

Genetic and environmental influences on victims, bullies and bully-victims in childhood

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Background: Three groups of children are involved in bullying: victims, bullies and bully-victims who are both bullies and victims of bullying. Understanding the origins of these groups is important since they have elevated emotional and behavioural problems, especially the bully-victims. No research has examined the genetic and environmental influences on these social roles. **Method:** Mother and teacher reports of victimisation and bullying were collected in a nationally representative cohort of 1,116 families with 10-year-old twins. Model-fitting was used to examine the relative influence of genetics and environments on the liability to be a victim, a bully or a bully-victim. **Results:** Twelve percent of children were severely bullied as victims, 13% were frequent bullies, and 2.5% were heavily involved as bully-victims. Genetic factors accounted for 73% of the variation in victimisation and 61% of the variation in bullying, with the remainder explained by environmental factors not shared between the twins. The covariation between victim and bully roles ($r = .25$), which characterises bully-victims, was accounted for by genetic factors only. Some genetic factors influenced both victimisation and bullying, although there were also genetic factors specific to each social role. **Conclusions:** Children's genetic endowments, as well as their surrounding environments, influence which children become victims, bullies and bully-victims. Future research identifying mediating characteristics that link the genetic and environmental influences to these social roles could provide targets for intervention. **Keywords:** Bullying, behavioural genetics, epidemiology, environmental influences, peer relationships, twins. **Abbreviations:** DZ: dizygotic, MZ: monozygotic.

Bullying is defined as intentional harm where there is a power differential between the bully and the victim (Rigby, 2002). Bullying is an important problem for society because of the distress and other difficulties that accompany and follow it. Studies across 25 countries have shown that between 9% and 54% of children are involved in bullying (Nansel, Craig, Overpeck, Saluja, & Ruan, 2004) and boys are more often involved than girls are (Nansel et al., 2001).

Bullies and victims of bullying are more likely than children uninvolved in bullying to suffer from a wide variety of problems, including low self-regard (Egan & Perry, 1998), depression and anxiety (Hawker & Boulton, 2000; Arseneault et al., 2006), and violent behaviours (Nansel, 2003). Bully-victims are a small group of children who engage in bullying both as victims and bullies. They experience the most severe problems: bully-victims are more depressed and anxious (Schwartz, 2000; Arseneault et al., 2006) and have higher rates of ADHD (Schwartz, 2000) compared to either children uninvolved in bullying, victims or bullies. They are more likely to be referred for psychiatric consultation and are more prone to school refusal than any other group involved in bullying (Kumpulainen et al., 1998).

Various studies have described characteristics associated with becoming a victim, bully or bully-victim (Olweus, 1993). Cross-sectional research has found that compared to families of normative children, those of bullies and victims are less functional (Rigby, 1994) and parents are more overcontrolling and less caring (Rigby, 2002). Longitudinal studies examining children who later became bullies found that their parents provided less cognitive stimulation, emotional support and allowed more TV exposure than other parents (Zimmerman, Glew, Christakis, & Katon, 2005). Boys who later became bully-victims had had harsh early home environments, witnessing and experiencing aggression, maternal hostility and restrictive discipline (Schwartz, Dodge, Pettit, & Bates, 1997).

However, methodological limitations have made it hazardous to draw firm conclusions about familial influences. The causal influences underlying these familial associations are unclear because genetic and environmental influences are confounded within families. For example, introverted parents could have introverted children as a result of genetic transmission, overcontrolling parenting, or both. Genetically informative studies can disentangle genetic and environmental influences. There have been no previous behavioural-genetic studies of victimisation or bullying, but several have examined antisocial behaviour which includes bullying

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behaviour. Meta-analyses of genetic and environmental influences on antisocial behaviour have reported that 40–50% of variation in antisocial behaviour is explained by genetic factors, 20% by environmental influences shared by twins and 30% by environmental influences unique to each twin (Rhee & Waldman, 2002; Moffitt, 2005). We expected similar estimates for bullying. However, there is no prior research to guide expectations of genetic influences on victimisation. Looking for evidence of genetic transmission does not imply that victimisation is a personality trait, but merely acknowledges that genetic factors can influence children's behaviour in such a way as to increase their likelihood of becoming victimised.

The present study aimed to examine the genetic and environmental influences on children's involvement in bullying as victims or bullies, and also on the co-occurrence between being a victim and a bully. First, we considered victimisation and bullying separately to examine the genetic and environmental influences on each role. Second, we considered victimisation and bullying simultaneously to examine the genetic and environmental influences on the covariation between the two. This method was used to examine influences on bully-victims, as distinct from 'pure' victims and 'pure' bullies.

Method

Participants

Participants were members of the Environmental Risk (E-Risk) Longitudinal Twin Study, which tracks the development of a birth cohort of 2,232 twins (1,116 pairs). This E-risk sample was drawn from a larger 1994–1995 birth register of twins born in England and Wales (Trouton, Spinath, & Plomin, 2002). The sample was constructed in 1999–2000, when 1,116 families with same-sex 5-year-old twins participated in home-visit assessments, forming the base cohort for the longitudinal E-risk study. Details of sample construction are reported elsewhere (Moffitt & the E-Risk Study Team, 2002). Follow-up assessments were conducted when the children were 7 and 10 years old. Follow-up home interview data were collected for 98% and 96% of the 1,116 families at ages 7 and 10 respectively. Teacher questionnaires (posted to the children's teachers, with parental permission) were obtained for 93% and 90% of participants respectively. For this study, we used measurements collected during the visits when the twins were age 10. Ethical approval was granted by the Maudsley Hospital Ethics Committee.

Measures

Victimisation. We asked mothers whether either twin had been bullied by another child since they were age 5, responding 'never' (0), 'yes' (1) or 'frequently' (2). The child's age at the time of victimisation was also reported by the mother, with the aid of a Life History Calendar, a

visual data collection tool for dating life events (Caspi et al., 1996). The present study focuses on incidents that took place at age 9 or 10. An inter-rater reliability study found that of 100 mothers who reported a child as being bullied, 70% of the children agreed in a separate self-report; of 100 children who self-reported being victimised, 60% of their mothers agreed independently.

We asked mothers to describe the victimisation experiences. Incidents reported by mothers included being called names, being teased, and being excluded from group, as well as physical abuse. We asked mothers whether the twins suffered physical harm (e.g., bruises, cuts) or psychological harm (e.g., school avoidance, fear) ('no' (0), 'yes' (1), or 'frequent' (2)). To create a severity of victimisation score index, we summed the three items. Data were positively skewed so we reduced this severity of victimisation index to a 3-category variable (0 'never victimised' 75.6%, 1–2 'moderately victimised' 12.7% and 3–6 'severely victimised' 11.7%). Complete data were available for 2,138 children (96% of the sample).

Bullying. We assessed bullying using the Child Behavior Checklist with mothers (Achenbach, 1991a) and teachers (Achenbach, 1991b). The items for mothers were 'cruel or nasty to other people', 'bullying or threatening people', and 'teases a lot' and for teachers they were 'cruelty, bullying, or meanness to others', 'teases a lot', and 'threatens people'. All informants rated each item as being 'not true' (0), 'somewhat or sometimes true' (1), or 'very or often true' (2). The reporting period was 6 months prior to the interview. Mothers' and teachers' reports of bullying were correlated (polychoric $r = .24$, $p < .01$). This modest correlation partly reflects the different contexts assessed by mothers and teachers. To include this context diversity, we summed mothers' and teachers' ratings to give a total bullying scale (0–12). The internal consistency reliabilities were .65 for mothers' ratings and .83 for teachers' ratings. Due to a positive skew in the data we reduced this scale to a 3 category variable (0 'never a bully' 48.5%, 1–2 'moderate bully' 38.2% and 3–12 'frequent bully' 13.3%). Complete data were available for 1,899 children, 85% of the sample, due to the combination of missingness from mother and teacher reports.

Analyses

We used STATA 9.1 (STATA, 2005) to examine the prevalence of victimisation and bullying. Because each study family contains two children, statistical analyses were corrected conservatively for the non-independence of the twin observations by using tests based on the sandwich or Huber/White variance estimator (Williams, 2000). We used genetic model-fitting to estimate the relative contribution of genetic and environmental influences to the variation in victimisation, variation in bullying, and the covariation between the two.

In twin methodology, the concordance of a particular trait within pairs of MZ and DZ twins is used to indicate the relative magnitude of genetic and environmental influences on the trait (Neale & Cardon, 1992). Because our data were ordinal and weighted, we used the Mplus programme, version 4.1 (Muthén & Muthén, 2006),

using the robust weighted least squares estimation option. A liability threshold model is assumed, which allows analysis of the dimensional liability to each phenotype. Consequently, all children are included in all analyses, rather than comparing one group of 'cases' to one group of 'controls'.

We first tested the fit of the full model to that of a baseline model of uncorrelated dependent variables. We used an adjusted χ^2 difference test (Muthén & Muthén, 2006) because the difference between two robust χ^2 goodness of fit statistics does not have a χ^2 distribution (Satorra, 2000). We also used the Comparative Fit Index because it is relatively unaffected by sample size (CFI $>.90$ indicates satisfactory fit), and the root mean square error of approximation (RMSEA) because it is less affected by sample size and it penalizes for lack of parsimony (RMSEA $<.05$ indicates good fit, $<.08$ indicates adequate fit) (Hu & Bentler, 1999). Next, we tested the full models against more parsimonious nested models.

In the standard univariate model, the phenotypic variation is decomposed into that explained by additive genetic (A), shared environmental (C) and nonshared environmental (E) factors. Shared environmental influences represent factors that have impacted both twins equally, while nonshared environmental influences represent factors that have impacted the twins differently. The relative magnitude of the model parameters (A, C and E) is inferred by comparing observed between-twin correlations to correlations predicted from a hypothesised model. Error of measurement is partitioned into the E parameter (Neale & Cardon, 1992). We first fitted these univariate models for victimisation and bullying data separately.

To examine the covariation of victimisation and bullying, we then fitted a bivariate Cholesky decomposition model. Bivariate models follow the same principles as univariate models, but decompose the covariance between the two phenotypes (victimisation and bullying) into bivariate A, C and E parameters. These parameters are estimated using the cross-trait cross-twin correlations (e.g., victimisation in twin 1 correlated with bullying in twin 2). We used methods based on Cholesky models with categorical data in Mplus (Prescott, 2004). We converted the results into a correlated factors solution for presentation. Thresholds were estimated separately for girls and boys. Our model estimates a correlation between the genetic parameter for victimisation and the genetic parameter for bullying, which is known as the genetic correlation (r_A), and represents the extent to which genetic factors that influence victimisation also influence bullying. The model also estimates correlations between the C and E parameters, r_C and r_E respectively (Figure 1a).

In the prevalence and univariate analyses, bully-victims count both as victims and as bullies, so there is no direct estimate of aetiological influences on 'pure' victims (who are victims only) or 'pure' bullies (who are bullies only). However, aetiological influences that do not correlate across the two phenotypes in the bivariate model influence one phenotype but not the other, indicating influences that contribute to 'pure' phenotypes.

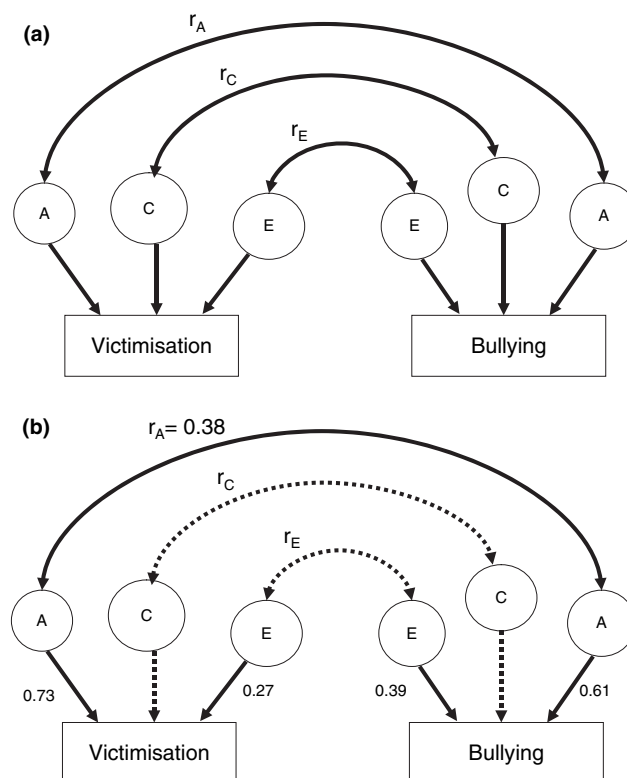


Figure 1 Correlated factors bivariate model for victimisation and bullying, full model (1a) and best-fitting model (solid lines indicate significant paths) (1b). r_A , r_C and r_E refer to the genetic, shared environmental and nonshared environmental correlations respectively. Other numbers refer to the percentage of univariate variance accounted for by each parameter

Results

Descriptive statistics

Many children were involved in bullying either as victims or as bullies (Table 1). Almost a quarter of children had been victimised between ages 9 and 10, but only 11.7% experienced severe victimisation. There were no significant sex or zygosity differences in the prevalence of victimisation. More than half of the children bullied others, but only 13.3% bullied others frequently. Boys were significantly more likely to bully others than were girls ($\chi^2 = 44.7$, $df = 1$, $p < .01$), but the prevalence did not differ by zygosity. Children who were both severe victims and frequent bullies accounted for 2.5% of the sample. This proportion did not significantly differ by zygosity (2.3% in MZ, 2.8% in DZ), but was considerably higher in boys (4.1%) than girls (1.1%) ($z = 3.0$, $p < .01$). These prevalences are within the range found for singletons (Nansel et al., 2004).

Heritability can be roughly estimated by calculating $2(r_{MZ} - r_{DZ})$, where r is the within-pair correlation. The polychoric between-twin correlations for victimisation and for bullying were higher for MZ than DZ twins (Table 2), suggesting genetic influences on the two phenotypes. These correlations also suggest

Table 1 Prevalence of victims and bullies at age 9 to 10 years

	Victims (<i>N</i> = 2,138)			Bullies (<i>N</i> = 1,899)		
	Never %	Moderate %	Severe %	Never %	Moderate %	Frequent %
Total sample	75.6	12.7	11.7	48.5	38.2	13.3
Boys	74.0	12.4	13.6	39.0	42.8	18.2
Girls	77.2	12.9	10.0	57.4	34.0	8.7
MZ	75.4	12.7	11.9	47.8	39.4	12.8
DZ	75.8	12.6	11.6	49.3	36.9	13.8

Percentages shown are corrected for sample weighting.

Table 2 Correlations between MZ and DZ twins for victimisation, bullying, and cross-trait

	Victimisation		Bullying		Cross-trait	
	<i>r</i>	95% CI	<i>r</i>	95% CI	<i>r</i>	95% CI
Total sample						
MZ	.72	.65-.79	.63	.55-.71	.26	.16-.37
DZ	.38	.25-.52	.33	.22-.44	.17	.06-.29
Within-person	–	–	–	–	.25	.18-.33
Boys						
MZ	.77	.68-.86	.58	.47-.70	.26	.12-.40
DZ	.41	.21-.61	.23	.06-.40	.23	.09-.38
Within-person	–	–	–	–	.26	.16-.36
Girls						
MZ	.65	.52-.77	.62	.50-.74	.21	.05-.36
DZ	.36	.18-.54	.41	.26-.55	.13	-.04-.29
Within-person	–	–	–	–	.23	.13-.34

CI = Confidence interval.

nonshared environmental influences because MZ correlations were less than 1.00 and indicate little influence from the shared environment because DZ correlations were only slightly higher than half MZ correlations.

Being a victim of bullying and being a bully were moderately correlated within individuals (polychoric $r = .25$, Table 2). Cross-twin correlations between victimisation and bullying were higher for MZ twins ($r = .26$) than DZ twins ($r = .17$), suggesting genetic influences on the covariation between the two phenotypes.

Behavioural genetic analyses on bullying and victimisation separately

We fitted univariate genetic models to victimisation and bullying data. The model for victimisation gave a satisfactory fit to the data ($\Delta\chi^2 = 26.05$, $df = 14$, $p = .03$, $CFI = .97$, $RMSEA = .06$). The significant $\Delta\chi^2$ value is possibly due to the large sample size. Model parameters could be equated across sex without significant loss of fit ($\Delta\chi^2 = 2.02$, $df = 3$, $p = .57$). The parameter estimates in the full model (with parameters equated across sexes) included a small non-significant influence from the shared environment (Table 3). Thus, the best-fitting model included genetic (73%) and nonshared environmental (27%) parameters only ($\Delta\chi^2 = .08$, $df = 1$, $p = .78$).

The univariate model for bullying was a good fit to the data ($\Delta\chi^2 = 13.20$, $df = 14$, $p = .51$, $CFI = 1.00$, $RMSEA = .00$). Model parameters could be equated across sex without significant loss of fit ($\Delta\chi^2 = 1.95$, $df = 3$, $p = .58$). The best-fitting model included genetic (61%) and nonshared environmental (39%) parameters only ($\Delta\chi^2 = .27$, $df = 1$, $p = .60$) (Table 3).

Behavioural genetic analyses on co-occurring bullying and victimisation

We fitted a bivariate model to victimisation and bullying data simultaneously, which fitted well ($\Delta\chi^2 = 48.15$, $df = 38$, $p = .13$, $CFI = .99$, $RMSEA = .03$). Although cross-trait correlations seem to suggest sex differences, model parameters could be equated across sex without significant loss of fit ($\Delta\chi^2 = 4.81$, $df = 9$, $p = .87$), which may be due to low power to detect sex differences. This model indicated that influences responsible for the within-child phenotypic correlation between victimisation and bullying (i.e., the existence of bully-victims) were mainly genetic (65% explained by *A* in Model 1, Table 4).

We dropped the univariate and bivariate *C* parameters from the model because the univariate models had indicated non-significant *C* influences on either phenotype separately. The fit was not significantly worse (Model 2, Table 4). We used this reduced *AE* bivariate model to test the influence of *A* and *E* factors on the overlap between victimisation

Table 3 Genetic and environmental parameter estimates and fit indices for the sex-equated univariate model of victimisation and the univariate model of bullying

Social role	Variance components			Fit indices (compared to full model)		
	A Additive genetic	C Shared environment	E Nonshared environment	$\Delta\chi^2$ (adjusted)*	Δ df	<i>p</i>
Victimisation						
Full model (ACE)	.69	.04	.27	–	–	–
AE model	.73	–	.27	.08	1	.78
CE model	–	.65	.35	19.72	1	<.001
Bullying						
Full model (ACE)	.54	.06	.40	–	–	–
AE model	.61	–	.39	.27	1	.60
CE model	–	.50	.50	14.25	1	<.001

*The difference between two robust χ^2 goodness-of-fit statistics does not have a χ^2 distribution, so an adjustment was used (Muthén & Muthén 2006).

Best-fitting models shown in bold.

Table 4 Genetic and environmental parameter estimates and fit indices for the sex-equated bivariate model of victimisation and bullying

Model	Univariate components	Bivariate components	Phenotypic correlation due to influences (%)			Bivariate correlation			Fit indices		
			A	C	E	r_A	r_C	r_E	$\Delta\chi^2$ (adjusted)*	Δ df	<i>p</i>
1.	ACE	ACE	65	32	2	.28	1	.02	–	–	–
2.	AE	AE	103	–	–3	.38	–	–.02	1.21 ^a	3	.75
3.	AE	A	100	–	–	.38	–	–	.07^b	1	.80
4.	AE	E	–	–	100	–	–	.76	28.17 ^b	1	<.001

*The difference between two robust χ^2 goodness-of-fit statistics does not have a χ^2 distribution, so an adjustment was used (Muthén & Muthén, 2006).

Best-fitting model shown in bold.

^a = compared to Model 1.

^b = compared to Model 2.

and bullying. The only significant bivariate parameter was the genetic (*A*) parameter ($\Delta\chi^2 = 28.17$, $df = 1$, $p < .001$). Therefore, the best-fitting model indicated *A* and *E* influences on the univariate components of victimisation and bullying, and only *A* influences on the covariation (Model 3, Table 4; Figure 1b).

Genetic factors therefore accounted for all of the phenotypic (within-child) overlap of victimisation and bullying. However, the genetic correlation (r_A) in the best-fitting model was .38 indicating that approximately 14% (.38²) of the genes that influence bullying also influence victimisation. The bivariate model also implies substantial genetic and non-shared environmental influences on 'pure' victimisation and 'pure' bullying; the univariate *A* and *E* parameters were significant but r_A and r_E were both less than 1.0. This indicates that there are some *A* and *E* factors that influence victimisation but not bullying, and others that influence bullying but not victimisation.

Discussion

This is the first study to examine the relative contributions of genetic and environmental factors to

the development of victims, bullies and bully-victims during childhood. We showed that children's tendencies to be victimised and to bully others are mainly genetically, but also partly environmentally, influenced. Children's tendency to be both a victim and a bully was influenced almost solely by genetic factors.

Victimisation

We found that genetic influences accounted for over two-thirds of individual differences in children's victimisation, with the remainder due to nonshared environmental influences. These genetic influences on childhood environmental exposures are a novel finding. Previous studies have found that children's susceptibility to maltreatment by adults is largely unrelated to genetic influences (Jaffee, Caspi, Moffitt, & Taylor, 2004; Dinwiddie et al., 2000). This comparison suggests that children's heritable characteristics are meaningful in evoking abuse from similar-aged peers whereas abuse from adults is related to characteristics of the aggressor or the situation.

Because victimisation is an exposure rather than a direct behaviour, genetic influences could be a reflection of heritable characteristics that influence children's vulnerability to victimisation. Introverted

personality, which is elevated in victimised children (Mynard & Joseph, 1997), could mediate genetic influences on victimisation. Previous research has indicated that personality moderates genetic influences on other types of environmental exposure such as negative life events (Saudino, Pedersen, Lichtenstein, McClearn, & Plomin, 1997). Other characteristics of victims that could be mediators of genetic influences could include social cognitive deficits (Camodeca & Goossens, 2005) and emotional regulation and emotional displays (Mahady Wilton, Craig, & Pepler, 2000).

Environmental factors influence children's victimisation via experiences that are unique to each twin (nonshared environmental factors) rather than experiences common to both twins. This might include experiences at home or at school, but ones that impact each child differently, including their unique friendship groups, random factors and bad luck (such as being in the wrong place at the wrong time) rather than factors common to both children such as household income. Family factors can also uniquely impact children's victimisation via the effect of nonshared environmental processes such as differential maternal treatment (Caspi et al., 2004).

Bullying

Bullying was strongly influenced by genetic factors and, to a lesser extent, by nonshared environmental factors. The genetic and environmental parameters found for bullying are in line with those typically reported for antisocial behaviours and aggressive conduct problems, except that we found no significant influence from common environmental factors. As for victimisation, there are likely to be characteristics that mediate the genetic and environmental influences on bullying such as verbal ability and self-regulation.

It has been suggested that socialisation via parenting (Patterson, Reid, & Dishion, 1992) and peers (Harris, 1998) contributes to shared and nonshared environmental influences on aggressive behaviour. Genetically influenced characteristics including aspects of personality such as impulsivity and sensation seeking may mediate the genetic influences on antisocial behaviour (Jacobson, Prescott, & Kendler, 2002). It is likely that some of these genetic and nonshared environmental factors influence bullying similarly. These factors may operate via mediating characteristics that are observed in bullies, including biases in social cognitions (Sutton, Smith, & Sweetenham, 1999), and low emotionality and poor emotional regulation (Sutton et al., 1999; Calkins, Gill, Johnson, & Smith, 1999; Dodge, Lochman, Hanish, Bates, & Pettit, 1997).

Bullying can be seen as a particular subset of aggressive behaviour (intentionally harming a weaker child, repeatedly over time) and may depend

on a specific set of social circumstances and abilities compared to antisocial behaviour. This may explain why we did not find significant effect from environmental influences that are shared by both twins in a pair.

Covariation between victimisation and bullying

The modest correlation between victimisation and bullying indicates that few children are bully-victims while more are 'pure' victims or 'pure' bullies. This corroborates previous research that has found similarly modest positive correlations between victimisation and aggression (Hodges & Perry, 1999; Crick & Bigbee, 1998). The correlation between victimisation and bullying was just as high in girls as in boys, despite significantly higher prevalences of bullies and bully-victims among boys. This indicates that one's chances of being a bully given that one is a victim (or vice versa) are just as high for girls as for boys.

The covariation between victimisation and bullying was explained solely by genetic factors, but the size of the correlation between the genes involved in victimisation and the genes involved in bullying was modest. This indicates that although there are some genetic factors that influence both traits ('common' genetic factors), there are more that influence only one trait ('specific' genetic factors). These 'specific' genetic influences are involved in the development of 'pure' victim or 'pure' bully roles.

The influence of the 'common' genetic factors on the covariation between victimisation and bullying may occur via two processes. Firstly, common mediating influences could make children more likely to be both bullied and to bully others, via heritable characteristics. One of the most likely candidates is emotional dysregulation, which is highly heritable (Kozak, Strelau, & Miles, 2005; Goldsmith, Lemery, Buss, & Campos, 1999), characterises both victims and bullies, and is most extreme in bully-victims (Schwartz, Proctor, & Chien, 2001). Secondly, the 'common' genetic factors may operate through phenotypic causality where genetic factors influence victimisation and the experience of being a victim makes a child more likely to become a bully (or vice versa). For example, a child who is bullied by someone older and stronger may seek to recoup self-esteem by bullying someone younger and weaker.

Lack of shared environmental influences

The absence of significant shared environmental influences on individual differences in victimisation, bullying and the covariation between both is unlikely due to low power because the sample size was large. In addition, the shared environmental terms were not merely nonsignificant, but only explained

around 5% of the variance in the univariate models. This suggests that previously reported effects of parenting on bullying roles do not reflect environmental influences. Correlations between parental characteristics (including parental styles and inter-parental aggression) and victimisation in offspring (Rigby, 1994; Rigby, 2002; Schwartz et al., 1997) may reflect genetic rather than environmental influences. For example, parents' anxiety may be associated with overcontrolling parenting, and risk of victimisation in offspring may be caused by genetically inherited anxiety traits rather than parenting style. This is a 'passive' correlation between genes and environments (Plomin, Defries, & Loehlin, 1977) since the parents' genes are correlated with the type of environment they provide to their children. The same sort of process could explain the correlations between family-level exposures with bully and bully-victim roles.

Limitations

The experience of victimisation may differ between twins and singletons. Having a co-twin may be similar to having a best friend and friendship quality has been shown to be protective against victimisation (Bollmer, Milich, Harris, & Maras, 2005). Twins may also be perceived to be unusual and may be more (or less) likely to be victimised than singletons overall. Comparison of our prevalence rates to those found in a sample of singletons from 25 different countries (Nansel et al., 2004) suggest no notable differences. Another potential problem could arise if the victimisation experiences reported by mothers were within twin pairs – if one twin was bullying his or her co-twin. If so, the cross-twin cross-trait correlations would be inflated for reasons other than shared genetic or environmental risks in the general population. However, mothers' descriptions of bullying incidents implicated bullies outside the family in virtually all cases.

This is the first study of the genetic and environmental influences on victimisation by peers during childhood. Our findings need to be replicated with similar populations using different measurement tools and interviewing different informants. Children's self-reports may index different severity levels and victims' subjective perceptions, which could be important factors in the development of psychopathology. This study examined school-age children in whom social roles are probably well established. Our measures are probably reasonable indicators of how they will adapt in secondary schools. Genetically informative studies on children of different ages would be valuable in understanding developmental trends.

The large *E* parameter found in our univariate models is likely to include some measurement error rather than just nonshared environmental influences. Measurement error probably accounts for

only a small proportion of the *E* parameter for bullying because the internal consistency reliability is satisfactory. However, although our victimisation score provided a detailed account of victimisation incidents, and was validated against self-reports, it came from one reporter only. Using multiple reporters would enhance accuracy, especially as children grow older and become more independent from their parents.

Implications for research and intervention

This study has indicated classes of aetiological factors responsible for individual differences in bullying involvement. Research should investigate heritable characteristics that may lead to victimisation and bullying. Such research could identify mediating characteristics that, if reduced in target children, could lead to reductions in both victimisation and bullying. Genetically informative and longitudinal samples will be useful to improve our understanding of emotional and cognitive mediating factors and possible phenotypic causality. Knowledge of mediating factors could also indicate whether psychopathology associated with involvement in bullying is caused by these social roles or is a consequence of the genetically influenced characteristics that make children vulnerable to the social roles in the first place.

The large genetic influences found do not imply that bullying involvement is immutable but instead point to children's characteristics to be used as targets for environmental intervention. Interventions should focus on victims and not just bullies because there are factors other than 'bad luck' that can evoke victimisation.

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