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Fertility history and use of antidepressant medication in late mid-life: a register-based analysis of Norwegian women and men

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ABSTRACT

Objectives: Life course influences on later life depression may include parenting trajectories. We investigate associations between number and timing of births and use of antidepressant medication in late mid-life using data on the whole Norwegian population.

Methods: We estimated logistic regression models to analyse variations in the purchase of antidepressants between 2004 and 2008 by timing of births and number of children among women and men aged 45−73, using Norwegian population register data. We controlled for age, education, marital and partnership status, and (in some models) family background shared among siblings.

Results: Mothers and fathers of two or more children were generally less likely to purchase antidepressants than the childless. Mothers who started childbearing before age 22 were an exception, although according to sibling models they were not *more* likely to purchase antidepressants. All models showed that women who became mothers before age 26 and had only one child had higher odds of medication purchase than the childless. Older age at first birth was generally associated with lower risks of antidepressant purchase.

Conclusion: This analysis of high-quality data for a national population indicates that early motherhood, childlessness and low parity are associated with higher usage of antidepressants in late mid-life. Our data did not allow identification of mediating pathways, and we lacked information on early mental and physical health and some other potentially important confounders not shared between siblings. Furthermore purchase of antidepressants is not a perfect indicator of depression. Those concerns aside, the results suggest complex effects of fertility on depression that merit further investigation.

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Antidepressants; fertility; Norway; prescription; register; sibling fixed effects

Introduction

Depression is one of the conditions contributing the most to the burden of disease in high- and middle-income countries (Ferrari et al., 2013), so understanding its determinants is important for public health. Life events and circumstances related to childbearing and childrearing are among the factors potentially affecting later-life depression. Indeed, the sense of meaning, purpose and responsibility parenthood may bring has been assumed to be protective for mental health since Durkheim's observation of low suicide rates among parents (Durkheim, 1897). However, responsibility for children may also lead to stresses, the impact of which may accumulate over time and increase with the number of children. As a reflection of such counteracting mechanisms, whose importance may vary across population groups and settings, and because of the variety of methods and (not always very good) data that have been used, conclusions from earlier analyses have been rather mixed, as described later in this section. In this study we analysed associations between depression at age 45−73 and a detailed indicator of timing and the number of children, using high-quality data for a whole national population that allow us to control for potentially important confounders.

Many studies on the association between fertility and depression have addressed ante- and post-natal depression. This has been estimated to affect 10%−20% of mothers in high-income countries and also is common among fathers (Fletcher, Matthey, & Marley, 2006; O'Hara & Swain, 1996). Other studies have considered depression beyond the post-natal period among women or men of reproductive age, many of whom have children that are relatively young and still co-resident (see details in sub-section on Analysis of same-sex siblings). Some of these studies have shown higher levels of depression among childless women (Rimehaug & Wallander, 2010) or men (Helbig, Lampert, Klose, & Jacobi, 2006) than among parents, while others have reached the opposite conclusion: one study found a high relative prevalence of depression among those with young children (Evenson & Simon, 2005), and another that unmarried parents were more likely to be depressed than the childless, while married fathers (but not mothers) were at an advantage (Nomaguchi & Milkie, 2003).

Possible longer-term effects of childbearing on the risk of being or becoming depressed have been investigated in studies considering a broad range of ages (McKenzie & Carter, 2013; Mirowsky & Ross, 2002; Pudrovska, 2008) and several which have focussed on people of post-reproductive age (Henretta, Grundy, Okell, & Wadsworth, 2008; Kruk & Reinhold, 2014; Plotnick, 2011), in some cases restricted to those aged 65 years and over (Kendig, Dykstra, van Gaalen, & Melkas, 2007; Spence, 2008; Zhang & Hayward 2001). These studies have also provided mixed evidence about the parenthood-depression association. Some have found that depression is less common

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among fathers than childless men, while no such relationship appears among women (Buber & Engelhardt, 2008; Huits, Kraaykamp, & Subramanian, 2013). Others have reported lower rates of depression among both mothers and fathers compared with the childless (McKenzie & Carter, 2013), while some have found no significant associations for either men or women (Agerbo, Mortensen, & Munk-Olsen, 2013; Hank, 2010; Kendig et al., 2007; Plaisier et al., 2008; Plotnick, 2011; Pudrovska, 2008; Read & Grundy, 2011; Spence, 2008).

A smaller number of studies of people in post-reproductive ages have considered the effects of number of children among those who have had at least one child. Some authors have found adverse effects of high parity among women (Kruk & Reinhold, 2014), some have found such adverse effects among men (Buber & Engelhardt, 2008; Plaisier et al., 2008; Pudrovska, 2008), and some have found no differences (Hank, 2010).

In addition to considering parenthood or number of children, some attention has been devoted to the possible effect of age at first birth. Some studies have found the highest prevalence of depression among those who became parents at a young age (Aras, Oral, Aydin, & Gulec, 2013; Carlson, 2011; Falci, Mortimer, & Noel, 2010; Henretta et al., 2008; Mirowsky & Ross, 2002; Spence, 2008), at least for one of the sexes (Read & Grundy, 2011), while other authors have seen no effect of a low age at first birth (Hank, 2010; Taylor, 2009). A *high* age at first birth has also been associated with depression (Carlson, 2011), at least for some sub-groups (Hank, 2010). Mirowsky and Ross (2002) reported that depression was more common among those having had their first child at an early or (for women) a late age than among the childless.

In this study, we analysed the association between fertility history and depression in late mid-life (ages 45−73) using purchase of antidepressant medication as our outcome indicator. A few earlier studies have considered the use of antidepressants (Dave, Petersen, Sherr, & Nazareth, 2010; Kruk & Reinhold, 2014), but most have relied on self-reporting of symptoms. The latter approach probably leads to more 'cases', but more heterogeneity in severity as antidepressants are typically prescribed for serious and moderate depression (Fournier et al., 2010; Hämäläinen et al., 2009). We used a very large register-based data set covering the entire Norwegian population and including 1.3 million observations (whereas earlier studies have typically included 1000−25,000). This enabled us to analyze associations by number of children (0, 1, 2, 3, 4+), age at first birth and the combination of both, which has not been considered before. Separate analyses were done for women and men because of known gender differences in depression risks, as well as possibly relevant gender differences in the experience of parenthood and childrearing.

Early life circumstances are among the factors that may influence both fertility and later depression. To take account of this we undertook some analyses in which we controlled for unobserved factors shared between siblings by estimating sibling fixed-effects models from a sub-sample of same-sex maternal siblings. Only a few other studies in this research area have taken into account unobserved confounders by means of sibling comparisons or panel data approaches (see below). Our data also allowed us to control better for marriage and partnership history, which to a large extent is intertwined with fertility, than in most previous studies.

Data and methods

Data

Our core data source was the Norwegian Central Population Register, which includes everyone who has lived in Norway since 1960. Information about year of birth, marital status at the beginning of each year, year of death, immigration or emigration (if any) and a spouse identifier were taken from the 2008 and older versions of the register. The register includes links to parents for more than 98% of the children born after 1953 and many of those born earlier. This means that, for almost every individual born after 1935 (and who is unlikely to have had children before 1935), there is complete information about the year of birth of all his or her children and identifiers of the other parent of each of these children. Using the parent identifiers, we constructed a sub-sample of siblings (see details below).

Education histories were added from the Educational Database (operated by Statistics Norway) and purchases of medicine were added from the Norwegian Prescription Database (NorPD) (Furu et al., 2011). NorPD was started in 2004 and covers all purchases of prescription medicine (defined by ATC codes) by Norwegian residents, except individuals living in institutions. As the proportion of individuals in institutions is very small even in the oldest age group we consider (only 2% of those aged 67−74 years [Ugreninov, 2005]), this is not a serious limitation.

Linkage of the various data files was done by Statistics Norway, using the unique personal identification number assigned to everyone included in the Central Population Register. Permission to establish and use the data was obtained from the data owners, the Regional Committees for Medical and Health Research Ethics and the Norwegian Data Protection Authority.

Outcome measure

Our indicator of depression was any purchase of antidepressants between 2004 and 2008. In accordance with the link between medicines and 32 specific diseases presented by Kuo et al. (2011), we considered all medicines with ATC codes starting with N06A, except N06AX01 and N06AX02, as being antidepressants.

Statistical analysis

Logistic models for the chance of purchasing antidepressants at least once during 2004−2008 were estimated for people born between 1935 and 1959 who were alive and resident in the country at the beginning of 2004 and the end of 2008.

In most models, three categories were defined for age at first birth: <22, 22−25 and 26+ for women, and <25, 25−28 and 29+ for men. In the total sample of parous, each of these three categories included about one-third of the observations. Parity (number of children) in the beginning of 2004 was our second fertility variable.

We also included variables expected to influence both fertility and depression risks. In addition to the person's year of birth (5-year categories), we included educational level and marital status at the beginning of 2004, and − among the married − we distinguished between those who had definitely experienced an earlier partnership disruption and others (see details in Results section). Educational level was

Table 1. Distribution of women and men in the study population (born in 1935–1959 and alive at the beginning of 2004 and end of 2008) by variables used in the analysis and number of women and men in the study population who purchased medicine for depression at least once during the years 2004–2008.

	Women		Men	
	Proportion in different categories (%)	Number of persons purchasing depression medicine	Proportion in different categories (%)	Number of persons purchasing depression medicine
Year of birth				
1935–1939	13.3	17,407	11.9	7979
1940–1944	17.0	21,193	16.6	11,381
1945–1949	22.7	30,618	23.2	18,204
1950–1954	22.7	30,922	23.4	18,628
1955–1959	24.3	31,148	24.9	18,987
Education				
10 years	27.7	45,031	24.1	23,087
11 years	32.3	42,184	24.3	19,016
12–13 years	14.3	17,688	21.9	15,313
14–17 years	22.6	23,620	20.7	13,366
18+ years	3.2	2765	9.0	4397
Marital status				
Never married	8.3	6771	13.3	11,342
Married	66.4	78,568	68.5	45,963
Widowed	6.5	9373	1.7	1475
Divorced/separated	18.7	32,576	16.5	16,399
Number of children, age at first birth				
0 child	9.7	12,754	14.2	12,346
1 child, low	2.5	4321	2.3	2099
1 child, medium	3.1	4831	2.8	2252
1 child, high	6.9	9273	6.9	5337
2 children, low	11.5	18,173	12.0	9785
2 children, medium	14.7	18,968	12.9	8660
2 children, high	14.3	14,975	13.2	8382
3 children, low	10.1	15,936	10.2	8176
3 children, medium	9.8	11,393	8.4	5456
3 children, high	5.8	5550	5.8	3583
4+ children, low	6.0	9130	6.0	5253
4+ children, medium	4.0	4436	3.5	2442
4+ children, high	1.6	1549	1.8	1409
	Total number under study		Total number under study	
	6,30,513	1,31,288 (20.8%)	6,28,277	75,179 (12.0%)

Note: For women, low age at first birth is below 22, medium is 22–25, and high is above 25; for men, low age at first birth is below 25, medium is 25–28, and high is above 28.

grouped into compulsory schooling (currently 10 years), short secondary education (11 years), completed secondary education (12–13 years), some college or university education (14–17 years) and the equivalent of a master's degree (18 or more years).

Distributions of the population by variables considered in the analysis are shown in Table 1. In total, 12.0% of the men and 20.8% of the women purchased antidepressant medication.

Sibling fixed-effects analysis

For women born between 1935 and 1959, we constructed a sub-sample consisting of those registered with at least one maternal sister (i.e. a woman having the same mother identifier) also born between 1935 and 1959. Mothers were identified for almost all those born after 1953, but for increasingly small proportions for older cohorts (as these were less likely to be still living in the parental home in 1970, which was the basis for establishing the parent–child link). The sample of female siblings thus includes rather few born before 1945. Models with and without sibling fixed effects were estimated from these sub-samples. A similar analysis was done for men, based on male maternal siblings.

The sibling analysis included 188,620 (30%) of the 630,513 women in the main analysis, while there were 235,835 men (38%), compared with 628,277 in the main analysis. (The number of sibling *groups* was lower than half of that as some had two or more siblings.) The reason for the larger number of men than women in the sibling sample is that men leave the parental home later (Chiuri & Del Boca, 2010) – partly because of their higher age at marriage – so that those born before 1953 are more likely to be registered with a link to their mother than are women born the same year.

Results

Number of children

Among women, those with one child were more likely to have purchased antidepressants in 2004–2008 than their childless counterparts (reference category), whereas those with two or more children had lower odds of medication purchase (Table 2, first column). Among men, purchase of antidepressants declined with parity up to two to three children and then increased such that the odds ratio for those with one child and those with four or more was only slightly below 1 (0.96, CI 0.93–0.98/0.99). For both men and women, purchase of antidepressants was inversely associated with increasing educational level, and was higher among those younger than age 60 than those older (not shown).

Age at first birth

High-parity women and men tend to have had their first birth relatively early. When this was taken into account in an analysis of the parous by adding age at first birth to the model, a

Table 2. Associations (with 95% confidence intervals) between number of children and parent's age at first birth and odds of using medicine for depression at least once during 2004−2008, among women and men aged 45−69 in 2004 (i.e. born in 1935−1959).

Women	Model 1	Model 2 (for the parous)	Model 3 (for the parous)
Number of children			
0 child	1		
1 child	1.13*** (1.11−1.16)	1	1
2 children	0.97** (0.95−0.99)	0.86*** (0.84−0.88)	0.81*** (0.80−0.83)
3 children	0.95*** (0.93−0.97)	0.84*** (0.83−0.86)	0.76*** (0.75−0.78)
4+ children	0.94*** (0.92−0.97)	0.83*** (0.81−0.85)	0.72*** (0.70−0.74)
Age at first birth			
−19			1.37*** (1.34−1.40)
20−21			1.17*** (1.15−1.19)
22−25			1
26−28			0.87*** (0.86−0.89)
29+			0.85*** (0.83−0.87)
Men	**Model 1**	**Model 2 (for the parous)**	**Model 3 (for the parous)**
Number of children			
0 child	1		
1 child	0.96*** (0.93−0.98)	1	1
2 children	0.84*** (0.82−0.86)	0.88*** (0.86−0.90)	0.86*** (0.84−0.88)
3 children	0.85*** (0.83−0.87)	0.89*** (0.87−0.91)	0.85*** (0.83−0.88)
4+ children	0.96** (0.93−0.99)	1.00 (0.97−1.03)	0.94*** (0.91−0.97)
Age at first birth			
−22			1.23*** (1.20−1.26)
23−24			1.08*** (1.05−1.11)
25−28			1
29−32			0.98* (0.95−1.00)
33+			0.99 (0.96−1.02)

*$p < 0.10$; **$p < 0.05$; ***$p < 0.01$.
Note: It was controlled for age and education.

more negative association between use of antidepressant medication and number of children was seen, especially for mothers, among whom the association between purchase of antidepressants and older age at first birth was most sharply negative (Table 2, last two columns).

Associations between purchase of antidepressants and a variable combining the number of children and the age at first birth are shown in Table 3. For women, odds of purchasing antidepressants were raised for those who had their first child early (<22). Women who had their only child at age 22−25 also had higher odds of medication purchase than the childless, as did one-child mothers who had their child at an older age (26+), but the difference in this case was small and no longer significant when marital status was controlled. Women with more than one child and a first birth after age 22 had lower odds of medication purchase than the childless. For example, the odds for those who had their first child at age 26 or later and had at least one subsequent birth were 19%−26% lower than for the childless. Controlling for marital status only weakened these associations to 15%−21%.

Among men, odds of medication purchase for fathers who had their first child early (<25) and had no more children was only raised by 5% and this difference ceased to be significant when marital status was controlled. Men who had their first child after age 28 and ended up with two or three children were, like women in this category, about 20% less likely to purchase antidepressants than the childless (odds being 22%−24% lower). The exception was men with four or more children. Controlling for marital status had only a rather small impact (odds then being 17%−18% lower than among the childless).

The small group (6%) of women who had their first child before age 26 and had no more children were more likely than other women to be divorced. (29% were divorced compared to, for example, 12% among those who had three or more children and had the first when they were older than 26.) Thus, when marital status was controlled, odds of purchase of antidepressants were lower, although the difference was still significant. This group might also be expected to include a higher proportion who had experienced union disruption, even if currently married. We therefore also controlled for an alternative marital-status variable where a distinction was made between married individuals with a registered earlier disruption of a marriage (only known if the current marriage started after 1970, the first year for which marital status is available) or who had had a child with someone other than their spouse (and thus presumably within a marriage or consensual union with that person) and all other married individuals. We refer to these two groups as having 'definitely experienced disruption' and 'others', respectively. Controlling for this gave only slightly reduced odds ratios for one-child mothers with an early birth (going down from 1.20 to 1.12 for those with a first birth before age 22 and from 1.15 to 1.14 among those with a first birth at age 22−25), and other estimates were also very similar (Table 3, third column).

Analysis of same-sex siblings

When the analysis was restricted to women and men with same-sex siblings, the results (Table 4, first column) were similar to those already reported for the total population. Among women, the positive associations were weaker and the negative ones stronger. Thus, the odds ratio for three- or four-child mothers who had their first child at age 26 or later were as low as 0.71 and 0.66, respectively. For men, the corresponding odds ratios were 0.71 and 0.75, as opposed to 0.76 and 0.96 in the full sample. When early life factors shared between siblings were controlled for by adding sibling fixed effects, the estimates were similar (Table 4, second and third column), although odds of antidepressant purchase were no longer

Table 3. Association (with 95% confidence intervals) between number of children and parent's age at first birth and odds of using medicine for depression at least once during 2004–2008, among women and men aged 45–69 in 2004 (i.e. born in 1935–1959).

Women			
Number of children, age at first birth			
0 child	1	1	1
1 child, low	1.28*** (1.23–1.34)	1.20*** (1.15–1.25)	1.17*** (1.12–1.22)
1 child, medium	1.21*** (1.16–1.26)	1.15*** (1.11–1.20)	1.14*** (1.10–1.19)
1 child, high	1.05*** (1.01–1.08)	1.02 (0.99–1.06)	1.04** (1.01–1.07)
2 children, low	1.18*** (1.15–1.21)	1.15*** (1.12–1.18)	1.14*** (1.11–1.17)
2 children, medium	0.97** (0.95–0.99)	0.97** (0.94–1.00)	0.98 (0.95–1.01)
2 children, high	0.81*** (0.79–0.83)	0.82*** (0.80–0.85)	0.85*** (0.82–0.87)
3 children, low	1.16*** (1.13–1.19)	1.14*** (1.11–1.18)	1.13*** (1.09–1.16)
3 children, medium	0.87*** (0.84–0.89)	0.88*** (0.86–0.91)	0.89*** (0.87–0.92)
3 children, high	0.74*** (0.72–0.77)	0.77*** (0.74–0.80)	0.79*** (0.77–0.82)
4 children, low	1.09*** (1.06–1.13)	1.07*** (1.03–1.11)	1.05*** (1.02–1.09)
4 children, medium	0.82*** (0.79–0.85)	0.84*** (0.80–0.87)	0.85*** (0.81–0.88)
4 children, high	0.74*** (0.70–0.78)	0.78*** (0.73–0.82)	0.80*** (0.75–0.85)
Marital status			
Never married		1.10*** (1.07–1.13)	
Married		1	
Widowed		1.21*** (1.18–1.24)	
Divorced/separated		1.58*** (1.55–1.60)	
Marital status			
Never married			1.16*** (1.13–1.19)
Married, definitely experienced disruption			1.47*** (1.43–1.50)
Married, other			1
Widowed			1.26*** (1.23–1.30)
Divorced/separated			1.66*** (1.63–1.69)
Men			
Number of children, age at first birth			
0 child	1	1	1
1 child, low	1.05** (1.00–1.11)	1.04 (0.99–1.09)	1.03 (0.97–1.08)
1 child, medium	0.95* (0.91–1.00)	0.95** (0.90–1.00)	0.94** (0.89–0.99)
1 child, high	0.92*** (0.89–0.95)	0.93*** (0.89–0.96)	0.93*** (0.90–0.97)
2 children, low	0.93*** (0.91–0.96)	0.96** (0.93–1.00)	0.96** (0.93–1.00)
2 children, medium	0.81*** (0.79–0.83)	0.84*** (0.81–0.87)	0.85*** (0.80–0.85)
2 children, high	0.78*** (0.76–0.80)	0.81*** (0.79–0.84)	0.83*** (0.80–0.85)
3 children, low	0.93*** (0.90–0.96)	0.96** (0.93–1.00)	0.95*** (0.92–0.98)
3 children, medium	0.79*** (0.77–0.82)	0.83*** (0.80–0.86)	0.83*** (0.80–0.87)
3 children, high	0.76*** (0.73–0.80)	0.81*** (0.77–0.84)	0.82*** (0.78–0.85)
4 children, low	1.02 (0.98–1.05)	1.04* (1.00–1.08)	1.00 (0.96–1.04)
4 children, medium	0.86*** (0.82–0.90)	0.90*** (0.86–0.94)	0.88*** (0.84–0.93)
4 children, high	0.96 (0.91–1.02)	1.02 (0.96–1.09)	1.02 (0.96–1.08)
Marital status			
Never married		1.14*** (1.11–1.27)	
Married		1	
Widowed		1.31*** (1.24–1.39)	
Divorced/separated		1.47*** (1.44–1.50)	
Marital status			
Never married			1.18*** (1.15–1.22)
Married, definitely experienced disruption			1.28*** (1.25–1.32)
Married, other			1
Widowed			1.36*** (1.28–1.44)
Divorced/separated			1.53*** (1.50–1.56)

*$p < 0.10$; **$p < 0.05$; ***$p < 0.01$.

Notes: For women, low age at first birth is below 22, medium is 22–25, and high is above 25; for men, low age at first birth is below 25, medium is 25–28, and high is above 28. It was controlled for age and education. Among women, 66% of the exposure time was in the married category (see Table 1); 7% was in the sub-category 'definitely experienced disruption' and 59% were in the 'other' sub-category. The corresponding proportions for men were 69%, 9% and 60%.

significantly higher among women who had their first child early than among the childless, except for mothers who had no further children (odds ratio 1.12, as opposed to point estimates 1.07, 1.08 and 1.01 at the higher parities).

Discussion

These results show that women and men with two or more children had a lower chance of purchasing, and thus presumably using, antidepressants in late mid-life than the childless and those with one child. For example, women and men who started childbearing relatively late (after age 25 and 28, respectively) and had two or more children were 15%–38% less likely to purchase antidepressants than the childless (varying across models and with the lowest odds ratios appearing among women of the highest parities). Some estimates suggested *high* usage compared to the childless among women with two or more children who started childbearing before age 22. However, when we controlled for family background using sibling fixed-effects models this disadvantage disappeared. We found a negative relationship – at each parity level – between age at first birth and the chance of using antidepressants. This association was stronger for mothers than for fathers.

The relatively small group of women who became mothers before age 22 and had only one child were more likely to use antidepressants than the childless, and this excess usage was evident even when partnership and family background

Table 4. Association (with 95% confidence intervals) between number of children and parent's age at first birth and odds of using medicine for depression at least once during 2004–2008, among women and men who were born in 1935–1959 and who were registered in the data with at least one maternal sibling of the same sex also born 1935–1959 (i.e. a sample of sibling sets consisting of two or more maternal same-sex siblings born in 1935–1959).

Women	Model without control for sibling fixed effects	Models with control for sibling fixed effects	
Number of children, age at first birth			
0 child	1	1	1
1 child, low	1.27*** (1.18–1.36)	1.15** (1.03–1.27)	1.12** (1.01–1.25)
1 child, medium	1.19*** (1.11–1.28)	1.16*** (1.05–1.28)	1.16*** (1.04–1.29)
1 child, high	1.02 (0.96–1.07)	0.98 (0.91–1.07)	1.00 (0.92–1.08)
2 children, low	1.12*** (1.07–1.18)	1.05 (0.97–1.12)	1.07* (0.99–1.16)
2 children, medium	0.93*** (0.89–0.98)	0.91*** (0.85–0.98)	0.95 (0.88–1.02)
2 children, high	0.78*** (0.74–0.82)	0.80*** (0.75–0.86)	0.85*** (0.78–0.91)
3 children, low	1.13*** (1.08–1.19)	1.05 (0.98–1.13)	1.08* (0.99–1.17)
3 children, medium	0.85*** (0.80–0.89)	0.86*** (0.80–0.93)	0.91 (0.83–0.99)
3 children, high	0.71*** (0.66–0.75)	0.70*** (0.64–0.77)	0.76*** (0.69–0.83)
4+ children, low	1.07** (1.01–1.13)	0.98 (0.90–1.07)	1.01 (0.92–1.11)
4+ children, medium	0.77*** (0.71–0.82)	0.77*** (0.69–0.85)	0.81*** (0.72–0.91)
4+ children, high	0.66*** (0.54–0.74)	0.62*** (0.53–0.73)	0.68*** (0.58–0.80)
Marital status			
Never married			1.20*** (1.12–1.28)
Married			1
Widowed			1.28*** (1.18–1.38)
Divorced/separated			1.57*** (1.50–1.63)
Men	**Model without control for sibling fixed effects**	**Models with control for sibling fixed effects**	
Number of children, age at first birth			
0 child	1	1	1
1 child, low	1.07* (0.99–1.16)	1.05 (0.93–1.18)	1.07 (0.95–1.21)
1 child, medium	0.95 (0.88–1.03)	0.89** (0.80–1.00)	0.91 (0.81–1.03)
1 child, high	0.92*** (0.87–0.97)	0.90*** (0.82–0.97)	0.92* (0.85–1.01)
2 children, low	0.92*** (0.87–0.96)	0.88*** (0.81–0.94)	0.94 (0.86–1.01)
2 children, medium	0.81*** (0.77–0.85)	0.83*** (0.78–0.89)	0.89*** (0.82–0.96)
2 children, high	0.76*** (0.72-0.79)	0.79*** (0.74–0.84)	0.85*** (0.78–0.91)
3 children, low	0.92*** (0.88–0.97)	0.93* (0.87–1.00)	1.00 (0.92–1.08)
3 children, medium	0.76*** (0.72–0.81)	0.80*** (0.74–0.87)	0.87*** (0.79–0.95)
3 children, high	0.71*** (0.67–0.75)	0.78*** (0.71–0.85)	0.84*** (0.76–0.93)
4+ children, low	0.97 (0.92–1.03)	0.95 (0.87–1.04)	1.01 (0.91–1.11)
4+ children, medium	0.75*** (0.69–0.81)	0.73*** (0.65–0.82)	0.79*** (0.70–0.89)
4+ children, high	0.75*** (0.67–0.84)	0.76*** (0.65–0.89)	0.82*** (0.70–0.97)
Marital status			
Never married			1.21*** (1.13–1.29)
Married			1
Widowed			1.45*** (1.24–1.68)
Divorced/separated			1.41*** (1.34–1.48)

*$p < 0.10$; **$p < 0.05$; ***$p < 0.01$.

Notes: For women, low age at first birth is below 22, medium is 22–25, and high is above 25; for men, low age at first birth is below 25, medium is 25–28, and high is above 28. It was controlled for age and education. There were 188,620 women in the study population (38,395 of whom purchased antidepressants) and 235,835 men (27,150 of whom purchased antidepressants).

shared between siblings was controlled for. For men, there were only weak indications of higher odds of usage compared to the childless among those who had their only child at a low age (before 25).

Potential causal influences

These observed associations between fertility history and depression probably reflect a combination of several causal effects and selective mechanisms that may counteract each other and perhaps even operate in a non-linear way. Starting with the potential causal effects, having offspring may confer a sense of meaning in life, and there are also emotional rewards from interactions with children (Eibach & Mock, 2011; Nelson, Kusklev, English, Dunn, & Lyubomirsky, 2013; White & Dolan, 2009). Parents may also be better integrated into the community (Bühler, 2008; Knoester & Eggebeen, 2006), and may benefit from emotional support and practical assistance from children (Brandt, Haberkern, & Szydlik, 2009; Wenger, Dykstra, Melkas, & Knipscheer, 2007). All this may reduce the chance of depression among parents, though there may not be proportionate increases in these benefits with each additional child. On the other hand, having children also involves stresses, such as economic concerns and worries about the child's well-being, which may have both immediate and longer-term effects on mental well-being (Manuel, Martinson, Bledsoe-Mansori, & Bellamy, 2012; Skipstein, Janson, Kjeldsen, Nilsen, & Mathiesen, 2012). These advantages and burdens may not be shared equally between mothers and fathers.

Some of these pathways potentially linking childbearing and depression may involve somatic health. Previous Norwegian studies have shown that parity is inversely associated with mortality and that, among parents, higher mortality is associated with earlier ages at first birth (Grundy & Kravdal, 2010). These associations probably reflect that childbearing affects physical health though social mechanisms such as those mentioned (and through a lower inclination for risky behaviour among parents (Wang, Kruger, & Wilke, 2009)), in addition to having some physiological effects for women. Physical health is in turn an important determinant of depression especially in later life (Kessler & Bromet, 2013).

Overall our results may reflect that the suggested beneficial effects of childrearing and involvement with children outweigh any negative influences in the form of increased stress, since most groups of parents had lower usage of antidepressant medication than the childless, even when controlling for

marital status (although there were not strong indications that having more than two children conferred additional advantages). The higher level of depression that was suggested among one-child mothers than among childless women – unless they had their child at a relatively old age – could, in theory, reflect a non-linear net negative effect of the number of children combined with a special burden for those starting early. Such a non-linearity may arise because economic or other burdens increase less when having additional children beyond the first (for example due to economies of scale), or because various emotional or other advantages derived from additional children for some reason are most pronounced at high parities.

The penalty that appears to be associated with an early first birth, at all parity levels, accords with earlier studies of the association between birth timing and depression in midlife. On the other hand, the use of antidepressants was not raised among those with a *later* first birth, which runs against the conclusion from some other investigations. The pattern we have observed is consistent with the hypothesis that maturity helps a parent deal with the challenges of childrearing, and it could also reflect that early parenthood contributes to disrupt education and work careers beyond what we capture with our education variable. The fact that the relationship between birth timing and use of antidepressants was stronger among women than men seems reasonable in the light of women's stronger involvement in childrearing.

There is no explicit information in these data about the character of the depression for which the medicine is used. However, the frequency of purchases may be seen as a very crude indicator. In supplementary analysis based on a multinomial model, we made a distinction between those who purchased antidepressants for 1 year, 2–3 years or 4–5 years within the study period. Unless those who purchased antidepressants only one year made purchases in years before or after the study period, it could indicate a short-lasting personal trauma. The conclusion was that parenthood has a less protective or (if an early birth) more adverse effect on the chance of using antidepressants for one year than on the chance of using these medicines for four or more years (not shown in tables). In other words, it seems that responsibility for or contact with children primarily helps people avoid the more long-lasting and possibly more serious cases of depression.

Selection

The relatively low chance of purchasing antidepressants among parents of two and more children, especially those with a late first birth, and the higher usage among some groups of one-child parents than among the childless, might also be partly or fully a result of selection. We could include rather few control variables in our analysis. Two of these were cohort (almost equivalent with age given the short period of observation) and current education. In principle, a relatively low education could to some extent be a *result* of high fertility or early parenthood, as mentioned in the preceding sub-section (see also Cohen, Kravdal, & Keilman, 2011), so one might suspect that we would be tapping out some of the causal effect of reproduction by including education. However, this is not an important issue in practice, as the control for education did not affect the key estimates very much.

We also controlled for marital status and to some extent partnership stability (though some people in the 'other' group may actually also have experienced disruption). Some other authors have also taken marital status into account (Bures, Koropeckyj-Cox, & Loree, 2009; Hank, 2010; Rimehaug & Wallander, 2010), but most have not. A reverse causality is even more likely with respect to such family variables, which is the reason why we showed effects both with and without control for marital status. However, the estimates did not differ much, which means that: (1) to the extent that we 'overcontrolled' by including marital status it is not a large problem, and (2) lack of control for marital status in earlier studies may not have been a serious limitation.

In a few previous studies, efforts have been made to reduce the selection problem by considering twin births (Kruk & Reinhold, 2014) or successful artificial fertilization (Agerbo et al., 2013) as an 'exogenous shock' to fertility. A few other authors have controlled for family-level unobserved confounders by comparing siblings (Pudrovska, 2008), as we do, or have used a fixed-effects longitudinal approach to control for constant individual-level unobserved factors, and thus essentially checked whether an increase in the number of children leads to a change in depression (McKenzie & Carter, 2013). Also Nomaguchi and Milkie (2003) used longitudinal data, but with control for the earlier situation rather than in a fixed-effects approach. These longitudinal studies have only shed light on the short-term responses to childbearing; however the focus of the present study was on long-term effects.

When sibling fixed-effects were included in the model in addition to cohort, education and marital status, our estimates did not change very much. In other words, characteristics that are shared between siblings – which include for example genetic factors, exposure to parents' attitudes, and socio-economic resources in childhood – do not appear to strongly affect both fertility and the chance of later depression (net of any effect through the socio-demographic variables in the model). A source of bias remains, of course, also in such sibling models, as there may be unobserved factors specific to each sibling that affect both that person's fertility and subsequent depression. Adult socio-economic resources above and beyond what is captured by the education variable is one example. In particular, women's and men's wages probably influence their chance of entering parenthood early (in complex ways) as well as their inclination to have additional children (Kravdal, in press), and may also have implications for their later mental health (Kessler & Bromet, 2013). Personality traits may also be a joint determinant of fertility (Skirbekk & Blekesaune, 2013) and depression. Moreover, there may be some aspects of the childhood family situation that are *not* shared between siblings, for example because parents treat their children differently or their resources change over time, and these could have implications both for fertility and later depression. However, they are not likely to be important confounders, given that the shared background factors appear to play such a small role.

Health in earlier years, including depression, is another possible confounder, even in the sibling fixed-effects models. More specifically, it seems likely that individuals who are depressed (including those suffering from post-natal depression) may be less interested than others in having (additional) children because they are more concerned about their ability to handle the burdens associated with childrearing and to appreciate the emotional rewards (Pinquart, Stotzka, & Silbereisen, 2008). They are, of course, also more likely to be depressed at a later age. On the other hand, there is also

some evidence suggesting that depressed individuals are more inclined than others to have their first child early, and that this explains the apparently adverse effect of teenage childbearing on depression, at least among the socio-economically disadvantaged (Mollborn & Morningstar, 2009). Similarly, poor physical health could affect the timing and quantum of fertility (Syse, Kravdal, & Tretli, 2007) and also lead to later depression. Moreover, having no or few children may – especially among the (previously) married – reflect impaired fecundity, which could be the result of diseases increasing the chance of later depression.

In additional analyses we tried to shed some light on the importance of earlier depression as a source of selection bias by estimating discrete-time hazard models for first-, second-, and higher-order births for the years 2006–2008, in a younger group of people aged 17–40. The models included the purchase of antidepressants during the previous year, age, duration since previous birth (if relevant) and interactions between these variables. We found that purchase of antidepressants was associated with increased subsequent first-birth rates among the youngest individuals, especially among women, while it was associated with lower first-birth rates among older individuals and generally lower second-birth rates (not shown). Thus, the relatively high usage of antidepressants among one-child mothers who had their child at an early age and the generally lower usage at higher parities for both sexes are consistent with the existence of this kind of selection mechanism (as judged from younger cohorts).

Strength and limitations of the study

The main limitation of this study is that potentially important confounders such as those mentioned earlier could not be controlled for. This problem has also hampered all earlier investigations. We approached the selection problem in a more advanced way than most previous studies as we (for a sub-sample) controlled for family background factors in a sibling fixed-effects approach.

Another limitation of our study, and any other study based on medication data, is that these data may not adequately reflect the actual prevalence of the disease. Rather, purchases of antidepressants reflect the combination of being depressed (or suffering from anxiety, as further discussed later in this section), going to the doctor, and getting a medication-based treatment (instead of another type of treatment or no treatment). Obviously, it is difficult to obtain a clear picture of how common it is for depressed people to use antidepressants or receive another type of treatment. According to studies from Sweden and Finland in the first years of the twenty-first century, only about one-fourth of those classified as depressed based on interviews used antidepressants, although the proportion was larger among the subgroup with the most severe depression (Hämäläinen et al., 2009; Henriksson, Asplund, Boëthius, Hällström, & Isacsson, 2006). In fact, there is probably underuse of all kinds of treatment (so studies based on health care usage would have similar limitations). Some authors have concluded that only half of the depressed receive any kind of acceptable treatment (Hämäläinen et al., 2009) and only two-thirds even among the severely depressed (Shim, Baltrus, Ye, & Rust, 2011). A Norwegian investigation showed an even smaller proportion seeking professional help for their depression, though the authors warned against quantifying this phenomenon (Roness, Mykletun, & Dahl, 2005). On the other hand, the proportion purchasing antidepressants in our data (16% during a five-year period) is close to the life-time prevalence according to Norwegian surveys (Mykletun, Knudsen, & Mathiesen, 2009), which suggests that we get a quite good impression of the prevalence of the disease by considering these purchases.

Could a difference in the tendency to consult a doctor explain some of the patterns that we see? Our main finding is that parents with at least two children have lower chance of purchasing antidepressants than the childless, and while this in theory could reflect that they do not suffer particularly little from depression, but tend not to go to the doctor and get prescription for these medicines, it does not seem a plausible explanation. Having a large family would rather be likely to increase the chance of seeking professional help. In support of that idea, a recent Norwegian study has shown particularly large underuse of medication for circulatory diseases among unmarried people (Kravdal & Grundy, 2014). Unfortunately, we are not aware of any similar evidence when it comes to the fertility-depression association.

It is also possible, in principle, that some of those with a low chance of using antidepressants are not actually less depressed or go less to the doctor, but that they have a tendency to prefer or be recommended other types of treatment than the medication-based. Conversely, a high chance of using antidepressants could reflect a preference for this kind of treatment more than a particularly high chance of actually being depressed. To our knowledge, however, there is so far no evidence of a link between fertility and the choice of treatment for depression.

An additional potential problem is that antidepressants are sometimes used for conditions other than depression, anxiety being the most common among these. According to some studies, as much as about one-third of antidepressants are prescribed for such reasons (Gardarsdottir, Heerdink, Van Dijk, & Egberts, 2007; Milea, Verpillat, Guelfucci, Toumi, & Lamure, 2010). In additional analyses, we therefore estimated associations between parity and the use of anxiolytics. This gave a similar pattern in the estimates, except that the use of this medicine decreased more with parity above one and with higher age at first birth, primarily among men (not shown). If the association between reproductive factors and the use of antidepressants for anxiety is similar to that between reproductive factors and the use of anxiolytics, it would mean that – for men – the reproductive factors are more weakly associated with the use of antidepressants for depression than with the total use of these medicines (i.e. the association we have estimated).

Finally, we lacked information on possible mediating pathways, and we have not taken into account possible variations in the effects of having children by the socio-economic resources of the family, whether both parents are strongly involved in the childrearing, the availability of social support, the characteristics of the children (e.g. sex, education, health), the quality of the child–parent relationship and various societal factors. A few earlier studies have addressed some of these interactions, reaching different conclusions (Hopcroft & McLaughlin, 2012; Huits et al., 2013; Manuel et al., 2012; Skipstein et al., 2012).

However, the study also has some important strengths, in addition to the use of a relatively advanced methods to control for antecedent factors shared among siblings. In particular, it is based on data for a whole population and so a large

analysis sample and lack of bias arising from non-response. Besides, a relatively complex fertility indicator is used and the outcome indicator is objectively measured (rather than self-reported). Because of these strengths the results may deserve considerable attention. The take-home message is that parenthood is associated with lower risk of antidepressant usage in late mid-life, except for women embarking on parenthood at an early age, and especially for those then having only one child. This pattern suggests rather complex underlying mechanisms which merit further investigation.

Ethical issues

The use of the register data for this research purpose has been approved by the Regional Committees for Medical and Health Research Ethics and the Norwegian Data Protection Authority.

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