4		1.20***(1.11-1.31)	1.00	1.00	(0.94-1.07)	1.02	(0.94-1.10)	
Circulatory diseases	**1**	1.97***(1.79-2.17)	1.56***(1.40-1.74)	1.00	0.99	(0.90-1.09)	1.20***	(1.081.33)	
	2	1.96***(1.78-2.17)	1.51***(1.36-1.68)	1.00	0.96	(0.88-1.06)	1.07	(0.97-1.20)	
	3	1.58***(1.41-1.77)	1.37***(1.23-1.53)	1.00	0.98	(0.90-1.08)	1.09	(0.98-1.21)	
	4		1.48***(1.32-1.65)	1.00	0.94	(0.85-1.03)	1.01	(0.90-1.12)	
Respiratory diseases	**1**	2.08***(1.74-2.49)	1.56***(1.28-1.91)	1.00	1.06	(0.89-1.26)	1.31***	(1.09-1.58)	
	2	2.04***(1.70-2.45)	1.48***(1.21-1.81)	1.00	1.02	(0.86-1.20)	1.12	(0.93-1.35)	
	3	1.35** (1.09-1.68)	1.24* (1.01-1.52)	1.00	1.05	(0.88-1.25)	1.16	(0.96-1.40)	
	4		1.32** (1.07-1.64)	1.00	0.97	(0.82-1.16)	1.01	(0.83-1.23)	
Alcohol-related	**1**	1.88***(1.54-2.31)	1.89***(1.54-2.32)	1.00	0.72**	(0.58-0.89)	0.72*	(0.56-0.94)	
	2	1.90***(1.55-2.34)	1.84***(1.50-2.26)	1.00	0.70***	(0.56-0.86)	0.65**	(0.50-0.84)	
	3	1.90***(1.52-2.38)	1.57***(1.27-1.93)	1.00	0.75**	(0.61-0.93)	0.72*	(0.55-0.94)	
	4		1.76***(1.42-2.18)	1.00	0.67***	(0.54-0.83)	0.58***	(0.45-0.77)	
Accidents & violence	**1**	1.85***(1.60-2.14)	1.58***(1.35-1.84)	1.00	0.95	(0.83-1.09)	0.87	(0.73-1.04)	
	2	1.82***(1.57-2.11)	1.57***(1.34-1.83)	1.00	0.95	(0.82-1.09)	0.85	(0.71-1.02)	
	3	1.56***(1.32-1.85)	1.35***(1.16-1.58)	1.00	1.00	(0.87-1.15)	0.91	(0.76-1.09)	
	4		1.43***(1.22-1.69)	1.00	0.95	(0.82-1.09)	0.83*	(0.69-1.00)	
Other diseases	**1**	3.29***(2.99-3.64)	1.73***(1.54-1.95)	1.00	0.82***	(0.74-.92)	0.93	(0.82-.06)	
	2	3.04***(2.75-3.35)	1.66***(1.47-1.87)	1.00	0.81***	(0.72-0.90)	0.84**	(0.73-0.96)	
	3	2.16***(1.92-2.43)	1.44***(1.29-1.63)	1.00	0.82***	(0.73-0.92)	0.85*	(0.74-0.97)	
	4		1.60***(1.43-1.81)	1.00	0.79***	(0.71-0.89)	0.81**	(0.70-0.93)	
All causes	**1**	1.78***(1.71-1.85)	1.44***(1.38-1.50)	1.00	0.96*	(0.92-0.99)	1.01	(0.97-1.05)	
	2	1.74***(1.68-1.81)	1.41***(1.35-1.47)	1.00	0.94**	(0.91-0.98)	0.94**	(0.90-0.98)	
	3	1.50***(1.43-1.56)	1.31***(1.25-1.36)	1.00	0.96*	(0.93-1.00)	0.97	(0.93-1.02)	
	4		1.37***(1.31-1.43)	1.00	0.94***	(0.90-0.97)	0.92***	(0.89-0.97)	

1: Controlling for age and year; **2**: Controlling for age, year, and level of education; **3**: Controlling for age, year, level of education, region of residence, logarithm of population size of municipality and marital status; **4**: Parous only; controlling for age, year, level of education, region of residence, logarithm of population size of municipality, marital status and age at first birth

* $p<0.05$; ** $p< 0.01$; *** $p< 0.001$

Table 4. Associations between parity and mortality from selected cause groups (Odds Ratios, 95% confidence intervals), Norwegian men aged 45-68.

Cause of death	Model	Parity						
		0	1	2	3		4+	
Lung cancer	**1**	1.59***(1.45-1.75)	1.29***(1.15-1.45)	1.00	1.03	(0.94-1.13)	1.16**	(1.04-1.29)
	2	1.40***(1.28-1.54)	1.21** (1.08-1.36)	1.00	1.01	(0.92-1.11)	1.06	(0.95-1.18)
	3	1.31***(1.16-1.47)	1.14* (1.02-1-29)	1.00	1.01	(0.92-1.11)	1.04	(0.93-1.16)
	4		1.18** (1.04-1.33)	1.00	0.97	(0.88-1.06)	0.96	(0.85-1.07)
Other cancers	**1**	1.27***(1.20-1.35)	1.19***(1.11-1.27)	1.00	0.95	(0.90-1.00)	0.97	(0.91-1.04)
	2	1.21***(1.14-1.28)	1.16***(1.09-1.25)	1.00	0.95*	(0.90-1.00)	0.94	(0.88-1.01)
	3	1.11** (1.03-1.20)	1.14***(1.06-1.22)	1.00	0.96	(0.91-1.01)	0.96	(0.90-1.03)
	4		1.18***(1.10-1.27)	1.00	0.94*	(0.88-0.99)	0.92*	(0.86-0.99)
Circulatory diseases	**1**	2.00***(1.91-2.10)	1.37***(1.29-1.46)	1.00	1.03	(0.98-1.09)	1.18***	(1.12-1.26)
	2	1.80***(1.72-1.89)	1.31***(1.23-1.39)	1.00	1.02	(0.97-1.07)	1.10**	(1.04-1.17)
	3	1.29***(1.21-1.38)	1.18***(1.11-1.25)	1.00	1.04	(0.99-1.09)	1.11***	(1.05-1.18)
	4		1.23***(1.16-1.31)	1.00	1.01	(0.96-1.06)	1.04	(0.98-1.11)
Respiratory diseases	**1**	3.52***(3.04-4.09)	1.69***(1.39-2.08)	1.00	1.01	(0.84-1.20)	1.28*	(1.05-1.57)
	2	2.91***(2.50-3.38)	1.57***(1.28-1.92)	1.00	0.98	(0.82-1.18)	1.15	(0.94-1.40)
	3	1.55***(1.27-1.90)	1.29* (1.05-1.58)	1.00	1.03	(0.86-1.23)	1.20	(0.98-1.46)
	4		1.39** (1.13-1.73)	1.00	0.98	(0.82-1.17)	1.10	(0.89-1.35)
Alcohol-related	**1**	2.96***(2.68-3.28)	1.98***(1.75-2.24)	1.00	0.84**	(0.74-0.96)	1.01	(0.87-1.17)
	2	2.61***(2.36-2.89)	1.86***(1.64-2.10)	1.00	0.83**	(0.73-0.94)	0.92	(0.79-1.07)
	3	1.40***(1.22-1.59)	1.30***(1.15-1.48)	1.00	0.89	(0.78-1.01)	0.94	(0.81-1.09)
	4		1.47***(1.29-1.67)	1.00	0.81***	(0.71-0.91)	0.79**	(0.68-0.92)
Accidents & violence	**1**	2.30***(2.13-2.49)	1.45***(1.32-1.61)	1.00	1.07	(0.98-1.17)	1.35***	(1.23-1.49)
	2	2.13***(1.97-2.31)	1.40***(1.27-1.55)	1.00	1.06	(0.97-1.16)	1.28***	(1.16-1.41)
	3	1.33***(1.20-1.48)	1.16** (1.05-1.29)	1.00	1.09*	(1.00-1.19)	1.28***	(1.16-1.42)
	4		1.21***(1.09-1.35)	1.00	1.06	(0.97-1.16)	1.22***	(1.10-1.36)
Other diseases	**1**	3.10***(2.89-3.32)	1.56***(1.42-1.71)	1.00	0.91*	(0.84-0.99)	1.09	(0.99-1.21)
	2	2.73***(2.55-2.94)	1.49***(1.36-1.63)	1.00	0.91*	(0.83-0.99)	1.03	(0.93-1.13)
	3	1.64***(1.50-1.80)	1.24***(1.13-1.36)	1.00	0.95	(0.88-1.04)	1.08	(0.98-1.20)
	4		1.35***(1.23-1.50)	1.00	0.92	(0.85-1.01)	1.02	(0.93-1.13)
All causes	**1**	2.07***(2.02-2.13)	1.40***(1.36-1.44)	1.00	0.99	(0.96-1.02)	1.12***	(1.09-1.16)
	2	1.86***(1.82-1.92)	1.34***(1.30-1.39)	1.00	0.98	(0.95-1.01)	1.05***	(1.02-1.09)
	3	1.33***(1.28-1.38)	1.19***(1.15-1.23)	1.00	1.00	(0.97-1.03)	1.07***	(1.04-1.11)
	4		1.25***(1.21-1.30)	1.00	0.97*	(0.94-0.99)	1.00	(0.97-1.04)

1: Controlling for age and year; **2**: Controlling for age, year, and level of education; **3**: Controlling for age, year, level of education, region of residence, logarithm of population size of municipality and marital status; **4**: Parous only; controlling for age, year, level of education, region of residence, logarithm of population size of municipality, marital status and age at first birth.

* p<0.05; ** p< 0.01; *** p< 0.001

Table 5. Associations between age at first birth and mortality from selected cause groups (Odds Ratios, 95% confidence intervals), parous Norwegian women aged 45-68

Cause of death	Model	Age at first birth					
		<20		**20-24**	**25-29**		**30+**
Breast cancer	1a	0.93	(0.81-1.06)	1.00	1.14**	(1.04-1.26)	1.41***(1.24-1.60)
	4	0.92	(0.81-1.06)	1.00	1.14**	(1.04-1.26)	1.41***(1.23-1.60)
Cancer of the	1a	1.05	(0.71-1.56)	1.00	0.79	(0.58-1.10)	0.30***(0.15-0.60)
uterus	4	0.99	(0.67-1.48)	1.00	0.84	(0.60-1.17)	0.32** (0.16-0.64)
Ovarian cancer	1a	1.01	(0.80-1.28)	1.00	0.97	(0.81-1.15)	0.57***(0.42-0.78)
	4	0.97	(0.76-1.22)	1.00	1.01	(0.84-1.21)	0.61** (0.44-0.84)
Cervical	1a	1.31*	(1.03-1.66)	1.00	0.58***(0.45-0.75)		0.57** (0.40-0.83)
cancer	4	1.05	(0.82-1.34)	1.00	0.68** (0.52-0.88)		0.70 (0.48-1.02)
Lung cancer	1a	1.90***(1.70-2.13)		1.00	0.63***(0.56-0.72)		0.52***(0.42-0.63)
	4	1.56***(1.39-1.75)		1.00	0.78*** (0.68-0.89)		0.69***(0.56-0.84)
Other cancers	1a	1.18***(1.09-1.27)		1.00	0.94	(0.88-1.01)	0.90* (0.82-1.00)
	4	1.11** (1.03-1.20)		1.00	1.00	(0.93-1.06)	0.98 (0.89-1.08)
Circulatory	1a	1.47***(1.33-1.61)		1.00	0.67***(0.61-0.74)		0.60***(0.52-0.69)
diseases	4	1.22***(1.10-1.34)		1.00	0.81*** (0.74-0.90)		0.78***(0.67-0.90)
Respiratory	1a	1.97***(1.67-2.32)		1.00	0.58***(0.48-0.71)		0.49***(0.36-0.66)
diseases	4	1.49***(1.26-1.76)		1.00	0.77** (0.63-0.93)		0.70* (0.51-0.94)
Alcohol-related	1a	1.83***(1.50-2.23)		1.00	0.41***(0.32-0.53)		0.38***(0.27-0.53)
	4	1.43***(1.17-1.75)		1.00	0.51*** (0.40-0.65)		0.53***(0.37-0.74)
Accidents &	1a	1.46***(1.25-1.70)		1.00	0.85*	(0.74-0.98)	0.79* (0.65-0.97)
violence	4	1.32***(1.13-1.55)		1.00	0.89	(0.77-1.03)	0.87 (0.70-1.07)
Other causes	1a	1.36***(1.20-1.53)		1.00	0.80***(0.71-0.89)		0.62***(0.52-0.73)
	4	1.14*	(1.01-1.29)	1.00	0.95	(0.85-1.06)	0.78** (0.66-0.92)
All causes	1a	1.36***(1.31-1.41)		1.00	0.82***(0.79-0.85)		0.75***(0.71-0.80)
	4	1.20***(1.15-1.25)		1.00	0.92*** (0.88-0.95)		0.88***(0.83-0.93)
Number of deaths		3,317		9,710	4,361		1,716

1a: controlling for age, year and parity; 4:controlling for age, year, parity, level of education, region of residence, logarithm of population size of municipality, and marital status.

* p<0.05; ** p< 0.01; *** p< 0.001

Table 6. Associations between age at first birth and mortality from selected cause groups (Odds Ratios, 95% confidence intervals), parous Norwegian men aged 45-68.[a]

Cause of death	Model	Age at first birth			
		<23	**23-28**	**29-34**	**35+**
Lung cancer	1a	1.61***(1.47-1.77)	1.00	0.92 (0.83-1.02)	0.84* (0.71-0.99)
	4	1.41***(1.28-1.54)	1.00	0.99 (0.89-1.10)	0.89 (0.75-1.05)
Other cancers	1a	1.19***(1.12-1.26)	1.00	0.92** (0.87-0.98)	0.85** (0.78-0.94)
	4	1.14*** (1.07-1.21)	1.00	0.94* (0.89-1.00)	0.87** (0.79-0.95)
Circulatory diseases	1a	1.39***(1.32-1.47)	1.00	0.88***(0.83-0.93)	0.87** (0.80-0.95)
	4	1.23***(1.17-1.30)	1.00	0.93* (0.88-0.99)	0.93 (0.85-1.01)
Respiratory diseases	1a	1.59***(1.34-1.90)	1.00	0.91 (0.75-1.10)	0.74 (0.54-1.01)
	4	1.31** (1.10-1.57)	1.00	1.00 (0.82-1.21)	0.77 (0.56-1.06)
Alcohol-related	1a	1.94***(1.73-2.16)	1.00	0.65***(0.56-0.75)	0.42***(0.33-0.54)
	4	1.48*** (1.32-1.66)	1.00	0.72***(0.62-0.83)	0.45***(0.35-0.58)
Accidents & violence	1a	1.32***(1.21-1.44)	1.00	0.94 (0.85-1.03)	0.74***(0.63-0.86)
	4	1.15** (1.05-1.26)	1.00	1.00 (0.91-1.10)	0.77** (0.66-0.90)
Other causes	1a	1.30***(1.20-1.41)	1.00	0.87** (0.80-0.95)	0.77***(0.67-0.89)
	4	1.13** (1.04-1.23)	1.00	0.93 (0.85-1.01)	0.82** (0.71-0.95)
All causes	1a	1.38***(1.34-1.42)	1.00	0.89***(0.86-0.91)	0.81***(0.77-0.85)
	4	1.22*** (1.19-1.26)	1.00	0.94*** (0.91-0.97)	0.84*** (0.80-0.88)
Number of deaths		6,836	14,996	5,656	1,747

1a: controlling for age, year and parity; 4: controlling for age, year, parity, level of education, region of residence, logarithm of population size of municipality, and marital status.

* p<0.05; ** p< 0.01; *** p< 0.001