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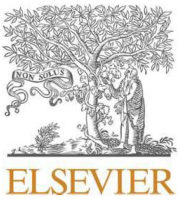

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The intergenerational transmission of risk and trust attitudes: Replicating and extending "Dohmen, Falk, Huffman and Sunde 2012" using genetically informed twin data

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ABSTRACT

This replication revisits an influential contribution on the intergenerational transmission of risk and trust attitudes, which, based on data from the *German Socioeconomic Panel* (GSOEP), reveals a positive correlation between parents' and children's attitudes. The authors of the original study argue that socialization in the family is important in the transmission process. The replication is motivated by mounting evidence indicating that within-family transmission has a considerable genetic component, which calls into question socialization as the main transmission pathway. To consider genetic transmission in addition to social transmission, the replication relies on the German twin family panel *TwinLife*. The findings reveal that, first, most of the variation in children's risk and social trust attitudes is attributable to differences in the non-shared environment, followed by genetic differences, whereas differences in the shared family environment – the main candidate for social transmission – do not matter. Second, correlations between parents' and children's attitudes essentially involve genetic similarity. Third, family conditions do not moderate these relationships. Thus, the findings do not support the socialization assumption.

1. Introduction

The intergenerational transmission of social positions is one of sociology's core themes, with decades of research documenting the relationship between parental characteristics and those of their offspring. While social mobility research with its focus on educational and occupational attainment has dominated the field (e.g., Blau and Duncan 1967; Breen and Jonsson 2005; Pfeffer 2008; Roksa and Potter 2011), other features have received less attention, such as the passing on of attitudes (e.g., Kretschmer 2018; Platt and Polavieja 2016). At the same time, recent empirical advances – oftentimes in neighboring disciplines – are chipping away at the narrative of intergenerational social transmission by highlighting the considerable heritability of dispositional characteristics and behavioral patterns (Kendler and Baker 2007; Plomin et al., 2016; Polderman et al., 2015; Turkheimer 2000). Studies using genetically informed research designs cast doubt on the assumption that social processes within families are the main transmission channel (Branje et al., 2020; Mills and Tropf 2020). Their findings suggest that assessments of social transmission might be inadequate, because some part of the correlation between parents' and their children's attitudes is genetic. Whenever genetic confounding has entered the sociological

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discussion on intergenerational transmission (e.g., Conley et al., 2015; Liu 2018; Mills and Tropf 2020), the evidence speaks for the consideration of social *and* genetic influences. Ignoring that parts of intergenerational correlations are genetic can have unintended consequences: from a purely scientific perspective, ignoring it may generate biased or even incorrect answers as to the reasons and sources of within-family resemblance. From a policy perspective, investing into measure that aim at affecting change in the wrong source of differences may at the very least be inefficient or even completely miss their mark - depending on the extent of genetic confounding.

In this article, we take advantage of data that enables us to integrate genetic transmission methodologically. We build upon a much-cited study by Dohmen, Falk, Huffman and Sunde on "The intergenerational transmission of risk and trust attitudes", published in 2012 in the *Review of Economic Studies*.[1] We duplicate their empirical work with the original data,[2] replicate it with a different data source that allows genetic transmission to be considered in addition to social transmission, and extend it in several respects.

Dohmen et al. (2012) examine attitudinal transmission using data from the *German Socio-Economic Panel (GSOEP)*. First, they show that parents' willingness to take risks and to trust others is positively related to their offspring's manifestations of these attitudes. Furthermore, the resemblance persists after taking into account a broad range of characteristics. Second, the authors provide evidence that the regional level of willingness to take risks and to trust others is positively related to children's attitudes over and above parental transmission. This finding indicates that transmission not only takes place within the family, but is also shaped by the local environment. Third, they illustrate that assortative mating of parents further contributes to the intergenerational transmission of risk and trust attitudes. The authors argue that the overlap in spouses' attitudes enhances their ability to convey these attitudes onto their children. In addition to these key results, Dohmen et al. (2012) elaborate on a range of family conditions that may influence the intergenerational transfer of attitudes. They show, for example, that the transmission of risk attitudes is more successful when parental attitudes are homogenous, in single-mother households, when children have fewer siblings, and when there are fewer fights in the family. According to the authors, these findings point to the importance of socialization in processes of attitude transmission.

Due to data restrictions, Dohmen et al. (2012) were not able to empirically examine the possibility that risk and trust attitudes are partly heritable. With our contribution, we aim at closing this gap by assessing whether and how their results, and the interpretations of these findings, change once we take into account genetic transmission.

TwinLife, a longitudinal twin family study conducted in Germany (Diewald et al., 2020), makes use of the fact that monozygotic (MZ) twins are genetically identical, thus sharing 100 percent of their DNA, whereas dizygotic (DZ) twins share on average 50 percent of their segregating DNA (Boomsma et al., 2002). Differences between a pair of MZ twins can only be due to their environment, whereas differences between a pair of DZ twins can be due to both their environment and to genetic influences (Boomsma et al., 2002; Falconer 1989). Whenever MZ twins resemble each other more than DZ twins, this difference in resemblance between the two types of twin pairs is likely to be genetic. The overall variation in a trait can be partitioned into: a (1) genetic component (or *genetic heritability*); (2) a component attributable to differences in the *shared environment* (such as provided by the family); and (3) a residual component, the *non-shared environment*, that captures all remaining influences that are neither covered by genes or the shared environment (i.e., essentially an error term; Conley et al., 2013: 416). Since *TwinLife* is located in Germany and draws on the questionnaires used in the *GSOEP*, we are able to replicate Dohmen et al.'s (2012) results for the same country, and we can use identical measures for many constructs. Hence, differences between our findings and those reported in the original study are unlikely to be driven by measurement inconsistencies and different societal contexts. It is, however, impossible to rule out the possibility that other differences between the two samples, such as their differential age composition, produce divergent results.

In accordance with advice for conducting replication studies found in the literature (e.g., Carsey 2014; Janz 2016; Janz and Freese 2021; King 1995, 2003), we first duplicate Dohmen et al.'s (2012) original study to determine whether using the same methods on the same data (i.e., the *GSOEP*) produces the published result (Janz and Freese 2021, p. 305). This step is also discussed as "reproducibility" and aims at the verification of the results of the original study (p. 305). Thereafter, we replicate these analyses using *TwinLife* data. In this replication, we follow the original study as closely as possible by considering the authors' decisions to restrict the sample, the measurements used, and their treatment of missing information. Relying on a different data source can be seen as robustness challenge (p. 306). In a third step, we extend these analyses in several respects. We adapt some of the measurements of the original study, include single-person households, and use multiple imputation to deal with missing information. With these adaptations, we seek to increase the scope of the findings (p. 305).

The most important feature of both the replication and the extension study is the use of a genetically informed research design. As will be explained below in more detail, this design is used to partition the variance in children's risk and trust attitudes into three different components, one of them being a genetic component. Aside from this additional focus on variance components, the regression methods implemented in the replication and the extension study do not substantially differ from those used in the original study.

We replicated this study with social science scholars in mind who are interested in intergenerational transmission processes but may not have familiarity with genetic arguments and approaches. Dohmen et al.'s study serves as an excellent starting point to showcase how genetic-informed reasoning can enhance our comprehension of intergenerational transmission. Although the primary findings of our replication and extension study may not be novel to researchers knowledgeable in behavioral genetics, they still offer valuable insights for social scientists new to this field. Our study demonstrates that the common practice of connecting parental traits

[1] Google Scholar reports about 1000 citations (April 2022).

[2] The duplication is based on the exceptionally detailed and well documented replication package as well as additional material and data sets provided by the authors of the original study. We are extremely grateful to the team of authors, especially Uwe Sunde, for their cooperation. We also would like to thank the SOEP team at the DIW for their support.

to those of their offspring and regarding it as social transmission falls short of the potential of this line of research.

2. The transmission of attitudes

2.1. Social transmission

We start by describing Dohmen et al.'s (2012) arguments on social transmission. In line with previous work, they use the term social transmission to capture a range of processes taking place within families and in families' social environments, whereby attitudes and behaviors of parents and other network members are – to some extent – adopted by the next generation. Empirically, transmission is reflected in a correlation between the attitudes and behaviors of parents (and network members) and those of their offspring.

Social transmission covers both direct and indirect processes. At home, children are exposed to parental attitudes and behaviors, learn from them, and to varying degrees, adapt their own attitudes and behaviors to those of their parents (Bandura 1989; Chick et al., 2002). In addition to these indirect processes, parents directly influence their offspring, for example, through explicitly teaching values and behaviors (Grusec et al., 2014). Socialization can also take place outside the household, involving network members such as peers, teachers and role models in the broader social context (Schoon and Eccles 2014). Direct and indirect processes oftentimes are indistinguishable in empirical studies, because available data sources do not include separate measures for them.

Fig. 1 depicts Dohmen et al.'s (2012) theoretical considerations, which are based on the work of Bisin and Verdier (2000). The two arrows (a) indicate transfers of the mother's and the father's attitudes onto their child, covering direct and indirect social transmission. Arrow (b) represents social transmission taking place outside the household via members of the social network. Dohmen et al. (2012) empirically capture this transmission channel by focusing on attitudes that are prevalent in the child's region of residence. The transmission of such contextual attitudes is probably predominantly indirect, in that children observe the behaviors and attitudes of others in their everyday social environments and may adjust their thinking and conduct accordingly.

Arrows (c) and (d) refer to within-family conditions that facilitate the transmission of parental attitudes. The idea is that social transmission is more efficient when parents share similar attitudes and are therefore better able to work in concert to transmit them. The double-headed arrow (c) indicates assortative mating (or homophily), according to which individuals seek out partners with characteristics similar to their own (Blossfeld 2009; Kalmijn 1998). The greater the overlap in parental attitudes, the stronger their ability to signal and transport them consistently.

Lastly, arrow (d) captures family characteristics that moderate social transmission. Dohmen et al. (2012) focus on five attributes that include parental characteristics (i.e., the couple's attitudinal homogeneity), aspects of the family structure (i.e., single-parent households, the number of siblings present, and the child's gender) and the family climate (i.e., quality of parent-child relations). The underlying rationale for focusing on these conditions is twofold: first, consistent and concordant signals sent from both parents provide a clear message to children and should therefore encourage adaptation; second, intergenerational transmission is more efficient when children are exposed to parental attitudes on a regular basis and encounter ample opportunities to observe their parents or to be tutored by them. Accordingly, the more homogeneous a couple's attitudes are, the stronger the cues are that they send to their offspring. Similarly, single-parent households, by definition, consist of a single person who will display homogenous attitudes. Regarding transmission opportunities, in families with more children, parents need to divide their time and attention between more heads, which reduces the average time available for each child. Furthermore, children may be less inclined to seek their parents' attention when conflict is present and parent-child relations are strained, which should reduce the time spent together and, thus, the opportunities for attitude transmission. Finally, Dohmen et al. (2012) expect that intergenerational transmission is more efficient in same-sex parent-child dyads, because of a preference for same-sex interactions, which should lead to more exposure to the attitudes of the same-sex parent.

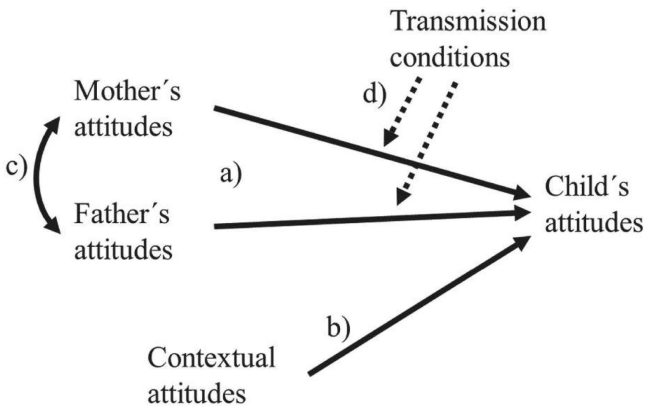

Fig. 1. Channels of social transmission according to Dohmen et al. (2012).

2.2. Integrating behavioral genetics

Like Dohmen et al. (2012), we are interested in assessing the social transmission pathway. For this purpose – and here we differ from the original study – we follow the literature arguing that, for an assessment of social transmission, it is necessary to take genetic transmission into account (Conley et al., 2015; Liu 2018; Mills and Tropf 2020). In accordance with this strand of research, we do not address the processes underlying genetic transmission in terms of specifying the genes or the genetic pathways that give rise to differences in risk and trust attitudes. Instead, we make use of indirect methods applied in behavioral genetics. In these models, genetic information is considered indirectly by making use of the fact that monozygotic (MZ) twins are genetically identical and thus share 100 percent of their DNA, whereas dizygotic (DZ) twins share on average 50 percent of their segregating DNA (Boomsma et al., 2002). Given assumptions detailed below, any difference between MZ and DZ twin pairs is likely to be genetic (Boomsma et al., 2002; Falconer 1989). A very basic metric for estimating genetic heritability is calculated as two times the difference between the correlation of MZ and DZ twins (i.e., Falconer's formula; Falconer 1989). The greater the difference in these correlations between MZ and DZ twins, the greater the heritability of a trait. Modern approaches still rely on this core idea of comparing differences in the degree of resemblance in a trait between MZ and DZ twins, but move beyond these basic metrics. In so-called ACE models, the differences in resemblance are used to decompose the total variance of a trait into an additive genetic component (A), a component of the shared environment (C), and a component capturing the non-shared environment (E; Conley et al., 2013: 416). Applied to our study on the transmission of risk and trust attitudes, A covers genetic differences, C represents the shared family environment – and thus is the likely candidate for social transmission – and E is an error term, which is akin to the standard residual error in regression models and captures measurement error and individual differences to which neither genes, nor the shared family environment are linked (i.e., the non-shared environment; Conley et al., 2013: 416). Note that the shared family environment is certainly not the only "social pathway" for socialization effects to generate within-family resemblance. Differential treatment by parents may also generate (dis-)similarity but would be part of the non-shared environment due to its uniqueness to one child but not another. However, the transmission literature's main focus is explaining differences *across* families (which therefore explicitly refer to the shared family environment C) and to a substantially lesser degree why the extent of resemblance varies *within* families (which stresses the importance of the non-shared environment). In the following, we summarize three of the "big" findings of behavioral genetic research that have been robustly replicated (Plomin et al., 2016: 3) and that have implications for our study on the intergenerational transmission of attitudes.

First, attitudes like other traits and behaviors are heritable to some degree (Kendler and Baker 2007; Plomin et al., 2016; Polderman et al., 2015; Turkheimer 2000). In fact, "all psychological traits show significant and substantial genetic influence" (Plomin et al., 2016: 4). In their meta-analysis based on fifty years of twin-studies, Polderman et al. (2015, Supplementary Table 16) report an average heritability of around 0.3 in social values (i.e., the trait category in their analyses that comes closest to attitudes), which suggests that around 30 percent of the differences between individuals are due to genetic differences. The remaining 70 percent fall into the other two components: the variation attributable to differences in the shared or family environment, and the variation capturing any other non-shared environmental source of individual differences. These findings suggest that the scope for social transmission within families is limited by the other two components, and that disregarding them may lead to inaccurate interpretations of similarities between the attitudes of parents and their offspring.

Second, measures of the shared environment, such as parenting, social support or other instruments used to capture social transmission processes within families, have been shown to possess a genetic component (Kendler and Baker 2007; Plomin et al., 2016). The provision of books at home, for example, is frequently used as an indicator of a stimulating home environment that provides access to a favorable scholarly culture (e.g., Evans et al., 2014). It is thus interpreted as a measure of the shared environment. However, books may be present in the household in the first place, because, due to their own genetic endowment, parents have a preference for reading and are therefore inclined to buy books (van Bergen et al., 2017: 147). Consequently, children's exposure to books at home cannot be considered exclusively as an indicator of a socially shaped environment, but as a feature that carries itself a genetic component.[3] If this genetic component is disregarded, social transmission cannot be not adequately assessed. In addition, measures of the shared environment can also be shaped by how individuals react to persons of different genetic endowments.[4] For instance, parenting styles have been shown to be affected by the genetically influenced behaviors of children (Avinun and Knafo 2014). If a child loves books due to a genetic disposition, parents may be inclined to buy more books to satisfy these preferences. These behaviors contribute to creating an environment that reflects the child's genetic features. Disentangling these feedback-like relationships between the behaviors of the child and the behaviors of the parents is difficult, suggesting that even perfectly replicable social transmission effects can be genetically confounded.

Third, heritability in certain traits and behaviors tends to change across the lifespan (Bergen et al., 2007; Haworth et al., 2010; Plomin et al., 2016). For example, Haworth et al. (2010) report that the genetic component of differences in cognitive ability increases with age, meaning that among older individuals, a larger proportion of the differences in cognitive ability is attributable to differences in genes than among younger individuals. An important explanation for this pattern refers to the guiding and constraining influence of parents and the social environment in childhood: with increasing age, as individuals become less dependent on their parents, parental influence and constraints weaken, and individuals can more easily self-select into environments and behaviors in line with their preferences or predispositions (Bergen et al., 2007: 424). They thus have a greater capacity to select, modify and create their own

[3] The example represents a *passive gene-environment correlation*, according to which parents give their children both the genes relevant to reading and an environment that is favorable for the development of this trait (Plomin, DeFries and Loehlin 1977: 310).

[4] Influences of this kind represent *reactive gene-environment correlations* (Plomin, DeFries and Loehlin 1977).

experiences (Haworth et al., 2010: 1112).[5] For example, a child may enjoy reading, but the family environment may provide few opportunities for reading, and consequently does not allow the child to act upon their preference. With increasing age, an adolescent or young adult can more easily satisfy their reading preferences, because they have more resources at her disposal to buy books, or because they may visit the library or friends who have books. Hence, social transmission is likely to be stronger at younger ages, where individuals have less control over their environment and are more dependent on their parents. For a study of the transmission of attitudes that is interested in detecting family influences, this reasoning is of importance, as it suggests that, for a sample of adults such as that used by Dohmen et al. (2012), it might be harder to identify social transmission processes compared to a sample of children or adolescents.

Given the substantial and robust evidence that has been accumulated throughout the past decades, there is broad consensus about these three "big" findings (in addition to several others; Plomin et al., 2016). Although we refer to them and highlight their relevance for a study of intergenerational attitude transmission, this should not be interpreted as our having a deterministic view of genetics. Instead, with our application, we follow the "interactionist consensus" according to which all traits are the result of genes and environments (Kitcher 2001). Accordingly, we neither pose the question of whether genetic transmission takes place, nor whether the social environment matters: we consider both as given, and as uncontroversial facts. Our motivation is to examine both transmission pathways, and use this knowledge for a better understanding of the correlation between parents' trust and risk attitudes and those of their offspring.

3. Data and methods

3.1. Data

For the duplication of Dohmen et al.'s (2012) original study, we use the code and the *GSOEP* data (v26 from 2009) provided by the authors. The *GSOEP*, an ongoing longitudinal survey launched in 1984, is based on a random sample of private households in Germany (Goebel et al., 2019). The questionnaires cover a range of topics, many of them measured on a yearly basis. They also include additional content measured at larger time intervals. Dohmen et al. (2012) focused mainly on the 2003 and 2004 waves, which include questions on risk and trust attitudes. Each wave covers roughly 22,000 individuals from about 12,000 households. In their calculations, Dohmen et al. (2012) applied listwise deletion of observations with missing information.[6] Depending on the set of variables included in the respective models, the number of observations considered in the different analyzing steps varied accordingly. In their first set of calculations, they included between 2583 and 3337 families (Dohmen et al., 2012: 654, Table 1). In the second set of analyses, where they focused on a selection of family conditions, they included between 427 and 3333 families (Dohmen et al., 2012: 670, Table 8).

For our replication of Dohmen et al.'s (2012) original study, we use data from the first two waves of the German twin family panel *TwinLife*, which were collected in 2014/2015 (wave 1) and 2016/2017 (wave 2; Diewald et al., 2020). *TwinLife* is based on a stratified random sample of administrative data (Lang and Kottwitz 2020). It covers four birth cohorts of monozygotic (MZ) and same-sex dizygotic (DZ) twins, who were aged around 5, 11, 17, and 23 at the time of the first survey. In the first wave, data on 4079 twin pairs were collected, equating to roughly 1000 cases in each cohort (Lang and Kottwitz 2020:132). *TwinLife* was designed to study the development of social inequality over the life course, and covers genetic, social, and psychological factors (Hahn et al., 2016). We restrict the analytical sample to the two oldest twin cohorts, because risk and trust attitudes were not surveyed in the two younger cohorts.

The random samples of households implemented in the *GSOEP* seems to be better suited to address the overall German situation than the cohort samples of twins. However, since *TwinLife* was designed to address family-related influences, it covers periods in which such processes most likely occur: when individuals are young and still live at home. Dohmen et al.'s (2012) analyses of *GSOEP* data refer to individuals aged 17 to 54. Half of them were older than 23, and around 40 percent no longer lived with their parents. In contrast, in the two *TwinLife* cohorts relevant to our replication study (i.e., individuals aged around 17 and 23), the share of individuals in our analytical sample who no longer live with their parents amounts to around 23 percent. A sample of older respondents comes with an additional drawback: as discussed above, heritability in certain traits tends to increase across the lifespan (Bergen et al., 2007; Haworth et al., 2010; Plomin et al., 2016), suggesting that in adulthood, parents' constraining influences decrease, and individuals have more opportunities to self-select into contexts and pursue behaviors in line with their genetic dispositions.[7] Apart from this major difference between the two samples' age composition, the *GSOEP* and *TwinLife* samples show similar distributions for most other characteristics (see Table A1 in the Appendix). Despite a great degree of overlap, it is nevertheless clear that any discrepancy between the two samples can create noise, which can translate into divergences between the results of the original and the replication study. It is, however, impossible to settle, which of the studies reflects the "true" relationships (Janz and Freese 2021: 306).

[5] These processes, by which individuals actively seek an environment related to their genetic dispositions, are known as *active gene-environment correlation* (Plomin, DeFries and Loehlin 1977).

[6] For some of the independent variables, they controlled for missing information (i.e., for education, residency of youth and denomination).

[7] To tackle this consideration, we reran Dohmen et al.'s (2012) analyses with a *GSOEP* subsample of individuals aged 17 to 25. Comparing these findings with those obtained in the original study (covering adults aged 17 to 54) confirms that it is easier to detect attitudinal transmission in a younger sample. The coefficients for the associations between parents' and children's attitudes increase in magnitude compared to the original study. The considerable loss in statistical power due to the smaller sample size does, however, result in fewer systematic differences. To illustrate these differences, we present the coefficients of the original study and the *GSOEP*-subsample in Figure A1 in the Appendix.

Table 1
GSOEP and *TwinLife* measures used in Dohmen et al. (2012) and in the replication study.

	Dohmen et al. (2012), *GSOEP* (2003 and 2004 waves)	Replication study, *TwinLife* (2014/15 and 2016/17 waves)
Dependent variables		
Risk attitudes	"Are you generally a person who is fully prepared to take risks or do you try to avoid risks?" 11-point scale ranging from 0 = "not at all prepared to take risks" to 10 = "very prepared to take risks"	
Trust attitudes	Based on three items: "In general, one can trust people.", "These days you cannot rely on anybody else.", and "When dealing with strangers, it is better to be careful before you trust them." 4-point scale ranging from 1 = "strongly disagree" to 4 = "strongly agree" Measured as an index of unrotated principal component scores (PCA) with standardized input variables. Standardization and PCA scores were calculated separately for children, mothers and fathers.	
Independent variables		
Sex	Child's sex with 1 = "female" and 0 = "male"	
Age	Age (in years)	
Height	Height (in cm)	
Denomination	1 = "Catholic", 2 = "Protestant", 3 = "other Christian", 4 = "other non-Christian", 5 = "no religious affiliation", 6 = "missing information"	Only available in wave 2
Residence	Child's residence before the age of 16 with 1 = "big city", 2 = "city", 3 = "countryside", 4 = "small town", 5 = "missing"	Child's current residence with 1 = "big city", 2 = "city", 3 = "countryside/small town"
Education	Years of education (+ control for missing information)	ISCED-97 codes converted to years of education based on Schneider's (2008b: 9–11) recommendations (+ control for missing information). For children currently enrolled in education, we use their grade level.
Household income	Gross annual household income (in ln Euro)	Gross monthly household income multiplied by 12 (in 1000 Euros; one value assigned to each household)
Health	"How would you describe your current health?" 5-point scale ranging from 1 = "very good" to 5 = "bad"	"How would you describe your state of health during the last 12 months, in general?" 6-point scale ranging from 1 = "excellent" to 6 = "very poor"
Region (fixed effects)	97 "Raumordnungsregionen"	Not available
Nationality	Child's nationality with 17 categories	Child's nationality with 1 = "foreign national", 0 = "German"
GDR experience	Lived in the GDR before 1989 with 1 = "lived in the GDR", 0 = "did not live in the GDR"	Born during the existence of the GDR with 1 = "born in the GDR", 0 = "not born in the GDR" (only for parents)
Independent variables on conditions of social transmission		
Homogeneity of parental attitudes	Only considered for a subsample: difference between parental risk/trust attitudes of less than one standard deviation (="homogenous parents") and of more than one standard deviation (="heterogeneous parents")	Difference between parental risk/trust attitudes (in standard deviations)
Single-parent household	1 = "single-mother", 0 = "mother in two-parent household" Only considered for a subsample	
Siblings	0 = "none", 1 = "one or more siblings"	0 = "twin sibling", 1 = "additional sibling"
Quality of relationship	Frequency of fighting with mother/father when growing up with 0 = "seldom or never", 1 = "very often, often or sometimes" (child's report); coded into 1 = "disharmonious", 0 = "harmonious"	Negative communication based on two items (child's report). "How often do the following things usually happen between you and your parents? 'Your mother/father yells at you because you did something wrong' and 'Your mother/father scolds you because she/he is angry'." 5-point scale ranging from 1 = "never" to 5 = "very often" Measured as a mean index of both input variables.

Notes: If not indicated otherwise, measures were available for the child, the mother, and the father. Collapsed cells indicate that the same measure was available in both surveys. With the exception of denomination, which was measured in wave 2, all remaining variables were measured in wave 1 (*TwinLife*).
Data sources: GSOEP (original study); *TwinLife* (replication and extension study).

In the replication study, we follow Dohmen et al.'s (2012) decisions about restricting the sample as closely as possible. Only in the extension study do we deviate from them. One of the decisions of the authors of the original study was to exclude all families from their analyses in which only one parent answered the questions on trust and risk attitudes, thereby limiting the account to households in which both parents participated in the survey and answered the questions of interest. As a consequence, not just households in which one of the partners did not respond to the risk and trust items were disregarded, but all households where one partner was absent (i.e., all single-parent households). If parents who score higher on risk attitudes are more likely to get divorced (for evidence, see Lowenstein 2005) – and therefore are more likely to raise their child as a single parent – are excluded from the analyses, this could distort the estimates of variables capturing social transmission. A similar issue relates to Dohmen et al.'s (2012) reasoning on the presence of homogeneous versus heterogeneous attitudes at home, and thus to a condition that is assumed to moderate social transmission. By definition, divergent parental attitudes can only be present in two-parent households. The exclusion of single parents from the sample may thus bias the estimate for homogenous versus heterogeneous attitudes. Dohmen et al.'s (2012) decision to focus on two-parent families was based on the *GSOEP*'s household-centered sampling strategy, according to which parents living outside the household are missing by design. *TwinLife*, in contrast, also covers parents who live in a different household.

Overall, *TwinLife* seems to be better suited to identifying social transmission processes, because its sample includes younger individuals who, for the most part, still live at home (relevant for the replication and the extension study), and because data is also available for parents who do not reside in the same household (relevant for the extension study). These represent advantages which come in addition to the key reason for considering *TwinLife* in the first place, that is that it offers the opportunity to consider social and genetic transmission.

3.2. Measures

TwinLife uses many scales and instruments which are also implemented in the *GSOEP*, which facilitates replication. Nevertheless, it is necessary to be aware of a number of differences between the measures covered in the two data sources. First, there is an issue of data availability regarding the regional indicators used in the original study. *TwinLife* lacks information on where the child and their parents lived before the age of 16 ("residence" in the original study), and it does not provide fine-grained regional information ("region"). Consequently, we are unable to replicate Dohmen et al.'s (2012) analyses on contextual influences. To assess the severity of this omission, we estimated multilevel models on the original data with individuals nested in regions. The results reveal that between two and three percent of the variance in risk and trust attitudes was attributable to regional differences. Thus, omitting the regional analyses from our account does not appear to seriously affect the validity of our study. Second, some measures can only approximate the latent constructs, and, given differences between the two data sources, may capture different facets of it. For example, while the *GSOEP* includes information on the "frequency of fighting with parents when growing up", *TwinLife* captures "negative communication" in the family. Both measures are used to assess the "quality of the relationship between parents and the child". Third, in some cases, sample characteristics required us to measure a construct differently. In our analyses of the *TwinLife* data, for example, we need to take into account the fact that most children still live with their parents, meaning that we cannot assign a measure of household income separately to the child, the mother and the father, as this would imply serious collinearity. We therefore use just one household-level measure of gross annual income. In addition, most children in *TwinLife* possess German citizenship, whereas foreign nationals make up a substantive share of the *GSOEP*. Instead of the detailed control of various nationalities that Dohmen et al. (2012) applied, we limit the account to a comparison of foreign nationals to Germans. Table 1 summarizes the variables used in the original (*GSOEP*) and the replication study (*TwinLife*). Table A1 in the Appendix, in addition, displays descriptive statistics for the original (*GSOEP*), the replication and the extension study (*TwinLife*).

For the extension study, we adapt some of the measures. Instead of years of education, we rely on the commonly used *International Standard Classification of Education* (ISCED; OECD, 1999; Schneider 2008a). For parents, we use the ISCED-97 coding with seven categories: primary (ISCED 1), lower secondary (ISCED 2a), upper secondary (ISCED 3a-c), post-secondary non-tertiary (ISCED 4a), tertiary first stage (ISCED 5a), tertiary second stage (ISCED 5b), and doctorate (ISCED 6). For the offspring, we consider a condensed measure, because many of them were still attending school (ISCED 1-2a, ISCED 3a-4, and ISCED 5a-6). Moreover, we consider children's personality characteristics based on the short version of the *Big Five Inventory* (BFI–S; Gerlitz and Schupp, 2005). In this way, we account for the possibility that risk and trust attitudes are an expression of dispositional characteristics (Nicholson et al., 2006; Freitag and Bauer 2016). Finally, we eliminate measures of height and parental denomination from the extension study, because it is unclear how these features contribute to the formation of risk and trust attitudes.

3.3. Analytical strategy

We begin with a duplication of Dohmen et al.'s (2012) original study. With this reproduction, we assess whether proceeding in the same way and using the same data (i.e., the *GSOEP*) produces the published result (Janz and Freese 2021: 305). Thereafter, we replicate their analyses using *TwinLife* data. In this step, we follow the original study as closely as possible by considering the authors' decisions to restrict the sample, the measurements used, and their treatment of missing information. Relying on a different data source allows the robustness of the published findings to be assessed (p. 306). In the subsequent extension study, we deliberately deviate from the original analyses by adapting some of the measurements; by including single-parent households, and by using multiple imputation to deal with missing information. With these adaptations, we attempt to increase the scope of the findings (p. 305). We refer to these empirical accounts as the original study, the replication study, and the extension study.

The key element of both the replication and the extension study is the use of a research design which enables us to consider genetic similarities between parents and their children – in addition to similarities arising from social conditions. For this purpose, we apply a twofold modelling strategy. In the first part, we partition the variance in children's risk and trust attitudes into three different components, a genetic component (A), a component attributable to differences in the shared environment (C), and a residual component (E; the non-shared environment). The findings from these analyses inform us about the different sources of variation in risk and trust attitudes. We learn, for example, that 33 percent of the overall variation in children's risk attitudes can be attributed to genetic differences. This knowledge about the sources of variation in risk and trust attitudes can then be used to interpret the results of our regression models, which comprise the second part of the modelling strategy. In this part, we estimate regression coefficients in line with the standard regression approach, and accordingly present correlations between the various independent variables and children's trust and risk attitudes – just like the original study by relying on a genetically-informed research design. This part of the analysis is thus equivalent to the proceedings implemented by Dohmen et al. (2012) and will, if the findings are robust, yield similar regression coefficients. While we do not "control for" genetic similarity in the regression part, we use the results of the variance partitioning to determine which of the independent variables contributes to an explanation of which variance component. For example, do parental attitudes explain variance associated with the shared environment component, or do they instead explain variance associated with the

genetic component? We assess these contributions by adding the variable of interest (in the above example parental attitudes) to the regression model, and then observe the change in variance components compared to the model without this variable. A decrease in a component indicates that the variable in question (partly) explains this component, whereas an unchanged component indicates that the variable does not contribute to its explanation. Thereby the additional feature of our study, which ensues from the genetically informed research design, is the variance partition. This partition informs our interpretation of the relationships assessed in the regression models. In the remainder of this section, we present the statistical methods and discuss the underlying assumptions.

Using multilevel modelling techniques, the variance decomposition can be extended to multivariate models. These extensions allow for the consideration of mean differences in outcomes by including additional random effects (Rabe-Hesketh et al., 2008). In effect, a multilevel model is set up in such a way that the original two-level data (twins [i] in families [k]) is reparametrized to a three-level model by inserting an additional artificial twin level (twins in twins in families [j]). Formally, this model is written as:

$$y_{ijk} = b_0 + \left\{ a_{jk} \left[\sqrt{\frac{1}{2}}\, \overline{MZ_k} \right] + a_k \left[MZ_k + \sqrt{\frac{1}{2}}\, \overline{MZ_k} \right] \right\} + c_k + \varepsilon_{ijk} \tag{1}$$

where MZ_k represents a dummy variable taking the value 1 for MZ twins and 0 for DZ twins (corresponding to $\overline{MZ_k}$). Note that this dummy variable enters the model as a random slope at the corresponding level but not as the main effect. The variance of c_k represents the shared environment component (C), whereas the variance of e_{ijk} represents the non-shared environment component (E). In this estimation, all variance components are mutually uncorrelated and the random slopes a_{jk} and a_k are constrained to have equal variances. The variances of a_{jk} or a_k represent the additive genetic component (A). Using this parametrization generates additive genetic components with a correlation of 1 for MZ and a correlation of 0.5 for DZ twins.

An advantage of this specification is the ease by which changes in assumptions about twin models can be incorporated statistically. If we suspect that assortative mating on risk and trust attitudes biases the results – which we do in view of Dohmen et al.'s (2012) findings – we can increase the assumed correlation of the DZ twin scores by changing the fractions of the square root terms in Equation (1) (e.g., changing 1/2 to 3/5 assumes a correlation of 0.6 rather than 0.5). In our application, we adjust the correlation of DZ twin scores to 0.52 for risk attitudes and to 0.56 for trust attitudes. These values are based on a procedure proposed by Loehlin et al. (2009).[8] This approach follows the notion that assortative mating increases the chances that genetic information contributing to the formation of risk and social trust attitudes is transmitted to the child. In contrast to these considerations on genetic transmission, Dohmen et al. (2012) discussed assortative mating from a social transmission perspective, arguing that parents with similar attitudes are better able to work in concert and are therefore more efficient in transmitting these attitudes.

We estimate the various models based on the Stata ado "ACELONG" (Lang 2017). To deal with missing information, in the extension study, we use multiple imputation with chained equations (50 imputed data sets). The replication code for our study is available at https://osf.io/dvg9n/(https://doi.org/10.17605/OSF.IO/DVG9N).

Twin studies and decomposition models (such as the ACE-model) rely on assumptions which need to be met to adequately assess the relative importance of genes, the shared environment and the non-shared environment. Violations of these assumptions result in biased estimates of the three components. One crucial assumption relevant to twin modelling is that parents mate randomly. If there is non-random mating, and various disciplines provide evidence that this is the case for many traits (e.g., Blossfeld 2009; Kalmijn 1998), there is more genetic similarity among parents than expected by random chance. As a consequence, DZ twins born to these parents share more than the average 50 percent of the genes for the trait in question. This violation leads to an underestimation of the A component (i.e., genetic heritability) and an overestimation of the C component (i.e., the shared environment; Røysamb and Tambs 2016: 40). As discussed above, the models used in our replication and extension study address the bias caused by assortative mating.

Another important assumption underlying twin modelling is that MZ and DZ twins share environmental influences to the same extent (i.e., the equal environment assumption; Conley et al., 2013; Polderman et al., 2015). This assumption implies, for example, that MZ twins are not treated more equally than DZ twin simply because they are MZ twins. A violation of this assumption will underestimate C (i.e., the shared environment) and overestimate A (i.e., genetic heritability), because some of the variation in DZ twins is due to differences in the environment rather than to genetic differences (Derks et al., 2006). However, evidence suggests that the equal environment assumption is not violated for a wide variety of traits (Conley et al., 2013; Polderman et al., 2015).

A third assumption is that most traits follow an additive genetic model. This means that so-called loci (i.e., specific positions of genes) contribute in an additive manner to a certain trait. Whenever a trait is influenced by dominant alleles, or if alleles at different loci interact with one another, this assumption is violated. The presence of non-additive effects can lead to an overestimation of A (i.e., genetic heritability; Zuk et al., 2012). Empirical findings indicate that roughly two-thirds of complex traits follow the assumption of additive genetic variation (Polderman et al., 2015).

Finally, estimates of the genetic component of a trait can also be biased due to gene-environment correlations (Sternberg 2013). For example, children may actively seek environments related to their genetic disposition (i.e., active gene-environment correlation), or parents may react to their child's genetic endowment and therefore provide a certain environment (i.e., reactive gene-environment correlation; Plomin et al., 1977). The presence of such gene-environment correlations can lead to an overestimation of A (i.e., genetic heritability; Sternberg 2013). Unfortunately, the extent of this overestimation is unclear. It is also an open question whether, in

[8] They calculate the DZ twin correlation with assortative mating as $0.5+(0.5*h^2*r_p)$, where h^2 corresponds to trait heritability and r_p to the trait correlation among parents. Based on *TwinLife*, we can estimate these values for risk and trust attitudes. For example, for risk attitudes, genetic heritability is 0.39 and the correlation among parents is 0.09, which results in a corrected DZ twin correlation of $0.05+(0.05*0.39*0.09) \sim 0.52$.

such instances, the shared environment or the non-shared environment are underestimated. Bivariate correlations among the MZ and DZ twin pairs can be found in Appendix Table A5.

4. Results

Our duplication of Dohmen et al.'s (2012) empirical study reproduces the published coefficients, and thus verifies the original results. For the replication and the extension study, we present findings from a baseline ACE model, in which we partition the overall variance in risk and trust attitudes into three components: additive genetic heritability (A), shared environment (C) and non-shared environment (E). In our application, social transmission in the family should be captured primarily by the shared environment. Table 2 presents the results for the replication and the extension study. We present the estimates side-by-side, while being aware that the models rely on different samples. For the extension study, we can make use of a considerably larger sample, since we not only consider families in which both parents are present, but also impute missing values. In the following, we refer to the estimates of the extension study, as they are very similar to those of the replication study.

The findings illustrated in Table 2 indicate that risk and trust attitudes are not attributable to differences in the family environment (C), suggesting that the within-family transmission pathway is irrelevant for the intergenerational transfer of attitudes. Instead, about 67 percent of the differences in risk attitudes are due to differences in non-shared environments and the remaining 33 percent are attributable to genetic differences. For social trust, these value amount to 54 percent for the non-shared environment and to 45 percent for the genetic component.

These results resemble those of earlier decompositions (for risk attitudes, see e.g. Cesarini et al., 2009; Cesarini et al., 2010; Le et al., 2010; for trust attitudes, see e.g., Kettlewell and Tymula 2021; Reimann et al., 2017; Sturgis et al., 2010; Oskarsson et al., 2012, Weinschenk and Dawes 2019) and consistently reveal the same pattern, according to which attitudinal differences are due mainly to differences in the non-shared environment (E), followed by genetic differences (A), while the shared environment (C) does not account for attitudinal differences. Unlike our study, these contributions do not address intergenerational transmission, and therefore do not broach the issue of whether interpretations regarding social transmission, such as those proposed by Dohmen et al. (2012), hold when a genetically informed research design is used based on data with similar measures from the same research context.

In Fig. 2, we present regression findings and plot the estimates of parents' risk and trust attitudes on their offspring's attitudes separately for the original study, as well as for our replication and extension study. The coefficients illustrate the degree to which parental attitudes are correlated with those of their children, while controlling for a range of independent variables. The main finding is that the estimates obtained in the replication and the extension study are considerably smaller than those of the original study. For risk attitudes, replication and extension coefficients are between 48 and 65 percent (for mothers) and around 75 percent (for fathers) smaller than the coefficients of the original study. The difference is less pronounced for social trust attitudes, with coefficients of between 17 and 28 percent (for mothers), and between 34 and 43 percent (for fathers) smaller than those obtained by Dohmen et al. (2012). While we find statistically significant coefficient estimates of the direct transmission pathway from parental attitudes on child attitudes in most cases, the sizes of these coefficients suggest that parents' attitudes influence their children's attitudes only to a minor extent. The strongest relationship exists between mothers' trust attitudes and those of their offspring: for a one-standard deviation increase in maternal attitudes, the child's trust score increases by 0.2 standard deviations.

Despite being reduced, the estimates illustrated in Fig. 2 still point to significant associations between parents' and their offspring's risk and trust attitudes. These relationships seem to suggest that social transmission takes place after all – a conclusion that seems to be at odds with the findings presented in Fig. 2. How can these seemingly divergent results captured in Fig. 2 and Table 2 be reconciled? To make sense of them, we move away from framing the association between parents' and children's attitudes solely in terms of the social transmission taking place within families. Instead, we examine, whether it also has genetic, and non-shared environment components. For this purpose, we investigate whether parental attitudes explain variance in the genetic, the shared, and the non-

Table 2
Variance components of risk and trust attitudes.

	Replication		Extension	
	Risk	Trust	Risk	Trust
Variance terms				
Additive genetic heritability (A)	1.62 (0.19)	0.25 (0.02)	1.66 (0.20)	0.24 (0.01)
Shared environment (C)	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)	0.03 (0.00)
Non-shared environment (E)	3.28 (0.18)	0.26 (0.01)	3.44 (0.18)	0.29 (0.01)
Percent total variance				
Additive genetic heritability (A)	33.0	49.0	32.5	42.8
95 %-confidence interval	[29.9; 35.4	[46.7; 50.8]	[29.1; 35.1]	[42.5; 43.1]
Shared environment (C)	0.0	0.0	0.0	5.4
95 %-confidence interval	[0.0; 0.0]	[0.0; 0.0]	[0.0; 0.0]	[5.3; 5.4]
Non-shared environment (E)	67.0	51.0	67.5	51.8
95 %-confidence interval	[64.6; 70.1	[49.2; 53.3]	[64.9; 70.9]	[51.5; 52.1]
n	2056	1965	4012	4012

Note: Standard errors in parentheses.
Data source: TwinLife.

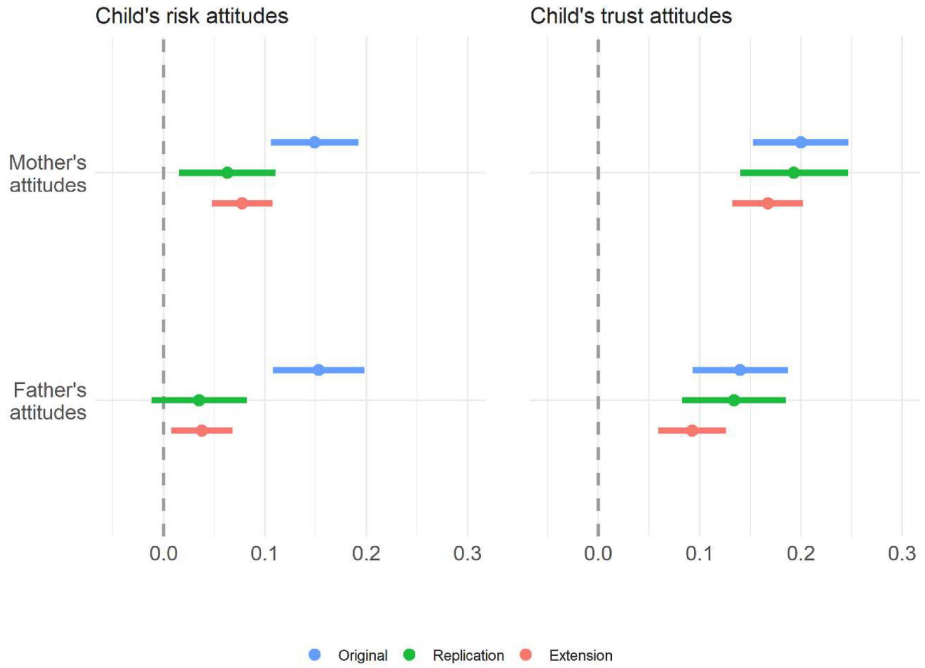

Fig. 2. Direct transmission coefficients of risk and trust attitudes from the original study, the replication study and the extension study.
Data sources: GSOEP (original study); *TwinLife* (replication and extension study).*Notes*: The dots show the regression coefficients, the horizontal lines the 95 percent confidence intervals. Original = Dohmen et al.'s (2012: 654) original study (based on Table 1, Model 3 for risk attitudes and Table 1, Model 7 for trust attitudes); Replication = replication of the original study with *TwinLife* data following the sample and variable selection of the original study as best as possible; Extension = replication study with additional features as specified above (i.e., adapted measurements, inclusion of single-parent households, multiple imputation). Table A2 in the Appendix displays the corresponding detailed findings for the replication study and the extension study.

shared environment components of children's corresponding attitudes.

For risk and trust attitudes, Fig. 3 shows two models. The first model depicts the baseline variance decomposition discussed earlier (i.e., the variance terms of the replication study presented in Table 2). In the second model, we add mothers' and fathers' attitudes. Comparing the variance components of the baseline model with those of the second model informs us whether and how variance components change once parental attitudes are included. If, for example, the genetic component decreases in the second model, this could imply that part of the relationship between parents' and children's attitudes involves genetic similarity (see section Analytical strategy on p. 18 for more detail). The key finding of this analyzing step is that adding parental attitudes leads to a decrease in the variance terms related to additive genetic heritability. This pattern applies to both risk and trust attitudes but is more pronounced for social trust.[9] Hence, the contradiction between the findings presented in Table 2 and Fig. 2 resolves to the conclusion that, what is commonly interpreted as evidence for social transmission within families, is actually due to genetic similarity between parents and their offspring. This finding should not distract from another important result: most of the differences between individuals are due to non-shared environmental conditions, that is, the chance encounters, unique experiences or relationships individuals have, which they do not share with their siblings.

With these results in mind, we turn to the replication of Dohmen et al.'s (2012) analyses on a range of family conditions that the authors assumed to moderate the intergenerational transmission of attitudes. Fig. 4 for risk attitudes, and Fig. 5 for trust attitudes present the corresponding coefficient estimates for the original, the replication, and the extension study. Each condition is depicted with its two manifestations; for example, the first panel presents attitudinally homogeneous versus attitudinal heterogeneous relationships. The dots in these figures stand for the coefficient estimates, and the lines crossing them horizontally indicate the corresponding 95 percent confidence intervals. For example, in the original study, a one-standard deviation increase in mothers' risk attitudes is associated with a 0.14 point increase in children's risk attitudes when the mother is in an attitudinally homogenous relationship. For mothers in heterogeneous relationships, this value amounts to 0.11 points. The confidence intervals of these two coefficients show a far-reaching overlap, indicating that the observed differences between the two conditions are unsystematic (statistical tests of coefficient differences are presented as shaded cells in Tables A3 & A4). The same holds true for the corresponding green markers that are used for the replication study, and the blue markers for the extension study.

Of all the models captured in Figs. 4 and 5, only three transmission conditions appears to moderate the relationship between parents' and their offspring's attitudes: in the original study, fathers' risk attitudes are more strongly related to their offspring's

[9] The slight increase in the variance component of the non-shared environment hints at the presence of more family-level variance than expected under random sampling (Hox 2010: 75).

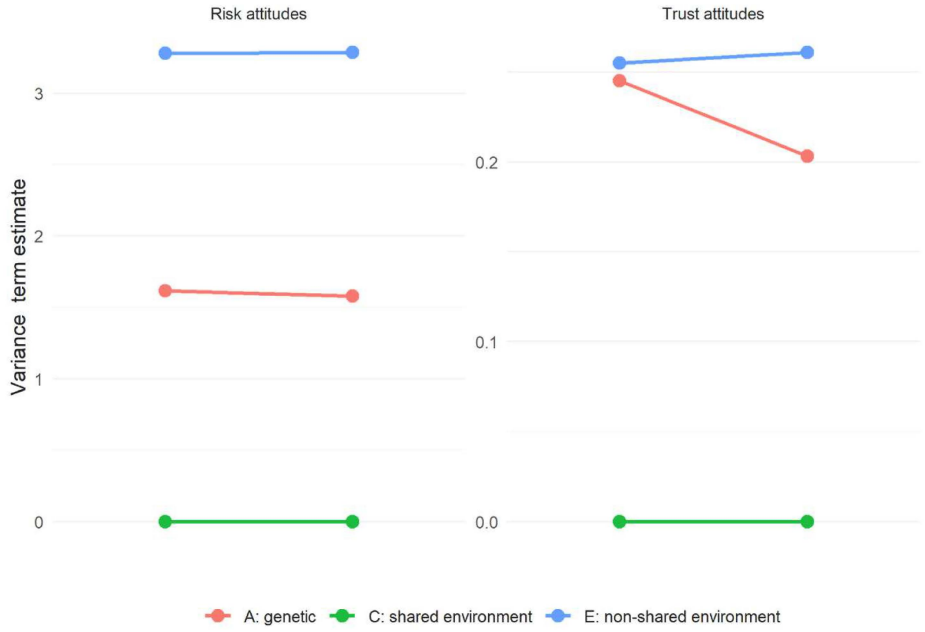

Fig. 3. Changes in variance components of risk and trust attitudes (replication study).
Data source: TwinLife.Notes: The variance estimates of the baseline models correspond to the estimates of the replication study presented in Table 2. The variance estimates of the subsequent models cover the additional influence of parental attitudes.

attitudes when children indicate that the relationship is harmonious, compared to constellations in which children fight with their parents; the same holds true for social trust and mother's attitudes in the replication study and in the extension study, mother's trust attitudes are more strongly related to their children's when fewer siblings are present. For all other 51 comparisons of transmission conditions, overlapping confidence intervals and z-tests suggest unsystematic differences across the original, the replication, and the extension study. As with the main results, the replication and extension analyses produce smaller coefficient estimates than the original analyses. Overall, neither the original study, nor our replication and extension studies provide substantive evidence for a moderation of the intergenerational transmission of attitudes via family conditions.

5. Discussion

In this article, we replicated a much-cited study by Dohmen et al. (2012), which examined the intergenerational transmission of risk and trust attitudes. The replication is based on data from the German *TwinLife* study. In our replication, we follow the analytical choices made by the authors of the original study as closely as possible. The additional extension study makes use of a larger analytical sample as a result of applying multiple imputation techniques to deal with missing observations, and of considering single-parent households. We also adapted some of the measurements.

The replication and the extension study reproduce the patterns of intergenerational attitude transmission identified in the original study. However, the estimates of the relationships between parents' and their children's' attitudes are considerably smaller. The reasons for this difference are difficult to pin down and vary depending on what is assumed to be the origin of the intergenerational transmission effects. According to socialization arguments, analyses of the replication sample of younger individuals still living with their parents should yield stronger effects than in the original study. One reason why this is not the case could be that the twins tend to be more influenced by each other, and this crowds out parental influence due to less exposure to them. In contrast, a more "gene-centric" explanation could attribute the stronger effect in the original study to the presence of gene-environment correlations, where genetic effects become more pronounced with increasing age, creating a stronger intergenerational correlation (Haworth et al., 2010).

More importantly, the underlying genetically informed research design allows for a different interpretation of these findings. Variance decomposition results suggest that variation in children's risk and trust attitudes is primarily related to differences in the non-shared environment and secondarily to genetic differences. According to these findings, there is little room for social transmission shared within families, as this transmission pathway should be captured in the shared environment component. We also show that family conditions do not moderate the relationships between parents' and their offspring's attitudes and therefore that the replication results do not support the socialization assumption.

Dohmen et al. (2012: 647, 668–669) discuss the implications of their findings in contrast to views which highlight the importance of genetic heritability and the non-shared environment, in addition to the shared environment. Rather than ruling out the possibility that genetic mechanisms may also play a role in the formation of children's risk and trust attitudes, the authors of the original study point to three results that, in their view, speak for social rather than genetic transmission. In the following, we discuss these findings and the interpretations offered by Dohmen et al. (2012) from a genetically informed transmission perspective. Through this discussion, we attempt to illustrate the ways in which their interpretations can be reconciled with a genetically informed view, and where the

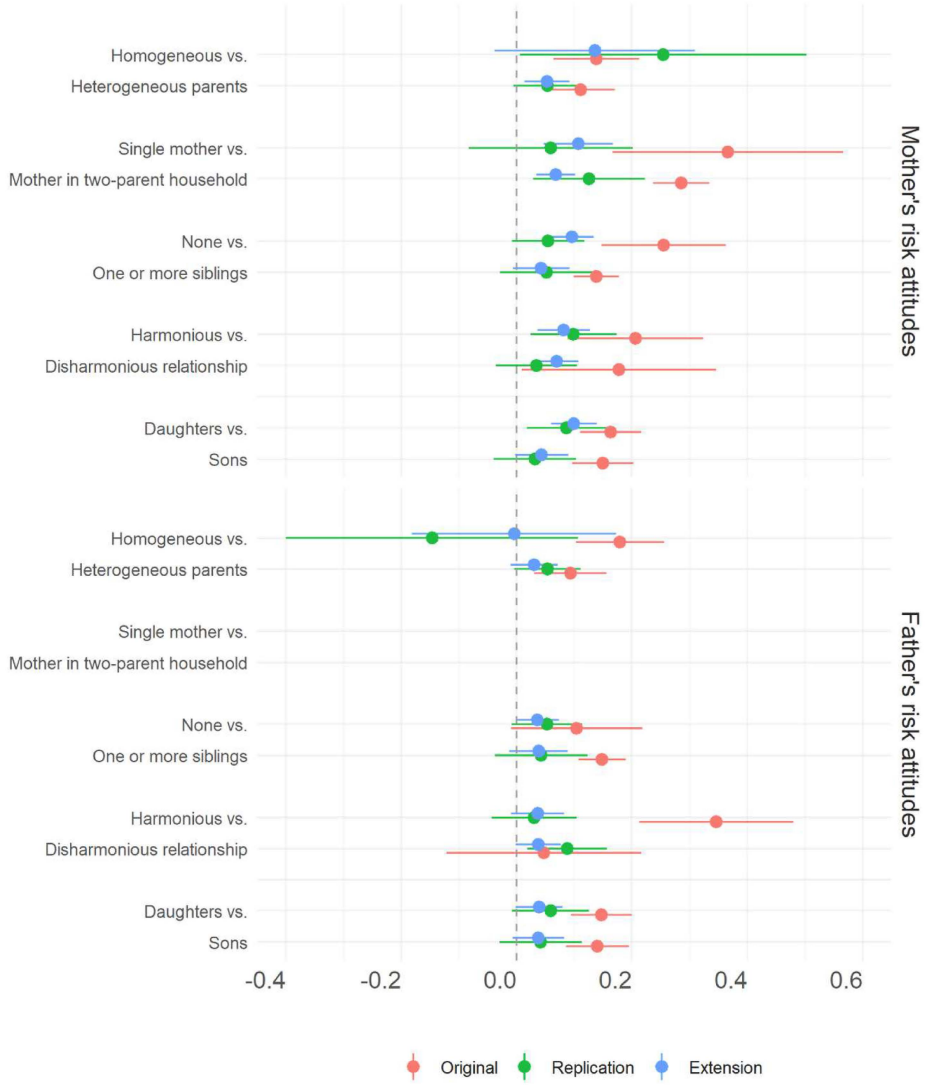

Fig. 4. Conditional transmission coefficients for risk attitudes from the original study, the replication study and the extension study.
Data sources: GSOEP (original study); *TwinLife* (replication and extension study).*Notes*: The dots show the regression coefficients, the horizontal lines the 95 percent confidence intervals. Original = Dohmen et al.'s (2012: 670) original study (based on Table 8, Panel A); Replication = replication of the original study with *TwinLife* data following the sample and variable selection of the original study as best as possible; Extension = replication study with additional features as specified above (i.e., adapted measurements, inclusion of single-parent households, multiple imputation). Table A.3 in the Appendix displays the corresponding detailed findings for the replication and the extension study.

genetically informed approach offers alternative interpretations.

The first finding the authors raise when arguing that transmission involves socialization rather than genetic mechanisms refers to the tendency for mothers to be more important than fathers in the transmission of trust attitudes (Dohmen et al., 2012: 669). In their view, this tendency "would not be expected based on genetic transmission but indicates a process of socialization in which parents have unequal impacts on child attitudes" (Dohmen et al., 2012: 669). Behavioral genetics offers an alternative interpretation of the relatively greater importance of the mother for a child's trait, according to which only biological mothers can exert prenatal environmental effects, while fathers enter the child's environment only after birth (Loehlin 2016). The presence of prenatal environmental effects has been illustrated for a range of outcomes, most of them focusing on children's health throughout different stages of their lives (e.g., Lumey et al., 2011; Niaura et al., 2001; Veenendaal et al., 2013). We are not aware of empirical contributions linking differences in prenatal environments to attitudes. Nevertheless, the presence of such effects in other domains, and empirical findings on such effects in animals (Muñoz-Villegas et al., 2017) hints at the possibility that they could also play a role in attitude formation. If this were the case, a genetically informed interpretation would still refer to the shared environment component, albeit – and this would be in contrast to the original study – pointing at influences taking place inside, rather than outside the womb. As a side note, the stronger correlation between mothers' and children's attitudes compared to the correlation between fathers' and their children's attitudes only appears for trust, but not for risk attitudes. Moreover, it is generally relatively small and could thus simply results from randomness for instance related to measurement error.

The second result highlighted by Dohmen et al. (2012: 668) is that regional attitudes are correlated with children's attitudes. Due to

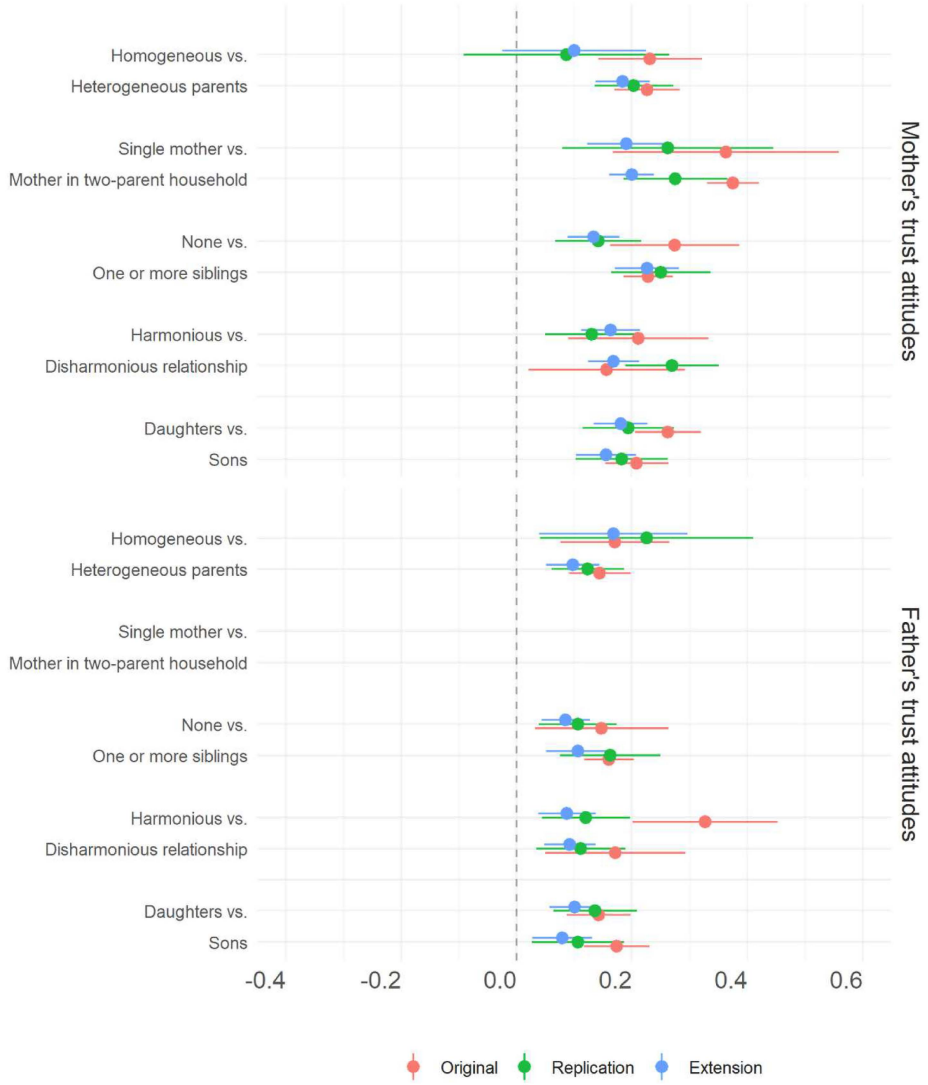

Fig. 5. Conditional transmission coefficients for trust attitudes from the original study, the replication study and the extension study.
Data sources: GSOEP (original study); *TwinLife* (replication and extension study).*Notes*: The dots show the regression coefficients, the horizontal lines the 95 percent confidence intervals. Original = Dohmen et al.'s (2012: 670) original study (based on Table 8, Panel B); Replication = replication of the original study with *TwinLife* data following the sample and variable selection of the original study as best as possible; Extension = replication study with additional features as specified above (i.e., adapted measurements, inclusion of single-parent households, multiple imputation). Table A.4 in the Appendix displays the corresponding detailed findings for the replication and the extension study.

restrictions in data access (i.e., fine-grained regional data is not available for reasons of anonymization), we are unable to replicate this aspect of the original study based on *TwinLife*. Nevertheless, it is possible to incorporate the corresponding argumentation of the authors of the original study into the decomposition framework. According to Dohmen et al. (2012: 669), "the impact of regional attitudes suggests an important role for genetically unrelated individuals as role models and is thus a clear indicator of socialization". This socialization takes place outside the family and so, in the terminology of behavioral genetics, would be part of the non-shared environment component. For example, in regions in which risk-prone attitudes are more prevalent, there may be more opportunities for exposure to risky behaviors and their consequences. These experiences, in turn, may shape the risk attitudes of children. Note that this reasoning does not necessarily imply systematic socialization, but is consistent with the view of idiosyncratic experiences that are captured by the non-shared environment component. We would therefore argue that the interpretation offered by the authors of the original study can be reconciled with an interpretation following a genetically informed perspective. At the same time, given that only 2 to 3 percent of the variance in attitudes was attributable to the regional level in the original study, the importance of regional differences should not be overstated.

The third finding that the authors of the original study interpret as evidence for the socialization assumption rather than genetic transmission is based on their examination of a set of family conditions which they expect moderate the intergenerational transmission of attitudes. Dohmen et al. (2012: 669) argue that such moderation effects "should be irrelevant from the perspective of genetic transmission". We disagree with this assessment for two reasons. First, with a genetically informed research design it is possible to include both the genetic, as well as the social transmission pathway. This design is also compatible with a moderation of attitudinal

transmission via family conditions. Second, in our view, the empirical results do not support the authors' claim that differences in family conditions are relevant to children's attitude formation, as the estimates of the original study are mostly insignificant and rather small. In the original study, with the exception of one condition (i.e., a stronger transmission of father's risk attitudes in harmonious versus disharmonious relationships), no systematic evidence was found for a within-family moderation effect. This result is in line with the decomposition findings obtained in the replication and extension study, which indicate that none of the variance in children's attitudes can be attributed to the shared-environment. Taken together, the three findings discussed as evidence of social transmission by Dohmen et al. (2012) can be reconciled with a genetically informed interpretation.

What should we conclude from the replication and extension study? First, they provide further evidence that an assessment of intergenerational transmission requires the consideration of genetic heritability in addition to socio-environmental influences (Conley et al., 2015; Liu 2018; Mills and Tropf 2020; Erola et al., 2022). Without separating these components, variations in children's risk and trust attitudes are assumed to be shaped by social conditions, and the estimates of social transmission within families are likely to be biased (Liu 2018: 279). Recognizing the possibility of genetic confounding is thus necessary to adequately assess socioenvironmental influences. Adopting this perspective by no means suggests a deterministic view: genes do *not* determine one's attitudes (or any other traits), but their effect depends on the social contexts to which individuals are exposed (Liu 2018: 279). Following this "interactionist consensus" according to which all traits are the result of genes and environments (Kitcher 2001), we would encourage researchers who do not rely on data sources that allow genetic heritability to be addressed directly, to follow a more tentative approach to interpreting their empirical findings. In their interpretations, researchers might do well to not only acknowledge the possibility that their estimates of social transmission probably contain genetic transmission, they could also reflect upon the available empirical evidence that consistently shows that the shared environment is of little importance to the intergenerational transmission of many traits (e.g., Turkheimer 2000), including risk and trust attitudes (for risk attitudes, see e.g. Cesarini et al., 2009; Cesarini et al., 2010; Le et al., 2010; for trust attitudes, see e.g., Kettlewell and Tymula 2021; Reimann et al., 2017; Sturgis et al., 2010; Oskarsson et al., 2012; Weinschenk and Dawes 2019).

Second, despite refuting Dohmen et al.'s (2012) claims on social transmission of trust and risk attitudes within families, we do not suggest that the social context does not matter. Quite to the contrary, the replication findings show that the largest part of the variance in children's attitudes is due to the non-shared environment. Future research therefore needs to concentrate on this component and address socio-environmental influences outside the nuclear family or other conditions that might make two twins growing up in the same family different from each other.

Third, genetics can help us to better understand intergenerational transmission. In addition to direct genetic effects, genes can operate indirectly – through the social environment. For example, children who are genetically prone to risky behaviors may actively seek environments related to this disposition, and therefore reinforce this tendency via their social environment by selecting friends with certain attributes, or by pursuing specific activities. In these instances, environmental exposure is connected to an individual's genetic make-up (i.e., gene-environment correlation; Plomin et al., 1977). Genes may also moderate environmental exposure so that the effect of a certain environment is conditional on an individual's genes and vice versa (i.e., gene-environment interaction; Erola et al., 2022). For example, an adolescent with a moderate genetic propensity for risky attitudes and behaviors may be more susceptible to peers' risky behaviors than an adolescent with a high or a low propensity level. In this scenario, the risky behavior of others only matters for those in the middle of the genetic risk distribution.[10] Indirect effects of this kind involve the social environment, and underline that social context matters, albeit via a different pathway than the within-family socialization route proposed by Dohmen et al. (2012). Addressing the interplay between genes and the social environment allows social scientists interested in questions of how social conditions shape the transmission of attitudes and behaviors to move forward collaboratively. This route acknowledges that the genetic code is responsive to the social environment. Pursuing it opens up the possibility of eliciting change in attitudes and behavior – via social influences that are malleable to policy interventions. A recent intervention study (Kuo et al., 2019) illustrates this reasoning. The authors show that a family-centered intervention during adolescence moderates the association between the genetic risk for alcohol dependence and an alcohol dependence diagnosis as a young adult. Whereas among participants in the intervention condition there was no association between the genetic risk and a later alcohol dependence diagnosis, in the control condition, the genetic risk for alcohol dependence was associated with a greater likelihood of receiving an alcohol dependence diagnosis during adulthood. These findings illustrate that modifying the social environment of individuals who are genetically vulnerable to alcohol dependence can reduce the likelihood of developing alcohol dependence (Kuo et al., 2019: 983). Rather than taking the genetic setup as a given about which nothing can be done, this example shows that considering the interplay between a genetic risk and social conditions is key to improving individuals' life chances.

CRediT authorship contribution statement

Christoph Spörlein: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing. **Cornelia Kristen:** Conceptualization, Formal analysis, Investigation, Methodology, Supervision, Writing – original draft, Writing – review & editing. **Regine Schmidt:** Conceptualization, Formal analysis, Investigation, Methodology, Software, Writing – original draft, Writing – review & editing.

[10] This exemplary scenario follows empirical findings on the interaction between the genetic disposition for alcohol consumption and peer drinking behavior (Guo et al., 2015).

Declaration of competing interest

None.

Appendix

Table A.1
Descriptive statistics for the original (n = 3337), the replication (n = 1960) and the extension study (4,014).

	Original			Replication/ Extension	Replication		Extension		Replication/ Extension
Variable	Range	Mean/ proportion	sd	Range	Mean/ proportion	sd	Mean/ proportion	sd	Percent missing
Dependent variables									
Risk attitudes	0–10	5.23	2.23	0–10	5.75	2.20	5.80	2.26	0.20/0.17
Trust attitudes	−3.37–3.92	0.00	1.31	−1.65–1.92	−0.08	0.71	0.00	0.73	1.28/1.27
Independent variables									
Mother's risk attitudes	0–10	3.75	2.30	0–10	4.24	2.23	4.33	2.37	n.a./5.28
Father's risk attitudes	0–10	4.57	2.37	0–10	5.33	2.25	5.31	2.27	n.a./43.82
Mother's trust attitudes	−3.34–3.99	0.00	1.27	−1.54–1.91	−0.08	0.69	0.00	0.72	n.a./7.68
Father's trust attitudes	−3.20–3.96	0.00	1.31	−1.53–2.10	−0.04	0.72	−0.02	0.73	n.a./44.77
Female	0–1	0.47		0–1	0.54		0.57		
Age	17–54	25.23	6.97	16–25	19.60	3.02	19.90	3.08	
Mother's age	34–84	51.64	8.25	37–64	50.19	4.78			0.00/n.a.
Father's age	33–87	54.49	8.62	38–79	52.72	5.54			0.00/n.a.
Height	140–210	174.23	9.27	100–204	173.58	9.77			7.40/n.a.
Mother's height	141–189	164.44	6.22	74–189	167.19	7.53			1.53/n.a.
Father's height	139–200	176.32	7.14	85–204	180.10	7.79			1.02/n.a.
Denomination									38.78/44.05
Catholic	0–1	0.26		0–1	0.21		0.19		
Protestant	0–1	0.29		0–1	0.23		0.19		
Other Christian	0–1	0.03		0–1	0.03		0.02		
Other non-Christian	0–1	0.06		0–1	0.03		0.03		
No religious affiliation	0–1	0.25		0–1	0.12		0.12		
Missing	0–1	0.12		0–1	0.39				
Mother's denomination									32.24/n.a.
Catholic	0–1	0.29		0–1	0.25				
Protestant	0–1	0.33		0–1	0.25				
Other Christian	0–1	0.03		0–1	0.02				
Other non-Christian	0–1	0.07		0–1	0.01				
No religious affiliation	0–1	0.26		0–1	0.14				
Missing		0.02		0–1	0.32				
Father's denomination									38.98/n.a.
Catholic	0–1	0.29		0–1	0.20				
Protestant	0–1	0.28		0–1	0.21				
Other Christian	0–1	0.03		0–1	0.01				
Other non-Christian	0–1	0.07		0–1	0.02				
No religious affiliation	0–1	0.31		0–1	0.18				
Missing	0–1	0.02		0–1	0.39				
Residence									0.00/0.10
Big city	0–1	0.19		0–1	0.21		0.19		
City	0–1	0.18		0–1	0.28		0.27		
Countryside	0–1	0.30		0–1	0.51		0.54		
Small town	0–1	0.20		0–1					
Missing	0–1	0.12		0–1					
Years of education	7–18	11.97	2.35	1–17.5	12.24	2.07			0.61/n.a.
Missing	0–1	0.22		0–1					
Mother's years of education	7–18	11.60	2.62	4–20	14.43	2.69			0.31/n.a.
Missing	0–1	0.02		0–1					
Father's years of education	7–18	12.17	2.86	4–20	15.03	2.83			0.82/n.a.
Missing	0–1	0.02		0–1					
Education									n.a./0.40
ISCED 1-2a				0–1			0.18		

(*continued on next page*)

Table A.1 (*continued*)

	Original			Replication/ Extension	Replication		Extension		Replication/ Extension
Variable	Range	Mean/ proportion	sd	Range	Mean/ proportion	sd	Mean/ proportion	sd	Percent missing
ISCED 3a-4				0–1			0.72		
ISCED 5a-6				0–1			0.09		
Mother's education									n.a./5.08
ISCED 1				0–1			0.02		
ISCED 2a				0–1			0.07		
ISCED 3a-c				0–1			0.39		
ISCED 4a				0–1			0.10		
ISCED 5a				0–1			0.22		
ISCED 5b				0–1			0.14		
ISCED 6				0–1			0.01		
Father's education									n.a./43.80
ISCED 1				0–1			0.02		
ISCED 2a				0–1			0.03		
ISCED 3a-c				0–1			0.37		
ISCED 4a				0–1			0.06		
ISCED 5a				0–1			0.34		
ISCED 5b				0–1			0.13		
ISCED 6				0–1			0.05		
Household income*	0–12.81	10.39	1.62	0–2744.96	78.69	142.26	55.81	106.46	
Household income Mother	0–13.08	10.02	2.36						
Household income Father	0–13.08	10.07	2.31						
Health*	1–5	2.06	0.81	1–6	2.40	0.94	2.48	0.98	0.51/0.47
Mother's health	1–5	2.75	0.92	1–6	2.92	0.90			0.71/n.a.
Father's health	1–5	2.75	0.88	1–6	2.82	0.90			0.31/n.a.
Foreign national*	0–1	0.09		0–1	0.01		0.01		
GDR experience*	0–1	0.29							
Mother's GDR experience	0–1	0.29		0–1	0.10		0.11		0.10/3.69
Father's GDR experience	0–1	0.29		0–1	0.09		0.09		0.20/38.63
Homogeneity of parental attitudes*dtbl									
Risk attitudes	0–1	0.46		0–1	0.34		0.34		n.a./48.68
Trust attitudes	0–1	0.61		0–1	0.49		0.49		n.a./50.42
Single-parent household	0–1	0.03		0–1	0.10		0.22		n.a./0.00
Sibling*	0–1	0.89		0–1	0.37		0.36		n.a./0.00
Disharmonious relationship*	0–1	0.47		1–5	2.13	0.82	2.15	0.85	0.71/13.26
Big Five									n.a./0.17
Agreeableness				1.67–7			5.52	0.97	
Openness				1–7			4.94	1.05	
Conscientiousness				1–7			5.22	10.6	
Extraversion				1–7			4.86	1.33	
Emotional stability				1–7			4.20	1.24	

Data sources: GSOEP (original study) and TwinLife (replication and extension study).

Notes: All variables without reference to the mother or the father either refer to the child, or apply to the household. n.a. = "not available", either because the measure has no missing information by design (e.g., parents' risk and trust attitudes in the original and replication study due to listwise deletion) or because it is not included in a study for other reasons (e.g., height in the extension study). * These variables were measured differently in *GSOEP* and *TwinLife* (see Table 1). In *TwinLife*, with the exception of denomination, which was measured in wave 2, all remaining variables were measured in wave 1.

Table A.2
Direct transmission coefficients of risk and trust attitudes from the replication and the extension study (illustrated in Fig. 2)

	Original		Replication		Extension	
	Coef.	s.e.	Coef.	s.e.	Coef.	s.e.
Risk attitudes						
Mother's risk attitudes	0.149	0.022	0.063	0.024	0.078	0.015
Father's risk attitudes	0.153	0.023	0.035	0.024	0.038	0.015
Trust attitudes						
Mother's trust attitudes	0.200	0.024	0.193	0.027	0.167	0.018
Father's trust attitudes	0.140	0.024	0.134	0.026	0.093	0.017

Data sources: GSOEP (original study); *TwinLife* (replication and extension study).

Notes: Original = Dohmen et al.'s (2012: 654) original study (based on Table 1, Model 3 for risk attitudes and Table 1, Model 7 for trust attitudes); Replication = replication of the original study with *TwinLife* data following the sample and variable selection of the original study as best as possible; Extension = replication study with additional features as specified above (i.e., adapted measurements, inclusion of single-parent households, multiple imputation). Case numbers: original (n = 3337), replication (n = 1960) and extension study (4,014).

Table A.3
Conditional transmission coefficients of risk attitudes from the original study, the replication study, and the extension study (illustrated in Fig. 4). *Data sources: GSOEP* (original study); *TwinLife* (replication and extension study).

	Original		**Replication**		**Extension**	
	Coef.	s.e.	Coef.	s.e.	Coef.	s.e.
Mother's attitudes						
Homogeneous vs.	0.138	0.038	0.254	0.127	0.135	0.089
Heterogeneous parents	0.111	0.030	0.053	0.030	0.052	0.020
Single mother vs.	0.366	0.102	0.059	0.073	0.107	0.031
Mother in two-parent household	0.285	0.025	0.125	0.049	0.068	0.017
None vs.	0.255	0.055	0.054	0.032	0.095	0.019
One or more siblings	0.138	0.020	0.052	0.041	0.042	0.025
Harmonious vs.	0.206	0.060	0.098	0.038	0.082	0.023
Disharmonious relationship	0.177	0.086	0.034	0.036	0.069	0.019
Daughters vs.	0.163	0.027	0.086	0.035	0.099	0.020
Sons	0.149	0.027	0.031	0.036	0.043	0.023
Father's attitudes						
Homogeneous vs.	0.179	0.039	-0.147	0.129	-0.005	0.090
Heterogeneous parents	0.093	0.032	0.053	0.029	0.030	0.021
Single mother vs. Mother in two-parent household						
None vs.	0.104	0.058	0.052	0.031	0.036	0.019
One or more siblings	0.148	0.021	0.042	0.041	0.038	0.026
Harmonious vs.	0.346	0.068	0.030	0.038	0.036	0.023
Disharmonious relationship	0.047	0.086	0.088	0.035	0.037	0.020
Daughters vs.	0.147	0.027	0.059	0.034	0.039	0.021
Sons	0.140	0.028	0.041	0.036	0.038	0.023

Notes: Original = Dohmen et al.'s (2012: 670) original study (based on Table 8, Panel A); Replication = replication of the original study with *TwinLife* data following the sample and variable selection of the original study as best as possible; Extension = replication study with additional features as specified above (i.e., adapted measurements, inclusion of single-parent households, multiple imputation). Total case numbers: original (n = 3337), replication (n = 1960) and extension study (4,014). Shaded cells denote statistically significant differences between tested conditions (z-test, Clogg et al., 1995).

Table A.4
Conditional transmission coefficients of trust attitudes from the original study, the replication study and the extension study (illustrated in Fig. 5). *Data sources: GSOEP* (original study); *TwinLife* (replication and extension study).

	Original		Replication		Extension	
	Coef.	s.e.	Coef.	s.e.	Coef.	s.e.
Mother's attitudes						
Homogeneous vs.	0.231	0.046	0.086	0.091	0.100	0.064
Heterogeneous parents	0.226	0.029	0.203	0.035	0.183	0.024
Single mother vs.	0.363	0.100	0.262	0.093	0.190	0.035
Mother in two-parent household	0.375	0.023	0.275	0.046	0.199	0.020
None vs.	0.274	0.057	0.141	0.038	0.133	0.023
One or more siblings	0.228	0.022	0.250	0.044	0.226	0.028
Harmonious vs.	0.211	0.062	0.130	0.041	0.163	0.026
Disharmonious relationship	0.156	0.069	0.269	0.041	0.168	0.023
Daughters vs.	0.262	0.029	0.193	0.040	0.180	0.024
Sons	0.208	0.028	0.182	0.041	0.155	0.027
Father's attitudes						
Homogeneous vs.	0.170	0.048	0.225	0.094	0.167	0.066
Heterogeneous parents	0.144	0.027	0.123	0.032	0.097	0.024
Single mother vs.						
Mother in two-parent household						
None vs.	0.147	0.059	0.106	0.034	0.085	0.021
One or more siblings	0.160	0.022	0.162	0.044	0.106	0.028
Harmonious vs.	0.327	0.064	0.120	0.039	0.087	0.026
Disharmonious relationship	0.171	0.062	0.111	0.039	0.092	0.023
Daughters vs.	0.142	0.028	0.136	0.037	0.101	0.023
Sons	0.173	0.029	0.106	0.041	0.079	0.027

Notes: Original = Dohmen et al.'s (2012: 670) original study (based on Table 8, Panel B); Replication = replication of the original study with *TwinLife* data following the sample and variable selection of the original study as best as possible; Extension = replication study with additional features as specified above (i.e., adapted measurements, inclusion of single-parent households, multiple imputation). Total case numbers: original (n = 3337), replication (n = 1960) and extension study (4,014). Shaded cells denote statistically significant differences between tested conditions (z-test; Clogg et al., 1995).

Table A5
Bivariate correlations among twins (n = 4012).

	MZ twins		DZ twins	
	correlation	95 %-CI	correlation	95 %-CI
Risk	0.34	[0.28; 0.39]	0.15	[0.08; 0.21]
Trust	0.45	[0.40; 0.50]	0.26	[0.20; 0.32]

Data source: TwinLife, extension data (m = 1).

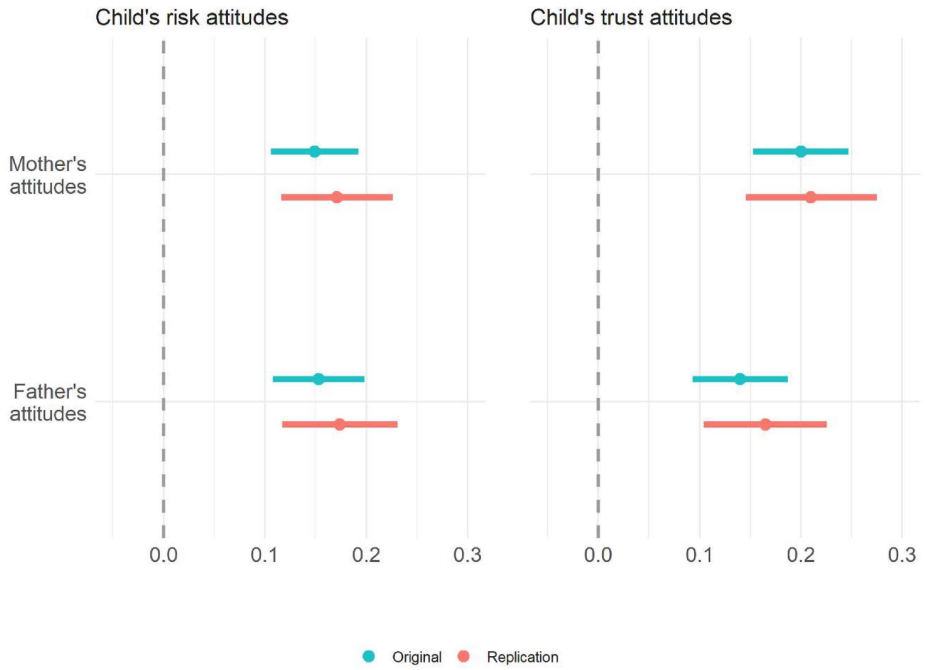

Fig. A.1. Direct transmission coefficients of risk and trust attitudes from the original study and from a *GSOEP*-subsample of individuals aged 17 to 25. *Data sources: GSOEP* (original study).
Notes: The dots show the regression coefficients, the horizontal lines the 95 percent confidence intervals. Original = Dohmen et al.'s (2012: 654) original study (based on Table 1, Model 3 for risk attitudes and Table 1, Model 7 for trust attitudes).

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