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Health effects of parental deaths among adults in Norway: Purchases of prescription medicine before and after bereavement



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ABSTRACT

We analyse effects of parental deaths on the health of women and men aged 18–59 in 2004–2008, indicated by purchases of prescription medicines. Register data covering the entire Norwegian population were used, and fixed-effects models were estimated to control for unobserved time-invariant individual factors. A parent's death seemed to have immediate adverse consequences in both main age groups considered (18–39, 40–59), although effects were lower in the older group. Some results suggested that this health disadvantage widened with increasing time since the parent's death. However, effects were weak: the annual number of different medicines purchased was only increased by 1–7% as a result of losing a parent. Death of a parent was associated with an immediate increase in purchases of medication for mental diseases, and there were indications of a physical response as well. As time since the parental death increased, there was a decline in the purchase of medication for mental diseases, but an opposite trend with respect to medication for other diseases. On the whole, maternal and paternal deaths had the same impact, and effects on daughters and sons were of the same magnitude.

1. Introduction

Relationships with close family members are important in most people's lives, and disruption of these relationships through death may have adverse consequences. Half of those who marry and do not divorce will eventually lose their spouse, and extensive research has identified excess mortality (Berntsen & \$2 Kravdal, 2012; Shor et al., 2012) and deteriorating physical (Carey et al., 2014) and mental (Sasson & \$2 Umberson, 2014) health following conjugal bereavement. However, a loss that an even larger proportion experience - in fact almost everyone in contemporary rich countries - is parental death. Obviously, a parent's death may be particularly harmful for young children, and the consequences of such early parental losses have attracted considerable scholarly attention (Li et al., 2014), but in contemporary societies it is far more usual that the death of a parent is experienced in adulthood. Although adults are clearly less dependent on their parents than children, strong interpersonal bonds and exchanges of support are nevertheless common (Attias-Donfut, Ogg, & \$2 Wolff, 2005; Bengtson, 2001; Hank, 2007), and disruption of these through death of the parent may have adverse effects on the health of adult offspring.

Results from the few and rather varied studies of adults' responses

to parental bereavement have been mixed. In two American investigations of short-term responses, the main conclusion was that parental deaths adversely affected both mental and physical health (Marks, Jun, & \$2 Song, 2007; Umberson & \$2 Meichu, 1994), although the opposite pattern appeared in some groups (Umberson & \$2 Meichu, 1994). In addition to observing a quite immediate effect on health, Marks et al. (2007) found that both the mental and physical health disadvantage increased over a five-year period for those whose parent had already died when this five-year period began. However, a Swedish register-based study focusing on mortality showed relatively low risks shortly after a parent's death, followed by an increase up to the level in the remaining population, or even above that among men who had lost their mother (Rostila & \$2 Saarela, 2011). Finally, a recent German study found that the death of a parent had, on the whole, a rather weak short-term adverse effect on subjective well-being, but no effect in the longer term except for daughters who lost their mother early (Leopold & \$2 Lechner, 2015).

A problem in all studies of health effects of loss of close family members is that a number of individual and societal factors may influence both the chance of such a death and the health of the individual under study (Boyle, Feng, & \$2 Raab, 2011; Van den Berg, Lindeboom, & \$2 Portrait, 2011). To mitigate these problems, the

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German study controlled for unobserved time-invariant individual characteristics, using a within-individual fixed-effects approach. Also the two American studies were based on measurements at different times for each person, but used another type of panel approach.

Our goal is to add to the knowledge about how the death of a mother or father affects adults' health by taking steps in three directions. First, we use register data for an entire national population. These include high-quality objective information on purchases of prescription medication, which we consider an indicator of health (although such purchases also reflect the use of health care as further discussed below). The only previous large register-based study (using Swedish data) focused on death, which is a rare outcome among young adults. Second, we make a distinction between mental and physical health responses, which was only done in the two American surveybased investigations, and we consider a quite long period of 10 years after a parental death, over which the relative importance of these two responses may change. We also examine medication use prior to the death of a parent in order to see if there is any effect which might arise from caregiving responsibilities and concerns over the parent's health. The German researchers also considered responses before death but not the other studies mentioned above. Third, we employ a longitudinal approach that controls for unobserved factors that have a constant effect on the health of the individual under study and may be associated with determinants of the parent's death. Such factors include genetically linked diseases or disease risks and certain traits and behaviours, for example health related behaviours and use of healthcare, which are likely to be similar within families. Furthermore, there is a large constant component of socioeconomic resources (also influenced by the family background and often not quite adequately measured in the data), which may operate through the mentioned factors as well as other channels. Constant time-invariant factors were also controlled for in the German study of effects on subjective well-being, but this approach could not be used in the Swedish study that, like ours, was based on national register data, as it was focused on death (which occurs only once for a person).

Our focus is on effects of parental deaths among adults younger than 59, as the data did not allow older individuals to be included. We consider two broad age groups and draw the dividing line at 40 years (almost in the middle of the 18–59 interval). Women and men are compared, but, for simplicity, we do not consider other interactive effects, such as how the responses to a parental death may vary with the person's own family situation.

2. Theoretical considerations

2.1. How the death of a parent may affect health

There are many reasons why an adult's health may be affected by the death of a parent. The grief and associated stress (Richardson et al., 2015) may cause mood and anxiety disorders (often difficult to distinguish from the intense sadness and anxiety that may be common features of the grief; Shear, 2012). The stress response, possibly in combination with poorer mental health, may also have implications for physical health, including the development and progression of cardiovascular diseases (Hamer, Endrighi, Venuraju, Lahiri, & \$2 Steptoe, 2012; Steptoe & \$2 Kivimäki, 2012). These reactions may compound each other as poorer physical health also increases the chance of depression and other mental diseases (Kessler & \$2 Bromet, 2013). Additionally, there may be more indirect health influences through grief- and stress-induced lifestyle changes, such as more use of alcohol, intensified smoking or a lower chance of quitting smoking (Kassel, Stroud, & \$2 Paronis, 2003; Umberson, 2003). Furthermore, many adults benefit from emotional and instrumental support from parents (Bengtson, 2001), and loss of this advantage may have adverse health effects. Also, the death of one parent may lead to increased responsibility and concern for the surviving spouse if there is one (Umberson,

2003).

In some circumstances these disadvantages may be offset by certain benefits. For example, caring for a close family member during terminal illness or earlier, or being very worried, may constitute a substantial burden on health (Christakis & \$2 Allison, 2006; Luhmann, Hofmann, Eid. & \$2 Lucas, 2012; Wolf, Raissian, & \$2 Grundy, 2015). and although this burden may leave a long-term mark (Moriarty, Maguire, O'Reilly, & \$2 McCann, 2015), it is also possible that the death to some extent may be felt as a relief, so that the person's health improves sometime afterwards (Rostila & \$2 Saarela, 2011). A combination of such a response and some of the mentioned longer-term disadvantages could, in theory, underlie the initial decline and subsequent increase in mortality observed by Rostila and Saarela (2011). Another possible beneficial effect is that bequests from deceased parents may improve the financial status of their children, with positive health implications (Rostila & \$2 Saarela, 2011). Additionally, experiencing a death in the near family may make people more conscious about their own health risks, leading to healthier behaviour (Marks et al., 2007; Umberson & \$2 Meichu, 1994). Finally, death may come as a relief if there has been an extremely difficult relationship with the parent (Umberson & \$2 Meichu, 1994).

Some of these responses may be more immediate than others. For example, the grief and associated stress reaction may influence mental health rather quickly, while it may take some time for physical health to be affected. Also, to the extent that there are bereavement-induced changes in lifestyle with implications for physical and perhaps mental health (through grief or, on the more positive side, inheritance from the deceased parent or stronger consciousness about own health risks), the process would likely be rather slow. Some responses may also be more transitory than others. In particular, effects triggered by a heavy care burden for a sick parent may wane quite shortly after the death. Besides, for many bereaved people, the grief and stress may become less intense after a while (Umberson, 2003), and to the extent that the death leads to an important loss of support, alternative sources may be found. Furthermore, if new lifestyle habits are developed as a result of a parent's death, it may be possible to reverse these. However, it is also possible that some of these problems are not quickly solved (e.g. inability to substitute support from parents with other kinds of support), and in the worst case a vicious circle may develop: One bad health condition may increase the chance of another, so that the health disadvantage actually increases as time goes on (as indicated by the studies by Marks et al. (2007) and Rostila and Saarela (2011)). Thus, it is far from obvious how the effects of parental loss should be expected to progress over time, except that the mental response probably comes earlier than the physical.

It is also difficult to predict how effects of maternal and paternal losses may differ. One possibility is that maternal deaths are felt more strongly because children, by and large, are more attached to their mother (Silverstein, Bengtson, & \$2 Lawton, 1997; Umberson, 2003). Two of the earlier studies do indeed point in that direction, but only for one of these sexes, as further discussed below (Rostila & \$2 Saarela, 2011; Leopold & \$2 Lechner, 2015).

2.2. Interactive effects

Parental deaths are likely to be particularly harmful to young children, for obvious reasons. Among adults, dependence on parents and the value of parental support may weaken as age increases. Indeed, as the parents themselves also become older, the direction of the net support stream may change. Furthermore, it is possible that dramatic and completely unexpected deaths have the most harmful implications (Merlevede et al., 2004; Rostila & \$2 Saarela, 2011), and these are more common at an early age. Overall, this would suggest that death of a parent has more adverse effects among younger compared with older, as reported in an earlier study (Leopold & \$2 Lechner, 2015).

Responses to parental deaths may differ also by gender, for example

because of variations in dependence on the deceased, or in vulnerability and coping strategies. The evidence is very diverse, however. A stronger effect for men than for women has been suggested in some studies (Rostila & \$2 Saarela, 2011), especially when considering physical health (Marks et al., 2007). Sex differences in this direction are consistent with an idea that women have other, and perhaps more efficient, coping strategies and also stronger alternative support networks (Umberson, 2003). However, daughters tend to have more contact with their parents as adults (Umberson, 2003; Silverstein et al., 1997), which could make them feel the loss particularly intensely. Daughters are also likely to be more involved in caregiving (Leopold, Raab & \$2 Engelhardt, 2014), which could work either way. Other studies have shown particularly strong responses for the child with the same sex as the parent who has died (Marks et al. 2007), and especially in mother-daughter dyads (Umberson & \$2 Meichu, 1994; Leopold & \$2 Lechner 2015), reflecting the suggested uniqueness of this relationship (Silverstein et al., 1997). It has also been reported that men who lose their mother are most strongly affected (Rostila & \$2 Saarela, 2011).

3. Data and methods

3.1. Data sources

The core data source for this study was the Norwegian Central Population Register, which includes everyone who has lived in Norway at any point since 1964. Information about year of birth, death, immigration and emigration (if any), as well as parent identifiers, was taken from the 2008 version of the register. The study population included those aged 18–59 in 2004 (i.e. born 1945–1986). Parent identifiers exist for almost everyone born after 1953, but for an increasingly smaller proportion as we go further back in time (see Appendix Table A1), so it would not be meaningful to include individuals born before about 1945. The time of death, if any, is known for all parents who are identified. Unfortunately, there was no information about the cause of death in the data available to us.

Information on purchases of prescription medicine was added from the Norwegian Prescription Database (NorPD; Furu et al., 2010). NorPD was started in 2004 and records all purchases of prescription medication - defined by Anatomical Therapeutic Code (ATC) codes - by all Norwegian residents except the few (in the age group considered in this study) who live in health care institutions.

3.2. Outcome variables

Several summary measures of morbidity based on administrative data have been suggested (Yurkovich, Avina-Zubieta, Thomas, Gorenchtein & \$2 Lacaille, 2015). These include medication-based indices, most of which reflect the idea that certain classes of medication can be seen as indicators of certain diseases or conditions ("diseases" is used below for simplicity), in the sense that they are used specifically for these and not for others. These diseases can then simply be counted (Sarfati, 2012), or one may construct a severity-weighted summary measure based on professional judgements of the severity of the diseases (von Korff, Wagner & \$2 Saunders, 1992; Fishman et al., 2003). Alternatively, one may want to take into account also the medicines that are not uniquely used for certain diseases, but may still provide information about the person's health. This may be done, for example, by considering the total number of different medicines purchased (Alibhai et al., 2008; Perkins et al., 2004; Schneeweiss et al., 2001). Using ATC codes, it would be reasonable to consider medicines as being different if the first five digits of the code (defining the active substance) are different.

This study was based primarily on the total number of different prescription medicines purchased within a year, but we also considered the total number of diseases within a year, inferred using Kuo et al.'s (2011) identification of 32 diseases treated by drugs uniquely prescribed for these diseases. Furthermore, we constructed another and original version of the latter variable by giving severity weights to each disease. These weights were taken as the effects of purchases of the respective medicines in the preceding year on all-cause mortality, according to a discrete-time model estimated for persons aged 30–79 (when there are more deaths) in 2005–2008. Thus, if the severity-weighted medication index is, for example, 0.2 higher for one person than for another, it means that there is a difference in overall medication use associated with a rise of 0.2 in the log-odds of dying in a general population at age 30–79. Additionally, we considered the total number of mental diseases and the total number of physical diseases. The former were depression, anxiety, psychosis and bipolar disorders, the first two of these being clearly the most common.

3.3. Models

The idea is to analyse how the outcome variable, which is defined for each year between 2004 and 2008 on the basis of purchases of prescription medicine, depends on whether the parent is dead at that time and how many years earlier the death took place. In the first part of the analysis, those whose parent was alive were divided into three groups: parent dead the next year, parent dead the year after that, and parent survived at least the next two years (in which case the parent may have died three or more year later or still be alive at the end of study period in 2008).

To be more specific, for women and men who were 18–59 years old in 2004 and for whom the father/mother was identified in the data, the following Poisson model was estimated, separately for the two age groups:

$$\log(E(Y_{it})) = b_0 + b_1 a_{it} + b_2 x_{it} + m_i$$
(1)

 $E(Y_{it})$ is the expectation value of the number of different medicines purchased during year t (between 2004 and 2008) by individual i, and m_i is a person-specific error term (fixed effect) picking up unobserved time-invariant factors that may be linked to factors affecting the father's/mother's chance of dying. \mathbf{x}_{it} is a vector of dummies for father's/mother's "status" at time t. The categories for this variable were "father/mother alive and not dead within the next two years" (reference category), "father/mother died two years later", "father/mother died one year later", "father/mother died the same year", "father/mother died one year earlier", "father/mother died two years earlier", etc. up to "father/mother died nine years earlier". Observations corresponding to the father/mother being dead for 10 or more years were left out.

Thus, individuals whose parent died before 1995 did not contribute in the analysis. Those whose parent died in 1995 contributed only an observation for 2004 (parent dead 9 years earlier), while those whose parent died in 1996 contributed observations for 2004 and 2005 (parent died eight years earlier and nine years earlier, respectively), and so on. Those whose parent died in 2007 or 2008 contributed observations in the reference category for 2004 or 2004-2005, respectively, and in other categories for the remaining years, while those whose parent was still alive by the end of 2008 contributed observations in the reference category for all years 2004-2008. Other individuals did not contribute observations in the reference category. (Individuals who had not lost their parent by the end of 2008 may, of course, have lost the parent later. Thus, their observations from 2007 and 2008 could in reality belong in the two categories "parent dead one year later" or "parent dead two years later". For that reason, all observations for 2007 and 2008 for individuals who had not lost their parent by the end of 2008 were excluded in supplementary estimation. This gave very similar results.).

 \mathbf{a}_{it} is a vector of dummies for the individual's age at time t, each representing a one-year age group. For an individual, a one-year increase in age goes hand in hand with a one-year later calendar year,

so these age effects also capture the weak secular increase in medication purchases. It is important to include age in the model, because as time since death increases the person also becomes older (and later calendar years are entered), and more medication is purchased because of that. Age effects are identified from the one-year observations in the reference category for the **x**-variable, for individuals for whom there are at least two such observations.

To provide an intuitive understanding of the fixed-effect approach, the effects of parental death and time since parental death are essentially estimated by comparing the change in medication purchases as the individuals "move" across the relevant categories of \mathbf{x} (e.g. from "parent dead next year" to "parent dead same year", or from "parent dead eight years ago" to "parent dead nine years ago") with the change in medication purchases over the same ages for those remaining in the reference category.

The reason for using one-year categories for time since a parent's death is that, if there are broader categories and the outcome variable really changes with time since death within each of these categories, this change will contribute to the age effect, which obviously has implications for the estimates of the duration effects. Preliminary estimation showed that the effects of long duration since death were considerably different if two- or three-year categories were used rather than one-year categories. For similar reasons, adding a broad category for 10 or more years could be potentially problematic, although the other parameter estimates were actually only moderately changed when this was done.

Obviously, only observations for years between 2004 and 2008 in which the individual was alive and resident in the country both at the beginning and end of the year could be included in the analysis. Mortality is low at the ages considered in the study. For example, in the total Norwegian population only 1.4% die before age 40 and 5.3% before age 59, according to the most recent life tables (Statistics Norway, 2016).

The models were estimated with the xtpoisson procedure in the Stata software, using the robust option to allow for within-individual clustering in the calculation of standard errors. Because of concern about possible overdispersion, some negative binomial models were also estimated, but the results were almost identical.

To avoid being drowned in parameter estimates when comparing results across outcomes and when considering the interactions with sex, we also estimated the simpler model:

$$\log (E(Y_{it})) = b_0 + \mathbf{b}_1 \, \mathbf{a}_{it} + b_2 \, d_{it} + b_3 d_{it} (t - t_0) + m_i \tag{2}$$

where d_{it} is 1 if the parent was dead at time t and otherwise 0 (i.e. the reference group now included also observations one or two years before death). t_0 is the year of death. Such models were estimated for the two main age groups as well as these groups combined, and for all outcome variables with one exception: When analysing the disease indicator based on severity weights, which is a continuous variable, linear models were estimated by means of the xtreg procedure. $log(E(Y_{it}))$ was then substituted by Y_{it} in (2) and an individual-level time-specific random term was added.

When considering the modifying impact of the child's sex s_i (1=female, 0=male), we also allowed the age effect to differ with sex. Thus, the model for a categorical outcome was:

$$\log (E(Y_{it})) = b_0 + \mathbf{b}_1 \mathbf{a}_{it} + b_2 d_{it} + b_3 d_{it}(t-t_0) + \mathbf{b}_4 \mathbf{a}_{it} s_i + b_5 d_{it} s_i + b_6$$

$$d_{it}(t-t_0) s_i + m_i$$
(3)

(The main effect of sex is subsumed in the fixed effect.) When analysing the severity-weighted disease indicator, log (E(Y_{it})) was, again, substituted by Y_{it} and an individual-level time-specific random term was added.

Because of the small range of values for some of the outcome variables (see details below), we also estimated some logistic models. Focusing on two of the outcomes, we estimated models for the

Table 1Effects (with standard errors) of parental deaths on the number of different medicines purchased within a year, among women and men aged 18–59.

	Loss of mother 18-39	Loss of mother 40-59	Loss of father 18-39	Loss of father 40-59
Alive and not dead within next two years	0	0	0	0
Dead 2 years				
later	0.0184** (0.0080)	0.0008 (0.0031)	0.0085 (0.0060)	0.0023 (0.0036)
Dead 1 year				
later	0.0322** (0.0084)	0.0005 (0.0033)	0.0220** (0.0062)	0.0057 (0.0037)
Dead same				
year	0.0437** (0.0087)	0.0074* (0.0034)	0.0328** (0.0064)	0.0159** (0.0039)
Dead				
1 year earlier	0.0334** (0.0094)	0.0126** (0.0038)	0.0372** (0.0070)	0.0182** (0.0043)
2 years earlier	0.0304** (0.0102)	0.0071 (0.0042)	0.0416** (0.0076)	0.0174** (0.0048)
3 years earlier	0.0285^{*}	0.0114*	0.0417**	0.0152^{**}
4 years earlier	(0.0110) 0.0216	(0.0045) 0.0123^*	(0.0082) 0.0396**	(0.0052) 0.0164^{**}
5 years earlier	$(0.0118) \ 0.0349^{**}$	$(0.0049) \ 0.0112^*$	(0.0088) 0.0395**	(0.0057) 0.0174**
6 years earlier	(0.0126) 0.0374^{**}	(0.0052) 0.0149^{**}	(0.0094) 0.0518**	(0.0061) 0.0227^{**}
7 years earlier	(0.0133) 0.0389**	(0.0056) 0.0173**	(0.0100) 0.0649**	(0.0063) 0.0233**
8 years earlier	(0.0142) 0.0419^{**}	(0.0060) 0.0209**	$(0.0105) \ 0.0657^{**}$	(0.0070) 0.0206^{**}
9 years earlier	$(0.0151) \ 0.0387^*$	(0.0064) 0.0220**	$(0.0111) \\ 0.0721**$	(0.0074) 0.0288^{**}
	(0.0160)	(0.0068)	(0.0117)	(0.0079)
Number of observations (millions)	6.08	4.25	5.78	3.04
Proportion (%) of observations in "alive" categories	95.8	70.0	91.2	54.6

Notes: Each column shows estimates from one model, which also includes age in oneyear categories and individual fixed effects.

probability of using at least two (or at least three) different medicines, and for the probability of having at least one mental disease. Besides, we estimated linear models for these two categorical outcomes. (In economics, the use of linear models is quite common even when the outcome variable is dichotomous.) The effects could, of course, not be expected to have the same absolute value as the effects in the Poisson models, but the signs were the same (not shown in tables).

In comparison, leaving out the fixed effects gave larger effects of parental deaths (not shown in tables), as one would expect. Without the fixed effects one should, of course, instead control for various observed factors that could be linked to parental deaths as well as affecting the person's health. For example, adding education reduced the effects, although they were still higher than according to the fixed-effects analysis.

3.4. Description of the samples

The proportions of the one-year observations that are in one of the

^{*} p & \$2lt; 0.05.

^{**} p & \$2lt; 0.01.

three "alive" categories (i.e. the reference category or the categories for parent died one or two years later) are shown in Table 1. For example, in the analysis of paternal deaths within the age group 40–59 (and where observations 10 or more years after the paternal death were left out, as explained earlier), 55 percent were in this category. In the analysis of maternal deaths, the corresponding proportion was 70 percent, reflecting women's higher life expectancy. The corresponding proportions were, of course, higher in the younger age group 18–39.

These figures accord reasonably well with some simple cross-sectional calculations for Norwegian residents in 2008: Among individuals aged 40–44 (the first five years of the oldest age group in our statistical analysis), 13% had lost a mother and 29% had lost a father, although many of those deaths could have taken place more than 10 years earlier (see Appendix Table A1, which shows results from calculations based on the same register data as used for the statistical analysis). The corresponding numbers for the age group 55–59 (the last five years of the oldest age group considered in the analysis) were 54% and 81%.

Individuals aged 18–39 on average purchased 2.1 different medicines per year during the study period. The standard deviation was 2.5. The corresponding average was 3.0 in the age group 40–59, and the standard deviation was 3.4. In the latter age group, 26% purchased no medicines, 17% purchased one, 14% purchased two, 11% purchased three, 9% purchased four, and 24% purchased five or more (of which only 5% purchased more than nine). The averages were 88% and 45% higher among women than men in the two age groups. Averages and standard deviations of all outcome variables are shown in Table 2. The lowest average, for mental diseases at age 18–39, is 0.11. (92% had no such disease as judged from the medication purchases, 6% had one and 2% had two or more.).

4. Results

4.1. Number of different medicines

Individuals of age 18–39 years who had lost their father the same year had somewhat poorer health than those with a father who was alive and not dying within the next two years (the reference category), as judged from the 3.3% higher number of different medicines purchased (Table 1). The number 3.3 is calculated by exponentiating the parameter estimate 0.0328 and subtracting 1. More medicine was also purchased in the year preceding the father's death, but not as much as in the year of the death itself. Over the subsequent 10 years, the effect of having lost a father first increased slowly and then more rapidly, reaching 7.5% higher number of medication purchases (=exp(0.0721)-1). For those losing their mother at this age, the

Table 2Average and standard deviation of outcome variables in the sample used to analyse effects of paternal deaths.

	Age 18-39		Age 40-5	59
	Average	Standard deviation	Average	Standard deviation
Total number of different medicines	2.09	2.54	2.99	3.36
Total number of diseases, severity weighted	0.10	0.39	0.19	0.54
Total number of diseases	0.57	0.98	1.09	1.47
Total number of mental diseases	0.11	0.39	0.18	0.50
Total number of diseases, except mental	0.46	0.82	0.91	1.27

Table 3Effects (with standard errors) of parental deaths on various medication-based health indicators, among women and men aged 18–59.

	Total number of different medicines	Total number of diseases, severity weighted	Total number of diseases	Total number of mental diseases	Total number of diseases, except mental
Loss of mo-					
ther					
Age 18– 59					
Dead	0.0089** (0.0019)	0.0079** (0.0012)	0.0089** (0.0022)	0.0641** (0.0047)	-0.0011 (0.0024)
Year	0.0013	0.0012)	0.0000	-0.0123***	0.0028**
since	(0.0006)	(0.0004)	(0.0007)	(0.0014)	(0.0007)
death					
Age 18– 39					
Dead	0.0172**	0.0102**	0.0144*	0.1179**	-0.0139
Year	(0.0052) -0.0006	(0.0023) 0.0005	(0.0070) -0.0048*	(0.0137) -0.0324**	(0.0078) 0.0032
since	(0.0015)	(0.0007)	(0.0020)	(0.0040)	(0.0032
death	(*****)	(0.000,)	(0.00=0)	(4144.14)	(****==)
Age 40– 59					
Dead	0.0075**	0.0073**	0.0085^{**}	0.0561**	0.0005
	(0.0020)	(0.0014)	(0.0023)	(0.0050)	(0.0025)
Year since	0.0014 [*] (0.0006)	0.0011** (0.0004)	0.0006 (0.0007)	-0.0087** (0.0015)	0.0028** (0.0007)
death	(0.0000)	(0.0004)	(0.0007)	(0.0013)	(0.0007)
Loss of					
<u>fa-</u>					
ther					
Age 18-					
59 Dead	0.0147**	0.0073**	0.0142**	0.0538**	0.0063*
_ 544	(0.0020)	(0.0011)	(0.0024)	(0.0051)	(0.0026)
Year	0.0019**	0.0005	-0.0007	-0.0114**	0.0018*
since death	(0.0005)	(0.0003)	(0.0007)	(0.0015)	(0.0008)
death					
Age 18– 39					
39 Dead	0.0195**	0.0085**	0.0232**	0.0829**	0.0079
	(0.0039)	(0.0017)	(0.0053)	(0.0106)	(0.0058)
Year	0.0038**	0.0008	-0.0012	-0.0189**	0.0034*
since death	(0.0011)	(0.0005)	(0.0015)	(0.0030)	(0.0015)
Age 40-					
59 Dead	0.0121**	0.0065**	0.0115**	0.0446**	0.0053
Deau	(0.0023)	(0.0014)	(0.0026)	(0.0057)	(0.0029)
Year	0.0009	0.0002	-0.0007	-0.0089**	0.0012
since	(0.0007)	(0.0004)	(0.0008)	(0.0017)	(0.0009)
death					

Notes: For each outcome variable and age group, each model includes whether the parent under consideration is dead (alive is reference category) and the time since death. Effects of these two variables are shown in the table. The models also include the person's age in one-year categories and individual fixed effects.

increase in medication purchases around the time of death was somewhat higher according to the point estimates (but confidence intervals were overlapping), followed by a decline and subsequent increase to almost the same level. Among those losing a parent at age 40–59, there was no clear upturn in the medication purchases before

^{*}p & \$2lt; 0.05.

^{**} p & \$2lt;0.01.

death. In the year of the parental death, there were only 0.7%-1.6% more purchases than in the reference category (and also a weaker response than at age 18–39 in absolute terms), but this was followed by an increase up to more than 2% over the 10-year period. On the whole, there was not a consistent difference in the 10-year increase between the two age groups.

Turning to the simpler models and pooling the two age groups, the additional medication purchases associated with a mother's death increased by 0.12% annually over the subsequent 10 years (the parameter estimate being 0.0012; Table 3, first column), while the corresponding increase after a father's death was 0.19%. However, confidence intervals overlap, so one cannot conclude that effects of paternal and maternal deaths are different (and not from the results for other outcomes either; see below). In comparison, medication purchases increased by 4.0% annually in the general population (not shown in the tables). Stated differently, when a person who has not lost a parent becomes one year older (and a new calendar year is entered), he or she purchases 4.0% more medicines. A person who has lost a mother or father purchases generally more medicine as a result of that loss, and the annual increase after that is not 4.0%, but 4.12% or 4.19%, respectively. Thus, one could say that those who have lost a parent have experienced a subsequent deterioration of health over age that is between 3% (0.12/4.0) and 5% (0.19/4.0) stronger than among other people (i.e., 4% overall, to make it simpler). Anyway, regardless of the perspective that is taken, the immediate response and subsequent further deterioration must be considered as rather weak.

4.2. Other medication-based outcome variables

Table 3 shows estimates also from models for the other medication-based indicators. An immediate adverse response appeared also when the total number of diseases, calculated with or without severity weights, was considered as an alternative indicator of the general health. Moreover, the disadvantage increased over time after maternal deaths in the age group 40–59 and the entire age group 18–59 when the severity weights were taken into account. (It is an absolute increase that is considered in these particular models, but predictions showed that there was also an increase in relative terms, such as considered in the other models.) There was an opposite trend after maternal deaths at age 18–39 when the severity weights were not taken into account.

Even more interestingly, when the sum of diseases was split into mental and other diseases, we observed an immediate increase of 4–13% in the use of medication for the former after a parental death (most sharply in the youngest age group) and some evidence of an increase with respect to the latter (only significant at 5% level after paternal deaths and when considering the entire age group 18–59). Moreover, there was a decline in the excess use of medicines for mental diseases over the subsequent 10 years, while there was an increase in the excess use of medicines for other diseases after maternal deaths in the age group 40–59 and after paternal deaths in the age group 18–39. In other words, the increase observed in the models for the total number of different medicines seems to be exclusively due to an increasing excess use of medication for other diseases than the mental.

4.3. Sex differences

Because of the modest variation across age that appeared in the other analysis, observations for both main age groups were used when analysing sex differences. According to the models for the total number of different medicines purchased, daughters only had about half as sharp immediate response to a father's death as did the sons (Table 4). (Also the absolute effect was somewhat weaker among women, who use more medicine than men, but not twice as many.) There were indications in the same direction with respect to maternal deaths (p & \$2lt;0.10). However, the subsequent increase after paternal deaths was restricted to the daughters. The point estimates suggest the same

Table 4Sex-specific effects (with standard errors) of parental deaths on various medication-based health indicators, among women and men aged 18–59.

	Total number of different medicines	Total number of diseases, severity weighted	Total number of diseases	Total number of mental diseases	Total number of diseases, except mental
Loss of mother, a	ge 18–59				
Dead	0.0128^{**}	0.0072^{**}	0.0115^{**}	0.0677^{**}	0.0036
	(0.0030)	(0.0015)	(0.0032)	(0.0081)	(0.0034)
Year	0.0000	0.0015^{**}	-0.0005	-0.0099**	0.0013
	(0.0009)	(0.0005)	(0.0010)	(0.0024)	(0.0010)
Dead×Female	-0.0068	0.0014	-0.0048	-0.0056	-0.0096^*
	(0.0039)	(0.0024)	(0.0044)	(0.0099)	(0.0048)
Year since	0.0018	-0.0010	0.0009	-0.0039	0.0029^{*}
dead×Femal-	(0.0011)	(0.0007)	(0.0013)	(0.0029)	(0.0014)
e					
Loss of father, age	e 18–59				
Dead	0.0201**	0.0057^{**}	0.0175^{**}	0.0478**	0.0127^{**}
	(0.0022)	(0.0014)	(0.0036)	(0.0084)	(0.0038)
Year	0.0002	0.0006	-0.0022^{*}	-0.0109^{**}	-0.0006
	(0.0009)	(0.0004)	(0.0010)	(0.0024)	(0.0011)
Dead×Female	-0.0093**	0.0033	-0.0063	0.0099	-0.0128^{*}
	(0.0041)	(0.0021)	(0.0048)	(0.0106)	(0.0052)
Year since	0.0027^{**}	-0.0003	0.0028^{*}	-0.0008	0.0046**
dead×Femal-	(0.0012)	(0.0006)	(0.0014)	(0.0031)	(0.0015)
e					

Notes: For each outcome variable, each model includes whether the parent under consideration is dead (alive is reference category), the time since death and interactions between these variables and the person's sex (Female=1 if individual is female, otherwise 0). Effects of these four variables are shown in the table. The models also include the person's age in one-year categories and individual fixed effects.

pattern after maternal deaths.

Also the total number of diseases increased more for daughters than for sons over the 10 years after a father's death. The particularly sharp increase in the disadvantage for daughters seems to be entirely a result of a physical health response that needs more time to build up for them (starting at a lower level but increasing more afterwards).

5. Discussion

This analysis showed that the loss of a parent has adverse health effects, as judged by the purchase of prescription medicine (which probably is a very good measure of the use). In addition to an immediate response, which was largest among the youngest, the excess use of medication increased among bereaved offspring with increasing time since the death in some situations (loss of mother at age 40-59 and loss of father at age 18-39). Taking the estimates for both age groups into account – and considering also the other outcome variables - it is impossible to conclude whether the response to a mother's death is different from the response to a father's death.

The effects of parents' death were rather small, however. The number of different medicines that was purchased increased by only 1–4% around the time of death, and over the subsequent 10 years the annual increase in the medication purchases was, on the whole, about 0.15 percentage points larger (and thus 4% larger in relative terms) than in the general population.

These results imply that the potential beneficial effects of a parental death, such as a stronger consciousness about own health risks and economic improvement because of inheritance, are more than outbalanced by various disadvantages. As mentioned, these disadvantages include loss of support and grief-induced stress (operating partly through life style changes), which may have implications both for

^{*} p & \$2lt; 0.05.

^{**}p & \$2lt; 0.01.

physical and mental health. Additionally, the health could be relatively poor when the burden of care and worries is most intense shortly before death, and this may leave a mark on health for some time after death. In support of that idea, there were indications of relatively poor health during the one or two years before death, although only among the youngest. While deaths of relatively young parents are often unexpected and not preceded by a long illness period, there is also a large proportion of the other early deaths that are a result of cancer, which to a particular extent may involve some years with great concerns over the person's health. Besides, it is possible that the youngest are generally more upset about any potentially fatal disease among parents because of the stronger dependence on the parents that was mentioned as a possible reason for their sharper reaction to a death.

As mentioned earlier, some types of response may come quickly, while others may need some time to build up. Furthermore, some may be transitory, while others are long-lasting or even accumulative. Predicting the overall outcome would be very difficult, but it seems at least likely that the impact on the mental health is more immediate than that on the physical health. In line with that idea, our analysis showed that the mental response was sharper than the physical shortly after death, but that the former became weaker over time, whereas the latter became stronger. However, it is far from obvious that one should expect a *steadily accumulating* disadvantage with respect to the physical health such as indicated by some observed increases in the excess total use of medication throughout the 10-year period under study.

Adverse health effects were seen also in the two American studies of parents' death (Marks et al., 2007; Umberson & \$2 Meichu, 1994), and one of them indicated a particularly strong worsening of the health situation over a five-year period for those who had lost their parents some time before this period (Marks et al., 2007). This accords with the increasing disadvantage we found. Also the results from the Swedish study of mortality bears some resemblance with ours, although there are also clear differences, as the authors reported a temporarily lower mortality after a parent's death, followed by an increase (Rostila & \$2 Saarela, 2011). However, the more careful analysis of the temporal pattern in the German study (Leopold & \$2 Lechner, 2015) did not reveal any lasting disadvantage, except in one case, but rather a shortterm worsening of subjective well-being. In other words, the pattern that we have observed with our presumably good data and control for unobserved time-invariant factors - and that is by no means theoretically implausible (see earlier discussion of a possible vicious circle) is quite different from what has been reported in the literature. There are no obvious reasons why an increasing disadvantage should appear only in a Norwegian setting, so a similar trend might appear also in other countries if the same kind of data and method were used.

Some earlier studies have suggested sharper effects of parental deaths at lower ages, but such a pattern was only moderately supported by our analysis. While the immediate response was sharpest at the youngest ages, and only the youngest had increased use of medication already before the parent's death, the subsequent medication trajectories did not vary consistently with age.

The existing evidence is quite mixed when it comes to the sex differences. In our analysis, the models for total number of different medicines showed a particularly strong immediate response among men after a parental death (one effect being significant and the other almost significant). However, the subsequent increase appeared to be restricted to daughters. This pattern seemed to be entirely due to a slower physical reaction to parental loss among women. Thus, there was no basis, on the whole, for concluding that daughters and sons were differentially affected by parental deaths, and no trace of a particularly sharp response to maternal deaths among daughters, as argued by some researchers.

5.1. Weaknesses

Estimates may be biased by selection even with this fixed-effects approach, as it only takes into account that certain unobserved factors of importance for a parent's mortality have a constant effect on the person's health. The higher usage of medication after a parent's death that is indicated by many of our estimates could, in theory, be the result of *time-varying* confounding factors. For example, acute dramatic family circumstances may have led to this death and also influence the health of the person under study negatively around the time of death and later.

Moreover, the increasing disadvantage after death could partly or fully be the result of childhood adversity or other factors that are linked to high parental mortality and also create an accumulative health disadvantage for the son or daughter. In other words, he or she may have been on a relatively steep upward trend in medication use already before the death, and might have remained on that path had the parent not died (yet). The education of the adult under study may capture some of this adversity, and we therefore added interactions between three educational categories and age in some supplementary models for those aged 40-59 (among whom a parental death up to 10 years earlier is not likely to have had any impact on the educational career). With such a model that allows for educational differences in the "basic age trend", the increase in the excess medication use after mother's or father's death was reduced by only about 8% (not shown). It remains to be known, of course, whether unobserved indicators of earlier adversity could have explained more of the increase.

Another weakness of the analysis is that it only includes the use of prescription medicine, not other types of medicine, which also reflect the health situation. Besides, the use of prescription medicine is not only a result of sickness and need for medicines, but also the use of health care services. Health care usage is partly a result of attitudes. which to some extent are constant and therefore controlled for. However, there is also likely to be variation in health care usage over time, and even a variation that is linked to parental death. To be more specific, relatives of a terminally ill parent may have been in more contact with health institutions than they otherwise would have been, which could increase their medication usage around the time of death. It could also continue to be high after the death if it is common to renew prescriptions even when the need is no longer so strong. Alternatively, there may instead be an underuse of medication shortly before a parent's death because there is not sufficient time to take care of one's own medical needs. One could always speculate whether the first-mentioned mechanism might be the stronger and contribute substantially to the observed increase in medication purchases shortly before and after a parent's death. However, it is hard to see how the widening excess medication purchases over the subsequent years could be explained by increasing use of health care.

5.2. Conclusion

To conclude, a parent's death has immediate adverse health consequences, as judged from the purchases of prescription medicines. Moreover, some results suggested a growing disadvantage among those who had lost a parent. These effects of parental deaths were generally rather weak, however. A parental loss affected the use of medication for mental conditions immediately, and there were indications of a physical response as well. While the mental response to parental loss weakened, the physical response became gradually stronger. Gaining further understanding of this apparently increasing disadvantage in physical health could be a goal for future research.

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Table A1Proportion having mother or father alive among women and men born in Norway and alive and residents in Norway 1 January 2008, by age.

Age	Number of	Proportion (%) with at least one parent not identified	Proportion (%) with at least one parent	Proportions (%) among the remaining			
individuais	individuals		emigrated	Both alive	Mother dead	Father dead	Both dead
18-19	116188	0.9	1.1	96.6	1.0	2.3	0.1
20-24	254331	0.8	1.4	95.1	1.4	3.4	0.1
25-29	241617	0.8	1.6	92.1	2.3	5.3	0.3
30-34	256280	0.8	1.7	87.3	3.5	8.6	0.7
35-39	302207	0.9	1.8	78.3	5.1	14.6	2.0
40-44	305835	1.1	1.8	63.4	7.0	23.8	5.8
45-49	284371	1.5	1.3	44.8	8.3	32.7	14.3
50-54	282536	2.3	0.7	26.8	8.2	36.9	28.1
55-59	270081	12.5	0.3	12.3	6.5	33.7	47.5

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Appendix

see Appendix Table A1

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