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# The Genetics of Partnership Dissolution

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**Abstract:** There is a genetic component to divorce risk, but little is known about which and how genetically influenced traits are involved. This study makes three major contributions to address these gaps. First, we link genetic data from the Norwegian Mother, Father, and Child Cohort Study (MoBa) to population register data and estimate the total influence of common genetic variants on partnership dissolution ($N = 121,408$). Then, we identify heritable traits associated with partnership dissolution using event-history analysis and a broad set of polygenic indices. Finally, we assess whether associations are robust to controls for confounding in within-sibling models. Significant heritability estimates were found for both females ($h2SNP = 0.09$; $SE = 0.01$; $p < 0.0001$) and males ($h2SNP = 0.03$; $SE = 0.01$; $p < 0.0001$). Genetic dispositions for educational attainment and other sociodemographic factors decrease the probability of partnership dissolution, whereas dispositions for internalizing symptoms and risk behavior increase the likelihood of partnership dissolution. Integrating genetics and sociodemographic approaches can shed new light on the causes of partnership dynamics by helping us understand what drives the selection processes throughout the life course.

**Keywords:** partnership dissolution; divorce; genetics; polygenic scores; fixed effects; MoBa

**Reproducibility Package:** See below for data availability statement. Access to administrative data from Statistics Norway can be applied for at Statistics Norway (http://www.ssb.no/mikrodata/) and access to MoBa Genetics can be applied for at the Norwegian Public Health Institute (http://www.fhi.no/studier/moba/). Code for data preparation and analysis is available at https://github.com/torkildl/genetics-dissolution

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PARTNERSHIP dissolution, whether in marital or non-marital relationships, is an increasingly common life event. Divorce rates have risen throughout the twentieth century, and in particular after the onset of what is often referred to as "the second demographic transition" (Goode 1993; Mortelmans 2020; Ruggles 1997). This trend started in rich countries but is now a common characteristic also of many middle- and low-income countries (Chen, Rizzi, and Yip 2021; Dommaraju 2016). Partnership dissolution now takes place in all human populations and in nearly all demographic groups.

Studying dissolution processes is important in its own right and also because dissolving a partnership can be highly consequential for both the couple and their children (Amato 2000; Leopold 2018; Leopold and Kalmijn 2016). The large research effort on partnership dissolution has to date not paid much attention to the role of genetically rooted individual differences. Here, we report an analysis of the genetic antecedents of partnership dissolution in a large Norwegian cohort that includes

data on couples' genotypes, showing that several antecedents of dissolution have genetic roots.

## *Genetics in Partnership Dissolution Processes*

Partnership dissolution rates are influenced by a complex combination of behaviors, attitudes, and social circumstances. The conventional approach to study partnership dissolution in sociology and economics has typically been to test predictions from a utility-maximization framework by comparing differential rates of dissolution (Härkönen and Dronkers 2006; Jalovaara 2001; Levinger 1976). Viewing partnership dissolution as the result of a dyadic process, caused by and affecting both partners in a couple, as well as their children and social surroundings, this line of research has documented many predictors of dissolution (Arpino, Le Moglie, and Mencarini 2021; Lyngstad and Jalovaara 2010). Factors such as a longer education, a higher age at marriage, and a man's higher income are associated with a lower divorce risk (Lyngstad and Jalovaara 2010). Other factors, such as doing shift work (Presser 2000), being in a ethnic or religiously heterogamous union (Smith, Maas, and van Tubergen 2012), and having experienced one's parents divorce (Amato and DeBoer 2001; Wolfinger 2009), are associated with a higher risk of own partnership dissolution.

From another research effort, in behavioral genetics, we have learned that human traits and behaviors are partly heritable and genetics contribute to individual differences in most human traits (Polderman et al. 2015; Turkheimer 2000). An implication of this is that socially contingent outcomes, such as partnership dissolution and its antecedents, are also partially influenced by genetic factors. Although these examples of antecedents are likely also genetically influenced, the knowledge of such genetic sources of variation is very limited in the extant literature. Educational gradients in divorce, for instance, may be partly reflective of genetically rooted individual differences that are rewarded in the educational system, and not necessarily of social or economic consequences of having completed a longer education.

Psychosocial family research has found that high subjective- and psychological well-being may have a protective effect against divorce, while divorce rates are higher in couples where one or both spouses report mental health issues (Butterworth and Rodgers 2008; Mastekaasa 1994). Risk -averse individuals are also less likely to divorce than their risk-tolerant counterparts, and higher risk tolerance is associated with more excessive divorce risk for women than for men (Light and Ahn 2010). Both subjective well-being and risk tolerance are partly attributed to individual genetic differences, with heritability estimates of ~30 percent to ~40 percent (Bartels 2015; Beauchamp, Cesarini, and Johannesson 2017; Harden et al. 2017; Nes and Røysamb 2017; Røysamb et al. 2018; Røysamb and Nes 2019).

The literature on antecedents of divorce includes many well-established correlations, but generally fewer causal effect estimates. Individuals' genetic profiles are fixed at conception, and when used in concert with appropriate statistical models and family demographic data, they offer an opportunity to better identify causal processes that contribute to partnership dissolution. Information on individuals' genetic profiles also let us explore dimensions of dissolution risks that are often

not covered by conventional data sets. For instance, measures of neuroticism, body mass index (BMI), subjective well-being, and number of sexual partners are not available in demographic data sources, but their genetic roots can be explored using genetic methods.

A small number of twin studies have demonstrated that genetics account for a substantial proportion of the variation in divorce, with heritability estimates ranging from 15 percent to 53 percent (D'Onofrio et al. 2007; Jerskey et al. 2010; McGue and Lykken 1992; Salvatore et al. 2018). Jockin, McGue, and Lykken (1996) studied the link between personality and divorce using a twin study design and found support that genetic effects on divorce went through personality. About 30 percent and 42 percent of the heritability of divorce for women and men, respectively, could be attributed to common genetic variants affecting personality variation in one spouse (Jocklin, McGue, and Lykken 1996). Genetic factors also contribute to the intergenerational transmission of divorce (D'Onofrio et al. 2007; Salvatore et al. 2018). An adoption study showed that adopted children resemble their biological parents to a larger extent than their adoptive parents in their history of divorce (Salvatore et al. 2018). Despite this, we still know very little about what the heritability of partnership dissolution contains, which genetic dispositions are involved, and how individuals' genetic traits are associated with partnership dissolution. Heritability estimates alone establish only that genetics *somehow* play a role in the process and are silent about any causal pathways from genetics via the wide range of likely mediators to partnership dissolution. Clearly, there is no such thing as a "divorce gene," as complex behavioral traits like partnership dissolution involve many–or all–facets of human psychology and physiology as well as interactions between partners and with the social environment. The aim of this study is to assess the role of genetics in partnership dissolution behavior, while acknowledging both its social complexity and its polygenicity.

## *The Contribution of This Study*

Using a linkage between the Norwegian Mother, Father, and Child Cohort Study (MoBa) and administrative register data, we performed a comprehensive study of the genetics of partnership dissolution. Our contribution is threefold. First, we quantify the total influence of common measured genetic variants on partnership dissolution in a relatively homogeneous Norwegian cohort using Linkage disequilibrium (LD) score regression. Second, we explore the nature of this genetic influence using the standard demographic approach to study partnership dissolution: In discrete-time event-history analyses, we prospectively assess associations between partnership dissolution and a range of polygenic indices (PGIs), such as education, demographic and sexual behavior, risk behaviors, and psychological characteristics as well as anthropometric traits. We consider males and females separately, but in supplementary analyses we also test the importance of within-partnership differences in PGIs, that is, dyadic genetic similarity (cf. online supplement figure 5).

Associations between PGIs and an outcome cannot be interpreted as causal effects due to the many potential confounding factors. For instance, Genome-

wide association studies (GWAS) results may reflect gene−environment correlation related to ancestry, geography, or socioeconomic status, and suffer from bias due to indirect genetic effects (Trejo and Domingue 2018). In a third analysis step, we therefore estimate sibling fixed-effect models to better assess to what extent genetic associations with partnership dissolution are causal. While the resulting within-family effects let us move closer to a causal interpretation, environmental selection and moderation are still crucial in how these genetics are linked to partnership dissolution. They are not–and should not be interpreted as–purely biological causes.

Although partnership dissolution is a relatively common phenomenon in Norway today, there has been a slight decline in divorce rates since 2005, due to fewer marriages, and consequently more selection into marriage (Statistics Norway 2024). Many couples cohabit over longer periods, and some never formally marry. A large share of break-ups therefore happen in non-marital partnerships. Because our data include both married and cohabiting couples, with at least one non-adopted child, we avoid restricting the analysis to only married individuals. Divorce and partnership dissolution rates are also highly dependent on normative and cultural environments (Furtado, Marcén, and Sevilla 2013). Dissolving a partnership may require more personal resources in conservative regimes than in less restrictive normative climates. Norway represents a context at the liberal end of this spectrum. By leveraging data on a large demographically and culturally homogeneous cohort, we avoid conflating very different life experiences in a single analysis. Studying the genetics of partnership dissolution in the Norwegian context can nevertheless provide important insights into the heritability of relationship stability and the potential impact of genetic variants on relationship outcomes.

# Data and Methods

## *Data and Study Population*

*The Norwegian Mother Father and Child Cohort Study (MoBa).* MoBa (Magnus et al. 2016) is a population-based pregnancy cohort study conducted by the Norwegian Institute of Public Health. Participants were recruited from all over Norway from 1999 to 2008. The women consented to participation in 41 percent of the pregnancies. The cohort includes approximately 114,500 children, 95,200 mothers, and 75,200 fathers. Blood samples were obtained from both parents during pregnancy and from mothers and children (umbilical cord) at birth. The current study is based on the version 12 of the quality-assured data files released for research in August 2018. The establishment of MoBa and initial data collection was based on a license from the Norwegian Data Protection Agency and approval from The Regional Committees for Medical and Health Research Ethics. The MoBa cohort is currently regulated by the Norwegian Health Registry Act.

*Genotype data.* Genotyping array data for MoBa were generated as described previously (Corfield et al. 2022). Phasing and imputation was performed with IMPUTE4.1.2_r300.335, using the publicly available Haplotype Reference Consortium release 1.1 panel as a reference. To identify a sub-population of European-associated

ancestry, principal component analysis was performed with 1000 genomes phase 1 after LD pruning (Corfield et al. 2022).

*Norwegian Administrative Register Data.* We linked the MoBa sample to Norwegian population register data through personal anonymized identification numbers. These data, collected continuously through administrative systems, have no attrition, and include information on both marital status changes as well as household changes, allowing us to obtain annual information on partnerships and dissolutions across the adult life course.

*Samples.* Our samples are restricted to individuals who are participants in MoBa and have been genotyped. In the GWAS, we include all genotyped adult individuals in MoBa, which includes 52,035 males and 76,441 females. All individuals were in a partnership at the birth of their MoBa-participating child. Individuals in same-sex partnerships were excluded. The share of partnership dissolutions happening during the observational period was 26 percent. This number is relatively low compared to the general population, and likely due to the sample consisting of couples with common children, for which union dissolution rates are lower. The MoBa sample also comprises a larger share of individuals with relatively high educational levels, which may contribute to the observed lower rate of partnership dissolutions during the period. The within-family analysis consisted of 20,622 adult individuals from MoBa who had an adult sibling also included in the data set. To maximize statistical power in the within-family analysis, we combine information on men and women. Online supplement 1 provides descriptive statistics of the samples.

## Measures

*Outcomes.* We use two measures of partnership dissolution. For the GWAS and in the within-sibship eller sibling analysis, we use a static measure indicating whether the individual ever has experienced partnership dissolution: Individuals who never have experienced a partnership dissolution are coded as 0 and individuals who have experienced a partnership dissolution are coded as 1. For the event-history analyses, we use a dynamic measure indicating if the couple experienced a partnership dissolution in a given year while being at risk of the event.

We combine information on marriage and non-marital cohabitation to construct both measures. Across the industrialized world, rates of non-marital cohabitation have increased sharply, and patterns in causes and consequences of non-marital partnership dissolution mirror those for formal divorce. Non-marital dissolution events are thus being included along with formal divorce in any measure of partnership dissolution.

*Predictors: Polygenic indices.* PGIs, aggregations of many genetic associations for a trait into one single genetic propensity score for every individual in our sample, were calculated for 16 different phenotypes. We used beta weights from large, publicly available up-to-date GWAS of educational attainment (EA) (without 23andme)

$N = 765,283$ (Okbay et al. 2022), cigarettes per day $N = 377,334$ (Liu et al. 2019), depression (without 23andme) $N = 500,199$ (170,756 cases and 329,443 controls) (Howard et al. 2019), height $N = 1,502,499$ (Yengo et al. 2022), Attention-deficit hyperactivity disorder (ADHD) $N = 225,534$ (Demontis et al. 2023), autism spectrum disorder $N = 46,350$ (Grove et al. 2019), being a morning person $N = 697,828$ (Jones et al. 2019), loneliness $N = 452,302$ (Day, Ong, and Perry 2018), age at first sex $N = 397,338$ (Mills et al. 2021), number of children ever born $N = 785,604$ (Mathieson et al. 2023), number of sex partners $N = 370,711$ (Karlsson Linnér et al. 2019), age at first birth $N = 542,901$ (Mills et al. 2021), subjective well-being $N = 298,420$ (Okbay et al. 2016), BMI $N = 695,648$ (Loic Yengo et al. 2018), drinks per week $N = 941,280$ (Liu et al. 2019), and neuroticism $N = 168,105$ (Turley et al. 2018).

PGI construction was done using LDpred software. LDpred takes a Bayesian approach that uses a prior on the expected polygenicity of a trait (assumed fraction of non-zero effect markers) and adjusts for linkage disequilibrium based on a reference panel to compute Single nucleotide polymorphism (SNP) weights. MoBa genotypes were first coordinated with the summary statistics. LD adjustment was performed using the European subsample of the 1000 genomes genotype data as the LD reference panel. The weights were estimated based on the heritability explained by the markers in the GWAS summary statistics and the assumed fraction of markers with non-zero effects.

## Statistical Methods

*SNP-based heritability of partnership dissolution.* First, GWAS was performed separately for males and females. Because MoBa includes relatives, we implemented mixed-linear models using *FastGWA* software with flags `-fastGWA-mlm-binary` and `-grm-sparse` to estimate SNP associations while controlling for a matrix of SNP-based genetic similarity, among other covariates (genotyping batch, chip, 10 principal components representing genetic ancestry, and age) (Jiang et al. 2019). Then, we calculated SNP heritability by inputting the GWAS summary statistics into LD score regression software (Bulik-Sullivan et al. 2015).

*Prospective event-history models of partnership dissolution risk.* To analyze associations between PGIs and partnership dissolution in a prospective design, we use discrete-time event-history analysis, the standard method in family demography for studying partnership dissolution (Lyngstad and Jalovaara 2010). The observational period was defined as the years from the birth of the MoBa child up to 2018. Every person is observed until the occurrence of the dissolution or until censoring at the end of the study period in 2018. To be amenable to discrete-time event-history analysis, the data set was converted to observations of person-years. In each of the included person-years, an individual is at risk of experiencing partnership dissolution. Logistic regression was used to estimate parameters in the discrete-time event-history models with the dynamic outcome variable coded 1 if dissolution took place that year and 0 otherwise. All PGIs were included simultaneously in the model, and men and women were assessed separately (but see the online sup-

plement for the analysis of squared differences in PGIs between partners). The analytical sample included 1,542,287 person-year observations.

The prospective event-history models were specified as follows:

$$\log\left(\frac{P(D_{it}=1)}{1-P(D_{it}=1)}\right) = \alpha + \sum_{j=1}^{n}\beta_j PGI_{ij} + \gamma_1 Batch_i + \gamma_2 \chi p_i + \sum_{k=1}^{10}\delta_k PC_{ik} + \varepsilon_{it}$$

where $P(D_{it}=1)$ is the probability of partnership dissolution at time $t$ for individual $i$; $\alpha$ is the intercept; $PGI_{ij}$ represents the $j$th polygenic index for individual $i$, with all PGIs included simultaneously; $\beta_j$ is the coefficient for the $j$th PGI; $Batch_i$ and $\chi p_i$ account for genotyping batch and chip effects, with coefficients $\gamma_1$ and $\gamma_2$; $PC_{ik}$ represents the $k$th principal component of ancestry for individual $i$, with $k = 1, \ldots, 10$ and corresponding coefficients $\delta_k$; and $\varepsilon_{it}$ represents the error term. This equation applies to both men and women, with the models run separately by sex in our analysis.

*Within-family models of prospective partnership dissolution risk.* We assess the potentially causal influence of genetics on partnership dissolution using sibling fixed-effect models. Some of the adults in MoBa have full siblings that can be identified through the administrative register data (i.e., they have the same mother and father identifiers). Sibling fixed-effect models control for factors that are shared between siblings, which may also influence their risk of partnership dissolution. Such shared factors could, for instance, be features of their family background, parents' own marital behavior and parenting styles. Siblings share 50 percent of their genetics, and their PGIs will also be correlated at 0.5 (assuming no assortative mating [AM]). Event-history analyses with sibling fixed effects are subject to problems of differential censoring (Allison 2005), and we therefore collapse the person-year data into individual-level data indicating whether or not a dissolution took place for that sibling in the follow-up period.

The sibling models were specified as follows:

$$\log(P(Y_{im}=1)) = \alpha + \sum_{k=1}^{K}\beta_k \cdot PGI_{imk} + \delta_i + \varepsilon_{im}$$

where $\alpha$ is an overall intercept and $\beta_k$ represents the coefficient for a specific PGI $k$, with additive effects summed over $K$ different PGIs. In the single PGI models, $K$ is equal to 1 and the model reduces to only have one $\beta$ term. The $\delta$ term is specific to each sibling group (i.e., a sibling fixed effect), and the $\varepsilon$ term represents an individual-specific error term.

The parameters in the within-family models are identified through differences in PGIs within sibling pairs. Such differences are randomly generated during meiosis at conception, and the resulting parameter estimates, obtained net of unobserved family factors, thus represent near-causal effects of PGIs on partnership dissolution.

# Results

## *The Heritability of Partnership Dissolution*

We estimated the SNP-based heritability of lifetime partnership dissolution in 52,035 males and 76,441 females in the MoBa study using LD score regression (Bulik-Sullivan et al., 2015). The input for this method was genome-wide association summary statistics derived from the `-fastGWA-mlm-binary` option in *fastGWA* software. Heritability estimates for dissolution using LD score regression (liability scale) were significant for both females ($h^2{}_{SNP} = 0.09$; $SE = 0.01$; $p < 0.0001$) and males ($h^2{}_{SNP} = 0.03$; $SE = 0.01$; $p < 0.0001$). The SNP-based genetic correlation for partnership dissolution in females and males (LD score regression) was close to 1 at 0.75 ($SE = 0.15$). In additional analyses, we estimated the liability scale SNP heritability of relationship dissolution in the combined sample of males and females to be 0.06 (0.01); however, the observations are not independent due to the presence of couples in the data set. See the online supplement figures 1–4 for more detail on this analysis.

## *Polygenic Indices Associated with Partnership Dissolution*

We followed men and women in MoBa in partnership records obtained from longitudinal Norwegian population register data to assess associations between dissolution and the genetics of one or both partners in the couple. We selected individuals who were either married or cohabiting at the start of data collection (the pregnancy of the MoBa-sampled child). Each individual was followed from the birth of their MoBa-sampled child and until dissolution or end of follow-up. The observation period ended in 2018. Based on the 16 different genome-wide association studies, we calculated PGIs for every individual in the sample. We used large-scale studies reflecting a broad range of phenotypes, covering behavioral and anthropometric traits as well as phenotypes considered antecedents of union dissolution in conventional sociological and demographic studies. Using event-history analysis, we assessed associations between the PGIs and risk of partnership dissolution. In total, data for the event-history analyses included 1,542,287 person-year observations.

Figure 1 shows changes in the OR for partnership dissolution with a one standard deviation (SD) increase in PGIs for women and men separately (with numerical results available in the online supplement 2). Note that the baseline annual risk of partnership dissolution is relatively low, so an odds ratio of partnership dissolution higher or lower than 1.0 does not necessarily entail a large increase in the absolute risk of dissolution. PGIs representing phenotypes that are known sociodemographic correlates of partnership dissolution, like a higher age at first birth and holding a higher education, were associated with a lower probability of partnership dissolution. One SD increase in the PGI for EA was associated with an approximately 10 percent lower annual odds of experiencing partnership dissolution (men: OR 0.899, 95 percent CI 0.880–0.918; women: OR 0.892, 95 percent CI 0.877–0.907). Weaker associations in the same direction were found for age at first sex (men: OR 0.95, 95 percent CI 0.93–0.97; women: OR 0.92, 95 percent CI 0.90–0.93), age at first birth (men: OR 0.94, 95 percent CI 0.92–0.97; women: OR 0.94, 95 percent CI 0.92–0.95),

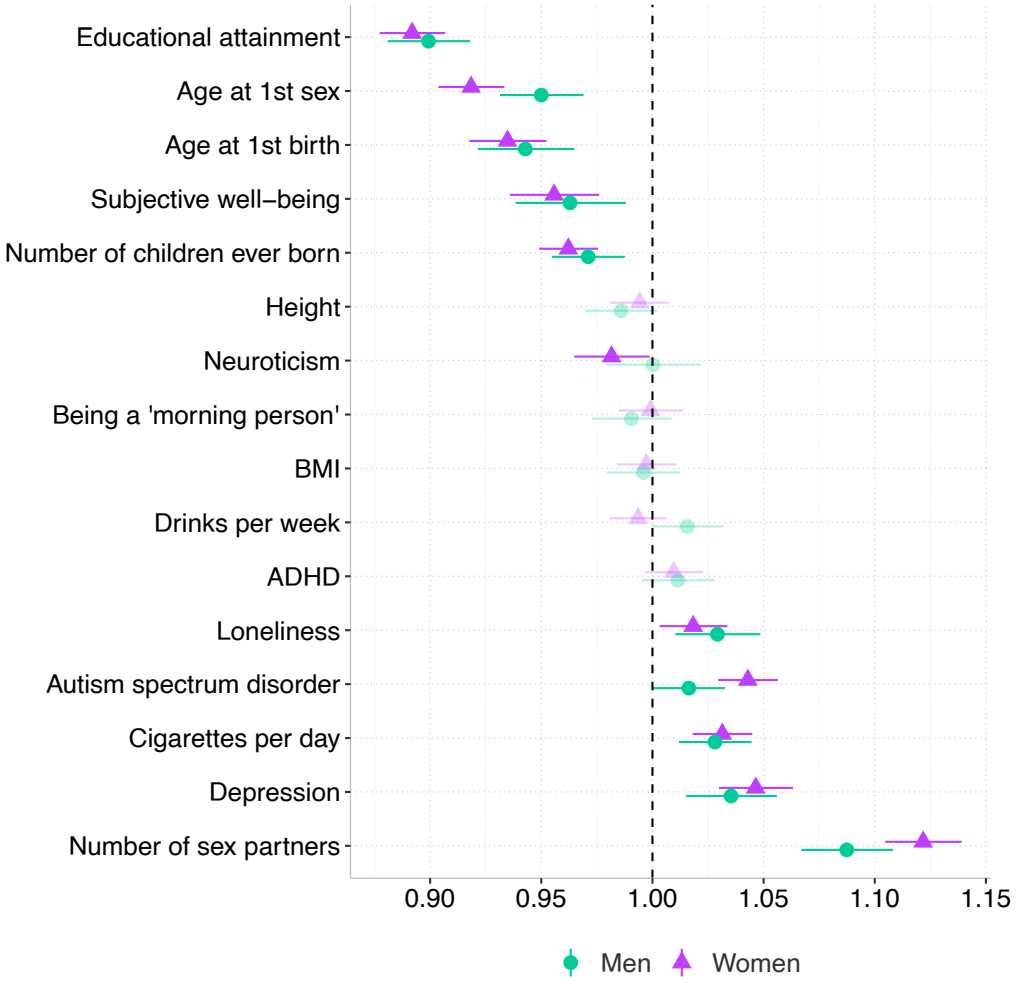


**Figure 1:** Results from discrete time event-history models for men and women showing OR for partnership dissolution by own PGI. Horizontal error bars represent 95 percent confidence intervals. All 16 PGIs were *z*-standardized and simultaneously included in the model. Non-significant estimates (CI includes 1.0) are shown in partial transparency. All models were adjusted for genotyping batch, chip, and 10 principal components of ancestry.

subjective well-being (men: OR 0.96, 95 percent CI 0.94–0.99; women: OR 0.96, 95 percent CI 0.94–0.98), and number of children ever born (men: OR 0.97, 95 percent CI 0.95–0.99; women: OR 0.96, 95 percent CI 0.95–0.97).

On the other hand, having a higher PGI for traits associated with risk behavior and mental health conditions such as number of sex partners, smoking, and depression was associated with increased partnership dissolution risk. Women who had a one SD higher PGI for number of sex partners had approximately a 12 percent higher annual odds of dissolving their partnership (OR 1.12, 95 percent CI 1.10–1.14), whereas the corresponding association for men was 9 percent higher (OR 1.09, 95 percent CI 1.07–1.11). The PGIs for depression, loneliness, cigarettes smoked per day, and autism spectrum disorder were also associated with a higher

odds of dissolution. For a number of other traits–BMI, ADHD, height, and "being a morning person"–weaker and non-significant associations were found.

As the process of partnership dissolution is in its nature dyadic, we tested whether the difference between ego's PGI and their partner's PGI predicted partnership dissolution, that is, whether similarity in genetic dispositions is linked to dissolving a partnership. In models with squared differences between partners' PGIs where all such differences were included, no parameter estimate was statistically significant. In single PGI models, "being a morning person," was significantly associated with partnership dissolution (OR 1.01, 95 percent CI 1.00–1.01).

### *Predicting Partnership Dissolution in Within-Family Models*

Associations between PGIs and partnership dissolution may not only emerge due to complex causal mechanism chains but also due to unmeasured confounding. To address this problem, we exploit sibling relationships in the data to estimate sibling difference models of partnership dissolution. Siblings share 50 percent of their genetics, and their genetic differences are random. Observed and unobserved family background differences are also attenuated between siblings because they share the same parents. In total, there are 10,280 parents in our sample who have a sibling who is also part of the study. In order to maximize limited statistical power in this analysis, we combine information on men and women and estimate logistic regression models of dissolution at any time throughout the follow-up period. This comes at the expense of the dyadic perspective but is justified by the largely similar pattern in estimates for men and women revealed in the previous analysis.

Figure 2 shows associations between PGIs and the risk of partnership dissolution during the follow-up period. We first regressed each PGI on the outcome in separate models. The resulting estimates are shown in blue color. Statistically significant associations with a lower odds of partnership dissolution were found for age at first sex (OR 0.72, 95 percent CI 0.65–0.80), subjective well-being (OR 0.74, 95 percent CI 0.67–0.81), EA (OR 0.74, 95 percent CI 0.67–0.82), age at first birth (OR 0.75, 95 percent CI 0.68–0.82), number of children ever born (OR 0.87, 95 percent CI 0.79–0.95), being a "morning" person (OR 0.88, 95 percent CI 0.80–0.96), and BMI (OR 0.90, 95 percent CI 0.82–0.99). Significant associations with a higher odds of partnership dissolution were found for number of sex partners (OR 1.18, 95 percent CI 1.07–1.29), cigarettes per day (OR 1.18, 95 percent CI 1.07–1.30), loneliness (OR 1.31, 95 percent CI 1.19–1.44), and depression (OR 1.34, 95 percent CI 1.22–1.48).

Several of the included PGIs have fairly strong genetic correlations, and effect estimates may therefore reflect horizontal pleiotropy. In an additional model (estimates in orange color), all PGIs were included simultaneously, which let us obtain effect estimates where such contributions are purged. In this model, four of the PGIs had statistically significant effects on partnership dissolution during the follow-up period: subjective well-being (OR 0.75, 95 percent CI 0.65–0.88), neuroticism (OR 0.82, 95 percent CI 0.72–0.94), age at first sex (OR 0.85, 95 percent CI 0.75–0.96), and EA (OR 0.86, 95 percent CI 0.76–0.98). These PGI associations obtained from within-family models have interpretations that are markedly closer to causal effects than the associations obtained in event-history models, given that they are associations

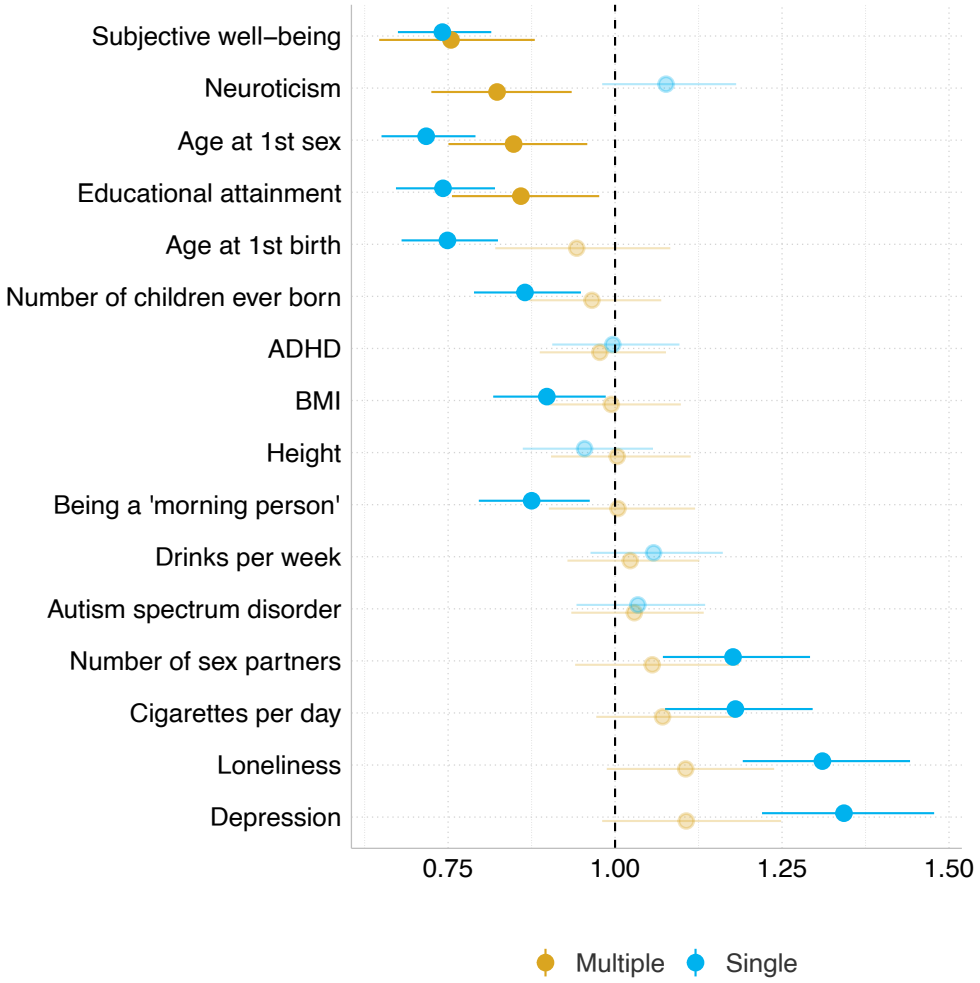


**Figure 2:** Associations between 15 PGIs and partnership dissolution during follow-up period. Error bars represent 95 percent CIs. PGIs were *z*-standardized. Blue color indicates that the PGIs were assessed separately. Orange color estimates are from models where all PGIs were assessed simultaneously. Non-significant estimates (CI includes 1.0) are shown in partial transparency.

estimated from what presumably is random sibling differences in PGIs. Numerical results are available in the online supplement 3.

## Discussion

Partnership dissolution has been a subject of enduring interest across social sciences (Goode 1993). Dissolving a partnership is a consequential life event, involving all kinds of social and psychological aspects, of which many (if not all) have genetic antecedents (McGue and Lykken 1992). This study adds three major contributions to the investigation of the genetics of partnership dissolution. First, we quantified the variance explained by genetic factors in dissolution by providing heritability estimates obtained from analysing measured genotypes. Second, we performed

event-history analyses linking PGIs for a broad range of traits to partnership dissolution, establishing associations of non-trivial magnitude. Third, we presented within-family estimates of PGI effects on prospective dissolution that more easily lend themselves to causal interpretations.

### *Partnership Dissolution is Linked with Genetic Variation*

The results from the first genomic study of partnership dissolution conformed to our expectations based on previous pedigree studies. Partnership dissolution was, to some extent, genetically influenced. The *h*2SNP estimates were ~0.09 and ~0.035 for females and males respectively, suggesting a polygenic nature of partnership dissolution. Nevertheless, the estimates were small, and notably smaller than those reported in previous twin and adoption studies, consistent with the anticipated difference between pedigree and SNP-based heritability estimates (D'Onofrio et al. 2007; McGue and Lykken 1992; Salvatore et al. 2018).

DNA-based studies of divorce have not been conducted previously, largely due to the absence of data. Conventional GWAS pool data from different countries and birth cohorts to maximize sample size. This implicitly assumes that the effects of individual variants are identical across populations. Historically, the rate of change in partnership dissolution behavior has varied greatly across contexts, and both acceptance of dissolution and the opportunity structures for dissolving a partnership are affected by developing normative climates and social−political regimes (Musick and Michelmore 2018). The role of genetics (or genetically rooted resources) for partnership dissolution is thus expected to vary across contexts. Access to register data linked to a large, genotyped sample from Norway allowed us to perform a well-powered SNP heritability analysis in a demographically homogeneous cohort, largely avoiding this issue.

Overall, the phenotypic variance explained by SNPs of partnership dissolution in our analysis was modest. Partnership dissolution is a dyadic process, involving much of both partners' psychological and social contexts, and their interactions. The causal chain from genetic propensities to partnership dissolution is therefore long and complex. Moreover, thousands of SNPs are likely to contribute to the heritability of partnership dissolution. Many different genetic variants may, in sum, contribute to, for example, slightly increased partnership dissolution risk via many heritable traits and behaviors. However, genetic factors are non-deterministic, and their manifestations can be modified through environmental exposures and interventions. Although our results do not provide insights into the pathways linking genetic variants to partnership dissolution, our analysis using PGIs enabled a more focused exploration of specific traits.

### *A Range of Heritable Traits are Associated with Relationship Breakup*

The event-history analysis revealed several associations between PGIs and partnership dissolution for both females and males. The pattern in associations by and large mirror associations published in the existing literature of antecedents of divorce (Amato 2010; Lyngstad and Jalovaara 2010; Mortelmans 2020). The PGIs of classical sociodemographic antecedents, and especially education and age at first

birth, are associated with lower risk of partnership dissolution. This corresponds to the phenotypic patterns, as in Norway and comparable countries, there is a clear association between spouses' EAs and age at first birth on the one hand and partnership dissolution on the other hand (Lyngstad and Jalovaara 2010). However, the magnitude of associations is mostly lower for the PGI results than what is observed in the literature for phenotypic associations. This is not surprising given the fact that PGIs are relatively noisy summary measures, and the measurement error alone will attenuate associations.

The PGIs related to risk tolerance and mental health (ever being a smoker, depression, and number of sexual partners) are associated with an increased risk of dissolving the union. This also resonates with previous findings and theories stating that risk-averse individuals are less likely to dissolve their unions (Light and Ahn 2010). In these event-history models, as the PGIs are based on GWAS results, the associations are prone to confounding from population stratification and indirect genetic effects (Morris et al. 2020). Stated differently, genetic associations may reflect social mechanisms and not a causal path from genetic variants through various stages of the life course including forming a union and thus coming under risk (a higher or lower) of dissolving the partnership.

### *Some Genetic Signals Survive a Stringent Test of Causality*

Within sibling pairs, both population structure and family background effects that are similar for both siblings (including indirect genetic effects) should be moot. There is a randomization of which alleles are transmitted from the parents to each sibling, and the resulting genetic deviation within siblings should therefore be independent of shared environmental influence (Selzam et al. 2019). As several of the included PGIs exhibit fairly strong genetic correlations, effect estimates may still reflect horizontal pleiotropy, where genetic variants have diverse effects on multiple traits. Because observed effects may not be specific to the trait of interest, but influenced by shared genetic factors affecting other traits, all PGIs were included simultaneously. The results from these within-sibling analyses therefore allow us to move closer toward causal interpretations. In the multiple-PGI sibling analysis, signals for lower odds of partnership dissolution remained for PGIs for subjective well-being, neuroticism, age at first sex, and EA.

Educational gradients in divorce are well-documented, but it is unclear exactly what social mechanisms these gradients reflect. Some argue that education enhances both human capital, cognitive and non-cognitive skills, leading to increased relationship stability through mechanisms such as more compatible partner matching or improved communication skills (Härkönen and Dronkers 2006). Our results imply that educational gradients in partnership dissolution may not solely be reflective of a person's social or economic resources or status but also reflect unobserved differences that emerge through selection mechanisms operating throughout the educational system.

The within-family estimates of partnership dissolution show that all associations are reduced, and most do not survive after adjustment for shared family background. Although the PGI estimates from regular OLS- and event-history

models are probably biased upward due to confounding, studies have suggested that within-family models suffer from the opposite challenge, yielding more conservative measures of PGI effects (Trejo and Domingue 2018). Taken together, the "between-family" event-history estimates and the within-family PGI effects may therefore serve as upper- and lower-bound estimates of the relationship between the various PGIs and partnership dissolution (Trejo and Domingue 2018). With larger sample sizes of siblings with genotype and relationship data, we are likely to see a greater number of PGIs survive controls for population structure and indirect genetic effects.

Although important confounding sources are mitigated in the sibling analyses, bias from AM may remain. In the presence of AM, siblings will be more genetically similar. As the within-family analysis relies on essential variation in the within-family deviation in PGIs between siblings, this would, however, also introduce downward bias in the estimates, and thereby increase the risk of type II errors. AM might also function as a mechanism or mediator influencing the likelihood of partnership dissolution. We know that partners exhibit positive genetic correlations for various traits, such as, for instance, EA (Sunde et al. 2024). In the within-family analyses, considering the presence of AM, the PGIs may reflect not only the causal effect of an individual's PGI but also the indirect effect of the partner's PGI, arising from AM.

### *Sex Differences: $h^2$, Genetic Correlation between Sexes, and PGIs*

Our results indicate some sex differences. Although the heritability estimates for partnership dissolution were significantly higher for women compared to men, both estimates were relatively small. The genetic correlation between women's and men's summary statistics was 0.75 (SE=0.1459), which also suggests moderate sex differences. Although we cannot offer any direct explanation of such differences, the pathways from genetic dispositions to dissolution may be different for women and men. They could for example emerge from women having a more active role in dissolution processes, which resonates with some previous findings showing that women more often take the initiative to divorce (Kalmijn and Poortman 2005). In contrast to the genome-wide association analysis, the polygenic score analysis did not reveal any major sex differences (two polygenic scores, number of sexual partners and age at first sex, had associations that visually differed somewhat by sex, but these differences were not statistically significant). Replications with even larger data are needed in order to better understand these results.

### *Limitations*

The study has several limitations worth addressing. First, given that MoBa is a pregnancy cohort, all three samples used in analyses are restricted to individuals in opposite-sex relationships with a common child, a group known for having lower partnership dissolution rates compared to the general population (Joyner, Manning, and Bogle 2017). In addition, MoBa participants tend to have higher educational levels than the average Norwegian population, a trait associated with a reduced likelihood of partnership dissolution. Second, there is likely population

stratification in the heritability estimates from our genome-wide association study. Although this could have been addressed by doing a within-sibship GWAS, this would have greatly compromised statistical power, and potentially lead to insufficient power to detect meaningful associations. Future research could combine MoBa with other cohorts in a large-scale GWAS similar to those published for EA. Such an undertaking would require careful statistical and demographic considerations, due to the large international and demographic variations in the prevalence and correlates of relationship breakups. Third, despite the increasing popularity of PGIs, it is important to recognize their limitations (Burt 2024). PGIs only explain a small portion of the variation in a given outcome and explain variance varies across GWASs. In addition, they only incorporate contributions from common genetic variants, and their predictive power depends on the heritability of the trait in question and the quality of the GWAS, which typically ignores potential variation in effects of single genetic variants across contexts. The associations between PGIs and partnership dissolution uncovered in this study should be seen as documenting the existence of links between individual genetics and the experience of dissolving a marital or coresidential union but do not provide a full picture on the magnitude of those links.

### *Implications for Theory and Research on Partnership Dissolution*

Sociological and demographic studies on partnership dissolution and family dynamics have traditionally placed little emphasis on individual differences and their genetic roots. Our results indicate that such differences may play a role in shaping partnership choices and individual life courses. This article is the first to broadly assess the genetic antecedents of partnership dissolution using data on measured genotypes. With the rapid increase in genomic data and analytical tools, there are growing opportunities to combine these with conventional social science data sources (Harden and Koellinger 2020). We encourage further research to investigate the complex pathways mediating genetic effects, including gene−environment interaction studies, and to examine other aspects of family life using SNP data—especially in contexts where normative restrictions on partnership dissolution are stronger.

In conclusion, it is important for family scholars to acknowledge that genetics may be a source of variation in family related behaviors and outcomes. Studies of union dynamics and other life course transitions could benefit from incorporating sociogenomic perspectives. Our study goes beyond classical twin and adoption studies of divorce. The findings contribute to a more nuanced understanding of partnership dissolution beyond conventional social scientific frameworks, highlighting the intricate and multifaceted nature of these processes. PGIs allow us to investigate multiple individual differences seldom available in phenotypic data sets and enable the examination of causal questions using within-family methods.

However, it remains crucial to recognize that for a process as complex as partnership dissolution, genetic factors are just some—and still small—pieces of the puzzle. Just as social influences cannot be fully understood without reference to partly heritable individual differences, genetic influences must be considered alongside social, cultural, political, and environmental factors that contribute to variation

in partnership dissolution rates (Musick and Michelmore 2018). This study of the genetic antecedents of partnership dissolution in the Norwegian context contributes to a more comprehensive understanding of the myriad factors shaping relationship dynamics.

## Data Availability Statement

The data analyzed in the study are administrative data maintained by Statistics Norway and genotype data from MoBa Genetics. The data are not publicly available but available to researchers upon application to the respective data owners. Such applications require approval by the appropriate ethics/research data access authorities. Access to administrative data from Statistics Norway can be applied for at Statistics Norway (http://www.ssb.no/mikrodata/) and access to MoBa Genetics can be applied for at the Norwegian Public Health Institute (http://www.fhi.no/studier/moba/). In Norway, the appropriate ethics and research data boards are the Regional Committee on Medical Research Ethics (REK) or SIKT. The consent given by the MoBa participants does not open for storage of data at individual level in repositories or journals.

## Code Availability Statement

The software used in the data preparation and analysis was R 4.2.3, LDPred 2.0, plink2, and fastGWA. Code for data preparation and analysis is available at https://github.com/torkildl/genetics-dissolution.

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