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Published in:
Social Science & Medicine

DOI:
10.1016/j.socscimed.2023.116070

Publication date:
2023

Document Version
Publisher's PDF, also known as Version of record

Social Science & Medicine 331 (2023) 116070

Contents lists available at ScienceDirect

Social Science & Medicine

journal homepage: www.elsevier.com/locate/socscimed

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

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ARTICLE INFO

Handling Editor: Social Epidemiology Editorial Office

Keywords:
Twin study
Gene-environment interaction
Single mothers
Substance use
Smoking
Drug use
Alcohol consumption

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 1762 twins, this study examines whether family living arrangements moderate the influence of genetic predispositions on health risk behavior. Derived from variance decomposition moderator models, three key findings emerge. Firstly, genetic contributions to drug use are significantly higher in single-mother families, indicating an amplified heritability potentially resulting from triggered genetic sensitivities or challenges in preventing genetic risks from unfolding. Secondly, unique environmental factors have a greater impact on drug use in single-mother families. Lastly, no heritability differences are found in smoking and excessive alcohol consumption between family types. These findings provide novel evidence of increased importance of genetic influences on drug use in single-mother families, shedding light on gene-environment interactions, and informing policy interventions that support vulnerable family arrangements.

1. Introduction

Health risk behavior related to substance use often initiates in adolescence (Umberson et al., 2010), and has far-reaching health implications for later life, such as higher chronic disease risks (Kaur et al., 2022) and lower life expectancies (Martikainen et al., 2014). Studies reporting declines in the prevalence of substance use among adolescents in Germany (Zeiher et al., 2018), 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 important to identify individuals who are at risk of using substances such as tobacco, alcohol, and other drugs. Previous studies have shown that individuals' risk of engaging in health risk behavior varies according to both social circumstances and genetic sensitivities. It has been shown that certain social environments associated with higher stress levels increase risks for substance use (Short and Mollborn, 2015). Other studies found substantial genetic influences on smoking, alcohol consumption, and drug use, meaning that traits closely associated with these forms of substance use are influenced by variations in a large number of genetic variants (Kreek et al., 2005; Li, 2008).

In addition to research focusing on either social or genetic influences, several studies analyzed the interplay of social and genetic influences to explain differences in substance use (e.g., Kendler et al., 2014; Pampel et al., 2015). These studies emphasized that environments can increase or decrease the influence of genetic predispositions on trait development, called *gene-environment interaction* (GxE) (see Horwitz and Neiderhiser, 2011). Thus, these studies argued that genetic risks for substance use are more likely to be realized under certain environmental conditions.

Examining social contexts that may influence the expression of genetic risk factors could help to identify social groups particularly vulnerable to health risk behavior. Although previous research has examined 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 et al., 2003; Kirby, 2002).

In this paper, we address this gap by examining whether adolescents in single-mother families who have experienced a parental separation

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https://doi.org/10.1016/j.socscimed.2023.116070
Received 14 February 2023; Received in revised form 23 May 2023; Accepted 1 July 2023
Available online 3 July 2023

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). Adolescents in single-mother families show higher rates of smoking (e.g., Griesbach et al., 2003), heavy drinking (e.g., Brown and Rinelli, 2010), and drug use (e.g., Cavanagh, 2008) than adolescents in two-biological-parent families.

There are three main theoretical approaches that address the impact of family living arrangements. First, the sociological *stress theory* (Pearlin, 1989) focuses on stress related to the conflict and instability associated with parental separation, which affects children by increasing their likelihood of engaging in risky behavior (Kirby, 2002) as a coping mechanism. These coping strategies might be learned from close environments in childhood (Umberson et al., 2010). 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 et al., 2008; Rattay et al., 2018). 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 drug use among adolescents from single-parent families can be explained by higher stress exposure; while Kirby (2002) found that parental separation increases adolescents' likelihood to 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 mothers having fewer social resources after 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 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 weakens ties and relationship quality between family members, increasing 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 preventing health risk behavior, especially substance use, in children through values, norms, and rules. Single mothers may face challenges in exerting control over their adolescent children due to high emotional distress levels, or because they need to work more because of 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).

The concepts of *socialization*, *social control,* and *social stress* are strongly associated. Adolescents experiencing particularly stressful situations or environments might be less responsive to parental control, and 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

Research has shown that genetic influences contribute to smoking behaviors, alcohol consumption, and drug use. However, estimates of genetic variation (termed heritability) vary substantially across studies (e.g., Verhulst et al., 2015). For instance, depending on the study and the indicator under consideration, heritability of alcohol misuse ranges from 16% to 20% (Walters, 2002); heritability of smoking ranges from 11% to 84% (Hall et al., 2002; Li et al., 2003); and heritability of drug use ranges from 6% to 76% for problem cannabis use (Verweij et al., 2010).

The substantial variation in the heritability of different types of substance use can partly be explained by the environment shaping the degree of realization of genetic risk (Boardman et al., 2011, p. 1518). Three potential GxE mechanisms 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 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 the presence of a positive and enriched environmental setting that positively affects individual functioning by preventing genetic risks from unfolding (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 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, these mechanisms 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 control, have been located in the individual's immediate surroundings (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 moderation of peer deviance on genetic contributions to drug use among adolescents.

2.3. The current study

Previous research has shown that the heritability of substance use can be shaped by the environment, particularly among adolescents. Although some studies looked at the relevance of social origin (e.g., Barr et al., 2018; Timberlake et al., 2007), 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 research reporting strong associations between family living arrangements and substance use, 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 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 genetic influences on health risk behavior to be 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, 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.

We consider the German context, where alcohol consumption among young adults is 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 14–17) binge drinking regularly (Zeiher et al., 2018). A comparable pattern of binge drinking (slightly over 10%) has been observed in the United States, where the percentage of adolescents and young adults who regularly binge drink is 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 11–17) 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 similar 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). In terms of drug use, 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 from 2014 to 2016, and includes 4097 twin pairs and their families residing in Germany. This total sample is composed of four age cohorts of approximately 500 monozygotic (MZ) and 500 same-sex dizygotic (DZ) twin pairs (Hahn et al., 2016). Face-to-face interviews were conducted with both 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 16–18 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 1762 twins nested in 881 twin pairs. Of these, 724 twin pairs were living with both biological parents, and 157 twin pairs were living in a single-mother household. Observations of twin pairs by family living arrangement, sex and zygosity are shown in Table 1.

3.2. Variables

3.2.1. Outcomes and moderator

We examine smoking, excessive alcohol consumption, and drug use as indicators of health risk behavior. These are derived from questions asked of all participants aged 16 or older at the time of the interview. The question on 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 to a dummy variable, with categories 1–4 representing current smokers (coded as 1) and categories 5–6 representing current non-smokers (coded as 0). This measure is widely used in research on adolescent smoking behavior (see Khlat et al., 2020; Kristjansson et al., 2009). The question on 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 recoded this information into a dummy variable with categories 1–4 representing regular excessive alcohol consumers (coded as 1) and categories 5–6 representing non-regular excessive consumers (coded as 0). This dummy-coding of excessive alcohol consumption based on monthly frequency is commonly used in previous research (Khlat et al., 2020; Rattay et al., 2018). The question on drug use was: "*Have you ever taken drugs (e.g., marijuana, hash, ecstasy, cocaine, etc.)? We're not referring to cigarettes or alcohol.*" This indicates whether the respondent had ever used illegal drugs (coded 1) or not (coded 0). Measures of lifetime drug use have been used widely in the literature on adolescent drug use (see Hayatbakhsh et al., 2006; Mandara et al., 2011). Nevertheless, to examine

Table 1
Twin pairs by family living arrangement, sex, and zygosity.

	Single-mother families		Two-parent families	
	MZ	DZ	MZ	DZ
Female	41	51	188	218
Male	33	32	145	173

the measurement sensitivity of our results, we conducted robustness checks focusing on drug use in the last 12 months, which confirmed the credibility of our results.

Our moderator variable is the family living arrangement of the twins. 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. To analyze theoretical reflections of potential effects of parental separation, we restrict the single-mother households to those that had been exposed to parental separation. In doing so, we closely followed the sample construction approach of Baier and Van Winkle (2021), who investigated heritability differences among family living arrangements in educational achievement based on the TwinLife data. In the initial sample, 66.2% were families with both biological parents in the household and 19.5% single mother families. We excluded stepfamilies (9%), single-father families (3.7%), and households in which family composition could not be assigned due to missing information (1.5%). Single-father families were excluded from the study sample due to the substantial differences in socio-demographic characteristics observed between single-mother and single-father families in previous research (Collings et al., 2014). Additionally, the limited sample size of single-father families prevented the conduct of stratified analyses. Among single mothers, we excluded those who reported being widowed (10.8%) and those without current marital status information (less than 1%). In addition, we excluded all families in which the twins have never shared the household with their biological father, which was the case for 9.4% of single-mother families.

3.2.2. Controls

In all models, we control for parental socioeconomic status (SES). Prior research has shown that there are socioeconomic inequalities in both substance use (e.g., Pampel et al., 2010) and family living arrangements (e.g., Jalovaara, 2003). Furthermore, investigating the heritability of substance use adjusted for SES strengthens the claim that potential differences in heritability are due to family living arrangements, and not to socioeconomic differences. We derive a family-SES score from net equivalent household income, parental education (International Classification of Education), and parental occupation (International Socio-Economic Index of Occupational Status) using confirmatory factor analysis. The derived SES scores are divided into terciles to indicate low, medium, and high SES families.

SES may explain increasing heritability of health risk behavior in single-mother families. Households with higher SES 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 without controlling for parental SES.

In addition, we control for sex, 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).

According to the classical twin design, 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, capturing the averaged effects of the genome on the outcome (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). In the univariate ACE model comparisons (see Appendix), we also tested ADE models that included both additive and non-additive genetic effects, but ADE models showed worse model fit for all outcomes. Accordingly, since we focus on the proportion of genetic variation that is due to additive genetic effects, we focus on *narrow-sense heritability*. However, we will refer to it in the following as *heritability*.

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 assumes random mating of the parents. To check how this assumption might bias our results, we conducted robustness checks with adjusted DZ-twin correlations following the approach of Loehlin et al. (2009), which is described in more detail 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 et al., 1999). Accordingly, MZ and DZ twin pairs are assumed to share homogeneous environmental influences to the same extent. The validity of this *Equal Environment Assumption* has been supported 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.

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 Fig. 1. Here, the influences of the latent variables A, C, and E 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.

To decompose the outcomes and obtain moderating effects by family living arrangements, we estimate the effects of the ACE and the GxE models based on Liability Threshold Models (Neale, 2005). These models estimate thresholds for the liability distribution. It is assumed that for binary variables, there is an underlying normal distribution of liability with a mean of 0 and variance of 1. Using moderator models, we restrict the variance to 1 for a specific expression of the moderator (Medland et al., 2009). Treating binary variables as continuous underestimates genetic and shared environmental variance components and overestimates non-shared environmental components (Verhulst and Neale, 2021). Our models are estimated in R using the twinflex (Ruks, 2022) and the umx package (Bates et al., 2019), both wrappers for OpenMx (Neale et al., 2016). While Fig. 1 shows the full GxE model, sub-models excluding certain paths may fit the data better. We compare the model fit values for all combinations of included or excluded parameters. Model comparisons for smoking, excessive alcohol consumption, and drug use are presented in the Appendix. We selected the most parsimonious model with the lowest Akaike Information Criterion (AIC) where the likelihood-ratio test indicated that it did not fit the data significantly worse. To account for the potential issue of inflated p-values in ACE models when comparing nested twin models using likelihood ratio tests based on chi-square distributions, we follow the recommendation by Dominicus et al. (2006) to halve the p-values obtained. However, as noted by Bates et al. (2017) this does not apply to GxE models. We handled missing values on the outcome variables using Full Information Maximum Likelihood estimators, requiring only deletion of observations with missing values on the family living arrangement.

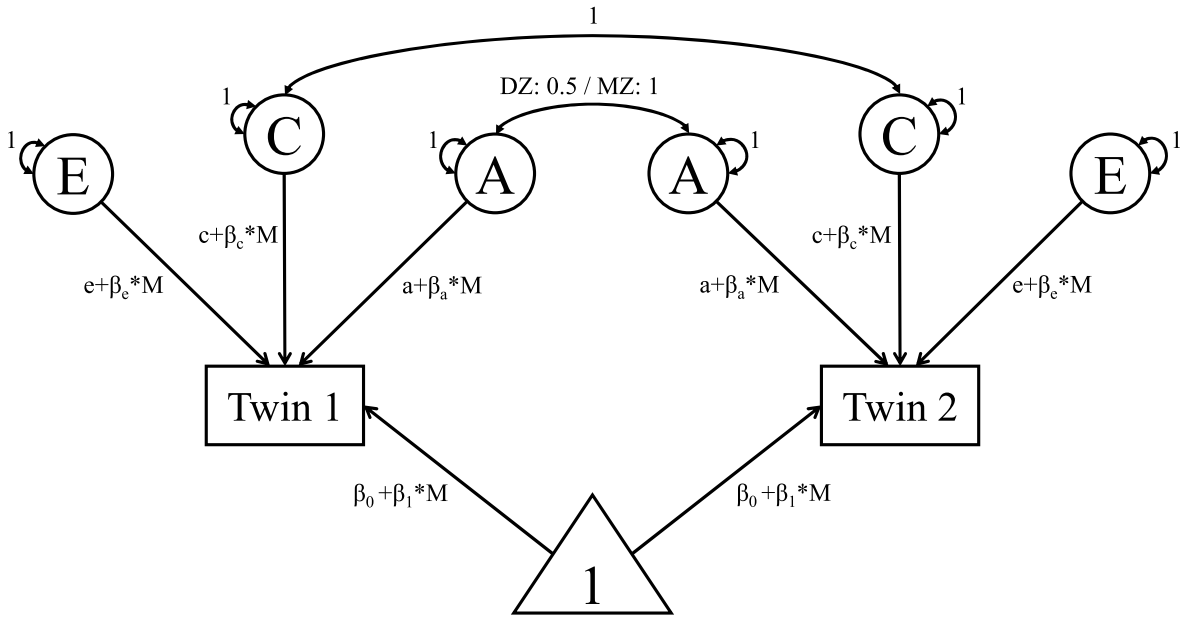

Fig. 1. Univariate Purcell moderation model (Bates et al., 2019).

4. Results

4.1. Descriptive results

Our sample includes 314 twins in single-mother families and 1448 twins in two-parent families (see Table 2). The majority of twins are female and dizygotic. Twins in both family types are 17 years old on average; while single mothers are slightly younger than partnered mothers. The mean net equivalent household income in the single-mother families is less than two-thirds that of the two-parent families and single mothers are less educated than partnered mothers, with a much smaller percentage having secondary or tertiary education. The proportion of current smokers is significantly higher among adolescents in single-mother families (27.8%) than in two-parent families (14.3%). However, while 32.4% of adolescents in single-mother families reported regularly consuming alcohol excessively, the percentage is only slightly lower in two-parent families (29.1%). The percentage of adolescents who reported that they have ever used illegal drugs is considerably higher in single-mother families (30.9%) than in two-parent families (21.0%). These differences are supported by regression analyses (see Appendix), showing significantly higher odds of smoking and drug use for adolescents in single-mother families compared to two-parent families, while differences in excessive alcohol consumption are insignificant. Finally, Table 2 shows that the proportion of mothers who currently smoke or have ever used illegal drugs is significantly higher among single mothers than among those living with the biological father of their children.

4.2. Quantitative genetic models

In this section, we present the effects and standardized variance components from ACE models. Subsequently, we examine GxE models to assess the moderation of A, C, and E by the family living arrangement.

Table 3 provides an overview of the additive genetic, shared environmental, and non-shared environmental effects on smoking, excessive alcohol consumption, and drug use among adolescents. Additionally, we present the standardized variance components, indicating the percentage of total variance attributed to genetic, shared, or non-shared environmental contributions to health risk behaviors. The standardized variance components add up to 100%, and their sizes vary as a function of each other (Mönkediek, 2022; Purcell, 2002). For all outcomes, we present the model with the best fit to the data; model comparisons are presented in the appendix. Sex and SES are included as covariates in all models.

Regarding smoking, the model without shared environmental effects fits the data best. The additive genetic effects are approximately three times stronger than the non-shared environmental effects. Considering the standardized variance components, smoking shows a high heritability, explaining about 90% of the total variance among adolescents.

Likewise, the model without shared environmental effects yielded the best fit for alcohol consumption. The additive genetic effect is the strongest, and standardized variance components indicate a heritability of 73%, while non-shared environmental effects explain about 27% of the total variance.

Table 2
Socio-demographic descriptives.

	Single-mother families	Two-parent families	Difference
Proportion female twins	58.6%	56.1%	
Proportion monozygotic twins	47.1%	46.0%	
Mean age twins	17.0	17.0	*
Mean age mother	47.6	48.0	
Mean net equivalent household income	1047.1	1633.2	***
Proportion tertiary education mother	33.5%	46.1%	***
Proportion currently smoking (adolescents)	27.%	14.3%	***
Proportion excessive alcohol	32.4%	29.1%	
Proportion ever taken drugs	30.9%	21.0%	***
Proportion mother currently smoking	38.1%	22.9%	***
Proportion mother excessive alcohol	10.9%	8.8%	
Proportion mother ever taken drugs	20.8%	11.8%	***
N twins (proportion)	314 (17.8%)	1448 (82.2%)	

Table 3
Estimates from univariate ACE models.

Path coefficients	Smoking		Alcohol consumption		Drug use	
	Estimate	CI	Estimate	CI	Estimate	CI
a	0.95	[0.92; 0.98]	0.85	[0.79; 0.91]	0.59	[0.34; 0.83]
c					0.69	[0.52; 0.87]
e	0.32	[0.22; 0.41]	0.52	[0.43; 0.62]	0.42	[0.31; 0.52]
Standardized variance components						
A	0.90	[0.84; 0.96]	0.73	[0.63; 0.83]	0.34	[0.06; 0.63]
C					0.48	[0.24; 0.72]
E	0.10	[0.04; 0.16]	0.27	[0.17; 0.37]	0.17	[0.09; 0.26]
N (twins)	1762		1762		1762	
N (twin pairs)	881		881		881	

For drug use, significant effects of genetics, shared- and non-shared environment are observed. Notably, the effect of the shared environment is the strongest. According to the standardized variance components, the heritability of drug use among adolescents is 34%. Thus, genetic effects explain approximately one-third of the total variance in drug use among adolescents.

We employed a GxE model to examine whether the additive genetic effects on the three outcomes were influenced by the family living arrangement (living with a single mother or with both biological parents). Table 4 presents the parameters a (additive genetic effects), c (shared environmental effects), and e (non-shared environmental effects), along with the interaction coefficients of these effects with the family living arrangement (β_a, β_c and β_e). We present the models with the best fit for each outcome, while model comparisons are provided in the appendix.

The best-fitting model for smoking indicated no shared environmental effects and no moderation of effects by the family living arrangement. Consequently, there are no systematic differences in the heritability of smoking between adolescents in single-mother and two-parent families.

Similarly, for alcohol consumption, the best-fitting model shows no shared environmental effects and no moderation of effects. Therefore, similar to smoking, there is no moderation by family living arrangement causing systematic differences in the genetic effects on excessive alcohol consumption.

The best-fitting model for drug use includes genetic effects, shared- and non-shared environmental effects, and moderations on both genetic and non-shared environmental effects by the family living arrangement. The interaction effects revealed that genetic effects on drug use among adolescents in two-parent families are significantly smaller than those in single-mother families, indicating higher heritability among adolescents in single-mother families. The effects of the non-shared environment are also significantly stronger in single-mother families.

Table 4
Estimates from univariate GxE moderator models (standard errors in parentheses).

	Smoking		Alcohol consumption		Drug use	
Best fitting model	No C and no moderation		No C and no moderation		No moderation on C	
a	0.95***	(0.02)	0.85***	(0.03)	0.90***	(0.06)
c					0.12	(0.17)
e	0.32***	(0.05)	0.52***	(0.05)	0.42***	(0.10)
β_a					−0.80***	(0.18)
β_c						
β_e					−0.50***	(0.15)
N (twins)	1762		1762		1762	
N (twin pairs)	881		881		881	

4.3. Robustness checks

To assess the robustness of our findings, we conducted additional analyses, with detailed results provided in the appendix.

We performed analyses in which we did not control for parental SES and analyses in which we accounted for a higher correlation in DZ twins due to parental assortative mating. Results align closely with the main analyses, indicating that our findings are not distorted either by assortative mating or by explaining away substantial parts of the mechanisms when controlling for parental SES.

To investigate the impact of our operationalization of drug use on the observed moderated genetic effect, we estimated an alternative model using drug use in the past 12 months as a binary-coded outcome. The results confirmed significant moderation on the genetic effect, further supporting the robustness of our findings based on lifetime drug use as the outcome.

Furthermore, descriptive information (Table 2) revealed significant differences in the substance use prevalence between single mothers and mothers in two-parent families. To test whether our results were driven by maternal substance use, we reran our analysis controlling for mother's substance use behavior. While the results remain consistent with the main models for smoking and alcohol consumption, no moderation effect is observed in the model focusing on drug use.

Finally, given the examination of three different substance use variables in separate models, we re-examined our results based on reduced p-value thresholds according to the Bonferroni correction. We divided the significance level of 0.05 by 3 (due to the three outcomes), testing the model comparisons against a significance threshold of $p < 0.017$. The best fitting models resulting from these comparisons remained consistent with those from the main analyses.

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. Drawing from existing literature on parental separation and health risk behavior, as well as the varying heritability estimates across different forms of substance use, we expected that parental separation could unravel genetic sensitivities. While social sciences have primarily attributed the increased risk of substance use among adolescents in separated families to coping mechanisms, our research focused on explanatory mechanisms related to increased responsiveness to genetic sensitivities due to parental separation. Based on German twin data, we applied quantitative genetic 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, our analyses revealed a significantly higher genetic contribution to drug use among adolescents in single-mother families compared to those in two-parent families. This aligns with our expectations of GxE, indicating that experiencing parental separation enhances heritability by triggering genetic sensitivities resulting from stressful experiences, a lack of parental control over child behaviors, and limited resources for compensating negative behaviors. Notably, the absence of this moderation when controlling for maternal substance use behavior suggests that social control plays the most important role in the interaction: it can be assumed that mothers with prior drug experiences are less successful in exerting social control on their children in this regard, even in two-parent families. On one hand, this could be attributed to mothers with their own experiences being less restrictive with their children. On the other hand, this could be attributed to children who are aware of their

mothers' experiences or current habits perceiving their constrictions as less consequential. This makes systematic differences in genetic influences disappear when maternal drug use is held constant. To our knowledge, this study is the first to provide evidence of increased genetic influences on drug use among adolescents in single-mother families. Our findings contribute to drug use related GxE studies that have demonstrated that genetic effects on drug use increase in the presence of negative life events (Covault et al., 2007) and insecure attachments (Olsson et al., 2013).

Second, we observed a significantly larger contribution of unique environmental factors to drug use among adolescents in single-mother families compared to those in two-biological-parent families. This suggests that the higher prevalence of drug use among adolescents in single-mother families might be attributed to individual coping responses to stress (Gustavsen et al., 2016) or experiences of 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, we found no systematic differences between single-mother and two-parent families in the relevance of individual or shared-environmental influences. This result aligns with our finding of no systematic differences in the prevalence of excessive alcohol consumption between single-mother and two-parent families. This can be attributed to the widespread social acceptance of alcohol in Germany and the low age threshold of 16 for purchasing alcoholic beverages, such as beer and wine.

Furthermore, we observed a surprisingly high heritability in smoking without moderations by family living arrangement. This could be due to the declining prevalence of smoking among German adolescents. Although smoking prevalence rose during the COVID-19 pandemic (Koopmann et al., 2021), our data were collected earlier. Consequently, reduced social incentives and increased financial and social sanctions on smoking may suggest that genetic predispositions primarily account for the variation in adolescents' smoking behavior.

Our findings showed consistently stronger genetic influences on drug use in single-mother families, even after adjusting the assumed DZ correlation for parental assortative mating. Furthermore, robustness checks indicated no substantial differences in the models that accounted for SES or did not control for it. Accordingly, socioeconomic changes potentially affected by parental separation did not have a substantial influence on the moderation of heritability by parental separation, or on systematic differences in the influences of unique environmental factors and experiences shared by twins.

Beyond the presented results, 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 excluded other pathways into single motherhood, such as widowhood, or other family arrangements, such as stepfamilies, due to their limited representation in the data. Examining stepfamilies would help determine if the increased substance use risk from triggered genetic sensitivity or the increased genetic sensitivity from missing social control can be compensated or if they cause additional conflict in the family. 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. Furthermore, we have no information on the mother's partnership behavior between these two points. Some adolescences may have experienced one or more maternal re-partnering or reconciling transitions and separations, while others lived constantly with their single mother. Hence, we could consider potential dynamics and duration of certain environmental conditions that may have affected adolescents' health risk behavior. However, our theoretical approaches suggest that we may underestimate the effect of parental separation on the heritability of health risk behavior, as more fragile trajectories may be associated with increased stress (Carr and Springer, 2010). Third, it is important to note that our focus is only on health risk behavior per se, and we cannot make any statements about the associated problems that we have briefly outlined in the introduction. Future research should investigate the extent to which the increased genetic variance proportion in drug use observed in single-mother families, resulting from correlations between genes and gene-environment correlations, is associated with genetic sensitivities to health problems associated with drug use.

Overall, 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. In addition, future research could examine how heritability of substance use influenced by parental separation evolves over time into young adulthood, as alcohol and drug use, in particular, may increase after moving out of the parental home. Also, sex-stratified analyses of the twins, which we were unable to conduct due to limited sample size, may provide further insight into the interplay of parental separation and genetic contributions to health risk behavior.

In conclusion, our work contributes to the literature on family-related effects on substance use and the investigation of how the environment, particularly the family, shapes the impact of genetic influences. Our study has shown for the first time that parental separation moderates the genetic contributions to health risk behavior, opening opportunities for future research to explore underlying mechanisms and the broader link between parental separation and genetic risks. 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 social control and compensation 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.

Declaration of competing interest

None.

Data availability

The TwinLife data is available for scientists as Scientific Use File (SUF) at the data catalogue of GESIS. The R-Code can be obtained on request from the authors.

Acknowledgements

We would like to thank Mirko Ruks as well as the two anonymous reviewers for their suggestions and comments. Philipp Dierker was supported by grants to the Max Planck – University of Helsinki Center from the Max Planck Society, Jane and Aatos Erkko Foundation, Faculty of Social Sciences at the University of Helsinki, and Cities of Helsinki, Vantaa and Espoo; and the European Union (ERC Synergy, BIOSFER, 101071773). Views and opinions expressed are, however, those of the author only and do not necessarily reflect those of the European Union or the European Research Council. Neither the European Union nor the granting authority can be held responsible for them. Further support provided from the International Max Planck Research School for Population, Health and Data Science (IMPRS-PHDS) is gratefully acknowledged. Bastian Mönkediek acknowledges funding from the German Research Foundation (DFG) as part of the longterm project TwinLife (grant number 220286500), awarded to Martin Diewald, Christian Kandler, Rainer Riemann and Frank M. Spinath.

Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.socscimed.2023.116070.

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