Reproductive history and mortality in late middle age among Norwegian men and women

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Family status has been linked with health and mortality in both theoretical and empirical studies for well over a century (1, 2). Extensive evidence shows health benefits of marriage, especially for men, and, to a lesser extent, suggests advantages of living with children, at least among those with a partner (3-8). The number of children born and the timing of births may also have implications for health in later life. It is well established that nulliparity and late childbearing are associated with higher risks of breast cancer, and high parity appears protective against some other hormonally related cancers (9-12). High parity and early childbearing are, however, associated with higher risks of diabetes and circulatory diseases, although underlying mechanisms are not clearly established (13). Apart from specific biological links between aspects of female reproductive history and particular diseases, there may be other biosocial pathways which link reproductive patterns and the later mortality of both women and men. For example, childrearing may provide an impetus to avoid health damaging behaviours and often involves greater social participation in the community (14,15). We would expect accumulated effects of such behavioural patterns to be apparent later in life. Additionally, social support from children is associated with health benefits at older ages (16, 17). Less positively, reproduction involves physical stress on mothers (18) and for both women and men childrearing, or certain patterns of childrearing, may involve increased exposures to infections; stress, with associated higher risks of depression; and substantial economic costs (19-21).

A number of studies of contemporary developed country populations have examined associations between aspects of reproductive history and later life mortality or health (22-29). Reviews of this literature have reported that, while most studies suggest later disadvantages for nulliparous and high parity women, the evidence is far from conclusive (30, 31). A few studies have also shown a disadvantage for mothers of one child only (9, 26) and a recent Finnish study of women with five or more children reported below average mortality for all cause mortality (32).

Interpretation of these studies is complicated by varying control for socio-economic and marital status and other potentially confounding factors. For example, socio-economic status is associated with differentials in the timing of fertility and total number of births, as well as with mortality, and should be controlled for (33, 34). Women (and men) who start childbearing at younger ages have higher total parities on average, so control for this is also desirable, especially as studies which have examined age at childbearing have consistently shown poorer later health and higher mortality among women with an early entry to motherhood (25, 26, 29, 35, 36). Later health disadvantages of early fatherhood have also been reported (25, 37, 38). Several studies have reported an opposite association finding lower mortality for those with one or more births at relatively old ages (26, 29, 39-42), although a few studies point in the other direction (43, 44).

Studies which include both women and men are potentially valuable in providing insight into the relative importance of social or biological factors underlying associations between reproductive history and later health (14). Most of the few such studies are of high fertility populations, several with unusual characteristics (39, 45-47). Identified studies of women and men in contemporary developed country populations are based on surveys, not always nationally representative, subject to varying degrees of non-response (24, 25, 39). Recent analysis of data on the mothers and fathers of children included in a long-term British cohort reported that these parents had lower mortality than the general population of the same age (39). Another British study of people in early old age found that women with four or more children had raised odds of poor health and disability and some suggestion of a similar effect for men (25).

In this study we use register data for a national population to analyse associations between reproductive history and mortality at ages 45-68 among both women and men. The aims of the study were to see whether parity was associated with later mortality risk, taking account of marital status and education and, among parents, also timing of maternity or paternity. Additionally we wanted to investigate associations between timing of childbearing and later mortality risk in their own right and, in a sub analysis, see whether such effects were modified by controlling for the educational level of subjects' own parents.

MATERIALS AND METHODS

Data

Norwegian data on population and vital events are combined in a Central Population Register which was established drawing on the 1960 Census. Official personal identification numbers were allocated to the whole population then in Norway and have subsequently been allocated at birth (or immigration). These numbers are required in all dealings with official agencies, including educational, health, housing, welfare and tax authorities, and many commercial ones, such as banks. A range of other registers include a register of level of education (48). Use of personal identification numbers allows linkage between these registers which have been widely used in epidemiological and demographic research (9-11, 34). In this study we include all men and women born 1935-1958 living in Norway in 1960 or at any subsequent point who therefore have a personal identification number. For these cohorts almost complete maternity and paternity histories can be assembled as parents' identification numbers have been recorded at registration of all births since 1965, when those included in this analysis were aged 7-30, and earlier births to the oldest members of the study can be captured through linkage of parent-child information from the 1960 and 1970 Censuses undertaken by Statistics Norway; further details have been reported elsewhere (49). The same method of parent-child linkage enabled identification of parental information for 81.3 percent of male and 77.1 percent of female sample members born 1950-58 (nearly all with missing data were born before 1953). The analysis was restricted to ages above 45 (i.e. no earlier than 1980), 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 (50). Socio-demographic status was controlled by including detailed information on marital status and own (and, where relevant and available, spouse's or parents') highest educational level.78,5317 men contributed 40,071 deaths during the 7.36 million person-years of follow-up, and 74,4784 women contributed 23,241 deaths and 7.20 million person-years of follow-up.

Variables and modeling strategy

Discrete time hazard models were estimated following standard procedures (51). In such models a series of observations for discrete time periods (in this case one year) are created for each person from a relevant starting point (here January the year the person turned 45) until the event of interest (in this case death) or censorship (at the end of follow-up in 2003 or through emigration) observations for all discrete time periods are then pooled. The observations include co-variates that can be time-invariant (here sex, parity, age at first and last birth) or time-varying (here age, period, marital status, educational qualifications). The outcome variable in our case is whether the person died within the year. Logistic regression is applied to the data to estimate how the co-variates are associated with the hazard of death. After excluding periods relating to temporary absences abroad, sex-specific models were estimated, firstly for all men and women and secondly for parents of at least one child. Models were also estimated for specific age and marital status groups. The Proc Logistic procedure in the SAS software was used.

Age at first birth and total parity may be influenced by childhood circumstances that also affect health. We partially control for this by fitting further models including information on the education of parents of the youngest cohorts (born 1950-58) included in the analysis whose mortality was observed between the ages of 45 and 53. After those with missing parental education had been excluded, there were 2,105 deaths and 929,385 person years of observation for men and 1,397deaths and 911,179 person years of observation for women.

Categorisation of age group at first birth was based on the distribution of these ages for women and men respectively. Calendar year and the person's age were included as continuous control variables in all models. A five fold classification of own and spouse's current level of education (i.e. in the year of observation) was derived distinguishing those with compulsory level education (10 years of schooling); lower secondary (11-12 years); higher secondary (13 years); higher (14-17 years); and postgraduate. Parental education (most recent available) was dichotomised into compulsory versus secondary or higher due to the very small proportions with advanced level qualifications.

In addition to including current marital status (never-married, currently married, divorced or separated, widowed) as a co-variate, we estimate stratified models because effects of reproductive history may be modified by marital status. The data do not allow identification of those in non-marital cohabiting unions. This could be a source of bias as the unmarried with children are probably more likely to be cohabitating than the unmarried childless. However in the cohorts and age groups we consider rates of cohabitation were low. In 1993, for example, fewer than 5% of 45-69 year olds, and approximately 15% of unmarried people of this age, were cohabiting (52).

Separate models were run for two broad age groups (45-54 and 55-68), in addition to including age in single years as a continuous co-variate. Current age, age at last birth, and current age of the youngest child are linearly dependent on each other and there is some evidence in the literature that co-residence with a child has beneficial effects on health and health related behaviours (3-8). This might confound associations between late age at maternity or paternity, one of the aspects of reproductive history we investigate, and mortality. We therefore wanted to see whether associations were similar in these two broad age groups as this potential problem would be much less relevant among those aged 55-68 than in the younger group (information on household composition was not available).

RESULTS

Characteristics of the sample are shown in Table 1. 63.4 percent of women and 60.6 percent of men had had two or three children and 11.3 percent of the former and 15.9 percent of the latter no children. Fewer than five percent had had five or more children. Among parents, mean ages at first and last birth respectively were 23.8 and 29.5 for women and 26.7 and 32.7 for men.

Table 2 shows results from modeling odds of death for women and men by number of children borne/fathered, marital status, and education. Mortality was inversely associated with years of education and was higher for the unmarried than for the married reference group, being highest among the divorced and separated. Also consistent with the literature, marital status differentials were greater among men than women. For both women and men odds of death were highest for the childless and next highest for those who had had only one child; in all sex and age groups both were significantly higher than for the reference category comprising parents of two children. Women with three or four children had lower mortality than those with two, although this difference was not statistically significant in the 45-54 year old age group. High parity (five or more children) was not significantly associated with mortality among either women or men, although among women odds ratios were below 1.

Stratified analyses were undertaken for separate marital status groups controlling for the same variables as above and for spouse's educational level for those currently married. Figure 1 shows odds ratios and 95 percent confidence intervals from these (fully adjusted) models for ever-married groups. Effects were similar in all groups. Thus childless men and women and those who had had only one child had elevated risks of mortality whether they were married, divorced/separated, or widowed. Married women with three, four, or five or more children had significantly lower mortality than those with two: formerly married men who had had five or more children also had lower mortality than fathers of two. In no case was higher parity significantly associated with higher mortality risk. Results for the never-married are not shown in the Figure because of the very different distribution by parity. Most never-married men and women were nulliparous (81.9 percent and 71.3 percent respectively) and small proportions had more than two children (2.5 percent of men, 4.2 percent of women). Model results nevertheless showed higher mortality among the childless never-married compared with never-married parents of two children (OR for women 1.59: 1.28-1.98; for men 1.99: 1.70-2.32) and gave no indication of disadvantage for parents of three or more children (odds ratios below 1).

Results from models taking into account timing of maternity or paternity and excluding the nulliparous are shown in Table 3. For mothers, results show an overall negative association between parity and mortality with mothers of five or more children having the lowest mortality (OR 0.88: 0.80, 0.96). Among fathers, the lowest mortality was observed for those with three children and the highest for those with only one. Mortality risks were raised for mothers and fathers who had had their first child in the youngest age band and tended to decrease with older age at first birth. Having had the last birth at age 40 or over was associated with reduced mortality among fathers aged 55-68 and mothers aged 45-54.

Table 4 shows results from the sub analysis of parous men and women born 1950-58 for whom information on their own parents' education was available. Neither maternal nor paternal educational level was significantly associated with the mortality of the parous women in this analysis (which included subjects' own educational level). Parous men whose mothers had higher or secondary educational qualifications had lower mortality than those whose mothers had lower levels of education but the association with paternal education was in the opposite direction with raised mortality among sons of more educated fathers. Women who had had their first child as a teenager and men who became a father before age 23 had higher odds of death than those with later ages at first birth even when these parental educational variables were controlled (OR for women: 1.16: 1.00, 1.33; for men: 1.17: 1.05, 1.29). Parents of one child only had higher odds of death than parents of two children, but there were no significant differences between those of higher parities and the reference group with two children. As the main analysis, results indicted a mortality advantage for those who had had a child at age 40 or older.

Finally, an additional series of models were run for women for mortality for all causes of death other than breast cancer. In all cases the associations reported above were still found to be statistically significant.

DISCUSSION

Results from this large population study including both women and men are consistent with those of other studies in suggesting later mortality disadvantages of childlessness and teenage childbearing among women; they additionally show a similar, although less strong, association between male mortality and childlessness or paternity before age 23 and also show consistently higher mortality among both women and men who had had only one child. Higher mortality for those who became parents at young ages was still observed when the educational level of their own parents was controlled, although this information was missing for a fifth of the sample included in this sub analysis and we lack information on other aspects of the childhood environment. Our results are also consistent with some other studies in showing an association between having a child at age 40 and above and lower mortality risk.

It must be recognised that these findings demonstrate associations rather than causality. We included detailed information on educational level in all models as it is known that education is strongly associated with both fertility and mortality but there are other potential confounders, such as religious orientation or rural residence, which may be associated both with mortality risk and with fertility which we have been unable to take account of. We also lack information on quality of relationships between parents and adult children or, importantly, on prior health status and health related behaviours. It is known that experience of disrupted and disadvantaged childhoods is associated with risky health related behaviours and poor health, as well as with young entry to parenthood (35-38) and this, rather than stresses consequent on young parenthood, could be the mechanism underlying the relationships between early parenthood and later mortality. Those with births at ages 40 and over may, conversely, be selected for good health. Age at menopause is associated with health related behaviours, health status and mortality (53-55). Although men do not experience menopause, male potency declines with age (56) and for both sexes late parenthood may be a marker of better health and slower ageing, not least because perceived health status is likely to influence decisions about mid-life parenthood. More generally, poor health may in some cases affect fecundity, otherwise restrict opportunities for parenthood, or lead parents to limit their family size (57, 58).Unlike several other studies of contemporary populations, and evolutionary theories which posit a trade off between reproduction and longevity (59), our results show no disadvantage for high parity women. On the contrary, we found significantly lower among higher parity mothers, especially married mothers (both with and without control for timing of first birth) and similar, although less marked, associations among men. Plausibly, differences in ages under observation might account for some variation between studies in associations between parity and post reproductive female mortality. Breast cancer accounts for a higher proportion of deaths among those in late middle age than at older ages when deaths from cardiovascular diseases predominate (60). As high parity is reported to be negatively associated with deaths from breast and some other cancers, but positively associated with cardiovascular disease deaths, this could mean that results would differ according to ages at follow-up. Further cause specific analyses would clarify this and provide more information on possible underlying mechanisms. However, the differentials we found were substantially unaltered when we reran analyses for causes of death other than breast cancer and our results differ quite markedly from two similar studies including women of very much the same age. One of these (26) analysed data from the England and Wales Longitudinal Study, a large nationally representative study including census and linked vital events data. For women aged 50-69 whose mortality was observed 1980-2000, a very similar age group and time period to that included in this study, models in which age, marital and socio-economic status were controlled showed excess risks for the nulliparous of a similar order, although slightly lower, to our results (OR 1.28: 1.10, 1.49). However, results also showed raised odds of death for women with five or more children (OR 1.25: 1.06, 1.46). An earlier Norwegian study (16) which analysed the mortality over a twenty year follow up period (1961-81) of women included in a breast screening programme also showed, for women aged 50 and over at entry to the study, raised mortality among nulliparous and high parity women (SMR for mothers of five or more children: 1.07: 1.03-1.12). These differences in results might reflect to some extent confounding by factors not controlled for in any of these analyses. Differences in period and cohort, and between countries, may also be relevant. It seems plausible, for example, that larger proportions of higher-order births were planned in the more recent cohorts we consider than in the earlier Norwegian study because of differences in the availability of contraception and legal abortion. There may also be some differences between our population and the England and Wales analysis in this regard. When births are planned, the argument that only the healthier have many children becomes more relevant; moreover unplanned childbearing may itself sometimes have negative consequences for psychosocial health (61). Additionally, in the contemporary Norwegian setting, the social benefits of having many children may outweigh the (physiological or socio-economic) penalties. The Nordic countries are special in having a very generous welfare system; child allowances are relatively large and during the 1980s and 1990s there were significant extensions in paid maternity (and paternity) leave, improved access to subsidised day care, and implementation of various work place reforms, all of which helped parents, particularly mothers, to combine paid work and family roles (62). It has been suggested that these supports explain why differentials in the health status of lone mothers and married mothers are lower in Norway than in Canada (63). Those in our study would have benefited from these developments to a greater extent than those in the earlier Norwegian analysis and have been advantaged in comparison to the women in England and Wales where family supports in the period considered were less generous (64). Possibly the mechanisms underlying the associations between reproductive history and later mortality are conditioned by such contextual factors implying that 'family friendly' policies may have long-term benefits for the health of parents. The similarity of our results for women and men suggests biosocial pathways underlying associations between reproductive history and health. These may include, for example, accumulated advantages of differences in health related behaviours and social participation, as suggested by studies which have found higher rates of smoking and alcohol abuse among childless and low parity individuals and higher rates of community activity among parents (8, 17, 31), and beneficial effects of support from adult children later in life (14-17). Research using datasets which include information on health related behaviours and health status throughout the lifecourse would help to clarify mechanisms underlying the associations reported here.

REFERENCES

1. Farr W. The influence of marriage on the mortality of the French people. Transactions of the National Association for the Promotion of Social Science 1858; 504-12.
2. Durkheim E. Suicide: a study in sociology. Glencoe IL: The Free Press, 1951. (Originally published, 1897).
3. Elstad JI. Inequalities in health related to women's marital, parental and employment status - a comparison between the early 1970s and the late 1980s, Norway. Soc Sci Med 1996; 42:75-89.
4. Hughes ME, Waite LJ. Health in household context: Living arrangements and health in late middle age. J Health Soc Behavior 2002; 43: 1-21.
5. Martikainen P. Women's employment, marriage, motherhood and mortality: a test of the multiple role and role accumulation hypotheses. Soc Sci Med 1995; 40: 199-212.
6. Roos E, Burström, Saastamoinen, Lahelma E. A comparative study of the patterning of women's health by family status and employment status in Finland and Sweden. Soc Sci Med 2005, 60:2443-51.
7. Ross CE, Mirowsky J, Goldsteen K. The impact of family on health: The decade in review. J Marriage Family 1990; 52:1059-78.
8. Weitoft GR, Burstrom B, Rosen M. Premature mortality among lone fathers and childless men. Soc Sci Med 2004; 59: 1449-59.

9. Kvale G, Heuch I, Nilssen S. Parity in relation to mortality and cancer incidence: a prospective study of Norwegian women, Int J Epidemiol 1994; 23**:** 691-9.
10. Albrektsen G, Heuch I, Hansen S, Kvale G. Breast cancer risk by age at birth, time since birth and time intervals between births: exploring interaction effects. Br J Cancer 2005; 92**:** 167-75.
11. Lochen ML**,** Lund E. Childbearing and mortality from cancer of the corpus uteri. Acta Obstet Gynecol Scand. 1997;76:373-7.
12. Teras LR, Patel AV, Rodriguez C et al. Parity, other reproductive factors, and risk of pancreatic cancer mortality in a large cohort of U.S. women. Cancer Causes Control. 2005;16:1035-40.
13. Lawlor DA, Emberson JR, Ebrahim S et al. Is the association between parity and coronary heart disease due to biological effects of pregnancy or adverse lifestyle risk factors associated with childrearing? Circulation 2003; 107: 1260-4.
14. Kravdal O. Relationship between childbearing and cancer incidence due to biology or lifestyle? Examples of the importance of using data on men. Int J Epidemiol 1996; 24: 477-84
15. Knoester C, Eggebeen DJ. The effects of the transition to parenthood and subsequent children on men's well-being and social participation. J Family Issues 2006; 27: 1532-60
16. Antonucci TC, Arjouch KJ, Janevic MR. The effect of social relations with children on the education-health link in men and women aged 40 and over, Soc Sci Med 2003; 56: 949-60.
17. Barefoot JC, Grønbǽk M, Jensen G et al. Social network diversity and risks of ischemic heart disease and total mortality: findings from the Copenhagen City Heart Study. Am J. Epidemiol 2005; 161: 960-7.

18. Christensen K, Gaist D, Jeune B, Vaupel J. A tooth per child? Lancet 1998; 352**:** 204.
19. Mastekaasa A. Parenthood, gender and sickness absence. Soc Sci Med 2000; 50:1827-42
20. D'Elio M, Ness R, Matthews K, Kuller L. Are life stress and social support related to parity in women? Behavioural Medicine 1997; 23:87-94.
21. Evenson RJ, Simon RW. Clarifying the relationship between parenthood and depression. J Health Soc Behav 2005; 46:341-58.
22. Beral V. Long term effects of childbearing on health. J Epidemiol and Community Health 1985; 39**:** 343-6.
23. Doblhammer G. Reproductive history and mortality later in life: a comparative study of England and Wales and Austria. Pop Studies 2000; 54**:** 169-76.
24. Friedlander N. The relation of lifetime reproduction to survivorship in women and men: A prospective study. Am. J. Human Biol 1996; 8**:** 771-83.
25. Grundy E, Holt G. Adult life experiences and health in early old age in Great Britain. Soc Sci Med 2000; 51**:** 1061-74.
26. Grundy E, Tomassini C. Fertility history and health in later life: a record linkage study in England and Wales. Soc Sci Med 2005; 61:217-28.
27. Kington R, Lillard L, Rogowski J. Reproductive history, socioeconomic status, and self-reported health status of women aged 50 years or older. Am J Public Health 1997; 87**:** 33-7.
28. Koski-Rahikkala H, Pouta K, Pietilainene K, Hartikainene AL. Does parity affect mortality among parous women? J Epidemiol Community Health 2006; 60: 968-73.
29. Lund E, Arnesen E, Borgan JK. Pattern of childbearing and mortality in married women-a national prospective study from Norway. J Epidemiol Community Health 1990; 44:237-40.
30. Hurt LS, Ronsmans C, Thomas SL. The effect of number of births on women's mortality: Systematic review of the evidence for women who have completed their childbearing. Pop Studies 2006; 60:55-71.

31. Kendig H, Dykstra P, van Gaalen RI. Health of parents and childless individuals. J Fam Issues; in press.
32. Hinkula M, Pukkala E, Kyyronen P, Kauppila A. Grand multiparity and the risk of breast cancer: population-based study in Finland. Cancer Causes Control 2001; 12:491-500.
33. Ni Brolchain M. New perspectives on fertility in Britain. Studies on Medical and Population Subjects 55. London: HMSO, 1993.
34. Kravdal Ø. The high fertility of college educated women in Norway: An artefact of the separate modelling of each parity transition. Demographic Research 2001; 5: 187-216 . Online at www.demographic-research.org/ Volumes/Vol5/6
35. Maughan B, Lindelow M. Secular change in psychosocial risks: the case of teenage motherhood. Psychological Medicine 1997; 27: 1129-44.
36. Mirowsky J, Ross CE. Depression, parenthood and age at first birth. Soc Sci Med. 2002; 54:1281-98.
37. Anda RF, Chapman DP, Felitti VJ et al. Adverse childhood experiences and risk of paternity in teen pregnancy. Obstet Gynecol 2002; 100: 37-45.
38. Sigle-Rushton W. Young fatherhood and subsequent disadvantage in the United Kingdom. J Marriage and the Family 2005; 67: 735-53.
39. Hyponnen E, Smith GD, Shepherd P, Power C. An intergenerational and lifecourse study of health and mortality risk in parents of the 1958 birth cohort: (II) mortality rates and study representativeness. Public Health 2005; 119:608-15.
40. Muller H, Chiou J, Carey J, Wang JL. Fertility and life span: Late children enhance female longevity. J Gerontol Series A-Biol Med Sci 2002; *57A*: B202-6.
41. Perls T, Alpert L, Fretts R. Middle aged mothers live longer. Nature 1997; 389: 133.
42. Yi Z, Vaupel JW. Association of late childbearing with healthy longevity among the oldest-old in China. Pop Studies 2004; 58:37-53.

43. Cooper G, Baird D, Weinberg C et al. Age at menopause and childbearing patterns in relation to mortality. Am J Epidemiol 2000; 151**:** 620-3.
44. Alonso A. Long term health consequences of delayed childbirth: NHANES III. Women's Health Issues 2002; 12**:** 37-45.
45. Hurt LS, Ronsmans C, Campbell OMR et al. Long-term effects of reproductive history on all-cause mortality among adults in rural Bangladesh. Stud Family Planning 2004; 35: 189-96.
46. McArdle PF, Pollin TI, O'Connell JR et al. Does having children extend life span? A genealogical study of parity and longevity in the Amish. J Gerontol Med Sci 2006; 61A: 190-5.
47. Smith KR, Mineau GP, Bean LL. Fertility and Post-Reproductive Longevity. Soc Biol 2002; 49: 185-205.
48. Longva S, Thomsen I, Severeide P. Reducing costs of Censuses in Norway through use of administrative records. Internat Statistical Review 1998; 2:223-4.
49. Rindfuss R, Guilkey D, Morgan SP et al. Child care availability and first-birth timing in Norway. Demography 2007; 44:
50. Statistics Norway(www.ssb.no/english/subjects/02/02/10/dode_en/)
51. Allison PD. Event history analysis: Regression for longitudinal event data. Newbury Park, CA: Sage Publications 1984.
52. Statistics Norway. Statistical Yearbook of Norway 2006. Oslo, Statistics Norway, 2006. www.ssb.no/english/subjects/02/01/20/samboer_en/)
53. McKinley SM, Bifano NL, McKinley JB. Smoking and age at menopause in women. Annals of Internal Medicine 1985;103: 350-66.
54. Jacobsen BK, Heuch I, Kvåle G. Age at natural menopause and all cause mortality: A 37- year follow-up of 19,731 Norwegian women. Am J Epidemiol 2003; 157:923-929.
55. Snowdon DA, Kane RL, Beeson WL et al. Is early natural menopause a biologic marker of health and aging? Am J Public Health 1989; *79***,** 709-14.
56. Hellstrom WK, Overstreet JW, Sikka SC et al. 2006. Semen and sperm reference ranges for men 45 years of age and older. J Androl 27; 421-28.

57. Kiernan K. Who remains childless? J Biosoc Sci 1989; 21: 387-98.
58. Sallmen M, Sandler DP, Hoppin JA et al. Reduced fertility among overweight and obese men. Epidemiology 2006; 17: 520-23.
59. Kirkwood T, Rose M. Evolution of senescence: late survival sacrificed for reproduction. Philosophical Transactions of the Royal Society of London*;* 1991; B332**:** 15-24.
60. Sonnenschein E, Brody JA. Effect of population aging on proportionate mortality from heart disease and cancer, U.S. 200-2050. J Gerontol Soc Sci 2005; 60B:S110-12.
61. Warner R, Appelby L, Whitton A, Faragher B. Demographic and obstetric risk factors for postnatal psychiatric morbidity. Br J Psychiatry 1996; 168: 607-11.
62. Rønsen M. Fertility and family policy in Norway- A reflection on trends and possible connections. Demographic Research 2004; 10: 265-286.
63. Curtis L, Phipps S. Social transfers and the health status of mothers in Norway and Canada. Soc Sci Med 2004; 58: 2499-507.
64. Aassve A, Mazzuco S, Mencarini L. Childbearing and well-being: a comparative analysis of European welfare regimes. J Europ Soc Pol 2005; 15: 283-9.

TABLE 1 Distribution of the sample by variables used in the analysis

	All women	All men_
Mean age (SD)_	52.04 (5.49)_	51.91 (5.42)_
Mean age 45-54_	48.98 (2.83)_	48.97 (2.83)_

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Marital status (%):Single_6.46_11.13_

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2_
3.8
5_

Me
an
(SD
)_
2.2
6
(1.3
1)_
2.1
3
(1.3
6)_

No.
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232
41_
400

71_

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196
610
_73
604
71_

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Ag
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up
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(%)
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<20
(F)
<23
(M)
_13
.51
_18
.81
_

20-
4
(F)
24-
8
(M)
_50
.16
_52

.32

–

25-
9
(F)
29-
34
(M)
_26
.18
_21
.22

–

30+
(F)
35+
(M)
_10
.14
_7.
65_

Me
an
(SD
)
age
1st
birt
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_23
.80
(4.3
4)_
26.
71
(5.0
7)_

Me
an
(SD
)
age
last
birt
h_2
9.5

0
(5.0
5)_
32.
68
(6.1
3)_

Las
t
birt
h
>39
(%)
_
3.1
0_1
3.0
4

No. of deaths

19106_29423_

Person years of observation_6380507_6189170

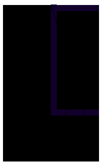

TABLE 2. Odds Ratios and 95% confidence intervals from fully adjusted discrete-time survival models of associations between number of children and mortality, all women and men aged 45-68.

Wo
me
n

Me
n_
_45
-
54_
55-
68_
45-
68_
45-
54_
55-
68_
45-
68_
Age
_1.0
98_
1.09
0-
1.10
5_1.
078
_1.0
72-
1.08
5_**1.**
088
_1.0
86-
1.09

1_1.
096
_0.9
72-
0.97
7_1.
106
_1.1
01-
1.11
1_**1.**
102
_1.1
00-
1.10
5_
Edu
cati
on:
___
___
___
___
_
Lo
wer
sec.
_0.7
35_
0.70
4-
0.76
7_0.
723
_0.6
94-

0.75
3_**0.**
730
_0.7
09-
0.75
2_0.
796
_0.7
69-
0.82
4_0.
795
_0.7
68-
0.82
3_**0.**
798
_0.7
79-
0.81
8_
Hig
her
sec.
_0.6
00_
0.55
3-
0.65
0_0.
656
_0.6
03-
0.71
5_**0.**

625
_0.5
90-
0.66
3_0.
647
_0.6
19-
0.67
6_0.
729

0.69	**0.66**
8-	**6-**
0.76	**0.70**
1_**0.**	**9_**
687	Hig
	her_
	0.51
	6_0.
	486
	-
	0.54
	9_0.
	530
	_0.4
	97-
	0.56
	5_**0.**

523
_0.5
01-
0.54
7_0.
512
_0.4
88-
0.53
7_0.
570
_0.5
42-
0.59
8_**0.**
540
_0.5
22-
0.55
9_
MS
c+_
0.40
1_0.

341
-
0.47
1_0.
466
_0.3
93-
0.52
0_**0.**
430
_0.3
83-
0.48
4_0.
415
_0.3
85-
0.44
6_0.
428
_0.3
97-
0.46
2_**0.**

422
_0.4
01-
0.44
5_
Mar
ital
stat
*us_*
____
____
____
____
Sin
gle_
1.39
4_1.
272
-
1.52
8_1.
610
_1.4
88-

1.74
4_1.
605
_1.5
23-
1.69
1_2.
024
_1.9
28-
2.12
5_1.
942
_1.8
36-
2.05
4_1.
989
_1.9
18-
2.06
4_
Div/
Sep

_1.7
21_
1.64
5-
1.80
1_1.
60
.532 **1.61**
- **4-**
1.68 **1.72**
3_**1.3**_2.
668 317
_2.2
39-
2.39
8_2.
072
_2.0
01-
2.14
5_**2.**
192
_2.1
39-

2.246_
Widowed_1.601_1.493-1.717_1.395_1.319-1.475_**1.394_1.330-1.462_**1.899_1.676-2.15

2_1.
594
_1.4
74-
1.72
3_**1.**
691
_1.5
83-
1.80
7_
No.
chil
dre
*n*__
___
___
___
__
0_1.
600
_1.5
04-
1.70

3_1.
396
_1.3
09-
1.48
9_1.
496
_1.4
31-
1.56
5_1.
423
_1.3
56-
1.49
2_1.
271
_1.2
07-
1.33
9_1.
348
_1.3
01-

1.39
6_
1_1.
363
_1.2
86-
1.44
4_1.
262
_1.1
88-
1.34
1_1.
312
_1.2
58-
1.36
8_1.
200
_1.1
45-
1.25
7_1.
193

_1.1
34-
1.25
4_1.
19
.157_3 40071
- 7360471
1.23
9

[redacted]

[redacted]

[redacted]

[redacted]

NS p>0.05; * p<0.05; **p<0.01; all other p<0.001

Reference categories: Compulsory education; married; 2 children. Year and dummy indicator of missing education also controlled.

TABLE 3 Odds Ratios and 95% confidence intervals from fully adjusted discrete-time survival models of associations between number of children and timing of parenthood with mortality, parous women and men aged 45-68

Wo
me
n

Me
n_
_45
-
54_
55-
68_
45-
68_
45-
54_
55-
68_
45-
68_
Age
_1.1
03_
1.09
5-
1.11
1_1.
084
_1.0
77-
1.09
1_**1.**
093
_1.0
90-
1.09

6_1.
01_
1.09
4-
1.10
7_1.
112
_1.1
06-
1.11
8_**1.**
110
_1.1
07-
1.11
2_
Mar
ital
stat
*us_*
Sin
gle_
1.42
9_1.
268
-
1.61
1_1.
430
_1.2
08-
1.69
3_**1.**
422
_1.2
90-

1.56
7_2.
055
_1.8
80-
2.24
6_1.
905
_1.6
51-
2.19
9_**1.**
984
_1.8
40-
2.13
8_
Div/
Sep
_1.6
53_
1.57
4-
1.73
5_1.
580
_1.5
03-
1.66
1_**1.**
620
_1.5
64-
1.67
7_2.
327

_2.2
43-
2.41
4_2.
053
_1.9
78-
2.13
0_**2.**
185
_2.1
29-
2.24
2_
Wid
owe
d_1.
349
_1.2
22-
1.48
9_1.
366
_1.2
87-
1.45
0_**1.**
358
_1.2
91-
1.42
9_1.
923
1.68 1.41
2- 8-

2.19 1.67
8_1.9_**1.**
543 **648**
_1.5
34-
1.77
0_
Pari
*ty*__
___
—_
___
—
1_1.
435
_1.3
51-
1.52
4_1.
309
_1.2
29-
1.39
5_**1.**

373
_1.3
15-
1.43
4_1.
265
_1.2
05-
1.32
9_1.
235
_1.1
73-
1.30
2_1.
255
_1.2
10-
1.30
0_
3_0.
926
**_
0.87

9-
0.97
5_0.
919
**_
0.87
4-
0.96
6_0.
922
_0.8
89-
0.95
6_0.
944
**_
0.90
6-
0.98
5_0.
988
NS_
0.94
9-

1.02
9_0.
965
*_0.
938
-
0.99
4_
4_0.
920
*_0.
854
-
0.99
1_0.
892
**_
0.83
6-
0.95
3_0.
904
_0.8
60-

0.94
9_1.
016
NS_
0.95
7-
1.07
8_1.
014
NS_
0.96
0-
1.07
1_**1.**
011
NS_
0.97
2-
1.05
3_
5+_
0.92
1N
.8280.80

- 1.025_0.877 **	4-0.957_**0.894** **_0.836-0.956_**0.973 NS_0.893-1.060_1.039 NS_0.965-1.118_**1.007 NS_0.95**

3-
1.06
5_
Age
at
1st
birt
*h_*
<20
/23_
1.17
8_1.
113
-
1.24
8_1.
232
_1.1
64-
1.30
5_**1.**
205
_1.1
58-

1.25
5_1.
224
_1.1
74-
1.27
5_1.
219
_1.1
68-
1.27
2_**1.**
223
_1.1
88-
1.26
0__
25-
9/29
-
34_
0.91
2**
_0.8

65-
0.96
3_0.
931
*_0.
884
-
0.98
0_0.
921
_0.8
87-
0.95
6_0.
918
**_
0.87
7-
0.96
1_0.
974
NS_
0.93
3-

1.01
8_0.
946
**_
0.91
7-
0.97
7__
30+
/35
+_0
.876
**_
0.80
8-
0.94
9_0.
910
*_0.
841
-
0.98
5_0.
890

_0.8
41-
0.94
1_0.
873
**_
0.80
6-
0.94
5_0.
928
NS_
0.85
8-
1.00
4_**0.**
897
_0.8
49-
0.94
8__
Last
birt
h at

age
40+
_0.7
94*
*_0.
687
-
0.91
7_0.
929
NS_
0.81
3-
1.06
2_**0.**
861
****_**
0.78
0-
0.94
9_0.
78
.740
- 0.91

0.83 1**
8 _
0.85
9-
0.96
7_**0.**
849
_0.8
14-
0.88
7__
No.
deat
hs
P.Y
ears
_92
52
443
498
5_9
854
194
552

2_**1**
910
6
638
050
7_1
442
6
434
689
7_1
499
7
184
227
3_**2**
942
3
618
917
0

NS p>0.05; * p<0.05; **p<0.01; all other p<0.001.
Reference categories: Married; 2 children; age at first birth 204 (F)/24-8 (M).
Educational status, year and dummy indicator of missing education also controlled.

TABLE 4. Odds Ratios and 95% confidence intervals from fully adjusted discrete-time survival models of associations between number of children and timing of parenthood among parents born 1950-58 controlling for educational level of subjects' parents (additionally to own education and marital status).

Women Men_

*Age*_1.155_1.005-1.328_1.093_1.069-1.118_

*Parental education*_____

Mother secondary/higher_0.994 NS_0.879-1.1

23_
0.8
44_
0.7
63-
0.9
33_
_Fa
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sec
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hig
her
_1.
030
NS
_0.
917
-
1.1
57_
1.1
42*
*_1
.04
0-
1.2
55_
_*N*
o.
chil
dre
*n*__
___
_1_
1.6
71_
1.4
57-
1.9
16_
1.3
70_
1.2
33-
1.5
45_
_3_
1.0
42
NS
_0.
911
-

1.1
91_
0.9
12
NS
_0.
814
-
1.0
20_
_4_
0.9
33
NS
_0.
733
-
1.1
18_
1.0
84
NS
_0.
911
-
1.2
90_
_5+
_1.
266
NS
_0.
867
-
1.8
50_
0.9
87
NS
_0.
725
-
1.3
44_
_A
ge
at
1st
birt
h
<20
/23
_1.
155
*_1

.00
5-
1.3
28_
1.1
68*
*_1
.05
4-
1.2
93_

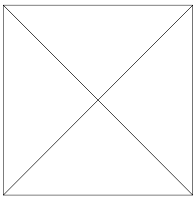
Last birth at age 40+	0.852 NS_0.622-1.167_0.708_0.613-0.816__No. of deaths Person Years_1397 911 179_2105

929
385

NS p>0.05; * p<0.05; **p<0.01; all other p<0.001.
Reference categories: Compulsory (parental) education; 2 children; age at first birth > 19 (F)/22- (M).
Marital status, own educational status, year, and dummy indicator of own missing education also controlled.

FIGURE 1 Mortality (ORs, 95% confidence intervals) among (A) ever-married women and (B) ever-married men by parity and marital status. (Controlling for age, year, own education, and, for married women, husband's education).

A)



B)