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Does parental separation moderate the heritability of health risk behavior among adolescents?

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Abstract

Social influences on adolescents' health risk behavior are well documented, but little is known about the interaction of parental separation with genetic sensitivities. Using data from a German sample of 1,824 twins, this study examines whether family living arrangements moderate the extent to which health risk behavior among adolescents is influenced by genetic predispositions. Derived from variance decomposition moderator models, the results provide evidence of a significantly larger genetic contribution to smoking among adolescents living in single-mother families than among adolescents living with both parents, but not of the moderation of heritability for drug use and excessive alcohol consumption. Thus, these findings indicate that the unfolding of genetic risk is increased for smoking, but not for other substances. However, the significantly stronger influences of individual experiences of drug use observed in single-mother families reveal the overall vulnerability of families who have experienced parental separation.

1 Introduction

Health risk behavior related to substance use often initiates in adolescence, and has far-reaching implications for later life (Short and Mollborn 2015; Umberson, Crosnoe, and Reczek 2010). Research has found associations between substance use and lower life expectancies (Martikainen et al. 2014) and higher chronic disease risks (Kaur, Kaur, and Kumar 2022), as well as increases in mental disorders (Flensborg-Madsen et al. 2011), school dropout rates (Bray et al. 2000), and adverse labor market outcomes (Böckerman, Hyytinen, and Maczulskij 2017).

Studies reporting declines in the prevalence of substance use among adolescents in Germany (Zeiher et al. 2018), and in other European countries (Looze et al. 2015) and the United States (Patrick and Schulenberg 2014), have emphasized the success of preventive public health measures in general. At the same time, it is particularly important to identify individuals who are vulnerable to using substances such as tobacco, alcohol, and other drugs. Previous empirical studies found that individuals' risk of engaging in health risk behavior differs depending on both their social

circumstances and their genetic sensitivities. It has been widely shown that for certain social groups and circumstances, the risk of substance use increases due to, among other factors, higher stress levels (Short and Mollborn 2015; Umberson et al. 2010). Other studies found substantial genetic influences on smoking, alcohol consumption, and drug use, which means that traits closely associated with these forms of substance use are affected by variations in a large number of genetic variants (Edenberg and Foroud 2013; Kreek et al. 2005; Li 2008).

In addition to research that focused on either social or genetic influences, several studies analyzed the interplay of social and genetic influences in order to explain differences in substance use (Kendler et al. 2014; Pampel et al. 2015; Timberlake et al. 2007). These studies were built on the fundamental understanding that genetic and social influences do not simply have additive effects on specific traits. Instead, they emphasized that environments can increase or decrease the influence of genetic predispositions, called *gene-environment interaction* (GxE) (Boardman and Fletcher 2021; Harden and Koellinger 2020; Mills and Tropf 2020). Thus, these studies argued that genetic risks for substance use are more likely to be realized under certain environmental conditions.

The examination of social contexts that might affect the expression of genetic risk factors could help to identify social groups particularly vulnerable to health risk behavior. Although previous research has investigated a variety of environmental moderators, such as levels of parental monitoring (e.g., Dick et al. 2007), most studies have ignored family living arrangements as a significant environmental context of individuals. This is surprising, given that parental separation is a major explanation for adolescents' health risk behavior (Griesbach, Amos, and Currie 2003; Kirby 2002; Wolfinger 1998).

In this paper, we address this gap, and study whether adolescents in single-mother families who have experienced a parental separation have a higher unfolding of genetic risk of health risk behavior than adolescents living in two-parent families. Using data from the German TwinLife study, we examine adolescent twin pairs in terms of their similarities and differences, and use structural equation modeling to investigate whether family living arrangements moderate the genetic influence of smoking, drug use, and excessive alcohol consumption.

2 Background

2.1 Parental separation and risk behavior

Studies have shown that adolescents are more likely to engage in health risk behavior when they are experiencing a parental separation or are living in a single-mother household (Umberson et al. 2010). Prior research has uncovered evidence that adolescents in single-mother families have higher rates of smoking (e.g., Griesbach et al. 2003; Kirby 2002; Wolfinger 1998), heavy drinking (e.g., Brown and Rinelli 2010; Flewelling and Bauman 1990; Isohanni et al. 1994), and drug use (e.g., Barrett and Turner 2006; Cavanagh 2008; Needle, Su, and Doherty 1990) than adolescents in two-biological-parent families.

Three main theoretical approaches to address the impact of family living arrangements have been put forward in the literature. First, the sociological *stress theory* (Pearlin 1989) focuses on the stress component related to the conflict and instability that accompanies parental separation, which also affects the children (Gustavsen, Nayga, and Wu 2016), increasing their likelihood of engaging in risky behavior (Kirby 2002). Thus, individuals may engage in certain behaviors, such as substance use, to cope with stress caused by unpleasant experiences or environments. These coping strategies might be learned from close environments in childhood (Umberson et al. 2010:145). Previous research has found a positive association between experiencing a parental separation or living in a single-parent family and stress in adolescents (Barrett and Turner 2005; Cavanagh 2008). Among the sources of stress that could induce adolescents to engage in risky behavior are economic deprivation, increased parent-child conflict, and having a less supportive parent-child relationship (Broman, Li, and Reckase 2008; Rattay et al. 2018). All of these stressors could lead adolescents to engage in risky health behavior as a coping strategy (Barrett and Turner 2006; Needle et al. 1990). This association has been found in previous research: Barrett and Turner (2006) showed that an increase in levels of drug use in adolescents from single-parent families can be explained by higher stress exposure; while Kirby (2002) found that parental separation increases the likelihood that adolescents will start smoking, and that this effect is partly mediated by increased depressed mood.

Second, the *socialization theory* implies that adolescents growing up in a single-mother family might be more likely to socialize with risk-taking peers, which could, in turn, reinforce their own risky behavior (Broman et al. 2008). This is explained by single mother having fewer social resources after the experience of a separation to provide a safe and stable environment for her

children, and to protect them from adverse peer environments (Kirby 2002). A large body of research has found that peer networks affect adolescents' health risk behavior (e.g., adams et al. 2022; Ellickson et al. 2003). It has, for example, been shown that peer drug use (Broman et al. 2008), peer deviance in general (Barrett and Turner 2006), and the number of friends who smoke (Kirby 2002) are important mediators between substance use in adolescents and family living arrangements. Rattay et al. (2018) found that the association between parental separation and adolescents' smoking and drinking behavior is partly explained by levels of family cohesion, as parental separation leads to weaker ties and worse relationship quality between family members, which can, in turn, increase the likelihood that adolescents will smoke or drink. Kristjansson et al. (2009) found that higher smoking and drinking prevalence among adolescents who experienced parental divorce is partly explained by time spent with parents. Furthermore, Broman et al. (2008) showed that the relationship between family living arrangements and drug use among adolescents is mediated by parental warmth and acceptance.

Third, *social control* refers to parents emphasizing values, norms, and explicit rules to prevent their children from engaging in health risk behavior, and especially in substance use. Single mothers may face challenges in exerting control over their adolescent children because they have high emotional distress levels, or because they need to work more due to financial constraints (Wolfinger 1998). Previous research has shown that single mothers are less able to exert social control or to monitor their children (Demo and Acock 1996), and that parental control serves as a mediator between family living arrangements and adolescents' smoking and drinking behavior (Brown and Rinelli 2010; Kristjansson et al. 2009).

Although the concepts of *socialization, social control,* and *social stress* are introduced separately in the literature, they are strongly associated. There might be a link between stress and social control in the sense that adolescents experiencing particularly stressful situations or environments might be less responsive to parental control. Adolescents who are subject to less parental control or are rejecting parental control may start to socialize with more deviant peers. As all of these concepts lead to the expectation that adolescents will engage in more risky behavior following parental separation, we do not expect the effect directions to differ even if the mechanisms are interrelated.

2.2 Heritability of health risk behavior and gene-environment interaction

A large body of research has shown that genetic influences contribute to smoking behaviors, alcohol consumption, and drug use. However, when looking at the health risk behaviors that have been linked to genetic variation (termed heritability), we see that heritability estimates vary substantially across studies (e.g., Sullivan and Kendler 1999; Verhulst, Neale, and Kendler 2015). For instance, depending on the study and the indicator under consideration, estimates of the heritability of alcohol misuse range from 16% to 20% (Walters 2002); estimates of the heritability of smoking range from 11% to 84% (Hall, Madden, and Lynskey 2002; Li et al. 2003); and estimates of the heritability of drug use range from 6% to 76% for problem cannabis use (Verweij et al. 2010).

It has been argued that the substantial variation in the heritability of different types of substance use can be explained in part by the environment shaping the degree of realization of genetic risk (Boardman et al. 2011:1518). Three potential GxE mechanisms – which are partly in line with the theoretical approaches presented above – have been put forward in the phenotypic literature: *social triggering*, *compensation*, and *social control*. *Social triggering* suggests that certain genetic vulnerabilities only show up in specific environments, or are more pronounced there (Diewald et al. 2015; Shanahan and Boardman 2009). For example, increased stress might trigger the development of genetic risk by interacting with personal predispositions for certain behaviors (Shanahan and Hofer 2005). The assumption that the heritability of risk factors increases in adverse environments due to exposure to greater amounts of stress is similar to the claims made by the sociological stress theory presented earlier (Barr et al. 2016). Thus, a genetic predisposition to health risk behavior could be triggered by stress, which may be higher in a single-mother family with fewer financial and social resources.

The second mechanism is *compensation*, which is not simply the absence of detrimental contexts, but is, instead, the presence of a positive and enriched environmental setting that positively affects individual functioning by preventing the expression of genetic risk (Shanahan and Hofer 2005). Compensation as a GxE mechanism for health risk behavior might be expressed as two parents having more resources to spend time with their child, which might, in turn, prevent the emergence of a genetic predisposition to engage in risky behavior.

The third potential GxE mechanism is *social control*, which is similar to compensation in that both compensation and social control provided by specific environments can prevent or reduce genetic expression (Shanahan and Hofer 2005). However, they also differ in that compensation

refers to the prevention of genetic risk through enriched environments, while social control refers to social norms and structural constraints that limit individuals' behavior and agency (Shanahan and Boardman 2009). In the case of health risk behavior, social control could limit the realization of genetic predispositions. For example, unlike many single mothers, two parents may be able to prevent genetic risk from unfolding in their child because they have sufficient time resources to pay attention to the peers their child associates with, and the ability to insist on certain rules and norms being followed in the household.

Previous research has investigated the extent to which the environment affects genetic contributions to substance use for adolescents and young adults using different environmental moderators. Some of these moderators, such as differences in legal regulations, have been located at the societal level (Boardman 2009); while others, such as levels of parental monitoring, have been located in the immediate surroundings of individuals (Dick et al. 2007). Several of these studies found that levels of social control affect the realization of genetic endowments for substance use, such as for smoking. It has, for example, been shown that low levels of parental monitoring increase genetic influences on smoking (Dick et al. 2007), and that the heritability of daily smoking among adolescents is lower in U.S. states with higher cigarette taxes and more controls on cigarette vending machines and advertising (Boardman 2009). There is also evidence that social norms affect genetic contributions to substance use. For example, Timberlake et al. (2006) observed lower levels of heritability of smoking in more religious populations. In addition, the results of studies that examined the role of social contagion indicate that the role of peers varies depending on the outcome. Boardman et al. (2008) found that genetic influences on daily smoking are highest in schools where the most popular students smoke, whereas Kendler et al. (2014) found no evidence for GxE when analyzing how peer deviance moderates genetic contributions to drug use among adolescents.

2.3 The current study

Previous research has consistently shown that the heritability of substance use can be shaped by the environment, particularly among adolescents. Although some of these studies looked at the relevance of social origin (e.g., Barr et al. 2018; Timberlake et al. 2007), it is striking that the family, as one of the closest environments of children and adolescents, has received little attention in research on GxE related to health risk behavior. Given the large body of literature reporting strong associations between family living arrangements and substance use, it is clear that family

composition should be considered as part of the environment. Therefore, this study assesses the impact of family living arrangements on the health risk behavior of adolescents from a genetically informed perspective.

We are specifically interested in the differences in the genetic contributions to substance use behaviors between adolescents living with a single mother, who have experienced a parental separation, and adolescents living with both biological parents. We expect to find that the genetic influences on health risk behavior are greater among adolescents in single-mother families than among adolescents in two-parent families. We argue that, first, parental separation, as well as living in a single-mother family, is associated with increased stress, which could *trigger* genetic predispositions to health risk behavior. Second, in line with the *socialization theory* discussed above, we argue that single parents may have less time to spend with their children due to resource scarcity, and thus that *compensation* for genetic risks through strategies of parental support might be less effective in single-parent families than in two-parent families. Third, we argue that compared to single mothers, parents in two-parent families may be better able to monitor their children's behavior (*social control*), and may therefore be more effective in counteracting genetic influences on health risk behavior.

Here, we consider the German context, which may differ from other country contexts in some respects. First, alcohol consumption among young adults is quite common and legal (e.g., beer) from age 16 onward. By age 17, about 87% of adolescents have already consumed alcohol, with 11.7% of adolescents (ages 14 to 17) binge drinking regularly (Zeiher et al. 2018). A comparable pattern has been observed in the United States, where the percentage of adolescents and young adults who regularly binge drink is also slightly over 10% (Chen and Yoon 2019). Smoking has become less socially accepted, and, since 2007, people under age 18 cannot legally purchase tobacco products. Thus, smoking rates among youth in Germany have declined over the past decade. The proportion of children and adolescents (ages 11 to 17) who smoke on a daily basis decreased from about 14% in 2003-2006 to about 4% in 2014-2017, while the age of smoking initiation increased over the same period (Zeiher et al. 2018). A comparable decline has been reported in the U.S., although the prevalence of smoking among high school students in 2018 was higher in the U.S., at 8.1% (Hammond et al. 2019). With respect to drug use, it is important to recognize that cannabis is the most commonly used drug among adolescents in Germany, after alcohol and tobacco (Lampert and Thamm 2007). While the consumption of cannabis remains (with some

exceptions for medical use) illegal, there are currently efforts to legalize it under certain conditions in Germany.

3 Data, Variables, Method

3.1 Data

Our analyses are based on the first wave of the German Twin Family Panel (TwinLife) (Diewald et al. 2022). TwinLife is a socially and regionally stratified probability-based register sample that allows researchers to analyze twin families from across the social spectrum (Lang and Kottwitz 2020). The first wave was collected between 2014 and 2016, and includes a total of 4,097 twin pairs and their families residing in Germany. This total sample is composed of four age cohorts of approximately 500 monozygotic (MZ) twin pairs and approximately 500 same-sex dizygotic (DZ) twin pairs (Hahn et al. 2016). Face-to-face interviews were conducted with both of the twins separately and with their parents. The target population of our analyses is comprised of twin pairs from the third birth cohort (1997/1998), aged 16 to 18 at the time of the first survey wave. We restrict the sample to families in which both twins were living in the same household with either both biological parents or a single mother. Our final analysis sample consists of 1,824 twins nested in 912 twin pairs. Of these, 716 twin pairs were living in a two-biological-parent family, and 196 twin pairs were living in a single-mother household.

3.2 Variables

3.2.1 Outcomes and moderator

We examine three different forms of substance use as indicators of health risk behavior: smoking, excessive alcohol consumption, and drug use. These are derived from questions posed to all TwinLife participants who were at least 16 years old at the time of the interview. The question regarding smoking behavior was "*Do you smoke?*" The response categories were: 1 "yes, I'm a heavy smoker," 2 "yes, I'm a smoker," 3 "yes, I'm a light smoker," 4 "yes, I'm a social smoker," 5 "no, I'm a former smoker (I don't smoke anymore, but I did smoke)," and 6 "no, I never smoked (I don't smoke and I never smoked in the past)." We recoded these categories to a dummy variable indicating whether the participant was or was not a current smoker at the time of the interview, and coded categories 1 to 4 as 1 (current smoker) and categories 5 and 6 as 0 (current non-smoker). The question regarding excessive alcohol consumption was: "*How often would you say that you drink a lot?*" The response categories were: 1 "daily," 2 "several times per week," 3 "once a week,"

4 “1 to 3 times per month,” 5 “less frequently,” and 6 “never.” We also recoded this information into a dummy variable indicating whether the respondent was or was not regularly consuming alcohol excessively, and coded categories 1 to 4 as 1 (consumes alcohol excessively on a regular basis) and categories 5 and 6 as 0 (does not consume alcohol excessively on a regular basis). The question about drug use was: “*Have you ever taken drugs (e.g., marijuana, hash, ecstasy, cocaine, etc.)? We’re not referring to cigarettes or alcohol*.” This variable indicates whether the respondent had ever used illegal drugs (coded 1) or not (coded 0). The variables are included in the quantitative genetic models as continuous variables indicating the probabilities for engaging in the respective forms of substance use.

The family living arrangements in which the twins were living is our moderator variable. As the vast majority of single-parent households in Germany are headed by women (Geisler and Kreyenfeld 2019), we focus on twins who 1) were living with their single mother and compare them with 2) twins who were living with their biological parents. In order to analyze the theoretical reflections of potential effects of parental separation, we further restrict our sample to adolescents in single-mother households who had been exposed to parental separation. Accordingly, we excluded families of widowed single mothers and those in which the biological father of the twins had never lived in the same household.

3.2.2 Controls

In all models, we control for parental socioeconomic status (SES). This is an appropriate control variable, since prior research has shown that there are socioeconomic inequalities in both substance use (e.g., Pampel, Krueger, and Denney 2010) and family living arrangements (e.g., Jalovaara 2003). Furthermore, by investigating the heritability of substance use types adjusted for SES, we can strengthen the claim that potential differences in heritability are due to family living arrangements, and not to socioeconomic differences. Using a latent variable approach, we derive a family SES score from information on household income (net equivalent household income), parental education (International Classification of Education, ISCED), and parental occupation (International Socio-Economic Index of Occupational Status, ISEI) using confirmatory factor analysis (CFA). Beforehand, we performed a principal component analysis (PCA) on the z-standardized variables (as recommended by Reinecke 2014), which showed that the three indicators load sufficiently high on a common latent factor. For the CFA, variance of the latent factor was restricted to 1, and missing values were considered using Full Information Maximum Likelihood

(FIML). The SES scores derived from CFA are divided into terciles to indicate low, medium, and high SES families. This allows us to consider a non-linear association.

SES may be an explanatory mechanism of increasing heritability of health risk behavior in single-mother families. Households with more socioeconomic resources might have advantages in preventing the unfolding of genetic risks, and parental separation may negatively affect these resources. Therefore, we perform additional robustness checks of all models (presented in the appendix) without controlling for parental SES.

In addition, we control for sex in all models. Sex is an appropriate control variable, since men are reportedly more likely than women to respond to stress with externalizing behavior such as substance use, while women are more likely to exhibit internalizing behavior such as depression (Simon 2014). Furthermore, we control for age effects by design through the restriction to one cohort.

3.3 Method

We use a twin-based approach to examine the extent to which family living arrangements moderate genetic influences on health risk behavior. Similar to molecular genetic approaches based on polygenic scores (PGS), advanced twin models, like the Purcell model applied in this paper, provide a flexible approach to studying gene-environment interaction. Although twin-based approaches presumably overestimate whole genome contribution, PGS typically do not comprehensively capture, and thus underestimate, the whole-genome effect, while confounding with environmental influences is unclear (Burt 2022). Thus, each of the approaches has advantages and disadvantages.

According to the classical twin design as shown in Figure 1, the observed traits of two twins within a twin pair depend on three latent variables per twin (A, C, and E) and the means μ1 and μ2 (Jöreskog 2021). The latent variable A refers to the additive genetic component, which captures the averaged effects of the genome on the outcome of interest (Neale and Maes 1996). Additive genetic effects have been shown to contribute the most to the heritability of most human traits, while non-additive genetic effects, such as dominance or epistasis, are postulated to contribute relatively little (Zhu et al. 2015).

The additive genetic component A can be derived, since the genetic relationship between the twins is 1 for MZ twins, due to their identical genetical makeup; and is 0.5 for DZ twins, since they share, on average, half of their genes. This is based on the assumption of random mating of

the parents. To check this assumption and the robustness of our results, we additionally run our analysis with an adjusted score for the genetic relatedness of the DZ twins (following the approach of Loehlin, Harden, and Turkheimer 2009). The results are presented in the appendix.

The latent random variable C captures all shared environmental influences that make twins more similar to one another, and is assumed to be 1 for MZ and DZ twin pairs (Rowe, Jacobson, and Van Den Oord 1999). In other words, MZ and DZ twin pairs are assumed to share homogeneous environmental influences to the same extent (called the *Equal Environment Assumption*). The validity of this assumption has been supported several times in previous research (see Conley et al. 2013; Mönkediek 2020).

While C reflects only the homogeneous effects of the twins' shared experiences (Freese and Jao 2017), the latent random variable E incorporates all non-shared environmental influences that increase their trait dissimilarity (Plomin and Asbury 2005). E can refer to different environments, such as different peer networks, and to differences in perceptions of objectively shared environments (Mönkediek 2022). In addition, in statistical models, E also contains the error term.

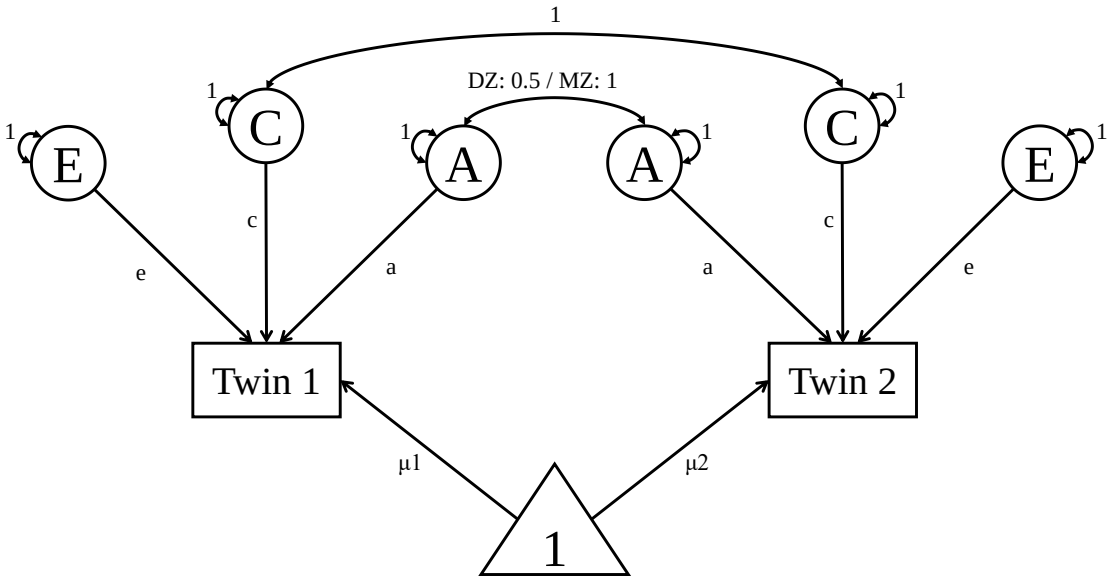

Figure 1: Classical twin design (Neale and Maes 1996)

The univariate GxE model by Purcell (2002) extends the classical twin model by incorporating a linear regression term on the path coefficients, as shown in Figure 2. Here, the influences of the latent variables A, C, and E, which are represented by a, c, and e in the classical twin design

in Figure 1, are extended by effects of the moderator M, and the coefficients of the moderator are represented by β_a, β_c, and β_e. The parameters a, c, and e thus refer to the effects when the moderator takes the value 0.

As described above, the dependent variables are continuous indicators of the probability of engaging in one of the substance use indicators, with the effects of socioeconomic status and sex partialized out. The models are estimated in R using version 4.10.5 of the umx package (Bates, Maes, and Neale 2019). While Figure 2 shows the full GxE model, it is possible that for the different outcomes considered here, sub-models that exclude certain paths fit the data better. We thus compare the model fit values for all possible combinations of models where certain parameters are included or excluded by fixing them to zero. The model comparisons for smoking, excessive alcohol consumption, and drug use as dependent variables are presented in the appendix. We identify the model with the best fit to the data based on the Akaike Information Criterion (AIC) in combination with a likelihood-ratio test (lrtest) (see Grasby et al. 2017). Here, we selected the most parsimonious model with the lowest AIC where the likelihood-ratio test indicated that it did not fit the data significantly worse.

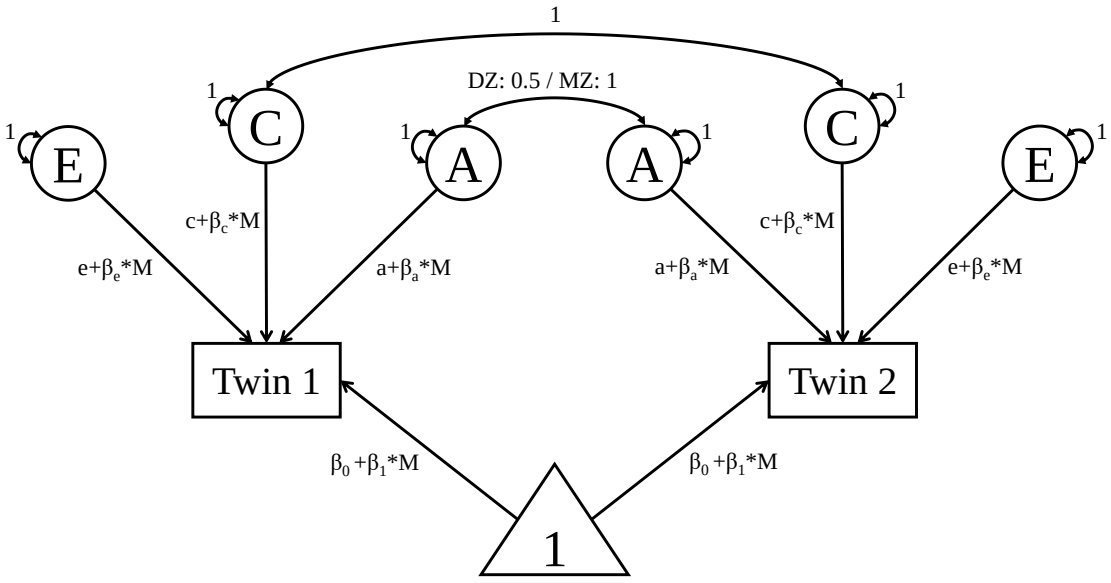

Figure 2: Univariate Purcell moderation model (Bates et al. 2019)

4 Results

Our study population includes 318 twins (18%) in single-mother families and 1,452 (82%) twins in two-parent families. Table 1 shows the descriptive statistics of our analytical sample by these two family types. The majority of twins in both family types are female (59.1% and 56%) and dizygotic (53.5% and 54%). The twins in both family types are, on average, 17 years old; while the single mothers are, on average, slightly younger (47.6 years) than the partnered mothers (48 years). As it can be expected, there are differences in income and education between the family types. The mean net equivalent household income in the single-mother families is less than two-thirds that of the two-parent families. In terms of educational attainment, single mothers are less educated than partnered mothers, with a much smaller percentage having secondary (31.6% versus 40.5%) or tertiary education (11.6% versus 14.6%).

Table 1: Socio-demographic descriptives

	Single-mother families	Two-parent families
Proportion female twins	59.1%	56.1%
Proportion monozygotic twins	46.5%	46.0%
Mean age twins	17.0	17.0
Mean age mother	47.6	48.0
Mean net equivalent household income	1047	1633
Proportion ISCED mother		
Primary education	3.2%	1.5%
Lower secondary education	8.4%	6.1%
Upper secondary education	43.2%	34.8%
Post-secondary, non-tertiary education	31.6%	40.5%
First stage of tertiary education	11.6%	14.6%
Second stage of tertiary education	1.9%	2.5%
N twins (proportion)	318 (18.0%)	1452 (82.0%)

Before we incorporate the genetic perspective, we first examine whether living in a single-mother family is associated with a higher likelihood of substance use for adolescents. As shown in Figure 3, there are both major and minor differences between twins living in single-mother and in two-parent families. For example, 27.5% of adolescents in single-mother families and 14.2% in two-parent families are current smokers; with the difference being significant. However, while 31.9% of adolescents in single-mother families reported regularly consuming alcohol excessively, the percentage is only slightly lower for adolescents in two-parent families, at 29.2%. The percentage of adolescents who reported that they have ever used illegal drugs is considerably higher in

single-mother families (30.5%) than in two-parent families (20.9%). In addition to this descriptive approach, we derived odds ratios from logit regression models (presented in the appendix). In line with the findings shown in Figure 3, significantly higher odds of smoking and drug use are found for adolescents in single-mother families than for adolescents in two-parent families, while no significant differences in excessive alcohol consumption are observed.

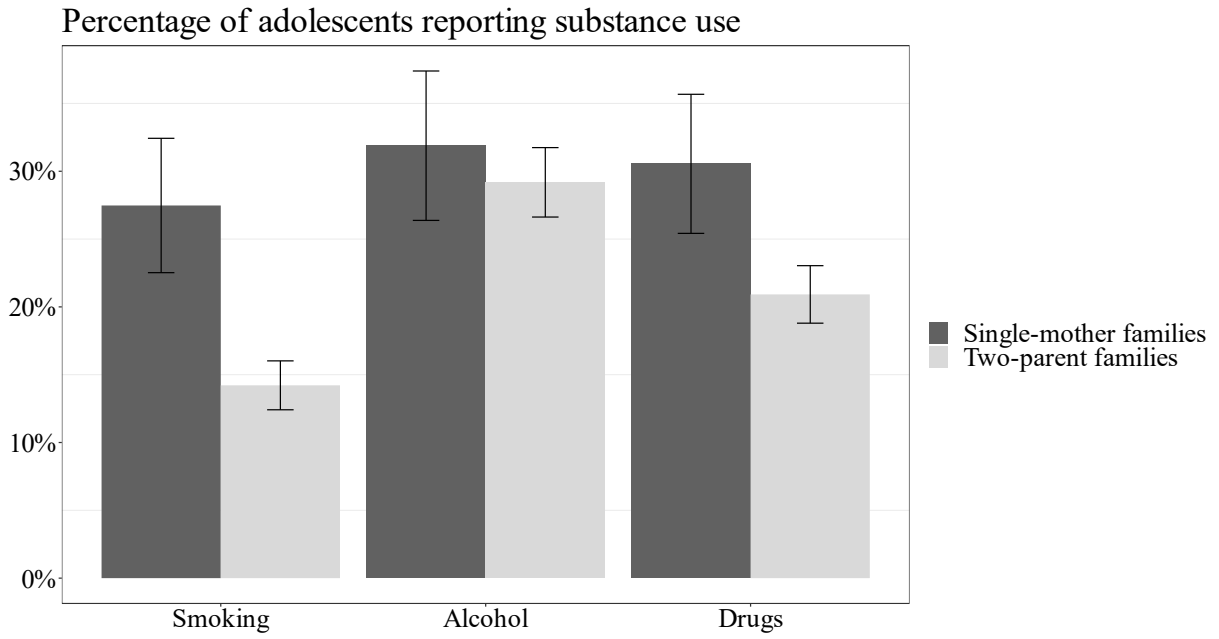

Figure 3: Descriptive proportions of adolescents reporting substance use

Table 3 presents the results of the base ACE variance decomposition of the three forms of substance use. The table contains both the standardized estimates (A, C, E) and the unstandardized estimates (a^2, c^2, e^2) and their confidence intervals (CI). Due to their better interpretability, we focus on the standardized estimates in the following.

As shown in Table S1 in the appendix, for smoking, the AE model without the shared environment component shows the best model fit. The standardized values in Table 3 indicate that across all families in our sample, 67% of the total variance in adolescents' smoking behavior relates to genetic variation, while 33% relates to the non-shared environment. For excessive alcohol consumption, the model assuming no shared environment component again has the best fit to the data (Table S2 Appendix): 48.7% of the total variance in excessive alcohol consumption relates to genetic variation, while 51.3% relates to non-shared environmental influences. For drug use, the model with all three variance components (ACE) has the best fit to the data (Table S3 Appendix). Here, the standardized estimates in Table 3 show that 38.7% of the total variance in drug use relates

to genetic variation, 22.1% relates to shared environmental influences, and 39.1% relates to non-shared environmental influences.

Taken together, the results indicate that smoking has the highest heritability, while alcohol consumption has the largest contribution of the non-shared environment component. Drug use is the only outcome for which a shared environment component is observed.

Table 2: Estimates and confidence intervals from ACE variance decomposition models of smoking, excessive alcohol consumption, and drug use

	Smoking		Alcohol consumption		Drug use	
Standardized variance component	Esti-mate	CI	Esti-mate	CI	Esti-mate	CI
Additive genetic (A)	0.670	[0.621;0.718]	0.487	[0.416;0.558]	0.387	[0.211;0.564]
Shared environment (C)					0.221	[0.073;0.370]
Non-shared environ-ment (E)	0.330	[0.282;0.379]	0.513	[0.442;0.584]	0.391	[0.331;0.452]
Unstandardized vari-ance component						
a^2	0.576	[0.505;0.647]	0.565	[0.461;0.669]	0.393	[0.211;0.575]
c^2					0.224	[0.071;0.378]
e^2	0.284	[0.246;0.322]	0.595	[0.515;0.675]	0.397	[0.340;0.454]
Total variance ($a^2+c^2+e^2$)	0.860		1.160		1.014	
N twins	1732		1378		1682	
N twin pairs	866		689		841	

Figures 4 to 6 summarize the results of the univariate Purcell models for the outcomes smoking, excessive alcohol consumption, and drug use. To facilitate interpretation of the results, both the unstandardized (a^2, c^2, e^2) and standardized variance components (A, C, E) are presented. The unstandardized components (a^2, c^2, e^2) are of particular interest to us because they show whether family living arrangements moderate genetic and environmental influences. If only the standardized components (A, C, E) were considered, such moderation effects would not necessarily be apparent because, first, the total variance may differ between the groups considered; and, second, the standardized components may vary as a function of each other (Mönkediek 2022; Purcell 2002). The point estimates of the path coefficients and the estimates for the unstandardized and the standardized variance components are additionally presented in the Tables S7 and S8 (Appendix).

For smoking, the "no C and no moderation on C" model has the best fit to the data (see Table S4 Appendix). The variance components shown in Figure 4 thus represent the estimated components of the models with estimates C and β_c set to zero. As shown by the unstandardized variance components, genetic influences on smoking are significantly higher among adolescents in single-mother families than among adolescents in two-parent families, while the differences in unshared environmental influences are not significant. This is not obvious when looking at the standardized variance components in the right graph of Figure 4, as the total variance differs between groups. Thus, the contributions of the genetic component and the non-shared environmental components do not differ between single-mother families and two-parent families. However, the overall variance in smoking behavior is significantly larger in single-mother families (see Table 8 Appendix).

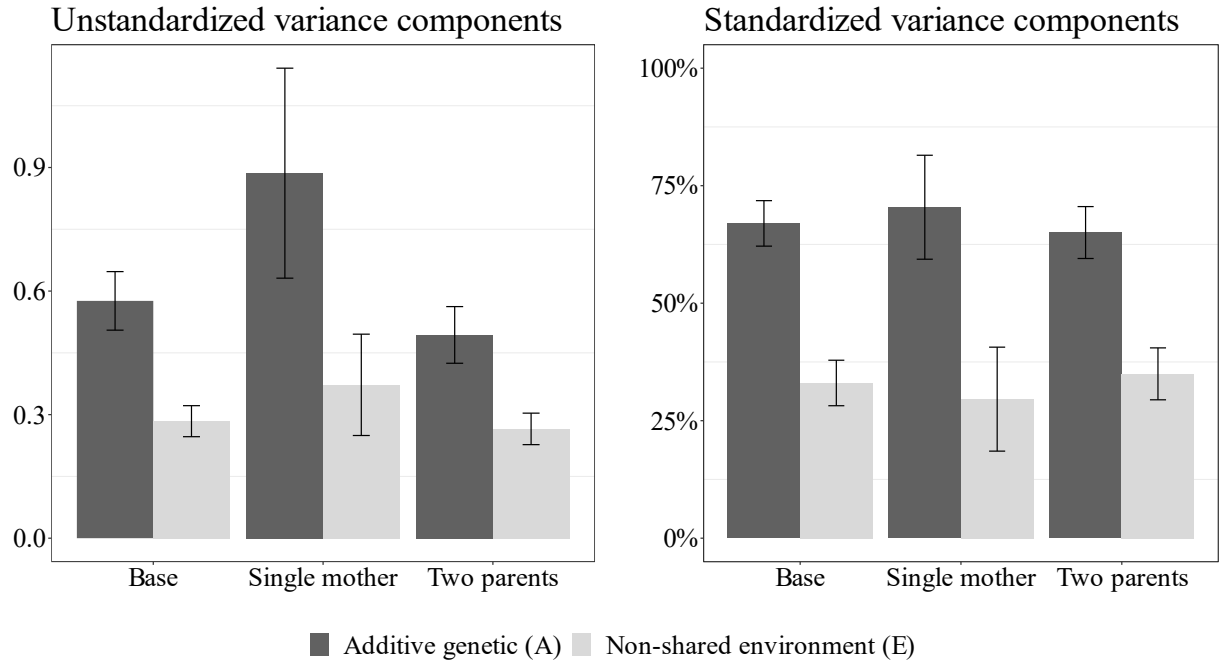

Figure 4: Unstandardized and standardized variance components for the baseline model and the univariate Purcell moderator models for smoking

As shown in Table S5 (Appendix), for excessive alcohol consumption, the "no C and no moderation" model has the best fit to the data. Here, the C estimate as well as the three moderation effects (β_a, β_c and β_e) are set to zero. Thus, as shown in Figure 5, the variance components do not differ between single-mother and two-parent families.

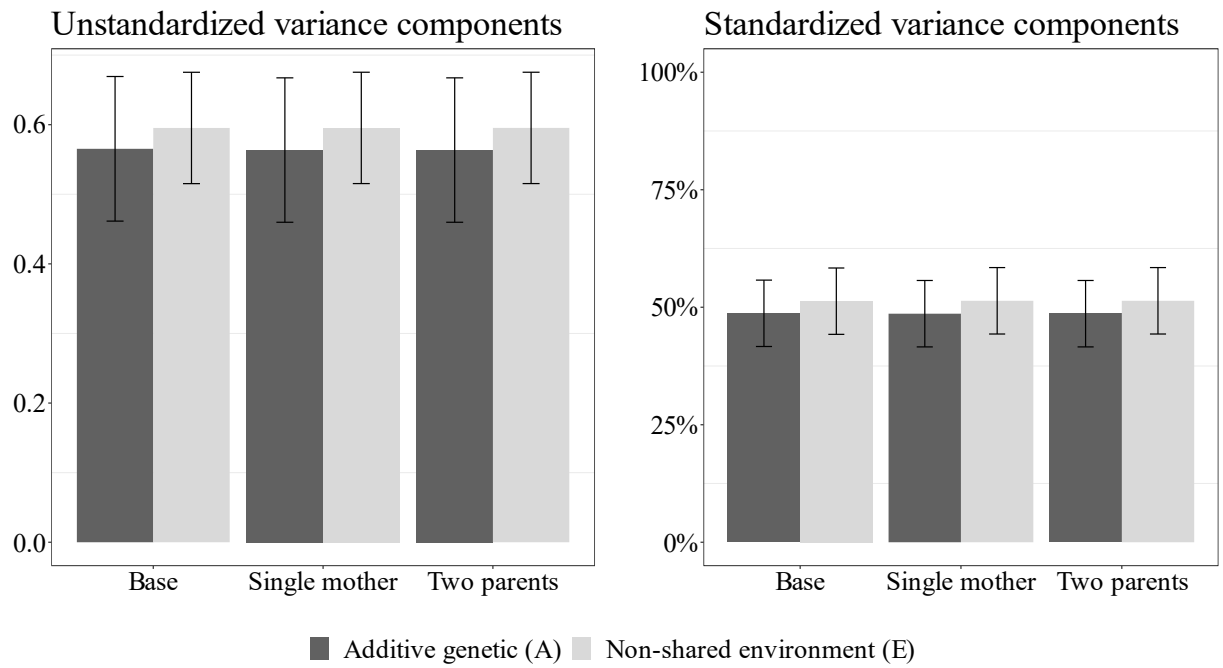

Figure 5: Unstandardized and standardized variance components for the baseline model and the univariate Purcell moderator models for excessive alcohol consumption

As shown in Table S6 (Appendix), the "no moderation on A and C" model has the best fit to the data when drug use is considered. Thus, in line with our base ACE model (Table 3), we find that all the variance components contribute to drug use. Since the results suggest that family living arrangements moderate only the contribution of the non-shared environment, β_a and β_c are set to zero. Figure 6 visualizes the results for adolescent drug use. As Figure 6 indicates, the contribution of the nonshared environment is significantly higher in single-mother families than in two-parent families. Although the family living arrangements do not alter genetic and shared environmental influences on drug use, the moderation of the non-shared environment component affects the proportions of all standardized variance components. These indirect effects result in a lower heritability and a lower relevance of the shared environmental component in single-mother families than in two-parent families.

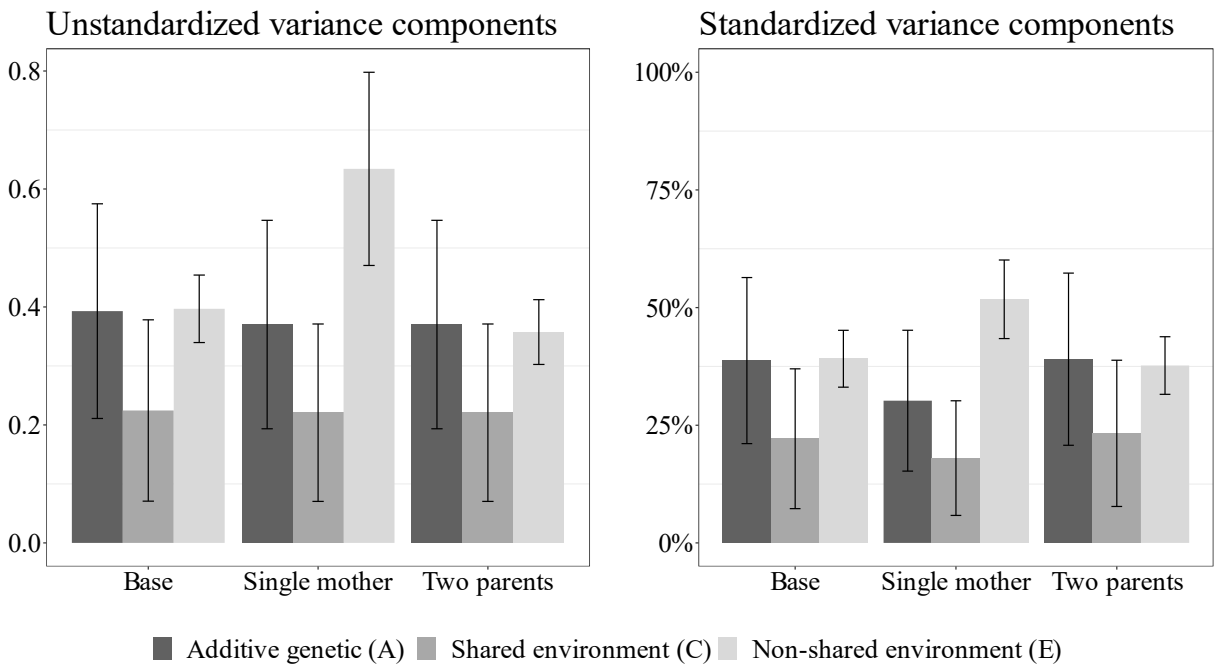

Figure 6: Unstandardized and standardized variance components for the baseline model and the univariate Purcell moderator models for drug use

The results of our robustness checks (presented in the appendix) do not substantially differ from the main analyses. Thus, they indicate that our results are not distorted either by assortative mating or by explaining away substantial parts of the mechanisms when controlling for parental SES. In all models with adjusted DZ correlation, as well as in those without controls for parental SES, a significantly larger genetically explained variance component was found for smoking, and a significantly larger variance component was explained by individual influences for drug use among adolescents in single-mother families.

5 Discussion

In this study, we sought to elucidate the role of parental separation in the unfolding of genetic risk for health risk behavior in adolescents. Based on previous findings from research on parental separation and health risk behavior, and the considerable variation in heritability estimates across different forms of substance use, we expected that parental separation might have induced the unraveling of genetic sensitivities. While social science research has primarily attributed the increased risk of substance use observed among adolescents in separated families to coping mechanisms, our research question focused on explanatory mechanisms related to increased responsiveness to genetic sensitivities due to parental separation. Based on German twin data, we applied

variance decomposition moderator models to investigate the genetic and environmental influences on smoking, excessive alcohol consumption, and drug use among adolescents in single-mother and two-parent families.

Our analyses generated three main findings. First, the results revealed a significantly higher genetic variance component in smoking among adolescents in single-mother families than among adolescents in two-parent families. This is consistent with our expectations of GxE: i.e., that experiencing parental separation increases heritability by triggering genetic sensitivities resulting from stressful experiences, a lack of parental control of child behaviors, and reduced opportunities to compensate for negative behaviors due to limited resources. Despite these different possible paths, all of these mechanisms led to the same outcome of adolescents who experienced parental separation being more likely to express genetic risks for smoking. To the best of our knowledge, our study is the first to show evidence of increased genetic influences on smoking among adolescents in single-mother families. However, our findings add to multiple studies on GxE on smoking that have demonstrated that smoking heritability is moderated by state restrictions (Boardman 2009), parental monitoring (Dick et al. 2007), peer effects (Boardman et al. 2008), and religiousness (Timberlake et al. 2006).

Second, while we found no genetic influences on drug use, but observed a significantly larger contribution of individual experiences to drug use among adolescents in single-mother families than among their peers in two-biological-parent families. This suggests that the higher prevalence of drug use among adolescents in single-mother families may be explained less by increased susceptibility to genetic risk than by individual coping responses to stress (Gustavsen et al. 2016) or peer networks (Kirby 2002).

Third, the results for excessive alcohol consumption show no differences in the variance components between single-mother and two-parent families. Accordingly, in this case, our expectation of larger genetic influences due to parental separation was not confirmed. Moreover, no systematic differences between single-mother and two-parent families in the relevance of individual or environmental influences shared by twins were detected.

Our findings of systematic differences between single-mother and two-parent families in the contributions of the variance components only for the outcomes of smoking and drug use were supported by the descriptive statistics and the additional binary regressions analysis (presented in the appendix), which showed a significantly higher prevalence of smoking and drug use, but not

of excessive alcohol consumption, in single-mother families. The results of the quantitative genetic models were also robust after adjusting the assumed DZ correlation for maternal assortative mating. Furthermore, our robustness checks showed no substantial differences in the models depending on whether SES was or was not controlled for. This leads us to conclude that socioeconomic changes did not have a substantial influence on the moderation of heritability by parental separation, or on systematic differences in the influences of individual experiences and experiences shared by twins.

The differences in the results for drug use and smoking might be explained by different thresholds of access to the substances. Since the threshold for access to cigarettes is rather low in Germany (tobacco products can be legally purchased from age 18 onward), while access to illicit drugs is more strictly regulated, it is possible that for drugs, genetic vulnerability is not as decisive as individual experiences, especially with regard to peer groups and opportunity structures. Similarly, the results for excessive alcohol consumption – which showed no evidence of increased excessive consumption in single-mother families or of moderation of any of the variance components by family living arrangements – can be explained by the prevalence and social acceptance of alcohol in Germany, and by the low age threshold of 16 for buying specific alcohol products, such as beer and wine. Taken together, while these results show that single-mother families resulting from parental separation were more likely to provide conditions that triggered genetic risk for smoking, they also highlight the relevance of individual experiences and opportunity structures for drug use. Therefore, while family living arrangements alone do not increase adolescents' risk of taking drugs, in combination with opportunity structures that provide access to drugs, single-mother families might be less efficient in offsetting health risk behaviors.

The results of our study highlight promising avenues for improving our understanding of the interplay of parental separation and genetic predispositions for health risk behavior. While recent research has shown that parental separation can negatively impact the realization of genetic potential in children (see Baier and Van Winkle 2021), in our study, we showed for the first time that it can also promote the unfolding of genetic predispositions for health risk behavior. Our results point to questions for future research that we could not address using our analytical approach, but that could provide further important insights into the interplay of family influences, genetic sensitivities, and health. In particular, the question of what the specific mechanisms are, which could not be conclusively answered from our results, would be an interesting starting point. While we can

assume, based on our robustness checks, that socioeconomic resources shaped by parental separation are not the main drivers of increased heritability, whether genetic influences are more likely to be triggered by stress associated with separation, or whether the absence of compensation or control plays a more important role, remains unclear.

Beyond the results presented here, three limitations of our research warrant further investigation. First, we focused on single-mother families resulting from parental separation in which only the mother was living with the children at the time of the interview. We did not consider other pathways into single motherhood, such as widowhood, or other family arrangements, such as stepfamilies, because only a few such families were present in the data. Second, the data we used on single-mother families did not provide information on precisely when the father moved out of the household. The sample restrictions we applied ensured that parental separation occurred between the birth of the twins and the interview, but the exact time point remains unknown. Therefore, we do not know how long certain environmental conditions that may have affected adolescents' health risk behavior prevailed. Third, as smoking, drug use, and alcohol consumption were operationalized differently in TwinLife, it is difficult to compare them. For example, in the case of drug use, the data only provide information on whether drugs had ever been used, but not on whether the respondent was still using them regularly at the time of the survey. Thus, in the drug use variable, respondents who had not used drugs for a long time could also be coded as users, whereas in the smoking and drinking variables, only respondents who were still regularly smoking or drinking excessively at the time of the survey were coded as users. Further research based on more comparable measures of substance use is therefore needed.

In conclusion, our work contributes to both an extensive literature on family-related effects on substance use and a growing body of work that investigates how environment in general and family in particular shapes the intensity of genetic influences. Our study has shown for the first time that parental separation moderates the extent to which health risk behavior is influenced by genetic predispositions, and thus offers considerable opportunities for future research to examine both the mechanisms behind this phenomenon, and to focus more closely on the link between parental separation and the expression of genetic risks in general. The broader implications of our findings contribute to the growing understanding that parental separation not only affects adolescents' behavior, but also shapes the extent to which genetic sensitivities for particularly risky behavior, such as substance use, unfold. Our findings inform policy strategies aimed at supporting

single mothers in protecting their children. In particular, explanations regarding the lack of compensation and missing social control point to the need for family policies that support vulnerable family arrangements, especially by counteracting insufficient social resources. Additionally, the stress-related triggering of increased genetic risks in the context of parental separation should be addressed by measures that support both adolescents and single mothers.

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7 Appendix

7.1 Main analyses

7.1.1 Binary regression model

We additionally applied binary logit models to investigate the prevalence of substance use among adolescents in our sample. All models include parental SES and sex of the twins as control variables. Due to difficulties associated with the interpretation of results, we transform the derived log odds to odds ratios and interpret the exponential of the logit coefficients accordingly (for more details see Long and Freese 2014:229).

Table A1 shows the odds ratios indicating the probability that the individual twins smoke, use drugs, or consume alcohol excessively. As indicated by the results, adolescents living in families with a single mother have higher odds of smoking (2.261 times larger), alcohol consumption (1.273 times larger) and drug use (1.972 times larger) compared to adolescents living in families with both biological parents. However, while results for smoking and drug use are significant, the result for alcohol consumption is not.

Table A1: Odds ratios of substance use

	Smoking		Alcohol consumption		Drug use	
	Odds ratios	CI	Odds ratios	CI	Odds ratios	CI
Living in a single mother household (Ref.: Two-parent household)	2.261***	[1.675;3.039]	1.273	[0.945;1.707]	1.972***	[1.473;2.633]
Female (Ref.: Male)	0.600***	[0.463;0.776]	0.498***	[0.396;0.625]	0.383***	[0.302;0.484]
Medium SES (Ref.: Low SES)	0.944	[0.695;1.283]	1.641***	[1.227;2.203]	1.370*	[1.021;1.841]
High SES (Ref.: Low SES)	0.788	[0.567;1.903]	1.513**	[1.124;2.046]	1.669***	[1.240;2.254]
N (individuals)	1747		1483		1718	

7.1.2 Quantitative genetic model

Tables A2-A4 show the model fit comparisons of the different ACE decomposition models with control for the sex of the twins as well as parental SES. The best fitting models are marked with an asterisk (*) for each outcome variable.

Table A2: Model parameters for the base ACE decomposition model considering smoking as outcome

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
ACE	4				4409.339	-
ADE	4	-0.145	0		4409.194	ACE
AE*	3	0	1	1.000	4407.339	ACE
CE	3	283.054	1	<0.001	4690.392	ACE
E	2	283.054	2	<0.001	4688.392	ACE

Table A3: Model parameters for the base ACE decomposition model considering excessive alcohol consumption as outcome

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
ACE	4				4325.722	-
ADE	4	1.920	0		4327.642	ACE
AE*	3	1.920	1	0.166	4325.642	ACE
CE	3	7.571	1	0.006	4331.292	ACE
E	2	120.066	2	<0.001	4441.788	ACE

Table A4: Model parameters for the base ACE decomposition model considering drug use as outcome

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
ACE*	4				4643.956	-
ADE	4	7.616	0		4651.572	ACE
AE	3	7.616	1	0.006	4649.572	ACE
CE	3	17.605	1	<0.001	4659.561	ACE
E	2	246.814	2	<0.001	4886.770	ACE

Tables A5-A7 show the model fit comparisons of the different univariate Purcell moderator models with control for the sex of the twins as well as parental SES. The best fitting models are marked with an asterisk (*) for each outcome variable

Table A5: Model parameters for the univariate Purcell moderator model considering smoking as outcome

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
GxE	8				4364.586	-
No mod. on A	7	5.692	1	0.017	4368.279	GxE
No mod. on C	7	0.139	1	0.709	4362.725	GxE
No mod. on E	7	3.330	1	0.068	4365.916	GxE
No mod. on AE	6	19.116	2	<0.001	4379.702	GxE
No mod. on AC	6	10.145	2	0.006	4370.731	GxE
No mod. on CE	6	3.734	2	0.155	4364.320	GxE
No moderation	5	31.867	3	<0.001	4390.453	GxE
No A no mod. on A	6	55.815	2	<0.001	4416.401	GxE
No C no mod. on C*	6	0.139	2	0.933	4360.725	GxE
No C no CE mod.	5	3.737	3	0.291	4362.323	GxE
No C no mod.	4	31.867	4	<0.001	4388.453	GxE

Table A6: Model parameters for the univariate Purcell moderator model considering excessive alcohol consumption as outcome

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
GxE	8				4328.817	-
No mod. on A	7	1.377	1	0.241	4328.194	GxE
No mod. on C	7	2.595	1	0.107	4329.412	GxE
No mod. on E	7	0.011	1	0.916	4326.828	GxE
No mod. on AE	6	1.864	2	0.394	4326.681	GxE
No mod. on AC	6	3.232	2	0.199	4328.049	GxE
No mod. on CE	6	2.897	2	0.235	4327.715	GxE
No moderation	5	3.245	3	0.355	4326.062	GxE
No A no mod. on A	6	8.840	2	0.012	4333.657	GxE
No C no mod. on C	6	4.342	2	0.114	4329.159	GxE
No C no CE mod.	5	4.533	3	0.209	4327.350	GxE
No C no mod.*	4	5.045	4	0.283	4325.862	GxE

Table A7: Model parameters for the univariate Purcell moderator model considering drug use as outcome

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
GxE	8				4619.041	-
No mod. on A	7	0.736	1	0.391	4617.777	GxE
No mod. on C	7	0.048	1	0.827	4617.089	GxE
No mod. on E	7	4.588	1	0.032	4621.630	GxE
No mod. on AE	6	12.895	2	0.002	4627.936	GxE
No mod. on AC*	6	2.073	2	0.355	4617.115	GxE
No mod. on CE	6	5.711	2	0.058	4620.752	GxE
No moderation	5	19.484	3	<0.001	4632.526	GxE
No A no mod. on A	6	17.933	2	<0.001	4632.974	GxE
No C no mod. on C	6	7.133	2	0.028	4622.174	GxE
No C no CE mod.	5	13.293	3	0.004	4626.335	GxE
No C no mod.	4	25.889	4	<0.001	4636.930	GxE

Table A8 shows point estimates with standard errors and 95% confidence intervals of univariate Purcell moderator models, and Table A9 shows the standardized and unstandardized variance components stratified by family living arrangements (single mother families vs. two parent families) with 95% confidence intervals.

Table A8: Point estimates, standard errors and confidence intervals of the univariate Purcell moderator models

	Smoking			Excessive alcohol consumption			Drug use		
Best-fitting model	No C no moderation on C			No moderation on A and C			No C no moderation		
Unstandardized estimates for the path coefficient	Estimate	SE	CIs	Estimate	SE	CIs	Estimate	SE	CIs
a	0.94	0.07	[0.803;1.077]	0.75	0.04	[0.672;0.828]	0.61	0.07	[0.473;0.747]
c							0.47	0.08	[0.313;0.627]
e	-0.61	0.05	[-0.708;-0.512]	0.77	0.03	[0.711;0.829]	-0.80	0.05	[-0.898;-0.702]
β_a	-1.64	0.07	[-1.777;-1.503]						
β_c									
β_e	1.13	0.05	[1.032;1.228]				0.20	0.05	[0.102;0.298]
Mean	0.08	0.08	[-0.077;0.237]	-0.04	0.07	[-0.177;0.097]	0.08	0.07	[-0.057;0.217]
Lin. Mean	-0.32	0.08	[-0.477;-0.163]	-0.11	0.08	[-0.267;0.047]	-0.28	0.08	[-0.437;-0.123]
N twins	1732			1378			1682		

Table A9: Unstandardized and standardized variance components, including confidence intervals, for the different univariate Purcell mod-erator models

	Smoking				Alcohol consumption				Drug use			
	Single mother		Two parents		Single mother		Two parents		Single mother		Two parents	
Standardized variance components	Estimate	CIs	Estimate	CIs	Estimate	CIs	Estimate	CIs	Estimate	CIs	Estimate	CIs
A	0.704	[0.594; 0.815]	0.65	[0.595; 0.706]	0.486	[0.416; 0.557]	0.486	[0.416; 0.557]	0.302	[0.153; 0.452]	0.390	[0.207; 0.573]
C									0.180	[0.058; 0.302]	0.233	[0.077; 0.388]
E	0.296	[0.185; 0.406]	0.35	[0.294; 0.405]	0.514	[0.443; 0.584]	0.514	[0.443; 0.584]	0.518	[0.434; 0.601]	0.377	[0.316; 0.438]
Unstandardized variance components												
a^2	0.886	[0.631; 1.141]	0.494	[0.425; 0.562]	0.564	[0.460; 0.667]	0.564	[0.460; 0.667]	0.370	[0.193; 0.547]	0.370	[0.193; 0.547]
c^2									0.221	[0.070; 0.371]	0.221	[0.070; 0.371]
e^2	0.372	[0.249; 0.495]	0.265	[0.227; 0.304]	0.595	[0.515; 0.675]	0.595	[0.515; 0.675]	0.634	[0.470; 0.798]	0.357	[0.303; 0.412]
Total variance ($a^2+c^2+e^2$)	1.258		0.759		1.159		1.159		1.225		0.948	
N twins	304		1428		252		1126		302		1380	

7.2 Robustness checks

7.2.1 No control for SES

Tables A10-A18 contain the results of the ACE decomposition models (A10-A13) and the univariate Purcell moderator models (A14-A18) without controlling for parental SES.

Table A10: Model parameters for the base ACE decomposition model considering smoking as outcome without controlling for SES

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
ACE	4				4427.785	-
ADE	4	-0.096	0		4427.688	ACE
AE*	3	0	1	1.000	4425.785	ACE
CE	3	60.168	1	< 0.001	4485.952	ACE
E	2	287.509	2	< 0.001	4711.294	ACE

Table A11: Model parameters for the base ACE decomposition model considering excessive alcohol consumption as outcome without control-ling for SES

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
ACE	4				4350.047	-
ADE	4	2.468	0		4352.515	ACE
AE*	3	2.468	1	0.116	4350.515	ACE
CE	3	6.679	1	0.010	4354.725	ACE
E	2	121.392	2	< 0.001	4467.439	ACE

Table A12: Model parameters for the base ACE decomposition model considering drug use as outcome without controlling for SES

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
ACE*	4				4664.575	-
ADE	4	7.749	0		4672.324	ACE
AE	3	7.749	1	0.005	4670.324	ACE
CE	3	18.000	1	< 0.001	4680.575	ACE
E	2	250.511	2	< 0.001	4911.086	ACE

Table A13: Estimates and confidence intervals from ACE variance decomposition models of smoking, excessive alcohol consumption, and drug use without controlling for SES

	Smoking		Alcohol consumption		Drug use	
Standardized variance component	Estimate	CI	Estimate	CI	Estimate	CI
Additive genetic (A)	0.672	[0.624;0.720]	0.487	[0.416;0.557]	0.389	[0.214;0.565]
Shared environment (C)					0.222	[0.074;0.370]
Non-shared environment (E)	0.328	[0.280;0.376]	0.513	[0.443;0.584]	0.389	[0.329;0.448]
Unstandardized variance component						
a^2	0.578	[0.508;0.649]	0.568	[0.464;0.671]	0.395	[0.214;0.576]
c^2					0.226	[0.073;0.379]
e^2	0.282	[0.245;0.320]	0.599	[0.519;0.679]	0.394	[0.338;0.451]
Total variance ($a^2+c^2+e^2$)	0.860		1.167		1.015	
N twins	1732		1378		1682	
N twin pairs	866		689		841	

Table A14: Model parameters for the univariate Purcell moderator model considering smoking as outcome without controlling for SES

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
GxE	8				4378.768	-
No mod. on A	7	5.717	1	0.017	4382.485	GxE
No mod. on C	7	0.160	1	0.689	4376.928	GxE
No mod. on E	7	3.581	1	0.058	4380.348	GxE
No mod. on AE	6	19.747	2	< 0.001	4394.515	GxE
No mod. on AC	6	10.553	2	0.005	4385.321	GxE
No mod. on CE	6	4.027	2	0.133	4378.795	GxE
No moderation	5	33.420	3	< 0.001	4406.187	GxE
No A no mod. on A	6	55.862	2	< 0.001	4430.630	GxE
No C no mod. on C*	6	0.160	2	0.923	4374.928	GxE
No C no CE mod.	5	4.040	3	0.257	4376.808	GxE
No C no mod.	4	33.420	4	< 0.001	4404.187	GxE

Table A15: Model parameters for the univariate Purcell moderator model considering excessive alcohol consumption as outcome without controlling for SES

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
GxE	8				4352.892	-
No mod. on A	7	1.683	1	0.195	4352.576	GxE
No mod. on C	7	3.344	1	0.067	4354.236	GxE
No mod. on E	7	0.001	1	0.976	4350.893	GxE
No mod. on AE	6	2.489	2	0.288	4351.382	GxE
No mod. on AC	6	4.293	2	0.117	4353.186	GxE
No mod. on CE	6	3.877	2	0.144	4352.769	GxE
No moderation	5	4.337	3	0.227	4351.230	GxE
No A no mod. on A	6	8.122	2	0.017	4357.014	GxE
No C no mod. on C	6	5.677	2	0.059	4354.569	GxE
No C no CE mod.	5	6.020	3	0.111	4352.912	GxE
No C no mod.*	4	6.732	4	0.151	4351.625	GxE

Table A16: Model parameters for the univariate Purcell moderator model considering drug use as without controlling for SES

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
GxE	8				4645.494	-
No mod. on A	7	0.553	1	0.457	4644.047	GxE
No mod. on C	7	0.050	1	0.823	4643.544	GxE
No mod. on E	7	4.409	1	0.036	4647.902	GxE
No mod. on AE	6	11.655	2	0.003	4653.149	GxE
No mod. on AC*	6	1.407	2	0.495	4642.901	GxE
No mod. on CE	6	5.549	2	0.062	4647.043	GxE
No moderation	5	16.605	3	< 0.001	4656.098	GxE
No A no mod. on A	6	18.146	2	< 0.001	4659.640	GxE
No C no mod. on C	6	7.535	2	0.023	4649.029	GxE
No C no CE mod.	5	13.432	3	0.004	4652.926	GxE
No C no mod.	4	23.394	4	< 0.001	4660.888	GxE

Table A17: Point estimates, standard errors and confidence intervals of the univariate Purcell moderator models without controlling for SES

	Smoking			Excessive alcohol consumption			Drug use		
Best-fitting model	No C no moderation on C			No C no moderation			No moderation on A and C		
Unstandardized estimates for the path coefficient	Estimate	SE	CIs	Estimate	SE	CIs	Estimate	SE	CIs
a	-0.94	0.07	[-1.080;-0.810]	0.75	0.04	[0.682;0.821]	-0.61	0.07	[-0.746;0.746]
c							-0.47	0.08	[-0.611;0.611]
e	-0.61	0.05	[-0.724;0.724]	0.77	0.03	[0.724;0.828]	0.78	0.05	[0.686;0.888]
β_a	1.64	0.07	[-0.387;1.790]						
β_c									
β_e	0.10	0.05	[0.000;1.243]				-1.38	0.06	[-1.503;0.291]
Mean	0.09	0.08	[-0.058;0.247]	-0.07	0.07	[-0.215;0.078]	0.05	0.07	[-0.091;0.199]
Lin. Mean	-0.34	0.08	[-0.500;-0.177]	-0.08	0.08	[-0.240;0.084]	-0.25	0.08	[-0.405;-0.089]
N twins	1732			1378			1682		

Table A18: Unstandardized and standardized variance components, including confidence intervals, for the univariate Purcell moderator mod-els without controlling for SES

	Smoking				Alcohol				Drugs			
	Single mother		Two parents		Single mother		Two parents		Single mother		Two parents	
Standardized variance components	Estimate	CIs	Estimate	CIs	Estimate	CIs	Estimate	CIs	Estimate	CIs	Estimate	CIs
A	0.704	[0.595; 0.814]	0.652	[0.597; 0.707]	0.486	[0.416; 0.557]	0.486	[0.416; 0.557]	0.310	[0.159; 0.461]	0.392	[0.211; 0.573]
C									0.186	[0.063; 0.309]	0.235	[0.081; 0.389]
E	0.296	[0.186; 0.405]	0.348	[0.293; 0.403]	0.514	[0.443; 0.584]	0.514	[0.443; 0.584]	0.504	[0.421; 0.587]	0.373	[0.313; 0.434]
Unstandardized variance components												
a^2	0.889	[0.636; 1.142]	0.492	[0.424; 0.560]	0.567	[0.463; 0.671]	0.567	[0.463; 0.671]	0.375	[0.119; 0.551]	0.375	[0.119; 0.551]
c^2									0.225	[0.075; 0.375]	0.225	[0.075; 0.375]
e^2	0.373	[0.251; 0.496]	0.263	[0.225; 0.301]	0.599	[0.519; 0.679]	0.599	[0.519; 0.679]	0.610	[0.453; 0.767]	0.357	[0.302; 0.412]
Total variance ($a^2+c^2+e^2$)	1.262		0.755		1.166		1.166		1.240		0.987	
N twins	304		1428		252		1126		302		1380	

7.2.2

7.2.3 Adjusted genetic DZ correlations for assortative mating

Tables A19-A27 contain the results of the ACE decomposition models (A19-A22) and the univariate Purcell moderator models (A23-A27) with an adjusted genetic correlation for DZ twins to account for parental assortative mating. The genetic similarity between DZ twins is 0.5 on average given the assumption of random mating. If information on the biological parents' health risk behavior (smoking, alcohol consumption, drug use) is available, it is possible to estimate an average genetic correlation for DZ twins adjusted for assortative mating. The correction term is $0.5+0.5*h_0^2*r_p$, with h_0^2 denoting the share of genetic influences estimated (standardized genetic variance component) without correction for assortative mating and r_p denoting the correlation of parents with respect to the trait under study.

Our sample contains families where no information about the biological father are present. Therefore, we cannot calculate the correlation of parents with respect to smoking, alcohol consumption and drug use for all families. Nevertheless, we can test the robustness of our results for

those families in which information on parental substance use is available for both biological parents. This approach requires the assumption that the correlation between parents does not differ systematically between two parent and single mother families.

Among the two parent families, r_p for smoking is 0.250 and the standardized additive genetic variance component is 0.670 according to the base ACE decomposition presented in Table 2 in the main paper. For excessive alcohol consumption, r_p is 0.156 and the standardized additive genetic variance component is 0.487 according to the base ACE decomposition presented in Table 2 in the main paper. For drug use, r_p is 0.303 and the standardized additive genetic variance component is 0.387 according to the base ACE decomposition presented in Table 2 in the main paper. This results in an adjusted genetic DZ similarity of 0.584 for smoking, of 0.538 for alcohol consumption, and of 0.559 for drug use. The results presented in Tables A19-A27 are based on this adjusted genetic correlation among DZ twins.

Table A19: Model parameters for the base ACE decomposition model considering smoking as outcome with genetic DZ correlations adjusted for assortative mating

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
ACE	4				4412.334	-
ADE	4	-3.140	0		4409.194	ACE
AE*	3	0	1	1.000	4410.334	ACE
CE	3	56.947	1	< 0.001	4467.281	ACE
E	2	280.059	2	< 0.001	4688.392	ACE

Table A20: Model parameters for the base ACE decomposition model considering excessive alcohol consumption as outcome with genetic DZ correlations adjusted for assortative mating

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
ACE	4				4325.722	-
ADE	4	1.061	0		4326.782	ACE
AE*	3	1.061	1	0.303	4324.782	ACE
CE	3	7.571	1	0.006	4331.292	ACE
E	2	120.066	2	< 0.001	4441.788	ACE

Table A21: Model parameters for the base ACE decomposition model considering drug use as outcome with genetic DZ correlations adjusted for assortative mating

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
ACE	4				4643.956	-
ADE	4	3.524	0		4647.480	ACE
AE*	3	3.524	1	0.060	4645.480	ACE
CE	3	17.605	1	< 0.001	4659.561	ACE
E	2	246.814	2	< 0.001	4886.770	ACE

Table A22: Estimates and confidence intervals from ACE variance decomposition models of smoking, excessive alcohol consumption, and drug use with genetic DZ correlations adjusted for assortative mating

	Smoking		Alcohol consumption		Drug use	
Standardized variance component	Estimate	CI	Estimate	CI	Estimate	CI
Additive genetic (A)	0.659	[0.608;0.710]	0.484	[0.413;0.554]	0.626	[0.573;0.679]
Shared environment (C)						
Non-shared environment (E)	0.341	[0.290;0.392]	0.516	[0.446;0.587]	0.374	[0.321;0.427]
Unstandardized variance component						
a^2	0.571	[0.497;0.645]	0.562	[0.458;0.666]	0.631	[0.647;0.715]
c^2						
e^2	0.295	[0.256;0.334]	0.600	[0.520;0.679]	0.377	[0.328;0.426]
Total variance ($a^2+c^2+e^2$)	0.866		1.162		1.008	
N twins	1732		1378		1682	
N twin pairs	866		689		841	

Table A23: Model parameters for the univariate Purcell moderator model considering smoking as outcome with genetic DZ correlations adjusted for assortative mating

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
GxE	8				4367.508	-
No mod. on A	7	5.334	1	0.021	4370.841	GxE
No mod. on C	7	0	1	1.000	4365.508	GxE
No mod. on E	7	4.062	1	0.044	4369.570	GxE
No mod. on AE	6	20.974	2	< 0.001	4384.482	GxE
No mod. on AC	6	8.781	2	0.012	4372.289	GxE
No mod. on CE	6	4.062	2	0.131	4367.570	GxE
No moderation	5	32.353	3	< 0.001	4393.860	GxE
No A no mod. on A	6	52.893	2	< 0.001	4416.401	GxE
No C no mod. on C	6	0	2	1.000	4363.508	GxE
No C no CE mod.*	5	4.062	3	0.255	4365.570	GxE
No C no mod.	4	32.353	4	< 0.001	4391.860	GxE

Table A24: Model parameters for the univariate Purcell moderator model considering excessive alcohol consumption as outcome with genetic DZ correlations adjusted for assortative mating

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
GxE	8				4328.817	-
No mod. on A	7	1.377	1	0.241	4328.194	GxE
No mod. on C	7	2.505	1	0.113	4329.323	GxE
No mod. on E	7	0.011	1	0.916	4326.828	GxE
No mod. on AE	6	1.864	2	0.394	4326.681	GxE
No mod. on AC	6	3.232	2	0.199	4328.049	GxE
No mod. on CE	6	2.836	2	0.242	4327.653	GxE
No moderation	5	3.245	3	0.355	4326.062	GxE
No A no mod. on A	6	8.840	2	0.012	4333.657	GxE
No C no mod. on C	6	3.454	2	0.178	4328.271	GxE
No C no CE mod.	5	3.691	3	0.297	4326.508	GxE
No C no mod.*	4	4.219	4	0.377	4325.036	GxE

Table A25: Model parameters for the univariate Purcell moderator model considering drug use as outcome with genetic DZ correlations adjusted for assortative mating

Model	EP	Δ -2LL	Δ df	p	AIC	Compare with model
GxE	8				4619.041	-
No mod. on A	7	0.736	1	0.391	4617.777	GxE
No mod. on C	7	0.098	1	0.754	4617.140	GxE
No mod. on E	7	4.777	1	0.029	4621.819	GxE
No mod. on AE	6	12.895	2	0.002	4627.936	GxE
No mod. on AC*	6	2.073	2	0.355	4617.115	GxE
No mod. on CE	6	6.217	2	0.045	4621.259	GxE
No moderation	5	19.484	3	< 0.001	4632.526	GxE
No A no mod. on A	6	17.933	2	< 0.001	4632.974	GxE
No C no mod. on C	6	3.341	2	0.188	4618.383	GxE
No C no CE mod.	5	9.797	3	0.020	4622.838	GxE
No C no mod.	4	22.250	4	< 0.001	4633.292	GxE

Table A26: Point estimates, standard errors and confidence intervals of the univariate Purcell moderator models with genetic DZ correlations adjusted for assortative mating

	Smoking			Excessive alcohol consumption			Drug use		
Best-fitting model	No C no moderation on C and E			No C no moderation			No moderation on A and C		
Unstandardized estimates for the path coefficient	Estimate	SE	CIs	Estimate	SE	CIs	Estimate	SE	CIs
a	-1.00	0.06	[-1.127;-0.881]	0.75	0.04	[0.677;0.817]	-0.65	0.08	[-0.790;0.790]
c							-0.41	0.11	[-0.583;0.583]
e	0.54	0.02	[-0.578;0.577]	0.77	0.03	[0.725;0.828]	-0.80	0.05	[-0.905;-0.699]
β_a	0.31	0.07	[0.186;0.444]						
β_c									
β_e							0.20	0.05	[0.100;0.308]
Mean	0.08	0.08	[-0.077;0.242]	-0.04	0.08	[-0.187;0.108]	0.08	0.07	[-0.064;0.230]
Lin. Mean	-0.32	0.09	[-0.491;-0.153]	-0.11	0.08	[-0.273;0.054]	-0.28	0.08	[-0.440;-0.122]
N twins	1732			1378			1682		

Table A27: Unstandardized and standardized variance components, including confidence intervals, for the univariate Purcell moderator mod-els with genetic DZ correlations adjusted for assortative mating

	Smoking				Alcohol				Drugs			
	Single mother		Two parents		Single mother		Two parents		Single mother		Two parents	
Standardized vari-ance components	Esti-mate	CIs	Esti-mate	CIs	Esti-mate	CIs	Esti-mate	CIs	Esti-mate	CIs	Esti-mate	CIs
A	0.773	[0.720; 0.825]	0.618	[0.562; 0.675]	0.483	[0.412; 0.554]	0.483	[0.412; 0.554]	0.342	[0.173; 0.512]	0.442	[0.235; 0.650]
C									0.140	[0.001; 0.279]	0.181	[0.002; 0.359]
E	0.227	[0.175; 0.280]	0.382	[0.325; 0.438]	0.517	[0.446; 0.588]	0.517	[0.446; 0.588]	0.518	[0.434; 0.601]	0.377	[0.316; 0.438]
Unstandardized vari-ance components												
a^2	0.993	[0.749; 1.236]	0.474	[0.404; 0.543]	0.560	[0.456; 0.664]	0.560	[0.456; 0.664]	0.420	[0.219; 0.620]	0.420	[0.219; 0.620]
c^2									0.171	[0.000; 0.343]	0.171	[0.000; 0.343]
e^2	0.292	[0.254; 0.330]	0.292	[0.254; 0.330]	0.600	[0.520; 0.680]	0.600	[0.520; 0.680]	0.634	[0.470; 0.798]	0.357	[0.303; 0.412]
Total variance ($a^2+c^2+e^2$)	1.285		0.766		1.160		1.160		1.225		0.948	
N twins	304		1428		252		1126		302		1380	