Fertility among Norwegian Women and Men with Mental Disorders

Abstract

Discrete-time models for first-, second-, and third-birth rates in 2010-2018 were estimated, using data from the Population Register and databases on primary and specialized health care consultations. These data sources cover the entire Norwegian population. Birth rates were relatively low among individuals who had at least one consultation for depression in primary or specialized health care in the calendar year before the preceding. According to a simulation, the reduction in births rates corresponds to a reduction in completed fertility from 1.60 among women without any of the mental disorders under study, to 1.34 among the depressed. The corresponding numbers for men were 1.41 and 0.90. The associations between anxiety and fertility were of similar magnitude, while fertility was lower among individuals with bipolar disorder, eating disorder or personality disorder. Completed fertility was lowest among women and men with schizophrenia (0.36 and 0.16, respectively). However, to the extent that individuals with these mental disorders enter parenthood, many of the estimates suggested that they do so at a relatively low age. The associations between mental disorders and fertility became weaker when partnership, education and income were controlled for, and when characteristics shared by siblings were taken into account in family fixed-effects models.

Keywords: Fertility; Men; Mental health; Register data; Women

(Main text excluding supplementary notes: 7947 words)

Please note: Numbers in brackets refer to "supplementary notes" that can be found in the Online supplementary material along with five appendices.

Introduction

The fertility among individuals with health problems is an important issue for several reasons. For example, old people with serious chronic diseases who have no or few children may need particularly much support from health institutions. Furthermore, although some individuals with serious health problems may have decided against childbearing because they think this is in their best interest under the circumstances (and the childlessness is not forced on them through disease-induced infecundity), not having children may nevertheless be felt as a loss adding to their health burden. One may also argue that the parenting quality may be reduced among parents with severe health limitations, and that the children may have increased risk of similar health problems because of social or genetic transmission. The higher the fertility in this group, the larger is the number of children affected. Giving this a slightly different twist, one may say that knowledge about the fertility of individuals with severe diseases contributes to our understanding of how the prevalence of these diseases and the genetic predisposition for them develops over time. Finally, if we ever enter a situation with marked changes in young adult health, knowledge about how health affects fertility will, in principle, help improve predictions of fertility at the population level.

However, the demographic literature on fertility determinants includes remarkably little about health, except in countries where HIV is widespread. Also, the relatively few studies of health effects have included indicators of general health rather than specific diseases (Dommermuth et al. 2011; Holton et al. 2011; Gray et al. 2013; Fiori et al. 2017;

Barclay and Kolk 2020; Alderotti and Trappolini 2021; Syse et al. 2022; Alderotti et al. 2023; Lazzerati 2023). In contrast, the medically oriented literature includes several papers on how the number of children appears to be influenced by disabilities (Bloom et al. 2017; Shandra et al. 2014; Namatova et al. 2021) or various specific diseases (Syse et al. 2007; Laursen and Munk-Olsen 2010; Wiebe et al. 2014; Ban et al. 2015; Ferraro et al. 2017; Power et al. 2013). In this literature, much attention has been paid to fecundity as a mediator, and there are also studies with fecundity as the endpoint (Gill et al. 2009; Gade et al. 2014; Pieczynska 2018; Dumanski and Ahmed 2019).

The research on mental diseases has shown low fertility among individuals with schizophrenia, and a less markedly reduced fertility associated with bipolar disorder or eating disorder (Laursen and Munk-Olsen 2010; Bundy et al. 2011; Power et al. 2013). Reduced fertility has also been observed among the depressed, although one study found this association only among men (Power et al. 2013), and some smaller investigations have shown no or (for relatively young women) a positive association (Jonsson et al 2011; Nilsen et al 2012; Grundström et al 2023).[1] Studies of individuals with internalizing disorders (which include depression and anxiety) also indicate reduced fertility primarily among men, and additionally suggest that the reduction occurs especially among the relatively old, while the birth rates at lower ages may be higher than among those without such disorders (Laursen and Munk-Olsen 2010; Jokela et al. 2014; Evensen and Lyngstad 2020). Even stronger evidence of relatively early childbearing (among individuals becoming parents) has appeared in analyses focused on externalizing behaviour (Jokela et al. 2014; Østergaard et al. 2017; Evensen and Lyngstad 2020).[2]

The aim of this study is to provide more knowledge about how individuals' mental health at a certain point in time is linked to their subsequent fertility, using register data covering the entire Norwegian population. We consider depression and anxiety, which appear

to have become more common over recent years among adolescents and young adults in Norway (Krokstad et al. 2022) and elsewhere (Bor et al. 2014; Centers for Disease Control and Prevention 2023), as well as schizophrenia, bipolar disorder, eating disorder and personality disorder. All these disorders are among the top 15 contributions[3] to the loss of healthy life-years at age 20-39 in Norway, except personality disorder and, among men, eating disorder (calculations by Carl Bavarelli based on the Global Burden of Diseases Collaborative Network [2022]). We do not include ADHD, which contributes much to the disease load among children and adolescents, but is hard to identify among adults.

Our health indicators are largely dichotomous and distinguish between i) individuals who consulted a general practitioner (GP) or a specialist for the disorder under consideration within a certain period and ii) all other individuals. Earlier register-based studies of health effects on fertility have been based on information only about specialized health care, which excludes a large proportion of cases[4] – cases that are likely less severe and where fertility may be higher. Taking also the primary care into account, the results are probably more generalizable to everyone with the mental disorder (many of whom never seek professional help). We check how this broader disease definition changes the results, and also show the implications of considering consultations over a longer period back in time.

Even more importantly, we add to the existing evidence about the association between mental health and fertility in the following ways: First, both sexes are considered, which has not always been done earlier. Second, we separate between first, second and later births, as in only one earlier study (Laursen and Munk-Olsen 2010), and we also distinguish between first births in different age groups. Third, previous studies have not always focused on associations between mental health at a certain point in time and subsequent fertility, but rather the associations between corresponding cumulative measures, which leaves more doubt about the direction of causality (Powers et al. 2013). Fourth, we check whether the

associations are partly driven by partnership status, education and income (which may be selection factors as well as mediators). The role of these factors has attracted little attention earlier. Fifth, we show the implications of including family fixed effects, to control for all constant characteristics shared by siblings. This has not been done in previous investigations of the associations between mental health and fertility. Sixth, we predict – by means of stochastic simulation – how the estimated regression coefficients for the different disorders add up to differences in average completed fertility, proportion childless at different ages, and average age at first birth. These differences may be more informative to many readers than the differences in birth rates. As part of this step, we also calculate how much the disorders in total contribute to completed fertility at the national level.

Theoretical Reflections

Main Determinants of Fertility

To simplify, a woman's probability of having a child may be considered as determined by fecundity[5], sexual activity and childbearing desires (both influenced by her partnership status), and whether adequate contraception is used[6] (Easterlin and Crimmins 1985). Purchasing power, direct and opportunity costs of childrearing, preferences for spending time and money on children rather than other sources of satisfaction ('tastes' for childbearing), and social expectations are key determinants of the woman's or (if she has a partner) the couple's childbearing desires. The same factors may be considered as driving men's probability of having a child.

It is not obvious theoretically whether high income for a man would lead him or a couple to want relatively many children[7], but empirical studies have at least shown non-negative effects of a man's income on actual fertility (Hart 2015; Trimarchi and van Bavel

2020). One would expect a different impact of women's high wages, as an income effect such as for men may be counteracted by higher opportunity costs (although less markedly in settings where childcare is purchased).[8] Some studies have shown negative effects of women's earnings (potential) on fertility, and others positive effects (Kornstad and Rønsen 2018; Trimarchi and van Bavel 2020).

When the focus instead is on fertility timing - which is particularly relevant for first births - the economic argumentation needs a twist.[9] Theoretically, one might expect that a couple want to delay childbearing if the man's income is low [10], while the effect of women's low wages just as well could go in the opposite direction[11]. A Norwegian study showed positive effects of both women's and men's income on first-birth rates (Hart 2015), but because no attention was paid to age interactions, it is not clear whether higher income speeds up entry into parenthood or increases the chance of ever becoming a parent. Kornstad and Rønsen (2018) also reported positive effects on men's first-birth rates, without distinguishing between age groups, but negative effects for women.

School enrolment is typically found to reduce the chance of having a child soon, especially among women (Kravdal 2007).[12] Another issue of relevance for fertility timing is that individuals who fear that the net non-economic gains from childbearing may be reduced over time (e.g., because of poorer health), or whose 'taste' or 'preference' for a child is relatively strong, may be eager to have a child soon.

How Mental Health May Affect Fertility

Most of the main fertility determinants mentioned above may contribute to associations between mental health and fertility. For example, mental disorders may restrict education, work or leisure activities (Kessler et al. 2008; Nordmo et al. 2022) – or even require some

sort or caregiving by other persons - making people with such disorders both less likely to meet potential partners and to be considered attractive as *mates* (MacCabe et al. 2009).[13] A reduced chance of forming and remaining in a partnership may be a more important causal channel for men than women, as their disorders may be more severe and lead to more social isolation (Laursen and Munk-Olsen 2010; Bundy et al. 2011; Power et al. 2013).[14]

Additionally, a disease or the treatment for it may lead to *sub- or infecundity*, including decreased libido. This mechanism is probably more relevant for physical[15] than mental diseases, but studies have shown associations between weak sexual desires and depressive symptoms (Lourenco et al. 2010), or reduced semen quality among men under psychological stress (Bhongade et al. 2015). Also, and regardless of fecundity, there may be concerns about whether the recommended medication for, for example, bipolar disorders or depression may have a teratogenic effect (Munk-Olsen et al. 2018).[16] Not having a child may then be seen as the best option.

Furthermore, people with poor health may see childbearing as less emotionally rewarding (i.e., they have less strong '*tastes'* for it) in a wide sense of that concept: They may fear that they will be too tired to enjoy the parental role or that the child will inherit the disease, or they may be burdened by doubts about whether they will care well enough for the child (Shover 2005; Schmidt et al. 2016; Ferraro et al. 2017; Meaney 2018; Harpe et al. 2022).[17] Such concerns may contribute both to a weaker desire for having a(nother) child and a reduced interest in having the child relatively fast, although the latter is less obvious, as some individuals with mental diseases may expect a worsening of the condition that makes parenthood even more burdening if delayed.[18]

To the extent that mental disorders lead to lower income and fewer years in school, this may affect fertility not only through partnership. As explained above, men's low income

may make a couple want a later entry into parenthood and fewer children, although there is much uncertainty especially about the latter, while there may be weaker or opposite effects of low wages among women. Because school enrolment is linked to low birth rates (especially among women), those with short education tend to have their children relatively early, but it is less obvious how the lower educational attainment itself affects fertility (through partnership, purchasing power, childbearing costs or otherwise). It possibly reduces fertility among men in particular (Kravdal 2007; Kravdal and Rindfuss 2008; Jalovaara et al. 2019).

It is also possible that some mental disorders reduce the chance of using adequate contraception, for various psychological reasons (Hall et al. 2015). Accordingly, unintended births have been shown to be relatively common among individuals with depression, anxiety or anorexia (James-Hawkins et al. 2014; Mikali et al. 2014; Hall et al. 2015)[19] and elevated abortion rates have also been reported (Laursen and Munk-Olsen 2010).

To summarize, a variety of causal pathways may contribute to fewer children and later births among individuals with mental diseases, but there are also opposite mechanisms, which may be most important for women. Furthermore, timing differences may be caused by other mechanisms than those mentioned above. First, the group with a mental health diagnosis consists of some individuals with very severe symptoms and others who are less affected. If, at any age, the chance of entering parenthood within the next year declines with worsening symptoms, those who have a diagnosis and are still childless at, say, age 30 will include a larger proportion with severe symptoms than those who have a diagnosis and are childless at, say, 20. Thus, the estimated effect of having a mental health diagnosis becomes more negative as age increases. Stated differently, to the extent that they enter parenthood at all, individuals with such a diagnosis will seem to do it relatively early. However, among individuals diagnosed already by age 20, the disease is particularly likely to have started

early, which is linked to high severity (Lahey et al. 1999; Kessler et al. 2001). This mechanism contributes in the opposite direction.[20]

Although the possibility of parity-dependent effects has attracted little attention (Laursen and Munk-Olsen 2010)[21], such variation is indeed plausible. For example, the chance of finding a partner – which is of special importance for the first-birth rates – may be more strongly linked to mental disorders than the chance of keeping a partner – which is relevant at higher parities. Furthermore, while having the first child typically is the most life-changing transition, it is possible that the burden of having another child – which may be particularly heavy for individuals with mental health problems – may be seen as larger at parity two than at parity one. Finally, there is a selection similar to that relevant for timing: Among individuals with a mental disorder, the disorder may be less severe among those who already have a child. Thus, the estimated effect of the disorder becomes weaker for second- and higher-order births.

Importantly, observed statistical relationships between health and fertility do not only reflect health effects on fertility, but also joint determinants. For example, personality is related to – and possibly also influencing - mental health, and may affect fertility as well, partly through social relations (Kotov et al. 2010). It is also possible that genetic liability for certain mental disorders may be linked to fertility, although this did not show up in a recent analysis of schizophrenia (Lawn et al. 2019). Additionally, the joint determinants likely include socioeconomic factors (Silva et al. 2016; Kinge et al. 2021), which one will typically never control perfectly for. As just mentioned, however, socioeconomic factors likely also mediate the health-fertility effect, which makes analysis difficult (see below). Similarly, partnership status – which is linked to socioeconomic factors – may influence both mental health and fertility, in addition to being causally between them. Furthermore, mental health problems may be partly a result of sub- or infecundity, which obviously influences later

fertility. Finally, there is an ongoing discussion about whether hormonal contraceptive use – which clearly may affect fertility – increases depression risks (Fruzzetti and Fidecicchi 2020).[22] In this study, we control for some observed and (in sibling models) unobserved characteristics of the family of origin, plus the index person's partnership status, education, and income (see details below). The remaining effects of mental health on fertility must then reflect other types of selection or causal channels.

Data and Methods

Data Sources

The key data sources were the Norwegian Population Register, the KUHR register, and the Norwegian Patient Register, from which data covering the period up to 1st January 2019 were extracted.

All persons who have ever lived in Norway after 1964 have been included in the Population Register and assigned a personal identification number (PIN) that is also used in other registers. The Population Register includes information about the person's sex, country of birth, and dates of birth and (if any) death. From 2005, there is information on marital and cohabitation status as of 1st January for everyone. Additionally, the data allow construction of almost complete birth histories for women and men born in Norway after 1935[23], and they include annual information on municipality of residence as of 1st January, with a missing code for residence abroad.

The KUHR register includes information about consultations with general practitioners (GPs) from 2006.[24] Up to two (or in a few cases three) diagnoses or symptoms, in the ICPC-2 system, are reported for each consultation. We considered only

face-to-face GP consultations and the diagnoses (not sympoms) when constructing the disorder indicators.

The Norwegian Patient Register (NPR) includes data on use of specialized health care from 2008 or, for some types of specialized care, a later year. We considered all consultations [25] and almost all information on main and secondary diagnoses.[26]

Additionally, the analysis was based on annual data from Statistics Norway on income[27], school enrolment, and education level.

Discrete-time Hazard Models

Discrete-time hazard models for first, second and third births (and in some parts of the analysis higher-order births) were estimated separately for women and men born in Norway 1965-2001.[28] In the analysis of first births, a series of 3-month observations (including January-March, April-June, July-September, or October-December) was constructed for each individual. The first observation (quarter) started 1st January the year the individual turned 17 or 1st January 2010, whichever came last. The last observation was the last quarter of the year when the individual turned 45, the last quarter of 2018, the quarter before the individual died, or the quarter when the first child was born, whichever came first. Each 3-month observation included some independent variables (described below) and an outcome variable, which was whether a first birth took place within the 3 months. The calendar year including the quarter is referred to as t below. Quarters in years when the individual did not live in Norway 1st January, and for individuals who had not lived in Norway 1st January each year since 2008, were excluded.[29] The logistic model

$$\log(p_{iq}/(1-p_{iq})) = \boldsymbol{\beta}_0 + \boldsymbol{\beta}_1 \mathbf{X}_{iq}$$

was estimated from all 3-month observations for all individuals. p_{iq} is the probability that individual i had a first birth within quarter q (within year t), $\boldsymbol{\beta}_0$ is a constant term, $\mathbf{X}_{iq}$ is a vector of categorical variables further described below, and $\boldsymbol{\beta}_1$ is the corresponding coefficient vector.

The analysis of second or third births was similar, except that the first 3-month observation was no earlier than the quarter after the previous birth.[30]

Parts of the analysis of first births was stratified by the age at the end of year t, using five age groups, each of which included about 1/5 of the first births. Variation in fertility effects across age provides information about timing. For example, if a disorder increases first-birth rates at ages 17-26 and reduces first-birth rates at higher ages correspondingly, so that the chance of ever becoming a parent remains unchanged, it means that individuals with the disorder become parents earlier. However, when effects on fertility are estimated from age-specific logistic models (and a simulation is based on these estimates; see below), one should not draw sharp conclusions about the timing. This is because estimates from logistic (as opposed to linear) models for different samples are not fully comparable (Mood 2010). In principle, (small) differences in the effect estimates across age may be just a result of age differences in unexplained variance.[31]

Because there are probably joint unobserved determinants of first, second and higher-order birth rates, one might consider it more reasonable to estimate one so-called multilevel-multiprocess model that includes all the parity transitions, such as in some other fertility studies published over the last couple of decades (Kravdal 2001). However, this much more complex analysis did not give markedly different results (Appendix 1).

Variables.

In most of the analysis, there was one dichotomous health indicator for each disorder. It refers to whether the disorder was included as at least one of the diagnoses for either a GP consultation or a specialist consultation in year t-2.[32] The diagnosis codes for the various disorders are shown in Table 1. Alternative health indicators were used in some models (see below).

(Table 1 about here)

In addition to the disorder indicators, age at the end of year t was included in all models, as well as duration since last previous birth (when relevant) and t itself.[33] In a first step of the analysis, it was checked whether controls for several sociodemographic characteristics of the index person's parents affected the estimates – which they did not (see below).[34]

Furthermore, we included the following sociodemographic variables for the index person in some models: partnership status (a combination of marital and cohabitational status) at the beginning of t-1, the highest education level attained as of 1st October in t-2, whether the individual was enrolled in school at that time, and the annual income in t-2.[35] Note that the year t-2 does not include the pregnancy leading up to the birth under study, if any, and that the situation in that year therefore cannot be a *result of* the pregnancy. The index persons' partnership status, education and income may influence both their mental health and fertility, in which case it would be reasonable to control for these factors. However, they may also be causally between mental health and fertility, so that we are 'taking away' a mediating channel by controlling for them. We therefore show estimates both

from models including and not including these variables. One might consider using longer lags for the sociodemographic variables, so that it would be more reasonable to consider them as selection variables, but this would be far from a perfect solution, because the health indicator based on consultations in t-2 may reflect a health problem that has lasted for some time.[36]

Note that the aim is not to quantify exactly how much partnership, education, and income contribute (as mediators or selection factors) to the associations between mental health and fertility. These factors operate in complex ways through each other, and one cannot even find out how much they contribute in total by simply comparing estimates from models with and without them. This is because of a so-called 'scaling problem': Effect coefficients for variables V in logistic models may change even when variables uncorrelated with V are added (Mood 2010). However, in such a case, the coefficients become larger rather than smaller, so comparison across models provides a conservative measure of how much the added variables contribute to the associations appearing in the simplest model.

Family Fixed Effects

Part of the analysis is based on family fixed-effects models, to control for constant (observed and unobserved) characteristics shared by maternal siblings. Such models have been used only in a few other studies of health effects on fertility, where the focus has been on physical diseases (Penovich 2000; Green et al. 2009; Wiebe et al 2014). It is not obvious that family fixed effects can be included in a discrete-time hazard model, but it has been shown that they can be included in a Cox model (Ridder and Tunali 1999). This is another (and continuous-time) type of hazard model, which we do not use as our main tool for convenience reasons,

and because only the discrete-time version includes a specified baseline hazard that makes it possible to do the simulation.

Fortunately, the estimates from Cox models[37] without family fixed effects were almost identical to those from the discrete-time models (see below). Before adding the fixed effects, we restricted the data set to individuals who had a same-sex maternal sibling under exposure for the same fertility transition within the relevant period.

In principle, estimates from a sibling model will be biased if fertility is affected by both own and a sibling's mental health, but as discussed in Appendix 2, there is little reason for concern about this.

Simulation

We generated birth histories for 100,000 women with, for example, depression. This was a sufficiently large sample, because we got the same results with twice as many individuals. Starting at age 17, a 3-month birth probability was predicted for a woman every third month up to age 45 on the basis of the demographic characteristics at that time (age, number of children, and duration since last birth), period set to 2018, the depression dummy set to 1, and the estimated effects of all these factors in the relevant model (i.e., model for first, second, third, fourth or fifth birth, depending on the number of children already born). A singleton birth was ascribed to the woman within the interval if a number drawn from a uniform distribution over [0,1] was less than the predicted birth probability. Based on these simulated birth histories, we calculated the average number of children at age 45, the proportion childless at different ages, and the average age at first birth among those who ever had a child. Similar simulations were carried out also for the other disorders, by setting the

respective disorder dummies to 1, while all disorder dummies were set to 0 in the simulation for women with none of the mental disorders. We then repeated the procedure for men.

Results

Main Patterns

Among women, 5.6% of the time under exposure for a first birth is in the depression category.[38] The corresponding proportions for anxiety, schizophrenia, bipolar disorder, eating disorder and personality disorder are 2.3%, 0.3% 0.6%, 0.9% and 0.8%, respectively (Table 2). The proportion with schizophrenia is higher among men than women, but all the other disorders, and especially eating disorders, are less common among men. The patterns in the distribution of the mental disorders are not very different among individuals under exposure for second or third births, although especially the proportion with schizophrenia is smaller at higher parities, because of the very low first-birth rates (Table 2).

To get an impression of how common the various disorders are, we also calculated the proportions who had at least one consultation for them between 2008 and 2016, among individuals at any parity who were 30 or 40 years old in 2018. The figures range from 0.1% with eating disorder among men to 25.3% with depression among women (Appendix Table A3.1). [39]

(Table 2 about here)

Depression and anxiety are associated with a 9% reduction in women's first-birth rate (Table 2).[40] However, the reduction is limited to the higher age groups; first-birth rates up

to age 23 are relatively high among women with these disorders (Table 3 and 'uncontrolled' in Figure 1). This means that, if entering motherhood at all, they tend to do so relatively early. Individuals with bipolar disorder, eating disorder, or personality disorder have even lower first-birth rates. Except for eating disorder, the lowest fertility compared to those without a mental disorder is again seen at the higher ages. Among women with schizophrenia, the first-birth rate is reduced by as much as 48% up to age 23 and about 85% from age 27. The age difference in the effect coefficients is larger for bipolar disorder and personality disorder than for schizophrenia and eating disorder. Stated differently, it is less clear that women with the latter diseases have a relatively early first birth. (See further comments on timing below, based on simulations.)

(Figure 1 and Table 3 about here)

The associations between mental disorders and first births are quite similar for men, but generally more negative or (at the lowest ages) less clearly positive. Note that controlling for sociodemographic family-of-origin variables had little impact (shown for first births in Appendix Table A3.2), so they were for simplicity left out in all final estimations.

There are also negative relationships between mental disorders and second- and third-birth rates, for both sexes (Table 2). Among women, the relationships with depression or anxiety are stronger for second births than first births (all ages pooled); among men, there is little difference. Otherwise, the relationships with mental disorders are weaker for second births than for first births, and even weaker for third births.

Estimates from an analysis of fourth and fifth births, which are quite uncommon, are displayed in Appendix Table A3.3. There are no negative relationships with mental disorders;

instead, some estimates point in the opposite direction. Fourth and fifth births were ignored in the remaining analysis, except in the simulation.

Simulations

The estimated effects on parity- and age-specific birth rates were translated into more intuitively informative fertility measures by means of simulation. Note that the underlying assumption in these simulations was that the individuals (very hypothetically) have at least one consultation for, for example, depression every year throughout the entire reproductive age span, but no consultation for the other disorders.

For women without any of the mental disorders, the simulated average number of births up to age 45 is 1.60 (Table 4). The number would have been slightly higher if sixth and higher-order births and multiple births had not been ignored. In comparison, the national total fertility rate for 2018 (including, of course, individuals with mental disorders) was 1.56 (Statistics Norway 2023a). The simulated childlessness at age 45 is 21.0%, and the average age at first birth among those having become mothers by age 45 is 30.7 years. Official fertility statistics does not include any corresponding numbers.

(Table 4 about here)

Among women with depression, the average number of children at age 45 is 1.34 and the proportion childless is 30.2. The average age at birth (30.2) is slightly lower than for mothers without a mental disorder – as one would expect from the estimates from the age-stratified models. Furthermore, and also in line with these estimates, the proportion childless

at age 25 is actually *lower* than among women without a mental disorder, while the proportion is higher from age 30. The same age pattern in childlessness is seen among those with anxiety, who also end up with almost the same number of children as the depressed.

The average number of children at age 45 among women with schizophrenia is 0.36, while 77.3% are childless. Among women with the other mental disorders, the number of children varies between 1.05 and 1.35. All groups except those with eating disorder have a slightly lower age at first birth than those without a disorder.

Among men without a mental disorder, the average number of children at age 45 is 1.41 and childlessness is 29.6%. In comparison, the national total fertility rate in 2018 was 1.41 (Statistics Norway 2023b), and if the contribution from ages 45-54 was subtracted, it was 1.35.[41] The figures for men with depression are 0.90 children and 49.2% childless, and those for men with schizophrenia are 0.16 children and 89.2% childless. In other words, the number of children is reduced from 1.60 among women without a mental disorder to 1.34 (i.e., 0.26) among those with depression, while the corresponding reduction among men is from 1.41 to 0.90 (i.e., 0.51), which is much more both in absolute and relative sense.

Men with anxiety, schizophrenia or personality disorder have a slightly lower age at first birth than those without a disorder, while those with eating disorder enter fatherhood later. As among women, the proportion childless at age 25 (which is very early in men's reproduction) is lower among those with depression or anxiety than among those without a disorder, while childlessness otherwise is most common among those with one of the disorders.

A similar simulation based on estimates from a model not including the disorder indicators gave a completed fertility of 1.56 for women and 1.37 for men (not shown in tables), which accords well with the national total fertility rates. When we left out 3-month

observations where at least one disorder indicator was one before the estimation, the corresponding numbers were 1.61 and 1.42 (which as expected are very close to the results for 'none of the disorders' reported above). Thus, one may say that, if (periods with) mental disorders very hypothetically had been eliminated, completed fertility would have been 0.05 higher for both sexes. Depression and anxiety contribute 70% to this increase (not shown).

Alternative Disease Indicators

Although it would seem reasonable to expect a less reduced fertility among individuals in contact with only primary – and not specialized - health care, there were only weak indications of such a pattern; the results were quite similar when we (like in earlier research) used information only about specialized health care. The time lag used for our health indicators matters more; the relationships with fertility were weaker if we considered the number of consultations over a period *before* t-2. The lowest fertility was observed among those who had consultations for the mental disorder both in t-2 and earlier. See details in Appendix 4.

The Role Played by Partnership, Education, and Income

According to models not stratified by age, the associations between mental disorders and birth rates become somewhat weaker when an indicator of partnership status is added, although almost all the associations that are significant in the simplest model remain significant. See Appendix Table A5.1.

The next step was to add also education level, school enrolment and income to the models. By and large, this further weakened the associations, but less for eating disorder than

for the other disorders. Again, almost all associations that were significant according to the simplest model remained significant, the most important exception being the associations between women's first-birth rates and depression or anxiety.

Turning to the age-specific models for first births (Figure 1, based on Appendix Table A5.2), there are less positive relationships between depression and fertility at low age when all the additional variables are included, while the relationships are less negative at the higher ages (and more because of the socioeconomic factors than partnership). In other words, the tendency among depressed women to become mothers relatively early seems to be partly due to the included sociodemographic variables. A similar development appears for the other disorders (except eating disorder), and for both sexes: Inclusion of the sociodemographic factors makes the associations at the lower ages less positive or more negative, while they become less negative at the higher ages.[42]

To make an attempt to quantify the contribution from the sociodemographic variables, let us return to the overall associations, without regard to age, and use bipolar disorder as an example: The coefficient in the model for women's first birth changes from 0.74 (i.e., the odds of a first birth are reduced by 26%) in the simplest model, to 0.76 when partnership is controlled for, and to 0.86 (i.e. odds reduced by 14%) when also the socioeconomic indicators are added. However, one cannot conclude that these variables in total account for 46% of the association (1-0.14/0.26), given the scaling problem arising in logistic models. Their contribution is somewhat larger. As an alternative assessment, we estimated corresponding linear probability models, which involve additive rather than multiplicative effects on fertility, and where there is not a similar scaling problem. Inclusion of sociodemographic variables in these models reduced the association by 56% (Appendix Table A5.3). Similar calculations for other disorders and parity transitions, and for both sexes, show that the reduction in the effect coefficients varies between 33% and 77%.

Family Fixed-effects Models

When we turned from discrete-time hazard models to Cox models, the estimated effects of the disorders were almost unchanged (shown for first births in Appendix Table A5.4). Leaving out those not having a same-sex sibling under exposure for the same fertility transition in the study period strongly reduced the sample, but the coefficients were not very different (Appendix Table A5.4). This indicates that results from a sibling analysis based on a 'sibling sample' have more general relevance.

When family fixed effects were added (Table 5), the associations between first-birth rates and anxiety, bipolar disorder (among women), and eating disorder were no longer significant (while, as pointed out above, inclusion of some observed family-of-origin characteristics did not matter). After further addition of partnership and socioeconomic factors, the associations with depression (in women) and personality disorder (in men) were no longer significant either. In the sibling analysis of second births, only the associations with depression (in both sexes) and anxiety (in women) are significant – and remain so when the sociodemographic factors are added - while no associations with third-birth rates are significant.

(Table 5 about here)

Discussion and Conclusion

Depression and Anxiety

Our analysis confirms the reduced fertility among men with depression reported elsewhere (Power et al. 2013), and also accords with investigations showing relatively low fertility

among men with internalizing behaviour (Jokela et al. 2014; Evensen and Lyngstad 2020). In fact, a negative association with depression is seen for all the first three parity transitions, and according to simulations from the model estimates, men who – very hypothetically - are depressed throughout the reproductive age span on average have only 0.90 children by age 45 (and 49% are childless), as opposed to 1.41 (30% childless) if they have none of the six mental disorders under study. In these simulations, it is assumed that the effects of depression are as estimated from the main models, where the depression indicators refer to the situation two years earlier. Our analysis based on alternative indicators suggests that the effects of being continuously depressed likely are stronger.

First- and second-birth rates are significantly reduced for men with depression even in a family fixed-effects analysis, which controls for constant characteristics shared by siblings. The associations with anxiety are somewhat weaker, and do not survive the inclusion of family fixed effects.

However, while earlier studies have provided less conclusive evidence of a fertility-reducing effect of depression and anxiety among women, our analysis shows that both disorders are associated with lower first-, second-, and third-birth rates for them also (and some of these associations remain significant in the sibling analysis). According to the simulations, the number of children among women with these disorders is 1.34 and 1.39, respectively, as opposed to 1.60 among those without a mental disorder. This difference is smaller than that among men, both in absolute and relative sense.

Among those who have become parents, the average age at first birth seems to be slightly lower among those with depression or anxiety than among individuals who do not have a mental disorder. (The wording is cautious because of the small differences, and because the conclusion hinges on comparison of estimates from logistic models estimated

from different age groups). However, compared to those without a mental disorder, individuals with depression or anxiety do not only have less reduced first-birth rates at low age than at high age; at low age, they have outright higher first-birth rates than this comparison group - and the proportion childless at age 25 is thus lower. Some earlier studies have suggested a similar pattern (Laursen and Munk-Olsen 2010; Jokela et al. 2014; Evensen and Lyngstad 2020).

The Less Common Mental Disorders

As reported also by others (Laursen and Munk-Olsen 2010; Bundy et al. 2011; Power et al 2013), individuals with schizophrenia have very low fertility, although it is less reduced among those who have already had a child. According to the simulations, women and men with schizophrenia have 0.36 and 0.16 children, respectively, at age 45, and the proportions childless are 77% and 89%. Among those who become parents, the average age at first birth is relatively low.

Bipolar disorder, eating disorder and personality disorder are also more strongly linked to fertility than are depression and anxiety, but fertility is less markedly reduced than in the case of schizophrenia. While the associations are significant both in the models for first births and, to lesser extent, higher-order births in the main analysis, not even all the associations with the first-birth rates are significant according to the sibling analysis. Women with bipolar disorder and men with personality disorder seem to have relatively early first births, while individuals with eating disorder tend to become parents later.

Mediation or Selection

Leaving the timing issue aside for the moment, the main picture is that the mental disorders are associated with reduced fertility. A quite small part of this association is due to a lower proportion in a union among those with mental disorders.[43] The coefficients become more markedly changed when also education level, school enrolment and income (which are linked with partnership status in addition to affecting fertility for other reasons) are added to the models. However, almost all relationships that are significant in the simplest models remain significant, two exceptions being the associations between depression or anxiety and first-birth rates among women. Unfortunately, the interpretation is not clear. Education and income may be joint determinants of mental health and fertility, but they may also be mediators, in which case we are 'tapping out' some of the causal effect of mental health on fertility by controlling for them. The associations between mental disorders and subsequent fertility that remain after the sociodemographic factors have been controlled for may reflect a variety of causal effects (and selection; see below). One possible explanation mentioned above is that women and men with a mental disorder may not find the strength to raise a(nother) child.

With regard to timing differences, one might expect that fewer years of school enrolment among individuals with mental disorder contributes to a relatively early age at first birth among those who become parents. A low income might have a similar effect among women, while the opposite perhaps is more likely for men. Our comparison of estimates from models with and without control for sociodemographic factors indeed supports the idea that low education and income among women contribute to push the age at first birth down (except with respect to eating disorder). The fact that inclusion of these factors has the same impact on the age-specific estimates for men may suggest that school enrolment plays a larger role as a selection factor or mediator than income.

What can explain the more negative associations between mental disorders (except eating disorder) and first-birth rates at the higher ages than at the lower ages that remain even after control for the mentioned factors, and which means that the average age at first birth is slightly lower than among those without a mental disorder? One explanation may be that some prefer to have their child relatively early because they suspect that their health will further deteriorate. Additionally, the pattern may reflect a selection mechanism: As the age increases, those who are still childless may to a larger extent include individuals with a particularly severe version of the disorder. It is also possible that the relatively early birth is a result of a higher chance of unplanned pregnancies, as indicated in some studies (James-Hawkins et al. 2014; Hall et al. 2014).

The generally less negative associations with mental disorders at higher parities may reflect a selection similar to the one just mentioned: Individuals with the least severe disorders are more likely to have become parents, and thus be among those under exposure for having a second or higher-order child. Alternatively, the step into parenthood may be seen as a particularly daunting transition, whereas having an additional child involves less challenging life changes.[44]

Relevance for Fertility at the Aggregate Level

The disorders may be considered as contribution quite modestly to the national fertility level, in the sense that a hypothetical elimination of them increases completed fertility by only 0.05 according to our simulation. Similarly, a doubling of their prevalence over some future years - which is not entirely implausible given the apparent increase in depression and anxiety in the recent decades - would reduce fertility by 0.05. However, the current prevalence is probably larger than indicated by our health care data. In that case, a hypothetical elimination

or doubling would have larger impact, although perhaps not proportionally larger, as fertility may be less affected among those not seeking professional help for their mental problems. Anyway, studies of mental health differences in fertility are potentially valuable even if actual changes in mental health matter quite little for the national fertility (see Introduction).

Strengths and Weaknesses

Our disorder indicators are based on high-quality register data on not only specialized health care, but also – unlike earlier investigations - primary care. We therefore capture a larger group of individuals with these disorders, which improves the precision of the key estimates and makes assessments of the aggregate-level fertility implications more reasonable. Also, because those using only primary health care likely have less severe symptoms, one might expect this larger group to be more representative of *all* those who have the mental disorders under study - including those who have not sought professional help for them. However, we got quite similar results if only the data on specialized health care were used, so it seems that those using only primary care in fact are no less affected in terms of fertility than those using specialized care. It may, of course, nevertheless be the case that those who do not consult any physicians have a fertility more similar to that of individuals without mental problems.

Another strength of our study is that we consider the associations between indicators of health at a certain time and *subsequent* birth rates, rather than for example associations between the number of births up to a relatively high age and having ever been registered with a disease before that age (Powers et al. 2013), which to a larger extent reflects a two-way causation. Furthermore, in addition to analysing differences in birth rates, we show how these differences produce differences in completed fertility. The latter has not been done in previous studies of birth rates among individuals with mental problems. Additionally, we

consider how fertility effects vary across parity (only done earlier by Laursen and Munk-Olsen 2010) and sex (not always done), and we explore how sensitive the results are to alternative lags in the mental health indicators (not considered earlier). We have also shown that most of the effects on the first-birth rates remain in a sibling comparison analysis (not done earlier).

One limitation, shared by many other studies, is that use of health care not only reflects a health problem, but also the inclination to consult health personnel for this problem. In principle, the threshold for seeking professional help may be linked to characteristics affecting fertility – negatively (in which case the effects of poor mental health on fertility would be less negative than suggested by our estimates) or positively; the direction of this bias is not obvious.

Another weakness in all such research is that several factors that cannot be adequately controlled for may affect mental health (or the use of health care for a mental health problem) as well as fertility. We have only controlled for some sociodemographic characteristics of the family of origin, which did not matter, and – in parts of the analysis – all constant characteristics shared by siblings, which mattered more. We have also controlled for some sociodemographic characteristics of the index person, although these may be causally intermediate rather than selection factors. While the remaining associations may partly reflect causal effects of mental health on fertility, they may also be a result of various characteristics that are unique to each sibling and affect both health and fertility. For example, personality, certain experiences in young adulthood, or a fecundity problem that sets in early may affect both mental health and subsequent birth rates.

The analysis is based on data from one particular country, where people on average have high purchasing power and are supported by a generous welfare state. It is possible that the link between mental disorders and fertility is somewhat different in other settings.

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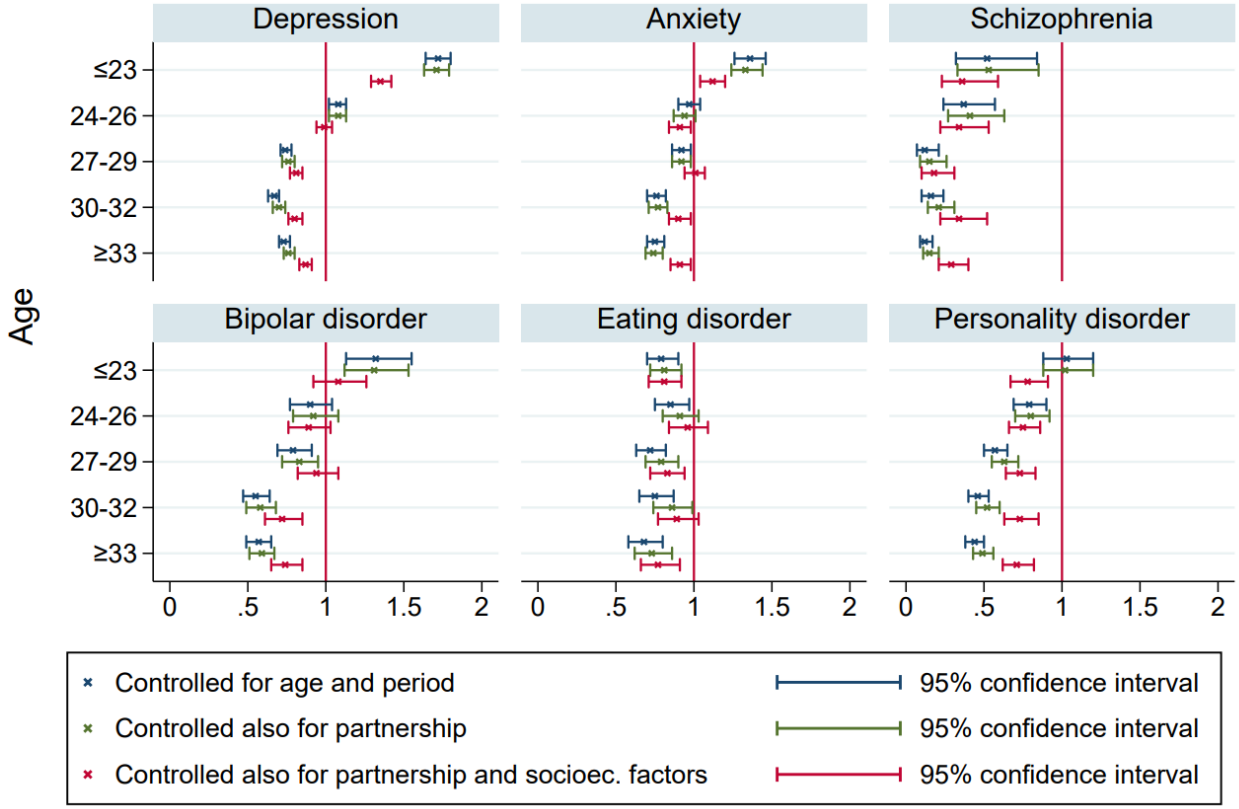
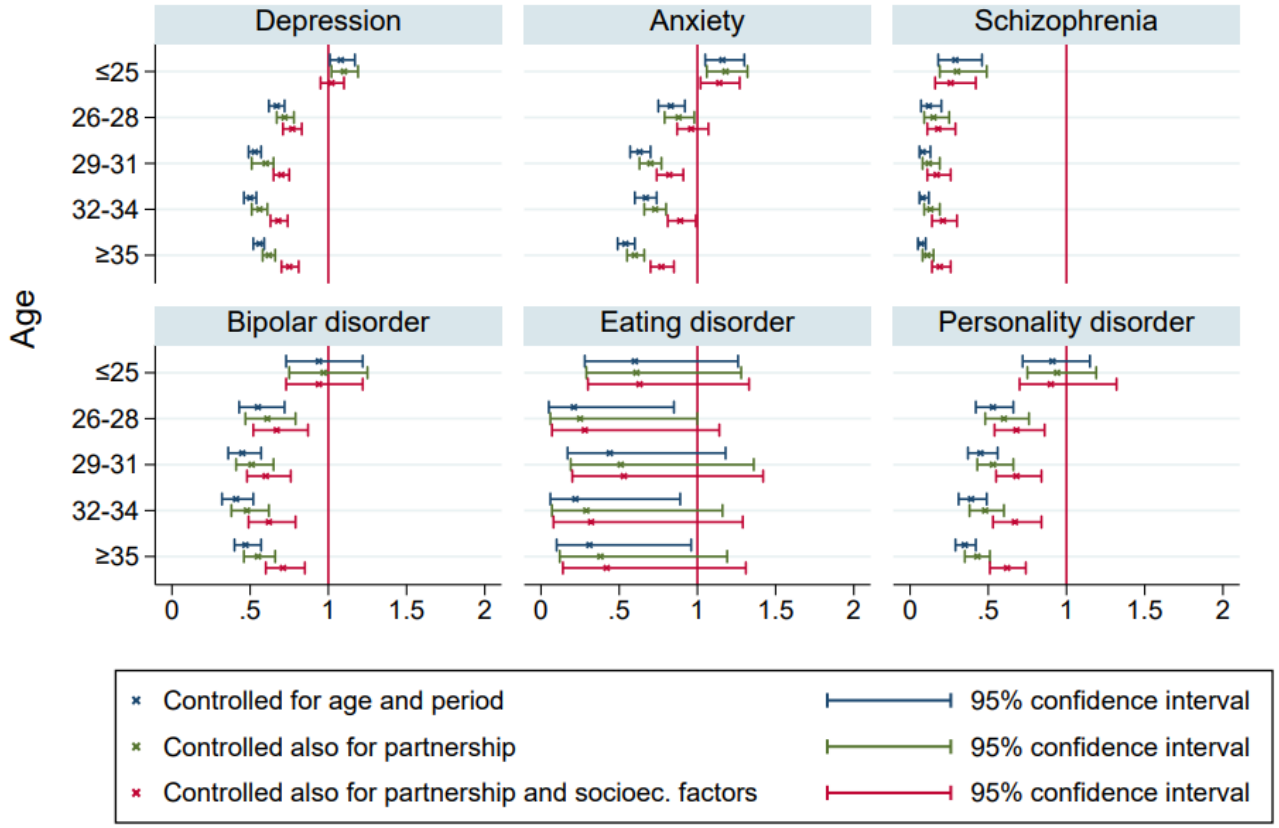
Figure 1. Effects (odds ratios with 95% CI) of disorder indicators in discrete-time hazard models for first-birth rates, by age, according to different models. Norwegian women and men 2010-2018 [a]

Women:



Men:



Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] Age and period are included with one-year categories. The graphs are based on estimates shown in Appendix Table A5.2.

Table 1. Definitions of mental disorder indicators based on ICPC-2 codes in primary health care and ICD-10 codes in specialized health care.

Dichotomous disorder indicators	ICD-10	ICPC-2
Depression	F32, F33	P76
Anxiety	F41	P74
Schizophrenia	F20	P72
Bipolar disorder	F31	P73
Eating disorder	F50	P86
Personality disorder	F60, F61	P80

Table 2. Effects (odds ratios with 95% CI) of disorder indicators in discrete-time hazard models for first-, second- and third-birth rates. Norwegian women and men 2010-2018.

Panel A: Women

	First births			Second births			Third births		
Dichotomous disorder indicators	Proportion of exposure time with this disorder (%)	Number of first births among those with this disorder	Effects on first-birth rates [a]	Proportion of exposure time with this disorder	Number of second births among those with this disorder	Effects on second-birth rates [b]	Proportion of exposure time with this disorder	Number of third births among those with this disorder	Effects on third-birth rates [b]
Depression	5.59	9161	0.91*** (0.89-0.92)	7.39	6335	0.70*** (0.69-0.72)	5.69	2656	0.87*** (0.83-0.90)
Anxiety	2.28	3883	0.91*** (0.88-0.94)	3.19	2681	0.76*** (0.73-0.79)	2.35	1130	0.89*** (0.83-0.94)
Schizophrenia	0.31	109	0.17*** (0.14-0.20)	0.21	56	0.36*** (0.28-0.47)	0.06	19	0.90 (0.57-1.42)
Bipolar disorder	0.61	908	0.74*** (0.69-0.79)	0.92	572	0.61*** (0.56-0.66)	0.57	217	0.85* (0.74-0.97)
Eating disorder	0.90	1069	0.78*** (0.74-0.83)	0.54	512	0.88** (0.81-0.97)	0.28	183	1.11 (0.96-1.28)
Personality disorder	0.84	1031	0.57*** (0.53-0.60)	1.00	625	0.69*** (0.64-0.75)	0.52	257	0.97 (0.86-1.10)
Number of births			162065			141826			54582
Exposure time (million person-quarters)			13.536			3.784			6.431

Panel A: Men

	First births			Second births			Third births		
Dichotomous disorder indicators	Proportion of exposure time with this disorder (%)	Number of first births among those with this disorder	Effects on first-birth rates [a]	Proportion of exposure time with this disorder	Number of second births among those with this disorder	Effects on second-birth rates [b]	Proportion of exposure time with this disorder	Number of third births among those with this disorder	Effects on third-birth rates [b]
Depression	3.03	3613	0.62*** (0.60-0.64)	3.83	2735	0.62*** (0.60-0.65)	2.81	1272	0.90*** (0.85-0.96)
Anxiety	1.46	1956	0.70*** (0.67-0.74)	1.84	1351	0.67*** (0.64-0.71)	1.18	542	0.88** (0.81-0.96)
Schizophrenia	0.58	125	0.09*** (0.08-0.11)	0.24	57	0.32*** (0.25-0.42)	0.07	13	0.44** (0.26-0.77)
Bipolar disorder	0.38	378	0.50*** (0.46-0.56)	0.46	286	0.69*** (0.61-0.77)	0.32	119	0.83* (0.69-0.99)
Eating disorder	0.05	18	0.38*** (0.24-0.60)	0.02	12	0.84 (0.47-1.49)	0.01	<10 [c]	0.53 (0.17-1.66)
Personality disorder	0.51	426	0.45*** (0.40-0.49)	0.57	286	0.55*** (0.49-0.62)	0.30	120	0.84 (0.70-1.01)
Number of births			160278			134863			52778
Exposure time (million person-quarters)			17.303			3.690			5.140

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The model also included age and period in one-year categories.
[b] The model also included age and period in one-year categories, as well as duration in the following categories (months): <6, 6-8, 9-11, 12-17, 18-23, 24-29, 30-35, 36-47, 48-59, 60-71, 72-83, 84-95, 96-107, 108-119, and ≥ 120.
[c] Numbers below 10 cannot be specified for data protection reasons

Table 3. Effects (odds ratios with 95% CI) of disorder indicators in discrete-time hazard models for first-birth rates, by age. Norwegian women and men 2010-2018.

Panel A: Women

Dichotomous disorder indicators	Age ≤ 23	Age 24-26	Age 27-29	Age 30-32	Age ≥ 33
Depression	1.72*** (1.64-1.80)	1.08** (1.02-1.13)	0.74*** (0.71-0.78)	0.67*** (0.63-0.70)	0.73*** (0.70-0.77)
Anxiety	1.36*** (1.26-1.46)	0.97 (0.90-1.04)	0.92* (0.86-0.98)	0.76*** (0.70-0.82)	0.75*** (0.70-0.81)
Schizophrenia	0.52*** (0.32-0.84)	0.37*** (0.24-0.57)	0.12*** (0.07-0.21)	0.16*** (0.10-0.24)	0.12*** (0.09-0.17)
Bipolar disorder	1.32*** (1.13-1.55)	0.90 (0.77-1.04)	0.79*** (0.69-0.91)	0.55*** (0.47-0.64)	0.57*** (0.49-0.65)
Eating disorder	0.79*** (0.70-0.90)	0.85* (0.75-0.97)	0.72*** (0.63-0.82)	0.75*** (0.65-0.87)	0.68*** (0.58-0.80)
Personality disorder	1.03 (0.88-1.20)	0.79*** (0.69-0.90)	0.57*** (0.50-0.65)	0.46*** (0.40-0.53)	0.44*** (0.38-0.50)
Number of first births	26976	31583	38949	32396	32161

Panel B: Men

Dichotomous disorder indicators	Age ≤ 25	Age 26-28	Age 29-31	Age 32-34	Age ≥ 35
Depression	1.08* (1.01-1.17)	0.67*** (0.62-0.72)	0.53*** (0.49-0.57)	0.50*** (0.46-0.54)	0.56*** (0.52-0.59)
Anxiety	1.16** (1.05-1.30)	0.83*** (0.75-0.92)	0.63*** (0.57-0.70)	0.67*** (0.60-0.74)	0.54*** (0.49-0.60)
Schizophrenia	0.29*** (0.18-0.46)	0.12*** (0.07-0.20)	0.08*** (0.06-0.13)	0.08*** (0.06-0.12)	0.07*** (0.05-0.10)
Bipolar disorder	0.94 (0.73-1.22)	0.55*** (0.43-0.72)	0.45*** (0.36-0.57)	0.41*** (0.32-0.52)	0.47*** (0.40-0.57)
Eating disorder	0.60 (0.28-1.26)	0.21* (0.05-0.85)	0.44 (0.17-1.18)	0.22* (0.06-0.89)	0.31* (0.10-0.96)
Personality disorder	0.91 (0.72-1.15)	0.53*** (0.42-0.66)	0.45*** (0.37-0.56)	0.39*** (0.31-0.49)	0.35*** (0.29-0.42)
Number of first births	26899	31613	36879	28948	35940

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The model also included age (within the broader age group) and period in one-year categories.

Table 4. Simulations of fertility based on estimated effects in birth rate models.

	Women					
	Average number of children at age 45	Percent childless at age 25	Percent childless at age 30	Percent childless at age 35	Percent childless at age 45	Average age at first birth among those who have at least one child at age 45
None of the disorders	1.60	88.1	58.2	32.5	21.0	30.7
Depression	1.34	83.6	60.7	40.4	29.1	30.2
Anxiety	1.39	86.3	59.8	38.4	27.5	30.4
Schizophrenia	0.36	94.6	88.2	81.4	77.3	30.4
Bipolar disorder	1.17	86.9	63.6	45.9	35.7	30.3
Eating disorder	1.35	90.0	66.2	43.5	32.2	31.0
Personality disorder	1.05	89.1	69.9	53.7	44.2	30.6

	Men					
	Average number of children at age 45	Percent childless at age 25	Percent childless at age 30	Percent childless at age 35	Percent childless at age 45	Average age at first birth among those who have at least one child at age 45
None of the disorders	1.41	94.2	72.2	46.6	29.6	32.5
Depression	0.90	93.7	79.5	63.3	49.2	32.5
Anxiety	1.01	93.2	76.4	57.7	45.2	32.0
Schizophrenia	0.16	98.3	95.5	92.1	89.2	32.3
Bipolar disorder	0.81	94.3	82.2	67.8	54.9	32.6
Eating disorder	0.59	96.4	88.2	77.6	67.6	33.0
Personality disorder	0.71	94.5	82.6	69.3	59.3	32.1

Notes:
The simulations were based on estimated effects in age-stratified equations for first births (shown in Table 3), equations for second and third births (Table 2), and an equation for fourth and fifth birth (Appendix Table A3.3). Births of sixth or higher order were ignored. All births were assumed to be singleton births. Calendar year was set to 2018 in the simulations. See main text for additional details about the simulation procedure.

Table 5. Effects (hazard ratios with 95% CI) of disorder indicators in sibling-comparison Cox hazard models for first-, second-, and third-birth rates. Norwegian women and men 2010-2018 who have a same-sex sibling under exposure for the same transition [a]

Panel A: Women

Dichotomous disorder indicators	Effects on first-birth rates	Effects on second-birth rates	Effects on third-birth rates
Depression	0.86*** (0.79-0.93)	0.78** (0.66-0.92)	1.00 (0.82-1.23)
Anxiety	1.04 (0.92-1.18)	0.61*** (0.48-0.79)	0.83 (0.62-1.12)
Schizophrenia	0.23*** (0.13-0.41)	0.69 (0.14-3.43)	0.45 (0.10-2.06)
Bipolar disorder	0.79 (0.62-1.02)	0.88 (0.54-1.41)	1.01 (0.55-1.86)
Eating disorder	0.90 (0.73-1.12)	1.01 (0.59-1.74)	0.71 (0.33-1.51)
Personality disorder	0.67*** (0.53-0.84)	0.85 (0.52-1.40)	1.21 (0.63-2.31)
Control also for partnership status and socioeconomic factors:			
Depression	0.94 (0.86-1.03)	0.84* (0.71-1.00)	0.99 (0.81-1.22)
Anxiety	1.07 (0.94-1.23)	0.62*** (0.48-0.81)	0.84 (0.62-1.14)
Schizophrenia	0.26*** (0.14-0.49)	0.83 (0.15-4.45)	0.42 (0.09-1.95)
Bipolar disorder	0.80 (0.62-1.05)	0.94 (0.58-1.52)	1.01 (0.55-1.86)
Eating disorder	1.00 (0.79-1.25)	0.99 (0.57-1.73)	0.72 (0.33-1.53)
Personality disorder	0.74* (0.58-0.94)	1.09 (0.65-1.83)	1.26 (0.65-2.43)
Number of births	70522	40669	18609

Panel B: Men

Dichotomous disorder indicators	Effects on first-birth rates	Effects on second-birth rates	Effects on third-birth rates
Depression	0.71*** (0.63-0.80)	0.65** (0.50-0.84)	0.85 (0.63-1.14)
Anxiety	0.86 (0.74-1.01)	0.95 (0.65-1.39)	0.88 (0.53-1.46)
Schizophrenia	0.13*** (0.08-0.22)	1.04 (0.36-3.04)	-
Bipolar disorder	0.49*** (0.35-0.69)	0.70 (0.29-1.71)	0.66 (0.25-1.73)
Eating disorder	0.32 (0.08-1.20)	-	-
Personality disorder	0.53** (0.38-0.74)	0.47 (0.21-1.04)	1.12 (0.41-3.06)
Control also for partnership status and socioeconomic factors:			
Depression	0.84** (0.74-0.96)	0.75* (0.57-0.98)	0.84 (0.62-1.14)
Anxiety	0.98 (0.83-1.17)	1.05 (0.72-1.54)	0.89 (0.53-1.49)
Schizophrenia	0.24*** (0.14-0.39)	1.40 (0.47-4.12)	-
Bipolar disorder	0.60** (0.41-0.88)	0.78 (0.32-1.95)	0.71 (0.27-1.86)
Eating disorder	0.32 (0.08-1.18)	-	-
Personality disorder	0.70 (0.48-1.01)	0.50 (0.22-1.12)	1.13 (0.41-3.13)
Number of births	75318	37956	16836

Notes: *p<0.05; ** p<0.01; *** p<0.001

-could not estimate because of too few observations

[a] The Cox models included year and (if relevant) duration since last previous birth and sociodemographic control variables, with the same categories as in the other analysis (see notes to Table 2 and Appendix Table A5.1).

SUPPLEMENTARY MATERIAL

Contents

Appendix 1: A multilevel-multiprocess model

As a supplement to the main analysis, we estimated a multilevel-multiprocess model that included seven equations – one for first-birth rates in each of five age groups, one for second-birth rates, and one for third-birth rates. These equations were as in the main analysis, except that the same individual-level normally distributed random term was added to each of them. The model was estimated from a 50% random sample because of constraints in the aML software (Lee and Panis 2003) that was used.

It has been argued that such joint modelling of first-, second-, and third-birth rates may give different estimates of the coefficients ("effects") for variables that are strongly linked to the first-birth rates, such as the educational level measured at a relatively high age (Kravdal 2001; Kravdal and Rindfuss 2008). The idea is that individuals have a set of unobserved characteristics with impact on fertility, for example a weak or strong interest in childbearing, or low or high fecundity. Let us, for simplicity, refer to these characteristics as one unobserved fertility determinant – scaled so that a high value of this determinant leads to high fertility. The determinant is represented by the random term, and its value at the start of the reproductive age span is the same, on average, for those who end up with high education and those who end up with low education. However, those among the better educated who ever become parents - and especially those who have their first child early - in spite of all the factors that tend to give the better-educated low fertility, must have higher-than-average value of the unobserved determinant, which also affects later parity transitions positively. That being said, one study showed that joint modelling has little impact on the results when the current (i.e., time-varying) educational level is in focus, rather than the education attained by a high age (Kravdal 2007).

It turned out that the effects of our six time-varying indicators of mental disorder (lagged two years) were unchanged when we estimated a multilevel-multiprocess. This was the case both for women (see Appendix Table A1.1) and men (not shown). The result may reflect that, although the disorders are linked with the chance of having a first child, they are not very strongly associated with the first-birth timing.

References

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Kravdal, Ø., & Rindfuss, R. R. (2008). Changing relationships between education and fertility: A study of women and men born 1940 to 1964. *American Sociological Review*, *73*(5), 854-873.

Appendix Table A1.1. Effects (log odds with standard errors) of disorder indicators in discrete-time hazard models for first-, second- and third-birth rates. 50% random sample of Norwegian women 2010-2018

Dichotomous disorder indicators	Separate models [a]	Joint models [b]
	Effects on first-birth rates, age 17-23	
Depression	0.527*** (0.032)	0.527*** (0.033)
Anxiety	0.355*** (0.052)	0.356*** (0.052)
Schizophrenia	-0.278 (0.303)	-0.278 (0.303)
Bipolar disorder	0.290* (0.115)	0.290* (0.115)
Eating disorder	-0.224* (0.091)	-0.224* (0.091)
Personality disorder	-0.039 (0.115)	-0.039 (0.115)
	Effects on first-birth rates, age 24-26	
Depression	0.105** (0.035)	0.106** (0.035)
Anxiety	-0.068 (0.054)	-0.067 (0.055)
Schizophrenia	-0.754** (0.288)	-0.754** (0.288)
Bipolar disorder	-0.224 (0.116)	-0.223 (0.117)
Eating disorder	-0.137 (0.088)	-0.137 (0.089)
Personality disorder	-0.329** (0.102)	-0.328*** (0.102)
	Effects on first-birth rates, 27-29	
Depression	-0.311*** (0.036)	-0.309*** (0.036)
Anxiety	-0.165** (0.051)	-0.164** (0.051)
Schizophrenia	-1.975*** (0.384)	-1.977*** (0.384)
Bipolar disorder	-0.222* (0.098)	-0.222* (0.098)
Eating disorder	-0.338*** (0.096)	-0.338*** (0.096)
Personality disorder	-0.500*** (0.095)	-0.500*** (0.096)
	Effects on first-birth rates, age 30-32	
Depression	-0.468*** (0.040)	-0.468*** (0.040)
Anxiety	-0.343*** (0.056)	-0.344*** (0.057)
Schizophrenia	-2.011*** (0.334)	-2.015*** (0.335)
Bipolar disorder	-0.407*** (0.108)	-0.408*** (0.108)
Eating disorder	-0.235* (0.106)	-0.237* (0.106)
Personality disorder	-0.692*** (0.105)	-0.693*** (0.105)
	Effects on first-birth rates, age 33-45	
Depression	-0.364*** (0.036)	-0.365*** (0.036)
Anxiety	-0.306*** (0.053)	-0.307*** (0.053)
Schizophrenia	-2.118*** (0.237)	-2.123*** (0.237)
Bipolar disorder	-0.609*** (0.103)	-0.612*** (0.103)
Eating disorder	-0.451*** (0.120)	-0.452*** (0.120)
Personality disorder	-0.875*** (0.102)	-0.878*** (0.102)
	Effects on second-birth rates	
Depression	-0.343*** (0.019)	-0.343*** (0.019)
Anxiety	-0.263*** (0.028)	-0.263*** (0.028)
Schizophrenia	-0.935*** (0.162)	-0.941*** (0.163)
Bipolar disorder	-0.493*** (0.058)	-0.495*** (0.059)
Eating disorder	-0.140* (0.063)	-0.141* (0.063)
Personality disorder	-0.336*** (0.057)	-0.337*** (0.057)
	Effects on third-birth rates	
Depression	-0.215*** (0.030)	-0.215*** (0.030)
Anxiety	-0.128** (0.040)	-0.128** (0.044)

Schizophrenia	0.063	(0.309)	0.061	(0.310)
Bipolar disorder	-0.077	(0.091)	-0.077	(0.091)
Eating disorder	0.180	(0.103)	0.181	(0.103)
Personality disorder	0.026	(0.087)	0.027	(0.087)

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The models and the samples were as in Table 2 and Table 3, except for the 50% random selection.
[b] The model and sample was as for the separate models, except that a random term was added to each of the equations, which were jointly estimated. The estimated standard deviation of the random term was 0.140 (0.030).

Appendix 2: A comment on the estimation of sibling models

In principle, sibling model estimates may be biased if an individual's fertility is influenced by whether the *sibling* has a mental disorder. Such an effect, which may reflect that a family member's mental disorder affects various aspects of the family environment, has indeed been suggested. More specifically, Power et al. (2013) found that sisters of individuals with schizophrenia or bipolar disorder and siblings of individuals with depression or substance abuse had slightly increased fertility, while brothers of individuals with schizophrenia or autism had reduced fertility. Bundy et al. (2011) reported an adverse effect of having a sibling with schizophrenia. However, these associations may reflect not only the mentioned causal effect, but also other mechanisms.

If the effect of the sibling's disease has the same sign as the effect of the person's own disease (although it likely is considerably weaker), there is a downward bias in the sibling model estimates of the latter effect (Sjölander et al. 2016). To get an impression of the relevance of this issue, we estimated some discrete-time hazard models including indicators of both own and oldest sibling's mental disorders. The oldest sibling may be a brother or a sister. Control for the sex had no impact on the results. The overall picture is that there are some rather weak associations – perhaps partly reflecting a causal effect such as mentioned - which go in the same direction as those between fertility and the person's own mental disorder (shown for first births in Appendix Table A2.1). This indicates that our sibling model estimates may be conservative.

References

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Sjölander, A., Frisell, T., Kuja-Halkola, R., Öberg, S., & Zetterqvist, J. (2016). Carryover effects in sibling comparison designs. *Epidemiology*, *27*(6), 852-858.

Appendix Table A2.1. Effects (odds ratios with 95% CI) of indicators of own and oldest sibling's disorders in discrete-time hazard models for first-birth rates. Norwegian women and men 2010-2018 who have at least one sibling [a]

Dichotomous disorder indicators	Women	Men
Own disorder		
Depression	0.91*** (0.88-0.95)	0.62*** (0.60-0.64)
Anxiety	0.91*** (0.88-0.95)	0.71*** (0.68-0.74)
Schizophrenia	0.17*** (0.14-0.21)	0.09*** (0.08-0.11)
Bipolar	0.75*** (0.70-0.80)	0.51*** (0.46-0.57)
Eating disorder	0.76*** (0.72-0.81)	0.35*** (0.21-0.58)
Personality disorder	0.56*** (0.53-0.60)	0.43*** (0.39-0.48)
Sibling's disorder		
Depression	1.01 (0.98-1.04)	0.95*** (0.93-0.98)
Anxiety	1.02 (0.96-1.06)	0.95* (0.92-0.99)
Schizophrenia	0.86** (0.78-0.94)	0.78*** (0.71-0.86)
Bipolar disorder	1.01 (0.93-1.08)	0.96 (0.89-1.03)
Eating disorder	0.93 (0.85-1.03)	0.94 (0.85-1.04)
Personality disorder	0.93* (0.87-1.00)	0.90** (0.83-0.96)
Number of births	152174	151185

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The models and the samples were as in Table 2, except that those without a sibling were excluded, and a disorder indicator for the sibling (the oldest if there was more than one) was included.

Appendix 3: Appendix Tables A3.1- A3.3

Appendix Table A3.1. Proportion (%) who had at least one consultation in primary or specialized health care for the disorder between 2008 and 2016, by age in 2018 and sex

Dichotomous disorder indicators	Women		Men	
	Age 30	Age 40	Age 30	Age 40
Depression	24.71	25.29	14.00	15.84
Anxiety	11.87	11.83	6.82	7.64
Schizophrenia	0.51	0.58	0.83	1.07
Bipolar	2.14	2.25	1.25	1.49
Eating disorder	3.11	1.42	0.15	0.13
Personality disorder	3.33	2.95	2.10	2.68

Appendix Table A3.2. Effects (odds ratios with 95% CI) of disorder indicators in discrete-time hazard models for first-birth rates. Norwegian women and men 2010-2018

	Women		Men	
Dichotomous disorder indicators	As Table 2	Also control for family-of-origin characteristics [a]	As Table 2	Also control for family-of-origin characteristics [a]
Depression	0.91*** (0.89-0.92)	0.89*** (0.87-0.91)	0.62*** (0.60-0.64)	0.63*** (0.60-0.65)
Anxiety	0.91*** (0.88-0.94)	0.89*** (0.86-0.92)	0.70*** (0.67-0.74)	0.71*** (0.68-0.74)
Schizophrenia	0.17*** (0.14-0.20)	0.17*** (0.14-0.21)	0.09*** (0.08-0.11)	0.09*** (0.08-0.11)
Bipolar disorder	0.74*** (0.69-0.79)	0.74*** (0.69-0.79)	0.50*** (0.46-0.56)	0.51*** (0.46-0.56)
Eating disorder	0.78*** (0.74-0.83)	0.80*** (0.75-0.85)	0.38*** (0.24-0.60)	0.38*** (0.24-0.60)
Personality disorder	0.57*** (0.53-0.60)	0.55*** (0.52-0.59)	0.45*** (0.40-0.40)	0.46*** (0.41-0.50)

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The model was as in Table 2, except for the inclusion of the following family-of-origin characteristics: whether the mother was found in the data (yes vs no, in which case she was put in the reference category for the other variables), her country of birth (Norway vs abroad), whether she was alive and living in Norway 1st January the year the child was 17 (yes vs. no, in which case she was put in the reference category for income and partnership), her educational level that year (missing, primary, lower secondary, higher secondary, lower tertiary, higher tertiary, and PhD), her income in NOK that year (missing, 0, 1-49999, 50000-99999, 100000-149999, etc up to 950000-999999, and ≥1000000), corresponding variables for the father, and whether the father and mother were marital or cohabitational partners 1st January the year the child was 17 (that year was before 2005 and the parents were not married with each other, before 2005 and married with each other, 2005+ and neither married nor cohabiting with each other, 2005+ and married with each other, 2005+ and cohabiting with each other).

Appendix Table A3.3. Effects (odds ratios with 95% CI) of disorder indicators in discrete-time hazard models for fourth- and fifth-birth rates. Norwegian women and men 2010-2018 [a]

	Women			Men		
Dichotomous disorder indicators	Proportion with this disorder (%)	Number of births among those with this disorder	Effects on birth rates	Proportion with this disorder (%)	Number of births among those with this disorder	Effects on birth rates
Depression	5.96	889	1.15*** (1.07-1.23)	3.06	536	1.26*** (1.15-1.38)
Anxiety	2.30	358	1.08 (0.97-1.20)	1.12	185	1.11 (0.95-1.28)
Schizophrenia	0.06	<10 [b]	0.87 (0.39-1.95)	0.05	10	1.31 (0.70-2.45)
Bipolar disorder	0.60	96	1.20 (0.98-1.48)	0.37	69	1.32* (1.04-1.68)
Eating disorder	0.25	46	1.15 (0.85-1.54)	0.01	<10 [b]	1.71 (0.54-5.37)
Personality disorder	0.54	101	1.18 (0.96-1.44)	0.29	61	1.27 (0.98-1.64)
Number of births			13007			13781

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The model also included age and period in one-year categories, parity (3 or 4), and duration as described in Table 2. There were 3.377 million observations among women and 2.504 million among men.
[b] Numbers below 10 cannot be specified for data protection reasons

Appendix 4: Alternative disorder indicators

As a first step of our analysis of alternative disorder indicators, we first checked the implications of considering only specialized health care. We found that, on the whole, those who two years earlier had a consultation for a certain mental disorder in primary health care exclusively had less reduced fertility than those with a consultation for this disorder in specialized health care (some of whom have also had a consultation in primary health care). However, the differences were not large. This is shown for first births in Appendix Table A4.1. Thus, our ability to include consultations in primary health care gives us a larger proportion of individuals in the disorder category under study (making the estimates more precise), but does not have much impact on the point estimates.

While our main focus is on a person's health care consultations two years earlier (i.e., t-2), we also estimated some models where the key independent variable was whether there had been consultations for the various diseases in at least one year between 2008 and *t-2* or between 2008 and t-3. The use of such an accumulative indicator makes, of course, a control for calendar year particularly important.

As an example of how large the various groups are, let us consider women who are under exposure for a first birth: 5.7% had a consultation for depression in t-2 (3.6% of whom also had such a consultation an earlier years), while 7.9% only had a consultation in an earlier year (not shown). If we focus on those under exposure for a first birth in 2018, 6.2% had a consultation for depression in t-2 (i.e., 2016), while 10.8% only had such a consultation an earlier year (i.e., one of the years 2008-2015, but not 2016). Among the 6.2% who had a consultation in t-2, 4.2% had a consultation also in one of the earlier years. The pattern was similar for anxiety, but the proportion with a consultation for one of the other disorders in 2008-2015, but not in 2016, was smaller than the proportion with a consultation in 2016 – reflecting the more lasting nature of these disorders.

As one might expect, the relationship between fertility and use of health care for mental disorders from 2008 to t-2 was somewhat less negative than that between fertility and the corresponding indicators for t-2 (shown for first births in Appendix Table A4.2). When we included both indicators for 2008 to t-3 and the usual ones for t-2, which would only be meaningful for the years from 2011, most of the coefficients for the latter indicators were stronger than those for the former, but not all of them (Appendix Table A4.3).

In a final step we included indicators of having consultations for the disease both sometime during the period from 2008 to t-3 and in t-2, which indicates a relatively long-lasting disease. As expected, the effect coefficients in these models were particularly strong (for example 0.84 for depression among women as opposed to 0.90 when the focus was on only t-2 (see Model 4 in Appendix Table A4.3).

Appendix Table A4.1. Effects (odds ratios with 95% CI) of disorder indicators in discrete-time hazard models for first-birth rates. Norwegian women and men 2010-2018 [a]

	Women		Men	
Dichotomous disorder indicators	Proportion of exposure time with this disorder (%)	Effects on first-birth rates	Proportion of exposure time with this disorder (%)	Effects on first-birth rates
Depression, only primary care	3.00	0.93*** (0.91-0.96)	1.84	0.64*** (0.61-0.66)
Depression, specialized care	2.59	0.87*** (0.84-0.90)	1.19	0.58*** (0.55-0.62)
Anxiety, only primary care	1.18	0.91*** (0.87-0.95)	0.90	0.70*** (0.66-0.74)
Anxiety, specialized care	1.10	0.91*** (0.87-0.95)	0.56	0.72*** (0.67-0.78)
Schizophrenia, only primary care	0.11	0.22*** (0.16-0.29)	0.19	0.10*** (0.08-0.14)
Schizophrenia, specialized care	0.20	0.15*** (0.11-0.19)	0.39	0.09*** (0.07-0.11)
Bipolar disorder, only primary care	0.22	0.75*** (0.68-0.84)	0.15	0.50*** (0.43-0.59)
Bipolar disorder, specialized care	0.39	0.73*** (0.67-0.79)	0.23	0.50*** (0.44-0.58)
Eating disorder, only primary care	0.11	0.77** (0.66-0.90)	0.01	0.43 (0.14-1.34)
Eating disorder, specialized care	0.78	0.79*** (0.74-0.85)	0.04	0.37*** (0.22-0.62)
Personality disorder, only primary care	0.12	0.55*** (0.46-0.65)	0.15	0.49*** (0.41-0.58)
Personality disorder, specialized care	0.72	0.57*** (0.54-0.61)	0.36	0.43*** (0.38-0.48)

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The models were as in Table 2, except for the more detailed disorder indicators. Each of them includes three categories. The category "only primary care" refers to whether there was at least one consultation for the disorder in primary care in the year t-2, but no consultation for the disorder in specialized care. The category "specialized care" refers to whether there was at least one consultation for the disorder in specialized care in the year t-2. There may or may not have been a consultation also in primary care. Those without any of the mental disorders in focus are in the reference category (not shown in the table).

Appendix Table A4.2. Effects (odds ratios with 95% CI) of disorder indicators in discrete-time hazard models for first-birth rates. Norwegian women and men 2010-2018 [a]

Dichotomous disorder indicators	Women		Men	
	Disorder indicator referring to t-2 (as Table 2)	Disorder indicator referring to the period from 2008 up to t-2 [a]	Disorder indicator referring to t-2 (as Table 2)	Disorder indicator referring to the period from 2008 up to t-2 [a]
Depression	0.91*** (0.89-0.92)	0.97*** (0.95-0.98)	0.62*** (0.60-0.64)	0.73*** (0.71-0.74)
Anxiety	0.91*** (0.88-0.94)	0.95*** (0.93-0.97)	0.70*** (0.67-0.74)	0.77*** (0.75-0.79)
Schizophrenia	0.17*** (0.14-0.20)	0.24*** (0.21-0.28)	0.09*** (0.08-0.11)	0.17*** (0.15-0.19)
Bipolar	0.74*** (0.69-0.79)	0.79*** (0.75-0.83)	0.50*** (0.46-0.56)	0.65*** (0.61-0.70)
Eating disorder	0.78*** (0.74-0.83)	0.90*** (0.87-0.94)	0.38*** (0.24-0.60)	0.53*** (0.42-0.66)
Personality disorder	0.57*** (0.53-0.60)	0.63*** (0.60-0.66)	0.45*** (0.40-0.49)	0.55*** (0.52-0.58)

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The model was as in Table 2, except for the definition of the disorder indicators.

Appendix Table A4.3. Effects (odds ratios with 95% CI) of disorder indicators in discrete-time hazard models for first-birth rates. Norwegian women and men 2011-2018 [a]

Panel A: Women

Dichotomous disorder indicators	Model 1[a]	Model 2 [b]	Model 3 [b]	Model 4 [b]
Indicators for having the disorder in t-2				
Depression	0.90*** (0.88-0.92)		0.92*** (0.90-0.94)	
Anxiety	0.90*** (0.87-0.93)		0.93*** (0.90-0.97)	
Schizophrenia	0.17*** (0.14-0.21)		0.38*** (0.30-0.48)	
Bipolar	0.74*** (0.69-0.80)		0.92* (0.84-0.99)	
Eating disorder	0.77*** (0.72-0.83)		0.81*** (0.75-0.87)	
Personality disorder	0.57*** (0.53-0.60)		0.75*** (0.69-0.81)	
Indicators for having the disorder between 2008 and t-3				
Depression		0.95*** (0.93-0.97)	0.98** (0.96-0.99)	
Anxiety		0.94*** (0.92-0.96)	0.96** (0.94-0.99)	
Schizophrenia		0.24*** (0.21-0.28)	0.39*** (0.33-0.46)	
Bipolar disorder		0.77*** (0.73-0.81)	0.81*** (0.75-0.86)	
Eating disorder		0.90*** (0.87-0.94)	0.97 (0.93-1.02)	
Personality disorder		0.63*** (0.60-0.67)	0.71*** (0.68-0.76)	
Indicators of having the disorder <u>both</u> between 2008 and t-3 <u>and</u> in t-2				
Depression				0.84*** (0.81-0.87)
Anxiety				0.84*** (0.80-0.88)
Schizophrenia				0.14*** (0.11-0.18)
Bipolar disorder				0.67*** (0.62-0.73)
Eating disorder				0.71*** (0.66-0.77)
Personality disorder				0.54*** (0.50-0.58)

Panel B: Men

Dichotomous disorder indicators	Model 1[a]	Model 2 [b]	Model 3 [b]	Model 4 [b]
Indicators for having the disorder in t-2				
Depression	0.61*** (0.58-0.63)		0.71*** (0.69-0.74)	
Anxiety	0.70*** (0.67-0.74)		0.85*** (0.81-0.90)	
Schizophrenia	0.09*** (0.08-0.11)		0.23*** (0.19-0.28)	
Bipolar	0.50*** (0.45-0.56)		0.73*** (0.65-0.83)	
Eating disorder	0.39*** (0.25-0.64)		0.56* (0.34-0.92)	
Personality disorder	0.44*** (0.39-0.48)		0.65*** (0.59-0.73)	
Indicators for having the disorder between 2008 and t-3				
Depression		0.72*** (0.70-0.73)	0.78*** (0.76-0.80)	
Anxiety		0.75*** (0.73-0.78)	0.79*** (0.77-0.82)	
Schizophrenia		0.18*** (0.16-0.20)	0.36*** (0.32-0.42)	
Bipolar disorder		0.66*** (0.61-0.71)	0.76*** (0.69-0.83)	
Eating disorder		0.53*** (0.41-0.68)	0.59*** (0.46-0.76)	
Personality disorder		0.55*** (0.51-0.58)	0.61*** (0.57-0.65)	
Indicators of having the disorder <u>both</u> between 2008 and t-3 <u>and</u> in t-2				
Depression				0.52*** (0.50-0.55)
Anxiety				0.63*** (0.59-0.67)
Schizophrenia				0.08*** (0.07-0.10)
Bipolar disorder				0.47*** (0.41-0.53)
Eating disorder				0.27*** (0.13-0.57)
Personality disorder				0.38*** (0.33-0.44)

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The model was as in Table 2, except for the shorter observation period (2011-2018 rather than 2010-2018)

[b] The models were as in Table 2, except for the shorter observation period (2011-2018 rather than 2010-2018) and the disorder indicators. Models 2 and 4 included one set of disorder indicators, while Model 3 included two sets of disorder indicators.

Appendix 5: Appendix Tables A5.1- A5.4

Appendix Table A5.1. Effects (odds ratios with 95% CI) of disorder indicators in discrete-time hazard models for first-, second- and third-birth rates. Norwegian women and men 2010-2018 [a]

Panel A: Women

Dichotomous disorder indicators	Effects on first-birth rates	Effects on second-birth rates	Effects on third-birth rates
Depression	0.91*** (0.89-0.92)	0.70*** (0.69-0.72)	0.87*** (0.83-0.90)
Anxiety	0.90*** (0.88-0.94)	0.76*** (0.73-0.79)	0.89*** (0.83-0.94)
Schizophrenia	0.17*** (0.14-0.20)	0.36*** (0.28-0.47)	0.90 (0.57-1.42)
Bipolar disorder	0.74*** (0.69-0.79)	0.61*** (0.56-0.66)	0.85* (0.74-0.97)
Eating disorder	0.78*** (0.74-0.83)	0.88** (0.81-0.97)	1.11 (0.96-1.28)
Personality disorder	0.57*** (0.53-0.60)	0.69*** (0.64-0.75)	0.97 (0.86-1.10)
Number of births	162062	141826	54582
Exposure time (million person-quarters)	13.536	3.784	6.431
Control also for partnership status:			
Depression	0.93*** (0.91-0.95)	0.75*** (0.73-0.77)	0.86*** (0.83-0.90)
Anxiety	0.90*** (0.87-0.93)	0.78*** (0.75-0.81)	0.89*** (0.83-0.94)
Schizophrenia	0.21*** (0.18-0.26)	0.44*** (0.34-0.58)	0.90 (0.57-1.42)
Bipolar disorder	0.76*** (0.71-0.81)	0.63*** (0.58-0.69)	0.84** (0.73-0.96)
Eating disorder	0.84*** (0.79-0.90)	0.93 (0.85-1.01)	1.10 (0.94-1.27)
Personality disorder	0.62*** (0.58-0.66)	0.76*** (0.70-0.83)	0.97 (0.85-1.10)
Control also for partnership status and socioeconomic factors:			
Depression	0.98 (0.96-1.00)	0.79*** (0.77-0.82)	0.90*** (0.86-0.93)
Anxiety	0.98 (0.95-1.01)	0.85*** (0.81-0.88)	0.92** (0.87-0.98)
Schizophrenia	0.26*** (0.22-0.32)	0.53*** (0.40-0.69)	0.98 (0.62-1.54)
Bipolar disorder	0.86*** (0.81-0.92)	0.69*** (0.63-0.75)	0.86* (0.75-0.98)
Eating disorder	0.88*** (0.83-0.93)	0.95 (0.87-1.04)	1.11 (0.95-1.28)
Personality disorder	0.73*** (0.68-0.77)	0.88*** (0.81-0.95)	1.02 (0.90-1.16)

Panel A: Men

Dichotomous disorder indicators	Effects on first-birth rates	Effects on second-birth rates	Effects on third-birth rates
Depression	0.62*** (0.60-0.64)	0.62*** (0.60-0.65)	0.90*** (0.85-0.96)
Anxiety	0.70*** (0.67-0.74)	0.67*** (0.64-0.71)	0.88** (0.81-0.96)
Schizophrenia	0.09*** (0.08-0.11)	0.32*** (0.25-0.42)	0.44** (0.26-0.77)
Bipolar disorder	0.50*** (0.46-0.56)	0.69*** (0.61-0.77)	0.83* (0.69-0.99)
Eating disorder	0.38*** (0.24-0.60)	0.84 (0.47-1.49)	0.53 (0.17-1.66)
Personality disorder	0.45*** (0.40-0.49)	0.55*** (0.48-0.62)	0.84 (0.70-1.01)
Number of births	160278	134863	52778
Exposure time (million person-quarters)	17.303	3.690	5.140
Control also for partnership status:			
Depression	0.68*** (0.66-0.71)	0.70*** (0.67-0.73)	0.89*** (0.84-0.94)
Anxiety	0.76*** (0.73-0.80)	0.72*** (0.68-0.76)	0.88** (0.81-0.96)
Schizophrenia	0.13*** (0.11-0.16)	0.39*** (0.30-0.51)	0.42** (0.24-0.73)
Bipolar disorder	0.57*** (0.52-0.63)	0.73*** (0.65-0.82)	0.80* (0.67-0.96)
Eating disorder	0.44*** (0.27-0.69)	0.86 (0.48-1.54)	0.52 (0.17-1.60)
Personality disorder	0.53*** (0.48-0.58)	0.63*** (0.56-0.71)	0.82* (0.68-0.99)
Control also for partnership status and socioeconomic factors:			
Depression	0.78*** (0.76-0.82)	0.77*** (0.74-0.80)	0.90*** (0.85-0.95)
Anxiety	0.90*** (0.86-0.94)	0.80*** (0.75-0.84)	0.89** (0.81-0.97)
Schizophrenia	0.19*** (0.16-0.22)	0.47*** (0.36-0.61)	0.42** (0.24-0.73)
Bipolar disorder	0.70*** (0.63-0.77)	0.80*** (0.71-0.90)	0.80* (0.66-0.96)
Eating disorder	0.47*** (0.30-0.75)	0.89 (0.50-1.60)	0.51 (0.16-1.59)
Personality disorder	0.68*** (0.61-0.74)	0.74*** (0.65-0.83)	0.82* (0.68-0.99)

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The estimates in the upper part of each panel are as in Table 2. All models included age, period and duration as described in Table 2. Some of them also included partnership status (married, widowed and not cohabiting, divorced and not cohabiting, separated and not cohabiting, never-married and not cohabiting, and cohabiting), and some additionally included the following sociodemographic factors: educational level (missing, primary or not completed higher secondary, higher secondary, lower tertiary, and higher tertiary), school enrolment (yes vs. no) and income in NOK, with the following categorization: missing, 0, 1-49999, 50000-99999, 100000-149999, etc up to 950000-999999, and ≥1000000.

Appendix Table A5.2. Effects (odds ratios with 95% CI) of disorder indicators in discrete-time hazard models for first-birth rates, by age. Norwegian women and men 2010-2018 [a]

Panel A: Women

Dichotomous disorder indicators	Age ≤ 23	Age 24-26	Age 27-29	Age 30-32	Age ≥ 33
Depression	1.72*** (1.64-1.80)	1.08** (1.02-1.13)	0.74*** (0.71-0.78)	0.67*** (0.63-0.70)	0.73*** (0.70-0.77)
Anxiety	1.36*** (1.26-1.46)	0.97 (0.90-1.04)	0.92* (0.86-0.98)	0.76*** (0.70-0.82)	0.75*** (0.70-0.81)
Schizophrenia	0.52*** (0.32-0.84)	0.37*** (0.24-0.57)	0.12*** (0.07-0.21)	0.16*** (0.10-0.24)	0.12*** (0.09-0.17)
Bipolar disorder	1.32*** (1.13-1.55)	0.90 (0.77-1.04)	0.79*** (0.69-0.91)	0.55*** (0.47-0.64)	0.57*** (0.49-0.65)
Eating disorder	0.79*** (0.70-0.90)	0.85* (0.75-0.97)	0.72*** (0.63-0.82)	0.75*** (0.65-0.87)	0.68*** (0.58-0.80)
Personality disorder	1.03 (0.88-1.20)	0.79*** (0.69-0.90)	0.57*** (0.50-0.65)	0.46*** (0.40-0.53)	0.44*** (0.38-0.50)
Number of first births	26976	31583	38949	32396	32161
Control also for partnership status					
Depression	1.71*** (1.63-1.79)	1.08** (1.02-1.13)	0.76*** (0.72-0.80)	0.70*** (0.66-0.74)	0.76*** (0.73-0.80)
Anxiety	1.33*** (1.24-1.44)	0.94 (0.87-1.01)	0.92* (0.86-0.98)	0.77*** (0.71-0.83)	0.74*** (0.69-0.80)
Schizophrenia	0.53*** (0.33-0.85)	0.41*** (0.27-0.63)	0.15*** (0.09-0.26)	0.21*** (0.14-0.31)	0.15*** (0.11-0.21)
Bipolar disorder	1.31*** (1.12-1.53)	0.92 (0.79-1.08)	0.83** (0.72-0.95)	0.58*** (0.49-0.68)	0.59*** (0.51-0.67)
Eating disorder	0.81** (0.72-0.92)	0.91 (0.80-1.03)	0.79*** (0.69-0.90)	0.86* (0.74-0.99)	0.73*** (0.62-0.86)
Personality disorder	1.02 (0.88-1.20)	0.80*** (0.70-0.92)	0.63*** (0.55-0.72)	0.52*** (0.45-0.60)	0.49*** (0.43-0.56)
Control also for partnership status and socioeconomic factors					
Depression	1.35*** (1.29-1.42)	0.99 (0.94-1.04)	0.81*** (0.77-0.85)	0.80*** (0.76-0.85)	0.87*** (0.83-0.91)
Anxiety	1.16** (1.12-1.20)	0.91* (0.84-0.98)	1.01 (0.94-1.07)	0.90*** (0.84-0.98)	0.91* (0.85-0.98)
Schizophrenia	0.36*** (0.23-0.59)	0.34*** (0.22-0.53)	0.18*** (0.10-0.31)	0.34*** (0.22-0.52)	0.29*** (0.21-0.40)
Bipolar disorder	1.08 (0.92-1.26)	0.89 (0.76-1.03)	0.94 (0.82-1.08)	0.72*** (0.61-0.85)	0.74*** (0.65-0.85)
Eating disorder	0.81** (0.71-0.92)	0.96 (0.84-1.09)	0.83** (0.72-0.94)	0.89 (0.77-1.03)	0.77** (0.66-0.91)
Personality disorder	0.78** (0.67-0.91)	0.75*** (0.66-0.86)	0.73*** (0.64-0.83)	0.73*** (0.63-0.85)	0.71*** (0.62-0.82)

Panel B: Men

Dichotomous disorder indicators	Age ≤ 25	Age 26-28	Age 29-31	Age 32-34	Age ≥ 35
Depression	1.08* (1.01-1.17)	0.67*** (0.62-0.72)	0.53*** (0.49-0.57)	0.50*** (0.46-0.54)	0.56*** (0.52-0.59)
Anxiety	1.16** (1.05-1.30)	0.83*** (0.75-0.92)	0.63*** (0.57-0.70)	0.67*** (0.60-0.74)	0.54*** (0.49-0.60)
Schizophrenia	0.29*** (0.18-0.46)	0.12*** (0.07-0.20)	0.08*** (0.06-0.13)	0.08*** (0.06-0.12)	0.07*** (0.05-0.10)
Bipolar disorder	0.94 (0.73-1.22)	0.55*** (0.43-0.72)	0.45*** (0.36-0.57)	0.41*** (0.32-0.52)	0.47*** (0.40-0.57)
Eating disorder	0.60 (0.28-1.26)	0.21* (0.05-0.85)	0.44 (0.17-1.18)	0.22* (0.06-0.89)	0.31* (0.10-0.96)
Personality disorder	0.91 (0.72-1.15)	0.53*** (0.42-0.66)	0.45*** (0.37-0.56)	0.39*** (0.31-0.49)	0.35*** (0.29-0.42)
Number of first births	26899	31613	36879	28948	35940
Control also for partnership status					
Depression	1.10* (1.02-1.19)	0.72*** (0.67-0.78)	0.60*** (0.51-0.65)	0.56*** (0.51-0.61)	0.62*** (0.58-0.66)
Anxiety	1.18** (1.06-1.32)	0.88* (0.79-0.98)	0.70*** (0.63-0.77)	0.73*** (0.66-0.80)	0.60*** (0.55-0.66)
Schizophrenia	0.30*** (0.19-0.49)	0.15*** (0.09-0.25)	0.12*** (0.08-0.19)	0.13*** (0.09-0.19)	0.11*** (0.08-0.15)
Bipolar disorder	0.97 (0.75-1.25)	0.61*** (0.47-0.79)	0.51*** (0.41-0.65)	0.48*** (0.38-0.62)	0.55*** (0.46-0.66)
Eating disorder	0.61 (0.29-1.28)	0.25* (0.06-1.00)	0.51 (0.19-1.36)	0.29 (0.07-1.16)	0.38 (0.12-1.19)
Personality disorder	0.94 (0.75-1.19)	0.60*** (0.48-0.76)	0.53*** (0.43-0.66)	0.48*** (0.38-0.60)	0.43*** (0.35-0.51)
Control also for partnership status and socioeconomic factors					
Depression	1.02 (0.95-1.10)	0.77*** (0.71-0.83)	0.70*** (0.65-0.75)	0.68*** (0.63-0.74)	0.75*** (0.70-0.81)
Anxiety	1.14* (1.02-1.27)	0.96 (0.87-1.07)	0.82*** (0.74-0.91)	0.89* (0.81-0.99)	0.77*** (0.70-0.85)
Schizophrenia	0.26*** (0.16-0.42)	0.18*** (0.11-0.29)	0.17*** (0.11-0.26)	0.21*** (0.14-0.30)	0.19*** (0.14-0.26)
Bipolar disorder	0.94 (0.73-1.22)	0.67** (0.52-0.87)	0.60*** (0.48-0.76)	0.62*** (0.49-0.79)	0.71*** (0.60-0.85)
Eating disorder	0.63 (0.30-1.33)	0.28 (0.07-1.14)	0.53 (0.20-1.42)	0.32 (0.08-1.29)	0.42 (0.14-1.31)
Personality disorder	0.90 (0.70-1.32)	0.68*** (0.54-0.86)	0.68*** (0.55-0.84)	0.67*** (0.53-0.84)	0.62*** (0.51-0.74)

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The estimates in the upper part of each panel are as in Table 3. All models included age and period as described in Table 3. Some of them also included partnership status (married, widowed and not cohabiting, divorced and not cohabiting, separated and not cohabiting, never-married and not cohabiting, and cohabiting), and some additionally included the following sociodemographic factors: educational level (missing, primary or not completed higher secondary, higher secondary, lower tertiary, and higher tertiary), school enrolment (yes vs. no) and income in NOK, with the following categorization: missing, 0, 1-49999, 50000-99999, 100000-149999, etc up to 950000-999999, and ≥1000000.

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Appendix Table A5.3. Effects (linear effects with standard errors) of disorder indicators in discrete-time hazard models for first-birth rates, when linear probability models are estimated from the 3-month observations. Norwegian women and men 2010-2018 [a]

Panel A: Women

Dichotomous disorder indicators	Effects on first-birth rates	Effects on second-birth rates	Effects on third-birth rates
Depression	-0.0012*** (0.0001)	-0.0092*** (0.0004)	-0.0011*** (0.0002)
Anxiety	-0.0013*** (0.0002)	-0.0072*** (0.0006)	-0.0010*** (0.0002)
Schizophrenia	-0.0126*** (0.0005)	-0.0119*** (0.0021)	-0.0005 (0.0014)
Bipolar disorder	-0.0041*** (0.0004)	-0.0102*** (0.0010)	-0.0012* (0.0005)
Eating disorder	-0.0024*** (0.0003)	-0.0036*** (0.0013)	0.0007 (0.0007)
Personality disorder	-0.0073*** (0.0003)	-0.0074*** (0.0010)	-0.0005 (0.0005)
Control also for partnership status and socioeconomic factors:			
Depression	-0.0004** (0.0001)	-0.0058*** (0.0004)	-0.0009*** (0.0002)
Anxiety	-0.0003 (0.0002)	-0.0036*** (0.0006)	-0.0007** (0.0002)
Schizophrenia	-0.0046*** (0.0005)	-0.0012 (0.0021)	-0.0001 (0.0014)
Bipolar disorder	-0.0017*** (0.0004)	-0.0067*** (0.0010)	-0.0011* (0.0005)
Eating disorder	-0.0014*** (0.0003)	-0.0018 (0.0013)	0.0007 (0.0007)
Personality disorder	-0.0032*** (0.0003)	-0.0010 (0.0010)	-0.0001 (0.0005)

Panel A: Men

Dichotomous disorder indicators	Effects on first-birth rates	Effects on second-birth rates	Effects on third-birth rates
Depression	-0.0042*** (0.0001)	-0.0117*** (0.0005)	-0.0010*** (0.0003)
Anxiety	-0.0034*** (0.0002)	-0.0096*** (0.0007)	-0.0012** (0.0004)
Schizophrenia	-0.0125*** (0.0003)	-0.0135*** (0.0020)	-0.0045** (0.0017)
Bipolar disorder	-0.0059*** (0.0004)	-0.0072*** (0.0014)	-0.0016* (0.0008)
Eating disorder	-0.0033** (0.0011)	-0.0044 (0.0076)	-0.0049 (0.0042)
Personality disorder	-0.0064*** (0.0003)	-0.0106*** (0.0013)	-0.0017* (0.0008)
Control also for partnership status and socioeconomic factors:			
Depression	-0.0020*** (0.0001)	-0.0065*** (0.0005)	-0.0010*** (0.0003)
Anxiety	-0.0009*** (0.0002)	-0.0044*** (0.0007)	-0.0012** (0.0004)
Schizophrenia	-0.0045*** (0.0003)	-0.0032 (0.0020)	-0.0049** (0.0017)
Bipolar disorder	-0.0027*** (0.0004)	-0.0036* (0.0014)	-0.0019* (0.0008)
Eating disorder	-0.0022* (0.0011)	-0.0021 (0.0076)	-0.0050 (0.0042)
Personality disorder	-0.0021*** (0.0003)	-0.0037** (0.0013)	-0.0018* (0.0008)

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The models and the samples were as in Appendix Table A5.1, except that they were linear probability models instead of logistic models

Appendix Table A5.4. Effects (odds ratios or hazard ratios with 95% CI) of disorder idicators in discrete-time hazard models or Cox models for first-birth rates. Norwegian women and men 2010-2018

Panel A: Full sample [a]

Dichotomous disorder indicators	Women		Men	
	Discrete-time hazard model (as Table 2)	Cox model	Discrete-time hazard model (as Table 2)	Cox model
Depression	0.91*** (0.89-0.92)	0.91*** (0.89-0.93)	0.62*** (0.60-0.64)	0.62*** (0.60-0.64)
Anxiety	0.91*** (0.88-0.94)	0.91*** (0.88-0.94)	0.70*** (0.67-0.74)	0.71*** (0.67-0.74)
Schizophrenia	0.17*** (0.14-0.20)	0.17*** (0.14-0.21)	0.09*** (0.08-0.11)	0.09*** (0.08-0.11)
Bipolar disorder	0.74*** (0.69-0.79)	0.74*** (0.70-0.79)	0.50*** (0.46-0.56)	0.51*** (0.46-0.56)
Eating disorder	0.78*** (0.74-0.83)	0.79*** (0.74-0.84)	0.38*** (0.24-0.60)	0.38*** (0.24-0.60)
Personality disorder	0.57*** (0.53-0.60)	0.57*** (0.54-0.61)	0.45*** (0.40-0.49)	0.45*** (0.41-0.49)
Number of births	162065	162065	160278	160278

Panel B: Including only those who have a same-sex sibling [b]

Dichotomous disorder indicators	Women		Men	
	Discrete-time hazard model	Cox model	Discrete-time hazard model	Cox model
Depression	0.93*** (0.90-0.96)	0.93*** (0.90-0.96)	0.63*** (0.60-0.66)	0.63*** (0.60-0.67)
Anxiety	0.94* (0.89-0.99)	0.94* (0.89-0.99)	0.75*** (0.70-0.80)	0.75*** (0.70-0.80)
Schizophrenia	0.18*** (0.13-0.24)	0.17*** (0.13-0.24)	0.09*** (0.07-0.12)	0.09*** (0.07-0.12)
Bipolar disorder	0.77*** (0.69-0.85)	0.77*** (0.69-0.85)	0.51*** (0.44-0.60)	0.51*** (0.44-0.60)
Eating disorder	0.77*** (0.70-0.84)	0.78*** (0.71-0.85)	0.50* (0.28-0.90)	0.47* (0.26-0.85)
Personality disorder	0.59*** (0.54-0.65)	0.60*** (0.54-0.66)	0.43*** (0.37-0.49)	0.43*** (0.37-0.50)
Number of births	70522	70522	75318	75318

Notes: *p<0.05; ** p<0.01; *** p<0.001

[a] The models and the samples were as in Table 2. The Cox models included year, but not (unlike the discrete-time hazard models) age, which was the "duration variable".
[b] The models were as those in Panel A, except that individuals who did not have a same-sex sibling who was also under exposure for first birth were excluded.

Supplementary notes

[1] Another study showed that subfertile women were less likely to initiate fertility treatment if they were depressed (Crawford et al. 2017), and Kalucza et al. (2015) found that an indicator of psychological problems, which included depression, was related to a reduced number of children at age 43 among men, but not among women.

[2] More specifically, externalizing behaviour was associated with relatively early first births (and often without being in a co-residential relationship) among men, but not women (Evensen and Lyngstad 2020), or generally higher fertility, and especially earlier transition to parenthood (Jokela 2014). One specific type of externalizing behaviour, ADHD, was associated with teenage fertility among both women and men, but also high permanent childlessness and (among men) lower completed fertility (Østergaard et al. 2017). In support of the idea that mental diseases may speed up fertility, a study of female teenagers showed that an indicator of "major mental illness" - defined as either major depression, bipolar disorder, or psychotic disorder - was associated with relatively high fertility (Vigod et al. 2014). Also, in an investigation of fertility among relatively young women, a positive association with hospitalization for a mental disorder (of any type) appeared (Selling et al. 2009).

[3] The ranking of disease burdens depends, of course, on how the various diseases are grouped together. The ranking referred to here is based on the so-called level-3 causes.

[4] In fact, among individuals who met diagnostic criteria for depression or anxiety in an interview, the proportion who had been registered with a mental diagnosis after a primary health care consultation within the two preceding years was twice as large as the proportion who had been registered with such a diagnosis in specialized health care (Torvik et al. 2018).

[5] To simplify the presentation, the word 'fecundity' refers to whether the woman and her sexual partner are able to conceive a child, possibly with help from artificial reproductive technology, and the pregnancy ends with a live birth.

[6] Inadequate contraceptive use among those who do not want a(nother) child (yet) may be a result of, for example, i) impulsivity, ii) not being able to use certain methods for medical reasons, or iii) poor access to or not accepting certain forms of contraception, although the latter is not common in a Nordic setting (while the use of abortion to compensate for inadequate contraception may not be acceptable to everyone).

[7] High purchasing power for a man may be expected to increase fertility desires if childbearing costs are fixed, but that is not necessarily the case. If the income is high, the parents may want to or feel obliged to spend more on each child.

[8] This idea is based on the assumption that it is primarily the mother who stays at home with the child after the period with paid parental leave (if any),

[9] One may argue that, given that another child is wanted, two main issues enter into the timing decision (Happel et al 1994; Gustafsson 2001). First, one may want to have the child later rather than sooner if the costs of childbearing would then be lower. Second, because it is costly to borrow money and there is a diminishing welfare return from increasing income, one may want to synchronize the childbearing costs with a period when the income is high, i.e. postpone childbearing if an income rise is expected.

[10] A rise in a man's income may be more likely if his current income is low, and if the current income is low, a given income rise may also be more likely to cause fertility postponement.

[11]When discussing effects of women's wages, an opportunity cost argument comes in addition: To the extent that currently low income increases the chance of a wage rise, it may also make a postponed birth more costly, at least when it comes to short-term opportunity costs.

[12] One reason for expecting that current school enrolment reduces the chance of having the child soon, is that those having a child while enrolled may not be able to reach their educational goals - with possibly long-term earnings implications. Individuals enrolled in school may also be less likely to be in a relationship, and especially to be married (Kravdal 1999).

[13] Not being able to or wanting to have a child because of mental disorders (as further discussed below) may be an additional reason for not being chosen as a partner.

[14] There has been more interest in the association between partnership and physical health or general health. Some of these studies have shown that it is more common for individuals with poor health to live alone (Wiik and Dommermuth 2014; Sandström et al. 2020), while other investigations have not revealed such a pattern (Lee and Panis 1996; Syse and Kravdal 2007; Syse 2008), or only observed an impact of health on partnership at the highest ages (Rapp 2018).

[15] Effects of physical health on fecundity have been shown, or at least speculated about, in several studies (Penovich 2000; Meirow and Nugent 2001; Gill et al. 2009; Green et al 2009; Gade et al. 2014; Wiebe et al. 2014; Derai et al. 2017; Pieczynska 2018; Dumanski and Ahmed 2019).

[16] This argument is also relevant for physical diseases (Leroy et al. 2015; Harpe et al. 2022).

[17] Doctors may also advise against pregnancy because they (more or less correctly) fear these types of effects.

[18] This point is made for serious diseases in general by Dommermuth et al. (2011).

[19] Studies of some physical diseases point in the same direction (Penovich 2000; Hall et al. 2015). Psychological factors may be involved and, in addition, doctors may in some cases discourage the use of certain contraceptive methods (Phillips et al. 2018).

[20] Another possible contribution in this direction is that it is less common to have a mental diagnosis at age 20 than at age 30. It may therefore be more stigmatising, which may strengthen some of the fertility-reducing effects mentioned above.

[21] For examples of studies of how fertility effects of physical or general health vary with parity, see Syse et al. (2007) or Baxter et al. (2013).

[22] With regard to physical health, risk factors such as alcohol, tobacco and obesity may influence fertility also, partly through fecundity (Eggert et al. 2004; Amiri and Therani 2020; Magnus et al. 2021; Salvio et al. 2022).

[23] The basis for doing this is the parents' PINs, which are included for almost everyone born in Norway after 1953.

[24] Primary health care personnel report consultations to KUHR in order to be reimbursed by the state. (KUHR is the acronym for The Norwegian Control and Distribution of Health Reimbursement Database). Additionally, KUHR includes some consultations with specialists. In the data extracted for the present analysis, 99.4% of the consultations were with physicians whom it may be reasonable to refer to as general practitioners. The few general practitioners who do not have a contract with the health authorities, and therefore do not benefit from public subsidies, do not report to KUHR.

[25] We included consultations (visits) in the following types of specialized health care: somatic hospitals (data available from 2008), private specialists in somatic medicine who had contract with the regional health authorities, and therefore benefitted from public subsidies ('AVTSOM', data available from 2009, but a sharp increase in the number of registrations up to 2013 suggests under-registration in 2009-2012), mental hospital for adults ('PHV', data available from 2008), private psychiatrists with adult patients ('AVTPHV', data available from 2008), mental hospitals for children and adolescents ('PHVBU', data available from 2008), and drug abuse treatment (data available from 2009, although possible under-registration 2009-2010).

[26] In most of these data files from NPR, up to two main diagnoses and 20 secondary diagnoses are reported. In other files, there are up to 10 secondary diagnoses (AVTSOM and AVTPHV), or no distinction is made between main and secondary diagnoses (PHBVU). Among the consultations reported in the latter file, 21% did not include any diagnosis at all, while this is uncommon in the other files (0.1% - 5%). For simplicity, we only took

into account 10 diagnoses, including all main diagnoses where such diagnoses are defined. There are very few consultations with a larger number of diagnoses reported (e.g., 0.8% of the hospital consultations).

[27] The income is the sum of labour income, entrepreneurial income, capital income, pensions and some benefits, minus tax deductible amounts.

[28] Immigrants were excluded because many of them may have children who have never lived in Norway, and who have therefore not been included in the Population Register (Kravdal 2021). This reduced the sample by about 1/5. However, the effects of mental health on fertility did actually not vary much depending on whether the immigrants were included.

[29] The reason for this restriction was that the number of health care consultations in one of the years back to 2008 (used to construct the key independent variables) is a less informative measure for persons not living in the country the whole year. Living in Norway both at the beginning of that year and the beginning of the next is considered an indication of living in the country the whole year.

[30] Almost identical estimates were obtained with 12-month observation intervals, so it would be unnecessary to use even shorter intervals than 3 months. In the analysis of first births, both singleton and multiple births were reckoned as first births. However, those who, for example, had a twin first birth did not contribute to the analysis of second births, but to the analysis of third and possibly higher-order births. Multiple births after the first were handled similarly.

[31] Including age interactions might have been an alternative to such an age stratified analysis, but this would also have been challenging, as interactions on a log-odds or odds scale do not necessarily translate into the same interactions at the probability scale (Ai and Norton 2003).

[32] Unlike Laursen and Munk (2010), we could not consider the time since disorder onset. The disease onset can only be identified for the cohorts who were young children in the first year covered by KUHR (2006) or NPR (2008 or later). Note that, even if the individual was not registered with the disorder in t-2, and therefore was in the reference category for that disorder indicator, they may have been registered with other mental disorders in t-2.

[33] It is potentially important to control for period, because i) unobserved characteristics that change over period are likely to affect fertility and ii) the health indicators are linked to period, partly because of changes in the registration practice (not least the apparently strong reduction in the under-registration of some types of specialized health care over the first few years after 2008).

[34] The following characteristics, measured when the index person was 17 years old, were included: whether the mother was identified in the data, whether she was alive and resident in Norway, her educational level, her income, her country of birth (Norway vs other), the corresponding variables for the father, and whether the parents were marital or cohabitational partners. Because one might also suspect that characteristics of the place of residence are important confounders, we included dummies for the index person's current municipality of residence in a supplementary analysis where we – to reach convergence within reasonable time – used linear probability models rather than logistic models. Inclusion of these dummies had no impact on the key estimates (not shown).

[35] Note that, in the analysis of second and higher-order births, the year t-2 may include a period when one or both parents earned relatively little because they stayed home (full- or part-time) to care for the most recently born child after the paid parental leave period (which most parents have). However, for other individuals the income in this year should be a reasonably good indicator of the purchasing power they have had for some time and can expect for some time into the future.

[36] Conversely, one might consider including a health indicator for t-3, so that it is more reasonable to consider the sociodemographic variables measured in t-2 as mediators, but the same issue then arises: For example, since earnings after all are relatively stable, a measurement in t-2 also reflects the earnings quite far back in time, which may have influenced the chance of having poor mental health in t-3.

[37] In the Cox models, age was the underlying time scale, and we assumed that exposures started at the beginning of the relevant quarters and ended (possibly with a birth "event") at the end of the quarters. Period, duration

since previous birth and all other variables were included with the same categories as in the discrete-time models.

[38] Being in this category means that, during the calendar year before the preceding, they had at least one consultation in primary or specialized healthcare where depression was registered as a diagnosis.

[39] If we instead base the disorder definition exclusively on primary care consultations, for which data exist over a longer period, the proportion who at age 20 (and pooled over all parities) have been registered with depression or anxiety two years earlier has increased over time (not shown). Among those aged 30, there has been no such increase. In both age groups, the proportions with the other disorders have remained quite constant, except for a small increase in bipolar disorders.

[40] Note that all disorder indicators were included in the same model. This means that we have taken into account, for example, that a person with depression also may have one of the other disorders. If depression had been included as the only disorder indicator, the effect would have been slightly stronger.

[41] One reason for the lower fertility among men than women is the surplus of males at birth, and therefore among individuals in the reproductive age group. An additional reason is that men tend to have children with women born a couple of years earlier, and that these younger cohorts have been smaller because of a fertility decline over the relevant decades (Kravdal 2021).

[42] A particularly good example is personality disorder among women, where a sharpening of the association with the first-birth birth rate over increasing age in the simplest models is changed to an almost age-independent association in the most complex model.

[43] The directions of causality are not clear. First, while it is plausible that partnership status is a determinant of fertility, it is also possible that plans about childbearing affect the chance of entering or remaining in a union. Second, mental health problems may reduce the chance of being in union, but the partnership status may also have implications for mental health. (As mentioned, the directions of causality would be difficult to establish even if the mental health indicators and partnership status were measured in different years.) To the extent that there is an adverse effect of mental disorders on the union entry rates, this may be a result of being less attractive as a mate because of lower income potential or other characteristics, or meeting relatively few potential partners because of less involvement in education or social activities.

[44] In principle, it would also make sense to expect that, as they proceed to higher parities in spite of the challenges they face, individuals with mental health problems would have an increasingly positive score on an unobserved constant fertility determinant that has the same distribution for everyone (regardless of mental disorders) at the start of the reproductive process. However, this idea was not supported by a multilevel-multiprocess model that deals with this type of selection; as mentioned in Appendix 1, the use of such a model did not change the results.

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