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Heritability of Bullying and Victimization in Children and Adolescents: Moderation by the KiVa Antibullying Program

Ada Johansson [a,b], Anne Huhtamäki[a], Miia Sainio[b], Anne Kaljonen[c], Michel Boivin[d], and Christina Salmivalli[b]

[a]Department of Psychology, Faculty of Arts, Psychology and Theology, Åbo Akademi University; [b]Department of Psychology and Speech-Language Pathology, University of Turku; [c]Department of Biostatistics, Faculty of Medicine, University of Turku; [d]School of Psychology, Université Laval

ABSTRACT

Objective: Bullying affects approximately a quarter of schoolchildren and is associated with numerous adverse outcomes. Although distinct risk factors for bullying and victimization have been identified, few studies have investigated the genetic and environmental underpinnings of bullying and victimization. The aims of this study were twofold: first, to examine the contributions of genetic and environmental factors to bullying and victimization, and second, to analyze whether the KiVa antibullying program moderated the magnitude of these contributions by comparing estimates derived from the KiVa versus control groups.

Method: The sample comprised students from schools that participated in the evaluation of the KiVa antibullying program in Finland during 2007–2009. Bullying and victimization were measured using peer nominations by classmates. The sample for the twin analyses comprised of 447 twins (107 monozygotic and 340 dizygotic twins) aged 7–15.

Results: Genetic contributions accounted for 62% and 77% of the variance in bullying and in victimization at pre-intervention, respectively. There was a post-intervention difference in the overall role of genetic and environmental contributions between the intervention and the control group for bullying and victimization, with non-shared environmental effects playing a lesser role (and genes a larger role) in the intervention than in the control group context.

Conclusions: This study replicates previous findings on the genetic underpinnings of both bullying and victimization, and indicates that a school-based antibullying program reduces the role of non-shared environmental factors in bullying and victimization. The results indicate that prevention and intervention efforts need to target both environmental and (heritable) individual level factors to maximize effectiveness.

Introduction

Bullying is a form of aggressive behavior where a child or a group of children repeatedly use their more powerful position to intentionally cause harm to a peer – often in the school context (Olweus, 1997). Bullying affects numerous children worldwide: a large-scale meta-analysis (Modecki, Minchin, Harbaugh, Guerra, & Runions, 2014) estimated that ~35% of children are involved in bullying either as victims or perpetrators, with some variation due to definitions or cultural context (Menesini & Salmivalli, 2017; Modecki et al., 2014). In addition, involvement in bullying is associated with a multitude of adverse outcomes for both victims and perpetrators of bullying (Bilodeau et al., 2018; Singham et al., 2017; Ttofi, Farrington, & Lösel, 2012).

Previous research has identified a host of school-, class-, family- and individual-level risk factors associated with bullying and victimization. Recent reviews (e.g. Álvarez-García, García, & Núñez, 2015; Nocentini, Fiorentini, Di Paola, & Menesini, 2019; Zych, Farrington, & Ttofi, 2019) reveal that bullying is associated with, for example, a negative school climate, negative self- and othe related cognitions, trouble resolving conflicts, low empathy, and externalizing behaviors. Family level risk factors include domestic violence, parental mental health problems, abuse/neglect and maladaptive parenting, and a negative family environment (Nocentini et al., 2019; Zych, Farrington, et al., 2019). Victimization, on the other hand, is associated with a negative school climate, low peer status and support, and low self-related (e.g. self-esteem, self-concept) and other-related (e.g. prosociality and social competence) personal competencies (Zych, Farrington, et al., 2019). Family risk factors for victimization include abuse/neglect, parental mental health, domestic violence (Nocentini et al., 2019), low

CONTACT Ada Johansson ada.johansson@abo.fi Department of Psychology, Åbo Akademi University, Tuomiokirkontori 3, Turku FIN 20500, Finland

parental support and a negative family environment (Zych, Farrington, et al., 2019).

Although classroom- and school-level factors contribute to bullying problems (Saarento, Garandeau, & Salmivalli, 2015), 85–95% of the variation in both bullying and victimization is due to inter-individual differences, rather than due to differences between classrooms or schools (Kärnä et al., 2011). Individual and family risk factors have been identified for both bullying and victimization, but little is known about the developmental processes underlying these associations. For instance, familial risk factors typically confound environmental and genetic sources of influence (i.e. besides influencing the home environment, parents also share part of their genetic makeup with their children), so that associations between risk factors and bullying or victimization can reflect underlying genetic, or environmental influences. Twin studies can help clarify these questions as they enable disentagling genetic from environmental source of variance for a given characteristic.

Few previous studies have investigated the heritability of bullying behavior. Ball et al. (2008) found the heritability of bullying perpetration to be 61%, and Veldkamp et al. (2019) ~70%, irrespective of type of bullying. In line with these estimates, Dunbar found in her Master's thesis a heritability of 55%. In addition, several twin studies of aggressive and antisocial behaviors, of which bullying is a specific form (Griffin & Gross, 2004), have found heritability estimates in the range of 40% to 80% (e.g., Brendgen et al., 2008; Burt, 2009; Polderman et al., 2015; Porsch et al., 2016).

As victimization is something done *to the child* rather than something done *by the child*, it is often perceived as being caused by environmental factors external to the child. However, factors assumed to be under environmental influence can also be under genetic influence (Jaffee & Price, 2007). Such genetic influence on environmental exposure is called gene-environment correlation (*r*GE), and can be observed, for example, when an individual evokes a reaction (e.g., bullying) from the environment partly due to his or her genetic disposition for an underlying individual characteristic or behavior (Plomin, DeFries, & Loehlin, 1977). The heritability of peer victimization has been investigated in such a context, sometimes with mixed results, with heritability estimates ranging from 0-77% (Ball et al., 2008; Boivin et al., 2013a; Bowes et al., 2013; Brendgen et al., 2011, 2008; Connolly & Beaver, 2016; Eastman et al., 2018; Shakoor et al., 2015; Silberg et al., 2016; Törn et al., 2015; Veldkamp et al., 2019). This variability across studies could be due to developmental differences having to do with the dynamic nature of peer relations/reputations and their assessments (see Boivin et al., 2013a). Heritability estimates might also vary as a function of the type of victimization. For instance, victimization of physical bullying was found to have the highest heritability when compared to, for example, verbal or social/relational bullying (Eastman et al., 2018; Veldkamp et al., 2019). Some evidence of measured genetic risk factors for victimization was recently found through a multi-polygenic score approach (Schoeler et al., 2019). Victimization was found to be associated with genetic risks relating to mental health problems, attention-deficit/hyperactivity disorder (ADHD), risk taking, body mass index (BMI), and intelligence (negative association).

Environmental factors may also moderate the role of genes; a process called gene-environment interaction (GxE) (Plomin et al., 1977). For example, genes are more likely to account for inter-individual differences in an environment free of risk factors than in a context where environmental risk factors differ substantially between individuals. Originally formulated for antisocial behavior as the "push" hypothesis (Raine, 2002), this form of GxE is also relevant for other phenotypes. According to this hypothesis, when psychosocial risk factors are prevalent in the environment, they may "push" a child toward antisocial behavior, and thus "mask" the role of genetic factors. However, when these psychosocial risk factors are less prevalent, biological factors may then play a larger role in accounting for individual differences in such behavior (Raine, 2002). In more general terms, this suggests that genetic factors are more likely to account for individual differences in the absence of environmental constraints (Ouellet-Morin et al., 2008). Accordingly, an antibullying intervention aimed at limiting the prevalence of bullying and victimization in the school context by reducing environmental risk factors, could moderate the contribution of genes to individual differences in bullying or victimization. The KiVa antibullying program is an evidence-based whole school intervention program based on the participant role approach, which relies on changing the behavior of bystanders in bullying situations to behavior that does not reinforce bullying, but instead makes it socially unacceptable (Salmivalli, Kärnä, & Poskiparta, 2011). Given that the KiVa antibullying program aims at changing the school climate (thus hypothetically reducing environmental variance), we hypothesized that genetic factors would account for a larger amount of post-intervention variance in bullying and victimization, in line with the push hypothesis (Raine, 2002). The randomized controlled design of the KiVa intervention study provides a unique opportunity to test this GxE hypothesis without the possible confound of *r*GE, which is often left uncontrolled in non-experimental studies (Keller, 2014).

Given the scarcity of twin analyses investigating the heritability of bullying, and the mixed results from twin

studies on victimization, the main aim of this study was to estimate genetic and environmental contributions on victimization and bullying in a population-based sample of twins aged 7 to 15 years. Based on previous results, a significant role of genes was expected for both bullying and victimization. In addition, we tested whether a school-based antibullying intervention (KiVa) moderated the magnitudes of environmental and genetic contributions to bullying and victimization. As the intervention was aimed at ameliorating the school climate, thus reducing environmental risk variance, we hypothesized that environmental factors would play a less important role, whereas genes would play a larger role in the intervention group compared to the control group.

Method

Sample

The twin sample was recruited from a sample consisting of students from 234 Finnish schools that took part in a one-year randomized controlled trial investigating the effects of the KiVa program in 2007–08 or 2008–09. Recruitment letters were sent to all schools providing basic education in mainland Finland. The volunteering schools were stratified by province and language (basic education is provided both in Finnish and Swedish) and half of the schools were randomized to participate in the KiVa intervention, while the other half served as controls in (more details on recruitement, participation and attrition rates have previously been published in Kärnä et al., 2011, 2013). Because the participating schools were located throughout the country and resembled other comprehensive schools in characteristics such as class size and proportion of immigrant students, they can be considered representative of Finnish schools at the time of data collection. Of the students, most were native Finnish (i.e. Caucasian), with <3% being immigrants. All participating students, as well as their parents or legal guardians, had provided active consent for participation, and the data collection was conducted in accordance with the 1964 Helsinki declaration of research with human participants. Consent was received from over 87% of the students enrolled in the participating schools.

Of the 24,820 students who had partaken in the project, 556 were identified as twins or triplets based on the same date of birth and biological parents according to the Finnish Population Register Center. The final twin sample consisted of 447 twins (50% girls) whose zygosity could be determined (intervention: monozygotic (MZ) n = 70, dizygotic (DZ) n = 185; control: n_{MZ} = 37, n_{DZ} = 155). The participants were in Grades 1– 9 (M_{grade} = 6.56, SD_{grade} = 2.24), and 7–15 years old at the beginning of the school year at the time.

Zygosity for same-sex pairs was determined based on self-reports on items concerning physical resemblance (Sarna, Kaprio, Sistonen, & Koskenvuo, 1978). Zygosity was also determined for a twin pair when only one twin in a pair responded to the zygosity questionnaire but both had phenotypic data available, if there was no contradiction within the responding twin's responses. Questionnaire based zygosity determination is widely used and shows good accuracy (e.g., Christiansen et al., 2003; Sarna et al., 1978). The zygosity questionnaire was sent to all identified same-sex twins (as opposite sex twins are always DZ). Responses were obtained from 309 (out of 400) same-sex twins, yielding a response rate of 77%.

Measurement of Bullying and Victimization

Students completed an online questionnaire about bullying and victimization before the start of the intervention and one year later, when the KiVa program had been implemented for ten months (see Kärnä et al., 2011). Bullying and victimization were measured using a version of the Participant Role Questionnaire (PRQ; Salmivalli & Voeten, 2004) in which students were asked to nominate an unlimited number of classmates who fit the item descriptions. A proportion score (0–1.0) was calculated for each student (indicating the percentage of classmates that nominated the child), and averaged across the three bullying and three victimization items, respectively.

Peer-reports for bullying were gathered using the following items: "*Starts bullying*", "*Makes the others join in the bullying*", and "*Always finds new ways of harassing the victim*". For victimization, the following items were used: "*He/she is being pushed around and hit*", "*He/she is called names and mocked*", and "*Nasty rumors are spread about him/her*". Students could reply "*No one*," if no classmate's behavior matched the item description. The Cronbach's alphas in the KiVa sample were .79 for victimization and .90 for bullying scales, respectively. The PRQ is a widely used measure and previous studies showing that it correlates with self-ratings of the same scales, and with teacher-reported aggression for bullies, indicates its validity (Salmivalli & Nieminen, 2002; Schäfer & Korn, 2004).

Statistical Analyses

Phenotypic Analyses

Descriptive statistics and twin intra-class correlations were analyzed using SPSS (version 24.0). The generalized estimating equations (GEE) method together with the robust

variance estimator was used to analyze whether school group status (intervention/control) was associated with post-intervention levels of bullying or victimization whilst including gender, grade and bullying or victimization at pre-intervention as covariates. Since the KiVa intervention is a whole school program, all students from the schools in which the twins attended were included in these analyses (n = 13 981). GEE was also used to analyze whether twin (n = 566) and non-twin individuals (n = 18 155) differed in bullying and victimization at pre-intervention with gender and grade level as covariates.

Twin Model Fitting Analyses

Twins can be used to disentangle the proportional contribution of genes and shared environmental, and non-shared environmental (residual variation) factors to an observed phenotype (P). This can be done by comparing the resemblance between MZ twins, who share 100% of their genes, with that between DZ twins, who share on average 50% of their segregating genes. Members in the same family can resemble each other due to genetic relatedness or shared environmental sources of influences. Environmental factors that make the twins more similar to each other (termed shared environmental factors; C) are assumed to affect MZ and DZ twins equally, and therefore, higher MZ than DZ twin correlations indicate that genes play a role in the trait in question.Variance in an observed P is seen as a sum of variance due to additive genetic factors (A; shared completely by MZ twins and on average 50% by DZ twins), non-additive genetic factors (D; shared 100% by MZ twins and 25% by DZ twins), shared environmental factors (C; 100% shared between twins in a pair), and non-shared environmental influences (E + measurement error). Using structural equation modeling (SEM), these variance components can be estimated, however, C and D cannot be estimated simultaneously using only twins and, therefore, researchers need to choose between an ACE and an ADE model. Inspection of twin correlations, as well as a comparison of the Akaike Information Criterion (AIC; Akaike, 1987), was used to select between these two models. For more information on the twin method, kindly see, for example, Posthuma et al. (2003).

SEM was conducted using univariate Cholesky Decomposition using OpenMx (Boker et al., 2018; Neale et al., 2016). Pre-intervention data were aggregated across the intervention and control groups whereas a multigroup approach was used post-intervention. Nested sub models were compared by calculating the difference in fit-function (−2 * Log-likelihood of data, −2LL) and the difference in degrees of freedom between competing models, which yields a significance-testable χ^2-value. Moderation of the variance components were tested by equating path estimates to be equal across intervention and control groups post-intervention and testing for significant decreases in −2LL. Residual scores were used for the twin analyses, in which the effect of covariates (grade, gender, group status) was removed for pre-intervention data by linear regression. There was an overall difference in the variance components between the intervention and the control group for bullying and victimization pre-intervention. Given the randomization into groups and that no intervention had yet taken place, this difference should reflect randomness. Therefore, the influence of bullying or victimization at pre-intervention were removed from the post-intervention data to take into account pre-intervention levels, in addition to the contributions of covariates. To avoid data loss due to listwise deletion, missing data was imputed using the EM procedure in SPSS. Imputations and residual scores were computed using the full sample for robustness (N = 24,820). Importantly, imputed data was used only for calculation of residual scores for the twin analyses for individuals with information on the dependent variable in question but potential missing data on an independent variable.

The majority of twins were in the same classroom (67%). In addition, the ratio between zygosity groups (MZ intervention, MZ control, DZ intervention, DZ control) did not differ between twin pairs being in the same vs. different classrooms, Fischer's exact test 5.18, p = .15. Neither were twins in the same classroom rated more alike than twins being in different classrooms (all p > .23). Therefore, the fact whether twins were in the same classroom or not, should not influence estimates of genetic or environmental factors.

Results

Phenotypic Analyses

Descriptive statistics are presented in Table 1. The post-intervention levels of bullying and victimization were significantly lower in intervention schools compared to control schools both for bullying, Wald χ^2 = 3.89, df = 1, p = .049, and victimization, Wald χ^2 = 6.94, df = 1, p = .008. No significant differences between twins and singletons were found regarding bullying, Wald χ^2 = 1.320, df = 1, p = .25, or victimization, Wald χ^2 = 0.342, df = 1, p = .56, as tested pre-intervention. Twin intraclass correlations (Table 2) suggest genetic contribution to both bullying and victimization.

Twin Model Fitting Analyses

The ACE and the ADE model fit the pre-intervention data equally well for both bullying and victimization (bullying AIC = − 1428.31; victimization AIC = − 1469.92), whereas

Table 1. Means (standard deviations) for bullying and victimization raw scores, shown for twins and all students separately.

	Bullying		Victimization	
	Twins	All students in schools of twins	Twins	All students in schools of twins
Pre-intervention				
Intervention	.05 (.08)	.06 (.11)	.07 (.07)	.06 (.09)
Control	.06 (.11)	.06 (.10)	.06 (.10)	.06 (.09)
Post-intervention				
Intervention	.04 (.07)	.04 (.08)	.04 (.06)	.05 (.07)
Control	05 (.08)	.05 (.09)	.06 (.08)	.06 (.08)

Bullying and victimization were measured using proportion scores based on peer nominations (range 0–1.0)

Table 2. Twin intra-class correlations [95% confidence intervals] for bullying and victimization.

	Pre-intervention	Post-intervention	
	All	Intervention	Control
Bullying			
MZ	.66 [.44, .81]	.52 [.24, .73]	.52 [.07, .79]
DZ	.37 [.20, .68]	.21 [.01, .40]	.07 [−.16, .29]
Victimization			
MZ	.66 [.44, .81]	.67 [.44, .82]	.22 [−.27, .61]
DZ	.42 [.26, .56]	.13 [−.17, .49]	.13 [−.08, .33]

MZ = monozygotic twins, DZ = dizygotic twins. Computed on residual scores where the effects of covariates (grade, gender, group status) and covariates plus pre-intervention level (for the post-intervention variables) were regressed out.

the *ADE* model fit the post-intervention data better for both phenotypes (bullying$_{ACE}$: AIC = − 2507.61; bullying$_{ADE}$: -AIC = − 2508.60; victimization$_{ACE}$: AIC = − 2577.22; victimization$_{ADE}$: AIC = − 2580.06). *ADE*-models were therefore chosen both for the pre- and post-intervention data.

Significant genetic contributions were found for pre-intervention bullying and victimization, comprising of both *A* and *D* components (Table 3). Significant genetic contributions were also revealed for post-intervention bullying and victimization after controlling for pre-intervention levels, but this time in the form of additive (*A*) components, except for victimization in the control group where both *A* and *D* components were significant. Magnitudes of the standardized variance components are presented in Table 4.

The test of invariance across conditions (intervention vs. control) revealed that the *A*, *D* and *E* components for both post-intervention bullying and victimization, respectively, could not be equalized simultaneously without significant deteriorations in model fit (Table 3), thus indicating that the magnitudes of the variance components differed between the intervention and control groups. We then tested this moderation separately for the genetic (*A* + *D*) and the non-shared environmental (*E*) components, and found that the *E* components differed across conditions (control vs. intervention), whereas the genetic components did not (Table 3).

Discussion

The present study aimed at examining the magnitude of genetic and environmental contributions to bullying and victimization, in a Finnish sample aged 7–15. In addition, the aim was to test whether the KiVa anti-bullying program would moderate these contributions. As expected, we found significant and substantial genetic contribution for both bullying and victimization in general, as well as a moderation through the antibullying intervention program of the ratio between genetic and non-shared environmental factors.

Broad sense heritability (H^2) for bullying was estimated at 62% (*A* = 23%, *D* = 39%) for pre-intervention, with non-shared environmental factors accounting for the rest of the variance. The dominance component has not been reported previously, however, twin analyses have limited statistical power to distinguish between additive and non-additive genetic effects, and estimates of the broad sense heritability are more stable (Eaves, 1972). In that sense, the broad sense heritability is very similar to the previous findings by Ball et al. (2008; 61% heritability for bullying), Veldkamp et al. (2019; ~70%), and Dunbar (2018; 55%), even though different informants were used (i.e. combined parents' and teachers' ratings in Ball et al., 2008, and teachers' ratings in Veldkamp et al., 2019). The present study is the first one to have estimated the heritability of bullying using peer nominations. The heritability estimates for bullying are in line with those found for antisocial behavior and aggression more generally (Brendgen et al., 2008; Burt, 2009; Polderman et al., 2015; Porsch et al., 2016).

Broad sense heritability for pre-intervention victimization scores was quite substantial at 77% (*A* = 50%, *D* = 27%), with non-shared environmental influences accounting for the rest of the variance. Again, this estimate is in the range of several previous studies (73% for Ball et al., 2008; 71–77% for Bowes et al., 2013; 70% for Connolly & Beaver, 2016; 67% for Törn et al., 2015; ~65% for Veldkamp et al., 2019). Other studies have reported lower heritability estimates: Brendgen et al. (2008) estimated the heritability of victimization to 0% in one study,

Table 3. Model fit statistics for the twin model fitting analyses.

Model	Tested against model	−2LL	df	AIC	Δ-2LL	Δdf	p
Pre-intervention[a]							
Bullying							
1. *ADE*		−742.31	343	−1428.31			
2. *AE*	1.	−733.88	344	−1421.88	8.43	1	.004
3. *E*	2.	−702.97	345	−1392.97	30.91	1	<.001
Victimization							
1. *ADE*		−783.92	343	−1469.92			
2. *AE*	1.	−777.46	344	−1465.46	6.46	1	.011
3. *E*	2.	−736.35	345	−1426.35	41.11	1	<.001
Post-intervention[b]							
Bullying							
1. $ADE_{\text{base model}}$		−1550.60	479	−2508.60			
Control:							
2. *AE*	1.	−1550.60	480	−2510.60	0	1	1.00
3. *E*	2.	−1544.28	481	−2506.29	6.31	1	.012
Intervention:							
4. *AE*	1.	−1550.11	480	−2510.11	0.49	1	.484
5. *E*	4.	−1534.44	481	−2496.44	15.66	1	<.001
Tests of moderation:							
6. $ADE_{\text{equalized between groups}}$	1.	−1538.58	482	−2502.58	12.02	3	.007
7. $AD_{\text{equalized between groups}}$	1.	−1550.32	481	−2512.32	0.28	2	.869
8. $E_{\text{equalized between groups}}$	1.	−1545.27	480	−2505.27	5.33	1	.021
Victimization							
1. $ADE_{\text{base model}}$		−1622.06	479	−2580.06			
Control:							
2. *AE*	1.	−1615.71	480	−2575.71	6.35	1	.012
3. *E*	2.	−1607.67	481	−2567.97	8.04	1	.005
Intervention:							
4. *AE*	1.	−1622.06	480	−2582.06	0.00	1	.986
5. *E*	4.	−1598.85	481	−2560.85	23.21	1	<.001
Tests of moderation:							
6. $ADE_{\text{equalized between groups}}$	1.	−1612.18	482	−2576.18	9.88	3	.020
7. $AD_{\text{equalized between groups}}$	1.	−1617.84	481	−2579.84	4.22	2	.121
8. $E_{\text{equalized between groups}}$	1.	−1616.15	480	−2576.15	5.91	1	.015

[a]Residual scores from which the effects of grade, gender and group status (intervention vs. control) were regressed out.
[b]Residual scores from which the effects of grade, gender, group status and pre-intervention level of bullying or victimization were removed. *A* = additive genetic effects, *D* = dominant genetic effects, *E* = non-shared environmental effects including error.

Table 4. Estimates of genetic and environmental effects on bullying and victimization residual scores.

Variable	Broad sense heritability (*A* + *D*)	*A [95% CI]*	*D [95% CI]*	*E [95% CI]*
Pre-intervention[a]				
Bullying	.62	.23*** [.13, .53]	.39** [.14, .59]	.38 [.25, .58]
Victimization	.77	.50*** [.25, .73]	.27* [.06, .44]	.23 [.15, .39]
Post-intervention[b]				
Bullying				
Intervention	.58	.48*** [.05, .74]	.10 [.00, .34]	.42 [.26, .70]
Control	.36	.36* [.00, .58]	.00 [.00, .31]	.64 [.42, .93]
Victimization				
Intervention	.72	.72*** [.38, .83]	.00 [00, .27]	.28 [.17, .47]
Control	.37	.00** [.00, .52]	.37* [.08, 52]	.63 [.30, 82]

*$p < .05$, **$p < .01$, ***$p < .001$.
[a]Residual scores from which the effects of grade, gender and group status were regressed out.
[b]Residual scores from which the effects of grade, gender, group status and pre-intervention levels of bullying or victimization were removed. Model fit statistics are presented in Table 3. The significance of *E* cannot be tested by dropping the path, since it also includes measurement error. *A* is tested after *D* has been fixed to 0. *A* = additive genetic effects, *D* = dominant genetic effects, and *E* = non-shared environmental effects, including measurement error.

and 26% in a later study (Brendgen et al., 2011), and Shakoor et al. (2015), Silberg et al. (2016), and Dunbar (2018) reported heritabilities of 35%, 45% and 48%, respectively. However, these variations could be accounted for by a variety of reasons. For instance, both Eastman et al. (2018) and Veldkamp et al. (2019) found that heritability estimates vary depending on the type of victimization; they found higher heritabilities for physical

(42% and 70%, respectively) versus social/relational (0% and 55%) and property-related victimization (0%, only in Eastman et al., 2018). In addition, the twin studies also seem to differ, for example, with regards to informants used and age of participants. The use of single informants may give rise to unstable or low estimates (due to larger measurement error), especially for self-ratings in younger children. When multiple informants were used in a latent model of peer victimization and rejection, a significant and substantial contribution of genes was seen for twins from Kindergarten to grade 4 (H^2 = 73–94%; Boivin et al., 2013a), which is in line with our results. Eastman et al. (2018) compared the genetic and environmental estimates derived from children (ages 9–14), versus adolescents (ages 15–20), and found differences in the magnitude and structure of genetic influences. The genetic contribution to victimization indicate that heritable characteristics in the child could evoke a negative reaction from peers and thus play a role in the likelihood of being bullied by others, a form of evocative *r*GE (Boivin et al., 2013a). Such characteristics could include reactive-impulsive aggression (Boivin et al., 2013b), but also depression, ADHD, risk taking, high BMI or low intelligence, as Schoeler et al. (2019) recently showed, through a polygenic risk score approach, that genetic risks for these characteristics were related to victimization. These modest associations need replications, as well as confirming evidence that they work through the mediating role of the putative child characteristics. Further evidence for the *r*GE hypothesis is also found in twin studies indicating partial overlap between the genes influencing victimization and those for social anxiety (Silberg et al., 2016), as well as depression/anxiety (Connolly & Beaver, 2016).

Significant genetic contributions to victimization and bullying were also found post-intervention, after pre-intervention levels were accounted for. This was true for both the intervention and the control group. Since pre-intervention levels were regressed out from the post-intervention levels, direct comparisons between heritability estimates between the time-points cannot be made. Crucial to the objective of the present study were the findings regarding the moderating role of the KiVa intervention. Moderation by the KiVa program would be indicated if the post-intervention estimates differed between the control and the intervention group after controlling for pre-intervention levels. This was true for both bullying and victimization. For both phenotypes, the general *ADE* pattern of estimates differed across groups (intervention vs control), essentially reflected by lower *E*-estimates in the intervention (bullying 42%, victimization 28%) compared to the control group (bullying 64%, victimization 63%), and thus leaving more room for genes to account for the remaining variance. The significantly smaller *E*-estimates in the intervention group could reflect a possible leveling out of environmental risk due to the KiVa intervention (e.g. through changing the behavior of bystanders and making bullying behavior less acceptable in the school setting), leaving a higher role to genetically influenced individual characteristics in that context. This pattern of findings is in line with the push hypothesis (Raine, 2002), which posits that genes will play a larger role in an environment freer of environmental risk factors. Evidence for such GxE findings have been found, for example, with respect to the moderating role of socioeconomic status on the genetic and environmental etiology of antisocial behavior (Tuvblad, Grann, & Lichtenstein, 2006) and the role of early adversity in physiological stress (Ouellet-Morin et al., 2008).

These results indicate the conditional nature of these environmental and genetic sources of individual differences, but they also point to the importance of providing contexts, through policies (e.g. early education) or intervention (i.e. KiVa), to create a more equitable social and learning environment for all children. When these interventions are successful in leveling out the environmental playing field, they may paradoxically identify individual factors, here genetic factors in the child, as more important for various outcomes. In doing so, they provide useful information in that they point to where the effort for change should be oriented. Specifically, even though the KiVa intervention is amongst the most effective antibullying interventions (Gaffney, Farrington, & Ttofi, 2019), it fails to stop all bullying. Our results indicate that genes play a significant role in accounting for post-intervention variance in bullying and victimization, and that the efficacy of the KiVa program might be enhanced by incorporating components targeting individual heritable characteristics. Previous research indicates that heritable individual characteristics that could evoke victimization from peers include mental health problems such as depression and anxiety (Connolly & Beaver, 2016; Schoeler et al., 2019; Silberg et al., 2016), ADHD, high BMI and low intelligence (Schoeler et al., 2019). Schoeler et al. (2019) suggested that one way to target such individual heritable characteristics with regards to victimization, could be to include components trying to reduce the stigma of mental health problems or other vulnerabilities such as high BMI, or to offer more support to children displaying internalizing or externalizing symptoms. Intervention components could either be universal (targeted at the entire school) such as in components

aiming to change the environment to be less discriminatory (e.g., toward people with mental health problems, neuropsychological difficulties, or high BMI), or individual (e.g. support and/or interventions for students at risk). In addition, one should also keep in mind that interventions not specifically aimed at reducing bullying victimization or perpetration, but rather aimed at reducing characteristics that increase the risk for victimization or perpetration could, in turn, also reduce bullying.

With respect to bullying perpetration, less is known about the specific underlying heritable characteristics, but such could include, for example, callous-unemotional traits and/or conduct problems (Viding, Simmonds, Petrides, & Frederickson, 2009; Zych, Ttofi, & Farrington, 2019). It is important to identify at-risk children early, and intervene not only in the school context but also by means of parenting programs (Waller, Hyde, Klump, & Burt, 2018). Further research is needed to identify specific heritable characteristics related to both the risk of victimization and bullying, especially victimization and bullying that persists after intervention efforts. However, as we know that standard interventions are not helpful in all cases (see also Kaufman, Kretschmer, Huitsing, & Veenstra, 2018), school personnel should always follow up after taking action to stop bullying, check whether their intervention was helpful, and to take further action when needed.

A strength of this study is that bullying and victimization were measured with proportion scores derived from peer nominations. In addition to there being multiple reporters, classmates can be considered to be more up to date on what is happening in the school setting (Boivin et al., 2013a; Stassen Berger, 2007), than for instance teachers or parents. Another clear strength of our study is the RCT-design, which effectively controls for potential confounds such as those that might arise from unaccounted *r*GE. The participating schools can be considered representative of Finnish schools at the time of data collection. In addition, twins did not differ from non-twin individuals on bullying or victimization, suggesting that the results are generalizable to non-twin individuals.

A limitation is the relatively small sample size, especially with regards to the post-intervention comparison between the intervention and the control group. The pre-intervention estimates are likely more robust, as these used data from all twins. We decided to do separate analyses for the pre- and post-intervention data for two reasons. First, even though a multi-group approach is warranted for the post-intervention data, it was preferable to analyze the pre-intervention data in a single group for increased statistical power. Second, an extension of the Cholesky decomposition to a multivariate case (i.e. analyzing both pre- and post-intervention) is problematic in multi-group approaches (Neale, Røysamb, & Jacobson, 2006). A multivariate approach would, however, allow a comparison of the variance components between the time-points, and therefore, replication efforts with larger sample sizes finding a solution to the multivariate approach would be welcome. A possible limitation of the twin method is whether the equal environments assumption holds. It has, however, been tested in a number of studies and appears to be valid (e.g. Derks, Dolan, & Boomsma, 2006; Kendler, Neale, Kessler, Heath, & Eaves, 1993).

Conclusion

In line with previous research, this study showed significant moderate to high heritability for both bullying and victimization. Furthermore, our results suggest that a change in the overall magnitude of genetic and environmental components underlying bullying and victimization can be induced through a school-based antibullying program. Taken together, our results indicate that an individual's genetic propensity for certain individual characteristics play a significant role in his/her likelihood for both bullying and being victimized, and that this role of genes can be amplified in explaining the remaining variance after a school-based antibullying intervention targeting changes in the school context. Thus, in order to most effectively combat bullying on multiple levels, it is crucial that both environmental and individual-level factors are taken into account and targeted in interventions and prevention efforts.

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ORCID

Ada Johansson http://orcid.org/0000-0003-1853-6981

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